

Optimized lesion geometry using very high-power short-duration ablation in catheter ablation adjacent to the phrenic nerve

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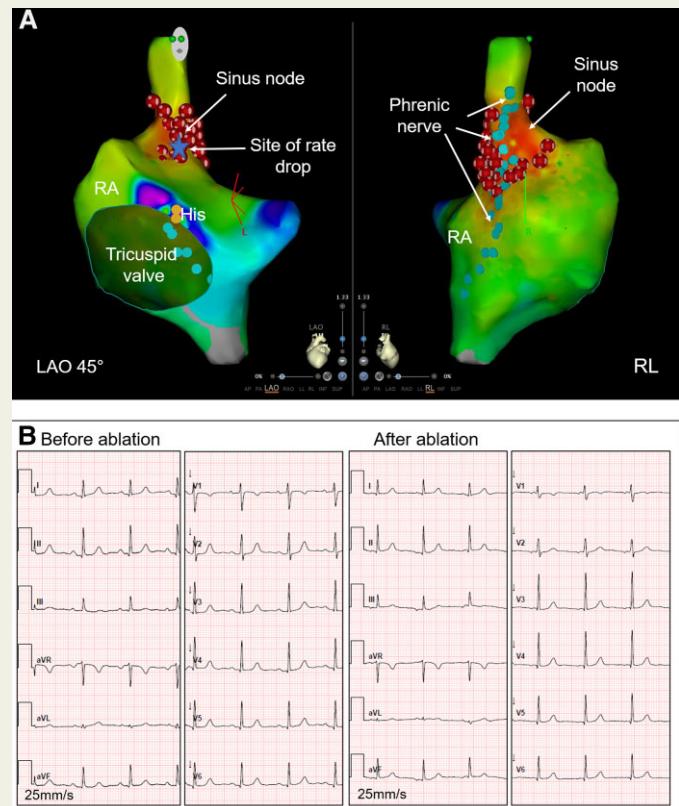
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Catheter ablation of atrial arrhythmias with high power and short applications (high-power short-duration, HPSD) emerged recently as an alternative to conventional ablation modes characterized by application of longer duration with low-power energy settings. During HPSD ablation, myocardial lesions are created by a greater amount of direct (resistive) rather than indirect (conductive) heating when compared with conventional ablation settings.¹ The HPSD may result in more effective, broader, and superficial lesion formation potentially avoiding collateral damage to adjacent structures like the oesophagus and the phrenic nerves (PNs).^{2,3} We present a case of right atrial (RA) ablation in an area with close relation to the PN which was facilitated by the use of HPSD.

A 38-year-old female patient with a history of symptomatic tachycardias and with diagnostic findings in line with inappropriate sinus tachycardia was referred to our department for electrophysiological therapy. Previously, medical therapy with beta-blockers, calcium channel blockers, and ivabradine was ineffective and not tolerated. On patients request, invasive evaluation and modulation of the sinus node area was planned. The patient presented with a basal heart rate (HR) of 80–110 b.p.m. at the start of sedation propofol, midazolam, and fentanyl. Electrophysiological study did not reveal inducibility of atrial arrhythmias as well as documentation of an accessory atrio-ventricular pathway and of dual atrioventricular node physiology.

High-density mapping of the RA was conducted using an electroanatomical 3D-mapping system (CARTO V7; Biosense Webster, Irvine, CA, USA) with a multi-electrode catheter (PENTARAY; Biosense Webster). Mapping was performed during basal HRs of 80–100 b.p.m. and the site of earliest atrial activation was identified (Panel A). Isoprenaline infusion (1 mL boli, continuous infusion with 15 mL/h) led to rapid increase of the HR to a maximum of 160 b.p.m. without induction of atrial arrhythmias. Afterwards, mapping of the earliest atrial activation during isoprenaline infusion was documented. The course of the PN was tagged via high output pacing from the mapping catheter along the lateral RA wall (Panel A). Ablation was targeted at the border zone of the sinus node area at sites of earliest RA activation during isoproterenol administration at maximum HR and without early activation during baseline conditions. To prevent PN paralysis (PNP), 'very HPSD' ablation was conducted with an ablation catheter facilitating temperature-controlled ablation with a maximum of 90 W over 4 s per application (Qdot with Qmode+; Biosense Webster). Ablation aimed at a target temperature of 60°C based on the maximum value of all four thermocouples and power settings as well as catheter irrigation were variable during ablation to avoid excessive temperature rise. A contact force of 5–15 g was aimed during ablation.

During ablation adjacent to the PN diaphragm movement was detected by manual palpation but no nerve paralysis occurred. Ablation resulted in a sudden HR drop with junctional escape rhythm (spot marked with star in figure). After recovery of SR, a significant decrease in HR to 80–100 b.p.m. with and without adrenergic stimulation could be observed. A total of 42 ablation applications with 168 s ablation time were delivered. At the day after the procedure, electrocardiogram revealed a change in P-wave morphology to a superiorly directed P-wave at 80 b.p.m. (Panel B). No radiographic and clinical signs of PNP were obvious. During follow-up, the patient reported on clinical improvement with marked reduction in inappropriate sinus tachycardias and without clinical signs of PNP.



This case highlights the potential of 'very HPSD' ablation to avoid collateral damage to adjacent structures beneath targeted myocardium due to optimized lesion formation for catheter ablation of atrial arrhythmias.

References

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