Wobble

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Spontaneous changes in the measured intracardiac intervals during tachycardia (wobble) are useful in determining the mechanism of arrhythmia but require careful interpretation. The changes may be seen at induction of tachycardia, prior determination, or just after pacing maneuvers are deployed. There are 3 reasons for difficulty with interpreting wobble:

- The measured interval may be a true conduction interval indicating the conduction time for activation progressing from the site giving rise to the first potential to the site giving rise to the second potential, or a pseudointerval, each with a different interpretation during wobble.
- More than 1 arrhythmia and >1 mechanism for a given interval may be operative.

When the direction of His activation is unclear, atrial decremental pacing or extrastimulus testing may lend clarity. With antegrade atrioventricular nodal conduction in the presence of pre-excitation, the A–H continues to increase with progressive incremental pacing or shorter coupled premature atrial contractions (PACs). At some point, the A–H becomes fixed and no longer decrements, and this finding is then diagnostic of retrograde activation of the His bundle. However, continued decrement is sometimes seen despite retrograde activation via an accessory pathway when the antegrade conducting accessory pathway itself is decremental.

Sequence of His Bundle Activation

When a multielectrode catheter is placed spanning the His bundle and proximal right bundle branch, the sequence of activation (proximal to distal or vice versa) should be indicative of whether the His is activated antegrade or retrograde. However, cautious interpretation is required with careful attention to whether the recorded sequence of electrograms is all in the proximal and distal right bundle branch or truly span the length of the His bundle itself.

Antegrade Activation With Distal-to-Proximal His Sequence of Activation

When the multielectrode catheter is placed on the right bundle distal to a site of proximal right bundle branch block, then antegrade conduction of the His will proceed through the left bundle and retrogradely activate the right bundle. Here, despite antegrade atrioventricular nodal conduction, a distal-to-proximal sequence will be seen on the recording electrodes.

Retrograde Activation of His Bundle but With Proximal-to-Distal Sequence of Activation

In either a pre-excited tachycardia or atrial pacing with maximal pre-excitation, if the accessory pathway connects to the proximal right bundle and the multipolar recording electrodes are all distal to the site of pathway insertion, then a proximal-to-distal conduction signal recording sequence will be seen despite the fact that the atrioventricular conduction system is activated retrograde.

In the case of Foreman et al, the sequence of activation could not be determined, as the His signal was recorded only on the distal electrode. With care to identify the exact location of the catheter, obtaining signals on the multiple electrodes and

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performing the incremental pacing maneuvers described previously, in most instances, one can identify whether the atrioventricular node and His are activated antegrade or retrogradely.

**V–H Interval**

Foreman et al describe how they used the change in the V–H interval to identify the mechanism of tachycardia in their patient. The interval between the onset of ventricular activation and the recorded His bundle electrogram is a true conduction interval with an antegrade conducting atrioventricular type of accessory pathway. Here, the V–H interval may prolong abruptly when retrograde right bundle branch block occurs, and the atrioventricular accessory pathway is located on the right side or with retrograde left bundle branch block and a left-sided atrioventricular accessory pathway. If with such V–H prolongation the tachycardia slows and there is lengthening of the V–A interval without a change in the activation sequence, the retrograde limb of the pathway can be diagnosed to be the atrioventricular conduction system (true antidromic tachycardia). However, with V–H prolongation, if the V–A interval remains fixed, then the retrograde limb is likely a second accessory pathway and the mechanism of tachycardia a pathway-to-pathway tachycardia.

With atriofascicular pathways, the V–H interval is a pseudo-interval since the ventricle is activated antegrade from the point of insertion of the pathway to the conduction system, and the His retrograde from the same point. However, as shown and discussed by Foreman et al, typically with antidromic atriofascicular reentry, the V–H interval is short, but if it prolongs abruptly there is likely retrograde right bundle branch block proximal to the site of insertion of the atriofascicular tract. Here again, similar logic can be applied to define the retrograde limb.

The authors importantly point out for us the diagnostic value of placing PACs on the right atrial free wall during tachycardia and showing that the tachycardia can be advanced without affecting septal atrial activation, with no change in QRS morphology, and without changing the retrograde sequence of activation (thus confirming antidromic reciprocating tachycardia). This observation is important because the observations of a retrograde V–H change, resetting the supraventricular tachycardia does not exclude the simultaneous existence of atrioventricular nodal reentrant tachycardia (AVNRT) and antidromic reciprocating tachycardia with an atriofascicular tract. In that case when the V–H is short, the antidromic reciprocating tachycardia cycle length is shorter than the intrinsic cycle length of the AVNRT and thus entrains the AVNRT. With retrograde bundle branch block and V–H lengthening, the AVNRT circuit is faster and the pathway is a bystander only responsible for the wide QRS complex.

