Continuous Rapid Atrial Pacing to Control Recurrent or Sustained Supraventricular Tachycardias Following Open Heart Surgery

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SUMMARY A technique is described to control recurrent or sustained supraventricular tachycardia associated with rapid ventricular rates following open heart surgery. The technique utilizes a pair of temporarily implanted atrial epicardial wire electrodes to pace the heart. In one group of patients with recurrent atrial flutter and 2:1 A-V conduction, continuous rapid atrial pacing at 450 beats/min produced and sustained atrial fibrillation. The ventricular response rate immediately slowed when compared to that during atrial flutter, and if further slowing was required, it was easily accomplished by the administration of digitalis. Another group of patients with different arrhythmias (recurrent paroxysmal atrial tachycardia, sustained ectopic atrial tachycardia, or sinus rhythm with premature atrial beats which precipitated runs of atrial fibrillation) was treated with continuous rapid atrial pacing to produce 2:1 A-V block. In all instances, the continuous rapid atrial pacing suppressed the supraventricular tachycardia and maintained the ventricular response rate in a therapeutically desirable range. It was demonstrated that the technique is safe, effective, and reliable.

SUPRAVENTRICULAR TACHYARRHYTHMIAS with associated rapid ventricular rates occur frequently in the period immediately following open heart surgery. Most of these arrhythmias are amenable to treatment with standard modes of therapy. However, in a small percentage of patients, these supraventricular tachyarrhythmias are resistant to the standard therapy. In this report, we describe results obtained utilizing continuous rapid atrial pacing to control the ventricular rate in 24 patients with recurrent or sustained supraventricular tachycardias associated with rapid ventricular response rates. In each patient, using temporarily implanted epicardial wire electrodes, the atria were paced for periods up to 72 hours at rates which increased atrioventricular (A-V) block and thereby slowed the ventricular rates.

Methods

Twenty-four patients were studied during recurrent or sustained supraventricular tachycardias which occurred following open heart surgery. Continuous rapid atrial pacing was utilized in all patients to slow the ventricular rate. In Group I patients, atrial pacing was used to produce and sustain atrial fibrillation and in Group II patients to produce and sustain 2:1 A-V block. Pertinent clinical data for the patients in each group are contained in tables I (Group I) and 2 (Group II). For each patient, the presenting arrhythmia was documented by recording both a bipolar atrial electrogram and a standard body surface electrocardiogram. At our hospital a pair of Teflon-coated wire electrodes are routinely implanted 0.5–1.0 cm apart on the atrial epicardium at the time of open heart surgery and are used in the postoperative period for both diagnosis and treatment.¹ They are removed one day prior to discharge. In the present study, these electrodes were utilized both to record bipolar atrial electrograms and to pace the atria. The electrodes were isolated from ground and from recording devices at all times. All electrograms and electrocardiograms were recorded with standard techniques.² ³ In selected cases, data were recorded on magnetic tape (Honeywell Tape System Model 5600) for later playback and analysis. The bipolar rapid atrial pacing was performed either with a Medtronic 1349A or 1379 battery-powered pacemaker.

Group I. Continuous Rapid Atrial Pacing to Precipitate and Sustain Atrial Fibrillation

The 13 patients in this group all had atrial flutter with 2:1 A-V block (table 1). For each patient, the atrial flutter initially was converted with rapid atrial pacing either to a sinus rhythm or to atrial fibrillation.⁴ However, for each of these patients, the atrial flutter with rapid ventricular response rate reoccurred, and despite one or more additional conversions with rapid atrial pacing, continued to recur. For 12 of these 13 patients, atrial pacing at a rate of 450 beats/min and for the other patient at 390 beats/min uniformly produced atrial fibrillation with an irregular ventricular response. This atrial fibrillation then was maintained by pacing the atria continuously at the same rapid rate. In a group of patients not part of this study who were being treated for a single episode of atrial flutter, we have observed that atrial fibrillation may not be precipitated by atrial pacing at rates up to 410 beats/min, whereas pacing at 450 beats/min always will precipitate atrial fibrillation. It was for this reason that we selected the rate of 450 beats/min to produce and sustain atrial fibrillation.

