Termination of Paroxysmal Junctional Tachycardia by Right Ventricular Stimulation

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SUMMARY

This report documents the use of right ventricular stimulation in the termination of a rapid junctional tachycardia. The tachycardia developed in the background setting of heart block and bradycardia. Right ventricular stimulation was effective at 94/min, a rate considerably slower than that of the tachycardia. Analysis of pacemaker stimuli during the tachycardia defined ventricular refractory and excitable periods. Within the ventricular excitable period there was a critical time interval during which stimulation produced premature ventricular depolarization and abrupt termination of the tachycardia. These findings support the hypothesis that junctional tachycardias in the absence of the Wolf-Parkinson-White syndrome may be sustained by reentry mechanisms. Moreover, right ventricular stimulation may be a valuable therapeutic adjunct in the treatment of such tachycardias.

Additional Indexing Words:
Heart block  Bradycardia  Reentry mechanisms

RECENT evidence has indicated that the technic of transvenous cardiac stimulation may terminate certain refractory tachycardias. The concept that an appropriately timed electrical stimulus delivered to the heart could terminate supraventricular tachycardias was first observed in patients with the Wolf-Parkinson-White syndrome. Subsequent studies have shown that supraventricular arrhythmias in the absence of pre-excitation may be abolished by atrial stimulation.

The patient described in this report had a refractory A-V junctional tachycardia, which developed in the setting of heart block and bradycardia. The tachyarrhythmia was abruptly terminated by right ventricular stimulation at a rate considerably slower than the rate of the tachycardia. Analysis of the records indicated that a critical time interval was present, during which a ventricular stimulus was effective. These observations support the concept that a reentry mechanism perpetuated the tachycardia in the absence of the Wolf-Parkinson-White syndrome. Moreover, right ventricular stimulation may be a useful means of acutely terminating such tachyarrhythmias.

Report of Case

W. T., a 45-year-old white man, was transferred from a local hospital for the treatment of recurrent episodes of tachycardia. He had had rheumatic fever at age 15 years and a documented heart murmur since age 20 years. In 1958, an electrocardiogram revealed first degree heart block with a P-R interval of 0.26 sec. In 1963, the P-R interval had widened to 0.40 sec. Since 1965, electrocardiograms revealed varying first, second, and rarely third degree heart block (fig. 1). Symptomatically, the patient complained...
Electrocardiograms. (Panel A) In June 1968, there is sinus rhythm at 75/min with first degree A-V block and a P-R interval of 0.44 sec. Second degree (2:1) A-V block occurred transiently and is shown in leads V_5 to V_6. The QRS interval is slightly prolonged at 0.10 sec, with a nonspecific intraventricular conduction disturbance. (Panel B) Electrocardiogram was taken during junctional tachycardia. The rate is 160 beats/min with occasional retrograde 2:1 A-V conduction (lead II). The QRS complexes are similar to those during intact antegrade conduction but for a minimal terminal conduction delay.

An electrocardiogram recorded during a typical episode of tachycardia revealed an A-V junctional tachycardia of 160 beats/min with occasional 2:1 retrograde atrial activation (fig. 1). Although the QRS complexes were prolonged, the ectopic tachycardia was felt to be junctional rather than ventricular in origin because of the close similarity between the QRS contour and duration during the tachycardia and during intact antegrade conduction on previous electrocardiograms (fig. 1). During the tachyarrhythmia, a no. 5 bipolar pacing catheter was inserted into the right antecubital vein and advanced under fluoroscopic control to the right atrium. Right atrial stimulation was performed at rates of 100 and 150/min at 5 to 10 ma using a Medtronic Model 5800 pulse generator. Atrial capture was produced, but of paroxysmal episodes of rapid heart action, frequently accompanied by substernal chest discomfort. Transient episodes of ectopic tachycardia had been recorded at an outside hospital. On February 21, 1969, the patient was referred to the Durham Veterans Administration Hospital with a 5-day history of recurrent tachycardia which was refractory to digitalis. Antiarrhythmic agents had not been administered because of the background history of heart block. The pertinent physical findings on admission included a rapid regular apical pulse of 160 beats/min and blood pressure of 100/65 mm Hg. The lungs were clear. Cardiac examination revealed cardiomegaly with moderate left ventricular enlargement and a grade II/VI diastolic decrescendo murmur along the left sternal border.
Figure 2

(Panel A) Lead II of the electrocardiogram during the junctional tachycardia at 160 beats/min. (Panel B) Right atrial stimulation at 100/min. (Panel C) Right atrial stimulation at 180/min. In both panels B and C intermittent atrial capture is evident. Although atrial capture was produced, the junctional tachycardia was not altered. Paper speed is 25 mm/sec with 0.04-sec time lines.

