Editorial Comments

Ventricular Tachycardia
Chipping Away at Finding Curative Therapy

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In clinical cardiology, therapy for the most part is still primarily palliative, whereas relatively few procedures are truly curative. In the early days of invasive electrophysiology, clinicians were excited about the ability to induce supraventricular tachycardia with single, critically timed atrial extrastimuli. Soon thereafter, a whole array of mechanisms that induced these tachycardias became evident. Tachycardia circuits could be categorized as those that were due to atrioventricular (AV) nodal reentrant circuits, those that incorporated extranodal bypasses, and those that included nodoventricular tracts. From this deeper understanding of reentrant circuits followed impressive surgical and, more recently, catheter ablative techniques designed as totally curative therapy. Now, for example, ablation of an accessory AV pathway without change to the normal AV node-His axis is an accepted, almost routine, surgical technique in many medical centers, and it allows for return of patients to a totally normal lifestyle without need for long-term antirhythmic therapy.

Recently, techniques have been developed allowing for reproducible induction of ventricular tachycardia, which has allowed for application of remarkable surgical and, in some instances, catheter ablative procedures for tachycardia control. These techniques, though often effective, still require open-heart surgery and extensive endocardial mapping during tachycardia. In a remarkable report in this issue of Circulation, Tchou et al have summarized their results, applying the technique of right bundle branch ablation to treat a subset of patients with ventricular tachycardia. Through excellent deductive reasoning, they showed that the mechanism of the tachycardia in these patients was most likely reentry involving the His-Purkinje system. If so, the right bundle branch is probably critical for maintenance of the tachycardia and, hence, the remarkable results achieved with catheter ablation of this structure. Such a landmark observation surely deserves critical appraisal before acceptance into our clinical arsenal.

1. Was His-Purkinje reentry proved by the authors?

The authors allude to two other possible tachycardia mechanisms in their patients; namely, AV nodal reentry with rate-related bundle branch or retrograde conduction over a concealed Mahaim tract. AV nodal reentry as a mechanism cannot be summarily dismissed because of the absence of two retrogradely conducted His deflections before tachycardia initiation because the initial retrograde His produced by the basic ventricular drive may have been obscured within the QRS complex or not recorded because of technical limitations. The following are strongly against AV nodal reentry: 1) sustained AV dissociation during tachycardia; 2) inability to produce the tachycardia from the atrium with reproducible induction with single premature ventricular extrastimuli; and 3) apparent absence of dual AV nodal conduction curves. Alternatively, could these tachycardias be explained by the presence of a retrogradely conducting concealed nodoventricular bypass tract? Shimizu et al recently provided excellent evidence for retrograde nodoventricular tract conduction in a patient with narrow complex tachycardia and AV dissociation. It should be emphasized that in the latter report, antegrade preexcitation through a nodoventricular bypass tract could be demonstrated by atrial pacing, whereas this was apparently not observed by Tchou et al. The possibility of all patients having such a rare tachycardia mechanism would appear to be very remote indeed.

2. Does catheter ablation of the right bundle branch prove that this structure is a critical component of the tachycardia circuit?

Unfortunately, present ablative procedures are not sufficiently precise to allow for sole disruption of target areas within the heart. Hence, putative ablation of the right bundle branch will likely result in associated damage to the summit of the ventricular septum as well as other portions of the specialized conduction system. Could the relatively non-selective, high-energy shocks disrupt an AV nodal reentrant circuit or a concealed right nodoventric
ular tract? The answer to these questions is obviously unknown, but the remarkable ability to consistently achieve tachycardia control parri-passu with disruption of right bundle branch conduction makes the authors’ hypothesis far more likely than the alternative possibilities.

My critical comments should in no way detract from the clinical importance of their observations. It provides clinical electrophysiologists with a potential Rosetta stone for more precise delineation of the tachycardia circuit for patients with ventricular tachycardia and for application of potentially curative techniques without resort to open-heart surgery. A word of caution is in order. Although catheter ablation of the right bundle branch is relatively simple, the ability to distinguish His-Purkinje reentry from other mechanisms is difficult even for very experienced clinical electrophysiologists. In addition, distinguishing the right bundle branch potential from the His-bundle deflection may be equally challenging in patients with intrinsic His-Purkinje disease. If, for example, high-energy shocks are erroneously applied to the His bundle proximal to the tachycardia turn-around point in the His-Purkinje system, the worst possible results may emerge, namely, induction of complete AV block without affecting the tachycardia circuit. Similarly, if early activation of the right ventricular summit is confused with a right bundle branch potential, high-energy shocks may result in producing a nidus for development of another ventricular tachycardia conceivably without affecting the original circuit. The report by Tchou et al represents a potentially important clinical breakthrough. Application of this technique is not for the novice!

References


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