Electrophysiologic Identification of Dual Atrioventricular Nodal Pathway Conduction in Patients with Reciprocating Tachycardia Using Anomalous Bypass Tracts

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SUMMARY In 67 consecutive patients with reciprocating tachycardia using an anomalous atrioventricular (AV) or nodoventricular (NV) bypass tract, electrophysiologic findings suggested the coexistence of dual AV nodal pathway conduction in eight patients. The evidence of coexistent dual AV nodal pathways and anomalous bypass tracts took three forms. In four patients, alternating short and long AV nodal conduction time (AH intervals), presumably caused by rate-dependent 2:1 conduction in the fast AV nodal pathway, were recorded during AV reciprocating tachycardia. Intravenous administration of atropine invariably resulted in 1:1 fast AV nodal pathway conduction during tachycardia in all patients. In three other patients who had anomalous AV bypass tracts capable of only retrograde conduction, discontinuous AV nodal conduction curves (A1A2, H,H,) were generated during atrial extrastimulation. The remaining patient had an anomalous NV bypass tract bridging the slow AV nodal pathway and the right ventricle. During atrial extrastimulation, antegrade block in the fast AV nodal pathway caused antegrade conduction across a pathway composed of the slow AV nodal pathway and the NV bypass tract. This suddenly produced right ventricular preexcitation with inscription of the His bundle deflection within the QRS complex, disrupting the AV nodal conduction curves (A1A2, H,H2). A sustained reciprocating tachycardia with a complete left bundle branch block pattern was subsequently initiated using the slow AV node-NV bypass tract pathway for antegrade conduction and the fast AV node-His-Purkinje system pathway for retrograde conduction. These observations suggest that dual AV nodal pathway conduction can be identified electrophysiologically in patients with reciprocating tachycardia involving anomalous bypass tracts, but its manifestations may take several forms.

DUAL ATGIOVENTRICULAR (AV) nodal pathway conduction is a common electrophysiologic phenomenon. Characteristically, discontinuous AV nodal conduction curves (A1A2, H1H2) are generated with chronic bifascicular block. (abstr) Am J Cardiol 43: 390, 1979

Patients and Methods

We reviewed data of 67 consecutive patients with reciprocating tachycardia that involved an anomalous bypass tract. Evidence suggestive of manifest and concealed anomalous bypass tract was electrophysiologically identified as previously described.2 4 The diagnosis of dual AV nodal pathways was based on the AV conduction patterns described by Denes et al.1 We assumed that the AV node could undergo functional longitudinal dissociation into two conducting pathways differing in electrophysiologic properties — fast and slow AV nodal pathways (short and long AH intervals, respectively) with different refractory periods. Eight of the 67 patients had electrophysiologic findings suggestive of the presence of dual AV nodal pathway conduction. Six were male and two female. Their ages ranged from 21–65 years (mean 39.2 years). All had palpitations and episodes of tachycardia documented electrocardiographically.

After obtaining an informed consent for the study, all cardiotonic and antiarrhythmic medications were discontinued 48–72 hours before the study. The study was performed with each patient in a postabsorptive, nonsedated state. High right atrial (HRA) activity was recorded through the proximal pair of electrodes of a hexapolar electrode catheter, and the left atrial (LA) electrogram was recorded with a quadripolar electrode catheter placed in the coronary sinus or in the left atrium through a patent foramen ovale.6 Using a conventional technique,4 the His bundle electrogram (HBE) was obtained through a tripolar electrode catheter, from which low septal right atrial (LRA) activity was recorded. The intracardiac electrograms were displayed simultaneously with standard electrocardiographic leads I, II, and V1 on a multichannel oscilloscopic photographic recorder (Electronics for Medicine DR-16, White Plains, New York) at a paper speed of 100 or 150 mm/sec, using filter settings between 40–500 Hz.

Through the remaining pairs of electrodes of the hexapolar and quadripolar electrode catheters, both the atria and the right ventricular endocardium were stimulated at one or two cycle lengths (A1, A2 or V1, V2) via a programmed digital stimulator (Biocelestronix, Miami Springs, Florida) that delivered stimuli (S1 and S2) 2.0 msec long at approximately twice diastolic threshold.5 After every eighth spontaneous or paced beat (A1 or V1) a single premature atrial or ventricular beat (A2 or V2) was delivered at progressively shorter coupling intervals until the effective refractory period of the atrium or the ventricle was encountered.

