Antegradé Slow Bypass Conduction After Closed-chest Ablation of the His Bundle in Permanent Junctional Reciprocating Tachycardia

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SUMMARY A case of permanent junctional reciprocating tachycardia in a 36-year-old woman successfully treated with closed-chest interruption of the His bundle is reported. Tachycardia had lasted for 14 years and showed a retrograde P wave (P') and RP' longer than PR' interval. The tachycardia used an anomalous pathway with a long conduction time in the retrograde direction. The atrial end of the anomalous pathway was located near the coronary sinus orifice. His ablation was accomplished by delivering a direct-current shock from a cardioversion unit to the nodal-His zone by means of a conventional electrode catheter percutaneously introduced via the femoral vein. Two shocks were necessary to obtain the desired results. After the procedure, complete atrioventricular block below the His bundle was induced, while antegrade conduction was assured through the anomalous pathway that showed decremental properties. During 7 months of follow-up, stable sinus rhythm with a long PR interval has been observed; the patient has remained free from tachycardia. Furthermore, she is not pacemaker-dependent and requires no cardioactive medication. This case demonstrates the therapeutic value of closed-chest ablation of the His bundle in a patient with permanent junctional reciprocating tachycardia, as well as demonstrating for the first time that the underlying accessory pathway is capable, in some instances, of antegrade conduction.

PERMANENT junctional reciprocating tachycardia (PJRT) is a rare supraventricular tachyarrhythmia that affects young people and is usually refractory to pharmacologic treatment. This arrhythmia can be characterized as being almost incessant, starting after a few sinus beats, without any prolongation of the PR interval. During tachycardia, the P wave (P') is negative in leads II, III, aVF, and the RP' is longer than the P'R interval. An unusual (i.e., fast-slow) variety of intranodal reentry^4,5 or reentry using an accessory pathway with a long conduction time^5 are the possible underlying substrates. We report a case of PJRT in a woman who was successfully treated with closed-chest interruption of the His bundle.

Case Report

The patient was a 36-year-old woman who had suffered for 14 years from PJRT. Pharmacologic therapy (quinidine, propranolol and amiodarone) were ineffective. During tachycardia, the heart rate was 130–180 beats/min and the surface ECG showed a retrograde P wave (P') and a P'R interval shorter than RP' interval (fig. 1). Sinus rhythm beats showed a normal PR interval and no evidence of a delta wave. Neither critical cycle length nor critical atrioventricular (AV) delay was found at tachycardia initiation (fig. 2).

During electrophysiologic investigation, antegrade and retrograde conduction curves could not be analyzed because of the presence of incessant tachycardia. Endocardial mapping of the right atrium and coronary sinus during tachycardia showed a septal sequence of retrograde atrial activation, the earliest retrograde electrogram being recorded at the coronary sinus orifice (fig. 3). Premature ventricular stimulation at coupling intervals of 390–325 msec interrupted tachycardia without resulting in atrial retrograde activation (fig. 3); atrial preexcitation was not observed, but the presence of an accessory ventriculoatrial pathway was suggested by the ability of premature ventricular depolarizations to interrupt tachycardia without propagating to the His bundle or atrium.

 Interruption of the His bundle was accomplished according to a previously described method. This technique consists of delivery of a direct-current shock from a cardioversion unit to the nodal-His zone by means of a conventional electrode catheter percutaneously passed via the femoral vein, as described by others.

 Two shocks were required. The first shock (200 J) resulted in disappearance of tachycardia for about 3 weeks. During that period, the surface ECG showed sinus rhythm with first-degree AV block (PR 360 msec). An electrophysiologic study performed 20 days after the first shock initially showed the absence of antegrade His activation. During the study, an antegrade Wenckebach phenomenon was observed, followed by a sudden change in the AV conduction pattern, characterized by evidence of His bundle activation with normal AV conduction intervals (fig. 4). A similar event occurred during atrial pacing at a cycle length of 560 msec. Three days later, tachycardia recurred.

 A second shock (280 J) resulted in first-degree AV block (PR 360 msec), the AH and HV intervals measuring 90 and 250 msec, respectively. We believe that antegrade block was present distal to the recorded His deflection; thus, all apparent HV intervals are spurious. An endocavitary restudy performed 30 days later.

