Paediatric Exercise Science and Medicine

SECOND EDITION

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OXFORD

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OXFORD
UNIVERSITY PRESS

Exercise testing in congenital heart disease

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Introduction

Exercise testing in adult cardiac patients has mainly focused on ischaemic heart disease. The results of exercise testing with ECG monitoring are often helpful in diagnosing the presence of significant coronary artery disease. In children with heart disease, the type of pathology is different. Ischaemic heart disease is very rare. The majority of the patients present with congenital heart defects, which affect exercise capacity. In patients with congenital heart disease, exercise tests are frequently performed to measure exercise function or to assess abnormalities of cardiac rhythm. The risk of exercise testing is very low in the paediatric age group.¹

To perform cardiopulmonary exercise testing in children, the same types of ergometers can be used as has been reported in adults (bicycle ergometer, treadmill).^{2–6} Differences with adult exercise testing procedures² are adaptations of the ergometer in order to fit the size of the child, and modifications of the exercise protocols due to cooperation (motivation, anxiety).³ Since the motivation of young children to sustain incremental exercise testing is lower than in adults, the duration of the exercise testing procedure should be shorter than in adults. Several protocols have been recommended for cycle exercise testing.^{1,3} However, in children, especially in younger children, treadmill exercise testing is preferred, since younger children are more used to walking than to cycling. The most widely used protocol is the Bruce test, where the inclination and speed of the treadmill are simultaneously increased every 3 min, until exhaustion.⁴ Normal values have been reported for the paediatric age group.^{4,6} Other frequently used protocols are the Balke protocol⁷ or variants where the speed is a function of the age of the child ($4.8 \text{ km}\cdot\text{hour}^{-1}$ for children below 6 years of age and $5.6 \text{ km}\cdot\text{hour}^{-1}$ for children above that age).^{8,9}

Review of commonly used parameters to assess exercise performance and aerobic exercise function in the paediatric age group

In adult exercise physiology, aerobic exercise performance is traditionally assessed by determining the maximal oxygen uptake. This reflects the highest level of oxygen ($\dot{V}\text{O}_2 \text{ max}$), which does not further increase despite an increase in exercise intensity. In

paediatric exercise testing, the $\dot{V}\text{O}_2 \text{ max}$ test is also frequently assessed. However, although the measurement of maximal oxygen uptake is useful, since it gives information about the maximal exercise tolerance, this physiological definition is not always met in children. Only about half of the children are able to reach such a plateau after repeated exercise tests (see ref. 6 and Chapter 8). Many children are not motivated to exercise till that point of exhaustion.¹⁰ Therefore, other criteria should be used to confirm a maximal exercise test such as (i) a respiratory gas exchange ratio (VCO_2/VO_2) > 1.10 ; (ii) a peak heart rate, which is close to $200 \text{ beats}\cdot\text{min}^{-1}$; and (iii) the subjective appearance of exhaustion. However, it should be noted that in patients with congenital heart disease the maximal heart rate cannot be used as a criterion in patients with chronotropic limitation as frequently observed in congenital heart disease.¹⁰

Other parameters of maximal exercise function are the measurement of the maximal work rate ($\text{kgm}\cdot\text{min}^{-1}$),⁵ which can be indexed by expressing it in relation to body mass and the maximal endurance time on a treadmill, while performing the Bruce protocol. Although the latter test has been shown to correlate with $\dot{V}\text{O}_2 \text{ max}$ (indexed per kg body weight; $r = 0.88\text{--}0.92$),⁶ this measurement is strongly influenced by the motivation of the child and by the encouragement of the investigator. Maximal exercise capacity is of little relevance in daily life, while performing mild to moderate physical activity.

Because maximal exercise tests may have several drawbacks in the paediatric population, clinical investigators have tried to use submaximal exercise test procedures for application in children. In the past, the heart rate response to exercise has frequently been used to assess cardiovascular exercise performance.¹¹ The advantage of this measurement is its submaximal nature and minimal ergometric equipment. However, in patients with congenital heart disease several drawbacks exist, as many patients may show a relative bradycardia during exercise, which is not associated with a high value for $\dot{V}\text{O}_2 \text{ max}$, as should theoretically be expected. For example, in patients after total surgical repair of tetralogy of Fallot or in patients with cardiac preload problems (Senning Mustard or Fontan operation), a reduced value for heart rate during graded exercise has been found, which was not associated with a normal value for ventilatory anaerobic threshold¹² or $\dot{V}\text{O}_2 \text{ max}$.¹³ Therefore, the use of heart rate response to exercise in the assessment of cardiovascular

exercise performance can be misleading in patients with congenital heart disease and cannot be considered to be a valid determinant of aerobic fitness.³

A more sensitive assessment of aerobic exercise function can be obtained by an analysis of the gas exchange. Therefore, considerable attention has been focused on the determination of the ventilatory anaerobic threshold in children (see refs 14–17 and Chapter 6). This reflects the highest exercise intensity at which a disproportionate increase in $\dot{V}\text{CO}_2$ elimination ($\dot{V}\text{CO}_2$) is found relative to $\dot{V}\text{O}_2$.^{18,19} Although in adult subjects a concomitant disproportional increase is found between the excessive CO_2 elimination and the lactate accumulation or bicarbonate decrease in the blood, a lot of experimental conditions exist, where this relationship can be disturbed [e.g. in patients with McArdle's disease who have an inability to produce lactate²⁰ and after glycogen depletion²¹]. However, despite this scientific debate, this exercise level has shown to be a very useful and reproducible indicator of aerobic exercise function also in the paediatric age group.^{16,19} Moreover, the recent development of breath-by-breath analysis of gas exchange, with rapid responding gas analysers or mass spectrometers, allows precise and reproducible measurements of this parameter. More specifically in the paediatric age group, no ventilatory anaerobic threshold can be detected in about 10 % of the children.^{16,18} Normal reference values have been reported for European^{15,22} and North American children.^{14,16}

More recently, newer concepts have been developed to assess dynamic changes of respiratory gas exchange during exercise, in patients with congenital heart disease. The study of the steepness of the slope of $\dot{V}\text{CO}_2$ versus $\dot{V}\text{O}_2$ above the ventilatory anaerobic threshold has been found to be a very sensitive and reproducible index for the assessment of cardiovascular exercise function in this patient group (Fig. 31.1).