AV conduction intervals and refractory periods were defined and measured as conventionally described.3 5 A1, H1 and V1 were low septal right atrial, His bundle, and ventricular responses induced by the driving stimuli (S1); A2, H2 and V2 were low septal right atrial, His bundle and ventricular responses, induced by the premature stimuli (S2).

Results

Electrophysiologic findings suggestive of dual AV nodal pathway conduction in these eight patients with reciprocating tachycardia that involved an anomalous bypass tract was classified into three forms:

Alternating Fast and Slow AV Nodal Pathway Conduction During AV Reciprocating Tachycardia

In four patients with AV reciprocating tachycardia that involved an anomalous AV bypass tract, atrial extrastimulation failed to induce characteristic dual AV nodal pathway conduction patterns. This was because the anomalous bypass tracts maintained antegrade conduction at short atrial premature coupling intervals, precluding disclosure of discontinuous AV nodal conduction curves (A1A2, H1H2). The antegrade effective refractory periods of the anomalous AV bypass tracts ranged from 280–340 msec at atrial pacing cycle lengths of 450–700 msec in these four patients. After failure of antegrade conduction over the anomalous AV bypass tracts, the AV nodal conduction time (AH intervals) of the atrial premature beats at which AV reciprocating tachycardia was induced ranged from 180–290 msec. During AV reciprocating tachycardia, there were alternately short and long cardiac cycles due to alternation of antegrade AV nodal conduction times (AH intervals) (fig. 1A). During AV reciprocating tachycardia, the RR intervals alternated between cycle lengths of 310 and 420 msec. The alternating short and long cardiac cycle lengths during AV reciprocating tachycardia were due to alternation of AV nodal conduction times (AH intervals) of 150 and 260 msec. The alternation between short and long AV nodal conduction times was presumed to reflect alternating AV conduction between fast and slow AV nodal pathways, consequent to the development of cycle length-dependent 2:1 conduction block in the fast AV nodal pathway during AV reciprocating tachycardia. Intravenous infusion of atropine (1.2 mg) invariably produced enhancement of antegrade fast AV nodal pathway conduction, resulting in 1:1 AV conduction via the fast AV nodal pathway during AV reciprocating tachycardia in all four patients (fig. 1B).

Induction of Discontinuous AV Nodal Conduction Curves in the Presence of a Concealed Anomalous AV Bypass Tract

During programmed atrial extrastimulation, discontinuous AV nodal conduction curves (A1A2, H1H2) characteristic of dual AV nodal pathway conduction were induced in three patients with anomalous AV bypass tracts capable only of retrograde conduction (concealed Wolff-Parkinson-White syndrome). Each time, antegrade AV conduction by way of a slow AV nodal pathway facilitated initiation of AV reciprocating tachycardia. The high right atrium was driven at a cycle length of 700 msec (fig. 2A). The A1A2, H1H2 and A1A2, A2H2 curves were discontinuous due to a sudden increase in AV nodal conduction time (A3H3) at an atrial premature coupling interval (A1A3) of 550 msec. This reflected failure of antegrade conduction over a fast AV nodal pathway with resultant slow AV nodal pathway conduction.
The antegrade effective refractory periods of the fast and slow AV nodal pathways were 550 and 330 msec, respectively. Induction of sustained AV reciprocating tachycardia coincided with the entire period of antegrade slow AV nodal pathway conduction between atrial premature coupling intervals of 550 and 340 msec. The representative tracings corresponding to figure 2A are shown in figure 3. The LA was activated before the low septal right atrium in the HBE as well as the HRA during AV reciprocating tachycardia, indicating the use of a left-sided anomalous bypass tract for retrograde conduction (fig. 3B and C). During AV reciprocating tachycardia, the phenomenon of alternating fast and slow AV nodal pathway conduction was not observed, because the tachycardia cycle length (390 msec) was within the antegrade effective refractory period of the fast AV nodal pathway (550 msec). As expected, the slow AV nodal pathway was used for antegrade conduction.

