Conversion of Supraventricular Tachycardias 
with Atrial Stimulation

Evidence for Re-entry Mechanisms

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SUMMARY
Atrial stimulation at rates less than the atrial rate was effective in terminating junctional tachycardia and atrial flutter in two patients. Analysis of the stimulus response in the patient with junctional tachycardia defined an atrial excitable and refractory period and a critical interval, the first 12 milliseconds of the excitable period, during which atrial stimulation terminated the tachycardia. The termination of atrial flutter appeared to occur simultaneously with capture of the atrial rate by the slower atrial stimulus. Atrial fibrillation did not occur. These observations suggest that a variety of tachycardias are supported by re-entry mechanisms and that the response to atrial stimulation may be useful in recognizing and treating such arrhythmias.

Additional Indexing Words:
Arrhythmias Atrial flutter Atrial pacing Junctional tachycardia

Synchronized direct current precordial shock is frequently used to terminate supraventricular tachycardias. However, Massumi and associates and Durrer and co-workers have terminated supraventricular tachycardias associated with the Wolff-Parkinson-White syndrome using a single atrial stimulus. They postulated that the mechanism of cardioversion was interruption of a re-entry pathway. Haft and associates have converted three patients with atrial flutter, using rapid (360 to 400 per minute) atrial stimulation.

We have studied two patients without the Wolff-Parkinson-White syndrome, one with a junctional tachycardia and one with atrial flutter, where sinus rhythm was established by stimulating the atrium at a rate lower than the atrial rate. These observations suggest that a single appropriately positioned atrial stimulus can be effective in terminating a variety of supraventricular tachycardias. Additionally, cardioversion in such a manner may prove useful in determining the underlying mechanism of the arrhythmia.

Report of Cases

Patient 1
H.H., a 60-year-old white man, experienced recurrent bouts of tachycardia for 14 years. These were more frequent during the preceding year, and refractory to digitalis, quinidine, procainamide, and propranolol. Multiple DC cardioversion had been necessary. Physical examination was within normal limits. A recent electrocardiogram during remission was within normal limits, with a P-R interval of 0.18 and no evidence of pre-excitation.

On October 19, 1967, during a bout of junctional tachycardia at a rate of 169 per minute (fig. 1), a bipolar pacing catheter was positioned in the mid right atrium, and stimulation at a
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Figure 1
A-V junctional tachycardia exhibited by case 1. All recordings for case 1 are lead II with 1 second photographic time lines; 50 mm/sec paper speed.

Figure 2

Figure 3
Case 1. Conversion of junctional tachycardia to a paced atrial rhythm. Capture occurs with stimulus 5.

Figure 4
Case 1. This is a continuous tracing, and is a continuation of figure 3. (A, upper left). Atrial capture is present, but there is delay in A-V conduction with eventual complete heart block. (B, upper right). Two junctional beats are present, the third QRS complex possibly represents an atrial capture beat followed by two ectopic atrial beats. (C, lower left). Pulse generator turned off after twelfth stimulus. T-wave distortion suggests coupled atrial beat. (D, lower right). Recurrence of junctional tachycardia follows a slow low atrial or junctional rhythm. Note T-wave distortion in second complex, suggesting a coupled atrial beat.

A rate of 108 per minute was initiated with a Medtronic Model 5800 pulse generator using a two to five milliampere output. On two occasions the arrhythmia was converted to a paced atrial rhythm (figs. 2 and 3). Upon turning off the pulse generator an unstable sinus rhythm was seen on both occasions. Figure 4 demonstrates varying degrees of A-V block, escape beats, and recurrence of the junctional tachycardia.

The record, consisting of lead II of the electrocardiogram written 50 mm per second with 1 second photographic time lines, was analyzed by measuring the pacemaker artifact to R-wave stroke (p-R) to the nearest 0.3 mm, then converting these to time intervals adjusted appropriately for paper drive speed. More critical measurements were taken from photographs of the record enlarged five times. Correlation of p-R intervals with the atrial response revealed the atrial refractory and excitable periods (fig. 5).

Eighty-nine pacing artifacts at a frequency of 108 per minute were available for analysis against a background of a junctional tachycardia of 169
Case 1. The large schematic electrocardiographic cycle illustrates three periods within the cycle which yield different atrial responses to atrial pacing. R-R cycle length = 366 msec. The remaining times shown are referred to as upstroke of R wave as time zero. p-R = pacemaker stimulus to R wave upstroke. Sixty-three pacemaker stimuli occurring 300 to 72 msec before the onset of R (speckled zone) fall in the atrial refractory period and yield no response, as illustrated in panel A. Twenty-six stimuli occur from 72 msec before to 66 msec after the R wave (clear zone and dotted zone, respectively). These stimuli produce excitation of the atrium, as evidenced by distortion or loss of the usual retrograde P wave (first pacemaker stimulus, panel B and panel C). The earliest two of these 26 stimuli, falling 72 to 60 msec before the R (clear zone) terminate the arrhythmia (panel B). The remaining 24 cause a variable amount of prematurity of the subsequent cycle length (resetting) but do not terminate the arrhythmia. P.M. = pacemaker stimulus.

beats per minute. Sixty-three discharges occurred 300 to 72 msec before the R wave and registered a refractory atrial response (fig. 5, panel A). The remaining 26 discharges occurred from 72 msec before to 66 msec after the R wave, producing atrial excitation with the exception of one at 42 msec (fig. 5, panels B and C).

