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How to ablate typical 'slow/fast' AV nodal reentry tachycardia

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Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) accounts for about 60% of the patients presenting with paroxysmal supraventricular tachycardia (PSVT). It is the result of functional dissociation of AV nodal conduction into a so-called 'fast pathway' (FP) and 'slow pathway' (SP). The fast pathway forms the normal physiological conduction axis. It connects to the atrium in the anterior (superior) septum, close to the recording of the most proximal His bundle potential (Fig. 1). The atrio-His (AH) interval during conduction over the fast pathway generally is not longer than 220 ms. Conduction over the slow pathway, connecting to the atrium in the posterior (inferior) septum, can be revealed when an atrial impulse is blocked in the fast pathway (which generally has a longer antegrade effective refractory period than the slow pathway) leading to a sudden prolongation of the AH interval. Often the wavefront migrates back to the atrium over the fast pathway resulting in an AV nodal echo beat. Slow pathway conduction can be demonstrated in the majority of people. However, one-to-one antegrade conduction over the slow pathway, i.e. a situation where every consecutive atrial impulse is conducted over the slow pathway to the His bundle, is unusual. Therefore, perpetuation of reentry leading to AVNRT is often not possible. Even in the presence of one-to-one antegrade slow pathway conduction, a necessary prerequisite for sustained AVNRT, tachycardia may be non-inducible because of absent or weak retrograde fast pathway conduction. If reentry evolves, causing AVNRT, it uses the slow pathway as an antegrade link and the fast pathway as a retrograde link in 90% of patients: therefore, this 'slow/fast' form of AVNRT is called 'typical' or 'common'. The earliest atrial activation will be seen at the insertion of the fast pathway with the atrium (i.e. at the anterior-superior septum), and the HA interval will be short (generally $\leq$60 ms). Therefore, the P-wave coincides with the QRS complex, or closely follows it. In 10% of the patients, the circuit may evolve in the reverse direction ('fast/slow'), or may even encompass reentry over two slow pathways ('slow/slow')[1]. These entities are denoted as 'uncommon' AVNRT. Although the slow pathway is the ablation target in all forms of AVNRT, this text will only focus on ablation of the typical, slow/fast, variant.

Methodology

Theoretically, the AVNRT circuit as described above could be interrupted both in the fast and the slow pathway. However, since the fast pathway constitutes the physiological conduction axis and since it is located in the proximity of the compact AV node and His bundle, targeting it carries a definite risk of creating complete AV block. Therefore, the so-called 'anterior approach' has been abandoned for the 'posterior approach', targeting the slow pathway. Indeed, it has been shown earlier that the insertion of the slow pathway in the atrium is often located 1 or 2 cm away from the compact AV node, extending into the region between the tricuspid annulus and the orifice of the coronary sinus. Recent histological work has added the notion that in a minority of patients the posterior extensions may also, or only, be situated on the left side of the septum (towards the mitral annulus). Although targeting the slow pathway clearly has proven to be safer than the anterior approach, ablation may also lead to inadvertent AV block. The goal of the procedure therefore is to be minimally aggressive and not to eliminate all slow pathway conduction but only to eliminate one-to-one

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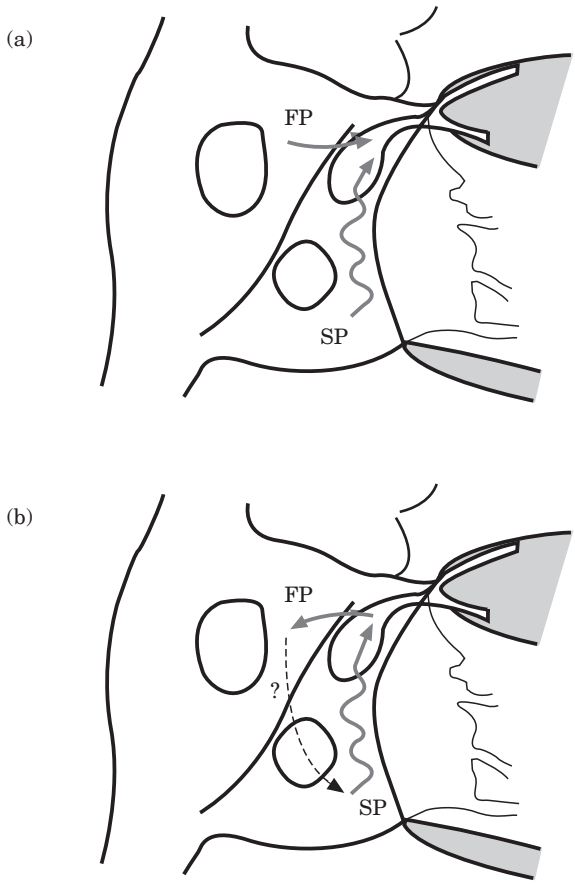

