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CURRENT OPINION
Congenital heart disease

Recommendations for participation in competitive sport in adolescent and adult athletes with Congenital Heart Disease (CHD): position statement of the Sports Cardiology & Exercise Section of the European Association of Preventive Cardiology (EAPC), the European Society of Cardiology (ESC) Working Group on Adult Congenital Heart Disease and the Sports Cardiology, Physical Activity and Prevention Working Group of the Association for European Paediatric and Congenital Cardiology (AEPC)

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Improved clinical care has led to an increase in the number of adults with congenital heart disease (CHD) engaging in leisure time and competitive sports activities. Although the benefits of exercise in patients with CHD are well established, there is a low but appreciable risk of exercise-related complications. Published exercise recommendations for individuals with CHD are predominantly centred on anatomic lesions, hampering an individualized approach to exercise advice in this heterogeneous population. This document presents an update of the recommendations for competitive sports participation in athletes with cardiovascular disease published by the Sports Cardiology & Exercise section of the European Association of Preventive Cardiology (EAPC) in 2005. It introduces an approach which is based on the assessment of haemodynamic, electrophysiological and functional parameters, rather than anatomic lesions. The recommendations provide a comprehensive assessment algorithm which allows for patient-specific assessment and risk stratification of athletes with CHD who wish to participate in competitive sports.

Keywords Congenital heart disease • Sports cardiology • Competitive sports • Participation recommendations

Introduction

Athletes with congenital heart disease (CHD) are likely to be encountered with increasing frequency. Improved clinical care of children with CHD has significantly increased survival to adulthood.[1] As a result, in developed countries, there are currently more adults than children with CHD, with one in 150 young adults affected and the number is expected to increase by 5% per year.[1,2] Whilst efforts to reduce long-term morbidity should focus on the actual congenital lesion,[3] similar to the general population there should also be emphasis on reducing the burden of cardiovascular risk factors, including a sedentary lifestyle.[4,5] The beneficial effects of exercise in individuals with CHD are well established as is the role of exercise as a preventive tool for developing acquired risk factors for cardiovascular disease.[6–9] The perceived risk of exercise-related complications, including adverse haemodynamic sequelae, accelerated disease progression, and sudden cardiac death (SCD), have often led to the adoption of a sedentary lifestyle amongst individuals with CHD. This is of particular importance when one considers that children with CHD are already more likely to be overweight because of physical inactivity compared with children without CHD.[10]

Target population

These recommendations apply to patients with CHD aged ≥16 years. Most children with CHD would have reached physical maturity by the age of 16 years. For the purpose of this document, competitive athletes are defined as individuals, who are engaged in exercise training on a regular basis in order to participate in regular official competitions. Official competition is defined as an organized team or individual sports event, at local, regional, national, or international level, that places a high premium on athletic excellence and achievement. A characteristic of competitive sports, regardless of the level of achievement, is the strong desire for participants to exert themselves physically to their limits and improve performance.[11,12]

Nature and aim of the recommendations

Published exercise recommendations for individuals with CHD are predominantly centred on individual anatomic lesions.[13–15] In 2013, the recognition of the challenges posed by the wide variation in the pathophysiology and functional status of patients with CHD led to the introduction of an individualized algorithm.[16] The recommendations for physical activity in adolescents and adults with CHD by the Section of Sports Cardiology & Exercise of the European Association of Preventive Cardiology Group (EAPC) and the European Society of Cardiology (ESC) Working on Adult CHD were based on haemodynamic and electrophysiological parameters, rather than focusing on specific anatomical defects, which do not correlate to exercise-associated risks. They provided a step-wise evaluation, which was based on the assessment of five parameters, namely (i) ventricular function, (ii) pulmonary artery pressure, (iii) aorta dimensions, (iv) presence of arrhythmias, and (v) arterial oxygen saturation at rest and during exercise. The type of exercise recommended was dictated by the static component, in accordance with the Mitchell *et al.*[17] classification, while the dynamic component was replaced by the relative intensity based on individual exercise performance on cardiopulmonary exercise testing (CPET). As such patients were provided with a wider, individually tailored choice of sports, in recognition of the fact that individualized exercise prescription, outside the context of competition, can accommodate different levels of intensity.

