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SPOTLIGHT

Cardioneuroablation for carotid sinus syndrome mediated by complete atrioventricular block

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Carotid sinus hypersensitivity is an enhanced response to pressure given to the carotid sinus at the carotid bifurcation, resulting in bradycardia, vasodilation, and hypotension which when accompanied by syncope is defined as the carotid sinus syndrome (CSS).[1] The mainstay treatment of cardioinhibitory-type CSS is implantation of a permanent pacemaker, although this procedure is not without short and long-term complications. We present a case of CSS patient who has refused pacemaker implantation and was successfully treated with cardioneuroablation.

A 77-year-old man presented with a 6-month history of repetitive syncope of unknown etiology occurring on weekly basis. Blood chemistry, including sodium, potassium, calcium, and magnesium levels, was insignificant, blood pressure was 110/70 mmHg, and the ECG demonstrated sinus rhythm at 70 bpm. The patient had a structurally normal heart, and the left ventricular ejection fraction was 60%. An invasive electrophysiological study was scheduled to assess sinoatrial and conduction system properties. The procedure was performed under conscious sedation with midazolam and fentanyl administered intravenously. All basal measurements were within normal range (basal cycle length: 825 msec, QRS duration: 98 msec), as were sinoatrial nodal recovery times and atrioventricular conduction properties (AH interval: 80 ms, HV interval: 48 ms, atrioventricular Wenckebach cycle length: 370 ms). During the right-sided carotid sinus massage, a reproducible complete AV block ensued without sinus arrest or sinus bradycardia. (Figure 1) Since atropine administration completely abolished the vagal response, upon discussion with the patient who was unwilling for pacemaker implantation, cardioneuroablation was planned. The next day biatrial cardioneuroablation procedure (Figure 2), with anatomical targeting, as previously described, was performed.[2] During the procedure, we started by mapping and ablation at the left atrium, selecting ablation targets by both empirical anatomical location of ganglionated plexi and the presence of >4 deflections on the local electrograms. Radiofrequency energy was delivered with power set to 30–35 W, for at least 30 s, with a target contact force of 8–20 g. Abolition of all local electrograms at targeted sites, as well as >20% decrease in basal cycle length, was utilized as endpoints. Postprocedural measurements were as follows: basal cycle length: 610 ms, AH interval: 76 ms, and HV interval: 48 ms. The carotid sinus massage performed after the procedure, the day following the procedure and 2 months later was negative and the patient reported complete resolution of all syncopal symptoms.

Vagal denervation by cardioneuroablation has been shown to achieve good clinical results with cardioinhibitory-type reflex syncope, although most of the studies are single-center observational reports.[2] Due to the similar efferent mechanism of the cardioinhibitory CSS mediated by vagal nerve, potential therapeutic effect of vagal denervation may be hypothesized. Indeed, there are rare case reports of cardioneuroablation in this condition.[3–5] We have performed biatrial cardioneuroablation in contrast to Palama et al., aiming for durable lesions and potentially less vagal reinnervation which was observed and reported by Zerpa Acosta et al.[3,5]

Optimal technique of cardioneuroablation in this patient population is unknown. In all previously mentioned reports, sinus bradycardia and arrest were the dominant clinical presentations; therefore, cardioneuroablation targeting both the sinus node and the atrioventricular node was performed.[3–5] Although we have also targeted all efferent input into both nodes, whether targeting atrioventricular efferent input only would suffice remains to be elucidated.

In conclusion, we have presented a patient with an atrioventricular block-mediated cardioinhibitory CSS, which was successfully treated by cardioneuroablation. Systematic studies are necessary to further demonstrate the clinical utility of cardioneuroablation in this setting.

