

## Post-exercise heart rate, blood pressure and oxygen uptake dynamics in pediatric patients with Fontan circulation Comparison with patients after right ventricular outflow tract reconstruction

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### Abstract

**Background:** Post-exercise heart rate (HR) and oxygen uptake ( $\dot{V}O_2$ ) recover more slowly in patients with the Fontan circulation, but little is known about the determinants of the delayed recovery.

**Purpose:** To evaluate the post-exercise cardiovascular dynamics and clinical profiles in these patients.

**Methods and results:** We studied 51 Fontan patients ( $14 \pm 4$  years) (atriopulmonary connection, APC=18 and total cavopulmonary connection, TCPC=33) and compared the results with 34 patients after right ventricular outflow tract reconstruction (RVOTR) with identical exercise capacity and arterial baroreflex sensitivity (BRS) ( $15 \pm 4$  years) and with 26 controls ( $14 \pm 4$  years). There were no differences in post-exercise HR or  $\dot{V}O_2$  declines between the Fontan and RVOTR groups. Although the systolic blood pressure (SBP) decline was delayed in the RVOTR group ( $p < 0.01$ ), its early decline in the Fontan group was rapid and equivalent to that in controls. In Fontan patients, BRS had a great impact on early HR decline ( $p < 0.05$ ) and early  $\dot{V}O_2$  decline was determined by peak  $\dot{V}O_2$ , age and cardiac index ( $p < 0.05-0.001$ ). TCPC and lower BRS were the main determinants of the slower SBP decline ( $p < 0.05$ ). In another study of repeated paired exercise tests before and after Fontan operation, post-exercise SBP decline became greater after the operation ( $p < 0.07$ ).

**Conclusions:** In the Fontan group, post-exercise HR and  $\dot{V}O_2$  declines are markedly delayed and are determined by cardiac vagal nervous activity, exercise capacity and age, respectively. Despite identical impaired hemodynamics and exercise capacity, post-exercise SBP decline is greater in the Fontan group, especially after APC, than in the RVOTR patients.

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**Keywords:** Autonomic nervous activity; Hemodynamics; Fontan operation; Exercise; Blood pressure

Cardiovascular recovery after exercise has been emphasized because indices derived from the post-exercise recovery period provide objective and useful information, not only to predict future cardiac events, but also to stratify functional cardiac status in both normal subjects and

patients with chronic heart failure [1–5]. Systolic blood pressure (SBP) recovery is also useful in evaluating adult patients with ischemic heart disease [6]. These indices are easily measured and, therefore, clinically useful. The number of adult congenital heart disease (CHD) patients is increasing and their long-term care after definitive repair will be focused on preventing late adverse cardiac events, making stratification of their post-operative functional cardiac status using relatively simple measurements clinically important. We demonstrated that cardiovascular

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recovery in patients after right ventricular outflow tract reconstruction (RVOTR) resembles that in both healthy individuals and adult patients with heart failure [7]. This similarity comes from the feasible stratification of biventricular CHD patients based on cardiac autonomic nervous activity (CANA) and neurohormonal factors [8]. However, these indices, especially CANA, are not related to functional status in patients after the Fontan operation [9]. Although delayed heart rate (HR) and oxygen uptake ( $\dot{V}O_2$ ) recovery were reported [10], their determinants remain unclear in patients with the Fontan circulation. Moreover, because there has been no study addressing the post-exercise blood pressure dynamics, it is also unclear whether there are unique characteristics of the blood pressure response after exercise.

We hypothesized that severely impaired CANA and reduced exercise capacity significantly impact the delayed HR and  $\dot{V}O_2$  recoveries [11–13]. In addition, we thought that the lack of a subpulmonary ventricle and the type of Fontan repair may also influence these recoveries, especially that of SBP, because of the reduced capacity to handle a

transient increase in cardiac venous return immediately after exercise. In this respect, we thought that comparison of Fontan patients with patients after RVOTR with identical exercise capacity would help clarify the differences in post-exercise HR, SBP and  $\dot{V}O_2$  recoveries.

## 1. Methods

### 1.1. Subjects

We retrospectively studied 51 patients with the Fontan circulation, 34 post-operative RVOTR patients and 26 age and body size-matched control subjects (Table 1). In addition to this cohort of study patients, we also reviewed 12 patients who had performed exercise tests before and after the Fontan operation.

