

# Left-sided accessory pathways with atrioventricular node-like properties in the wolff-parkinson-white syndrome

## Editorial

Unlike the conduction through the normal atrioventricular (AV) nodal pathway, the conduction over an accessory pathway (AP) in the Wolff-Parkinson-White (WPW) syndrome usually occurs at a constant interval in its anterograde and retrograde direction.<sup>1-5</sup> Nevertheless, the presence of AP with long conduction times and decremental conduction was also clearly demonstrated.<sup>6,7</sup> AV node-like conduction over an AP was mainly associated with the presence of Mahaim fibers and the permanent form of junctional reciprocating tachycardia.<sup>8-10</sup> Posteroseptal AP had the highest incidences of anterograde and retrograde decremental conduction. However, decremental conduction seldom occurred in both the anterograde and retrograde directions within the same AP.<sup>11</sup> Although, most of the reported cases of AP with decremental conduction were in right-sided pathways, we have previously demonstrated in the study of 55 patients with left-sided AP,<sup>12</sup> that 7% of anterograde conduction and 15% of retrograde conduction had decremental conduction over the AP with a maximal decrement of  $\geq 30$  ms.<sup>12</sup> Nevertheless, we have found that an additional 27% of the left-sided AP in the anterograde direction, and 29% in the retrograde direction had minimal decremental conduction of less than 30 ms.<sup>12</sup> Therefore, our electrophysiological studies revealed that it is not uncommon to observe minimal decremental conduction over left-sided AP, and stress that care should be taken in the evaluation of conduction over these connections.

An interesting question that comes to mind is where does the prolongation in conduction occur, at the atrial or ventricular junction to the AP? In this regard, O'Callaghan WG et al.,<sup>13</sup> demonstrated AP decremental properties at both the ventricular and atrial connections of the AP of a patient with WPW syndrome by means of standard bipolar electrode recordings.<sup>13</sup> Retrograde block was observed at the distal site, namely, at the AP-atrial junction after a prolongation of 10 ms occurred at this site. These are findings that support the concept of "impedance mismatch" as the cause of conduction block, and additionally support our findings of minimal decremental conduction within the AP.<sup>14</sup>

Although decremental conduction seldom occurred in a bilateral direction within the same AP, Nakamura K et al.<sup>15</sup> recently published a very interesting paper of a unique case with a posterolateral left-sided AP with anterograde and retrograde decremental conduction associated with the coronary sinus musculature.<sup>15</sup> They observed interesting electrogram recordings throughout the electrophysiological study prior to ablation. During sinus rhythm or atrial pacing before AP ablation, they recorded continuous low-amplitude and fragmented electrograms between the coronary sinus musculature and the left ventricle along the coronary sinus suggesting the presence of a conduction delay at this interface.<sup>15</sup> Of interest, was the fact that the anterograde conduction between the coronary sinus musculature and the left ventricle was so slow that resulted in the absence of pre-excitation during sinus rhythm despite the continuous presence of anterograde AP conduction.<sup>15</sup> In a retrospective study, Murdock CJ et

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al.<sup>11</sup> arbitrarily defined decremental conduction over the AP as a rate dependent prolongation of atrial-delta wave or VA intervals by more than 30 ms.<sup>11</sup> With this definition in mind, they found an incidence of decremental AP of 7.6%, which is very similar to the incidence we found in anterograde conduction in left-sided AP, namely 7%.<sup>12</sup> They have found that the highest incidence of anterograde decremental conduction was observed in right parietal pathways, and left parietal pathways had the highest incidence of retrograde decremental conduction.<sup>11</sup> They also found that decremental conduction was present in one direction only, while it was absent in the other. However, like in the case of Nakamura K et al.<sup>15</sup> we observed that 4 out of 55 patients (8%) with left-sided AP demonstrated both anterograde and retrograde decremental conduction.<sup>12</sup> In addition, we found that the anterograde decremental conduction zone was  $91 \pm 55$  ms. The retrograde decremental conduction zone was  $72 \pm 47$  ms. The comparison of maximal ventriculoatrial conduction time with the maximal decrement revealed a positive correlation ( $r = 0.63$ ;  $p < 0.01$ ).<sup>10</sup> Moreover, we have found a significant inverse correlation between the AP effective refractory period and the maximal decrement ( $r = 0.42$ ;  $p < 0.05$ ). These findings are very important since they may reflect slow conduction of early extra stimuli during partial recovery of AP excitability. The shorter the AP effective refractory period is, the higher the probability for decremental conduction to occur.<sup>12</sup> This may account for the fact that none of our patients with intermittent WPW syndrome presented with decremental conduction, since all of them had longer AP refractory periods.<sup>12</sup>

The magnitude of the prolongation of the conduction through the AP is related to refractoriness, anatomical and electrophysiological factors. Conduction time over the AP depends on fiber orientation, on the conduction velocity, and on the length and geometry of the AP.<sup>12-17</sup> The most likely explanation for AP decremental conduction is slow and anisotropic conduction across a tortuous AP and through its connections to the atrial and ventricular myocardium.<sup>18</sup> Several possible mechanisms for decremental conduction over an AP have been proposed. First, since AP located in close proximity to the AV node had the highest incidence of decremental conduction, this fact raises the

possibility that the pathway is an AV node-like structure.<sup>19,20</sup> Indeed, AV node-like cells were identified in pathological studies in some patients with AP that demonstrated decremental conduction. Second, AP geometry and fiber orientation may play a role in decremental conduction over the pathway.<sup>21</sup> This fact may also explain why there is decremental conduction in one direction but not in the other. AP histological studies in patients with the permanent form of junctional reciprocating tachycardia showed a tortuous course of the AP with a concomitant change in axial resistance, providing further support that AP orientation may play a role in decremental conduction.<sup>22</sup> Third, role for impedance mismatch between the accessory pathway and the atrium or ventricle has also been suggested as the cause of conduction delay and block.<sup>14</sup>

## Conclusion

In conclusion, our previously reported data revealed in left-sided AP that there is a significant inverse correlation between the AP effective refractory period and the maximal decrement, which may reflect slow conduction of early extra stimuli during partial recovery of AP excitability. Indeed, the shorter the AP effective refractory period is, the higher the probability for decremental conduction to occur in left-sided AP. And, that minimal decremental conduction over left-sided AP is not an uncommon finding and stress that care should be taken in evaluation of conduction over these connections.

