In this installment of Teaching Rounds in Electrophysiology, King et al clearly demonstrate and succinctly discuss a classical maneuver to identify the mechanism of a wide complex tachycardia occurring in a patient with known preexcitation. The similar axis of tachycardia and the sinus rhythm preexcited ECG suggests a preexcited tachycardia, and antidromic tachycardia was indeed the final diagnosis. However, the presence of some unusual features, such as lack of effect of adenosine, suggested the possibility of some less common causes, such as pathway-to-pathway tachycardia or preexcitation, resulting from a bystander antegrade conducting pathway. Their discussion emphasizes the importance of the location of the pacing site for placing atrial premature beats during tachycardia and the difference in interpretation based on this location—probable diagnosis versus definite diagnosis.

Why Are Wide QRS Tachycardias More Difficult to Define?

Most electrophysiologists are thoroughly familiar of the response to premature ventricular contractions placed during a narrow complex tachycardia, but finding interpreting the analogous maneuver of PACs during a wide complex QRS tachycardia more challenging.

Atrioventricular Node Has No Recordable Signals

The key to most diagnostic maneuvers is defining whether conduction through or involvement of the AV node in a tachycardia is present. However, despite the tremendous advances in mapping technology over the last 4 decades, we are unable to directly record signals from and electrically identify the location, participation of, and conduction through the AV node. As a result, we use an inexact surrogate, that is, the atrial myocardium in the vicinity of the normally located AV node. One needs to appreciate the need for specifically located myocardium to input to the AV node, as well as knowledge of the location of both the site of PAC placement and the site of the septal myocardium recording electrodes. Unexpected findings and exceptions can occur if the AV node has an unusual location.

Intra-Hisian Sequence

We routinely record His bundle electrograms on multielectrode catheters. Is the His bundle recording not a reliable surrogate for the AV node when PACs are placed during WQRS tachycardia? The His bundle may be activated either antegrade via the AV node or retrograde via an antegrade conducting pathway during wide QRS tachycardia, and therefore, a single His bundle recording does not reliably indicate whether
antegrade engagement of the AV node occurred when a PAC was introduced.

What about the intra-Hisian sequence? With multielectrode recording, we should be able to distinguish antegrade from retrograde His bundle activation. For example, in the case discussed by King et al,1 the intra-Hisian sequence should have distinguished antidromic reentry with retrograde His and AV node engagement from AV nodal reentrant tachycardia with bystander pathway presence (antegrade His sequence). However, the sequence alone on multipolar His bundle recordings is also not definite because some of the electrodes may be recording the right bundle branch (or left bundle branch) electrograms, and in the presence of proximal right bundle branch block in the antegrade or retrograde direction, sequences are difficult to interpret. For example, in this situation, an antegrade His bundle sequence may be found when the recording electrodes are on the right bundle during antidromic tachycardia with a Mahaim-type pathway and pathway insertion to the proximal right bundle.3

Black Boxes
Because of these fundamental limitations of our techniques in recording AV nodal activation, black boxes, or essentially undefinable mechanisms of arrhythmia, exist and are often the focus of controversy. For example, a PAC placed on the right atrial free wall during a wide QRS tachycardia that does not affect the septal atrial electrogram but advances the QRS without a change in morphology and pre-exites the tachycardia although also advancing the next septal atrial electrogram is said to be diagnostic of antidromic tachycardia, such as with an antegrade Mahaim fiber. This finding is said to exclude a nodoventricular tachycardia (because of the need to engage the septal myocardium).2 We also think that Mahaim fibers are not found on the septum. However, in the absence of definitive histological evidence, and based on the way we diagnose Mahaim antidromic reentry tachycardia (should not advance the septal A...), we would never know whether these accessory AV node-like structures may indeed be found at or near the septum and simply be called nodoventricular tracts. In addition, abnormal placement of the AV node outside of the triangle of Koch or extensions from a portion of the compact AV node, such as a slow pathway input directly into the ventricular myocardium, partial isolation, and entrance block to the atrial myocardium near the AV node, can only be conjectured and not defined with existing maneuvers.

Extrapolation
Although trainees sometimes question the value of detailed understanding of the classical maneuvers applied to rare arrhythmias, an understanding of these maneuvers in these situations can facilitate understanding and the use of maneuvers in more common settings.

Location and AF Maneuvers
A common error when pacing to identify the presence of right superior pulmonary vein potentials is to pace from the distal coronary sinus. The far-field electrograms seen on a circumferential mapping catheter placed in the right superior pulmonary vein are from the right atrium. Pacing from the distal CV may create a wavefront that simultaneously enters the right superior pulmonary vein and the right atrium, resulting in overlapping signals and misidentifying entrance block into this vein. Pacing close to the connection and ascertaining whether the electrograms at an adjacent site were advanced or not clarifies the source of different electrograms.4,5

Location and Identifying Ventricular Tachycardia Substrate
When multiple ventricular tachycardias or hemodynamically unstable rhythms are induced, a substrate-based approach can be effective in eliminating clinical and presently nonclinical but future arrhythmogenic sites.6,7 Substrate mapping involves identifying late potentials, fragmented signals, and careful measurement of the local electrogram amplitude. However, late potentials or slow conduction (fragmented electrograms) may be absent during sinus rhythm because of multiple wavefronts entering into the arrhythmogenic substrate or from a particular location for pacing because of unidirectional conduction block to the culprit sites.8 Choosing the right location for pacing and comparing maps produced by pacing at different sites may be time consuming, but may help clarify conduction pathways. The lessons derive from an appreciation of the importance of pacing site location in the classical maneuvers.

Location, Output, and Timing
In addition to recognizing the importance of pacing site location, it is important to realize that varying the output for stimulation and the timing relative to activation during tachycardia is critical. For example, para-Hisian pacing leverages the differences in response based on output, but exact location of pacing may be critical when interpretation is not straightforward: as when pacing captures the atrium directly or when there is block in the proximal right bundle branch and the pacing site is distal to a site of block. Timing (or pacing rate) may also determine the findings and can be a critical factor when interpretation is not straightforward. Similarly, pacemapping at or near the conduction system when identifying ventricular fibrillation substrate or fascicular tachycardia requires careful analysis of the remarkable interplay between the location, output, and timing when defining targets for ablation.9,10

King et al1 elegantly discuss the relevance of location when performing a common diagnostic maneuver for defining wide complex tachycardia mechanism. We encourage the student of electrophysiology to routinely consider the importance of pacing site location for diagnostic maneuvers because an in-depth understanding will facilitate your ability to reach the correct diagnosis and identify the appropriate ablation target for a variety of arrhythmias.

Disclosures
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References

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