Systolic dysfunction after Fontan is provoked by pulmonary high flow before procedure

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Background.
For causes of systolic dysfunction of systemic ventricle (SDSV) innate factors were reported in Fontan patients, such as right ventricle and regurgitation of atrio-ventricular valve. However, we predicted pulmonary high flow before Fontan was much related to SDSV in Fontan patients. We investigated what factors were connected with SDSV in Fontan patients.

Methods.
The medical records of 162 Fontan patients were reviewed aged from 1.7 to 42.9 years. They underwent cardiac catheterization between 2010 and 2015. We defined SDSV as ejection fraction of SV 0.40 or below (n=21). Clinical features were determined which would affect SDSV.

Results.
In monovariate analysis SDSV was associated with following 7 factors: study ages (> 6.0 yrs), Fontan ages (> 2.9 yrs), ventricular type (single right ventricle), 1st strategy (native pulmonary stenosis before Glenn), catheter intervention (No history of coil embolization), pulmonary artery index ($\geq$ 350 mm/m$^2$), and Fontan procedure (Non-fenestrated Fontan). After multivariate analysis SDSV was independently associated with odds ratio of 11.7 for larger pulmonary-artery index, 6.1 for single right ventricle, and 5.5 for elder ages at study. Explanatory coefficient was 0.42. Contrary to our expectation, the frequencies of regurgitation of atrio-ventricular valve or regurgitation of aortic valve, long sustentation of which would lead to SDSV finally, were not different between Fontan patients with SDSV and without SDSV. Afterloads, much increasing of which would cause SDSV particularly in Fontan circulation, were not different between two groups, such as systolic pressure of major ventricle and systolic pressure of aorta. The rate of isomerism heart was not different between two groups. Clinically, Fontan patients with SDSV had low cardiac output. Unexpectedly, indexes for liver function on blood test were inferior in patients with SDSV, such as gamma-glutamyl transpeptidase and total bilirubin.

Conclusion.
In Fontan patients SDSV was related to strategies before Fontan that suggested pulmonary high flow. Pulmonary high flow augments loads to ventricle. We might not raise pulmonary artery too large before Fontan in consideration of loading to ventricle. Additionally, liver congestion subsisted in SDSV patients. We should pay much attention to venous system failure in Fontan patients with SDSV.