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EDITORIAL

Slow and Unsteady in the Fast Lane

See Article by Chen et al

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Bundle branch reentry (BBRT) should never have existed as an arrhythmia. Sustained reentry requires either a long physical circuit length or slow conduction. Given the limited, relatively small, size of the human heart, reentrant ventricular tachycardia requires either discrete or diffuse slow conduction. The His-Purkinje system, including the bundle branches, are made for fast conduction and should be the last anatomic site where one would expect to find the culprit location for sustained reentrant tachycardia. Not surprisingly, the original descriptions[1] emphasized the abnormal ventricular myocardial components of this circuit along with markedly pathological conduction tissue making possible this unique arrhythmia. Less commonly, BBRT may occur without any obvious myocardial structural disease, and the mechanism is thought to be related to isolated but severe His-Purkinje dysfunction.[2] Chen et al[3] describe the electrophysiological characteristics, ablation outcomes, and long-term follow-up of 9 patients with BBRT and without structural heart disease. Generally, the right bundle is targeted for ablation because of its limited insulation and superficial course in the right ventricle. Unique in Chen's et al[3] series is the targeted ablation of the left bundle, a structure hitherto thought difficult to ablate because of its myocardial depth and significant insulation.

The patients described were not felt to have any structural heart disease based on normal cardiac magnetic resonance imaging and normal voltage mapping. However, all of the patients did have abnormalities seen in the His-Purkinje system during electrophysiology study. The principle findings from this study need to be understood in the context of needing an exact understanding of what is and is not in the BBRT circuit, recognizing the presently insurmountable difficulties with measuring conduction system velocity, and the concept of pseudointerval.

DO WE NEED THE HIS?

There is an inherent paradox in our current construct for BBRT. It is generally taught that the H-H intervals from one His bundle recording to the next should precede changes in the ventricular cycle length. At the same time, however, we recognize that the His bundle is not a part of the BBRT circuit. In the common variant of BBRT, conduction travels down the right bundle, transits through the septal myocardium, and travels retrograde through the left bundle.[2] The turnaround point from the left bundle to the right bundle is inferior to and does not include the bundle of His (Figure). One reason for this discrepancy is the term His bundle is used incorrectly synonymously with the right bundle branch (RBB) signal, which is indeed part of the circuit and cannot be dissociated from the tachycardia.[4]

Key Words: Editorials ■ anatomy ■ anisotropy ■ bundle-branch block ■ bundle of His ■ ventricular tachycardia

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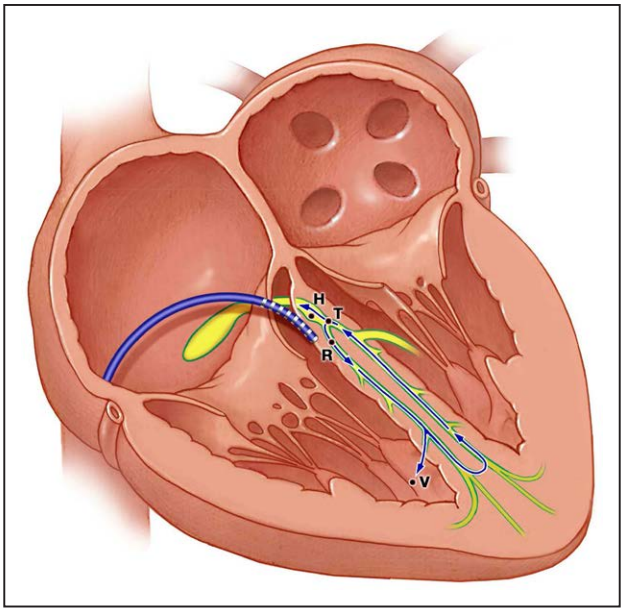

Figure. Diagram of mechanism of bundle branch reentry tachycardia. Diagram demonstrates conduction antegrade down the right bundle branch (R), exiting into the ventricle (V), traversing the septum, and conducting retrograde up the left bundle branch. The conduction then reaches a turnaround point (T) where it begins to go back down the right bundle branch. An octapolar recording catheter is shown that could record signals from the His (H) or the right bundle branch (R). Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.

PSEUDOINTERVALS

During sinus rhythm, the H-V interval represents the true conduction time from depolarization of the His bundle to the ventricular exit from the distal bundle branches. During BBRT, however, the H-V interval represents the relative conduction time between the turnaround point of the circuit retrograde to the His bundle recording catheter and from the turnaround point antegrade to ventricular exit and thus represents a pseudointerval.[5] There is therefore no predictable relationship between the H-V interval during BBRT and in sinus rhythm. Unsurprisingly, Chen et al[3] in their study found some patients with a longer H-V interval in tachycardia and others longer in sinus rhythm when compared with tachycardia.

CAN WE EFFECTIVELY MEASURE CONDUCTION NETWORK DELAY?

An important feature of Chen's et al[3] study is their attempt to precisely identify the site of conduction delay within the BBRT circuit. Intuitively, we may expect this delay to be at the proximal turnaround site with extreme anisotropy. However, the authors surprisingly determined in these patients with no myocardial disease the main delay site was at the interface of the distal RBB and the myocardial exit. This is an intriguing finding that may suggest some nonobvious myocardial disease process contributing to the genesis of BBRT in otherwise structurally normal hearts.

Importantly, however, conduction velocity determinations that presume a linear conduction system is near impossible to accurately define in the 3-dimensional filigree-like Purkinje network present in human hearts. Two recording sites may simply be measuring the relative times from a conduction breakthrough site in between the recording locations falsely underestimating the extent of conduction delay that may be present in the direction of propagation being measured.

ARE WE NOW BETTER AT ABLATING THE LEFT BUNDLE?

BBRT can be cured by ablating either the RBB or left bundle branch. Traditionally, we ablate the RBB because it's superficial and it was thought that the insulated, deeper-located left bundle branch could not be readily ablated.[6,7] Chen et al[3] used a standard, 4-mm-tip, nonirrigated catheter with a mean of 2.5 radiofrequency lesions to readily ablate the left bundle branch. This approach is attractive as we can leave what remains of relatively normal atrioventricular conduction through the RBB.

The findings from this study serve to enhance our understanding of His-Purkinje-related arrhythmia ablation that now includes the spectrum of BBRT, fascicular reentry, Purkinje triggers for ventricular fibrillation, and perhaps the substrate for ventricular fibrillation itself.[8–10]

ARTICLE INFORMATION

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Disclosures

None.

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