

## Evaluation of Fast Pathway Function: The Importance of Autonomic Tone

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There is no consensus on the effect of slow pathway ablation on the electrophysiologic properties of the fast pathway in patients with AV nodal reentrant tachycardia. Some investigators have demonstrated that ablation of the slow pathway results in shortening of the fast pathway effective refractory period, while other investigators have not made this observation.<sup>1-13</sup> In the setting of sympathetic stimulation, slow pathway ablation does not result in shortening of the fast pathway effective refractory period.<sup>10</sup> The results from studies not performed in the setting of autonomic blockade have been variable.<sup>2-7,12,13</sup> However, the majority of studies performed in the setting of autonomic blockade have demonstrated shortening of the fast pathway effective refractory period after complete, but not partial, elimination of slow pathway function.<sup>8-11</sup> Among the four studies that have included autonomic blockade as part of the study design, three demonstrated that the fast pathway effective refractory period decreases after complete elimination of slow pathway function.<sup>8-10</sup> In one study, shortening of the fast pathway effective refractory period after slow pathway ablation in the setting of atropine and propranolol was not observed.<sup>11</sup> In this report, complete autonomic blockade was not maintained throughout the study, as evidenced by large changes in the sinus cycle length before and after the ablation procedure.<sup>11</sup> If shortening of the fast pathway effective refractory period can be obscured by alterations in autonomic tone, then complete autonomic blockade throughout the entire study is required. Given that the changes in fast pathway function occur only after complete elimination of slow pathway function and that sympathetic stimulation can obscure these

changes, it is not surprising that the effect of slow pathway ablation on fast pathway function in previous studies that did not perform complete autonomic blockade has been variable.

In this issue of *JCE*, Shen and colleagues<sup>14</sup> present another study addressing the effect of slow pathway ablation on fast pathway refractoriness. The authors evaluated fast pathway function in the baseline state and during infusion of isoproterenol in 40 patients before and after slow pathway ablation. Autonomic blockade was not utilized, although the authors address this limitation by demonstrating that the sinus rate was not significantly different after elimination of slow pathway function as compared with before ablation, and by stating that a constant level of sedation was maintained throughout the ablation procedure. As in prior studies in which the influence of changes in autonomic tone were not controlled, no significant changes in fast pathway function were demonstrated after successful slow pathway ablation. These investigators conclude that fast pathway function does not change after slow pathway ablation, and that the fast and slow pathways are functionally independent.

The authors provide two major reasons for the disparity in their results as compared with the results of the investigators who performed studies in the setting of complete autonomic blockade.<sup>8-10</sup> The first is that the effect of slow pathway ablation on the fast pathway is variable. We agree that shortening of the fast pathway effective refractory period is not a universal finding, although the mean fast pathway effective refractory period decreases significantly if there is no residual slow pathway function, and alterations in fast pathway function are observed in a large percentage of patients without residual slow pathway function.<sup>8-10</sup> Shortening of the fast pathway effective refractory period was observed in 7 of 10 patients,<sup>8</sup> and in 11 of 13 patients<sup>9</sup> in whom complete elimination of slow pathway function resulted from ablation. Individual patient data were not published in the third study.<sup>10</sup> The second reason stated for the disparity is that

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a larger number of patients were studied. The present study included 40 patients, whereas the three studies with autonomic blockade included 10 to 20 patients.<sup>8-10</sup> Given the important influence of autonomic tone on fast pathway function, the ideal evaluation of AV node function includes autonomic blockade; otherwise, changes in autonomic tone will have an unpredictable effect on the results. In fact, based on the results of prior studies, if the authors had conducted their study with autonomic blockade, it is very possible that shortening of the fast pathway effective refractory period would have been found.<sup>8-10</sup>

