

Cardiopulmonary exercise test in the evaluation of exercise capacity, arterial hypertension, and degree of descending aorta stenosis in adults after repair of coarctation of the aorta

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Abstract

Background: *Despite effective repair of coarctation of the aorta (CoAo), arterial hypertension (AH) and early coronary artery disease that may result in heart failure. The aim of the study was to evaluate exercise capacity by a cardiopulmonary exercise test in patients after of CoAo repair, and to determine relations between these parameters and the presence of AH, residual stenosis of the descending aorta (AoD) and the patient's age at the time of the surgery.*

Methods: *74 patients at mean age 31.2 ± 9.8 years. The controls: 30 at mean age 32.2 ± 6.6. Descending aorta (AoD) gradient was evaluated by echocardiography. The group with residual AoD stenosis: ≥ 25 mm Hg (AoD+) 32 patients and AoD–: 41 patients. Subgroups without AH (AH–, n = 32), exercise-induced AH (AHex, n = 10), persistent AH (AH+, n = 32). The maximum exercise test was performed.*

Results: *A comparison of the study and control groups: VO₂max: p = 0.0001, VO₂max%: p = 0.0001 and VE/VCO₂: p = 0.001. Negative correlation: between VO₂max and the age at the time of surgery: p = 0.004) and a positive: between VE/VCO₂ and age at surgery: p = 0.005. No differences were observed between the AoD+ and AoD– groups with respect to cardiopulmonary parameters. A comparison of the AH+ and AH– groups revealed: VO₂max: p = 0.01, VO₂max%: p = 0.02 and VE/VCO₂: p = 0.003. A comparison of the AHex and AH– groups showed VE/VCO₂: p = 0.01.*

Conclusions: *The exercise capacity of adults after surgical CoAo repair is reduced. This is more pronounced in patients with AH and those operated on at a more advanced age, but not in AoD+. (Cardiol J 2007; 14: 76–82)*

Key words: coarctation of the aorta in adults, cardiopulmonary exercise test

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Introduction

The history of effective repair of coarctation of the aorta (CoAo) reaches back over fifty years [1–5]. Long-term monitoring has shown that the outcome of the original repair is more favourable in subjects

operated on early in the childhood [2, 3, 6], although the repair carried out in neonates has been associated with a greater incidence of residual stenosis of the descending aorta (AoD) [7–12]. However, even successful repair does not prevent a reduction in life expectancy [5, 9, 11]. Following repair of CoAo a considerable number of adults present with arterial hypertension (AH) and early coronary artery disease, then progressing to heart failure, which is one of the causes of early death in this population [2–4]. Satisfactory evaluation of exercise capacity is reported by most patients; however, individual judgment of exercise performance does not fully reflect a patient's actual clinical condition [2, 4, 12]. It may therefore be essential to evaluate oxygen uptake (peak $\dot{V}O_2$) with a maximum stress test combined with spirometry, referred to as a cardiopulmonary exercise test, which is the objective tool for measuring exercise tolerance. The results of the cardiopulmonary stress test are established predictors of poor prognosis in heart failure [13, 14].

The aim of the study was to assess exercise capacity measured with a cardiopulmonary stress test in adults after successful repair of CoAo and to investigate potential associations of cardiopulmonary stress test indices and AH, residual stenosis of the AoD, and age at the time of surgery.

Methods

The study population was selected from a group of 107 patients with a history of repair of CoAo who attended the Congenital Heart Disease Outpatient Clinic at Department of Cardiology No. 1 of the University of Medical Sciences in Poznan. The patients included in the study fulfilled the following criteria: they were eligible to complete an a cardiopulmonary exercise test, they did not present ischaemic heart disease or respiratory disorders and there was no significant aortic regurgitation. Finally the study involved 74 patients (29 females and 45 males) aged from 19 to 61 (mean 31.2 ± 9.8) years, who had been operated on at the age of 0.5–34 (mean 10.4 ± 6.8) years. Surgery had been performed between 5 and 34 years ago (mean 21.4 ± 6.2 years). The control group consisted of 30 healthy individuals (12 females and 18 males) aged from 26 to 46 (mean 32.2 ± 6.6) years. The majority of patients ($n = 65$, 88%) were operated on by the same cardiac surgeon using a synthetic patch, three had been treated with subclavian flap aortoplasty (4%) and another six (8%) using the end-to-end anastomosis approach, while one had had a St. Jude 24 mechanical prosthetic valve

implanted. Eight study patients underwent reoperation owing to stenosis of the AoD; non-invasive treatment of re-coarctation was applied in a further case and one patient underwent implantation of a stent graft in the AoD because of aneurysm. The following congenital heart diseases were additionally diagnosed in some of the patients: ventricular septal defect operated on in the past ($n = 6$), occluded persistent arterial duct ($n = 5$) and trivial mitral insufficiency ($n = 19$). All patients were classified as NYHA functional class I and all had sinus rhythm. Creatinine serum concentrations did not exceed $140 \mu\text{mol/l}$ and aspartate aminotransferase levels did not exceed double normal values.

