

Cardioneuroablation instead of pacemaker implantation in a young patient suffering from permanent 2:1 atrioventricular block after a slow pathway ablation



Alan Bulava, MD, PhD,^{*†} Thiago Guimarães Osório, MD,[‡] Jiri Hanis, MD,[†]
Carlos Thiene C. Pachón, MD,[§] José Carlos Pachón, MD, PhD,[§]
Carlo de Asmundis, MD, PhD, FHRS[‡]

From the ^{*}Faculty of Health and Social Sciences, the University of South Bohemia, Ceske Budejovice, Czech Republic, [†]Department of Cardiology, Ceske Budejovice Hospital, Ceske Budejovice, Czech Republic, [‡]Heart Rhythm Management Centre, Postgraduate Course in Cardiac Electrophysiology and Pacing, Vrije Universiteit Brussel, Universitair Ziekenhuis Brussel, Brussels, Belgium, and [§]São Paulo University and São Paulo Heart Hospital, São Paulo, Brazil.

Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common regular supraventricular tachycardia. Invasive treatment of the arrhythmia consists of the modification of the slow pathway (SP) using radiofrequency (RF) catheter ablation.¹ Although such treatment has a success rate close to 98%, atrioventricular (AV) block can occur in up to 2%–3% of patients during or after catheter ablation procedures.²

“Cardioneuroablation” (CNA) is a method initially proposed to modulate cardiac parasympathetic tone with the goal of removing or attenuating the functional cardioinhibitory response in cardioinhibitory syncope.^{3–5}

Here, we report on the case of a young patient who was successfully treated using CNA, after an inadvertent 2:1 AV block, which occurred after a second SP ablation that resulted in permanent bradycardia with an indication for a pacemaker.

Case report

A 19-year-old woman underwent a SP ablation owing to recurrent AVNRT in a different center. Two months after the first procedure, she presented with a recurrence of the arrhythmia. A redo ablation resulted in a permanent 2:1 AV block with a heart rate (HR) of 46 beats per minute (bpm). Since she was able to tolerate the bradycardia, which

KEYWORDS Cardioneuroablation; AV conduction block; Parasympathetic denervation; AV nodal reentry tachycardia; Complications; Catheter ablation (Heart Rhythm Case Reports 2020;6:261–264)

Alan Bulava and Thiago Guimarães Osório contributed equally to the article as first authors. **Address reprint requests and correspondence:** Dr Alan Bulava, Department of Cardiology, Ceske Budejovice Hospital, Ceske Budejovice, B. Němcové 54, České Budějovice, 370 01, Czech Republic. E-mail address: alanbulava@seznam.cz.

KEY TEACHING POINTS

- Cardioneuroablation may be an effective alternative method to definitive pacemaker implantation for the treatment of atrioventricular conduction disturbances caused by radiofrequency catheter ablation of the slow pathway in patients with atrioventricular nodal reentry tachycardia.
- A significant functional reserve of the atrioventricular node must be determined by the atropine test prior to cardioneuroablation.
- The extent of vagal denervation during cardioneuroablation (ie, acute efficacy) should be evaluated based on the presence of a cardioinhibitory reaction during extracardiac vagal stimulation.

presented as occasional dizziness, but never a syncope, she refused implantation of a pacemaker at that time. At the age of 25, she expressed a desire to become pregnant and was referred to our center by a gynecologist, for permanent pacemaker implantation.

Investigations

Initially, a 12-lead surface electrocardiogram (ECG) (Figure 1, left side) and a 24-hour ECG Holter were performed. The Holter monitoring showed almost permanent type 2 second-degree AV block with a minimum documented HR of 32 bpm during sleep. The minimum waking HR was