Group II. Continuous Rapid Atrial Pacing to Precipitate and Sustain 2:1 A-V Block

There were 11 patients in this group (table 2). Four had paroxysmal atrial tachycardia which always could be interrupted with standard rapid atrial pacing techniques,⁶ but which always recurred. Six patients had an ectopic atrial tachycardia which could not be interrupted with rapid atrial
pacing at rates up to 100 beats/min faster than the intrinsic atrial rate. One patient had sinus rhythm with frequent premature atrial beats which often precipitated recurrent bouts of atrial fibrillation. For the latter patient, although the episodes of atrial fibrillation were not sustained, they were symptomatically troublesome to the patient and were associated with clinically unacceptable rapid ventricular rates. Atrial pacing at rates up to 120 beats/min failed to suppress the bouts of atrial fibrillation. In eight patients in group II, standard pharmacological therapy to suppress the arrhythmia also was attempted, but was unsuccessful. This was an additional reason for atrial pacing at rates sufficiently rapid to achieve and maintain 2:1 A-V block.

**Results**

**Group I**

In this group of 13 patients, continuous atrial pacing was initiated at a rate sufficiently rapid to produce and sustain atrial fibrillation. Table 3 summarizes and table 1 contains the individual data for these patients, and figure 1 illustrates a representative example (patient #8). The preoperative rhythm was sinus in ten patients and atrial fibrillation in three, but the presenting rhythm postoperatively was atrial flutter in all 13. During the atrial flutter, each patient had 2:1 A-V block, with the ventricular rates varying between 140 and 165 beats/min. Atrial pacing at a rate of 450 beats/min (except in patient #6 in whom the pacing rate was 390 beats/min) promptly caused the ventricular rate to fall to levels of 100–140 beats/min. With the precipitation of atrial fibrillation, the ventricular rate became 120 beats/min or less in all but patients #3 and #7, in whom the ventricular rates were 130 and 140 beats/min, respectively. The administration of digitalis to the latter two patients readily produced further slowing of the ventricular rate, as it regularly did in each of those other patients in whom further slowing was desirable.

The duration of continuous rapid atrial pacing in group I patients was 5–40 hours, the average duration being 24 hours. Two patients (#11 and #13) each had two episodes of recurrent atrial flutter separated by three and four days, respectively, which required two different periods of continuous rapid atrial pacing. For all patients, the duration of continuous rapid atrial pacing was dictated by the re-

**Table 2. Pertinent Clinical Data on Patients in Group II**

<table>
<thead>
<tr>
<th>Pt/Age (yrs)/sex</th>
<th>Preop rhythm</th>
<th>Operative procedure</th>
<th>Spontaneous postop rhythm</th>
<th>Paced postop rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Dx</td>
<td>At rate*</td>
</tr>
<tr>
<td>14/26/F</td>
<td>Sinus</td>
<td>AoVR, MVR, TVR</td>
<td>PAT</td>
<td>175</td>
</tr>
<tr>
<td>15/54/F</td>
<td>At Fib</td>
<td>AoVR, MVR</td>
<td>At Tach</td>
<td>140</td>
</tr>
<tr>
<td>16/54/M</td>
<td>At Fib</td>
<td>AoCSVG - 3V</td>
<td>Sinus w/PACs &amp; recurrent A Fib</td>
<td>85</td>
</tr>
<tr>
<td>17/52/F</td>
<td>Sinus</td>
<td>AoCSVG - 1V</td>
<td>PAT</td>
<td>180</td>
</tr>
<tr>
<td>18/26/M</td>
<td>Sinus</td>
<td>AoVR</td>
<td>At Tach</td>
<td>140</td>
</tr>
<tr>
<td>19/55/M</td>
<td>Sinus</td>
<td>AoVR</td>
<td>At Tach</td>
<td>150</td>
</tr>
<tr>
<td>20/58/F</td>
<td>At Fib</td>
<td>MVR</td>
<td>At Tach, TV plastic, A-V block, 200</td>
<td>140-150</td>
</tr>
<tr>
<td>21/52/F</td>
<td>At Tach</td>
<td>MVR</td>
<td>PAT</td>
<td>180</td>
</tr>
<tr>
<td>22/40/F</td>
<td>At Fib</td>
<td>MVR</td>
<td>At Tach</td>
<td>200</td>
</tr>
<tr>
<td>23/14/F</td>
<td>Sinus</td>
<td>VSD repair</td>
<td>At Tach</td>
<td>180</td>
</tr>
<tr>
<td>24/60/M</td>
<td>Sinus</td>
<td>AoCSVG - 2V</td>
<td>PAT</td>
<td>180</td>
</tr>
</tbody>
</table>

