What is wrong with the right ventricle after surgical closure of a ventricular septal defect?

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The outcomes of patients after surgical closure of a ventricular septal defect (VSD) performed during infancy or childhood is considered to be excellent with low long-term morbidity and mortality.1–3 In most congenital heart centres, patients after surgical VSD closure are discharged from follow-up if there are no known significant residual lesions such as a residual VSD, aortic regurgitation, or subaortic stenosis. This probably explains why there are very few long-term follow-up studies on one of the most common indications for congenital heart surgery.

The study by Heiberg et al.4 looked at a small group of adult patients who underwent surgical closure of a VSD during infancy or childhood. The authors studied left and right ventricular structure and function using cardiac magnetic resonance imaging (cMRI) in 27 young adult patients (mean age 20.5 years) who underwent surgical VSD closure around 1.9 years of age. All patients were clinically asymptomatic. The authors observed that the RVs in the patients were slightly larger and more hypertrophied (higher RV mass index) when compared with normal controls. They also observed a slightly lower fractional area change suggesting a lower RV ejection fraction. Using feature tracking cMRI analysis, they found a small increase in radial RV strain with normal longitudinal RV strain. The left ventricular dimensions and function were within normal range. These data suggest incomplete right ventricular remodelling in patients after VSD closure with residual RV hypertrophy and dilation associated with increased radial deformation. The clinical significance and long-term implications of these findings are currently unknown, but the residual changes are interesting and require further investigation. The paper can be criticized for the methodology used to analyse RV size and function. The authors did not quantify RV volumes and ejection fraction, arguing that the more prominent RV trabeculations in the VSD group precluded a reliable RV endocardial tracing. This is a missed opportunity as MRI is generally considered to be the best technique for assessing RV volumes and other groups have reported reliable results in the presence of RV hypertrophy and dilation.5,6 Interestingly the authors decided to measure RV mass index, which theoretically would have the same technical problems as RV volumetric calculations. They also missed the opportunity to obtain detailed echocardiographic data, which could have provided further important information on RV structure and function.

Despite these methodological limitations, the findings are interesting and somewhat surprising.

The residual RV changes are difficult to explain, as you would expect RV dimensions and function to normalize after VSD closure. None of the patients included in the study had evidence of a residual VSD or of increased RV pressure at rest. A possible explanation is provided by data obtained in patients after VSD closure during exercise. Möller et al. found that a significant number of patients after atrial septal defect (ASD) or VSD closure with normal RV systolic pressure at rest had an abnormal increase in RV systolic pressure during exercise when compared with normal controls.7 This can be explained by an abnormal response in pulmonary vascular resistance to exercise. While some of the patients included in the study by Heiberg et al. were operated relatively late in childhood, none of them had evidence of pulmonary hypertension at rest. In adult patients late after surgical closure of atrial septal defects (ASD), Van de Bruaene et al. also found residual RV dilatation and decreased RV longitudinal deformation in the apical segment of the RV free wall.8 This was associated with decreased exercise capacity and an abnormal response in pulmonary vascular resistance to exercise.9 All these data seem to suggest that the residual changes in RV size and function could be related to residual changes in the pulmonary vascular bed. While these changes are not obvious at rest, an abnormal response in pulmonary vascular resistance to exercise could cause intermittent RV pressure loading and result in incomplete RV remodelling. In VSD patients RV hypertrophy is common prior to surgical closure in patients with large defects and elevated RV pressures. After VSD closure residual concentric hypertrophy may be triggered by an increase in pulmonary artery pressures during exercise. Patients with an ASD are exposed to chronic RV volume loading resulting in eccentric RV remodelling. After ASD closure generally the RV size normalizes. Residual RV dilatation could also be related to increased pulmonary vascular resistance during exercise. The RV hypertrophy described by Heiberg et al. was mild and was not associated with significant functional RV abnormalities. The increased radial strain pattern with preserved longitudinal strain could be a physiological adaptation to chronic pressure

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loading\textsuperscript{10} and does not indicate a pathological remodelling. It would be interesting to further study RV diastolic properties, as RV hypertrophy can be associated with increased RV stiffness or decreased compliance. It is reassuring that the right atrial dimensions observed in the VSD patients were within normal range.

Overall the data obtained by Heiberg et al. are reassuring and confirm that patients after VSD closure do well with some minor changes in RV dimensions and wall thickness that will need further investigation but probably will not affect their long-term outcomes. Nevertheless further long-term follow-up data are needed. The suggested changes in the pulmonary vascular bed long-term after ASD and VSD closure require further investigation and may influence decision-making during childhood on when to close an atrial or ventricular defect.

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References