Antidromic Tachycardia Utilizing Decremental, Latent Accessory Atrioventricular Fibers: Differentiation From Adenosine-Sensitive Ventricular Tachycardia

JEFFREY J. GOLDBERGER, MD, FACC, DAVID N. PEDERSON, MD,* ROGER S. DAMLE, MD, YOU-HO KIM, MD, ALAN H. KADISH, MD, FACC
Chicago, Illinois and San Antonio, Texas

Objectives. We studied two patients with latent, decremental atrioventricular (AV) fibers in whom pre-excitation could be demonstrated only during wide complex tachycardia.

Background. The presence of decremental AV fibers participating in antidromic AV reentrant tachycardia is usually suspected by the presence of pre-excitation either in sinus rhythm or during atrial pacing.

Methods. Two patients were referred for evaluation and treatment of wide complex tachycardia whose configuration suggested ventricular tachycardia that could be terminated with adenosine infusion. They underwent standard electrophysiologic studies.

Results. Baseline AH and HV intervals were normal. No pre-excitation was noted with atrial overdrive at multiple sites or during atrial extrastimulation. Retrograde conduction was present with a sequence compatible with AV node conduction. Sustained wide complex tachycardia was induced with ventricular overdrive pacing. Late atrial premature depolarizations during tachycardia pre-excited the subsequent ventricular activation. Earlier atrial premature depolarizations delayed the subsequent ventricular activation. In one patient, early atrial premature depolarizations terminated the tachycardia without activating the ventricle. In the other patient, spontaneous tachycardia termination was accompanied by ventriculoatrial block. The earliest ventricular activation was at the annulus in the posteroseptal region in one patient and at the left posterior region in the other. Atrioventricular node reentry and atrial tachycardia with bystander AV fibers were also excluded. These findings establish the diagnosis of antidromic AV reentrant tachycardia utilizing a slow, decrementally conducting AV pathway.

Conclusions. This is the first report describing the presence of latent, decremental accessory AV pathways in which conduction was manifest only during antidromic AV reentrant tachycardia. To differentiate these wide complex tachycardias from adenosine-sensitive ventricular tachycardia, we recommend that atrial premature depolarizations be applied during tachycardia to rule out the presence of a latent, decremental AV fiber even in patients who do not otherwise have pre-excitation with atrial pacing techniques.

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Antidromic atrioventricular (AV) reentrant tachycardia may occur in patients with AV fibers. Because ventricular activation during antidromic AV reentrant tachycardia occurs over an accessory AV pathway, the electrocardiographic (ECG) configuration of these tachycardias resembles that of ventricular tachycardia. However, the diagnosis of antidromic AV reentrant tachycardia is usually suspected by the presence of pre-excitation either in sinus rhythm or during atrial pacing. We report two cases in which a clinical diagnosis of adenosine-sensitive ventricular tachycardia was made. In addition, during electrophysiologic studies, no pre-excitation could be demonstrated in sinus rhythm or during atrial pacing or adenosine infusion. Nevertheless, the tachycardias were demonstrated to be antidromic AV reentrant tachycardia. The diagnostic and therapeutic implications of these unusual cases is discussed.

Methods

Patient characteristics. Two patients referred for evaluation and treatment of wide complex tachycardia (Fig. 1 and 2) are the subject of this report. Their clinical characteristics are displayed in Table 1. Neither subject had structural heart disease. On the basis of the configuration of the tachycardia (1), the presence of a narrow QRS complex during sinus rhythm and termination of clinical episodes of tachycardia with intravenous adenosine, a clinical diagnosis of adenosine-sensitive ventricular tachycardia was made. The patients were referred for electrophysiologic studies and catheter ablation.

