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2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease

The Task Force on sports cardiology and exercise in patients with cardiovascular disease of the European Society of Cardiology (ESC)

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† We would like to pay tribute to Professor Galderisi who passed away in March 2020.

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The disclosure forms of all experts involved in the development of these Guidelines are available on the ESC website www.escardio.org/guidelines

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Table of Contents

Abbreviations and acronyms 5
1 Preamble 6
2 Introduction 8
3 Identification of cardiovascular disease and risk stratification in individuals participating in recreational and competitive sports 9
3.1 Introduction 9
3.2 Definitions of recreational and competitive athletes 9
3.3 Exercise-related major adverse cardiovascular events 9
3.4 Incidence of sudden cardiac death in athletes 10
3.5 Aetiology of sudden cardiac death during exercise 10
3.6 Screening modalities for cardiovascular disease in young athletes 10
3.7 Screening for cardiovascular disease in older athletes 10
4 Physical activity, leisure exercise, and competitive sports participation 11
4.1 General introduction 11
4.1.1 Definition and characteristics of exercise interventions 11
4.1.1.1 Type of exercise 11
4.1.1.2 Exercise frequency 12
4.1.1.3 Exercise intensity 12
4.1.1.4 Training volume 12
4.1.1.5 Type of training 12
4.1.2 Classification of exercise and sports 13
4.2 Exercise recommendations in individuals with cardiovascular risk factors 14
4.2.1 General introduction 14
4.2.2 Obesity 18
4.2.3 Hypertension 19
4.2.4 Dyslipidaemia 19
4.2.5 Diabetes mellitus 20
4.2.5.1 Effect of exercise on diabetic control, risk factors and outcomes 20
4.2.5.2 Recommendations for participation in exercise in individuals with diabetes mellitus 20
4.2.5.3 Cardiac evaluation before exercise in individuals with diabetes mellitus 20
4.3 Exercise and sports in ageing 21
4.3.1 Introduction 21
4.3.2 Risk stratification, inclusion/exclusion criteria 21
4.3.3 Exercise modalities and recommendations for exercise and sport in the elderly 21
5 Exercise in clinical settings 22
5.1 Exercise programmes for leisure-time and competitive sport participation in chronic coronary syndrome 22
5.1.1 Individuals at risk of atherosclerotic coronary artery disease and asymptomatic individuals in whom coronary artery disease is detected at screening 23
5.1.1.1 Recommendations for sports participation 23
5.1.2 Established (long-standing) chronic coronary syndrome 24
5.1.2.1 Antithrombotic treatment 26
5.1.3 Myocardial ischaemia without obstructive disease in the epicardial coronary artery 26
5.1.4 Return to sport after acute coronary syndrome 26
5.1.4.1 Competitive athletes 26
5.1.4.2 Recreational athletes 26
5.1.5 Anomalous origin of coronary arteries 26
5.1.5.1 Background 26
5.1.5.2 Eligibility for sports 26
5.1.6 Myocardial bridging 28
5.1.6.1 Background 28
5.1.6.2 Eligibility 28
5.2 Exercise recommendations in individuals with chronic heart failure 28
5.2.1 Background: rationale for exercise in chronic heart failure 28
5.2.2 Risk stratification and preliminary evaluation 28
5.2.3 Exercise modalities and sports participation in heart failure 29
5.2.3.1 Aerobic/endurance exercise 29

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5.2.3.2 Resistance exercise 29
5.2.3.3 Respiratory exercise 29
5.2.3.4 Aquatic exercise 29
5.2.4 Sports participation and return to sports 30
5.2.4.1 Competitive sports 30
5.2.4.2 Recreational sports 30
5.2.5 Heart failure with preserved ejection fraction 31
5.2.5.1 Exercise modalities and sports participation 31
5.2.6 Exercise in individuals after heart transplantation 31
5.2.6.1 Exercise modalities and sports participation 31
5.3 Exercise recommendations in individuals with valvular heart disease 32
5.3.1 Introduction 32
5.3.1.1 General principles in assessment and risk stratification of individuals with valvular heart disease prior to leisure exercise or competitive sports 32
5.3.1.2 Surveillance 32
5.3.2 Aortic valve stenosis 32
5.3.3 Aortic valve regurgitation 33
5.3.4 Bicuspid aortic valve 34
5.3.5 Primary mitral regurgitation 35
5.3.5.1 Mitral valve prolapse 36
5.3.6 Mitral stenosis 36
5.3.7 Tricuspid regurgitation 37
5.4 Exercise recommendations in individuals with aortopathy 37
5.4.1 Introduction 37
5.4.2 Risk of dissection 38
5.4.3 Sporting disciplines 38
5.4.4 Effect on aortic diameter and wall stress 38
5.4.5 Recommendations 39
5.5 Exercise recommendations in individuals with cardiomyopathies, myocarditis, and pericarditis 39
5.5.1 Hypertrophic cardiomyopathy 39
5.5.1.1 Risk stratification in hypertrophic cardiomyopathy 39
5.5.1.2 Baseline assessment of patients with HCM 39
5.5.1.3 History 39
5.5.1.4 Resting and ambulatory ECG 40
5.5.1.5 Echocardiography 40
5.5.1.6 Cardiac magnetic resonance imaging 40
5.5.1.7 Exercise testing 40
5.5.1.8 Genetic testing 40
5.5.1.9 ESC risk score in HCM 40
5.5.1.10 Exercise recommendation 40
5.5.1.11 Special considerations 40
5.5.1.12 Follow-up 40
5.5.2 Arrhythmogenic cardiomyopathy 41
5.5.2.1 Risk stratification in arrhythmogenic cardiomyopathy 41
5.5.2.2 Baseline assessment of patients with arrhythmogenic cardiomyopathy 41
5.5.2.3 History 42
5.5.2.4 Resting and ambulatory ECG 42
5.5.2.5 Echocardiography and cardiac magnetic resonance imaging 42
5.5.2.6 Exercise testing 42
5.5.2.7 Genetic testing 42
5.5.2.8 Exercise recommendations 42
5.5.2.9 Special considerations 42
5.5.2.10 Follow-up 42
5.5.3 Exercise recommendations in individuals with left ventricular non-compaction 43
5.5.3.1 Risk stratification 43
5.5.3.2 Follow-up 43
5.5.4 Exercise recommendations in individuals with dilated cardiomyopathy 43
5.5.4.1 Baseline assessment of patients with dilated cardiomyopathy 44
5.5.4.2 Special considerations 44
5.5.5 Exercise recommendations in individuals with myocarditis and pericarditis 44
5.5.5.1 Myocarditis 44
5.5.5.2 Diagnosis 45
5.5.5.3 Risk stratification 45
5.5.5.4 Exercise recommendations for individuals with myocarditis 45
5.5.6 Pericarditis 45
5.5.6.1 Diagnosis 45
5.5.6.2 Risk stratification 45
5.5.6.3 Exercise recommendations for individuals with pericarditis 46
5.6 Exercise recommendations in individuals with arrhythmias and channelopathies 46
5.6.1 A general management framework 46
5.6.2 Atrial fibrillation 46
5.6.2.1 Patients without atrial fibrillation 46
5.6.2.2 Prognostic and symptomatic relevance of AF during sports 47
5.6.2.3 Impact of continuing sport on the natural progression of atrial fibrillation after ablation 47
5.6.3 Supraventricular tachycardia and Wolff-Parkinson-White syndrome 48
5.6.3.1 Prognostic and symptomatic relevance of paroxysmal supraventricular tachycardia without pre-excitation 48
5.6.3.2 Prognostic and symptomatic relevance of pre-excitation 48
5.6.4 Premature ventricular contractions and non-sustained ventricular tachycardia 49
5.6.4.1 Relation between number of premature ventricular contractions and risk 49
5.6.4.2 Morphology of premature ventricular contractions 49
5.6.4.3 Premature ventricular contractions: response to exercise 49
5.6.4.4 Practical management of cardiac patients with premature ventricular contractions or non-sustained ventricular tachycardia who want to engage in sports 50
5.6.5 Long QT syndrome 50
5.6.6 Brugada syndrome 51
5.6.7 Following device implantation 52
5.6.7.1 Pacemakers 52
5.6.7.2 Implantable cardioverter defibrillators 52
5.7 Exercise recommendations in individuals with adult congenital heart disease 53

Downloaded from https://academic.oup.com/eurheartj/advance-article/doi/10.1093/eurheartj/ehaa605/5898937 by guest on 29 August 2020

5.7.1 Introduction 53
5.7.2 The increasing numbers of athletes with congenital heart disease 53
5.7.3 Non-cardiac abnormalities in congenital heart disease and Paralympic sport 53
5.7.4 General considerations in the congenital heart disease athlete 53
5.7.5 Sudden death during sport 54
5.7.6 Exercise in athletes with congenital heart disease: current guidelines and recommendations 54
5.7.7 Assessment of the athlete with congenital heart disease 54
6 Key messages 56
7 Gaps in evidence 57
8 Sex differences 58
9 'What to do' and 'what not to do' messages from the Guidelines 59
10 Supplementary data 62
11 Appendix 62
12 References 64

List of tables

Table 1 Classes of recommendations 7
Table 2 Levels of evidence 7
Table 3 Characteristics of exercise 12
Table 4 Indices of exercise intensity for endurance sports from maximal exercise testing and training zones 14
Table 5 Cardiovascular risk categories 17
Table 6 Potential risks for older people during exercise 22
Table 7 Exercise prescription in the elderly 22
Table 8 Exercise activities for older people according to exercise type and intensity 22
Table 9 Borderline or uninterpretable ECG findings 23
Table 10 Factors determining risk of adverse events during intensive exercise and competitive sports in asymptomatic individuals with long-standing coronary artery disease 24
Table 11 High-risk features for exercise-induced adverse cardiac events in patients with atherosclerotic coronary artery disease 24
Table 12 Optimal exercise training dose for patients with chronic heart failure 29
Table 13 Factors influencing decreased exercise capacity (peak VO_2) and reduced cardiac output in individuals with heart transplants 31
Table 14 Classification of risk to perform sports in patients with aortic pathology 38
Table 15 Findings during an invasive electrophysiological study (with the use of isoprenaline) indicating an accessory pathway with increased risk of sudden death 48
Table 16 Baseline parameters for assessment in congenital heart disease 55

List of figures

Figure Central illustration Moderate physical activity should be promoted in all individuals with cardiovascular disease 8
Figure 1 Components for expression of physical fitness 11
Figure 2 Sporting discipline in relation to the predominant component (skill, power, mixed and endurance) and intensity of exercise. Intensity of exercise must be individualized after maximal exercise testing, field testing and/or after muscular strength testing 13
Figure 3a and 3b SCORE charts for European populations of countries at HIGH and LOW cardiovascular disease risk 15
Figure 4 Proposed algorithm for cardiovascular assessment in asymptomatic individuals with risk factors for and possible subclinical chronic coronary syndrome before engaging in sports for individuals aged >35 years 18
Figure 5 Clinical evaluation and recommendations for sports participation in individuals with established coronary artery disease 25
Figure 6 Schematic representation of the most frequent anomalous origin of coronary arteries and associated risk of sudden cardiac death 27
Figure 7 Schematic representation of a myocardial bridge 28
Figure 8 Specific markers of increased risk of sudden cardiac death with mitral valve prolapse 36
Figure 9 Pre-participation assessment of individuals with congenital heart disease. 55

Tables of recommendations

General recommendations for exercise and sports in healthy individuals 17
Recommendations for cardiovascular evaluation and regular exercise in healthy individuals aged >35 years 18
Special considerations for individuals with obesity, hypertension, dyslipidaemia, or diabetes 21
Recommendations for exercise in ageing individuals 22
Recommendations for exercise in individuals at risk of atherosclerotic coronary artery disease and asymptomatic individuals in whom coronary artery disease is detected at screening 24
Recommendations for exercise in individuals with long-standing chronic coronary syndrome 25
Recommendations for return to exercise after acute coronary syndrome 26
Recommendations for exercise in young individuals/athletes with anomalous origins of coronary arteries 27
Recommendations for exercise/sports in individuals with myocardial bridging 28
Recommendations for exercise prescription in heart failure with reduced or mid-range ejection fraction 30
Recommendations for participation in sports in heart failure 30
Recommendations for exercise and participation in sport in individuals with heart failure with preserved ejection fraction 31
Recommendations for exercise and participation in sport in heart transplant recipients 32
Recommendations for exercise and participation in recreational/leisure-time sports in asymptomatic individuals with aortic stenosis 33
Recommendations for participation in competitive sports in asymptomatic individuals with aortic stenosis 33
Recommendations for participation in recreational/leisure-time sports in asymptomatic individuals with aortic regurgitation 34
Recommendations for participation in competitive sports in asymptomatic individuals with aortic regurgitation 34

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Recommendations for participation in recreational/leisure-time sports in asymptomatic individuals with mitral regurgitation 35
Recommendations for participation in competitive sports in asymptomatic individuals with mitral regurgitation 35
Recommendations for participation in recreational/leisure-time sports in individuals with mitral stenosis 37
Recommendations for participation in competitive sports in asymptomatic individuals with mitral stenosis 37
Recommendations for exercise and participation in sports in individuals with aortic pathology 39
Recommendations for exercise and sports participation in individuals with hypertrophic cardiomyopathy 41
Recommendations for exercise and sports participation in individuals with arrhythmogenic cardiomyopathy 42
Recommendations for exercise in individuals with left ventricular non-compaction cardiomyopathy 43
Recommendations for exercise in individuals with dilated cardiomyopathy 44
Recommendations for exercise in individuals with myocarditis 46
Recommendations for exercise and sports participation in individuals with pericarditis 46
Recommendations for exercise in individuals with atrial fibrillation ... 47
Recommendations for exercise and sports participation in individuals with paroxysmal supraventricular tachycardia and pre-excitation 49
Recommendations for exercise in individuals with premature ventricular contractions or non-sustained ventricular tachycardia 50
Recommendations for exercise in long QT syndrome 51
Recommendations for exercise in Brugada syndrome 51
Recommendations for exercise in individuals with pacemakers and implantable cardioverter defibrillators 53
Recommendations for exercise in individuals with congenital heart disease 56

Abbreviations and acronyms

ACE	Angiotensin-converting enzyme
ACHD	Adults with congenital heart disease
ACM	Arrhythmogenic cardiomyopathy
ACS	Acute coronary syndromes
AED	Automatic external defibrillator
AHA	American Heart Association
AF	Atrial fibrillation
AFL	Atrial flutter
AMI	Acute myocardial infarction
AN-SUD	Autopsy-negative sudden unexplained death
AP	Accessory pathway
AOCA	Anomalous origin of coronary arteries
AR	Aortic valve regurgitation
ARVC	Arrhythmogenic right ventricular cardiomyopathy
AS	Aortic valve stenosis
ASI	Aortic size index
AVNRT	Atrioventricular nodal re-entrant tachycardia
AVRT	Atrioventricular re-entrant tachycardia
BAV	Bicuspid aortic valve
BMI	Body mass index
BP	Blood pressure
BrS	Brugada syndrome
CAC	Coronary artery calcium
CAD	Coronary artery disease
CCS	Chronic coronary syndrome
CCTA	Coronary computed tomography angiography
CHD	Congenital heart disease
CKD	Chronic kidney disease
CMD	Coronary microvascular dysfunction
CMR	Cardiac magnetic resonance
CPET	Cardiopulmonary exercise test
CPR	Cardiopulmonary resuscitation
CT	Computed tomography
CV	Cardiovascular
CVA	Cerebrovascular accident
CVD	Cardiovascular disease
DBP	Diastolic blood pressure
DCM	Dilated cardiomyopathy
EACPR	European Association for Cardiovascular Prevention and Rehabilitation
EAPC	European Association of Preventive Cardiology
ECV	Extracellular volume
ECG	Electrocardiogram
EDS	Ehlers Danlos syndrome
EF	Ejection fraction
EP	Electrophysiological
ESC	European Society of Cardiology
Ex-R	Exercise-related
exCR	Exercise-based cardiac rehabilitation
FFR	Fractional flow reserve
FITT	Frequency, intensity, time, and type
HCM	Hypertrophic cardiomyopathy
HDL	High-density lipoprotein
HF	Heart failure
HIIT	High-intensity interval training
HR	Heart rate
HFmrEF	Heart failure with mid-range ejection fraction
HFpEF	Heart failure with preserved ejection fraction
HFrEF	Heart failure with reduced ejection fraction
HRmax	Maximal heart rate
HRR	Heart rate reserve
HTAD	Hereditary thoracic aortic disease
HTx	Heart transplant
ICD	Implantable cardioverter defibrillator
IMT	Intima–media thickness
INOCA	Ischaemic and non-obstructive coronary artery disease
LBBB	Left bundle branch block
LDL	Low-density lipoprotein
LEAD	Lower extremity artery disease
LGE	Late gadolinium enhancement
LV	Left ventricular
LVEDD	Left ventricular end-diastolic diameter
LVEF	Left ventricular ejection fraction
LVNC	Left ventricular non-compaction
LVOT	Left ventricular outflow tract
LQTS	Long QT syndrome

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MACE	Major adverse cardiovascular events
MB	Myocardial bridge/bridging
MCE	Moderate continuous exercise
MET	Metabolic equivalent
MFS	Marfan syndrome
MI	Myocardial infarction
MR	Mitral regurgitation
MS	Mitral stenosis
MVA	Mitral valve area
MVP	Mitral valve prolapse
NSVT	Non-sustained ventricular tachycardia
NYHA	New York Heart Association
OAC	Oral anticoagulants
PA	Physical activity
PAD	Peripheral arterial disease
PAP	Pulmonary artery pressure
PCI	Percutaneous coronary intervention
PCSK-9	Proprotein convertase subtilisin/kexin type 9
PET	Positron emission tomography
PH	Pulmonary hypertension
PM	Pacemaker
PSVT	Paroxysmal supraventricular tachycardia
PVC	Premature ventricular contraction
PVI	Pulmonary vein isolation
RBBB	Right bundle branch block
RM	Repetition maximum
RPE	Rating of perceived exertion
RT-PCR	Reverse transcriptase polymerase chain reaction
RV	Right ventricular
RVOT	Right ventricular outflow tract
SBP	Systolic blood pressure
SCA	Sudden cardiac arrest
SCAD	Spontaneous coronary artery dissection
SCD	Sudden cardiac death
SCORE	Systematic Coronary Risk Evaluation
sPAP	Systolic pulmonary artery pressure
SPECT	Single-photon emission computed tomography
TIA	Transient ischaemic attack
TR	Tricuspid regurgitation
T2DM	Type II diabetes mellitus
US	United States
VA	Ventricular arrhythmia
VAD	Ventricular assist device
VF	Ventricular fibrillation
VT	Ventricular tachycardia
VO_2	Oxygen consumption
VO_{2max}	Maximum oxygen consumption
VO_{2peak}	Peak oxygen consumption
WADA	World Anti-Doping Agency
WPW	Wolff-Parkinson-White

1. Preamble

Guidelines summarize and evaluate available evidence with the aim of assisting health professionals in proposing the best management strategies for an individual patient with a given condition. Guidelines and their recommendations should facilitate decision making of health professionals in their daily practice. However, the final decisions concerning an individual patient must be made by the responsible health professional(s) in consultation with the patient and caregiver as appropriate.

A great number of Guidelines have been issued in recent years by the European Society of Cardiology (ESC), as well as by other societies and organizations. Because of their impact on clinical practice, quality criteria for the development of guidelines have been established in order to make all decisions transparent to the user. The recommendations for formulating and issuing ESC Guidelines can be found on the ESC website (http://www.escardio.org/Guidelines-&-Education/Clinical-Practice-Guidelines/Guidelines-development/Writing-ESC-Guidelines). The ESC Guidelines represent the official position of the ESC on a given topic and are regularly updated.

In addition to the publication of Clinical Practice Guidelines, the ESC carries out the EurObservational Research Programme of international registries of cardiovascular diseases and interventions which are essential to assess, diagnostic/therapeutic processes, use of resources and adherence to Guidelines. These registries aim at providing a better understanding of medical practice in Europe and around the world, based on high-quality data collected during routine clinical practice.

Furthermore, the ESC has developed and embedded, in some of its guidelines, a set of quality indicators (QIs) which are tools to evaluate the level of implementation of the Guidelines and may be used by the ESC, hospitals, healthcare providers and professionals to measure clinical practice as well as used in educational programmes, alongside the key messages from the Guidelines, to improve quality of care and clinical outcomes.

The Members of this Task Force were selected by the ESC, including representation from its relevant ESC sub-specialty groups, in order to represent professionals involved with the medical care of patients with this pathology. Selected experts in the field undertook a comprehensive review of the published evidence for management of a given condition according to ESC Committee for Practice Guidelines (CPG) policy. A critical evaluation of diagnostic and therapeutic procedures was performed, including assessment of the risk–benefit ratio. The level of evidence and the strength of the recommendation of particular management options were weighed and graded according to predefined scales, as outlined below.

The experts of the writing and reviewing panels provided declaration of interest forms for all relationships that might be perceived as real or potential sources of conflicts of interest. Their declarations of interest were reviewed according to the ESC declaration of interest rules and can be found on the ESC website (http://www.escardio.org/guidelines). This process ensures transparency and prevents potential biases in the development and review processes. Any changes in declarations of interest that arise during the writing period were notified to the ESC and updated. The Task Force received its entire financial support from the ESC without any involvement from the healthcare industry.

The ESC CPG supervises and coordinates the preparation of new Guidelines. The Committee is also responsible for the endorsement process of these Guidelines. The ESC Guidelines undergo extensive review by the CPG and external experts. After appropriate revisions the Guidelines are approved by all the

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Table 1 Classes of recommendations

Classes of recommendations	Definition	Wording to use
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective.	Is recommended or is indicated
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.	
Class IIa	Weight of evidence/opinion is in favour of usefulness/efficacy.	Should be considered
Class IIb	Usefulness/efficacy is less well established by evidence/opinion.	May be considered
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.	Is not recommended

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Table 2 Levels of evidence

Level of evidence A	Data derived from multiple randomized clinical trials or meta-analyses.
Level of evidence B	Data derived from a single randomized clinical trial or large non-randomized studies.
Level of evidence C	Consensus of opinion of the experts and/or small studies, retrospective studies, registries.

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experts involved in the Task Force. The finalized document is approved by the CPG for publication in the European Heart Journal. The Guidelines were developed after careful consideration of the scientific and medical knowledge and the evidence available at the time of their dating.

The task of developing ESC Guidelines also includes the creation of educational tools and implementation programmes for the recommendations including condensed pocket guideline versions, summary slides, booklets with essential messages, summary cards for non-specialists and an electronic version for digital applications (smartphones, etc.). These versions are abridged and thus, for more detailed information, the user should always access to the full text version of the Guidelines, which is freely available via the ESC website and hosted on the EHJ website. The National Cardiac Societies of the ESC are encouraged to endorse, adopt, translate and implement all ESC Guidelines. Implementation programmes are needed because it has been shown that the outcome of disease may be favourably influenced by the thorough application of clinical recommendations.

Health professionals are encouraged to take the ESC Guidelines fully into account when exercising their clinical judgment, as well as in the determination and the implementation of preventive, diagnostic

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or therapeutic medical strategies. However, the ESC Guidelines do not override in any way whatsoever the individual responsibility of health professionals to make appropriate and accurate decisions in consideration of each patient's health condition and in consultation with that patient or the patient's caregiver where appropriate and/or necessary. It is also the health professional's responsibility to verify the rules and regulations applicable in each country to drugs and devices at the time of prescription.

2. Introduction

Exercise recommendations and eligibility criteria for sports participation in competitive athletes with cardiovascular disease (CVD) were originally published by the Sports Cardiology Section of the European Society of Cardiology (ESC) in 2005[1] and some aspects were subsequently updated in 2018 and 2019.[2,3] The overarching aim of these recommendations was to minimize the risk of adverse events in highly trained athletes. It is important to recognize, however, that most of the exercising population engages in leisure sport and solo recreational exercise and, unlike elite athletes, these individuals have a higher prevalence of risk factors for atherosclerosis and established CVD.

Regular physical activity (PA), including systematic exercise, is an important component of therapy for most CVDs and is associated with reduced cardiovascular (CV) and all-cause mortality. In an era where there is an increasing trend towards a sedentary lifestyle and a rising prevalence of obesity and associated CVDs, the promotion of PA and regular exercise is more crucial than ever and at the forefront of priorities for all scientific CV societies. Even during routine

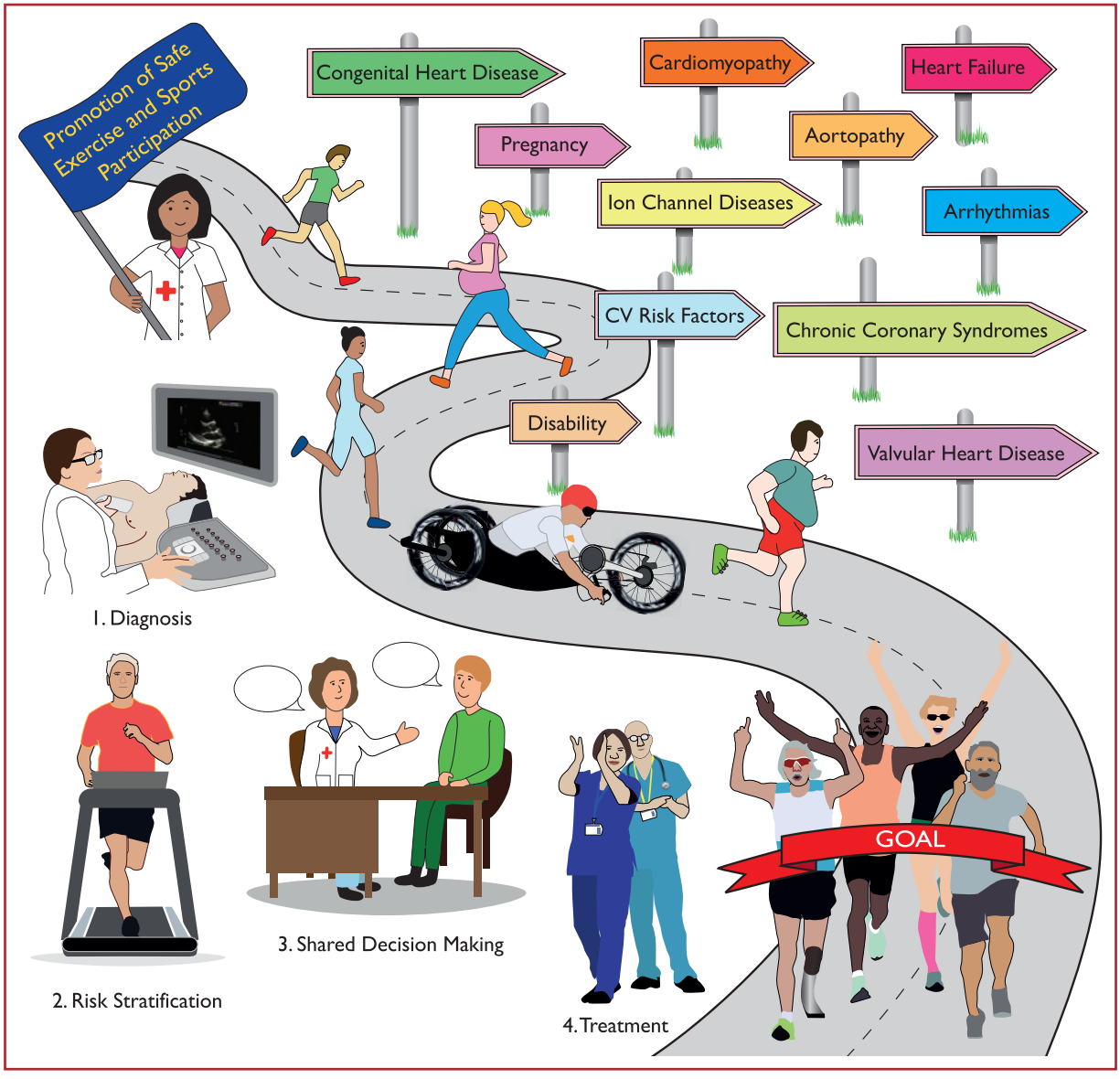

Figure Central illustration Moderate physical activity should be promoted in all individuals with cardiovascular disease. Appropriate risk stratification and optimal therapy are essential for providing exercise prescription for more vigorous activity. Individuals should be involved in the decision making process and a record of the discussion and exercise plan should be documented in the medical records.

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consultations for other considerations, physicians are encouraged to promote exercise in all patients.

Although proportionately scarce, exercise may paradoxically trigger sudden cardiac arrest (SCA) in individuals with CVD, particularly those who were previously sedentary or have advanced CVD.[4,5] In parallel with the drive to promote exercise in all individuals,[6] it is anticipated that physicians will be confronted with an increasing number of enquiries from individuals with established risk factors for coronary artery disease (CAD) or established CVDs about participation in exercise programmes and recreational sports activities. Such consultations need to strike a balance between the multiple benefits of exercise, the small risk of sudden death, and the patient's goals for cardiorespiratory fitness and ongoing participation in relatively strenuous exercise following a CV diagnosis.

The current Guidelines for exercise and sports participation in individuals with CVD are the first of a kind by the ESC. Sports cardiology is a relatively novel and emerging sub-speciality, therefore the evidence base for the natural history of disease progression or risk of death during intensive exercise and competitive sport among individuals with CVD is relatively sparse. This is reflected by the fact that a disproportionately large number of recommendations are reliant on the wisdom and vast experience of the consensus group rather than on large prospective studies. We acknowledge the inherent difficulties in formulating recommendations for all scenarios in a heterogeneous population with a diverse spectrum of CVDs in light of the limited availability of evidence. Therefore, these recommendations should not be considered as legally binding and should not discourage individual physicians from practising outside the remit of this document, based on their clinical experience in sports cardiology.

Where possible, the Guidelines have included the most up-to-date research in exercising individuals with CVD. The current Guidelines also draw upon existing ESC Guidelines for the investigation, risk assessment, and management of individuals with CVDs to aid physicians when prescribing exercise programmes or providing advice for sports participation. We hope that the document will serve as a useful clinical guide but also as an incentive for future research to challenge established wisdom.

In line with good clinical practice, the present document encourages shared decision making with the athlete patient and respects the autonomy of the individual after provision of detailed information about the impact of sports and the potential risks of complications and/or adverse events (*Central illustration*). Similarly, all exercise prescription and related discussions between the individual and the physician should be documented in the medical report.

3. Identification of cardiovascular disease and risk stratification in individuals participating in recreational and competitive sports

3.1 Introduction

Higher levels of PA and fitness are associated with lower all-cause mortality, lower rates of CVD, and lower prevalence of several known malignancies.[7–16] Despite the substantial health benefits provided by regular PA, intense exercise may paradoxically act as a trigger for life-threatening ventricular arrhythmias (VAs) in the presence of underlying CVD. Indeed, sudden cardiac death (SCD) is the leading cause of sports and exercise-related mortality in athletes.[17–19] CV safety during sports participation for individuals at all levels and ages is imperative to avoid catastrophic and often preventable SCD and has become a common goal among medical and sports governing organizations.[20–24]

Pre-participation CV screening aimed at the detection of disorders associated with SCD is universally supported by major medical societies.[20–22,25,26] However, the best method for CV screening of young competitive athletes (<35 years old) remains controversial, and limited data are available to guide recommendations in master athletes (≥35 years old)

Screening strategies must be tailored to the target population and the specific disorders with highest risk. SCD in young athletes is caused by a variety of structural and electrical disorders of the heart, including cardiomyopathies, ion channel disorders, coronary anomalies, and acquired cardiac conditions.[17,27,28] In adult and senior athletes, atherosclerotic CAD is the primary condition leading to major adverse cardiovascular events (MACE).[28,29]

3.2 Definitions of recreational and competitive athletes

The ESC defines an athlete as 'an individual of young or adult age, either amateur or professional, who is engaged in regular exercise training and participates in official sports competition'.[1,30] Similarly, the American Heart Association (AHA) and others define a competitive athlete as an individual involved in regular (usually intense) training in organized individual or team sports, with an emphasis on competition and performance.[31,32] Athletes involved in competitive sports span the age spectrum and can compete at the youth, high school, academy, university, semi-professional, professional, national, international, and Olympic levels. As a distinction, a recreational athlete engages in sports for pleasure and leisure-time activity, whereas a competitive athlete is highly trained with a greater emphasis on performance and winning. In a proposed classification of athletes based on the minimum volume of exercise, 'elite' athletes (i.e. national team, Olympians, and professional athletes) generally exercise ≥10 h/week; 'competitive' athletes [i.e. high school, college, and older (master) club level athletes] exercise ≥6 h/week; and 'recreational' athletes exercise ≥4 h/week.[33] This distinction is somewhat arbitrary since some recreational athletes, such as long-distance cyclists and runners, engage in exercise at higher volumes than some professional athletes participating in skill sports.

3.3 Exercise-related major adverse cardiovascular events

Exercise-related MACE include SCA and SCD; acute coronary syndromes (ACS) such as myocardial ischaemia and myocardial infarction (MI); transient ischaemic attacks (TIA) and cerebrovascular accidents (CVA); and supraventricular tachyarrhythmias.

SCA is defined as an unexpected collapse due to a cardiac cause in which cardiopulmonary resuscitation (CPR) and/or defibrillation is provided in an individual regardless of the survival outcome.[17,27,32]

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SCD is defined as a sudden unexpected death due to a cardiac cause, or a sudden death in a structurally normal heart at autopsy with no other explanation for death and a history consistent with cardiac-related death (i.e. requiring cardiac resuscitation).[17,27,32] In order to compare previously reported data on SCA and SCD using variable definitions, the timing of the event should be categorized as occurring during the episode, within the first hour post-exercise, or between 1 to 24 h post-exercise.[30] The activity at the time of the event can be further characterized as occurring during training or competition, at rest, or during sleep.[30]

Exercise-induced ACS are most likely to affect adult and senior athletes and result from atherosclerotic plaque disruption and coronary thrombosis in most cases.[34,35] More than 50% of patients who experience acute MI (AMI) and SCA do not have pre-existing symptoms or a known history of CAD.[36,37] In long-term endurance athletes, SCA and myocardial ischaemia can also occur from 'demand' ischaemia due to an imbalance between oxygen supply and demand resulting from stable calcified plaque and a fixed stenosis.[38] In a study of United States (US) marathon and half-marathon races, none of the runners with SCA with serious (>80% coronary artery stenosis in a proximal left coronary artery or three-vessel disease) coronary atherosclerosis had angiographic evidence of acute plaque rupture or thrombus.[38]

3.4 Incidence of sudden cardiac death in athletes

Current estimates of the incidence of SCD in competitive athletes range from almost 1 in a million to 1 in 5000 athletes per year.[17,39,40] Differences in current estimates are largely due to inconsistent study methodology and heterogeneous population comparisons.

Because reporting of SCD in athletes is not mandatory in most countries, studies risk underestimating the true incidence due to incomplete case ascertainment. For instance, studies using media reports as their main source to detect incidents of SCD identify only 5 - 56% of cases, even in high-profile competitive athletes.[41–44] Similarly, use of catastrophic insurance claims as the only method for case identification missed 83% of SCD cases and 92% of all SCA cases in Minnesota high school athletes.[40,45]

The athlete population being studied also needs to be precisely defined. Census population statistics, cross-sectional surveys, and self-reported athlete participation data all produce less reliable calculations. Other study details should also be considered. Does the study include all cases of SCA (survivors plus deaths) or only SCD? Does the study include cases occurring at any time (i.e. during exercise, rest, or sleep), or only those that occur during sports? Studies indicate that 56 - 80% of SCA in young athletes occurs during exercise with the remainder non-exertional.[17,18,46]

Evidence supports that some athletes display a higher risk for SCA based on sex, race, or sport.[17,40,41,45–50] Incidence rates are consistently higher in male athletes than in female athletes, with a relative risk ranging from 3: 1 to 9: 1 (male: female).[17,45,47–49,51,52] Black athletes of African Caribbean descent also have a higher risk than white athletes. In US college athletes, males had a higher risk than females (1 in 38 000 vs. 1 in 122 000), and black athletes had a 3.2 times higher risk than white athletes (1 in 21 000 vs. 1 in 68 000).[17] Male basketball players had the highest annual risk of SCD (1 in 9000), and male black basketball players had a risk of 1 in 5300.[17] Based on available studies and a systematic review of the literature, a generally accepted annual incidence of all SCA is approximately 1 in 80 000 in high school-aged athletes and 1 in 50 000 in college-aged athletes.[50] Male athletes, black athletes, basketball (US) and soccer (Europe) athletes represent higher risk groups. Limited estimates are available for youth, professional, and master athletes.

