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Cryo-Balloon Ablation of the Right Superior Pulmonary Vein Involving the Anterior Right Ganglionated Plexus for Speech and Breathing Induced Atrial Tachycardia

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Abstract

Targeting ganglionated plexi (GP) during catheter ablation of atrial fibrillation (AF) is associated with improved outcome. We present a patient with speech and breathing induced atrial tachycardia (AT) originating in the superior vena cava (SVC) and the right superior pulmonary vein (RSPV), near the anatomical location of the anterior right GP (ARGP). The trigger for the arrhythmia appeared to be vagal discharge from the GP, possibly induced by local stretch. Ablation with a 28 mm cryo-balloon advanced to the RSPV orifice through a patent foramen ovale (PFO) abolished the arrhythmia, probably involving the underlying parasympathetic influx to the SVC and RSPV myocardial sleeves.

Introduction

Speech induced AT is a rare and peculiar condition .[1] Very few cases of respiratory cycle dependent AT (RCAT) have been described .[2] Both etiologies implicate involvement of the cardiac autonomic nervous system (ANS) in the initiation of the arrhythmia, however their concomitant occurrence has not been reported. The origin of RCATs was shown to be in the base of the heart, in close proximity to the ARGP,[2] which is probably involved in the mechanism of arrhythmia. Hence, ablation of the GP in these patients may improve outcome. Similarly, in patients with atrial fibrillation, addition of GPs ablation to PV isolation conferred significantly better outcomes than PV isolation alone .[3]

Case Report

A 46-year-old male patient otherwise healthy, presented with repetitive short bouts of AT occurring constantly while speaking and at mid inspiration. The arrhythmia was provoked even by social talking, and was not associated with stress or physical activity. Electrocardiography (ECG) during the tachycardia revealed a P wave biphasic in V1, positive in the inferior leads and isoelectric in L1, suggesting origination from the SVC or the RSPV. A tall P wave in L2 favored an SVC origin [4] (figure 1A). Holter monitoring revealed 50-700 premature atrial contractions (PACs) per hour and more than 3000 short episodes of AT lasting 3-16 beats. Beta-blockers and class 1C antiarrhythmic drugs (AAD) were ineffective. The patient underwent an electrophysiological study (EPS) under deep sedation.

