Cardioneuroablation: don't underestimate the posteromedial left atrial ganglionated plexus.

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Abstract

Introduction Cardioneuroablation (CNA) is a technique used to modulate cardiac parasympathetic tone in patients with sino-atrial (SA) and atrio-ventricular (AV) vagally mediated syncope. We describe the case of a patient who developed AV block after a first procedure of CNA, requiring a second procedure. Case presentation A 47-Year-old man presented with recurrent syncope (daily episodes) associated with high vagal tone conditions. An ECG monitoring showed frequent episodes of sinus bradycardia and sinus arrest, with pauses up to 17 seconds. AV node conduction impairment was never identified. A CNA procedure targeting the right superior and posterior ganglionated plexi (GPs), both from the left and right atrium, was performed with acute success. The subsequent night, repetitive episodes of AV block with normal sinus rate were observed. A second procedure was performed targeting the posteromedial left GP. Follow-up at 4 months showed no recurrent syncopal event and no bradyarrhythmia episode on the implantable loop recorder. Conclusion This case report demonstrates that ablation limited to the right superior and posterior GPs may not be enough for neurocardiogenic syncope and a more systematic approach, extending the ablation to the posteromedial left GP, should be considered.

Cardioneuroablation: don't underestimate the posteromedial left atrial ganglionated plexus.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

FUNDING

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DATA AVAILABILITY STATEMENT

Data of this study are available upon reasonable request from the corresponding author.

ABSTRACT

Introduction

Cardioneuroablation (CNA) is a technique used to modulate cardiac parasympathetic tone in patients with sino-atrial (SA) and atrio-ventricular (AV) vagally mediated syncope. We describe the case of a patient who developed AV block after a first procedure of CNA, requiring a second procedure.

Case presentation

A 47-Year-old man presented with recurrent syncope (daily episodes) associated with high vagal tone conditions. An ECG monitoring showed frequent episodes of sinus bradycardia and sinus arrest, with pauses up to 17 seconds. AV node conduction impairment was never identified. A CNA procedure targeting the right superior and posterior ganglionated plexi (GPs), both from the left and right atrium, was performed with acute success. The subsequent night, repetitive episodes of AV block with normal sinus rate were observed. A second procedure was performed targeting the posteromedial left GP. Follow-up at 4 months showed no recurrent syncopal event and no bradyarrhythmia episode on the implantable loop recorder.

Conclusion

This case report demonstrates that ablation limited to the right superior and posterior GPs may not be enough for neurocardiogenic syncope and a more systematic approach, extending the ablation to the posteromedial left GP, should be considered.

Keywords: Cardioneuroablation, catheter ablation, syncope, vagal denervation, ganglionated plexi.

CASE

A 47-year-old man, with a medical history of OSAS and gastric bypass surgery for obesity presented repetitive episodes of reflex syncope. These episodes first appeared in 2010 and were associated with vegetative symptoms. Tilt table testing in another institution was able to reproduce the reflex syncope and demonstrated a VASIS type 1 profile (mixed response with drop in blood pressure and bradycardia, no asystole). In 2018 episodes progressively increased in frequency, and additional neurological investigation with an EEG and a brain MRI were performed, without any abnormal findings. Syncopal episodes became extremely frequent and recurred on a daily basis by the end of 2021. In November 2021 the patient underwent a video EEG, during which vasovagal sinus arrest was shown to precede the syncopal event (Figure 1 in the data supplement). The patient was then referred to our institution.

Physical examination was completely normal, blood pressure was 120/80 mmHg. The ECG showed sinus rhythm at 67 bpm, normal AV and IV conduction without repolarization alterations. Blood tests were unremarkable. He was under budesonide/formoterol and salbutamol inhalation therapy for asthma. During the ECG monitoring, different episodes of sinus bradycardia and symptomatic sinus pauses were documented, lasting up to 17 seconds at night. Since all the episodes were concomitant to high vagal status conditions and associated with parasympathetic signs and symptoms, a CNA procedure under general anaesthesia was scheduled.

The patient was brought to the EP laboratory on January 26th, 2022. He was in sinus rhythm at 59 bpm. A preliminary EP study showed borderline SA nodal function (after a 1 minute of atrial pacing at 600 ms

SNRT was 1460 ms, cSNRT was 420 ms). AV and HV intervals were respectively 50 and 46 ms, and the AV Wenckebach point (WP) was 540 ms.

The vagal nerve stimulation with a quadripolar catheter placed at the level of the right jugular vein (Micropace system, frequency: 30 Hz, pulse width: 0,5 ms, current intensity: 25 mA) elicited a moderate response with a heart rate drop of 17 bpm (from 63 bpm to 46 bpm).

A map of both right and left atria (Figure 1) was obtained using a multipolar catheter (PentaRay, Biosense Webster Inc, Diamond Bar, CA). Ablation of the right superior and posterior ganglionated plexi (GP) was performed using a CT-guided anatomical approach, both from left and right atria. The CT segmentation was merged with the bi-atrial electro-anatomical map.