3.5 Aetiology of sudden cardiac death during exercise

SCD in young athletes is usually caused by a genetic or congenital structural cardiac disorder.[17–19,42,53,54] However, autopsy-negative sudden unexplained death (AN-SUD), also referred to as sudden arrhythmic death syndrome, is reported on post-mortem examination in up to 44% of presumed SCD cases depending on the study population.[17,28,42,53–56] In apparently healthy young athletes the prevalence of cardiac disorders associated with SCD is approximately 0.3%, and this figure is supported by multiple studies using non-invasive evaluation tools to detect cardiac disorders at elevated risk of SCD.[20,57–65]

In athletes >35 years of age, more than 80% of all SCD is due to atherosclerotic CAD, and vigorous physical exertion is associated with an increased risk of AMI and SCD.[34,66–70] The athletes at greatest risk are those with little or no background in systematic training.

3.6 Screening modalities for cardiovascular disease in young athletes

Most experts believe that early detection of potentially lethal disorders in athletes can decrease CV morbidity and mortality through risk stratification, disease-specific interventions, and/or exercise modifications.[22,57,58,71] CV screening by history and physical examination or by electrocardiogram (ECG) presents unique challenges and limitations. Several studies have documented the low sensitivity and high positive response rate of pre-participation history questionnaires.[64,65,72–75] In CV screening studies in which experienced clinicians use contemporary ECG interpretation standards, ECG screening outperforms history and physical examination in all statistical measures of performance.[58,59,62,64,65,74,76]

While echocardiography may identify additional structural disorders, there is insufficient evidence to recommend an echocardiogram for routine screening.[77]

3.7 Screening for cardiovascular disease in older athletes

The recommendations and evidence base for CV screening in athletes >35 years of age are limited. CV screening in adult and senior athletes must target the higher prevalence of atherosclerotic CAD. However, routine screening for ischaemia with exercise testing in asymptomatic adults has a low positive predictive value and a high number of false-positive tests and is not recommended.[78–80]

A screening ECG may still discover undiagnosed cardiomyopathies and primary electrical disorders in older athletes, and risk factor assessment for CVD may identify higher risk individuals who warrant additional testing. Thus, consistent with a 2017 ESC position paper on pre-participation CV screening, exercise ECG testing should be reserved for symptomatic athletes or those deemed at high risk of

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CAD based on the ESC Systematic Coronary Risk Evaluation (SCORE) system (see chapters 4 and 5).[6,81]

Exercise testing may also be useful to evaluate the blood pressure (BP) response to exercise, the occurrence of exercise-induced arrhythmias, and to assess symptoms or physical performance and its relation to exercise training.[81] In adult and elderly individuals, especially those naïve to moderate to vigorous PA, exercise testing or cardiopulmonary exercise testing (CPET) is a useful means to assess overall CV health and performance, allowing individualized recommendations regarding sports and exercise type and intensity, as will be discussed in subsequent sections.[82]

4. Physical activity, leisure exercise, and competitive sports participation

4.1 General introduction

Recommendations for prescription of exercise require a basic knowledge of physiological responses to exercise, along with an understanding of concepts and characteristics of PA, exercise interventions, and their implications for sports participation. Although exercise and PA are often used interchangeably, it is important to recognize that these terms differ. PA is defined as any bodily movement produced by the skeletal muscle that results in energy expenditure. Exercise or exercise training, on the other hand, by definition, is PA that is structured, repetitive, and purposeful to improve or maintain one or more components of physical fitness.[83]

Physical fitness may be expressed by five major components (*Figure 1*):[83] a morphological component (body mass relative to height, body composition, subcutaneous fat distribution, abdominal visceral fat, bone density, and flexibility);[84] a muscular component (power or explosive strength, isometric strength, muscular endurance);[85] a motor component (agility, balance, coordination, speed of movement);[85] a cardiorespiratory component (endurance or submaximal exercise capacity, maximal aerobic power, heart function, lung function, BP); and a metabolic component (glucose tolerance, insulin sensitivity, lipid and lipoprotein metabolism, substrate oxidation characteristics).[86]

4.1.1 Definition and characteristics of exercise interventions

The basic tenets of exercise prescription have been described using the 'FITT' concept (frequency, intensity, time, and type). The mode of exercise (*Table 3*) is also an important characteristic. The following sections will describe each of these components related to aerobic exercise followed by components of strength exercise.

4.1.1.1 Type of exercise

Traditionally, different forms of exercise are classified in binary terms as endurance or resistance (strength) exercise. However, this classification is somewhat oversimplified. Further classifications of exercise are metabolically related (aerobic vs. anaerobic exercise) or those related to the type of muscle contraction: isotonic [contraction against resistance in which the length of the muscle shortens (concentric) or lengthens (eccentric)] and isometric (static or without change in length of the muscle).

Aerobic exercise refers to activity performed at an intensity that allows metabolism of stored energy to occur mainly through aerobic glycolysis. Besides the glycolytic pathway, fat metabolism (β-oxidation) is also involved during aerobic exercise. Aerobic exercise involves large muscle groups performing dynamic activities, resulting in

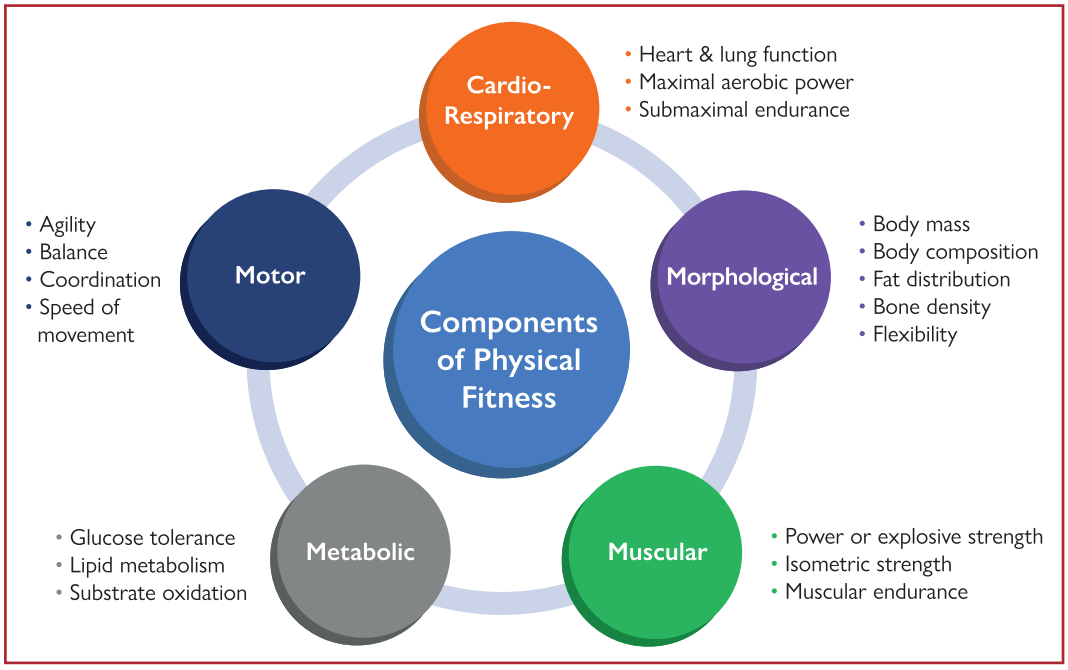

Figure 1 Components for expression of physical fitness.

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Table 3 Characteristics of exercise

Frequency:
- Sessions/week
- Bouts of exercise

Intensity:
- Endurance: %VO_2 peak or % peak HR or %HRR
- Strength or Power: % 1RM or % 5RM or %peak HR or %HRR for mixed exercise

Time:
- Duration of
  - exercise programme in weeks or months
  - training days per week
  - training session times per day
  - duration of training session in hours.

Type:
- Endurance (running, cycling, rowing, walking, swimming)
- Strength or resistance training
- Speed and speed endurance
- Flexibility (sit & reach, back stretch test, lateral mobility test)
- Coordination and balance

Mode of exercise training:
- Metabolic: *aerobic vs. anaerobic*
- Muscular work:
  isometric – isotonic
  dynamic (concentric, eccentric) vs. static
  continuous vs. interval
  large or small muscular groups

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HR = heart rate; HRR = heart rate reserve; RM = repetition maximum; VO_2 = oxygen consumption; VO_{2peak} = peak oxygen consumption.

substantial increases in heart rate and energy expenditure. Examples of aerobic exercise include cycling, running, and swimming performed at low to moderate intensity.[84] In contrast, anaerobic exercise refers to movement performed at high intensity unsustainable by oxygen delivery alone and requiring metabolism of stored energy to be processed largely by anaerobic glycolysis. A sustained isometric muscle action that is not working maximally but does not necessarily depend entirely upon oxygen during the muscle contraction is an example of anaerobic exercise. Another example of anaerobic exercise is intermittent high-intensity exercise.[85]

4.1.1.2 Exercise frequency

Exercise frequency is usually expressed as the number of times an individual engages in exercise per week. Guidelines suggest that moderate exercise should be performed most days of the week, amounting to a minimum of 150 min/week.

4.1.1.3 Exercise intensity

Of all the basic elements of exercise prescription, exercise intensity is generally considered to be the most critical for achieving aerobic fitness and to have the most favourable impact on risk factors.[86,87] Absolute intensity refers to the rate of energy expenditure during exercise and is usually expressed in kcal/min or metabolic equivalents (METs).[84,88] Relative exercise intensity refers to a fraction of an individual's maximal power (load) that is maintained during exercise and is usually prescribed as a percentage of maximal aerobic capacity (VO_{2max}) on the basis of a CPET.[88] Training intensity can also be expressed as a percentage of maximal heart rate (HR_{max}) recorded during an exercise test[89] or predicted on the basis of the equation [HR_{max} = 220 - age].[90] The use of prediction equations for HR_{max} is not recommended, because there is a large standard deviation around the regression line between age and HR_{max}.[91] Alternatively, exercise intensity can be expressed relative to a percentage of a person's HR reserve (HRR), which uses a percentage of the difference between HR_{max} and resting HR and adds it to the resting HR (Karvonen formula).[92] There are caveats to the use of HR for prescribing and evaluating exercise intensity in persons using beta-blockers.[93] Ideally, the HR derived for training should only be used if functional capacity was determined (an exercise test was performed) while taking the medication. Intensity is also commonly monitored using the rate of perceived exertion scale (e.g. 12 - 14 on the Borg 6 - 20 scale) or 'talk test', e.g. 'to be able to talk while exercising'.[91,94] General zones for various exercise intensities are shown in *Table 4*.

4.1.1.4 Training volume

Exercise intensity is inversely related to exercise time. Their product (in kcal or kJ) defines the volume of each training unit, which in turn multiplied by frequency provides an estimate of the energy expenditure of the training bout or session. The frequency of training sessions and the duration of the training period provide total energy expenditure of a training programme. Meeting the minimal activity guidelines equates to approximately 1000 kcal/week or about 10 MET/h/week (the product of MET level and duration in hours per week). Training volume should increase weekly either by 2.5% in intensity[95] or 2 mins' duration,[95] although the rate of progression should be individualized according to the biological adaptation of the individual. Training adaptation is also influenced by age, genetics,[96] fitness, and environmental factors, such as hydration, heat, cold, and altitude.[97]

4.1.1.5 Type of training

Aerobic training. Aerobic exercise training can either be continuous or interval based. There is a plethora of evidence and many guidelines on continuous aerobic exercise, but there is also strong evidence emerging about the benefits of interval-type training. The interval design involves the completion of short bouts of exercise at high intensities, interspersed with recovery periods. When compared with continuous training, this approach provides a greater challenge to the cardiopulmonary, peripheral, and metabolic systems and results in a more efficient training effect.[98] Interval training has been reported to be motivating, since the traditional continuous training can often be tedious. Interval training should be employed only in stable cardiac patients because it places a higher stress on the CV system.[99] Since intermittent training exposes subjects to near maximal effort, rest intervals of appropriate duration, preferably active ones, are recommended.[100] The exercise to rest ratio varies.[101] There are a number of different approaches used, which should be individualized according to fitness and comorbidities.

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Resistance training. *Exercise intensity:* The intensity of resistance exercise is typically prescribed in terms of one repetition maximum (1 RM). One RM is defined as the maximum amount of weight a person can lift throughout a range of motion with one repetition. Even though the performance of 1 RM appears to be a safe approach for evaluating strength[102] and no significant CV events have been reported using this approach,[103] for convenience and compliance reasons the use of multiple (usually five) repetitions (5 RM) is suggested. Five RM is the maximum amount of weight that can be performed five times. It has been reported that 1 RM can be accurately estimated from multiple repetitions and that 5 RM is an appropriate reflection of maximal strength.[104]

Exercise training zones: Resistance training using less than 20% 1 RM is generally considered aerobic endurance training. With more than 20% 1 RM, the muscular capillaries become compressed during muscle contraction resulting in a hypoxic stimulus responsible for training effects. The number of repetitions should be inversely related to the training intensity. A moderate training intensity of 30–50% 1 RM with 15–30 repetitions is considered muscular endurance training. Higher training intensities of 50–70% 1 RM with 8–15 repetitions are optimal for strength gains.

Training volume: Optimal strength gains occur when resistance training is performed 2–3 times per week. Approaches to resistance training often follow either a *station* or a *circuit* approach. In the former approach, individuals typically complete all of the sets for a given exercise per muscle group before moving to another exercise and muscle group. In the latter approach, individuals typically perform one set of a given muscle group and then rotate to another exercise and muscle group until the full set of exercises in completed per muscle group. One to three sets of 8–15 repetitions should be performed including flexion and extension of each muscle group. Multiple sets are superior to a single set.[105] A variety of 8–10 resistance exercises should be prescribed to cover most of the muscular groups.[88] Muscular power is best maintained when 3–5 min rest intervals are used instead of short rest intervals (<1 min).[106]

Mode of training: Resistance training can either be isometric (i.e. unchanged muscle length without joint movement) or dynamic (contraction with change in length of the muscle and movement of the joint throughout a range of motion). Isometric (static) muscle actions may induce a Valsalva manoeuvre at moderate to high loads, if it is not intentionally prevented by regular breathing, and may lead to an unnecessary fluctuation of BP. Dynamic training may include constant or variable resistance through the range of motion using either free weights or weight machines. In both of these modes, the type of contraction and the velocity of movement vary throughout the range of motion. This type of muscle activity mirrors muscle loading faced in daily activity. Muscles can contract in a *concentric* fashion, in which muscle shortening is exhibited during the movement, or *eccentric* fashion, in which a lengthening of the muscle occurs. Resistance training is an advanced application in which participants carry out a series of rapid concentric and eccentric muscle actions often at a relatively high load.

4.1.2 Classification of exercise and sports

A precise classification of sports by using the different components of FITT is difficult because of the differences in the type of muscular work, the mode, and the volume and intensity of exercise. Moreover, most sports consist of an isotonic and isometric muscular

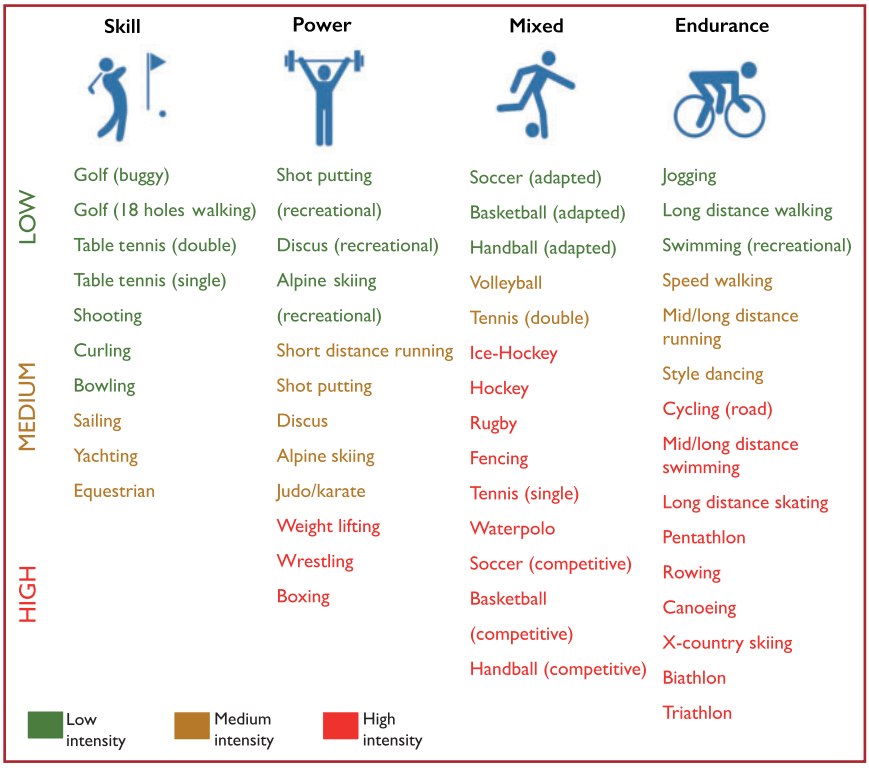

Figure 2 Sporting discipline in relation to the predominant component (skill, power, mixed, and endurance) and intensity of exercise. Intensity of exercise must be individualized after maximal exercise testing, field testing and/or after muscular strength testing (*Table 2*).

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component. For example, resistance activities can be performed in a predominantly dynamic manner or a predominantly static manner. Some sports require a high motor control component and level of skill, whereas other sports are performed at a low, moderate, high, or very high intensity. These intensities can vary depending upon the type of sport or the professional, amateur, or recreational level of performing the sports.

When providing advice regarding an exercise programme or sports participation, the physician should indicate: (i) the type of sport; (ii) frequency and duration of the exercise programme; and (iii) the intensity that appears most appropriate to the individual.

(1) Regarding the choice of the most convenient sport, the physician may indicate the type of sport as illustrated in *Figure 2* (skill, power, mixed, or endurance), with specification of the frequency, duration, and intensity of muscular work to be preferentially maintained during the exercise programme.
(2) In order to adequately prescribe the individual intensity of an endurance or mixed type of exercise or sports, the individual should perform a maximal exercise test with 12-lead ECG recording or preferentially, if possible, with simultaneous measurement of respiratory gas exchange (CPET).

Knowing a person's maximal exercise capacity allows the health professional to determine a personally tailored exercise programme that is safe and most likely to be effective. The exercise test permits the formulation of the appropriate exercise prescription based on well-recognized indices including heart rate reserve (HRR = HR_{max} - HR_{rest}), VO_2 reserve, the ventilatory threshold, or percentage of work rate for a given individual.

The exercise test also permits an assessment of any abnormal CV responses that might not otherwise be apparent during usual daily activities (including symptoms, ECG abnormalities, arrhythmias, or abnormal BP response). Based on the exercise testing results, the physician may indicate the intensity, mode, and duration of exercise that appears most suitable to the individual patient (see *Table 4*).

For power sports or resistance training, additionally maximal muscular testing is warranted in order to determine 1 RM or 5 RM. Percentage of these values, number of repetitions, and number of series will enable determination of the CV and muscular demand. Additionally, field tests will facilitate appropriate prescriptions, mainly for team sports.

When prescribing power sports for individuals with CVD, one should also consider the type of muscular work: isometric (static) or isotonic (dynamic) strength exercises. Additionally, the type and amount of exercise training, when preparing for a sport, is very important. The amount of exercise work should be adapted gradually according to the subject's actual exercise tolerance and to the anticipated level of performance.

4.2 Exercise recommendations in individuals with cardiovascular risk factors

4.2.1 General introduction

Exercise has a positive effect on several risk factors for atherosclerosis.[6] Regular exercise reduces the risk of many adverse health outcomes irrespective of age, sex, ethnicity, or the presence of comorbidities. Indeed, there is a dose–effect relationship between exercise and CV and all-cause mortality, with a 20–30% reduction in adverse events compared with sedentary individuals.[107,108] Consequently, European Guidelines recommend that healthy adults of all ages should perform a minimum of 150 min of moderate-intensity endurance exercise training over 5 days or 75 min of vigorous exercise per week over 3 days, with additional benefit derived by doubling the amount to 300 min of moderate-intensity or 150 min of vigorous-intensity aerobic PA per week.[6]

While exercise is also beneficial in patients with established CVD, the risk associated with vigorous exercise and sports in these individuals is increased. Importantly, CVD may be subclinical and unrecognized; therefore, consideration should be given to pre-participation assessment of risk in individuals with a higher likelihood of CVD. Individuals with multiple risk factors are more likely to develop CVD. Assessment of the individual likelihood of subclinical CVD may be performed by calculating the accumulated risk through established risk scores such as the SCORE risk charts (*Figure 3*) and considering

Table 4 Indices of exercise intensity for endurance sports from maximal exercise testing and training zones

Intensity	*VO_2max (%)*	*HRmax (%)*	*HRR (%)*	*RPE Scale*	*Training Zone*
Low intensity, light exercise[a]	<40	<55	<40	10–11	Aerobic
Moderate intensity exercise[a]	40–69	55–74	40–69	12–13	Aerobic
High intensity[a]	70–85	75–90	70–85	14–16	Aerobic + lactate
Very high intense exercise[a]	>85	>90	>85	17–19	Aerobic + lactate + anaerobic

HR_{max} = maximum heart rate; HRR = heart rate reserve; RPE = rate of perceived exertion; VO_{2max} = maximum oxygen consumption.
[a]Adapted from refs [84,85] using training zones related to aerobic and anaerobic thresholds. Low-intensity exercise is below the aerobic threshold; moderate is above the aerobic threshold but not reaching the anaerobic zone; high intensity is close to the anaerobic zone; and very intense exercise is above the anaerobic threshold. The duration of exercise will also largely influence this division in intensity.

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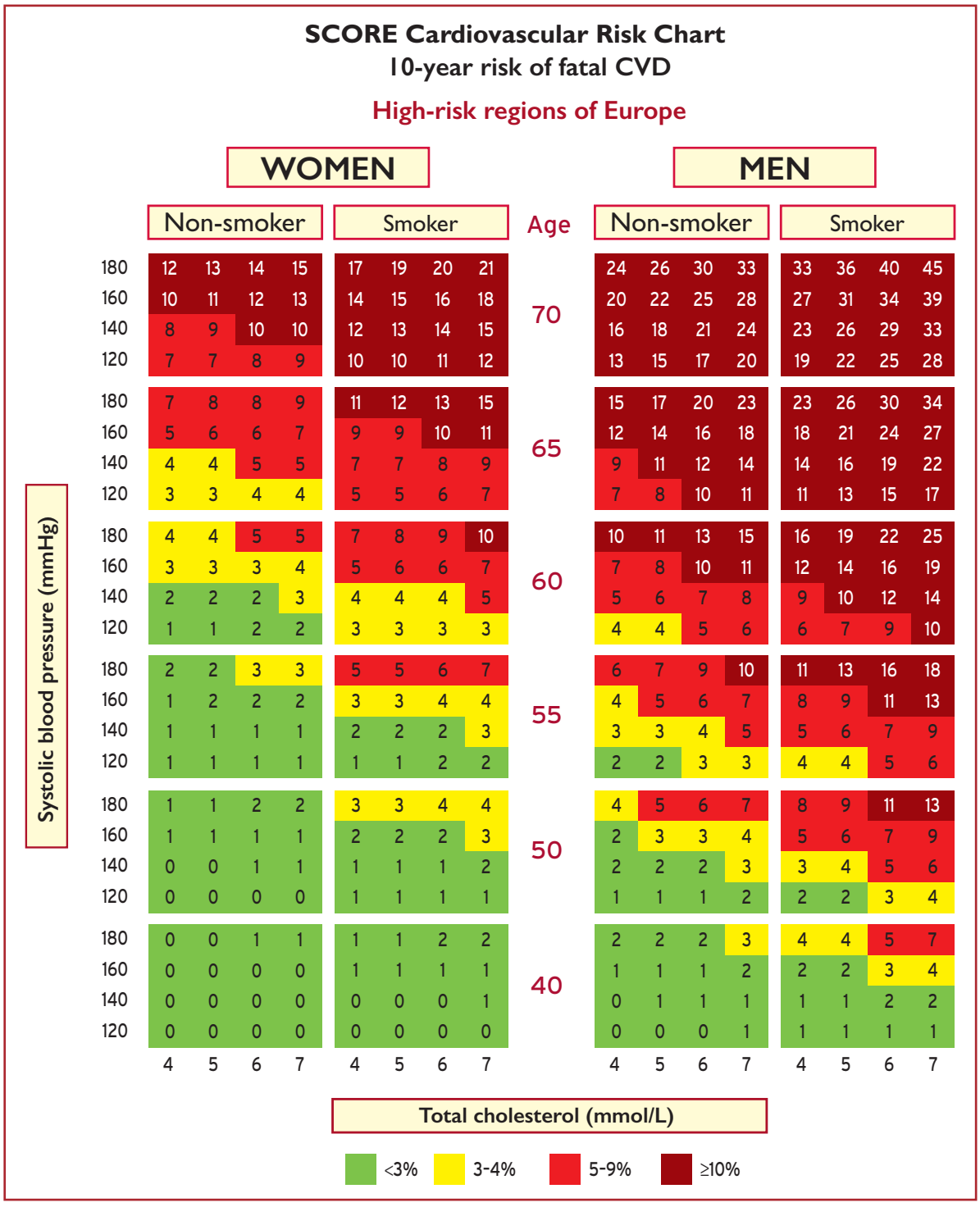

Figure 3a *SCORE charts for European populations at high cardiovascular disease (CVD) risk*[109]. *The 10-year risk of fatal CVD in populations at high CVD risk is based on the following risk factors: age, gender, smoking, systolic blood pressure, and total cholesterol. To convert the risk of fatal CVD to risk of total (fatal + non-fatal) CVD, multiply by 3 in men and 4 in women, and slightly less in older people. Note: the SCORE chart is for use in people without overt CVD, diabetes (type 1 and 2), chronic kidney disease, familial hypercholesterolaemia, or very high levels of individual risk factors because such people are already at high risk and need intensive risk factor advice. Cholesterol: 1 mmol/L = 38.67 mg/dL. The SCORE risk charts presented above differ slightly from those in the 2016 ESC/EAS Guidelines for the* management of dyslipidaemias and the 2016 European Guidelines on cardiovascular disease prevention in clinical practice, in that: *(i) age* has been extended from 65 to 70 years; *(ii) the interaction between age and each of the other risk factors has been incorporated, thus reducing the overestimation* of risk in older persons in the original SCORE charts; *(iii) the cholesterol band of* 8 mmol/L has been removed since such persons will qualify for further evaluation in any event. SCORE = Systematic Coronary Risk Evaluation.

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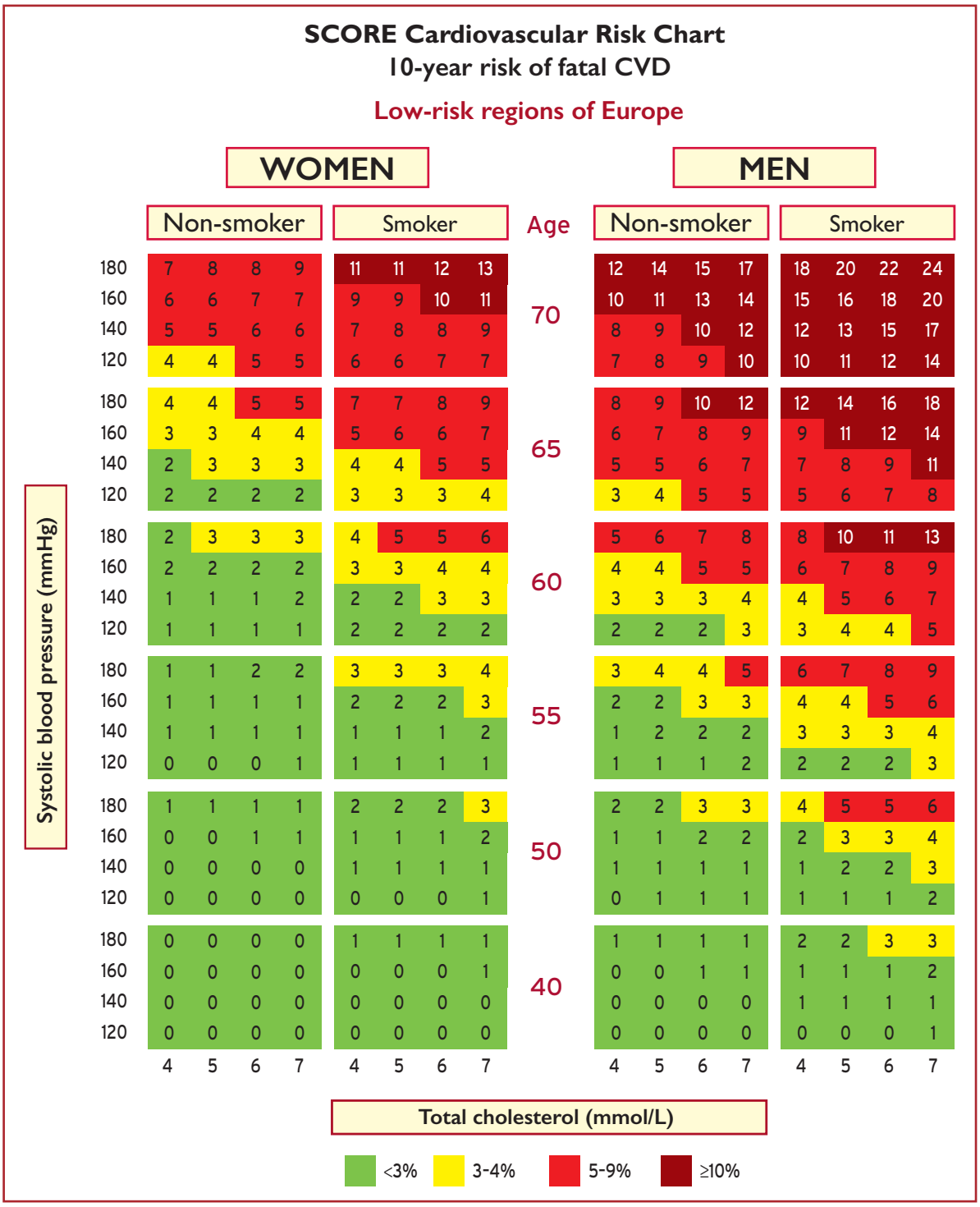

Figure 3b SCORE chart for European populations at low cardiovascular disease (CVD) risk. The 10-year risk of fatal CVD in populations at low CVD risk is based on the following risk factors: age, gender, smoking, systolic blood pressure, and total cholesterol. To convert the risk of fatal CVD to risk of total (fatal + non-fatal) CVD, multiply by 3 in men and 4 in women, and slightly less in older people. Note: the SCORE chart is for use in people without overt CVD, diabetes (type 1 and 2), chronic kidney disease, familial hypercholesterolaemia, or very high levels of individual risk factors because such people are already at high risk and need intensive risk factor advice. Cholesterol: 1 mmol/L = 38.67 mg/dL. The SCORE risk charts presented above differ slightly from those in the 2016 ESC/EAS Guidelines for the Management of Dyslipidaemias and 2016 European Guidelines on cardiovascular disease prevention in clinical practice, in that: (i) age has been extended from 65 to 70 years; (ii) the interaction between age and each of the other risk factors has been incorporated, thus reducing the overestimation of risk in older persons in the original SCORE charts; (iii) the cholesterol band of 8 mmol/L has been removed since such persons will qualify for further evaluation in any event. SCORE = Systematic Coronary Risk Evaluation.

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individual risk factors such as very high total cholesterol and low-density lipoprotein (LDL), diabetes mellitus, or a strong family history of CVD.[6] Based on this assessment the individual CV risk can be categorized from low to very high risk (*Table 5*).

Preliminary evaluation should consist of a self-assessment relating to symptoms and calculation of SCORE. Individuals who are habitually active and at low or moderate risk should not have any restrictions for exercise including competitive sports. Sedentary individuals and individuals at high or very high risk may engage in low-intensity exercise without further evaluation. Sedentary individuals and/or those at high or very high risk planning to undertake high-intensity exercise as well as selected individuals planning to undertake moderate-intensity exercise should undergo a physical examination, 12-lead ECG, and exercise stress test. The aim of the exercise test is to identify prognostically important CAD and to assess the presence of exercise-induced arrhythmias. Individuals with symptoms, abnormal findings on physical examination, abnormal ECG, or abnormal exercise test should be investigated further according to current ESC Guidelines for chronic coronary syndromes.[110] Following normal investigations, there should be no restrictions to sports participation. All individuals should, however, be thoroughly informed that development of symptoms during exercise should prompt reassessment.

While a normal exercise test and a high exercise capacity is associated with a good prognosis, the test has limited sensitivity in diagnosing mild to moderate obstructive CAD.[111,112] Currently there is no evidence for incorporating routine cardiac imaging in pre-participation screening among asymptomatic individuals aged >35 years old with a normal exercise stress test. However, in asymptomatic adults considered to be at high risk or very high risk (diabetes, strong family history of CAD, previous risk assessment suggesting high risk for CAD) a functional imaging test or coronary computed tomography angiography (CCTA) should be considered in the risk assessment (*Figure 4*).[110] Identification of atherosclerotic CAD should prompt aggressive management of risk factors and preventive medical treatment. Among individuals with proven obstructive CAD, further assessment and treatment is indicated.

Table 5 Cardiovascular risk categories

Very high-risk	People with any of the following: • Documented ASCVD, either clinical or unequivocal on imaging. Documented ASCVD includes previous ACS (MI or unstable angina), stable angina, coronary revascularization (PCI, CABG, and other arterial revascularization procedures), stroke and TIA, and peripheral arterial disease. Unequivocally documented ASCVD on imaging includes those findings that are known to be predictive of clinical events, such as significant plaque on coronary angiography or CT scan (multivessel coronary disease with two major epicardial arteries having >50% stenosis), or on carotid ultrasound. • DM with target organ damage,[a] or at least three major risk factors, or early onset of T1DM of long duration (>20 years). • Severe CKD (eGFR <30 mL/min/1.73 m^2). • A calculated SCORE ≥10% for 10-year risk of fatal CVD. • FH with ASCVD or with another major risk factor.
High-risk	People with: • Markedly elevated single risk factors, in particular TC >8 mmol/L (>310 mg/dL), LDL-C >4.9 mmol/L (>190 mg/dL) , or BP ≥180/110 mmHg. • Patients with FH without other major risk factors. • Patients with DM without target organ damage,[a] with DM duration ≥10 years or another additional risk factor. • Moderate CKD (eGFR 30–59 mL/min/1.73m^2). • A calculated SCORE ≥5% and <10% for 10-year risk of fatal CVD.
Moderate-risk	Young patients (T1DM <35 years; T2DM <50 years) with DM duration <10 years, without other risk factors. Calculated SCORE ≥1% and <5% for 10-year risk of fatal CVD.
Low-risk	Calculated SCORE <1% for 10-year risk of fatal CVD.

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General recommendations for exercise and sports in healthy individuals

Recommendations	Class[a]	Level[b]
At least 150 min/week of moderate-intensity, or 75 min/week of vigorous-intensity aerobic exercise, or an equivalent combination thereof is recommended in all healthy adults.[113–118]	I	A
A gradual increase in aerobic exercise to 300 min/week of moderate-intensity, or 150 min/week of vigorous-intensity aerobic exercise, or an equivalent combination is recommended for additional benefits in healthy adults.[114,116]	I	A
Regular assessment and counselling to promote adherence and, if necessary, to support an increase in exercise volume over time are recommended.[119]	I	B
Multiple sessions of exercise spread throughout the week, i.e. on 4–5 days a week and preferably every day of the week, are recommended.[113,114]	I	B

[a]Class of recommendation.
[b]Level of evidence.