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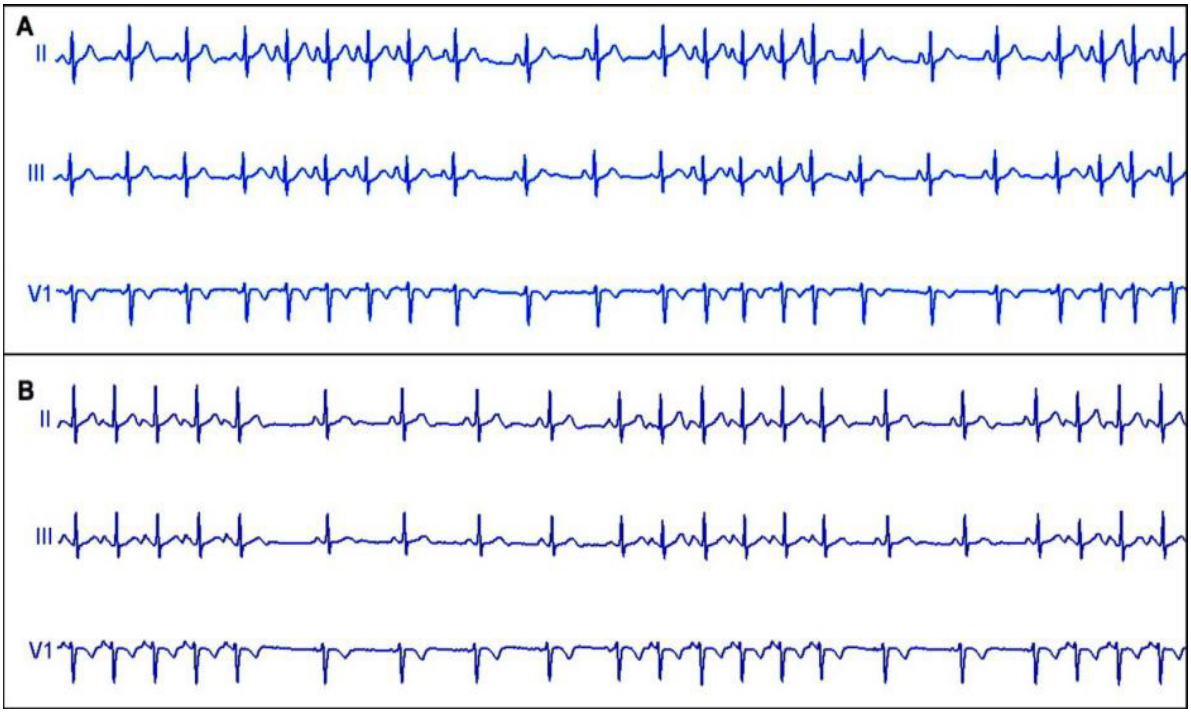
Body surface ECG and endocardial electrograms were monitored continuously with a computerized multichannel recording system (EP Tracer, CardioTek B.V. , Maastricht, The Netherlands) at a sample setting of up to 1000 Hz. The arrhythmia could not be induced by programmed electrical stimulation in the RA or the CS, nor by isoproterenol infusion and withdrawal, given alone or with concomitant pacing. The only mode of induction of the tachycardia was by waking the patient and asking him to speak. While awake, AT started with talking and ceased even between sentences. AT following the respiratory rate could also be demonstrated. Interestingly, the arrhythmia was not uniform and other morphologies of the P wave during tachycardia could be documented. A tridimensional map of the RA was performed using a nonfluoroscopic electro-anatomic mapping system (NavX, St. Jude Medical, St. Paul, Minnesota). The clinical arrhythmia was mapped to the postero-medial aspect of the SVC (figure 2A). Using a radio-frequency (RF) generator (IBI-1500, St Jude Medical), focal RF delivery to that region eliminated the AT and significantly decreased the frequency of ectopic activity. Several weeks later, the patient reported that symptoms reoccurred. ECG during the tachycardia revealed a positive P wave in V1 and a broad notched P wave in L2, favoring an RSPV origin (4) (figure 1B). A second attempt was made to ablate the tachycardia. The patient was kept awake and talking. Frequent short bouts of AT were demonstrated during the whole study, concomitant with speaking or following the respiration rate. As in the first study, AT was not uniform and had several morphologies. The left atrium (LA) was accessed through a previously unknown PFO. The site of earliest activation of the second clinical tachycardia was mapped to the antero-lateral aspect of the RSPV (figure 2B, video 1). Focal RF energy applied to sites with early and fractionated electrograms failed to terminate the tachycardia. Prolongation of the sinus cycle length by 20-30% and frequent sinus pauses were observed during RF delivery. Thereafter, a 28 mm diameter cryo-balloon (Arctic front, Medtronic, Minneapolis, Minnesota) was advanced into the LA through the PFO, using a 15F outer diameter deflectable sheath (FlexCath, Medtronic), over a 20 mm circular mapping catheter (Achieve, Medtronic) placed through its central lumen. Cryo-ablation of the RSPV orifice and antrum was performed as published [5] (Figure 3). The arrhythmia disappeared a few seconds after the temperature measured at the balloon's base

Figure 1: MECG Strips of Short Events of AT



A - The P wave is tall in L2 and isoelectric in V1, suggesting SVC origin. **B** – the P wave is broad in L2 and positive in V1, suggesting RSPV origin