What mechanism accounts for the shortening of the fast pathway effective refractory period after complete, but not partial, elimination of slow pathway function? A reasonable explanation for the shortening of the fast pathway effective refractory period is elimination of an electrotonic influence of the slow pathway on the fast pathway. Electrotonus is the phenomenon by which passive current flows secondary to voltage gradients between cells.<sup>15</sup> The refractory periods of adjacent cells may prolong due to subthreshold depolarizations from this passive current flow.<sup>15</sup> The presence of the slow pathway before ablation or of residual slow pathway function after ablation may allow for electronic interaction of the slow pathway with the fast pathway, resulting in lengthening of the fast pathway effective refractory period. Complete destruction of slow pathway function may eliminate this electrotonic interaction and result in shortening of the fast pathway effective refractory period. This explanation is consistent with a computer model of dual AV nodal physiology, which predicted an electrotonic interaction between the fast and slow pathways.<sup>16</sup>

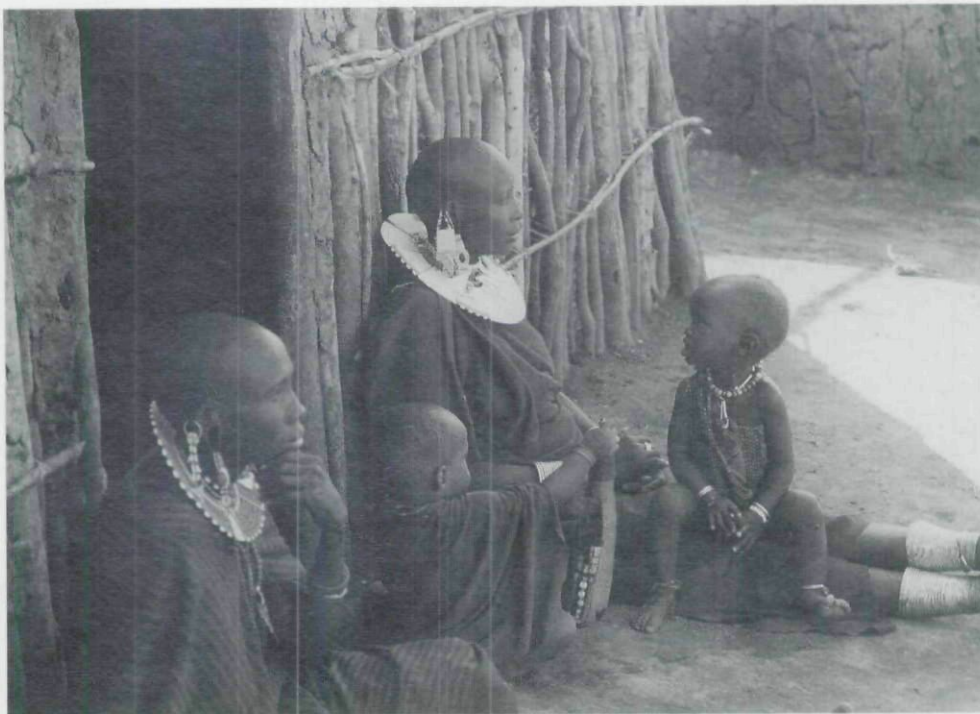
The authors discount the possibility of an electrotonic interaction between the fast and slow pathways because the AV block cycle length of the fast pathway did not change after slow pathway ablation, and because of the uniform response to isoproterenol that was observed. However, refractoriness and conduction are separate and distinct properties. Slow pathway ablation could affect refractoriness without affecting conduction. Isoproterenol administration may not be an ideal agent to aid in the identification of an electrotonic interaction between the fast and slow pathways. Sympathetic stimulation may improve fast pathway function to such a degree that the electrotonic effect of the slow pathway may be attenuated, thereby obscuring the shortening of the fast pathway that occurs after slow pathway ablation.

In conclusion, we believe that fast pathway function is sensitive to alterations in autonomic tone, and that fast pathway function should therefore routinely be evaluated in the setting of autonomic blockade. Under the condition of autonomic blockade, complete elimination of slow pathway function results in shortening of the fast pathway effective refractory period. The mechanism most likely to explain this observation is removal of an electrotonic interaction of the slow pathway on the fast pathway.

