Using an Accelerated Junctional Rhythm to Predict the Success and Safety of Slow AV Nodal Pathway Ablation

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Atrioventricular nodal reentrant tachycardia (AVNRT) has been successfully treated by targeting the slow atrioventricular nodal pathway for ablation going on 30 years (1-4). It has long been recognized that successful ablation of the antegrade slow AV nodal pathway using radiofrequency (RF) current delivered along the tricuspid annulus near, or slightly superior to, the coronary sinus ostium is almost always associated with an accelerated junctional rhythm in patients with typical slow-fast AVNRT (1-4). For example, Yu and colleagues observed an accelerated junctional rhythm with 42 of 43 RF applications that successfully eliminated the slow AV nodal pathway (1). While the sensitivity of an accelerated junctional rhythm as a marker for successful slow pathway ablation is very high (98%), the specificity is rather low, as approximately 45% if unsuccessful RF applications also induce an accelerated junctional rhythm (1). Mapping of the site of earliest atrial activation during the accelerated junctional rhythm induced by RF targeting the slow AV nodal pathway has shown that retrograde conduction occurs at the superior aspect of Koch's triangle where retrograde fast pathway conduction is recorded in response ventricular extrastimuli. This strongly suggests that RF energy applied to the region of the slow pathway stimulates AV nodal cells which conduct antero-superiorly through the fast pathway to the atria at the apex of Koch's triangle. Animal studies using isolated, perfused porcine and rabbit hearts have identified an area in, or close to, the compact AV node that is stimulated by heat (5). Microelectrode recordings have revealed that the accelerated junctional rhythm in response to heating arises in nodal-type cells with increased phase 4 depolarization slope and shortening of the action potential duration (6). Irregularity of the evoked accelerated junctional rhythm was shown to be due to interaction of multiple foci and the presence of conduction block between these foci and the His bundle (3). These findings are compatible with studies of accelerated junctional rhythm during slow pathway ablation in which the retrograde His-atrial conduction interval during RF-induced junctional rhythm is significantly shorter than the His-atrial interval during slow-fast AVNRT (57 + 24 vs 68+ 21 ms, $P < 0.01$) (7).

Sugumar and colleagues (8) prospectively analyzed the effects of each RF application on antegrade and retrograde AV nodal conduction in 67 consecutive patients with typical slow-fast AVNRT referred for slow pathway ablation to determine predictors of success. Every RF application was analyzed if the duration was at least 5 seconds or if at least one accelerated junctional beat was induced. An RF application was discontinued if no junctional rhythm occurred after 30 seconds. RF applications were also immediately discontinued if any degree of AV or accelerated junctional to atrial (JA) block occurred or when the accelerated junctional rhythm cycle length was <350 msec. An accelerated junctional rhythm was observed during 178 of 301 total RF applications (59%). Successful slow pathway modification, defined as no more than one AV nodal echo beat in response to programmed atrial stimulation, was achieved in 66 of 67 patients (99%). Among these, complete elimination of antegrade slow pathway conduction was achieved in 30 patients (46%). These authors found that an accelerated junctional rhythm was observed in all 66 patients for whom successful slow pathway ablation was achieved. However, like previous studies, the presence of an accelerated junctional rhythm was not a specific indicator of successful slow pathway ablation (1). However, the number of junctional beats observed was significantly greater for successful than unsuccessful RF applications (26.0 $\pm$ 26.7 vs 13.4 $\pm$ 16.1, p< 0.001). This may have been related, in part, to the fact that successful RF applications were of longer duration than unsuccessful applications (38.5 $\pm$ 25.6 vs 26.9 $\pm$ 18.4 sec, p=0.002). The cycle length of the induced junctional rhythm did not predict RF application success. Importantly, junctional-atrial block was observed during 19 RF applications, 14 of which resulted in successful slow pathway ablation and 5 of which did not. The authors conclude: 1) that an accelerated junctional rhythm during RF is a requirement for successful slow pathway ablation; and 2) junctional-atrial block is a strong predictor of an application that eliminates or modifies slow pathway conduction.

The present study, while prospectively and carefully analyzing each and every RF application, largely confirms the accumulated wisdom of electrophysiologists who have performed slow pathway ablation for many years. Electrophysiologists have long recognized that an accelerated junctional rhythm is necessary to successfully modify slow pathway conduction. Most operators will discontinue an RF application if an accelerated junctional rhythm is not observed within 10 seconds. While being necessary for successful slow pathway modification, the presence of an accelerated junctional rhythm during RF is not sufficient for a successful procedure. Thus, the sensitivity of an accelerated junctional rhythm is high, but the specificity of this finding is relatively low. And while the presence of junctional-atrial block during RF predicts slow pathway modification or block, this finding should prompt immediate cessation of an RF application due to the risk of causing both antegrade fast and slow AV nodal block. The presence of junctional-atrial block is likely an indicator that the compact AV node is in jeopardy of thermal injury. It should also be emphasized that these observations may not apply to atypical forms of AVNRT that utilize slowly conducting pathways which may connect to either the left or the right atria relatively remote from the compact AV node (9,10). Therefore, an accelerated junctional rhythm is likely not a reliable predictor of ablation success for these slowly conducting pathways underlying atypical forms of AVNRT.

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