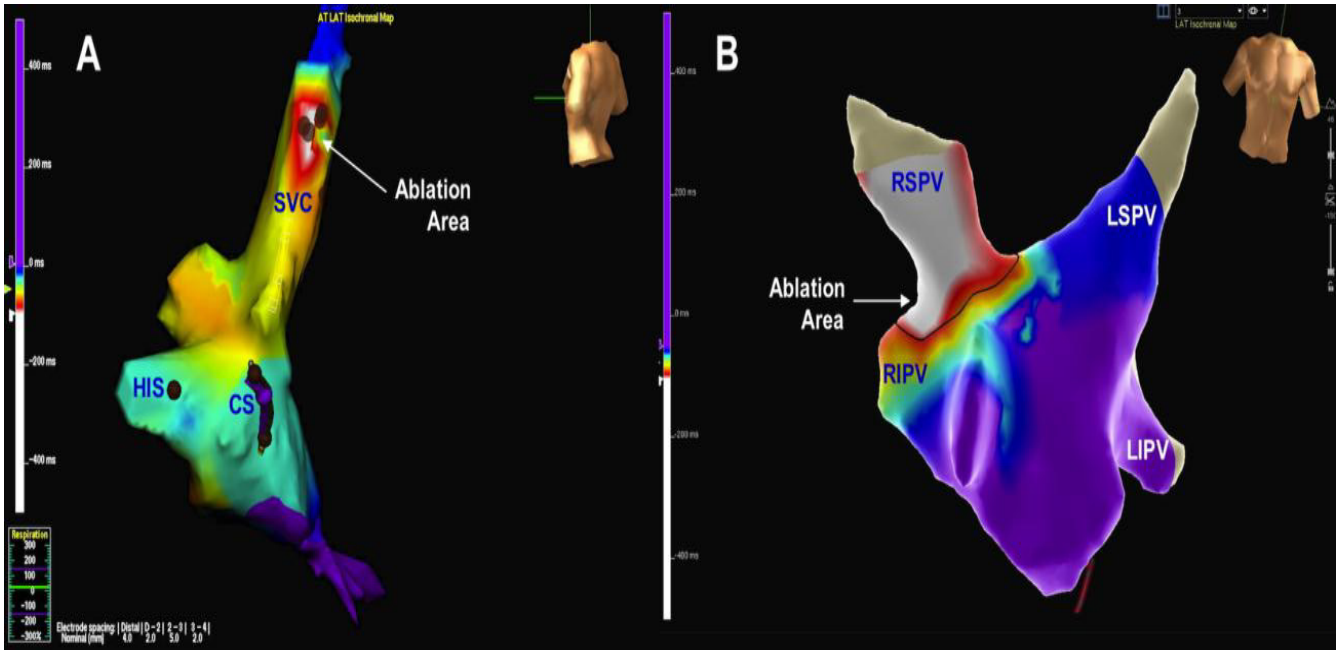
dropped below freezing. The patient tolerated the procedure well, including right phrenic stimulation during cryo-ablation to avoid nerve injury, albeit while awake. During the next 24 hours of continuous monitoring, sinus tachycardia up to 100 beats per minutes was observed. It persisted for a few months, very gradually subsiding. The patient remained arrhythmia free.

Discussion

We present a patient with co-occurrence of speech and respiratory induced AT, implying a common etiology most probably involving the ANS. The origin of arrhythmia was mapped to the SVC and RSPV area, similar to the findings of Yamamoto et al in seven patients with RCAT.[2] The epicardial adipose tissue in this area contains a network of nerves and ganglia comprising most of the ANS traffic to the heart . Using electron micrographs from human preparations, Pausa et al demonstrated nerve fibers in a sulcus between the SVC and the RSPV, which synapse within the closely located ARGP with other nerve fibers apparently innervating the sinus node and other RA tissue .[6] This was the anatomical ablation area in our patient. Biopsies of epicardial fat collected at this location from eight human patients during cardiothoracic surgery were analyzed for different neurotransmitters .[7] It was found that the GP has a complex anatomy, which includes cholinergic and nitrergic phenotype for most of the neurons, noradrenergic markers in some neurons, and other neurochemical inputs. Nevertheless, electrical GP stimulation in animals or humans elicits a strong vagal response.[8]

Near the ARGP location, the heart and the great vessels are attached to the mediastinum by the pericardial reflections, and hence may be susceptible to local stretch, like movements of the diaphragm or the trachea while speaking or breathing. Notably, in our patient, the arrhythmia started literally with the first word and subsided even between sentences. It was initiated at mid-inspiration as well. It is possible that activation of stretch receptors in the great vessels or in the carotid sinus induced a fast hypothalamic reflex within the nucleus of the solitary tract, with efferent parasympathetic discharge through the dorsal medulla to the cardiac ganglia.[9] Local physical irritation of a ganglion cannot be excluded as well. Studies in dogs demonstrate that electrical and cholinergic stimulation of the ARGP cause shortening of the

Figure 2: Three-Dimensional Mapping with NavX of the RA and SVC (A), and the LA and Pulmonary veins (B)



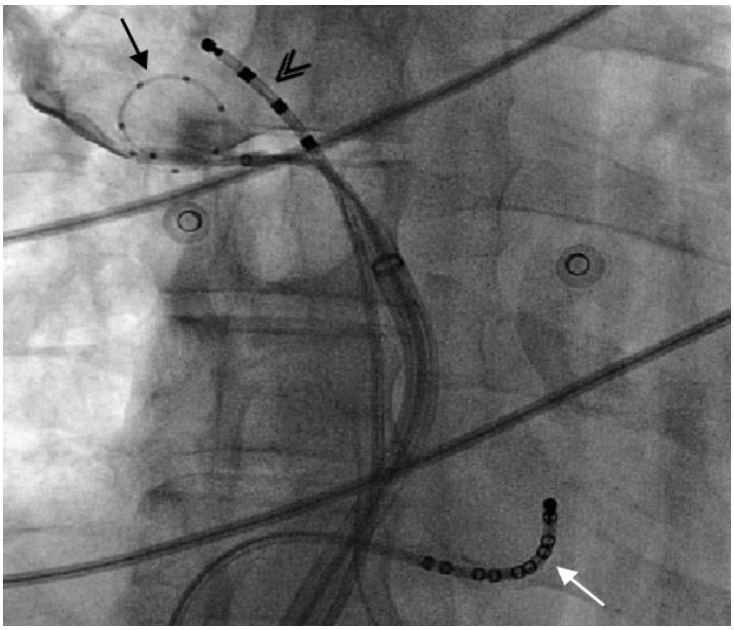
The site of earliest activation, white colored, is postero-medial in the SVC and antero-lateral in the RSPV (see torso, right upper corner). These two regions are across each other, the RSPV lines behind the SVC. CS - coronary sinus, LIPV - left inferior pulmonary vein, LSPV - left superior pulmonary vein, RIPV - right inferior pulmonary vein, RSPV - right superior pulmonary vein, SVC – superior vena cava.