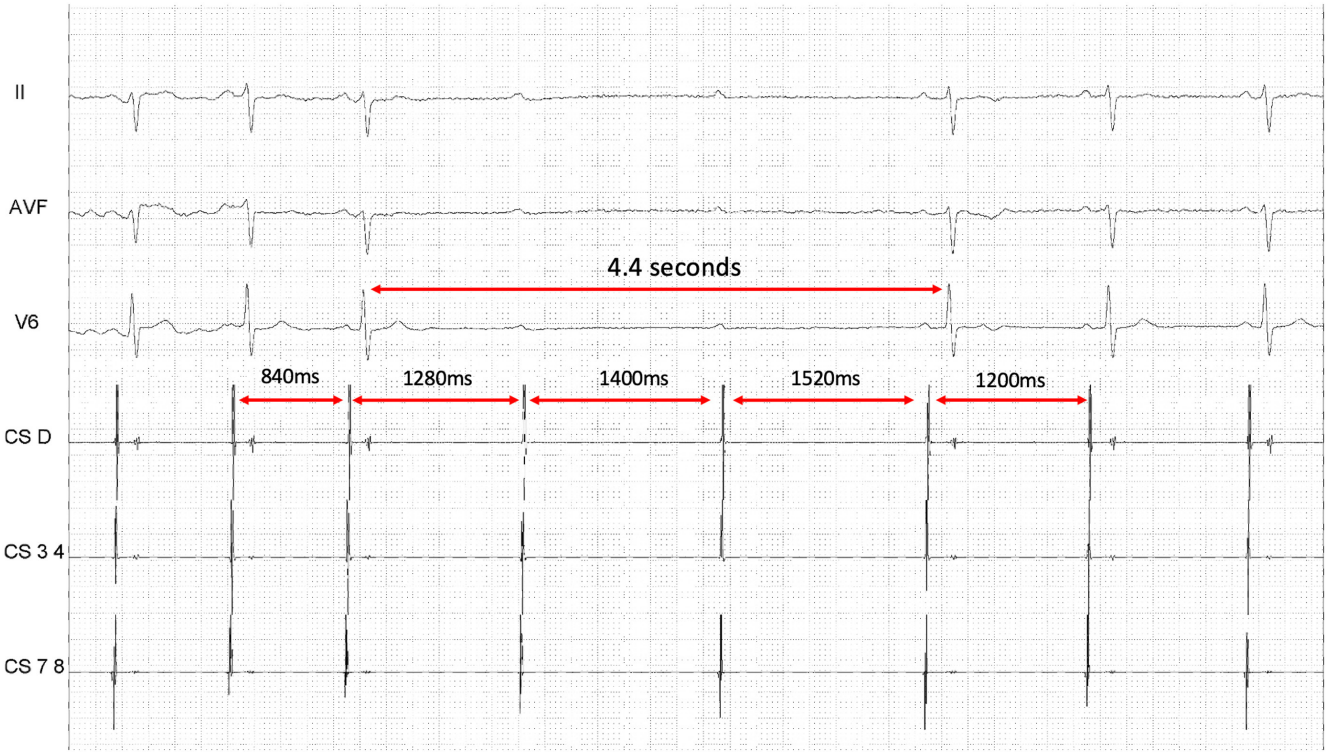

FIGURE 1 Intracardiac recordings obtained at the onset of the carotid sinus massage. The figure depicts three surface electrocardiogram recordings (DII, AVF, and V6) and recordings from a diagnostic catheter inside the coronary sinus (CSD, CS34, and CS78). At the onset, sinus node slows down, albeit without sinus pause and there is complete atrioventricular block which recovers after carotid sinus massage is stopped.

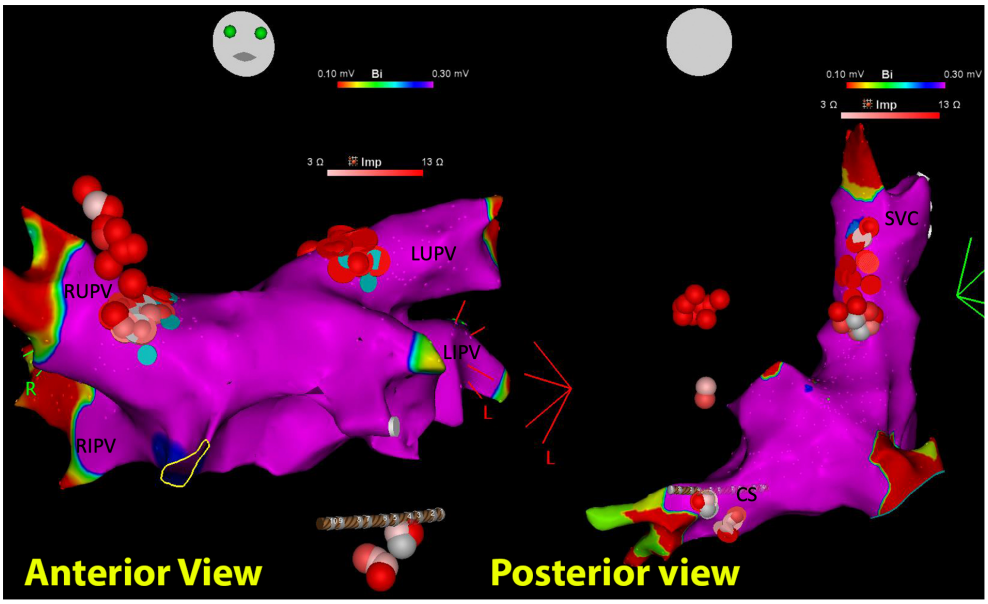

FIGURE 2 Electroanatomic maps of the left (anterior view) and right (posterior view) atria as well as ablation targets. We have targeted all major parasympathetic ganglia including ganglia located posterior to the coronary sinus which is the major efferent input into the atrioventricular node. Radiofrequency ablation was performed with power set to 30–35 W for at least 30 seconds. Red tags depicted on the figures are automatic ablation tags color-coded by impedance drop (VisiTag, Carto 3, Biosense-Webster, Diamond Bar, CA, USA). Pulmonary vein anterior antrums are marked by blue tags. At the end of the procedure, there was no vagal response to carotid sinus massage. CS, coronary sinus; LIPV, left inferior pulmonary vein; LUPV, left superior pulmonary vein; RIPV, right inferior pulmonary vein; RUPV, right superior pulmonary vein; SVC, superior vena cava.

CONFLICT OF INTEREST STATEMENT

We report no conflict of interest.

ETHICS STATEMENT

Ethics approval was obtained from local committee.

CONSENT FOR PUBLICATION

The patient was informed about the study procedure and provided a written consent to participate.

CLINICAL TRIAL REGISTRATION

N/A.

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