Tohoku J. Exp. Med., 1993, **169**, 113-119

Acute Hemodynamic Changes after Fontan Operation: An Experimental Study

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HANEDA, K., KONNAI, T., SATO, N., NICOLOFF, N.N. & MOHRI, H. *Acute Hemodynamic Changes after Fontan Operation : An Experimental Study.* Tohoku J. Exp. Med., 1993, **169** (2), 113-119 —— Seven adult mongrel dogs with an average body weight of 11.3 kg were subjected to experimental Fontan operation in order to evaluate acute hemodynamic changes at the Fontan circulation. The Fontan circulation was established by occlusion of the tricuspid valve with tightening a purse-string suture which was placed around the valve under inflow occlusion and was passed through the anterior right atrial wall, and by connection with a 10 mm Gore-Tex graft between right atrium and pulmonary artery. When the Fontan circulation was established, aortic pressure and cardiac output (CO) decreased from 94.9±22.0 mmHg to 41.9±6.0 mmHg and 0.907±0.179 liter/min to 0.259±0.072 liter/min, respectively. There were no significant changes in heart rate and systemic vascular resistance between normal and the Fontan circulations. On the other hand, pulmonary vascular resistance (PVR) increased from 11.6±7.3 units in the normal circulation to 21.8±13.0 units in the Fontan circulation. From this study it was concluded that main changes in hemodynamic parameters immediately after establishing the Fontan circulation were marked decrease in systemic arterial pressure and CO, and an increase in PVR. Therefore, one should consider a possibility of an increase in the PVR to the critical level when the Fontan operation was applied to patients with pulmonary hypertension. ——
Fontan operation; tricuspid atresia; pulmonary hypertension

Fontan operation has been widely performed in many complex cardiac anomalies including tricuspid atresia, univentricular heart or hypoplasia of the right ventricle since the first report in 1971 (Fontan and Baudet). Although surgical indication for the Fontan operation had been tightly set by Fontan's Ten Commandment (Choussat et al. 1978) initially, recently the procedure has been challenged successfully in patients even with pulmonary hypertension greater than 20 mmHg of mean pulmonary artery pressure or with pulmonary vascular

Received July 6, 1992; revision accepted for publication January 16, 1993.

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Supported by Grant-in-Aid for General Science Research (C) 03670649 from the Japanese Ministry of Education, Science and Culture.

resistance greater than 4 units • m^2. Postoperative hemodynamic status after the Fontan operation in such patients, however, are often critical and the mortality rates are high. In order to develop adequate postoperative management methods, it is necessary to understand the precise hemodynamic conditions following abrupt flow changes created by the Fontan circulation in which right ventricular kick is absent. This study was conducted to evaluate acute hemodynamic changes and its effects on cardiac functions when the pulmonary blood flow is altered from normal circulation to the Fontan circulation in a canine experimental model.

Materials and Methods

Seven adult mongrel dogs of both sexes were used in this experiment. Body weight ranged from 7.8 to 14.8 kg with an average weight of 11.3 kg. Anesthesia was induced with thiopenthal sodium in a dose of 25 mg/kg and maintained with 0.5% of halothane in oxygen. Animals were ventilated with a volume control ventilator (Model SN-480-3; Shinano, Tokyo) at a tidal volume of 20 ml/kg and a rate of 25 breaths/min. The heart was approached through the right fourth intercostal thoracotomy extended to the left thorax with a sternal transection. The pericardium was left widely open during the experimental procedure but it was suspended to the thorax cage in order to maintain the heart position. After systemic heparinization, a purse-string suture with a 4-0 polypropylene was placed around the tricuspid valve through a small longitudinal right atrial incision under normothermic inflow (superior and inferior venae cavae) occlusion. The suture was passed through the anterior right atrial wall and applied to a tourniquet to be tied later for complete obstruction of the tricuspid valve orifice (Fig. 1). This procedure required 1-2 min without significant blood loss and severe hemodynamic derangement. Subsequently, the anterior wall of pulmonary artery (PA) trunk was connected to the right atrial appendage with a 10 mm of ringed graft (expanded polytetrafluoroethylene, Gore-Tex) (Fig. 2). The graft was left cross clamped until the tricuspid valve was closed by tightening the tourniquet.

