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Heart Rhythm Society SM

2024 HRS expert consensus statement on arrhythmias in the athlete: Evaluation, treatment, and return to play

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ABSTRACT

Youth and adult participation in sports continues to increase, and athletes may be diagnosed with potentially arrhythmogenic cardiac conditions. This international multidisciplinary document is intended to guide electrophysiologists, sports cardiologists, and associated health care team members in the diagnosis, treatment, and management of arrhythmic conditions in the athlete with the goal of facilitating return to sport and avoiding the harm caused by restriction. Expert, disease-specific risk assessment in the context of athlete symptoms and diagnoses is emphasized throughout the document. After appropriate risk assessment, management of arrhythmias geared toward return to play when possible is addressed. Other topics include shared decision-making and emergency action planning. The goal of this document is to provide evidence-based recommendations impacting all areas in the care of athletes with arrhythmic conditions. Areas in need of further study are also discussed.

KEYWORDS Arrhythmogenic diseases; athletes; electrophysiology; emergency action plans; return to play; risk assessment; sports cardiology; sudden cardiac arrest; sudden cardiac death; shared decision-making

(Heart Rhythm 2024;■:1–97)

ABBREVIATIONS AAD = antiarrhythmic drug; AAOCA = anomalous aortic origin of coronary arteries; ABiMVP = arrhythmogenic bileaflet mitral valve prolapse; ACM = arrhythmogenic cardiomyopathy; AED = automated emergency defibrillator; AF = atrial fibrillation; AN-SUD = autopsy-negative sudden unexplained death; AP = accessory pathway; APERP = accessory pathway effective refractory period; ARVC = arrhythmogenic right ventricular cardiomyopathy; ATP = antitachycardia pacing; AV = atrioventricular; AVRT = atrioventricular reciprocating tachycardia; CHD = congenital heart disease; CI = confidence interval; CK = creatine kinase; CMR = cardiac magnetic resonance imaging; COR = class of recommendation; CPET = cardiopulmonary exercise testing; CPP = cardiac physiologic pacing; CPR = cardiopulmonary resuscitation; CPVT = catecholaminergic polymorphic ventricular tachycardia; CRT = cardiac resynchronization therapy; CT = computed tomography; CTA = computed tomographic angiography; CTI = cavotricuspid isthmus; DCM = dilated cardiomyopathy; Dx = diagnosis; EAP = emergency action plan; ECG = electrocardiogram; EICR = exercise-induced cardiac remodeling; EMS = emergency medical services; EP = electrophysiology; HCM = hypertrophic cardiomyopathy; IAS = inherited arrhythmia syndrome; ICD = implantable cardioverter-defibrillator; LAAO = left atrial appendage occlusion; LCSD = left cardiac sympathetic denervation; LOE = level of evidence; LQTS = long QT syndrome; LTE = life-threatening events; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; PAC = premature atrial contraction; PVC = premature ventricular contraction; PVI = pulmonary vein isolation; RVOT = right ventricular outflow tract; RWI = relationships with industry; SCA = sudden cardiac arrest; SCA/D = sudden cardiac arrest and death; SCD = sudden cardiac death; SCT = sickle cell trait; SPERRI = shortest preexcited R-R interval; SQTS = short QT syndrome; TTM = targeted temperature management; VF = ventricular fibrillation; WPW = Wolff-Parkinson-White

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Top 10 Take-Home Messages

1. For many arrhythmogenic disease entities, current data in athletes, while often not large enough to be definitive, have not confirmed increased arrhythmic risk of continuing sports participation for athletes who are appropriately risk-assessed and treated, and thus the approach to return to play is one of individualized shared decision-making.
2. The overarching goal in caring for athletes should be facilitating the athlete's return to sport if this is the desired outcome, through appropriate risk assessment and athlete-focused management of their arrhythmic condition. Restriction from sport is not benign.
3. Both venue-based and individualized emergency action plans including plans for early defibrillation are critical to survival of athletes with sudden cardiac arrest.
4. Disease-specific and guideline-based risk assessment and treatment of arrhythmogenic conditions prior to return to play are critical.
5. For patients with underlying complex arrhythmias, appropriate strategies for sudden death prevention and arrhythmia suppression are needed prior to return to play, including confirmation of suppression of arrhythmia during exercise.
6. Treatment decisions—including those regarding antiarrhythmic medications, ablation, and devices—should take athletic performance and training into consideration.
7. Exercise stress testing in athletes for diagnostic purposes or defining therapeutic efficacy should mimic the athlete's sport where possible and be terminated based on maximal effort, symptoms, and/or documentation of arrhythmia.
8. Endurance exercise in particular may contribute to arrhythmogenic conditions such as atrial fibrillation and genotype-negative arrhythmogenic right ventricular cardiomyopathy; risks and benefits of continued participation in endurance sports should be carefully weighed in athletes with these conditions.
9. The choice of pacemaker or defibrillator form factor and programming parameters should take into consideration the type of sport and training required so as to minimize risk of damage to the system.

10. Athletes with a diagnosis of Wolff-Parkinson-White pattern or syndrome should be allowed to return to play pending timely expert evaluation and treatment, as there is lack of conclusive evidence of increased risk of life-threatening arrhythmias with athletic participation.

Section 1 Introduction

1.1 Preamble

The Heart Rhythm Society (HRS) has developed scientific and clinical documents guiding the management of cardiac arrhythmias since 1996. This HRS-led expert consensus statement was developed in collaboration with the American College of Cardiology (ACC), the American Heart Association (AHA), the American Medical Society for Sports Medicine (AMSSM), the Asia Pacific Heart Rhythm Society (APHRS), the European Heart Rhythm Association (EHRA), the Latin American Heart Rhythm Society (LAHRS), and the Pediatric and Congenital Electrophysiology Society (PACES). This international expert consensus statement is intended to educate clinicians providing arrhythmia-related care to athletes, foster acceptance of the shared decision-making model for these populations, and promote research in best approaches for prevention, diagnosis, and management of arrhythmias in athletes.

1.2 Document scope and rationale

Sports cardiology is a rapidly evolving field. In the past decades, both youth sports, and adult individuals participating in organized sports such as marathons, have increased exponentially. Understanding cardiac care of the athlete requires specialized expertise, as sports participation in itself leads to changes that can be both adaptive and potentially maladaptive, such as the increased incidence of atrial fibrillation (AF) well documented in endurance athletes. Further, treatment decisions may be influenced by the desire to return to sports. To meet these needs, the field of sports cardiology is growing rapidly. As sports cardiology grows, attention to arrhythmic issues in the athlete needs to grow in parallel.

The overarching goal of this document is to provide evidence-based and expert consensus recommendations on the diagnosis, treatment, and management of arrhythmias in athletes of all ages, with an emphasis on shared decision-making. Participation in sports has innumerable benefits, both physical and psychological. Restriction from sports is not benign, with significant deleterious impact on psychological well-being and quality of life. While not always achievable, the goal should be facilitating the athlete's return to sport if this is the desired outcome, through appropriate risk assessment and athlete-focused management of their arrhythmic condition, and concerted efforts to achieve equity of care for all athletes.

1.3 Editorial independence

This expert consensus statement is sponsored by the HRS and was developed without commercial support. All writing committee members volunteered their time to the writing and review efforts.

1.4 Organization of the writing committee

The writing committee consisted of internationally recognized experts from 5 countries in the fields of clinical electrophysiology (EP), cardiology, pediatric EP and cardiology, genetic cardiology, sports cardiology, sports medicine, and clinical research science. Each writing committee member served as a representative of either HRS or the collaborator society and was nominated according to each organization's processes. HRS strives to ensure that the writing committee contains both requisite expertise and diverse representation from the broader medical community. This is achieved by selecting participants from a wide range of backgrounds representing different geographic regions, genders, races, ethnicities, intellectual perspectives, and scopes of clinical practice and by inviting organizations and professional societies with related interests and expertise to participate as collaborators. In addition, a patient partner was included in the writing committee to ensure a focus on delivering optimal patient care that is in alignment with patients' wants, needs, and preferences.

HRS has rigorous policies and methods to ensure that documents are developed without bias or improper influence. The HRS policy on relationships with industry (RWI) and other entities can be found in the *HRS Code of Ethics and Professionalism:* Appendix C and in the *HRS Clinical Document Development Methodology Manual and Policies*. A majority of the writing committee was free of relevant RWI throughout the development of the document, and sections with recommendations were written by the writing committee members who were free of relevant RWI. For full transparency, Appendix 1 is a comprehensive list of RWI (both relevant and nonrelevant to the document topic) disclosed by the writing committee members. Appendix 2 is a comprehensive list of RWI disclosed by the peer reviewers.

1.5 Evidence review and formulation of recommendations

This expert consensus statement was developed in accordance with the clinical practice methodology processes detailed in the *HRS Clinical Document Development Methodology Manual and Policies: Executive Summary,*[1] and with the standards issued in 2011 by the Institute of Medicine (now National Academy of Medicine).[2]

The writing committee reviewed evidence gathered by electronic literature searches (MEDLINE, PubMed, Embase, Cochrane Library, Ovid). No specific year was chosen for the oldest literature. Literature searches focused whenever possible on randomized controlled trials, but systematic reviews, nonrandomized and registry studies, cohort studies, and case series were included. Evidence tables are included in Appendix 3 and summarize the evidence used by the writing committee to formulate recommendations. References are representative of the totality of data and are not meant to be all-inclusive. Limitations of the evidence base are discussed in individual sections.

To assess consensus after discussions, the writing committee members participated in surveys. A predefined threshold of 70% approval for each recommendation was required, with

Table 1 ACC/AHA recommendation system: Applying class of recommendation and level of evidence to clinical strategies, interventions, treatments, and diagnostic testing in patient care (updated May 2019)*

CLASS (STRENGTH) OF RECOMMENDATION	
CLASS 1 (STRONG)	**Benefit >>> Risk**
Suggested phrases for writing recommendations: • Is recommended • Is indicated/useful/effective/beneficial • Should be performed/administered/other • Comparative-Effectiveness Phrases†: – Treatment/strategy A is recommended/indicated in preference to treatment B – Treatment A should be chosen over treatment B	
CLASS 2a (MODERATE)	**Benefit >> Risk**
Suggested phrases for writing recommendations: • Is reasonable • Can be useful/effective/beneficial • Comparative-Effectiveness Phrases†: – Treatment/strategy A is probably recommended/indicated in preference to treatment B – It is reasonable to choose treatment A over treatment B	
CLASS 2b (WEAK)	**Benefit ≥ Risk**
Suggested phrases for writing recommendations: • May/might be reasonable • May/might be considered • Usefulness/effectiveness is unknown/unclear/uncertain or not well-established	
CLASS 3: No Benefit (MODERATE) **(Generally, LOE A or B use only)**	**Benefit = Risk**
Suggested phrases for writing recommendations: • Is not recommended • Is not indicated/useful/effective/beneficial • Should not be performed/administered/other	
CLASS 3: Harm (STRONG)	**Risk > Benefit**
Suggested phrases for writing recommendations: • Potentialy harmful • Causes harm • Associated with excess morbidity/mortality • Should not be performed/administered/other	

LEVEL (QUALITY) OF EVIDENCE‡	
LEVEL A	
• High-quality evidence‡ from more than 1 RCT • Meta-analyses of high-quality RCTs • One or more RCTs corroborated by high-quality registry studies	
LEVEL B-R	**(Randomized)**
• Moderate-quality evidence‡ from 1 or more RCTs • Meta-analyses of moderate-quality RCTs	
LEVEL B-NR	**(Nonrandomized)**
• Moderate-quality evidence‡ from 1 or more well-designed, well-executed nonrandomized studies, observational studies, or registry studies • Meta-analyses of such studies	
LEVEL C-LD	**(Limited Data)**
• Randomized or nonrandomized observational or registry studies with limitations of design or execution • Meta-analyses of such studies • Physiological or mechanistic studies in human subjects	
LEVEL C-EO	**(Expert Opinion)**
• Consensus of expert opinion based on clinical experience	

COR and LOE are determined independently (any COR may be paired with any LOE).

A recommendation with LOE C does not imply that the recommendation is weak. Many important clinical questions addressed in guidelines do not lend themselves to clinical trials. Although RCTs are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

* The outcome or result of the intervention should be specified (an improved clinical outcome or increased diagnostic accuracy or incremental prognostic information).

† For comparative-effectiveness recommendations (COR 1 and 2a; LOE A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

‡ The method of assessing quality is evolving, including the application of standardized, widely-used, and preferably validated evidence grading tools; and for systematic reviews, the incorporation of an Evidence Review Committee.

COR indicates Class of Recommendation; EO, expert opinion; LD, limited data; LOE, Level of Evidence; NR, nonrandomized; R, randomized; and RCT, randomized controlled trial.

Adapted with permission from the American College of Cardiology (ACC) and the American Heart Association (AHA).

a minimum quorum of two-thirds of the writing committee. An initial failure to reach consensus was resolved by subsequent discussions, revisions as needed, and re-voting. Writing committee members with RWI did not vote on recommendations concerning relevant topics.

1.6 Class of recommendation and level of evidence

Recommendations in this expert consensus statement are designated with both a class of recommendation (COR) and a level of evidence (LOE). The COR denotes the strength of the recommendation based on the assessment of the magnitude and certainty of the benefits in proportion to the risks. The LOE reflects the quality of the evidence that supports the recommendation based on type, quantity, and consistency of data from clinical trials and other sources (Table 1).[3]

For clarity and usefulness, each recommendation is linked to the supportive evidence through the specific references from the literature used to justify the LOE rating, which are also summarized in their evidence tables (Appendix 3). Each recommendation is accompanied by explanatory text. Flow diagrams and appropriate tables provide a summary of the recommendations and are intended to assist clinicians at the point of care.

1.7 Document review and approval

The HRS invites public and stakeholder involvement in document development. In addition to patient representation in

Table 2 Relevant clinical practice documents

Title	Publication year
PACES/HRS Expert consensus statement on the management of the asymptomatic young patient with a Wolff-Parkinson-White (WPW, ventricular preexcitation) electrocardiographic pattern[4]	2012
AHA/ACC Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: Preamble, principles, and general considerations[5]	2015
International criteria for electrocardiographic interpretation in athletes: Consensus statement[6]	2017
Sports cardiology: Core curriculum for providing cardiovascular care to competitive athletes and highly active people[7]	2017
AHA/ACC/HRS Guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death[8]	2017
ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease[9]	2020
AHA/ACC Guideline for the diagnosis and treatment of patients with hypertrophic cardiomyopathy[10]	2020
APHRS/HRS expert consensus statement on the investigation of decedents with sudden unexplained death and patients with sudden cardiac arrest, and of their families[11]	2020
Recommendations for participation in leisure-time physical activity and competitive sports in patients with arrhythmias and potentially arrhythmogenic conditions. Part 1: Supraventricular arrhythmias[12]	2021
Recommendations for participation in leisure-time physical activity and competitive sports of patients with arrhythmias and potentially arrhythmogenic conditions. Part 2: Ventricular arrhythmias, channelopathies, and implantable defibrillators[13]	2021
ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death[14]	2022
ESC Guidelines for the management of cardiomyopathies[15]	2023
ACC/AHA/ACCP/HRS Guideline for the diagnosis and management of atrial fibrillation[16]	2023
HRS/APHRS/LAHRS Guideline on cardiac physiologic pacing for the avoidance and mitigation of heart failure[17]	2023
2024 AHA/ACC/AMSSM/HRS/PACES/SCMR Guideline for the Management of Hypertrophic Cardiomyopathy[18]	2024

the writing committee, draft recommendations were posted for public comment, and contribution was solicited from regulatory agencies and patient organizations.

This expert consensus statement was approved by the writing committee and underwent internal review by the HRS Scientific and Clinical Documents Committee. The document underwent external peer review by reviewers appointed by HRS and each of the collaborating societies, and revisions were made by the chairs. A record of writing committee response to reviewer comments and rationale is maintained by the HRS.

1.8 Document updates

The HRS Scientific and Clinical Documents Committee reviews each clinical practice document for currency at least every 5 years, or earlier in the event of newly published data. The literature is routinely monitored to evaluate the continued validity of recommendations.

1.9 Relevant clinical practice documents

Clinical practice documents relevant to the topic of diagnosing, managing, and treating arrhythmias in athletes were used to inform the development of this consensus statement. Table 2 lists the relevant clinical practice guidelines and consensus statements that the writing committee considered as fundamental to the development of this document.

Section 2 General concepts and principles

2.1 Definitions

The key terms related to athletes used in this consensus statement are defined in Table 3.

Definition of an athlete

Although there is no universal definition of "athlete" in the medical literature,[19] in this document, athletes are defined as individuals who are exposed to regular and high cardiovascular stress demands due to habitual vigorous exercise training for the purposes of obtaining a high level of fitness and for competition, occupation, or recreation. This definition of the athlete is broader than that used in other clinical practice documents on

Table 3 Definitions

Term	Definition
Athletes	Individuals who are engaged in habitual and vigorous training for the purposes of obtaining a high level of fitness. This includes competitive athletes, high-level recreational exercise enthusiasts, and occupational (tactical) athletes.
Age domains in athletes	Given the complexity of the interaction between age and different arrhythmic diseases, rather than use arbitrary age cut points, athletes are considered in different age domains based on stages of development. **Young** = prepubertal (< / ≈ 12 years old) and adolescent (≈ 13-17 years old) **Young adult** ≈ 18-24 years old **Adult** > / ≈ 25 years old **Master** > / ≈ 35 years old
Return to play/return to sport	These terms refer to returning to the desired level or intensity of recreational or competitive sport participation.

sports eligibility in athletes with cardiovascular diseases.[5,20–22] Specifically, the 2015 AHA/ACC scientific statement[5] defines a "competitive" athlete as "one who participates in an organized team or individual sport that requires regular competition against others as a central component, places a high premium on excellence and achievement, and requires some form of systematic (and usually intense) training." However, athletes may also be noncompetitive "exercise enthusiasts" or people who engage in regular, vigorous exercise training for purposes of recreation or other health benefits. Moreover, there is emerging recognition of the "tactical" athlete, such as members of law enforcement, military, or fire departments.[23] These individuals are also exposed to regular and high cardiovascular stress demands, not for competition or recreation, but for service and occupation. As issues related to management of arrhythmias may be similar for these types of athletes, this document addresses all of these groups. Throughout this document, "return to play" and "return to sport" refer to a return to the desired level or intensity of recreational, occupational, or competitive sport participation.

This document applies to both adult and pediatric athletes. Age considerations in the context of the clinical management of many arrhythmias are complex and do not fit well using a binary adult versus pediatric age cut point (eg, 18 or 21 years old) or a consensus-based lower pediatric age cut point (prior consensus cardiovascular recommendations for sports eligibility[5] and guidelines for the interpretation of the athletic electrocardiogram [ECG][6] used 12 years of age as a lower pediatric age cut point). Additionally, emphasis on competitive youth sports beginning at younger ages is increasing and genetic heart conditions, especially those with high penetrance, can affect young children. In this document the management of athletes with arrhythmias is considered in terms of the age domains defined in Table 3. The term "Masters athlete" is often used to indicate either formally, an athlete over 35 years, or informally, an older athlete.[7,24] "Young" is used in this document to refer to prepubertal and adolescent athletes. In areas where no age-group is specified, recommendations do not vary by age.

2.2 Clinical considerations for athletes with arrhythmias

Recommendations for clinical considerations for athletes with arrhythmias

COR	LOE	Recommendations
1	C-EO	1. In athletes with symptoms of arrhythmias, clinical evaluation should include exercise history and history of performance-enhancing drugs.
1	C-EO	2. In athletes with symptoms of arrhythmias, differential diagnosis should include consideration of etiologies specific to their sport.
1	C-EO	3. In athletes with symptoms of arrhythmias, evaluation should be performed by clinicians with an understanding of unique electrical and structural adaptations specific to the athlete (ECG or cardiac structural) and of the differentiation of "grey zone" cardiac phenotypes.
1	C-EO	4. In athletes with symptoms of or concern for arrhythmias, exercise stress testing should be based on maximal effort and/or symptom reproduction rather than heart rate or protocol completion.
1	C-EO	5. In athletes with symptoms of or concern for arrhythmias, exercise stress testing should be performed based on sport type and situation where symptoms are elicited.
1	C-LD	6. In athletes with arrhythmogenic conditions returning to play, a stress test should be performed prior to return to play.[25,26]
1	C-EO	7. In athletes with arrhythmias, clinical management strategies should consider limitations on athletic performance caused by the arrhythmia or by the pharmacological treatments for the arrhythmia, to optimize return to play if desired.
1	C-EO	8. In athletes with arrhythmias, clinical management strategies should take into account athlete- and sport-specific considerations including impact of therapeutic options on timing of return to play and any sport-specific restrictions.
1	C-EO	9. In athletes with arrhythmias who are not returning to competitive sports, plans for other levels of exercise should be discussed.