This document presents an update of the recommendations for competitive sports participation in athletes with cardiovascular disease published by the Sports Cardiology & Exercise section of the EAPC in 2005.[13] It moves away from specific anatomical defects and introduces an individualized assessment approach based on the principles of the 2013 recommendations for physical activity in CHD.[16] The recommendations, however, recognize that in the context of competitive sports, athletes will push themselves to their limits (maximum intensity) and as such the notion of relative intensity-based exercise prescription is not practical. In addition, the classification of

sports has been updated. Instead of the Mitchell *et al.*[17] classification, sports are classified according to the haemodynamic changes associated with exercise training and the long-term impact on cardiac morphology. In this regard, sport disciplines are divided in four major groups: skill, power, mixed, and endurance (*Figure 1*).[18] While this classification offers more clarity, we recognize that the four pre-defined categories may only partially reflect the nature of some sports, which may in fact cross categories (i.e. table tennis is a skill sport with significant dynamic component). Additional factors such as environmental conditions, psychological, and/or emotional demands should also be taken into consideration. Finally, following the accumulation of recent experience, some of the definitions in *Table 1* have been adjusted and some of the recommendations are more liberal compared to the document in 2013.[16]

In the absence of robust evidence, these recommendations are based on expert opinion (level of evidence C) and should not be discouraging of physicians to practice outside the remit of this document, based on their scientific and professional experience. In line with good clinical practice, the decision-making process should always include the athlete and respect her/his autonomy.

Assessment

Assessment should incorporate the general ESC guidelines for adult congenital heart disease (*Figure 2*).[20]

Step 1: History and physical examination

A comprehensive medical and surgical history is necessary with particular emphasis on the underlying primary CHD diagnosis, surgical and catheter interventions and their timings and non-cardiac comorbidities and prescribed and non-prescribed medication and nutritional supplements. The physician should take a detailed account of (i) cardiac symptoms such as exertional chest pain, dizziness, and syncope; (ii) functional status, with attention to exertional symptoms or changes in exercise capacity; (iii) the type of sport, volume and intensity of training and level competition, and (iv) the environment that

