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EHJ BRIEF COMMUNICATION
Heart failure/cardiomyopathy

Splanchnic nerve block for decompensated chronic heart failure: splanchnic-HF

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Introduction

Maladaptive volume redistribution is a potential cause of acute decompensated heart failure (ADHF).[1,2] Elevated sympathetic tone may cause decreased vascular compliance of the main storage compartment of intravascular blood volume (splanchnic compartment) and thus precipitate ADHF.[3,4] The splanchnic nerve has been identified as a potential target for patients with ADHF and preliminary haemodynamic/functional outcomes ($N = 5$) from this study were previously published.[5] Here, the completed Splanchnic-HF trial results are presented including data on haemodynamic changes, functional outcomes, comprehensive mechanistic evaluation of autonomic tone, vascular stiffness and volume shifts, and structural assessment of the heart.

Methods

We performed a prospective, open-label, interventional study of patients hospitalized for ADHF at Duke University Hospital between April 2017 and May 2018. Study flow is presented in *Figure 1A*. Eligible patients had ADHF, New York Heart Association Class III/IV symptoms, and a pulmonary capillary wedge pressure (PCWP) >15 mmHg (>12 mmHg if on inotropes) on baseline right heart catheterization. Patients on oral anticoagulants or $P2Y_{12}$ inhibitors were excluded. The study intervention was a bilateral temporary percutaneous splanchnic nerve block (SNB) with lidocaine.[5]

Results

Thirteen patients were enrolled and 11 underwent the procedure. No procedural or haemodynamic complications were observed for 48 h. The average age was 64 ± 13 years, 8 of 11 patients were male and 6 of 11 were black. Ischaemic disease was present in seven patients. All patients had advanced systolic/diastolic HF with a left ventricular ejection fraction (LVEF) of ≤30% in 10 of 11 patients and an LVEF of 45% in one patient. Six patients were on inotropic agents (milrinone or dobutamine).

Bilateral SNB resulted in temporary reduction of invasive haemodynamics such as mean PCWPs from 30 ± 7 mmHg at baseline to 22 ± 7 mmHg at 30 min, $P < 0.001$ (*Figure 1B*). The cardiac index increased from 2.17 ± 0.74 L/min/m^2 at baseline to 2.59 ± 0.65 L/min/m^2 at 30 min ($P = 0.007$).

Splanchnic nerve block temporarily reduced the cardiac sympathetic tone as measured by heart rate variability parameters at 30 min without significant changes at the end of the 90 min (*Figure 1C*). Similar changes were observed for surrogate markers of the sympathetic tone such as catecholamines. Following SNB, we observed a trend towards a decrease in central vascular pulse wave velocity, an index of vascular stiffness and thoracic fluid content, measured with bioelectrance technology (*Figure 1C*).

There was a decrease in mean left atrial volume index following SNB (76 ± 23 mL vs. 64 ± 12 mL; $P = 0.043$) without changes in left ventricular size or diastolic function. Finally, patients reported an acute improvement in symptoms during the procedure (*Figure 1D*) and had an increase in average 6-min walk distance of 8.7 ± 51.6 m (range -115 to 71 m) from before to after the procedure ($P = 0.606$) and 24.7 ± 31.2 m (range -28 to 71 m) 24 h after the procedure ($P = 0.045$).

Discussion/Conclusion

This first-in-man proof-of-concept study tested a new therapeutic approach to the treatment of ADHF. This study supports the splanchnic nerve as a potential therapeutic target in ADHF.

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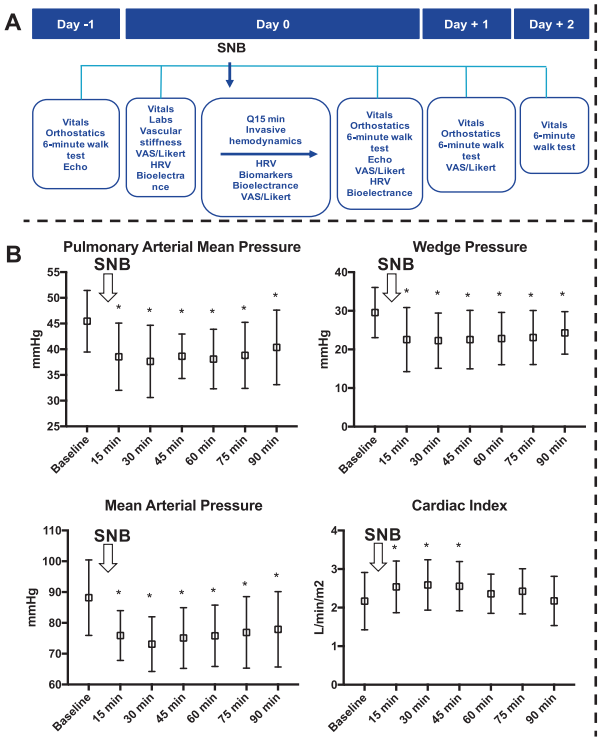

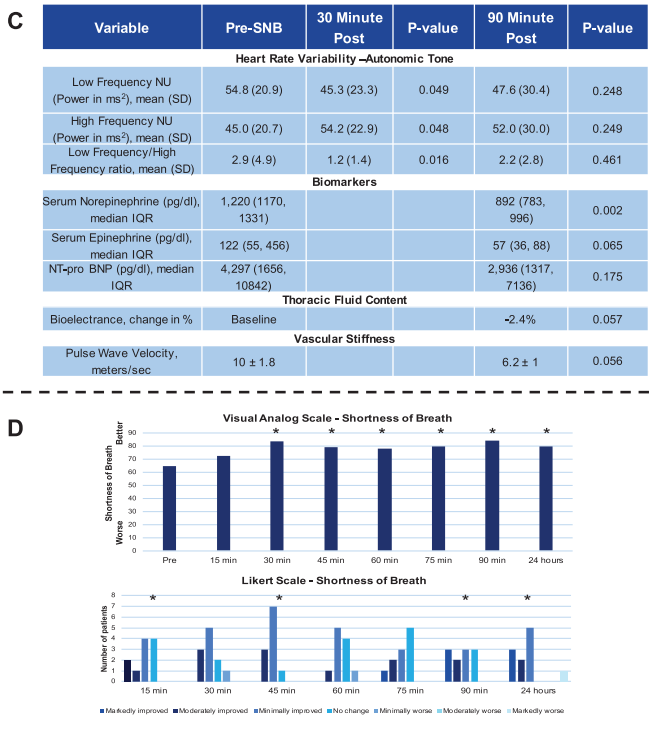

Figure 1 Splanchnic nerve block in heart failure. (*A*) Study flow diagram; (*B*) Effects of the splanchnic nerve block on invasive haemodynamics. **P* < 0.05 for a pairwise comparison with the baseline value. (*C*) Biomarker profile before and after splanchnic nerve block. (*D*) Effect of the splanchnic nerve block on shortness of breath using the Visual Analog Scale and Likert Scale, **P* < 0.05 for a pairwise comparison with the baseline value.

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