Furthermore, the use of time constants for the assessment of the initial response of $\dot{V}\text{O}_2$, $\dot{V}\text{CO}_2$ and pulmonary ventilation ($\dot{V}_E$) during constant rate exercise, together with the recovery (half-time measurement for $\dot{V}\text{O}_2$), have been found to be sensitive indicators for the evaluation of aerobic exercise function (see refs 23–25 and Chapter 22). Also the calculation of the normalized oxygen deficit, which reflects the oxygen debt at the onset of exercise, subtracted from the steady-state value, reached after 6 min of constant work rate exercise and expressed as a percentage of the total oxygen cost of a 6-min exercise test, has been found to be a useful parameter for the assessment of cardiovascular exercise function²⁶ in patients with poor exercise performance and when anaerobic threshold cannot be determined (Fig. 31.2).

Parameters of aerobic exercise function, such as the steepness of the slope of $\dot{V}\text{CO}_2$, time constants, and oxygen deficit and recovery kinetics, have the advantage that they all study the dynamic change of the cardiovascular response during constant work rate exercise. The evaluation of the non-steady-state phase of the cardiorespiratory response to exercise is much more relevant to activities during daily life than maximal exercise testing. Particularly, paediatric patients and normal children perform a lot of activities during non-steady-state exercise. The speed of the response of the cardiovascular system will affect exercise tolerance and consequently the ability to perform a subsequent bout of exercise.

However, when evaluating exercise tolerance in patients with congenital heart disease, it is recommended that each laboratory determines its own normal values, because geographical differences may influence the results of aerobic exercise performance. For example, in the classical studies of Astrand²⁷ on Swedish children,

Fig. 31.1 Typical response of CO_2 output ($\dot{V}\text{CO}_2$) versus oxygen uptake ($\dot{V}\text{O}_2$) during graded treadmill exercise in an 11-year-old boy after total repair for tetralogy of Fallot.

Data represent average values for breath-by-breath measurements of $\dot{V}\text{O}_2$ and $\dot{V}\text{CO}_2$ in 10-s intervals. Exercise intensity was increased until a heart rate of 170 beats·min⁻¹ was reached. S_1 is the calculated slope for increase of $\dot{V}\text{CO}_2$ versus $\dot{V}\text{O}_2$ from onset of exercise to ventilatory anaerobic threshold (VAT). S_2 is the slope between VAT and the respiratory compensation point. S_3 is the slope between VAT and exercise intensity reached at a heart rate of 170 beats·min⁻¹. Reproduced from Reybrouck *et al.*,¹⁹ with permission.

these subjects (both adults and children) always showed superior values for maximal aerobic power, which may be related to a more active lifestyle in the Scandinavian countries.

Assessment of habitual level of physical activity in patients with congenital heart disease

Different methods for assessing the daily level of physical activity in paediatric patients have been applied. These vary from history taking, questionnaires and interviews for more direct observations, measurements of heart rate, $\dot{V}\text{O}_2$, and even long-term video recording.²⁸ More sophisticated methods are pedometers and actometers, which record the number of steps and vertical displacements of the body. Each of these methods has its limitations. For recall questionnaires, a low reliability and low objectivity have been experienced. When patients or parents were asked to classify themselves or their children as active or non-active, in about 30% of the cases a misclassification was found when the data were correlated with objective measurements of maximal oxygen uptake.^{29,30} The same holds true for a self-keeping log. Even more sophisticated methods do not give a precise estimation of energy expenditure. For example, the measurement of $\dot{V}\text{O}_2$ is cumbersome, video recording is limited to a specific space and hard to quantify. Holter and ECG recordings are complex devices and require individual calibration. Furthermore, pedometers do not give information about vertical

Fig. 31.2 Normalized oxygen deficit. Data for oxygen uptake were first collected during a 3-min rest period, followed by 6-min treadmill exercise at a speed of 5 km.h^{-1} and inclination of 4% and finally a recovery period for 3 min. The normalized oxygen deficit was calculated at onset of exercise as the difference between the single breath values and the steady-state value. Each point represents a five breaths moving average value for oxygen uptake, expressed as $\text{mL.kg}^{-1}.\text{min}^{-1}$. Data were cumulated and expressed as a percentage of the total oxygen cost for 6 min of exercise. A typical example is presented for (a) a normal child with an oxygen deficit of 8% and (b) a patient with Fontan circulation with an oxygen deficit of 14%.

displacement and do not give information about the nature of the activity (e.g. walking or running). Therefore, these devices may underestimate at high activity levels. A review of advantages and disadvantages of different systems has been published by Bar-Or²⁸ (see also refs 31,32 and Chapter 10).

However, despite these limitations and lack of accuracy of questionnaires, we found that the use of a standardized questionnaire, with questions about school sport, leisure time physical activity, formal sports participation during leisure time was reproducible in young children (9–13 years of age; $r = 0.98$). Furthermore, these questionnaires were able to show reduced levels of physical activity in patients with congenital heart disease and subnormal exercise tolerance.²⁹

In conclusion, although the use of a standardized questionnaire to assess the daily level of physical activity is superior to the classical medical history taking, it cannot replace the objective assessment of aerobic exercise function by performing an exercise test. It should rather be considered as a complementary information which is useful in the interpretation of the exercise results.

Cardiorespiratory response to exercise in specific congenital heart defects

Left-to-right shunts

Atrial septal defect

Children with atrial septal defect (ASD) usually have a normal or near normal exercise capacity. These children can attain normal values for $\dot{V}O_2$ max or near normal values.^{1,33} A number of haemodynamic abnormalities to exercise have been documented. The increase of cardiac output during exercise may be smaller than normal and maximal heart rate response has been found to be lower than normal. In those who underwent surgical closure of the ASD, the age at the time of surgery has been shown to influence exercise performance. In a consecutive series of 50 patients with ASD or ventricular septum defect, evaluated in our laboratory, the ventilatory anaerobic threshold (as an estimate of aerobic exercise performance) was at the lower limit of normal ($89 \pm 14.4\%$ of normal).⁹ When studying the exercise response in children who underwent surgical closure of an ASD, a normal value was found in children who underwent surgery before 5 years of age, whereas a significantly lower value was found in children operated after that age.³⁵

In general, abnormalities detected in children either with unoperated or surgically closed ASD are usually minor and do not result in major limitations in exercise performance. Unless arrhythmia is a complication, these children should be encouraged to perform physical exercise and participate in all sports at all levels. Exercise testing is generally indicated if symptoms of arrhythmia or dyspnoea on exercise are reported.

