Background: One of the main goals of corrective surgery of congenital heart defects in children is the improvement of quality of life, which in young children is predominantly determined by exercise capacity. It is not known whether this goal can be achieved in school-aged children who have undergone cardiac surgery in infancy.

Objectives: To determine if primary school-aged children who underwent surgery to correct congenital heart defects in infancy are physically as fit as their peers.

Methods: We examined 84 children with congenital heart defects, aged 4 to 11 years, after surgical repair. Fifty-two children had simple defects (ie, atrial or ventricular septal defect, coarctation of the aorta). Thirty-two children had complex defects (ie, tetralogy of Fallot, pulmonary atresia with ventricular septal defect). All patients underwent exercise testing performed on a specially modified bicycle ergometer. Ninety-eight sex- and age-matched healthy children served as the control group.

Results: There was no gender difference, either in healthy children or in the group with congenital heart defects, regarding exercise testing and that the healthy children reached a mean ± SD normalized maximal performance of 2.8 ± 0.3 W/kg. The same range was found for the children who had undergone surgery to correct simple heart defects. The children operated on to correct complex heart defects showed significantly impaired mean normalized maximal performance, although this tended to be lower in the group that had pulmonary atresia with a ventricular septal defect than in the group with tetralogy of Fallot (mean normalized maximal performance, 1.9 W/kg vs 2.3 W/kg).

Conclusions: The goal of normal exercise capacity in childhood after heart surgery is achieved in those with simple heart defects only. In children with complex heart defects impaired exercise performance persists, depending on the severity of the heart defect and probably on chronotropic incompetence.

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THE GOAL OF SURGICAL CORRECTION OF CONGENITAL HEART DEFECTS IN CHILDREN IS NOT ONLY TO ENSURE SURVIVAL BUT ALSO TO ENABLE A LIFE AS NORMAL AS POSSIBLE. IN PARTICULAR IN SCHOOL-AGED CHILDREN THIS INCLUDES MEASURING PHYSICAL PERFORMANCE AGAINST THAT IN HEALTHY PEERS. EXERCISE TESTS ARE WIDELY USED AS DIAGNOSTIC AND THERAPEUTIC TESTS IN ADULTS. THESE DYNAMIC TESTS EVALUATE EXERCISE TOLERANCE AND ADAPTATIONS, ESSENTIALLY THOSE THAT ARE CARDIORESPIRATORY. THEY ARE ALSO USEFUL FOR DIAGNOSIS AND ASSESSMENT OF ABNORMAL SYMPTOMS DURING EXERCISE AND FOR INITIATION AND INDIVIDUALIZATION OF EXERCISE TRAINING. WE, THEREFORE, POSED THE QUESTION: "ARE PRIMARY SCHOOL-AGED CHILDREN WHO UNDERWENT SURGERY TO CORRECT CONGENITAL HEART DEFECTS IN INFANCY PHYSICALLY AS FIT AS THEIR PEERS?" THIS QUESTION IS ALL THE MORE IMPORTANT BECAUSE ADOLESCENTS AND YOUNG ADULTS WHO HAVE BEEN OPERATED ON TO CORRECT HEART DEFECTS HAVE DEFICITS IN PHYSICAL PERFORMANCE.1-13 THE FEW AVAILABLE STUDIES ON THIS SUBJECT ARE, TO SOME EXTENT, CONTRADICTORY.1,5

METHODS

PATIENTS

Data for 182 children aged 4 to 11 years were evaluated. The study was performed from July 1, 2000, to August 31, 2003, at the University Children’s Hospital, Göttingen, Germany.

Our patients were 52 children who had undergone surgery to correct a simple heart defect, that is, ventricular septal defect (VSD) or atrial septal defect (ASD; n=24) or coarctation of the aorta (CoA; n=28), and 32 children who had undergone surgery to correct a complex heart defect, that is, tetralogy of Fallot (TOF; n=23) or pulmonary atresia with VSD (PA+VSD; n=9).

The control group included 98 healthy children who were matched with the patients for sex and age. They had no heart disease and were chosen from patients examined to clarify an innocent heart murmur or chest pain.

