Mapping and Ablation of Ventricular Tachycardia From the Left Upper Fascicle
How to Make the Most of the Fascicular Potential?

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Triggered activity or localized reentry in the fascicular system can give rise to premature impulses or ventricular tachycardia (VT). The diagnosis of fascicular rhythms relies on the recording of the His bundle potential before the onset of surface ventricular activation.1 Radiofrequency catheter ablation can be performed successfully by identification of the earliest fascicular potential (FP).2 The following cases illustrate how the comparison of the FP-V interval between sinus rhythm (SR) and VT can help to identify the successful ablation site.

Case Description

Case 1
A 50-year-old man presented with symptoms of fatigue and frequent premature ventricular contractions (PVCs) that were refractory to medical therapy. Left ventricular systolic function was normal as measured by echocardiography. On a 12-lead ECG, the PVCs were relatively narrow with an incomplete right bundle branch block morphology and normal axis, suggestive of origin from the proximal left fascicular system (Figure 1A). Electrophysiology study and mapping were performed during PVCs with a 4-mm catheter electrode via a retrograde aortic approach. Right bundle branch block was noted because of inadvertent mechanical block of the right bundle branch during manipulation of the right ventricular catheter. The ablation catheter was positioned near the distal His bundle or proximal left bundle with H-V (His-V) interval of 52 ms during SR and 24 ms during PVCs (Figure 2A). The catheter was then positioned at the proximal left anterior fascicle (LAF) with recordings of FP-V (fascicular potential-V) interval of 38 ms during SR and PVCs (Figure 2C). Radiofrequency was applied at this site with 30 W at 60°C that resulted in complete elimination of PVCs and LAF block (Figure 1B). The proximal left posterior fascicle (LPF) and distal LAF were also mapped before radiofrequency application. FP-V interval was measured as 29 ms during SR versus 17 ms during PVCs at the site of the proximal LPF (Figure 2B). The FP-V interval was the same (18 ms) at the distal LAF during SR and PVCs (Figure 2D). At 4-month follow-up, the patient was free of ventricular arrhythmias without the use of any antiarrhythmic drugs.

Case 2
A 54-year-old man without structural heart disease was referred with a 6-year history of junctional tachycardia that was initially treated with diltiazem, metoprolol, and flecainide. These medications did not resolve his symptoms but caused significant fatigue and were, therefore, discontinued. Subsequently, his palpitations became incessant. The 12-lead ECG demonstrated a relatively narrow QRS tachycardia with atrioventricular dissociation and capture beats with normal QRS morphology (Figure 3A, arrow). Electrophysiology study and mapping were performed with a 4-mm ablation catheter via a retrograde aortic approach. The H-V interval was 35 ms during tachycardia and 65 ms during capture beats (Figure 3B). FP-V interval was recorded at 40 ms both during beats of tachycardia and capture beats at the proximal LAF (Figure 3C). Radiofrequency was applied at the proximal LAF with 30 W at 60°C during overdrive atrial pacing from the coronary sinus at a shorter cycle length than tachycardia to monitor atrioventricular nodal conduction. QRS morphology was noted to change with development of left axis deviation during radiofrequency application (Figure 4). VT termination was observed after cessation of atrial pacing. The patient has remained free from tachycardia in follow-up without the use of antiarrhythmic drugs for over 4 years.

Discussion
Left upper septal fascicular VT often presents as a relatively narrow QRS complex tachycardia. The His bundle electrogram usually precedes the QRS complex during tachycardia or PVCs with a significantly shorter H-V interval compared with the H-V interval during SR.2 The difference in H-V interval between SR and tachycardia or PVCs reflects the distance between the His bundle and ectopic site of origin, assuming a focal source or area of microreentry. The greater the difference, the more distal the site of origin within the fascicular system. This also assumes that the antegrade and retrograde conduction velocities within the fascicular system are similar. Detailed mapping of the proximal LAF and LPF should be performed with comparison of FP-V interval between SR and tachycardia or PVCs. A similar FP-V
interval between SR and tachycardia with earliest activation time is an indication of the site of origin. The FP-V intervals were measured at different sites within the left bundle branch during SR and PVCs in case 1. The FP-V interval was identical during SR and PVCs at both the proximal and distal LAF, with a relatively shorter FP-V interval at the distal LAF (Figure 2D). However, when the mapping electrode was placed at the LPF, there is a shorter FP-V interval during the PVCs compared with SR (Figure 2B). Ablation sites, where the FP-V interval is ≥20 ms less than the H-V interval during SR, may have less risk of producing atrioventricular block or left bundle branch block. Catheter ablation can produce LAF or LPF block without eliminating tachycardia or PVCs indicating that the ectopic origin is located more proximal in the fascicle or in a different branch. Ablation during tachycardia may carry a higher risk of atrioventricular block because of proximity of the His bundle region and the risk of atrioventricular block during ablation. The measurement and comparison of the FP-V interval during SR and tachycardia is of significant utility and establishes a proximal versus distal site of origin, while facilitating accurate mapping of the tachycardia focus.

