CASE REPORTS

His-Ventricular Dissociation in a Patient with Reciprocating Tachycardia and a Nodoventricular Bypass Tract

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SUMMARY A patient with recurrent bouts of atrial fibrillation and wide-complex regular tachycardia underwent electrophysiologic studies. Premature atrial stimulation or atrial pacing during sinus rhythm resulted in gradual lengthening of the PR and AH intervals, narrowing of the HV interval and progressive preexcitation with a left bundle branch block and left-axis contour. Induction of tachycardia was dependent on critical delay in the atrioventricular interval and was associated with attainment of a maximal preexcitation pattern. During tachycardia, the ventriculoatrival interval was constant, whereas the interval from His bundle deflection to the ventricular complex was variable. We postulate that the tachycardia circuit involved reciprocation within the atrioventricular node and that a nodoventricular bypass tract was present in close anatomic or functional association with the slow atrioventricular nodal pathway. Our data suggest that both the nodoventricular bypass tract and the His-Purkinje system may be passive "bystanders" rather than essential components of the tachycardia circuit. In addition, although HV dissociation usually implies ventricular tachycardia, this case demonstrates that HV dissociation during wide-complex regular tachycaryrrhythmia is not diagnostic of ventricular tachycardia.

RECIPOCATING TACHYCARDIA in patients with nodoventricular bypass tracts has rarely been reported in detail. Some investigators have concluded that the initiation or maintenance of the tachycardia involves antegrade conduction over the bypass tract and retrograde conduction via the His-atrioventricular node axis.1-8 However, Ward et al.7 suggested that the tachycardia circuit is confined to the atrioventricular node and that the bypass tract plays no role in either initiation or maintenance of the tachycardia. We report a patient with reciprocating tachycardia in whom the tachycardia circuit involved the atrioventricular node, but not the nodoventricular bypass tract or the His-Purkinje system. His-ventricular (HV) dissociation was present during atrioventricular nodal reciprocating tachycardia. This case demonstrates that HV dissociation is not diagnostic of ventricular tachycardia because it may also occur in patients with atrioventricular nodal reciprocating tachycardia and a nodoventricular bypass tract.

Case Report

A 30-year-old man was admitted to the hospital because of a history of recurring episodes of palpitations since age 19 years. The episodes occurred every 2-3 months and were usually spontaneous in onset; at times, the palpitations were triggered by emotional excitement or physical exertion. During the episodes, he would experience dyspnea, weakness and light-headedness; he had no history of syncope. The palpitations would usually last 4-8 hours. At times, the episodes could be terminated by carotid sinus massage or propranolol, 40 mg orally. During the past 10 years, intermittent treatment with digitalis and quinidine did not seem to affect the frequency of these episodes. At the time of his evaluation, he was taking no medications. There was no family history of episodic palpitations, syncope, heart disease or sudden death.

The patient was a well-nourished, well-developed man with a blood pressure of 110/70 mm Hg and a regular heart rate of 72 beats/min. The cardiac examination and the remainder of the physical examination showed no abnormalities.

The chest roentgenogram, baseline 12-lead ECG and M-mode echocardiogram showed no abnormalities. Numerous 12-lead ECGs recorded during episodes of palpitations showed regular tachycardia at a rate of 215 beats/min; the QRS morphology was that of a left bundle branch block and the QRS axis was 0°. No P waves could be seen. ECGs recorded during atrial fibrillation showed a rapid ventricular response of up to 200 beats/min; the QRS morphology was identical to that recorded during episodes of regular tachycardia.

