EP CASE REPORT

Cardioneuroablation in a patient with atrioventricular nodal re-entrant tachycardia

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Radiofrequency (RF) catheter ablation of ganglionic plexi—cardioneuroablation—has been proposed as a new treatment option for recurrent neurally mediated syncope and functional disorders of the sinoatrial (SA) and the atrioventricular (AV) nodes.[1–3]

This case report describes a 32-year-old female patient without any significant comorbidity who was referred due to recurrent sustained palpitations and chronic fatigue associated with presyncopal spells. The physical examination was unremarkable. She had a normal echocardiogram and negative tilt test. The electrophysiological study revealed the presence of dual AV nodal physiology with very easily inducible typical AV nodal re-entrant tachycardia (AVNRT). Furthermore, intermittent significant sinus and junctional bradycardia were also observed. Unexpectedly, transient high degree AV block developed during the first RF application at the AV nodal slow pathway region. As a result, the procedure was abandoned. Subsequent atropine administration increased the heart rate from 45 to 98 b.p.m., suggesting a functional disorder of the SA node and presumably concomitant functional disorder of the AV node.

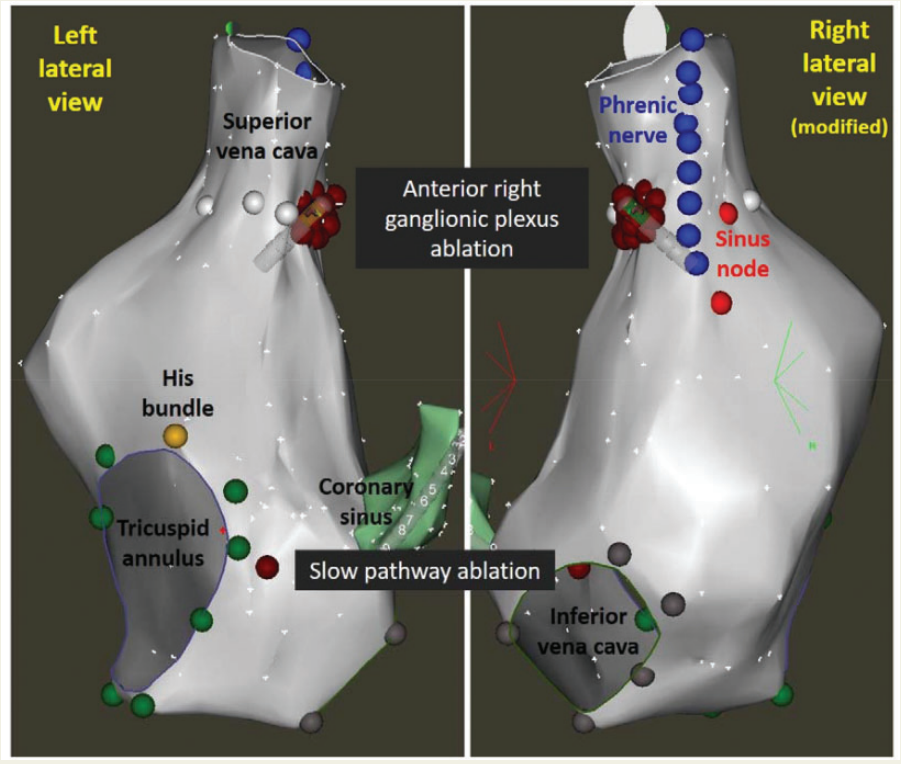

Figure 1 Map of the right atrium with ablations of the anterior right ganglionic plexus and slow pathway of the AV node.

The patient was offered a repeat procedure in a tertiary centre with the prospect of modulating the SA and AV nodal function with cardioneuroablation and subsequently ablating the slow pathway. At the start of this procedure, typical AVNRT was repeatedly induced by catheter manipulation in the AV nodal region, as well as by single atrial extrastimulus. A 3D mapping system and contact force sensing catheter (CARTO 3, ThermoCool® SmartTouch®, Biosense Webster) were used for mapping and ablation. Firstly, the right atrium was mapped and the sinus node and area of phrenic nerve capture were tagged. The former was defined as the area of earliest atrial activation and the phrenic nerve was found by pacing with an output of 10 mA leading to diaphragm contraction. Secondly, energy was delivered (25 W in power-controlled mode, 15 mL/min, total RF time: 180 s) at the posterior right atrial-superior vena cava junction, i.e. the empirical site of the anterior right ganglionic plexus (*Figure 1*). Ablation accelerated the sinus rhythm from 40–60 to 80 b.p.m., prolonged the AH interval from 80 to 100 ms, and increased the Wenckebach point from 80 to 130 b.p.m. Given such favourable effect of limited cardioneuroablation

on AV node conduction properties, ablation of the inferior left atrial ganglionic plexi was considered unnecessary. Surprisingly, the inducibility of AVNRT was significantly modified after the aforementioned ablation, with isoproterenol challenge and two atrial extrastimuli being required for induction. Finally, a single RF lesion at the AV nodal slow pathway was performed, which rendered AVNRT non-inducible. No AV conduction disturbances were observed. The patient has been completely asymptomatic for more than 1 year of follow-up, with no significant palpitations or chronic fatigue and no further documentation of diurnal bradycardia.

In conclusion, limited cardioneuroablation in the right atrium facilitated uneventful and safe ablation of the slow pathway in a patient with typical AVNRT and functional SA and AV nodal disorders. Associated symptoms suggestive of low cardiac output due to intermittent sinus or junctional bradycardia were also ameliorated by cardioneuroablation. Finally, this case nicely demonstrated how the inducibility of AVNRT can be influenced by modulation of the neural input to the AV node.

Supplementary material

Supplementary material is available at *Europace* online.

Conflict of interest: T.R. and D.W.: none declared. J.K.: Advisory board member - Bayer, Biosense Webster, Boehringer Ingelheim, Boston Scientific, Medtronic, MSD, St Jude Medical - Abbott, Microport.

References

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