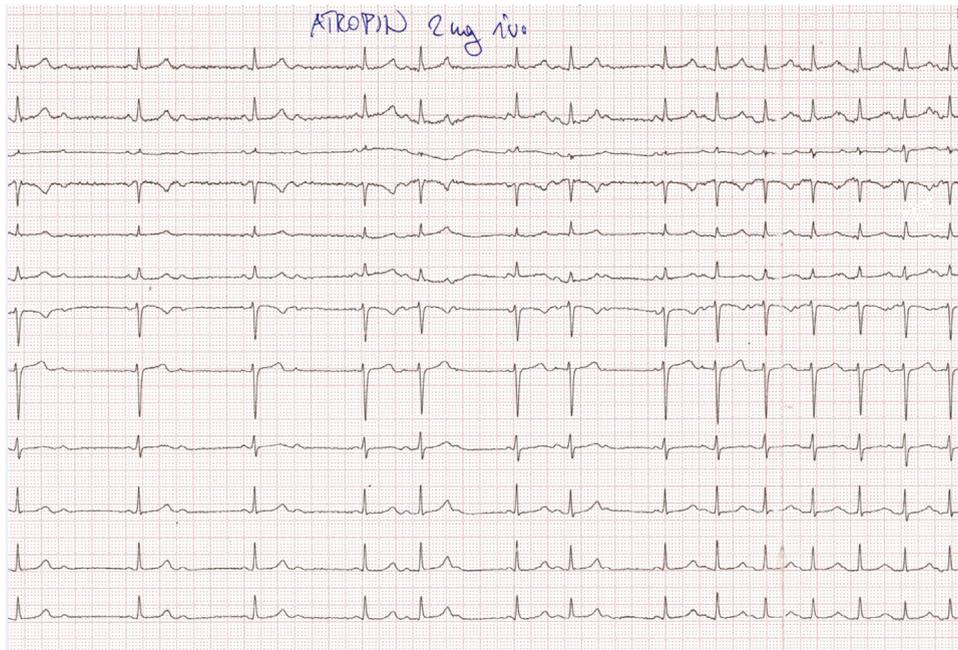


Figure 1 Initial 12-lead electrocardiogram shows constant 2:1 atrioventricular block (left side), which ceases after administration of 2 mg atropine intravenously (right side).

44 bpm. The maximum documented HR was 162 bpm during exercise. Continuing the investigation, an atropine test with 2 mg intravenous was performed; during the test, normal AV conduction was seen, suggesting a functional AV conduction block (Figure 1, right side).

Management

The patient was scheduled for CNA complemented with extracardiac vagal stimulation (ECVS), which was performed under general anesthesia in early February 2019. To avoid an increase in HR, we commenced the ablation in the antrum of the left superior pulmonary vein. During the application of RF current, we observed a vagal reaction manifesting as a complete AV dissociation (Figure 2A). Normal 1:1 AV conduction was first regained during ablation in the area of the left ridge between the left inferior pulmonary vein and the auricle (the presumed area of neurovegetative innervation input to the left atrium accompanying the vein [or ligament] of Marshall, Figure 2B). After this ablation, the Wenckebach point (WP) was 750 ms. We then continued with the RF applications at the inferior septum—close to the roof of the coronary sinus—and further shortening of AH interval was observed from 75 to 45 ms, and WP increased to 460 ms. We finished the procedure once there was no more AV block during atrial pacing after ECVS (Figure 3) in both the right and left vagus. Afterward, we administered 2 mg of atropine intravenously, which alone increased the HR to 100 bpm but had no effect on the WP, which remained unchanged (ie, 460 ms), suggesting a maximum WP achievable by the cardiac denervation. Incremental atrial pacing from the coronary sinus catheter up to a cycle length of 200 ms was performed at the very end of the procedure to

exclude any proarrhythmic effect of the ablation lesions. No arrhythmias were induced.

Follow-up

Two weeks following the procedure, the patient was in normal sinus rhythm and reported an improvement in her quality of life (QoL), tolerance to physical exercise, and a complete absence of fatigue and dizziness. Her resting ECG showed normal sinus rhythm: HR 77 bpm, PQ 160 ms, QRS 90 ms with no repolarization abnormalities.

After 10 months, the patient was still asymptomatic and reported further QoL improvement. Since February 2019, she has undergone 3 24-hour postprocedural ECG Holter recordings, with almost identical results: average HR 82–84 bpm, minimum HR 37 bpm (always between 2:00 am and 4:00 am, ie, at night during sleep), maximum HR 180 bpm (waking hours). No drops of HR below 60 bpm or the presence of Wenckebach-type second-degree AV block was documented during waking hours. No blood pressure abnormalities were noted during all in-office follow-ups.