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Recommendations for cardiovascular evaluation and regular exercise in healthy individuals aged >35 years

Recommendations	Class[a]	Level[b]
Among individuals with low to moderate CVD risk, the participation in all recreational sports should be considered without further CV evaluation.	IIa	C
Cardiac screening with family history, symptoms, physical examination, and 12-lead resting ECG should be considered for competitive athletes.	IIa	C
Clinical evaluation, including maximal exercise testing, should be considered for prognostic purposes in sedentary people and individuals with high or very high CV risk who intend to engage in intensive exercise programmes or competitive sports.	IIa	C
In selected individuals without known CAD who have very high CVD risk (e.g. SCORE>10%, strong family history, or familial hypercholesterolaemia) and want to engage in high- or very high-intensity exercise, risk assessment with a functional imaging test, coronary CCTA, or carotid or femoral artery ultrasound imaging may be considered.	IIb	B

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CCTA = coronary computed tomography angiography; CV = cardiovascular; CVD = cardiovascular disease; SCORE = Systematic Coronary Risk Evaluation.
[a]Class of recommendation.
[b]Level of evidence.

4.2.2 Obesity

A person with a body mass index (BMI) >30 kg/m^2 or (preferentially) a waist circumference >94 cm for males and >80 cm for females (both for European Caucasians) is considered obese.[120,121]

European guidelines for obese individuals recommend that a minimum of 150 min/week of moderate-intensity endurance exercise training should be combined with three weekly sessions of resistance exercise.[121] Such intervention leads to a reduction in intra-abdominal fat mass, increments in muscle and bone mass, attenuation in the weight loss-induced decline of resting energy expenditure, reduction in BP and chronic inflammation, and improvement in glucose tolerance, insulin sensitivity, lipid profile, and physical fitness.[121,122] There is also a positive influence on the long-term maintenance of weight reduction, general well-being and self-esteem, and reduction in anxiety and depression.[121] The impact of exercise intervention alone on fat mass is modest.[123] According to a series of large randomized controlled trials a high endurance-type exercise volume, >225 min/week, is required to maximize fat mass loss in obese individuals.[124]

A pre-participation CV assessment is warranted in obese individuals who intend to engage in high-intensity exercise (*Figure 4*), due to associated comorbidities such as type 2 diabetes, hypertension, dyslipidaemia, and CV and respiratory diseases.[121] Obese individuals with a normal CV assessment should not have any restrictions on exercise. There is evidence from healthy, non-obese and non-athletic individuals that running, and abrupt increases in training volume, contribute to musculoskeletal injuries.[125–127] Therefore, it may be reasonable to consider that obese individuals should limit high-volume weight-bearing exercises on a hard surface (i.e. <2 h/day) until a considerable

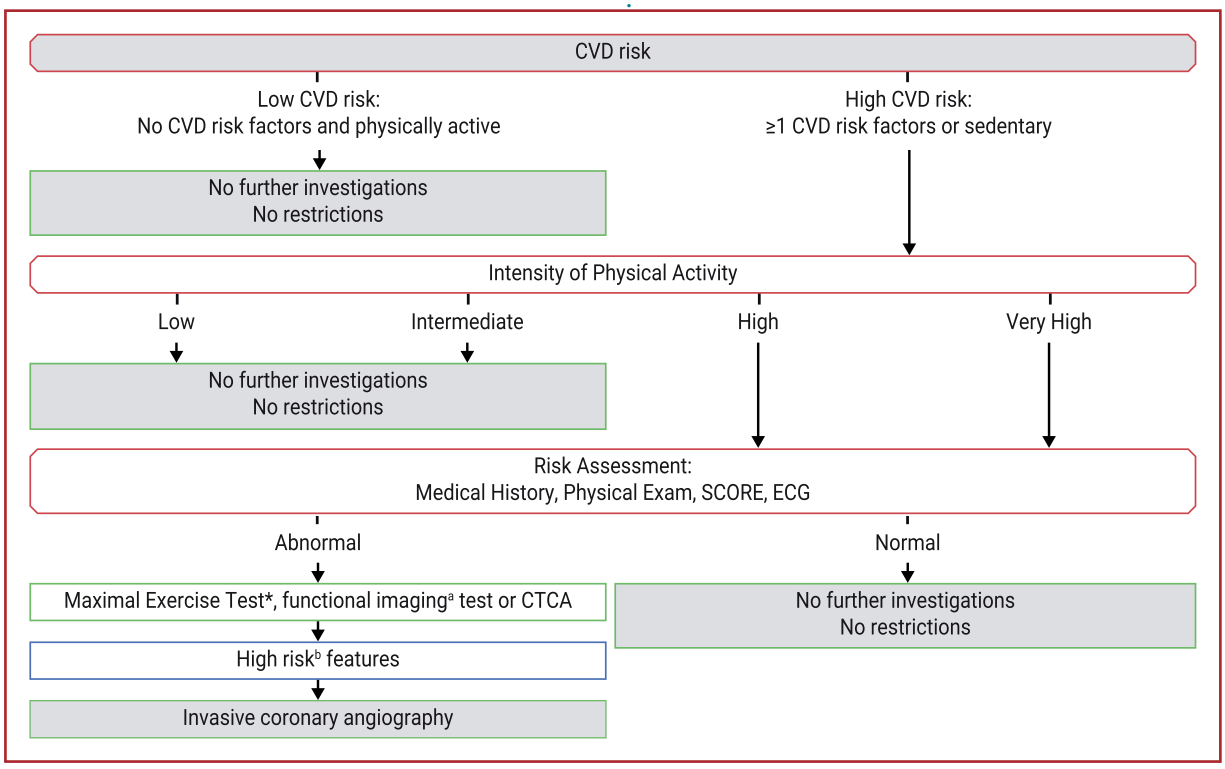

Figure 4 Proposed algorithm for cardiovascular assessment in asymptomatic individuals aged >35-years-old with risk factors for cardiovascular disease and possible subclinical chronic coronary syndrome before engaging in sports. *Consider functional test or CCTA if exercise stress test is equivocal or the ECG is uninterpretable. [a]See text for examples of functional imaging. [b]Single-photon emission computed tomography: area of ischaemia ≥10% of the left ventricular myocardium; stress echocardiography: ≥3 of 16 segments with stress-induced hypokinesia or akinesia; stress cardiovascular magnetic resonance: ≥2 of 16 segments with stress perfusion defects or ≥3 dobutamine-induced dysfunctional segments; coronary computed tomography angiography (CCTA): three-vessel disease with proximal stenoses; left main disease; proximal left anterior descending disease.[110] CVD = cardiovascular disease; ECG = electrocardiogram; SCORE = Systematic Coronary Risk Evaluation.

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reduction in body weight is achieved. Moreover, if high-volume exercise (>2 h/day) is desired, a sufficient recovery time should be allowed for between periods of exercise (optimally 48 h). It is important to emphasize that good physical and muscular fitness and neuromuscular coordination may protect obese individuals from musculoskeletal injuries, hence non-weight-bearing exercises such as cycling or swimming[128] may be beneficial. Finally, there is no compelling evidence that resistance training, when executed properly, will increase the risk for musculoskeletal injuries or provoke musculoskeletal symptoms in obese individuals.[129]

4.2.3 Hypertension

A person with a persistent systolic BP (SBP) ≥140 mmHg and/or diastolic BP (DBP) ≥90 mmHg is considered hypertensive.[130,131] Hypertensive individuals should participate in at least 30 min of moderate-intense dynamic aerobic exercise (walking, jogging, cycling, or swimming) for 5−7 days per week.[132] Such exercise intervention is associated with a mean reduction in SBP of 7 mmHg and DBP of 5 mmHg.[133] Additional resistance training is highly effective in reducing BP further and resistance training 2−3 days per week is also advised.[132] Indeed, the BP-lowering effect of resistance and isometric exercise may be comparable to, or even greater than, that of aerobic exercise.[134]

If high-intensity sports participation is desired, a pre-participation CV assessment is warranted to identify athletes with exercise-induced symptoms, excessive BP response to exercise[130], and the presence of end organ damage. Individuals with symptoms suggestive of CAD require further assessment and optimization of medical therapy before participation in sports. If arterial hypertension is poorly controlled (resting SBP > 160 mmHg), a maximal exercise test should be postponed until the BP is controlled.

Non-pharmacological measures should be considered as the first step in the management of hypertension in athletes, including: restriction of salt intake and alcohol consumption, weight reduction if applicable, balanced diet (e.g. Mediterranean diet), and cessation of smoking. Aerobic exercise programmes should herein complement the individual's training schedule.[131] If such lifestyle changes do not lower BP after 3 months, antihypertensive drugs should be commenced if SBP remains >140 mmHg. Antihypertensive therapy alongside lifestyle intervention should be considered in all individuals aged >65 years but <80 years, provided it is well tolerated.[131,132] It is important to consider that beta-blockers are prohibited in certain competitive skill sports such as shooting [see World Anti-Doping Association (WADA) for complete list[135]], and can induce bradycardia and/or lower aerobic exercise capacity.[131] Diuretics are prohibited in all competitive sports.[135]Angiotensin-converting enzyme (ACE) inhibitors, angiotensin II receptor blockers, and calcium antagonists are the preferred drugs of choice in exercising individuals. It is noteworthy that the use of non-selective non-steroidal inflammatory drugs for musculoskeletal pain may contribute to raised BP.[136]

When BP is uncontrolled, temporary restriction from competitive sports is recommended, with the possible exception of skill sports.[131] In individuals with a high-risk profile, including those with end organ damage [left ventricular (LV) hypertrophy, diastolic dysfunction, ultrasound evidence of arterial wall thickening or atherosclerotic plaque, hypertensive retinopathy, increased serum creatinine (men 1.3−1.5 mg/dL, women 1.2−1.4 mg/dL), and/or microalbuminuria] in whom BP is controlled, participation in all competitive sports is possible, with the exception of the most intensive power disciplines such as discus/javelin throwing, shot-putting, and weightlifting (see *section 4.1*).[131]

During sports participation, regular follow-up is recommended depending on the severity of hypertension and the category of risk. In individuals with borderline BP readings, regular ambulatory assessment of BP should be considered. In individuals with low or moderate CV risk and well-controlled BP, there should be no restrictions to sports participation, however, intensive heavy weightlifting, especially when this includes substantial isometric (static) muscle work, can have a marked pressor effect and should be avoided. In this context, avoiding the Valsalva manoeuvre in particular is warranted because breath holding during muscular contraction is associated with a greater elevation in SBP and DBP.[135] When executed correctly, high-intense dynamic resistance training (up to 80% of 1 RM), with a low number of repetitions (n < 10) does not induce greater increments in BP compared with low-intense dynamic resistance training (<50% of 1 RM) with a high number of repetitions (n ≥ 20).[137−142]

Some individuals who are normotensive at rest will have an exaggerated BP response to exercise. An exaggerated BP response to exercise increases the risk for incident hypertension in highly trained and normotensive athletes over a middle-term period.[143] If SBP rises to >200 mmHg at a workload of 100 W during exercise testing,[144] antihypertensive medical therapy should be optimized and clinical evaluation, including ECG and echocardiography, should be considered, even if the athlete is normotensive at rest.[131] Moreover, in young Olympic athletes a peak SBP of >220 mmHg in males and >200 mmHg in females measured during cycle ergometry are beyond the 95th percentile.[131]

4.2.4 Dyslipidaemia

Physical activity has favourable effects on lipid metabolism by reducing serum triglycerides by up to 50% and increasing high-density lipoprotein (HDL) cholesterol by 5−10%.[85,145] Exercise may also reduce LDL cholesterol by up to 5% and shift the more atherogenic small, dense LDL fraction towards larger LDL particles in a dose-dependent fashion.[146] These metabolic improvements can be achieved through 3.5 - 7 h of moderately vigorous PA per week or 30 - 60 min of exercise on most days.

In individuals with hypertriglyceridaemia or hypercholesterolaemia, a higher intensity of exercise is recommended, as this may

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improve the lipid profile and reduce CV risk. Before embarking on high-intensity exercise, a clinical assessment should be performed including symptomatic status, and a maximal exercise stress test, functional imaging test, or CCTA may be considered in the risk assessment[110] (*Figure 4*), particularly in individuals with familial hypercholesterolaemia. Among athletes with hypercholesterolaemia, regular exercise will rarely reduce LDL cholesterol to normal or near-normal values; therefore, guidelines on pharmacological treatment in primary and secondary prevention should be followed strictly. Individuals with dyslipidaemia should be assessed at least every 2–5 years for primary prevention and annually for secondary prevention.

Pharmacological intervention, particularly with statins, is superior to exercise and lifestyle intervention alone for reducing LDL cholesterol and improving prognosis.[147] Despite the minor effects of endurance exercise on serum LDL cholesterol, the clinically beneficial relationship between increased physical fitness and reduced CV events remains beyond the effects of statins.[147,148]

Physically active individuals with dyslipidaemia may experience muscle pain and soreness or tendinopathy accompanied by elevated muscle enzymes.[149] In these cases, measures such as stopping medication temporarily followed by repeat challenge with another statin drug, with or without an alternate day regimen, or introducing other lipid-lowering agents such as ezetimibe or proprotein convertase subtilisin/kexin type 9 (PCSK-9) inhibitors should be considered.[109] Individuals who develop rhabdomyolysis due to a statin should be prescribed an alternative lipid-lowering agent.

4.2.5 Diabetes mellitus

Physical inactivity is a major cause of type 2 diabetes mellitus (T2DM).[150] The risk of developing T2DM is 50–80% higher in individuals who are physically inactive compared to their active counterparts. However, exercise does not entirely compensate for the effect of obesity.[151–154] Diabetes is also independently associated with an accelerated decline in muscular strength and, partly because of hyperglycaemia, may lead to reduced joint mobility.

4.2.5.1 Effect of exercise on diabetic control, risk factors and outcomes

Aerobic exercise in patients with T2DM improves glycaemic control and reduces visceral fat and insulin resistance. Exercise also has beneficial effects on BP and lipid profile, and leads to modest weight loss.[155,156] Both aerobic and resistance training promote prolonged adaptations in skeletal muscle, adipose tissue, and the liver associated with enhanced insulin action.[157] Observational studies have shown lower mortality with exercise in both type 1 diabetes mellitus and T2DM.[158]

In patients with pre-diabetes or metabolic syndrome, both aerobic and resistance exercise may prevent the development of overt diabetes.[159–162] Intensity of exercise seems to be of greater importance than the volume of exercise; individuals who exercise at moderate or high intensity have a lower risk of developing metabolic impairment compared with those who have a similar energy expenditure at a lower intensity.[160,163]

The effects on muscle insulin sensitivity are observed with a relatively low volume of exercise (400 kcal/week) in previously sedentary adults, but increase with higher volumes of exercise.[164] The optimal combination of duration and intensity is not well established. High-intensity interval training may be superior to moderate aerobic training in achieving metabolic effects and improvement in exercise capacity; however, whether long-term results are superior is unknown.[165,166]

Diabetes is a cause of coronary microvascular dysfunction (CMD), which is associated with lower exercise capacity and adverse outcomes[167,168] and can be improved by exercise training.[162,169–171] Large randomized trials have confirmed the beneficial effect of exercise intervention on glycaemic control and risk factors, but this has not translated into a significant improvement in survival, partly because of suboptimal long-term maintenance of lifestyle changes.[172]

During an acute bout of exercise, glucose uptake in the muscles is increased for up to 2 h afterwards through mechanisms that are independent of insulin. The exercise-induced hypoglycaemic effect can be diminished by performing resistance training or interval training in patients with type 1 diabetes.[173] There is a dose–response relationship between intensity and volume of exercise and duration of glucose uptake by skeletal muscle that may last up to 48 h after exercise. These factors must be considered in individuals with diabetes who are undertaking intensive exercise or competitive sports in order to avoid hypoglycaemia.

4.2.5.2 Recommendations for participation in exercise in individuals with diabetes mellitus

Both aerobic and resistance training are effective for glycaemic control, BP reduction, weight loss, peak exercise capacity, and dyslipidemia.[174] A programme combining aerobic and resistance training has been shown to be superior in terms of glycaemic control, whereas the effect on other outcomes is unproven.[174–176]

The ideal exercise programme to achieve the full potential of benefits in patients with diabetes is daily exercise of at least moderate intensity, e.g. brisk walking, for at least 30 min, resistance training for 15 min on most days, and lighter-intensity activities (standing, walking) every 30 min. This can be supplemented by flexibility and balance exercise, particularly in older individuals or patients with microvascular complications due to their diabetes.

4.2.5.3 Cardiac evaluation before exercise participation in individuals with diabetes mellitus

Individuals with diabetes have a priori a higher likelihood of subclinical CAD; therefore, all individuals with diabetes should undergo CV assessment as outlined in *Figure 4* before taking up an exercise programme of high intensity. This should be supplemented by an evaluation of glycaemic status, including risk factors for hypoglycaemia, history of hypoglycaemic episodes, presence of autonomic neuropathy, and antidiabetic treatment.[177]

Asymptomatic individuals with diabetes mellitus and a normal CV assessment and maximal exercise test may engage in all sports but should be warned about the potential risk of iatrogenic

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hypoglycaemia in the event of inadequate caloric intake. Importantly, all patients with diabetes should be aware of warning symptoms and attention should be given to chest discomfort or unusual breathlessness during exercise as this may be indicative of CAD.

Special considerations for individuals with obesity, hypertension, dyslipidaemia, or diabetes

Recommendations	Class[a]	Level[b]
In obese individuals (BMI≥30 kg/m^2 or a waist circumference >80 cm for females or >94 cm for males) resistance training ≥3 times per week, in addition to moderate or vigorous aerobic exercise (at least 30 min, 5–7 days per week) is recommended to reduce CVD risk.[121]	I	A
In individuals with well-controlled hypertension, resistance training ≥3 times per week in addition to moderate or vigorous aerobic exercise (at least 30 min, 5–7 days per week) is recommended to reduce blood pressure and CVD risk.[132]	I	A
Among individuals with diabetes mellitus, resistance training ≥3 times per week in addition to moderate or vigorous aerobic exercise (at least 30 min, 5–7 days per week) is recommended to improve insulin sensitivity and achieve a better CVD risk profile.[176,178]	I	A
Among adults with well-controlled hypertension but high risk and/or target organ damage, high-intensity resistance exercise is not recommended.	III	C
In individuals with uncontrolled hypertension (SBP>160 mmHg) high-intensity exercise is not recommended until blood pressure has been controlled.	III	C

CVD = cardiovascular disease; SBP = systolic blood pressure.
[a]Class of recommendation.
[b]Level of evidence.

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4.3 Exercise and sports in ageing

4.3.1 Introduction

The elderly are defined as adults aged above 65 years. Similar to the general population, higher exercise capacity in this age group is also associated with reduced mortality.[179] A physically active lifestyle maintained through middle and older age translates into better health[180] and longevity.[181–185] Commencing a new exercise regimen among sedentary elderly individuals has shown significant health improvements[180,186] including cognitive capacity.[187–190] Moreover, regular exercise exerts beneficial effects in reducing the risk of developing CV and metabolic disease through improved control of CV risk factors,[191,192] also preserving cognitive function.[187–190] Importantly, exercise helps to preserve neuromuscular competence,[193,194] thus maintaining balance and coordination, which reduces the risk of falling.[195,196]

4.3.2 Risk stratification, inclusion/exclusion criteria

Moderate-intensity exercise is generally safe for older healthy people and medical consultation before starting or progressing the level of exercise programme is not usually required.[81,197] The general recommendation for exercise implementation for the general population also applies to healthy elderly people.

Nevertheless, due to potential risks of exercising among the elderly (*Table 6*), the European Association of Preventive Cardiology (EAPC) recommends self-assessment by a brief questionnaire[81] to determine the need for advice from health professionals, but this approach has not been tested prospectively.

Community-dwelling frail or sedentary older adults may have a slightly increased risk of falls during exercise; however, there is no evidence of serious adverse outcomes, injury, or CV events.[195,196,198,199] Exercise interventions to improve balance in those diagnosed with dementia bring numerous benefits without an increased risk of adverse outcomes.[200] Resistance exercise in older adults is rarely associated with adverse events.[201,202] No major risks have been reported in older individuals performing low- and moderate-intensity aerobic exercise, and even more intense aerobic activities are associated with a relatively small risk.[203–205] CV events during intense exercise occur at a rate of around 1 event per 100 years of vigorous activity.[206] Risks are highest during the first few weeks of beginning vigorous exercise; therefore both exercise intensity and duration should be increased gently (for example, every 4 weeks).[81,197,207–210] Among older individuals who are well prepared and accustomed to intense exercise, participation in competitive vigorous sports does not confer higher risk compared with younger adults.[38,211]

4.3.3 Exercise modalities and recommendations for exercise and sport in the elderly

The physical exercises for elderly persons should be designed according to their biological age, exercise experience, functional capacity, safety, ageing trajectories, comorbidity, lifestyle habits, and previous experience of exercise.

Elderly people should perform endurance and strength exercise, and specific exercises for flexibility and balance (*Table 7*).[201,212,213] Endurance exercise exerts beneficial effects on the cardiorespiratory system and resistance exercise prevents the decrease in muscle mass and sarcopenia.[192] Achieving >150 min/week moderate-intensity aerobic exercise (i.e. walking or other moderate intensity aerobics-type activities) is associated with at least 30% lower risk of morbidity, mortality, disability, frailty, and dementia compared with being inactive.[212,214,215] The strength exercises for the major muscle groups should be performed at least twice a week (8–10 different exercises, 10–15 repetitions).

Accustomed senior athletes should continue performing exercise and sports activities, without any predetermined age limit.[38,211,216] Sports activities for older people according to exercise type and intensity are reported in *Table 8*. Annual clinical assessment including a maximal exercise test (preferably with simultaneous CPET) is recommended in master athletes performing a high level of sports and exercise programmes.[217]

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Recommendations for exercise in ageing individuals

Recommendations	Class[a]	Level[b]
Among adults aged 65 years or older who are fit and have no health conditions that limit their mobility, moderate-intensity aerobic exercise for at least 150 min/week is recommended.[212,214,215]	I	A
In older adults at risk of falls, strength training exercises to improve balance and coordination on at least 2 days a week are recommended.[201,212,214,215]	I	B
A full clinical assessment including a maximal exercise test should be considered in sedentary adults aged 65 years or older who wish to participate in high-intensity activity.	IIa	C
Continuation of high- and very high-intensity activity, including competitive sports, may be considered in asymptomatic elderly athletes (master athletes) at low or moderate CV risk.	IIb	C

CV = cardiovascular.
[a]Class of recommendation.
[b]Level of evidence.

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Table 6 Potential risks for older people during exercise

- Arrhythmias, increase in blood pressure, myocardial ischaemia
- Musculoskeletal injuries and fractures
- Muscle soreness or swollen joints
- Increased risk of falls and subsequent injuries

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Table 7 Exercise prescription in the elderly

Aerobic work
- *Frequency:* Moderate exercise for 5 days per week or vigorous exercise for 3 days per week.
- *Intensity:* 5–6 points (for the modified 10 point Borg scale) for moderate exercise or 7–8 points for vigorous
- *Duration:* 30 minutes per day for moderate or at least 20 minutes for continuous exercise.

Strength training (all major muscle groups)
- *Frequency:* at least twice a week
- *Number of exercises:* 8–10
- *Number of repetitions:* 10–15

Exercises for flexibility and balance
- At least twice a week

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Table 8 Exercise activities for older people according to exercise type and intensity

Age-related moderate effort activities
- walking
- water aerobics
- ballroom and line dancing
- riding a bicycle on level ground or with few hills
- doubles tennis
- pushing a lawn mower
- canoeing
- volleyball

Age-related intense effort activities
- jogging or running
- aerobics
- swimming fast
- riding a bicycle fast or on hills
- singles tennis
- football
- hiking uphill
- energetic dancing
- martial arts

Muscle-strengthening activities
- carrying or moving heavy loads
- groceries activities that involve stepping and jumping
- dancing
- heavy gardening, such as digging or shovelling
- exercises that use your body weight for resistance, such as push-ups or sit-ups
- yoga
- pilates
- lifting weights

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5. Exercise in clinical settings

5.1 Exercise programmes for leisure-time and competitive sport participation in chronic coronary syndrome

Atherosclerotic CAD is the predominant cause of exercise-related (Ex-R) cardiac events including ACS, AMI, and SCA in individuals with established chronic coronary syndrome (CCS), or SCD as a primary presentation in individuals >35 years of age.[218] In addition to atherosclerotic CAD, other entities, including an anomalous origin of a coronary artery (AOCA),[219] myocardial bridge (MB),[220] and spontaneous coronary artery dissection (SCAD),[221] are also associated with myocardial ischaemia, and potentially with Ex-R SCD.

Physical inactivity is a risk factor for CAD, but somewhat paradoxically, vigorous physical exertion transiently increases the risk for AMI[66] and SCD.[216] Overall, the benefits of regular exercise greatly outweigh the Ex-R risk, even in individuals with CCS. Moderate- to vigorous-intensity exercise is strongly associated with a reduced

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incidence of adverse outcomes from CAD, but prolonged, high-intensity endurance exercise has been associated with increased coronary artery calcium (CAC), a marker of atherosclerosis,[58,222] and coronary plaques[58] but without an increase in mortality[112] in the medium term. Importantly, the diagnosis of myocardial injury is also more complex in athletes because intense exercise may increase serologic markers of myocardial injury, including cardiac troponin I and T.[223,224]

5.1.1 Individuals at risk of atherosclerotic coronary artery disease and asymptomatic individuals in whom coronary artery disease is detected at screening

Athletes or individuals participating in sports or regular exercise training may have risk factors for CAD and/or subclinical CCS.[225] Such individuals may be identified by routine pre-participation screening as recommended by the ESC[21] or by pre-evaluation of master athletes, as suggested by the European Association for Cardiovascular Prevention and Rehabilitation (EACPR) 2011[207] and the AHA.[226]

In addition to the SCORE risk stratification described earlier (*Table 5*), the increasing use of cardiac imaging techniques allows the identification of a greater number of individuals with asymptomatic CCS,[227] including competitive master athletes.[227]

Newer predictive measures, such as high-sensitive C-reactive protein and carotid intima–media thickness (IMT) add little to the traditional risk factors.[110] The exception is CAC, which provides additional predictive information in individuals with a moderate-risk profile,[228] dividing them into low- or high-risk individuals. The most prudent and cost-effective method of utilizing CAC may thus be additive to the traditional risk factors,[229] as suggested by the EAPC.[230]

Clinical evaluation of asymptomatic individuals with possible subclinical CCS should include (*Figure 4*):[112]

(1) Assessment of risk of CVD[110] (*Table 5*)
(2) Consideration of intensity of intended exercise programme
(3) Clinical evaluation, including maximal exercise stress test
(4) Further diagnostic testing in selected individuals.

Many middle-aged individuals in the general population can be expected to have some level of subclinical CCS as assessed with imaging techniques. Anatomical coronary imaging alone does not provide information relating to the coronary flow and reserve, which is important in assessing the risk of Ex-R ischaemia or SCD/SCA; therefore functional evaluation is necessary. Several methods of stress testing (e.g. cycle ergometry or treadmill testing), stress echocardiography, adenosine or dobutamine stress cardiac magnetic resonance (CMR), or positron emission tomography (PET)/single-photon emission computed tomography (SPECT), can be used to detect inducible myocardial ischaemia[231]. Exercise stress-echo is preferred in athletes because it is free from radiation and does not involve administration of drugs.

Exercise testing is the most widely available functional test, and provides information on exercise capacity, heart rate, and BP response, and detection of exercise-induced arrhythmias,[2] but has a lower specificity for myocardial ischaemia than other functional tests, especially in asymptomatic and low-risk individuals. It is recommended that a truly maximal exercise test[232] (with or without CPET) should be performed when evaluating individuals with possible subclinical (or clinical) CCS who intend to or are participating in systematic exercise including recreational or competitive sports. Whether the initial exercise test includes imaging or not depends on factors such as the baseline ECG (*Table 9*) and feasibility of performing functional imaging tests in a given institute.

Table 9 Borderline or uninterpretable ECG findings

(i) ST depression of ≤ -0.15 mV; in one lead only
(ii) not typically ascending/down sloping ST segment
(iii) pre-existing left bundle branch block (LBBB)
(iv) ventricular pacing

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- If the clinical assessment, including a maximal exercise test is *normal*, the presence of 'relevant CAD' is assumed to be unlikely (*Figure 4*).
- In the event of a *borderline* or uninterpretable exercise test result, it is recommended that a more specific imaging stress test is performed such as stress-echocardiography, CMR perfusion imaging, or SPECT. Maximal exercise SPECT and exercise echocardiography or nuclear perfusion techniques utilizing exercise rather than pharmacological stress may preferentially be used, depending on availability and local expertise.
- If the exercise test is *positive*, an invasive coronary angiogram should be performed to confirm the presence, extent, and severity of CAD (*Figure 4*).

5.1.1.1 Recommendations for sports participation

Individuals at risk of CAD and asymptomatic individuals in whom CAD is detected at screening should have aggressive management of risk factors for atherosclerosis.[6,131,132,202] Considering the benefits of exercise on primary and secondary prevention of CCS,[6,234] individuals with risk factors should be restricted from competitive sport only when there is substantial risk of an adverse event, as indicated by functional tests, or when there is evidence of disease progression during serial evaluations.[233] Exercise recommendations should be individually tailored based on the intensity of the exercise and the sporting discipline. Participation in competitive endurance, power, and mixed disciplines (see *sections 4.2 and 5.1.3*) generally requires vigorous effort and is more likely to induce myocardial ischaemia, whereas leisure sports or intentional recreational exercise allows for greater control of physical effort. Individuals with a high risk of atherosclerotic CAD and asymptomatic individuals in whom CAD is detected at screening who participate in intensive exercise should be assessed with a maximal exercise test or functional imaging test on an annual basis.

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Recommendations for exercise in individuals at risk of atherosclerotic coronary artery disease and asymptomatic individuals in whom coronary artery disease is detected at screening

Recommendations	Class[a]	Level[b]
Among individuals with asymptomatic CCS, defined as CAD without inducible myocardial ischaemia on a functional imaging or conventional exercise stress test, [233] participation in all types of exercise, including competitive sports, should be considered based on individual assessment.	IIa	C

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CAD = coronary artery disease; CCS = chronic coronary syndrome.
[a]Class of recommendation.
[b]Level of evidence.

5.1.2 Established (long-standing) chronic coronary syndrome

All individuals with established (long-standing) CCS should be encouraged to perform the minimal PA recommendations for general and CV health.[235] This applies to individuals with stable angina, asymptomatic and symptomatic individuals stabilized <1 year after ACS, or individuals with recent revascularization, and asymptomatic and symptomatic individuals >1 year after initial diagnosis or revascularization.[110] Advice on intensive exercise and participation in most competitive sports in asymptomatic individuals with long-standing CCS should be based on several factors, which are determined through clinical history, exercise stress testing, or functional imaging and echocardiography (*Table 10*).

Individuals with long-standing CCS who do not show any abnormalities on a maximal exercise test or functional imaging test, or have unimpaired LV function, may be considered as low risk for an exercise-induced adverse event[236–238] (*Table 11*). Such individuals may engage in all competitive sports on an individual basis (*Figure 5*). Some restrictions may apply for high-intensity power, mixed, and endurance sports (see *Figure 2*, *section 4.1.2*) for older patients (>60 years old) with CCS. This is due to the fact that age is an additional, strong predictor of adverse events during exercise. There are no restrictions in low-risk patients for skills sports regardless of age (*Figure 2*).

Individuals with inducible ischaemia during functional testing, despite adequate treatment, should undergo coronary angiography; those with high-risk lesions on coronary angiography (*Table 11*) should have revascularization prior to considering high-intensity exercise programmes or competitive sport (*Figure 5*). Individuals with high-risk coronary features may gradually return to sport 3–6 months after successful revascularization pending a normal maximal exercise or functional imaging test.

When ischaemia cannot be treated despite adequate therapy, including revascularization, the individual should be restricted from competitive sports, with the possible exception of individually recommended low-intensity skill sports. Such individuals may engage in regular recreational exercise of low and moderate intensity provided risk factors and symptoms are treated adequately and there is regular clinical surveillance. These individuals may also participate in leisure sports, 2–3 times/week, in selected cases, if the intended activity is below (around 10 beats) the ischaemic threshold and below the level of arrhythmias.[231]

Table 10 Factors determining risk of adverse events during intensive exercise and competitive sports in asymptomatic individuals with long-standing coronary artery disease

Type and level of sports competition
Fitness level of the individual patient
Profile of cardiovascular risk factors
Presence of exercise-induced myocardial ischaemia
Exercise-induced arrhythmia
Evidence of myocardial dysfunction

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Table 11 High-risk features for exercise-induced adverse cardiac events in patients with atherosclerotic coronary artery disease[233]

• Critical coronary stenosis, >70% in a major coronary artery or >50% in the left main stem on coronary angiography, and/or FFR <0.8 and/or iFR <0.9
• Basal left ventricular ejection fraction ≤50% and wall motion abnormalities
• Inducible myocardial ischaemia on maximal exercise testing
• NSVT, polymorphic or very frequent ventricular premature beats, at rest and during maximal stress
• Recent ACS ± PCI or surgical revascularization (<12 months)

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ACS = acute coronary syndrome; FFR = fractional flow reserve; iFR = instant flow reserve; NSVT = non-sustained ventricular tachycardia; PCI = percutaneous coronary intervention.

5.1.2.1 Antithrombotic treatment

Individuals with CAD should receive conventional antithrombotic treatment for secondary prevention, according to published guidelines for the general population.[233,239,240] Individuals taking dual antiplatelet agents should avoid sports with bodily collision, especially when they are combined with oral anticoagulants, due to the risk of haemorrhage.[241]

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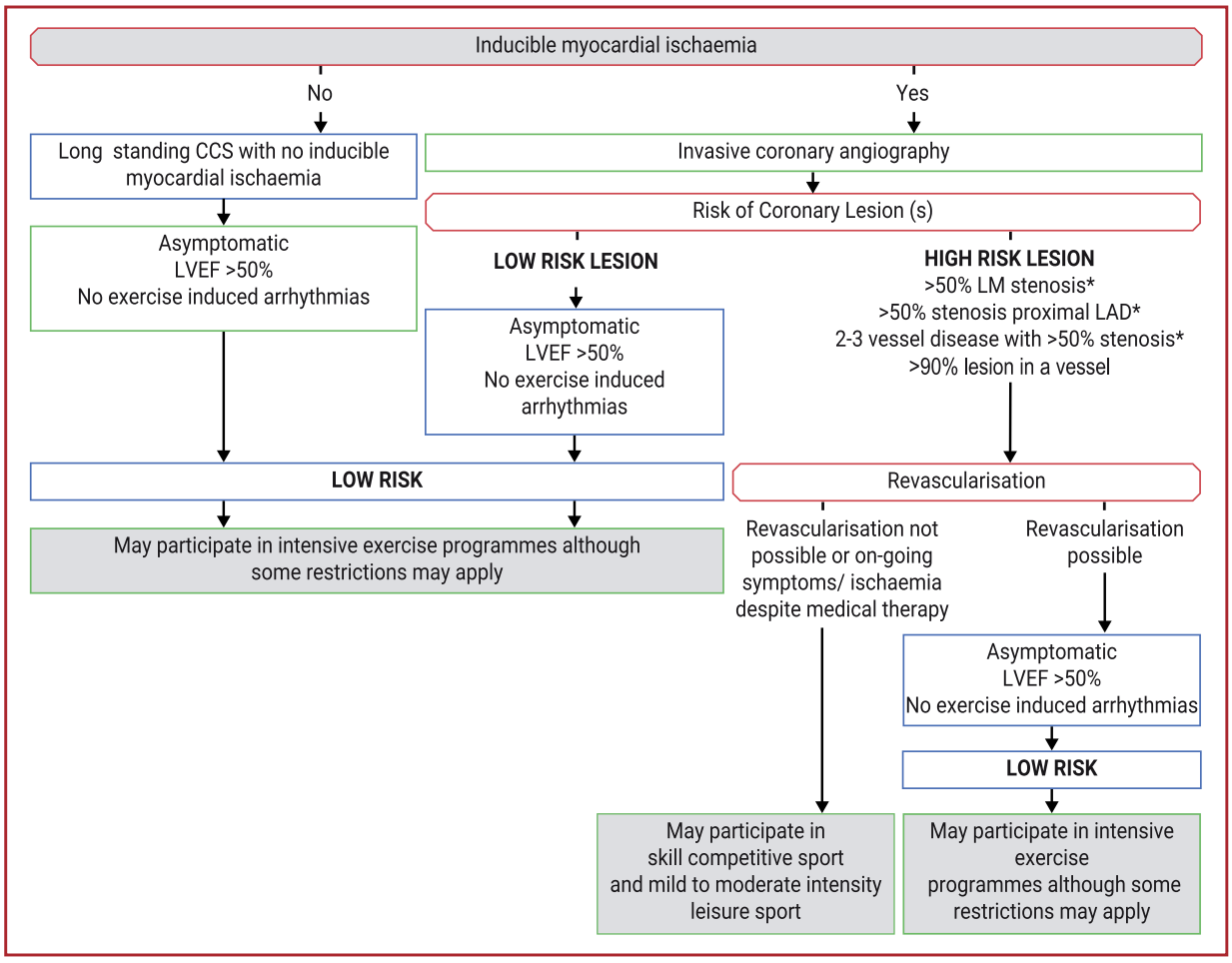

Figure 5 Clinical evaluation and recommendations for sports participation in individuals with established coronary artery disease. CCS = chronic coronary syndrome; LAD = left anterior descending coronary artery; LM = left main coronary artery; LVEF = left ventricular ejection fraction. *With documented ischaemia or a haemodynamically relevant lesion defined by FFR <0.8 or iFR <0.9.

