Measure twice, cut once: pitfalls in the diagnosis of supraventricular tachycardia

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In atrioventricular nodal and atrioventricular reentrant tachycardia, the relative timing of atrial and ventricular activation may sometimes be very similar, even during electrophysiological studies, and this may lead to an erroneous diagnosis and inappropriate treatment. As examples, we describe two cases that were recently referred to our hospital for a second opinion and treatment of paroxysmal supraventricular tachycardia. In both, the original diagnosis of the referring centres was common-type atrioventricular nodal reentrant tachycardia. Catheter ablation in those centres was unsuccessful. During our electrophysiological studies, however, an atrioventricular reentrant tachycardia was demonstrated, using a concealed accessory pathway for retrograde conduction in both patients. The accessory atrioventricular connection was successfully ablated and on follow-up both patients remained free of symptoms without medication. These findings illustrate the importance of complete electrophysiological analysis even for apparently simple supraventricular arrhythmias. (Neth Heart J 2010;18:78-84.)

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Typical atrioventricular nodal reentrant tachycardia (AVNRT) and atrioventricular reentrant tachycardia (AVRT) are common types of regular supraventricular tachycardias (SVT). Sometimes, the relative timing of atrial and ventricular activation is very similar and this may lead to an erroneous diagnosis and inappropriate treatment when the arrhythmia is only studied superficially. As examples, we describe two cases of AVRT using a concealed accessory pathway for retrograde conduction that were originally diagnosed and treated as AVNRT, jeopardising the normal atrioventricular (AV) node conduction system.

Patient 1

Clinical history

A young man (16 years) was referred with a three-year history of supraventricular tachycardia with sudden onset and end. The ECG during tachycardia showed the same QRS morphology as during sinus rhythm and no retrograde P waves were discernable. The patient had previously undergone an electrophysiological study elsewhere. During that study, a supraventricular tachycardia was induced and characterised as typical AVNRT. Radiofrequency (RF) ablation was performed in the AV-nodal area. However, the tachycardia remained inducible, and ablation had caused complete right bundle branch block (RBBB). The patient's symptoms had worsened after this first ablation procedure.

Electrophysiology study

After informed consent was obtained, the electrophysiological study was performed in a fasting, non-sedated state. Antiarrhythmic drugs were discontinued at least five half-lives before the study. Using a right femoral venous access, four catheters were inserted: a quadripolar electrode catheter was positioned in the high right atrium (HRA) and on the His bundle. A screw-in catheter (6416, Medtronic Inc, MN) was fixed in the right ventricular (RV) septum for RV stimulation and as reference for non-fluoroscopic catheter localisation (Localisa,

Medtronic Inc, MN). Finally an octapolar electrode catheter was placed in the coronary sinus. The baseline His-ventricular (HV) interval was 40 ms. To study ventriculoatrial (VA) conduction properties, a programmed ventricular extra-stimulus (S_2) with decreasing coupling interval was introduced after every eighth beat of a paced rhythm with a basic cycle length of 600 ms. During this stimulation protocol, retrograde conduction was present with a stable VA interval that did not prolong with decreasing ventricular S_2 interval. Earliest atrial activation was detected on the HRA catheter. During programmed atrial stimulation, no evidence of dual AV-nodal physiology was found: a decrease in S_2 interval of 10 ms never resulted in an increase in atrial-His (AH) interval of more than 50 ms.

Tachycardia characteristics

The tachycardia was reproducibly induced with an atrial extra-stimulus and also occurred spontaneously after atrial ectopy (figure 1). Apparently, only a small prolongation of the AH time was required to start the tachycardia. During tachycardia, the QRS complex was identical to that during sinus rhythm, suggesting anterograde conduction over the normal conduction system. Earliest retrograde atrial activation was recorded just after the QRS complex, as in common-type AVNRT. However, the earliest retrograde atrial activation was not recorded in the His bundle area, as would have been the case in common-type AVNRT, but at the HRA. During tachycardia, a premature ventricular stimulus was delivered in the tachycardia cycle at a time when the His bundle was still refractory. This extra-stimulus reproducibly resulted in termination of the tachycardia in the retrograde limb of the tachycardia cycle. This proved that atrioventricular reentry via a concealed accessory pathway was the tachycardia mechanism (figure 2). Tachycardia termination was due to refractoriness of a concealed accessory AV pathway when reached by premature ventricular activation.