Ventricular septal defect

A small ventricular septal defect (VSD) will transmit only a small amount of blood from the left to the right side of the heart. Also during exercise the shunt will remain small. Haemodynamic studies in this patient group showed that during graded exercise, patients with a VSD had a higher pulmonary circulation than systemic circulation, as could be expected. However, the relative shunt fraction decreased with increased exercise intensity.³⁶ Subnormal values for cardiac output were found in this patient group. Exercise performance, assessed by the measurement of maximal endurance time, maximal work rate on the cycle ergometer, and maximal heart rate were slightly reduced, when compared to normal controls ($90.8 \pm 1.6\%$).³⁷ Studies during submaximal exercise testing, using gas exchange measurements, showed suboptimal values for ventilatory anaerobic threshold in a consecutive series of 43 patients with an unoperated VSD, evaluated in our laboratory. This value averaged $90 \pm 15.3\%$ of normal and was below the lower limit of 95% confidence interval.⁹ In this patient group, the decreased level of exercise capacity was correlated with a decreased level of habitual physical activity. In another series of patients, studied after surgical closure of a VSD, a significantly lower value was found for the ventilatory threshold ($86 \pm 12\%$ of the normal value), which remains stable at re-evaluation about 3 years later on average (see Fig. 31.3 and ref 34). Finally, in a group of 18 patients who underwent surgical closure of a large VSD with pulmonary hypertension, before 1 year of life, the value for aerobic exercise performance was at the lower limit of normal ($92 \pm 17\%$ of normal).³⁸ In a retrospective study about quality of life, Meijboom *et al.*³⁹ reported a normal exercise capacity in 84% of the patient group who underwent surgical closure of a VSD ($N = 109$). This shows that surgical

Fig. 31.3 Serial evaluation of cardiovascular exercise performance in children with congenital heart disease. Reproduced from Tuley,²⁵ with permission.

correction of a congenital heart defect early in life can normalize the child's exercise performance.

Patent ductus arteriosus

Similarly in this group of patients, results of exercise testing will generally be normal if the size of the shunt is moderate or small. These subjects will ordinarily be asymptomatic. In most conditions, these defects will be closed surgically or percutaneously, at an age when exercise testing is not feasible. Exercise testing will add little to the routine clinical evaluation of these patients.¹

Valvular heart lesions

Aortic stenosis

Exercise testing in patients with aortic stenosis (AS) may show ST segment changes on the ECG, reflecting ischaemia, a drop in blood pressure or an inadequate rise in blood pressure with increasing exercise intensity, and eventually arrhythmia during exercise testing. The major haemodynamic determinant of ST segment changes during exercise is the inadequate oxygen delivery to the left ventricle. After surgical relief of the gradient, improvement of ST segment changes on the ECG during exercise has been reported.¹⁰ A critical AS can be identified by clinical findings and confirmed by echo-Doppler examination and eventually by cardiac catheterization.

During exercise testing, most of the patients show a reduced aerobic exercise performance,³⁴ which may be improved after surgery. This may be related both to the inability of the cardiac output to increase adequately during exercise and further also to the effect of a medically imposed restriction of heavy physical activity and competitive sports. Sudden cardiac death during exercise has been reported and has been ascribed to malignant arrhythmias. Fortunately, this is unlikely to happen in asymptomatic patients, with mild to moderate stenosis.³³ In our experience, it has never occurred in over 200 patients in follow-up with mild to moderate

AS (Doppler gradient < 60 mmHg). It allows them to perform up to moderate exercise, including recreational sports.

Pulmonary valve stenosis

Similar to AS, pulmonary valve stenosis results in a right ventricular overload. This may lead to a diminished pulmonary flow. In mild cases (PIG < 30 mmHg) normal or near normal values for ventilatory threshold have been found.³⁴

During exercise, the transvalvular pressure gradient in pulmonary stenosis (PS) may increase during graded exercise testing.¹ In mild cases (gradients < 30 mmHg), values for ventilatory anaerobic threshold have been found to be at the lower limit of normal.³⁴ In cases with moderate to severe PS, right ventricular pressures may rise considerably during exercise, which may limit exercise capacity.⁴⁰ In patients with mild to moderate PS (Doppler > 50 mmHg), balloon valvuloplasty (or very rarely surgery) will be performed. However, exercise performance may be limited in cases with severe pulmonary incompetence.⁴¹

Cyanotic heart disease

Tetralogy of Fallot

Postoperative children, who are felt to have good results (no residual VSD and a pressure gradient between right ventricle and pulmonary artery below 20 mmHg), are generally asymptomatic at rest. However, a variety of abnormalities may be brought out by intensive exercise.⁴² These include

- ♦ a high right ventricular pressure with values as high as 100 mmHg during maximal exercise, caused by a pressure gradient between right ventricle and pulmonary artery;
- ♦ a blunted increase in stroke volume and heart rate; and
- ♦ appearance of ventricular arrhythmia.

Despite these abnormalities, children who underwent total surgical repair for tetralogy of Fallot are usually well during daily

life. However, formal exercise testing has repeatedly shown subnormal values for maximal oxygen uptake and also for ventilatory anaerobic threshold in this patient group.^{13,19,43} Moreover, some individuals may reach normal values (100% normal or even higher than normal values). Furthermore after training, patients with this type of pathology can increase maximal work capacity by 25%.⁴⁴ When the adequacy of the oxygen transport during exercise in patients with tetralogy of Fallot repair was assessed by calculation of the slope of $\dot{V}O_2$ versus exercise intensity, reduced values have been found in patients after repair of tetralogy of Fallot.⁴⁵ This was associated with increased values for the physiological dead space ventilation during exercise or the slope of ventilation versus carbon dioxide output.^{46,47} This is mostly attributed to significant residual haemodynamic abnormalities, such as severe pulmonary regurgitation and right ventricular dysfunction.

Postoperative tetralogy of Fallot patients may have ventricular ectopy during exercise (exercise-induced arrhythmia). Exercise-induced ventricular arrhythmias are mainly seen in patients with late repair and poor right ventricular function.³³ Patients with important residual haemodynamic abnormalities such as those mentioned above are at risk for cardiovascular events.^{41,48}

Transposition of the great arteries

In simple transposition of the great arteries (TGA), the aorta arises from the right ventricle, while the pulmonary artery originates from the left ventricle. This results in severe cyanosis, as desaturated systemic venous blood is pumped into the systemic circulation, while the pulmonary venous return is pumped via left atrium and left ventricle into the lungs. Since this blood is already fully oxygenated, no more oxygen will be added to the blood.