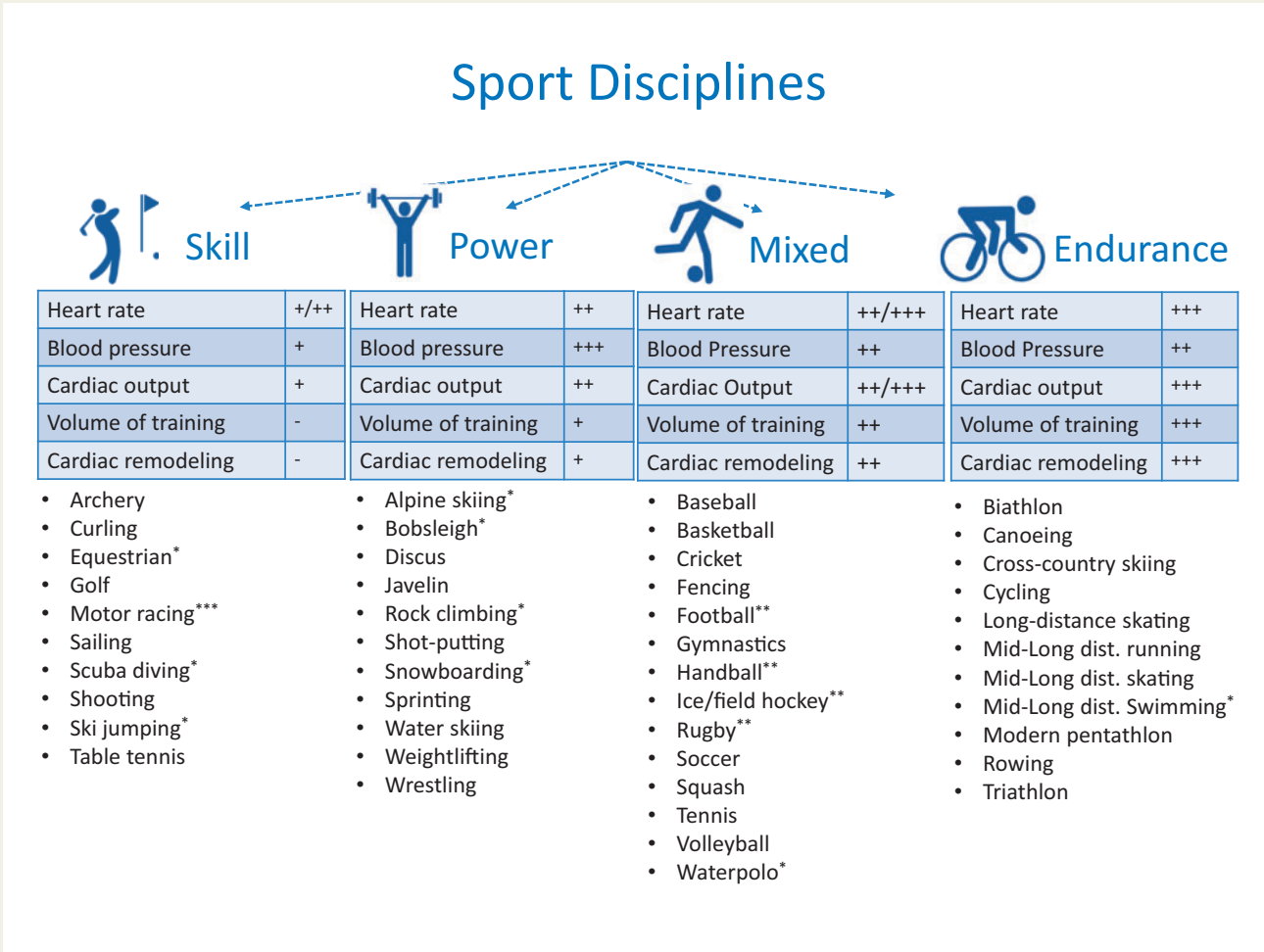


Figure 1 Schematic representation of the four different types of sport disciplines, modified after Pelliccia *et al.*[18] The common haemodynamic changes and cardiac remodelling occurring as a consequence of long-term training are indicated for each type of sport. *Symbols*: **indicates sport with increased risk of bodily collision. *indicates sport with intrinsic risk of serious harm or death for athlete and/or spectators in the event of syncope.

Table 1 Definition of variables

Variables	Definitions
Ventricles[a]:	
Ventricular dysfunction	*Left and right ventricles*:
• No dysfunction	EF ≥ 55%
• Mild dysfunction	45% ≤ EF < 55% (or normal systemic RV function)
• Moderate dysfunction	30 ≤ EF < 45%
• Severe dysfunction	EF < 30% (or impaired systemic RV function)
Ventricular hypertrophy	*Left ventricle*:
• No hypertrophy	Wall thickness (cm): ♂ <1.1 ♀ <1.0 or LV mass (g/m^2): ♂ 50–102, ♀ 44–88
• Mild hypertrophy	Wall thickness (cm): ♂ 1.1–1.3 ♀ 1.0–1.2 or LV mass (g/m^2): ♂ 103–116 ♀ 89–100
• Moderate hypertrophy	Wall thickness (cm): ♂ 1.4–1.6 ♀ 1.3–1.5 or LV mass (g/m^2): ♂ 117–130, ♀101–112
• Severe hypertrophy	Wall thickness (cm): ♂ ≥1.7 ♀ ≥1.6 or LV mass (g/m^2): ♂ ≥131 ♀ ≥113
	Right ventricle:
	Qualitative and quantitative echocardiographic evaluation
Ventricular pressure overload	*Left and right ventricles*:
• No pressure overload	No significant LVOT or RVOT gradient (PSV < 2.6 m/s), no obstruction in great arteries
• Mild pressure overload	2.6 m/s ≤ PSV <3 m/s for LVOT and RVOT obstructions and PPS; for CA, peak arm-leg gradient <20 mmHg
• Moderate pressure overload	3 m/s ≤ PSV ≤4 m/s for LVOT and RVOT obstructions and PPS
• Severe pressure overload	PSV >4 m/s for LVOT and RVOT obstructions and PPS; for CA, peak arm-leg gradient ≥20 mmHg
Ventricular volume overload[b]	*Left and right ventricles*:
• No volume load	Absent or mild to moderate valve regurgitation or shunt
• Volume load without remodelling	Severe valve regurgitation or shunt with non-dilated RV and LV [RV EDA (cm^2/m^2) ♂≤12.6, ♀≤11.5; LV EDV (mL/m^2) ♂≤74, ♀≤61] and preserved systolic function
• Volume load with mild remodelling	Severe valve regurgitation or shunt with RV or LV dilatation with preserved systolic ventricular function
• Volume load with severe remodelling	Severe valve regurgitation or shunt with RV or LV dilatation with impaired systolic ventricular function
• Ventricle physiology	Single- or bi-ventricular circulation
	Systemic LV or systemic RV
Pulmonary artery pressure	
• No evidence of PH	TVRV[c] ≤2.8 m/s and no additional echocardiographic findings suggestive of PH or mPAP<25 mmHg on right heart catheterization
• PH with no RV dilatation or dysfunction	mPAP≥25 mmHg on right heart catheterization without RV dilatation or dysfunction
• PH with RV dilatation or dysfunction	mPAP≥25 mmHg on right heart catheterization with RV dilatation or dysfunction
Aorta	
• No/mild dilatation	Normal (≤35 mm) or borderline sizes (≥35 to <40 mm) of the aorta, *z*-score ≥2 to <3[d]
• Moderate dilatation	Aorta size ≥40 to <45 mm, *z*-score ≥3 to <4
• Severe dilatation	Aorta size ≥45 to <50 mm, *z*-score ≥4
• Size reaching indication for repair	Aorta size ≥50 mm
Arrhythmia	
• No arrhythmia	Absence of or infrequent arrhythmias (<500/24 h) PVC on a Holter monitor, which do not worsen with exercise
• Mild arrhythmia burden/non-malignant arrhythmia	Frequent or coupled PVC or controlled atrial fibrillation/atrial flutter, which do not worsen with exercise
• Significant arrhythmia burden/potentially malignant arrhythmia	Atrial fibrillation/atrial flutter, which worsen with exercise
	Non-sustained or sustained ventricular tachycardia or PVC burden that increases during exercise