Recommendations for exercise in individuals with long-standing chronic coronary syndrome

Recommendations	Class[a]	Level[b]
Risk stratification for exercise-induced adverse events is recommended in individuals with established (long-standing) chronic coronary syndrome (CCS) prior to engaging in exercise.[233]	I	C
Regular follow-up and risk stratification of patients with CCS is recommended.[233]	I	B
It is recommended that individuals at high risk of an adverse event from CAD are managed according to the current Guidelines on CCS.[233]	I	C
Competitive or leisure sports activities (with some exceptions such as older athletes and sports with extreme CV demands) should be considered in individuals at low risk of exercise-induced adverse events (*Table 11*).[233]	IIa	C
Leisure-time exercise, below the angina and ischaemic thresholds, may be considered in individuals at high risk of exercise-induced adverse events (*Table 11*), including those with persisting ischaemia.[233]	IIb	C
Competitive sports are not recommended in individuals at high risk of exercise-induced adverse events or those with residual ischaemia, with the exception of individually recommended skill sports.[233]	III	C

CAD = coronary artery disease; CCS = chronic coronary syndrome; CV = cardiovascular.
[a]Class of recommendation.
[b]Level of evidence.

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5.1.3 Myocardial ischaemia without obstructive disease in the epicardial coronary artery

Ischaemia and non-obstructive CAD (INOCA) is an under-recognized entity associated with increased risk of adverse events[242] that is usually detected during evaluation of anginal symptoms. Stress CMR and PET can detect abnormal coronary flow reserve and suggest coronary microvascular dysfunction with non-critical lesions. There are no established treatments for microvascular angina. However, the panel suggests adhering to the same exercise recommendations as for long-standing CCS.

5.1.4 Return to sport after acute coronary syndrome

Exercise-based cardiac rehabilitation (exCR) reduces cardiac mortality, hospital readmission,[234234] and anxiety.[243] Individuals who have experienced an ACS, cardiac surgery, or percutaneous intervention should be referred to an early exCR programme,[235,242] soon after the discharge,[6,235,244] for 8–12 weeks after the cardiac event.[235,244] Every week that exercise is delayed requires an additional month of exercise to accomplish the same level of benefit.[245]

Exercising individuals with CAD may start performing low- to moderate-intensity recreational sporting activities in parallel with participation in the structured progressive exercise programmes. All types of sports activities may be considered, at an appropriate intensity level; however, careful attention should be paid to the development of new symptoms.[218]

In general, structured outpatient exercise programmes, for 3–6 months, are required to achieve the appropriate level of activity for sports participation in patients with CAD. In individuals with non-ST segment elevation MI or CCS who have had complete revascularization and do not have residual ischaemia, exercise training can be progressed at a faster pace until the recommended exercise level is reached.

5.1.4.1 Competitive athletes

Careful individual evaluation is required before starting high-intensity competitive sports. In competitive athletes, an echocardiogram, maximal exercise test with 12-lead ECG recording or CPET is recommended for risk stratification before return to sports (see *section 5.1.2*). CPET specifically adds information on aerobic and anaerobic thresholds, guiding exercise intensity prescription and progression (see *section 4.2*).

5.1.4.2 Recreational athletes

For individuals intending to participate in non-competitive, recreational sports and leisure-time activity, similar principles apply regarding risk stratification. A symptom-limited/maximal exercise test should precede the return to sports. Higher-risk patients with CCS (*Table 11*) are not eligible for competitive sports (see *section 5.1.2*); however, low-intensity skill sports, such as golf, may be considered, at intensities below the angina threshold. If aerobic exercise is not tolerated, predominantly strength-related sports with a small amount of muscular work are recommended (*Figure 2*, *section 4.1.2*).

Recommendations for return to exercise after acute coronary syndrome

Recommendations	Class[a]	Level[b]
Exercise-based cardiac rehabilitation is recommended in all individuals with CAD to reduce cardiac mortality and rehospitalization.[234]	I	A
During the initial period, motivational and psychological support, and individualized recommendations on how to progress the amount and intensity of sports activities, should be considered in patients with CAD.	IIa	B
All sports activities should be considered, at an individually adapted intensity level in low-risk individuals with CCS.	IIa	C

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CAD = coronary artery disease; CCS = chronic coronary syndrome.
[a]Class of recommendation.
[b]Level of evidence.

5.1.5 Anomalous origin of coronary arteries

5.1.5.1 Background

The prevalence of AOCA (left and right coronary artery) is 0.44% in the general population of adolescents.[246] AOCA is considered to be a common cause of SCD in young athletes[17,18,247,248] but is rarely implicated in individuals >40 years of age.[249,250]

Chest pain, exertional syncope and SCD may be the first manifestation of AOCA,[251] however, over two thirds of patients are asymptomatic.[252] Mechanisms leading to SCD likely include repeated bursts of ischaemia with consequent increase in myocardial fibrosis and a proclivity to develop VAs during exercise. Ischaemia may result from the compression of the anomalous vessel coursing between the aorta and the pulmonary artery and/or from the acute angled take-off from the aorta and/or the proximal intramural course of the anomalous vessel (*Figure 6*).[253] Both left and right anomalous coronary origins have been implicated in Ex-R SCD, although the risk has traditionally been thought to be considerably higher with an anomalous left coronary artery origin.[252] Exercise testing rarely reveals myocardial ischaemia and multislice contrast-enhanced CT, CCTA, or CMR are the mainstay of diagnosis.

5.1.5.2 Eligibility for sports

Eligibility for competitive sports is based on the anatomical type of AOCA and on the presence of ischaemia. A highly positive inotropic and positive chronotropic exercise stress test is the best approach to demonstrate or rule out ischaemia. AOCA with acute angled take-off from the aorta resulting in a slit-like orifice with reduced lumen and anomalous coursing between the aorta and the pulmonary artery is associated with the greatest risk for SCA/SCD whether or not the anomalous artery originates from the left or right sinus of Valsalva, and strong consideration should be given to surgical correction of such an anomaly

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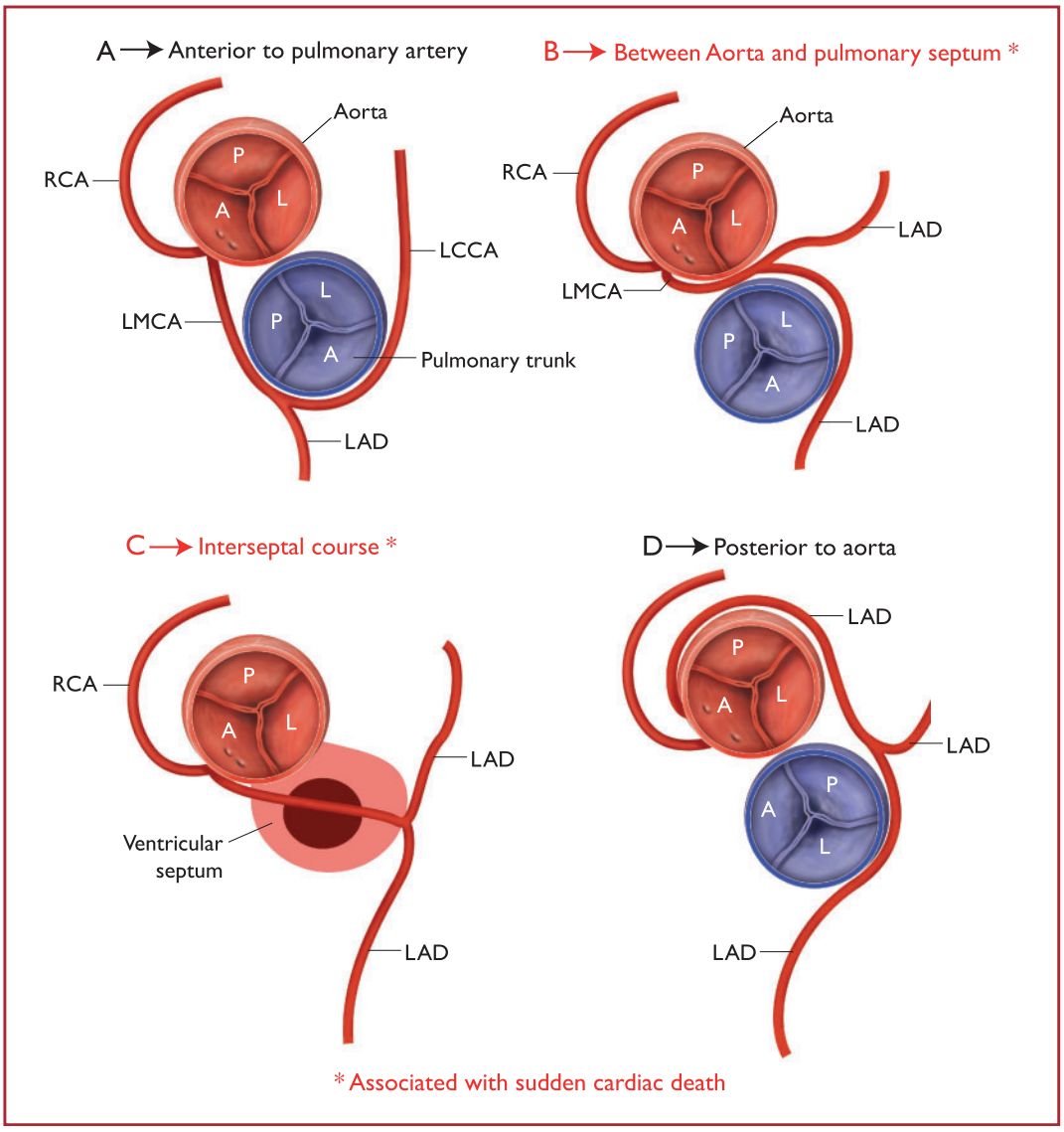

Figure 6 Schematic representation of the most frequent anomalous origin of coronary arteries and associated risk of sudden cardiac death. RCA = right coronary artery; LMCA = left main coronary artery; LAD = left anterior descending artery; LCCA = left circumflex coronary artery.

in symptomatic individuals. Prior to successful correction, participation in sports, other than low-intensity skill sports, is discouraged regardless of symptoms. We are unable to provide exercise or sport recommendations for older patients (>40 years) with AOCA, due to the paucity of studies. However, recreational exercise of moderate intensity seems reasonable, but a cautious approach is advised to more vigorous exercise.

Recommendations for exercise in young individuals/athletes with anomalous origins of coronary arteries

Recommendations	Class[a]	Level[b]
When considering sports activities, evaluation with imaging tests to identify high-risk patterns and an exercise stress test to check for ischaemia should be considered in individuals with AOCA.	IIa	C
In asymptomatic individuals with an anomalous coronary artery that does not course between the large vessels, does not have a slit-like orifice with reduced lumen and/or intramural course, competition may be considered, after adequate counselling on the risks, provided there is absence of inducible ischaemia.	IIb	C
After surgical repair of an AOCA, participation in all sports may be considered, at the earliest 3 months after surgery, if they are asymptomatic and there is no evidence of inducible myocardial ischaemia or complex cardiac arrhythmias during maximal exercise stress test.	IIb	C
Participation in most competitive sports with a moderate and high cardiovascular demand among individuals with AOCA with an acutely angled take-off or an anomalous course between the large vessels is not recommended.[c]	III	C

AOCA = anomalous origin of coronary arteries.
[a]Class of recommendation.
[b]Level of evidence.
[c]This recommendation applies whether the anomaly is identified as a consequence of symptoms or discovered incidentally, and in individuals <40 years of age.

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5.1.6 Myocardial bridging

5.1.6.1 Background

Myocardial bridging (MB) refers to a condition when some of the myocardium overlies a segment of an epicardial coronary artery (referred to as a tunnelled artery) and most commonly affects the left anterior descending artery (*Figure 7*). The prevalence of MB varies from 0.5–12% and up to 5–75% according to diagnostic angiography or CT scan series.[254] MBs are traditionally considered as benign; however, the association between myocardial ischaemia and MBs has increased their clinical relevance. MB may be discovered at imaging after an abnormal exercise ECG and should also be suspected in individuals who present with exertional angina or syncope. Coronary artery compression together with a Venturi (suction) effect are the potential underlying mechanisms for exercise-induced ischaemia.[248]

Evaluation of individuals with MB aims primarily at assessing the morphologic characteristics of the anatomical anomaly (i.e. number of MB, depth and overall length of the tunnelled vessel) and the presence of inducible ischaemia. A positive inotropic and positive chronotropic stress test is the best approach to demonstrate myocardial ischaemia. MB without other underlying associated diseases [e.g. hypertrophic cardiomyopathy (HCM)] and with no evidence of inducible myocardial ischaemia has a good prognosis.[255] However, in adult/senior individuals, it has been shown that the arterial compression in MB may be directly related to the atherosclerotic burden, proximal to the MB.[256] These individuals should be considered in the same category as individuals with CAD and treated appropriately if necessary, although the vast majority of MB is clinically silent. Beta-blockers should be used when patients are symptomatic or myocardial ischaemia is established. Surgical repair may be considered, while coronary stenting is discouraged.[255]

5.1.6.2 Eligibility

Patients with MB and evidence of ischaemia should be restricted from participation in competitive sports and should receive appropriate advice regarding leisure-time activities.

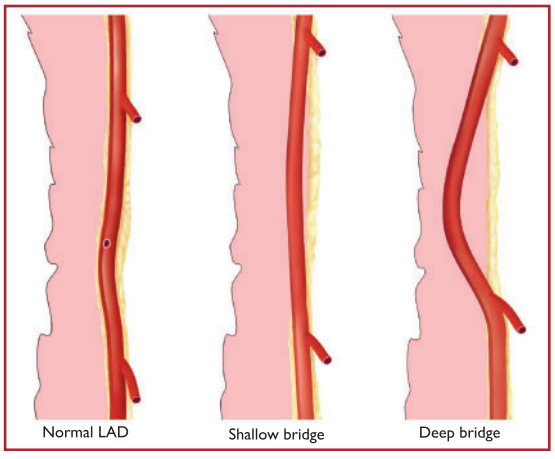

Figure 7 Schematic representation of a myocardial bridge. LAD = left anterior descending coronary artery.

Recommendations for exercise/sports in individuals with myocardial bridging

Recommendations	Class[a]	Level[b]
Participation in competitive and leisure-time sports should be considered in asymptomatic individuals with myocardial bridging and without inducible ischaemia or ventricular arrhythmia during maximal exercise testing.	IIa	C
Competitive sports are not recommended in individuals with myocardial bridging and persistent ischaemia or complex cardiac arrhythmias during maximal exercise stress testing.	III	C

[a]Class of recommendation.
[b]Level of evidence.

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5.2 Exercise recommendations in individuals with chronic heart failure

5.2.1 Background: rationale for exercise in chronic heart failure

Most of the evidence regarding exercise in chronic heart failure (HF) is derived from studies implementing exercise training programmes that are considered safe and highly recommended in stable patients on optimal medical therapy.[257–260] Meta-analyses of these studies have demonstrated a significant improvement in exercise tolerance and quality of life and a modest effect on all-cause and HF-specific mortality and hospitalization.[261–267]

5.2.2 Risk stratification and preliminary evaluation

Exercise intervention should only be initiated in a clinically stable individual after medical therapy for HF has been optimized. Key components before commencing an exercise programme and sports participation include:

(1) *Exclusion of contraindications to exercise:* Contraindications to initiating an exercise programme in chronic HF include hypotension or hypertension at rest or during exercise, unstable cardiac disease, deteriorating symptoms of HF, myocardial ischaemia despite therapy (exercise may be permitted up to ischaemic threshold), or severe and suboptimally treated pulmonary disease.[258]

(2) *Performing a baseline assessment:* A thorough cardiological evaluation is required, including assessment of comorbidities and HF severity (e.g. by assessment of blood natriuretic peptides and echocardiography). A maximal exercise test (preferably CPET) is important to assess functional capacity, exercise-induced arrhythmias or haemodynamic abnormalities and for prescription of exercise intensity, based on VO_{2peak}, or on resting and maximal heart rate during exercise [e.g. HRR or Borg's rating of perceived exertion (RPE)].[265,266]

(3) *Optimizing medical therapy:* All individuals with HF should be treated according to current Guidelines,[257] including device implantation when required.[267]

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The exercise session should be individually tailored for several weeks, according to symptoms and objective findings during exercise testing such as maximal exercise capacity, heart rate response, or arrhythmias. In atrial fibrillation (AF), exercise can only be monitored by power or Borg's RPE.

High-risk patients should be counselled more frequently during the initial phases. Ideally exercise should be supervised through an exercise-based cardiac rehabilitation programme while non-supervised home-based sessions should be gradually added.[260] When all these measures are followed, the overall risk of exercise is low, even during higher-intensity exercises and in patients with more severe HF.[268,269]

Follow-up examinations for exercise recommendations should be scheduled at least every 3–6 months. Intervals between examinations should depend on disease severity and comorbidities, setting of the sessions (supervised vs. home-based), patient's age and adherence.

5.2.3 Exercise modalities and sports participation in heart failure

Following risk factor control and therapy optimization, the individual with HF should be encouraged to start exercise programmes without delay.[242,244,270] Initially home-based exercise programmes may also be prescribed and monitored.[270,271]

In uncomplicated cases low to moderate-intensity recreational sporting activities may be considered in parallel to the structured exercise programme. When prescribed, maximal exercise intensities should be monitored, for example, by heart rate monitors. If monitoring does not reveal any exercise-induced arrhythmias or other abnormalities, then all types of recreational sports activities are permitted (see *Figure 2*, *section 4.1.2*).

5.2.3.1 Aerobic/endurance exercise

Aerobic exercise is recommended for stable patients [New York Heart Association (NYHA) class I–III], because of its well-demonstrated efficacy and safety.[260] Recommendations on optimal exercise dose have been previously described in ESC and AHA Guidelines.[242,270–272] The most commonly evaluated exercise mode is moderate continuous exercise (MCE).[242,270–272] In patients in NYHA functional class III, exercise intensity should be maintained at a lower intensity (<40% of VO_{2peak}), according to perceived symptoms and clinical status during the first 1–2 weeks. This should be followed by a gradual increase in intensity to 50–70% VO_{2peak}, and if tolerated, up to 85% VO_{2peak} as the primary aim.[270,271]

Recently, high-intensity interval training (HIIT) programmes have been considered as an alternative exercise modality for low-risk patients.[269] The most recent meta-analysis showed that HIIT was superior to MCE in improving VO_{2peak} in individuals with HF with reduced (<40%) ejection fraction (HFrEF) in the short term.[273] However, this superiority disappeared in subgroup analysis of isocaloric protocols. HITT programmes may be recommended initially to prepare low risk patients with stable HF who want to return to high intensity aerobic and mixed endurance sports (*Figure 2*, *section 4.1.2*).

5.2.3.2 Resistance exercise

Resistance exercise training may complement, but not substitute, aerobic exercise training because it reverses skeletal muscle mass loss and deconditioning without excessive stress on the heart.[270,274] The training intensity can preferably be set at the level of resistance at which the patient can perform 10–15 repetitions at 15 on Borg's RPE scale (*Table 12*).[242,270] In patients with altered skeletal muscle function and muscle wasting, exercise training should focus initially on increasing muscle mass by using resistance programmes.[275,276]

Resistance programmes may specifically be considered for low-risk stable patients, who want to return to strength-related power sports, e.g. weightlifting (*Figure 2*, *section 4.1.2*). A meta-analysis showed that resistance exercise as a single intervention has the capacity to increase muscle strength, aerobic capacity, and quality of life in HFrEF patients who are unable to participate in aerobic exercise programmes.[277] Also, in advanced HF or in patients with very low exercise tolerance, resistance exercise can be safely applied if small muscle groups are trained.[270,277,278]

Table 12 Optimal exercise training dose for patients with chronic heart failure

	Aerobic exercise	Resistance exercise
Frequency	3–5 days/week, optimally daily	2–3 days/week; balance training daily
Intensity	40–80% of VO_{2peak}	Borg RPE <15 (40–60% of 1RM)
Duration	20–60 min	10–15 repetitions in at least 1 set of 8–10 different upper and lower body exercises
Mode	Continuous or interval	
Progression	A progressively increasing training regimen should be prescribed with regular follow-up controls (at least every 3–6 months) to adjust the duration and the level of the exercise to the reached level of tolerance	A progressively increasing training regimen should be prescribed with regular follow-up controls (at least every 3–6 months) to adjust the duration and the level of the exercise to the reached level of tolerance

1 RM = one repetition maximum; RPE = rating of perceived exertion; VO_{2peak} = peak oxygen consumption.

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5.2.3.3 Respiratory exercise

Inspiratory muscle training improves VO_{2peak}, dyspnoea, and muscle strength,[279–282] and it typically involves several sessions per week with intensity ranging from 30% to 60% of maximal inspiratory pressure, and duration from 15–30 min for an average of 10–12 weeks.[279] This training modality should be recommended to the most severely deconditioned individuals as an initial alternative who may then transition to conventional exercise training and sports participation, to optimize cardiopulmonary benefits.[280]

5.2.3.4 Aquatic exercise

Aquatic exercise has not been recommended for individuals with HF, due to concerns that the increase in central blood

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volume and cardiac preload as a consequence of hydrostatic pressure may not be tolerated.[283] However, a recent meta-analysis has shown that aquatic exercise training may be safe and clinically effective.[284]

5.2.4 Sports participation and return to sports

In addition to risk stratification (*section 5.2.3*), the evaluation for participation in sports includes intensity and type of sports (competitive vs. recreational), and determining the individual fitness level.

5.2.4.1 Competitive sports

Participation in competitive sports may be considered in a group of selected low-risk individuals. A thorough individual evaluation using a maximal exercise test (or preferably CPET) is recommended before returning to sports, particularly before starting moderate- to high-intensity sports, mixed and power sports (*Figure 2*, *section 4.1.2*).

Asymptomatic individuals with preserved (≥50%) EF (HFpEF) or with mid-range (≥40−59%) EF (HFmrEF) who are optimally treated may be eligible to participate in some competitive sports in the absence of exercise-induced arrhythmias or exercise-induced hypotension. In such cases, a progressive increase in exercise dose is recommended. The duration of this process is dependent upon the functional capacity and perceived symptoms. Some restrictions may apply to high-intensity endurance, mixed and power sports with high demands, especially in older patients. No restrictions should apply for skill-related sports.

Asymptomatic patients with HFrEF who are optimally treated may only be considered safe to perform specific low-intensity skill sports at a competitive level (*Figure 2*). Higher-risk patients including those who are suboptimally treated, those that remain in NYHA II or III despite optimal therapy, and those with exercise-induced arrhythmias or exercise-induced hypotension should not participate in competitive sports, particularly those sports with moderate to high cardiopulmonary strain during training or competition.

5.2.4.2 Recreational sports

For patients intending to participate in recreational sports and leisure-time activity, similar principles apply regarding risk stratification. A progressive increase in exercise dose is recommended. Low- to moderate-intensity skill, power, mixed, and endurance sports may be considered in all asymptomatic individuals.

As with competitive sports, high-intensity recreational sports should only be considered in asymptomatic individuals with HFmrEF (EF 40−49%) who do not have exercise-induced arrhythmias or exercise-induced hypotension. Asymptomatic individuals with HFrEF who are optimally treated may engage in low- to moderate-intensity skill-related recreational sports, and selectively in low-intensity endurance sports (*Figure 2*).

In patients with HFrEF with very low exercise tolerance, frequent decompensation, or patients with LV assist devices (see *Supplementary Data*), participation in low-intensity skill-related sports is possible, if tolerated. Regular low-intensity endurance activities, e.g. walking or cycling, should generally be recommended to improve basic exercise capacity.

Recommendations for exercise prescription in heart failure with reduced or mid-range ejection fraction

Recommendations	Class[a]	Level[b]
Regular discussion about exercise participation and provision of an individualized exercise prescription is recommended in all individuals with heart failure.[260,261,285]	I	A
Exercise-based cardiac rehabilitation is recommended in all stable individuals to improve exercise capacity, quality of life, and to reduce the frequency of hospital readmission.[260,261,285]	I	A
Beyond annual cardiac assessment, clinical reassessment should be considered when the intensity of exercise is increased.	IIa	C
Motivational and psychological support and individualized recommendations on how to progress the amount and intensity of sports activities should be considered.	IIa	C
Low- to moderate-intensity recreational sporting activities and participation in structured exercise programmes may be considered in stable individuals.	IIb	C
High-intensity interval training programmes may be considered in low-risk patients who want to return to high-intensity aerobic and mixed endurance sports.	IIb	C

[a]Class of recommendation.
[b]Level of evidence.

Recommendations for participation in sports in heart failure

Recommendations	Class[a]	Level[b]
Before considering a sport activity, a preliminary optimization of heart failure risk factor control and therapy, including device implantation (if appropriate), is recommended.	I	C
Participation in sports activities should be considered in individuals with heart failure who are at low risk, based on a complete assessment and exclusion of all contraindications, in stable condition for at least 4 weeks, optimal treatment, and NYHA functional class I status.	IIa	C
Non-competitive (low- to moderate-intensity recreational) skill, power, mixed, or endurance sports may be considered in stable, asymptomatic, and optimally treated individuals with HFmrEF.	IIb	C
High-intensity recreational sports, adapted to the capabilities of the individual patient, may be considered in selected stable, asymptomatic, and optimally treated individuals with HFmrEF with an age-matched exercise capacity beyond average.	IIb	C

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Non-competitive (low-intensity recreational skill-related sports) may be considered (when tolerated) in stable, optimally treated individuals with HFrEF.	IIb	C
High-intensity power and endurance sports are not recommended in patients with HFrEF irrespective of symptoms.	III	C

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HFmrEF = heart failure with mid-range ejection fraction; HFrEF = heart failure with reduced ejection fraction; NYHA = New York Heart Association.
[a]Class of recommendation.
[b]Level of evidence.

5.2.5 Heart failure with preserved ejection fraction

Exercise-based cardiac rehabilitation programmes are a cornerstone in the holistic prevention and management of HFpEF.[260,285] Exercise intervention for 12–24 weeks increases functional capacity and quality of life.[286–292] The beneficial effects seem to be mediated by improvement in oxidative muscle metabolism and vascular function.[293] In obese patients, weight reduction has been shown to have similar effects to exercise alone,[288] therefore a stable weight reduction of 10% over 2–4 years is recommended.[294]

5.2.5.1 Exercise modalities and sports participation

Higher endurance intensities such as HIIT (4 × 4 min at 85–90% peak heart rate, with 3 min active recovery) have revealed positive effects on myocardial function, but data are limited to a small group of patients with diabetes.[295] HIIT performed over 4 weeks significantly improved VO_{2peak} and LV diastolic function.[296] Higher-intensity exercise should be limited to stable patients and could be gradually introduced after 4 weeks of MCE.

Exercise sessions should start with short phases of 10 min of endurance and 10 min of resistance exercises, which should gradually be extended in time over a period of 4 weeks. The final aim should be at least 30–45 min for ≥3 days per week. Depending on the patient's symptomatic status and functional capacity, intervals of higher intensity may be introduced.

Duration of intervention seems to be important for inducing functional and structural CV changes in HFpEF. Interventions over 2 years in healthy individuals reversed early signs of diastolic dysfunction.[297,298] Regarding sports participation refer to *section 5.2.3*.

Recommendations for exercise and participation in sport in individuals with heart failure with preserved ejection fraction

Recommendations	Class[a]	Level[b]
Moderate endurance and dynamic resistance exercise, together with lifestyle intervention and optimal treatment of cardiovascular risk factors (i.e. arterial hypertension and type 2 diabetes) are recommended.[287,289–292,299]	I	C
Competitive sports may be considered in selected stable patients without abnormalities on maximal exercise testing.	IIb	C

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[a]Class of recommendation.
[b]Level of evidence.
Refer also to the recommendation in *section 5.2.5*.

5.2.6 Exercise in individuals after heart transplantation

The exercise capacity in heart transplantation (HTx) recipients is reduced by 50–60% compared with healthy age and sex-matched individuals in the general population,[300–302] due to several factors (*Table 13*).[303] Exercise reduces CV risk induced by post-transplantation immunosuppressive medical therapy,[304] and increases physical performance,[305] enabling HTx patients to achieve levels comparable to age-matched controls.[306] HTx recipients participating in exercise-based cardiac rehabilitation programmes reveal a favourable outcome with respect to hospital readmission and long-term survival.[305,307,308]

Improvements in exercise capacity are primarily dependent on the volume of exercise. Increased functional capacity is primarily due to peripheral adaptations in the skeletal muscle including increased oxidative capacity and capillary conductance. Cardiac allograft neural reinnervation also contributes to improved functional capacity in the first year.[304,309,310] If these occur, training can be performed at high levels, enabling selected HTx patients to perform marathon runs or triathlons.[304,309,310]

5.2.6.1 Exercise modalities and sports participation

A combination of endurance and resistance exercise is considered to be the preferred exercise programme. Mean endurance exercise intensity should start at a moderate intensity (60% VO_{2peak}), which can later be increased to 80% of VO_{2peak}, a regimen level applied in the majority of exercise intervention studies in HTx.[305] In uncomplicated cases these intensities can be increased to maximum levels.

It is recommended that individuals should perform up to five bouts of 30 min of exercise per week; however, exercise duration and frequency have ranged from 30 min to 90 min for 2 to 5 times per week, in previous HTx studies.[305,311] Both endurance and resistance training is included in these training sessions; however, an additional 2–3 sessions of resistance training may be performed each week.

Resistance exercise should focus on large muscle groups using own body weight exercises or exercises on weight machines. Upper body resistance exercise should start at least 3 months after surgery, and intensity should gradually increase from low to moderate but can

Table 13 Factors influencing decreased exercise capacity (peak VO_2) and reduced cardiac output in individuals with heart transplants

Decreased exercise capacity (peak VO_2) and reduced cardiac output in heart transplant patients is determined by:

- cardiac allograft denervation
- diastolic dysfunction of the transplanted left ventricle
- reduced peak exercise end-diastolic and stroke volume by 20%
- increased pulmonary capillary wedge pressure/end-diastolic volume index ratio during upright maximal ergometry
- myocardial ischaemia due to cardiac allograft vasculopathy
- impaired peripheral vascular endothelial function
- increased systemic vascular resistance by 50%
- decreased skeletal muscle oxidative fibres, mitochondrial volume, enzyme activity and capillary density
- reduced arteriovenous oxygen difference by 25%
- elevated sympathetic activation

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also be performed up to submaximal intensities, in case of uncomplicated disease (see *section 4.1.1*).

A major limitation of endurance exercise is the reduced chronotropic response to exercise because of allograft denervation. Apart from chronotropic incompetence, other pathophysiological changes present after HTx should also be considered when prescribing and conducting an exercise programme (*Table 13*). Exercise-induced ischaemia from cardiac allograft vasculopathy should be considered, particularly when performing higher-intensity exercise, which has been advocated to have some superior effects on improving exercise capacity in these patients.[311,312]

Feasibility and safety of sports participation in stable asymptomatic HTx patients, after therapy optimization, has been reported. Therefore, participation in competitive sports, avoiding high-intensity power and endurance disciplines, may be considered in selected individuals.

Recommendations for exercise and participation in sport in heart transplant recipients

Recommendations	Class[a]	Level[b]
Regular exercise through cardiac rehabilitation, combining moderate-intensity aerobic and resistance exercise, is recommended to revert pathophysiology to pre-transplantation time, reduce cardiovascular risk induced by post-transplantation medical treatment, and improve clinical outcome.[305–312]	I	B
Recreational (low-intensity recreational) sports participation should be considered and encouraged in stable, asymptomatic individuals after therapy optimization.	IIa	C
Eligibility for competitive sports involving low- and moderate-intensity exercise may be considered in selected, asymptomatic individuals with an uncomplicated follow-up.[304,309,310]	IIb	C

[a]Class of recommendation.
[b]Level of evidence.

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5.3 Exercise recommendations in individuals with valvular heart disease

5.3.1 Introduction

Valvular heart disease affects approximately 1–2% of young exercising individuals in the general population. Reports on the natural history of valvular heart disease in athletes are sparse; however, there is a theoretical possibility that a large stroke volume, coupled with vigorous mechanical contractions of the heart, and an increased chronotropic state induced by exercise may accelerate valve dysfunction. The ensuing effects on chronic stenotic or regurgitant lesions may cause compensatory cardiac hypertrophy, impaired ventricular function, myocardial ischaemia, cardiac arrhythmias, and possibly SCD.

5.3.1.1 General principles in assessment and risk stratification of individuals with valvular heart disease prior to leisure exercise or competitive sports

There are no prospective studies examining the impact of exercise on the progression of valvular disease; therefore, general guidance presented in this section is based on consensus opinions and long-term follow-up studies from non-athletic populations. Most individuals with valvular heart disease are asymptomatic or mildly symptomatic and some may aspire to engage in regular exercise programmes including leisure and competitive sports. The management of these individuals requires assessment of the symptomatic status, functional capacity, the nature of the valvular lesion, and impact of the resulting loading conditions on cardiac structure and function. All individuals should be assessed with a clinical history, physical examination, ECG, echocardiography, and exercise stress test. The clinical history should enquire about cardiac symptoms and functional capacity. Echocardiography should focus on the valve morphology and function with particular attention to the severity and the impact of cardiac chamber size and function. Exercise testing should resemble the intensity of the sport being engaged in and should focus on inducibility of symptoms, arrhythmias, myocardial ischaemia, and the haemodynamic (BP) response to exercise. Some individuals may require exercise echocardiography to assess the severity of the valve defect.

Asymptomatic individuals with mild to moderate valvular dysfunction who have preserved ventricular function and show good functional capacity without exercise-inducible myocardial ischaemia, abnormal haemodynamic response, or arrhythmias are considered to be at low risk and may participate in all sports. Indeed, mild valvular regurgitation (mostly tricuspid and pulmonary) are common among trained athletes and likely represent a feature of the athlete's heart. Conversely, individuals with exertional symptoms, moderate or severe valvular dysfunction, left or right ventricular dysfunction, pulmonary hypertension, and exercise-induced cardiac arrhythmias or abnormal haemodynamic response are considered to be at high risk and should be considered for invasive intervention.