Discussion

To the best of our knowledge, this is the first case report that shows a correction of an AV block after SP ablation with CNA. Briefly, according to the current guidelines, treatment for this patient should be a dual-chamber pacemaker.⁶ We report here, after a meticulous investigation and thorough discussion with the patient, a different approach to the treatment of an AV block that avoids pacemaker implantation into a young patient and all the potentially negative consequences stemming from the long-term presence of a cardiac stimulation system. The reason why we believed that CNA would be beneficial in this particular patient was, apart from the

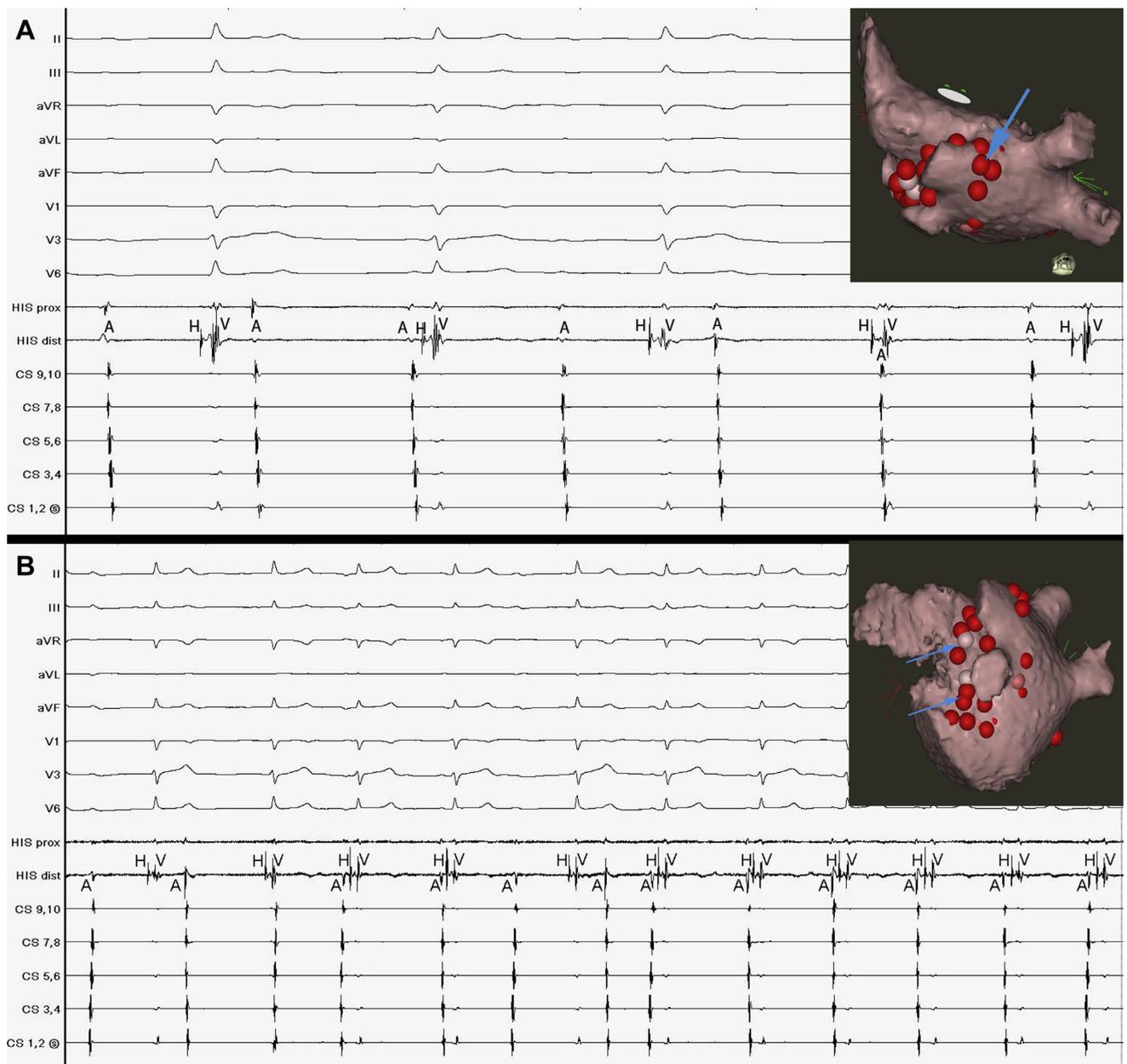


Figure 2 A: After ablation in the roof portion of the left superior pulmonary vein (blue arrow), complete atrioventricular (AV) dissociation occurred. B: Normal 1:1 AV conduction was first regained during ablation in the area of the left ridge between the left inferior pulmonary vein and the auricle (blue arrows). A = atrial signal; H = His bundle signal; V = ventricular signal.