Echocardiographic examination was performed using Vivid 7 with a 2.5 MHz probe in two-dimensional, M and Doppler modes. The evaluation included the assessment of cardiac anatomy and left ventricular systolic function using the Simpson biplane method (normal values of the latter parameters were additional inclusion criterion). Peak aortic valve gradient was determined by continuous-wave Doppler echocardiography (AoGrmax). The severity of residual stenosis of the AoD was assessed from a suprasternal notch view. Restenosis was diagnosed when the AoD gradient was equal to or greater than 25 mm Hg [2]. This measurement was used to divide the study population into the subgroup without residual stenosis of the AoD (AoD–) and that with residual stenosis of the AoD (AoD+). Echocardiography was performed by the same independent professional investigator.

All patients carried out a maximum symptom-limited (fatigue and/or dyspnoea) treadmill exercise test according to a modified Bruce protocol (adding to the standard Bruce protocol stage 0–3 min; 1.7 km/h, at 5% grading), whereas the control subjects were tested according to the standard Bruce protocol. Patients were encouraged to continue with the test for as long as their respiratory quotient (RQ) exceeded one. The maximum oxygen uptake (peak $\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), and minute ventilation (VE) were measured using breath-by-breath gas analysis (Sensor Medics, model Vmax29). The system was calibrated with a standard gas mixture of standard concentration before each test. Spirometry was performed in all subjects before the cardiopulmonary exercise test, including the measurement of forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) and was calculated as a percentage of predicted values, taking into account age and sex. The best achieved results from 3–4 spirometry cycles were selected for analysis. Peak $\dot{V}O_2$ was determined as

an average value within the last 20 s of exercise and expressed as mL/kg/min, and as the percentage of predicted peak oxygen uptake. The ventilation/carbon dioxide slope (VE/VCO₂ slope), reflecting the increase of ventilation relative to carbon dioxide production and obtained from linear regression analysis, was calculated automatically by the Vmax29 computer system.

Blood pressure at rest was registered in all participants by a cuff sphygmomanometer. The measurement was repeated immediately after exercise. Exercise-induced AH was diagnosed if systolic blood pressure at peak exercise was above 200 mm Hg [10] in patients with normal baseline blood pressure. Blood pressure exceeding 140/90 mm Hg was classified as AH according to ESH/ESC guidelines (2003) [15]. The study involved hypotensive agent naive patients and patients in whom antihypertensive therapy could be withdrawn at least two weeks prior to the treadmill test.

Statistical analysis

Statistical analysis was performed with Student's *t*-test for unpaired samples for variables following normal distribution. In case of variables not following normal distribution the Mann-Whitney U test was used. To determine the relationships between variables Spearman's rank order correlations were analysed.

Informed consent was obtained from each patient and the study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in *a priori* approval of the institution's Human Research Committee.

Results

The results of the cardiopulmonary exercise test are detailed in Table 1. We observed that the exercise performance of adults after repair of CoAo was considerably decreased: peak oxygen uptake (maxVO₂) in the study patients was lower than in healthy individuals (26.77 ± 6.75 vs. 35.27 ± 7.6 mL/kg/min, *p* = 0.0001) and likewise when expressed as percentages (maxVO₂[%] of 70.09 ± 13.39 vs. 90.3 ± 15.35, *p* = 0.0001). The ventilation/carbon dioxide slope (VE/VCO₂) was higher in the study group (33.14 ± 5.33 vs. 29.73 ± 3.32, *p* = 0.001). Heart rate at maximum workload in the study patients was reduced in comparison to controls (167.91 ± 21.25 vs. 180.83 ± 12.93 bpm, *p* = 0.0001) and the same was observed for HRmax[%] (88.25 ± 10.17 vs. 100.33 ± 7.11, *p* = 0.0001). The RQ in patients after surgery was lower than in controls (1.06 ± 0.07 vs. 1.13 ± 0.09, *p* = 0.001). Diastolic blood pressure at rest and during exertion did not differ between the two groups, whereas systolic blood pressure, both resting (136.58 ± 18.92 vs.