5.3.1.2 Surveillance

All individuals with valvular heart disease should be assessed on a regular basis. The frequency of the assessment may vary from 6 monthly to 2 yearly depending on symptomatic status and the severity of valve dysfunction.

5.3.2 Aortic valve stenosis

Aortic valve stenosis (AS) is most frequently the result of an age-dependent degenerative process causing progressive thickening, calcification, and reduced mobility of the cusps.[313] AS causes an increase in transvalvular pressure gradient and LV workload, with consequent LV hypertrophy, fibrosis, and increased myocardial oxygen demand. Left ventricular ejection fraction (LVEF) is usually preserved. Affected individuals may have a normal cardiac output at rest and even during exercise, therefore, some individuals with AS are capable of good exercise performance. Nonetheless, severe AS is associated with increased risk of heart failure and SCD from mechanical outflow obstruction, malignant VAs, or coronary hypoperfusion.[18,314]

The diagnosis and grading of AS during echocardiography is based on well-established criteria.[315] Specifically, severe AS is defined by: (i)

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a transvalvular Doppler velocity ≥4.0 m/s; (ii) a mean gradient ≥40 mmHg; and (iii) a calculated aortic valve area <1.0 cm^2 or an indexed area (recommended in athletes) <0.6 cm^2/m^2.[315] In cases with a low gradient (<40 mmHg) and calculated valve area <1.0 cm^2, with EF < 50% and stroke volume index <35 mL/m^2, low-dose dobutamine stress echocardiography is recommended to identify pseudo-severe AS or true severe AS.[315,316] Assessment of the aortic valve calcium score with CT can be useful in borderline cases where the severity of AS remains unclear.[313,316]

Exercise testing is particularly important to assess the haemodynamic response in AS and to serve as a guide to exercise prescription in cases of asymptomatic moderate and severe AS. A progressive drop in SBP with exercise, or failure to increase SBP by at least 20 mmHg, identifies subjects at higher risk.[317] Exercise-induced ventricular tachycardia should also be considered a criterion for exercise restrictions.

Asymptomatic individuals with mild AS may participate in all sports. Asymptomatic athletes with severe AS should not participate in any competitive or leisure sports with the exception of low-intensity skill sports. However, low-intensity aerobic exercise could be encouraged in asymptomatic individuals to improve functional capacity and general well-being.

Individuals with symptomatic AS should not participate in any competitive sport or recreational sport/exercise and valve replacement is recommended. Mild exercise, that does not provoke symptoms, may be considered in these individuals for general health benefits.

Recommendations for exercise and participation in recreational/leisure-time sports in asymptomatic individuals with aortic stenosis

	Aortic stenosis[c]		
	Recommendation	**Class[a]**	**Level[b]**
Mild	Participation in all recreational sports, if desired, is recommended.	I	C
Moderate	Participation in all recreational sports involving low to moderate intensity, if desired, should be considered in individuals with LVEF≥50%, good functional capacity, and normal exercise test.	IIa	C
Severe	Participation in all recreational sports/exercise involving low intensity, if desired, may be considered in individuals with LVEF≥50% and normal BP response during exercise.	IIb	C
	Participation in competitive or recreational sports/exercise of moderate and high intensity is not recommended.	III	C

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BP = blood pressure; LVEF = left ventricular ejection fraction.
[a]Class of recommendation.
[b]Level of evidence.
[c]For mixed valvular disease, the recommendation for the predominant lesion (stenotic or regurgitant) should be followed.

Recommendations for participation in competitive sports in asymptomatic individuals with aortic stenosis

	Aortic stenosis[c]		
	Recommendation	**Class[a]**	**Level[b]**
Mild	Participation in all competitive sports, if desired, is recommended.	I	C
Moderate	Participation in all competitive sports involving low to moderate effort, if desired, may be considered in individuals with LVEF≥50%, good functional capacity, and normal BP response during exercise.	IIb	C
Severe	Participation in low-intensity skill sports may be considered in a select group of individuals with LVEF≥50%.	IIb	C
	Participation in sports or exercise of moderate or high intensity is not recommended.	III	C

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BP = blood pressure; LVEF = left ventricular ejection fraction.
[a]Class of recommendation.
[b]Level of evidence.
[c]For mixed valvular disease, the recommendation for the predominant lesion should be followed.

5.3.3 Aortic valve regurgitation

Aortic valve regurgitation (AR) is usually caused by a congenitally abnormal valve (i.e. bicuspid valve), degeneration of a tricuspid valve, or loss of coaptation due to aortic root enlargement.[313,318] Less common causes of AR include infective endocarditis or aortic dissection.

The haemodynamic consequence of chronic AR is characterized by a pressure and volume overload that typically leads to a dilated and hypertrophied LV. To accommodate the concomitant forward flow from the mitral valve and the backward flow from the aortic valve during diastole, the LV progressively increases in size and mass. This remodelling may occasionally be difficult to distinguish from cardiac adaptation in athletes, especially in males with a large body size who engage in endurance activities, and therefore LV size should be interpreted in the context of the sport participated in, and the gender and body surface area of the individual.[319] Males with a LV end-diastolic diameter >35 mm/m^2 or a LV end-systolic diameter >50 mm, and females with a LV end-diastolic diameter >40 mm/m^2 or a LV end-systolic diameter >40 mm, should be considered to have pathological LV enlargement, irrespective of the level of physical training. These individuals should be closely monitored for a progressive increase in LV end-systolic diameter.

In individuals with suboptimal echocardiographic images, CMR has the advantages of providing an accurate assessment of LV volume and EF, flow calculations and detecting the presence of myocardial scar,[319] in individuals with severe AR. Furthermore, the whole thoracic aorta can be visualized during the same examination.

Asymptomatic individuals with mild and moderate AR may participate in all sports. Asymptomatic individuals with severe AR, moderately dilated LV, and good LV systolic function may participate in

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sports involving low and moderate intensity and may be considered for more intensive exercise on an individual basis. Such individuals require more frequent surveillance on a 6-monthly basis to assess LV function. In asymptomatic individuals with severe AR and reduced LVEF, surgical valve replacement/repair is indicated, and they should not participate in competitive sports but may participate in leisure sports involving only low-intensity exercise. Surgery is recommended in symptomatic individuals with severe AR. These individuals should not participate in competitive or leisure sports; however, low-intensity aerobic exercise activity is encouraged to improve functional capacity and general well-being.

5.3.4 Bicuspid aortic valve

Bicuspid aortic valve (BAV) is a common congenital abnormality with a prevalence of 1−2% in the general population.[320] BAV may be associated with AS or AR and increased risk for ascending aortic aneurysm or dissection, and SCD.[28,321] Compared with Marfan syndrome, the risk of aortopathy is lower; nonetheless BAV is much more frequent, and the relative risk of aortic dissection has been reported to be eight times greater than with a tricuspid aortic valve.[321] BAV may not be identified during physical examination in the absence of valve dysfunction;[58,322] however, the outcome of young individuals without valvular dysfunction is good.[323,324]

It is unclear whether intensive exercise accelerates aortic dilatation in the long term. A previous study comparing athletes with BAV, non-athletes with BAV, and athletes with a normal aortic valve reported that athletes with BAV showed a 0.11 ± 0.59 mm/year increase in aortic size at the sinuses of Valsalva and 0.21 ± 0.44 mm/year for the proximal ascending aorta, which was not dissimilar to non-athletes with a BAV.[325] Currently, expert consensus panels advise a cautious approach to sports activities when the ascending aorta is above the normal limits (see *section 5.4*). In the absence of aortopathy, exercise recommendations for individuals with BAV are identical to those in individuals with tricuspid aortic valve dysfunction.

Recommendations for participation in recreational/leisure-time sports in asymptomatic individuals with aortic regurgitation

	Aortic regurgitation[c]		
	Recommendation	**Class[a]**	**Level[b]**
Mild	Participation in all recreational sports, if desired, is recommended.	I	C
Moderate	Participation in all recreational sports, if desired, should be considered in asymptomatic individuals with a non-dilated LV with LVEF>50% and normal exercise stress test.	IIa	C
Severe	Participation in all recreational sports involving low and moderate intensity, if desired, may be considered with a mild or moderately dilated LV with LVEF>50% and normal exercise stress test.	IIb	C
	Participation in any moderate- or high-intensity recreational exercise is not recommended with LVEF≤50% and/or exercise-induced arrhythmias.	III	C

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LV = left ventricle; LVEF = left ventricular ejection fraction.
[a]Class of recommendation.
[b]Level of evidence.
[c]For mixed valvular disease, the recommendation for the predominant lesion should be followed.

Recommendations for participation in competitive sports in asymptomatic individuals with aortic regurgitation

	Aortic regurgitation[c]		
	Recommendation	**Class[a]**	**Level[b]**
Mild	Participation in all competitive sports, if desired, is recommended.	I	C
Moderate	Participation in all competitive sports, if desired, should be considered in individuals with LVEF>50% and normal exercise test.	IIa	C
Severe	Participation in most competitive sports involving low to moderate intensity may be considered in individuals with a mild or moderately dilated LV with LVEF>50% and normal exercise stress test.	IIb	C
	Participation in any moderate- or high-intensity competitive sports is not recommended in individuals with severe AR and/or LVEF≤50% and/or exercise-induced arrhythmias	III	C

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AR = aortic regurgitation; LV = left ventricle; LVEF = left ventricular ejection fraction.
[a]Class of recommendation.
[b]Level of evidence.
[c]For mixed valvular disease, the recommendation for the predominant lesion should be followed.

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5.3.5 Primary mitral regurgitation

Most individuals with mitral valve disease have primary mitral regurgitation (MR) from myxomatous disease.[326] MR is confirmed and quantified by echocardiography. General recommendations regarding exercise and sports are based on symptomatic status, severity of MR, LV function, systolic pulmonary artery pressure (sPAP), and the presence or absence of arrhythmias during exercise. Both athletic training and MR may be associated with an enlarged LV cavity; however, an enlarged LV that is disproportionate to the level of exercise may be suggestive of severe MR and an indication to refrain from competitive or leisure sports involving moderate- or high-intensity exercise.

Asymptomatic individuals with mild or moderate MR may compete in all sports if they have good functional capacity, preserved LV function, sPAP < 50 mmHg and absence of complex arrhythmias during exercise. Individuals with symptomatic MR and reduced exercise capacity or individuals with MR with exercise-induced complex arrhythmias should not participate in competitive or leisure sport; however, low-intensity aerobic exercise should be encouraged to improve functional capacity and general well-being. Individuals on long-term anticoagulation therapy for AF should not engage in contact/collision sport.

Recommendations for participation in recreational/leisure-time sports in asymptomatic individuals with mitral regurgitation

	Mitral regurgitation[c,d]		
	Recommendation	**Class[a]**	**Level[b]**
Mild	Participation in all sports, if desired, is recommended.	I	C
Moderate	Participation in all recreational sports, if desired, should be considered in individuals fulfilling the following: • LVEDD<60 mm[327] or <35.3 mm/m^2 in men and <40 mm/m^2 in women • LVEF≥60% • Resting sPAP<50 mmHg • Normal exercise test	IIa	C
Severe	Participation in all recreational sports involving low and moderate intensity, if desired, may be considered in individuals fulfilling the following: • LVEDD<60 mm[327] or <35.3 mm/m^2 in men and <40 mm/m^2 in women • LVEF≥60% • Resting sPAP<50 mmHg • Normal exercise test	IIb	C

LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; MR = mitral regurgitation; sPAP = systolic pulmonary artery pressure.
[a]Class of recommendation.
[b]Level of evidence.
[c]For mixed valvular disease, the recommendation for the predominant valve lesion should be followed.
[d]No collision or body contact sports if anticoagulated for atrial fibrillation.

Recommendations for participation in competitive sports in asymptomatic individuals with mitral regurgitation

	Mitral regurgitation[c,d]		
	Recommendation	**Class[a]**	**Level[b]**
Mild	Participation in all competitive sports, if desired, is recommended.	I	C
Moderate	Participation in all competitive sports, if desired, should be considered in individuals fulfilling the following: • LVEDD<60 mm[327] or <35.3 mm/m^2 in men and <40 mm/m^2 in women • LVEF≥60% • Resting sPAP<50 mmHg • Normal exercise test	IIa	C
Severe	Participation in competitive sports involving low exercise intensity, if desired, may be considered in individuals fulfilling the following: • LVEDD<60 mm[327] or <35.3 mm/m^2 in men and <40 mm/m^2 in women • LVEF≥60% • Resting sPAP<50 mmHg • Normal exercise test	IIb	C
	Participation in competitive sports is not recommended in individuals with a LVEF<60%	III	C

LVEDD = left ventricular end-diastolic diameter; LVEF = left ventricular ejection fraction; MR = mitral regurgitation; sPAP = systolic pulmonary artery pressure.
[a]Class of recommendation.
[b]Level of evidence.
[c]For mixed valvular disease, the recommendation for the predominant valve lesion should be followed.
[d]No collision or body contact sports if anticoagulated for atrial fibrillation.

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5.3.5.1 Mitral valve prolapse

Mitral valve prolapse (MVP) is characterized by fibro-myxomatous changes of the mitral valve leaflets and has a prevalence of 1–2.4%.[328,329] The diagnosis of MVP is defined as >2 mm displacement of one or both leaflets of the mitral valve beyond the annulus within the left atrium in end-systole.[330] MVP is generally benign with a 10-year mortality risk of 5%.[331] The majority of individuals are identified incidentally during cardiac auscultation, or echocardiography.

The most common complication of MVP is the progression to chronic severe MR, in 5–10% of individuals with MVP. Other complications include HF from chronic MR, pulmonary hypertension, infective endocarditis, supraventricular and VAs, and, occasionally, SCD.[313] In the Italian cardiac pathology registry of 650 SCDs in young adults, 7% were attributed to MVP.[332] Most decedents showed scarring in the infero-basal wall and papillary muscles and bi-leaflet prolapse. Myocardial scarring, mitral valve annular disjunction (i.e. an abnormal atrial displacement of the mitral valve leaflet hinge point during systole),[333] T-wave inversion in the inferior leads and VAs arising from the LV [right bundle branch block (RBBB) morphology] were high-risk features for SCD.[334] The mechanical strain of MVP on papillary muscles and adjacent myocardium is thought to be responsible for the myocardial scarring, which may be a possible mechanism for life-threatening arrhythmias in some individuals.[335,336]

In general, exercising individuals with MVP have an excellent prognosis. In a recent Italian study of 7449 young competitive athletes, MVP was identified in 2.9%. During a follow-up period of 8 ± 2 years there were no fatalities.[337] Adverse events, including progressive MR with LV dilatation, ischaemic stroke, and AF occurred at a rate of 0.5% per annum and were more common in older athletes with baseline mitral valve disjunction or VAs.

Individuals with MVP should be evaluated with an exercise test and 24-hour ECG. Individuals with inferior T-wave inversion or ventricular premature beats arising from the LV should undergo a CMR imaging scan to check specifically for myocardial fibrosis affecting the infero-basal wall. Other potentially high-risk markers include evidence of mechanical dispersion detected by speckled tracking echocardiography,[338] coexisting prolongation of the QT interval and mitral annular disjunction.[333]

Given the relatively benign nature of MVP, asymptomatic patients with mild or moderate MR can participate in all competitive sports and leisure sports in the absence of the aforementioned risk factors (*Figure 8*). Asymptomatic patients with severe MR but none of the above high-risk markers may compete in low- to moderate-intensity sports after detailed discussion with their specialist in the presence of LV end-diastolic diameter (LVEDD) <60 mm (or <35.5 mm/m^2 in men and <40 mm/m^2 in women) with LVEF ≥ 60%, resting sPAP < 50 mmHg, and normal exercise test.

Symptomatic patients with MVP and any of the aforementioned high-risk features (*Figure 8*) should not participate in recreational or competitive sports; however, low-intensity aerobic exercise should be encouraged to improve functional capacity and general well-being.

5.3.6 Mitral stenosis

Although rheumatic valve disease is uncommon in the western world, the increase in emigration patterns means that cardiologists may encounter individuals with rheumatic mitral stenosis (MS) who aspire to exercise. Individuals with advanced MS are usually symptomatic and incapable of engaging in exercise regimens with a high CV demand. The risk stratification of exercising individuals with MS is based largely on a detailed echocardiogram with specific interest in the severity of the lesion and accompanying systolic PAP. In addition, assessment should include a maximal exercise stress test to identify concealed symptoms and functional capacity.

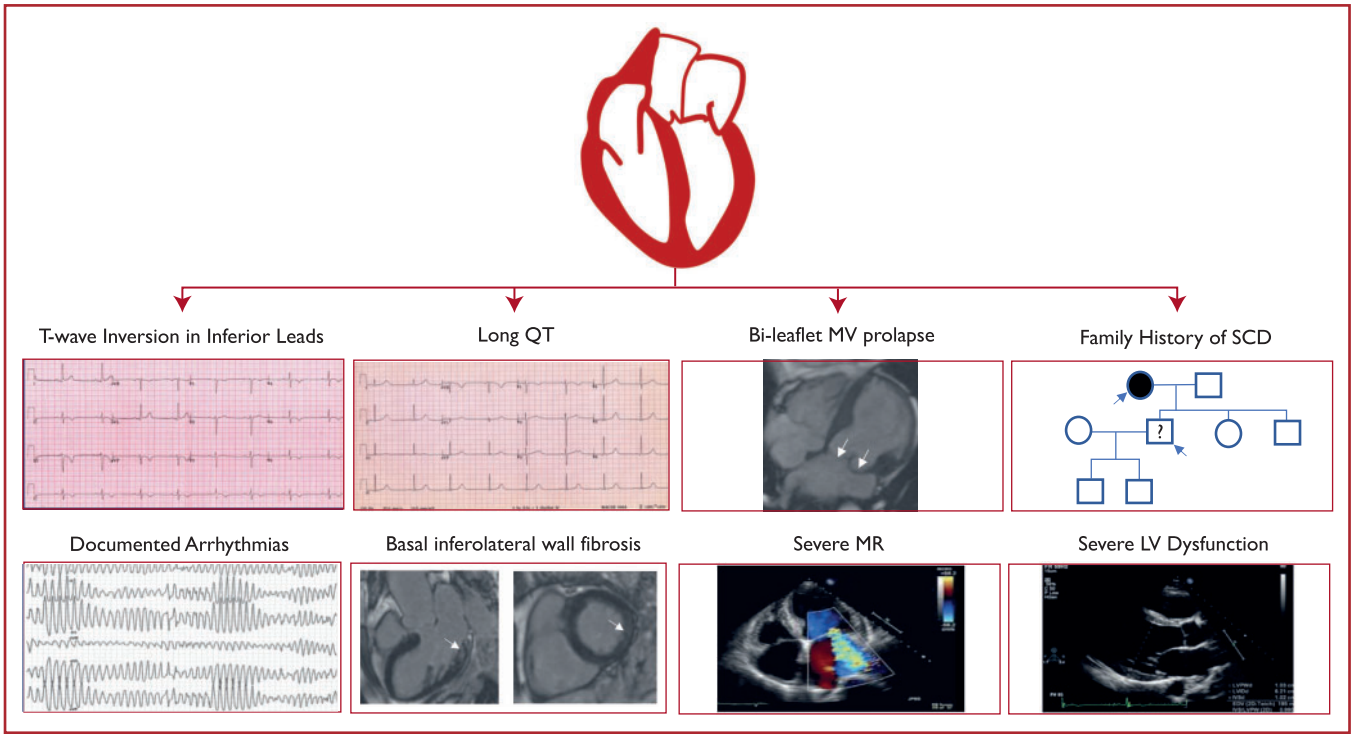

Figure 8 Specific markers of increased risk of sudden cardiac death (SCD) with mitral valve prolapse. LV = left ventricular; MR = mitral regurgitation; MV = mitral valve. Adapted from Gati *et al.*[336]

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Asymptomatic individuals with mild MS [mitral valve area (MVA) 1.5–2.0 cm^2] and moderate MS (MVA 1.0–1.5 cm^2) who are in sinus rhythm and demonstrate good functional capacity on exercise testing and a normal sPAP may participate in all competitive and leisure sports. Mildly symptomatic individuals with severe MS (MVA < 1.0 cm^2) may only participate in leisure exercise involving physical effort of low intensity. Individuals with symptomatic MS should be referred for intervention and advised to abstain from participation in sports and recreational exercise of moderate or high intensity. Individuals with AF should be anticoagulated and avoid contact/collision sport. In cases of balloon mitral valvuloplasty with good results (i.e. MVA > 2.0 cm^2) regular exercise and competitive sport may be considered in asymptomatic individuals with good functional capacity.

Mild TR is common in athletes and accompanied by physiological dilatation of the inferior vena cava, which is easily collapsible with inspiration. Severe TR is characterized by increasing tricuspid annular dilatation and RV remodelling that eventually leads to RV dysfunction and a non-reactive inferior vena cava. Individuals with severe TR may also have a reduced exercise capacity due to an impaired cardiac output response with exercise.[339] Furthermore, they may experience increased right- and left-sided filling pressures during exercise, the latter being due to diastolic ventricular interaction.[340]

In general, asymptomatic patients with TR who have good functional capacity, non-dilated right ventricle, preserved ventricular function, sPAP<40 mmHg, and absence of complex arrhythmias may compete in all competitive and recreational sports.

Recommendations for participation in recreational/leisure-time sports in individuals with mitral stenosis

	Mitral stenosis[c,d]		
	Recommendation	**Class[a]**	**Level[b]**
Mild (MVA 1.5–2.0 cm^2)	Participation in all recreational sports, if desired, is recommended in individuals with a resting sPAP<40 mmHg and normal exercise test.	I	C
Moderate (MVA 1.0–1.5 cm^2)	Participation in all recreational sports involving low and moderate intensity, if desired, may be considered in individuals with resting sPAP<40 mmHg and a normal exercise test.	IIb	C
Severe (MVA<1 cm^2)	Participation in leisure sports of moderate or high intensity is not recommended.	III	C

MVA = mitral valve area; sPAP = systolic pulmonary artery pressure.
[a]Class of recommendation.
[b]Level of evidence.
[c]For mixed valvular disease, the recommendation for the predominant valve lesion should be followed.
[d]No collision or body contact sports if anticoagulated for atrial fibrillation.

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Recommendations for participation in competitive sports in asymptomatic individuals with mitral stenosis

	Mitral stenosis[c,d]		
	Recommendation	**Class[a]**	**Level[b]**
Mild (MVA 1.5–2.0 cm^2)	Participation in all competitive sports, if desired, is recommended in individuals with a resting sPAP<40 mmHg and a normal exercise test.	I	C
Moderate (MVA 1.0–1.5 cm^2)	Participation in all competitive sports involving low intensity may be considered in individuals with a resting sPAP<40 mmHg and normal exercise test.	IIb	C
Severe (MVA<1.0 cm^2)	Participation in competitive sports is not recommended.	III	C

MVA = mitral valve area; sPAP = systolic pulmonary artery pressure.
[a]Class of recommendation.
[b]Level of evidence.
[c]For mixed valvular disease, the recommendation for the predominant valve lesion should be followed.
[d]No collision or body contact sports if anticoagulated for atrial fibrillation.

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5.3.7 Tricuspid regurgitation

Tricuspid regurgitation (TR) is usually secondary to left heart disease, pulmonary hypertension, or right ventricular (RV) dysfunction. In most patients with secondary TR, exercise limitations relate to the underlying pathology.

5.4 Exercise recommendations in individuals with aortopathy

5.4.1 Introduction

Thoracic aortic aneurysms are largely asymptomatic until a sudden and catastrophic event, including aortic rupture or dissection, occurs,

which can be rapidly fatal. Advanced age, male sex, long-term history of arterial hypertension, and the presence of aortic aneurysm confer the greatest population attributable risk for aortic dissection. However, patients with genetic connective tissue disorders such as Marfan (MFS), Loeys Dietz, Turner, or Ehlers Danlos (EDS) syndromes, and patients with BAV are at increased risk at a much younger age. BAV has a prevalence of about 1–2% in the general population. These patients have a relatively low risk for aortic dissection in comparison with patients with hereditary thoracic aortic disease (HTAD). Having a family history of aortic dissection or sudden death is a risk factor and a larger diameter of the aorta carries a higher risk, although dissection can occur at any diameter and especially in EDS patients there is no clear association with aortic diameter.[341–343]

A dilated aortic root (>40 mm) is not a feature of athlete's heart, with only a small minority of young athletes (0.3%) having an enlarged aortic root diameter.[344–347] During follow-up no progressive enlargement of the aortic diameter was observed in these athletes, and no aortic events occurred during a 5-year period.[347]

5.4.2 Risk of dissection

Because of the increase in BP and wall stress associated with intensive exercise and sports, such activities are potentially associated with an enhanced risk of expansion of the aorta and acute aortic dissection. However, daily exercise is important in maintaining an ideal BP, heart rate, and body weight and a sedentary lifestyle is an important modifiable risk factor for CV disease and mortality. Physical activity is advised in all patients with aortic pathology, even when the aorta is dilated.

There are no randomized controlled trials on competitive sports in patients with thoracic aortic disease, or any prospective data regarding the risks of competitive athletics in patients after surgical correction; however, even after aortic root replacement, patients with MFS and other HTAD remain at risk of aortic complications. One small prospective cohort study evaluated the feasibility and effects of a 3-week rehabilitation training programme in 19 MFS patients with a mean age of 47 years. During the 1-year follow-up, there were no adverse events but there was improvement in physical fitness and reduction in psychological distress. These effects were detectable after 3 weeks of rehabilitation, and mostly persisted through the 1-year follow-up. Unfortunately, no information on aortic diameters was provided.[348]

5.4.3 Sporting disciplines

Exercise-related acute thoracic aortic dissections are described in the literature in a total of 49 case reports. Of these, 42 patients suffered Stanford type A thoracic aortic dissections. In the majority (26/49) weightlifting was associated with aortic dissection.[349] Furthermore, a recently published retrospective cohort study of 615 patients with acute type A aortic dissection found that 4.1% cases were related to sports activities. The type of sports most often reported was golf (32%), but this was not corrected for the percentage of participants in the sport and probably reflects that golfers are frequently older with an increased risk of hypertension and hence the potential for dissection.[350]

5.4.4 Effect on aortic diameter and wall stress

One cross-sectional study, which included 58 competitive athletes with BAV, showed no correlation between aortic dimensions and duration of training.[351] Two studies compared athletes and sedentary individuals with BAV and reported no difference in aortic growth rate between the two groups.

Two MFS mouse models investigating the effects of mild-moderate dynamic exercise on the aortic wall showed a reduction in the growth rate of the aortic diameter in mice with MFS that performed mild to moderate dynamic exercise compared to sedentary mice with MFS.[352,353] Among exercising mice, the aortic wall became stronger and a larger mechanical stress was required to induce aortic rupture. An optimum protective effect was found at a training intensity level of 55–65% of maximum oxygen uptake (VO_{2max}).

Table 14 Classification of risk to perform sports in patients with aortic pathology

	Low risk	Low-intermediate risk	Intermediate risk	High risk
Diagnosis	• Aorta <40 mm in BAV or tricuspid valve • Turner syndrome without aortic dilatation	• MFS or other HTAD syndrome without aortic dilatation • Aorta 40–45 mm in BAV or tricuspid valve • After successful thoracic aorta surgery for BAV or other low risk situation	• Moderate aortic dilatation (40–45 mm in MFS or other HTAD; 45–50 mm in BAV or tricuspid valve, Turner syndrome ASI 20–25 mm/m², tetralogy of Fallot <50 mm) • After successful thoracic aorta surgery for MFS or HTAD	• Severe aortic dilatation (>45 mm in MFS or other HTAD, >50 mm in BAV or tricuspid valve, Turner syndrome ASI >25 mm/m², tetralogy of Fallot >50 mm) • After surgery with sequelae
Advice	• All sports permitted with preference for endurance over power sports	• Avoid high and very high intensity exercise, contact, and power-sports. • Preference for endurance over power sports	• Only skill sports or mixed or endurance sports at low intensity	• Sports are (temporarily) contra-indicated
Follow-up	Every 2–3 years	Every 1–2 years	Every 6 months to 1 year	Re-evaluation after treatment

ASI = aortic size index; BAV = bicuspid aortic valve; HTAD = hereditary thoracic aortic disease; MFS = Marfan syndrome.

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5.4.5 Recommendations

Regular exercise has a well-documented benefit for fitness, psychological well-being, and social interaction, as well as a positive effect on hypertension and concomitant future risk of dissection. Most individuals with aortic pathology benefit from a certain minimal exercise programme and can at least participate in recreational sports (*Table 14*). Some lesions are not compatible with endurance training and athletic sports, due to their high risk of dissection or rupture. Recommendations for exercise and sports should be individualized and based on the underlying diagnosis, the aortic diameter, family history for dissection or sudden death (risk factor), and the pre-existing fitness and experience. An exercise test with an assessment of blood pressure response is recommended before engaging in sports.

Recommendations for exercise and participation in sports in individuals with aortic pathology

Recommendations	Class[a]	Level[b]
Prior to engaging in exercise, risk stratification, with careful assessment including advanced imaging of the aorta (CT/CMR) and exercise testing with blood pressure assessment is recommended.	I	C
Regular follow-up including risk assessment is recommended.	I	C
Dynamic exercise should be considered more suitable than static exercise.	IIa	C
Participation in competitive or leisure-time sports activities (except power sports) should be considered in low-risk individuals (*Table 14*).	IIa	C
Participation in individualized leisure exercise programmes may be considered in high-risk individuals (*Table 14*).	IIb	C
Competitive sports are not recommended in individuals who are at high risk (*Table 14*).	III	C

CMR = cardiac magnetic resonance; CT = computed tomography.
[a]Class of recommendation.
[b]Level of evidence.

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5.5 Exercise recommendations in individuals with cardiomyopathies, myocarditis, and pericarditis

Cardiomyopathies are an important cause of SCD/SCA in young individuals and exercise has been implicated as a trigger for fatal arrhythmias.[17–19,28] The detection of a cardiomyopathy in an individual has important implications with respect to ongoing participation in exercise. The advent of preventive strategies for SCD has led to a significant expansion in the number of predominantly asymptomatic young patients with cardiomyopathies who aspire to exercise. When advising such individuals, it is essential to strike a balance between protecting patients from the potentially adverse effects of exercise and depriving them of the multiple benefits of exercise.

5.5.1 Hypertrophic cardiomyopathy

The diagnosis of HCM is based on the presence of unexplained LV hypertrophy, defined as a maximum end-diastolic wall thickness ≥15 mm, in any myocardial segment on echocardiography, CMR, or CT imaging.[355] HCM may also be considered in individuals with a lesser degree of LV hypertrophy (wall thickness ≥13 mm) in the context of a family history of definite HCM or a positive genetic test.[355]

5.5.1.1 Risk stratification in hypertrophic cardiomyopathy

Circumstantial evidence and a large systematic collection of young SCDs in sports in the US suggest that exercise increases the risk of SCD/SCA in individuals with HCM.[18] Consistently, previous consensus recommendations have restricted all athletes with HCM from competitive sports.[1,356,357]

More recently, relatively small longitudinal clinical studies indicate that the risk of SCD during exercise may be considerably lower than initially considered. Lampert *et al.* reported that individuals with HCM who continued participating in sports after implantable cardioverter defibrillator (ICD) implantation did not reveal an increased number of shocks during exercise.[358,359] In a cross-sectional study of 187 patients with HCM, vigorous exercise was not associated with the occurrence of VAs.[358] Pelliccia *et al.* reported outcomes in a cohort of 35 athletes with HCM, engaged in training and competitions for 5 to 31 years (mean 15 ± 8). During a 9-year follow-up period, there were no differences in the incidence of symptoms or major events between athletes who ceased exercise (n = 20) compared with athletes who continued competitive sports (n = 15).[360] In a post-mortem series, only 23% of 194 deaths from HCM occurred during sport, and affected males with a mean age of 30 years.[361] Finally, individuals with HCM who participated in rehabilitation programmes demonstrated a significant improvement in functional capacity without adverse events.[362,363]

In conclusion, there is limited evidence to indicate that all individuals with HCM are vulnerable to fatal arrhythmias during exercise and sport participation. In this regard, systematic restriction from competitive sports in all affected individuals is probably unjustified and a more liberal approach to sports participation is reasonable in some individuals after careful evaluation.[3] This is particularly important for the majority of individuals with HCM who wish to participate in amateur sports or leisure-time exercise to maintain their physical and psychological well-being.

5.5.1.2 Baseline assessment of patients with HCM

A systematic approach is required when assessing an individual with HCM who requests exercise advice. The baseline evaluation should include a comprehensive personal and family history with consideration of the age of the individual and years of exercise prior to diagnosis, assessment of the severity of the HCM phenotype, and the presence of any conventional risk factors for SCD/SCA. In older patients with HCM, the physician should review the presence of cardiac comorbidities such as hypertension and ischaemic heart disease, which may confer a worse prognosis in HCM.[364,365]

5.5.1.3 History

The presence of symptoms attributed to HCM should prompt more conservative exercise recommendations. Individuals with a history of cardiac arrest or unheralded syncope and individuals with exercise-

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induced symptoms should be advised to engage in low-intensity recreational sports only.

5.5.1.4 Resting and ambulatory ECG

The resting 12-lead ECG has limited value in risk stratification. Ambulatory ECG monitoring, preferably for 48 h, is important for detecting ventricular and supraventricular arrhythmias. The monitoring period should include an exercise session. Asymptomatic non-sustained ventricular tachycardia (NSVT) confers considerable risk of SCD in younger individuals (≤35 years).[355] Paroxysmal supraventricular arrhythmias may pose significant implications for functional capacity and, in the case of AF, for stroke prevention.[366]

5.5.1.5 Echocardiography

In relation to risk stratification for SCD the clinician should assess the following echocardiographic indices: (i) LV wall thickness; (ii) LV outflow tract (LVOT) gradient; and (iii) left atrial diameter.[355] All individuals should have the LVOT gradient assessed at rest, during the Valsalva manoeuvre, on standing suddenly, and after light exercise on the spot, such as repeated squats. By convention, LVOT obstruction is defined as a peak pressure gradient ≥30 mmHg at rest or during physiological provocation. A gradient ≥50 mmHg is considered to be haemodynamically important. Exercise stress echocardiography should be considered in individuals with exertional symptoms who have resting systolic anterior motion of the mitral valve leaflets but who do not reveal LVOT obstruction or show only mild to moderate LVOT obstruction with the aforementioned manoeuvres.

5.5.1.6 Cardiac magnetic resonance imaging

CMR imaging is increasingly recognized as a necessary tool for confirming diagnosis and to assess risk stratification in individuals with HCM. Late gadolinium enhancement (LGE), indicative of myocardial fibrosis, may be present in up to 75% of patients with HCM and, by itself, is a poor discriminator of outcomes. However, the presence of extensive (≥15% of LV myocardium) LGE may identify individuals at increased risk of ventricular tachyarrhythmias and SCD.[367–370]

5.5.1.7 Exercise testing

Exercise testing (or CPET) should be part of the routine evaluation to assess functional capacity in an individual with HCM who intends to exercise. In addition, an abnormal BP response to exercise (defined as <20 mmHg increase in SBP from baseline, or exercise-induced hypotension)[371,372] and the presence of exercise-induced symptoms or arrhythmias are markers of high risk and should result in more conservative exercise recommendations.

5.5.1.8 Genetic testing

Currently genetic testing is reserved for familial cascade screening. It does not inform decisions relating to the risk of SCD/SCA and should not be performed for exercise risk stratification.

5.5.1.9 ESC risk score in HCM

The ESC risk score uses seven variables (age, syncope, family history of SCD from HCM, maximal LV wall thickness, left atrial diameter, LV outflow obstruction, NSVT) to assess the risk of SCD of patients with HCM.[355,373] This information can be inserted into an online calculator (https://doc2do.com/hcm/webHCM.html) to estimate individualized 5-year risk to provide guidance on whether a prophylactic ICD is indicated. For the purposes of these Guidelines the risk of SCD is defined as low if <4%, moderate if between ≥4% and <6%, and high if ≥6% in 5 years.