The 182 children included in this study had New York Heart Association functional class I disease, that is, no limitation of physical activity; ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea (shortness of breath) (Table 1). Mean age at operation was 21 to 30 months; only the children with CoA were operated on significantly earlier, at age 7 months.
The types of surgery, palliative therapy, and reoperations performed are listed in Table 2. Because of the heart defect, 8 patients in the TOF or PAcVSD group received an aortopulmonary shunt as a palliative measure before corrective surgery. In 14 children in the TOF group a transannular plastic patch of the pulmonary valve was used. In the remaining 9 patients a transvalvular gradient.

In addition to a thorough clinical examination, all patients underwent 2-dimensional and M-mode echocardiography through a transesophageal approach with a diagnostic ultrasound system (Sonos 2000; Hewlett-Packard; Andover, Mass) interfaced with a multifrequency megahertz transducer. End-systolic diameter (ESD) and end-diastolic diameter (EDD) of the left ventricle were determined at echocardiography from a short axis view with M-mode echocardiography. Fractional shortening ([EDD − ESD]/EDD) was used as a parameter for pump function of the left ventricle. The velocities of tricuspid valve regurgitation and antegrade pulmonary valve flow were measured with continuous-wave Doppler ultrasonography and served to estimate the right ventricular (RV) systolic pressure and the transvalvular gradient.

A standard 12-lead electrocardiogram was recorded to analyze the cardiac rhythm and to determine the QRS complex du-

### Table 1. Anthropometric Data in Healthy Children and Patients With Congenital Heart Defects After Corrective Surgery*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Healthy Children (n = 98)</th>
<th>TOF (n = 23)</th>
<th>PAcVSD (n = 9)</th>
<th>CoA (n = 28)</th>
<th>ASD/VSD (n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boys, No.</td>
<td>49</td>
<td>12</td>
<td>5</td>
<td>18</td>
<td>12</td>
</tr>
<tr>
<td>Girls, No.</td>
<td>49</td>
<td>11</td>
<td>4</td>
<td>10</td>
<td>12</td>
</tr>
<tr>
<td>Age, y</td>
<td>7.8 ± 1.8 (4.3-11)</td>
<td>8.2 ± 2.0 (5-11)</td>
<td>8.0 ± 2.5 (5.1-11)</td>
<td>7.9 ± 2.2 (4.4-10.8)</td>
<td>7.7 ± 1.7 (4-10.5)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>27.3 ± 5.7 (15-41)</td>
<td>27.7 ± 9.4 (15-50)</td>
<td>27.8 ± 9.5 (17-48)</td>
<td>26.1 ± 7 (15-47)</td>
<td>25 ± 5 (18-35)</td>
</tr>
<tr>
<td>Height, cm</td>
<td>129 ± 11 (109-159)</td>
<td>130 ± 14 (105-155)</td>
<td>130 ± 15 (111-156)</td>
<td>127 ± 13 (105-148)</td>
<td>127 ± 10 (105-145)</td>
</tr>
</tbody>
</table>

Abbreviations: ASD, atrial septal defect; CoA, coarctation of the aorta; PAcVSD, pulmonary atresia with VSD; TOF, tetralogy of Fallot; VSD, ventricular septal defect.

*Data are given as the mean ± SD (range) unless otherwise indicated.