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revealed a similar AV conduction pattern. Wenckebach AV block spontaneously appeared and 2:1 conduction occurred either after atropine administration or during atrial pacing at a cycle length of 580 msec. However, there was no evidence of the change in antegrade conduction pattern that had been observed during the previous study under the same conditions.

A third electrophysiologic study, performed 2 months after the second shock, revealed the same pattern of AV conduction during sinus rhythm: Atrial pacing at a cycle length of 430 msec resulted in 2:1 AV block with an AH interval of 95 msec and an HV interval of 150–230 msec (fig. 5). Ventricular pacing at a cycle length of 480 msec showed the earliest atrial retrograde activation at the coronary sinus orifice, without retrograde activation of the His bundle; a retrograde Wenckebach phenomenon, followed by 2:1 ventriculoatrial conduction, appeared at a cycle length of 380 msec (fig. 6). Since the second shock, the patient has been free from tachycardia. The follow-up is 7 months; during this period, the surface ECG has shown stable sinus rhythm, with a long PR interval and a QRS pattern of posterior septal preexcitation (fig. 7). The patient is not pacemaker-dependent; she is being followed regularly by a transtelephonic monitoring system.

Discussion

Since 1967, a few examples of PJRT have been reported. 1-9 Coumel 1 ascribed the mechanism of the tachycardia to the presence of longitudinal dissociation in the AV node as well as a part or in the whole of the His bundle. Subsequently, some clinical cases have been shown to result from intranodal reentry of the fast-slow variety, 6-8 but the possibility that the underlying mechanism of this tachycardia may be a reentry that uses an accessory pathway has been documented as well. 2,3,7,9 The findings in our case are most consistent with utilization of an accessory pathway with decremental behavior in the posterior septal region of the heart. Thus, premature ventricular complexes introduced during tachycardia at intervals that did not result in retrograde depolarization of the His bundle terminated PJRT without an atrial response; furthermore, earliest retrograde atrial activity during PJRT was lo-

**FIGURE 1.** ECG during sinus rhythm (top) and during tachycardia (bottom). Sinus rhythm beats show a normal PR interval and no evidence of a delta wave. During tachycardia the P wave is negative in leads 2, 3 and aVF and the RP interval is longer than the PR interval.

**FIGURE 2.** Reciprocating tachycardia showing a PR interval shorter than the RP interval. Neither a critical PP cycle nor a critical atrioventricular delay is evident at tachycardia initiation. Numbers indicate milliseconds.
Figure 3. Endocardial mapping of the right atrium and coronary sinus during reciprocating tachycardia (first two beats) and its interruption by premature ventricular stimulation. The atrial retrograde depolarization with the earliest activation occurring on the proximal coronary sinus (PCS) indicates that the accessory pathway has its atrial end near the coronary sinus orifice. Premature ventricular stimulation interrupts the tachycardia circuit in the retrograde limb, since no retrograde atrial activation occurs. The His deflection was undisturbed. The fourth beat is a sinus rhythm beat. HB = His bundle electrogram; RA = right atrial electrogram; DCS = distal coronary sinus electrogram; PCS = proximal coronary sinus electrogram. Numbers indicate milliseconds.

Figure 4. Surface ECG (L1, L3, V1) and endocavitary recordings obtained 20 days after the first shock. Sinus rhythm with Wenckebach-type block and no antegrade Hisian potential are present (first five complexes). At the end of the Wenckebach phenomenon, a change in the atrioventricular conduction pattern appears, with normal intervals and right bundle branch block. See text for further details. HBE = His bundle electrogram; RA = right atrial electrogram; DCS = distal coronary sinus electrogram; PCS = proximal coronary sinus electrogram. Numbers indicate milliseconds.