In each patient, the presence of a left-sided anomalous AV bypass tract functioning in a ventriculoatrial direction was further supported by the programmed ventricular stimulation study. As shown in figure 2B, the right ventricle (RV) was driven at a cycle length of 700 msec. The sequence of retrograde atrial activation was first the LA followed by the LRA in the HBE and the HRA, identical to that seen during reciprocating tachycardia. Furthermore, the ven-
triculoatrial conduction curves (V1V2, V2A2) showed rather constant ventriculoatrial conduction time despite progressively premature ventricular stimulation characteristic of retrograde conduction via an anomalous AV bypass tract. Reciprocating tachycardia zones could be defined between ventricular premature coupling intervals of 380-270 msec.

**Disruption of AV Nodal Conduction Curves with an Anomalous Nodovenousicular Bypass Tract Participating in the Genesis of Reciprocating Tachycardia**

In the remaining patient, electrophysiologic findings suggested an anomalous nodovenousicular bypass tract associated with dual AV nodal pathway conduction. Clinically this patient had an associated Ebstein's anomaly and electrophysiologically the anomalous nodovenousicular bypass tract bridged the slow AV nodal pathway and the right ventricle, participating in the genesis of reciprocating tachycardia (fig. 4). Characteristically, the reciprocating tachycardia could be elicited with either programmed atrial or ventricular extrastimulation, and it used the slow AV node-nodovenousicular bypass tract pathway for antegrade conduction and the fast AV node-His-Purkinje system pathway or the distal common AV node-His-Purkinje system pathway for retrograde conduction.

The high right atrium was driven at a cycle length of 600 msec; the A1A2, H1H2 and A1A2, A2H2 curves were compatible with AV nodal conduction properties (fig. 5A). However, between atrial premature coupling intervals of 320-270 msec, an atrial premature complex was either conducted with an incomplete to complete right bundle branch block or a complete left bundle branch block pattern. When the right bundle branch block pattern appeared, it was always preceded by a His bundle deflection with an HV interval of 50-65 msec. The A1A2, H1H2 and A1A2, A2H2 curves were continuous. Nevertheless, when the complete left bundle branch block pattern appeared, His bundle deflection suddenly disappeared, with further prolongation of the AV interval. The A1A2, H1H2 and A1A2, A2H2 curves thus became abruptly disrupted. This was followed by a sustained reciprocating tachycardia with QRS complexes of a complete left bundle branch block pattern (the representative tracings corresponding to figure 5A are shown in figure 6A to D). We believed that intermittent failure of conduction by way of a fast AV nodal pathway oc-
**Figure 3.** Atrial premature stimulation with induction of sustained atrioventricular (AV) reciprocating tachycardia. S₁ and S₂ are pacing and premature stimuli. A and H are atrial and His bundle electrograms, respectively. The high right atrium (HRA) is driven at a cycle length (S₁S₂) of 700 msec. Panel A) An atrial premature beat at a coupling interval of 560 msec lengthens AV nodal conduction time (AH) from 110 to 140 msec. Panel B) An atrial premature beat at a coupling interval (S₁S₂) of 550 msec suddenly prolongs AV conduction time (AH) to 200 msec, resulting in discontinuous AV nodal conduction curves (A₁A₂, H₁H₂, fig. 2A). Moreover, this is followed by initiation of a sustained AV reciprocating tachycardia. Note that the sequence of retrograde atrial activation is first the left atrium (LA) followed by the low septal right atrium (in HBE) and then the high right atrium (HRA), indicating the use of a left-sided anomalous AV bypass tract for retrograde conduction during the reciprocating tachycardia. Panel C) Further shortening of the premature coupling interval (S₁S₂) to 330 msec (A₁A₂; 340 msec) results in progressive lengthening of AV nodal conduction time (AH) to 340 msec. In each instance, it is followed by the initiation of an AV reciprocating tachycardia using a left-sided anomalous AV bypass tract for retrograde conduction. Panel D) At the premature coupling interval (S₁S₂) of 320 msec (A₁A₂; 330 msec), the AV node becomes refractory. Thus, the antegrade effective refractory periods of the fast and slow AV nodal pathways are 550 and 330 msec, respectively, and the induction of sustained AV reciprocating tachycardia coincides with the slow AV nodal pathway conduction curve.
curred between atrial premature coupling intervals of 320–270 msec. This resulted in intermittent impulse transmission by way of a slow AV nodal pathway. The presence of an anomalous nodoventricular bypass tract resulted in the impulse transmission directly from the slow AV nodal pathway to the right ventricle producing right ventricular preexcitation (complete left bundle branch block pattern with a sudden disappearance of the His bundle deflection). Antegrade conduction through the slow AV node-