Two discharges yielding atrial capture 68 and 62 msec before the R wave were followed in the subsequent discharge by a paced atrial rhythm, indicating termination of the arrhythmia (fig. 5, panel B). The uniqueness of their response was matched by the uniqueness of the timing when further analyzed. These were the two earliest from the group of 26 beats that generated atrial interference.

Patient 2

R. C. was a 73-year-old construction worker who was transferred from a local hospital for further evaluation of chest pain. Serial electrocardiograms and serum enzymes had been within normal limits. Medications had included warfarin and digitoxin. He had recently been digitalized and experienced nausea and vomiting for the previous 3 days.

The pertinent physical findings were frequent premature beats, pulse 80, blood pressure 160/90, clear lungs, and a grade II/VI systolic ejection murmur at the left sternal border. The initial electrocardiogram showed sinus rhythm, with frequent premature atrial beats, and nonspecific ST and T-wave changes. Shortly after admission the pulse increased to 150, and a repeat electrocardiogram revealed atrial flutter at a rate of 300 with 2-to-1 A-V block. Over the next 24 hours he received lidocaine for frequent premature beats that probably represented supraventricular beats with aberrant conduction. He also received a beta adrenergic blocking agent, MJ 1999, which slowed the ventricular response but not the atrial rate.

Since the patient was suspected of having had a recent myocardial infarct and because of recent symptoms of digitalis intoxication it was elected to attempt restoration of sinus rhythm by atrial pacing rather than by conventional DC countershock. A hydrogen electrode was passed via an antecubital vein into the right atrium. Panel A in figure 6 is a rhythm strip of V₁ and panel B is a hydrogen electrode tracing from the right atrium. Unipolar pacing was possible by attaching the hydrogen electrode to a Grass stimulator. Pacemaker (PM) stimuli were delivered at 3 volts through an isolation unit. Stimuli at a rate of 120 for 1 minute failed to interrupt the tachycardia. The pacing rate was then increased to 180. Panel D demonstrates the point of cardioversion. The P-wave morphology following the PM stimulus indicated by the arrow in panel D is changed. At that point the PM stimulus appears to capture the atrial rate producing slight shortening, 0.19 to 0.17 seconds, and then termination of the atrial flutter. Each PM stimulus is then followed by a paced P wave. The PM-P and P-R intervals vary initially, however as seen at the end of panel D and in panel E there is a period of constant 2-to-1 A-V block prior to discontinuing the pacing. Cardioversion thus occurred while pacing at a rate less than the atrial rate. Atrial fibrillation did not occur.

The post-cardioversion tracings again showed frequent premature atrial beats. Quinidine sulfate, 1.2 g daily, abolished the premature beats. There was no recurrence of atrial flutter.

*Generously supplied by Mead Johnson Laboratories, Evansville, Indiana.
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Figure 6

Case 2. Cardioversion of atrial flutter with right atrial pacing. A is a surface recording of lead V₁, and B is a right atrial cavity recording obtained with a hydrogen electrode. C and D are continuous recordings of the precardioversion and cardioversion period. PM, panel D, refers to the large pacemaker artifact produced by unipolar stimulation with the hydrogen electrode, stimulation rate 180 per minute. The diagram, panel D, illustrates the pacemaker artifact, P- and R-wave relationship prior to the point of cardioversion (arrow) and following the cardioversion. Panel E represents right atrial pacing 2-to-1 A-V block followed by a sinus mechanism when the pacing was stopped. Paper speed was 50 mm/second in all tracings.

Discussion

In an experimental animal, Moe and associates\textsuperscript{5} terminated a paroxysmal A-V nodal tachycardia by placing two premature atrial beats at a critical time in the cardiac cycle. The arrhythmia was induced by atrial stimulation and was felt to be supported by a reciprocal passage over a dual A-V pathway. They postulated that a single premature stimulus, sufficiently close to the A-V node or re-entry site, would be adequate to interrupt the pathway.

In humans, recent investigators\textsuperscript{1,2} have utilized atrial pacing to terminate arrhythmias in patients with the Wolff-Parkinson-White syndrome. They proposed a re-entry mechanism as the basis for these arrhythmias, and felt that the termination by atrial pacing supported this concept. Massumi and associates\textsuperscript{1} with a single atrial stimulus produced
two atrial premature beats that led to conversion. Durrer and co-workers\textsuperscript{2} were able to effect cardioversion with one premature beat.