The surgical approach to TGA from the late 1960s to the early 1980s involved baffling or rerouting the systemic venous return (from the superior and inferior vena cava) to the mitral valve and left ventricle (Mustard or Senning procedure). The desaturated blood will then be pumped through the pulmonary artery (arising from the left ventricle) to the lungs. The interatrial septum was removed and the pulmonary venous return (arterial blood) was drained to the right ventricle, and pumped into the aorta (arising from the right ventricle). In these atrial switch procedures, the right ventricle functions as the systemic ventricle. However, two major problems exist: (i) there have been extensive atrial incisions and (ii) the right ventricle is left as the systemic ventricle. Long-term problems include sinus node dysfunction, slow junctional rhythms, supraventricular tachycardias, depressed right ventricular function, right ventricular failure, and tricuspid valve insufficiency. Furthermore, because of extensive atrial surgery, the reservoir function of the atrium is seriously compromised. During ventricular filling, blood cannot 'just drop' into the ventricle, but has to come along a much longer way. Any tachycardia will shorten diastole and may critically impair ventricular filling, especially if an obstruction to the pathway is present. This may result in syncope or sudden death.⁴⁹

Exercise testing following the atrial switch procedures has shown a variety of abnormalities even in patients who were asymptomatic at rest. Decreased endurance times, decreased $\dot{V}O_2$ max, subnormal $\dot{V}O_2$ during submaximal exercise and subnormal values for ventilatory anaerobic threshold have been reported.^{10,19,50} In addition, a variety of arrhythmias have been documented during exercise testing (junctional rhythm, premature atrial contractions, and premature ventricular contractions). In addition to these abnormal-

ities, potentially detrimental effects of vigorous training in these patients have been reported. It is unknown whether the right ventricle can dilate and hypertrophy to endurance training as does the left ventricle in highly trained young athletes.⁴² For these reasons, high intensive isometric exercise or high intensive dynamic exercise and competitive sports are discouraged.

Nowadays, the arterial switch operation is the current surgical technique for TGA in the majority of the cases. Normal or near normal values for exercise performance and normal ST on ECG have been reported in this patient group.^{51,52} With earlier techniques coronary problems early and late after surgery were not exceptional. Improved surgical techniques appear to have resolved these problems. However, the length of the follow-up with this procedure is still limited.¹

Fontan circulation

In tricuspid atresia, there is a congenital absence of the tricuspid valve. In a Fontan circulation, the caval veins are currently connected directly to the pulmonary arteries, bypassing the right heart. This means that there is no effective right heart pump. Although the survival and also preoperative exercise performance of these patients improves dramatically, most of these subjects still have a limited exercise tolerance.^{40-42,53-56} The circulatory output after Fontan is primordially regulated by the pulmonary vasculature, which limits ventricular preload. The ventricle usually 'will pump whatever it gets'. The heart rate response is usually blunted, but appropriate for the level of ventricular preload. A very fast heart rate with a limited preload would result in a decreased stroke volume with hypotension, syncope, and eventually death. The lower oxygen saturation in the arterial blood may be due to residual venous or atrial shunting.⁵⁶ In our series exercise performance estimated by ventilatory threshold amounted from 50% to 70%.⁵⁵

Rhythm disturbances and conduction defects

Congenital complete atrioventricular block

In congenital complete atrioventricular block, the atrial rate increases normally, during exercise, but ventricular rate does not accelerate adequately. In some cases, these patients may develop dizziness and syncope.⁵⁷ Exercise testing in these patients shows subnormal values for $\dot{V}O_2$ max or ventilatory anaerobic threshold and even for the increase of oxygen uptake versus exercise intensity.⁵⁸ This results from the lack of acceleration of heart rate during exercise, one of the major components to increase cardiac output and consequently oxygen delivery to the exercising tissues. In some cases with severe bradycardia and syncope a pacemaker is inserted. It is obvious that these children should avoid competitive sports and physical activities with a danger of body collision in order to avoid damage to the wiring system that connects the pacemaker with the heart.

In the paediatric population, the frequency and significance of arrhythmia differs from adults.⁵⁷ As a general rule, the assessment of cardiac arrhythmia during exercise is useful in the management of these patients. If arrhythmia disappears with increased exercise intensity, the prognosis of this type of arrhythmia is usually benign.

Congenital complete atrioventricular block after surgery

The anatomic structure of the sinus node is vulnerable to damage following cardiac surgery. Damage of the sinus node has been

observed after surgical procedures that require extensive manipulations and sutures in the atria. Specific defects include D-TGA, repaired by atrial baffling procedures.⁵⁹ However, fortunately surgically acquired complete atrioventricular block, despite extensive surgery in the atria as for D-TGA is relatively uncommon. In surgically acquired atrioventricular block there is usually no escape rhythm. In this case safety pacing with redundant leads is recommended. Furthermore, extra caution is warranted not to damage the pacing system with external forces. Pacemaker technology when applied in young patients has some limitations, because the upper limit of a DDD-R pacemaker (dual chamber, rate responsive: DDD-R) is often too low for these youngsters, to achieve a physiological normal value for heart rate during exercise.⁵⁹

Natural evolution of aerobic exercise performance and daily level of physical activity in patients with congenital heart disease

To study the natural evolution of aerobic exercise performance during medium term follow-up in patients with congenital heart disease, exercise performance tests were compared in patients who underwent exercise testing at least twice with a time interval of about 3 years. Between 1980 and 1992, at our department of Paediatric Cardiology, 1982 exercise tests were performed. In 79 patients from this database,³⁴ exercise tests were performed at least twice in the same patient, with satisfactory respiratory gas exchange measurements on a breath-by-breath basis and a time interval of at least 2 years. These patients were divided into six subgroups. Three groups were studied for a non-operated congenital heart defect. Fourteen patients were followed for a VSD, which did not require surgical closure. Twelve patients were followed for a mild PS, with a gradient of less than 41 mmHg (average 17 ± 11 mmHg), and 12 patients with a mild AS, with a mean gradient of 36 ± 17 mmHg. Furthermore, three groups of patients who underwent surgical repair of a congenital heart defect were also studied twice. Sixteen patients who underwent total correction for tetralogy of Fallot (TF-PO) were studied, 13 patients who underwent surgical closure of a VSD (VSD-PO) and finally 12 patients with a Fontan circulation for tricuspid atresia were studied 1.9 ± 1.1 years after the Fontan operation.

