A functional discordance of conductive and refractile properties within atrionodal and AV nodal tissues is thought to be the basis for AV nodal reentrant tachycardia (AVNRT). Contemporary therapy aimed at curing AVNRT without materially damaging normal AV conduction began with surgical AV node modification.\textsuperscript{1} Catheter based techniques with DC shocks and later with radiofrequency energy have been shown to be equally effective. The recognition of the posteriorly placed usual exit site of the slow pathway then led to the evolution of techniques of selective slow pathway ablation aimed at reducing the risk of complete AV block.\textsuperscript{2,3} One approach to slow pathway ablation has therefore been anatomical, with radiofrequency energy sequentially applied to the postero-inferior interatrial septum and, later, if required, to the mid-septum. Published data show that the success rates usually achieved (> 90\%) require a high number of radiofrequency energy applications (range of medians 4-14).\textsuperscript{4,5} The electrophysiological approach on the other hand uses either retrograde slow pathway mapping or recognition of pathway potentials to guide ablation.\textsuperscript{5-17} As retrograde slow pathway conduction is elicitable only in a minority of patients such mapping is not commonly used in spite of its reported specificity.\textsuperscript{6}

**Spikes potentials**

A spike-like “Asp” potential accompanying a lower amplitude A potential has been noted in the vicinity of the coronary sinus ostium in patients with AVNRT.\textsuperscript{7} These potentials have been shown to reverse their temporal relation during retrograde slow pathway conduction, and Asp to be separately advanced by late atrial premature extrastimuli. In our experience, the activation sequence of this double potential is strictly dependent on the site of origin of the atrial activity and, therefore, indirectly of the retrograde nodal exit.\textsuperscript{1} Such dual atrial potentials (including spike potentials) have been found in 98\% of a group of patients with no manifest AVNRT.\textsuperscript{8}

Using the Asp potential as a guide, AVNRT was successfully eliminated in 99\% of patients with a median of two applications of radiofrequency current, without affecting normal AV conduction. Animal studies, however, have shown that Asp-like potentials arise from atrial muscle cells possessing rapid upstroke action potentials.\textsuperscript{7}

**Slow potentials**

Electrical activity ascribed to the AV node was noted on extracellular recordings in dogs in 1956.\textsuperscript{8} However, it was Scher \textit{et al} who in 1959 described “slowly developing” potentials from the AV node region in a canine model.\textsuperscript{9} Other contemporaries also described similar potentials in dogs, calves, and pigs (variously termed N potentials, slow deflections, and nodal hump), and characterised their behaviour in response to vagal stimuli and pharmacological agents.\textsuperscript{10,11} In humans such potentials were recorded for the first time in 1969 by Damato \textit{et al}\textsuperscript{12} and attributed to the compact AV node.\textsuperscript{12}

In the course of studies involving radiofrequency ablation for AVNRT, we described slow potentials recordable during antegrade AV conduction along the tricuspid valve at the mid and posterior part of the septum—at a significant distance from the anatomic site of the AV node—in 95\% of patients with AVNRT and their previously unreported rate-dependent behaviour.\textsuperscript{13} Radiofrequency energy applied at this site characteristically curtailed the ability of atrial stimulation to prolong critically the AH interval. The tachycardia was thus rendered non-inducible with a median of two (presently one) applications of radiofrequency energy. Similar potentials were found in 80\% of patients without AVNRT or echo beats.

These low amplitude slow potentials are concealed within or prolong the atrial electrogram, and occupy some or all of the AV diastolic interval. Requiring high amplification (0.1-0.2 mV/cm) and atrial pacing, these potentials are readily recorded as the catheter is withdrawn posteriorly from the His bundle position. They are however absent or difficult to detect in about 20\% of patients. Most importantly, with progressively rapid atrial pacing, these potentials separate from preceding atrial electrograms, decline in amplitude and slope, and prolong in duration, frequently culminating in their disappearance. We believe that slow mid-septal potentials persisting with atrial pacing at the end of AV intervals during antegrade slow pathway activation represent activity of the compact AV node.

Studies in pigs and dogs have shown that similar double potentials with a prominent second low frequency component arise from and around the site of retrograde slow pathway activation (sensitivity 100\%), and originate in superficial transitional cells with nodal type action potentials.\textsuperscript{7,16,17} Recent evidence suggests that such cells situated in the region of the posterior AV nodal approaches participate in retrograde slow pathway conduction such as during reverse echo beats.\textsuperscript{14} Such slow potentials were also recorded in rabbits along a discrete bundle distinct from the crista terminalis input.\textsuperscript{18}

These potentials can, however, persist at the ablation site even after successful slow pathway ablation. Mapping studies consistently describe a rather diffuse distribution of such potentials, suggesting that only in the appropriate anatomic context (the paratricuspid mid-posterior septum) do these potentials represent slow pathway activation. These nodal transitional cells probably also form the
first defence of the ventricles against an atrial tachycardia, followed thereafter by the compact AV node. As there is evidence that multiple atrionodal connections may exist in the form of a network, the recognition of electrophysiological parameters distinguishing the actively participating slow pathway activation is important. Unfortunately, this is difficult in clinical conditions primarily owing to catheter instability. Also important is the separation of potentials produced by bystander cells/dead end tracts from those produced by cells actively involved in AV conduction.

In the interim, until such distinctions are achieved, we believe that both the anatomic and pathway potential approaches are equally successful and safe, but the pathway potential approach appears more parsimonious.

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