Physiologic parameters including electrocardiograms, and pressures in the aorta, PA, right atrium (RA) and left atrium (LA) were monitored on a Polygraph recording system (142-8; San-Ei, Tokyo). Cardiac output (CO) was measured with a Nihon Koden electro-

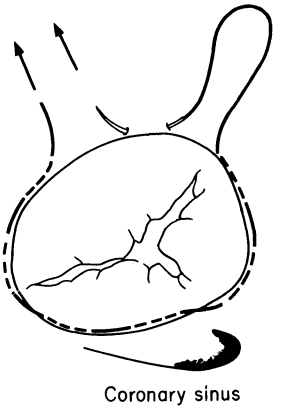

Fig. 1. Experimental procedure. A purse-string suture was placed around the tricuspid valve. The suture was passed through the anterior right atrial wall and applied to a tourniquet.

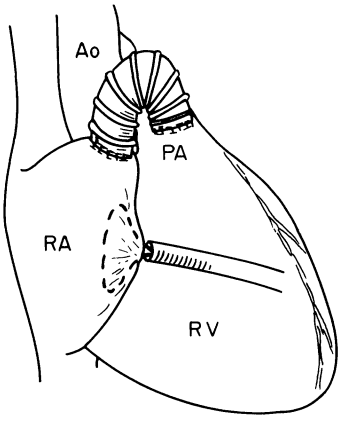

Fig. 2. The Fontan operation model. A conduit is placed between the right atrial appendage and the pulmonary artery trunk. The Fontan circulation was established by tightening the tourniquet around the tricuspid valve. Ao, aorta; PA, pulmonary artery; RA, right atrium; RV, right ventricle.

magnetic blood flowmeter and an electromagnetic flow probe of 12-14 mm in diameter which was placed around the ascending aorta.

Systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR) were calculated as follows:

SVR (units) = (MAP − mean RAP)/CO (liter/min)
where MAP = mean arterial pressure (mmHg), RAP = right atrial pressure

PVR (units) = (mean PAP − mean LAP)/CO (liter/min)
where PAP = pulmonary artery pressure, LAP = left atrial pressure

The Fontan circulation was established by tightening the suture string tourniquet placed around the tricuspid valve and by declamping the graft between PA and RA. Hemodynamic data were collected before (Control) and immediately after establishing the Fontan circulation without fluid augmentation and without administration of any cardiotonic drugs such as catecholamines and calcium chloride. The reproducibility of the hemodynamic changes was tested in 4 animals by re-establishing the normal and the Fontan circulation. At the end of experiment, complete occlusion of the tricuspid valve was confirmed through the RA.

All hemodynamic data were expressed as mean ± S.D. Results were compared using Student paired or nonpaired t test, and differences between before and after the Fontan circulation were considered to be significant if $p < 0.05$.

Results

All animals tolerated the procedure well. Hemodynamic data were presented in Figs. 3, 4 and 5.

Heart rate. Sinus rhythm was maintained throughout the procedure in all animals. Heart rates did not change significantly by creating the Fontan circulation (160.7 ± 31.0 vs. 131.4 ± 46.3, respectively).

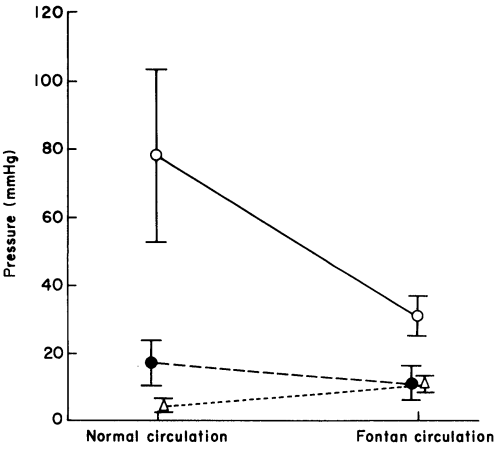

Fig. 3. Acute changes in mean arterial pressure (○, $p<0.01$), mean pulmonary artery pressure (●, $p<0.05$) and mean right atrial pressure (△, $p<0.01$) between the normal circulation and the Fontan circulation. mean±S.D.

