‘Slow pathway’ ablation in patients with atrioventricular nodal reentrant tachycardia: do we understand what we are doing?

In the field of interventional electrophysiology, radiofrequency catheter modulation/ablation of the slow pathway in patients with common type atrioventricular nodal reentrant tachycardia has proven to be highly effective and safe[1]. Being one of the few true curative techniques, catheter ablation can successfully abolish atrioventricular nodal reentrant tachycardia in up to 97% of patients[1,2]. Inadvertent complete or second-degree atrioventricular block are the most common complications and occur in 1–1.3% of patients[1,2]. Catheter ablation can be performed effectively and safely, even in the very elderly[3].

These remarkable developments have rekindled interest in the anatomy and electrophysiology of the atrioventricular node. Success rates of almost 97% suggest a comprehensive understanding of arrhythmogenesis in the atrioventricular junction. In contrast, most of the concepts of atrioventricular nodal functioning are still controversial. Dissociation in two (or more) functionally distinct pathways, each with different conduction velocities and refractory periods is widely accepted as the basis of atrioventricular nodal reentry. However, the precise pathway of reentrant excitation has not yet been defined. There is no convincing anatomical evidence for cable-like, histologically discrete dual or multiple pathways within the atrioventricular nodal area. Evidence from ablation studies suggest reentrant circuits far outside the specialized atrioventricular nodal tissue[4]. In recent experiments performed on isolated, blood-perfused canine hearts, however, Loh et al. could show that the reentrant pathway during ventricular echoes, which are thought to represent a ‘single beat expression’ of atrioventricular nodal reentrant tachycardia, is confined to the atrioventricular node[5]. Intranodal ‘microreentry’ during single echo beats and some forms of atrioventricular nodal reentrant tachycardia, and ‘macroreentry’ involving perinodal tissue during other forms of atrioventricular nodal reentrant tachycardia could explain this apparent discrepancy[6]. An accurate model of atrioventricular nodal reentrant tachycardia in an experimental setting is needed to assess these issues.

Radiofrequency catheter modulation/ablation of the ‘slow pathway’ usually targets the inferior to midseptal area of the inter-atrial septum. But what is the anatomical substrate of the slow pathway? Inferior atrionodal connections with nodal-type electrophysiology, as well as the inferior nodal extension have been proposed as possible substrates[7,8]. And what is the mechanism of ‘slow’ conduction through this pathway? Apart from normal conduction through just a longer path, slowing of conduction due to the characteristics of depolarizing membrane currents, electrical cell-to-cell uncoupling, and discontinuous conduction due to branching tissue architecture have been discussed as possible mechanisms[9].

As pointed out above, the risk of atrioventricular nodal damage during catheter ablation in patients with atrioventricular nodal reentrant tachycardia and normal atrioventricular conduction parameters is low. However, in patients with a prolonged PR interval at baseline, one would intuitively assume that the risk of atrioventricular block is higher. A prolonged PR could indicate impaired or absent antegrade function of the fast pathway, and radiofrequency ablation targeting of the slow pathway would subsequently lead to atrioventricular block. This reasoning, however, is far too simplistic. The conduction delay can take place anywhere in the conducting axis between the atrium and the His bundle without affecting the limbs of the reentrant circuit. In addition, more than two inputs to the atrioventricular node certainly exist. Indeed, several studies have suggested that slow pathway ablation can be safely performed in patients with atrioventricular nodal reentrant tachycardia and prolonged PR intervals[10,11].

The prospective study by Li et al.[12] in this issue is a welcome addition. Li et al.[12] describe the acute results of radiofrequency catheter ablation and the follow-up in a study population of 346 patients presenting with typical and atypical atrioventricular nodal reentrant tachycardia. Out of this group, 18 patients had a prolonged PR interval at baseline. Interestingly, impaired conduction was not restricted to the atrioventricular node (A-H 156 ± 28 vs 83 ± 16 ms, P<0.001; Wenckebach cycle length 378 ± 72 vs 320 ± 60 ms, P=0.003), but extended also to the His-Purkinje system (H-V 52 ± 7.4 ms vs 45.8 ± 9.1 ms, P<0.001) and intraventricular conduction (QRS 121 ± 28 vs 103 ± 17 ms, P=0.01). Patients...

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with a prolonged PR interval at baseline were older (62 ± 7 vs 52 ± 15 years, P<0.0001) and had a higher incidence of structural heart disease (8/18 vs 19/328, P<0.001). Acute success could be achieved without complications in all but one patient. Atrioventricular conduction was monitored by 24 hour Holter recordings 1 day before radiofrequency ablation and 1 day, 1 week and 1, 3, and 6 months after the procedure.

While none of the 328 patients with normal atrioventricular conduction revealed any abnormalities, in the subgroup with impaired atrioventricular conduction at baseline, 5/18 patients newly developed late asymptomatic atrioventricular block, and 1/18 patients developed late symptomatic atrioventricular block requiring pacemaker implantation. Atrioventricular block was documented after 1 day in three patients, after 1 week in two patients and after 1 month in one patient. These are important observations which may have been missed in prior studies without careful Holter follow-up. The mechanism of delayed impairment of atrioventricular conduction is unclear. Radiofrequency lesions may cause an increase in fibrous tissue between myocytes, which decreases cell-to-cell connections and may lead to conduction slowing and block.

However, before we withhold potentially curative treatment from older patients with structural heart disease and impaired atrioventricular conduction, a few points need to be addressed to place these observations in a clinical context. First, when the need for a pacemaker is used as an end-point, the data did not show any statistical significance. Secondly, the chosen end-point of the ablation procedure was non-inducibility of dual physiology and atrioventricular nodal reentrant tachycardia. If dual atrioventricular nodal pathways still manifested as an AH jump or atrioventricular nodal echoes (one or more), at least two additional radiofrequency impulses were applied. Complete slow pathway ablation was assumed if the antegrade atrioventricular nodal effective refractory period after radiofrequency ablation was longer than the cycle length of the atrioventricular nodal reentrant tachycardia. According to this definition, total elimination of the slow pathway was achieved in 7/18 patients with prior atrioventricular conduction disturbances, while sole modulation of the slow pathway was done in 11/18 patients.

It seems important that 5/7 patients with complete elimination of the slow pathway developed late atrioventricular conduction impairment, while only 1/11 patients with slow pathway modulation had late atrioventricular block. This raises an intriguing question: how aggressive should we be in tackling something that we do not completely understand — the slow pathway? In patients with diseased conduction tissue, complete elimination of the functional slow pathway should not be vigorously pursued, and this should even be extended to patients with normal PR intervals. There is abundant clinical evidence that residual slow pathway conduction does not predict recurrence of atrioventricular nodal reentrant tachycardia as long as no more than a single atrioventricular nodal echo is accepted during isoproterenol infusion[13–16]. The risk of complications, including late occurrence of atrioventricular block, may in fact be related to the cumulative effect of the energy employed during the procedure. Therefore, unnecessary radiofrequency applications should be thoroughly avoided.

In conclusion, although the anatomical and physiological correlates are not completely understood, patients with atrioventricular nodal reentrant tachycardia can be effectively and safely cured. The information from the study by Li et al[15] offers important insights into the clinical management of patients with atrioventricular nodal reentrant tachycardia and pre-existing disease of the atrioventricular conducting axis. Ablative therapy can be considered the initial treatment of choice for these patients. However, the ideal end-point during slow pathway ablation should be the abolition of tachycardia with preservation of dual atrioventricular nodal physiology.

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References