**FIGURE 5.** A) $A_1A_2, H_1H_2 (A_1A_2, V_1V_2)$ and $A_1A_2, A_2H_2 (A_1A_2, A_1V_2)$ curves during high right atrial (HRA) pacing at a cycle length (CL) of 600 msec. There is gradual prolongation of atrioventricular (AV) nodal conduction time ($A_2H_2$) as the atrial premature coupling interval ($A_1A_2$) progressively shortens, compatible with AV nodal properties. However, between premature coupling intervals ($A_1A_2$) of 320 and 270 msec, an atrial premature beat ($A_2$) is conducted to the ventricles with either an incomplete-to-complete right bundle branch block pattern (open square) or a complete left bundle branch pattern (closed square). Of note, when the incomplete-to-complete right bundle branch block pattern appears, it is always preceded by a His bundle potential with a HV interval of 50–65 msec; when the complete left bundle branch block pattern appears, the His bundle potential suddenly disappears and it is followed by initiation of a sustained reciprocating tachycardia (RT). B) $V_1V_2, V_2A_2$ and $V_1V_2, A_1A_2$ curves during right ventricular (RV) pacing at a CL of 600 msec. Note that the sequence of ventriculoatrial conduction is the slow septal right atrium (LRA) first followed by the HRA and then the left atrium (LA) compatible with retrograde conduction by way of the AV node–His-Purkinje system. Additionally, the progressive ventriculoatrial conduction delay as the ventricular premature coupling interval ($V_1V_2$) gradually shortens is in keeping with the retrograde conduction properties of the AV node–His-Purkinje system. Reciprocating tachycardia (RT) could be elicited between ventricular premature coupling intervals of 260 and 230 msec.
nodoventricular bypass tract pathway was followed by retrograde ventriculoatrial conduction by way of the fast AV node–His-Purkinje system, establishing the reciprocating tachycardia circuit (fig. 4).

The RV was also driven at a cycle length of 600 msec, and the V1V2, A1A2 and V1V2, V2A2 curves were compatible with retrograde AV node–His-Purkinje system conduction properties (fig. 5B). Between ventricular premature coupling intervals of 260–230 msec, progressive prolongation of ventriculoatrial conduction was followed by initiation of a sustained reciprocating tachycardia (the representative tracings
Figure 6. Atrial premature stimulation with intermittent induction of sustained reciprocating tachycardia. The high right atrium (HRA) is driven at a cycle length \((S_2S_4)\) of 600 msec. The atrioventricular (AV) nodal conduction time \((AH)\) lengthens progressively as the atrial premature coupling interval \((S_1S_2)\) gradually shortens (fig. 5A). Sustained reciprocating tachycardia could be intermittently induced between premature coupling intervals \((S_1S_4)\) of 320 and 270 msec. A) An atrial premature beat at a coupling interval \((S_1S_2)\) of 320 msec is conducted to the ventricles with an incomplete right bundle branch block pattern. The corresponding AH, HV intervals are 120 and 50 msec, respectively. B) An atrial premature beat at the same coupling interval \((S_1S_2)\) of 320 msec conducted to the ventricles with a complete left bundle branch block pattern followed by initiation of a sustained reciprocating tachycardia (arrow). Note sudden disappearance of the His bundle potential and prolongation of AV conduction time \((A_4V_4)\) before ventricular activation with a complete left bundle branch block pattern. C) An atrial premature beat at a coupling interval \((S_1S_2)\) of 270 msec is conducted to the ventricles with a complete right bundle branch block pattern with a superior axis. The corresponding AH, HV intervals are 130 and 65 msec, respectively. D) An atrial premature beat at the same coupling interval \((S_1S_2)\) of 270 msec is conducted to the ventricles with a complete left bundle branch block pattern similar to that seen in panel B. During reciprocating tachycardia (both panels B and D), the low septal right atrial electrogram cannot be clearly separated from the ventricular electrogram. However, the mid-right atrial (MRA) activation precedes left atrial (LA) activation, and the ventriculoatrial conduction time is constant after initiation of tachycardia.