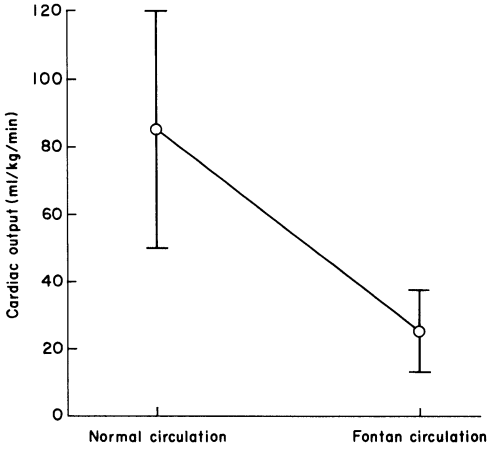

Fig. 4. Acute changes in cardiac output between the normal circulation and the Fontan circulation. $p<0.01$. mean±S.D.

Aortic pressure. Systolic pressure changed significantly ($p<0.01$) from 94.9±22.0 mmHg in the Control to 41.9±6.0 mmHg in the Fontan circulation (44.2% of control). MAP in the Control was 76.9±25.1 mmHg, whereas it dropped to 29.9±6.2 mmHg in the Fontan circulation ($p<0.01$).

PA pressure. Mean PA pressure decreased significantly from 16.9±6.2 mmHg in the Control to 11.1±5.0 mmHg in the Fontan circulation ($p<0.05$).

RA pressure. Mean RA pressure in the Control was 4.1±0.9 mmHg. When the Fontan circulation was established, it increased significantly to 10.9±2.3 mmHg ($p<0.01$) with a prominent "a" wave and reached equivalent level to the

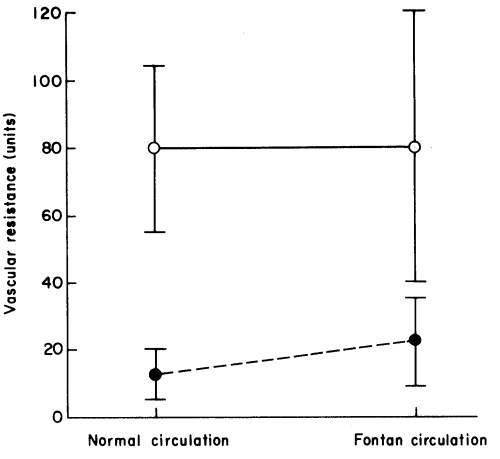

Fig. 5. Acute changes in systemic vascular resistance (○, n.s.) and pulmonary vascular resistance (●, $p<0.05$) between the normal circulation and the Fontan circulation. mean±S.D.

mean PA pressure.

LA pressure. Mean LA pressure decreased from 6.1±1.9 mmHg in the Control to 3.9±2.2 mmHg in the Fontan circulation ($p<0.01$).

CO. CO dropped to 0.259±0.072 liter/min (24.6±10.2 ml/kg/min) in the Fontan circulation from 0.907±0.179 liter/min (86.2±34.0 ml/kg/min) in the Control ($p<0.01$).

SVR. There were no significant changes in the SVR between the Control and the Fontan circulation (80.1±23.9 units vs. 81.0±40.0 units, respectively).

TABLE 1. *Acute hemodynamic changes at repeated creation of the Fontan circulation*

	1st		2nd	
	Control 1	Fontan 1	Control 2	Fontan 2
Heart rate	162.5±33.8	144.8±34.3	157.3±28.4	143.0±28.1
MAP (mmHg)	82.5±9.6	30.5±5.2	68.5±8.2	27.0±6.0*
Mean PAP (mmHg)	15.1±7.1	10.7±5.4	13.8±5.3	7.4±2.0
Mean RAP (mmHg)	4.3±1.0	11.3±3.0	4.0±2.9	8.8±1.7
Mean LAP (mmHg)	5.9±2.3	4.0±2.7	3.4±2.2*	2.8±1.7
CO (liter/min)	0.908±0.161	0.283±0.054	0.775±0.117	0.288±0.094
SVR (units)	86.9±7.6	69.8±17.2	84.1±16.1	68.9±17.9
PVR (units)	11.1±8.6	23.7±16.2	13.3±6.2	17.7±9.9

MAP, mean aortic pressure; PAP, pulmonary artery pressure; RAP, right atrial pressure; LAP, left atrial pressure; CO, cardiac output; SVR, systemic vascular resistance; PVR, pulmonary vascular resistance.