Electrophysiologic study. After written informed consent was obtained, an electrophysiologic study was done in the fasting, mildly sedated state. Quadripolar catheters were positioned in the right atrium, His bundle region and right ventricular apex through sheaths inserted percutaneously in the femoral vein. A multipolar electrode catheter was also
positioned in the coronary sinus through a sheath placed either in the left subclavian vein (Patient 1) or in the femoral vein (Patient 2). A large-tipped steerable catheter (Mansfield-Webster) was also used for left ventricular pacing and mapping in Patient 2 and was positioned using the retrograde approach across the aortic valve through a sheath placed in the femoral artery. Multiple ECG leads, as well as the intracardiac electrograms, were displayed on a multichannel oscilloscope. Electrograms were bandpass filtered from 40 to 400 Hz. Recordings were obtained at a paper speed of 100 mm/s. Incremental atrial and ventricular pacing were performed to assess anterograde (AV) and retrograde (ventriculoatrial [VA]) conduction properties, respectively. Pacing was performed at twice the diastolic threshold, with a stimulus duration of 2 ms. Anterograde and retrograde refractory periods were assessed using the extrastimulus technique, after an 8-beat drive train. Sustained wide complex tachycardia, reproducing the clinical tachycardia, was induced in both patients using the techniques described earlier.

The diagnosis of antidromic AV reentrant tachycardia was confirmed by the response of the tachycardia to the application of single atrial premature depolarizations (see Results). Orthodromic supraventricular tachycardia was ruled out by the absence of a His bundle deflection before ventricular activation, the appearance of a retrograde His bundle activation during the QRS complex or earliest ventricular activation occurring at the base of the heart. Ventricular tachycardia was ruled out by the response of the tachycardia to single atrial premature depolarizations. Atrial tachycardia and AV junctional reentrant tachycardia with an innocent bystander AV pathway were also ruled out (see Results).

Evaluation of potential anterograde conduction over an accessory AV pathway was evaluated with incremental right and left atrial pacing, atrial extrastimuli from the right and left atrium and infusion of adenosine. In Patient 1, atrial stimulation was also performed in the low posterior right atrium, near the coronary sinus, at the presumed site of the accessory pathway. Retrograde conduction was evaluated in both patients with incremental right ventricular pacing as well as application of premature ventricular depolarizations from the right ventricle. In addition, ventricular stimulation near the presumed ventricular insertion site was performed in each patient. Decremental conduction was defined as delay in conduction at more rapid rates or with increasing prematurity, regardless of the underlying mechanism for the delay (2).

Because of the diagnostic complexities and patient discomfort, only limited attempts at catheter ablation were made at the initial diagnostic study. Both patients subsequently underwent a second procedure for catheter ablation of the AV pathway. Because there was no evident anterograde or retrograde conduction in the accessory AV pathway except during antidromic AV reentrant tachycardia, mapping was performed during tachycardia. The earliest ventricular activation relative to the onset of the QRS complex was used to guide catheter
ablation. At each site, radiofrequency energy was delivered as a continuous, unmodulated sine wave output from an electrical surgical unit at 350 kHz (Radionics RFG-3C lesion generator). Energy was applied for 15 to 20 s at 20 to 30 W. The end point of catheter ablation was the inability to induce lachycardia. If tachycardia was not inducible at baseline, isoproterenol was infused, and repeat attempts at induction of tachycardia were made.

**Results**

**Baseline electrophysiologic data.** In both patients, the baseline rhythm was sinus, with normal conduction intervals and no pre-excitation (Table 1). During incremental right and left atrial pacing, there was evidence of decremental AV node conduction. The AH interval prolonged from 60 and 65 ms at baseline to 130 and 120 ms during overdrive atrial stimulation in Patients 1 and 2, respectively. The HV interval remained constant. The AV node Wenckebach cycle lengths were 340 and 245 ms. Application of atrial extrastimuli also resulted in progressive increase of the AH interval, with no change in the HV interval and no evidence of pre-excitation. The AV node effective refractory periods were 220 ms and <210 ms (at a drive cycle length of 40 ms). Typical recordings during atrial stimulation are displayed in Figure 3. During ventricular pacing there was one-to-one VA conduction. The earliest retrograde atrial activation was in the His bundle electrogram, consistent with retrograde AV node conduction; there was no change in retrograde activation sequence between pacing and tachycardia. There was no His bundle activation preceding the ventricular activation. On the basis of the QRS pattern during tachycardia, no evidence of anterograde His-Purkinje conduction during tachycardia and lack of any pre-excitation during atrial pacing, these tachycardias could be diagnosed as ventricular tachycardia.