Continued

Table 1 *Continued*

Variables	Definitions
Arterial oxygen saturation at rest/during exercise	
• No central cyanosis	No clinical signs; transcutaneous saturations in the range of 96–100%, at rest and during exercise
• Mild cyanosis	Transcutaneous oxygen saturations between 90% and 95%, at rest or during exercise
• Severe cyanosis	Transcutaneous oxygen saturations <90%, at rest or during exercise

CA, coarctation; EDA, end-diastolic area; EDD, end-diastolic diameter; EDV, end-diastolic volume; EF, ejection fraction; LV, left ventricle; LVEDD, left ventricular end-diastolic diameter; LVESD, left ventricular end-systolic diameter; LVOT, left ventricular outflow tract; PAP, pulmonary artery pressure; PPS, peripheral pulmonary stenosis; PSV, peak systolic velocity; PVC, premature ventricular complex; RV, right ventricle; RVEDD, right ventricular end-diastolic diameter; RVOT, right ventricular outflow tract; TVRV, tricuspid valve regurgitation velocity. Reference values from Lang *et al.* [19].
[a]Interpretation of chamber wall thickness, size, and function should take into consideration the athlete's demographics and sporting discipline.
[b]Serial imaging is necessary, particularly when uncertainty exists relating to the severity and haemodynamic impact of specific lesions and exercise regimes.
[c]In individuals with a systemic right ventricle the values refer to mitral valve regurgitation velocity.
[d]To follow common practise, z-score values should be used over absolute values if they fall into different categories in an individual patient–athlete.

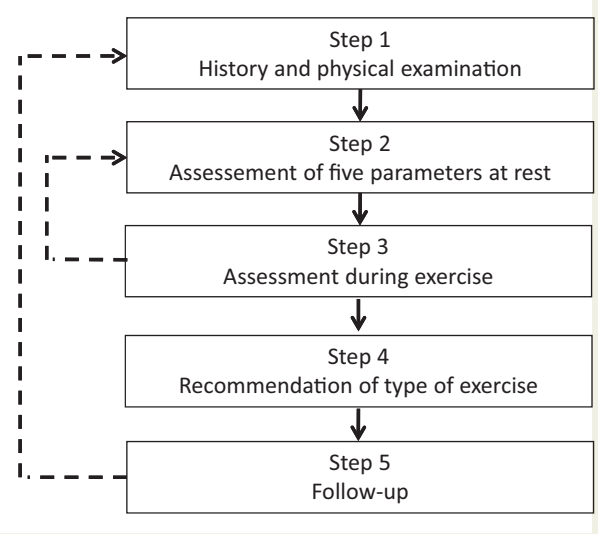


Figure 2 Flow chart depicting the five steps when evaluating competitive athletes with congenital heart disease.

sports will be performed. Finally, the physician should perform a thorough physical examination with particular reference to resting heart rate and rhythm, blood pressure, features of ventricular dysfunction, and the presence or absence of central cyanosis.

