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Electrocardiographic Findings of Fascicular Ventricular Tachycardia Versus Supraventricular Tachycardia With Aberrancy

Why the Difference?

See Article by Michowitz et al

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Idiopathic ventricular tachycardia (VT) using the left posterior fascicle can be easily mistaken for supraventricular tachycardia (SVT) with right bundle branch block (RBBB) and left anterior fascicular block (LAFB), and distinguishing these entities via ECG analysis is essential for appropriate management. Discussion of treatment options and risks and procedural planning depend on accurate ECG diagnosis. Preparation for an approach in the event of noninducibility at electrophysiological study must also be considered—empirical slow pathway ablation might be considered in some patients with SVT and dual-atrioventricular nodal physiology,¹⁻⁴ while a linear ablation strategy can be used for left posterior fascicular VT (LPF-VT).⁵ Numerous well-known ECG criteria have been developed to distinguish VT from SVT with aberrancy,⁶⁻⁹ though LPF-VT may lack some features typically ascribed to VT, by virtue of involvement of the conduction system. In fact, these criteria were recently shown to have reduced sensitivity for differentiating idiopathic VT in patients without structural heart disease from SVT with aberrancy.¹⁰ In 39 patients with idiopathic VT and a RBBB morphology, 79% received a correct diagnosis of VT based on conventional ECG criteria, while 21% were deemed indeterminate, all based on conflicting morphological criteria between leads V1 and V6. On the other hand, 14% of ECGs showing wide-complex SVT were misclassified as VT or felt to be indeterminate.

In this issue of *Circulation: Arrhythmia and Electrophysiology*, Michowitz et al¹¹ analyzed 183 ECG tracings with LPF-VT and 61 ECG tracings with RBBB and LAFB to determine distinguishing characteristics. The authors combined 144 ECG tracings of LPF-VT confirmed via electrophysiological study from the literature with 39 from their own ablation experience, excluding patients with structural heart disease or poor-quality ECGs. The RBBB and LAFB tracings were chosen from patients in sinus rhythm, including 16 who underwent electrophysiological study with atrial pacing at rates ≥ 100 beats per minute. Four variables were found to be independently associated with a diagnosis of LPF-VT: positive QRS in aVR, QRS width ≤ 140 ms, R/S ratio ≤ 1 in V6, and atypical V1 morphology (no rsR', or R larger than R' in V1). A nomogram was created based on logistic regression, assigning a point value to each criterion and a probability of LPF-VT based on total points; using a threshold probability cutoff of 0.59 yielded a sensitivity of 82.1% and specificity of 78.3% for predicting LPF-VT. No patients with LPF-VT had only 0 or 1 of the 4 described criteria.

Compared with traditional ECG criteria for distinguishing VT, several interesting findings were noted. The QRS duration in LPF-VT was shorter than with RBBB and LAFB, in contrast to the conventional wisdom for VT when associated with structural heart disease. Atypical RBBB morphology was included in the prediction model, but more than half of the patients with LPF-VT demonstrated a typical morphology in

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V1. Similarly, while an R/S ratio ≤ 1 in V6 supported a diagnosis of LPF-VT, it was also noted in 59% of patients with RBBB and LAFB—the S wave in V6 can be deeper because of apical to basal depolarization of the LV during VT or because of LAFB. An R/S ratio < 0.15 in V6 was exclusively diagnostic of LPF-VT, as was a QRS axis $< -100^\circ$.

Overall, the authors reported lower sensitivity and specificity for their algorithm when compared with traditional algorithms for differentiating wide-complex tachycardia in structural heart disease. Ultimately, the sensitivity for LPF-VT was similar to that found using traditional criteria in a cohort of patients with idiopathic VT of varied etiologies (including right ventricular outflow tract, left ventricular outflow tract, LPF-VT, and other sites or origin such as the posterior papillary muscle).¹⁰ However, the exclusive focus on LPF-VT by Michowitz et al¹¹ was, by design, more challenging, as was purposeful exclusion of atrioventricular dissociation as a criterion.

The authors report that patients with RBBB and LAFB who underwent atrial pacing ≥ 100 beats per minute had similar ECG findings to the nonpaced sinus rhythm control group. However, heart rates were still significantly lower compared with the LPF-VT examples. In our view, this represents an excellent area for additional investigation. It has been previously shown that most patients with preexisting bundle branch block demonstrate significant changes in QRS configuration with rapid atrial pacing, though many of these patients also had structural heart disease.¹² The likelihood of seeing significant QRS changes increased with heart rate, and half of the patients had major changes at rates > 150 beats per minute. Common changes in a baseline RBBB pattern in response to overdrive pacing included decrease in the amplitude of the initial R wave in V1 (and V2) and increase in the amplitude and duration of R' (sometimes even becoming a monophasic R wave with or without a notch and sometimes with shortening of the QRS duration). Obscuration of initial QRS morphology by the preceding T wave at rapid rates can also occur.⁸ These changes could feasibly cause a typical RBBB pattern that might otherwise be correctly identified as aberrancy at lower rates to meet 2 of the criteria for LPF-VT at higher rates: atypical appearance in V1 and narrower QRS.