### Table 2. Characteristics of Patients With Congenital Heart Defects After Corrective Surgery and of Healthy Children*

<table>
<thead>
<tr>
<th>Variable</th>
<th>TOF (n = 23)</th>
<th>PAcVSD (n = 9)</th>
<th>CoA (n = 28)</th>
<th>ASD/VSD (n = 24)</th>
<th>Healthy Children (n = 98)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at operation, mo</td>
<td>21 ± 16</td>
<td>30 ± 25</td>
<td>7 ± 11</td>
<td>29 ± 23</td>
<td>...</td>
</tr>
<tr>
<td>Follow-up after operation, y</td>
<td>6.4 ± 2.6</td>
<td>5.5 ± 2.7</td>
<td>7.3 ± 2.5</td>
<td>5.3 ± 2.6</td>
<td>...</td>
</tr>
<tr>
<td>Palliation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortopulmonary shunt, No. (%)</td>
<td>5 (21)</td>
<td>3 (33)</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Type of operation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TAP</td>
<td>14</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>No TAP</td>
<td>9</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>RV-PA conduit implantation</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Septal direct closure</td>
<td></td>
<td>...</td>
<td>...</td>
<td>2</td>
<td>10</td>
</tr>
<tr>
<td>Septal patch closure</td>
<td>23</td>
<td>9</td>
<td>...</td>
<td>14</td>
<td>...</td>
</tr>
<tr>
<td>End-to-end anastomosis</td>
<td></td>
<td>...</td>
<td>...</td>
<td>28</td>
<td>...</td>
</tr>
<tr>
<td>Reoperation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary valve replacement, No. (%)</td>
<td>1 (4)</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Conduit exchange, No. (%)</td>
<td>...</td>
<td>4 (44)</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Ventricular septal closure, No. (%)</td>
<td>...</td>
<td>...</td>
<td>1 (3)</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Echocardiography</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular end-diastolic diameter, mm</td>
<td>39.2 (36-42)</td>
<td>38.8 (34-43)</td>
<td>39.4 (37-41)</td>
<td>38.7 (37-40)</td>
<td>40 (36-44)</td>
</tr>
<tr>
<td>Fractional shortening, %</td>
<td>36 (33-38)</td>
<td>34 (27-39)</td>
<td>38 (36-41)</td>
<td>36 (35-38)</td>
<td>36 (30-40)</td>
</tr>
<tr>
<td>Tricuspid insufficiency velocity, m/s</td>
<td>2.4 (2.1-2.7)†</td>
<td>3.2 (2.7-3.8)†</td>
<td>1.9 (1.4-2.4)</td>
<td>2 (1.9-2.3)</td>
<td>1.9 (1.8-2.0)</td>
</tr>
<tr>
<td>Pulmonary valve flow velocity, m/s</td>
<td>1.9 (1.7-2.1)†</td>
<td>3 (2.3-3.8)†</td>
<td>0.06 (0.9-1.0)</td>
<td>1 (0.7-1.3)</td>
<td>0.98 (0.8-1.2)</td>
</tr>
<tr>
<td>Flow velocity in descending aorta, m/s</td>
<td>1.3 (1.1-1.4)</td>
<td>1.1 (0.9-1.4)</td>
<td>2.1 (1.9-2.3)†</td>
<td>1.3 (1.2-1.4)</td>
<td>1.2 (1.0-1.4)</td>
</tr>
<tr>
<td>Electrocardiography</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>QRS duration (range), ms</td>
<td>133 (126-141)†</td>
<td>134 (119-148)†</td>
<td>83 (79-86)</td>
<td>86 (81-90)</td>
<td>80 (76-84)</td>
</tr>
</tbody>
</table>

Abbreviations: ASD, atrial septal defect; CoA, coarctation of the aorta; PAcVSD, pulmonary atresia with VSD; TOF, tetralogy of Fallot; VSD, ventricular septal defect.

†P < .001.

Significant correlation shown in boldface type.

The types of surgery, palliative therapy, and reoperations performed are listed in Table 2. Because of the heart defect, 8 patients in the TOF or PAcVSD group received an aortopulmonary shunt as a palliative measure before corrective surgery. In 14 children in the TOF group a transannular plastic patch of the pulmonary valve was used. In the remaining 9 patients a transannular patch was not necessary. In these patients the ventriculotomy was either closed directly or with a patch after commissurotomy of the pulmonary valve, resection of the hypertrophic infundibulum, and patch occlusion of the VSD. In 1 child the pulmonary valve was replaced with a homograft. All patients with PAcVSD were fitted with a right ventricle–pulmonary artery conduit. In 4 patients a conduit exchange was performed.

Coarctation of the aorta was corrected in all patients with end-to-end anastomosis. Two patients also had VSDs, which were closed at the same time. In 1 child the remaining VSD subsequently required surgical revision. In the ASD or VSD group the septal defect was repaired with a patch in 14 children and was directly closed in 10 children.