Left upper septal ventricular tachycardia is caused by either triggered activity or localized reentry within the fascicular system. Changes in QRS axis and morphology following ablation were noted in both cases indicating block in the proximal LAF. The observation of similar FP-V interval between SR and tachycardia or PVCs at the ectopic origin may not apply to tachycardia or PVCs originating from the distal LAF or LPF if the mechanism of tachycardia is macroreentry or delayed conduction within the fascicular branches.

Conclusion
We describe 2 cases of left upper septal VT that were successfully ablated at the proximal left anterior fascicle. Mapping of this region can be challenging because of proximity of the His bundle region and the risk of atrioventricular block during ablation. The measurement and comparison of the FP-V interval during SR and tachycardia is of significant utility and establishes a proximal versus distal site of origin, while facilitating accurate mapping of the tachycardia focus.

Disclosures
None.

References

Key Words: catheter ablation ■ electrophysiology ■ ventricular arrhythmia

Figure 1. A, Twelve-lead ECG recording of premature ventricular beats with a relatively narrow QRS and incomplete right bundle branch block pattern. B, Twelve-lead ECG recording after ablation demonstrates block within the left anterior fascicle.

Figure 2. Intracardiac recording during SR and PVCs with mapping electrode positioned at the His bundle (A) and LPF (B). The H-V interval (A) and FP-V interval (B) during PVCs were shorter than measured during SR indicating that the ectopic site originates below the His bundle or along a different branch of the fascicle. Note also in A, the reversal of polarity in the His potential between a conducted sinus beat and PVC. Mapping electrode placed at the site of successful ablation in the proximal LAF (C) and the distal LAF (D). During mapping of the proximal and distal LAF, the FP-V interval during PVC is identical to the SR but is earliest at the proximal LAF. Solid circle represents the ectopic focus. ABL indicates ablation electrode; AVN, atrioventricular nodal; CS, coronary sinus; HB, His bundle; H-V, His bundle to onset of surface ventricular; FP-V, fascicular potential to onset of surface ventricular; LAF, left anterior fascicle; LPF, left posterior fascicle; PVC, premature ventricular contraction; RB, right bundle; RBB, right bundle brunch; and SR, sinus rhythm.
Figure 3. ECG (A) and His bundle recordings (B) during VT. The QRS morphology during VT is relatively narrow with atrioventricular dissociation and a capture beat (A, arrow). With a capture beat, the H-V interval is longer than that measured during VT (B). VT was successfully ablated at left ventricular upper septum. At this site, the FP-V interval is 40 ms during VT, identical to the interval during a capture beat (C). ABL indicates ablation electrode; CS, coronary sinus; FP-V, fascicular potential to onset of surface ventricular; H-V, his bundle to onset of surface ventricular; and RVA, right ventricle apex.

Figure 4. Intracardiac recording during radiofrequency application at the proximal left anterior fascicle with overdrive atrial pacing from the CS. Note the change in QRS morphology with development of greater left axis deviation during RF application, indicating block in the LAF (arrow). ABL indicates ablation electrode; CS, coronary sinus; HRA, high right atrium; RF, radiofrequency; and RVA, right ventricle apex.
EDITOR’S PERSPECTIVE

How does one teach a concept or procedure when even experts do not know how best to address the problem at hand? An expert teacher overcomes this difficulty not by presenting a solution but rather presenting the problems. In this issue of *Circulation: Arrhythmia and Electrophysiology*, Shehata et al provide an elegant new look at how to use routinely recorded electrograms to identify mechanisms and pick an appropriate ablation target in a form of fascicular tachycardia. They highlight for the student the reasons why fascicular arrhythmia is difficult to ablate and why such creative techniques are required.

Why Is Fascicular Arrhythmia Difficult to Ablate?

Fascicular tachycardias can be due to macroreentry, microreentry, or, less commonly, automaticity. Familiar mapping methods to identify an appropriate ablation target include finding the earliest site of activation, entrainment mapping, and comparing activation sequence in tachycardia, assessed by the electrocardiogram or intracardiac electrograms, with those during pacing from a target site. 