Synopsis

The clinical evaluation of arrhythmias that could be life-threatening when provoked during exercise is generally the same for all patients, whether they are considered an athlete or not. Similarly, "red flag" symptoms, such as unheralded syncope during exercise, lead to a similar differential diagnosis and high clinical concern for both athletes and nonathletes. However, compared with nonathletes, there are several

reasons why athletes come with distinct considerations in the approach to and the evaluation of arrhythmias, as well as in the determination of treatment options.

Recommendation-specific supportive text

1. Some arrhythmias (eg, AF, ventricular tachycardia in the setting of arrhythmogenic cardiomyopathy [ACM]) have independent associations or direct causal links with exercise training habits.[27,28] As a result, a detailed exercise history, including both during and outside of organized sports, may provide clues to the diagnosis of different arrhythmia syndromes. Exercise history includes type of sport, frequency, and duration, and type of training and intensity of training and work demands for the tactical athlete. A history of performance-enhancing drug use is also important, as these can have arrhythmic effects (Table 4).[29–33] Thus, for athletes, differential diagnoses may be uniquely guided by exercise or training habits.
2. Unique sport-specific factors must be considered in determining the differential diagnosis for symptomatic athletes. For example, dietary habits within the culture of some sports (eg, eating disorders) can precipitate arrhythmic symptoms or lead to a high burden of ectopic beats and should be ascertained during the clinical evaluation. Also, performance-enhancing drugs can impact cardiac structure and electrical function (Table 4).[29–33] Additionally, exercise-associated collapse with prior prodromal symptoms immediately after finishing prolonged exercise such as a distance race is consistent with a benign presyncopal or syncopal event.
3. Interpretation of electrical and cardiac structural alterations in athletes can impact the consideration of the diagnosis of arrhythmias in athletes, and thus it is critical that clinicians evaluating athletes have appropriate training in how vigorous exercise influences cardiac structure and function and how these changes manifest on cardiac testing.[7,28] Overdiagnosis of cardiac abnormalities in the athlete is frequent due to lack of recognition of these changes. It is well understood that habitual and vigorous exercise training and the subsequent changes in autonomic tone and hemodynamic physiologic stresses can lead to cardiovascular adaptations.[28,34] These physiologic changes can lead to alterations on the surface 12-lead ECG[6] and also changes in cardiac phenotypes, termed exercise-induced cardiac remodeling (EICR) (Table 5) or, colloquially, the "athlete's heart,"[28] which can overlap with cardiac pathology, termed "grey zone" phenotypes. Exercise history, as above, is critical to understand expected adaptations (Figure 1).[7,28] High vagal tone may manifest on the 12-lead ECG as sinus bradycardia or sinus arrhythmia or first-degree and Mobitz I second-degree atrioventricular (AV) block.[6] Vigorous exercise can lengthen repolarization, including to levels which may indicate risk.[35] EICR is a sport-specific phenomenon. All vigorous sports involve combinations of dynamic (endurance/isotonic) and static (strength/isometric) physiology in a continuum.[7] Dynamic exercise requires increases in oxygen delivery, and thus endurance training, dynamic exercise over long periods of time, represents a volume hemodynamic challenge to the heart, with resultant chamber dilatation. Static exercise increases afterload, and thus strength training presents a pressure challenge, in which, over time, cardiac hypertrophy occurs. In endurance or mixed endurance-strength sports, the pattern of hypertrophy is generally eccentric with concomitant biventricular and biatrial enlargement. Advanced imaging such as cardiac magnetic resonance imaging (CMR) can be helpful in differentiating EICR from pathology in "grey zone" athletes.[36,37]
   Recognition of the consequences of these phenotypic alterations is important for several reasons. The first is diagnosis: arrhythmias in the context of EICR may have very different implications than those occurring in pathological states, and thus, recognition of "grey zone" phenotypic crossover between extreme forms of adaptive EICR versus mild forms of cardiomyopathy—hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), or ACM—is critical.[28,38] Next, changes in cardiac structure over time, in particular, left atrial dilatation in Masters athletes, may predispose an athlete to atrial arrhythmias (see Section 8). Finally, for some cardiovascular disease entities, specifically some of the arrhythmogenic cardiomyopathies (see Section 7), endurance exercise with its increased volume challenge may increase progression of disease and thus need to be discussed as part of the shared decision-making process.
4. Athletes may present with symptoms only at very high exercise intensities. As such, it is critical that in the clinical evaluation of the symptomatic athlete, exercise stress testing proceeds to maximal volitional effort (or symptoms) and is not terminated based on heart rate thresholds (eg, 85% maximum predicted heart rate). Maximal effort may be determined by the rating of perceived exertion (eg, Borg scale) or maximum heart rate achieved, although heart rate alone should not be considered a stopping point. Cardiopulmonary exercise testing (CPET) is also useful in adjudicating maximal effort through measured VO_{2max}, maximum exercise heart rate, surpassing the ventilatory threshold, and the respiratory exchange ratio. CPET that incorporates quantitative gas and metabolic analyses may enhance understanding of a patient's particular physiology and limitations. Exercise protocols may vary, but generally require a more accelerated ramp effort beyond standard protocols (eg, Bruce protocol).[39,40] In small studies, a "burst" protocol involving a sudden high workload has shown increased sensitivity for detecting abnormalities in catecholaminergic polymorphic ventricular tachycardia (CPVT)[41] and HCM.[42] Reported protocols include a first stress test, in which the treadmill speed/grade at maximum heart rate is determined, and then a second test, in which the treadmill exercise is started at this speed/grade. For athletes in

Table 4 Potential arrhythmic effects of certain stimulants and performance-enhancing drugs[29–33]

Stimulant/PED	Potential arrhythmic effect
Cocaine	Ectopy, QT prolongation, early repolarization, myocardial fibrosis and infarction, Na^+, K^+, and Ca^{2+} channel dysfunction, atrial arrhythmias, ventricular arrhythmias, bradyarrhythmias, SCD
Amphetamines	Ectopy, QT prolongation, atrial arrhythmias, ventricular arrhythmias, K^+ and Ca^{2+} channel dysfunction. atrial arrhythmias, ventricular arrhythmias, SCD
Marijuana	Ectopy, QT prolongation, atrial arrhythmias, ventricular arrhythmias, myocardial infarction, cardiomyopathy, atrial arrhythmias, stroke
Ecstasy	Ectopy, atrial arrhythmias, ventricular arrhythmias, myocardial infarction, valvular heart disease, cardiomyopathy, myocardial infarction, SCD
Caffeine	Ectopy, sinus tachycardia
Anabolic steroids	Myocardial dysfunction, coronary artery disease, myocardial fibrosis, left ventricular hypertrophy, atrial fibrillation

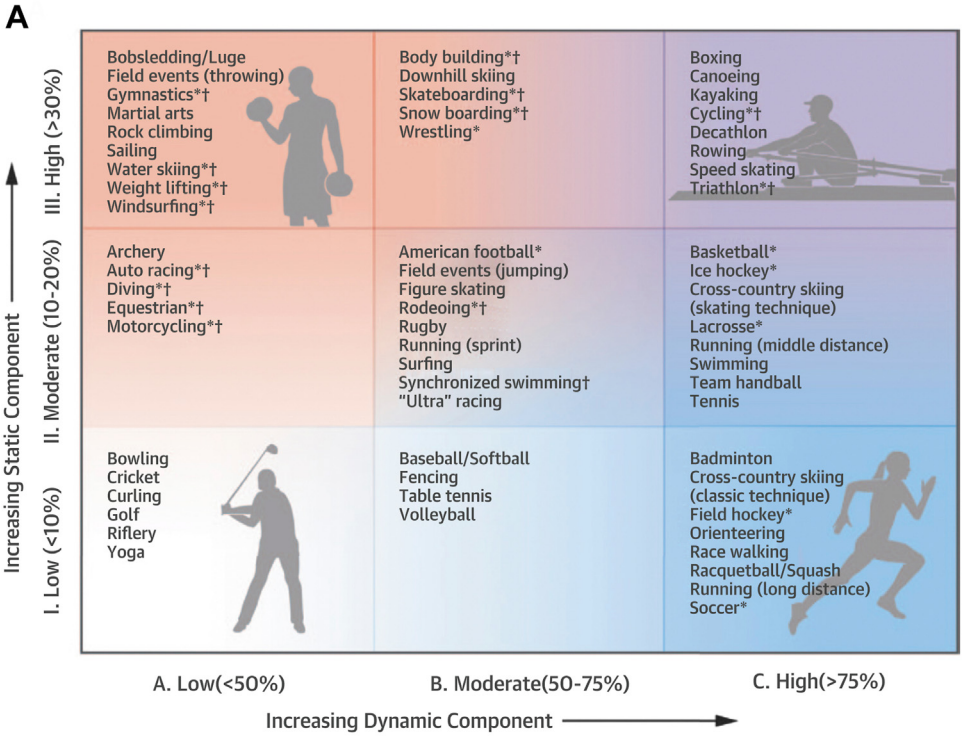
Ectopy refers to premature atrial contractions (PACs) and/or premature ventricular contractions (PVCs). PED = performance-enhancing drug; SCD = sudden cardiac death.

whom stress testing does not invoke symptoms and/or symptoms are rare, external/internal monitoring during the athlete's usual training and competition situation may be a next step, as described in Section 5 in more detail.

5. It is also important to test the athlete with the appropriate exercise modality based on the type of athlete and the environment in which symptoms are elicited. To appropriately assess the athlete, use a treadmill (sometimes with additional sprint intervals), cycle ergometry, and rowing ergometry (if available).
6. Using the stress test to determine arrhythmia suppression for athletes returning to play after diagnosis and treatment of an arrhythmic condition has been reported as part of return-to-play protocols for conditions including CPVT and other genetic heart diseases, with long-term results suggesting overall safety of the protocol.[26] It is used in the general population for determining beta blocker response,[25,43] and efficacy of surgery for anomalous aortic origin of coronary arteries (AAOCA). Discussion of the exercise stress test for guiding programming of implantable cardioverter-defibrillators (ICDs) and pacemakers appears in more detail in those sections. For some entities, such as Wolff-Parkinson-White (WPW), where an identifiable cure for arrhythmia exists, exercise stress testing prior to return to play is not needed, but for most arrhythmic entities, complete cure is difficult to determine and documentation of arrhythmia suppression is needed.
7. In addition to the accepted medical standards of care, consideration of limitations on athletic performance, related to symptomatology and/or potential treatments, become more relevant for athletes. For example, symptoms from an arrhythmia or subsequent pharmacotherapy choices (eg, beta blockers, antiarrhythmic drugs) can limit performance and thus may require procedural treatment strategies. Overall, clinical management options in all of these scenarios, particularly when taking into consideration return to play, can be complex, and therefore require an expert-guided and shared-decision-making approach.
8. For professional and high-level collegiate athletes, there will likely be heightened emphasis on the timing of return to play given the financial investment from the team/sponsors and/or concomitant income concerns from the athlete. Prior to initiation of pharmacological treatments, it should be determined whether medications under consideration require disclosure to supervising bodies and/or influence eligibility. Data on performance-enhancing effects of beta blockers are minimal, with one study in marksmen showing improved shooting performance,[44] another no improvement in archers, with resulting prohibition of these agents by the World Anti-Doping Agency for sports requiring precision and accuracy such as archery and skiing.[45,46] Tactical athletes, such as divers, may face unique challenges due to demands of the job and environment.

Table 5 Exercise-induced cardiac remodeling (electrical and structural)[28]

High autonomic/vagal tone	Electrocardiographic findings
	• Sinus bradycardia • Sinus arrhythmia • Junctional rhythm • Ectopic atrial rhythm • Early repolarization • First-degree atrioventricular block • Mobitz I second-degree atrioventricular block
Hemodynamic challenge	**Cardiac structural findings**
Isotonic exercise	• Eccentric left ventricular hypertrophy • Symmetric right ventricular enlargement • Biatrial dilation • Low-normal/mildly reduced left ventricular systolic function • Enhanced left ventricular diastolic function
Isometric exercise	• Concentric left ventricular remodeling • Normal right ventricular dimensions • Normal biatrial dimensions • No changes in left ventricular function



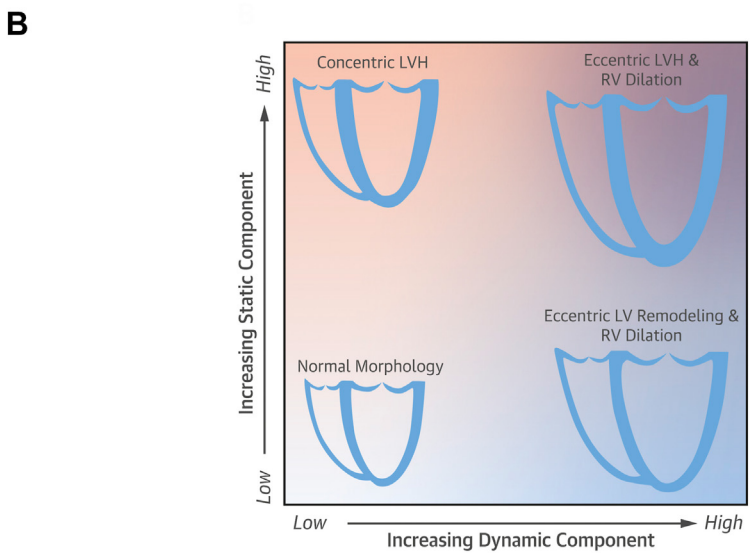


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Figure 1
Expected ventricular adaptation from static and dynamic stressors on the heart. (A) Physiological classification of common sporting disciplines representing relative contributions of static and dynamic physiology. (B) Anticipated structural cardiac adaptations that develop as a function of the static and dynamic stressors. *Danger of bodily collision. †Increased risk if syncope occurs. LV = left ventricular; LVH = left ventricular hypertrophy; RV = right ventricular. Reprinted with permission from Baggish et al.[7]

9. For athletes who do not return to play and cease a competitive athletic career, regardless of the diagnosis or condition, an exercise prescription should be discussed and encouraged. Maintaining a healthy and high level of cardiorespiratory fitness improves overall health and reduces the risk of other forms of cardiovascular disease. Athletes not returning to competitive sports should not be discouraged from exercising. CPET can be used in determining an exercise prescription, and detailed discussions with the athlete on their specific exercise goals should be a part of this process. Transition away from competitive sports can be psychologically difficult for athletes, particularly if not voluntary, with loss of athletic identity leading to depression or other psychological distress. Career-ending injuries can impact athletes for 10 years after retirement. Working with the athlete's care network, including team physicians and sports psychologists with experience in these issues, is important for the athlete's well-being.[47]

2.3 Shared decision-making and clinical management determination

This document presents a shared decision-making approach to return to play, for decisions both around whether to return to play and around treatments tailored to facilitating that decision. Historically, return to play decisions have used algorithm-like approaches based on specific cutoffs for clinical variables and sport classifications. However, for many disease entities, current prospective data in athletes have not confirmed increased arrhythmic risk of continuing sports participation for athletes who are appropriately risk-assessed and treated, emphasizing the importance of individualized shared decision-making. Clinical characteristics indicating higher risk of sudden cardiac arrest (SCA), which then guide treatment such as ICD and other disease-specific therapies, have been described for most cardiovascular disease entities. Whether there are clinical characteristics that interact with vigorous exercise to increase arrhythmic risk has not yet been delineated for most entities. Thus, this document focuses on risk assessment and treatment using shared decision-making that should take into account well-described disease-specific risk factors for SCA as well as sport-specific factors as described in Section 2.3.

Participation in vigorous athletic activity has multiple benefits, both psychological and physical, which have been reviewed elsewhere.[48] Restriction from sports is not benign and is associated with significant impact on quality of life and psychological distress. Among athletes diagnosed through screening, loss of athletic identity is a large component of distress, and those disqualified from sports show the highest level of distress.[49,50] For adolescents receiving defibrillators, restriction from sports can be the most life-altering and distressing aspect of the device, and athletic adults with HCM experience lasting psychological difficulty adjusting to exercise restriction.[51,52]

Recommendations for shared decision-making and clinical management determination

COR	LOE	Recommendations
1	C-LD	1. In athletes with arrhythmogenic conditions, determination of clinical treatment options should be made through shared decision-making, prioritizing preferences, values, and goals of the athlete.[53]
1	C-EO	2. In athletes with arrhythmogenic conditions, the fundamentals of shared decision-making should be grounded in core principles of knowledge, humility, respect and trust, teamwork with key stakeholders, and transparent communication.

Synopsis

The preferences and values of individual athletes are key factors when determining clinical management strategies. These values may differ from those of nonathletic patients and need to be elicited from shared risk discussions and as part of the shared decision-making process. Shared decision-making with patients is grounded in the core principles of knowledge, humility, respect and trust, and transparent communication with all parties involved, including 3rd party stakeholders that generally come with ascending levels of competitive sport.

Recommendation-specific supportive text

1. The clinical evaluation and treatment decisions for athletes are founded on shared principles and disclosed risks between the practitioner and patient. As such, shared decision-making is not just a core tenet that underlies the determination of sports eligibility for athletes with cardiovascular disease,[54,55] but it is also a core principle in considering all the various treatment options. In a shared decision-making paradigm, the clinician counsels the patient on the potential clinical management options. Then, in concert with the patient's personal preferences, morals, and values, treatment strategies are constructed. Shared decision-making is widely advocated throughout all medicine. Levine and Stray-Gundersen first introduced the concept of individual responsibility for athletes in 1994 in the context of determining sports eligibility.[56] In sports cardiology, the shift away from medical paternalism stemmed from several key factors that included the publication of data challenging assumptions of increased risk of sports for individuals with cardiovascular disease,[57,58] and the ethical imperative for patient-centered care as mandated by the Institute of Medicine, the American College of Cardiology, and others.[59] Moreover, other updated disease-specific guidelines, such as for HCM,[10] acknowledge ongoing clinical uncertainties and advocate for individualized shared decision-making. The lack of randomized controlled trials and robust data evaluating the safety of sports participation in various cardiovascular conditions emphasizes the need to include shared decision-making with the athlete in discussions involving their own medical care. Shared decision-making regarding exercise has been shown to decrease decisional conflict and decisional regret.[53] As described in more detail elsewhere, shared decision-making for return-to-play decision-making is consistent with legal precedents which have affirmed the "team physician" model.[55]
2. Medical treatment options for athletes diagnosed with arrhythmias are generally affected by a desire to return to play, return to regular vigorous exercise habits, or, for occupational athletes, an expedited return to a physically demanding occupation. As such, although evidence-based best shared decision-making practices with athletes remain uncertain,[60] inclusion of shared decision-making remains of paramount importance in the guidance and determination of treatment options and in ensuring equity of care for all athletes. The fundamentals of shared decision-making with

athletes are grounded in several core principles: knowledge of the disease/condition and implications of potential interventions, both positive and negative; humility; respect for the athlete's values, goals, and preferences; and trust. Clear, transparent communication regarding risk, including when risk is unknown, is critical. The physician should ensure that risk is understood by the athlete and family. Discussion of all treatment options with the athlete and other key parties throughout the shared decision-making process is critical.[55]

An additional and unique aspect of shared decision-making with athletes is the influence of key 3rd party stakeholders that come with ascending levels of competitive sport and in certain occupations. For all young athletes, schools, teams, and leagues are all involved in the decision to return to play. For higher-level athletes, elite collegiate and professional further stakeholders are involved (eg, athletic directors, general managers, owners, sponsors), and 3rd party influences play a key role in return-to-sports decision-making. Collaborative discussion with team physicians is paramount. However, the goal of the shared decision-making process between the practitioner and athlete and/or athlete's family is still a patient-centered focus, regardless of level of the athlete. The privacy of the athlete must also be maintained to the extent possible in communication with stakeholders. For tactical athletes, such as those in the military, governing bodies will determine eligibility.

Collaborative discussion among the athlete, family, treating physician, team physician, additional experts, and other institutional stakeholders (athletic departments and athletic trainers) is critical to balancing risk tolerances and institutional resources/responsibilities,[55] as shown in Figure 2. The shared decision-making model does not offer "clearance" (implying no risk) but rather outlines a decision-making process and documents understanding and acceptance of risk by athlete and physician. All counseling the athlete should also make clear their support for the athlete should they decide to not return to play. With clear communication and discussion and understanding of risk, athlete-centered outcomes can be achieved in most clinical management and, ultimately, return-to-play decisions.