## References

1. Haissaguerre M, Gaita F, Fischer B, et al: Elimination of atrioventricular nodal reentrant tachycardia using discrete slow potentials to guide application of radiofrequency energy. *Circulation* 1992;85:2162-2175.
2. Jackman WM, Beckman KJ, McClelland JH, et al: Treatment of supraventricular tachycardia due to atrioventricular nodal reentry by radiofrequency catheter of slow-pathway conduction. *N Engl J Med* 1992;327:313-318.
3. Jazayeri MR, Hempe SL, Sra JS, et al: Selective transcatheter ablation of the fast and slow pathways using radiofrequency energy in patients with atrioventricular nodal reentrant tachycardia. *Circulation* 1992;85:1318-1328.
4. Kay GN, Epstein AE, Dailey SM, et al: Selective radiofrequency ablation of the slow pathway for the treatment of atrioventricular nodal reentrant tachycardia: Evidence for involvement of perinodal myocardium within the reentrant circuit. *Circulation* 1992;85:1675-1688.
5. Mitrani RD, Klein LS, Hackett FK, et al: Radiofrequency ablation for atrioventricular node reentrant tachycardia: Comparison between fast (anterior) and slow (posterior) pathway ablation. *J Am Coll Cardiol* 1993;21:432-441.
6. Takahashi A, Iesaka Y, Igawa M, et al: Atrioventricular nodal physiology after slow pathway ablation. *PACE* 1994;17:2137-2142.
7. Simmers TA, Wever EF, Wittkampf FH, et al: Change in delay of atrioventricular conduction after radiofrequency catheter ablation for atrioventricular nodal reentry tachycardia. *Br Heart J* 1995;73:442-444.
8. Natale A, Klein G, Yee R, et al: Shortening of fast pathway refractoriness after slow pathway ablation: Effects of autonomic blockade. *Circulation* 1994;89:1103-1108.
9. Strickberger SA, Daoud E, Niebauer M, et al: Effects of partial and complete ablation of the slow pathway on fast pathway properties in patients with atrioventricular nodal reentrant tachycardia. *J Cardiovasc Electro-physiol* 1994;5:645-649.
10. Strickberger SA, Weiss R, Daoud EG, et al: Ventricular rate during atrial fibrillation before and after slow-pathway ablation: Effects of autonomic blockade and

- adrenergic stimulation. *Circulation* 1996;94:1023-1026.
11. Blanck Z, Dhala AA, Sra J, et al: Characterization of atrioventricular nodal behavior and ventricular response during atrial fibrillation before and after a selective slow-pathway ablation. *Circulation* 1995;91:1086-1094.
  12. Kreiner G, Heinz G, Siostrzonek P, et al: Effect of slow pathway ablation on ventricular rate during atrial fibrillation: Dependence on electrophysiological properties of the fast pathway. *Circulation* 1996;93:277-283.
  13. Markowitz SW, Stein KM, Lerman BB: Mechanism of ventricular rate control after radiofrequency modification of atrioventricular conduction in patients with atrial fibrillation. *Circulation* 1996;94:2856-2864.
  14. Shen WK, Munger TM, Stanton MS, et al: Effects of slow pathway ablation on fast pathway function in patients with atrioventricular node reentrant tachycardia. *J Cardiovasc Electrophysiol* 1997;8:627-638.
  15. Antzelevitch C: Electrotonus and reflection. In Rosen MR, Janse MJ, Wit AL, eds: *Cardiac Electrophysiology: A Textbook*. Futura Publishing Company, Inc., Mount Kisco, NY, 1990, pp. 491-516.
  16. Lesh MD, Gibb WJ, Epstein LM, et al: A computer model of dual AV nodal pathways: Evidence for electrotonic interactions. *Comput Cardiol* 1992:39-42.



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