Mapping and RF ablation

A steerable quadripolar mapping/ablation catheter with a 4 mm distal electrode (Biosense Webster Inc., Diamond Bar, CA) was inserted in the right atrium and visualised on the Localisa system. During RV stimulation at a cycle length of 400 ms, the site of earliest retrograde atrial activation was found superoanteriorly, at 1 to 2 cm distance from the bundle of His. RF current was delivered at a power of 50 Watt and a temperature limit of 62 °C. Interruption of retrograde VA conduction occurred within 4 sec after RF onset. During a 30-minute observation period, retrograde accessory pathway conduction remained absent and no other tachycardias could be induced. Antegrade conduction through the AV node remained unaffected.

Patient 2

Clinical history

A 57-year-old man was referred to our hospital with recurrent drug refractory palpitations for a second opinion. The patient had undergone two electrophysiological studies in the referring centre. In both studies, the diagnosis was common-type AVNRT and modification of the inferior AV node was attempted with multiple RF applications. However, ablation was unsuccessful. Finally, pacemaker implantation and ablation of the bundle of His was proposed.

The electrogram showed sinus rhythm with narrow QRS complexes. The patient had undergone coronary artery by-pass grafting for stable angina at age 52.

Electrophysiology testing

The electrophysiological study was performed in a fasting, non-sedated state, after informed consent had been obtained. Antiarrhythmic drugs were discontinued at least five half-lives before the study. The setup for the electrophysiological study was the same as in the previous patient, only this time the screw-in catheter was placed in the atrial septum to serve as positional reference for Localisa. A quadripolar catheter was placed in the RV apex. The baseline intracardiac recordings showed sinus rhythm with a normal HV time of 35 ms. As described above, VA conduction properties were studied. A programmed ventricular extra-stimulus with decreasing coupling interval was introduced after every eighth beat of a paced rhythm with a basic cycle length of 600 ms. Earliest retrograde atrial activation was detected at the screw-in catheter in the right atrial septum and the VA interval did not prolong with decreasing coupling interval.

During programmed atrial stimulation, we found no evidence of dual AV node physiology. However, an extra atrial response reproducibly occurred at an S_2 <470 ms, with earliest activation again in the atrial septum (figure 3). Adenosine administration (18 mg) during ventricular stimulation did not result in either VA block or in VA interval prolongation. This suggested VA conduction via a concealed septal accessory pathway. The presence of such an accessory pathway can be further investigated by para-Hisian pacing.¹ With a catheter on the proximal right bundle, just inside the RV, stimulation strength is gradually increased while monitoring QRS morphology and VA conduction (figure 4). At relatively low output, only local (septal) ventricular myocardium is captured resulting in a broad QRS complex. At higher output, the proximal right bundle can be captured too, leading to fusion of ventricular activation via the antegrade conduction system and local ventricular activation, resulting in narrowing of the QRS complex. In the

Figure 1. Supraventricular tachycardia in patient 1. After two sinus complexes, a spontaneous premature atrial extra-systole causes a small prolongation of the atrioventricular conduction time and the tachycardia starts with earliest retrograde atrial activation on the high right atrial (HRA) electrogram, not on the mapping (MAP) catheter that is positioned in the His (H) bundle area.

Figure 2. Patient 1. Delivery of a right ventricular (RV) extra-stimulus simultaneous with anterograde His (H) bundle activation. Refractoriness of the His bundle would prevent retrograde conduction through the atrioventricular node. The ventricular extra-systole, however, stops the tachycardia without retrograde atrial activation. This was reproducible and proves that the ventricle is part of the reentrant circuit and thus that an accessory pathway (AP) is present.

Figure 3. Right atrial (RA) pacing in patient 2. At a basic cycle length of 600 ms, an extra-stimulus (S_2) <470 ms reproducibly resulted in atrial activation that is earliest in the atrial septum (RA sept) and proximal coronary sinus (CS) electrogram with electrodes 7 and 8 near the CS ostium.

absence of a septal accessory pathway, retrograde conduction to the atrium via the His bundle and AV node is faster when the specialised conduction system is stimulated directly, because the activation wave front does not have to take the detour via the more distally located Purkinje network to reach the AV node. Thus, capture of the proximal right bundle causes narrowing of the QRS complex together with shortening of retrograde conduction delay to the atrium. In the presence of a septal accessory pathway, however, stimulation of the ventricular septum close to the bypass insertion usually results in (fast) retrograde atrial activation, irrespective of right bundle capture because retrograde conduction via a septal accessory pathway is typically much faster than via the AV node. In our patient, a higher output led to narrowing of the QRS while the VA interval remained relatively short and constant (figure 4).