A critical question raised by this and other studies remains: how does depolarization of the ventricles actually differ between LPF-VT and SVT with RBBB and LAFB? Furthermore, should we expect uniform surface ECG properties for either entity? In the case of LPF-VT, earliest activation of the left ventricle typically arises from the LPF, but contribution of the left anterior fascicle (LAF), left septal fascicle, and even the right-bundle branch are not necessarily excluded. Liu et al¹³ postulated a macroreentrant circuit comprising a decremental P1 fiber that connects to the LPF at its distal portion and serves as the antegrade limb, a portion of the LPF, septal ventricular myocardium that serves as the retrograde limb, and a

zone of slow conduction between the myocardium and the proximal portion of P1 (Figure). They found that the site of merged P1 and P2 (the latter representing the LPF) could be predicted by the V-H interval during tachycardia—the longer the V-H interval, the longer the P1 segment along the left ventricular septum and the more distal the location of the earliest P2. In such a model where bystander retrograde conduction can continue along the portion of the LPF proximal to the connection of P1, varying degrees of ventricular depolarization and slight surface ECG fusion from antegrade LAF, left septal fascicle, and right-bundle branch conduction during tachycardia are feasible. Such fusion might even be expected to generate a slightly narrower QRS than might otherwise be seen with pure LPF activation, a concept that may be further supported by the demonstration of less common but relatively narrow QRS nonreentrant VT arising from the LPF.¹⁴ Earlier activation of the lateral LV via the LAF could also contribute to positivity in aVR.

Conversely, in SVT with RBBB and LAFB, our natural inclination is to assume complete conduction block in

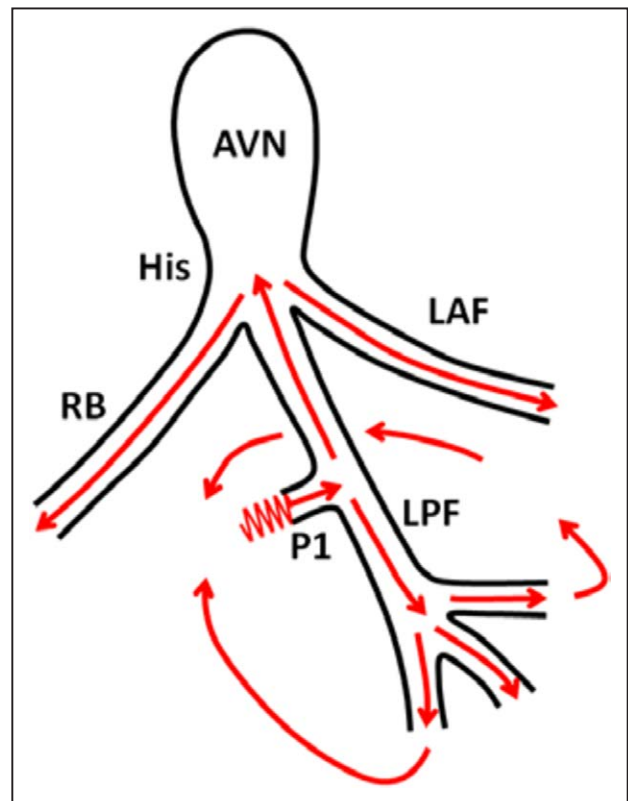


Figure. Schematic diagram of the left posterior fascicular tachycardia (LPF-VT) reentry circuit.

Adapted from Liu et al¹³ with permission of the publisher. Copyright © 2016, American Heart Association, Inc. Authorization for this adaptation has been obtained both from the owner of the copyright in the original work and from the owner of copyright in the translation or adaptation. AVN indicates atrioventricular node; LAF, left anterior fascicle; LPF, left posterior fascicle; and RB, right bundle branch.

the right bundle branch and left anterior fascicle, with ventricular depolarization initiated entirely via antegrade conduction of the LPF and left septal fascicle. However, it is possible that some residual slow conduction in the fascicles remains. Analogous pseudoblock is often discovered when treating bundle branch reentrant VT—even in many patients with presumed complete antegrade left bundle branch block, elimination of right bundle conduction via ablation induces a RBBB ECG pattern rather than complete atrioventricular block. It stands to reason, therefore, that depolarization of the ventricle (and the resulting surface ECG pattern) with RBBB and LAFB can vary based on the degree of residual slow conduction in either the right-bundle branch or the LAF and how the differential conduction properties of the fascicles may vary based on heart rate. Anatomic variability in the morphology of the left fascicular bundles, particularly with regard to site of origin of a left septal fascicle, may further contribute to variability in ventricular depolarization.¹⁵ All of these factors may help explain some of the nonuniformity of surface ECG findings between patients.

The authors are to be congratulated for adding another valuable tool to our armamentarium for correctly distinguishing LPF-VT from SVT with RBBB and LAFB, and particularly for its applicability in clinical practice—the criteria specified are simple, easy to remember, non-ambiguous, and not dependent on identifying dissociated atrial activity (the presence of which, in practice, is not infrequently debated among different observers). It will be important to see how the algorithm performs prospectively (especially in the case of more rapid SVT with aberrancy, when the RBBB pattern may change) and whether variations in LPF-VT morphology can help predict the site of successful ablation.

AFFILIATIONS

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DISCLOSURES

None.

FOOTNOTES

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