Acute effects of unilateral temporary stellate ganglion block on human atrial electrophysiological properties and atrial fibrillation inducibility

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BACKGROUND In experimental models, stellate ganglion block (SGB) reduces the induction of atrial fibrillation (AF), while data in humans are limited.

OBJECTIVE The aim of this study was to assess the effect of unilateral SGB on atrial electrophysiological properties and AF induction in patients with paroxysmal AF.

METHODS Thirty-six patients with paroxysmal AF were randomized in a 2:1 order to temporary, transcutaneous, pharmaceutical SGB with lidocaine or placebo before pulmonary vein isolation. Lidocaine was 1:1 randomly infused to the right or left ganglion. Before and after randomization, atrial effective refractory period (ERP) of each atrium, difference between right and left atrial ERP, intra- and interatrial conduction time, AF inducibility, and AF duration were assessed.

RESULTS After SGB, right atrial ERP was prolonged from a median (25th–75th percentile) of 240 (220–268) ms to 260 (240–300) ms ($P < .01$) and left atrial ERP from 235 (220–260) ms to 245 (240–280) ms ($P < .01$). AF was induced by atrial pacing in all 24 patients before SGB, but only in 13 patients (54%) after the intervention ($P < .01$). AF duration was shorter after SGB: 1.5 (0.0–5.8) minutes from 5.5 (3.0–12.0) minutes ($P < .01$). Intra- and interatrial conduction time was not significantly prolonged. No significant differences were observed between right and left SGB. No changes were observed in the placebo group.

CONCLUSION Unilateral temporary SGB prolonged atrial ERP, reduced AF inducibility, and decreased AF duration. An equivalent effect of right and left SGB on both atria was observed. These findings may have a clinical implication in the prevention of drug refractory and postsurgery AF and deserve further clinical investigation.

KEYWORDS Stellate ganglion; Block; Atrium; Electrophysiology; Atrial fibrillation

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Introduction

Paroxysmal atrial tachycardia or atrial fibrillation (AF) in animal models are preceded by increased extrinsic cardiac nerve activity recorded in left stellate ganglion.[1] Additional experimental studies suggest that stimulation of the stellate ganglion increases sinus rate and predisposes to atrial arrhythmias.[2,3] While unilateral electrical stimulation of the stellate ganglion facilitates the induction of AF and aggravates atrial electrical remodeling in canine models, unilateral ganglionectomy reduces AF induction.[4]

In humans, unilateral right stellate ganglion block (SGB) has a combined sympathetic and parasympathetic influence on sinus node, resulting in a decrease in sinus rate.[5,6] After SGB, a decrease in low frequency and low/high frequency ratio of heart rate variability has been observed, suggesting a shift in cardiac autonomic balance toward the predominance of parasympathetic over sympathetic activity.[7] Although left SGB has been proposed as an effective treatment for drug refractory ventricular tachycardia or fibrillation, the effect of this technique on atrial arrhythmias has not been extensively studied.[8,9]

The aim of the present randomized, placebo-controlled study was to assess the acute impact of unilateral SGB on human atrial electrophysiological properties and AF inducibility, as well as to compare the effects of left and right SGB, in patients with paroxysmal AF.

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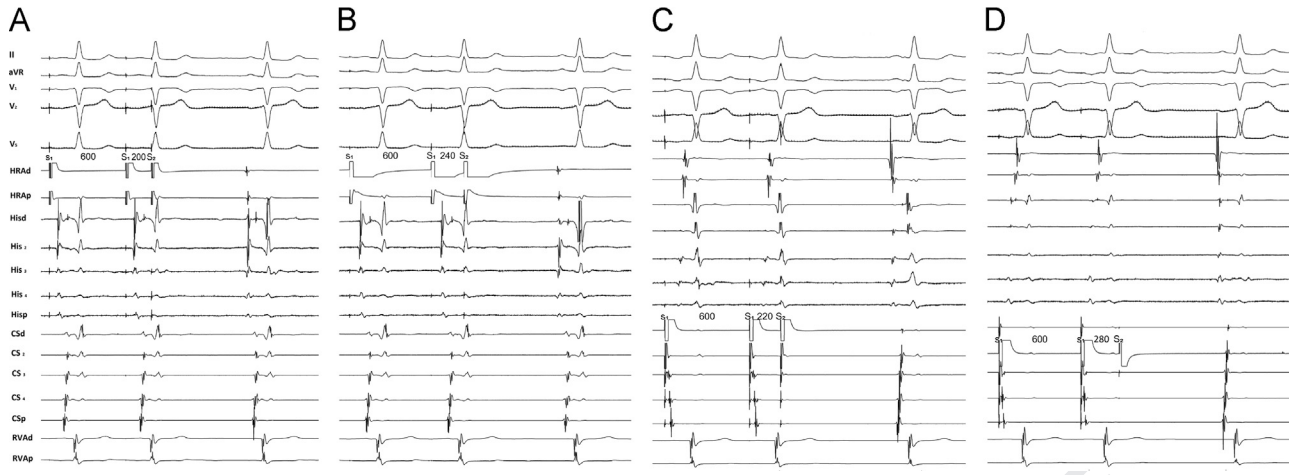