corresponding to figure 5B are shown in figs. 7A–C). The reciprocating tachycardia had a complete left bundle branch block QRS morphology with a cycle length of 370 msec identical to that elicited during atrial extrastimulation. In this situation, we felt that the slow AV node–nodoventricular bypass tract pathway had a retrograde effective refractory period longer than that of the fast AV node–His–Purkinje system pathway. Retrograde ventriculoatrial conduction proceeded over the fast AV node–His–Purkinje system pathway during ventricular extrastimulation. At critical premature coupling intervals between 260–320 msec, a ventricular premature beat being blocked in the slow AV node–nodoventricular bypass tract pathway was conducted over the fast AV node–His–Purkinje system pathway in a retrograde direction, and in turn, activated the slow AV node–nodoventricular bypass tract pathway in an antegrade direction, establishing the reciprocating tachycardia circuit. In addition, similar to that described for AV nodal reentrant tachycardia and AV reciprocating tachycardia involving an anomalous AV bypass tract, a decrease in the ventricular pacing cycle length without ventricular extrastimulation could in itself trigger an onset of reciprocating tachycardia in this patient (fig. 7D).

Atrial flutter-fibrillation was deliberately induced using rapid atrial stimulation. When the QRS complex was of a complete right bundle branch block pattern, it was constantly preceded by a His bundle potential (figs. 8A–C). In contrast, when it was of a complete left bundle branch pattern, the His bundle potential suddenly disappeared. This observation further substantiated the theory of dual AV conduction pathways — the fast AV node–His–Purkinje system pathway that resulted in functional right bundle branch block and the slow AV node–nodoventricular bypass tract pathway that resulted in the complete left bundle branch block pattern (right ventricular preexcitation).

During reciprocating tachycardia, the QRS complexes were of the complete left bundle branch block pattern and they were characteristically not preceded by His bundle deflections, as the slow AV node–nodoventricular bypass tract pathway was used for antegrade conduction (figs. 6B and D, 7B–D). The atrium could be prematurely captured without disturbing the tachycardia, suggesting that the atrium was not a necessary link. At appropriately timed coupling intervals, the tachycardia could be terminated by an atrial premature beat without being conducted to the ventricles or by a ventricular premature beat without being conducted to the atria (figs. 9A–C). These findings suggested that the reentrant circuit was located between the atria and the ventricles.

One might argue that the site of reentry was within the AV node with ventricular preexcitation by way of the nodoventricular bypass tract. The following observations suggested that the right ventricle was a necessary link of the tachycardia circuit after antegrade slow AV nodal pathway conduction: 1) whenever the tachycardia was reset by an atrial or ventricular premature beat, it began with a constant ventriculoatrial response with constant ventriculoatrial conduction time (figs. 9A–C); and 2) constant ventriculoatrial response with constant ventriculoatrial conduction time was also consistently observed during the “warming-up” phase of the tachycardia (fig. 7D). Therefore, we concluded that the slow AV node–nodoventricular bypass tract was utilized as the antegrade conduction limb, and the fast AV node–His–Purkinje system or the distal common AV node–His–Purkinje system as the retrograde limb of the tachycardia circuit (fig. 4).

Discussion

Several investigators have suggested coexistence of dual AV nodal pathways in patients with anomalous bypass tracts, and have implicated that AV nodal reentry may cause tachyarrhythmias in these patients. Recent studies have indicated that differentiation between AV nodal reentrant tachycardia and AV reciprocating tachycardia involving an anomalous bypass tract requires characterization of
Figure 7. Induction of reciprocating tachycardia during programmed ventricular stimulation. From A to C, the right ventricle is driven at a cycle length ($S_1S_1$) of 600 msec. Ventricular premature beats ($S_2$) at coupling intervals of 300, 260 and 230 msec induce retrograde atrial activation with a gradual prolongation of ventriculoatrial conduction time ($S_2-LRA_2 = 150, 220$ and $250$ msec, respectively). In B and C, the ventricular premature beats ($S_2$) also elicit sustained reciprocating tachycardia (arrows). Retrograde His bundle potentials are recorded in B and C. Retrograde AV nodal conduction time ($H_2A_2$) is 45 msec. Note constant ventriculoatrial response with constant ventriculoatrial conduction time after initiation of tachycardia (arrow) despite variable RR intervals between the ventricular premature beat ($S_2$) and the first beat of reciprocating tachycardia. D. Rapid ventricular pacing at a cycle length ($S_1S_1$) of 400 msec can in itself initiate an onset of reciprocating tachycardia (arrow). Note constant ventriculoatrial response with constant ventriculoatrial conduction time (absence of AV dissociation) during the "warming-up" phase of reciprocating tachycardia.
the sequence of retrograde atrial activation as well as the functional properties of the conduction pathways which constitute the reentrant circuit. None of the 67 consecutive patients with anomalous bypass tracts in our study had a reentrant circuit confined within the AV node. Nevertheless, electrophysiologic findings suggestive of dual AV nodal pathway conduction could be identified during induction of reciprocating tachycardia in eight of these patients.

