Teaching electrophysiology is mostly about preparation, learning as much as possible about a tachycardia before delivering the energy source for ablation. This includes the history, the physical examination, careful review of the ECG and recorded tracings, and analysis and correct execution of maneuvers to identify tachycardia mechanisms accurately. However, at times the electrophysiologist learns while burning. Slowing or acceleration of an automatic tachycardia, decrease in electrogram amplitude, or evidence for collateral damage while ablating are all important to recognize. In this segment of Teaching Rounds in Electrophysiology, Kumar and Gehi discuss what we can learn when ablating reentrant arrhythmia based on changes noted in the tachycardia characteristics and activation sequence: a change in the cycle length of tachycardia without a change in the activation sequence, abrupt changes in the activation sequence but tachycardia continuing at the same rate, or occasionally changes in both rate and sequence despite the original tachycardia continuing.

Change in the Sequence of Tachycardia but the Same Cycle Length
While burning, when the tachycardia cycle length stays exactly the same, likely the same tachycardia continues, but why would there be an abrupt change in the activation sequence?

- Bystander loop: In patients with previous surgery and with increasing frequency, previous multiple atrial ablations, several areas of slow conduction, scar, and potential for circuits exist. For example, during a left atrial perimital flutter, there may be passive entrainment of the cavitricuspid isthmus (CVTI). Here, conduction enters from the left atrium posteriorly in the vicinity of the coronary sinus and activates the CVTI in a counterclockwise fashion. There may be simple bystander conduction across the CVTI or actual induction and continuous resetting (entrainment) of the induced CVTI flutter by the more rapid left atrial flutter. When ablating across the cavitricuspid isthmus with a multielectrode catheter placed on the lateral and inferolateral right atrial wall, an abrupt change in activation sequence (from counterclockwise to clockwise) may occur as a result of obtaining block across the CVTI. However, the tachycardia continues at the same rate. The change in the ECG may be subtle, but the local electrogram sequence where the multielectrode catheter has been placed is easily seen.
- Focal driver: In a similar fashion, automatic tachycardias, including from the pulmonary vein or superior vena cava, may be the inducing and entraining source for a protected isthmus-dependent flutter (eg, CVTI). Again, during ablation, the local activation sequence changes; however, the underlying tachycardia continues unabated at the same cycle length.

Just as with ablating a bystander accessory pathway (eg, AVNRT (atrioventricular nodal reentrant tachycardia) with bystander accessory pathway), when tachycardia continues despite evidence of local ablation, it is probably best to continue the ablation until the local electrograms have sufficiently fragmented or decreased amplitude because this site may be a primary source of tachyarrhythmia even without the focal driver or primary reentrant loop.

Change in the Cycle Length While Maintaining an Identical Activation Sequence
When ablating across a critical isthmus for reentrant tachycardia, the width of the isthmus progressively narrows. As an isthmus narrows, wavefront curvature increases, conduction velocity slows, and the cycle length increases while maintaining the same sequence of activation distal to the slow site. When the ablation line is completed, anchored to a scar or anatomic obstacle, the tachycardia terminates.

Occasionally, however, there may be a fairly sudden change in the cycle length, and even with continued ablation to an anchor point, the tachycardia does not terminate. For example, when ablating between the mitral annulus and the ablation circle around the left lower pulmonary vein for perimital flutter, the wavefront (say, clockwise) can enter through the progressively narrower isthmus being created or through gaps in the perivenous ablation circle that may be present. If the slow zone created with ablation is slower than conduction through the gaps in the ablation circle, then the tachycardia wavefront goes through these perivenous gaps and completes the mitral isthmus circuit. Depending on the location of the recording electrodes, no discernible change in activation sequence may be seen. Despite continuing the ablation line appropriately with anchors to the mitral annulus and the left lower pulmonary vein ablation circle, the tachycardia will continue. Entrainment characteristics along the perimital region remain the same (concealed entrainment), and if the operator does not
recognize the possibility that ≥2 gaps in the ablation circle are present and being used for maintenance of the perimtrial flutter, then unnecessary extensive ablation along the mitral isthmus may be continued.

**Change in the Cycle Length and Activation Sequence**

In general, if both the cycle length and activation sequence of an arrhythmia change, then the arrhythmia itself is likely changed. Occasionally, however, identical tachycardia may change rate and sequence when ablating as a result of some variant or manifestation of longitudinal dissociation along an isthmus. For example, with previous ablation of the CVTI, the isthmus itself may be split longitudinally into an anterior periventricular component and a posterior pericaval component. If the ablation electrodes are placed on the anterior portion of the isthmus and the ablation line carried back from the tricuspid annulus, a block along the anterior isthmus may occur, resulting in abrupt change in activation sequence, and because conduction now goes posteriorly, possibly through a potentially slower zone of conduction, the tachycardia also slows. With the mapping electrodes (closely spaced multielectrode catheter) spanning the entire CVTI up to and above the Eustachian ridge, this situation can be clarified. Rarely, the Eustachian ridge may serve as a line of longitudinal separation of a pre-Eustachian and post-Eustachian component for the CVTI-dependent flutter.

Kumar and Gehi present an interesting discussion on how there can be transient, near reversal of conduction shown with relatively wider spaced electrodes along the isthmus. As they point out, both multiple barriers of conduction and slow conduction at ≥1 site along the isthmus are likely necessary for this phenomenon. For the student of electrophysiology, it must be remembered that the most common cause of a reversal (not just a change) in activation sequence when ablating a reentrant tachycardia is inadvertent movement of the mapping electrodes across a line of conduction block. For example, if a multielectrode catheter placed along the CVTI along the lateral wall is moved behind the crista terminalis or when placed more inferiorly behind the Eustachian ridge, a complete reversal in activation sequence may be seen. Similar dramatic changes may also occur when a catheter along a lateral wall scar in a patient with reentry involving a previous atriotomy incision moves from one side of the incision to the other. The operator should be alert to the possibility that the line of block may represent the substrate around which the circuit is propagating, and entrainment maneuvers on either side will determine whether this activation sequence is part of the circuit or a bystander loop.

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**References**


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Editor's Perspective: Spinning Wheels: What Goes Up Cannot Come Down?
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