

## The Fast Zone for Reentry

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Reentry is the most common mechanism for arrhythmia in patients treated in the electrophysiology laboratory. Mapping and defining the exact circuit to then allow targeting an ideal site for ablation is challenging.<sup>1</sup> However, as reentry usually involves a relatively large circuit often coupled with a discrete slow zone of conduction, we are able to use the classical techniques of entrainment mapping to find and ablate the circuit isthmus containing the slow zone ablation for permanent elimination.<sup>2,3</sup>

### See Article by Liu et al

Typically, slow zones for conduction occur as a result of diseased myocardium in the vicinity of scars in patients with structural heart disease. Reentry seen in normal hearts often has the atrioventricular node serving as the primary slow zone of the tachycardia circuit, for example, in atrioventricular node reentrant tachycardia and accessory pathway-related tachycardia. Indeed, nodoventricular tachycardia, although a rare tachycardia, exists because of atrioventricular nodal participation unlike patients with a fasciculoventricular tract where no tachycardia has been described likely because of the absence of a slow zone.

Reentry that involves the conduction system is in some ways paradoxical because the Purkinje system is specialized tissue with fast conduction velocity. Bundle branch reentrant tachycardia does occur but almost always in severely diseased hearts with both slowed conduction through the Purkinje tissue and diseased myocardium forming parts of the circuit.<sup>4</sup>

Left posterior fascicular tachycardia is an enigmatic arrhythmia that occurs in normal hearts. The involvement of the conduction system with no apparent slow zone and success with discrete ablation at apparently normal tissue yet with clear characteristics of reentry based on entrainment makes this arrhythmia unique and difficult to approach.<sup>5</sup> Treating the arrhythmia as a small circuit reentry akin to a focal source is also of limited value because the earliest site of ventricular activation is simply the exit site of some part of the infra-Hisian conduction system. Mapping the earliest conduction tissue signal often fails because there is no truly early site given the reentrant nature of this tachycardia.

In this installment of Teaching Rounds in Cardiac Electrophysiology, Liu et al<sup>6</sup> build on the pioneering work of Nogami et al<sup>5,7,8</sup> and provide an exemplary analysis of 3 patients' fascicular tachycardia. Their submission is rife with teaching points that may well take the student of

electrophysiology multiple, careful readings to fully appreciate. Their figures and discussion clearly describe the fast zone components of the tachycardia with intriguing observations on what may constitute the return limb of the tachycardia circuit back into the conduction system and a possible slow zone for reentry. This submission exemplifies the 2 main attributes of great teaching—pointing out observations that may otherwise escape notice (details of conduction tissue signal sequences in tachycardia and pacing) and physiology-based deduction on the behavior of these signals when perturbed with pacing maneuvers. Undoubtedly, students will be inspired to observe and analyze more carefully when tackling the mysteries in our field, but in doing so, it is worthwhile examining how important the fundamental tools available to the electrophysiologist are necessary to unravel and expose the essence of difficult arrhythmias.

### Reset

Paramount in understanding the analysis provided by Liu et al<sup>6</sup> is the careful interpretation of where, when, and how much reset of the tachycardia circuit occurs during pacing and insertion of premature beats. The surface ECG is rarely sufficient for this analysis because reset does not need to be global. In fact, discerning which electrograms are advanced or delayed when a premature beat is introduced versus those that are not is a fundamental tool in piecing together the parts of the relevant circuit as opposed to bystander sites. For example, in Figure 1B, advancing the P1 and P2 signals without change in the proximal conduction system and His bundle electrograms was instrumental in appreciating 2 disparate fast zones of conduction as part of the reentrant circuit.

### Sequence of Activation

Analyzing reset without meticulous attention to possible changes in global and regional sequence of activation may lead to erroneous conclusions. Even a common maneuver such as placing premature ventricular contractions during a narrow complex tachycardia will miss the fact that an identified accessory pathway is a bystander without appreciating that the His refractory premature ventricular contraction advances the next set of atrial electrograms but with a distinct change in the retrograde activation sequence.

