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What to Do When Bumping Impairs Conduction of an Accessory Pathway?

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A 30–year-old lady underwent a repeated electrophysiology study for documented left bundle branch block (LBBB) QRS morphology tachycardia. During her first procedure antidromic atrioventricular re-entry tachycardia (AVRT) was diagnosed utilizing a right sided, anterograde-only accessory pathway (AP) with decremental conduction (Figure 1). There was no evidence of pre-excitation during sinus rhythm; however, pacing from the right atrial free wall revealed the AP conduction. During mapping at the tricuspid annulus using a steerable catheter, AP conduction disappeared and did not recover during prolonged waiting, precluding ablation of the AP. Palpitations soon returned and recurrence of AVRT was documented. At the beginning of the second procedure, the same tachycardia was easily induced; however, during mapping with an ablation catheter around the tricuspid annulus at the free wall, bumping of the AP impaired its conduction again. After this, AVRT was no longer inducible and high right atrial (HRA) pacing was conducted mainly with normal QRS complexes. Fully pre-excited, LBBB-like QRS complexes were seen only occasionally and unpredictably. At this point the question was: what can be done to be able to successfully ablate the AP?

1 | Commentary

The diagnosis of antidromic AVRT was established by a late premature atrial stimulus delivered close to the lateral tricuspid annulus that reproducibly reset the tachycardia by delaying the subsequent ventricular activation, without affecting septal atrial timing. Ventricular activation at the lateral tricuspid annulus is relatively late and occurs after retrograde His-bundle activation (Figure 1), pointing to a long, atriofascicular AP. Localizing atriofascicular fibers can be achieved by mapping the tricuspid annulus for the His-like AP potential during antidromic AVRT or HRA pacing [1]. However, bumping the AP during mapping is a common occurrence that can hinder effective ablation [2]. Intermittent, fully pre-excited QRS complexes, only seen with anterograde AV nodal block, can result due to prolongation of AP conduction by the mechanical trauma, in conjunction with retrograde, concealed penetration of the AP by impulses travelling through the AV node. This latter mechanism is thought to underlie some cases of unidirectional conduction in APs [3] and has been shown in a case of an atriofascicular pathway [4].

The atriofascicular pathway is a long fiber, extending from the lateral tricuspid annulus to the right ventricular apex. Several lines of evidence suggest that its distal insertion is directly into the right bundle branch (RBB) [5], by way of the moderator band [6]. Ablation is also possible at the distal insertion site [7], and this can spare mechanical AP block during mapping.

To promote anterograde conduction through the atriofascicular AP and impede it over the AV node, ventricular extrastimulation was utilized, which consistently elicited antidromic echo beats (Figure 2A). The premature ventricular stimulus conducts retrogradely through the AV node, making it refractory. At the same time the ventricular part of the AP is retrogradely penetrated by the stimulus, but this happens properly early compared to the event of the impulse, which conducts retrogradely through

Abbreviations: Abl, ablation catheter; AP, accessory pathway; AV, atrioventricular; AVRT, atrioventricular re-entry tachycardia; CS, coronary sinus; HRA, high right atrial; LBBB, left bundle branch block; RBB, right bundle branch; RF, radiofrequency; RV, right ventricular.

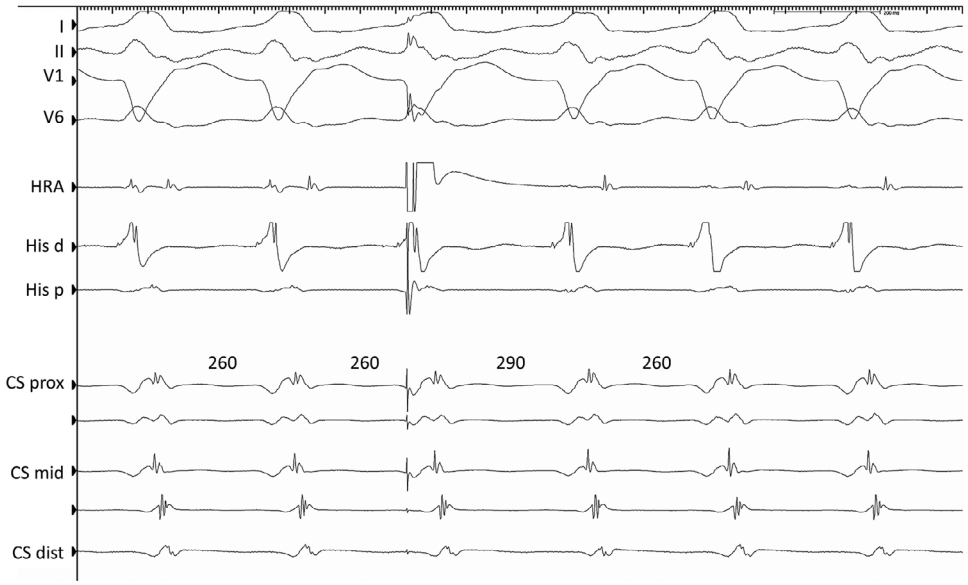

FIGURE 1 | A late atrial extrastimulus delivered from the high right atrium (HRA), close to the tricuspid annulus, at a time when septal atrial activation is committed (no change in timing of the immediate atrial signal on proximal coronary sinus (CS) recording, shown by numbers) resets (delays) wide QRS, preexcited tachycardia. Surface ECG leads I, II, V_1 and V_6 are shown, together with intracardiac recordings from the high right atrium (HRA), proximal and distal His-bundle region (His) and coronary sinus (CS).