Figure 3

On the left of panel A, the junctional tachycardia, at 160 beats/min may be seen. The effect of right ventricular stimulation at 94/min is shown. The first stimulus produced a fusion beat as indicated by the single asterisk. The second stimulus (double asterisk) produced ventricular capture and termination of the arrhythmia with a subsequent paced ventricular rhythm of 94/min. Following termination of ventricular stimulation (panel B), 2 sec of ventricular asystole occurred, followed by resumption of the tachycardia. Atrial activity with a P-P interval of 1.04 sec was seen during ventricular asystole but was no longer evident with the new onset of the tachycardia. Paper speed was 25 mm/sec with 0.04-sec time lines.

interruption or alteration of the tachycardia was not observed (fig. 2).

The electrode catheter was then advanced to the apex of the right ventricle. Stimulation of the
right ventricle at a rate of 94/min at 1 ma abruptly terminated the tachycardia on several occasions. The electrocardiograms at the time of conversion are illustrated in figures 3, 4, and 5. When ventricular pacing was discontinued, a transient ventricular asystole was followed by recurrence of the paroxysmal tachycardia (figs. 3 and 4) or by complete heart block (fig. 5).

The patient was placed in a coronary care unit, where constant electrocardiographic monitoring could be performed. The use of antiarrhythmic agents, quinidine sulfate, and propanolol, and ventricular pacing at various rates in both the demand and fixed rate modes were evaluated. The combination of quinidine sulfate, 300 mg every 6 hours, and fixed rate ventricular pacing at 84/min was found to be the most successful regimen in preventing recurrences of the ectopic tachycardia. On March 11, 1969, a permanent transthoracic cardiac pacemaker was inserted with good result. After 8 weeks of follow-up, the patient has remained free of symptoms and has experienced no further recurrences of the tachyarrhythmia.

**Results**

An analysis was made of stimuli delivered to the right ventricle in the setting of the ectopic tachycardia. Right ventricular stimulation was at 94/min; the ventricular rate of the tachycardia was 160 beats/min (figs. 3 to 5). Ten stimuli were available for analysis. Paper speed was 50 mm/sec for study of 8 stimuli and 25 mm/sec for 2 stimuli. Time lines were 0.04 sec. Measurements were made of the interval from the onset of the R wave to the pacemaker stimulus (R-Pm interval) and correlations were made with these intervals and the resultant effect on the arrhythmia.

*Figure 4*

Panel A illustrates the Junctional tachycardia at 160 beats/min. In panel B, right ventricular stimulation at 94/min in the setting of the tachycardia is shown. The second, third, fifth, and sixth stimuli find the ventricle refractory. The first and fourth stimuli (single astersisk) produce premature ventricular depolarization and reset the tachycardia. The seventh stimulus (double asterisk) produces a premature ventricular contraction and abruptly terminates the tachycardia.

Panel C illustrates ventricular pacemaker capture at 94/min. In panel D, after right ventricular stimulation is stopped, 2 sec of ventricular asystole followed by resumption of the tachycardia may be seen. For the first three beats the tachycardia is slightly irregular but becomes regular at 160 beats/min. Atrial activity with a P-P interval of 0.94 sec is seen during the transient ventricular asystole. Paper speed is 50 mm/sec with 0.04-sec time lines.

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Refractory and excitable periods during a given R-R interval were found. Four stimuli with R-Pm intervals of 0.03 to 0.21 sec fell within the ventricular refractory period. Six stimuli with R-Pm intervals from 0.25 to 0.35 sec produced ventricular capture. No stimuli were recorded with an R-Pm interval from 0.21 to 0.25 sec, and it could not be determined whether this 40-msec interval belonged to the ventricular refractory or excitable periods. During the excitable period, three stimuli with R-Pm intervals of 0.31 to 0.35 sec produced premature ventricular depolarization and either early resetting of the tachycardia or a fusion beat. Three stimuli falling within a critical time interval of 50 msec (R-Pm interval of 0.25 to 0.30 sec) produced premature ventricular depolarization and abrupt termination of the tachycardia. The effects of right ventricular stimulation on the junctional tachycardia are graphically illustrated in figure 6.

Atrial activity presumably representing the sinus node cycle can be seen with A-V dissociation during ventricular pacemaker capture and immediately following the cessation of pacemaker stimulation. The P-P interval varied from 0.94 to 1.04 sec and is readily visualized during transient ventricular asystole in figures 3 and 4 and during A-V block in figure 5. With resumption of the junctional tachycardia (fig. 3, line B; fig. 4, line D), well-defined atrial activity is no longer present. During the tachycardia only intermittent, probably retrograde P waves are seen.

**Discussion**

A recent review of A-V junctional arrhythmias has emphasized the importance of slowing of the primary cardiac pacemaker.
with or without A-V block in the genesis of paroxysmal tachycardias. With bradycardia there is temporal dispersion of repolarization along with areas of unidirectional block in the conducting system, factors which set the stage for rapid paroxysmal reentry arrhythmias. The tachycardia illustrated in the present case originated in the setting of well-documented A-V block and bradycardia. The disappearance of the sinus P wave with the onset of the tachycardia suggests that the arrhythmia was initiated by an atrial echo and that this coupled reciprocal response became perpetuated as a reciprocating junctional tachycardia.