### CLINICAL INVESTIGATION

In addition to a thorough clinical examination, all patients underwent 2-dimensional and M-mode echocardiography through a transesophageal approach with a diagnostic ultrasound system (Sonos 2000; Hewlett-Packard; Andover, Mass) interfaced with a multifrequency megahertz transducer. End-systolic diameter (ESD) and end-diastolic diameter (EDD) of the left ventricle were determined at echocardiography from a short axis view with M-mode echocardiography. Fractional shortening ([EDD − ESD]/EDD) was used as a parameter for pump function of the left ventricle. The velocities of tricuspid valve regurgitation and antegrade pulmonary valve flow were measured with continuous-wave Doppler ultrasonography and served to estimate the right ventricular (RV) systolic pressure and the transvalvular gradient.

A standard 12-lead electrocardiogram was recorded to analyze the cardiac rhythm and to determine the QRS complex du-
ration. In each patient all examinations were performed on the same day.

**BICYCLE ERGOMETRY**

We used a bicycle ergometer (ERG 401; DIMEQ, Delft, Holland) for exercise testing. This ergometer is designed for adults and functions independently of rotational speed with eddy current brakes. To permit exercise testing in children aged 4 years and older, a child’s bicycle was mounted on the ergometer and attached to it with a drive belt.

During the measuring process the child should achieve a steady rotational speed (about 80 rpm). The special feature of this bicycle ergometer is that it is possible to gradually increase the workload, normally measured in watts, thus averting erratic increases in strain, which in young children is often disconcerting and results in abandonment of the exercise. We set 1 W/kg of body weight as the basic load and increased the strain every 10 seconds by 1 W. All patients exercised to maximum exercise capability. Criteria for abandoning exercise were the occurrence of significant rhythm or repolarization dysfunction, inadequate increase in pulse, exhaustion, pallor, dyspnea, dizziness, and headache.3

The maximal workload (P<sub>max</sub>, in watts) reached at the end of the session was determined, and the total workload (TW) (workload X exercise duration [W<sub>max</sub>]) was automatically calculated. The children’s heart activity was kept under surveillance with a 6-channel electrocardiographic monitor with a permanent indicator. Blood pressure was measured with an oscillometer (Critikon Dinamap Vital Systems Monitor 1846; Critikon Corporation, Tampa, Fla) before and immediately at the end of exercising. The latter measurement started up during the last 30 seconds of the exercise performance.

**EXCLUSION CRITERIA**

Based on findings at medical examination, the contraindications for exercise were assessed before each session using the guidelines of the American Heart Association.3 Patients receiving cardiac medication were also excluded.

**INFORMED CONSENT**

After adequate explanation of the purpose of the study, informed consent and assent was obtained from all patients and their parents. The study was designed retrospectively and was approved by the institutional review board.

**STATISTICAL ANALYSIS**

The results were analyzed using a computer with commercially available software (Excel 2000; Microsoft, Redmond, Wash; Prism; GraphPad Software Inc, San Diego, Calif). The group comparison was calculated on the base mean value plus or minus the 95% confidence interval. For comparison of individual parameters we used the mean±SD. Because most variables were not normally distributed, the nonparametric technique (Mann-Whitney test) was used. For all analyses, a value of P<.01 was considered statistically significant.

**RESULTS**

**PATIENTS**

As listed in Table 1 there were no significant differences in the mean age, weight, and height of the children in all groups. Neither did the postoperative observation period in the group who underwent cardiac surgery differ significantly (Table 2).

**ELECTROCARDIOGRAPHY**

All patients were in sinus rhythm. There were no patients with cardiac pacemakers. Seven children (78%) with PAcVSD and 20 children (83%) with TOF had right bundle branch block. All of the remaining children had a normal QRS interval. During and after the exercise test no cardiac complications (repolarization dysfunction, arrhythmias) were found in any child.