Figure 5. Surface ECG (L1, L3, V1) and endocavitary recordings after the second shock. During sinus rhythm, complete block below the His bundle is present, ventricular activation being assured by the anomalous pathway with a long atrioventricular conduction time. Atrial pacing at a cycle length of 430 msec induces 2:1 block in the anomalous pathway. See text for further explanations. HBE = His bundle electrogram; RA = right atrial electrogram; DCS = distal coronary sinus electrogram; PCS = proximal coronary sinus electrogram. Numbers indicate milliseconds.
calized to the orifice of the coronary sinus after a long ventriculoatrial interval — features consistent with our previous reports.\(^1\)\(^,\)\(^3\) The AV conduction pattern after the shocks also supports such interpretation. After the first shock, in the presence of a His bundle reversibly damaged, a sudden change from an abnormal (long PR) to a normal (normal PR) AV conduction pattern was observed at the end of Wenckebach phenomenon. In fact, no antegrade activation of the His bundle occurred during conduction over the anomalous pathway with Wenckebach phenomenon (fig. 4); at that time antegrade conduction over the AV node–His bundle was probably inhibited by the occurrence of concealed retrograde conduction. At the end of the Wenckebach cycle, conduction in the AV node–His bundle resumes, so the His deflection precedes normal QRS complexes with an HV interval of 50 msec. This situation is analogous to our findings during retrograde conduction studies in another patient with PJRT. We reported\(^2\) retrograde Wenckebach over the anomalous pathway during ventricular pacing, with sudden appearance of 1:1 retrograde conduction over the normal conduction system at shorter intervals, due to disappearance of antegrade concealed conduction after the blocked beat of the Wenckebach period.

After the second shock, when the nodal–His conduction was irreversibly damaged, antegrade conduction only occurs through the anomalous pathway, in the presence of either spontaneous Wenckebach-type block or 2:1 AV block induced by atrial pacing (fig. 5). According to this interpretation, the His bundle deflection results from antegrade conduction over the normal AV tissue, which is interrupted below the His bundle. Thus, the apparent HV interval is seen to be extremely long and variable; furthermore, numerous attempts to pace the His bundle region failed to alter atrial or ventricular activity. Ventricular activation occurs over the anomalous pathway, which now conducts in the antegrade direction with decremental properties. We believe that this is the first demonstration of the capability of such an anomalous pathway to conduct in the antegrade direction. Actually, after the second shock,
the surface ECG shows that the QRS forces are directed superiorly (negative in L₂, L₃) and are isoelectric in V₁; this corresponds to the pattern of a posterior septal accessory pathway, in agreement with the location predicted from the study of the retrograde atrial activation sequence. After the second ablation, the anomalous pathway is the only one capable of retrograde conduction (fig. 6). During ventricular pacing the earliest retrograde atrial depolarization occurs near the coronary sinus orifice and no retrograde activation of the His bundle appears. In fact, the His deflection results from antegrade conduction over the normal AV tissue with infra-Hisian complete block. Figure 6 also demonstrates the decremental properties of this kind of accessory pathway, as shown by retrograde Wenckebach phenomenon and 2:1 conduction in the course of ventricular pacing. According to previously reported data,²-⁴,¹⁷ such an accessory pathway has its atrial insertion close to the coronary sinus orifice.

Our case prompts speculation about what causes tachycardia initiation in PJRT. In the presence of a concealed anomalous pathway, if tachycardia started after a critical shortening of the PP cycle, it would be reasonable to believe that, as a consequence of reduction in atrial refractoriness, the first retrograde activation could take place, thereby initiating tachycardia. Another initiating mechanism could be the occurrence of intraventricular delay on the side ipsilateral to the concealed accessory pathway.²⁰,²¹ In our case, no change in PP cycle and no ventricular aberration was evident at the onset of tachycardia.

Thus, tachycardia initiation in this patient could be ascribed to a spontaneous change in atrial refractoriness, a critical change in the retrograde conduction of the pathway or due to block of concealed (antegrade or retrograde) conduction into the region of the accessory pathway.

Decremental conduction in accessory pathways has been reported,¹⁹,²⁰ although the magnitude of decrement observed in cases of PJRT is considerably greater than that encountered with typical accessory AV fibers of the Kent bundle variety.

In cases in which tachycardia is resistant to pharmacologic and electrical treatment, closed-chest interruption of the His bundle may represent a useful therapeutic means. In our patient, antegrade conduction was assured by an anomalous pathway with nodal-like properties such that the patient is not pacemaker dependent. She also does not have the theoretical disadvantages related to the presence of an accessory pathway in the absence of the nodal-His conduction. Careful follow-up is being performed to observe possible block of conduction in the anomalous pathway, which could result in complete AV block.

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