Methods

The patient was studied in a fasting, nonsedated state. Using the Seldinger technique, three quadrripolar electrode catheters were inserted into the right femoral vein and positioned against the high lateral right atrium, across the tricuspid valve and against the right ventricular apex, respectively. An additional quadrripolar electrode catheter was inserted into a left
antecubital vein and manipulated into the coronary sinus. Surface leads V₁, I and II and the high right atrial, His bundle, coronary sinus and right ventricular electrograms were displayed on a DR-12 oscilloscope and recorded on an Electronics for Medicine recorder at a paper speed of 100 mm/sec. Programmed atrial and ventricular electric stimulation was achieved by means of a programmable stimulator (Bloom, Inc.) with a pulse duration of 2 msec at two to three times the diastolic threshold. Incremental right atrial, left atrial (coronary sinus) and right ventricular overdrive pacing was performed to a minimal paced cycle length of 300 msec. Early in the study, atrial pacing resulted in unstable atrial arrhythmias, i.e., atrial fibrillation and atrial tachycardia; 700 mg of procainamide was administered intravenously to stabilize the atrial rhythm during pacing. Single atrial or ventricular stimuli at 10-msec decrements were induced throughout the respective diastolic cycles. Similarly, during induced tachycardia, the atrium and ventricle were scanned with progressively premature atrial or ventricular extrastimuli. Because of the patient’s propensity to develop atrial fibrillation during atrial pacing, a total of 1.4 g of procainamide was infused intravenously during the study to stabilize the atrial rhythm. The patient was placed on a regimen of procainamide, 750 mg every 4 hours, and digoxin, 0.25 mg/day.

Results

Evidence for a Nodoventricular Bypass Tract

During control recordings, the atroventricular nodal conduction time (AH), infranodal conduction time (HV) and QRS duration were 55, 50 and 80 msec, respectively. Incremental atrial extrastimuli induced during right atrial drive at a cycle length of 500 msec was associated with gradual prolongation of the AH interval. No abrupt increase in the AH interval was observed. Atrial extrastimuli induced from 310–260 msec after the last driven atrial complex were associated with progressive shortening of the HV interval concomitant with development of a left bundle branch contour (fig. 1). Comparable pacing from the coronary sinus resulted in a lessened increment of the AH interval without evidence of preexcitation. During atrial fibrillation, normal, fusion and fully preexcited (left bundle branch block) complexes were recorded (fig. 2). The HV interval varied with the degree of fusion and fully preexcited complexes were never preceded by a His bundle deflection. Spontaneous junctional extrasystoles had a contour of left bundle branch block that was similar to preexcited complexes and were not preceded by a His bundle deflection. These findings are characteristic of a nodoventricular bypass tract inserting into the right ventricle.

Initiation of Tachycardia

Incremental atrial overdrive pacing (cycle length 600–325 msec) resulted in incremental increases in the AH interval from 60 to 120 msec. At a paced cycle length of 300 msec, atrioventricular nodal Wenkebach conduction appeared and the AH interval varied from 110–170 msec. This was accompanied by progressive shortening of the HV interval and tachycardia was initiated at the time of maximal ventricular preexcitation (fig. 3). Tachycardia initiation was therefore dependent on critical lengthening of the atrioventricular interval and was associated with maximal preexcitation. These data suggest that block of a

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Simultaneous recordings from surface leads V₁, I and III from intracardiac positions in the high right atrium (HRA) and coronary sinus (CS), and the His bundle electrogram (HBE). During right atrial (RA) pacing at a cycle length (CL) of 500 msec, an atrial extrastimulus (S₂) is introduced with a coupling time of 270 msec. There is prolongation in atroventricular nodal conduction time (AH) from 80 to 165 msec and shortening in infranodal conduction time (HV) from 50 to 25 msec. The ventricular complex that results from S₂ (V₂) has a left bundle branch block contour. Prolongation in atroventricular conduction time associated with shortening of the HV time and development of a left bundle branch block contour are typical of a right-sided nodoventricular bypass tract. Time lines in this and following figures represent 1 second.
pathway in the atrioventricular node enabled conduction down another atrioventricular nodal pathway in close proximity to the bypass tract. Reciprocity could either be totally within the atrioventricular node or involve the nodoventricular bypass and His-Purkinje system.

Incremental right ventricular overdrive pacing during sinus rhythm at cycle lengths of 500–400 msec resulted in gradual prolongation of retrograde ventriculoatrial (VA) conduction; retrograde VA Wenckebach was observed at a paced cycle length of 300 msec. Tachycardia could not be initiated during either right ventricular overdrive or programmed right ventricular stimulation.