Step 2: Assessment of five parameters at rest

Assessment of ventricular structure and function

Transthoracic echocardiogram is usually sufficient to evaluate left and right ventricular function, valvular lesions, mechanical obstruction, and intracardiac shunts. Functional assessment by echocardiography should follow published guidelines[19] but need to take into account the specifics of ventricular pathophysiology in CHD, and the central role of the right ventricle (RV) in many CHD pathologies.[21] Cardiovascular magnetic resonance (CMR) may be required to assess right and left ventricular (LV) volumes and function, myocardial scar (which may act as a surrogate for arrhythmia risk), regurgitant fraction, visualization of prosthetic materials (conduits), and detailed morphological studies (e.g. pulmonary veins, coronary arteries) that can all impact on ventricular function in athletes with CHD. Computerized tomography (CT) is the imaging modality of choice for the delineation of small anatomical structures such as coronary arteries and collateral arteries and for imaging parenchymal lung pathology. Serial imaging is necessary, particularly when uncertainty exists relating to the severity and haemodynamic impact of specific lesions and exercise regimes. Interpretation of chamber size and function and wall thickness should also take into consideration the athlete's demographics, gender, and sporting discipline (*Table 1*).[22] It is well established that competitive athletes may exhibit a degree of LV hypertrophy and dilatation of all four cardiac chambers and differentiation of what constitutes physiological adaptation or sequelae of the CHD lesion may be challenging and requires joint assessment by an experienced sports and congenital cardiologist.

Assessment of pulmonary artery pressure

Pulmonary arterial hypertension usually occurs in the setting of a long-standing intra- or extracardiac communication that allows unrestricted volume and pressure overload on the RV. Over time this can result in mildly elevated or even fixed supra-systemic pulmonary artery pressures, elevated pulmonary vascular resistance, and reversal of shunting (Eisenmenger syndrome) leading to RV but also LV dysfunction. In addition, pulmonary venous hypertension secondary to systemic ventricular failure becomes more common in the ageing CHD population. A transthoracic echocardiogram is usually sufficient to evaluate the pulmonary artery pressure in individuals with tricuspid regurgitation (TR). Pulmonary hypertension (PH) is excluded when TR velocity is ≤2.8 m/s, and there are no additional echocardiographic variables suggestive of PH present. In cases, where a high index of suspicion for PH persists, particularly when restriction from some or all competitive sport is contemplated, right heart catheterization should be performed.[23]

Assessment of the aorta

Aortic diameters in athletes should be measured by echocardiography using standard methodology.[24] If dilated, serial measurements

are paramount as the rate of increase in aortic diameter is important for risk stratification.[25] While absolute aortic diameters are most commonly used in clinical practice (*Table 1*), indexed values or z-scores may be used to define normal values ($-2 < z > 2$).[24,26,27] Borderline or pathological values require additional cross-sectional imaging by CT or CMR and regular follow-up.[28] Although aortic pathologies such as dilatation or aneurysms can be secondary to CHD,[29] a primary aortopathy should be considered in cases where clinical findings or family history raises suspicion of genetic disease. Athletes with a primary aortopathy should be assessed using the specific guidelines.[24]

Assessment of arrhythmia

Sudden cardiac death is an important cause of mortality in CHD.[3,30] Sudden cardiac death during physical exertion accounts for 10% of all cases of SCD in CHD,[31] therefore, the assessment of arrhythmia in the athlete with CHD is important. Baseline investigations should include a 12-lead electrocardiogram (ECG), a 24-h or prolonged ECG monitoring, including periods of training and competition, and an exercise stress 12-lead ECG. Further investigations may be required depending on the presence of risk factors for arrhythmic events, which may include a CMR to assess myocardial fibrosis, an implantable loop recorder, and an electrophysiology study. Assessment for supraventricular or ventricular arrhythmias will primarily guide exercise prescription (*Table 1*). Additional risk factors for arrhythmia to consider include; extensive atrial or ventricular surgery due to scarring,[32] prolonged QRS duration, QRS fragmentation, QT dispersion, and moderately to severely impaired systemic or sub-pulmonary ventricular function.[31,33]