Table 1. Characteristics and comparison of cardiopulmonary parameters in study and control groups.

	Study group (n = 74)	Control group (n = 30)	p
Current age (years)	31.2 ± 9.8	32.2 ± 6.6	NS
Age at surgery (years)	10.4 ± 6.8	–	NS
Body mass index [kg/m ²]	29.4 ± 2.21	30.2 ± 3.12	NS
VO ₂ [ml/kg/min]	26.77 ± 6.75	35.27 ± 7.6	0.0001
VO ₂ [%]	70.09 ± 13.39	90.3 ± 15.35	0.0001
VE/VCO ₂	33.14 ± 5.33	29.73 ± 3.32	0.001
HR [bpm]	167.91 ± 21.25	180.83 ± 12.93	0.0001
HRmax [%]	88.25 ± 10.17	100.33 ± 7.11	0.0001
FVC [L]	4.12 ± 0.95	8.03 ± 0.53	0.02
FVC [%]	93.19 ± 6.49	103.2 ± 9.3	0.001
FEV ₁ [L]	3.30 ± 0.80	3.76 ± 0.86	0.02
FEV ₁ [%]	87.86 ± 14.4	98.37 ± 10.65	0.0004
Respiratory quotient	1.06 ± 0.07	1.13 ± 0.09	0.001
SBP at rest [mm Hg]	136.56 ± 18.92	126.66 ± 11.87	0.004
DBP at rest [mm Hg]	85.76 ± 8.61	83.33 ± 8.23	NS
SBP ex [mm Hg]	198.25 ± 25.29	167.93 ± 15.67	0.001
DBP ex [mm Hg]	88.82 ± 1.40	86.50 ± 9.94	NS

VO₂ — maximum oxygen uptake, VE/VCO₂ — ventilatory equivalent for carbon dioxide, HR — heart rate, HRmax — peak exercise heart rate, FVC — forced vital capacity, FEV₁ — forced expiratory volume in 1 s, SBP — systolic blood pressure, DBP — diastolic blood pressure. Please refer to the text for the remaining abbreviations

Table 2. Characteristics and comparison of cardiopulmonary parameters in patients in relation to the presence of re-coarctation.

	Study group AoD- (n = 41)	Study group AoD+ (n = 32)	p
Current age (years)	29.47 ± 9.13	33.45 ± 10.58	NS
Age at surgery (years)	8.98 ± 5.94	12.45 ± 7.49	0.03
VO ₂ [ml/kg/min]	27.04 ± 6.33	26.61 ± 7.48	NS
VO ₂ [%]	71.96 ± 12.79	68.78 ± 12.52	NS
VE/VCO ₂	26.13 ± 5.50	33.72 ± 5.40	NS
HR [bpm]	167.18 ± 21.74	163.82 ± 20.93	NS
HRmax [%]	87.29 ± 10.66	91.75 ± 10.38	NS
Respiratory quotient	1.07 ± 0.09	1.05 ± 0.07	NS
FVC [L]	4.25 ± 1.00	3.97 ± 0.88	NS
FVC [%]	92.73 ± 12.83	96.14 ± 16.48	NS
FEV ₁ [L]	3.40 ± 0.80	3.16 ± 0.82	NS
FEV ₁ [%]	86.38 ± 13.27	88.45 ± 15.34	NS
SBP at rest [mm Hg]	136.31 ± 19.99	138.39 ± 18.31	NS
DBP at rest [mm Hg]	85.57 ± 0.06	84.64 ± 9.32	NS
SBP ex [mm Hg]	201.63 ± 27.15	196.89 ± 21.94	NS
DBP ex [mm Hg]	87.36 ± 12.12	88.50 ± 10.75	NS

AoD — descending aorta; VO₂ — maximum oxygen uptake, VE/VCO₂ — ventilatory equivalent for carbon dioxide, HR — heart rate, HRmax — peak exercise heart rate, FVC — forced vital capacity, FEV₁ — forced expiratory volume in 1 s, SBP — systolic blood pressure, DBP — diastolic blood pressure. Please refer to the text for the remaining abbreviations

126.66 ± 11.87 mm Hg, $p = 0.004$), and during exercise (198.25 ± 25.29 vs. 167.94 ± 15.67, $p = 0.001$) was higher in the study patients. The parameters of pulmonary elasticity, including FVC and FVC[%], were lower in the patients studied than in controls: 4.12 ± 0.95 vs. 8.03 ± 0.53 ($p = 0.02$) and 93.19 ± 16.49 vs. 103.2 ± 9.3 respectively ($p = 0.001$). Moreover, the parameters used to identify airway obstruction, including FEV₁: 3.30 ± 0.80 L and FEV₁[%]: 87.86 ± 14.41 were significantly lower in post-surgical patients than in controls: FEV₁: 3.76 ± 0.86 L and FEV₁[%]: 98.37 ± 10.65 ($p = 0.02$, $p = 0.0004$ respectively).