5.5.1.10 Exercise recommendation

On completion of the baseline evaluation, the physician should consider: (i) the presence of symptoms; (ii) ESC risk score; (iii) presence of resting or inducible LVOT obstruction during exercise; (iv) the haemodynamic (BP) response to exercise; and (v) the presence of resting or exercise-induced arrhythmias before recommending the appropriate form and intensity of exercise.

Although these Guidelines advocate for a more liberal approach to sports participation, it is indisputable that even the absence of all major risk factors does not convey immunity to SCD.[374] In addition, the ESC risk score relies on evidence derived from predominantly non-athletic cohorts.[373] Whereas there are no data to suggest that this approach to risk estimation is less valid in athletic individuals, one has to consider that it may not accurately reflect the risk of SCD in individuals exposed to the haemodynamic and metabolic stresses of high-intensity sports. Therefore, when advising an individual with HCM regarding participation in high-intensity exercise programmes and competitive sports, this consideration should be an integral part of the discussion during the shared decision-making process.

5.5.1.11 Special considerations

The age of an individual may have an impact on risk. The mean age of death in the largest series of SCD from the US was 18 years, with 65% of deaths occurring in athletes ≤17 years.[354] Although young age should not exclude an individual from high-intensity exercise in the absence of additional risk factors, it should be considered in the discussion with the individual and the parents or guardians. In addition, specific sports may pose a higher risk for SCD, such as highly dynamic, start–stop sports like basketball and football.[17,58]

Individuals who have a positive genotype but who do not reveal any phenotypic structural or arrhythmia features of HCM may engage in all sports. Such individuals should be assessed annually for phenotypic features and risk stratification purposes.

5.5.1.12 Follow-up

Annual follow-up is recommended for most individuals with HCM who exercise on a regular basis. More frequent (6-monthly) follow-up should be considered in adolescent individuals and young adults whose phenotype, and therefore risk of SCD, may still be evolving and who are more vulnerable to exercise-related SCD.[58,239]Follow-up evaluation should focus on assessment of disease progression and risk stratification. New symptoms should prompt interruption of exercise and re-evaluation.

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Recommendations for exercise and sports participation in individuals with hypertrophic cardiomyopathy

Recommendations	Class[a]	Level[b]
Exercise recommendations		
Participation in high-intensity exercise/competitive sports, if desired (with the exception of those where occurrence of syncope may be associated with harm or death), may be considered for individuals who do not have any markers of increased risk[c] following expert assessment.	IIb	C
Participation in low- or moderate-intensity recreational exercise, if desired, may be considered for individuals who have any markers of increased risk[c] following expert assessment .	IIb	C
Participation in all competitive sports, if desired, may be considered for individuals who are gene positive for HCM but phenotype negative.	IIb	C
Participation in high-intensity exercise (including recreational and competitive sports) is not recommended for individuals who have ANY markers of increased risk[c].	III	C
Follow-up and further considerations relating to risk		
Annual follow-up is recommended for individuals who exercise on a regular basis.	I	C
Six-monthly follow-up should be considered in adolescent individuals and young adults who are more vulnerable to exercise-related SCD.	IIa	C
Annual assessment should be considered for genotype-positive/phenotype-negative individuals for phenotypic features and risk stratification purposes.	IIa	C

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BP = blood pressure; ESC = European Society of Cardiology; HCM = hypertrophic cardiomyopathy; LVOT = left ventricular outflow tract obstruction cardiomyopathy; SCD = sudden cardiac death.
[a]Class of recommendation.
[b]Level of evidence.
[c]Markers of increased risk include: (i) cardiac symptoms or history of cardiac arrest or unexplained syncope; (ii) moderate ESC risk score (≥4%) at 5 years; (iii) LVOT gradient at rest >30 mmHg; (iv) abnormal BP response to exercise; (v) exercise-induced arrhythmias.
Refer to *Table 4* for different indices of exercise intensity and training zones.

5.5.2 Arrhythmogenic cardiomyopathy

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is defined pathologically by the presence of fibro-fatty replacement of the right ventricle and clinically by life-threatening VAs. The condition was initially recognized as a predominantly RV disease, and diagnosis is currently based on probabilistic Task Force Criteria that encompass electrophysiological, anatomical, functional, and clinical features of the disease.[375] Since its first description, the concept of ARVC has evolved to include concealed or subclinical phenotypes and biventricular disease. It is now well established that both ventricles are affected in most cases.[376–378] This has led to the development of a new term, arrhythmogenic cardiomyopathy, that embraces an array of diagnostic terms for different (genetic and acquired) pathologies. Although the definition of 'arrhythmogenic cardiomyopathy' is yet to be agreed, it can be considered as an umbrella term for a family of diseases that are characterized by biventricular myocardial abnormalities, including fibro-fatty infiltration and scarring, identified by pathological examination and/or cardiac imaging and VA.

The term arrhythmogenic cardiomyopathy (ACM) is used throughout these recommendations; however, it is important to recognize that most of the literature on the influence of exercise on disease progression and risk of SCD is derived from cohorts with classical ARVC. This is reflected in the recommendations provided in these Guidelines. It is possible therefore that the recommendations may not accurately reflect predominantly LV disease, which constitutes a small proportion of the disease spectrum where the impact of exercise on disease phenotype and risk is less clarified than the RV variant. Where appropriate, guidance is provided for other conditions that can be reasonably considered under the umbrella of ACM [including subtypes of dilated cardiomyopathy (DCM)].

5.5.2.1 Risk stratification in arrhythmogenic cardiomyopathy

ACM accounts for a significant proportion of SCDs in young and athletic individuals.[28] Established risk factors for SCD that should prompt consideration for an ICD include aborted SCD, unheralded syncope, ventricular tachycardia, and impaired RV and/or LV systolic function.[379] A novel risk prediction model for VAs has recently been proposed but is yet to be validated.[380] Regular and high-intensity exercise programmes are associated with acceleration of the disease process and worse outcomes.[381–389]

In an experimental model of heterozygous plakoglobin-deficient mice, exercise training accelerated RV dysfunction and arrhythmias.[382] Similar results have been confirmed in human desmosomal mutation carriers participating in vigorous (>70% VO_{2max}) endurance sports.[384] Similar findings were reported in patients with ACM and asymptomatic gene-positive family members, despite a more conservative definition of athletic status (exercise with intensity ≥6 METs for ≥4 h/week for ≥6 years).[386] Recently, the results from the North American multidisciplinary study reported that patients engaging in competitive sports were at two-fold increased risk of ventricular tachyarrhythmias or death and earlier presentation of symptoms, compared with patients who participated in recreational sports and sedentary individuals.[385] Among patients engaging in competitive sports, early age of sports initiation was associated with premature presentation of symptoms and adverse clinical profile. Reducing exercise intensity was associated with a substantial decrease in the risk of ventricular tachyarrhythmias or death, to the same level as inactive patients.[385] Finally, in a multinational registry of 393 competitive athletes implanted with an ICD who continued to participate in regular competitions, 20% of athletes with ACM received a shock during exertion compared to 10% at rest, during a median follow-up of 44 months. The diagnosis of ACM was the only variable associated with receiving appropriate shocks during competition.[359,389]

5.5.2.2 Baseline assessment of patients with arrhythmogenic cardiomyopathy

A systematic approach is required when assessing individuals with ACM who request exercise advice. The baseline evaluation should include a comprehensive history of symptoms and family history of ACM or SCD, assessment of the severity of the ACM phenotype, and the presence of any conventional risk factors for SCD/SCA.

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5.5.2.3 History

Syncope due to presumed arrhythmia is an important risk marker for SCD/SCA and a predictor of future appropriate ICD therapies.[390–394] The presence of symptoms attributed to ACM should reinforce the conservative exercise recommendations. Individuals with a history of cardiac arrest or unheralded syncope and individuals with exercise-induced symptoms should be advised to engage only in low-intensity recreational exercise programmes.

5.5.2.4 Resting and ambulatory ECG

Apart from its diagnostic utility, the 12-lead ECG may provide useful information relating to risk stratification in ACM. The presence of extensive T-wave inversion affecting ≥3 precordial leads or T-wave inversion in two of the three inferior leads confers some additional risk for SCD/SCA.[395,396]

Ambulatory ECG monitoring is important for detecting VAs. Every effort should be made for the monitoring period to include the proposed exercise session. The presence of NSVT or significant burden of ventricular ectopy (≥1000/24 h), even in asymptomatic individuals, confers an increased risk of fatal arrhythmias.[392,393,397]

5.5.2.5 Echocardiography and cardiac magnetic resonance imaging

In relation to risk stratification for SCD, the clinician should assess the severity of RV and LV involvement in terms of ventricular dilatation and systolic dysfunction. CMR imaging is more useful than echocardiography for assessing RV wall motion abnormalities and can also quantify the degree of myocardial fat infiltration and/or scar. The more extensive the disease the higher the arrhythmic risk.[398,399]

5.5.2.6 Exercise testing

Exercise testing should be part of the routine assessment of every individual with ACM who wishes to exercise, as it can provide information regarding functional capacity and risk stratification. Exercise testing in patients with ACM should not be performed during 'hot phases'. The presence of exercise-induced symptoms or arrhythmias should result in more conservative recommendations.

5.5.2.7 Genetic testing

Genotype may also be of prognostic value. In the ARVC variant, a number of studies have reported that carriers of multiple pathogenic variants in the same desmosomal gene or mutations in ≥2 genes may have an almost four-fold higher arrhythmic risk than those with a single mutation.[400] Particular genotypes such as DSP and TMEM43, but also LMNA and FLNC, associated with other ACM phenotypes (see *section 5.5.4*) have a propensity for high arrhythmic burden that can pre-date the structural phenotype.[401,402]

5.5.2.8 Exercise recommendations

The overall scientific evidence supports the concept that in patients with ACM participation in high-intensity sports should be discouraged, because it is associated with accelerated disease progression, greater risk of VAs and major events. This recommendation is also applicable to genetic carriers of pathogenic variants for ACM even in the absence of overt disease phenotype.

5.5.2.9 Special considerations

Young age of presentation and male sex are associated with increased risk of malignant arrhythmias in ACM.[379] Although young age should not exclude an individual from moderate-intensity exercise in the absence of high-risk features, age should be considered in the discussion with the patient and the parents. In addition, one should consider that specific highly dynamic, start–stop sports, such as basketball and football, may pose a higher risk of SCD particularly in athletes who compete at the highest level.[17,365]

5.5.2.10 Follow-up

An annual follow-up is recommended for most individuals with ACM who exercise on a regular basis. More frequent (6-monthly) follow-up should be considered for adolescent and young adults whose ACM phenotype, and therefore risk of SCD, may still be evolving, particularly if they engage in moderate- to high-intensity exercise. More frequent follow-up should also be considered in individuals with high arrhythmic risk genotypes such as DSP, TMEM43, and carriers of multiple pathogenic variants. New symptoms should prompt interruption of exercise and re-evaluation.

Recommendations for exercise and sports participation in individuals with arrhythmogenic cardiomyopathy

Recommendations	Class[a]	Level[b]
Exercise recommendations		
Participation in 150 min of low-intensity exercise per week should be considered for all individuals.	IIa	C
Participation in low- to moderate-intensity recreational exercise/sports, if desired, may be considered for individuals with no history of cardiac arrest/VA, unexplained syncope, minimal structural cardiac abnormalities, <500 PVCs/24 h and no evidence of exercise-induced complex VAs.	IIb	C
Participation in high-intensity recreational exercise/sports or any competitive sports is not recommended in individuals with ACM, including those who are gene positive but phenotype negative.[384,386]	III	B
Follow-up and further considerations relating to risk		
Annual follow-up is recommended for individuals who exercise on a regular basis.	I	C
Six-monthly follow-up should be considered in adolescent individuals and young adults who are more vulnerable to exercise-related SCD.	IIa	C
Annual assessment should be considered for genotype-positive/phenotype-negative individuals for phenotypic features and risk stratification purposes.	IIa	C
Six-monthly follow-up should also be considered in individuals with high arrhythmic risk genotypes such as DSP, TMEM43, and carriers of multiple pathogenic variants.	IIa	C

ACM = arrhythmogenic cardiomyopathy; PVC = premature ventricular contraction; SCD = sudden cardiac death; VA = ventricular arrhythmia.
[a]Class of recommendation.
[b]Level of evidence.
Refer to *Table 4* for different indices of exercise intensity and training zones.

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5.5.3 Exercise recommendations in individuals with left ventricular non-compaction

LV non-compaction (LVNC) is an unclassified cardiomyopathy characterized by prominent trabeculation and deep recesses that communicate with the LV cavity.[403,404] Clinical presentation of LVNC includes progressive LV systolic dysfunction, ventricular tachyarrhythmias, and thromboembolic events.[404]

Athletes often demonstrate LV hypertrabeculation and up to 8% fulfil the echocardiographic criteria for a diagnosis of LVNC.[405] It is hypothesized that an increased cardiac preload may unmask LV trabecular morphology.[406] Therefore, among athletic individuals, the suspicion of LVNC should only be considered in those who fulfil echocardiographic criteria for LVNC but also have either LV systolic dysfunction (EF < 50%), symptoms suggestive of cardiac disease, or a positive family history of LVNC.[407–409] Additional echocardiographic criteria include a very thin compacted epicardial layer (5 mm in end-diastole on CMR, or <8 mm in systole) and abnormal myocardial relaxation (average E' < 9 cm/s on tissue Doppler imaging).[404,405,410,411] Such athletes will require further assessment with CMR, exercise echocardiography, and Holter monitor to assess the presence of LV fibrosis, cardiac thrombi, contractile reserve, and exercise-induced complex arrhythmias.[405,406]

5.5.3.1 Risk stratification

The clinical outcomes of LVNC are determined by the presence of symptoms, severity of LV dysfunction, and the nature of the VAs. There are no reported adverse cardiac events in the absence of LV dysfunction regardless of the severity of LV trabeculation.[405–409]

5.5.3.2 Follow-up

Regular follow-up is recommended for individuals with LVNC. New symptoms should prompt interruption of exercise and re-evaluation.

Recommendations for exercise in individuals with left ventricular non-compaction cardiomyopathy

Recommendation for diagnosis	Class[a]	Level[b]
A diagnosis of LVNC in athletic individuals should be considered if they fulfil imaging criteria, in association with cardiac symptoms, family history of LVNC or cardiomyopathy, LV systolic (EF<50%) or diastolic (E'<9 cm/s) dysfunction, a thin compacted epicardial layer (<5 mm in end-diastole on CMR, or <8 mm in systole on echocardiography), or abnormal 12-lead ECG.[404,405,410,411]	IIa	B
Exercise recommendations		
Participation in high-intensity exercise and all competitive sports, if desired, with the exception where syncope may cause serious harm or death, may be considered in asymptomatic individuals with LVNC and LVEF≥50% and absence of frequent and/or complex VAs.	IIb	C
Participation in recreational exercise programmes of low to moderate intensity, if desired, may be considered in individuals with LVEF 40–49% in the absence of syncope and frequent or complex VAs on ambulatory Holter monitoring or exercise testing.	IIb	C
Participation in high- or very high-intensity exercise including competitive sports, if desired, may be considered for individuals who are gene positive for LVNC but phenotype negative (with the exception of lamin A/C or filamin C carriers).	IIb	C
Participation in high-intensity exercise or competitive sports is not recommended in individuals with any of the following: symptoms, LVEF<40% and/or frequent and/or complex VAs on ambulatory Holter monitoring or exercise testing.	III	C
Follow-up and further considerations		
Annual assessment for risk stratification is recommended for individuals with LVNC and genotype-positive/phenotype-negative individuals who exercise on a regular basis.	I	C

CMR = cardiac magnetic resonance; ECG = electrocardiogram; EF = ejection fraction; LV = left ventricular; LVEF = left ventricular ejection fraction; LVNC = left ventricular non-compaction; VA = ventricular arrhythmia.
[a]Class of recommendation.
[b]Level of evidence.

5.5.4 Exercise recommendations in individuals with dilated cardiomyopathy

DCM is characterized by LV or biventricular systolic dysfunction with or without dilatation that are not explained by abnormal loading conditions or CAD. Possible causes include genetic predisposition, myocarditis, drugs, toxins, peripartum cardiomyopathy, and, in some cases, the cumulative effect of more than one factor.[412]

The clinical spectrum of disease may range from a mild phenotypic expression characterized by absence of symptoms, isolated LV dilatation and normal or low-normal systolic function, to an overt disease phenotype with limiting symptoms and significant systolic dysfunction. Ventricular arrhythmias are common in DCM, particularly in individuals with previous myocarditis, or with lamin A/C mutations and filamin C mutations.[413,414] The risk of SCD in DCM is 2–3% per year and increases with lower EF and higher NYHA class.[415] Exercise training improves functional capacity, ventricular function, and quality of life in patients with DCM and should therefore be considered as an integral part of the management of affected individuals.[416,417] However, intensive exercise and competitive sports are reported as a cause of SCD in DCM.[28,46,58,413,418]

LV cavity enlargement in trained individuals that is not associated with systolic dysfunction and outside the context of a familial disease, represents a benign physiological adaptation if it is consistent with the type of sports participated (usually, endurance sports) and the body size of the athlete. Conversely, a mildly reduced EF (45–50%) in an athlete with an enlarged LV cavity should not merely be considered as a normal adaptation. In such cases assessment of LV function during exercise may provide important diagnostic clues.[319] Failure to increase EF at peak exercise by >10% compared with the baseline value may suggest a pathological condition.[319,419,420] The presence of diastolic dysfunction or reduced peak oxygen consumption on CPET may also provide supporting information for the differential diagnosis. CMR has emerged as an important tool for the diagnosis and risk stratification of DCM.

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Specifically, the presence of LGE, with the typical mid-wall distribution, has been associated with increased risk of VAs and SCD.[319,419,421–424]

5.5.4.1 Baseline assessment of patients with dilated cardiomyopathy

Clinical evaluation of affected individuals who request exercise advice should aim to: (i) ascertain the potential aetiology; (ii) assess the clinical status including exercise history and functional capacity; (iii) review the degree of LV dilatation and dysfunction; (iv) assess the haemodynamic response to exercise; and (v) assess the presence of exercise-induced symptoms or arrhythmias.

In general, symptomatic individuals with DCM should abstain from most competitive and leisure sports or recreational exercise associated with moderate or high exercise intensity. A select group of asymptomatic individuals with DCM who have mildly impaired LV function (LVEF 45–50%) without exercise-induced arrhythmias or significant myocardial fibrosis may participate in most competitive sports.

5.5.4.2 Special considerations

Although the natural history of most pathogenic variants is unknown, it would be reasonable to permit intensive exercise and competitive sports in most individuals with pathogenic variants implicated in DCM in the absence of overt features of DCM. Special consideration, however, should be given to individuals with pathogenic variants that are associated with an increased risk of life-threatening arrhythmias such as lamin A/C or filamin C mutations. There is emerging evidence that exercise may have an adverse effect on cardiac function and risk for potentially fatal arrhythmias in individuals harbouring pathogenic variants in lamin A/C.[425–427] Affected individuals should not engage in any competitive sports or recreational exercise of high or very high intensity irrespective of the severity of LV dysfunction and dilatation.[428,429]

5.5.4.3 Follow-up.

Regular follow-up is recommended for most individuals with DCM. New symptoms should prompt interruption of exercise and re-evaluation

Recommendations for exercise in individuals with dilated cardiomyopathy

Recommendations	Class[a]	Level[b]
Participation in low- to moderate-intensity recreational exercise should be considered in all individuals with DCM, regardless of the EF, in the absence of limiting symptoms, and exercise-induced VAs.	IIa	C
Participation in high- or very high-intensity exercise including competitive sports (with the exception of those where occurrence of syncope may be associated with harm or death) may be considered in asymptomatic individuals who fulfil all of the following: (i) mildly reduced LV systolic function (EF 45–50%); (ii) absence of frequent and/or complex VAs on ambulatory Holter monitoring or exercise testing; (iii) absence of LGE on CMR; (iv) ability to increase EF by 10–15% during exercise; and (v) no evidence of high-risk genotype (lamin A/C or filamin C).	IIb	C
Participation in all competitive sports may be considered in individuals with DCM who are genotype positive and phenotype negative, with the exception of carriers of high-risk mutations (lamin A/C or filamin C).	IIb	C
Participation in high- or very high-intensity exercise including competitive sports is not recommended for individuals with a DCM and any of the following: (i) symptoms or history of cardiac arrest or unexplained syncope; (ii) LVEF<45%; (iii) frequent and/or complex VAs on ambulatory Holter monitoring or exercise testing; (iv) extensive LGE (>20%) on CMR; or (v) high-risk genotype (lamin A/C or filamin C).	III	C
Follow-up recommendations		
Annual follow-up is recommended for individuals with DCM who exercise on a regular basis.	I	C
Six-monthly follow-up should be considered in individuals with high-risk mutations and adolescent individuals and young adults whose DCM phenotype may still be evolving and who are more vulnerable to exercise-related SCD.	IIa	C
Annual assessment should be considered for genotype-positive/phenotype-negative individuals for phenotypic features and risk stratification purposes.	IIa	C

CMR = cardiac magnetic resonance; DCM = dilated cardiomyopathy; EF = ejection fraction; LGE = late gadolinium enhancement; SCD = sudden cardiac death; VA = ventricular arrhythmia.
[a]Class of recommendation.
[b]Level of evidence.

5.5.5 Exercise recommendations in individuals with myocarditis and pericarditis

5.5.5.1 Myocarditis

Myocarditis is a non-ischaemic inflammatory disease of the myocardium, which may cause cardiac dysfunction and arrhythmias. Myopericarditis is defined as a primary pericarditis with associated myocardial inflammation and biomarker evidence of myocyte necrosis.[430,431] The aetiology of myocarditis is heterogenous, but viral infection is the most common cause in the developed world. Enterovirus, Coxsackie B virus, parvovirus B-19, and human herpesvirus 6 are the most frequently responsible infectious pathogens.[432,433] In the context of young individuals, toxins such as cocaine and amphetamine-based supplements should also be evaluated in the clinical history.[430]

The clinical presentation is highly variable and the diagnosis can be challenging. The illness may be proceeded by coryzal symptoms and athletic individuals may present with non-specific features of general malaise, fatigue, or diarrhoea.[430,431] At the other extreme, myocarditis may simulate MI or present with symptomatic supraventricular and VAs unexplained by other causes, HF, cardiogenic shock, or SCD.

Approximately 50% of individuals reveal full resolution of LV function within 30 days, 25% show persistent cardiac dysfunction, and 12–25% progress to fulminant HF. LV dysfunction is an important prognostic factor in the long term.[28,434]

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5.5.5.2 Diagnosis

Serum cardiac troponin is usually elevated in myopericarditis and is a sensitive marker of cardiac inflammation-induced myocyte necrosis.[435]

The ECG has low sensitivity and electrical anomalies are non-specific. ECG patterns vary from non-specific T-wave and ST-changes to ST-segment elevation mimicking MI, left bundle branch block (LBBB), or frequent and/or complex ventricular and supraventricular arrhythmias, or atrioventricular block, or low QRS voltages in the presence of a pericardial effusion.[436]

Recognized echocardiographic features of overt myocarditis include a non-dilated LV cavity with increased myocardial wall thickness (when oedema is present), or mildly dilated LV cavity with a thinned myocardial wall, usually with regional wall motion abnormalities.[437] The global LV systolic function may range from being almost normal to severely depressed. Regional wall motion abnormalities may be present.[438]

CMR is the most useful diagnostic tool and has excellent sensitivity for detecting myocardial hyperaemia, inflammation, oedema and/or focal scar.[439,440] The Lake Louise Criteria and LGE are now complemented by CMR techniques of T1/T2 mapping and extracellular volume fraction (ECV).[440–442] The extent and distribution of LGE with non-ischaemic pattern are independent predictors of CV events during follow-up.[439,440,443–447] Namely, a 10% increase of LGE volume conveys a 79% increase in the risk of major CV events.[448,449]

Endomyocardial biopsy is the gold standard for the diagnosis of myocarditis.[450,451] A histological diagnosis allows distinction between the different types of inflammatory processes, (i.e. giant cell myocarditis), and guides treatment in life-threatening presentations.[430,445,452] The diagnostic yield of endomyocardial biopsy can be improved by analysing the viral genome through DNA-RNA extraction and reverse transcriptase polymerase chain reaction (RT-PCR) amplification, which has the advantage of identifying the disease-causing pathogen.[445]

5.5.5.3 Risk stratification

Case series have established myocarditis as a risk factor for SCD, which accounts for up to 2–20% of sudden death in athletes.[17,18,28,430,453,454] Murine models have shown that daily exercise in mice infected with Coxsackie virus is associated with increased viral titres, fulminant myocarditis, and sudden death.[455] These animal models provide some insight into the mechanisms of SCD with exercise, which appears to cause an accelerated and progressive inflammatory response.[455–458]

5.5.5.4 Exercise recommendations for individuals with myocarditis

Athletic individuals with a probable or definitive diagnosis of recent myocarditis should be advised to abstain from competitive sports or leisure sports while active inflammation is present, regardless of age, sex, or extent of LV systolic dysfunction.[459,460]

The duration of myocardial inflammation can be highly variable and may take several months for full resolution. Both the ESC and AHA recommend abstinence from moderate- to high-intensity exercise for a period of 3–6 months,[459,460] although the precise timing for return to competitive or recreational sports involving moderate- or high-intensity exercise may be guided by the presence of inflammation on T2-weighted images and LGE uptake on CMR.[3,461]

Individuals with myocarditis should have a comprehensive evaluation after complete recovery to assess the risk of exercise-related SCD. Imaging studies, exercise stress test, and Holter monitor provide essential information for risk stratification. Depressed LV function, presence of LGE and complex VAs during exercise or Holter monitoring are recognized risk markers for adverse outcomes.[455,462,463]

Repeat evaluation should consist of measurement of troponin and biomarkers of inflammation, echocardiography, and prolonged ECG monitoring. Individuals without evidence of ongoing inflammation should undergo an exercise stress test. A CMR should be repeated if myocardial oedema or LGE was present during the acute illness. Return to sporting activities should be considered, in asymptomatic individuals, with normal troponin and biomarkers of inflammation, normal LV systolic function on echocardiography and CMR, no evidence of ongoing inflammation or myocardial fibrosis on CMR, good functional capacity, and absence of complex arrhythmias during exercise on prolonged ECG monitoring .[430,434,453,459,460,464]

Individuals with previous myocarditis are at risk of recurrence and silent clinical progression, and the presence of LGE during the acute presentation is associated with increased incidence of major adverse cardiac events; therefore, periodic re-evaluation is advised on an annual basis.[443,445,454,463]

Among individuals with healed myocarditis with persistence of LGE on CMR but no myocardial oedema at 3–6 months, those who are asymptomatic, with normal troponin and biomarkers of inflammation, normal LV systolic function, no evidence of ongoing inflammation on CMR, and absence of complex arrhythmias during exercise on prolonged ECG monitoring (48 h Holter ECG and exercise stress testing), should be evaluated on a case by case basis and may return to competitive sports on an individual basis. In contrast, individuals with extensive myocardial scar (>20% LGE) and persistent LV dysfunction should abstain from exercise programmes and sports activities involving moderate or high physical intensity.

5.5.6 Pericarditis

Pericarditis is defined as an inflammatory disorder of the pericardium,[430,465] which may be preceded by upper respiratory or gastrointestinal symptoms. As with myocarditis, viral pathogens are the most commonly implicated pathogens in the western world.

5.5.6.1 Diagnosis

The ECG is non-specific but may reveal characteristic concave ST-segment elevation in most leads and/or PQ depression in the acute phase. Echocardiography may reveal a pericardial effusion. CMR should be considered in individuals with raised cardiac troponin levels to assess for concomitant myocardial inflammation. Furthermore, CMR will identify active inflammation of the pericardium, thickened pericardial layers, and any signs of pericardial constriction.

5.5.6.2 Risk stratification

Pericarditis is generally associated with an excellent prognosis.[430,465,466] However, there are a subset of patients who may be at greater risk of recurrence and these include individuals with a temperature >38°C at

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presentation, large pericardial effusion, and those who are resistant to therapy with non-steroidal anti-inflammatory drugs.[465,466]

5.5.6.3 Exercise recommendations for individuals with pericarditis

Exercise should be avoided in individuals during active pericarditis. Individuals can return to exercise after complete resolution of the active disease.[467] Individuals with a milder clinical course and rapid resolution can return to sporting activities within 30 days. However, in more severe cases, it may be necessary to wait for a period of 3 months for complete resolution followed by re-evaluation before returning to sports.

Asymptomatic individuals with a small pericardial effusion occasionally detected on echocardiography in the absence of clinical correlates should be monitored with periodic surveillance, but should not be restricted from sports participation. Competitive sports and/or moderate- to high-intensity leisure-time activities should be avoided in individuals with constrictive pericarditis. Individuals with myopericarditis should be managed according to the recommendations for myocarditis.

Recommendations for exercise in individuals with myocarditis

Recommendations	Class[a]	Level[b]
Comprehensive evaluation, using imaging studies, exercise stress test and Holter monitoring, is recommended following recovery from acute myocarditis to assess the risk of exercise-related SCD.[455,462,463]	I	B
Return to all forms of exercise including competitive sports should be considered after 3–6 months in asymptomatic individuals, with normal troponin and biomarkers of inflammation, normal LV systolic function on echocardiography and CMR, no evidence of ongoing inflammation or myocardial fibrosis on CMR, good functional capacity, and absence of frequent and/or complex VAs on ambulatory Holter monitoring or exercise testing.[430,434,453,459,460,464]	IIa	C
Among individuals with a probable or definitive diagnosis of recent myocarditis, participation in leisure-time or competitive sports while active inflammation is present is not recommended.[459,460]	III	C
Participation in moderate- to high-intensity exercise for a period of 3–6 months after acute myocarditis is not recommended.[459–461,467]	III	B
Participation in leisure exercise or competitive sports involving high intensity in individuals with residual myocardial scar and persistent LV dysfunction is not recommended.	III	C

CMR = cardiovascular magnetic resonance; LV = left ventricular; SCD = sudden cardiac death; VA = ventricular arrhythmia.
[a]Class of recommendation.
[b]Level of evidence.

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Recommendations for exercise in individuals with pericarditis

Recommendations	Class[a]	Level[b]
Return to all forms of exercise including competitive sports is recommended after 30 days to 3 months for individuals who have recovered completely from acute pericarditis, depending on clinical severity.[459,460]	I	C
Participation in leisure-time or competitive sports is not recommended for individuals with a probable or definitive diagnosis of recent pericarditis while active inflammation is present, regardless of age, sex, or extent of LV systolic dysfunction.[459,460]	III	C
Participation in moderate- to high-intensity exercise, including competitive sports, is not recommended for individuals with constrictive pericarditis.	III	C

LV = left ventricle.
[a]Class of recommendation.
[b]Level of evidence.

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5.6 Exercise recommendations in individuals with arrhythmias and channelopathies

5.6.1 A general management framework

When individuals with known arrhythmias or with a potentially arrhythmogenic condition want to engage in sports activity, three principle questions should guide management: (1) is there an increased risk for life-threatening arrhythmias?; (2) how does one control symptoms due to arrhythmias, during sports, but also at rest?; and (3) what is the impact of sports on the natural progression of the arrhythmogenic condition? The general view on the association between sports and arrhythmias is that exercise sets the stage for an arrhythmia in the context of an underlying and pre-existing condition, be it structural, electrical, inherited, or acquired. Moreover, regular exercise programmes may induce or accelerate the progression of ARVC,[382,384] even among those without underlying mutations.[383,387,468] Conceptually, all the structural and functional cardiac adaptations to regular intensive exercise may contribute to the development of arrhythmias, at the atrial, nodal, and ventricular level.[469] This concept explains why recommendations for sports participation in individuals with arrhythmogenic conditions are so complex.

5.6.2 Atrial fibrillation

5.6.2.1 Patients without atrial fibrillation

Moderate, regular PA is a cornerstone in the prevention of AF through modifying many of its predisposing factors.[297,470–473] Patients at risk of AF should therefore be motivated to exercise (see *section 4.2*). Conversely, AF is more prevalent in active and former male master athletes and those performing high-intensity endurance sports, suggesting a U-shaped relationship between habitual exercise and AF.[471,474–477 478–481] This association has not been confirmed in women.[474]

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5.6.2.2 Prognostic and symptomatic relevance of AF during sports

Underlying structural heart disease or pre-excitation should always be excluded before advising sports activity in individuals with recognized AF. It is also important to exclude hyperthyroidism, alcohol abuse, and (illicit) drug use. Intensive sports participation should be temporarily stopped until an identified underlying cause is corrected.

Rapid atrioventricular nodal conduction of AF during exercise may lead to symptoms, including dizziness, syncope, fatigue, or impaired physical performance. Therefore, the individual should be instructed to stop PA on the emergence of symptoms and rate control should be optimized. Rapid 1: 1 conduction can occur, especially during atrial tachycardia or atrial flutter (AFL); therefore, if AFL has been documented, prophylactic cavo-tricuspid isthmus ablation should be considered. If there is evidence for adequate rate control while in AF through an exercise stress test or ECG monitoring during training or competition, all sports participation is possible in asymptomatic individuals.

Achieving adequate rate control can be difficult, however. Beta-blockers are the logical choice but may not be tolerated due to their impact on physical performance. Calcium-channel blockers and digitalis are usually not potent enough when used alone. Often a combination of individually titrated negatively chronotropic agents is needed, while avoiding sinus bradycardia at rest or chronotropic incompetence during exercise.

Rhythm control is equally complicated. Class III antiarrhythmic drugs are usually insufficient for control (sotalol) or relatively contraindicated in a young population (amiodarone). Although class I drugs may be able to prevent recurrences of AF, they should not be used in monotherapy, since these may increase the propensity to develop AFL ('class I AFL'), which in the absence of adequate rate control may lead to 1: 1 atrioventricular conduction, high ventricular rates, and very profound intraventricular conduction slowing, with haemodynamic compromise.[482,483] Therefore, prophylactic cavo-tricuspid-isthmus ablation should be considered if class I drugs are prescribed in monotherapy in athletes.

In patients with sporadic AF, class I drugs may be considered only for acute cardioversion, i.e. as a 'pill-in-the-pocket' approach. These patients should refrain from sports as long as AF persists, and until two half-lives of the antiarrhythmic drug have passed.[484]

Prescription of oral anticoagulants (OAC) depends on the clinical risk profile (mainly CHA_2DS_2-VASc score).[485] Sports with direct bodily contact or prone to trauma should be avoided in patients on OAC.[486]

Catheter ablation by pulmonary vein isolation (PVI) should be considered if drug therapy fails or as first-line therapy if drug therapy is not desired.[487] Several small series have shown that the outcome of PVI in athletes with paroxysmal AF is similar to that in non-athletic patients.[488,489]

5.6.2.3 Impact of sports continuation on the natural progression of atrial fibrillation after ablation

If there are no recurrences of AF within 1 month of a successful ablation procedure, sports activity may be resumed. It is unknown whether continuation of sports after successful PVI might progress the disease process and lead to recurrence of non-pulmonary vein-dependent AF in the future. Therefore, no firm recommendation can be made about the 'safe' dose of sports after ablation.

Recommendations for exercise in individuals with atrial fibrillation

Recommendations	Class[a]	Level[b]
Regular physical activity is recommended to prevent AF.[297,470–473]	I	A
Evaluation and management of structural heart disease, thyroid dysfunction, alcohol or drug abuse, or other primary causes of AF is recommended before engaging in sports.[485]	I	A
Counselling about the effect of long-lasting intense sports participation on (recurrence of) AF is recommended in individuals with AF who exercise vigorously for prolonged periods, especially in middle-aged men.[471,475,481,490]	I	B
AF ablation is recommended in exercising individuals with recurrent symptomatic AF, and/or in those who do not want drug therapy, given its impact on athletic performance.[488,489]	I	B
The ventricular rate while exercising with AF should be considered in every exercising individual (by symptoms and/or by ECG monitoring), and titrated rate control should be instituted.	IIa	C
Participation in sports without antiarrhythmic therapy should be considered in individuals without structural heart disease, and in whom AF is well tolerated.	IIa	C
Cavo-tricuspid isthmus ablation should be considered in those with documented flutter who want to engage in intensive exercise, to prevent atrial flutter 1 : 1 atrioventricular conduction.	IIa	C
Prophylactic cavo-tricuspid isthmus ablation to prevent flutter should be considered in individuals with AF who want to engage in intensive exercise and in whom class I drug therapy is initiated.	IIa	C
The use of class I antiarrhythmic drugs as monotherapy, without proof of adequate rate control of AF/AFL during vigorous exercise, is not recommended.[482,483]	III	C
After ingestion of pill-in-the-pocket flecainide or propafenone, participation in intensive sports is not recommended until two half-lives of the antiarrhythmic drug have elapsed (i.e. up to 2 days).[484]	III	C
Sports with direct bodily contact or prone to trauma are not recommended in exercising individuals with AF who are anticoagulated.[485]	III	A

AF = atrial fibrillation; AFL = atrial flutter; ECG = electrocardiogram.
[a]Class of recommendation.
[b]Level of evidence.