The results of this study showed that at the initial evaluation all patients were in class I of the New York Heart Association. Aerobic exercise performance, assessed by the determination of the ventilatory anaerobic threshold, was at the lower limit of the normal mean value or below (Fig. 31.3). Significant differences for aerobic exercise performance were found between the different pathologies. The lowest values were found in the patients with the Fontan circulation and the highest values in patients with a VSD.

At reassessment, about 3 years later, all patients remained in NYHA class I, except for two patients with a Fontan circulation, who belonged to class II and III at re-evaluation. For patients in whom no medical restriction of physical activity was imposed (VSD, PS, and VSD-PO), no significant change was found for the value of ventilatory anaerobic threshold (expressed as a percentage of the mean value obtained in a pool of normal controls of the same age and gender) (Fig. 31.3). At variance, in patients with AS, with a medically imposed restriction of heavy physical exercise and

competitive sports, a decrease was found for aerobic exercise performance, of about 8%. In patients with surgical repair of tetralogy of Fallot, a decrease in the value of ventilatory anaerobic threshold of about 9% was found over the same time interval, which was related to residual haemodynamic lesions, such as pulmonary valve incompetence.⁴³ Finally also in the patients with a Fontan circulation, a significant decrease in aerobic exercise performance was found over this time interval. Similarly, on reassessment the lowest values were found in this patient group. This was related to the fact that these patients were unable to perform intensive physical exercise during daily life activities and also to residual haemodynamic dysfunction.

The daily level of physical activity, assessed by a standardized questionnaire, was significantly lower both at the first and second evaluation in patients with AS, surgical repair of tetralogy of Fallot and Fontan repair. These subnormal values for the daily physical activity level were associated with a significant decrease of aerobic exercise function at reassessment. In the other patient groups (VSD, PS, VSD-PO), no significant change was found for aerobic exercise performance between the two assessments (Fig. 31.3).

These data show the combined effect of residual haemodynamic lesions and hypoactivity on the evaluation of aerobic exercise performance in these groups of patients. In children and adolescents with AS significantly lower values for aerobic exercise performance were found both at first and second assessment. Similar data have been reported by Driscoll *et al.*³⁷ In this group of patients heavy physical exercise and competitive sports are discouraged because of the risk of ischaemia and arrhythmia. In the other patient groups residual haemodynamic dysfunction may impede the child to perform aerobic exercise of heavy intensity, which is necessary for the normal development of the oxidative metabolism. In these patient groups (Fontan and TF-PO), subnormal values for increase in $\dot{V}O_2$ during graded exercise have also been observed.^{19,47,55}

The results of these studies show that the suboptimal aerobic exercise performance in children and adolescents with congenital heart disease is to some extent attributable to residual haemodynamic lesions after corrective surgery of the defect and also to some degree of hypoactivity, which results from overprotection of the parents and the environment. However, in some patients there may also be an increase in the severity of the disease, which may impede the individual to perform the same amount of physical exercise as healthy peers. Therefore, except for some cases with the progression of the severity of the disease and medically imposed restriction of intensive dynamic or static physical exercise, children and adolescents with congenital heart disease and their parents should be strongly encouraged to be more active and to prevent the deleterious effect of physical deconditioning.

Exercise recommendations and rehabilitation of patients with congenital heart disease

Nowadays most children with congenital heart disease, both before and after surgery, are encouraged to be fully active and to participate in all recreational sport activities. These recommendations are based on clinical findings, which have shown that physical exercise in children with congenital heart disease has beneficial effects on the physical, psychological, and social levels both for the children

and also for the parents. In the majority of the cases, these children do not need to participate in a formal rehabilitation programme, but they should be encouraged to participate in recreational physical activities in leisure time and at school. Even after corrective surgery, a formal rehabilitation programme is mostly restricted to the hospitalization period and consists mainly of chest physiotherapy (breathing exercises) and early mobilization. As soon as the children are discharged from the hospital, they are encouraged to resume their normal physical activities at home.

In the majority of the cases of congenital heart disease, there are only a few contraindications for physical exercise both for the non-operated cardiac defects and for the operated ones.⁴¹ The final decision to allow the child with congenital heart disease to participate in physical exercise should always be based on a full cardiological examination.

A few controlled exercise studies in patients with congenital heart disease have shown that maximal exercise capacity can be improved following a period of physical training.^{44,61} However, $\dot{V}O_2$ max was not improved in all subjects. The improvement of maximal exercise performance (expressed in Watts or $\text{kgm}\cdot\text{min}^{-1}$, assessed during cycle ergometry) without an increase in $\dot{V}O_2$ max represents an improved mechanical efficiency during exercise. This

may be beneficial for the patients, since the same level of exercise will be perceived as easier to perform and will induce less dyspnoea. Furthermore, especially in young children, the measurement of $\dot{V}O_2$ max is often difficult as it depends on the motivation of the child. In fact, a plateau in $\dot{V}O_2$ with increasing exercise intensity, which is a prerequisite for a true $\dot{V}O_2$ max is difficult to obtain in young children⁶² (and see chapter 8).

Cumulative medical experience has shown that the potential risk of physical exercise in patients with congenital heart disease is very low.⁴¹ In fact, only a few heart defects have been associated with sudden cardiac death, during sports participation. These include mainly hypertrophic cardiomyopathy, severe AS, congenital anomalies of the coronary arteries, Marfan's syndrome, and myocarditis. Fortunately, these anomalies represent only a small percentage of the total number of congenital heart defects.