Maximum aortic valve gradient ranged from 4.11 to 28.53 mm Hg (mean 11.75 ± 6.19). In 32 patients (AoD+) residual stenosis of the AoD was found, and the pressure gradient ranged from 25.0 to 60.2 mm Hg (mean 36.66 ± 9.81); in the remaining 41 patients (AoD-) it ranged from 5.51 to 24.0 mm Hg (mean 15.91 ± 5.08). The comparison of cardiopulmonary parameters between the AoD+ and AoD- groups is detailed in Table 2 and did not reveal any significant differences. AoD+ patients were operated on later in life (12.45 ± 7.49) than AoD- patients (8.98 ± 5.94, $p = 0.03$).

Thirty-two patients had normal resting blood pressure (AH-) and 10 had AH induced by exercise activity (ATex); the remaining group of 31 subjects (AH+) had persistent AH (Table 3). The assess-

ment of AT- and AHex showed that patients with exertional AH had a higher ventilation-to-perfusion ratio: VE/VCO₂ (28.31 ± 4.56 vs. 33.89 ± 5.02, $p = 0.01$) but reached lower heart rates at peak exercise (181.00 ± 13.05 vs. 171.24 ± 15.76 bpm, $p = 0.001$). A comparison of the AH- and AH+ subgroups showed that persistently hypertensive patients had a reduction in the following parameters: peak oxygen uptake: VO₂max (26.30 ± 15.08 vs. 28.05 ± 7.9 mL/kg/min, $p = 0.01$), VO₂max[%] (66.17 ± 14.58 vs. 74.75 ± 12.16, $p = 0.02$), heart rate at peak exercise: HRmax (162.46 ± 21.76 vs. 181.00 ± 13.05 bpm, $p = 0.0001$), HR[%] (84.46 ± 10.58 vs. 94.31 vs. 7.91, $p = 0.0004$) and RQ (1.04 ± 0.5 vs. 1.08 ± 0.09, $p = 0.03$), whereas the ventilation-to-perfusion ratio was higher: VE/VCO₂ (28.31 ± 4.56 vs. 34.06 ± 4.78, $p = 0.003$). A comparison between the AHex and AH+ subgroups showed patients with persistent AH had a lower HRmax (162.46 ± 21.73 vs. 171.24 ± 15.76 bpm, $p = 0.001$), HR[%] (84.46 ± 10.58 vs. 92.30 ± 6.11, $p = 0.01$) and RQ (1.04 ± 0.05 vs. 1.09 ± 0.07, $p = 0.02$). No differences were observed between any of the groups with respect to spirometric parameters.

Patients with persistent AH were older than the AH- group (35.59 ± 11.02 vs. 28.62 ± 7.78 years, $p = 0.004$) and exercise-induced hypertensive individuals (35.59 ± 11.02 vs. 25.20 ± 5.07 years, $p = 0.006$). The age at the time of surgery of

Table 3. Characteristics and comparison of cardiopulmonary parameters in patients in relation to the presence of arterial hypertension.

