

Ablative care of atrioventricular block: Uncovering new horizons or chasing ghosts



Melvin M. Scheinman, MD

From the University of California, San Francisco, San Francisco, California.

In a fascinating report Tuohy et al¹ demonstrated quite logically that the atrioventricular (AV) block in a patient was due to a concealed nodoventricular (N-V) pathway and that ablation successfully restored AV conduction. A series of keen observations led to these innovative and exciting conclusions. They first noted the presence of junctional premature complexes (JPCs) that were either manifest or concealed (no anterograde conduction to the ventricle), but resulted in AV block. They further discerned that JPCs were always linked to the preceding conducted ventricular complex. They further found that oral flecainide (a potent class 1C agent) treatment clearly increased the density of JPCs. While the effects of flecainide on abnormal automaticity or triggered rhythms is not uniform,^{2,3} it clearly should not be expected to increase His bundle or fascicular activity, which led them to invoke the possibility of a reentrant mechanism and the possibility of an N-V pathway.

Conventional analyses would suggest that the AV block was due to either concealed junctional extra systoles or dual AV nodal conduction with slow pathway or distal AV nodal block. Both these hypotheses were excluded by inability to record a His bundle potential before the blocked P wave and by the finding that prior slow pathway ablation failed to secure normal AV conduction. They postulated the presence of a concealed N-V pathway, which explained the absence of the His deflection during AV block. Subsequent careful mapping and ablation in the ventricle adjacent to the coronary sinus just below the His bundle remarkably resulted in restoration of AV conduction and disappearance of JPCs.

Certain points deserve emphasis. For example, the site of successful ablation on the ventricular side of the annulus starting inferiorly at the level of the coronary sinus and incrementing towards the His bundle sounds suspiciously close to the slow pathway. Is it possible that the initial SP ablation was incomplete and that slow pathway ablation was, in fact, eliminated by the second ablation? This observation would not explain the response to flecainide therapy. In addition,

inability to record a consistent His bundle potential might be due to technical factors related to catheter movement. For studies in which His and/or right bundle branch block potentials are critical to establish a proper diagnosis, it is well to consider the use of closely spaced multipolar (decapolar) electrode catheters or even use of 2 catheters to span the His bundle region. Finally, none of the observations provided exclude the possibility of a concealed nodofascicular pathway rather than an N-V pathway, but this does not in any way take away from the splendid logic of their analyses.

Having the benefit of hindsight, are there any additional maneuvers to help in diagnoses? Conceivably, other pharmacological challenges might be helpful; for example, the use of adenosine might be expected to reduce the frequency of JPCs owing to N-V reentry while increasing AV block. The authors used intravenous procainamide at a dose of 10 mg/kg and failed to note any effects on JPCs or AV block. Perhaps a higher dose would have been more effective. Unfortunately, intravenous flecainide is not available in the United States but is available in other countries. With the use of flecainide, one could examine both the JPC density and the coupling interval between the JPC and the conducted complex. Moreover, if they are correct, then conduction block in the right bundle branch (RBB) for conducted complexes should result in lengthening of the coupling interval between conducted RBB complexes and the JPC. This can be done in the laboratory by gentle bumping of the RBB. The same considerations apply for pathways with putative left-sided concealed N-V in terms of the effects of a left bundle branch pattern. Moreover, insertion of closely coupled atrial premature beats should encourage AV nodal delay and enhance reentry involving a concealed N-V pathway.

The authors approach to ablation of the putative pathway involves a great deal of serendipity and good fortune since they could not be guided by recording a discrete potential. Others have described successful ablation of nodofascicular or N-V pathways guided by discrete potentials.^{4,5} Inability to record a discrete N-V potential is likely due to the fact that the potential is obscured by simultaneous ventricular activation during sinus rhythm. One consideration is use of low-energy ventricular pacing in the general region of the pathway, hoping for capture of muscle, but not the pathway potential, hoping to make the recording of a potential more accessible after the completion of local ventricular activation.

Address reprint requests and correspondence: Dr Melvin M. Scheinman, Cardiac Electrophysiology, University of California, San Francisco, 500 Parnassus Avenue, San Francisco, CA 94143. E-mail address: scheinman@medicine.ucsf.edu.

This technique would be similar to discernment of a His potential with low energy during para-Hisian pacing.⁶

In this era where we are inundated by studies often involving hundreds or thousands of patients, it is refreshing to follow the logic and mode of successful treatment for a single patient. Does this study truly open new horizons in the treatment of patients with AV block or is this just a “one and done” observation? Time will tell.

References

1. Tuohy S, Saliba W, Pai M, Tchou P. Catheter ablation as a treatment for atrioventricular. *Heart Rhythm* 2018;15:90–96.
2. Wit AL, Wellens HJ, Josephson ME. *Electrophysiological Foundations of Cardiac Arrhythmias: A Bridge Between Basic Mechanisms and Clinical Electrophysiology*. Cardiotext Publishing; 2017: Chapter 4, p. 189.
3. Wit AL, Wellens HJ, Josephson ME. *Electrophysiological Foundations of Cardiac Arrhythmias: A Bridge Between Basic Mechanisms and Clinical Electrophysiology*. Cardiotext Publishing; 2017: Chapter 6, p. 253.
4. Hluchy J, Schlegelmilch P, Schickel S, Jörgen U, Jurkovicova O, Sabin GV. Radiofrequency ablation of a concealed nodoventricular Mahaim fiber guided by a discrete potential. *J Cardiovasc Electrophysiol* 1999;10:603–610.
5. Badhwar NS, Kang MH, Dressing T, Tchou P, Josephson MJ, Scheinman MM. Successful site of ablation for patients with concealed nodofascicular tachycardia. *Heart Rhythm* 2007;4:S75.
6. Hirao K, Otomo K, Wang X, Beckman KJ, McClelland JH, Widman L, Gonzalez MD, Arruda M, Nakagawa H, Lazzara R, Jackman WM. Para-Hisian pacing: a new method for differentiating retrograde conduction over an accessory AV pathway from conduction over the AV node. *Circulation* 1996;94:1027–1035.