*Beats per minute.

Abbreviations: AoVR = aortic valve replacement; MVR = mitral valve replacement; TVR = tricuspid valve replacement; VSD = ventricular septal defect; PAT = paroxysmal atrial tachycardia; At Fib = atrial fibrillation; At Tach = ectopic atrial tachycardia; PAC = premature atrial beats; Dx = diagnosis; TV plastic = tricuspid valvuloplasty.
currence of the atrial flutter upon an attempt at cessation of pacing, as well as by the clinical circumstances (e.g., we rarely stopped the pacing during the nighttime hours). Following final termination of rapid atrial pacing, spontaneous atrial fibrillation was present in all 13 patients. The atrial fibrillation persisted in the three patients with preoperative atrial fibrillation but spontaneously reverted to sinus rhythm in a short time (within 5 seconds to 3 hours) in the ten patients with preoperative sinus rhythm. Thus, in every patient the rhythm present following successful termination of atrial pacing was the same as the preoperative rhythm.

**Group II**

In this group of patients, continuous atrial pacing was initiated at rates sufficiently rapid to achieve 2:1 A-V block. Table 4 summarizes and table 2 contains the individual data for the 11 patients in this group, and figures 2-4 illustrate a representative example (patient #14).

Four patients had recurrent paroxysmal atrial tachycardia with ventricular rates of 175-180 beats/min. Continuous atrial pacing at rates of 200-240 beats/min produced 2:1 A-V block with ventricular rates of 100-120 beats/min, and thus satisfactorily controlled the rapid ventricular rates. These data confirm a previous observation in one patient (7) in whom the atria were paced at a rate of 200 beats/min for two hours and 15 minutes to produce 2:1 A-V block in order to suppress recurrent paroxysmal atrial tachycardia following open heart surgery.

Five other patients had sustained ectopic atrial tachycardia with 1:1 A-V conduction at rates between 130-200 beats/min. In the four adult patients, continuous atrial pacing at rates from 180 to 230 beats/min produced 2:1 A-V block with ventricular rates between 90 and 115 beats/min. For the fifth patient (#23), a 16-month-old child, the atria were paced at a rate of 340 beats/min because clinically the optimal ventricular rate appeared to be 170 beats/min.

One patient (#21) had sustained atrial tachycardia at 200 beats/min with variable A-V block which resulted in a ventricular response rate of 140-150 beats/min. For this patient the optimal ventricular rate was 90-115 beats/min. During atrial pacing at an atrial rate of 200 beats/min, the ventricular rhythm was 90-115 beats/min.