Atrial premature depolarizations were applied during both tachycardias. Figures 5 and 6 demonstrate the results of this maneuver. In Figure 5A and B, an atrial premature depolarization was applied from the low posterior right atrium in Patient 1 and from the left atrium (catheter in the coronary sinus) in Patient 2. In Figure 5B, the stimulus artifact is seen to occur after the onset of the atrial electrogram in the His.
consistent with retrograde activation through the atrioventricular node. Tachycardia cycle length ~ 4211 ms. Other abbreviations as in Figure 3.

Figure 4. Surface electrocardiographic leads I, II, and V1 and intracardiac electrograms from the high right atrium, distal and proximal His bundle electrograms, proximal and distal coronary sinus electrograms (CSp and Csd, respectively) and right ventricular apex (RVA) during ventricular overdrive stimulation (S) at 370 ms, which induced wide complex tachycardia in Patient 1. Earliest atrial activation is indicated by the arrow and occurs in the His bundle electrogram, consistent with retrograde activation through the atrioventricular node. Tachycardia cycle length = 420 ms. Other abbreviations as in Figure 3.

bundle recording suggesting that the AV node is refractory. Of note, the atrial premature depolarizations resulted in pre-excitation of the following ventricular depolarization by 20 to 25 ms. Because the atrial premature depolarizations advance the QRS complex when the AV node is refractory, there must be an accessory AV connection. Atrial premature depolarizations were also able to delay the subsequent QRS complex in both cases. Figure 6 demonstrates an atrial premature depolarization that resulted in delay of the following QRS complex by 30 ms. This finding establishes that the accessory AV pathways have decremental properties. The ability of atrial premature depolarizations to advance (particularly when the His bundle is refractory) and delay the QRS complex without a change in configuration excludes ventricular tachycardia. Figure 7A demonstrates termination of the second patient's tachycardia by an even earlier atrial premature depolarization. This finding was reproduced several times. Termination of the tachycardia without conduction to the ventricles also rules out ventricular tachycardia as the mechanism of the tachycardia. In addition, in Patient 1, spontaneous tachycardia termination reproducibly occurred simultaneously with VA block (Figure 7B), also mitigating against ventricular tachycardia. Finally, atrial pacing during tachycardia entrained the tachycardia without a change in the QRS configuration. During tachycardia, atrial pacing produced conduction block in the accessory pathway at a cycle length of 330 ms in Patient 1 and 340 ms in Patient 2.

At Patient 1 had evidence of dual AV node physiology, whereas Patient 2 did not. Atrioventricular junctional reentrant tachycardia utilizing an innocent bystander bypass tract was ruled out by the fact that the tachycardia could be reset by atrial premature depolarizations with an identical retrograde atrial activation sequence and VA time as during the tachycardia. This finding suggests that retrograde activation is part of the reentrant pathway. Additionally, in Patient 2, catheter manipulation in the left ventricle resulted in transient left bundle branch block. During the presence of left bundle branch block there was a slowing of the tachycardia by 30 ms, also suggesting that this was an AV nodal reentrant tachycardia utilizing a left-sided accessory pathway. Because of the presence of dual AV node physiology in Patient 1, the possibility of inducible AV nodal reentrant tachycardia also was evaluated after isoproterenol infusion. The same wide complex tachycardia was induced and again AV nodal reentrant tachycardia was ruled out, as seen earlier. Of note, the tachycardia cycle length shortened to 300 ms and the AV interval shortened by 30 ms. Atrial tachycardia with an innocent bystander bypass tract...
is unlikely because no pre-excitation could be elicited during atrial pacing, and the VA time after atrial overdrive stimulation was fixed (4).