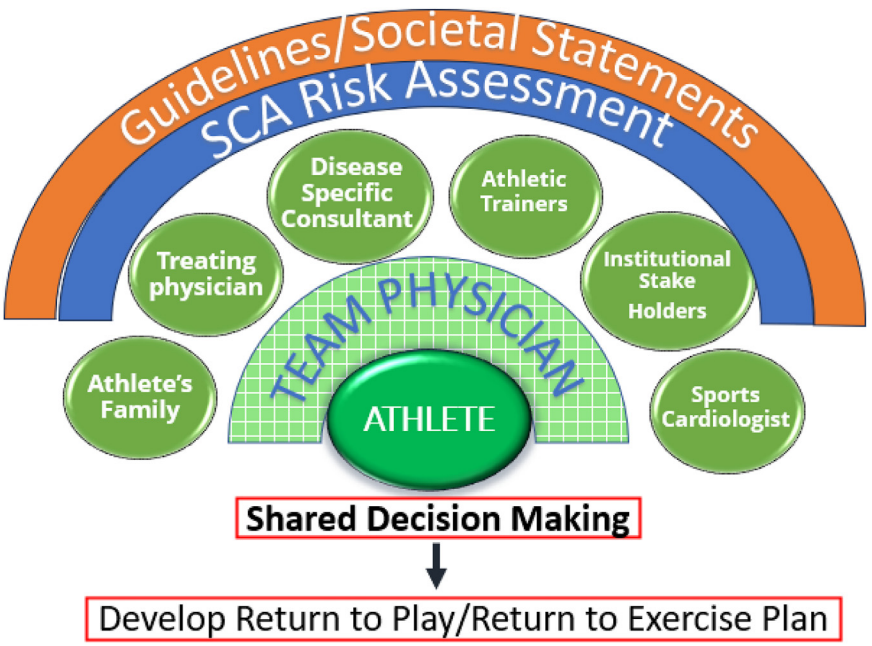

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Figure 2
Model for shared decision-making for athletes with cardiovascular disease. In the team-physician-led decision-making process, physicians incorporate shared decision-making, guided by respect for patients' goals and preferences, while integrating collaborative discussion among the athlete, family, treating physician, team physician, additional experts, and other institutional stakeholders (athletic departments and athletic trainers) in balancing risk-tolerances. SCA = sudden cardiac arrest. Reprinted with permission from Martinez et al.[55]

2.4 Emergency action planning for sudden cardiac arrest

Recommendations for emergency action planning

COR	LOE	Recommendations
1	B-NR	1. For athletes training or competing at schools, recreational facilities, or other athletic venues, an emergency action plan (EAP) should be in place to respond to acute medical and cardiac events to improve survival from SCA.[61–65]
1	B-NR	2. For athletes training or competing at schools, recreational facilities, or other athletic venues, steps for rapid and early cardiopulmonary resuscitation and defibrillation should be included in an EAP to improve survival from SCA.[61–65]
1	B-NR	3. For athletes competing at interscholastic levels or in other organized leagues, relevant governing bodies should put into place policies and direct resources toward increasing the effectiveness of EAPs.[61–65]
1	B-NR	4. For athletes training or competing at schools, recreational facilities, or other athletic venues, there should be medical support and infrastructure that enables all athletes and team-affiliated staff to learn cardiopulmonary resuscitation (CPR) and be familiar with automated emergency defibrillators (AEDs).[61–67]

Synopsis

Effective emergency action planning, inclusive of immediate SCA recognition and CPR initiation and rapid defibrillation, is critical in all competitive sports participation and must be in place in venues in which athletes practice and compete. Legislative efforts that support EAP efforts must remain a point of emphasis. Key components of effective EAPs are listed in Table 6.

Recommendation-specific supportive text

1. EAPs to facilitate recognition and response to cardiac arrest in athletic venues are considered best practice, as time to defibrillation is the most important factor influencing survival.[61–65,68,69] As most young individuals suffering SCA do not have a prior diagnosis, venues need to be prepared for SCA in any athlete. The EAP is a written document, and regularly rehearsed. Specific individuals, including physicians, athletic trainers, and coaches, who are regularly present when athletes are training or competing, should be trained in rapid assessment for potential SCA and certified in CPR. An emergency communication system should be in place integrating plans for early defibrillation and communication with emergency medical services (EMS), as well as logistics for transfer to the closest appropriate medical center after SCA resuscitation. EAPs should be individualized to each sport and venue.[70]
2. Early CPR and defibrillation has been demonstrated to save lives in the general population.[61–65] Time to defibrillation is a critical factor in survival of SCA. In venues where SCA may occur such as schools and venues for training and sporting events, there should be an accessible AED within 3 minutes.[71–73] Among athletes, survival from cardiac arrest is significantly higher if an AED is used.[69] Athletes at club-sponsored events have lower levels of resuscitation and survival compared with school-sponsored events.[61] AEDs should be placed near fields or courts where an arrest may occur.
3. Increasing the effectiveness of EAPs requires directed efforts and resources.[61–65] Ensuring EAPs are in place at the secondary school level generally requires statewide legislative policies. In addition, dedicated financial resources for schools are necessary to ensure standardized education and rehearsals of EAP processes, maintenance of EAP equipment, and regular updates of key EAP tenets.
4. As described in detail above, early CPR and early defibrillation save lives.[61–65] Training in CPR and AED use have been demonstrated to improve use of these.[66,67] In an analysis of the CARES (Cardiac Arrest Registry to Enhance Survival) dataset,[67] an EMS-based registry for out-of-hospital cardiac arrests, states with laws requiring CPR training have higher rates of CPR in out-of-hospital cardiac arrests. Survey data also show that active and more extensive training in CPR increases willingness to perform it.[66]

Table 6 Key components of emergency action plans

Components
✓ A written EAP for the recognition and treatment of SCA has been reviewed and rehearsed by key personnel at least annually.
✓ Those likely to be first responders in the event of SCA (teachers, administrators, coaches, strength and conditioning coaches, athletic trainers, team physicians, etc) have received training and are up to date in CPR and AED use.
✓ A communication system has been established for a rapid and coordinated response to cardiac arrest.
✓ AEDs are placed strategically to achieve < 3-minute retrieval time.
✓ EAPs are individualized to the sport (facility-based, running, water sports, etc).
✓ Signage indicating the location of AEDs is clear and visible.
✓ AEDs are accessible (never in locked cabinets or behind locked doors) and are regularly checked for proper battery charge and functional electrode pads.
✓ Emergency medical service entrance and exit to facilities is predetermined, accessible, and secure.
✓ Ideally, EAPs should be reviewed and practiced with local emergency medical services.

AED = automated external defibrillator; CPR = cardiopulmonary resuscitation; EAP = emergency action plan; SCA = sudden cardiac arrest.

Section 3 Sudden cardiac arrest in athletes

3.1 Epidemiology of sudden cardiac arrest and death in athletes

Sudden cardiac arrests (SCAs) are rare in the young athlete, with most series describing a rate of 1-2 per 100,000 person-years.[74,75] SCAs are more common as athletes age, predominantly due to the increased prevalence of coronary disease. Many states mandate AEDs and EAPs at schools, sporting events, and gymnasiums. Sporting activities may increase the odds of an arrest due to several factors, including direct effects of autonomic changes, increased heart rate, myocardial ischemia, and cardiac dilation.

SCA is an important cause of death in athletes of all ages. However, attempts to define both incidence and causes have been limited by methodological issues. Accurate estimation of incidence requires all cases of both SCA with survival and SCA with sudden cardiac death (SCD) (numerator) in a specific cohort to be identified. Many studies on SCA are retrospective, do not include cases with survival, have estimated or inexact cohorts (denominators), and do not have the necessary granularity to accurately represent risk among different populations. For example, risk of SCA varies drastically by sex; therefore, inclusion of both sexes in risk calculations underrepresents risk in males and overrepresents risk in females.[74,76–82] Risk also appears to differ based on sport and reported race.[79–84] Further, the specific causes of SCA change with age.[85–88] Hereditary and congenital conditions are the predominant causes of SCA in younger athletes, and acquired causes, specifically coronary artery disease, are the principal causes in Masters athletes.[85,88,89] Estimates of SCA that include wide age ranges with different primary etiologies give imprecise representations of risk. Finally, many studies examining SCA in athletes include only sports- or exercise-related SCA, which is a subset of all SCA. While some studies suggest that up to 90% of deaths in young athletes occur during exercise,[90] others suggest that number is closer to 50%.[79,82,91,92] A tool designed to decrease bias in reporting and conduction of studies of SCA in athletes has recently been developed and should improve the quality of these data.[93]

Incidence

Two recent systematic reviews and meta-analyses reported the rate of sudden cardiac arrest and death (SCA/D) in young athletes as 1.7-1.9 deaths per 100,000 person-years (1 death per 52,000-58,000 person-years).[74,75] They included a heterogeneous group of studies that included both sexes, had wide age ranges, and had both SCD occurring at any time and sports- or exercise-related SCD. Most did not include cases of SCA survivors. Incidence numbers must be interpreted in the context of the above limitations. Table 7 includes the incidence of SCA/D in athletes aged 14-26 years and includes studies assessed at low or intermediate risk of bias.[93,94] When multiple reports of the same database appeared, the most recent or comprehensive study was included in the table. The overall incidence of SCA/D in this population is generally reported as 1 death in 50,000 person-years in college-age athletes with slightly lower rates in high school athletes (likely an artifact of less coverage in media reports in the high school age range) with higher rates in males and certain sports. Table 8 includes the incidence of sports- and exercise-related SCA including information on how cases were identified (numerator) and how the population was defined (denominator). Rates of death in sports- and exercise-related SCA/D tend to be lower: around 1 death in 200,000 person-years. Many of these studies use population-based registries that accurately identify deaths; however, clearly defining and identifying athletes can be challenging. A more comprehensive list of all studies on the incidence of SCA/D in athletes that includes those with wider age ranges is in Table 9. This table highlights the wide range of estimates of incidence and the existence of groups that are potentially at much higher risk of SCA/D including males, Black athletes, and athletes playing men's basketball, men's soccer, and American football.

Commotio cordis (also discussed in Section 4) is SCA precipitated by a blunt, nonpenetrating blow to the anterior precordium at a specific point during the cardiac cycle in a structurally normal heart.[95] Commotio cordis is sometimes included in databases of SCA/D or sudden death but may be differentiated as a traumatic death, as there is no underlying cardiac condition. Commotio occurs most often in ball sports but can also occur with a blow to the chest in martial/fighting arts or other contact sports. In the Commotio Cordis Registry, there were 167 cases in athletes of all ages (68% died) over 42 years (1970-2012), or about 4 cases a year.[96] At a national cardiac pathology referral center in the United Kingdom, 6 cases of commotio cordis were reported in athletes over 28 years[92] and there were two cases of commotio cordis found over 20 years in National Collegiate Athletics Association (NCAA) athletes with an incidence of 1 in 4,553,258 athlete-years.[79,82]

The incidence of SCA/D in older or Masters athletes is equally difficult to ascertain. Studies vary by the definition of an athlete, and it is often difficult to clearly define the population (denominator) of older exercising or competitive athletes. Most information on the incidence of SCA in those aged > 35 years is exercise-related SCA/D and comes from data at long distance races and other events. In a 2012 study of 10.9 million runners, there were 59 arrests, for an incidence rate of 0.54 per 100,000 participants with a 29% survival rate.[97] A recent study of deaths during 46 long distance races with over 1,000,000 participants reported a rate of 2.33 per 100,000 runners with major cardiovascular events with a 90% resuscitation rate.[98] While it is well established that cardiorespiratory fitness lowers overall mortality and other adverse health outcomes,[99] even among the most fit, the immediate risk of SCA/D is increased during and shortly after physical exertion.[100,101]

Causes of SCA

As with incidence rates, there are limitations of the currently available studies that report on causes of SCA. The United States has no coordinated system for postmortem evaluation, instead relying on a patchwork of medical examiners and coroners with variable expertise and often limited by poor funding in the testing performed. Specialized referral centers exist in some parts of the United States and other parts of the world, but studies from those centers may reflect ascertainment

Table 7 Incidence of sudden cardiac arrest and death in athletes in low or intermediate risk of bias studies with sex, race, and sport rates*

Study	SCD or SCA/D?	Age range (years), no. of cases	Overall incidence of SCA/D	Male SCA/D incidence	Female SCA/D incidence	Black SCA/D incidence	White SCA/D incidence	Sex and sport-specific incidence	Risk of bias
High school age (~14-18 years)									
Toresdahl et al, 2014[78]	SCA/D	14-18, N=44	1:88,000	1:58,000	1:323,000				Low
Drezner et al, 2014[106]	SCA/D	14-18, N=13	1:71,000					Male, basketball 1:21,000	Intermediate
Malhotra et al, 2018[84]	SCD	15-17, N=8						Male, soccer 1:14,794	Low
Harmon et al, 2016[80]	SCA/D	14-18, N=104	1:67.000	1:45,000				Male, basketball 1:37,000	Intermediate
Peterson et al, 2021[81]	SCA/D	14-18, N=204	1:66,000	1:44,000	1:204,000			Male, ice hockey 1:24,000 Male, basketball 1:40,000 White, football 1:20,000	Low
College/university age (~18-25 years)									
Peterson et al, 2021[81]	SCA/D	18-24, N=39	1:51,000	1:35,000	1:123,000	Black, male 1:18,000	White, male 1:39,000	Black, male, basketball 1:4800 White, male, basketball 1:15,000 Black, football 1:28,000 White, football 1:20,000	Low
Petek et al, 2023[82]	SCD	17-26, N=143	1:64,000	1:43,000	1:165,000	1:27,000	1:75,000	Football 1:32,000 Male, basketball 1: 12,000 Male, cross-country 1:38,000 Male, Div 1, White, basketball 1:6000 Male, Div 1, Black, basketball 1:8000	Low

When multiple reports of the same database appeared, the most recent or comprehensive study was included in the table. Div 1 = National Collegiate Athletics Association Division 1; SCA = sudden cardiac arrest; SCA/D = sudden cardiac arrest and death; SCD = sudden cardiac death.
*Includes studies assessed at low or intermediate risk of bias.[94]

bias, with a larger proportion of undiagnosed or structurally normal cases being referred. Conversely, it has been suggested that less specialized centers are more likely to overdiagnose structural or myocardial disease.[92] Most studies reporting on the causes of SCA include only those athletes that died and therefore may be overrepresenting cardiac conditions that are more lethal; electrical abnormalities are rarely diagnosed after death because the heart is structurally normal. Those diagnosed with autopsy-negative sudden unexplained death (AN-SUD) represent a variety of pathologies including channelopathies such as long QT syndrome (LQTS), CPVT, Brugada syndrome, and conduction abnormalities like WPW. Genetic testing (molecular autopsy) has been shown to be helpful in these cases, with one study detecting pathogenic variants in 44% of young people with exertion-related sudden unexplained death.[102] In addition, our understanding of pathology is evolving. There is an increasing prevalence of left ventricular hypertrophy with or without fibrosis noted that may represent a cardiomyopathy not yet phenotypically expressed, or the fibrosis may be an underlying primary trigger for arrhythmia. Table 10 includes studies of the causes of SCA/D in young people and the relative contributions of each entity in the study. AN-SUD appears to be the most frequent cause of SCA/D in young athletes, followed by cardiomyopathies and coronary artery abnormalities. In athletes aged > 30–35 years, coronary artery disease is the most common cause of SCA/D, with the incidence increasing with age (Figure 3).

Exertional death in athletes with sickle cell trait (SCT) occurs suddenly and is included in some registries of sudden death.[103] Although cardiac conditions may coexist in athletes with SCT, the primary mechanism of exertional death in athletes with SCT is thought to be most likely explosive rhabdomyolysis.[104] When differentiating SCD from death due to SCT, one must consider the presence of underlying cardiac pathology and contextual information regarding the collapse.[105] Collapse in SCA/D includes loss of consciousness, but the athlete with SCT typically experiences a conscious collapse. Death due to SCT should not be included as a cardiac cause.

Summary

The incidence of SCA/D is higher in males, Black athletes, and certain sports such as basketball, American football, and soccer. The causes of SCA/D vary in younger and older athletes, with structural or electrical conditions the primary causes in younger athletes and coronary artery disease in older athletes (Figure 3). Future research should focus on inclusion of both SCA and SCD and should take into account age, sex, race, and sport.

Table 8 Incidence of sudden sports-related SCD in population-based studies

Study	Study design and population	Case identification (numerator)	Population definition (denominator)	SCD or all SCA/D?	Study years	Age range (years), no. of cases	Annual incidence
Holst et al, 2010[107]	Retrospective cohort; athletes and general population in Denmark	Review of death certificates, Cause of Death Registry, and National Patient Registry in Denmark	Interview data of people aged 16-35 years from the National Danish Health and Morbidity Study	SCD	2000-2006	12-35, N=15 12-35, N=428	*Athletes* 1:83,000 *General population* 1:27,000
Marijon et al, 2011[108]	Prospective cohort; general population in France	Data from emergency medical system	General population statistics, data from the Minister of Health and Sport to estimate young competitive athlete population	SCA/D	2005-2010	10-75, N=820 10-35, N=50	*General population* 1:217,000 *Young competitive athlete* 1:102,000 *Young noncompetitive athlete* 1:455,000
Risgaard et al, 2014[109]	Retrospective cohort; competitive and noncompetitive athletes in Denmark	Review of death certificates and the Danish National Patient Registry	Competitive and noncompetitive athlete populations in Denmark estimated based on survey data from the Danish National Institute of Public Health	SCD	2007-2009	12-35, N=44	*Competitive athlete* 1:213,000 *Noncompetitive athlete* 1:233,000
Bohm et al, 2016[110]	Prospective cohort; sports-related SCD in all persons in Germany	Voluntary reporting to German National Registry, web-based media search, regional institutes	Physical activity estimated from the German Health Update study and extrapolated to population data from the German Federal Statistical Office	SCD	2012-2014	10-79, N=144	*Sports participants* 1:1,200,000
Grani et al, 2016[111]	Retrospective; sports-related SCD in all persons in German-speaking Switzerland	Forensic reports	Physical activity estimated from survey on sports participation by the Swiss Federal Office of Sports	SCD	1999-2010	10-39, N=69	*Sports participants* Competitive 1:90,000 Recreational 1:192,000
Weizman et al, 2023[112]	Retrospective; sports-related SCD in 3 European registries	Review of death certificates and medical records	Population areas of the 3 registries	SCA/D	2006-2014	19-96, N=760; Female N=56, Male N=704	*People with SCA during sports* Female 1:5,263,000 Male 1:380,000

SCA = sudden cardiac arrest; SCA/D = sudden cardiac arrest and death; SCD = sudden cardiac death.