Characteristics of the Tachycardia

The tachycardia cycle length varied between 300–360 msec and showed a complete left bundle branch block contour that was identical to that during spontaneous atrial fibrillation and during atrial overdrive at rates more rapid than the tachycardia (fig. 4). The atrial deflection was inscribed during the QRS complex, and the relationship between the onset of ventricular activation, as measured from the surface leads, to the septal atrial electrogram was 100 msec. Variations in tachycardia cycle lengths were a result of spontaneous variations in the interval from atrial to ventricular complexes. The septal atrial electrogram always preceded that from either the coronary sinus or the right atrium (fig. 4). In contrast to the constancy of the VA interval, the His deflection was obviously dissociated from either the atrial or the ventricular complexes. The His deflection occurred slightly before, within or just after the ventricular complex (fig. 5).

The normal retrograde atrial activation sequence during tachycardia and the decremental pattern of VA conduction during ventricular pacing argue against the involvement of a retrogradely conducting extranodal accessory pathway in the tachycardia circuit. In addition, the HV dissociation excludes participation of the His-Purkinje system in the retrograde limb of the tachycardia circuit. These findings are consistent with reciprocation confined to the atrioventricular node. However, retrograde conduction via a His-atrial bypass tract would also result in a constant VA conduction time in the presence of variable atrioventricular conduction times.

Response to Atrial or Ventricular Stimulation During the Tachycardia

Premature atrial stimulation on one occasion resulted in advancement of the next ventricular complex and was not followed by a compensatory pause.

Figure 2. During atrial fibrillation, the ventricular complexes displayed in surface leads V₁, I and III include fusion and fully preexcited beats. In the fully preexcited beat (last QRS complex on right), a His bundle deflection (H) does not precede the ventricular complex. In the fusion beats, the HV intervals vary from 0–45 msec, depending on the degree of fusion. See figure 1 for definition of abbreviations.

Figure 3. With right atrial pacing at a cycle length of 300 msec, there is atrioventricular nodal Wenckebach conduction. As the AH interval prolongs, there is concomitant shortening in the HV interval associated with progressive development of ventricular preexcitation with a left bundle branch block contour. The tachycardia is initiated (arrow) when there is maximal ventricular preexcitation; at this time, the His deflection no longer precedes the ventricular depolarization. S₁ = premature ventricular extrastimulus; see figure 1 for definition of other abbreviations.
A very late diastolic ventricular extrastimulus during tachycardia resulted in advancement of ventricular activation without change in the atrial cycle length (fig. 6). This ventricular premature depolarization was associated with a fully compensatory pause. Earlier ventricular extrastimuli advanced the tachycardia and were associated with incremental increases in VA conduction. Because only the very late diastolic ventricular premature depolarization was not associated with a change in the atrial cycle length, one cannot completely rely on this observation to exclude the ventricles from the tachycardia circuit. The tachycardia could be consistently terminated only by two closely coupled ventricular depolarizations.

The patient was treated with procainamide to abolish premature complexes that might trigger atrial fibrillation or the regular tachycardia; in addition, digoxin therapy was instituted to interfere with the presumed atrioventricular nodal reciprocation. The patient has had no episodes of tachycardia (5 months).

**Discussion**

The findings of progressive prolongation of the PR and AH intervals associated with a decrease in the HV interval and of ventricular preexcitation with a left bundle branch block configuration are characteristic of a nodoventricular bypass tract with insertion into the right ventricle. Tachycardia was only initiated by attainment of a critical atrioventricular delay and was associated with maximal preexcitation. These findings are best explained by the presence of dual atrioventricular nodal pathways with differing conduction and refractory properties and a close anatomic or functional link between the slow atrioventricular nodal pathway and the nodoventricular bypass tract (fig. 7). During sinus rhythm, conduction always occurred via the fast atrioventricular nodal pathway and preexcitation was never present. With incremental atrial pacing, physiologic delay in the fast pathway or a progressive block in retrograde penetration of the slow pathway enabled conduction over the slow pathway, and hence, over the bypass tract. At a critical atrial rate, block in the fast pathway enabled induction of a reciprocating tachycardia with maximal preexcitation.