Once the diagnosis of antidromic AV reentrant tachycardia was established, further attempts were made to characterize the accessory pathway. The ability of late atrial premature depolarizations to pre-excite the tachycardia suggests an atrial insertion of the pathway (rather than AV node). The ventricular insertion was identified by mapping ventricular activation during tachycardia. In the first patient, the earliest ventricular activation was identified 20 ms before the QRS onset in the right posteroseptal area at the tricuspid annulus. In the second patient, the earliest ventricular activation was identified 20 ms before the QRS onset at the left posterior mitral annulus, ~2 cm from the coronary sinus os. Mapping of ventricular activation at sites away from the AV rings demonstrated later activation times than the earliest sites on the annulus. The ECGs during tachycardia (Figs. 1, 2) that exhibit maximal pre-excitation are consistent with a ventricular insertion near the annulus. Functionally, anterograde conduction was slow and decremental, as demonstrated by the delay of ventricular activation with early atrial premature depolarizations. In addition, in Patient 1, when the tachycardia was slowed with a shift of the retrograde pathway from fast to slow AV node conduction (VA time 135 vs. 195 ms), the interval from earliest atrial activation to QRS onset shortened from 245 to 215 ms. Retrograde conduction over these fibers could not be demonstrated, even with stimulation near the ventricular insertion. A summary of the electrophysiologic data during tachycardia is displayed in Table 1. In the first patient, attempts at catheter ablation in the posteroseptal area (predominantly from the atrial aspect of the annulus) were not successful in eliminating tachycardia. She has been treated with acebutolol without recurrence (13-month follow-up). Catheter ablation was successful in the second patient, with application of 16 lesions for a total duration of 420 s at 20 to 30 W, using a retrograde transaortic approach. No tachycardia could be induced after ablation, even after the administration of isoproterenol.

To explain the lack of pre-excitation with atrial pacing near the presumed insertion site of the accessory pathway, the AV times were compared during pacing from that site at approximately the tachycardia cycle length and during tachycardia. In both patients, the AV times during atrial pacing at approximately the tachycardia cycle lengths, when conduction proceeded through the AV node, were 140 ms. During tachycardia, when conduction proceeded through the accessory AV fiber, the AV times were 245 and 230 ms in Patients 1 and 2, respectively. Thus, at approximately the tachycardia cycle length, AV node conduction was 90 to 105 ms more rapid than conduction through the accessory AV fiber.
Discussion

We believe that this is the first report of decremental, latent accessory AV pathways in which conduction was manifest only during antidromic AV reentrant tachycardia. These fibers were shown to be slowly and decrementally conducting AV accessory pathways. Multiple attempts to demonstrate preexcitation with atrial pacing and adenosine infusion were unsuccessful, rendering these pathways difficult to diagnose with routine electrophysiologic maneuvers. This finding has important implications because these tachycardias could be easily misdiagnosed by both clinical and electrophysiologic criteria as ventricular tachycardia. Our findings suggest that patients with wide complex tachycardia and one-to-one VA conduction who have a clinical and ECG pattern consistent with antidromic tachycardia need to have this diagnosis excluded using atrial pacing maneuvers during tachycardia. The absence of preexcitation in sinus rhythm, during atrial pacing or after adenosine infusion is inadequate to rule out the presence of these atypical AV pathways. The true incidence of these types of fibers is not known as to date, their presence has not been studied systematically.