Table 9 Incidence of sudden cardiac arrest and death in athletes

Study	Study design and population	Case identification (numerator)	Population definition (denominator)	Sports-related SCD o all SCD?	SCD or all SCA/D?	Study years	Age range (years), no. of cases	Annual incidence	Risk of bias
Van Camp et al, 1996[113]	Retrospective cohort; high school and college athletes	National Center for Catastrophic Sports Injury Research and media reports	Data from NCAA, NFHS, NAIA, and NJCAA, added together with conversion factor (1.9 for high school and 1.2 for college) used to account for multisport athletes "based on discussions with representatives from the national organizations"	Sports-related	SCD	1983-1993	13-24, N=160	*College and high school* Overall 1:188,000 Male 1:134,000 Female 1:752,000 *High school* Overall 1:213,000 Male 1:152,000 Female 1:861,000 *College* Overall 1:94,000 Male 1:69,000 Female 1:356,000	High
Corrado et al, 2003[114]	Prospective cohort; athletes and nonathletes in the Veneto Region of Italy	Mandatory reporting of sudden death	Registered athletes in the Sports Medicine Database of the Veneto Region of Italy and the Italian Census Bureau	All	SCD	1979-1999	12-35, N=51 12-35, N=208	*Athletes* Overall 1:47,000 Male 1:41,000 Female 1:93,000 *Nonathletes* Overall 1:143,000	Low
Drezner et al, 2005[115]	Retrospective survey; college athletes	Survey of NCAA Division 1 institutions (244/326 responded)	Reported number of athletes	All	SCD		N=5	*College* Overall 1:67,000	Intermediate
Maron et al, 2009[116]	Retrospective cohort; amateur and competitive athletes	Registry for Sudden Death in Athletes	An estimated 10.7 million participants per year $\leq$ 39 years of age in all organized amateur and competitive sports	All	SCA/D	1980-2006	8-39, N=1046	*Athletes* 1:164,000	High
Drezner et al, 2009[63]	Cross-sectional survey; high school athletes	Survey of 1710 high schools with AEDs	Reported number of student athletes	All cases occurring on campus	SCA/D	2006-2007	14-17, N=14	*High school* 1:23,000 (SCA/D) 1:64,000 (SCD)	Intermediate
Steinvil et al, 2011[117]	Retrospective cohort; athletes in Israel	Retrospective review of 2 Israeli newspapers	Competitive athletes registered in the Israel Sport Authority in 2009; extrapolated this data for prior 24 years based on the growth of the Israeli population (age 10-40 years) from the Central Bureau of Statistics; allowed for a presumed doubling of the sporting population over 24 years	All	SCD	1985-2009	12-44, N=24	*Athletes* 1:38,000	High
Maron et al, 2013[118]	Retrospective cohort; Minnesota high school athletes	Registry for Sudden Death in Athletes	Minnesota State High School League statistics (estimated using conversion factor of 2.3 to account for multisport athletes)	All	SCD	1986-2011	12-18, N=13	*High school* Overall 1:150,000 Male 1:83,000 Female 0	Intermediate
Toresdahl et al, 2014[78]	Prospective observational; high school students and student athletes	2149 high schools monitored for SCA events on school campus	Reported number of students and student-athletes	All cases occurring on school campus	SCA/D	2009-2011	14-18, N=44	*Student athlete* Overall 1:88,000 Male 1:58,000 Female 1:323,000 *Student nonathlete* Overall 1:326,000 Male 1:286,000 Female 1:357,000	Low

(*continued*)

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Table 9 Continued

Study	Study design and population	Case identification (numerator)	Population definition (denominator)	Sports-related SCD o all SCD?	SCD or all SCA/D?	Study years	Age range (years), no. of cases	Annual incidence	Risk of bias
Drezner et al, 2014[106]	Retrospective cohort; Minnesota high school athletes	Public media reports	Minnesota State High School League statistics (sum of unduplicated athletes 2003-2004 through 2011-2012 school years)	All	SCA/D	2003-2012	14-18, N=13	*High school* Overall 1:71,000 Female 0 Male, basketball 1:21,000	Low
Harmon et al, 2015[79]	Retrospective cohort; college athletes	Parent Heart Watch database, NCAA Resolutions list, catastrophic insurance claims	Participation data from the NCAA	All	SCD	2003-2013	17-26, N=79	*College* Overall 1:53,000 Male 1:38,000 Female 1:122,000 Black 1:21,000 White 1:68,000 Football 1:36,000 Male, soccer 1:24,000 Male, black 1:16,000 Male, basketball 1:9,000 Male, black, basketball 1:5,300 Male, Div. 1 basketball 1:5,200	Low
Maron et al, 2016[119]	Retrospective cohort	Records of the Medical Examiner	Data from the Minnesota Department of Education, National Center for Education Statistics, and the Minnesota State High School League for Hennepin County, Minnesota	All	SCD	2000-2014	14-23, N=27	*Nonathlete* 1:39,000 *Athlete* 1:121,000	High
Harmon et al, 2016[80]	Retrospective cohort, United States high school athletes	Media reports	NFHS participation statistics	All	SCA/D	2007=2013	14-18, N=104	*High school* Overall 1:67,000 Male 1:45,000 Female 1:237,000 Male, basketball 1:37,000	Intermediate
Chatard et al, 2018[120]	Prospective, Pacific Island athletes who were screened	Prospectively followed	Defined cohort of 1450 athletes		SCD	2012-2015	10-40, N=3	*Pacific Island athletes* 1:2,416	High
Malhotra et al, 2018[84]	Prospective	Followed from time of screen to 2016	Defined cohort of 11,168 elite soccer athletes	All	SCD	1996-2016	15-17, N=8	*Elite male soccer athletes* 1:14,794	Low

Peterson et al, 2021[81]	Prospective	National Center for Catastrophic Sports Injury Research	Has defined cohort for high school and college athletes	All	SCA/D	2014-2018	14-18, N=204 18-24, N=39	*High school* Overall 1:66,000 Male 1:44,000 Female 1:204,000 Male, ice hockey 1:24,000 Male, basketball 1:40,000 *College* Overall 1:51,000 Male 1:35,000 Female 1:123,000 Black, male, basketball 1:4,800 White, male, basketball 1:15,000	Low
Petek et al, 2023[82]	Retrospective cohort study	National Collegiate Athletic Association resolutions list, Parent Heart Watch database and media reports, National Center for Catastrophic Sports Injury Research database, and insurance claims	Participation data from the NCAA	All	SCD	2002-2022	17-26 N=143	Overall 1:63,682 Male 1:43,348 Female 1:164,504 Football 1:32,000 Male, basketball 1:12,000 Male, cross-country 1:38,000 Male, Div 1, White, basketball 1:6,000 Male, Div 1, Black, basketball 1:8,000	Low

AED = automated emergency defibrillator; Div 1 = National Collegiate Athletics Association Division 1; NAIA = National Association of Intercollegiate Athletics; NCAA = National Collegiate Athletics Association; NFHS = National Federation of State High School Associations; NJCAA = National Junior College Athletic Association; SCA = sudden cardiac arrest; SCA/D = sudden cardiac arrest and death; SCD = sudden cardiac death.

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Table 10 Studies of the causes of sudden cardiac arrest and death in athletes and young people*

Study	Years of study	Athletes or young people	Methods	Autopsy	Country	Sports-related or all deaths	Age range (years)	Cases	HCM	Idiopathic LVH/ fibrosis	Coronary artery anomalies	ACM/ ARVC	DCM	AN-SUD**	CAD	Myocarditis related	Aortic dissection	Commotio cordis	Other
Corrado et al[121]	1979-1999	Athletes	Prospective, mandatory reporting, all deaths in Veneto region	Standard procedure at referral center	Italy	All	12-35	46	2%	0%	13%	26%	2%	2%	22%	11%	2%		17%
Maron et al[116]	1980-2006	Athletes	Retrospective, registry, media reports	Review of available autopsy	United States	All; includes SCA	8-39	1114	23%	5%	11%	3%	1%	35%	2%	4%	2%	6%	9%
Holst et al[107]	2000-2006	Young people	Retrospective, death certificates	Autopsy reports, hospital records	Denmark	Sports-related	12-35	14	0%	7%	7%	29%	0%	29%	14%	7%	0%		7%
Suarez-Mier et al[122]	1995-2010	Young people	SCD referred to National Institute of Forensic Sciences of Madrid	Standard procedure at referral center	Spain	Sports-related	9-35	81	10%	9%	6%	15%	0%	23%	14%	5%	0%		19%
Bohm et al[110]	2012-2014	Young People	Retrospective	Media reports, registry	Germany	Sports-related	10-34	29	7%	3%	10%	3%	3%	17%	21%	31%	0%		3%
Harmon et al[80]	2007-2013	Athletes	Retrospective, media reports	Autopsy reports	United States	All	14-18	50	14%	28%	8%	2%	0%	18%	6%	14%	0%		12%
Morentin et al[123]	2010-2017	Young people	Retrospective	Standard procedure at referral center	Spain	Sports-related	15-24	14	14%	21%	0%	36%	0%	0%	0%	21%	0%		7%
Thiene et al[124]	1980-2015	Athletes and young people	Prospective	Standard procedure at referral center	Italy	All	< 40	75	5%	0%	16%	27%	0%	11%	23%	4%	0%		15%
Wisten et al[125]	2000-2010	Young people	Retrospective	Death certificates, autopsy, and medical records	Sweden	Exercise-related SCD	<35	62	16%	10%	0%	13%	6%	24%	11%	11%	0%		8%
Egger et al[126]	2014-2018	Athletes	Media reports, registry	Autopsy reports and interviews	Many	SCA/D	<35	104	11%	9%	13%	4%	1%		14%	13%	0%	7%	42%
Peterson et al[81]	2014-2018	Athletes	Media reports, reports to NCCSIR	Autopsy reports	United States	SCA/D	11-29	209	21%	13%	12%	6%	3%	10%	2%	4%	3%		16%
Bohm et al[127]	2012-2019	Young people	EMS and web-based screening	Autopsy reports	Germany, France	SCA/D	18-35	25 with autopsy	8%	4%			4%	24%	20%	16%			24%
Finocchiaro et al[92]	1994-2022	Athletes	All cases SCD referred to CRY	Standard procedure	United Kingdom	All	18-35	128	3%	12%	9%	8%	3%	52%	1%	0%	0%	5%	7%
Petek et al[82]	2002-2022	Athletes	NCAA database	Autopsy reports	United States	All	18-26	118	13%	17%	8%	5%	2%	19%	6%	7%	4%	2%	13%
Total								**2002**	**17%**	**8%**	**10%**	**6%**	**2%**	**29%**	**6%**	**5%**	**1%**	**4%**	**12%**

ACM = arrhythmogenic cardiomyopathy; AN-SUD = autopsy-negative sudden unexplained death; ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; CRY = Cardiac Risk in the Young; DCM = dilated cardiomyopathy; EMS = emergency medical services; HCM = hypertrophic cardiomyopathy; LVH = left ventricular hypertrophy; NCAA = National Collegiate Athletics Association; NCCSIR = National Center for Catastrophic Sport Injury Research; SCA = sudden cardiac arrest; SCA/D = sudden cardiac arrest and death; SCD = sudden cardiac death.

*If studies used same/similar or subsets of database most recent or complete included.

**Reported Wolff-Parkinson-White and long QT syndrome included in AN-SUD.

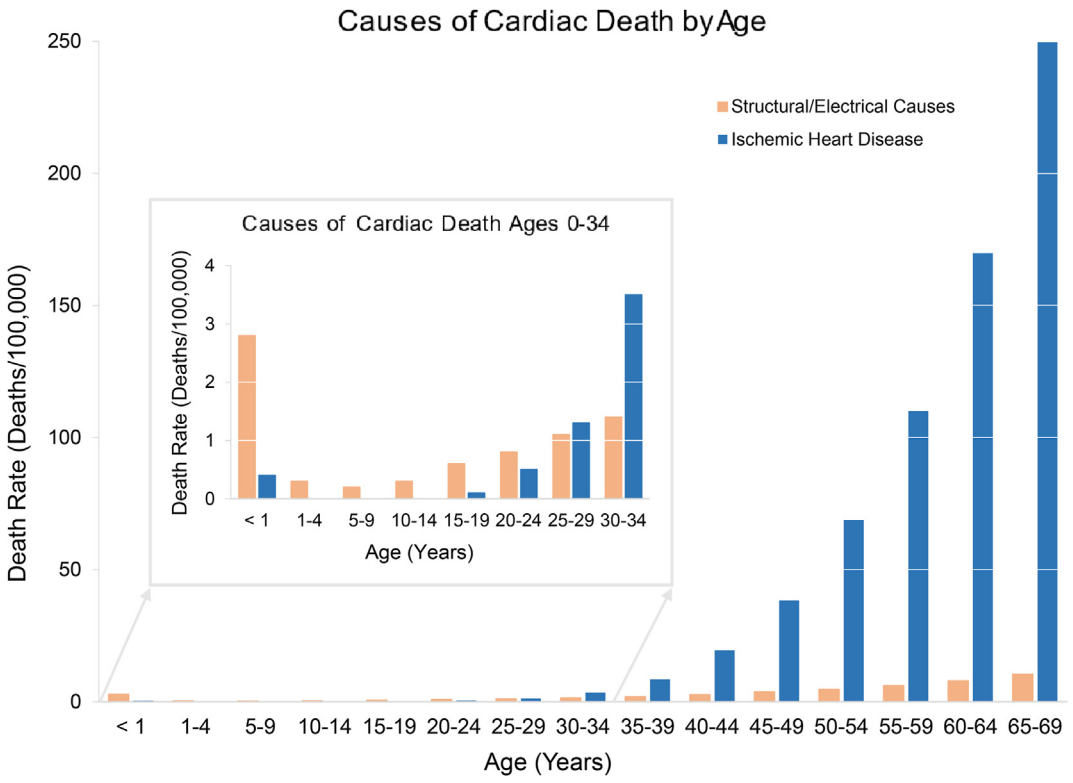

Figure 3

Coronary artery disease and other structural/electrical causes of cardiac death by age (1999-2020) from CDC Wonder.[88] ICD-10 codes for other structural/electrical causes include I40.0 (Infective myocarditis), I40.1 (Isolated myocarditis), I40.8 (Other acute myocarditis), I40.9 (Acute myocarditis, unspecified), I42.0 (Dilated cardiomyopathy), I42.1 (Obstructive hypertrophic cardiomyopathy), I42.2 (Other hypertrophic cardiomyopathy), I42.3 (Endomyocardial (eosinophilic) disease), I42.4 (Endocardial fibroelastosis), I42.5 (Other restrictive cardiomyopathy), I42.8 (Other cardiomyopathies), I42.9 (Cardiomyopathy, unspecified), I45.6 (Pre-excitation syndrome), I46.1 (Sudden cardiac death, so described), I46.9 (Cardiac arrest, unspecified), and Q24.5 (Malformation of coronary vessels).[88]

3.2 Sudden cardiac arrest prevention strategies

The prevention of SCA in athletes involves the detection of underlying cardiovascular conditions that may put a young athlete at risk including inherited cardiomyopathies, channelopathies, conduction abnormalities, and other congenital conditions. Approximately 1 in 300 young athletes will have an underlying cardiac condition that may predispose to SCA.[84,128–132] SCD is the leading medical cause of death in young athletes and the leading cause of death while exercising. In athletes aged > 30 to 35 years, coronary artery disease is the most likely entity to cause SCA.[85,88,89] Athletes aged > 25 years should have their coronary risk factors appropriately assessed.[133,134]

Recommendations for sudden cardiac arrest prevention strategies

COR	LOE	Recommendations
1	C-EO	1. In athletes, periodic preparticipation evaluations including screening for SCD risk is recommended.

Synopsis

There is no generally agreed upon screening strategy, with the AHA/ACC recommending a preparticipation cardiovascular screen using a 14-point history and physical examination,[135–137] other professional societies recommending the addition of a 12-lead ECG,[138] and still others advocating performance of an ECG just for competitive athletes or based on available resources.[70,139–141] A full discussion of the nuances of preparticipation cardiovascular screening in young athletes is beyond the scope of this document; however, if preparticipation screening is performed, it is recommended that the evaluation include screening for SCD risk.

Recommendation-specific supportive text

1. Guidance for cardiovascular screening strategies varies but includes, at a minimum, a history and physical examination.[142–146] The ECG added to the history and physical examination has been shown to be complementary and to outperform history and physical alone in detecting some conditions leading to SCD,[129] and evolution of ECG screening criteria has significantly decreased false positive rates.[147] The need for experienced readers, the potential financial costs, and the limited utility in specific conditions and populations (eg, anomalous coronary

arteries) are major limitations of ECG screening.[136,142–146] Many studies show increased diagnostic yield of ECG over history and physical exam.[90,129] ECG screening has not been clearly shown to improve mortality[90,117] in population studies. Whether history and physical improves mortality has not been tested. An in-depth review of the ECG screening debate is beyond the scope of this consensus document.

As described in Section 2, athletic training leads to electrical and structural adaptation reflected in the ECG, and if a 12-lead screening ECG is performed, it should be evaluated by an individual with expertise in interpretation of young athletes' ECGs using modern criteria.[148]

Athletes over 25 years of age need appropriate evaluation based on cardiovascular risk factors for atherosclerotic heart disease.[133,134]

Echocardiography as a screening modality has been suggested[149] and is in use in some professional leagues. Very small studies show an increased yield of cardiac diagnoses compared with history and physical examination alone.[150] This modality is not widely recommended as a screening test.[151,152]

3.3 Diagnostic evaluation of sudden cardiac arrest

As shown in Figure 4, the initial diagnostic workup of an athlete with cardiac arrest begins in the emergency department, ideally in consultation with a cardiologist/electrophysiologist, with history (including prior diagnosis, medications, and family medical history), vital signs, physical examination, laboratory work, and ECG.[11] Although toxicology screen is less often positive in sports-related SCA than non-sports-related,[153] occasionally a positive test leading to diagnosis is found. Important steps to understand the event include obtaining AED strips, questioning any witnesses, and reviewing any available video of the event. A history of left chest impact immediately prior to the event can point to a commotio cordis event after other entities are evaluated and excluded. Exercise-related events are more common in the arrhythmogenic right ventricular cardiomyopathy (ARVC) form of ACM and in CPVT.[27,154–156] Patients with anomalous coronary arteries nearly exclusively die secondary to exercise-induced triggering of arrhythmias,[82] quite likely mediated via acute coronary ischemia and resultant ventricular arrhythmias. If there is fever in the resuscitated athlete, hyperthermia-related collapse should be considered. Blood pressure could be low due to dehydration or high due to untreated hypertension. In the physical examination, stigmata of Marfan syndrome should be sought, as should murmurs that may be present in HCM and valvular disease.

The ECG is abnormal in approximately 90% of patients with HCM (left ventricular hypertrophy, T-wave abnormalities, pseudo–myocardial infarction patterns).[157–159] Similarly, patients with ARVC forms of ACM often, although not always, have abnormal ECGs with right bundle branch blocks, epsilon waves, and T-wave inversions in the anterior leads.[160] Patients with Brugada syndrome may present with the classic type I covered pattern with T wave abnormalities in V1-V2,[161,162] although the pattern can be intermittent. Patients with WPW have a delta wave, although the preexcitation can be subtle in young patients with fast AV nodes, particularly with left-sided pathways. For many conditions, the ECG abnormalities may be inconsistent, and it is important to obtain serial ECGs. Patients with early repolarization syndrome present with ventricular fibrillation (VF) and early repolarization on 12-lead ECGs.[163] Patients with genetic cardiomyopathies and myocarditis will often have nonspecific ST and T wave abnormalities. Patients with commotio cordis often have precordial ST elevation on their presenting ECG.[164] Short QT on ECG or short-coupled premature ventricular contractions (PVCs) on ECG or telemetry may also lead to a diagnosis.[165–167] ECGs are typically normal in CPVT, anomalous coronary arteries, and idiopathic VF. ECGs often show prolonged QTc in resuscitated patients,[168–170] so the initial ECGs should not automatically lead to a diagnosis of LQTS. In a small study with repeated ECGs, most had returned to normal within 3-5 days, although 20% remained abnormal at discharge.[168–170] After the initial workup in the emergency department, next steps could include the cardiac catheterization laboratory if there is ST elevation consistent with an acute infarct or targeted temperature management (TTM)[171] if they remain unconscious. Further workup can wait until TTM is completed. All patients should be admitted and placed on cardiac telemetry.

Hospitalization continues until the diagnostic workup is complete, and secondary preventive measures are implemented as directed by the diagnosis. Workup includes cardiac echocardiogram in all patients who survive a cardiac arrest. This can show abnormalities in athletes with HCM, ACM, and other cardiomyopathies as well as congenital heart disease (CHD). Bileaflet prolapse points to further evaluation for arrhythmic mitral valve prolapse with CMR.[172] Echocardiograms are typically normal in myocarditis, CPVT, LQTS, WPW (although some can have Ebstein anomaly of the tricuspid valve), early repolarization, and commotio cordis. Echocardiograms should be interpreted in the context of cardiac adaptation to exercise. If the diagnosis is still in doubt, patients should receive CMR, which is more sensitive in diagnosis of structural heart disease such as HCM, ACM, or arrhythmic mitral valve prolapse or noncompaction as well as myocarditis.

Exercise stress testing can be helpful in diagnosing CPVT if it induces ventricular arrhythmias. Burst exercise stress testing may increase sensitivity for CPVT.[41] Stress testing can also be helpful in LQTS, as the QT will often lengthen in recovery[173]; epinephrine can also bring out QT lengthening.[174] Ajmaline, procainamide, or flecainide administration can bring out the abnormalities of Brugada syndrome.