**ECHOCARDIOGRAPHY**

The left ventricular EDD and fractional shortening in all patient groups were in the normal range. With the exception of the children who had undergone surgery to correct CoA, the patients with simple heart defects showed no remarkable residual defects at echocardiography. In patients with CoA we found increased flow velocity in the descending aorta (mean±SD, 2.1±0.5 m/s), indicative of slight narrowing in this region after corrective surgery. Owing to the velocity of tricuspid regurgitation, the estimated RV systolic pressure in the TOF (2.4±0.03 m/s) and PAcVSD (3.2±0.03 m/s) groups was significantly higher than in the ASD or VSD (2.1±0.02 m/s) or CoA (1.9±0.04 m/s) groups. In addition, slight to moderate stenosis and insufficiency of the pulmonary valve or the conduit valve were seen in all patients in the TOF and PAcVSD groups (Table 2).

**BICYCLE ERGOMETRY**

**Control Group**

In healthy children we saw no difference between boys and girls for load duration, P<sub>max</sub>, normalized maximal performance (P<sub>max/kg</sub>), TW, heart frequency, and blood pressure. For this reason these data were collated without reference to gender and were used as control.

Various parameters can be used to assess physical capacity: P<sub>max</sub>, P<sub>max/kg</sub>, or TW = [Σ(performance X time)] in all stages of performance (W X minutes).

Figure 1 shows the P<sub>max</sub> subject to body surface area in the control group. It is clear that P<sub>max</sub> in the range ex-
Normalized maximal performance ($P_{\text{max}}/\text{kg}$) proves to be a parameter that is largely independent of growth in the examined range. Its mean value in healthy children is 2.8 W/kg (Figure 2). This means that healthy children of primary school age can, on average, achieve a $P_{\text{max}}/\text{kg}$ of 2.8 W/kg under our exercise conditions.

**Patients**

Table 3 gives the ergometry data for all children included in this study. The mean load duration in the TOF and PAcVSD groups of 8.5 minutes and 7.9 minutes, respectively, is significantly shorter than in healthy children, whereas that in the patients in the CoA and ASD or VSD groups, at 9.5 minutes and 9.2 minutes, is in the normal range. The patients with complex heart defects showed significantly reduced maximal heart rate during exercise, whereas this value was in the normal range in the patients with CoA or with ASD or VSD (Table 3).

Owing to technical conditions, blood pressure was measured in the left arm and was, on average, lower in the CoA group than in healthy children. This can be interpreted as a consequence of the operation, because the left subclavian artery is frequently included in the stenosis and the area of the operation.

In the TOF and PAcVSD groups, $P_{\text{max}}$ of 63 W and 54 W, respectively, was significantly below the norm. This was also true for TW (mean, 392 Wmin and 330 Wmin, respectively), because in these children the load duration was also below normal. In contrast, the parameters in the patients in the CoA and ASD or VSD groups exhibited no significant differences from those in healthy children: $P_{\text{max}}$ 72 W and 70 W; TW, 480 Wmin and 448 Wmin, respectively. The differences can be clearly demonstrated with the parameter $P_{\text{max}}/\text{kg}$. The mean value in children after CoA correction or ASD or VSD correction is 2.8 W/kg and is thus normal (Figure 3). The corresponding mean values after surgery to correct TOF or PAcVSD were 2.3 W/kg and 1.9 W/kg, respectively, and are thus significantly lower than those in healthy children and children who were operated on for correction of CoA or of ASD or VSD (Figure 4).

**Figure 5** shows the difference between the various patient groups more clearly. The $P_{\text{max}}/\text{kg}$ is recorded on the y-axis, and the patient collective on the x-axis. It can be seen that the children who underwent surgical correction of CoA or septal defects can be normally stressed, whereas the exercise capacity of children after surgery to correct TOF or PAcVSD is markedly reduced.

Because ergometry cannot be easily performed with standard means in children of primary school age and valid spiroergometry is not practical in this age group, methodologic problems arise when attempting to objectively answer the question, “Is the cardiopulmonary capacity normal in primary school–aged children who have undergone cardiac surgery?” Apart from the motivation of the children, the availability of a suitable ergometer has a decisive role, which is why we modified our bicycle ergometer. With this ergometer we exercised 87 children aged 4 to 11 years who had undergone cardiac surgery with a standardized protocol. The underlying heart defects were confined to 4 main groups, of which 2 are primarily assigned as noncyanotic cardiac defects (CoA, ASD or VSD) and 2 as cyanotic cardiac defects (TOF or PAcVSD). These cardiac defects account for 50% to 75% of all congenital cardiac defects.

