Adenosine and Atrial Tachycardia
Learning Before Burning?

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Atrial tachycardia (AT) may be encountered in a variety of settings in the electrophysiology laboratory. It constitutes a differential diagnosis in patients presenting for an electrophysiological evaluation and ablation for paroxysmal supraventricular tachycardia. These patients typically do not have any evidence of structural heart disease. Patients may also present with AT in setting of an atrial myopathic process, related to acquired (eg, rheumatic heart disease) or congenital heart disease, cardiac surgery, or an idiopathic process. Finally, patients with atrial fibrillation (AF), especially the persistent tachycardia, triggered activity, or microreentry/localized reentry.

Although the prevalence of precise arrhythmia mechanism differs in these contexts, broadly speaking, there are 2 possibilities: focal activation and macroreentry. Focal arrhythmias may be further characterized as being caused by abnormal automaticity, triggered activity, or microreentry/localized reentry.

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Defining the operative mechanism is important because it helps tailor the ablation approach. For example, targeting the site of origin of a focal AT should be sufficient in preventing recurrence. In contradistinction, the end point during a procedure for macroreentrant tachycardia includes not only arrhythmia termination during radiofrequency energy delivery, but also demonstration of linear block. Lerman et al have previously explored these various possibilities and found that adenosine infusion helped distinguish between the various mechanisms responsible for AT in patients.

In this issue of the Journal, Dr Lerman and his colleagues present their experience with adenosine in delineating the mechanism of AT in a large series of patients. Among the 59 patients, approximately one third had undergone a prior procedure for either AF or AT. The criteria for macroreentry included accounting for >90% of the tachycardia cycle length (CL) and documenting intersection of early and late areas on 3-dimensional (3D) mapping. A focal mechanism was diagnosed upon demonstration of centrifugal activation on 3D mapping. Triggered activity was diagnosed if the tachycardia was inducible with programmed stimulation, and the electrogram at the target site was discrete, that is, accounted for <20% of the tachycardia CL. If mapping accounted for >75% of the tachycardia CL, and the electrogram at the target site were fractionated (specifically, responsible for >35% of the tachycardia CL), the mechanism was deemed to be microreentry. Abnormal automaticity was the mechanism for ATs that required isoproterenol infusion (but not programmed stimulation) and demonstrated warm-up and cool-down phenomena.

Based on the results of 3D activation and entrainment mapping, a focal mechanism was established in 49 and macroreentry in the remaining 10 patients. Adenosine had no effect in any of the patients with macroreentry. Among the 49 patients with focal tachycardias, triggered activity was ascribed the mechanism in 26 patients, microreentry in 17 patients, and automaticity in 6 patients. Adenosine terminated the tachycardia in all 26 patients with triggered activity, had no effect in any of the patients with microreentry, and slowed or suppressed the tachycardias in all 6 patients with automaticity. The authors concluded that insensitivity to adenosine identifies reentrant ATs. Their data also suggest that termination of an AT with adenosine establishes a triggered mechanism. They go on to suggest that a diagnostic algorithm based on adenosine infusion does not require pacing to establish a reentrant mechanism and that adenosine could be infused as the initial step, even before mapping. The findings of this study confirm the utility of adenosine in diagnosing triggered arrhythmias. The accuracy of the proposed algorithm, of course, depends on the precise definition of the various mechanistic subtypes in patients with focal ATs. One could, therefore, argue that requirements of electrogram duration are somewhat arbitrary and subjective. On the other hand, it could also be argued that in the context of a clinical electrophysiological procedure and in the absence of a gold standard, the proposed diagnostic criteria are reasonable and clinically practical. In the electrophysiology laboratory, it is not possible to definitively prove triggered activity, for example. This would require demonstration of delayed after depolarizations during intracellular recordings. Mapping with catheters capable of recording monophasic action potentials has been performed clinically and does offer unique insights into arrhythmia mechanisms. However, perturbations in monophasic action potentials are not felt to accurately reflect actual transmembrane recordings. Classically, during induction of triggered arrhythmias, there is a direct relationship between the pacing rate and the coupling interval of the first beat of the tachycardia, that is, the interval between the last paced complex and the first beat of the tachycardia. Although this criterion does not require specialized equipment during a clinical electrophysiology procedure, it may not be demonstrable if the tachycardia is not reproducibly inducible.
Because this was not a randomized study, it cannot be definitively concluded that adenosine infusion helps expedite an ablation procedure for AT as compared with a standard approach. So how does one assimilate findings in this study in the context of our conventional mapping armamentarium, which includes the ECG, 3D activation mapping, and entrainment mapping? The clinical context is also important because it greatly influences the type of arrhythmia encountered. The most important step, in agreement with the authors, is to evaluate the possibility of macroreentry. With multipolar mapping catheters, it should be possible to create a high-density map of the atrium within a few minutes. However, findings of activation mapping should be confirmed with entrainment mapping because, especially in the presence of prior ablation, scar, and conduction block, activation patterns may not be reliable. Demonstration of return cycles approximating the tachycardia CL from opposite walls of the chamber (ie, anterior and posterior, or septal and lateral, etc) helps confirm a macroreentrant mechanism. This definition helps avoid confusion that may be encountered with other algorithms, which rely on subjective circuit dimensions and others.