positive reaction to atropine, the fact that no complete AV block was observed after AVNRT ablation and that the patient was able to regain 1:1 AV conduction during strenuous exercise. AV conduction impairment definitely had an iatrogenic origin resulting from structural injury caused by the previous SP ablation, not excessive parasympathetic tone alone; however, reducing even “normal” parasympathetic tone resulted in normalization of AV conduction, at least during waking hours and during the majority of the sleeping cycle. Our case confirms that apart from iatrogenic damage to the AV node area, parasympathetic tone may also play a role in patients with AV conduction disturbances after SP ablation. The borderline between reversible or nonreversible

changes caused by SP ablation is, however, definitely difficult to establish. A positive reaction to atropine, a young age with no structural heart disease, and at least intermittent 1:1 AV conduction during exercise might be a clue. A recently published case of successfully reduced parasympathetic tone using CNA, which allowed for a subsequent successful ablation of the SP in a young patient with AVNRT, suggests that increased parasympathetic tone may also play a role in AV node conduction disturbances after SP ablation.⁷

The initially proposed indication for the CNA procedure was reflex cardioinhibitory syncope. The ablation procedure is usually performed in both atria, and the targets are the ganglionated plexi and the atrial parasympathetic

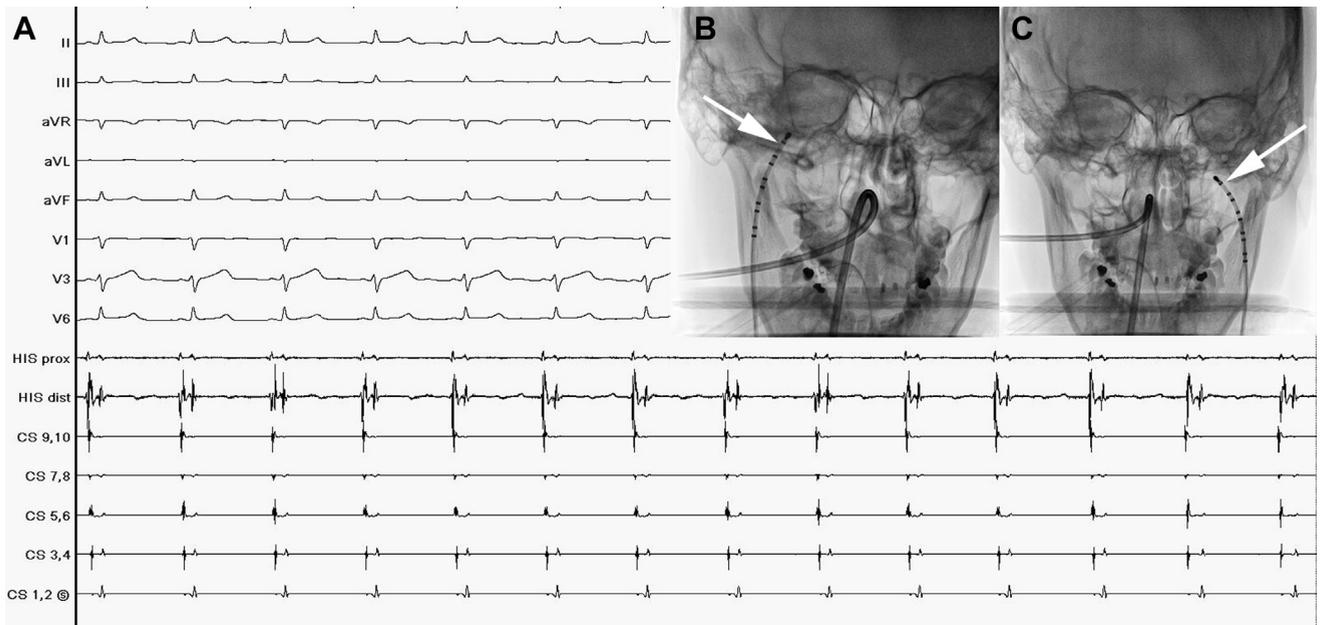


Figure 3 No atrioventricular (AV) block was observed during vagal stimulation (50 Hz, 50 μ s pulses, 5 s), both from the right and from the left jugular vein at the end of the procedure. **A:** 1:1 AV conduction was fully reestablished and persisted on the surface 12-lead electrocardiogram during follow-up. **B,C:** A standard decapolar steerable catheter (white arrow) introduced high into the jugular vein was used for vagal stimulation (B: position of the catheter in the right jugular vein; C: position of the catheter in the left jugular vein).

innervation. The ablation can be guided by different methods, including high-frequency stimulation, iodine-123 meta-iodobenzylguanidine imaging (D-SPECT), fractionation mapping, and/or spectral mapping, but recently the empiric anatomical approach has been increasingly applied in most procedures. Nevertheless, the extent of CNA procedures should be determined by its acute efficacy, which is evaluated based on the presence of a cardioinhibitory reaction during ECVS. During the vagal stimulation, a standard electrode is introduced either to the right or left jugular vein close to the anatomical position of the vagal nerve and 50 Hz alternate current of specific waveform and amplitude is applied for the time of 5 s (see also Figure 3). Vagal irritation usually causes either a complete asystole (sinoatrial and AV block) or rarely, and only when pacing on the left side, sinus bradycardia accompanied by complete AV block.