Genetic testing is increasingly useful in the workup of patients with cardiac arrest, especially in younger patients and those with idiopathic VF or idiopathic cardiomyopathies. A pathogenic or likely pathogenic variant can be found in approximately 65% of patients with HCM, 60% of patients with ARVC forms of ACM, 75-80% of patients with LQTS,[175]

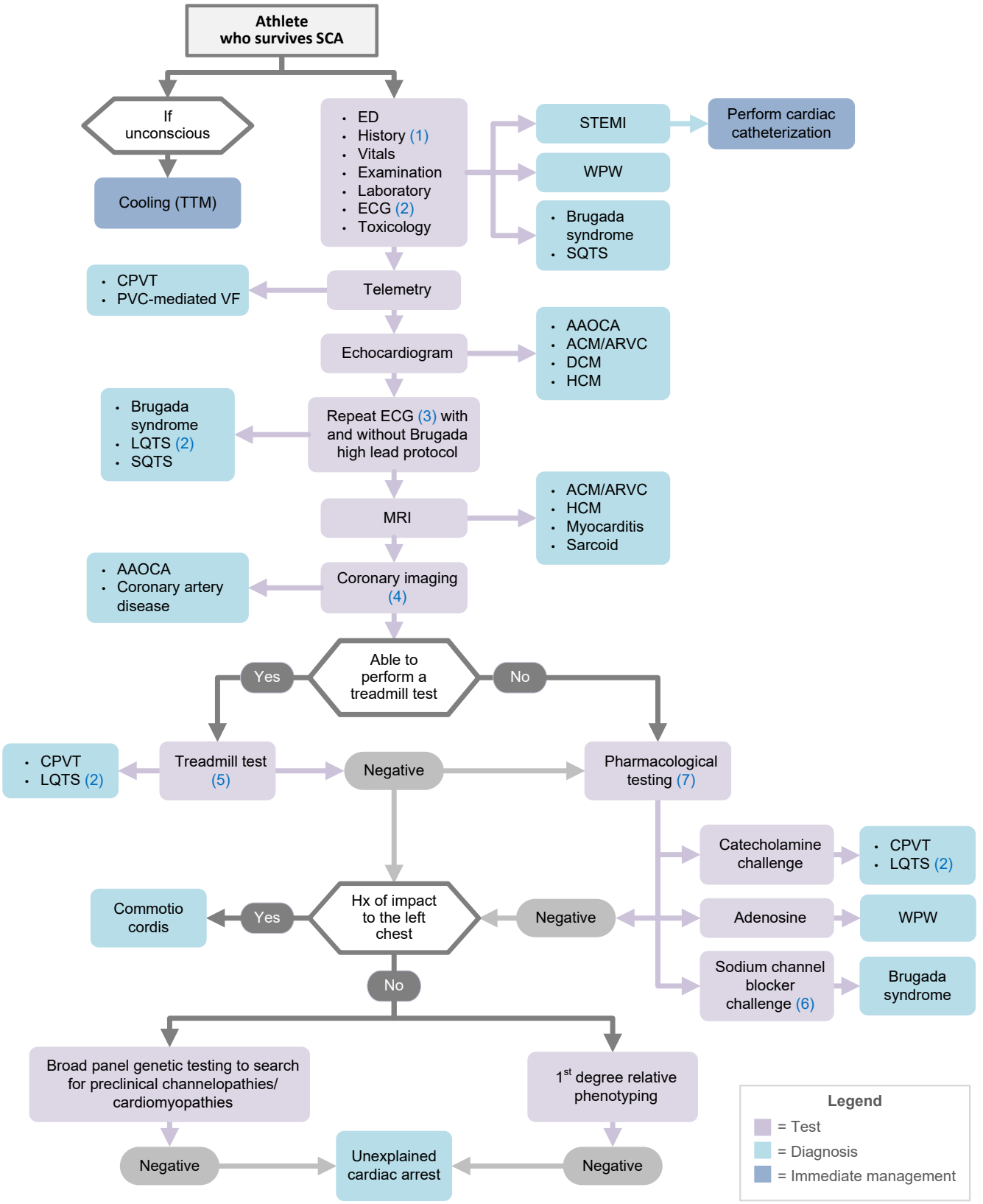

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Figure 4

Diagnostic algorithm for the evaluation of sudden cardiac arrest (SCA) in athletes. (1) History should include circumstances of arrest, past medical history, and family medical history. The circumstances of the arrest may drive the clinical investigation. For example, if the patient was struck in the chest, commotio cordis is high in the differential. If the arrest is associated with a high adrenergic state or noise/startle/emotion, then adrenergic testing should be performed. If the arrest happened while the patient is at rest or sleeping, then Brugada syndrome is in the differential, so high precordial lead ECG and sodium blocking pharmacologic testing should be performed.[162] Initial ECG may be diagnostic of Wolff-Parkinson-White (WPW) syndrome, Brugada syndrome, or ST elevation myocardial infarction (STEMI). (2) If the post-arrest electrocardiogram ECG (after 3-4 days) shows no persistent QT prolongation, use caution in interpreting catecholamine challenge or stress test results. (3)

60% of patients with CPVT, 20%-30% of patients with Brugada,[176] and up to 30% of patients with genetic cardiomyopathies. Variants in RYR2, suggestive of calcium deficiency release syndrome, which is electrically silent, may be seen on genetic testing.[177] Variants of uncertain significance are of little use.[178–183] ECGs of family members should be obtained. The finding of a pathogenic variant will also impact family member cascade screening (see also Section 7.3).

3.4 Emergency action plan for and immediate treatment of sudden cardiac arrest

Prompt recognition of SCA, implementation of an EAP, and time to defibrillation are crucial to survival.[68] In addition to anecdotal examples of public resuscitation of athletes, data have also demonstrated the importance of execution and importance of EAPs in saving the life of athletes. In sports-related SCA in high school athletes, survival rates of 85% are reported when the arrest was witnessed and an AED was used.[64,69] EAPs have been widely recommended at all levels of sport,[70–73,140,184] although recreational sporting leagues may be less prepared.[185] EAPs also save lives in all attending the events—coaches, referees, and spectators. The first step to an efficient emergency response is prompt recognition of SCA. Any athlete who is collapsed and nonresponsive should be assumed to have SCA until proven otherwise. Treatment of SCA involves activation of the EMS, early cardiopulmonary resuscitation, and early defibrillation. AEDs should be placed strategically to achieve < 3-minute retrieval time. AEDs need to be accessible in an unlocked cabinet. Coaches and other potential first responders should be trained in CPR and AED use, and plans should be practiced at least annually. A system that requires regular checks to ensure proper operation of AEDs is critical. Organizations should coordinate plans with local EMS providers. When EAPs are practiced and rehearsed regularly, SCA can be survived[64,69] (see Table 6). In addition to general planning for venues, when athletes with known cardiovascular disease are returning to play, individualized EAPs should also be in place.

Recommendations for emergency action plan for and immediate treatment of sudden cardiac arrest sudden cardiac arrest

COR	LOE	Recommendations
1	B-NR	1. In athletes who have collapsed and are nonresponsive, SCA should be presumed and acted upon until proven otherwise.[61,64]
1	C-EO	2. For athletes with a known SCA-predisposing heart condition who are returning to play, an individualized EAP should be in place.
2b	C-EO	3. In athletes with a known SCA-predisposing heart condition (and/or their families), obtaining and carrying a personal AED with their personal athletic equipment may be considered as part of their EAP made via shared decision-making.

Synopsis

EAPs and early access to defibrillation are crucial to the survival of SCA. An acutely collapsed, unconscious athlete should be assumed to be in SCA until proven otherwise. AEDs should be available at all venues with reasonable risk of SCA occurring including recreational venues. Athletes with known cardiac conditions that may predispose them to SCA should have individualized EAPs with consideration for a personal AED.

Recommendation-specific supportive text

1. Because prompt resuscitation efforts improve survival in athletes,[61–65] immediate recognition is critical to implement life-saving interventions. Collapsed athletes often have tonic-clonic movements of arms and limbs, which can be mistaken for a seizure or other noncardiac cause of collapse, critically delaying treatment.[63,64] Likewise, agonal gasping can be mistaken for respirations and assessment of the presence or absence of pulse can be difficult in emergency situations,

ECGs early after an arrest will often have long corrected QT intervals (QTc) as well as other transient abnormalities that include transient QT prolongation, transient T-wave inversions, or other repolarization abnormalities. (4) For the younger patient without risk factors for coronary artery disease, a computed tomography coronary angiogram protocol would identify those with anomalous coronary arteries. For those more likely to have coronary disease, a cardiac catheterization may be used. (5) In the evaluation of CPVT, a burst exercise stress test is more likely to bring on the arrhythmias of CPVT.[41] (6) Specific sodium channel blocker tests may include ajmaline, procainamide, and/or flecainide. Ajmaline has more false-positive test results. (7) Pharmacological testing based on clinical suspicion. AAOCA = anomalous aortic origin of coronary arteries; ACM = arrhythmogenic cardiomyopathy; ARVC = arrhythmogenic right ventricular cardiomyopathy; CPVT = catecholaminergic polymorphic ventricular tachycardia; DCM = dilated cardiomyopathy, Dx = diagnosis; ED = emergency department; HCM = hypertrophic cardiomyopathy; Hx = history; LQTS = long QT syndrome; MRI = magnetic resonance imaging; PVC = premature ventricular contraction; TTM = targeted temperature management.

especially for lay responders. Therefore, any athlete with acute collapse who is unconscious should be assumed to have suffered a cardiac arrest and AED pads applied. Application of an AED is not dangerous, and a shock will not be discharged in the absence of an abnormal shockable rhythm. Delay in CPR due to efforts to prevent "tongue swallowing" and clear the airway have been described—this is not necessary and CPR should not be delayed.[186]

2. Athletes with a known condition predisposing for SCA or those returning to sport after SCA should have an individualized EAP that addresses ready access to an AED and any factors (equipment, location, potential responders) that may complicate resuscitation. The plan should be coordinated by medical staff and reviewed and practiced with potential responders.[71–73] AEDs should be placed near fields or courts where an arrest may occur. Sports that practice or compete outside a defined venue such as cross-country should consider how EMS will be activated and accessed. An EAP in this case may include running with a partner, carrying a phone, and awareness of location for directing EMS. EAPs in unique environments such as swimming facilities should be reviewed and practiced. Those with SCA should be removed from the pool, but most AEDs are self-grounded and safe to use on wet athletes, in wet environments (such as pool decks or on snow and ice), or on metal grates. Rowing provides particular challenges including extraction from a shell or boat that should be considered and practiced annually.[187] Equipment can present barriers to resuscitation. EAP rehearsals in sports with pads or other protective equipment should include instruction and practice on how to remove pads and quickly access the chest for compressions and electrode pad application.
3. A personal AED may benefit athletes with a known SCA-predisposing heart condition,[188] whose risk may not warrant an implantable device but is not zero. A personal AED has been recommended by PACES, based on the known imprecision of risk stratification, non-risk-free nature of the ICD, particularly in children, and effectiveness of AEDs.[188] The risks and benefits, including psychological burden, as well as the importance of upkeep of the AED, need to be discussed with the patient and family as part of a shared decision-making process.[54] Avoidance of economic disparities requires coverage of personal AEDs when recommended.

3.5 Sudden cardiac arrest treatment and implantable cardioverter-defibrillator management in athletes

The treatment of an athlete who has had a SCA has many similarities to that for nonathletes, but there are some important differences in that management decisions take into consideration the impact of treatment (eg, choice of medication or type of ICD) on sports performance. Also, return to play after a cardiac arrest requires confirmation that the underlying arrhythmic vulnerability has been identified and treated appropriately.

Recommendations for sudden cardiac arrest treatment and implantable cardioverter-defibrillator management in athletes

COR	LOE	Recommendations
1	C-EO	1. In athletes who have experienced SCA, a comprehensive evaluation and shared decision-making discussion of potential risks of sports participation with an expert provider is recommended.
1	C-EO	2. In athletes who have experienced SCA, further evaluation including addressing psychological readiness should be completed prior to return to play.
1	A	3. In athletes who have experienced SCA, an ICD should be implanted as indicated based on underlying disease entity.[189–191]
2a	B-NR	4. In athletes with an ICD, return to play is reasonable, in the context of shared decision-making that includes underlying disease entity, clinical characteristics, and sport type.[192–194]
2a	B-NR	5. In athletes who have experienced SCA and are undergoing ICD implant, it is reasonable to consider sport type and disease entity in the decision regarding ICD type and location.[192,195]
2a	C-EO	6. For athletes undergoing ICD implantation who will be returning to play, a waiting period of 4-6 weeks after a new implant or 2 weeks after generator replacement is reasonable.
1	B-NR	7. In athletes who have experienced SCA and have an ICD, ICD detection criteria should be programmed to long duration and a high rate cutoff to prevent unnecessary shocks.[192,193,196,197]
1	C-EO	8. In athletes who have experienced SCA, exercise stress testing at maximum effort should be performed to measure heart rate, assess for arrhythmias during exertion, and confirm appropriate sensing by the ICD (if applicable) prior to return to vigorous exercise.
1	C-EO	9. In athletes who have experienced SCA, an individualized EAP should be in place prior to return to play.
1	A	10. In athletes who have experienced SCA, regular EP follow-up is recommended, including remote monitoring of ICDs (if applicable).[198–200]
1	C-EO	11. In athletes with an ICD who experience an ICD shock, an evaluation of the cause, treatment of the underlying etiology, and confirmation of appropriate device function should be done prior to return to play.

Synopsis

As summarized in the recommendation algorithm in Figure 5, return to play requires shared decision-making, individual EAPs, and appropriate follow-up and care with a cardiologist with experience in athletes and ICDs. Physical and psychological readiness need to be confirmed. Decision-making around treatment after SCA is based on the underlying disease. Patients with most of the underlying diseases causing SCA benefit from medical therapy, ablation, and/or an ICD. Patients with LQTS respond well to beta blockers, and beta blockers are likely beneficial for patients with CPVT as well. Patients with genetic cardiomyopathies and a depressed ejection fraction are generally treated with guideline-directed medical therapy and an ICD. For those receiving an ICD, device type and programming should be personalized to the athlete. Patients with WPW who survived a cardiac arrest usually should undergo ablation (see Section 9). Efficacy of treatment, whether medical, procedural, or device-based, should be confirmed by exercise stress testing prior to return to play, as should an individual EAP via discussion with stakeholders.

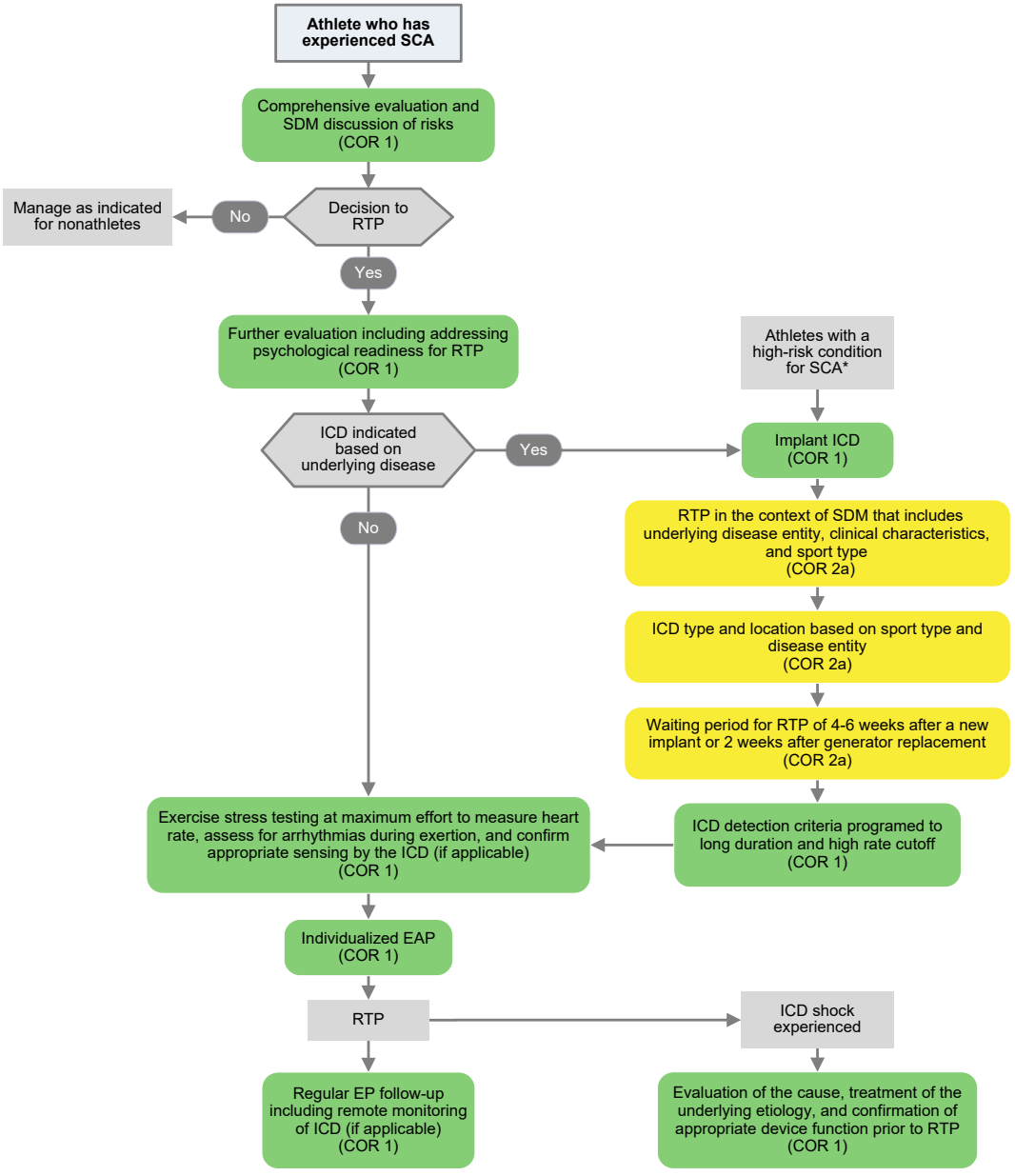

Figure 5

Algorithm for the management of sudden cardiac arrest (SCA) in athletes. Colors correspond to the class of recommendation (COR) in Table 1. *See Section 7. EAP = emergency action plan; EP = electrophysiology; ICD = implantable cardioverter-defibrillator; RTP = return to play; SDM = shared decision-making.

Recommendation-specific supportive text

1. Comprehensive evaluation to fully understand potential risks and benefits of return to play with a cardiologist who is not only an expert in the disease entity but also knowledgeable regarding the demands of sports settings is important.[54–56,201,202] The risk of recurrent SCA, underlying heart disease, type of sport, and wishes of the athlete are all important considerations. In addition, other stakeholders such as team physicians, parents, coaches, and institutions should be involved in the decision-making process.
2. Any athlete who has had SCA should have a full and complete evaluation, as discussed in detail in Section 3.3 and Figure 4, to determine causation, if possible, and assess risk before return to play.[11] This workup should also include an assessment for psychological readiness. Survivors of SCA in general have high rates of subsequent psychological distress, including depression, anxiety, and post-traumatic stress.[203] Return to play even after less life-altering events can be psychologically difficult for athletes.[47] Cognitive, emotional, and behavioral responses to injury, such as resilience and optimism, vary in athletes, and these factors have been demonstrated to determine outcomes in athletes returning to sport after injury. Building confidence, providing support, and fostering autonomy have all been associated with improved outcomes with return to play after injury. How psychological factors impact return to sport after a cardiac event has not been studied, but these likely have a similar impact. Specific instruments to assess readiness are undergoing investigation in the sports medicine community.[204] Athletes returning to play after SCA need attention to both illness-related and sports-related emotional needs.
3. Medical decisions around treatment of the athlete surviving SCA, including device implantation, are based on the underlying disease and should be directed by an expert in that disease entity. As shown in Figure 6, most conditions leading to SCA in athletes are treated with an ICD, with the exception of those with reversible causes such as a ST elevation myocardial infarction, ablated WPW syndrome, commotio cordis, or surgically corrected anomalous coronary arteries.[8,205] Other electrical disorders can occasionally be treated medically or with ablation, without a device, under expert care. Decisions regarding ICDs in CPVT require an electrophysiologist experienced with this entity, as ICD shocks can be arrhythmogenic and there are some data suggesting medical therapies and/or sympathetic denervation are effective, as detailed in Section 7.1.[206] Other than these exceptions, randomized controlled trials in the general population have demonstrated that survivors of a ventricular tachycardia or VF SCA have improved long-term survival when randomized to an ICD compared with medical therapy.[189–191] These trials excluded those thought to have a reversible cause such as myocardial infarction, severe potassium disorders, commotio cordis, etc.
4. Several retrospective and prospective observational studies have demonstrated lack of high risk for adverse events in athletes continuing to participate with an ICD. The ICD sports registry[192,193] enrolled 440 athletes participating in competitive (N=393) or dangerous (N=47) sports, including 77 engaged in varsity/junior varsity/traveling team competition, and followed them for a median of 44 months. There were no tachyarrhythmic deaths or resuscitated SCAs during sports participation and no injuries related to arrhythmias during sports. Appropriate and inappropriate shocks occurred. More athletes experienced shocks during physical activity than rest, but there was no difference between competition/training and other physical activity. Most ventricular arrhythmias occurring during sports were converted

Reversible cause – ICD not indicated	Consider other therapeutic options	Situational dependent factors	Irreversible cause – ICD indicated
• Commotio cordis • WPW, treated with ablation	• CPVT • LQTS type 1	• Anomalous coronary, surgically repaired • STEMI • Myocarditis	• Cardiomyopathy – ACM, HCM, DCM • LQTS, non type I • Brugada syndrome • Unexplained cardiac arrest • Arrhythmogenic bileaflet mitral valve prolapse

Figure 6

Treatment of sudden cardiac arrest based on disease entity. ACM = arrhythmogenic cardiomyopathy; CPVT = catecholaminergic polymorphic ventricular tachycardia; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter-defibrillator; LQTS = long QT syndrome; WPW = Wolff-Parkinson-White.

with the first programmed shock, with multiple shocks occurring rarely.[192,193] A recent retrospective series of elite athletes included 8 with ICDs, with similar findings.[194]

5. For some sports, the subcutaneous implantable cardioverter-defibrillator may have advantages over the transvenous implantable cardioverter-defibrillator, although more research is needed. The main issue with transvenous ICDs is lead durability. In theory, sports involving significant arm/shoulder movement, such as rowing, swimming, or weightlifting, may create wear on the lead insulation and connections, potentially causing inappropriate shocks. The subcutaneous ICD lead is placed subcutaneously and outside the thorax, which is a potential advantage for athletes in sports with repetitive arm use. However, the lead being outside the thorax may be a disadvantage for athletes in contact or ball-based sports. A secondary analysis of the ICD Sports Registry data[207] found that sports with repetitive arm use and contact sports were not associated with lead malfunction. However, the sample size and follow-up time in this analysis were limited, and more research is needed to accurately assess this risk. To date, no studies have been published on the experience of athletes with subcutaneous ICDs, but research is ongoing. If choosing a subcutaneous ICD, sensing testing should be performed in different body positions. The newly approved extravascular ICD may offer other benefits, as the lead is implanted substernally.[208] Figure 7 shows available ICD systems with advantages and disadvantages.