Tachycardia characteristics

Tachycardia with a cycle length of 340 ms was reproducibly induced under isoproterenol infusion using a single atrial extra-stimulus (S_2) following a drive train (S_1) of eight stimuli. During tachycardia, retrograde atrial activation occurred shortly after the QRS complex. The earliest retrograde atrial activation was detected on the screw-in catheter in the atrial septum with a VA interval of 120 ms. Premature ventricular stimulation during the

tachycardia at the time of His-bundle refractoriness reproducibly resulted in advancement of retrograde activation of the atria (figure 5). This confirmed the presence of an accessory pathway. The advanced atrial activation also resulted in termination of the tachycardia, apparently due to AV nodal refractoriness (figure 5).

Mapping and RF ablation

Again, Localisa was used for 3D catheter localisation and labelling of important sites. Mapping of the earliest atrial activation during RV stimulation showed that the atrial insertion of the accessory pathway was very close to the bundle of His (figure 6). Ablation at that site carries a definite risk of the creation of heart block.^{2,3} While the optimal ablation site of a concealed accessory pathway can only be determined during retrograde accessory pathway conduction, delivery of RF energy while stimulating the RV will impede monitoring of anterograde AV nodal conduction. One way to eliminate the accessory pathway could be ablation during orthodromic tachycardia while monitoring both AV nodal conduction and retrograde AP conduction. Very often, however, interruption of the accessory pathway and termination of the tachycardia leads to dislocation of the ablation catheter and this too may cause accidental His bundle ablation. We therefore decided to mark the atrial insertion site, determined during RV stimulation,

Figure 4. Para-Hisian pacing in patient 2. Continuous stimulation on the proximal right bundle with increasing stimulus strength. At low output, only ventricular septal myocardium is captured while at higher output, both ventricular myocardium and the bundle of His are captured together. The latter results in a shorter stimulus-QRS delay and a narrower QRS complex. In the absence of a septal concealed accessory atrioventricular pathway, retrograde conduction to the atrium would be faster with direct His capture. This, however, does not occur, suggesting the presence of a septal retrogradely conducting accessory pathway.

Figure 5. Delivery of a single ventricular stimulus during tachycardia in patient 2. The extra-stimulus is delivered while the bundle of His is refractory. Advancement of ventricular excitation also advances retrograde atrial activation, proving the presence of an accessory ventricular-atrial connection. The tachycardia stops, most likely due to anterograde atrioventricular nodal refractoriness.

Figure 6. Image of the Localisa screen during mapping and ablation in patient 2. The location of the coronary sinus (CS) is visualised with the coronary sinus catheter and green dots, while the position of the bundle of His is marked with light blue dots. The purple and golden dots represent the site of early and earliest retrograde atrial activation via the accessory AV pathway respectively, during right ventricle (RV) stimulation. A single successful RF pulse was delivered at the location of the red dot a few mm superior to the bundle of His.

on Localisa and to perform the ablation during atrial stimulation while monitoring anterograde AV nodal conduction only. As monitoring tool, we did not rely on the relatively low speed electrograms of an electrophysiology system. Instead, we used a custom, high speed (1000 mm/sec), triggered electrogram monitoring system (Trema); a standard component of our EP armature that allows accurate, real time, beat-to-beat monitoring of activation intervals. RF power was gradually increased, starting at 5 Watt up to a maximum of approximately 15 Watt. After this application, ventricular stimulation was performed to investigate the presence or absence of the concealed accessory pathway. Retrograde conduction was absent, confirming successful ablation. Electrophysiological testing continued during a 30-minute observation period that included isoproterenol infusion, but tachycardias could no longer be induced and the procedure was terminated.

Follow-up

Neither of the two patients have reported any palpitations without antiarrhythmic medication and additional conduction disturbances were not detected during two-year follow-up.

Discussion

Initially, both patients were incorrectly diagnosed and treated for AVNRT, probably because of a short VA delay during tachycardia, rather similar to that of common-type AVNRT. We cannot exclude the presence of dual atrioventricular nodal electrophysiology during the initial EP studies in both patients, but VA conduction via a concealed accessory pathway was missed. Consequently, catheter ablation remained unsuccessful. In the first patient, symptoms even increased after the creation of RBBB and increased conduction delay to the right ventricle that facilitated retrograde conduction through the right-sided AP.

Diagnostic 12-lead electrocardiographic criteria have been developed to differentiate between AVNRT and AVRT⁴⁻⁷ and they can reach an accuracy of 84 to 90%. A right-sided accessory pathway, however, can lead to a short VA delay during AVRT very similar to that found during AVNRT. The characteristics, though, of those two arrhythmias are very different and so is the target site for ablation. These cases demonstrate the importance of complete and careful electrophysiological investigation in patients with SVT even in apparently 'simple' cases. ■

Author's note

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