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5.6.3 Supraventricular tachycardia and Wolff-Parkinson-White syndrome

The term paroxysmal supraventricular tachycardia (PSVT) includes (i) atrioventricular nodal re-entrant tachycardia (AVNRT; most common); (ii) atrioventricular re-entrant tachycardia (AVRT) involving an accessory pathway; or (iii) atrial tachycardia.

Ventricular pre-excitation on the resting ECG is due to an accessory pathway (AP) with antegrade conduction. The prevalence of pre-excitation in the general population varies from 0.1–0.3%.[491,492] Wolff-Parkinson-White (WPW) syndrome is defined as the presence of paroxysmal arrhythmias in a patient with pre-excitation.

5.6.3.1 Prognostic and symptomatic relevance of paroxysmal supraventricular tachycardia without pre-excitation

PSVT without pre-excitation and without associated structural heart disease is not life-threatening, although the arrhythmia may result in dizziness and exhaustion that requires cessation of exercise. Syncope is uncommon. Pre-excitation, however, may be associated with sudden death (see later); therefore, it is important to exclude the presence of latent pre-excitation, by performing carotid sinus massage or an adenosine-test in sinus rhythm.[493]

Athletes with PSVT should stop exercise in the event of palpitations since rapid heart rates may cause (pre)syncope. Individuals with proven PSVT without pre-excitation should be educated on how to safely perform vagal manoeuvres (such as carotid sinus massage or, preferably, Valsalva manoeuvre) to facilitate termination of the arrhythmia.[494] Exercise may be resumed after termination of the arrhythmia. Prophylactic drug treatment with beta-blockers or calcium antagonists with atrioventricular nodal blocking properties can be considered, although it has limited efficacy. Class I drugs have no role in the management of PSVT, since they can cause life-threatening arrhythmias (see earlier).

If competitive athletic activity is desired, curative treatment by ablation should be considered. Ablation outcome is equally safe and has similar acute success rates in athletes and non-athletes.[495] If the PSVT is only sporadic and transient and not associated with haemodynamic consequences, even when it develops during exercise, or in cases where ablation is not desired or unsuccessful, sports activity is permissible when there is no increased risk of a fatality from a potential loss of consciousness (such as motorsports drivers, parachute jumpers, divers, and so on).

5.6.3.2 Prognostic and symptomatic relevance of pre-excitation

It has been estimated that one third of patients with WPW syndrome may develop AF and, in such cases, rapid conduction over the AP can lead to ventricular fibrillation (VF) and sudden death. Given the fact that AF is more common in athletes, pre-excitation constitutes a prognostic concern in athletes. The risk for sudden death in patients with pre-excitation varies in population-based studies from 0.15–0.20%, and usually presents during exercise or emotional stress.[496]

Evaluation of the athlete with ventricular pre-excitation should exclude associated structural cardiac disease, such as HCM or Ebstein anomaly. Minimal or 'latent' pre-excitation can be unmasked on a 12-lead ECG during sinus rhythm by vagal manoeuvres or intravenous administration of adenosine. Prolongation of the PR interval without a change in the QRS morphology, or transient atrioventricular block, excludes non-intermittent latent pre-excitation. Pre-excitation may be intermittent, which usually indicates low risk properties of the pathway. However, some accessory pathways may be potentiated by adrenergic stimuli. Therefore, exercise testing excluding pre-excitation at peak exercise is recommended before clearance for sports.

Ablation of the AP is recommended in competitive and recreational athletes with pre-excitation and documented arrhythmias. In the event of transient, infrequent well-tolerated arrhythmia (even during exercise), good anticipation of an ablation procedure with increased risk (e.g. anteroseptal AP), or reluctance of the athlete to undergo ablation, management should be guided by assessment of the antegrade conduction characteristics of the AP using either non-invasive tests or an invasive electrophysiological (EP) study.

Non-invasive investigation examines for intermittent pre-excitation on ECG or Holter, for abrupt disappearance of pre-excitation after administration of a low dose of class I drugs, or for its abrupt disappearance during an exercise test.[497] In cases of a long refractory period and hence low risk for sudden death, continuation of sports activity is permitted without ablation on the understanding that sporting activity should be stopped in the event of recurrence of palpitations.

In competitive athletes with asymptomatic pre-excitation an EP study is warranted to evaluate the risk for sudden death. In the event of a high-risk finding (*Table 15*), ablation of the AP is recommended. For athletes who refuse ablation, or if the procedure is associated with high risk, such as an anteroseptal accessory pathway, participation in competitive sports activities can be discussed on an individual case by case basis including the use of pharmacological therapy, although there are currently no data about its efficacy. Sports in which the potential loss of consciousness could be fatal should be discouraged.

In recreational athletes with asymptomatic pre-excitation, risk assessment may first be pursued via non-invasive testing.[497] The sensitivity of non-invasive screening for AP properties that facilitate a fast ventricular response to AF/AFL is good, but its specificity is low.[498]

Of note, in children younger than 12 years, the risk of AF-induced VF and sudden death is very low. Generally, a conservative approach

Table 15 Findings during an invasive electrophysiological study (with the use of isoprenaline) indicating an accessory pathway with increased risk of sudden death

Findings
Inducibility of AVRT or AF[499]
A pre-excited R-R during AF ≤250 ms[498]
An antegrade refractory period ≤250 ms[498]
Presence of multiple accessory pathways[493]
Septal location of the accessory pathway (mainly posteroseptal and midseptal)[493, 497]

AF = atrial fibrillation; AVRT = atrioventricular re-entrant tachycardia.

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is recommended in this age group, although one study[499] suggested that prophylactic assessment and ablation reduces the risk of sudden death. There is a knowledge gap in the benefit/risk ratio of this approach and large-scale studies are required to address the issue.

Leisure-time and low- to medium-intensity exercise programmes can generally be resumed 1 week after ablation if there is no particular risk of recurrence of arrhythmia.

Resuming competitive sports is possible after 1–3 months, with further ECG follow-up at 6 months and 1 year (given the very small risk for late recurrence of pre-excitation).

Although there may be an association between (type of) AVNRT and history of sports, there are no data about higher recurrence rate post ablation when sports are resumed or not, and hence no reason to limit exercise programmes for such reason.

Recommendations for exercise and sports participation in individuals with paroxysmal supraventricular tachycardia and pre-excitation

Recommendations	Class[a]	Level[b]
In individuals with palpitations, a comprehensive assessment to exclude (latent) pre-excitation, structural heart disease, and VAs is recommended.[500]	I	B
Participation in all sports activities is recommended in individuals PSVT without pre-excitation.[500]	I	C
Ablation of the accessory pathway is recommended in competitive and recreational athletes with pre-excitation and documented arrhythmias.[500]	I	C
In competitive/professional athletes with asymptomatic pre-excitation, an EP study is recommended to evaluate the risk for sudden death.[497,500]	I	B
In competitive athletes with PSVT but without pre-excitation, curative treatment by ablation should be considered.	IIa	C

EP = electrophysiological; PSVT = paroxysmal supraventricular tachycardia; VA = ventricular arrhythmia.
[a]Class of recommendation.
[b]Level of evidence.

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5.6.4 Premature ventricular contractions and non-sustained ventricular tachycardia

5.6.4.1 Relation between number of premature ventricular contractions and risk

Only a minority of athletes exhibit frequent or complex VA with a prevalence similar to that of their sedentary counterparts.[502–505] Premature ventricular contractions (PVCs) may be a marker of underlying heart disease, the presence of which would confer an unfavourable prognosis even in asymptomatic individuals. Specific characteristics of the PVCs, including morphology (origin from the apex or free wall of the LV or RV), high burden, complexity (e.g. couplets, triplets, or non-sustained runs), multifocal origin, and/or increasing frequency with exercise should alert to the possibility of electrical, ischaemic, or structural heart disease.[505,506]

There is no absolute threshold of the number of PVCs that can be used as a cut-off for underlying disease. One study has shown that in asymptomatic athletes with >2000 PVCs per day, there was a 30% chance of finding an underlying structural or cardio-genetic disease.[503]

5.6.4.2 Morphology of premature ventricular contractions

The morphology of the PVCs may provide important prognostic information since some foci of origin are recognized as benign. The most prevalent entity in this respect are PVCs originating from the right or left ventricular outflow regions (RVOT/LVOT), showing a clear inferior axis with high voltages in the inferior limb leads. Early precordial transition (in V2, and certainly when V1 shows a right bundle branch morphology) suggests a left-sided origin.[507] RVOT/LVOT PVCs are thought to be the result of triggered activity, i.e. a local cellular cause, which has no negative prognostic implications. Although these RVOT/LVOT arrhythmias usually occur in structurally normal hearts, they may be the expression of subclinical arrhythmogenic cardiomyopathy. Cardiac imaging tests can help exclude structural heart disease in such athletes.

Less common locations of focal PVCs are around the mitral or tricuspid annulus, most often in a postero-septal location. These have a superior axis with LBBB or RBBB morphology. The PVCs originating from the His-Purkinje system typically have relatively narrow QRS complex with RBBB morphology and either left anterior or left posterior hemi-block. Lastly, intramyocardial foci may occur, often related to the papillary muscles or moderator band.[508]

PVCs of differing morphologies from the RV (i.e., wide LBBB and superior axis) in individuals with normal LV function should prompt investigations to exclude arrhythmogenic cardiomyopathy or sarcoidosis. Similarly, wide RBBB pattern, with superior axis and multifocal PVCs of LV origin should trigger investigations for non-ischaemic cardiomyopathy.

Very rarely, otherwise 'benign' PVCs arising from the Purkinje network may give rise to polymorphic ventricular tachycardia (VT) or VF due to their short coupling interval.[509,510] In such patients, the malignant electrical presentation mandates aggressive treatment. Finally, frequent but otherwise benign PVCs (usually defined as >10–15% of the total number of beats per 24 h) can impair LV function over time (PVC-induced cardiomyopathy), which may be reversible with medical treatment or catheter ablation.[511,512]

5.6.4.3 Premature ventricular contractions: response to exercise

Reduction or resolution of PVCs with increasing exercise load is typical of idiopathic and benign VAs, particularly those with an outflow tract morphology.[513,514] PVCs induced by exercise should be considered as a 'red flag', because VAs associated with heart diseases are often made worse by adrenergic stimulation.[19,502,511,512,515–520] A higher prevalence of myocardial substrates (mainly mid-wall or subepicardial non-ischaemic LV scars) was found in a CMR study among athletes with exercise-induced PVCs compared to those with exercise-suppressed VAs (56% vs. 21%).[516]

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Of note, exercise-induced isolated or repetitive PVCs with multiple morphologies, especially with beat-to-beat alternating morphologies (so-called 'bi-directional' pattern), may be the expression of catecholaminergic polymorphic VT, which can degenerate into VF.[518,521]

5.6.4.4 Practical management of cardiac patients with premature ventricular contractions or non-sustained ventricular tachycardia who want to engage in sports

The most important task in individuals with PVCs or NSVT who want to engage in sports is to exclude underlying structural or familial arrhythmogenic conditions, since sports activity may trigger sustained VT. It has been suggested that the presence of ≥2 PVCs on a baseline ECG (or even ≥1 PVC in the case of high-endurance athletes) should prompt a more thorough evaluation.[522]Work-up includes a family history, assessment of the number, morphology, and complexity of PVCs by Holter and 12-lead ECG, inducibility by exertion (via exercise test or long-term ECG recording during sports activities), and tailored additional imaging.[1] Further diagnostic evaluation with molecular genetic testing may be indicated in selected cases if the suspicion for familial disease is high. Finally, repeat evaluation may be needed after 6 months to 2 years. Recommendations for sports participation of athletes with PVC should be individualized based on evaluation for underlying cardiac conditions as described earlier, and often requires shared decision making.

Recommendations for exercise in individuals with premature ventricular contractions or non-sustained ventricular tachycardia

Recommendations	Class[a]	Level[b]
In exercising individuals with ≥2 PVCs on a baseline ECG (or ≥1 PVC in the case of high-endurance athletes) thorough evaluation (including a detailed family history) to exclude underlying structural or arrhythmogenic conditions is recommended.[503,522]	I	C
Among individuals with frequent PVCs and non-sustained VT a thorough investigation with Holter monitoring, 12-lead ECG, exercise test, and suitable imaging is recommmended.[503]	I	C
It is recommended that all competitive and leisure-time sports activities are permitted, with periodic re-evaluation in individuals without familial or structural underlying disease.[503]	I	C

ECG = electrocardiogram; PVC = premature ventricular contractions; VT = ventricular tachycardia.
[a]Class of recommendation.
[b]Level of evidence.

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5.6.5 Long QT syndrome

The QT and corrected QTc intervals vary by sex and physical training. Congenital long QT syndrome (LQTS) should be distinguished from acquired forms, i.e. due to circumstances, which can be reversed and prevented. Once acquired LQTS is established, sports activity should be prohibited until the underlying cause is corrected.

A definitive diagnosis of congenital LQTS is often difficult.[523] Congenital LQTS should be suspected on a routine ECG or 4 min into recovery after an exercise stress test,[524] if the corrected QTc interval according to Bazett's formula is ≥470 ms or ≥480 ms in asymptomatic male or female athletes, respectively.[525] A QTc of ≥500 ms is diagnostic.[526] In the case of a borderline long QTc interval and a negative personal and familial history, subclinical arrhythmias should be excluded by exercise testing and long-term ECG recording.

Since the risk of cardiac events during sports activities is largely gene-specific, genetic testing and cascade screening of family members should be performed following a clinical diagnosis of LQTS. Individuals with LQT1 are at highest risk during stressful exercise.[527,528]

Symptomatic athletes should not engage in competitive sports. Individuals with LQT1 should not engage in sports that involve diving into cold water since this is associated with increased risk of arrhythmias. General precautions include avoidance of QT-prolonging drugs, dehydration, and electrolyte imbalance. Beta-blocker therapy is extremely effective in LQT1 and additional therapies are only needed to control more severe cases or specific genotypes.[529]

Survivors of SCA (certainly while taking beta-blocker therapy) should be referred for an ICD. Similarly, individuals who have experienced sudden syncope despite beta-blocker therapy should also be referred for an ICD or sympathetic cardiac denervation.[530] ICD implantation does not constitute clearance for intensive or competitive sports. Continued sports participation with an ICD is possible, but specific recommendations apply (see *section 5.5.6*). American guidelines are more lenient with respect to participation in competitive sport (except for LQT1), provided that precautions include the presence of an automatic external defibrillator (AED) 'as part of the athlete's personal sports safety gear'.[531] We consider such obligation to be impractical (e.g. winter sports, water sports), and it places an added responsibility on clubs or other bystanders, which cannot be justified by a medical recommendation for an individual athlete. Moreover, although LQTS-related cardiac arrest is uncommon, even during competitive sports,[527] AED efficacy is not 100% in such cases.[532]

In asymptomatic LQTS mutation carriers without a prolonged QT interval, i.e. <470 ms in men and <480 ms in women ('genotype positive/phenotype negative'), shared decision making is required, balancing the risk for arrhythmias with psychological well-being. A negative exercise stress test has no predictive value.

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Recommendations for exercise in long QT syndrome

Recommendations	Class[a]	Level[b]
It is recommended that all exercising individuals with LQTS with prior symptoms or prolonged QTc be on therapy with beta-blockers at target dose.[529]	I	B
It is recommended that exercising individuals with LQTS should avoid QT prolonging drugs (www.crediblemeds.org) and electrolyte imbalance such as hypokalaemia and hypomagnesaemia.[529]	I	B
Shared decision making should be considered regarding sports participation in patients with genotype-positive/phenotype-negative LQTS (i.e. <470/480 ms in men/women). Type and setting of sports (individual vs. team), type of mutation, and extent of precautionary measures should be considered in this context.	IIa	C
Participation in high-intensity recreational and competitive sports, even when on beta-blockers, is not recommended in individuals with a QTc>500 ms or a genetically confirmed LQTS with a QTc≥470 ms in men or ≥480 ms in women.	III	B
Participation in competitive sports (with or without ICD) is not recommended in individuals with LQTS and prior cardiac arrest or arrhythmic syncope.	III	C

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ICD = implantable cardioverter defibrillator; LQTS = long QT syndrome.
[a]Class of recommendation.
[b]Level of evidence.

5.6.6 Brugada syndrome

The Brugada syndrome (BrS) is an inherited cardiac ion channel disorder with an elevated risk of VF and SCD in individuals with a structurally normal heart.[533,534] Although BrS was initially described as a purely electrical disease, minor structural RV abnormalities have been described,[535–538] suggesting that the disease may be the result of early repolarization or delayed depolarization.[509] The diagnosis is based on the presence of the type 1 Brugada pattern on the 12-lead ECG (coved-type ST-segment elevation ≥2 mm followed by a negative T-wave in ≥1 mm of the right precordial leads positioned in the fourth, third, or second intercostal space), either spontaneously or following provocation by a sodium ion channel blocker.[523,539,540]

Most individuals with BrS remain asymptomatic throughout their lives. In the majority of cases, events occur during sleep or rest, during febrile states or, occasionally, from heat stroke.[541–549] Patients who have suffered a SCA or an arrhythmic syncope should undergo ICD implantation.[247,511] Risk stratification in the asymptomatic population with spontaneous type 1 ECG pattern is more challenging.[247,511] There is controversial evidence that exercise testing showing an aggravating phenotype during exercise or early recovery, or EP study, are efficient at detecting individuals at risk of SCD. In asymptomatic patients with only inducible type 1 Brugada ECG pattern, preventive measures are recommended, such as avoidance of triggering drugs (www.brugadadrugs.org), electrolyte imbalance, and increases in core temperature >39°C (e.g. by minimizing immersion in hot tubs, saunas, and steam rooms; by avoiding sports in warm/humid conditions; or by abstaining from prolonged endurance events such as triathlons and marathons). During febrile illness, fever should be treated aggressively.[247,511]

One could speculate that an enhanced vagal reaction during recovery[550] and a predominant vagal tone at rest[551] may increase the susceptibility of highly trained individuals to develop arrhythmias during recovery or at rest. However, there are no reports directly linking exercise or sports training to cardiac events and there are no large prospective studies evaluating the effect of exercise and sports in BrS.

Asymptomatic patients with the spontaneous type I BrS ECG pattern may compete in all sports except endurance sports associated with an increase in core temperature >39°C (e.g. marathon running and triathlons). Similar rules apply to asymptomatic genotype-positive/phenotype-negative individuals and those with the concealed form of BrS.

If treated with an ICD, provided precautionary measures are taken and the patient has been asymptomatic for ≥3 months, resumption of all sport, including competitive sport, may be considered, after shared decision making, and also taking into consideration the findings from the ICD registry (see later).

Recommendations for exercise in Brugada syndrome

Recommendations	Class[a]	Level[b]
ICD implantation is recommended in patients with BrS with episodes of arrhythmic syncope and/or aborted SCD.[247]	I	C
Following implantation of an ICD, resumption of leisure or competitive sports should be considered after shared decision making in individuals who have not experienced recurrent arrhythmias over 3 months after ICD implantation.	IIa	C
In asymptomatic individuals with BrS, asymptomatic mutation carriers and asymptomatic athletes with only an inducible ECG pattern, participation in sports activities that are not associated with an increase in core temperature >39°C (e.g. endurance events under extremely hot and/or humid conditions) may be considered.	IIb	C
Prescription of drugs that may aggravate BrS[c], electrolyte abnormalities, and sports practice that increases core temperature >39°C are not recommended in individuals with overt BrS or phenotypically negative mutation carriers.	III	C

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BrS = Brugada syndrome; ECG = electrocardiogram; ICD = implantable cardioverter defibrillator; SCD = sudden cardiac death.
[a]Class of recommendation.
[b]Level of evidence.
[c]For example, www.brugadadrugs.org.

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5.6.7 Following device implantation

5.6.7.1 Pacemakers

Pacemaker (PM) implantation is common. Generally, individuals implanted with a PM have less severe disease and comorbidities than patients with an ICD. Moreover, there is less risk of malfunction for a PM than for an ICD during sports.[552] For all these reasons, recommendations for sports practice are more liberal in patients with a PM than in patients with an ICD. Patients with a PM may participate in competitive or recreational sports in the absence of structural or other heart disease for which exercise may be prohibited.

In the first weeks after device implantation, sports activities that increase the risk of lead dislocation (e.g. strong upper extremity movements) should be avoided. Exercise testing and/or Holter monitoring during sports may improve individualized programming of the upper sensor and the tracking rate and exclude inappropriate rate acceleration in other circumstances (e.g. horse riding).[553]

For all patients with cardiac devices (PM, cardiac resynchronization therapy, and ICD), sports activities associated with a risk of chest trauma should be avoided.[554–557] Some sports such as soccer, basketball, and baseball may be possible while wearing appropriate padding. It is noteworthy that sports with pronounced arm movements such as volleyball, basketball, tennis, golf, and climbing may increase the risk for late lead damage due to subclavian crush (with insulation or conductor failure).[554,558,559] Implantation on the contralateral side of the dominant arm, fixation within the pocket, or submuscular placement may improve durability of the system. It is not known whether subcostal or epicardial implant techniques provide long-term benefit. Electromagnetic interference is unlikely with modern devices, and no cases have been reported, but it should always be suspected and closely evaluated in specific athletic environments with electronic equipment (e.g. fencing). Myopotential inhibition may result in inhibition of pacing, a problem that is more common with unipolar electrodes, although it can usually be corrected with appropriate reprogramming of the device.[560,561] Bipolar leads are less sensitive to this problem, but may have a reduced longevity.

5.6.7.2 Implantable cardioverter defibrillators

A large multinational ICD Sports Safety Registry has shown that after a median follow-up of 44 months, there were no deaths or arrhythmia or shock-related physical injury in 440 athletes who continued organized competitive or high-risk sports after ICD implantation.[359,389] An additional analysis in 82 non-professional recreational athletes confirmed these reassuring outcomes,[562] which is relevant for the many ICD recipients who want to continue recreational sports activities after implantation. Therefore, shared decision making is appropriate when deciding whether or not to continue sports and the level of participation with an ICD.

However, three important considerations come into play. First, if sport is contraindicated because it can contribute to the progression of the underlying disease (such as in arrhythmogenic cardiomyopathy or lamin A/C mutations),[384,425] an ICD cannot be considered as a substitute for sports restriction, and participation in moderate- and high-intensity exercise should be discouraged. Recommendations in such circumstances should have a lifetime perspective and be based on the optimal preservation of structural cardiac integrity. The ICD, however, may allow for light to moderate exercise without concerns about risk of arrhythmia or shocks, and may help individuals to regain autonomy and overcome fears relating to exercising unattended.

The second consideration is that ICD shocks in general, even when appropriate and safe, will have a psychological impact on the athlete; 30–40% of the athletes who experienced shocks in the multinational ICD Sports Safety Registry stopped participation, at least temporarily, out of fear of repeat shocks.[389] Moreover, the proportion of athletes with appropriate and inappropriate shocks during exercise was higher in competitive than in recreational sports, emphasizing the known triggering effect of high-intensity exercise.[562] Considering that ICD therapy is lifelong, where quality of life is not only dependent on the ability to perform sports activities, but also and mostly on continued trust in the device, physicians should be aware that their own belief in the effectiveness and safety of ICD therapy during sports should not implicitly put pressure on the athlete to continue sports. Again, informed decision making needs to re-evaluate all options of the athlete aftershocks, including continuing, reducing, or stopping sports.[563,564]

Third, situations where loss of focus or loss of consciousness could cause harm to a third party or the athlete (such as in motor sports, diving, mountain climbing, even cycling) should be avoided.

The athlete must be aware of the programmed detection rate cut-offs to be able to avoid reaching those during exercise. Conversely, detection zones need to be programmed sufficiently high to allow for adequately high heart rates during the desired exercise levels. This practice proved safe and reduced the occurrence of shocks in the ICD Sports Safety Database.[565] The most common cause of inappropriate shocks in individuals with an ICD is the occurrence of sinus tachycardia and supraventricular arrhythmias.[566,567] Underlying heart disease and endurance sport itself carries a higher risk for developing AF.[474,480,568] Implantation of a dual system ICD for the sole reason of atrial arrhythmia detection and discrimination is not warranted because it is usually not effective.[565,569–572] Given the fact that many of these athletes are young, there is a higher risk for long-term lead complications when more leads are implanted; therefore, implantation of more complex ICD systems, and their indication, should be weighed in every patient. Routine inclusion of athletes in remote monitoring programmes is highly recommended.

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Recommendations for exercise in individuals with pacemakers and implantable cardioverter defibrillators

Recommendations	Class[a]	Level[b]
It is recommended that individuals with implanted devices with/without resynchronization and underlying disease follow the recommendations pertaining to the underlying disease.[384,425]	I	B
Participation in sports and exercise (except collision sports) should be considered in individuals with pacemaker therapy who do not have pathological substrates for fatal arrhythmias.	IIa	C
Prevention of direct impact to the implanted device by adapting the site of lead and/or device implantation, padding, or restricting direct impact sports should be considered.	IIa	C
Holter recordings and device interrogation during and after resuming sports should be considered to allow appropriate tailoring of rate-responsive pacing parameters, exclusion of myopotential or electromagnetic inhibition, and detection of VAs.	IIa	C
Shared decision making should be considered during decisions relating to continuation of intensive or competitive sports participation in individuals with an ICD, taking into account the effect of sports on the underlying substrate, the fact that intensive sports will trigger more appropriate and inappropriate shocks, the psychological impact of shocks on the athlete/patient, and the potential risk for third parties.	IIa	C
An ICD is not recommended as a substitute for disease-related recommendations when these mandate sports restrictions.	III	C

ICD = implantable cardioverter defibrillator; VA = ventricular arrhythmia.
[a]Class of recommendation.
[b]Level of evidence.

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5.7 Exercise recommendations in individuals with adult congenital heart disease

5.7.1 Introduction

Congenital heart disease (CHD) has a prevalence of 8–9 per 1000 live births and is the most common birth defect.[573] Most children with CHD survive to adulthood, including over 85% with complex disease. Regular exercise is important for adults with congenital heart disease (ACHD) and exercise participation should be discussed at every patient encounter.[574] However, CHD represents a spectrum of conditions with widely varying physiological consequences. Individualized assessment is essential before advising on sports participation. This requires detailed understanding of the congenital heart defect, its physiological consequences, and the effect of surgical or transcatheter intervention.

5.7.2 The increasing numbers of athletes with congenital heart disease

Athletes with CHD include those with minor unoperated lesions and palliated and repaired CHD. Some athletes will be diagnosed with CHD for the first time during pre-participation screening. Approximately 1 in 150 adults have a congenital heart defect. This is an increase of over 50% in the last 10 years and reflects improved diagnosis and long-term survival.[575] Overall 90% of adults with CHD are in NYHA functional class I or II.[576] The number of professional and recreational athletes with CHD is unknown but the elite athlete population is likely to be small. In a study of 2352 Olympian athletes, only nine (0.4%) had a congenital heart defect.[577] However, many more CHD patients compete at a recreational level. It is likely that the number of athletes with CHD is increasing due to improved survival, better understanding of the benefits of exercise, and the increased number of professional athletes from countries where poorly developed healthcare systems are associated with under-diagnosis of CHD.[573,574]

5.7.3 Non-cardiac abnormalities in congenital heart disease and Paralympic sport

CHD has a multifactorial aetiology, but there is a significant genetic component and a recurrence risk of 2–5%.[578] The genetic origin may be due to a chromosome anomaly, a heritable syndrome with Mendelian pattern, or sporadic with variable penetrance.[578] Approximately 14% of patients with CHD have additional non-cardiac abnormalities.[579] Thus, 44% of ACHD patients have lung function abnormalities.[580] This can make advising sports participation for the ACHD athlete more difficult. There is no specific classification for CHD in Paralympic sport.

5.7.4 General considerations in the congenital heart disease athlete

There is considerable variation in the haemodynamic consequences and prognosis of different CHD lesions. Furthermore, the consequences of any individual lesion can vary hugely between individuals. Thus, Ebstein's anomaly may present in infancy with pulmonary hypoplasia due to compression by a giant right atrium or may present in late middle age with supraventricular tachycardia and minimal haemodynamic consequences. Similarly, a ventricular septal defect may be small and asymptomatic, or large presenting with HF in infancy, or may present with extreme cyanosis, reduced exercise capacity, and Eisenmenger syndrome in the adult. Consequently, these recommendations are based on a physiological assessment rather than underlying anatomic diagnosis. An understanding of the range of severity and a lesion-based knowledge of potential complications is essential. The cardiologist should work in conjunction with a CHD specialist. Athletes with CHD who participate in regular training and competitive sports should undergo a comprehensive annual assessment that includes clinical examination, ECG, echocardiogram, and exercise stress test, ideally CPET.

Many athletes with CHD will have undergone corrective or palliative surgery. In young adults with post-surgical CHD, valve

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incompetence and arrhythmias are common problems, but with increasing age arrhythmias and HF predominate. In master athletes, problems related to previous corrective or palliative surgery become prevalent. These include cardiac arrhythmias, systemic ventricular dysfunction, valvar incompetence, and prosthetic conduit obstruction. Redo valve or conduit surgery and arrhythmia ablation due to re-entry arrhythmias (secondary to surgical scar) are frequent in this age group. In addition, with increasing age, acquired CVD may develop.[581,582] This is of particular relevance in the assessment of the master athlete with CHD.

5.7.5 Sudden death during sport

SCD in CHD is rare (<0.1% per year)[583] and only 8% of deaths occur during exercise.[584] Many complex CHD patients at highest risk of SCD have reduced exercise tolerance and cannot participate in significant athletic activity. However, some diagnostic groups, e.g. post tetralogy of Fallot repair, carry a risk of SCD but can still compete in elite sport.[585] Other high-risk congenital lesions such as anomalous coronary origin, mitral valve prolapse, and aortopathy are described in *sections 5.1, 5.3, and 5.4*, respectively. In a large population-based study of SCD in CHD patients under 35 years, 87% of SCD was due to a presumed arrhythmia and 41% occurred in patients with undiagnosed CHD.[586]Activity-related SCD was more common in the undiagnosed group (18% vs. 4%), which supports the case for pre-participation athletic screening.[586] It is not known whether exercise intensity is a risk factor for SCD in CHD. There is reasonable evidence that moderate to vigorous exercise is safe in most ACHD patients even when symptomatic (NYHA II–III).[576,587,588] However, cardiac arrhythmias are a common cause of hospital admission in ACHD and extreme exercise may expose a latent arrhythmic substrate.[589,590]

5.7.6 Exercise in athletes with congenital heart disease: current guidelines and recommendations

Regular structured exercise is safe and effective therapy for most patients with CHD. This is true for most diagnostic groups, including symptomatic patients, and includes aerobic and strength-based exercise.[588,591–596] Exercise intolerance in CHD is a strong predictor of both outcome and SCD.[576] Specific precautions are necessary in extreme environments including underwater sports and this is considered below (see *Supplementary Data, section 4*). This is particularly true if there is a potential for a right to left shunt. Guidelines are available for exercise assessment and prescription in both children and adults with CHD, although these are not specifically designed for the athlete.[597,598] The paediatric guidelines are based primarily on the underlying anatomical diagnosis, but the teenage and young adult CHD guidelines have taken a functional approach based on underlying haemodynamics and arrhythmia risk. This latter approach is better suited for assessment of the ACHD athlete (*Figure 9*).

5.7.7 Assessment of the athlete with congenital heart disease

The guidelines for exercise prescription in adolescents and adults with CHD use a structured methodology described by Budts *et al.*[597] This can be modified for use in the assessment of athletes with CHD.

Stage 1. A full history and physical examination are carried out. This should include details of underlying CHD diagnosis, any transcatheter or surgical interventions, current medications and CV symptoms (at rest and on exercise). Attention should be paid to any associated non-cardiac diagnoses including pulmonary dysfunction. A full exercise and sports participation history should be taken including precise details of current training schedule and any dietary supplements. Details of the planned or current sports activity should be established to include an assessment of the static component and intensity as described in *section 4.1* (see *Figure 2*). If necessary, advice should be sought from a sports fitness coach or sports medicine specialist.

Stage 2. The following five baseline parameters should then be evaluated[597] (*Table 16*).

(1) Ventricular function

Assessment of ventricular function can usually be achieved using echocardiography. The aim is to establish whether function is reduced (EF < 55%), and if so, is it mild (45–55%), moderate (30–45%), or severe (<30%). This is used for baseline assessment and subsequent monitoring of the effects of exercise training. Echocardiography can also evaluate inflow and outflow abnormalities, which may become more severe during acute exercise (e.g. LV outflow obstruction or systemic atrioventricular valve regurgitation). CMR scanning may be a preferable modality in complex disease. This has the additional benefit of evaluating intracardiac scar, which may inform the assessment of arrhythmia risk.[599,600]

(2) Pulmonary artery pressure

Pulmonary hypertension (PH) is diagnosed when the mean PAP is >20 mmHg.[601] PH may occur in the context of a chronic left–right shunt (e.g. atrial septal defect, ventricular septal defect, patent ductus arteriosus) that allows unrestricted volume/pressure overload. Eventually this can result in supra-normal PAP with reversal of shunting and elevated pulmonary vascular resistance (Eisenmenger syndrome). An increased RV afterload limits the ability to increase cardiac output by increasing stroke volume and can impair LV function through disruption of normal RV–LV interaction.

Few CHD patients with significant PH will take part in competitive sports due to reduced exercise capacity. However, some athletes with CHD will have mild elevation of pulmonary vascular resistance that may be exacerbated by factors such as altitude training.[602] PAP rises during exercise. This rise is accentuated with increasing age and may be exaggerated in CHD athletes due to elevated pulmonary vascular resistance.[603] In addition, valvar pulmonary stenosis or distal stenoses in branch pulmonary arteries may cause exercise-related RV hypertension. The non-invasive assessment of PH can be difficult and full assessment guidelines have been published.[604,605] Further information can be gained by CPET and a VO_2 of >25.2 mL/min/kg makes significant PH unlikely.[606] As PH can also be a late postoperative complication after surgical correction of CHD[607] the assessment of PAP should be part of every echocardiographic examination in athletes with CHD. Although exercise training is generally regarded as safe in PH, competitive sport is not recommended.[1,608]

(3) Aortic assessment

Many CHD patients are at risk of aortic dilatation, in particular, patients with tetralogy of Fallot, coarctation of the aorta and certain

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Table 16 Baseline parameters for assessment in congenital heart disease

Parameter	Comments
Ventricular function	Usually by echocardiogram. In complex conditions CMR may be preferable[597,599,600]
Pulmonary pressure	Use tricuspid regurgitation velocity, pulmonary regurgitation velocity on echocardiography. May require cardiac catheterization for accurate measurement[599,604]
Aortic size	Usually by echocardiography or CMR. Coarctation should be excluded[609, 618]
Assessment of arrhythmia	12 lead ECG with low threshold for 24-hour ambulatory ECG. Additional tests may be required if symptomatic[611,612]
Assessment of saturations	Pulse oximetry at rest/on exercise[602,614]

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CMR = cardiac magnetic resonance; ECG = electrocardiogram.

syndromes such as 22q11 microdeletion and Turner syndrome. However, aortic dissection is very rare in CHD.[609] Athletes have mildly increased aortic dimensions in comparison to sedentary controls, but it is not known if this has a cumulative effect in CHD athletes with aortic dilatation.[345] The presence of ascending aorta dilatation should lead to assessment for coarctation of the aorta as this can be associated with severe coarctation, which may be missed on clinical assessment but may cause severe exercise-related hypertension.[610] Exercise risks in aortopathy are described in detail in *section 5.4*. Contact sports should be avoided in patients with dilated aortas >5 cm.