Mapping the Earliest Ventricular Electrogram (V)

Comparing an inter-electrogram interval obtained in tachycardia with the same interval measured with pacing can be a useful technique to identify the ability to capture the tissue generating the local signal that is being questioned as to whether it is in the circuit or not. Because of the overlap of the fascicular tissue and the ventricular myocardium, capturing one without the other is difficult and not reproducibly done. Thus, one may compare the fascicle, the ventricular myocardium, or both, and this may vary from beat to beat. One may be entraining within a fascicular reentrant circuit, but because of local myocardial capture, manifest fusion may be noted.

Comparing Intervals

Comparing an inter-electrogram interval obtained in tachycardia with the same interval measured with pacing can be a useful technique to identify an appropriate target site. For example, in a macroreentrant myocardial ventricular tachycardia, if the local electrogram to the onset of the QRS approximates the same interval when pacing and capturing the tissue giving rise to the local electrogram, then the pacing site is likely in the reentrant circuit. This is similar to comparing the retrograde His-A interval during ventricular pacing versus tachycardia to help distinguish junctional tachycardia from a conducted rhythm or AV node reentry. Once again, however, with fascicular arrhythmia, these maneuvers are not straightforward to perform because local capture typically involves the underlying fascicle, as well as the adjacent myocardium. 

Entrainment Mapping

With fascicular arrhythmias, the reentrant circuits may be complex, ranging from rather localized microreentry through a continuum to large macroreentrant circuits such as bundle branch reentry. Entrainment mapping is potentially useful for interrogating sites in complex circuits but requires the ability to capture the tissue generating the local signal that is being questioned as to whether it is in the circuit or not. Because of the overlap between the fascicular tissue and the ventricular myocardium, capturing one without the other is difficult and not reproducibly done. Thus, one may capture the fascicle, the ventricular myocardium, or both, and this may vary from beat to beat. One may be entraining within a fascicular reentrant circuit, but because of local myocardial capture, manifest fusion may be noted.

Earliest Fascicular Signal

Since mapping the earliest V is unlikely to work when ablating fascicular arrhythmia, what about mapping the earliest Purkinje potential or fascicular signal? This approach is potentially limited for 2 reasons. When macroreentrant, the earliest site has no relevance, as one needs to identify a critical component of the circuit to target for ablation. Secondly, although microreentrant tachycardia may be considered to be a localized or focal source, because of the diverse ramifications of the infra-Hisian conduction system, very late activation from a previous beat can be difficult to distinguish from a truly early site of activation. For example, if a signal on a papillary muscle is found 200 ms prior to the onset of the surface QRS, and another signal on the anterior septum is found 180 ms or 100 ms prior to the surface QRS, one does not know whether the origin is on the anterior septum, and activation of the papillary muscle fascicle is late (after inscription of the surface QRS) or very early. Thus, although identifying a fascicular signal at the site is a prerequisite for successful ablation, it is difficult to know whether an apparently early signal is from the arrhythmia substrate or simply a bystander.

Three caveats, however, should be noted. (1) There is an assumption that antegrade and retrograde conduction in any given limb of the infrahisian conduction system is about equal. While they note that there is a difference in the contralateral, presumably with the geometric complexity of the fascicles (wavefront curvature, concealed penetration, etc), this may not always be the case. (2) Importantly, a conducted sinus beat may reach the V through the right bundle or any branch of the left bundle. Thus, if there is delayed conduction through the arrhythmogenic fascicle, then the H-V (or F-V) during sinus rhythm reflects conduction to the earliest ventricular site which may be distant to the earliest ventricular exit site in tachycardia such that analysis of these intervals is not a reliable guide to ablation. Perusal of the QRS morphology can suggest when this limitation is present, for example, left bundle branch block in sinus rhythm and right bundle superior axis during tachycardia. In the cases presented by Shehata et al, the QRS morphology during sinus rhythm and fascicular tachycardia is similar, possibly related to either intrinsic or in one case inadvertent catheter trauma to the right bundle branch. (3) For an automatic focus in the fascicle, sites below the focus will have a matching sinus and tachycardia F-V intervals. Shehata et al elegantly demonstrate a classic tenet of a successful teacher on clinical rounds. They expose and explore present limitations in mapping methods for this difficult arrhythmia and suggest a new and useful method that can potentially solve the problem for some cases.

References

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