Confronting an entrenched atrioventricular node: Ablation strategies for an elusive target

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Introduction

Prior cardiac surgery and intracardiac instrumentation can create significant anatomical barriers to ablation, requiring the use of alternative ablation strategies. We describe a challenging case of atrioventricular (AV) node ablation in a patient with prior atrial septal repair, 2 patent foramen ovale closure devices, tricuspid valve ring annuloplasty, bioprosthetic mitral valve replacement, and a pacemaker with a His lead.

Case report

We present a 71-year-old male patient with significant structural heart disease. He underwent surgical repair of septum secundum atrial septal defect with recurrence requiring transcatheter Amplatzer closure. He developed atrial fibrillation/flutter (AF/AFL) and underwent radiofrequency ablation. Secondary to myxomatous mitral valve disease with bileaflet prolapse, moderate mitral stenosis, and moderate-to-severe tricuspid regurgitation, he required bioprosthetic mitral valve replacement, tricuspid ring annuloplasty repair, maze procedure, and patent foramen ovale closure. Owing to significant postoperative bradycardia, he underwent implantation of a dual-chamber permanent pacemaker with His bundle pacing. For recurrent atrial septal defect, he underwent repeat transcatheter Amplatzer closure. In follow-up, he was found to be in drug-refractory symptomatic persistent AF/AFL with recurrent episodes of rapid ventricular response. Owing to his complex history, he was deemed a poor candidate for repeat AF/AFL ablation. After extensive discussion, he was scheduled for AV nodal ablation.

A 4 mm Navigation catheter (CARTO; Biosense Webster, Irvine, CA) was advanced into the right atrium, and 3D mapping of the cardiac chambers was performed. The patient's tricuspid valve ring annuloplasty, septal closure devices, and His lead (Figure 1) constituted significant physical barriers to positioning the ablation catheter near the compact AV node, as the His signal was difficult to appreciate. In the presence of extensive hardware, moreover, mapping and appreciation of electrograms altogether was challenging. Specifically, mapping the septum was hindered by multiple septal closure devices, and mapping the anterosuperior area was hindered by the His lead. Subsequently, high-output His bundle pacing was attempted in order to localize the His bundle and was unsuccessful. As multiple attempts at ablation near the presumed anatomical compact AV node failed to achieve heart block, retrograde aortic access with ablation in the septal area just below and in the noncoronary cusp was performed. Mapping was performed without the advantage of a multispline multipolar mapping catheter, and in the setting of AF, the His signal was not appreciated retrogradely. Despite this maneuver, only transient heart block was achieved.

Subsequently, the coronary sinus and its ostium were mapped, and anatomical slow pathway ablation (superior and anterior to coronary sinus os) was performed, with accelerated junctional beats noted. With continued ablation through the accelerated junctional beats, the rhythm became fast, and ultimately complete heart block with an escape rhythm of 30 beats per minute (bpm) was achieved (Figure 2). This rhythm persisted after a waiting period of 20 minutes. Postprocedurally, the patient's device and lead function were normal. The patient was programmed at DDDR 90 bpm and was subsequently seen in the outpatient setting with satisfactory clinical progress.

Discussion

AV junction ablation is a relatively simple procedure commonly used for refractory rapid AF to control the ventricular rate.[1] It can occasionally, however, prove to be a technically challenging procedure. Typically, AV junction ablation is performed by targeting the compact AV node. As the current recording systems are not capable of recording the electrical signals from the AV node, the His signal is therefore used as a surrogate for the AV node. Usually, an ablation catheter is used to map and record the His region, and an

