Response to Letter Regarding Article, “Variable Clinical Features and Ablation of Manifest Nodofascicular/Ventricular Pathways”

We thank Drs Papagiannis and Kanter1 for their interest and keen attention to our recent article. We shall address each of the points raised in their letter.

Case 1: There is no difference between your description of the mechanism of case I and that provided in the article.

Case 2: There is again no substantive difference with regards to tachycardia mechanism. You are correct. Figure 4A shows an obvious error in that we clearly show that stim A-V increases with A2. Thank you for pointing out this oversight.

Case 3: We are well aware of the differential diagnoses of mechanisms resulting in reverse activation of the His bundle, 2 but these points are moot for reasons detailed in the article and reemphasized in your comments. It may be difficult to distinguish atrioventricular nodal reentrant tachycardia with a bystander manifest nodofascicular (MNF) from a MNF critical to the circuit. We did consider both options but concluded in the article that persistence of the MNF in response to ventricular extrastimulus while the tachycardia was no longer inducible was evidence that the manifest nodofascicular pathway was a bystander. It is possible that both atrioventricular nodal reentrant tachycardia with a MNF bystander and MNF pathway critical to circuit were capable of sustaining tachycardias.

Case 4: There are obvious disagreements in interpretation of the data for case 4. We agree with you that the distal His most likely represents the right bundle branch potential. With respect to Figure 7B, note that the RB–V interval decreases comparing the sinus beat with premature beats associated with a left bundle branch block pattern.

Similarly, for Figure 7D, the RB–V decreases with the left bundle branch block patterns. There should be no reason for such changes if this was due, as you suggest, to atrioventricular nodal reentrant tachycardia or AV reentrant tachycardia with left bundle branch block aberrancy. With regards to the premature beats shown in Figure 7C, note that the second beat is coincident with a His deflection. We interpret this as a junctional beat with pre-excitation, which is most compatible with a manifest nodoventricular tract. If this was because of premature ventricular contractions from the right ventricle (as you suggest), then the His deflection should be buried within the QRS. In addition, as emphasized in the article, after ablation of the slow pathway, the aberrant beats disappeared and the septal accessory pathway was located and ablated over the anterior superior tricuspid annulus. Taken together, this is our rationale for suggesting that a manifest nodoventricular pathway was present but not part of the tachycardia.

In summary, we find no substantive differences in the interpretations for cases 1 to 3. We respectfully differ with your interpretations of case 4, but welcome the thoughtful discussion of this fascinating arrhythmia.

Disclosures

None.

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References


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