After ruling out macroreentry, then one is left with the various mechanistic possibilities for centrifugal tachycardias. Although it may seem obvious, it is critical to define the P wave onset, which greatly facilitates mapping of focal tachycardias. If the P wave is obscured by the T wave or the QRS, ventricular pacing is helpful in unmasking atrial activity. An early site on a 3D activation map may not reflect the site of origin because it may only represent a breakthrough from the contralateral chamber. The early region is then explored in detail to further constrain the target site before energy delivery. The electrogram at the target site may or may not be fractionated, depending on the mechanism as elucidated by the authors. After tachycardia termination during ablation, it is common practice to deliver additional radiofrequency energy to consolidate the effective lesion and prevent recurrence. Barring anatomical constraints, this is reasonable to do irrespective of the tachycardia mechanism, microreentry, triggered activity, or enhanced automaticity. If long-duration electrograms are encountered at the target site, suggesting the presence of a small reentrant circuit (in the parlance of post AF arrhythmias), it is not unreasonable to map and ablate adjacent sites, which harbor similar electrograms or to extend the lesion to anatomic obstacles, such as pulmonary veins or prior lines.

Adenosine infusion during nonmacroreentrant ATs does afford mechanistic insights. The current contribution by Dr Lerman and colleagues has codified the mechanistic approach to the diagnosis of AT, will be frequently referenced even by the seasoned electrophysiologist, and be a great teaching aide for trainees. It also reminds us of the maxim, learning before burning. But this ideal may not be always possible, practical, or in some cases, necessary. From a clinical standpoint, if we are targeting the earliest site with respect to the P wave, does it matter if the mechanism is triggered activity or abnormal automaticity? This distinction could be made by infusion of adenosine, but the result would not alter the mapping approach. Even if one were dealing with microreentry or a small reentrant circuit, documenting the lack of termination with adenosine does not get us any closer to the target site. One could also supplement the results of activation mapping with entrainment mapping to verify a reentrant mechanism.

Adenosine administration may yield equivocal results if the tachycardia is not reproducibly inducible. It may also be difficult to distinguish between suppression (for automatic rhythms) and termination (for triggered arrhythmias). As is well known, adenosine may, by shortening atrial refractoriness, facilitate the induction of AF. Because some post-AF arrhythmias are difficult to induce, conversion of a stable AT to AF secondary to adenosine infusion for diagnostic purposes would be most unwelcome.

In patients with paroxysmal supraventricular tachycardia, it is mandatory to establish the mechanism because the ablation approach varies for the various possibilities. But in other cases, for example, patients with documented typical atrial flutter who present to the laboratory in sinus rhythm, it would be reasonable to perform ablation of the target site without inducing the arrhythmia and proving the mechanism of ishmus dependence. Similarly, after ruling out macroreentry in patients with AT, the simple diagnostic approach outlined herein is probably sufficient in most cases and does not rely on pharmacological perturbation, which is unlikely to improve outcomes, and may be associated with potential drawbacks.

Disclosures

None.

References


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