During programmed atrial extrastimulation in patients with anomalous bypass tracts, the assessment of AV nodal conduction properties is frequently handicapped by AV conduction over the anomalous bypass tract (ventricular preexcitation) obscuring the recording of the His bundle potential. Therefore, discontinuous AV nodal conduction curves (A1A2, H1H2 and A1A2, A3H3) characteristic of dual AV nodal pathway conduction may, thus, become unrecognized. In the present study, four patients had anomalous AV bypass tracts with relatively short antegrade refractory periods. The AV nodal conduction curves could not be precisely assessed. However, the AV reciprocating tachycardia manifested alternating short and long cycle lengths due to alternation of AV nodal conduction times (fig. 1A), suggesting alternate use of fast and slow AV nodal pathways in an antegrade direction. This observation was first reported by Friedberg and Schamroth and subsequently by Spurrell, Kirkler and Sowton. We assumed that there was 2:1 conduction block in the fast AV nodal pathway related to the critical cycle length of AV reciprocating tachycardia. Intravenous atropine, by improving conduction in the fast AV nodal pathway (shortening of its antegrade refractory period), resulted in 1:1 fast AV nodal pathway conduction, abolishing the alternating short and long cycle length phenomenon (fig. 1B).

Patients with concealed Wolff-Parkinson-White syndrome provide a unique situation for studying AV nodal conduction properties as the anomalous AV bypass tract is capable only of retrograde conduction. Farshidi et al. identified dual AV nodal pathway conduction in five of their 12 patients with concealed anomalous AV bypass tracts. We demonstrated in three patients that the induction of discontinuous AV nodal conduction curves (A1A2, H1H2) was followed by utilization of a left-sided anomalous AV bypass tract in a retrograde direction, establishing an AV reciprocating tachycardia circuit (figs. 2A and 3). The presence of a retrograde conducting anomalous AV bypass tract in each patient was supported by: 1) recording of abnormal sequence of retrograde atrial activation during tachycardia, in which the left atrium was activated before the low septal right atrium in the vicinity of the AV node and the high right atrium; and
Figure 9. Resetting and termination of reciprocating tachycardia by an atrial or ventricular premature beat. A) Atrial premature stimulation (St) at an atrial premature coupling interval of 290 msec resets the reciprocating tachycardia. B) During atrial driving at a cycle length (S1S2) of 600 msec, an atrial premature beat (coupling interval of 240 msec) elicits a reciprocating tachycardia with a cycle length of 370 msec. However, an atrial premature stimulation (St) at an atrial premature coupling interval of 270 msec that is not conducted to the ventricles terminates the tachycardia. C) During reciprocating tachycardia (cycle length 370 msec, a ventricular premature stimulation with a ventricular premature coupling interval of 300 msec resets the tachycardia. However, a ventricular premature stimulation (St) with a premature coupling interval of 230 msec without ventriculoatrial conduction terminates the tachycardia. In both panels A and C, note that the first beat of the tachycardia after resetting (the fifth and sixth QRS complexes, respectively, in panels A and C) has a constant ventriculoatrial conduction time.

2) demonstration of rather constant ventriculoatrial conduction time with relative lack of refractory-dependent conduction delay in the retrograde limb of the tachycardia circuit during programmed ventricular extrastimulation (fig 2B). These findings further stress the importance of recording the sequence of atrial activation along with the assessment of conduction pathway properties so that AV nodal reentry vs AV reciprocation can be differentiated.