The ergometry data for 98 healthy children who had been examined following the same protocol were used as control. In this group we saw no difference between boys and girls for load duration, $P_{\text{max}}$, and TW. This confirms previous research in children aged 5 to 12 years.

For the anthropometric data, we found no significant difference between the healthy children and those who had undergone heart surgery (Table 1). This indicates in the first instance that children in this age group who had undergone surgery, even those with TOF or PAcVSD, showed no growth deficit when they started school. However, analysis of the ergometry data revealed clear differences between primary school–aged children with noncyanotic cardiac defects and those with cyanotic cardiac defects. The load duration, $P_{\text{max}}$, $P_{\text{max}}/\text{kg}$, and TW in the children in the CoA and ASD or VSD groups did not differ in the mean value from the corresponding parameters in healthy children, whereas all ergonomic parameters in the children in the TOF and PAcVSD groups were significantly below the norm. This means that children who have undergone surgery to correct complex cardiac defects already exhibit measurable deficits in TW duration and maximal exercise capacity at primary school age. Such deficits are also seen in adolescents and young adults with these cardiac defects. One can, therefore, assume that the reduced deficiency seen at the primary school level will persist over the long-term.

Our data show that patients operated on to correct simple cardiac defects have, on average, normal exercise capacity in childhood. However, this is diminished in patients who undergo surgery to correct complex cardiac defects, and can be explained as follows.

First, after corrective surgery in patients with TOF and PAcVSD, one must usually expect residual defects, such as...
as pulmonary valve stenosis or insufficiency. These result in chronic RV overload. In addition, owing to surgical intervention, right bundle branch block develops in most of these patients. Assessing the function of the RV is challenging because of its complex anatomy. Difficulties are compounded by irregularities in the ventricular cavities and abnormalities in wall motion in these patients after cardiac surgery. Thus, the complex shape of the RV makes quantification difficult.19 In our cohort we did not routinely measure the diameter of the RV in all patients, but the well-known relationship between RV dilatation and QRS interval (right bundle branch block)20,21 allows us to conclude that the reduced exercise capacity is also affected by RV dysfunction.

Table 3. Comparison Between Healthy Children and Patients With Congenital Heart Defects After Corrective Surgery With Regard to Cardiopulmonary Exercise Testing

<table>
<thead>
<tr>
<th>Variable</th>
<th>TOF</th>
<th>PAcVSD</th>
<th>CoA</th>
<th>ASD/VSD</th>
<th>Healthy Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Load duration, min</td>
<td>8.5† (7.4-9.3)</td>
<td>7.9† (5.7-10)</td>
<td>9.5 (8.3-10.6)</td>
<td>9.2 (8.1-10.2)</td>
<td>10 (9.5-10.5)</td>
</tr>
<tr>
<td>Pmax, W</td>
<td>63† (55-71)</td>
<td>54† (40-67)</td>
<td>72 (66-79)</td>
<td>70 (64-76)</td>
<td>78 (74-81)</td>
</tr>
<tr>
<td>Pmax/kg, W/kg</td>
<td>2.3§ (2.2-2.4)</td>
<td>1.9§ (1.7-2.2)</td>
<td>2.8 (2.7-2.9)</td>
<td>2.8 (2.7-2.9)</td>
<td>2.8 (2.7-2.9)</td>
</tr>
<tr>
<td>Total work, W/min</td>
<td>392† (310-474)</td>
<td>338† (171-490)</td>
<td>480 (388-573)</td>
<td>448 (368-529)</td>
<td>533 (490-577)</td>
</tr>
</tbody>
</table>

Heart rate

Before ergometry, per min

TOF 83 (78-87)  
PAcVSD 93 (34-103)  
CoA 86 (82-90)  
ASD/VSD 85 (80-89)  
Healthy Children 91 (89-94)  