Since children and especially children who perform competitive sports may be exposed to high levels of physiological stress a classification of sports has been made by the American College of Cardiology and American College of Sports Medicine⁶³ (Table 31.1). This table classifies sport activities according to the type of exercise (dynamic vs. static) and according to the intensity of exercise, which reflects the cardiovascular stress. This classification can be used for

Table 31.1 Classification of sports

	(A) Low dynamic	(B) Moderate dynamic	(C) High dynamic
(I) Low static	Bowling Cricket Golf Rifery	Fencing Table tennis Tennis (doubles) Volleyball Baseball*/softball*	Badminton Race walking Running (marathon) Cross-country skiing (classic) Squash*
(II) Moderate static	Auto racing*** Diving** Equestrian*** Motorcycling*** Gymnastics* Karate/Judo* Sailing Archery	Field events (jumping) Figure skating Lacrosse Running (sprint)	Basketball* Biathlon Ice hockey* Field hockey* Rugby* Soccer* Cross-country skiing (skating) Running (mid/long) Swimming Tennis (single) Team handball*
(III) High static	Bobsledding*** Field events (throwing) Luge*** Rock climbing*** Waterskiing*** Weight lifting* Windsurfing***	Body building* Downhill skiing*** Wrestling* Snow boarding***	Boxing* Canoeing, Kayaking Cycling*** Decathlon Rowing Speed skating Triathlon***

Adapted and modified after Mitchell *et al.*⁶⁴

*Danger of bodily collision.

**Increased risk if syncope occurs.

counselling children and adolescents with congenital heart disease. An overview of the guidelines for sport participation in specific congenital heart defects, recommended by the Task Force of the European Society of Cardiology is presented in Table 31.2.

A restriction of heavy physical exercise and competitive sports is imposed in moderate to severe AS, in left-to-right shunts with pulmonary hypertension, hypertrophic cardiomyopathy, pulmonary hypertension, and arrhythmia which worsens during exercise. As a general rule, cardiopulmonary exercise testing is advised in children with congenital heart defects, before sport participation is allowed.

Summary

- ♦ As the majority of the patients with congenital heart disease belong to the paediatric age group, exercise testing equipment and exercise protocols have to be adapted for children.

Functional performance should be assessed by performing exercise testing with measurement of gas exchange. Nowadays, new concepts for exercise testing in the paediatric age group are analysis of gas exchange during the non-steady state of exercise and determination of the kinetics of gas exchange during the recovery phase of exercise.

- ♦ In some groups of patients with congenital heart disease, sub-optimal values have been found for aerobic exercise capacity, which can be ascribed to haemodynamic dysfunction or residual haemodynamic lesions after surgery (e.g. in TGA, tetralogy of Fallot and Fontan repair for univentricular heart). In other types of pathologies medically imposed restriction of intensive physical exercise or competitive sports may determine to some extent a subnormal value of exercise performance.
- ♦ Finally in some other types of congenital heart disease without overt haemodynamic dysfunction (e.g. VSD or ASD, with nor-

Table 31.2 Recommendations for sport participation in congenital heart diseases

Lesion	Recommendation
ASD (closed or non-significant or PFO)	No restrictions Scuba diving should be avoided in those with remaining shunt, due to the risk of paradoxical embolism
VSD (closed or non-significant)	No restrictions
PDA (closed or non-significant)	No restrictions
AVSD (successfully repaired)	No restrictions
Moderate MVR	Low to moderate dynamic and static sports
PAPVC/TAPVC (successfully repaired)	No restrictions
Pulmonary stenosis (mild)	No restrictions
Moderate	Low to moderate dynamic and static sports
Aortic stenosis (mild)	Low to moderate dynamic and static sports
Moderate	Low to moderate dynamic and static sports No competitive sport if left ventricular dysfunction of symptoms
CoA (successfully repaired)	No restrictions*
TOF (successfully repaired)	Low to moderate dynamic and static sports*
Residual disease	Low dynamic and sports*
TGA	
aso TGA (successfully repaired)	No restrictions
iar TGA, cc TGA	Low to moderate dynamic and static sports**
Ebstein anomaly	Low to moderate dynamic and static sports**
Univentricular hearts/Fontan circulation	Low to moderate dynamic and static sports** Low to moderate dynamic and static sports**
Eisenmenger's syndrome	Low dynamic sports**
Congenital coronary artery anomalies	No restrictions

For definitions, risk stratification, and follow-up see text. ASD = atrial septal defect; PFO = patent foramen ovale; VSD = ventricular septal defect; MVR = mitral valve regurgitation; PAPVC/TAPVC = partial or total anomalous pulmonary venous connection; CoA = coarctation of the aorta; TOF = tetralogy of Fallot; TGA = transposition of the great arteries; aso = arterial switch operation; iar = intra-atrial repair; cc = congenitally corrected.

Updated guidelines for exercise recommendation in patients with congenital heart disease (from Hirth *et al.*,³³ with permission).

*Those with conduit, interposed graft, or on anticoagulant drugs should avoid sports with the risk of bodily collision.

**No competitive sport.

mal pressures in the pulmonary circulation) a suboptimal value for aerobic exercise capacity is often related to overprotection of the parents or environment of the child. Therefore, except for some cases with medically imposed restriction of intensive physical exercise, most patients are encouraged to be fully active during leisure time and to participate at all types of physical exercise at school.

