Editor’s Perspective

The Isthmus of Uncertainty

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Whether in the atrium or the ventricle, macro–reentrant tachyarrhythmias are among the most challenging arrhythmias to eliminate with ablation. Namdar et al.,1 in this installment of Teaching Rounds in Cardiac Electrophysiology, present and explain insightful findings from a patient with a complex macro–reentrant circuit encountered after prior cardiac surgery. They take us through a step-by-step approach and their thought process in identifying the eventual curative ablation target.

Is Early Good?

Does identifying a site of early activation from an activation map of the arrhythmia facilitate ablation? Unlike focal tachycardias, reentrant arrhythmias have no true early or late sites of activation. Timing is based completely on the empirical choice of a reference electrogram and the setting of the mapping window.2 Although this principle is apparent and most experienced electrophysiologists will either map and define the entire circuit of tachycardia or use entrainment mapping to identify appropriate sites for ablation, several corollaries to this principle are equally important but not as readily recognized.3,4

P-Wave Morphology

P-wave vector and morphology (or QRS morphology in the case of ventricular tachycardia) can be invaluable in estimating location of origin for a focal arrhythmia. With macro–reentry however, there is not necessarily a true onset for the P wave (or QRS). Thus, although patterns that have been identified as characteristic for cavotricuspid isthmus–dependent flutter are suggestive of the arrhythmia mechanism, general analysis of the flutter-wave morphology for other macro–reentrant arrhythmias and even common cavotricuspid isthmus–dependent arrhythmias in the presence of extensive atrial disease or prior surgery or ablation is often of little value.

When an isoelectric segment is noted in multiple leads, a focal mechanism for tachycardia is usually present. As the authors point out, however, in certain patients, macro–reentry may also manifest with an isoelectric P-wave segment and suggest an origin for the tachycardia. If a true onset for the P wave is clearly recognized, analysis of the vector may indeed help identify an exit site for the tachycardia. Exit is meant to define a transition from an area of slow conduction to relatively normal atrium from which the wavefront of activation progresses quickly. The preceding conduction through the slow zone is not identified on the surface ECG, and thus an isoelectric segment is present. Importantly, in diseased or scarred hearts, multiple exits may be present, and the isthmus relevant for ablation may be far removed from the diagnosed exit.

Activation Sequence and Interpretation

Another corollary to the fact that no early site exists in macro–reentrant arrhythmia is that regional activation sequences have to be interpreted with caution. For example, if there is distal to proximal activation in the coronary sinus, one cannot assume left atrial origin or that left atrial targets for ablation exist because there are always earlier sites in the left atrium. These in turn may be preceded by activation of the interatrial septum and the right atrium from a right atrial tachycardia.

Micro–Reentrant Tachycardia

An isoelectric interval on the surface 12-lead may signify no electric activation, focal mechanism, activation vector perpendicular to the sensed vector of that ECG lead (in this case, other leads will not be isoelectric with macro–reentry), or the electric activation is simply low amplitude and cannot be detected in the surface ECG. In an extreme case, slow conduction is present in one part of the atrium. The majority of the reentrant surface may be housed within that region such that an isoelectric period in multiple leads occurs just before the excitation wavefront exits to the rest of the atrium. Here, at the early site of activation, a highly fragmented and abnormal electrogram is found in contrast to mostly normal local activation at the early site of an automatic focal tachycardia.

Thus, in patients with prior cardiac surgery or atrial myopathy, the relation of the P-wave morphology to the activation sequence is not straightforward.

Postpacing Interval in Hearts with Multiple Areas of Abnormality

Even when one is familiar with the principles of entrainment mapping, globally abnormal and scarred atria create challenges. Decrement in areas of slow conduction (both

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arrhythmogenic and bystander sites), as well as additional loops of reentry, can complicate interpretation.

**Inordinately Long Postpacing Interval**

In the patient discussed by Namdar et al., when entrainment was attempted from the right atrial freewall, the postpacing interval (PPI) was almost twice the cycle length of the tachycardia. Although this is possible purely from combined distance and temporal delay in diseased hearts, the operator should suspect an intermediary area of block and forced conduction through a slow zone. Because slow conduction and block, if unidirectional, represent the substrate for reentry, a second reentry circuit, as with double-loop flutter, may be present between the primary flutter circuit and the pacing site. Other causes include a focal tachycardia inducing a reentrant flutter and exit block from the flutter circuit to the chamber being paced without entrance block.

**Inordinately Short PPI**

When the PPI is slightly shorter than the tachycardia cycle length, triggered activity or measuring the PPI to the wrong electrogram (eg, a far-field component) are potential causes. In diseased hearts with multiple scars, however, particularly in combination with decrement through a critical slow zone or an area of slow conduction between the pacing site and the arrhythmogenic circuit, the PPI may be so long that the last electrogram accelerated to the pacing cycle length fall very late, even >2 tachycardia cycle lengths after the last stimulus, and an intervening earlier electrogram that should not be used for PPI measurement is present.