After the ablation, the EP parameters were tested again, with an improvement of SNRT (1060 ms, pacing at 600 ms) and WP at 360 ms. Right vagal stimulation no longer elicited a significant heart rate drop.

During the first night after the procedure, the patient had a recurrent episode of syncope with high grade AV block (Figure 2 in the data supplement). Sinus bradycardia or arrest was no longer recorded.

The patient was brought back to the EP laboratory the next day for a second procedure, this time under conscious sedation. Basal HR was 83 bpm. AH, HV, and PR intervals were respectively 50, 54, and 138 ms, the AV WP was 320 ms and AV ERP was 250 ms for a baseline cycle length of 600 ms. Since general anaesthesia was not available vagal nerve stimulation was not performed. The posteromedial left atrial GP was targeted this time, at the level of the coronary sinus ostium, from the right and left atria (Figure 2). After the ablation there was no sensible change in basal HR, the PR interval was slightly reduced to 120 ms (AH 32 ms, HV 56 ms), the WP decreased to 290 ms and AVN ERP decreased to below 200 ms.

A loop recorder (Biotronik BIOMONITOR) was implanted before discharge. At 4 months of follow-up, no bradyarrhythmias or recurrent syncopal episode were documented, and the patient has had no recurrent syncope.

LEARNING OBJECTIVES

To define the effect of different GPs on SA and AV function.

To understand the importance of ablating ganglia converging both on the SA node and AV node.

To not overestimate the acute success indicators of CNA.

DISCUSSION

Cardioneuroablation is a treatment for neurocardiogenic syncope, consisting of autonomic denervation via catheter ablation of GPs in both left and right atria[1]. Mainly described as a technique to modulate sinoatrial bradyarrhythmia, recent evidence has also shown efficacy for the treatment of vagally mediated AV block[2].

GPs are embedded in epicardial fat pads[3] and there is no general consent on nomenclature. The ones commonly targeted, according to the classification made by Armour et al[3], are the superior right atrial GP (SRGP) and the posterior right atrial GP (PRGP), also referred as a single element, the right atrial GP (RAGP), respectively on the posterior superior surface of the right atrium adjacent to the superior vena cava and on the posterior surface of the right atrium adjacent to the interatrial groove, the superior left GP (SLGP) on the posterior surface of the left atrium between the pulmonary veins, the inferior left GP (ILGP) located in the inferoposterior area around the root of the left inferior pulmonary vein, the posterior right atrial GP (PRGP) on the posterior surface of the right atrium adjacent to the interatrial groove and the posteromedial left atrial GP (PMLGP) located between coronary sinus ostium and lower part of the LA[3,4].

In canine models, there was a predominance of right vagal projections ending on SA nodal tissue[5]. The posterior and superior right GPs have been demonstrated to mediate vagal influences preferentially via the SA node [5,6], and SRGP stimulation in humans has shown to affect the SA node activity without AH interval prolongation [7,8]. In animal models, the sole ablation of the SRGP has been shown to mitigate both the

right and left vagal nerve stimulation-induced bradycardia, but to reduce only right vagal nerve mediated AH interval prolongation, without significant effect on the left vagal nerve influence[9]. In humans, there were no statistical differences in heart rate modification after SLGP, ILGP, and RIGP ablation, whereas heart rate increased significantly after SRGP (which was referred as right anterior GP) ablation[10]. For this reason, Right Superior and Right Posterior GPs are considered by some authors as the primary targets of cardioneuroablation.

On the other hand, stimulation of the left vagus nerve elicited a greater change in AV conduction time than did right vagal stimulation[11]. In addition, supramaximal left vagal stimulation is more likely to produce severe AV block than right vagal stimulation[12]. Based on different canine studies, this effect seems to be mediated through the Postero-Medial Left GP (PMLGP), located at the inferior vena cava - left atrial junction, in close proximity to the CS ostium[5,6].

Our patient had repetitive episodes of SA bradycardia and SA block, without AV conduction alterations. There is currently no consensus on how to perform CNA, hence our initial approach was conservative, and anatomically guided ablation[13] was restricted to the Right Superior and Right Posterior GPs, which innervate the SAN. For the same principle and the mentioned physiological reasons, vagal stimulation was performed only from the right side.

This case report demonstrates that ablation restricted to the SRGP/PRGP may not be enough for neurocardiogenic syncope due to sinus arrest, even after what could be considered a good acute outcome. Functional AV block may be masked by the concomitant SA bradyarrhythmia. A more systematic approach, extending the ablation to the PMLGP, should be considered.

