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Response to Kataoka *et al.*'s 'How to assess haemodynamic impact of atrial fibrillation'

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Authors' response to the commentary of Kataoka et al., https://doi.org/10.1093/ehjopen/oead123.

Response

We appreciate the opportunity given by the editor to address concerns raised in the letter from Kataoka *et al.* regarding our paper titled 'Haemodynamic changes after atrial fibrillation initiation in patients eligible for catheter ablation: a randomized controlled study.'

In the letter to the editor, Kataoka *et al.* warrant a more extensive examination to enhance the comprehensiveness of our findings.

After conducting a study and analysing data, it is usual to reflect on potential areas for improvement or further thoroughness. This is, of course, applicable in our study, as in many others. In this paper, we cannot do more than described and interpret our results according to an approved study protocol.

The primary concern raised by Kataoka *et al.* concerns the haemodynamic assessment conducted during general anaesthesia and the potential impact on the autonomic nervous system response to haemodynamics. We would like to clarify that, as explicitly stated in our paper,[1] all measurements were executed under mild conscious sedation, with both study groups undergoing identical sedation protocols during the procedure.

Another raised issue is that the average heart rate was increased by 43 beats per minute during atrial fibrillation (AF), with a suggestion that the control group should have been paced to achieve a comparable heart rate elevation. We acknowledge this concern and note that, given our specific focus on AF haemodynamics and the randomized nature of our research design, pacing the control group was not incorporated into the study protocol. This question has previously also been addressed by Lau *et al.*[2]

The authors also rightly point out that intraatrial septal defects can influence haemodynamic effects on biatrial pressures.[3] In our study, all patients underwent transseptal puncture, as outlined in the methodology section. While acknowledging the potential impact of septal defects, we assert that the transeptal sheaths were carefully positioned through the foramen ovale during measurements. The tissue enveloping the sheaths, combined with the randomized setting, leads us to conclude that this does not pose a significant issue.

Lastly, we concede that acute haemodynamic changes may not persist into later phases of AF, as appropriately discussed in the limitations section of our paper.

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