Assessment of arterial oxygen saturation

All patients with CHD should have a record of arterial oxygen saturations at rest. Central cyanosis is largely excluded when transcutaneous saturations are >95%, at rest and during exercise. Arterial oxygen saturations can be reduced not only by known or expected right-to-left shunts but also by yet undiagnosed shunting post-surgery via patch or baffle leaks, as well as systemic-to-pulmonary venous collaterals or pulmonary arteriovenous fistula. Therefore, when arterial oxygen saturations are reduced, comprehensive evaluation for the underlying pathophysiology, including evaluation for PH must be conducted.[8]

Step 3: Assessment during exercise

Cardiopulmonary exercise testing provides invaluable information relating to physiological sequelae of anatomical lesions, risk of morbidity and mortality, and timing of intervention.[34–36] It is an important tool for assessing the baseline fitness of individual athletes and helps to inform decision-making on different types of sport. Serial CPET assessments are required to monitor disease progression and the effect of exercise training, particularly when one considers that the physical fitness of competitive athletes with CHD may mask the significance of haemodynamically important lesions which left untreated might be detrimental. Physicians should adhere to published assessment guidelines[37] and reference values for the adult CHD population[38] and CPET should be accompanied by 12-lead ECG monitoring. Where CPET is not available, regular exercise ECG testing to assess for arrhythmias and ischaemic risk, should be performed, accepting its limitations, particularly in complex CHD.

Important parameters to assess during CPET are: (i) cardiopulmonary indices; peak-oxygen consumption (peak VO_2) is one of the best predictors of morbidity and mortality in patients with CHD.[34,35,39] In addition, the following parameters can help quantify exercise capacity: heart rate reserve as an outcome predictor particularly in the presence of cyanosis[40]; ventilatory efficiency slope as a useful parameter in the context of sub-maximal testing[34]; O_2 pulse to assess stroke volume; and gas exchange threshold to detect disturbances in aerobic and anaerobic metabolism. (ii) Arrhythmias or conduction disease; detection of arrhythmias during exercise, increases the risk of sudden death by 6.6-fold.[31] Chronotropic incompetence in CHD patients is often a symptom of ventricular dysfunction or ischaemia.[41] (iii) Ischaemia; ischaemia can occur in CHD and is likely to become a more significant problem in an ageing CHD population.[4] (iv) Arterial oxygen saturation; continuous monitoring of transcutaneous saturation or arterial blood gases can also be performed during CPET to detect progressive desaturations secondary to intracardiac shunts or pulmonary pathology. (v) Blood pressure; blood pressure response to exercise is also a useful adjunct to the assessment of patients with coarctation of the aorta or systemic outflow tract obstruction. A normal blood pressure response during exercise includes a rise in systolic blood pressure by ≥25 mmHg, to a maximum of 220 mmHg (men) and 200 mmHg (women), an attenuated response or a drop of systolic blood pressure requires further assessment.[42,43] A slight decrease in diastolic blood pressure can be seen in healthy individuals during exercise.[44] Individual centres may use their own criteria to determine abnormal blood pressure response. Abnormalities detected during CPET should prompt further evaluation and, if required, appropriate treatment or intervention before the athlete can re-enter the assessment algorithm at Step 2 (*Figure 2*) for repeat assessment of all five variables.

Exercise echocardiography has recently been shown to be a sensitive tool to detect early ventricular pathology in CHD.[45] Exercise imaging by echocardiography or CMR should be considered in selected cases and in centres where expertise in exercise imaging are available. Exercise imaging may demonstrate impaired contractile reserve, increased valvular gradients and relevant exercise-induced increase in pulmonary arterial pressure, all of which can be associated with exercise intolerance and arrhythmias.

Step 4: Recommendation on type of exercise

The physician should assess each of the parameters 1–5 and assign the individual athlete with CHD to a specific route which will dictate the recommended sporting disciplines (*Figure 3*). When all parameters fall within normal limits or there is evidence of mild hypertrophy or mild pressure or volume load (green route) athletes can participate in all competitive sports. When one of the parameters is outside these limits, restriction applies to endurance disciplines that are likely to pose the highest haemodynamic demands and require high volumes of training (orange route) or athletes should be restricted to skill sports only (brown route). Athletes with severe structural, haemodynamic or electrophysiological sequelae (red route) or symptomatic limitation should be restricted to recreational sport. Athletes