Anatomically there are two varieties of anomalous nodoventricular bypass tracts — one emerging from the transitional cell zone and the other from the compact portion of the AV node to connect to the ventricles.21 The exact locations of the fast AV nodal pathway, the slow AV nodal pathway and the nodoventricular bypass tract in the case presented can only be speculative. The present study, however, has demonstrated electrophysiologic findings suggestive of...
two AV conduction pathways — the fast AV node-His-Purkinje system pathway and the slow AV node-nodoventricular bypass tract pathway: 1) During programmed atrial extrastimulation, there is abrupt (rather than gradual) disappearance of the His bundle potential followed by the development of ventricular preexcitation; in this situation the AV nodal conduction curves (A₁A₂, H₁H₂ and A₁A₃, A₃H₃) become suddenly disrupted reflecting antegrade failure of fast AV nodal pathway with resultant slow AV nodal pathway-nodoventricular bypass tract conduction (figs. 5A and 6A-D); and 2) during atrial flutter-fibrillation, intermittent antegrade slow AV node-nodoventricular bypass tract pathway conduction, in contrast to antegrade fast AV node-His-Purkinje system pathway conduction, is also characterized by abrupt disappearance of the His bundle potential with subsequent ventricular preexcitation (figs. 8A-C). Additionally, the presence of an anomalous AV bypass tract (Kent bundle) is excluded by the findings of normal sequence of retrograde atrial activation and absence of atrial preexcitation demonstrated during reciprocating tachycardia and during programmed ventricular extrastimulation (figs. 5B and 7A-D).

Although it has been suggested that an anomalous nodoventricular bypass tract may coexist with dual AV nodal pathways,14, 22, 23 participation of such an anomalous nodoventricular bypass tract in the genesis of reciprocating tachycardia is unusual, and conclusive evidence is lacking. Ward et al.14 reported that the tachycardia circuit in these patients is usually confined to the AV node (AV nodal reentry) with occurrence of ventricular preexcitation by way of the anomalous nodoventricular bypass tract. Although this possibility cannot be totally excluded, the case presentation in this study provided more electrophysiologic findings suggestive of the presence of dual AV nodal pathways and the involvement of a slow AV node-nodoventricular bypass tract pathway in the genesis of reciprocating tachycardia. These findings included:

1) The absence of ventricular fusion during sinus rhythm as evidenced by a normal PR interval (0.16 second) and a normal HV interval (50 msec). Ventricular fusion, in this situation, is determined by the conduction time of the fast AV node-His-Purkinje system pathway relative to that of the slow AV node-nodoventricular bypass tract pathway. If the former has a shorter conduction time than the latter, no ventricular fusion (or preexcitation) is expected to occur.

2) Induction of sudden disappearance of the His bundle potential along with right ventricular preexcitation by critically timed atrial premature beats (figs. 5, 6B and D). This observation suggested antegrade failure of a fast AV nodal pathway resulting in slow AV node-nodoventricular bypass tract pathway conduction, consistent with the theory postulated by Denes et al.1 atrial flutter-fibrillation which produced intermittent slow AV node-nodoventricular bypass tract pathway conduction further supported this observation (fig. 8A–C).

3) The first beat of reciprocating tachycardia elicited by either an atrial or ventricular premature beat had constant ventriculoatrial response with constant ventriculoatrial conduction time (figs. 6B and D and 7B and C); and

4) The absence of AV dissociation during the "warming-up" phase of reciprocating tachycardia as a result of constant ventriculoatrial response with constant ventriculoatrial conduction time (fig. 7D). These latter observations suggested that the right ventricle, which was activated following the slow AV nodal pathway conduction, was a necessary link of the reciprocating tachycardia circuit.

We have demonstrated the electrophysiologic manifestations of dual AV nodal pathway conduction in our patients with reciprocating tachycardia involving an anomalous bypass tract. These include: 1) alternation of tachycardia cycle lengths; 2) demonstration of discontinuous AV nodal conduction curves (A₁A₂, H₁H₂) when an anomalous AV bypass tract is capable only of retrograde conduction; and 3) disruption of AV nodal conduction curves (A₁A₂, H₁H₂), followed by ventricular preexcitation when a nodoventricular bypass tract connects a slow AV nodal pathway to the ventricles. Dual AV nodal pathways coexisting with an anomalous bypass tract may take several forms. A detailed electrophysiologic analysis is required to identify their coexistence.

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