**Table 3. Summary of Data for Group I (13 patients)**

<table>
<thead>
<tr>
<th>Preop Rhythm</th>
<th>Postop Ventricular Rate</th>
<th>Duration of Rapid Atrial Pacing</th>
<th>Post Rapid Atrial Pacing Rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus (10)</td>
<td>during atrial flutter</td>
<td>5-40 hours; avg 24</td>
<td>Sinus (10)</td>
</tr>
<tr>
<td>At Fib (3)</td>
<td>during rapid atrial pacing</td>
<td></td>
<td>At Fib (3)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ventricular Rate*</th>
<th>Spontaneous</th>
<th>Paced E:1 response</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAT (4)</td>
<td>175-180</td>
<td>100-120; avg 113</td>
</tr>
<tr>
<td>At Tach</td>
<td>130-150</td>
<td>90-115; avg 99</td>
</tr>
<tr>
<td>Adults (4)</td>
<td>200</td>
<td>170</td>
</tr>
<tr>
<td>Children (1)</td>
<td>140-150</td>
<td>110</td>
</tr>
<tr>
<td>Sinus with PACs</td>
<td>85</td>
<td>110</td>
</tr>
</tbody>
</table>

*In beats/min.

Numbers in parentheses are number of patients in each group.

Numbers in parentheses are number of patients in each group.

Abbreviations: At Fib = atrial fibrillation; At Tach = atrial tachycardia; PAT = paroxysmal atrial tachycardia; PAC = premature atrial beat; avg = average.

**Table 4. Summary of Data for Group II (11 patients)**

<table>
<thead>
<tr>
<th>Preop Rhythm</th>
<th>Ventricular Rate*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus (6)</td>
<td>110-130; avg 113</td>
</tr>
<tr>
<td>At Fib (4)</td>
<td>100-120; avg 113</td>
</tr>
<tr>
<td>At Tach with A-V Block (1)</td>
<td>110-130; avg 113</td>
</tr>
</tbody>
</table>

**Figure 1.** Top panel: ECG lead recorded simultaneously with a bipolar atrial electrogram documents that the rhythm is atrial flutter with 2:1 A-V block. The atrial rate is 320 beats/min. Bottom panel: ECG lead recorded simultaneously with a stimulus artifact during atrial pacing at a rate of 450 beats/min. The rapid atrial pacing has produced atrial fibrillation and reduced the ventricular rate from 160 to 120 beats/min. Time lines are at one second intervals. S = stimulus artifact.
patient, continuous atrial pacing at 220 beats/min resulted in 2:1 A-V block with a ventricular response rate of 110 beats/min. One patient (#16) had sinus rhythm with frequent premature atrial beats which precipitated recurrent bouts of atrial fibrillation with clinically unacceptable rapid ventricular response rates. Continuous atrial pacing at a rate of 220 beats/min completely suppressed the premature atrial beats and established a ventricular response of 110 beats/min.

The duration of continuous rapid atrial pacing to achieve 2:1 A-V block in group II patients ranged from 15 minutes to 72 hours with an average duration of 21 hours. The patient (#20) whose atria were paced for only 15 min illustrates an important point. The low cardiac output present in this very ill patient improved somewhat when the rapid atrial pacing at 200 beats/min slowed her ventricular rate from 130 to 100 beats/min. However, because the cardiac output was still clinically too low, continuous intravenous administration of epinephrine (0.01 mcg/kg/min) was initiated. Because the medication enhanced A-V conduction, the ventricular response rate to the atrial pacing at 200 beats/min increased. Since neither the spontaneous rhythm nor atrial pacing at rates up to 240 beats/min was associated with sufficiently slow ventricular rates, atrial fibrillation was precipitated deliberately with rapid atrial pacing, and digitalis was administered to control the ventricular rate. For this patient, the atrial fibrillation then sustained itself, making continuous rapid atrial pacing unnecessary. In one other patient (#23) rapid atrial pacing also was terminated, in that instance because complete A-V block developed. For the other nine patients in group II, the rhythm which appeared or developed after termination of continuous rapid atrial pacing was the same as the preoperative rhythm.