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**Figure 1.** The tributarized isthmus. **A.** The classic concept of isthmus. **B.** Tributaries within an isthmus that show the actual portion being used for maintaining flutter and other potential sites that may become arrhythmogenic if the primary tributary is ablated. **C.** Because ablation eliminated the prior isthmus portion being used for tachycardia and the adjacent tributary had poorer conduction, the flutter continues but at a slower rate.

**Figure 2.** **A** to **F.** Cartoon descriptions of actual anatomic components that may create a tributarized isthmus (see text for description). LAA indicates left atrial appendage; LIPV, left inferior pulmonary vein; LSPV, left superior pulmonary vein; MV, mitral valve; RIPV, right inferior pulmonary vein; and RSPV, right superior pulmonary vein.
Knowing Your Boundaries
Namdar et al.1 excellently emphasize as a teaching point the importance of prior knowledge of the location of surgical incisions for successful ablation in their patient. Surgical scars, dense myocardial infarct, postmyocarditic fibrosis, cardiac veins and arteries, and the fibrous skeleton of the heart (including the annulus) serve as obstacles to electric conduction and thus ideal locations to anchor ablation lines when treating macro-reentrant tachycardia. In patients with markedly abnormal hearts, generalized slow conduction throughout the myocardium (or ventricle) may be present such that even with careful mapping it is difficult to find an area of slow conduction where ablation will be successful. Slow zone ablation may terminate the arrhythmia, but unless ablation lines are anchored to an anatomic obstacle, other flutters, possibly faster and nonsustained, may continue to use the existing channel (isthmus) and be a source of symptoms. Cardiac MRI with late gadolinium enhancement images, careful review of prior surgical reports, and, in some cases, intracardiac echocardiography during the procedure may all be of value in allowing the operator to anticipate both areas of slow conduction and identify boundaries to anchor lines.5–7

 Tributarized Isthmus
The classic understanding of an isthmus in electrophysiology is a relatively narrow band of myocardium activation, through which the propagating wavefront is constrained by 2 adjacent and bordering electrically inactive sites. Perhaps the best known is the relatively broad area of usually normal myocardium between the tricuspid valve and the inferior vena cava.8

In diseased hearts and when surgical incisions and multiple anatomic obstacles exist, the concept of an isthmus is far more nuanced and complex. Nevertheless, understanding these variations on the primary theme of needing to transect an isthmus completely to block conduction through a necessary, critical region for maintenance of reentrant tachycardia is useful to appreciate. Figure 1A shows the classic concept of the isthmus. In Figure 1B, longitudinal scar, paucity of myocardium, or anatomic ridges compartmentalize and longitudinally dissociate portions of the isthmus. In turn, multiple tributaries of the primary isthmus are present. Each of these tributaries will have differing conduction properties, and only one is necessary for continuing the flutter. If ablation is done over one of these isthmuses, the flutter may continue by using one of the tributaries, albeit with slower conduction and thus a longer cycle length with the vast majority of the circuit being unchanged (Figure 1C). Because there is some change in the activation pattern, however, electrocardiographic and electrogram characteristics may show differences which in some cases may be fairly profound (see below). Eventually, all components of the isthmus will require ablation to abolish that particular flutter.

The same concept can be expanded to appreciate why ablation may be successful ablation at sites far from an isthmus component where entrainment was otherwise perfect (Figure 2). The tributaries of the isthmus could be considered in this case to be distant from each other and separated by scars, the pulmonary veins, other ablation lines, and ablation circles. If a flutter is entrained on the isthmus between the mitral annulus and the left pulmonary vein, an ablation line perfectly placed between the mitral valve and the vein orifice will simply allow the flutter to continue through the pulmonary vein. Now, if that pulmonary vein is isolated, another tributary between the pulmonary veins can be used. If a wide-area ablation around both veins is done, it is possible for flutters still entrained from the primary isthmus to continue down the posterior wall and medial to the left-sided circle. A roof line may then prevent this isthmus tributary, but the circuit could use the right-sided pulmonary vein, myocardium, etc. Not coincidentally, both pulmonary vein circles anchored to each other by the roof line and in turn anchored to the mitral annulus would be required to prevent all but the rarest macro-reentrant atrial tachycardias that can be entrained on the mitral isthmus.

Therefore, when ablating in diseased, scarred atria, an abrupt change in the cycle length with some components, including entrainment characteristics, remaining similar should prompt a search not only for additional loops of reentry but also for other components of the initial isthmus.

Summary
Circulation Arrhythmia & Electrophysiology readers and students of electrophysiology will indeed appreciate the approach and elucidation of the steps to successfully ablate even complex flutters occurring in postsurgical atria presented in this issue by Namdar et al.1 The student should become familiar with what exactly is meant by the terms early site, exit, boundary, and isthmus.

Disclosures
Dr Asirvatham receives no significant honoraria and is a consultant with Abiomed, Attriecure, 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.

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