Moe and co-workers experimented induced paroxysmal nodal tachycardia in a dog by premature atrial stimulation. These investigators proposed that dissociated pathways within the A-V node sustained a circus movement tachycardia. Moreover, it was demonstrated that premature excitation of the atrium or ventricles could successfully terminate a paroxysm of tachycardia.

Patients with the Wolf-Parkinson-White syndrome have paroxysmal junctional tachycardias which are perpetuated by reentry mechanisms. Massumi and associates and Durrer and co-workers have successfully converted A-V junctional tachycardias in these patients by the production of atrial premature beats. Moreover, Cobb and associates described the successful conversion of a junctional tachycardia in a patient with the Wolf-Parkinson-White syndrome by both atrial and ventricular stimulation at rates slower than that of the tachycardia.

Hunt and co-workers were the first to demonstrate that an A-V junctional tachycardia in a patient without the Wolf-Parkinson-White syndrome could be interrupted by appropriately timed atrial stimuli. These authors felt that the critical timing necessary for atrial stimulation to abolish this rhythm was in keeping with a reentry process. Furthermore, Massumi and associates have briefly described the use of catheter-induced premature ventricular contractions to terminate supraventricular tachycardias which developed in two patients during cardiac catheterization.

The patient discussed in this report was unique in that the junctional tachycardia did not respond to atrial stimulation but responded dramatically to ventricular pacing at a rate slower than that of the tachycardia. The sequence of events strongly suggests a reentry mechanism as the underlying basis for the arrhythmia. Analysis of ventricular stimuli defined ventricular refractory and excitable periods. Within the excitable period, two intervals became apparent. Stimuli falling at 0.31 sec or greater after the onset of the previous R wave produced ventricular depolarization and reset the arrhythmia. Presumably, retrograde impulses entered excitable reentry pathways and sustained the tachycardia with the subsequent junctional beat.
Panel A is a tracing of figure 4, panel B. The line drawings in panels B and C diagrammatically illustrate theoretical reentry circuits sustaining the tachycardia. In panel B, the reentry cycle is depicted entirely confined to A-V junctional tissue. In panel C, the mechanism is illustrated including the ventricles in the reentry circuit. The first pacemaker stimulus (PM) occurs 0.31 sec after onset of the previous QRS and produces premature ventricular depolarization which conducts retrograde to reset and perpetuate the reentry tachycardia. The subsequent junctional beat occurs prematurely by 0.03 sec. The second and third stimuli occur when the ventricle is refractory and have no effect on the tachycardia. The fourth stimulus occurs 0.29 sec after the onset of the previous QRS and produces premature ventricular depolarization which terminates the tachycardia. The retrograde impulse penetrates the reentry path making it refractory to the reentrant impulse from the preceding beat. However, as the entire reentry pathway had not recovered excitability, the circuit could not be completed and the tachycardia was not perpetuated.

occurring prematurely. Three stimuli falling earlier in the excitable period between 0.25 and 0.30 sec after the onset of the preceding R wave produced ventricular depolarization and abrupt termination of the tachycardia. It is hypothesized that stimulai during this critical 50-msec interval produced retrograde impulses entering a reentry pathway which was only partially excitable. Retrograde impulses could penetrate the reentry path making it refractory to the reentrant impulse from the preceding beat, but could not complete the reentry circuit, as the entire pathway had not yet recovered excitability. This hypothesis is diagrammatically illustrated in figure 7.

A junctional origin for the arrhythmia with reentry entirely within junctional tissue or possibly involving the ventricles is postulated (fig. 7). Although a ventricular origin for the tachycardia cannot be excluded with absolute certainty, the minimal change in the QRS contour and duration from previously conducted complexes would favor a junctional origin for the arrhythmia. The minimal terminal conduction disturbance in the QRS complexes during the tachycardia may actually represent participation by the ventricles in the reentry circuit (fig. 7, panel C).

In the patient described in this report, a reentry A-V junctional tachycardia occurred in the presence of antegrade A-V block. The presence of A-V block would explain the inability of atrial stimulation to interrupt the reciprocating rhythm. Although earlier views proposed that the atria participated in the circus pathway of reciprocating junctional beats, Mignone and Wallace provided data that echo beats may occur by reentry pathways entirely within the A-V node and that the atria are not an essential link for this circuit.

The combination of rapid paroxysmal tachycardias in the setting of heart block and bradycardia frequently poses a difficult therapeutic problem. The use of chronic ventricular pacing in combination with antiarrhythmic drugs in the prevention of such life-threatening tachycardias is well documented. This report documents the use of right ventricular stimulation in the acute termination of a rapid junctional tachycardia in addition to its use in the chronic prevention of the arrhythmia by maintaining an adequate ventricular rate.

The value of transvenous cardiac stimulation in the acute therapy of refractory tachyarrhythmias is apparent. The technic has proven effective in both atrial flutter and A-V junctional tachycardias in patients with and without the Wolf-Parkinson-White syndrome. The method obviates the requirements for general anesthesia normally used with standard DC cardioversion and the anti-inotropic effects of most antiarrhythmic agents. This technic would seem to be especially useful in patients in whom tachycardias evolve from background bradycardia and in patients with recurrent paroxysmal arrhythmias.

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