Figure 1 **(a) Schematic organization of the AV node, showing the location of the fast pathway (FP) inserting in the atrium anteroseptally, close to the compact AV node and His bundle, and the slow pathway (SP) inserting in the atrium between the coronary sinus and tricuspid annulus. The precise anatomical substrate for the slow pathway remains a matter of debate. (b) During typical slow/fast AVNRT, antegrade conduction evolves over the slow pathway, while the fast pathway is retrogradely activated. Most likely, the atrium forms the remaining part or the circuit.**

conduction over the slow pathway. Abolishing one of the key requirements for sustained AVNRT as described above is sufficient to achieve full clinical success.

Diagnostic work-up before ablation

In the patient with documentation of PSVT suggesting AVNRT, or with a typical history of PSVT, the absence of an accessory pathway must first be verified. This can easily be done with 'para-Hisian pacing', which is always performed as the very first pacing protocol in our laboratory[2]. It also allows the omission of a separate ventricular pacing catheter during the study, since pacing is performed from the tip of the octapolar His bundle catheter. The antegrade and retrograde conduction properties of the AV node are evaluated using decremental pacing and extrastimuli. Special attention is given to the prerequisites for AVNRT, i.e. (1) a sudden prolongation of the A_2H_2 interval on shortening of the A_1A_2 coupling interval (typically by 50 ms or more, although many patients present with more 'gradual' A_2H_2 curves) indicating the presence of the slow pathway, (2) one-to-one antegrade conduction over the slow pathway, and (3) robust retrograde conduction over the fast pathway (without which typical AVNRT is almost never seen, in contrast with atypical variants).

During these manoeuvres, tachycardia may start. If atrial burst pacing is also not effective for initiation, Isoprenaline is given in incremental doses of $0{\cdot}5\ \mu g\ .\ min^{-1}$ (up to a maximum of 3–4 $\mu g\ .\ min^{-1}$) to facilitate nodal conduction. After tachycardia has been started, ventricular extrastimuli may be delivered to exclude further the presence of a concealed accessory pathway, showing the absence of atrial resetting as long as the antegrade His bundle activation is unchanged. Moreover, we systematically map the site of earliest atrial activation during tachycardia which for typical slow/fast AVNRT should be anteroseptal (about 3 mm posterior and atrial from the recording site of the most proximal His bundle potential) and similar to the earliest atrial activation during ventricular pacing.

Radiofrequency delivery and evaluation

Multiple measures are taken during ablation to avoid inadvertent complete AV block: (1) ablation is started at least 5 min after cessation of Isoprenaline infusion to eliminate hyperdynamic cardiac contractility; (2) it is advisable to ablate during sinus rhythm and never during tachycardia since the strong myocardial contraction following the cessation of tachycardia may lead to dislocation of the ablation tip; (3) good anatomical landmarks demarcating the triangle of Koch are mandatory (Fig. 2). The His bundle catheter forms the key in this respect. We also prefer to position a catheter in the proximal coronary sinus clearly to indicate its ostium. An additional atrial pacing catheter (e.g. in the right atrial appendage) is not strictly necessary, since the coronary sinus (CS) catheter (or even the ablation catheter) can be used for this purpose. As mentioned earlier, ventricular pacing can be performed from the tip of the His bundle catheter.

There are two main schools of thought for localizing the site of radiofrequency (RF) delivery. One approach is purely anatomical, using the landmarks of Koch's triangle to determine the site of RF applications. The other approach is to use the recording of electrograms on the (posterior) septum to indicate the presence of the slow pathway. These may be the 'slow potentials' as described by Haïssaguerre *et al.*[3] or the 'slow pathway potentials (A_{SP})' as described by Jackman *et al.*[4]. These potentials follow a preceding 'atrial' deflection and are recorded on the mid-septum or the region between the coronary sinus ostium and the tricuspid annulus on the posterior (inferior) septum, respectively. It has been shown that this electrogram-based approach is more

