Electrocardiographic Diagnosis of Dual AV Nodal Pathways Complicating the Wolff-Parkinson-White Syndrome

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The diagnosis of dual AV nodal pathways complicating concealed or manifest preexcitation, is usually made at the time of electrophysiologic study in patients with recurrent paroxysmal supraventricular tachycardia. In these patients, electrophysiologic studies demonstrate discontinuous antegraded AV nodal conduction curves (dual AV nodal pathways, fast and slow), as well as a unidirectionally conducting anomalous pathway. In some of these patients, one or more tachycardias are induced. The tachycardias induced can include a fast AV reentrant tachycardia (antegrade fast pathway and retrograde anomalous pathway), a slow AV reentrant tachycardia (antegrade slow and retrograde anomalous pathway), and the usual variety of AV nodal reentrant paroxysmal tachycardia (antegrade slow pathway and retrograde fast pathway). In these cases, one then has to retrospectively examine electrocardiograms in an attempt to correlate induced and clinical tachycardias.

In the present study, we report a patient with known Wolff-Parkinson-White syndrome, in whom dual pathways were diagnosed from electrocardiographic findings prior to electrophysiologic study. In this patient, the presence of dual pathways was clinically relevant, in that the patient suffered from two different spontaneous paroxysmal tachycardias.

Case Report

The patient was a 39-year-old white man with known preexcitation, referred to the University of Illinois for evaluation of recurrent paroxysmal palpitation. Other than the history of palpitation, there was no history suggesting cardiovascular disease. Physical examination, as well as extensive noninvasive work-up, revealed no complicating organic heart disease.

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Electrocardiograms

Electrocardiograms during sinus rhythm revealed intermittent typical preexcitation. Preexcited QRS complexes were characterized by negatively directed delta waves in leads 2, 3, and aVF, and a positively directed delta wave in lead V₁ (Fig 1). The QRS morphology was suggestive of a left posterior or left posteroseptal anomalous pathway.

Immediately following a graded treadmill test, the patient developed spontaneous paroxysmal supraventricular tachycardias. These tachycardias were characterized by narrow QRS, and an easily visible retrograde P wave with an RP interval of approximately 0.12 second (Fig 2). Two distinct tachycardias could be noted, one with a heart rate of approximately 160 to 170 beats/min and the second with a heart rate of approximately 120 to 130 beats/min (Fig 2 and 3). Spontaneous conversion from fast to slow and slow to fast tachycardia was noted. These conversions were characterized by an abrupt change in cycle length, reflecting an abrupt change in PR interval without any change in RP interval (Fig 3).

Scrutiny of these electrocardiograms suggested a diagnosis of dual antegrade AV nodal pathways in a patient with preexcitation, with a Kent bundle capable of both antegrade and retrograde conduction. The tachycardias were felt to be orthodromic, with antegrade conduction via the AV node and His bundle, and retrograde conduction via the anomalous pathway. The two cycle lengths were explained by postulating the existence of dual AV nodal pathways, with the faster tachycardia reflecting antegrade conduction via the fast AV nodal pathway, and the slow tachycardia reflecting antegrade conduction via the slow AV nodal pathway.

Electrophysiologic Study

Electrophysiologic studies were performed using standard techniques. The patient had a left posterior anomalous pathway, with poor capability for antegrade conduction and excellent capacity for retrograde conduction (intact anomalous pathway conduction up to ventricular paced rates of 210 beats/min). Electrophysiologic study of AV nodal properties revealed dual AV nodal pathways. The findings most germane to the tachycardias demonstrated after treadmill testing were those obtained during isoproterenol infusion (2 μg/min). Atrial extrastimulus testing to a sinus cycle length of 600 msec revealed discontinuous AV nodal conduction curves (A₁, A₂, H₁, H₂), diagnostic of dual antegrade AV nodal pathways. The fast pathway...
effective refractory period was 380 msec and the slow pathway effective refractory period was less than 220 msec. Fast pathway AH intervals ranged from 100 to 140 msec and slow pathway AH intervals ranged from 180 to 240 msec. Two AV reentrant tachycardias were induced, one characterized by fast pathway antegrade conduction and retrograde anomalous pathway conduction (cycle length of 320 to 350 msec), and the other characterized by antegrade slow pathway and retrograde anomalous pathway conduction (cycle length of 470 msec).