	Study group AH- (n = 32)	Study group AHex (n = 10)	Study group AH+ (n = 31)	P (AH- and AHex)	P (AH- and AH+)	P (AH+ and AHex)
Current age (years)	28.62 ± 7.78	25.20 ± 5.07	35.59 ± 11.02	NS	0.004	0.006
Age at surgery (years)	9.12 ± 5.83	6.50 ± 3.71	12.95 ± 7.53	NS	0.02	0.01
VO ₂ [ml/kg/min]	28.05 ± 7.9	27.97 ± 6.68	26.30 ± 15.08	NS	0.01	NS
VO ₂ [%]	74.75 ± 12.16	71.00 ± 13.49	66.17 ± 14.58	NS	0.02	NS
VE/VCO ₂	28.31 ± 4.56	33.89 ± 5.02	34.06 ± 4.78	0.01	0.003	NS
HRmax [bpm]	181.00 ± 13.05	171.24 ± 15.76	162.46 ± 21.73	0.001	0.0001	0.001
HRmax [%]	94.31 ± 7.91	92.30 ± 6.11	84.46 ± 10.58	NS	0.0004	0.001
Respiratory quotient	1.08 ± 0.9	1.09 ± 0.07	1.04 ± 0.05	NS	0.03	0.02
FVC [L]	4.72 ± 0.75	4.42 ± 0.77	4.07 ± 1.12	NS	NS	NS
FVC [%]	92.79 ± 19.10	91.50 ± 9.61	94.21 ± 15.9	NS	NS	NS
FEV ₁ [L]	3.22 ± 0.65	3.38 ± 0.65	3.21 ± 0.95	NS	NS	NS
FEV ₁ [%]	89.89 ± 14.71	86.40 ± 8.34	86.28 ± 15.98	NS	NS	NS
SBP at rest [mm Hg]	126.20 ± 15.50	132.00 ± 8.21	149.71 ± 8.98	NS	0.0001	0.01
DBP at rest [mm Hg]	80.51 ± 8.48	82.50 ± 3.53	91.17 ± 2.74	NS	0.0001	0.001
SBP ex [mm Hg]	169.48 ± 19.88	212.00 ± 11.98	210.23 ± 23.21	0.0001	0.0001	NS
DBP ex [mm Hg]	85.17 ± 9.77	87.00 ± 14.18	95.46 ± 7.09	NS	0.0001	0.003

AH — arterial hypertension; AHex — with exercise-induced arterial hypertension; VO₂ — maximum oxygen uptake, VE/VCO₂ — ventilatory equivalent for carbon dioxide, HR — heart rate, HRmax — peak exercise heart rate, FVC — forced vital capacity, FEV₁ — forced expiratory volume in 1 s, SBP — systolic blood pressure, DBP — diastolic blood pressure. Please refer to the text for the remaining abbreviations

the AH+ patients was higher in comparison to both the AH- (12.95 ± 7.53 vs. 9.12 ± 5.83 years, p = 0.02) and AHex subgroups (12.95 ± 7.53 vs. 6.50 ± 3.71, p = 0.01). At the same time the gradient through the AoD did not differ between the respective groups (AH-: 25.52 ± 14.02 mm Hg, AHex: 30.69 ± 15.3 mm Hg, and AH+: 24.68 ± 13.4 mm Hg).

Linear correlation analysis, including current age, age at operation, time elapsing after the operation, blood pressure and cardiopulmonary parameters in the study group as a whole showed a significant negative correlation between the present age of patient and VO₂max (r = -0.328, p = 0.005), which was positive with respect to VE/VCO₂ (r = 0.337, p = 0.006), and a negative correlation was found between age at surgical repair and VO₂max (r = -0.343, p = 0.004) (Fig. 1), positive with respect to VE/VCO₂ (r = 0.335, p = 0.005) (Fig. 2). No significant correlations were observed between exercise capacity parameters and blood pressure. A statistically significant correlation was observed between VO₂max and HR [%] (r = 0.485, p = 0.00003) (Fig. 3).

Discussion

Even though adults with previous repair of CoAo assess their exercise capacity as satisfacto-

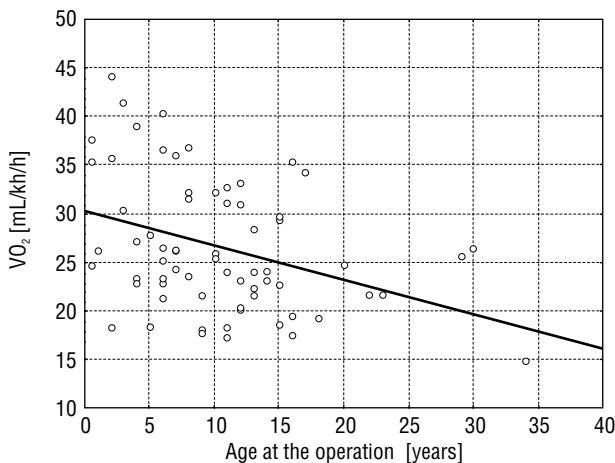


Figure 1. Correlation between age at surgical repair and maximum oxygen consumption: VO₂max (r = -0.343, p = 0.004)

ry, as observed by number of investigators [2, 3, 12], our objective evaluation with the use of a cardiopulmonary exercise test showed that it is considerably impaired. Balderston et al. [16] reported differently with regard to children and Markham et al. [8, 9] with adolescents. Our conclusion remains consistent with Rhodes et al. [17] and the results of



