Teaching Rounds in Cardiac Electrophysiology

Mapping Reentry
In, Out, Into, or In Two?

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Among the most difficult but fulfilling challenges to meet in the invasive electrophysiology laboratory is defining a reentrant circuit and choosing an effective ablation strategy. In this installment of teaching rounds, Ip et al elegantly discuss the deductive process involved when analyzing activation and entrainment data in a particularly instructive reentrant circuit involving both atria.

See Article by Ip et al

Centrifugal Spread
Can activation mapping define the mechanism of tachycardia? In general, a centrifugal spread from an apparent point source suggests an automatic focus, whereas the absence of such a spread would favor reentry. However, there are important exceptions that occur frequently enough that such a simple construct is rarely of sufficient practical value.

- Small-circuit reentry (microreentry) in which extremely slow conduction in a focal area sustains the reentry also presents with centrifugal activation. Unlike automaticity, however, the local bipolar electrograms at the site of the apparent focal source are fragmented and abnormal with microreentry.
- When mapping 1 chamber, say the right atrium, but a macroreentry circuit is located within the left atrium, there may be a discrete breakout from the left atrium to the right atrium, such as through Bachmann bundle, and from this breakout, centrifugal spread mimicking an automatic focus may be found.
- With macroreentry in highly diseased hearts, although the circuit may be large, there may be a slow area of conduction within the circuit. As a result when exiting from the slow zone of the circuit, centrifugal activation of the rest of the chamber in addition to continued conduction through the circuit will be present and visually difficult to distinguish from the anticipated pattern with focal tachycardia.
- Reentrant arrhythmias contained within the cul-de-sacs of the heart, such as the pulmonary veins, supravalvar myocardium of the great arteries, and the papillary muscles, may have discrete exits from the cul-de-sac to the rest of the myocardium, again producing a pattern of centrifugal activation. Conversely, with focal tachycardia occurring in cardiac chambers with multiple scars, previous surgery or ablation, etc, the point source origin is not allowed to spread centrifugally and may follow a pattern that visually may suggest reentry.

Thus, although the general appearance of an activation map should be considered when attempting to diagnose the mechanism of tachycardia, additional and often confirmatory value will be found with entrainment mapping.

Disparate but Similar
With entrainment mapping, the extent of the difference between the postpacing interval (PPI) measured at the site of stimulation and the tachycardia cycle length (TCL) is an approximate measure of the distance from the mapping site to the circuit. It follows that 2 distant or disparate sites are unlikely to have similar or equal PPIs. As described by Ip et al, some reentrant circuits involve both atria where entrainment may be perfect in both the lateral tricuspid annulus and the lateral mitral annulus. Patients with previous cardiac maze or extensive ablation may have, for example, a circuit that involves the right atrium entering the left atrium through Bachmann bundle involving the left atrial myocardium entering the coronary sinus possibly via the vein of Marshall and through the coronary sinus musculature reentering the right atrium. Similar and small PPI-TCL differences may be found in the roof of the left atrium, the cavotricuspid isthmus, and indeed the lateral right atrium in this circumstance.

The electrophysiologist should also be aware that ≥3 sites that show exactly the same PPI although they do not equal the TCL strongly suggests that all of those mapped sites are within the tachycardia circuit. The reason for this is that the tachycardia circuit may exhibit decremental properties when pacing faster than the tachycardia. As a result, the PPI is always slightly more than the TCL. This difference may be more when pacing at relatively shorter cycle lengths. Because ≥3 sites are unlikely to be exactly equidistant from the actual tachycardia circuit, when equal PPIs are noted at multiple sites, there should be a strong suspicion that all or most of the points are within the tachycardia circuit itself.

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Together but Dissimilar
In some patients, sites that are close together may show marked differences in the PPI. This finding usually signifies a region of anatomic, functional, or pathological block (or slow conduction) between the 2 sites mapped. For example, a lower loop reentry circuit behind the Eustachian ridge, the cavotricuspid isthmus anterior to the Eustachian ridge will show PPIs that are large (perhaps in excess of 150 ms when compared with the TCL), yet a few millimeters posterior to the Eustachian ridge entrainment will exhibit a perfect PPI–TCL match. Other sites where this may be found include within the left inferior pulmonary vein versus just anterior to the vein for mitral annular flutter or in a patient cavotricuspid isthmus flutter and a previous right lateral atriotomy, posterior to the atriotomy scar versus anterior to it (between the scar and tricuspid annulus).

Close sites with marked differences in the postspacing cycle length can help to quickly identify a potential reentrant circuit. For instance, if perfect entrainment (entrainment with concealed fusion and PPI=TCL) is noted on the midinferior mitral annulus, either mitral annular flutter or a flutter circuit using the posterior wall, a small part of mitral annular myocardium, and then completing the circuit around the left- or right-sided pulmonary veins may be present. A small move from the initial entrainment map site posteriorly will show a major increase in the PPI if the circuit is a perimital flutter but continue to show a short PPI–TCL difference if the circuit involves the posterior wall, such as with a flutter around one set of pulmonary veins.

Two Circuits, One Chamber
In their presentation, Ip et al.1 explain how >1 chamber may be involved in the same tachycardia circuit. Other examples include ventricular tachycardias where the slow zone is within tissue shared by both ventricles, such as the posterior right ventricular outflow tract that represents the outflow tract septum or the inflow septum, separated by scar, for example, in sarcoidosis.

Less commonly, 2 circuits may be found within a single chamber and produce confusing activation and entrainment mapping results. In dual loop reentry, 2 distinct reentrant circuits are present, one with a fundamental cycle length shorter than the other, thus determining the TCL and in turn either passively conducting into the second slower putative circuit or entraining a second tachycardia in the secondary circuit. Even less common is true figure-of-8 reentry where a common isthmus between 2 possible reentrant loops is present and neither loop alone is capable of sustaining tachycardia. In such a tachycardia when the common isthmus has been activated, the circuit exits alternating (and entering) between the 2 limbs producing a figure-of-8 pattern of activation. Here, perfect entrainment may be found in either limb of the figure-of-8 circuit and in the common isthmus, whereas in dual loop reentry, perfect entrainment will only be found when entraining from the dominant loop.

Fusion and Mapping 3-Dimensional Mapping Systems
The authors importantly point out in their discussion the need for multiple electrodes or activation mapped points to identify the sometimes small regions where an antidromic wavefront during entrainment mapping may be present and otherwise missed and thus leading to the inexact diagnosis of perfect concealed entrainment.1 Newer mapping systems where a few beats of tachycardia or pacing can allow for the creation of dense electroanatomic maps may enable us to identify subtle differences in activation patterns when entraining close to versus within a circuit and at different sites within a macroreentry circuit.

Students of electrophysiology will appreciate the process of careful deduction demonstrated by Ip et al.1 integrating activation, entrainment, and anatomic information that leads to identifying the reason for unusual entrainment results even with a seemingly common arrhythmia.

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References

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