When choosing a transvenous ICD for an athlete, other factors should be considered. Dual-chamber devices offer the ability to pace the atrium, but this is rarely necessary for athletes. Better discrimination of supraventricular arrhythmias has not been proven in clinical trials.[209] In addition, dual-chamber devices have a higher rate of lead complications.[195] Therefore, unless atrial pacing is needed, athletes should receive a single-chamber device. For athletes participating in collision sports, who are not represented in the ICD Sports Registry, it is unknown whether system damage might be higher. One small series has described a minimally invasive epicardial device with the generator placed posterior to the

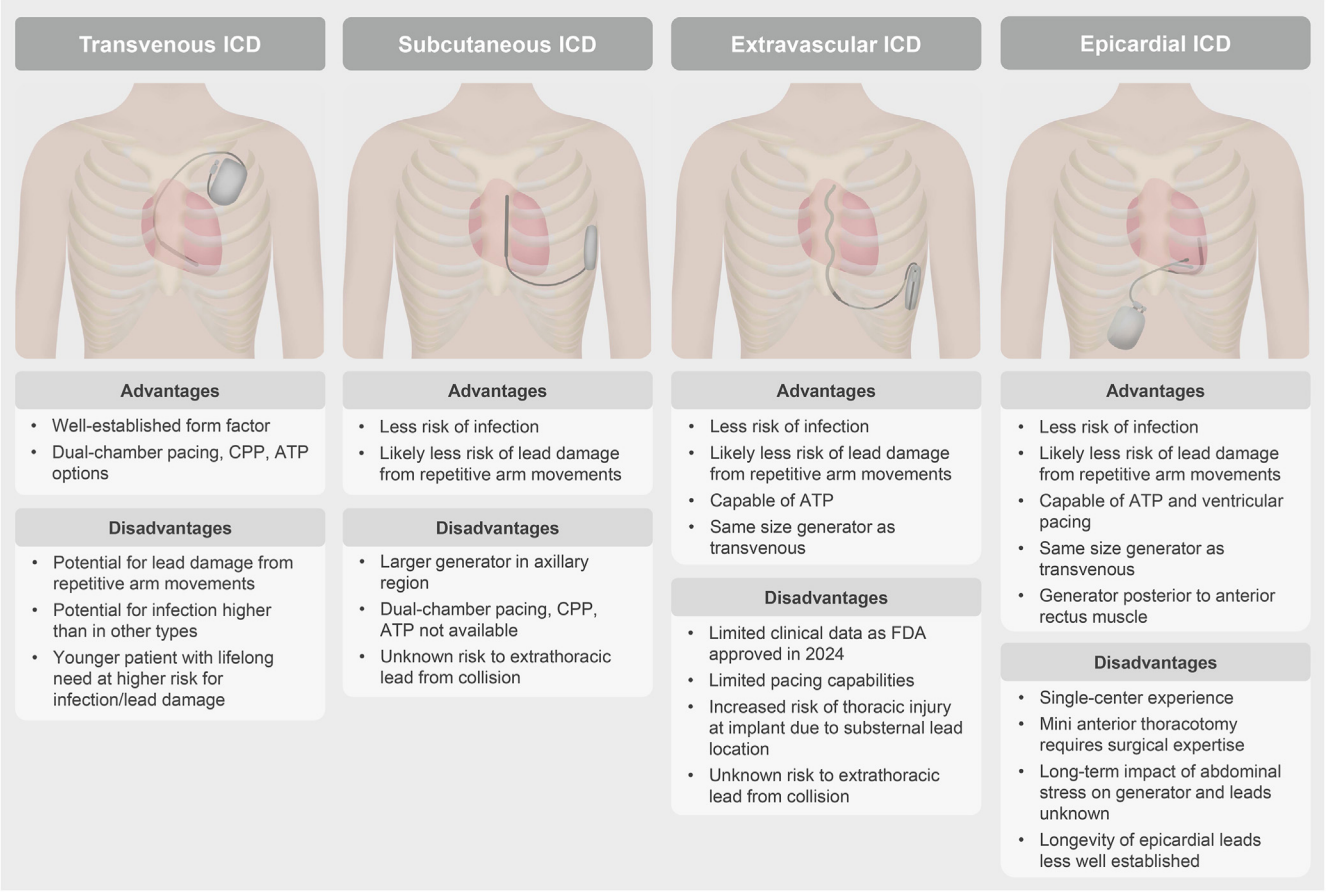

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Figure 7
Athlete-specific advantages and disadvantages of 4 different options for ICD form factors. ATP = antitachycardia pacing; CPP = cardiac physiologic pacing; FDA = Food and Drug Administration; ICD = implantable cardioverter defibrillator.

anterior rectus muscle in the subcostal region. This system can be considered in high-collision-sport athletes, although data are not yet available in athletes.[210]

6. Avoidance of reaching or lifting for 4-6 weeks after implantation of transvenous leads has been traditionally recommended to avoid lead dislodgment before lead endothelization is complete. Several small studies of resistive range-of-motion exercise[211] and early removal of arm restriction[212,213] have not shown arm/shoulder movement to increase dislodgment or other complications, and an ongoing randomized trial of lenient versus strict arm restriction after device implant is ongoing.[214] However, at this time, as randomized data are not available and the published small studies did not investigate vigorous exercise/arm motion in athletes, continuation of the traditional advisory is recommended. Two weeks should be allowed for healing of the skin incision.
7. Young athletes can achieve very rapid heart rates (in general 220 – age). In the nonathlete, programming ventricular tachycardia detection rates > 200 bpm has been shown to reduce inappropriate shocks and increase longevity.[196] In the athlete, data have shown that high rate cutoffs and long duration programming decreased both appropriate and inappropriate shocks with no increase in syncope.[197]
8. Exercise stress testing that recreates the demands of an athlete's sport as closely as possible including maximal effort should be performed prior to return to sport to evaluate treatment efficacy and ICD programming. Protocols with exercise intensity targeting nonathletes may not be sufficient to recreate the training demands of an athlete. Stress testing of athletes is addressed in detail in Section 2.3
9. EAPs that take into account the needs of the specific athlete, their sport, and their setting are important to consider and should be coordinated by medical staff.[73,215–217] These action plans could include a personal AED that is with the athlete at all times, particularly if ICD was not indicated, knowledge of the athlete's condition by the coach and medical staff, and surrounding personnel trained in CPR and AED use. In addition, special considerations related to settings where the athlete practices or competes (ie, outside of an arena or gymnasium) should be understood such as water sports (eg, swimming, rowing, canoeing) or sports in outdoor settings (cross-country or winter sports), including how to contact and provide timely access to an athlete by EMS. Individual EAPs are discussed in more detail in Section 3.4
10. Most athletes with SCA have an underlying arrhythmia etiology requiring long-term follow-up, and most will have an ICD. Because athletes, particularly in college, may live far from their primary home, and from their primary EP team, maintaining continuity of care requires a purposeful care plan. Although there are no specific data in athletes, remote monitoring of ICDs improves outcomes and is recommended for all ICD patients.[218] Effective remote monitoring requires a defined physician or physician group that is responsible for this remote monitoring.[219] The primary EP team could include physicians where the athletes lives or where they go to school, but the athlete should at least have knowledge of a local care team with experience in ICDs. The monitor should follow the patient to the school and back during long breaks. The covering personnel should have the contact information of the patient.
11. Patients with ICD shocks and/or antitachycardia pacing therapy require an evaluation before returning to play to determine whether the therapy is appropriate or inappropriate. For inappropriate shocks, the leads and system should be evaluated and revised if indicated. If oversensing is the etiology, programming should be optimized. ICD therapy is increased by activity.[192,220] For athletes with appropriate shocks, ventricular arrhythmias should be treated as indicated with medications and/or ablation, and treatment efficacy should be confirmed prior to return to play.

Section 4 Commotio cordis

Commotio cordis—sudden death with relatively innocent chest wall impact—was described in the 19th century,[221] but only in the last 15 years has it become more widely known. Current estimates of incidence range from 10 to 20 events a year in the United States.[95,222] In some series, commotio cordis was the second leading cause of sudden death in young athletes in the United States when it was included as a cause of athletic deaths,[223] although more recent data from the NCAA describe just 2 of the 127 SCAs over 20 years as being due to commotio cordis.[82] Commotio cordis is also increasingly described in European nations.[224,225] Commotio cordis has also been described in nearly every sport, and even in nonsport activity due to collision with body parts such as elbows, fists, and knees.

Based on human and experimental data, commotio cordis is likely due to immediate VF triggered by chest wall impact. A necessary confluence of patient characteristics (age, chest wall compliance, location of impact) and impact object variables (timing, hardness, shape) is what underlies the relatively rarity of commotio cordis.[226,227] Susceptible patients are young and typically male. There may also be a genetic predisposition or protection from commotio cordis.[228]

Successful resuscitation cases, originally thought rare in commotio cordis, are now routinely reported, likely due to increased recognition that VF is the cause of sudden collapse after chest wall impact and the increased prevalence of AEDs and knowledge of CPR in the community (see Table 6).[229,230] Prompt recognition and early defibrillation are paramount. The critical importance of emergency action planning for sporting venues is described in Section 2.4.

4.1 Commotio cordis prevention and diagnosis

Recommendations for commotio cordis prevention		
COR	LOE	Recommendations
1	C-EO	1. In athletes who have experienced SCA with a history of blow to the chest, underlying heart disease should be excluded prior to diagnosing commotio cordis.
1	C-EO	2. For athletes participating in sports for which chest protectors are used, chest protectors should meet sports- and position-appropriate standards.
2a	C-EO	3. In athletes under age 13 years, the use of age-appropriate safety baseballs is reasonable to reduce the risk of commotio cordis.

Synopsis

Athletes suffering SCA after a blow to the chest need full evaluation to exclude the possibility of underlying heart disease prior to making a diagnosis of commotio cordis. The risk of commotio cordis can likely be decreased. Coaching and rules should be amended to not allow blocking the ball with the chest in sports. Chest protectors were present in one-third of the individuals who suffered commotio cordis during competitive sports.[231,232] In some sports such as hockey, the arms were lifted, pulling the chest protector upward and thus exposing the chest.[232] However, in other sports such as lacrosse and baseball, the ball struck the chest protector, which was directly over the heart, and commotio cordis still resulted. Age-appropriate safety (ie, softer) baseballs will decrease the risk of VF with chest wall impact.[233]

Recommendation-specific supportive text

1. Commotio cordis is a rare cause of SCA in athletes. Athletes suffering SCA after a blow to the chest need full evaluation to exclude the possibility of underlying heart disease prior to making that diagnosis. Commotio cordis stems from a rare external event—a blow to the chest timed in milliseconds to the cardiac cycle—and it does not carry risk of recurrence. It is critical to exclude other potential etiologies that may require secondary prevention of SCA with an ICD and/or arrhythmia suppression.
2. To facilitate the development of effective chest protectors and ensure their quality, based on data from the experimental model[233] and other data, a mechanical surrogate to assess the ability of chest wall protectors to decrease the risk of commotio cordis was developed.[234] This has been licensed to the National Operating Committee on Standards for Athletic Equipment (NOCSAE)[235] and is in use by such organizations as the National Federation of State High School Associations (NFHS),[236] the NCAA Baseball Rules Committee,[237] and USA Lacrosse.[238] Similar standardization should be implemented worldwide.
3. A reduction in the risk of commotio cordis is likely possible with age-appropriate safety baseballs. Commotio cordis is rarely seen with safety baseballs, and these balls have been tested in an experimental model and were found to lower the risk of VF with ball impact.[233] Similar modifications have not been attempted in other sports.

Section 5 Symptoms of arrhythmias in athletes

5.1 Syncope in athletes

Syncope is a transient loss of consciousness, associated with the inability to support postural tone, with rapid and spontaneous recovery, secondary to diffuse cerebral hypoperfusion.[239] This clinical symptom, with a spectrum of underlying mechanisms and etiologies, can be classified as cardiogenic, neurally mediated, or orthostatic syncope. Neurally mediated syncope occurs when neural reflexes modify heart and blood pressure inappropriately, which can occur in response to multiple vagal stimuli including strong emotion, micturition, defecation, swallowing, and coughing. Orthostatic syncope occurs with a loss of vascular tone resulting in cerebral hypoperfusion. In athletes this most often occurs after the abrupt cessation of strenuous exercise and pooling of blood in the extremities. Loss of consciousness in athletes can also be related to nonsyncopal causes including head trauma, heat illness, hyponatremia, and SCT.[240]

Loss of consciousness in both athletes and nonathletes can also be due to systemic causes such as diabetes, seizures, or intoxication or psychogenic causes, which should be included in the differential diagnosis.

Nontraumatic loss of consciousness while exercising needs to be considered cardiac until proven otherwise. As described in detail below, only a small fraction of those with exertional syncope will ultimately be diagnosed with cardiac disease but an appropriate workup is essential, as these entities can be life-threatening. Exertional syncope should be investigated to rule out cardiac substrates including cardiomyopathies such as HCM or ACM anomalous coronary artery origin, or electrical abnormalities leading to ventricular arrhythmia such as LQTS or CPVT, all of which are associated with sudden death.[240] An algorithm for the approach to syncope in the athlete is shown in Figure 8.

5.1.1 Etiologies (cardiac/electrophysiology differential)

There are many underlying cardiac causes, either structural or electrical, that can be responsible for syncope in athletes. The

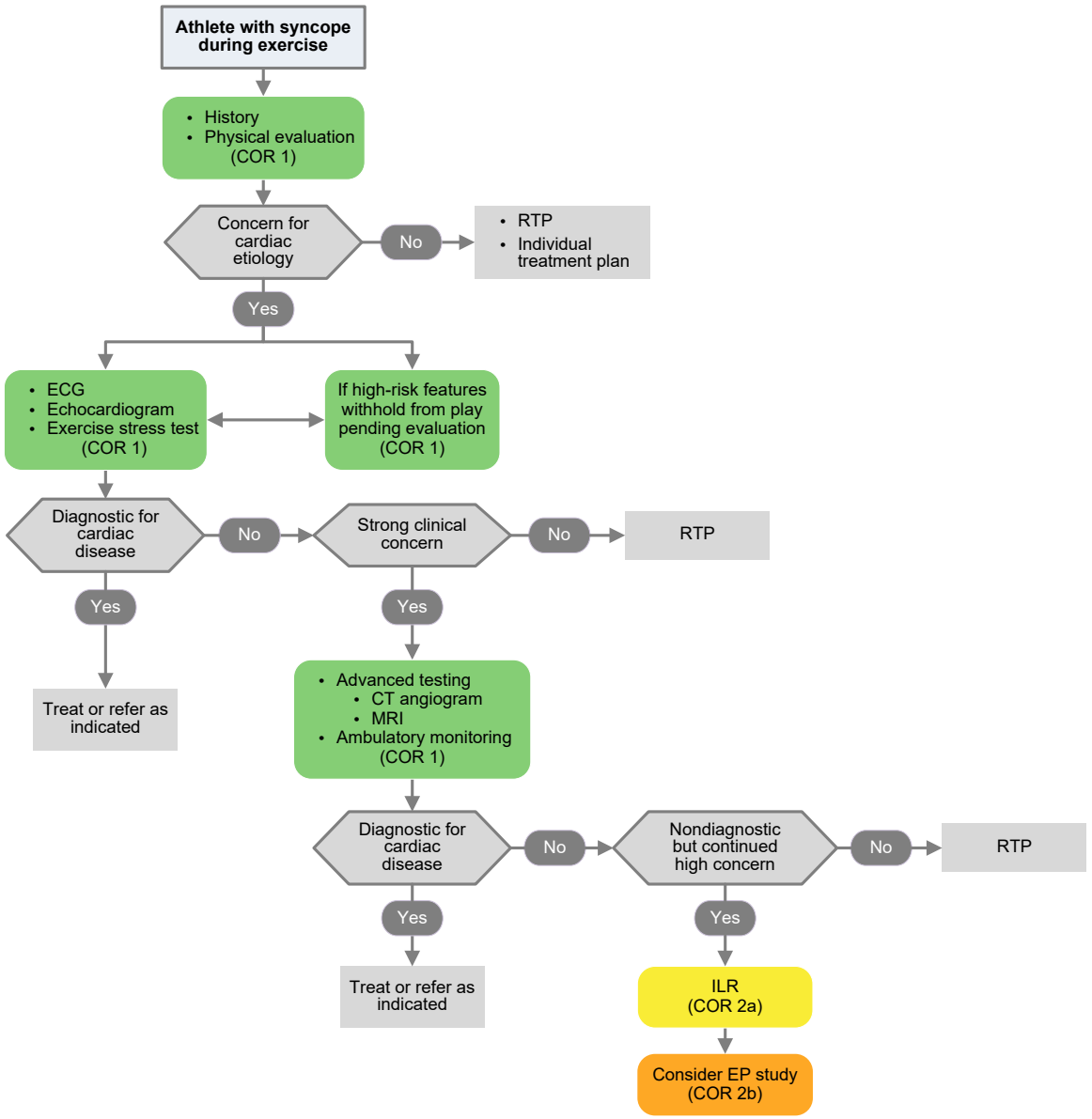

Figure 8
Algorithm for the evaluation of syncope during exercise. Colors correspond to the class of recommendation (COR) in Table 1. CT = computed tomography; ECG = electrocardiogram; EP = electrophysiology; MRI = magnetic resonance imaging; RTP = return to play.

diagnostic approach for athletes with syncope should be focused to exclude these cardiac substrates for ventricular tachyarrhythmia and SCD. Syncope in athletes that occurs during exercise is more likely to be associated with structural cardiac pathology potentially associated with SCD than syncope that occurs post-exercise or is not exercise-related, although this remains a rare cause.[241] Colivicchi et al[242] describe a cohort of 7568 athletes of whom 474 (6.2%) reported at least 1 syncopal episode. In the majority of athletes, syncope was unrelated to exercise (86.7%); syncope was post-exertional in 57 (12.0%) and exertional in 6 (1.3%). All episodes of nonexertional or post-exertional syncope had the typical features of neurally mediated fainting with a prodrome and were not related to cardiac disease. Of the 6 athletes with exertional syncope, 1 had HCM and 1 had right ventricular outflow tract (RVOT) ventricular tachycardia. Although this study showed a relatively high rate of cardiac causes, subsequent series have shown cardiac etiologies in 8-10% of exercise-related syncope.[242–247] Table 11 shows series of studies in athletes that looked at the etiology of syncope during exercise. From a total of 6 studies, 82 athletes manifested syncope during exercise. Most of the episodes (75 of 82 [91.5%]) were not associated with structural heart disease. The remainder (8.5%) occurred in athletes diagnosed with cardiovascular disease, which included HCM in 1 of 7 (14.2%), RVOT ventricular tachycardia in 1 of 7 (14.2%), mitral valve prolapse in 3 of 7 (42.8%), angina in 1 of 7 (14.2%), and anomalous coronary artery in 1 of 7 (14.2%). The majority of these events did not occur in the setting of underlying structural or electrical disease; however, because these diseases can be life-threatening, workup is indicated.

Table 11 Series of athletes with syncope

Study	Study design (N)	Age	Syncope		Findings
			Timing	N (%)	
Colivicchi et al, 2004[242]	Preparticipation screening N=7568 athletes, 474 reported prior syncope in the past 5 years	16 ± 3 years	During exertion	6 (1.3%)	1/6 (16.7%) HCM; 1/6 (16.7%) RVOT VT; 4/6 (66.7%) noncardiac
			Post-exertion	57 (12.0%)	100% noncardiac
			Non-exercise-related	411 (86.7%)	100% noncardiac
Colivicchi et al, 2002[243]	Cohort study N=33 athletes with recurrent exercise-related syncope	Mean age 21.4 ± 3.2 years	During exertion	33 (100%)	2/33 (6%) mitral valve prolapse; 4/33 (12.1%) hypotension during maximal exercise; 22/33 (66.6%) head-up tilt testing positive
			Post-exertion	NR	NR
			Non-exercise-related	NR	NR
Holtzhausen et al, 1994[245]	Prospective series in 56 km ultramarathoners N=111 athletes, 46 collapsed during or after the race	33 ± 8 years	During exertion	8	3/8 (37.5%) hypoglycemia; 3/8 (37.5%) gastroenteritis; 1/8 (12.5%) angina; 1/8 (12.5%) asthma
			Post-exertion	38	Diagnosed exercise-associated collapse
			Non-exercise related	NR	NR
Kaiser-Nielsen et al, 2017[246]	Retrospective series in athletes referred for any symptom N=201 athletes, 38 with syncope	Median age 27 years	During exertion	10 (26.3%)	Reported no cardiac syncope, specific diagnoses NR
			Post-exertion	13 (35.2%)	Reported no cardiac syncope, specific diagnoses NR
			Non-exercise-related	15 (39.5%)	Reported no cardiac syncope, specific diagnoses NR
McKinney et al, 2017[247]	Preparticipation screening N=1419 athletes, 16 reported prior exertional syncope	12–35 years	During exertion	16 (100%)	1/16 (6.3%) mitral valve prolapse
			Post-exertion	NR	NR
			Non-exercise-related	NR	NR
Gier et al, 2023[244]	Retrospective series in college athletes with syncope/presyncope N=55 athletes, 15 with syncope related to exercise	19.7 ± 1.5 years	During exertion	9 (25.0%)	1/9 (11.1%) anomalous aortic origin of coronary artery
			Post-exertion	6 (16.7%)	No underlying cardiac condition
			Non-exercise-related	NR	NR

HCM = hypertrophic cardiomyopathy; RVOT VT = right ventricular outflow tract ventricular tachycardia; NR = not related.