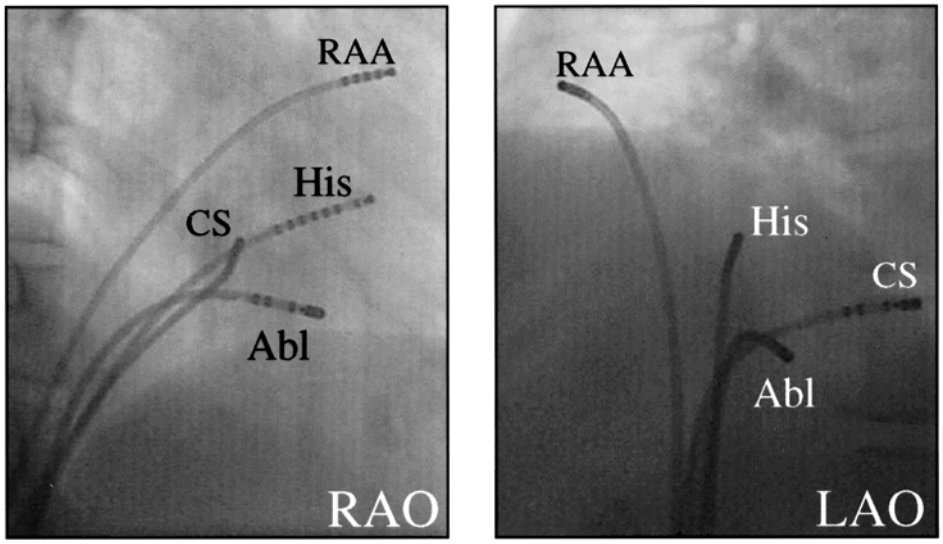

Figure 2 **Catheter positions during ablation of AVNRT. Radiographs taken in the right and left anterior oblique views are shown (RAO & LAO). The distal bipole of the octapolar His bundle catheter (His) is also used for ventricular stimulation. A catheter in the proximal coronary sinus (CS) helps to delineate the triangle of Koch. The ablation catheter (Ab1) is positioned in the right posteroseptal (inferoseptal) area, recording a large and sharp 'slow pathway potential (A_{SP})' between the ostium of the coronary sinus and the tricuspid annulus. This site, which is far from the compact AV node, was used to start RF delivery (see Fig. 3). A fourth catheter, positioned in the right atrial appendage (RAA) is used during atrial programmed stimulation.**

effective and associated with fewer applications, a higher likelihood of success, and a lower risk for inadvertent AV block[1]. Since the A_{SP} potential is recorded farthest away from the AV node, which may reduce the risk for inadvertent complete AV block, preference is given to the largest, highest frequency and latest A_{SP} to determine commencement of RF delivery (Fig. 3 (a)). Often this A_{SP} is located more than 2 cm away from the compact AV node, and used as an RF target leads to successful ablation with one or a few RF applications. If not, further applications may be guided anatomically, progressively higher up the septum. Thereby, special attention is given to covering the tricuspid edge of the ostium of the coronary sinus. Often, targeting this edge by moving the ablation catheter tip slightly in and out of the CS, is the clue to successful ablation. Moving higher up on the septum could be a next step and if this also fails, one could opt to put one or two burns inside the first centimetre of the CS. In some patients this will ablate the slow pathway as probably it is then dependant on left-sided posterior extensions of the AV node. Even the application of RF energy to the left side of the septum (using a retrograde approach) has been described, although it is only very rarely necessary. Using the described approach, elimination of one-to-one antegrade slow pathway conduction and non-inducibility of AVNRT can be achieved in 98 to 99% of subjects. However, while moving more anteriorly (superiorly) on the septum during consecutive burns, one should always weigh the potential benefit of this more aggressive approach against the increasing risks for complete heart block.

Since the A_{SP} is recorded between the CS ostium and the tricuspid annulus, starting with a 4 mm catheter with a slightly extended reach is usual. In smaller hearts a shorter curve may be more suitable. For AVNRT, low power settings are usually sufficient, e.g. 20 W, which can be increased up to 30 W during the application. Antegrade AV nodal conduction is evaluated by incremental atrial pacing immediately before and after energy delivery, to have a good comparison between both measurements. The shortest cycle length with one-to-one antegrade fast and slow pathway conduction is recorded. As mentioned, the elimination of one-to-one antegrade slow pathway conduction is the end-point for ablation. We even allow two or three consecutive AV nodal echo beats provided that the conduction time over the slow pathway is progressively lengthening from beat to beat before final block. Complete elimination of all antegrade slow pathway conduction is not essential for clinical success, and is associated with a higher risk of AV block. Nonetheless, after elimination of one-to-one antegrade SP conduction, we also evaluate the absence of induction of tachycardia (if it was reliably induced before), since in 1 or 2% of the patients tachycardia induction may persist even in the absence of demonstrable one-to-one antegrade slow pathway conduction, requiring further RF delivery.