Decremental, accessory AV fibers. Although the term Mahaim fiber has been used to describe a variety of different types of accessory pathways with similar physiology (5–8), the rapid changes in our understanding of the anatomic and functional characteristics of these fibers (9) has led to the realization that our terminology is confusing and may be misleading. After the initial description by Mahaim et al. (10) of accessory fibers originating in the AV node region, it was assumed that accessory pathways with nodelike properties were all nodoventricular fibers. It has become clear that many of these fibers, despite similar physiologic properties, are accessory atriofascicular or AV fibers with nodelike properties (5,8,9). To avoid further confusion, it is probably best to describe these fibers by their functional and anatomic characteristics. Thus, the two pathways presented in this report were both latent, slowly conducting, decremental AV pathways. Although latent accessory pathways may occur commonly, the uniqueness of the present report is that these pathways were not manifest by pacing maneuvers and drug infusions that typically reveal preexcitation.

Decremental AV fibers are usually diagnosed by evidence of ventricular pre-excitation with atrial pacing techniques that produce AV node conduction delay. Because these fibers also demonstrate both slow conduction and decremental conduction, it is likely that conduction was not manifest in the AV fibers in our patients during routine atrial stimulation because their conduction times and refractory periods were always longer than those of the AV node at all the conditions tested. This is supported by the finding of longer AV intervals during tachycardia than during atrial pacing at the same rate from near the atrial origin of the pathway. Similarly, adenosine may have depressed conduction in these pathways to a similar or greater degree as in the AV node. During ventricular pacing, retrograde block in these fibers with delayed, retrograde conduction through the AV node allowed for antegrade conduction by means of the AV fibers and induction of tachycardia. Induction of atrial fibrillation also may have allowed conduction in the decremental, latent AV fiber to be manifest. However, even during atrial fibrillation, conduction would most likely have been predominantly through the AV node because it had superior conduction characteristics. Occasional conduction by means of the AV fiber and perhaps fusion between AV node conduction and AV pathway conduction would most likely have suggested the diagnosis of premature ventricular depolarizations rather than the presence of a decremental, latent AV fiber because there was no preexcitation with atrial pacing.

Although most decremental AV fibers have been reported to be right-sided, AV fibers with slow and decremental conduction have been reported in the posteroseptal region and left free wall. In a compilation of several series that have identified the location of these fibers (5,8,11–13), 20 (80%) of 25 were right sided, 4 (16%) of 25 were in the posteroseptal region, and only 1 (5%) was left sided. Two other reports (14,15) have described a total of three left-sided “nodoventricular” fibers, indicating the rarity of this finding; two of these fibers were not associated with inducible tachycardias. These numbers may not truly reflect the incidence of left-sided decremental AV fibers for a number of reasons. The relative incidence of left-sided decremental AV fibers may be overestimated because of a relative underreporting of the more commonly occurring right-sided decremental AV fibers. Additionally, left-sided decremental AV fibers may be underdiagnosed if they are latent, even with electrophysiologic techniques that typically produce pre-excitation, as in the present report. This report documents the presence of both posteroseptal and left-sided decremental AV fibers that participate in antidromic AV reentrant tachycardia and establishes that they may be latent even with routine electrophysiologic testing.

Adenosine-sensitive wide complex tachycardia. On the basis of the morphology of the clinical tachycardias, termination with adenosine and lack of baseline pre-excitation, a presumptive clinical diagnosis of adenosine-sensitive ventricular tachycardia was made in the two study patients before invasive electrophysiologic testing. Antidromic tachycardia using a decremental AV fiber was also considered in the differential diagnosis because these tachycardias may frequently be misdiagnosed as ventricular tachycardia because of the lack of baseline pre-excitation (16). However, during electrophysiologic testing the presence of pre-excitation with the introduction of atrial extrastimuli or decremental atrial pacing suggests the diagnosis of antidromic tachycardia. As suggested by Lerman et al. (17) in a study of adenosine-sensitive ventricular tachycardia, absence of these findings has been considered sufficient to rule out the presence of a decremental AV fiber. Thus, using these criteria in the present report would have confirmed the clinical diagnosis of adenosine-sensitive ventricular tachycardia. Only the application of atrial premature depolarizations during tachycardia resulted in making the correct diagnosis, specifically, antidromic AV reentrant tachycardia through a decremental AV fiber.
References