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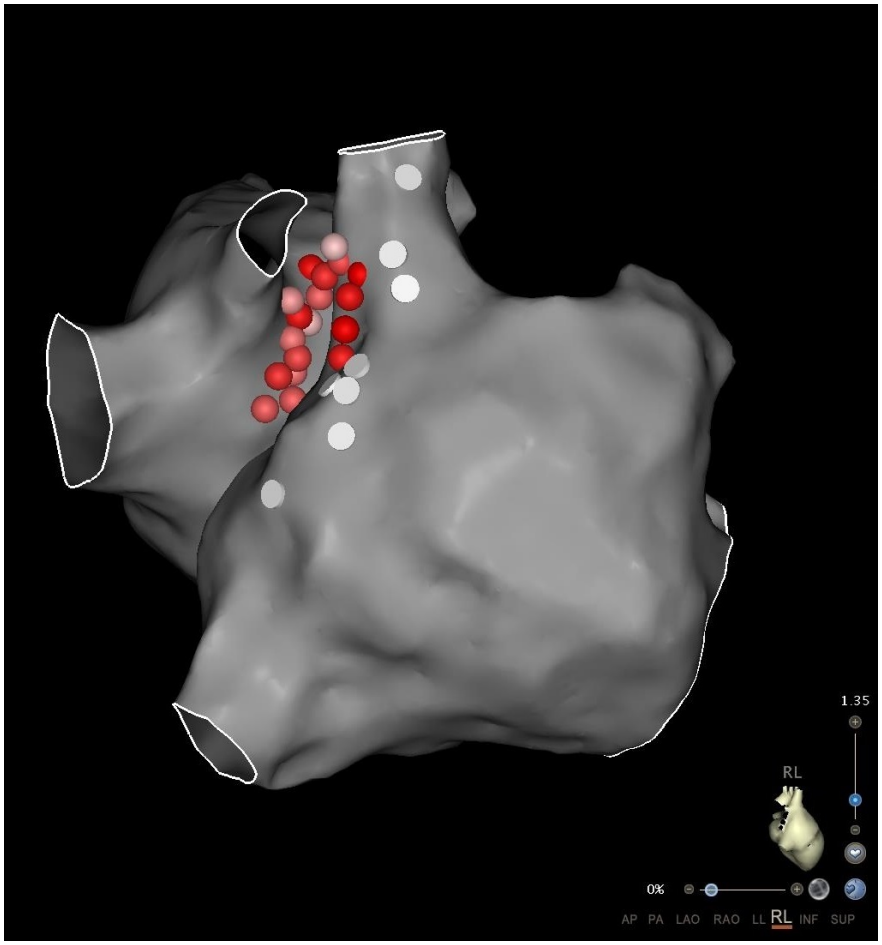

Figure 1

Three-dimensional mapping of the right and left atria at the time of the first procedure. White dots represent phrenic nerve capture at high output pacing, red dots represent ablation sites.

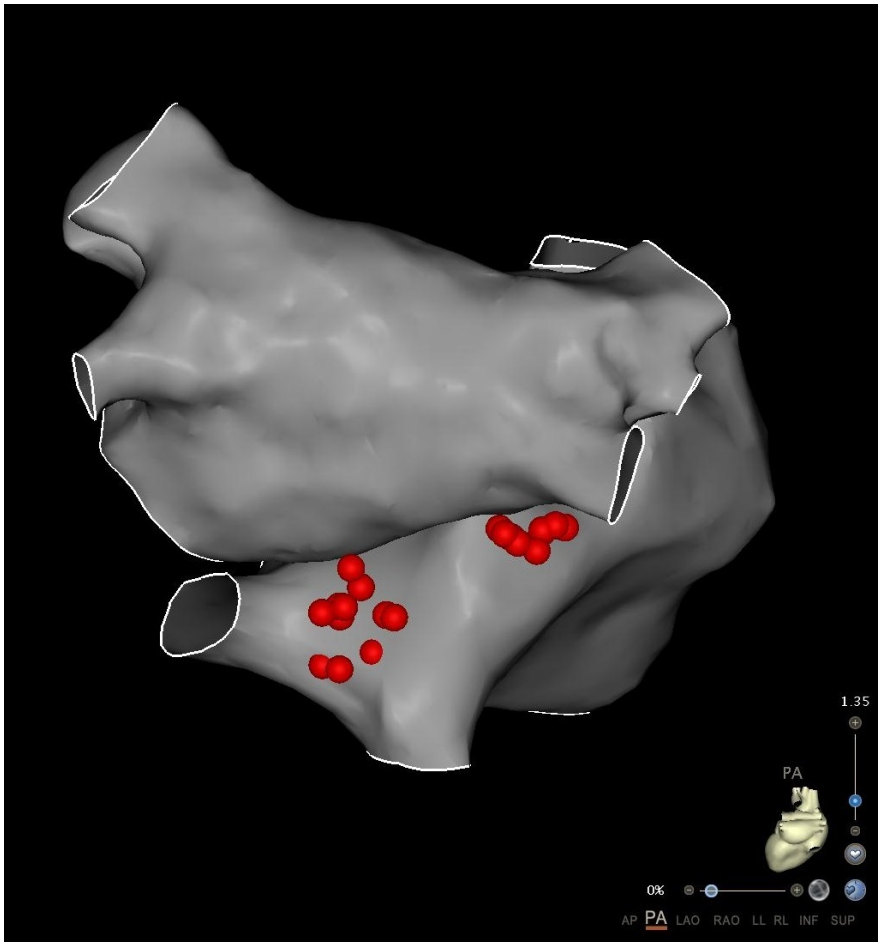

Figure 2

Three-dimensional mapping of the right and left atria at the time of the second procedure. Red dots represent ablation sites.