**H–A and Right Bundle–A Interval**

The H–A interval is a pseudointerval (no conduction from His to A) in most tachycardias. For example, in AVNRT, the H–A interval is determined from relative conduction times to the His and the A from a central site of activation, and in atrial tachycardia, the relative time relating conduction through the atrioventricular node and the inherent rate of the tachycardia itself will determine the H–A interval. The H–A interval is a true conduction interval in antidromic tachycardia, including those using an atriofascicular pathway that inserts distal to the recorded His bundle electrogram. However, the right bundle-to-A interval may be a pseudointerval in atriofascicular and atriohisian pathway-related antidromic reciprocating tachycardia when the pathway inserts proximal to the right bundle or when an atriofascicular pathway inserts distal to the right bundle but retrograde right bundle branch block is present.

**H–V Interval and Right Bundle–V Interval**

The H–V interval is a true conduction interval and part of the circuit in orthodromic reciprocating tachycardia. The H–V is also a true conduction interval in the rare occurrence of an atriohisian pathway being used for antegrade conduction with retrograde atrioventricular nodal block, and the retrograde limb of the tachycardia being completed by another accessory pathway conducting in the retrograde conduction (pathway-to-pathway tachycardia with retrograde atrioventricular block). The H–V interval is unrelated to the tachycardia circuit with atrioventricular node reentry, atrial tachycardia, junctional tachycardia, etc. Notably, the H–V interval is a pseudointerval with bundle branch reentry and with antidromic tachycardia associated with an atriofascicular tract. This is because with bundle branch reentrant tachycardia the H and V are activated from a central point representing the turnaround of the tachycardia circuit near the proximal right bundle, and in atriofascicular related tachycardia, the H and V are activated from a central point representing the insertion site of the atriofascicular tract. However, the right bundle–V interval is a true conduction interval with bundle branch reentry because the right bundle is part of the circuit for this arrhythmia.

**Atrial Stimulation**

The authors highlight the critical element of the maneuver described by Tchou and Akhtar when PACs are placed during a wide complex tachycardia to elucidate mechanism. The PACs need to be delivered when the atrial septum in the vicinity of the atrioventricular node is refractory to exclude the possibility that the stimulated wavefront conducts antegrade over the atrioventricular node. The atrial septum is used as a surrogate for atrioventricular nodal activation because the His bundle may be activated antegrade or retrograde, and we are unable to record signals directly from the atrioventricular node.

**Difficult Diagnoses**

Despite careful measurement and exact analysis of the spontaneous changes in the intracardiac intervals during tachycardia (wobble), diagnoses cannot always be made. Coexisting tachycardias, as mentioned by Foreman et al, are particularly difficult to diagnose. When one tachycardia such as an automatic atrial tachycardia induces and entrains another such as a reentrant atrial flutter, diagnostic maneuvers will simply show evidence of both (entrainment and overdrive suppression). When wobble occurs in this situation, however, in addition to the usual interpretations described above, one must need to consider temporary termination.
of one of the tachycardias and reinduction or alternation or change in which the faster tachycardia is entraining the slower tachycardia circuit.

Another difficult diagnosis involves septal decremental pathways. When PACs can advance the next V and reset the tachycardia without a change in the activation sequence only when pre-exciting the septal A, we consider a nodoventricular or nodofascicular tract as the likely diagnosis. However, this maneuver does not exclude a septal decremental pathway, including a septal Mahaim fiber. Some electrophysiologists describe Mahaim fibers only on the free wall, but this may be an artifact for how we diagnose this arrhythmia. Importantly, Foreman et al describe finding the successful site that they targeted for ablation by identifying the pathway potential and showing that the pathway potential participated in the tachycardia. This is an important teaching point for students of electrophysiology, even with the difficult situation of a decremental fiber located on the septum or dual tachycardias. If the pathway potential can be definitively diagnosed and shown to participate in the tachycardia, an excellent target for possible ablation has been found.

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, and Elsevier. Dr Stevenson is coholder of a patent on needle ablation that is consigned to Brigham and Women’s Hospital.

References

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