#### 1.1.1. Fontan patients

Of the Fontan patients, a total cavopulmonary connection (TCPC) was created in 33 and an atriopulmonary con-

Table 1  
Clinical characteristics of the study patients

| Group  | Fontan (n=51)                                     |  | RVOTR (n=34)           | Control (n=26) |
|--|---|--|------------------------|----------------|
|  | APC (n=18)  | TCPC (n=33)  | –                      | –              |
| Age (years)  | 15±4  | 14±4   | 15±4                   | 14±4           |
| Body weight (kg)                                   | 42±11   | 41±12  | 43±13                  | 44±11          |
| Follow-up (years)                                  | 10±3 <sup>##,!!!</sup>                            | 7±3  | 6±5                    | –              |
| Disease  | TA [8], UVH [2]<br>DORV [1], MA [3]<br>Others [4] | TA [5], UVH [11]<br>DORV [8], MA [6]<br>Others [3] | TOF [27], DORV [7]     | Hx of KD [26]  |
| Hemodynamics                                       | (n=16)  | (n=31)   | (n=34)                 | (n=25)         |
| Central venous pressure (mm Hg)                    | 13±3 <sup>***,!!!</sup>                           | 12±3 <sup>***,!!!</sup>                            | 7±3 <sup>***</sup>     | 3±1            |
| Pulmonary artery pressure (mm Hg)                  | 12±2 <sup>!!!</sup>                               | 12±3 <sup>!!!</sup>                                | 19±6 <sup>***</sup>    | 13±2           |
| SV end-diastolic pressure (mm Hg)                  | 8±3 <sup>*,!!!</sup>                              | 9±3 <sup>*,!!!</sup>                               | 12±5                   | 11±3           |
| SV end-diastolic volume index (ml/m <sup>2</sup> ) | 74±30 <sup>!!</sup>                               | 85±35 <sup>!</sup>                                 | 103±33 <sup>***</sup>  | 77±13          |
| SV ejection fraction (%)                           | 50±12 <sup>***,!</sup>                            | 50±13 <sup>***,!!</sup>                            | 58±12 <sup>*</sup>     | 66±8           |
| Cardiac index (L/min/m <sup>2</sup> )              | 2.1±0.4 <sup>***,!!!,#</sup>                      | 2.5±0.6 <sup>***,!</sup>                           | 2.9±0.6 <sup>***</sup> | 3.6±0.8        |
| Pulmonary artery resistance (U·m <sup>2</sup> )    | 2.5±0.7   | 1.9±0.8 <sup>!!!</sup>                             | 3.2±2.2 <sup>***</sup> | 1.7±0.6        |
| Systemic artery resistance (U·m <sup>2</sup> )     | 37±7 <sup>***,!!!,#</sup>                         | 31±9 <sup>**</sup>                                 | 30±8 <sup>*</sup>      | 24±6           |
| Arterial oxygen saturation (%)                     | 96±1 <sup>*,##</sup>                              | 93±5 <sup>***,!!!</sup>                            | 98±1                   | 98±1           |
| Natriuretic peptides                               |   |  |                        |                |
| Atrial natriuretic peptide (pg/ml)                 | 127±70 <sup>***,!!!,####</sup>                    | 51±47 <sup>!</sup>                                 | 81±63 <sup>***</sup>   | 24±10          |
| Brain natriuretic peptide (pg/ml)                  | 95±66 <sup>***,!,##</sup>                         | 38±82  | 56±79 <sup>**</sup>    | 5±4            |
| Cardiac autonomic nervous activity                 |   |  |                        |                |
| BRS (ms/mm Hg)                                     | 3.1±3.1 <sup>***</sup>                            | 3.4±3.2 <sup>***</sup>                             | 3.0±2.6 <sup>***</sup> | 19.0±6.3       |
| Plasma norepinephrine (pg/ml)                      | 277±176 <sup>***,!!</sup>                         | 245±116 <sup>***,!!</sup>                          | 169±90                 | 159±78         |

BRS=arterial baroreflex sensitivity, DORV=double outlet right ventricle, Hx of KD=history of Kawasaki disease, MA=mitral atresia, UVH=univentricular heart, SV=systemic ventricle, TA=tricuspid valve atresia.

Values are mean±S.D.

\*  $p<0.05$  vs. control.

\*\*  $p<0.01$  vs. control.

\*\*\*  $p<0.001$  vs. control.

#  $p<0.05$  vs. TCPC.

##  $p<0.01$  vs. TCPC.

###  $p<0.001$  vs. TCPC.

!  $p<0.05$  vs. RVOTR.

!!  $p<0.01$  vs. RVOTR.

!!!  $p<0.001$  vs. RVOTR.

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