After ergometry, per min

TOF 164§ (156-171)  
PAcVSD 166§ (154-177)  
CoA 173 (167-180)  
ASD/VSD 176 (171-182)  
Healthy Children 181 (178-184)  

Blood pressure, mm Hg (range)

Systolic

Before ergometry

TOF 102 (96-107)  
PAcVSD 102 (96-108)  
CoA 95 (91-98)  
ASD/VSD 101 (94-107)  
Healthy Children 104 (102-107)  

After ergometry

TOF 132 (124-141)  
PAcVSD 126 (114-137)  
CoA 109 (103-115)  
ASD/VSD 129 (121-137)  
Healthy Children 123 (120-128)  

Diastolic

Before ergometry

TOF 59 (55-62)  
PAcVSD 59 (54-64)  
CoA 56 (53-58)  
ASD/VSD 59 (56-63)  
Healthy Children 60 (58-63)  

After ergometry

TOF 68 (63-73)  
PAcVSD 66 (62-70)  
CoA 61 (57-65)  
ASD/VSD 66 (61-71)  
Healthy Children 65 (63-68)  

Abbreviations: ASD, atrial septal defect; CI, confidence interval; CoA, coarctation of the aorta; PAcVSD, pulmonary atresia with VSD; Pmax, maximal workload; Pmax/kg, normalized maximal performance; TOF, tetralogy of Fallot; VSD, ventricular septal defect.

*Significant correlation shown in boldface type. Data are given as mean percentage (95% CI).
†P<.01.
‡P<.005.
§P<.001.

as pulmonary valve stenosis or insufficiency. These result in chronic RV overload. In addition, owing to surgical intervention, right bundle branch block develops in most of these patients. Assessing the function of the RV is challenging because of its complex anatomy. Difficulties are compounded by irregularities in the ventricular cavities and abnormalities in wall motion in these patients after cardiac surgery. Thus, the complex shape of the RV makes quantification difficult.19 In our cohort we did not routinely measure the diameter of the RV in all patients, but the well-known relationship between RV dilatation and QRS interval (right bundle branch block)20,21 allows us to conclude that the reduced exercise capacity is also affected by RV dysfunction.

Second, significantly reduced maximal heart rates in patients with complex heart defects may indicate that chronotropic incompetence, failing to reach the age-adjusted maximum heart rate, is a major problem in the pathophysiology of reduced exercise capacity in this patient group. It has been shown that chronotropic incompetence may be due to postsynaptic desensitization of the β-adrenergic receptor pathway during chronic heart failure3 and may be related to abnormalities in autonomic regulation.22-24 Whether this mechanism already begins in early childhood requires further investigation.

Third, in about 25% of patients palliation by means of an aortopulmonary shunt had been initially performed, leading to changes in the pulmonary vascular system, which in most of these children is, in any case, pathologically altered. It is also the case that repeat operations are frequently necessary in this group, which can, in turn, negatively affect exercise capacity.

Figure 3. Normalized maximal performance (Pmax/kg) in children after corrective operation of aortic coarctation (CoA), ventricular septal defect (VSD), and atrial septal defect (ASD). Gray area shows the normalized maximal performance (Pmax/kg) in healthy children. BSA indicates body surface area.

Figure 4. Normalized maximal performance (Pmax/kg) in children after corrective operation of tetralogy of Fallot (TOF) and pulmonary atresia with ventricular septal defect (PAcVSD). Gray area shows the Pmax/kg in healthy children. BSA indicates body surface area.
The type of cardiac defect and subsequent surgical correction have an effect on the exercise capacity in these patients, even at the primary school level. Children with complex and primary cyanotic heart defects show verifiable deficits in exercise capacity. This loss of efficiency is, on the one hand, a consequence of residual defects after cardiac surgery (eg, right ventricular) and, on the other hand, due to a more or less already damaged pulmonary vascular system (eg, previous aortopulmonary shunt procedure) and chronotropic incompetence. However, after corrective surgery, children with simple and primary noncyanotic cardiac defects are as physically fit as their peers. Further observation and studies are necessary to determine whether this trend continues into adulthood.

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