5.1.2 Etiologies (noncardiac differential)

Noncardiogenic causes of syncope can be broken down to orthostatic syncope and neurally mediated (reflex) syncope.[239,248] A thorough and complete history can suggest the cause of syncope and direct the workup. The most common cause of syncope in athletes is post-exertional syncope due to the abrupt decrease in venous return that occurs when activity is stopped and there is a relaxation of the "muscular pump," a form of orthostatic syncope.[249] With prolonged exertion there is significant vasodilation, and the sudden loss of pressure by skeletal muscle causes pooling of the blood in the extremities, which leads to loss of consciousness. Training-induced increases in vagal tone may increase likelihood of this occurrence. Post-exertional orthostatic syncope can be witnessed at any time; a common example occurs in long-distance running often as athletes cross the finish line, exiting "the chutes." Likewise, post-exertional orthostatic syncope is common in rowing, particularly when training on dry land on a rowing ergometer while doing set workouts or "pieces." Post-exertional syncope does not reflect underlying life-threatening disease, and it can be treated with attention to volume, electrolyte, and hydration status as well as a slow cool-down period. In some cases, compression stockings, either knee or thigh high, may be helpful. Inadequate nutrition may exacerbate this type of syncope, and eating regularly or consultation with a nutritionist can be helpful.[250] In athletes with heat-related post-exercise syncope who have not responded to nonpharmacological treatment, one small series has suggested an H1-receptor antagonist may be helpful.[251] Neurally mediated syncope occurs when neural reflexes modify heart and blood pressure inappropriately, which can occur in response to strong emotion, micturition, defecation, swallowing, and coughing in both athletes and nonathletes.

As shown in Table 12, loss of consciousness can occur from other athletic-related conditions such as head trauma, heat illness, hyponatremia, and exertional collapse related to SCT.[249] History and context should inform these diagnoses. For heat illness, treatment is emergent and includes rapid

cooling, best accomplished in an ice bath with evaluation of rectal temperature. Cooling is critical and should occur before transport to emergency care. Hyponatremia should be suspected in situations where significant consumption of water has occurred. This is typical in a long-distance race or marathon in a participant with a slower pace and longer overall duration of exercise. Hyponatremia is associated with incoordination and altered mental status. One in 12 Black individuals are heterozygous for SCT, which is associated with an increased risk of sudden death.[103] This is distinct from sickle cell disease, which is generally incompatible with strenuous physical activity. The pathogenesis of exercise-associated collapse associated with SCT is sickling of red blood cells in the microvasculature leading to muscle death, massive release of potassium, and fatal arrhythmia. Exercise-associated collapse associated with SCT occurs only when athletes are unable to stop and recover and are pushed beyond their physical capacity, which is almost always when training. Any struggling and collapsing athlete should not be pushed and should be allowed to recover. If the athlete is known to have SCT, physical activity should be halted if the athlete is struggling. If they collapse, EMS should be activated and the emergency room informed of likely etiology. Finally, loss of consciousness in both athletes and nonathletes can be due to seizures, intoxication, and psychogenic causes and should be included in the differential diagnosis.

Table 12 Noncardiac causes of loss of consciousness

Cause	History	Pathogenesis	Treatment	Workup
Post-exertional/ orthostatic	Occurs after heavy exercise, usually after abrupt cessation of heavy exertion	With cessation of exercise, the muscle pump is lost and blood pools in extremities	• Supine position with legs lifted • Attention to hydration, electrolytes, and nutrition • Consider compression stockings	• With classic history, no workup is necessary • ECG may be considered
Neurally mediated	• Strong emotion • Micturition • Defecation • Swallowing • Coughing	Inappropriate heart and blood pressure response	None	• With classic history, no workup is necessary • ECG may be considered
Heat illness	• High wet-bulb globe index* or temperature/humidity • Altered mental status • Incoordination • Conscious with progressive collapse	Increased core body temperature	Immediate cooling	• Immediate rectal temperature • Comprehensive metabolic profile, LFTs, CK, and ECG if patient does not respond immediately to cooling
Hyponatremia	• Endurance race • Slower times • High water consumption • Conscious, progressive collapse • Altered mental status	Low sodium	Sodium administration, dependent on severity, should be done in the emergency room	Comprehensive metabolic panel
Exertional collapse associated with SCT	• Athlete with SCT (1 in 12 Black individuals) • Not all individuals with SCT are aware • Extreme exertion when athlete feels they cannot stop to recover, typically in settings of training, timed run, or not letting teammates down	RBC sickle in peripheral vasculature "clogging" capillaries leading to muscle necrosis, massive potassium and myoglobin release, and arrhythmia or renal failure	• Stop activity when athlete is struggling • If recovery is not immediate, consider: ○ Oxygen therapy ○ Transport to hospital ○ Make emergency room aware that immediate electrolytes are needed ○ Supportive care with attention to kidney function	Comprehensive metabolic panel

CK = creatine kinase; ECG = electrocardiogram; LFT = liver function test; RBC = red blood cell; SCT = sickle cell trait.
*Measure of heat stress in direct sunlight, taking into account temperature, wind speed, humidity, sun angle, and cloud cover.

5.1.3 Diagnostic and monitoring strategies for syncope in athletes

Recommendations for diagnostic and monitoring strategies for syncope in athletes

COR	LOE	Recommendations
1	B-NR	1. In athletes with syncope, a detailed history and physical examination should be performed to guide further diagnostic evaluation.[129,131,252]
1	B-NR	2. In all athletes with syncope during exertion, an ECG, exercise stress test, and transthoracic echocardiogram should be performed.[129,131,253–262]
1	C-EO	3. In athletes with syncope during exertion with high-risk features, withholding from sports participation pending evaluation is indicated.
1	B-NR	4. In athletes with syncope during exertion with high-risk features and with negative primary evaluation, advanced imaging should be performed.[254,263,264]
1	C-EO	5. In athletes with syncope, tests should be interpreted in the context of EICR.
1	B-R	6. In athletes with unexplained syncope or when arrhythmic syncope is suspected, ambulatory ECG monitoring is beneficial.[265–267]
2a	B-NR	7. In athletes with a high suspicion of arrhythmic etiology, unexplained after initial testing, and/or whose symptoms are rare, loop recorder implantation can be useful.[268,269]
2b	C-LD	8. In athletes with syncope with high-risk features and negative initial evaluation, an EP study may be considered.[270,271]
3: Harm	C-LD	9. In athletes with syncope, tilt table testing is not recommended because of high prevalence of false positives leading to inappropriate interventions.[272–274]
3: No benefit	C-EO	10. In athletes with a history suggestive of noncardiac syncope, further evaluation is not indicated.

Synopsis

Syncope in an athlete requires a thorough evaluation including history, physical, and advanced testing as appropriate. While the majority of syncope during exertion is noncardiac, cardiac workup is indicated to rule out life-threatening conditions. Identification of the cause enables appropriate treatment, shared decision-making, and return to play when appropriate. The characteristics of high risk as described below will determine appropriate workup as well as timing of return to play. Use of diagnostic specific modalities is described below.

Recommendation-specific supportive text

1. An algorithm for the evaluation of syncope with exercise is shown in Figure 8. Most important are the history and physical exam, which determine the etiology of syncope in the majority of cases.[239] The history should be obtained from the athlete, bystanders, athletic trainers, and a review of video evidence when available. For athletes, determining whether true loss of consciousness occurred, versus near syncope, and determining whether the event occurred during or after exercise are the most important features determining likelihood of a life-threatening etiology, as described in Section 5.1 and Table 11. Characteristics of higher-risk syncope appear in Table 13 and include acute face plant or other manifestation of acute or abrupt loss of consciousness and postural tone without protective reflexes during exertion, abrupt palpitations, prior history of syncope, shortness of breath that limits their ability to exercise, and chest pain. Typical situational triggers and symptoms suggestive of vaso-vagal origin should be queried.[239] A social history related to the use of recreational drugs or performance-enhancing drugs should be obtained (see Table 4). Family history of sudden, young, or unusual death also indicates need for more extensive workup.
2. The ECG is the principal tool for the detection of cardiovascular abnormalities in athletes with syncope. There are a wide range of expected findings that occur as a result of physical conditioning that need to be taken into account when evaluating an athlete's ECG, as outlined in the International Criteria for Electrocardiographic Interpretation in Athletes consensus statement.[148] Many of these findings would be considered abnormal in nonathletes and if unrecognized may result in additional testing and unnecessary restriction from exercise.[148] Echocardiography for the assessment of structural heart disease is important, as the ECG does not have complete sensitivity for many of the conditions that may lead to syncope, including cardiomyopathy and anomalous origin of a coronary artery. Stress testing is critical for identifying exercise-induced arrhythmias, which may be diagnostic and/or indicate need for advanced imaging.[275,276] As described

Table 13 High-risk features for cardiac syncope

High-risk features
Absence of prodrome
Acute face plant during exertion
Abrupt palpitations
Shortness of breath that limits the ability to exercise
Chest pain

in detail in Section 2.3, exercise stress testing should mimic the activity during which syncope occurred. Standard exercise protocols that are intended to elicit ischemia are not appropriate for the assessment of syncope in the athlete.

3. Those athletes with syncope during exercise who present with high-risk features (Table 13) such as acute face plant during exertion, abrupt palpitations, prior history of syncope, shortness of breath that limits their ability to exercise, chest pain, and/or a family history of sudden death, require cessation of sports activity until the necessary testing can be obtained.
4. In athletes with an initial nondiagnostic evaluation, advanced imaging, such as CMR or cardiac computed tomographic angiography (CTA), provide increased sensitivity for coronary artery anomalies and cardiomyopathies. CMR has unique abilities to characterize tissue. Late gadolinium enhancement is a marker of fibrosis or extracellular protein deposition. T1 and T2 mapping allow further quantification of fibrosis or infiltration, as well as edema. These increase sensitivity for diagnosis of HCM, ACMs, and arrhythmogenic bileaflet mitral valve prolapse (ABiMVP), even when the echocardiogram or ECG does not demonstrate these.[277] Angelini et al[263] described a series of 5169 children aged 11 to 18 years undergoing screening including CMR, with probable high-risk cardiovascular conditions found in 76 (1.4%) of the participants, in whom ECG showed no abnormalities. CMR could be useful in athletes with syncope and suspected structural abnormalities, including AAOCA. Prevalence of AAOCA is reported in 1.5%-1.7% of CTAs done for a variety of indications; the most common AAOCA was an origin of the right coronary artery from the left coronary sinus, followed by an origin of the left circumflex artery arising from the right coronary sinus.[278,279] In one small study of patients with syncope,[264] CTA revealed AAOCA after prior workup was unrevealing. CTA is currently considered a "gold standard" for diagnosis of AAOCA, identifying both the ostia and course of the coronary arteries, and ongoing technical advancements have decreased acquisition time and radiation exposure.[280] CMR is emerging as an alternative to CTA, particularly as data on tissue characteristics and cardiac structure can be obtained at the same time.[281] If AAOCA is found, invasive coronary angiography, flow measurements, and assessment of ischemia is generally needed to determine functional significance.
5. As discussed in detail in Section 2, normal EICR leads to structural and electrical changes that are not pathological. Understanding these changes is critical to interpretation of testing in athletes to avoid incorrect diagnosis of pathological entities.
6. Rhythm monitoring strategies are an important part of the assessment for syncope during exertion particularly in the instance where a cardiac condition is suspected or the activity that triggered the symptoms could not be reproduced on an exercise stress test. Table 14 describes features of available systems to guide selection of the appropriate device depending on the frequency and duration of the symptoms, including the device configuration, advantages/limitations of the device, and sports-specific considerations. In the absence of prodrome, a device with autodetection such as a loop/event recorder or implantable loop recorder should be considered. In the SYNARR-Flash study of general patients with syncope,[265] 4-week external ECG monitoring had a diagnostic yield of 71.6%. Holter monitoring has a good diagnostic performance, especially in those patients with daily symptoms.[282] In one cohort of 654 athletes with symptoms or signs of arrhythmias, including syncope, or an abnormal ECG, Holter monitoring established a diagnosis of arrhythmia in a substantial proportion of the patients. If immediate results are needed, a continuous ECG monitor is a better option than a Holter monitor or certain patch monitors that need to be returned for processing. Multiple channels are helpful if activity may create artifact on a single lead.
7. Implantable loop recorders have been found to be useful in several studies in the general population for establishing the etiology of syncope especially in cases where infrequent episodes fall outside the recording window of other modalities (eg, 2-week patch monitor).[268,269,283] In addition, the subcutaneous location allows continuous recordings for athletes during any sport, whether in the water or during periods of high perspiration.
8. There is no evidence about the usefulness of an EP study including arrhythmia induction, voltage mapping, and drug infusion in athletes with syncope. The 2017 AHA/ACC guidelines on syncope[239] recommend an EP study in the general population with syncope and suspected arrhythmic disease, although the yield in those with no structural heart disease is low (2%-9%).[270,271] The yield of an EP study in diagnosis in athletes when there is no obvious etiology after a thorough evaluation is unknown.
9. Specificity of the tilt test in athletes is low, with a positive test in 47% of patients with arrhythmic syncope, and presyncope with typical hemodynamic features can be induced on tilt even in patients without a syncopal history.[273] Studies of healthy athletes with no syncope history show rates of positive tilt testing in 20%-100% of asymptomatic endurance runners.[284–286] This high frequency of false positives can bias medical judgment and make true diagnosis more difficult[287] and may lead to unnecessary therapeutic interventions such as medications or procedures.
10. Neurally mediated or orthostatic syncope is common in athletes with noncardiogenic syncope. If the patient's clinical context is characteristic of a neurally mediated episode or orthostatic syncope, no further workup is necessary.

5.1.4 Treatment of neurally mediated or orthostatic syncope in athletes

Neurally mediated syncope and orthostatic syncope are common in athletes. Treatment plans can be similar to those for nonathletes. The management of neurally mediated syncope or orthostatic syncope begins with nonpharmacological treatment and education of both the athlete and the athletic personnel on recognizing the triggers of syncopal episodes as well as the implementation of physiological measures to reduce the likelihood of syncope. Special attention is required in the athlete to reduce the risk of injury based on the sport and/or the circumstances.

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Table 14 Features of ambulatory monitors, including external and implantable recorders

Monitor type	Device configuration	Recording time	Advantage	Limitations	When to use	Sports-related considerations
Holter monitor (wire based)	Small, battery-operated recorder with 2 -3 leads for recording	Continuous recording for 24-48 hours	Continuous recordings of multiple channels (2-3); inexpensive for clinics	Time-limited recording; device must be returned and processed creating delays	Frequent symptoms (daily)	Bulky and gets in the way of most activities; not waterproof and needs to be removed to shower or bathe; still useful for burden (eg, PVCs) assessment
External loop/ event monitor	Small, battery-operated recorder with 1-3 leads for recording	Records up to 30 days of rhythm data before and after triggered event	May be worn continuously or applied during symptoms; some devices transmit real-time data to a monitoring center	Some require activation of recording and others are auto-triggered to record; some devices must be returned and processed creating delays	Less frequent symptoms (weekly to monthly)	Bulky and gets in the way of most activities; not waterproof and not useful without autodetection in syncope without prodrome
Patch continuous monitor	All-in-one adhesive device with continuous recording; no wires or battery packs; some have Bluetooth capability	Similar to Holter, records up to 30 days of patient-triggered events along with auto-triggered events	All-in-one adhesive device without wires	Typically, only a single lead, which can suffer from signal distortion; can cause skin irritation	Daily, near daily, or weekly	Smaller profile and less obtrusive than Holter or loop/event monitor; some are waterproof
Implantable loop recorder	Small, subcutaneously implanted monitoring device; Bluetooth or smartphone app communication	Long-term monitor that lasts up to 3-4 years	Records rhythm when activated and has autodetection algorithms that can be programmed; continuously recording useful in infrequent syncope without prodrome	MRI conditional (safe under specific MRI environments); can impact MRI image quality; false or noisy signals can be an issue; requires procedure to remove	Less frequent symptoms (monthly to yearly) that may lead to syncope	Subcutaneous and generally unaffected by activity (including swimming) or perspiration
Mobile cardiac telemetry	Patch or lead-based sensor transmits continuous ECG information to monitoring center via cellphone technology	Can be worn up to 30 days; transmits real-time rhythm data to a monitoring center for immediate review	Auto-trigger algorithms that detect abnormal rhythms and transmit data to the monitoring center; records all cardiac activity so useful in frequent syncope without prodrome	Generally, more expensive; not readily available	Less frequent palpitations (weekly to monthly)	Nonpatch versions are bulky and not waterproof; requires cell tower connection so limited in some areas
Commercially available wearables	Fitness trackers, smartwatches, handheld electrodes, chest strap	ECG displayed and stored directly on device and can be sent wirelessly and shared with care team	Commercially available (does not require a prescription); algorithms available for autodetection of arrhythmias, easy to use	Ability to integrate into electronic health record still a challenge	Palpitations lasting long enough to manually record	Challenging to record mid-exercise and not usable in all conditions; more data needed on effect of sports on quality of tracings and alerts; cost prohibitive for some

ECG = electrocardiogram; MRI = magnetic resonance imaging; PVC = premature ventricular contraction.

Recommendations for treatment of neurally mediated or orthostatic syncope in athletes		
COR	LOE	Recommendations
1	C-EO	1. In athletes with neurally mediated or orthostatic syncope, providing education on the diagnosis, nonpharmacological treatment, prognosis, and triggers of syncope is recommended.
1	C-EO	2. In athletes with neurally mediated or orthostatic syncope, communication among the medical and athletic teams for preparation to manage a syncopal event is recommended.

Synopsis

Most of the pharmacological and nonpharmacologic measures for the athlete are similar to those for the nonathlete, with athlete-specific considerations as noted below.

Recommendation-specific supportive text

1. The identification of triggers and application of the countermeasures can reduce the rate of recurrence. Athletes should be trained to recognize their prodromal symptoms and stop physical activity and execute physical counterpressure maneuvers (sitting down, bending the head forward, leg crossing)[288] in an attempt to abort the episode. A cool-down period after intense exercise, rather than stopping abruptly, is critical. The National Athletic Trainers' Association recommends[289] that athletes should maintain hydration (+1% to –1% of body mass) for best exercise performance and should not lose more than 2% of body mass after exercise. It is possible to have oral prehydration of no more than 2% of body mass and to consume sodium chloride supplements during exercise to keep an adequate plasma volume and vascular tone.[289] Knee-high compression socks attenuated the drop in cardiac stroke volume and cerebral blood flow velocity, suggesting that these may be an effective countermeasure to reduce the incidence of post-exercise syncope.[290] Reassurance that these are not life-threatening conditions and can most often be managed with conservative measures may reduce anxiety in athletes with these symptoms. Cardioneuroablation is emerging as a promising therapy for vagal syncope but is not yet standard of care.[291]
2. All athletes with neurally mediated syncope or orthostatic syncope require close medical monitoring to reduce the rate of syncopal episodes that impact quality of life and sports performance of the athlete. Measures should be put in place to reduce the risk of syncopal episode in environments such as a high dives, skating rinks, and cycling paths or in other similar high-risk scenarios where loss of consciousness could endanger the life of the athlete.

5.2 Palpitations in athletes

Palpitations are the perception of rapid heartbeats, extra beats, or skipped beats. They may be related or unrelated to exercise in athletes and can be associated with cardiac pathology, systemic pathology, or normal physiological processes. Athletes are often in tune with their body, and anything out of the ordinary, such as palpitations, can cause significant distress. Palpitations in an athlete thus require further evaluation.

5.2.1 Etiology of palpitations

Similar to nonathletes, the most common causes of palpitations in athletes are sinus tachycardia or premature atrial and/or premature ventricular beats. Arrhythmic conditions such as supraventricular tachycardia (SVT), AF/atrial flutter, and ventricular tachycardia are less common (Table 15).[282,292–295] In a retrospective cohort of 6579 elite athletes,[282] 659 underwent Holter monitoring, of whom 162 reported symptoms (palpitations, syncope, dizziness, chest pain). The most common arrhythmic findings in this group of athletes were premature atrial contractions (62%), PVCs (39%), nonsustained ventricular tachycardia (1.5%), and AF/atrial flutter (< 1%).[282] Importantly, the vast majority of symptomatic palpitations were not associated with structural cardiac abnormalities.