*$p<0.05$ between 1st and 2nd data.

PVR. The Fontan circulation demonstrated an increase in the PVR to 1.9 times of the Control (11.6 ± 7.3 units in the Control vs. 21.8 ± 13.0 units in the Fontan circulation, $p < 0.05$).

Reproducibility of the hemodynamic changes. Hemodynamic changes before and after the Fontan circulation between the first and the second trials were shown in Table 1. In comparison of the hemodynamic parameters between 2 trials (Control 1 vs. Control 2 and Fontan 1 vs. Fontan 2), there were no significant differences in hemodynamic parameters except for a few points.

Discussion

Experimental Fontan models in dogs have been reported by several investigators (Haller et al. 1966; Shemin et al. 1979; Matsuda et al. 1981). In these experimental models, the Fontan circulation was established using cardiopulmonary bypass by total occlusion of the tricuspid valve and by RA-PA conduit or by side-to-side anastomosis between superior vena cava and PA. Although the experimental method used in this study was similar to others reported in terms of creation of the total occlusion of the tricuspid valve and the use of a RA-PA conduit, the operative procedures utilized were different from those in literatures. In our experimental model, the simple caval occlusion method was utilized instead of cardiopulmonary bypass to place purse-string suture around the tricuspid valve and the tricuspid valve was occluded from the outside of RA with a tourniquet when the hemodynamic condition was stable. This operation technique has several advantages over other methods such as 1) minimum untoward hemodynamic effects on the experimental animals since cardiopulmonary bypass was not utilized and 2) furnishing the ease of turning on-off the Fontan circulation.

Precise investigations regarding hemodynamic changes before and after the Fontan operation have not been reported yet. In our experimental study, hemodynamic changes immediately after establishing the Fontan circulation were drastic and reproducible. Main changes in hemodynamic parameters were marked decrease of the systemic arterial pressure and CO, and an increase in PVR while SVR remained unchanged. Without contractive force of the right ventricle, systemic arterial pressure and CO decreased to 30-40% of control values and PVR increased almost twice as high as in the normal circulation. Several experimental studies regarding pumping capability of the RA in the Fontan circulation could not demonstrate the actual pumping function from the RA into the pulmonary circulation regardless of the coexistence of right atrial hypertrophy (Murphy et al. 1978; Shemin et al. 1979; Matsuda et al. 1981; de Leval et al. 1988). Although some pulse-pressure amplitude exists in the PA pressure in this experimental model, it was negligible level and can be said as non-pulsatile pressure. Shepard et al. (1966) and Mavroudis (1978) documented the advantages in pulsatile flow compared to non-pulsatile flow with respect to peripheral circulation

stating that pulsatile flow had high energy 2.3 times as much as in non-pulsatile flow at the same mean arterial pressure, and this extraenergy in pulsatile flow was thought to maintain the peripheral perfusion by keeping capillary beds open and aiding in lymph flow. Because SVR does not change significantly despite of decreased systemic arterial pressure and CO, it is suspected that less energy in the non-pulsatile PA flow decreases the perfusion area in the intrapulmonary capillary beds and results in increase in the PVR.

In the clinical situations, fluid augmentation to maintain high RA pressure and/or pharmacological cardiac support is necessary in order to sustain an adequate hemodynamic condition immediately after the Fontan operation. Because the PVR significantly increases immediately after the Fontan operation, the PVR in patients with pulmonary hypertension is suspected to increase to the critical level after the Fontan operation. Therefore, it might be necessary to apply some mechanical cardiac support systems in addition to fluid augmentation and pharmacological support in order to overcome a high PVR for extension of the indication for the Fontan operation.

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