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Atrioventricular coupling before and after pulmonary valve replacement in patients with tetralogy of fallot.

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Topic(s):
Cardiac Magnetic Resonance: Systolic and Diastolic Function

Citation:
European Heart Journal - Cardiovascular Imaging (2019) 20 (Supplement 2), ii206

Funding Acknowledgements:
Swedish Heart–Lung foundation, Lund University, Skåne University Hospital, Region Skåne and Swedish Research Council

Background
The timing and indications of pulmonary valve replacement (PVR) in patients with repaired Tetralogy of Fallot (rToF) and pulmonary regurgitation (PR) is debated and new functional measures are needed to guide therapy and indicate treatment success.

Purpose
To understand the changes in pumping after PVR in patients with rToF and PR. Therefore, we aimed to investigate how the right ventricular (RV) longitudinal function correlates with atrial filling and if it differs between patients with rToF and PR and healthy controls and if changes occur after PVR.

Methods
Ten patients with rToF and PR>35% were prospectively examined with CMR before and 1 year after PVR. Fifteen healthy controls were used as controls for comparison. Segment software (http://segment.heiberg.se) was used for the analysis. Short axis cine bSSFP images were used for atrial and ventricular volumes. Stroke volume (SV) was defined as end-diastolic volume minus end-systolic volume. 3- and 4-chamber cine bSSFP images were used to semi-automatically quantify RV atrioventricular plane displacement (AVPD). Tricuspid plane displacement (TPD) contribution to SV was calculated as the RVAVPD multiplied with the end-systolic area of the right atrium above the AV-plane. 2D and 4D flows were used to quantify the blood flow in the caval veins.

Results
Venous return through the caval veins occurred to a lower extent during systole in patients, 44±18% of the total SV, compared to controls, 63±9%, p=0.0015, and was not affected by PVR, 41±19%, p=0.95. The caval blood flow during systole correlated well with the atrial volume change during systole for patients before PVR, r=0.83; p=0.0049 and after PVR, r=0.93; p=0.0007 as well as for controls, r=0.94; p<0.0001. Also, there was a good correlation between the right atrial volume change during systole and the TPD contribution to SV in patients before PVR and in controls, but no correlation was found in patients after PVR, Figure 1. TPD contribution to SV, measured as fraction of SV, was lower in patients with rToF and did not change after PVR, Figure 1.

Conclusion
Patients with rToF and PR had reduced atrial reservoir function and elevated conduit function compared to controls both before and after PVR. The atrioventricular coupling was impaired after PVR. RV longitudinal contribution to SV was lower in patients before PVR and did not normalize within a year after PVR. Further studies are needed to investigate the reason for the impaired atrioventricular coupling after PVR and to see if
longitudinal function improves at long time follow up.