Figures 2–4 illustrate a representative example of the rhythm which prompted the initiation of sustained atrial pacing to achieve 2:1 A-V conduction. In figure 2, ECG leads II and III have been recorded simultaneously with a bipolar atrial electrogram during a bout of paroxysmal atrial tachycardia in which the ventricular rate was 175 beats/min. Using standard atrial pacing techniques, the rhythm was converted to a sinus rhythm; however, premature atrial beats, which could not be suppressed even during atrial pacing at rates up to 120 beats/min (fig. 3), reprecipitated the paroxysmal atrial tachycardia. Continuous atrial pacing at rates up to 120 beats/min failed to terminate the paroxysmal atrial tachycardia once it was reestablished (fig. 3). When atrial pacing was initiated at a rate of 200 beats/min (fig. 4), 1:1 atrial capture with 2:1 A-V conduction and a ventricular rate of 100 beats/min was produced. For this patient (#14) continuous atrial pacing was maintained for 20 hours, after which the cessation of pacing was followed by sustained normal sinus rhythm.

Limitations of the Technique

Although continuous rapid atrial pacing to treat recurrent or sustained supraventricular tachycardia was uniformly successful whenever tried, the following examples in some of our patients not included either in group I or II illustrate some limitations of the technique. We have observed one instance in which rapid atrial pacing techniques used to treat an episode of atrial flutter following open heart surgery precipitated atrial fibrillation during which the ventricular response rate was greater than that during the atrial flutter (fig. 5). Preoperative and postoperative ECGs recorded from this patient during sinus rhythm demonstrated a P-R inter-

**Figure 2.** ECG leads recorded simultaneously with a bipolar atrial electrogram during an episode of paroxysmal atrial tachycardia in Patient #14. The atrial and ventricular rates are 175 beats/min. Timelines are at one second intervals.

**Figure 3.** ECG leads recorded simultaneously with a stimulus artifact during atrial pacing at 120 beats/min: same patient as in figure 2. A premature atrial beat (fifth beat) precipitated paroxysmal atrial tachycardia which was not interrupted by atrial pacing at 120 beats/min. Timelines are at one second intervals. S = stimulus artifact.
val of only 0.12 seconds. Thus, we suggest that rapid atrial pacing to produce and sustain atrial fibrillation may be hazardous in patients who have a short P-R interval during sinus rhythm. Furthermore, as it is well known that the ventricular response rate to atrial fibrillation in undigizedal patients may be quite rapid (e.g., 150 to 180 beats/min), one should anticipate that the immediate ventricular response rate following deliberate precipitation of atrial fibrillation in undigizedal patients may be quite rapid.

In patients with marked sinus tachycardia, rapid atrial pacing to achieve 2:1 A-V block and thereby slow the ventricular rate may not be possible. In two patients with sinus tachycardia of 150 beats/min, we have observed that brief bursts of rapid atrial pacing (used during the initial diagnostic assessment of the rhythm) produced 1:1 A-V conduction at rates up to 220 beats/min.

Discussion

Deliberate precipitation of atrial fibrillation with a brief period of rapid atrial pacing in order to permit relatively easy control of the ventricular rate in patients with supraventricular tachyarrhythmias has been amply described by others, as has utilization of a brief period of rapid atrial pacing to interrupt selected supraventricular tachycardias. In the present study we extended these concepts to treat recurrent or sustained supraventricular tachycardias associated with rapid ventricular response rates in patients following open heart surgery. Our results demonstrate that continuous rapid atrial pacing to produce and then sustain either atrial fibrillation or 2:1 A-V block is a safe and effective therapy. Continuous rapid atrial pacing not only permits the ventricular response rate to be slowed and controlled, but also provides time for any required pharmacologic treatment. However, the data also demonstrate that there are at least three circumstances in which the technique either should not be used or used with great caution: 1) in patients with a previously documented short P-R interval during sinus rhythm; 2) in patients with sinus tachycardia; and 3) in patients during administration of drugs which enhance A-V conduction.

The selection of continuous rapid atrial pacing either to produce and sustain atrial fibrillation or to produce and sustain 2:1 A-V block depends upon the presenting arrhythmia.

