Ventricular interaction provokes diastolic dysfunction of left ventricle in patients with tetralogy of Fallot after repair

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Background.
Intensive overload to right ventricle (RV) metamorphoses geometry of left ventricle (LV), which induces LV dysfunction (ventricular interaction). Patients with tetralogy of Fallot (TOF) after repair often possess intensive overloads on right heart. We sometimes encounter diastolic dysfunction of LV (DDLV) in repaired-TOF patients. We predicted DDLV would be provoked by RV overloads in repaired TOF patients.

Methods.
The medical records of 117 repaired-TOF patients aged from 1.3 to 53.0 years were reviewed. We performed cardiac catheterizations to grasp hemodynamic status between 2010 and 2015. We defined DDLV (n=38) as end-diastolic pressure of LV (LVEDP) 13 mmHg or over. First, indexes were determined which would affect DDSV. Second, we compared clinical features between repaired-TOF patients with and without DDLV.

Results.
After multivariate analysis DDLV was independently associated with odds ratio of 35.7 (p<0.001) for RVEDP (≥ 14 mmHg), 3.6 (p=0.038) for ejection fraction of RV (≤ 52%), and 3.1 (p=0.044) for end-diastolic volume of RV (≥ 163 ml/m2). In monovariate analysis DDLV was additionally related to end-systolic volume of RV (≥ 79 ml/m2). Contrary to expectance, elevated end-systolic pressure of RV had no association with DDLV. Explanatory coefficient was high (0.49). In DDLV group, patients were operated at higher age (≥ 3.0 years; 57% vs. 34%, p=0.014), and advanced in age (≥ 17.0 years; 63% vs. 25%, p<0.0001). They had lower cardiac output (< 2.6 L/min/m2; 57% vs. 16%, p<0.0001), and low ejection of LV (≤ 50%; 28% vs 7%, p=0.0052). The rate of patients with DDLV was only 16%, if patients had risk factors 0 or 1; it was 53%, if patients had risk factors 2 or more. Clinically, repaired-TOF patients with DDLV had some form of symptoms more than those without DDLV (39% vs. 6%, p<0.0001).

Discussion.
Our study showed DDLV in repaired-TOF patients was independently associated with abnormal RV function: elevated EDP, decreased ejection fraction, and increased end-diastolic volume. Overloads of RV in repaired-TOF patients would be related to DDLV through ventricular interaction. Patients with DDLV fell into lower cardiac output. We should attention DDLV in repaired-TOF patients with intensive RV overloads.