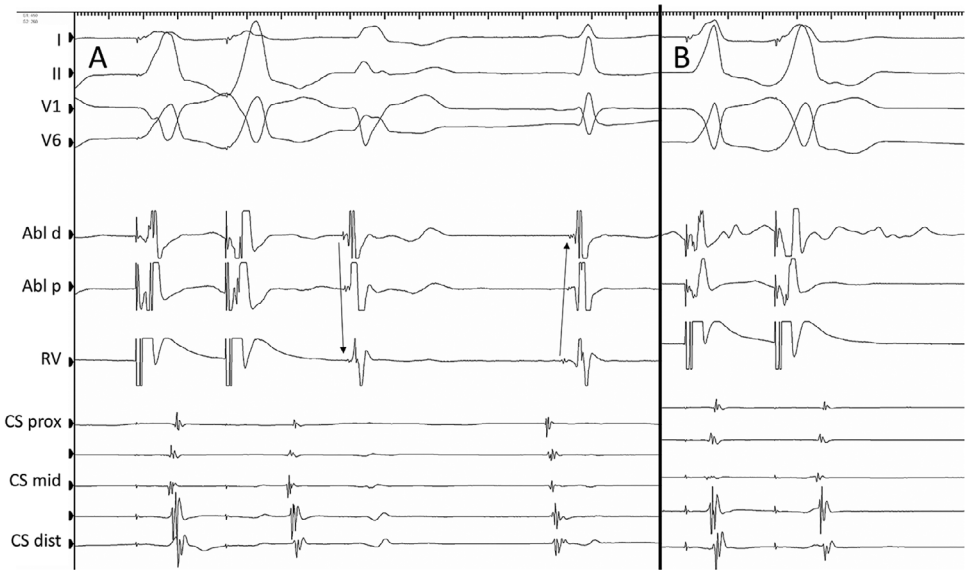

FIGURE 2 | Ventricular extrastimulation from the right ventricular septum (RV) reproducibly elicits antidromic echobeats (A). The position of the ablation catheter (Abl) is shown in Figure 3. Activation of the right bundle branch (shown by arrows) is distal to proximal during accessory pathway conduction and reversed during AV nodal conduction from a sinus beat. The echo beats have identical QRS-morphology to the tachycardia. After starting RF delivery, echobeats no longer can be induced (B). Other leads and abbreviations are the same as in Figure 1.

the AV node, reaching the atrial interface of the AP. This time difference allows recovery of excitability along the course of the atriofascicular AP and anterograde conduction of the impulse. The combined effect of increased AV nodal-, while "peeling back" of AP-refractoriness allows anterograde conduction exclusively over the damaged atriofascicular fiber.

To avoid further mechanical trauma to the AP mapping was undertaken in the right ventricle during antidromic echo beats following ventricular extrastimuli. Localization of the insertion of the atriofascicular pathway into the RBB was approached by a combination of activation mapping the earliest Purkinje potentials and anatomical imaging by intracardiac echocardiography. The ablation site was selected at a location where the right ventricular moderator band joins the septomarginal trabeculations (Figure 3). A single radiofrequency (RF) pulse eliminated AP conduction without damage to the RBB (Figure 2B). The patient has been without recurrence for nine months.

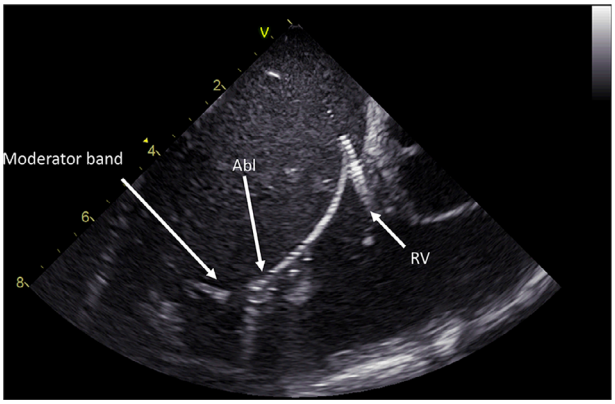

FIGURE 3 | Intracardiac echocardiography image from the right atrium ("home view"). The ablation catheter (Abl), with distal (d) and proximal (p) electrode pairs, is placed at the junction of the moderator band with the septomarginal trabeculations. Also shown is the right ventricular catheter (RV) at the basal septum.

2 | Conclusion

Catheter-induced trauma may be less likely when targeting the atriofascicular pathway at its distal insertion site, compared to annular mapping [7]. However, the development of RBB block can be a complication of ablating at the site of earliest ventricular activation [7]. Complementing activation mapping with anatomic imaging using intracardiac echocardiography to target the septal end of the moderator band may spare such a complication. When bump-suppression has already occurred, retrograde, concealed penetration from AV nodal impulses can contribute to loss of preexcitation beyond prolonged AP conduction. This may be overcome by ventricular extrastimulation increasing AV nodal and "peeling back" AP refractoriness to elicit antidromic echo beats. These allow activation mapping, and their elimination serves as a procedural endpoint when all else fails.

Author Contributions

Gábor Bencsik contributed to the conception, drafting, and review of the manuscript. Róbert Pap contributed to the drafting and editing of figures and the interpretation of data.

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The authors have nothing to report.

Ethics Statement

This study was approved by the Ethics Committee of the University of Szeged.

Consent

The patient provided written informed consent.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data supporting the findings of this paper are available within the manuscript. Additional data are available upon request from the corresponding author.

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