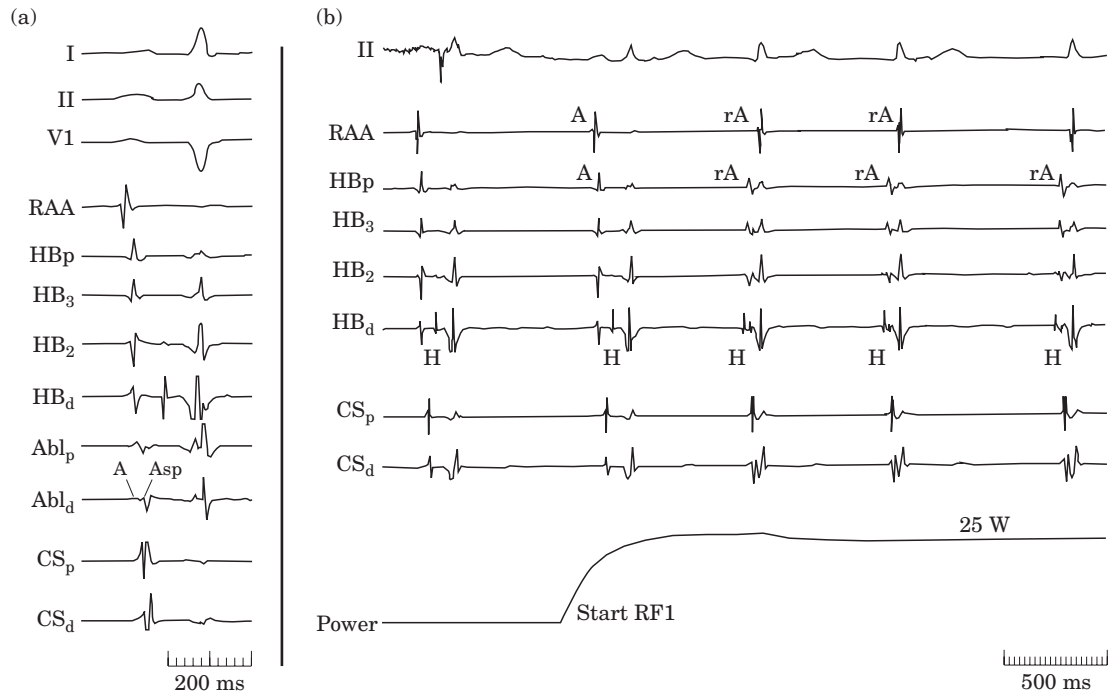

Figure 3 **(a) Electrograms recorded from the catheters as shown in Fig. 2. The electrogram at the distal bipole of the ablation catheter ($Ab1_d$) shows a large and sharp 'slow pathway potential (A_{SP})', preceded by a small 'atrial deflection (A)', and followed by a large ventricular deflection. Note that the A_{SP} is recorded after atrial activation in the proximal coronary sinus (CS). RAA=right atrial appendage; HB=His bundle, proximal to distal. (b) Radiofrequency (RF) energy delivered at this site immediately resulted in the induction of an accelerated junctional rhythm. Note the retrograde atrial activation over the fast pathway (rA) during each consecutive junctional beat, ensuring 'online' integrity of the physiological AV nodal conduction axis. Later during RF delivery, the junctional rhythm slowed down and gave way to sinus rhythm. After this single application (60 s), one-to-one antegrade slow pathway conduction was eliminated, and AVNRT was no longer inducible.**

In many cases, successful ablation of the slow pathway is associated with a marked shortening of the antegrade refractory period and one-to-one conduction at shorter cycle length over the fast pathway. The explanation for these findings remains elusive but the observation may be helpful in confirming the end-point of the procedure.