Both the spontaneous and the numerous induced episodes of tachycardia showed a fully preexcited pattern. In addition, during tachycardia, the His
deflection was dissociated from the ventricular complex. These findings are best explained by the following tachycardia circuit: antegrade conduction via the slow atrioventricular nodal pathway with associated passive conduction over the nodoventricular pathway, with the reentrant circuit confined to the atrioventricular node (fig. 7). The His-Purkinje system is clearly not involved in the tachycardia circuit, and the HV dissociation is explained by varying physiologic delay in the His-Purkinje system or in the atrioventricular node distal to the takeoff of the nodoventricular pathway. Because premature atrial stimulation during tachycardia advanced the ventricular complex, an atrial link in the tachycardia circuit cannot be excluded.

A ventricular premature depolarization induced late in the tachycardia cycle was not associated with advancement of the atrial depolarization. Although this supports lack of involvement of the ventricles in the tachycardia circuit, these data are not conclusive, for the premature ventricular depolarization could have been associated with slight reciprocal intraventricular or VA conduction delay, enabling a compensatory atrial cycle length. Earlier ventricular premature depolarizations clearly advanced the atrial electrogram and were associated with incremental VA conduction delays suggestive of retrograde penetration via the atrioventricular node. Although unlikely, we cannot definitely exclude the presence of a reentrant circuit confined to a longitudinally dissociated nodoventricular fiber or involving antegrade and retrograde conduction over separate nodoventricular fibers. Similarly, retrograde conduction over an accessory atrioventricular pathway with node-like properties cannot be excluded.

Previous studies of patients with nodoventricular bypass tracts yielded different interpretations of the tachycardia circuit. For example, Ward et al.7 concluded that the circuit was confined to the atrioventricular node. In one patient in their series who received i.v. disopyramide during tachycardia, preexcitation disappeared, but the tachycardia continued without change in cycle length. In contrast, Gallagher et al.8 recently reported six patients with nodoventricular bypass tracts and reciprocating tachycardias and concluded that the nodoventricular bypass was a necessary component of the tachycardia circuit because fusion or normal complexes were never seen during tachycardia. They also concluded that the His-
Purkinje system was involved in the retrograde portion of the circuit because in three patients the His deflection (presumed to be retrograde) always followed ventricular activation by a constant time interval. If one assumes a close anatomic link between the slow atrioventricular nodal pathway and the nodule-ventricular bypass tract and if atrioventricular nodal reciprocation occurs in the usual manner (i.e., antegrade via the slow pathway and retrograde via the fast pathway), then the ventricular complex during tachycardia would always be maximally preexcited. Similarly, registration of the His deflection after the ventricular complex does not prove that this deflection is retrogradely conducted from the ventricle. During tachycardia in our patient, for example, the His deflection was at times recorded after the ventricular complex. The variation in the HV interval may have resulted from either variations in conduction over the distal portion of the atrioventricular node or changes in His-Purkinje conduction due to pretreatment with intravenous procainamide.

Our observations have several important clinical implications. First, if the tachycardia circuit is confined to the atrioventricular node, as suggested by our findings, surgical ablation of the nodoventricular bypass tract would not terminate the arrhythmia. In addition, others have clearly shown that tachycardias associated with nodoventricular bypass tracts may mimic ventricular tachycardia, in that broad QRS complexes are not preceded by a His bundle deflection, and atrioventricular dissociation may be present. Our findings extend these observations to show that HV dissociation during wide-complex tachycardia is similarly not diagnostic of ventricular tachycardia. Ventricular tachycardia was conclusively eliminated in our patient because complexes identical to those recorded during tachycardia were inscribed during premature atrial stimulation, atrial overdrive pacing and atrial fibrillation. Similarly, during tachycardia the VA interval was constant and the HV interval was variable.

Finally, a recent study by Benditt et al. concluded that in patients with reciprocating atroventricular nodal tachycardia, the interval from the onset of ventricular activation to the inscription of the septal atrial electrogram was usually less than 61 msec and less than 95 msec to the high right atrial electrograms. These intervals were suggested as important criteria for distinguishing between atrioventricular nodal reentry and reentry involving an accessory atrioventricular nodal bypass. These criteria are clearly not applicable to patients with atrioventricular nodal reciprocation and nodoventricular bypass tracts, because during tachycardia the ventricles may be preexcited via the bypass tract and thus result in a longer apparent VA interval.

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