For example, it is not useful to treat recurrent atrial flutter with continuous atrial pacing to achieve 2:1 A-V block. While in the present study we precipitated and sustained atrial fibrillation only in patients with recurrent atrial flutter, this technique could have been applied to the patients in group II, i.e., to patients with recurrent or sustained supraventricular tachycardia other than atrial flutter. In fact, with a brief burst of rapid atrial pacing, we deliberately did precipitate atrial fibrillation (which then sustained itself without further need of pacing) in one patient (20) in Group

![Figure 4](https://example.com/figure4.png)

**Figure 4.** ECG leads recorded simultaneously with a stimulus artifact from the same patient as in figures 2 and 3 during atrial pacing at a rate of 200 beats/min which produced 2:1 A-V block. Timelines are at one second intervals. **S** = stimulus artifact.

![Figure 5](https://example.com/figure5.png)

**Figure 5.** Top panel) Bipolar atrial electrogram recorded simultaneously with ECG lead during spontaneous atrial flutter with 2:1 A-V block. The atrial rate is 320 beats/min and the ventricular rate is 160 beats/min. Bottom panel) Bipolar atrial electrogram recorded simultaneously with ECG lead during atrial fibrillation precipitated by rapid atrial pacing. The ventricular rate of 190 beats/min during the atrial fibrillation is more rapid than the ventricular rate during the atrial flutter.
II. However, whenever possible, it is preferable to initiate continuous atrial pacing to produce and sustain 2:1 A-V block because a desirable ventricular rate generally will be achieved promptly without the need to induce further slowing of the rate with digitalis, the ventricular rate can be manipulated up or down quickly and easily as desired simply by changing the atrial pacing rate, and the potential need to perform DC cardioversion of pacing-induced atrial fibrillation is not present.

Other Possible Uses of Continuous Rapid Atrial Pacing

Although the present report describes the temporary use of continuous rapid atrial pacing by way of fixed electrodes, the same principles can be employed using catheter electrodes inserted pervenously into the right atrium. In fact, using a bipolar catheter electrode so placed, we recently have successfully treated two patients with continuous rapid atrial pacing, the first for 72 hours in order to suppress recurrent paroxysmal atrial tachycardia, and the second for 36 hours in order to suppress sustained ectopic atrial tachycardia. In each patient, by pacing the atria continuously at 200 beats/min, we provided a satisfactory ventricular rate by producing and sustaining 2:1 A-V conduction. If one elects to initiate continuous rapid atrial pacing utilizing a catheter electrode, however, the secure and safe placement of the catheter within the atrium is essential in order that inadvertent rapid ventricular pacing does not occur.

Intermittent use of rapid atrial pacing in selected patients via a permanently implanted electrode and pacemaker system has been reported to suppress recurrent supraventricular tachycardia. Continuous use of rapid atrial pacing with such a system is feasible. However, the technological limitations of the presently available atrial electrodes and the present nonavailability of an appropriate pacemaker power system make such application impractical at this time. Furthermore, although there is no apparent reason to expect that prolonged rapid pacing should be associated with untoward effects, the technique has not been used for periods of more than 72 hours and possible harmful effects must yet be excluded.

Use of Temporarily Implanted Epicardial Atrial Wire Electrodes

Seven of the 24 patients in this study had atrial fibrillation prior to surgery. It previously has been suggested that the presence of atrial fibrillation preoperatively is a reason for not placing temporary atrial wire electrodes. However, as demonstrated in this study, the preoperative rhythm is not a useful index of the need for the implantation of atrial wire electrodes for diagnostic and therapeutic use in the postoperative period. Therefore, we routinely implant a pair of wire electrodes in all patients at the time of open heart surgery. The ease of implantation and the absence of any mortality or significant degree of morbidity associated with their use, coupled with their demonstrable diagnostic and therapeutic value, provide sufficient reasons for their continued routine use.1–5, 15–19

References

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