Figure 2. Correlation between age at surgical repair and ventilatory equivalent for carbon dioxide: VE/VCO_2 ($r = 0.335$, $p = 0.005$)

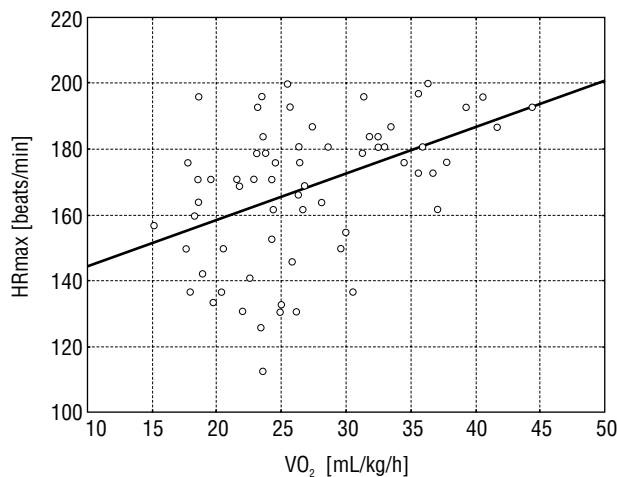


Figure 3. Correlation between maximum oxygen consumption (VO_2) and heart rate: HR_{max} ($r = 0.485$, $p = 0.00003$)

a comprehensive analysis carried out by investigators from the Royal Brompton Hospital in London of the adult population with congenital heart disease, which also included patients after surgical repair of CoAo [12]. These authors demonstrated that VO_{2max} of up to 15.5 mL/kg/min was an independent predictor of hospitalisation and death in patients with congenital heart disease. None of our patients failed to reach such a low result of peak oxygen uptake. Abnormal chronotropic reaction and dependence between chronotropism and exercise capacity impairment were reported [9, 12, 18], as observed in adults with different congenital heart diseases and also in this study. This observation was not supported by Swan et al. [7] and Balderston

et al. [16], although their populations only comprised children.

Restrictive and obstructive respiratory disorders were found in the study patients. Potential underlying pathomechanisms include reduced pulmonary compliance, heart enlargement, and pulmonary fibrosis secondary to chronic pulmonary congestion and respiratory myopathy [12, 19]. However, the severity of respiratory function impairment did not differ between subgroups with respect to re-coarctation, persistent AH and exercise-induced AH.

Exercise capacity compromise was seen to be dependent on the patient's current age [13, 14], while at the same time it was inversely related to the age at the time of coarctation repair. A similar relation to age at operation in 335 adults with congenital heart disease was observed by the Royal Brompton Hospital investigators referred to above [12]. This finding lends support to efforts to repair this anatomical defect early [2–5, 10].

The severity of exercise capacity impairment in patients with re-coarctation was similar to those without residual stenosis of the AoD. It should be highlighted that the study population with residual stenosis of the AoD did not present increased blood pressure, as compared to the patients free of re-coarctation, findings that are in accordance with the results of numerous analyses [19–21] and in disagreement with others [1, 7–10]. The occurrence of isolated AH was not unrelated to exercise capacity. Significantly worse cardiopulmonary parameters in terms of peak oxygen uptake and ventilation-to-perfusion ratio were observed in hypertensive patients than in normotensive ones. Additionally, an increase in ventilation-to-perfusion rate was found in patients with exercise-induced AH in comparison with normotensives. The severity of chronotropic impairment was also significantly different in the subgroups. The results presented here were undoubtedly influenced by the greater age of the patients with persistent AH, as well as their age at repair in comparison to the patients without hypertension. Normotensive patients did not differ from those with exercise-induced hypertension with regard to age and the age at the time of the operation; however, the latter group presented poorer exercise capacity. This suggests that, in contrast to the hypothesis of Swan et al. [7] and Leonardo et al. [22], which challenge the need to select the group of subjects with “latent hypertension” after repair of CoAo, patients with exercise-induced AH require particular clinical monitoring [1–4, 7] and, according to some authors, earlier introduction of antihypertensive agents [5].

Conclusions

The exercise capacity of adults after surgical repair of coarctation of the aorta is reduced. This reduction is more pronounced in patients with arterial hypertension and is not affected by residual stenosis of the descending aorta. Exercise capacity reduction is also more pronounced in patients operated on when older, which confirms the importance of earlier surgical correction.

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