Effects of ventricular assist device with a fenestration in the failing Fontan circulation: a theoretical analysis using a lumped parameter model

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Background: Fenestration is surgically created at the time of Fontan operation, in which the extracardiac conduit is connected to the atrium through a small communication. Since the fenestration has been reported to increase venous return to the ventricle and improve the hemodynamics, the fenestration is often made when the risk of postoperative Fontan failure is high. In the failing Fontan circulation, the implantation of ventricular assist device (VAD) is sometimes required as a bridge-to-transplantation therapy. However, it has been controversial whether a closure of fenestration is necessary or not.

Purpose: To evaluate the effects of fenestration on the hemodynamics under VAD in the failing Fontan circulation.

Methods: A computational model of Fontan circulation was developed using a time-varying elastance cardiac chamber model and a modified 3-element Windkessel vasculature model. Pressure gradient through fenestration was calculated by the simplified Bernoulli’s equation. The relationship between pressure head and rotational frequency of VAD was modeled as a non-linear function. To simulate Fontan failure, ventricular end-systolic elastance was set at 1.0 mmHg/ml and then pulmonary vascular resistance index (PVRI) was varied from 3.0 to 6.8 WU m².

Results: In the Fontan failure model with PVRI of 3.0 WU m², mean blood pressure (MBP), cardiac index (CI) and central venous pressure (CVP) were 58.4 mmHg, 1.7 l/min/m² and 19.2 mmHg, respectively. When MBP was maintained at the same value, the 4-mm fenestration rarely affected CI (1.7 l/min/m²) and CVP (18.1 mmHg), but significantly decreased oxygen saturation (SpO₂, 84.0 %). The introduction of VAD at 4000 rpm significantly increased MBP and CI in both models with/without fenestration (Fenestration (+): 93.7 mmHg, 3.2 l/min/m², Fenestration (-): 94.5 mmHg, 3.2 l/min/m²). In the model with fenestration, SpO₂ recovered to 91.6 % after the VAD initiation. However, CVP slightly changed after the VAD initiation in both models with/without fenestration (18.5 and 18.9 mmHg, respectively). When stressed blood volume (SBV) was decreased until end-systolic ventricular volume became around 0 ml, CVP was decreased to the normal range (Fenestration (+): 8.3 mmHg, Fenestration (-): 10.0 mmHg). Then SBV was significantly lower in the model with fenestration than that without fenestration (-150 ml). The effects of fenestration on CVP and SBV were larger in the model with high PVRI of 6.8 WU m² (CVP: -5.5 mmHg, SBV: -460 ml) than those with PVRI of 3.0 WU m² (CVP: -1.7 mmHg, SBV: -150 ml).

Conclusions: Although fenestration decreases SpO₂ at the onset of Fontan failure, fenestration can significantly decrease CVP and SBV under VAD support. These effects are more significant in the patients with higher PVRI. This study demonstrates that fenestration did not impair the hemodynamics under VAD support, indicating the closure of fenestration may not be necessary.