Figure 1 Right and left effective refractory period (RAERP and LAERP) before and after stellate ganglion block (SGB). During pacing at 600-ms cycle length from the high right atrium, RAERP was prolonged from 200 ms before (**A**) to 240 ms after (**B**) block. Similarly, LAERP was 220 ms before (**C**) and 280 ms after (**D**) SGB during pacing from the distal coronary sinus. Electrocardiographic leads II, aVR, V_1, V_2, and V_5, along with electrograms from the high right atrium distal (HRAd) and proximal (HRAp), His area distal to proximal (Hisd, His_2, His_3, His_4, Hisp), coronary sinus distal to proximal (CSd, CS_2, CS_3, CS_4, CSp), and right ventricular apex distal (RVAd) and proximal (RVAp), are presented.

Methods

Patients and randomization

Thirty-six consecutive patients with paroxysmal AF admitted for electrical pulmonary vein isolation (PVI) were included in the study. Antiarrhythmic drug, except amiodarone, was interrupted 1 week before admission. Patients with coronary artery disease, cardiac valve dysfunction, or other systematic disease such as diabetes mellitus, renal or hepatic failure, and neurological diseases were excluded.

Before PVI ablation, all patients were randomized in a 2:1 order to temporary, transcutaneous, pharmaceutical SBG or placebo treatment. In patients randomized to SBG, either the left or the right stellate ganglion was blocked in a 1:1 sequence with paratracheal infusion of 8 mL of 1% lidocaine solution. The efficacy of SBG was confirmed by the transient appearance of Horner sign, tearing, hoarse voice, difficulty in swallowing, or warmth of the ipsilateral upper extremity. In the arm of placebo (controls), 8 mL of sodium chloride 0.9% was paratracheally injected with the same technique.

Electrophysiological parameters

After local anesthesia, quadripolar diagnostic catheters were percutaneously advanced to the high right atrium (HRA) and at the His bundle area so as to record upper lateral and anteroseptal atrial electrograms, along with His electrogram. A decapolar catheter was inserted into the coronary sinus (CS) so that the distal bipole reached the left atrial appendage area and could provide stable left atrial pacing, while the proximal bipole was located at the entrance of CS. Before sedation for the transeptal puncture and ablation procedure, the following parameters were assessed by programmed atrial pacing: (1) right atrial effective refractory period (RAERP) at a paced cycle length of 600 ms, defined as the longest coupling interval that failed to propagate to the atrium; (2) left atrial effective refractory period (LAERP) at the same paced cycle length; (3) difference between RAERP and LAERP (dERP); (4) right intra-atrial conduction time by pacing from the distal bipole of the HRA catheter and measured from the onset of the local atrial electrogram to the onset of the atrial electrogram at the His area; (5) left intra-atrial conduction time by pacing from the proximal bipole of the CS catheter and measured from the onset of the local atrial electrogram at the entrance of CS to the onset of the atrial electrogram at the distal bipole; (6) right to left interatrial conduction time ($IACT_{R-L}$) by pacing from the distal bipole of the HRA catheter and measured from the onset of the local atrial electrogram to the onset of the distal CS electrogram; and (7) left to right interatrial conduction time ($IACT_{L-R}$) by pacing from the distal bipole of the CS catheter and measured from the onset of the local atrial electrogram to the onset of the distal HRA electrogram (Figures 1 and 2). All measurements were performed at a paced cycle length of 600 ms in order to achieve stable atrial capture and avoid inhibition because of intrinsic extrasystoles.

After these measurements, AF induction was attempted by pacing from the distal CS or the HRA in 15 seconds lasting atrial bursts at a fixed cycle length of 200 ms or by decremental pacing starting at 200 ms and decrementing by 10 ms with a 10-second interval before each decrement to the shortest cycle length that resulted in 1:1 atrial capture or until AF was induced (Figure 3). Episodes of AF longer than 10 seconds were included in the analysis, and the duration of arrhythmia was assessed. Episodes lasting more than 20 minutes were terminated by cardioversion.

After SGB, all the aforementioned parameters and AF inducibility were reassessed and compared to initial ones. The physician who performed the measurements was not aware of which group each patient belonged to. Before enrollment, all patients gave their informed consent and the protocol of the study was approved by the hospital ethics committee.