**DISCUSSION**

In the present case, the diagnosis of electrophysiologic mechanism of tachycardias was made with relative certainty prior to the performance of the actual electrophysiologic study. The patient had known preexcitation, and a history of paroxysmal palpitation, without previously documented paroxysmal tachycardias. Following a graded treadmill test, the patient developed spontaneous paroxysmal tachycardias. This development of tachycardias during or post treadmill test is an unusual event in patients with the Wolff-Parkinson-White syndrome.

Scrutiny of the induced tachycardias revealed narrow QRS complexes, implicating the normal AV pathway as the antegrade limb of his circus movement. In addition, there was an easily visualized retrograde P wave following QRS complexes of the tachycardias, with an RP interval of approximately 120 msec. In patients without the Wolff-Parkinson-White syndrome, this location of retrograde P waves suggests the utilization of an anomalous pathway as a retrograde limb of the circus movement.

Thus, the antegrade limb of the circus movement was established as the normal pathway and retrograde limb as almost certainly being anomalous.

**Figure 2.** Electrocardiogram demonstrating "slow" A-V reentrant paroxysmal tachycardia. Note easily visible retrograde P wave following narrow QRS complexes (see text for discussion).

**Figure 3.** Abrupt transition from fast to slow A-V reentrant paroxysmal tachycardia. Note fixed V-A interval, with change in cycle length of tachycardia reflecting sudden change in PR.
pathway. One then had to explain the changes in cycle length of tachycardia. Specifically, two paroxysmal tachycardias were seen. These were identical in regard to QRS morphology, retrograde P wave morphology, and ventriculo-atrial conduction time (RP). These tachycardias were thus characterized by either a fast antegrade conduction time (shorter PR interval), or a slow antegrade conduction time (longer PR interval). The change from fast to slow tachycardia and from slow to fast tachycardia was abrupt. The most reasonable explanation for the above events was the postulation of dual antegrade AV nodal pathways, with both pathways being suitable participants for sustained conduction during circus movement tachycardia. The abrupt change in cycle length would reflect sudden failure of the fast AV nodal pathway, with subsequent slow pathway conduction (change from fast to slow tachycardia), or abrupt facilitation of fast pathway conduction during slow pathway tachycardia, producing the faster tachycardia. Slight variations in autonomic tone probably produced changes in refractoriness of the fast pathway, accounting for its intermittent functioning.

The postulated mechanisms of paroxysmal tachycardia in this preexcitation patient were verified by electrophysiologic testing. As expected, AV reentrant tachycardias were inducible in the catheterization laboratory (we have previously reported a close relationship between electrophysiologically induced and clinically occurring paroxysmal tachycardias). Dual antegrade AV nodal pathways were identified, both having ability to sustain paroxysmal tachycardia, as was suspected clinically. During electrophysiologic study, the antegrade limb of tachycardia was noted to be either fast or slow pathway as postulated above, with the anomalous pathway being utilized as a retrograde limb.

Clinical Implications

The diagnosis of dual AV nodal pathways in the present case has clinical implications. To achieve cure of paroxysmal tachycardias in these patients, one should be cognizant of the two tachycardias seen. Effective prophylaxis for tachycardias in the present patient could consist of achieving pharmacologic depression of retrograde anomalous pathway function, so that this pathway became incapable of the sequential retrograde conduction necessary for circus movements to exist. This task is somewhat complicated by the presence of dual AV nodal pathways, since the retrograde anomalous pathway is not under much stress during the slow tachycardia, because of the relative long cycle length of this rhythm. To eliminate the slow tachycardia utilizing pharmacologic depression of retrograde anomalous pathway function, this depression would have to be of significant magnitude (this might or might not be achievable with drugs such as procainamide, quinidine, or disopyramide phosphate).

Another approach to prophylaxis of tachycardias in this patient could be to attempt to achieve pharmacologic depression of both fast and slow antegrade AV nodal pathways, so that neither could function as the antegrade limb for circus movement tachycardias. Currently available oral agents that depress antegrade fast and slow pathway conduction include cardiac glycosides, propranolol, disopyramide phosphate, and occasionally disopyramide phosphate. A readily available means for assessing drug effectiveness prior to initiation of chronic prophylactic therapy, would be the use of serial studies with programmed stimulation.

During electrophysiologic study in this patient, one would have to be cognizant of the possibility that a third tachycardia could occur, this being the usual variety of AV nodal reentrance, which consists of antegrade slow and retrograde fast pathway conduction. This additional variety of reentrance should be looked for during electrophysiologic study (it was looked for and not found).

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