An important learning point from Liu et al's<sup>6</sup> descriptions is their use of multiple electrodes with high fidelity

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recordings to expose the detailed regional conduction system sequence of activation.<sup>6</sup> Although minimalist approaches may be useful to decrease cost and increase efficiency for well understood arrhythmias, complex cases need as much information as possible. However, this information must be reliable with stable contact electrograms and sufficient sampling rate and dynamic range for the displayed recorded signals to be of value in noting sometimes subtle changes in activation.

Because reset with fusion defines reentry, a principle reason for having multiple electrodes is to identify fusion that would not be apparent otherwise. For instance, in all 3 cases, both ventricular electrograms and conduction tissue-related electrograms have some sites of identical sequence as tachycardia during pacing, whereas other sites are clearly different with reset defining fusion and, thus, an entrainable reentrant tachycardia.

### Varied Stimulation

To determine reset with or without an activation sequence change, pacing during tachycardia is the key maneuver. However, with complex arrhythmias such as fascicular tachycardia where overlapping conduction tissue and ventricular myocardium is the rule and indeed different parts of the conduction system lie in close proximity, P1- and P2-related signals, it is impossible to identify and integrate components into a putative circuit without varying the pacing stimulation. Electrophysiologists have 3 ways to do this—by changing the site of pacing, the rate of pacing, or the output when pacing at a constant rate or stable site.

### Pacing Site

Nogami et al, other early investigators,<sup>8</sup> and the authors of this article<sup>6</sup> pace from right ventricular myocardium, myocardium closer to the left-sided conduction system, and cleverly utilized atrial pacing so as to have early sole conduction system-related stimulation in the ventricle to help differentiate myocardial and conduction sites that may be part of this hard-to-identify circuit.

### Pacing Rate

From a given site, the degree of prematurity required to reset a tachycardia is useful although indirect measure of how far the pacing site is from the arrhythmogenic circuit. Some prematurity is essential to reset the tachycardia, but placing beats too early in the cycle may change or terminate the tachycardia or in the case of conduction tissue-related arrhythmias, change local sequences making it impossible to differentiate bystanders from closely approximated arrhythmogenic signals.

### Pacing Output

Because of the superimposition of conduction tissue and ventricular myocardium, changing the pacing output with fixed prematurity and stable location can be useful to distinguish whether the fascicular signals being recorded at a probable circuit site are part of the tachycardia or simply present at a myocardial site involved in reentry. In the

proximal conduction system including the His bundle, high output pacing is required to penetrate the fibrous covering of these structures, whereas low output fails to capture the conduction system and results in capture of only the ventricular myocardium. Thus, if high output pacing resets with fusion, and low output pacing near the proximal conduction system produces a wide QRS that does not, then fascicular involvement in the tachycardia is likely. However, in the distal Purkinje network, capture of only the conduction tissue may be possible with low output pacing and small bipolar electrodes, whereas high output pacing would capture both ventricular myocardium and the Purkinje network. Therefore, when high output pacing resets a tachycardia but low output pacing with capture does not, the myocardium is likely necessary for the tachycardia.

As pointed out by Liu et al,<sup>6</sup> fascicular tachycardia seems to involve 2 sets of conduction tissue, fast conducting tissue and intervening proximal myocardium.<sup>6</sup> The variation between their cases also stresses that real-time identification of the tachycardia circuit and defining a possible channel for ablation require thorough appreciation of their teaching points and understanding the need for varied stimulation.

### Significance and Summary

As with great teaching from any field of scientific learning, Liu et al<sup>6</sup> provide for us instructive observations and analysis for one of the most difficult arrhythmias to comprehend and logically target for ablation, while simultaneously providing principles and guidelines that permeate all aspects of electrophysiology when dealing with reentry that involves fast and slow zones.

### Disclosures

Dr Asirvatham receives no significant honoraria and is a consultant with Abiomed, Atricure, Biosense Webster, Biotronik, Boston Scientific, Medtronic, Spectranetics, St. Jude, Sanofi-Aventis, Wolters Kluwer, Elsevier, and Dr Stevenson is coholder of a patent on needle ablation that is consigned to Brigham and Women's Hospital.

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