Reply to Koestenberger and Ravekes

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We would like to thank Koestenberger et al. [1] for their comments regarding our manuscript titled ‘Disparity in right vs left ventricular recovery during follow-up after ventricular septal defect correction in children’ [2]. We fully agree with Koestenberger et al. [1] that it would be interesting to gain further knowledge regarding ventricular performance in patients with different subtypes of ventricular septal defects (VSDs). The location of the VSD and implanted patch and its relation to the tricuspid valve ring may certainly influence right ventricular (RV) free wall motion. Both tricuspid annular plane systolic excursion (TAPSE) and RV peak systolic tissue Doppler imaging velocity (S) are assessed at the base of the RV free wall, close to the tricuspid valve ring. Accordingly, both TAPSE and RV S could be different in patients with different subtypes of VSDs.

Hence, we appreciate the suggestion made by Koestenberger et al. [1] to calculate the z scores of TAPSE and RV S, as previously defined [3, 4], to shed further light on the subtype analysis. The calculation of preoperative z scores yielded a mean ± standard deviation of TAPSE of 0.9 ± 2.3 and a mean RV S z score of 0.6 ± 1.5. One day postoperatively, z scores significantly decreased, displaying a mean TAPSE z score of −5.7 ± 1.3 and a mean RV S z score of −4.5 ± 1.2. At medium-term follow-up, z scores were still low, with a mean TAPSE z score of −1.6 ± 1.4 and RV S z score of −1.5 ± 0.9. Similar to previous results, one-way analysis of variance of these z scores did not reveal significant differences between subtypes of VSD patients preoperatively or at medium-term follow-up. Yet, considering the small number of patients and relatively large standard deviations of z scores, a lack of statistical power could still be the cause of this absence.

Accordingly, these additional analyses could not shed further light on the differences between subtypes. However, current z score calculations do further validate the conclusion of our manuscript, of a persistent impairment of RV performance after surgery for a VSD. In a previous study by our group, including patients with a variety of congenital heart defects, a similar decrease in RV performance following surgery was observed [5]. Furthermore, a longer aortic cross-clamp time was associated with a lower RV S in this study. These results emphasize the detrimental role of cardiopulmonary bypass in ventricular deterioration after surgery and highlight the importance of follow-up of RV performance after surgery. Hence, we join Koestenberger et al. [1] in their hope for routine RV function analysis in postoperative VSD patients.

REFERENCES