An accelerated junctional rhythm is often seen during RF applications (Fig. 3 (b)). It is important in two respects. First, its induction is strongly correlated with successful applications, although this relationship is not specific. Second and more important, the junctional rhythm provides an alternative way to assess integrity of the fast pathway, like the AH interval during sinus rhythm: each junctional beat should be accompanied by retrograde conduction over the fast pathway to the atrium (a luxury which is often absent during ablation of the uncommon forms of AVNRT). RF delivery should be stopped immediately after a first non-conducted beat, which in itself is rare using the described approach. Employing this cautionary guidance, development of complete and permanent antegrade AV block can almost surely be avoided.

Because of the right-sided nature of the procedure, aspirin is used as an antithrombotic agent (75–100 mg . day^{-1}), started the day before and continued up to 6 weeks after ablation. Full heparinization during the procedure is not mandatory.

Follow-up

Recurrence of AVNRT after a successful ablation procedure is rare, ranging from 1 to 5%. Dependent on the frequency of tachycardia episodes and the responsiveness to drugs, one may opt for a repeat procedure. It is important to note however, that some patients complain of palpitations after the procedure that are not due to recurrence of AVNRT but to inappropriate sinus tachycardia (which usually disappears after a few weeks), the persistence of atrial premature beats (a possibility which should be discussed with the patients before discharge) or the emergence of another supraventricular arrhythmia (like atrial flutter or sino-atrial reentrant tachycardia). Electrocardiographic documentation of the palpitations after ablation is thus paramount.

In a minority of patients no tachycardia can be induced during an electrophysiological study, although it may have been documented clinically and one-to-one antegrade slow pathway conduction is present. If no

Table 1 The essence of slowlfast AVNRT ablation

Diagnostic work-up
Exclusion of a concealed accessory pathway
Evidence for 1-to-1 antegrade slow pathway conduction
Evidence for retrograde fast pathway conduction
Mapping of the earliest atrial activation in the anterior (superior) septum, close to the His bundle
Ablation end-points
Elimination of 1-to-1 antegrade slow pathway conduction
Non-inducibility of AVNRT
Radiofrequency energy delivery
Catheter: 4 mm tip with a curve to reach the isthmus between tricuspid annulus and CS ostium
Power-controlled mode might be safer (20–25 W)
Based on the mapping of 'slow pathway' electrograms
If unsuccessful: extended anatomically to ostium CS, proximal CS, and/or low midseptum
Precautions to avoid complete heart block
RF delivery during sinus rhythm, without administration of isoprenaline
Continuous evaluation of the AH interval during sinus rhythm or retrograde fast pathway conduction during junctional rhythm

AH=atrio-His; AVNRT: atrioventricular nodal reentrant tachycardia; CS=coronary sinus; RF=radiofrequency.

other arrhythmia mechanism can be revealed, ablation of the slow pathway has been shown to lead to clinical long-term success in these patients. In 2% of our patients, there was neither inducible PSVT nor one-to-one slow pathway conduction, although PSVT suggestive of AVNRT was previously documented. We applied empirical RF energy at the slow pathway in these patients, based on the A_{SP} mapping. After a follow-up of 17 ± 12 months, none of them has had a recurrence of palpitations.

In 1–2% of the patients, the ablation may be abandoned at a certain stage (out of fear of creating AV block) without complete elimination of one-to-one antegrade slow pathway conduction or even elimination of tachycardia, but with the evidence that the conduction properties over the slow pathway have definitely changed (e.g. by lengthening of the Wenkebach cycle length, by eliminating the longest AH intervals, by decreasing the window of cycle lengths with one-to-one slow pathway conduction, and/or by decreasing the number of consecutive beats with conduction over the slow pathway before a final beat is blocked).

Complications

With an electrogram-guided posterior approach, induction of complete AV block during RF application is very rare. We observed it in only 1% of the patients and it was transient in all, with recovery of conduction within a few seconds, and 2 min in one patient. It may take a few hours, however, before the PR-interval returns to normal. Moreover, late third-degree AV block (occurring after the procedure but within 24 h) has been described. Close telemetry is thus required after transient AV block during the procedure or other indications of AV nodal 'collateral damage'. The incidence of permanent AV block requiring pacemaker implantation should be less than 1%, but can be higher[5]. Complications related to vascular access are usually mild. Cardiac perforation is extremely uncommon.

Indications

In this usually young patient population ablation should be advised only after a failed drug trial and/or the resolute refusal to take drug therapy by the patient, given the small but definite risk for complete AV block requiring pacemaker implantation which can never fully be excluded.

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