References

- Gibbons RJ, Balady GJ, Beasley JW, Bricker JT, Duvernoy WFC, Froelicher VF, Mark DB, Marwich TH, McCallister BD, Thompson PD, Winters WL, Yanowitz FG (1997). ACC/AHA Guidelines for Exercise Testing. A Report of the American College of Cardiology/American Heart Association Task force on Practice Guidelines (Committee on Exercise Testing). *J Am Coll Cardiol* **30**, 260–315.
- Pina IL, Balady GJ, Hanson M, Labovitz AJ, Madonna DW, Myers J (1995). Guidelines for Clinical Exercise Testing Laboratories. A Statement for Healthcare Professionals from the Committee on Exercise and Cardiac Rehabilitation, American Heart Association. *Circulation* **91**, 912–21.
- Washington RL, Bricker JT, Alpet BS, Daniels SR, Decelbaum RJ, Fisher EA, Gidding SS, Isabel-Jones J, Kavey REW, Marx GR, Strong BW, Teske DW, Wilmore JH, Winston M (1994). Guidelines for exercise testing in the pediatric age group. *Circulation* **90**, 2166–79.
- Cumming GR, Everatt D, Hastman L (1978). Bruce treadmill test in children: Normal values in a clinic population. *Am J Cardiol* **41**, 69–75.
- James FW, Blomqvist CG, Freed MD, Miller WW, Moller JH, Nugent EW, Riopel DA, Strong WB, Wessel HU (1982). Standards for exercise testing in the pediatric age group. *Circulation* **66**, 1377A–97A.
- Rowland TW (1993). Aerobic exercise testing protocols. In: Rowland TW (ed.), *Pediatric laboratory exercise testing. Clinical guidelines*, pp. 19–41. Human Kinetics, Champaign, IL.
- Riopel DA, Taylor AB, Hohn AR (1979). Blood pressure, heart rate, pressure-rate product and electrocardiographic changes in healthy children during treadmill exercise. *Am J Cardiol* **44**, 697–704.
- Chandramouli B, Ehmke DA, Lauer RM (1973). Exercise-induced electrocardiographic changes in children with congenital aortic stenosis. *J Pediatr* **87**, 725–30.
- Reybrouck T, Weymans M, Stijns H, van der Hauwaert LG (1986). Ventilatory anaerobic threshold for evaluating exercise performance in children with congenital left-to-right intracardiac shunt. *Pediatr Cardiol* **7**, 19–24.
- Pianosi PT, Driscoll DJ (2008). Exercise testing. In: Allen HE, Driscoll D, Shaddy RE, Feltes TF (eds.), *Moss' heart disease in infants, children, and adolescents*, pp. 81–94. Wolters Kluwer–Lippincott Williams and Wilkins, Philadelphia, PA.
- Adams FH, Linde LM, Niyake H (1961). The physical working capacity of normal school children. *Pediatrics* **28**, 55–64.
- Reybrouck T, Weymans M, Stijns H, van der Hauwaert LG (1986). Exercise testing after correction of tetralogy of Fallot: The fallacy of a reduced heart rate response. *Am Heart J* **112**, 998–1003.
- Wessel H, Paul M (1999). Exercise studies in tetralogy of Fallot: A review. *Pediatr Cardiol* **20**, 39–48.
- Cooper DM, Weiler-Ravell D, Whipp BJ, Wasserman K (1984). Aerobic parameters of exercise as a function of body size during growth in children. *J Appl Physiol* **56**, 628–34.
- Reybrouck T, Weymans M, Stijns H, Van der Hauwaert LG (1985). Ventilatory anaerobic threshold in healthy children. *Eur J Appl Physiol* **54**, 278–84.
- Washington RL, van Gundy JC, Cohen C, Sondheimer HM, Wolfe RR (1988). Normal aerobic and anaerobic exercise data for North American school-age children. *J Pediatr* **112**, 223–33.
- Washington RL (1999). Cardiorespiratory testing: Anaerobic threshold/respiratory threshold. *Pediatr Cardiol* **20**, 12–16.
- Wasserman K, Beaver WL, Whipp BJ (1990). Gas exchange threshold and the lactic acidosis (anaerobic) threshold. *Circulation* **81** (Suppl. 11), 14–30.
- Reybrouck T, Mertens L, Kalis N, Weymans M, Dumoulin M, Daenen W, Gewillig M (1996). Dynamics of respiratory gas exchange during exercise after correction of congenital heart disease. *J Appl Physiol* **80**, 458–63.
- Hagberg JM, Coyle EF, Carroll JE, Miller JM, Martin WH, Brooke MH (1982). Exercise hyperventilation in patients with Mc Ardle's disease. *J Appl Physiol* **52**, 991–4.
- Heigenhauser GJF, Sutton JR, Jones NL (1983). Effect of glycogen depletion on ventilatory response to exercise. *J Appl Physiol* **54**, 470–4.
- Schulze-Neick I, Wessel HU, Lange P (1992). Heart rate and oxygen uptake response to exercise in children with low peak exercise heart rate. *Eur J Pediatr* **151**, 160–6.
- Cooper DM, Kaplan MR, Baumgarten L, Weiler-Ravell D, Whipp BJ, Wasserman K (1987). Coupling of ventilation and CO₂ production during exercise in children. *Pediatr Res* **21**, 568–72.
- Zanconato S, Cooper DM, Armon Y (1991). Oxygen cost and oxygen uptake dynamics and recovery with 1 min of exercise in children and adults. *J Appl Physiol* **71**, 993–8.
- Turley KR (1997). Cardiovascular responses to exercise in children. *Sports Med* **24**, 241–57.
- Mertens L, Reybrouck T, Eyskens B, Daenen W, Gewillig M (2003). Slow kinetics of oxygen uptake in patients with a Fontan-type circulation. *Pediatr Exerc Sci* **15**, 146–55.
- Åstrand PO (1952). *Experimental studies of physical working capacity in relation to sex and age*. Munsgaard, Copenhagen.
- Bar-Or O (1982). Clinical implications of pediatric exercise physiology. *Ann Clin Res* **14** (Suppl. 34), 97–106.
- Rogers R, Reybrouck T, Weymans M, Dumoulin M, Van der Hauwaert L, Gewillig M (1994). Reliability of subjective estimates of exercise capacity after total repair of tetralogy of Fallot. *Acta Paediatr* **83**, 866–9.
- Barber G, Heise CT (1991). Subjective estimates of exercise ability: Comparison to objective measurements. *Pediatr Exerc Sci* **3**, 327–32.
- Saris WH, Binkhovst RA (1977). The use of pedometer and actometer in studying daily physical activity in man. Part II. Validity of pedometer and actometer in measuring the daily physical activity. *Eur J Appl Physiol* **37**, 229–35.
- Rowland T (1996). *Developmental exercise physiology*. Human Kinetics, Champaign, IL.
- Hirth A, Reybrouck T, Bjarnason-Wehrens B, Lawrenz W, Hoffman A (2006). Recommendations for participating in competitive and leisure sports in patients with congenital heart disease: A consensus document. *Eur J Cardiovasc Prev Rehab* **13**, 293–9.
- Reybrouck T, Rogers R, Weymans M, Dumoulin M, Vanhove M, Daenen W, Gewillig M (1995). Serial exercise testing in patients with congenital heart disease. *Eur J Pediatr* **154**, 801–6.
- Reybrouck T, Bisschop A, Dumoulin M, Van der Hauwaert LG (1991). Cardiorespiratory exercise capacity after surgical closure of atrial septal defect is influenced by the age at surgery. *Am Heart J* **122**, 1073–8.
- Bendien C, Bossina KK, Buurma AE, Gerding AM, Kuipers JRG, Landsman MLJ, Mook GA, Zijlstra WG (1984). Hemodynamic effects of dynamic exercise in children and adolescents with moderate-to-small ventricular septal defects. *Circulation* **70**, 929–34.
- Driscoll DJ, Wolfe RR, Gersony WM, Hayes CJ, Keane JF, Kidd L, O'Fallon M, Pieroni DR, Weidman WH (1993). Cardiorespiratory responses to exercise of patients with aortic stenosis, pulmonary stenosis, and ventricular septal defect. *Circulation* **87** (Suppl. 1), I-102–13.
- Reybrouck T, Mertens L, Schulze-Neick I, Austenat I, Eyskens B, Dumoulin M, Gewillig M (1998). Ventilatory inefficiency for carbon