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KEY TEACHING POINTS

- Prior cardiac surgery and intracardiac instrumentation can create significant anatomical barriers to ablation, requiring the use of alternative ablation strategies. We describe a challenging case of atrioventricular (AV) node ablation in a patient with prior atrial septal repair, 2 patent foramen ovale closure devices, a tricuspid valve ring, and a His lead.
- In our patient, extensive mapping from the right side, high-output His bundle pacing, and a left-sided approach failed to localize the His bundle and resulted in unsuccessful attempts at AV nodal ablation. This difficulty was compounded by the need to ablate proximally to the His bundle area to avoid block distal to the His lead.
- Whereas the slow pathway potential was not mapped because of atrial fibrillation, the anatomic slow pathway area anterior and superior to the coronary sinus ostium was ablated using 50 W. Initially, slow junctional beats were noted, and the ablation was continued through the junctional rhythm, which subsequently accelerated and became rapid. The patient not only developed complete heart block but demonstrated a favorable junctional escape rhythm at 30 beats/min.
- It is possible that the fast pathway or even compact AV node might have been displaced posteriorly and inferiorly, and ablation in the presumed anatomical slow pathway area would have resulted in the ablation of these structures.
- Therefore, the anatomical slow pathway area can be targeted in a difficult-to-localize AV node. Whereas inducing rapid accelerated junctional rhythm during slow pathway ablation for AV nodal reentrant tachycardia is typically avoided, in selected instances this exact mechanism may be harnessed to achieve heart block in an otherwise difficult-to-ablate AV node.

ablation is performed in the area just posterior to where a His signal is recorded. During difficult AV node ablations, various strategies, including the use of long and steerable sheaths, irrigated-tip catheters, and left-sided AV junction ablations, have been shown to be helpful. One of the reasons for difficulty ablating the AV junction is failure to obtain a clear His bundle signal on the electrode catheters. AF per se, as well as rapid ventricular rates, can also interfere and obfuscate the His signal. Unsuccessful ablation attempts,

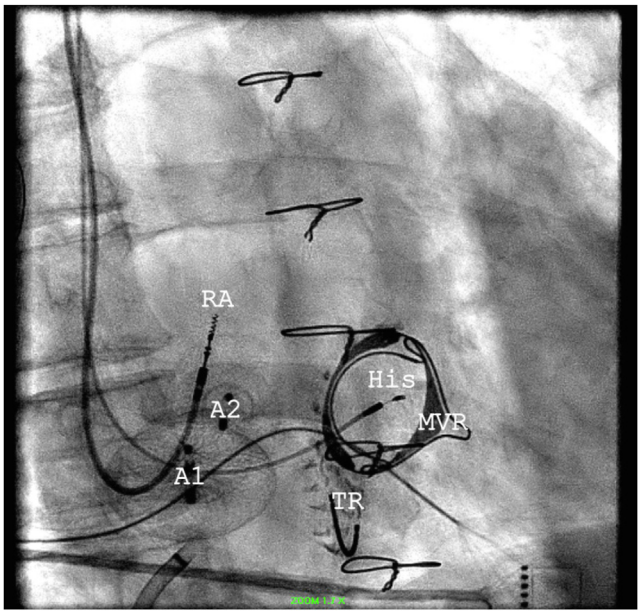

Figure 1 Fluoroscopic imaging demonstrating the patient's extensive cardiac instrumentation in the setting of known structural heart and conduction system disease. RA = right atrial lead fixated in the right atrial appendage. A1, A2 = Amplatzer atrial septal occluder devices; His = His lead fixated in the His bundle position; MVR = Edwards bioprosthetic mitral valve replacement; TR = Medtronic tricuspid annuloplasty ring.

moreover, can induce tissue edema, which in turn further attenuates the His bundle signal. Ablation from the left side using a transseptal or retroaortic approach has been proposed in patients who have unsuccessful ablation from the right side.[2] However, both procedures carry an increased risk of arterial injury and stroke. High-output His bundle pacing can also help localize the His bundle by producing a narrower QRS complex at the pacing site.[3–6]

In our patient, extensive mapping from the right side, high-output His bundle pacing, and a left-sided approach failed to localize the His bundle and resulted in unsuccessful attempts at compact AV nodal ablation. Our patient had undergone multiple surgical and transcatheter procedures, including septal closure surgery, 2 septal closure devices, mitral valve replacement, and tricuspid valve repair. In his postsurgical course, the patient underwent His bundle lead implantation. These procedures and implants created anatomical barriers to accessing the His region safely. Specifically, given the vicinity of the 2 septal closure devices and the His bundle lead, unclear signals and catheter instability created significant difficulty in mapping. It also became crucial to ablate proximally to the His bundle area to avoid causing block distal to the His lead. It is possible that multiple surgeries and presence of hardware may have displaced the compact AV node more posteriorly and inferiorly. This could also explain why heart block could not be achieved using a left-sided retrograde approach. After multiple attempts failed to safely localize and ablate the AV node, it was decided to perform ablation anterior and superior to the coronary sinus ostium (the anatomical slow pathway). The objective was to modify the AV node to slow the conduction during rapid