1. Ventricles	No systolic dysfunction No/mild hypertrophy No/mild pressure load No volume load	Mild systolic dysfunction Volume load without remodelling	Moderate systolic dysfunction Moderate hypertrophy Moderate pressure load Volume load with mild remodelling Single ventricle physiology Systemic right ventricle	Severe systolic dysfunction Severe hypertrophy Severe pressure load Volume load with severe remodelling
2. Pulmonary artery pressure	Low probability of pulmonary hypertension	PH without RV dilatation or dysfunction		PH with RV dilatation or dysfunction
3. Aorta	No/mild dilatation	Moderate dilatation	Severe dilatation	Dilatation approaching indication for repair
4. Arrhythmia at rest/during exercise	No arrhythmia	Mild arrhythmic burden Non-malignant arrhythmia		Significant arrhythmic burden Malignant arrhythmia
5. Saturation at rest/during exercise	No central cyanosis		Mild central cyanosis	Severe central cyanosis
	A	B	C	D
	When all applicable	When ≧1 parameters applicable AND no parameter falls within columns C or D	When ≧1 parameters applicable AND no parameter falls within column D	When ≧1 parameters applicable
Choice of competitive sport	All sports	Skill, Power, or Mixed sports	Skill sports only	NO COMPETITIVE SPORT

Figure 3 Flow chart depicting in detail Steps 2–4. Following assessment of the five variables at rest and during exercise, an individualized recommendation can be provided.

with aortic dilatation exceeding the mild range should avoid competing in sporting disciplines with a high static component, which includes most power sports and some of the skill sports such as car racing. Athletes with significant aortic dilatation should also exercise caution regarding the choice of sport, as disciplines at high risk of impact may pose considerable risk. In athletes where participation in competitive sports is not recommended, participation in recreational sports should be considered in line with the 2013 recommendations.[16]

Step 5: Surveillance

Athletes with CHD engaging in regular competition should be re-evaluated by cardiologists with expertise in CHD and sports cardiology every 6–12 months depending on the underlying lesion, the haemodynamic and electrophysiological sequelae and the characteristics of the sport. Athletes should go through the proposed algorithm at each visit including exercise assessment. Changes in functional status or symptoms should prompt temporary suspension of competition pending the results of repeat evaluation.

Special considerations

Exceptions

The recommendations are not applicable to all patient–athletes with CHD and in particular to athletes with concomitant congenital or inherited rhythm or conduction disorders, cardiomyopathies, isolated congenital coronary artery anomalies, and systemic arterial hypertension. These pathologies are addressed under the section recommendations of arrhythmias and potentially arrhythmogenic disorders, cardiomyopathies,[46] coronary artery disease,[47] and arterial hypertension,[48] respectively.