ERP in the SVC and the RSPV myocardial sleeves, resulting in early after-depolarizations, rapid local firing and atrial tachyarrhythmia .[10,11] This appears to be the mechanism of arrhythmia in our patient, who had clinically identical ATs originating from both the SVC and the RSPV. Interestingly, two out of the seven patients with RCAT reported by Yamamoto et al also had two different arrhythmias, initiating from the same locations .[2] It is likely that a common trigger induced both ATs.

The arrhythmia in our patient occurred only during talking or breathing, had different morphologies, did not respond to AAD and could not be induced during EPS by programmed electrical stimulation. All of these suggest triggered activity as the mechanism of the arrhythmia. The lack of response to beta-blockers and the inability to induce AT under isoproterenol infusion exclude a sympathetic etiology. Vagal response was evidenced during RF ablation at the site of AT origin by sinus pauses and increased sinus cycle length. Such a response to electrical stimulation is used to identify ganglion sites during AF ablation.[3,8] Similarly, the ARGP was probably stimulated by the RF energy, being in close proximity to the tip of the catheter and therefore, ablation at this location was expected to involve the ganglion. Sinus tachycardia, which occurred after the ablation and lasted for a few months, is compatible with the expected vagal withdrawal after ablation of the ARGP, which mediates the parasympathetic traffic to the SA node .[6] Similarly, inappropriate sinus tachycardia has been reported in 23.5% of patients who underwent successful GP ablation as an adjunct to AF ablation.[3]

Balloon cryo-ablation has been developed as an alternative to RF ablation for PV isolation in AF patients. To the best of our knowledge, this is the first report of using a cryo-balloon to treat AT near a single PV, or of manipulating this balloon through a PFO. The large contact area with the atrial wall adjacent to the ARGP location probably caused plexus ablation, involving the underlying pre-ganglionic vagal nerves running between the RSPV and the SCV.[6] A reduction in heart rate variability parameters compatible with modulation of the intrinsic cardiac ANS has been reported after cryo-balloon PV isolation for the treatment of AF

Figure 3: Balloon Cryo-Balloon Ablation of the RSPV Ostium



Note the contrast material in the vein. Arrowheads - electrode in SVC for phrenic nerve stimulation. Black arrow - "Achieve" lasso electrode. White arrow - electrode in CS

Conclusions

We present a patient with AT induced by speech and by respiration. The trigger of the arrhythmia appears to be parasympathetic discharge from the ARGP, mediated either by a fast ANS reflex or by another local mechanism. These vagal stimuli in-

duced rapid firing in the SVC and the RSPV myocardial sleeves and initiated ATs in both sites. Balloon cryo-ablation around the orifice of the RSPV, probably involving the ARGP and the pre-ganglionic vagal nerves, abolished the arrhythmia. Thus, targeting the cardiac ANS appears to have an important role in AT/AF ablation. Other mechanical stimuli have been reported as triggers of atrial tachyarrhythmias, including cough, swallowing or esophageal reflux.[1,13] As in our patient, these maneuvers may stretch the ARGP area, induce vagal discharge and initiate AT/AF. This possible etiology should be kept in mind while ablating arrhythmia in these patients.

We showed that balloon cryo-ablation might be used for the treatment of AT originating near the orifice of a single PV if a large contact area with the atrial wall is needed or if focal ablation failed. The feasibility of handling a large deflectable 15F (outer diameter) sheath and a 28 mm diameter Arctic Front cryo-balloon through a PFO is hereby demonstrated, as well as the good tolerability of the procedure in a non-sedated patient.

Disclosures

No disclosures relevant to this article were made by the authors.

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