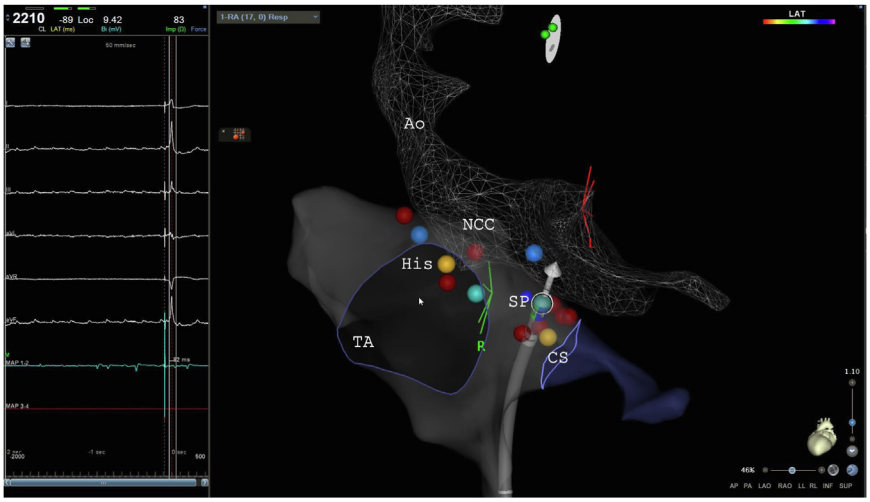

Figure 2 CARTO (Biosense Webster) 3D map demonstrating the 3 ablation strategies used. First, the area posterior to the His bundle (yellow marker next to "His" label) was ablated, indicated by teal and red markers. Second, the septal area below the noncoronary cusp (NCC) was ablated, indicated by the blue markers. Third, the anatomical slow pathway (SP), located superior and anterior to the coronary sinus os (CS), was ablated, indicated by ablation catheter and the teal marker outlined in white. On the left, the electrogram demonstrates ventricular pacing after the ablation of the slow pathway, further confirming complete heart block. TA = tricuspid annulus.

AF. Once the slow pathway was localized anatomically, 50 W ablation was performed in the area. Initially, slow junctional beats were noted, and ablation was continued through the junctional rhythm; while ablation was continued, the junctional beats became very rapid, and heart block was ultimately achieved.

One of the feared complications of slow pathway ablation is complete heart block. The occurrence of accelerated junctional beats during ablation is a nonspecific response to the heating of a slow pathway, fast pathway, or even a compact AV node. Although accelerated junctional beats are a marker of efficiency during slow pathway ablation for AV nodal reentrant tachycardia, an operator ceases ablation when accelerated junctional beats become too fast or there is loss of 1:1 retrograde AV conduction—ie, predictors of impending heart block. In our patient, we used the same principle to achieve complete heart block with a favorable junctional escape rhythm at 30 bpm. As accelerated junctional beats were not noted during ablation at prior sites, it is to be acknowledged that the fast pathway or even the compact AV node could have been displaced posteriorly and inferiorly to the area where the slow pathway is typically located. Given that the His signal was not recorded, however, definitive localization of the compact AV node in such a situation is difficult. Moreover, it is difficult to differentiate between accelerated junctional beats of the slow vs fast pathway. Because the slow pathway potential could not be appreciated in the setting of AF, the assumption of slow pathway ablation is based on the anatomic location anterior and superior to the coronary sinus os. It has therefore been demonstrated in this case that continued anatomical slow pathway ablation through an accelerated junctional rhythm can be an extremely helpful strategy to achieve heart block in a challenging AV nodal ablation.

Conclusion

This case highlights significant technical challenges to AV nodal ablation in a patient with numerous prior surgical and invasive cardiac interventions and device implantations. Ultimately, whereas a fast accelerated junctional rhythm during slow pathway ablation for AV nodal reentrant tachycardia is typically avoided, this exact mechanism was harnessed to achieve complete heart block in an otherwise difficult-to-localize-and-ablate compact AV node.

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