## References

- Wellens HJ, Durrer D. Patterns of ventriculoatrial conduction in the Wolff-Parkinson-White syndrome. *Circulation*. 1974;49(1):22–31.
- Narula OS. Retrograde pre-excitation: Comparison of anterograde and retrograde conduction intervals in man. *Circulation*. 1974;50(6):1129–1143.
- Centurión OA, Shimizu A, Isomoto S, et al. Mechanisms for the genesis of paroxysmal atrial fibrillation in the Wolff-Parkinson-White syndrome: Intrinsic atrial muscle vulnerability vs. electrophysiological properties of the accessory pathway. *Europace*. 2008;10(3):294–302.
- Centurión OA, Isomoto S, Hayano M, et al. Evidence of quadruple anterograde AV nodal pathways in a patient with atrioventricular node reentry. *J Electrocardiol*. 1994;27(1):71–78.
- Centurión OA, Isomoto S, Konoe A, et al. Electrophysiologic demonstration of anterograde fast and slow pathways within the His bundle in patients with normal intraventricular conduction. *Int J Cardiol*. 1994;44(3):251–260.
- Goldberger JJ, Pederson DN, Damle RS, et al. Antidromic tachycardia utilizing decremental, latent accessory atrioventricular fibers: differentiation from adenosine sensitive ventricular tachycardia. *J Am Coll Cardiol*. 1994;24(3):732–738.
- Johnson CT, Brooks C, Jaramillo J, et al. A left free-wall, decrementally conducting, atrioventricular (Mahaim) fiber: diagnosis at electrophysiological study and radiofrequency catheter ablation guided by direct recording of a Mahaim potential. *Pacing Clin Electrophysiol*. 1997;20(10 Pt 1):2486–2488.
- Gallagher JJ, Smith WM, Kasell JH, et al. Role of Mahaim fibers in cardiac arrhythmias in man. *Circulation*. 1981;64(1):176–189.
- Lerman BB, Waxman HL, Proclemer A, et al. Supraventricular tachycardia associated with nodoventricular and concealed atrioventricular bypass tracts. *Am Heart J*. 1982;104(5 Pt 1):1097–1102.
- Bardy GH, German LD, Packer DL. Mechanism of tachycardia using a nodoventricular Mahaim fiber. *Am J Cardiol*. 1994;74:1140–1141.
- Murdock CJ, Leitch JW, Teo WS, et al. Characteristics of accessory pathways exhibiting decremental conduction. *Am J Cardiol*. 1991;67(6):506–510.
- Centurión OA, Fukatani M, Shimizu A, et al. Anterograde and retrograde decremental conduction over left-sided accessory atrioventricular pathways in the Wolff-Parkinson-White syndrome. *Am Heart J*. 1991;125(4):1038–1047.
- O'Callaghan WG, Colavita PG, Kay GN, et al. Characterization of retrograde conduction by direct endocardial recording from an accessory atrioventricular pathway. *J Am Coll Cardiol*. 1986;7(1):167–171.
- De la Fuente D, Sasyniuk B, Moe GK. Conduction through a narrow isthmus in isolated canine atrial tissue. A model of Wolff-Parkinson-White syndrome. *Circulation*. 1971;44(5):803–809.
- Nakamura K, Naito S, Kaseno K, et al. Antegrade and retrograde decremental conduction properties of an accessory pathway associated with the coronary sinus musculature. *Indian Pacing Electrophysiol J*. 2015;15(1):55–61.
- Inoue H, Zipes DP. Conduction over an isthmus of atrial myocardium in vivo: a possible model of Wolff-Parkinson-White syndrome. *Circulation*. 1987;76:637–647.
- Lunel AA. Significance of annulus fibrosus of the heart in relation to AV conduction and ventricular activation in cases of Wolff Parkinson-White syndrome. *Br Heart J*. 1972;34(12):1263–1271.
- Becker AEZ, Anderson RH, Durrer D, et al. The anatomical substrates of Wolff-Parkinson-White syndrome. A clinicopathologic correlation in seven patients. *Circulation*. 1978;57(5):870–879.
- Klein GJ, Prystowski EN, Pritchett ELC, et al. Atypical pattern of retrograde conduction over accessory atrioventricular pathways in the Wolff-Parkinson-White syndrome. *Circulation*. 1979;60(7):1477–1486.
- Denes P, Kehoe R, Rosen KM. Multiple reentrant tachycardias due to retrograde conduction of dual atrioventricular bundles with atrioventricular nodal-like properties. *Am J Cardiol*. 1979;44(1):162–170.
- Jackman WM, Friday KJ, Yeung-Lai-Wah JA, et al. New Catheter technique for recording left free wall accessory atrioventricular pathway activation: Identification of pathway fiber orientation. *Circulation*. 1988;78(3):598–610.
- Critelli G, Gallagher JJ, Monda V, et al. Anatomic and electrophysiologic substrate of the permanent form of junctional reciprocating tachycardia. *J Am Coll Cardiol*. 1984;4(3):601–606.