Dr Jaster referred to the importance of the afferent feedback from the heart via the sympathetic nervous system with respect to the formation of medullary ischemic lesions in the solitary tract nuclei and mentioned the potential development of severe cardiac arrhythmias via the disruption of brain automatic regulations. I read his comments with interest.

However, I would like to point out that the objective of our study was not to prevent life-threatening cardiac events as he mentioned but to confirm the cardiac function-improving effect of adaptive servo-ventilation (ASV) therapy in patients with chronic heart failure (CHF). Although not indicating the superiority of ASV therapy to guideline-directed medical therapy (GDMT) in the cardiac function-improving effect (the primary endpoint), our study showed a clinical status-improvement effect and indicated a given level of clinical benefit in the studied patients with CHF through a significant improvement in the clinical composite response (CCR) as corroborated by improvements in New York Heart Association class and in the post hoc analysis of our study according to the possible combination and backward elimination methods in order to examine the associations of explanatory variables with CCR. Consequently, the apnea-hypopnea index was not included in the best combination of explanatory variables.

Furthermore, our study did not intend to treat sleep-disordered breathing as with the studies mentioned by him, but was conducted based on the previously estimated mechanisms of ASV to improve pulmonary congestion, including a reduction in cardiac preload through reduced venous return and the inhibition of sympathetic nerve overactivity. We conducted the post hoc analysis of our study according to the possible combination and backward elimination methods in order to examine the associations of explanatory variables with CCR. Consequently, the apnea-hypopnea index was not included in the best combination of explanatory variables (Table).

In addition, our understating is that the major cause of sudden death in patients with CHF is not ischemic lesions in the medulla oblongata but fatal ventricular arrhythmias with the underlying pathology of sympathetic overactivity as mentioned in the guideline. We consider that this comprehension is clinically supported by the fact that β-blockers are effective for reducing the risk of death and improving the cardiac function of patients with CHF. ASV, a nonpharmacological modality, has been shown to improve sympathetic overactivity and our study indicated a tendency of suppression of HF deterioration. Therefore, we recognize the potential improvement in prognosis by ASV. Nevertheless, a well-controlled clinical study will be required to verify the potential.

Disclosures
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References

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