- dioxide during exercise in patients with pulmonary hypertension. *Clin Physiol* **18**, 337–44.
39. Meijboom F, Szatmari A, Utens E, Deckers JW, Roelandt JRTC, Bos E, Hess J (1994). Long-term follow-up after surgical closure of ventricular septal defect in infancy and childhood. *J Am Coll Cardiol* **24**, 1358–64.
 40. Rowland TW (1995). Congenital obstructive and valvular heart disease. In: Goldberg B (ed.), *Sports and exercise for children with chronic health conditions*, pp. 225–36. Human Kinetics, Champaign, IL.
 41. Graham TW Jr, Bricker T, James FW, Strong WB (1994). Congenital heart disease. *Med Sci Sports Exerc* **26** (Suppl.), S246–53.
 42. Fahey JT (1995). Congenital heart disease-shunt lesions and cyanotic heart disease. In: Goldberg B (ed.), *Sports and exercise for children with chronic health conditions*, pp. 208–24. Human Kinetics, Champaign, IL.
 43. Rowe SA, Zakha KG, Manolio TA, Hornheffer PJ, Kidd L (1991). Lung function and pulmonary regurgitation limit exercise capacity in postoperative tetralogy of Fallot. *J Am Coll Cardiol* **17**, 461–6.
 44. Goldberg B, Fripp RR, Lister G, Loke J, Nicholas JA, Talner NS (1981). Effect of physical training on exercise performance of children following surgical repair of congenital heart disease. *Pediatrics* **68**, 691–9.
 45. Reybrouck T, Mertens L, Bruselle S (2000). Oxygen uptake versus exercise intensity: A new concept in assessing cardiovascular exercise function in patients with congenital heart disease. *Heart* **84**, 46–52.
 46. Gilljam T, Eriksson BD, Six R (1998). Cardiac output and pulmonary gas exchange at maximal exercise after atrial redirection for complete transposition. *Eur Heart J* **19**, 1035–84.
 47. Reybrouck T, Boshoff D, Vanhees L, Defoor J, Gewillig M (2004). Ventilatory response to exercise in patients after correction of cyanotic congenital heart disease: Relation with clinical outcome after surgery. *Heart* **90**, 215–16.
 48. Roos-Hesselink JW, Perlroth MG, McGhie J, Spitaels S (1995). Atrial arrhythmias in adults after repair of tetralogy of Fallot. Correlation with clinical, exercise, and echocardiographic findings. *Circulation* **91**, 2214–19.
 49. Gewillig M, Balaji S, Mertens B, Lesaffre E, Deanfield J (1991). Risk factors for arrhythmia and death after Mustard operation for simple transposition of the great arteries. *Circulation* **84**, 187–92.
 50. Reybrouck T, Gewillig M, Dumoulin M, van der Hauwaert LG (1993). Cardiorespiratory exercise performance after Senning operation for transposition of the great arteries. *Br Heart J* **70**, 175–9.
 51. Reybrouck T, Eyskens B, Mertens L, Defoor J, Daenen W, Gewillig M (2001). Cardiorespiratory exercise function after the atrial switch operation for transposition of the great arteries. *Eur Heart J* **22**, 1052–9.
 52. Massin M, Hövels-Gürich H, Däbritz S, Messmer B, von Bernuth G (1998). Results of the Bruce treadmill test in children after arterial switch operation for simple transposition of the great arteries. *Am J Cardiol* **81**, 56–60.
 53. Driscoll DJ, Danielson GK, Puga FJ, Schaff HF, Heise CT, Staats BA (1986). Exercise tolerance and cardiorespiratory response to exercise after Fontan operation for tricuspid atresia or functional single ventricle. *J Am Coll Cardiol* **7**, 1087–94.
 54. Gewillig M (1994). The Fontan circulation: Late functional results. *Semin Thoracic Cardiovasc Surg* **6**, 56–63.
 55. Mertens L, Rogers R, Reybrouck T, Dumoulin M, Vanhees L, Gewillig M (1996). Cardiopulmonary response to exercise after the Fontan operation—A cross sectional and longitudinal evaluation. *Cardiol Young* **6**, 136–42.
 56. Gewillig M, Lündstrom R, Bull C, Wyse RKH, Deanfield JE (1990). Exercise responses in patients with congenital heart disease after Fontan repair: Patterns and determinants of performance. *J Am Coll Cardiol* **15**, 1424–32.
 57. Park MK (1996). *Pediatric cardiology for practitioners* (3rd ed.). Mosby, St Louis, MO.
 58. Reybrouck T, Vanden Eynde B, Dumoulin M, Van der Hauwaert LG (1989). Cardiorespiratory response to exercise in congenital complete atrioventricular block. *Am J Cardiol* **64**, 896–9.
 59. Parridon SM (1997). Congenital heart disease: Cardiac performance and adaptations to exercise. *Pediatr Exerc Sci* **9**, 308–23.
 60. Reybrouck T, Rogers R, Weymans M, Dumoulin M, Vanhove M, Daenen W, Van der Hauwaert L (1995). Serial cardiorespiratory exercise testing in patients with congenital heart disease. *Eur J Pediatr* **154**, 801–6.
 61. Balfour IC, Drimmer AM, Nouri S, Pennington DG, Hemkens C, Harvey LL (1991). Pediatric cardiac rehabilitation. *Am J Dis Child* **145**, 627–30.
 62. Armstrong N, Welsman J, Winsley R (1996). Is peak $\dot{V}O_2$ a maximal index of children's aerobic fitness? *Int J Sports Med* **17**, 356–9.
 63. American College of Sports Medicine and American College of Cardiology (1994). Recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. *Med Sci Sports Exerc* **26** (Suppl.), 223–76.
 64. Mitchell JH, Haskell W, Snell P, Van Camp S (2005). Task Force 8: Classification of sports. *J Am Coll Cardiol* **45**, 1364–7.