(4) Arrhythmia assessment

Arrhythmias are responsible for 25% of CHD hospital admissions. Over 80% are atrial but life-threatening VAs can occur.[611] Independent risk factors include increasing age, male gender, double outlet right ventricle, atrioventricular septal defect, HF, obstructive sleep apnoea, transposition of the great arteries, congenitally corrected transposition, and tetralogy of Fallot.[599] Assessment of the CHD athlete should include a symptom history with evaluation of palpitations, presyncope and syncope, particularly during exercise.

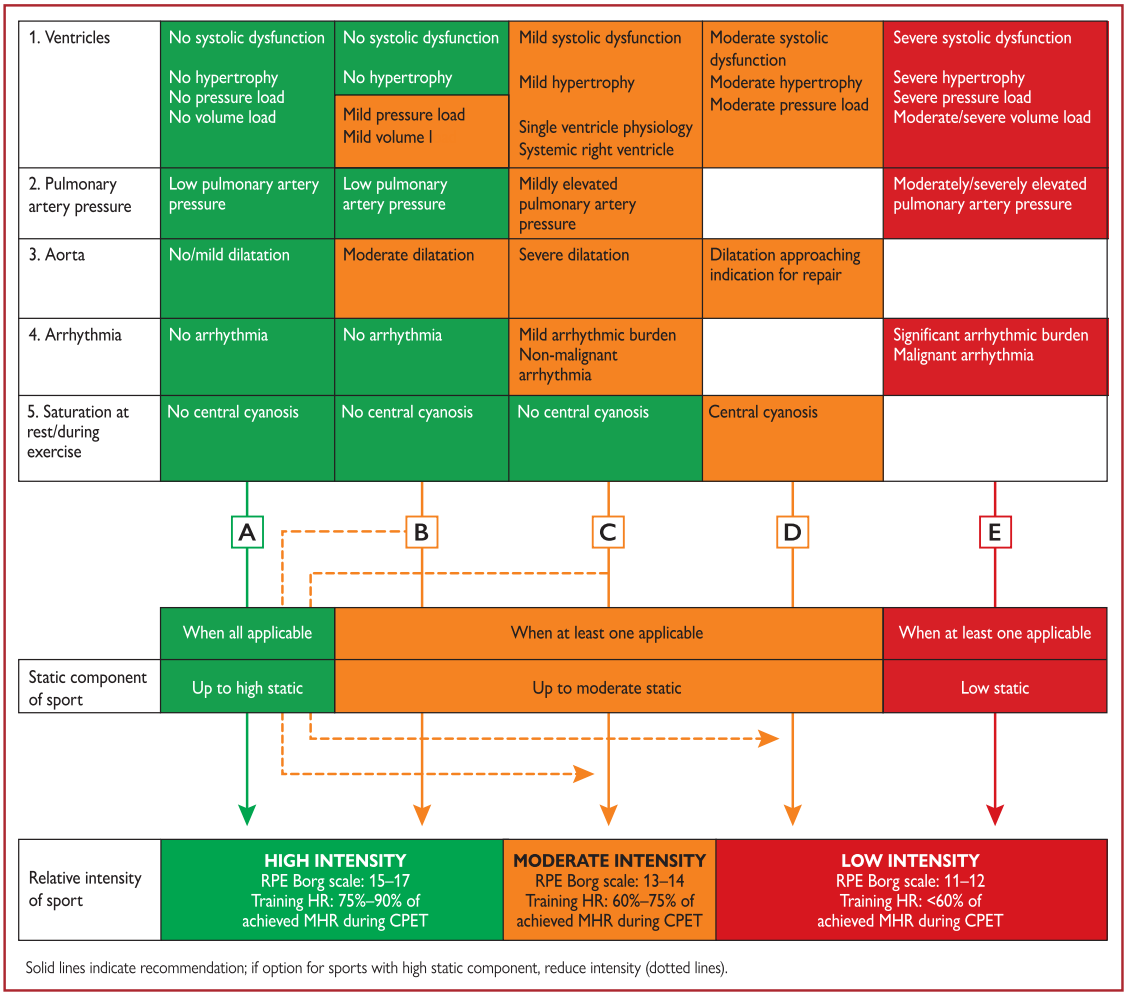

Figure 9 Pre-participation assessment of individuals with congenital heart disease.[598] CPET = cardiopulmonary exercise test; HR = heart rate; MHR = maximum heart rate; RPE = rate of perceived exertion. A–E represent pathways linking static and intensity components for each column. After assessment of CPET and the five variables (*Table 16*), an individual recommendation can be given (solid arrow). If a higher static level sport is chosen, then a lower intensity level is advised (dotted arrow).

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Arrhythmias may be the first sign of underlying haemodynamic deterioration and new-onset arrhythmias should lead to a full haemodynamic assessment. Detailed guidelines for the assessment and treatment of arrhythmias in CHD are available.[612] In the athlete, arrhythmia therapy may be complicated by the need to minimize the negative chronotropic effects of antiarrhythmic treatment, which may affect performance. If symptomatic arrhythmias are present then an exercise ECG, prolonged ECG monitoring, loop recorder implantation, and even electrophysiology testing might be required. This should be coordinated by an electrophysiologist with expertise in CHD.

(5) Assessment of saturations/lung function

CHD athletes should be assessed for the potential of an underlying intracardiac right to left shunt. This can be assessed using pulse oximetry, but a resting saturation >95% does not exclude exercise-related central cyanosis, and exercise assessment is essential. The potential for a pulmonary cause of cyanosis must be considered and lung function should be assessed as part of a cardiopulmonary exercise test. Even after surgical correction of the cardiac defect there may be residual intracardiac shunting.

Stage 3. Cardiopulmonary exercise testing

CPET is invaluable in risk stratifying the adult with CHD and can predict outcome.[613,614] It is also extremely useful in the evaluation of the athlete with CHD as it allows an assessment of PAP, respiratory problems, cardiac output, exercise-related haemodynamics, and arrhythmias. This should be used in conjunction with an evaluation of effort such as the Borg scale.[615] Normal CPET values are available for non-athlete CHD patients.[616] CPET should be carried out in conjunction with a 12-lead ECG to allow detection of arrhythmias and evaluate chronotropic incompetence. Reduced VO_{2max} and peak oxygen pulse may reflect reduced stroke volume and are found in complex CHD as well as in other forms of CHD, including repaired tetralogy of Fallot, aortic regurgitation, and coarctation of the aorta. In CHD, ventilatory anaerobic threshold may be reduced and this impairs gas exchange efficiency in dynamic and endurance sports. This may relate to previous thoracotomy or lung disease. Similarly, there may be an elevated minute ventilation/carbon dioxide production (VE/VCO_2) slope,[601] indicating an inadequate pulmonary vascular bed.[610,611,613–616] Reduced skeletal muscle mass is common in adults with CHD and can contribute to decreased tissue oxygen uptake. The extent to which this is modified in athletes with CHD has not been evaluated. However, even in complex disease, regular resistance exercise participation improves muscle mass and regular exercise participation is a dominant factor in determining long-term outcome.[593,617]

Stage 4. Exercise intensity and prescription

An assessment of the athlete's sports and exercise participation should take place as discussed in the introduction (*Table 16*). This should include an assessment of the intensity, aerobic and resistance components (isometric and dynamic), and the overall volume of exercise, which should include training and competition. The haemodynamic consequences should be considered in light of an understanding of known lesion-specific consequences and individual athlete-specific changes identified on assessment.

Stage 5. Follow-up and repeat assessment

In the CHD athlete, serial assessment should be carried out—usually on an annual basis—while participating in sport. This recognizes the age-related changes that occur in the CHD athlete and the potential for onset of degenerative CVD.

Exercise recommendations for individuals with congenital heart disease

Recommendations	Class[a]	Level[b]
Participation in regular moderate exercise is recommended in all individuals with CHD.[588,591–594,619]	I	B
A discussion on exercise participation and provision of an individualized exercise prescription is recommended at every CHD patient encounter.[574,597,598,617]	I	B
Assessment for ventricular function, pulmonary artery pressure, aortic size, and arrhythmia risk is recommended in all athletes with CHD.[342,348,573,597]	I	C
Competitive sports participation should be considered for CHD athletes in NYHA class I or II who are free from potentially serious arrhythmias after individual tailored evaluation and shared decision making.[573,595,597,598]	IIa	C
Competitive sports are not recommended for individuals with CHD who are in NYHA class III–IV or with potentially serious arrhythmias.[605,608]	III	C

CHD = congenital heart disease; NYHA = New York Heart Association.
[a]Class of recommendation.
[b]Level of evidence.

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6. Key messages

CV screening before participation in recreational and competitive sports is aimed at the detection of disorders associated with SCD and has the potential to lower CV risk through disease-specific and individualized patient management.

CV screening in adult and senior athletes should target the higher prevalence of atherosclerotic CAD including an assessment of CVD risk factors and exercise stress test. CAC scoring may be performed in asymptomatic athletes with a moderate atherosclerotic risk profile.

Healthy adults of all ages and individuals with known cardiac disease should exercise on most days, totalling at least 150 min/week of moderate-intensity exercise.

Individuals with CAD, at low risk for exercise-induced adverse events, should be considered eligible for competitive or leisure sports activities, with few exceptions.

Competitive sports are not recommended in individuals with CAD, at high risk of exercise-induced adverse events or those with residual ischaemia, with the exception for individually low-intensity skill sports.

Exercise programmes in HF improve exercise tolerance and quality of life and have a modest effect on all-cause and HF-specific mortality, and all-cause hospitalization and HF-specific hospitalization.

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Asymptomatic individuals with mild valvular heart disease may participate in all sporting activities including competitive sports.

A select group of asymptomatic individuals with moderate valve disease who have good functional capacity and no evidence of myocardial ischaemia, complex arrhythmias, or haemodynamic compromise on a maximal exercise stress test may be considered for competitive sports after careful discussion with an expert cardiologist.

Implementation of healthy lifestyle behaviours including sports participation decreases the risk of CV events and mortality in individuals with aortopathies.

Individuals with acute myocarditis or pericarditis should abstain from all sports while active inflammation is present.

Individuals with cardiomyopathy or resolved myocarditis or pericarditis, who wish to participate in regular sports, should undergo comprehensive evaluation, including an exercise test, to assess the risk of exercise-induced arrhythmias.

Individuals who are genotype positive/phenotype negative or have a mild cardiomyopathy phenotype and absence of symptoms or any risk factors, may be able to participate in competitive sports. A notable exception is arrhythmogenic cardiomyopathy where high-intensity exercise and competitive sports should be discouraged.

Managing sports participation in individuals with arrhythmogenic conditions is guided by three principles: (i) preventing life-threatening arrhythmias during exercise; (ii) symptom management to allow sports; and (iii) preventing sports-induced progression of the arrhythmogenic condition. In each case, these three basic questions need to be addressed.

Pre-excitation should be excluded in all athletes with PSVT, and ablation of the accessory pathway is recommended if present.

In individuals with PVCs who want to engage in sports, underlying structural or familial arrhythmogenic conditions must be excluded, since sports activity may trigger more malignant arrhythmias if those underlying conditions are present.

Athletes with electrical abnormalities of genetic origin, such as the inherited ion channelopathies, require assessment and shared decision making in which cardiogeneticists are involved, given the complex interplay of genotype, phenotype, potential modifiers, and exercise.

Individuals with pacemakers should not be discouraged from participation in sport because of the device but need to tailor their sports participation according to the underlying disease.

Participation in leisure-time and competitive sport in patients with an ICD may be considered, but requires shared and individualized decision making, based on a higher likelihood of appropriate and inappropriate shocks during sports, and the potential consequences of short episodes of loss of consciousness.

Patients with CHD should be encouraged to exercise and should be given a personalized exercise prescription.

7. Gaps in evidence

Outcomes in exercising individuals with cardiovascular diseases. The natural history and absolute risk of conditions associated with SCD in athletes identified through CV screening is largely unknown, making it difficult to quantify short- and long-term risk. Prospective outcomes data, including the occurrence of major CV events and other CV morbidity, is needed to better guide risk stratification, management, and eligibility recommendations for athletes diagnosed with CVD.

Cardiovascular evaluation in master athletes. Current methods for screening individuals for atherosclerotic CAD are based on symptoms and a maximal exercise test; however, they do not identify individuals with mild to moderate atherosclerotic plaques. More knowledge on the optimal pre-participation screening algorithm for identifying individuals at risk of adverse cardiac events during exercise is required.

Safety of high-intensity exercise in cardiovascular disease. There are limited data on the safety of high-intensity exercise training and sports participation in healthy individuals with a high burden of risk factors; more information can be gained through high-quality prospective studies in the future.

Risk of anomalous origins of the coronary arteries in older individuals. Although AOCA from the opposite sinus of Valsalva are a recognized cause of mortality in young athletes, more knowledge is required on the risk of high-intensity exercise in older individuals (>40 years old), with this congenital abnormality.

Risk of myocardial bridge. The precise significance of MB in causing MI or sudden death during exercise is unknown.

Benefit of regular exercise on survival in chronic heart failure. Although exercise improves functional capacity and quality of life in individuals with HF, strong evidence on the benefit of regular exercise on survival is still lacking.

Role of high-intensity exercise in chronic heart failure. The impact of high-intensity exercise in asymptomatic individuals with HF with reduced and preserved systolic function is unknown and large multicentre studies are required to facilitate exercise prescription in the future.

Initiation of an exercise programme after decompensated heart failure. The start time of an exercise programme of moderate-high intensity and return to sports after an acute event in HF is unknown.

Aquatic exercise in heart failure. The safety of aquatic exercise in HF needs to be confirmed by larger trials.

Effect of exercise on the natural history of valvular heart disease. There is a paucity data on the effects of regular intensive exercise in individuals with valvular heart disease. Large-scale longitudinal studies are required to provide more evidence-based guidelines on exercise prescription in individuals with valvular heart disease.

Effect of exercise on the natural history of aortopathies. There is a lack of knowledge on the impact of sport on progression of aortic disease and risk of dissection or aortic rupture among individuals with aortopathy. The impact of beta-blockers on progression of aortic disease among individuals with aortopathy who participate in regular exercise is unknown and should be investigated in a randomized controlled trial.

Optimal safe exercise dose in cardiomyopathy. With an exception for arrhythmogenic cardiomyopathy, current practice relating to exercise recommendations in individuals with cardiomyopathy or following myopericarditis are largely based on circumstantial evidence. Large, adequately powered randomized prospective studies are necessary to provide evidence-based recommendations for optimal exercise prescription without compromising safety. Such

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studies should also prove useful for validating current risk stratification protocols derived from a relatively sedentary population.

Exercise and atrial fibrillation. The threshold lifetime sports activity for increasing the risk of developing AF is unknown. It is also unknown whether ongoing participation in vigorous exercise at the same intensity after successful AF ablation is associated with a higher risk of AF recurrence.

Benefit of invasive electrophysiological studies in adolescents with accessory pathways. The benefit/risk ratio of early invasive EP evaluation of AP conduction properties in young athletes with asymptomatic pre-excitation (<12–14 years old), and prophylactic ablation, remains unclear and requires large-scale studies for clarification.

Exercising with an implantable cardioverter defibrillator. Although an international registry has shown absence of sports-related sudden death or injury in individuals with an ICD who perform competitive, high-intensive recreational or high-risk sports, many data are sparse and have not examined specific athletic populations with reduced LV function, catecholaminergic polymorphic VT. Moreover, the long-term physical and psychological impact of appropriate and inappropriate shocks, which become more frequent with increasing sports intensity, is unknown.

Arrhythmic risk of exercise in adult congenital heart disease. The relationship between intensity of exercise and risk of arrhythmias in individuals with CHD is unknown and further research is needed. Although exercise prescription is recommended in CHD patients, further studies are necessary to identify the optimal method of improving adherence to an exercise prescription.

8. Sex differences

For many years sports participation, especially at the highest echelons, has been dominated by males, but the last four decades have witnessed an increasing number of females participating in a large variety of sporting disciplines at elite level. Females constituted 45% of all athletes at the 2016 Rio Olympics and participated in 26 of the 28 different sporting disciplines including those that were traditionally considered to be 'male sports', such as footfall, rugby, and boxing. Although the guidelines in this document are applicable to both sexes, there are some pertinent sex-based differences relative to exercise and sport including: (i) the incidence of SCD during exercise; (ii) quantitative differences in CV adaptation to regular intensive exercise and the overlap with cardiomyopathy; (iii) the predilection of adverse events with specific CVDs in women; and (iv) the additive haemodynamic effects of exercise in pregnant women with a structurally abnormal heart.

Current evidence suggests a significantly lower prevalence of exercise-related SCD in females who represent a small minority of athletes dying suddenly with a male to female ratio ranging from 3 to 10: 1.[17,18,46,49,52,620] This disproportionately lower incidence of SCD among young competitive female athletes also holds true for older recreational athletes where deaths in males are 20-fold greater than in females.

Apart from a lower incidence of exercise-related SCD, there appear to be sex differences related to the diseases predisposing to SCD. Unlike males, female athletes rarely succumb to SCD from HCM. In the US National Registry, females comprised only 3% of the 302 individuals who died from HCM.[621] Potential determinants of this disproportionate prevalence of death in males may include a lower absolute volume and intensity of training load in females, which could make them less susceptible to ventricular tachyarrhythmias. However, it is also plausible that certain protective metabolic or hormonal mechanisms could reduce the arrhythmic risk during intense physical exertion in females with HCM. This observation is relevant to prescription of intensive exercise or competitive sports in HCM, which is relatively conservative, but could be of less concern and more liberal in affected females in the future. The diagnosis of HCM in an athlete is also relevant in this regard, because male athletes, particularly those competing in endurance sports, show quantitatively great structural changes within the heart including LV hypertrophy. Approximately 2% of white male athletes and 13% of black male athletes show a LV wall thickness of 12–15 mm that overlaps with morphologically mild HCM and can pose a diagnostic dilemma. In contrast female athletes rarely reveal a LV wall thickness >12 mm or concentric LV remodelling, hence the diagnosis of HCM is clear-cut.[3]

In contrast, a different scenario is represented in females with MVP, where recommendation for participation in intensive exercise warrants a more detailed risk stratification. In the Italian pathology registry of 650 SCDs, 7% were attributed to MVP. Of these, the majority (60%) were female who presented a marked elongation of both leaflets due to an extensive myxomatous degeneration.[332]

It is also noteworthy that the majority of exercise-related SCDs in young females are associated with a structurally normal heart at autopsy. This observation from several registries suggests that genetic electrical diseases (namely, LQTS, Brugada syndrome, or catecholaminergic polymorphic VT) are likely responsible for a significant proportion of these deaths. Differences in cardiac repolarization between men and women have been reported in healthy subjects and in individuals with LQTS.[622] Healthy females also have a longer QTc interval compared with males. Therefore, they are more often clinically affected by the syndrome than men, despite the equal sex distribution of the disease genotype. Females are also at a higher risk than males of developing arrhythmias in response to QT-prolonging drugs, and electrolyte disturbances compared with males. Furthermore, female sex is an independent risk factor for cardiac events in LQTS.[623] Unfortunately, little is known about the influence of sex hormones on cardiac repolarization, except that androgens may shorten the QTc interval. Based on these considerations, we recommend particular caution when advising competitive sports in women with a probable or definite diagnosis of LQTS.

Pregnancy is associated with a 50% increase in plasma volume and cardiac output. The stresses of exercise in pregnant women with structural heart disease have the potential to cause haemodynamic compromise. Moderate aerobic exercise is generally safe in all women and is associated with a lower prevalence of excessive weight gain, post-partum obesity, gestational diabetes, and pre-eclampsia. Female athletes may continue training intensively during pregnancy, although it is recommended that women do not exceed a heart rate of >90% of the maximum predicted for age to reduce the risk of foetal bradycardia. Pregnant women with known structural heart disease require evaluation before embarking on intensive exercise programmes. Exercise or sports involving forceful physical contact, risk of falling or abdominal trauma, heavy lifting, scuba diving, and

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exercising at high altitude in an unacclimatized state are not recommended.

Finally, females represent the largest population of obese individuals worldwide and females with T2DM have a higher prevalence of cardiovascular complications and death compared with men. In this regard all women should be incentivized to engage in regular PA and exercise programmes irrespective of age, ethnicity, and CV morbidities.

9. 'What to do' and 'what not to do' messages from the Guidelines

Recommendations: What to do and what not to do for exercise and sports in healthy individuals	Class[a]	Level[b]
General recommendations for exercise and sports in healthy individuals		
At least 150 min/week of moderate-intensity, or 75 min/week of vigorous-intensity aerobic exercise or an equivalent combination thereof is recommended in all healthy adults.	I	A
Regular assessment and counselling to promote adherence and, if necessary, to support an increase in exercise volume over time are recommended.	I	B
Multiple sessions of exercise spread throughout the week, i.e. on 4–5 days a week and preferably every day of the week, are recommended.	I	B
Special considerations for individuals with obesity, hypertension, dyslipidaemia, or diabetes		
In obese individuals (BMI≥30 kg/m^2 or a waist circumference >80 cm for females or >94 cm for males) resistance training ≥3 times per week, in addition to moderate or vigorous aerobic exercise (at least 30 min, 5–7 days per week) is recommended to reduce CVD risk.	I	A
In individuals with well-controlled hypertension, resistance training ≥3 times per week in addition to moderate or vigorous aerobic exercise (at least 30 min, 5–7 days per week) is recommended to reduce blood pressure and CVD risk.	I	A
Among individuals with diabetes mellitus, resistance training ≥3 times per week) in addition to moderate or vigorous aerobic exercise (at least 30 min, 5–7 days per week) is recommended to improve insulin sensitivity and achieve a better CVD risk profile.	I	A
In individuals with uncontrolled hypertension (SBP>160 mmHg) high-intensity exercise is not recommended until BP has been controlled.	III	C
Recommendations for exercise in ageing individuals		
Among adults aged 65 years or older who are fit and have no health conditions that limit their mobility, moderate-intensity aerobic exercise for at least 150 min/week is recommended.	I	A
In older adults at risk of falls, strength training exercises to improve balance and coordination on at least 2 days a week are recommended.	I	B
Recommendations for exercise in individuals with coronary artery disease		
Recommendations for exercise in individuals with long-standing chronic coronary syndrome		
Risk stratification for exercise-induced adverse events is recommended in individuals with established (long-standing) CCS prior to engaging in exercise.	I	C
Competitive sports are not recommended in individuals at high risk of exercise-induced adverse events or those with residual ischaemia, with the exception of individually recommended skill sports.	III	C
Recommendations for return to exercise after acute coronary syndrome		
Exercise-based cardiac rehabilitation is recommended in all individuals with CAD to reduce cardiac mortality and rehospitalization.	I	A
Recommendations for exercise in young individuals/athletes with anomalous origins of coronary arteries		
Participation in most competitive sports with a moderate and high CV demand among individuals with AOCA with an acutely angled take-off or an anomalous course between the large vessels is not recommended.	III	C
Recommendations for exercise/sports in individuals with myocardial bridging		
Competitive sports are not recommended in individuals with myocardial bridging and persistent ischaemia or complex cardiac arrhythmias during maximal exercise stress testing.	III	C
Recommendations for exercise in chronic heart failure		
Recommendations for exercise prescription in heart failure with reduced or mid-range ejection fraction		
Regular discussion about exercise participation and provision of an individualized exercise prescription is recommended in all individuals with HF.	I	A
Exercise-based cardiac rehabilitation is recommended in all stable individuals to improve exercise capacity and quality of life and to reduce the frequency of hospital readmission.	I	A

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Recommendations for participation in sports in heart failure		
Before considering a sport activity, a preliminary optimization of HF risk factor control and therapy, including device implantation (if appropriate), is recommended.	I	C
High-intensity power and endurance sports are not recommended in patients with HFrEF irrespective of symptoms.	III	C
Recommendations for exercise and participation in sports in individuals with heart failure and preserved ejection fraction		
Moderate endurance and dynamic resistance exercise, together with lifestyle intervention and optimal treatment of CV risk factors (i.e. arterial hypertension and type 2 diabetes) are recommended.	I	C
Recommendations for exercise and participation in sports in individuals with heart transplant recipients		
Regular exercise through cardiac rehabilitation combining moderate-intensity aerobic and resistance exercise is recommended to revert pathophysiology to pre-transplantation time, reduce CV risk induced by post-transplantation medical treatment, and improve clinical outcome.	I	B
Recommendations for exercise in asymptomatic individuals with valvular heart disease		
Participation in competitive or recreational sports/exercise of moderate and high intensity is not recommended in individuals with severe aortic stenosis.	III	C
Participation in competitive or recreational sports/exercise of moderate and high intensity is not recommended in individuals with severe aortic regurgitation with LVEF≤50% and/or exercise-induced arrhythmias.	III	C
Participation in competitive sports is not recommended in individuals with severe mitral regurgitation with a LVEF<60%.	III	C
Participation in any competitive sport, or leisure sports/exercise of mild to moderate intensity, is not recommended in individuals with severe (MVA<1 cm^2) mitral stenosis.	III	C
Recommendations for exercise in individuals with aortic pathology		
Prior to engaging in exercise, risk stratification, with careful assessment including advanced imaging of the aorta (CT/CMR) and exercise testing with BP assessment, is recommended.	I	A
Competitive sports are not recommended in individuals who are at high risk (*Table 14*).	III	C
Recommendations for exercise in individuals with cardiomyopathy		
General recommendation		
Annual assessment for risk stratification is recommended in all individuals with cardiomyopathy who exercise regularly.	I	C
Recommendations for exercise and sports participation in individuals with hypertrophic cardiomyopathy		
Participation in high-intensity exercise (including recreational and competitive sports) is not recommended in individuals who have ANY markers of increased risk [(i) cardiac symptoms or history of cardiac arrest or unexplained syncope; (ii) moderate ESC risk score (≥4%) at 5 years; (iii) LVOT gradient at rest >30 mmHg; (iv) abnormal BP response to exercise; (v) exercise-induced arrhythmias].	III	C
Recommendations for exercise and sports participation in individuals with arrhythmogenic cardiomyopathy		
Participation in high-intensity recreational exercise/sports or any competitive sports is not recommended in individuals with ACM including those who are genotype positive and phenotype negative.	III	C
Recommendations for exercise and sports participation in individuals with left ventricular non-compaction cardiomyopathy		
Participation in high-intensity exercise or competitive sports is not recommended in individuals with symptoms, LVEF<40% and/or frequent and/or complex VAs on ambulatory Holter monitoring or exercise testing.	III	C
Recommendations for exercise and sports participation in individuals with dilated cardiomyopathy		
Participation in high- or very high-intensity exercise including competitive sports is not recommended for individuals with a DCM and any of the following: (i) symptoms or history of cardiac arrest or unexplained syncope; (ii) LVEF<45%; (iii) frequent and/or complex VAs on ambulatory Holter monitoring or exercise testing; (iv) extensive LGE (>20%) on CMR; or (v) high-risk genotype (lamin A/C or filamin C).	III	C
Recommendations for exercise in individuals with myocarditis and pericarditis		
Comprehensive evaluation, using imaging studies, exercise stress test, and Holter monitoring, is recommended following recovery from acute myocarditis to assess the risk of exercise-related SCD.	I	B
Return to all forms of exercise including competitive sports is recommended after 30 days to 3 months of rest for individuals who have recovered completely from acute pericarditis, depending on clinical severity.	I	C
Among individuals with a probable or definitive diagnosis of recent myocarditis or pericarditis, participation in leisure-time or competitive sports while active inflammation is present is not recommended.	III	C
Participation in moderate- to high-intensity exercise for a period of 3–6 months after acute myocarditis is not recommended.	III	B
Participation in moderate- to high-intensity exercise, including competitive sports, is not recommended for individuals with constrictive pericarditis.	III	C

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Recommendations for exercise in individuals with arrhythmias and implantable cardiac devices		
Exercise recommendations in individuals with atrial fibrillation		
Regular physical activity is recommended to prevent AF.	I	A
Evaluation and management of structural heart disease, thyroid dysfunction, alcohol or drug abuse, or other primary causes of AF is recommended before engaging in sports.	I	A
AF ablation is recommended in exercising individuals with recurrent, symptomatic AF and/or in those who do not want drug therapy, given its impact on athletic performance.	I	B
Sports with direct bodily contact or prone to trauma are not recommended in exercising individuals with AF who are anticoagulated.	III	C
Recommendations for exercise and sports participation in individuals with paroxysmal supraventricular tachycardia and pre-excitation		
In individuals with palpitations, a comprehensive assessment to exclude (latent) pre-excitation, structural heart disease, and VAs is recommended.	I	B
Ablation of the accessory pathway is recommended in competitive and recreational athletes with pre-excitation and documented arrhythmias.	I	C
In competitive/professional athletes with asymptomatic pre-excitation, an EP study is recommended to evaluate the risk for sudden death.	I	B
Exercise recommendations in individuals with premature ventricular contractions or non-sustained ventricular tachycardia		
In exercising individuals with ≥2 PVCs on a baseline ECG (or ≥1 PVC in case of high-endurance athletes) a thorough evaluation (including a detailed family history) to exclude underlying structural or arrhythmogenic conditions is recommended.	I	C
Among individuals with frequent PVCs and non-sustained VT a thorough investigation with Holter monitoring, 12-lead ECG, exercise test, and suitable imaging is recommended.	I	C
Exercise recommendations in long QT syndrome		
It is recommended that all exercising individuals with LQTS with prior symptoms or prolonged QTc be on therapy with beta-blockers at target dose.	I	B
It is recommended that exercising individuals with LQTS should avoid QT-prolonging drugs (www.crediblemeds.org) and electrolyte imbalance such as hypokalaemia and hypomagnesaemia.	I	B
Participation in high-intensity recreational and competitive sports, even when on beta-blockers, is not recommended in individuals with a QTc>500 ms or a genetically confirmed LQTS with a QTc≥470 ms in males or ≥480 ms in females.	III	B
Participation in competitive sports (with or without ICD) is not recommended in individuals with LQTS and prior cardiac arrest or arrhythmic syncope.	III	C
Exercise recommendations in Brugada syndrome		
ICD implantation is recommended in patients with BrS with episodes of arrhythmic syncope and/or aborted SCD.	I	C
Prescription of drugs that may aggravate BrS, electrolyte abnormalities, and sports practices that increase core temperature >39°C are not recommended in individuals with overt BrS or phenotypically negative mutation carriers.	III	C
Exercise recommendations in individuals with implantable cardiac electronic devices		
It is recommended that individuals with implanted devices with/without resynchronization and underlying disease follow the recommendations pertaining to the underlying disease.	I	B
An ICD is not recommended as a substitute for disease-related recommendations when these mandate sports restrictions.	III	C
Recommendations for patients with congenital heart disease		
Participation in regular moderate exercise is recommended in all individuals with CHD.	I	B
A discussion on exercise participation and provision of an individualized exercise prescription is recommended at every CHD patient encounter.	I	B
Competitive sport is not recommended for individuals with CHD who are in NYHA class III–IV or with potentially serious arrhythmias.	III	C
Recommendations for exercise in pregnancy		
Among pregnant women without medical or obstetric contraindications, participation in at least 150 min/week of moderate-intensity aerobic exercise before, during, and after pregnancy is recommended.	I	B
Re-evaluation before continuing exercise or training is recommended in pregnant woman if they experience excessive shortness of breath, severe chest pain, dizziness or syncope, regular painful contractions, vaginal bleeding, or amniotic fluid leakage.	I	A
Among women with CVD who were habitually engaged in strength training or power sport disciplines before pregnancy, discussing the option with the medical team before continuing and avoiding the Valsalva manoeuvre is recommended.	I	A
Exercise or sport involving forceful physical contact, risk of falling or abdominal trauma, heavy lifting, scuba diving, or exercising at high altitude when unacclimatized are not recommended.	III	C

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Vigorous exercise associated with a maximal predicted heart rate >90% of the predicted heart rate is not recommended during pregnancy.	III	B
Exercising while lying supine on a hard surface is not recommended after the first trimester due to the risk of decreased venous return and uterine blood flow.	III	B
Recommendations for exercise in chronic kidney disease		
Low- to moderate-intensity aerobic exercise training (up to 150 min/week), and low- to moderate-intensity resistance exercise training (2 day per week, 8–12 exercises, 12–15 repetitions), and flexibility exercises are recommended in all individuals with CKD.	I	A
Among patients with established osteodystrophy/osteoporosis, or coagulopathies, participation in contact sports is not recommended.	III	C
Participation in sport is not recommended in the following circumstances: electrolyte abnormalities, recent changes to the ECG, excess inter-dialysis weight gain, changing or titration of medication regimen, pulmonary congestion, and increasing peripheral oedema.	III	C
Recommendations for exercise in individuals with cancer		
Regular exercise during and after cancer therapy is recommended to reduce cancer-related fatigue, and improve quality of life, physical fitness, and prognosis.	I	A
Among individuals treated with cardiotoxic medications, echocardiography before participation in high-intensity exercise is recommended.	I	A
Recommendations for exercise in individuals with spinal cord injury		
In adults with spinal cord injury participation in 20 min of moderate- to vigorous-intensity aerobic exercise at least 3 times a week, along with moderate-intensity resistance training 2–3 times per week, is recommended for cardiorespiratory fitness, cardiometabolic health, and muscle strength benefits.	I	A
Artificial methods of inducing autonomic dysreflexia by causing intentional pain to the lower half of the body ('boosting') through obstruction of an indwelling urinary catheter, overly tight leg straps and electrical shocks or other methods of pain to the genitalia or lower limbs can be life-threatening and are not recommended.	III	C
Recommendations for exercise in individuals with ventricular assist devices		
Regular exercise, through cardiac rehabilitation, combining moderate-intensity aerobic and resistance exercise, is recommended in individuals with a ventricular assist device (VAD).	I	A
Sports that may potentially affect any of the VAD components (e.g. with body contact) are not recommended.	III	C
Physical activity and sports recommendations in patients and athletes with peripheral arterial disease		
It is recommended that patients with atherosclerotic PAD perform regular exercise (at least 150 min/week of moderate aerobic exercise or 75 min/week of vigorous aerobic exercise or a combination thereof) as part of the secondary prevention strategy.	I	A
In patients with symptomatic LEAD, supervised exercise training programmes including walking to the maximal or submaximal distance for at least 3 h/week are indicated.	I	A
Continuation of competitive sports is recommended in athletes with traumatic or non-traumatic PAD following recovery after successful open surgery or percutaneous revascularization.	I	C

ACM = arrhythmogenic cardiomyopathy; AF = atrial fibrillation; AOCA = anomalous origin of coronary arteries; BP = blood pressure; BMI = body mass index; BrS = Brugada syndrome; CAD = coronary artery disease; CCS = chronic coronary syndrome; CHD = congenital heart disease; CKD = chronic kidney disease; CMR, cardiovascular magnetic resonance; CT = computed tomography; CV = cardiovascular; CVD= cardiovascular disease; EEG = electrocardiogram; EP = electrophysiological; ESC = European Society of Cardiology; EP = electrophysiological; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; ICD = implantable cardioverter defibrillator; LEAD = lower extremity artery disease; LQTS = long QT syndrome; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; MVA = mitral valve area; NYHA = New York Heart Association; PAD = peripheral arterial disease; PVC = premature ventricular contractions; SCD = sudden cardiac death; SBP = systolic blood pressure; VA = ventricular arrhythmia; VAD = ventricular assist device; VT = ventricular tachycardia.

[a]Class of recommendation.

[b]Level of evidence.

10. Supplementary data

Supplementary Data with additional Supplementary Figures, Tables, and text complementing the full text are available on the *European Heart Journal* website and via the ESC website at www.escardio.org/guidelines.

11. Appendix

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