

Response by Fujisawa et al to Letter Regarding Article, “Pulmonary Artery Denervation by Determining Targeted Ablation Sites for Treatment of Pulmonary Arterial Hypertension”

In Response:

We appreciate the interest and comments from Drs Kuebler and Friedberg on our article.¹ They pointed out that a reduction in heart rate improved right ventricular failure in an experimental model of pulmonary arterial (PA) hypertension. In addition, they suggested that tachycardia would worsen the ventricular diastolic failure and that the negative chronotropic interventions may be beneficial for ventricular function and vascular remodeling. In our patient,¹ there was no reduction in heart rate (100–105 bpm) shortly after the PA denervation procedure. Heart rate decreased to 90 bpm at 1 and 4 months after the procedure, whereas mean PA pressure decreased from 65 to 56 mmHg. Daily average heart rate was not evaluated, but the patient did not receive drugs with negative chronotropic effects, such as β -blockers or calcium channel blockers. In the study by Zhou et al,² heart rate did not decrease after PA denervation in an animal model. In addition, in a case report from Zhang et al,³ there was no change in heart rate after PA denervation in a patient with pulmonary hypertension secondary to left heart disease, whereas hemodynamics improved. Conversely, in a phase II study,⁴ patients without PA hypertension-related events at the 1-year follow-up after PA denervation had a slower resting heart rate. Thus, changes in heart rate after PA denervation seem to be inconsistent. Future studies on a large number of cases are needed to elucidate heart rate changes after PA denervation. The slower heart rate could be brought about by an autonomic effect of sympathetic nerve denervation or by an improvement in right-sided heart failure. The causality between decreased heart rate and PA denervation remains to be clarified.

Disclosures

None.

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