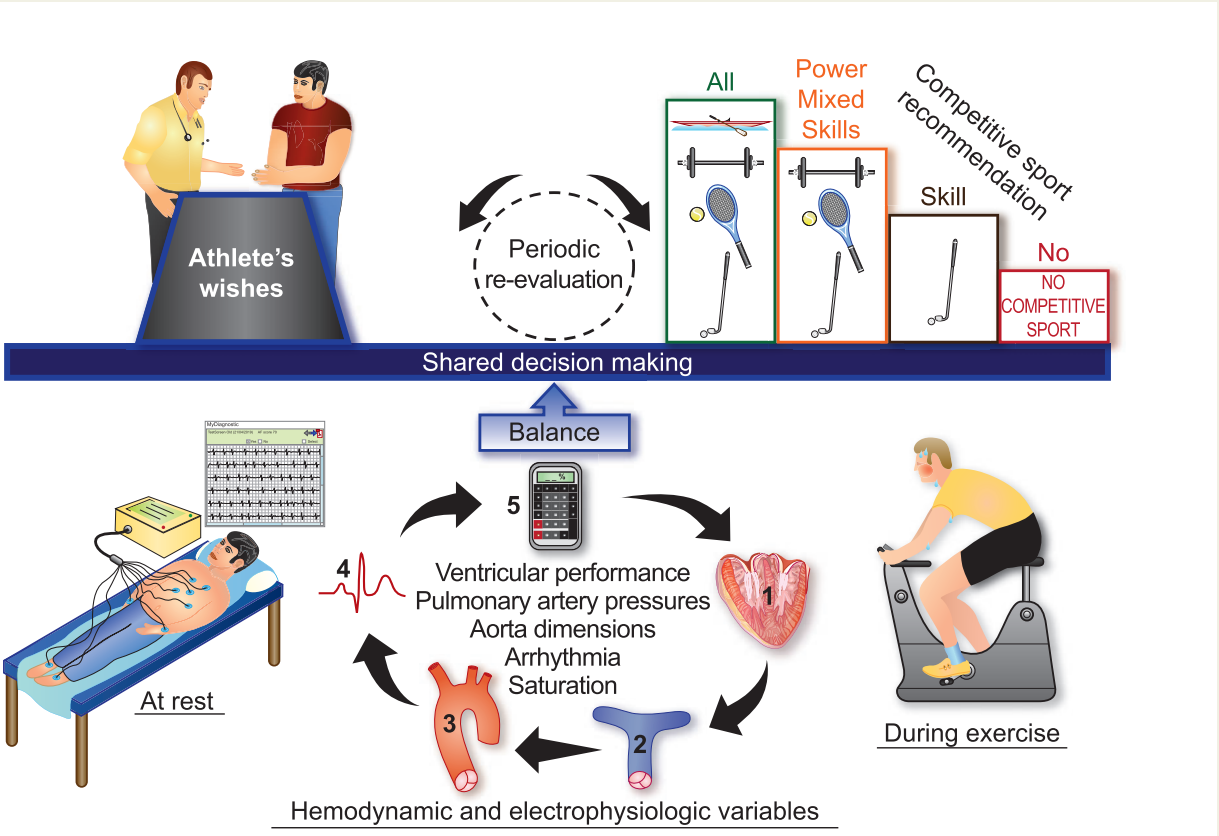
Surgical and catheter interventions

In the athlete with CHD, the indications and timings of surgical and transcatheter interventions should follow published guidelines.[20,49]

Although the health of the athlete is the foremost objective that should guide medical decisions, the expectations and goals of the athlete with CHD may differ from non-athlete patients in terms of functional status and physical performance level. This should be taken into consideration by healthcare professionals, and decisions on the timing of interventions in athletes should involve experts in sports and congenital cardiology. The aim is to provide the athlete with the best possible advice in order to make an informed decision.

Patients with implantable cardioverter-defibrillator and pacemakers

Athletes with an implantable cardioverter-defibrillator (ICD) or a pacemaker on a background of CHD should follow the guidelines for athletes with devices,[50] however, decision-making on eligibility will be determined by the proposed algorithm presented here. Important to



Take home figure Athletes with congenital heart disease who wish to participate in competitive sports should undergo comprehensive evaluation of the five key parameters at rest and during exercise, as appropriate. The proposed algorithm allows for individualized recommendations which should be balanced with the athlete's wishes and aspirations. Once a plan is agreed it should be reviewed periodically. Every review should re-address the balance depending on objective clinical markers form re-evaluation of all five parameters and changes in the athlete's goals.

consider when assessing athletes with CHD and an ICD or pacemaker are the higher rates of both, appropriate and inappropriate shocks and increased rate of lead complications in patients with CHD.[51]

Exercise at high altitude

Hypobaric hypoxia leads to decreased oxygen consumption, changes in cardiac volumes and output, tachycardia, hypocapnia-mediated decrease in stroke volume, and a rise in pulmonary artery pressures in the acclimatization phase. The increase in pulmonary vascular resistance associated with moderate and extreme high altitude can lead to impaired oxygenation, reduced cardiac output, and hypercoagulability. In addition, there are concerns of increased risk of arrhythmias at moderate and high altitudes.[52] Patients with cyanotic, unrepaired, or palliated complex CHD or CHD with associated PH should be advised against competitive sport at moderate or high altitude (above 1500 m).

Anticoagulation

Many patients with CHD receive temporary or life-long anticoagulation. Athletes with CHD on anticoagulation treatment should be advised against participation in contact sports.

Conclusion

The recommendations provide a comprehensive assessment algorithm which allows for individualized advice in athletes with CHD who wish to participate in competitive sports. At the same time, this document may form the platform for more uniform advice between healthcare professionals, while acknowledging the important role of individual physician experience. Only athletes with CHD in whom intense exercise may have a detrimental effect should be restricted from competitive sport participation. In such cases providing tailored exercise prescription will encourage physical activity and participation in less demanding recreational sports.[53]

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