Patient 1 differed from the above cases in demonstrating no evidence of the Wolff-Parkinson-White syndrome. However, several features suggest that a re-entry mechanism was the basis of this arrhythmia. T-wave distortion seen in beat one (fig. 4C) and beat two (fig. 4D) suggests atrial premature beats with a fixed coupling interval. Cycle length shortening was seen following premature atrial excitation (fig. 7) on numerous occasions. Both of these phenomena are well explained by a re-entry process.\textsuperscript{2, 6–8} The presence of A-V conduction delay shown in patient 1 should be conducive for re-entry involving the A-V node by allowing more time for a retrograde pathway to recover excitability.\textsuperscript{8, 9}

Figure 4D shows an instance of recurrence of the tachycardia. An atrial premature beat, apparent as T-wave distortion in beat 2 of figure 4D, initiated the tachycardia. The longer cycle length between the second and third complexes relative to subsequent beats may be a function of prematurity of the atrial beat and incomplete recovery of nodal excitability due to length of the preceding cycle. This set-

![Figure 7](image)

Case 1. Proposed mechanism of arrhythmia and its termination by atrial pacing, taken from record shown in figures 4 and 5B. \( A = \text{atrium}; \ A-V = \text{A-V node}; \ V = \text{ventricle}. \) Pacemaker stimuli designated 1 through 6. Re-entry path is arbitrarily shown confined to the A-V node; normal QRS duration suggests that the ventricle was not involved in the re-entry path, but involvement of the atrium is indeterminate. Stimuli 1 and 3 fall during the atrial refractory period. Stimulus 2 causes premature atrial excitation and is sustained by excitable orthograde and retrograde paths to reset but not terminate the rhythm. Stimulus 4 occurs earlier than stimulus 2, traverses the atrium, and is dissipated within the A-V node on a part of the path that has not recovered excitability, thus terminating the arrhythmia. Stimuli 5 and 6 produce an atrial-paced rhythm.

The critical timing necessary for atrial stimulation to abolish this rhythm is in keeping with termination of a re-entry process. Stimuli falling before this time in the cycle would meet a refractory atrium. After this time, stimuli would cause premature (atrial) excitation. Only when the atrial stimulus entered the node early enough to encounter a refractory orthograde path would the process end. Figure 7 depicts the proposed mechanism of cardioversion.

Interruption of a rapidly firing pacemaker by a properly timed atrial stimulus cannot be ruled out. There is no literature bearing on the response of drug-induced tachycardias, as may be produced by aconitine, to pacing.\textsuperscript{10} Initial studies in our laboratory indicate aconitine tachycardias are not abolished or reset by external pacing. The temporary suppression of a physiological pacemaker by pacing it at a faster rate is a well-recognized phenomenon (overdrive suppression).\textsuperscript{11–18} This phenomenon was not operative in our cases, as the pacing rate was slower than the atrial rate.

Atrial flutter has been produced in the mammalian heart by mechanical, chemical, and electrical stimulation.\textsuperscript{10, 14, 15} Although the basic mechanism remains unknown, the two major theories that have been proposed are rapid firing of an ectopic pacemaker and circus movement or re-entry.\textsuperscript{16, 17} Atrial flutter generally is easily terminated with DC countershock. Drug therapy has been less effective. Haft and associates\textsuperscript{5} recently reported the use of atrial pacing to terminate atrial flutter in three patients, using an atrial pacing rate of 400 per minute. Conversion to sinus rhythm occurred only after varying periods of atrial fibrillation. They postulated that cardioversion may have resulted from overdrive suppression in two of the patients.

Our second patient is a typical example of atrial flutter with an atrial rate of 300 per minute. Cardioversion occurred while stimulating the atrium at a rate of 180 per minute. Cardioversion also appeared to be effected
by a single critically positioned stimulus. Atrial fibrillation did not occur.\textsuperscript{6,8} Cardioversion thus appeared to occur in a manner similar to our first patient and to those reported by Massumi, Durrer, Moe and their associates.\textsuperscript{1,2,5} It would seem reasonable to postulate that a similar mechanism, that of re-entry, was present. A rapid-firing pacemaker would not be expected to be suppressed by pacing at a slower rate.

The feasibility of this method in widespread use for reversion of supraventricular tachycardias is yet to be tested. The simplicity of the method, requiring no more than a pacing catheter and pulse generator, is attractive. Furthermore, there is no requirement for general anesthesia, and the problem of post-countershock arrhythmias\textsuperscript{18,19} may be reduced or eliminated. In addition, this method of cardioversion would lend itself readily in the treatment of arrhythmias during cardiac catheterization. There are certain patients with recurrent tachycardias refractory to conventional forms of therapy. If pacing were proved effective in terminating these arrhythmias, consideration might be given to an implantable atrial pacemaker triggered by a rapid rate or an external source. Finally, this technique may prove to be of diagnostic importance in separating the arrhythmias due to ectopic pacemakers from those due to re-entry. Theoretically, the latter might be abolished by a slower pacing stimulus, the former should not.

References
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