Our endpoint, in this case, was the complete abolishment of a functional AV block induced by ECVS in both the right and left jugular vein, during atrial pacing, which prevented the atria from total standstill owing to sinoatrial impulse suppression when vagally stimulated. We avoided ablating in the anatomical areas correlated with sinus node vegetative innervation. In this manner, we avoided significant increases in HR and possible worsening of the AV block in the event that disruption of the AV node parasympathetic innervation failed.

Conclusion

In this case, CNA was an effective alternative method to definitive pacemaker implantation for the treatment of AV block with significant functional reserve. The ECVS was easy to

perform and made it possible to define an appropriate endpoint during the procedure (ie, extensive selective AV node parasympathetic denervation), a fact confirmed by the complete absence of a response to atropine in the AV nodal territory at the end of the procedure. We strongly believe that by avoiding pacemaker implantation, we dramatically improved not only the patient's QoL but also her long-term prognosis.

Acknowledgments

The authors would like to thank Tom Secrest for proofreading the manuscript. The authors would also like to thank the Foundation Cardiac Center Ceske Budejovice for their financial support covering publication fees.

References

1. Bulava A, Peichl P, Pandozi C, et al. Atrioventricular nodal reentrant tachycardia: anatomical and electrophysiological considerations. *Ital Heart J* 2003;4:163–172.
2. Clague JR, Dagues N, Kottkamp H, Breithardt G, Borggrefe M. Targeting the slow pathway for atrioventricular nodal reentrant tachycardia: initial results and long-term follow-up in 379 consecutive patients. *Eur Heart J* 2001;22:82–88.
3. Pachon JC, Pachon EI, Pachon JC, et al. "Cardioneuroablation" - new treatment for neurocardiogenic syncope, functional AV block and sinus dysfunction using catheter RF-ablation. *Europace* 2005;7:1–13.
4. Pachon JC, Pachon EI, Cunha Pachon MZ, Lobo TJ, Pachon JC, Santillana TG. Catheter ablation of severe neurally mediated reflex (neurocardiogenic or vasovagal) syncope: cardioneuroablation long-term results. *Europace* 2011;13:1231–1242.
5. Pachon JC, Pachon EI, Santillana TG, et al. Simplified method for vagal effect evaluation in cardiac ablation and electrophysiological procedures. *JACC Clin Electrophysiol* 2015;1:451–460.
6. Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay. *J Am Coll Cardiol* 2019;140:e382–e482.
7. Roubicek T, Wichterle D, Kautzner J. Cardioneuroablation in a patient with atrioventricular nodal re-entrant tachycardia. *Europace* 2018;20:2044.