Table 15 Series of athletes with palpitations, arrhythmias, and cardiomyopathies documented

Study	Study design/population	Age	No. patients with palpitations	Arrhythmic findings	Structural conditions	Monitoring strategy
Boraita et al, 2022[282]	A retrospective cohort of elite athletes; 654/6579 (9,9%) had any sign/symptom related to arrhythmia or abnormal resting/exercise ECG, and they were evaluated with Holter including at least one hard training session	Median age 24 years (interquartile age range 19–28)	162/654 reported symptoms (palpitations, syncope, dizziness, chest pain)	Sinus bradycardia (96% of cases), Premature atrial and ventricular beats (61.9% and 39.4%, respectively), sinus pauses ≥3 s, high-grade atrioventricular blocks, and AF/atrial flutter were rare (<1%). Polymorphic PVCs (1.4%) and idioventricular rhythm (0.005%). PVC couplets (10.7%) (PVC triplets 1.8%; sustained ventricular tachycardia 0.0%; and NSVT 1.5%)	HCM (6), DCM (2), ACM (2), LV noncompaction (8), ICM (1)	Most underwent single Holter evaluation, 19% underwent 2 or more evaluations
Jewson et al, 2022[293]	Case series; 6 athletes with symptoms related to exercise; 4/6 palpitations	Ages 16-28 years	4/6	Athletes with palpitations (2 patients with SVT, 1 patient with AF, and 1 patient with normal ECG). Athletes without palpitations (1 with atrial flutter, 1 with normal traces)	None found	6 leads, Smartphone ECG AliveCor Kardia device
Peritz et al, 2015[294]	Case series; 6 college athletes	Ages 18-21 years	6	No arrhythmias detected	None found	Single-lead ECG by AliveCor Heart monitor
Sciarra et al, 2022[295]	Athletes with palpitations and unknown origin with external loop recorder/cohort study; 61 athletes/ 61 sedentary controls with palpitations at least once monthly	Median age 24 years (age range 18-37 years)	61	7 (sustained SVT), 2 (NSVT), 4 (PVCs), 3 (PACs), 28 (no arrhythmic findings during palpitations), 11 (negative symptoms and findings)	None found	The median duration of ECG monitoring was 12 days
Biffi et al, 2002[292]	Observational study (Long-Term Clinical Significance of Frequent and Complex Ventricular Tachyarrhythmias in Trained Athletes); 355 athletes with palpitations or 3 or more PVCs in resting ECG; they were evaluated with 24-hour ambulatory Holter	Group A ages 24 ± 10 years; group B ages 24 ± 10 years; group C ages 25 ± 11 years	18/355	8/18 (≥ 2000 PVCs and ≥ 1 NSVT) for group A; 10/18 (≥ 100 to < 2000 PVCs) for group B	Group A (≥ 2,000 PVCs and ≥ 1 NSVT) (7 ARVC, 6 mitral valve prolapse, 4 myocarditis, 4 DCM); group B (≥ 100 to < 2,000 PVCs) (5 mitral valve prolapse); group C (< 100 PVCs) nonstructural condition	24-hour ambulatory (Holter) ECG

ACM = arrhythmogenic cardiomyopathy; AF = atrial fibrillation; DCM = dilated cardiomyopathy; ECG = electrocardiogram; HCM = hypertrophic cardiomyopathy; IC = ischemic cardiomyopathy; LV = left ventricular; NSVT = nonsustained supraventricular tachycardia; PAC = premature atrial contraction; PVC = premature ventricular contraction; SVT = supraventricular tachycardia.

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5.2.2 Diagnostic strategies in athletes with palpitations

Diagnostic strategies in athletes with palpitations		
COR	LOE	Recommendations
1	B-NR	1. In athletes with palpitations, a history, physical examination, and resting 12-lead ECG are recommended.[129]
1	B-NR	2. In athletes with palpitations associated with exercise, exercise stress testing should be performed to mimic the athlete's sport, or monitoring during exercise should be performed.[282]
2a	B-NR	3. For athletes in a team setting, supplying athletic trainers with smartphone-based ECGs for onsite assessment of palpitations can be useful.[294]
2b	C-LD	4. In athletes with palpitations, a personal (portable or wearable) ECG may be considered as a diagnostic tool.[293]

Synopsis

When evaluating an athlete with palpitations, it is important to obtain a detailed history along with a physical examination and 12-lead ECG. It is critically important to know the frequency of palpitations, the relation with exercise, and if there are associated symptoms such as chest pain or lightheadedness. When considering a monitoring strategy for athletes, it is important to take the sport into consideration and select a monitor that has adequate data recording but doesn't impede the athletes' ability to participate in the sport.

Recommendation-specific supportive text

1. Athletes with palpitations should have a comprehensive evaluation including history, physical examination, and 12-lead ECG.[7,129,296] Description of symptoms such as heart racing with abrupt onset/offset, versus gradual ramp up and ramp down, favor sustained arrhythmia including supraventricular or ventricular tachycardia. Irregularity suggests ectopic beats or AF. The association with exercise and any associated symptoms of lightheadedness, chest pain, or syncope are important. A family history of cardiovascular disease increases the likelihood of underlying heart disease. During the physical examination, it is important to assess the pulse for the presence of ectopic beats or irregularity. The ECG may reveal ectopy or evidence of potential underlying cardiac conditions.[7,129,296] When evaluating the athlete's ECG, it is important to recognize that the changes that may be considered abnormal in nonathletes can occur commonly in athletes due to physical adaptation.[148] The routine use of echocardiography in evaluation of palpitations, in the absence of documented arrhythmia or other concerning personal or family history, is not supported by data, and recommendations regarding this use vary,[297] with use in unselected populations felt not appropriate, although some have recommended its use in the athlete with exertional symptoms.[36]
2. In athletes with palpitations occurring during exercise, it is important to identify the rhythm associated with symptoms. The standard exercise stress test has the benefit of availability, a controlled environment, and recording of the rhythm with 12-lead ECGs but often doesn't mimic the activity that triggered the symptoms such as swimming, jumping, or climbing. Stress testing in athletes is discussed in more detail in Section 2.3 In circumstances where the trigger is hard to reproduce in the typical exercise laboratory or stress testing does not invoke the symptoms, then a monitoring strategy that enables rhythm determination during the activity can be useful. Table 14 describes features of available systems to guide selection of monitoring strategy. When selecting a monitor for an athlete, the frequency and the duration of the palpitations should be considered.[298] There are also sport-specific considerations such as whether the recording device is waterproof (swimming) or gets in the way of the athletic activity (eg, Holter monitor in rowing and climbing). If monitoring with the selected device captures the symptom and demonstrates a normal rhythm, then reassurance is indicated to allay the concerns of the athlete.[7,296]
3. Athletic trainers are often present at sporting events, and if provided with a smartwatch or handheld ECG recording devices, they can record the athlete's heart rhythm at the time they are symptomatic. This strategy has been shown to decrease need for cardiology evaluation, as most often the rhythm during symptoms is sinus.[294]
4. In athletes who have palpitations that are infrequent or difficult to capture with standard recording devices, commercially available wearable devices that allow rhythm recordings, such as a smartwatch or handheld electrodes, can be helpful. These devices also have limitations, including accuracy and the requirement that the user have the device always available.[293,294,298,299]

Section 6 Ventricular arrhythmias

Ventricular arrhythmias are similarly prevalent in athletes and nonathletes[282,300–302] and are 5- to 10-fold less common in women athletes as compared with men.[262,302,303] There is no clear correlation between the amount and type of exercise and ventricular arrhythmias.[282,300,301] PVCs or other ventricular arrhythmias may be identified when an athlete presents with palpitations, or asymptomatic PVCs may be identified during an ECG obtained for preparticipation screening[6,70] or for other purposes. Typical PVCs are those of an outflow tract or fascicular morphology. Complex ventricular arrhythmias include PVCs of atypical morphology and/or that are multifocal, have a short-

Table 16 Risk features from history and tests used to identify structural or electrical heart disease

Low-risk features	Higher-risk features
Clinical	
Asymptomatic	Presyncope, syncope, dyspnea, or sudden-onset exercise intolerance
Palpitations suggestive of simple PVCs	Sustained rapid palpitations
No history suggestive of inherited heart disease	Family history of collapse, syncope, sudden death, or cardiomyopathy
Electrocardiographic	
Monomorphic PVCs with outflow tract or fascicular morphology ("typical morphology")	Polymorphic PVCs or with non-outflow-tract or fascicular morphology ("atypical morphology")
Normal 12-lead ECG (other than PVCs)	Abnormal ECG (in addition to PVCs), eg, low voltage
Normal PVC coupling interval	Short PVC coupling interval
Single PVCs	Couplets, triplets, or nonsustained ventricular tachycardia
Low burden of PVCs on Holter ECG	High-burden PVCs (> 10% burden)
Exercise stress testing	
Suppression of PVCs with exercise	Nonsuppression or emergence of PVCs with exercise or emergence of PVCs during recovery
No symptoms and normal hemodynamics	Symptoms of sudden-onset exercise intolerance associated with emergence of arrhythmias
Echocardiography/stress echocardiography	
Normal cardiac structure and function for an athlete (ie, inclusive of athlete's heart changes)	Increased wall thickness or excessive ventricular dilation, reduced systolic function, or segmental abnormalities
Clear augmentation of biventricular function with exercise	Reduced contractile reserve during exercise (of the left or right ventricle)
Cardiac magnetic resonance imaging	
Normal structure and function	Increased wall thickness or excessive ventricular dilation, reduced systolic function, or segmental abnormalities
No evidence of post-contrast enhancement	Delayed gadolinium enhancement, particularly mid-wall or epicardial enhancement
Invasive electrophysiology	
Focal arrhythmogenic site	Multiple inducible arrhythmias
Catecholamine triggering of focal site	Catecholamine triggering of rapid polymorphic arrhythmias
Normal electroanatomical mapping	Low-voltage regions (noting a tendency to epicardial pathology in endurance athletes)

ECG = electrocardiogram; PVC = premature ventricular contraction; RVOT = right ventricular outflow tract.

coupling interval, or occur as multiples, including couplets, triplets, or nonsustained ventricular tachycardia. High-risk ventricular arrhythmias are defined as those with morphological as well as clinical features including nonsuppression with exercise suggestive of higher risk of malignant prognosis, as shown in detail in Table 16. Benign PVCs and benign idiopathic ventricular tachycardia are defined as those occurring in the absence of structural heart disease, identified electrical disease, or high-risk electrical features, as noted above.

Inherited cardiomyopathies (HCM, ACM, and DCM), ABiMVP, inherited channelopathies (LQTS, Brugada, and CPVT), and acquired pathologies (coronary artery disease, myocarditis, and sarcoid) are all heart diseases associated with ventricular arrhythmias. Identifying whether one of these pathologies is present is critical, as the presence of underlying heart disease is the primary determinant of prognosis and outcomes in ventricular arrhythmias.

6.1 Evaluation of ventricular arrhythmias in athletes

The primary initial objective in the assessment of an athlete with PVCs is to determine whether these are benign focal premature beats or the potential sign of structural or electrical heart disease, which can be associated with SCA. In addition to underlying heart disease, a syndrome of acquired heart disease has been described in highly trained endurance athletes who develop ventricular arrhythmias in the absence of inherited heart disease. Heidbuchel et al[304] reported that ventricular arrhythmias in endurance athletes (predominantly professional cyclists) most frequently arose from the right ventricle, were often associated with mild structural or functional abnormalities, and could be life-threatening despite an absence of clinical or genetic evidence of inherited heart disease.[304,305] This entity was initially termed "exercise-induced ARVC," and validation of the observation of an "ARVC-like" syndrome in endurance athletes without evidence of inheritance has also been referred to as genotype negative ARVC.[306] More recently, Venlet et al[307] provided an invasive electrophysiological description of this syndrome, noting that endurance athletes often had epicardial-based scar originating around the RVOT that was associated with very rapid, potentially life-threatening ventricular tachycardia. There have also been descriptions of arrhythmogenic subepicardial scar in the left ventricle of endurance athletes, again in the absence of inherited heart disease.[308,309]

Thus, in the athlete presenting with ventricular arrhythmias, the clinician must consider the possibility of 3 main categories, each with different prognostic and therapeutic

implications. Two of these entities, benign ventricular ectopy in the absence of structural heart disease and ventricular arrhythmias associated with structural heart disease (inherited or acquired), have features that are similar to nonathletic populations, with the caveat that it can be difficult to distinguish between the structural and functional changes of "athlete's heart" and subtle pathology. The third entity, exercise-induced ACM, is unique to endurance athletes.

Recommendations for the evaluation of ventricular arrhythmias in athletes

COR	LOE	Recommendations
1	B-NR	1. In athletes with symptoms suspicious for ventricular arrhythmias, a resting 12-lead ECG and ambulatory ECG monitor are recommended to assess ventricular arrhythmia burden and complexity.[310,311]
2a	B-NR	2. In athletes with symptoms suspicious for suspected ventricular arrhythmias, exercise stress testing is reasonable to assess ventricular arrhythmia occurrence, characteristics, and morphology.[255,312]
1	C-EO	3. In athletes with ventricular arrhythmias, taking a history of supplements and performance-enhancing drug use is recommended to identify potential triggers for ventricular arrhythmias.
1	C-LD	4. In athletes with 2 or more asymptomatic typical PVCs, or 1 atypical PVC, on a 12-lead ECG, further evaluation with ambulatory monitoring and cardiac imaging is recommended.[292]
2b	C-EO	5. In athletes with 1 asymptomatic typical PVC (single outflow tract or fascicular morphology) on a 12-lead ECG, further evaluation may be considered.
1	C-EO	6. In athletes with PVCs of a single outflow tract or fascicular morphology, assessment of cardiac structure and function with echocardiography is recommended to exclude underlying pathology.
1	B-NR	7. In athletes with higher-risk ventricular arrhythmias and/or abnormal primary testing, comprehensive cardiac imaging including CMR and exercise stress testing is recommended to assess for underlying structural heart disease and behavior of PVCs with exercise.[88,313,314]
2b	C-LD	8. In adult athletes with ventricular arrhythmias with higher-risk but nondiagnostic features after comprehensive examination, EP study with voltage mapping may be useful in defining the extent and location of the arrhythmogenic substrate.[307,315]
1	C-EO	9. In athletes with ventricular arrhythmias with higher-risk features, withholding from sports participation pending evaluation is recommended.

Synopsis

Ventricular arrhythmias, including PVCs and ventricular tachycardia, can be diagnosed in athletes with a range of presentations from asymptomatic benign ectopy to SCD. For the athlete with PVCs, the challenge is to accurately differentiate between the healthy heart (inclusive of the spectrum of physiological changes associated with athletic training) as opposed to underlying structural heart disease that constitutes a risk for malignant arrhythmias.

Accurate diagnosis requires an assessment of clinical history and cardiac imaging in a facility sufficiently experienced with the range of EICR that can be observed in the well-trained athlete. Table 16 provides a hierarchical flow from simpler, less expensive, noninvasive tests through increasingly specialized examinations. Low-risk features are compared with higher-risk features that can assist in discriminating between the normal athlete and the athlete who may have prognostically significant structural or electrical heart disease. Higher-risk features will direct the extent and timing of evaluation. An algorithm of the recommendations for evaluating ventricular arrhythmias in athletes is shown in Figure 9.

Recommendation-specific supportive text

1. A 12-lead ECG is a critical initial assessment tool, as it provides information on the characteristics of the ventricular arrhythmia.[292,316–318] The morphology of the PVC enables the clinician to determine whether it is from a common and benign site (outflow tract or fascicular morphology) as opposed to complex PVCs of uncommon (non-outflow-tract) or multiple sites, that have a short-coupling interval (< 300 ms), or occur in multiples (couplets, triplets, or nonsustained ventricular tachycardia).[319] High-risk PVCs are defined as those with morphological as well as clinical features suggestive of higher risk of malignant prognosis (Table 16).
The suspicion of underlying cardiac pathology is increased in athletes in whom there are multiple consecutive ventricular beats (couplets, triplets, or nonsustained ventricular tachycardia) and/or multifocal PVCs.[282,292,300–302]
Although there are no studies specific to athletic populations, ambulatory ECG monitoring is important to determine the burden of PVCs given the association between very high burden ectopy (10% burden) and left ventricular dysfunction.[311,316] Longer-term monitoring may be needed if symptoms are less frequent. Features of available monitors are shown in Table 14.
2. Exercise stress testing is a valuable means of attempting to reproduce an athlete's symptoms and evaluate for the presence, location, and complexity of ventricular arrhythmias. There is some evidence to suggest that exercise stress tests of greater intensity and duration may be

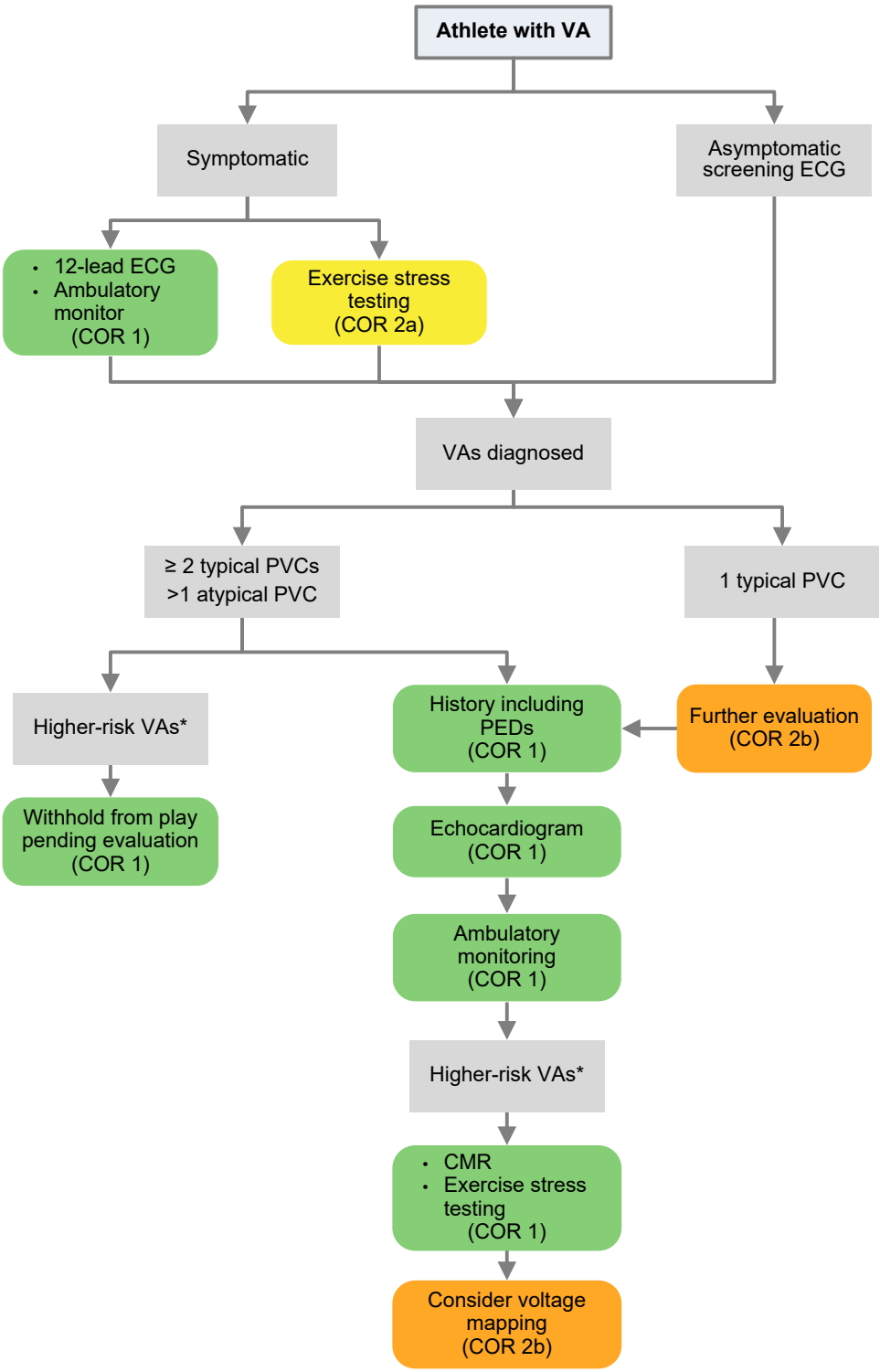

Figure 9
Evaluation of athletes with ventricular arrhythmias (VAs). Colors correspond to the class of recommendation (COR) in Table 1. *See text and Table 16 for definition of higher risk. CMR = cardiac magnetic resonance imaging; ECG = electrocardiogram; PED = performance-enhancing drug; PVC = premature ventricular contraction; RVOT = right ventricular outflow tract.

more sensitive in identifying ventricular arrhythmias[262] and that the disappearance of PVCs during exercise is less likely associated with structural heart disease,[312] whereas the appearance of PVCs during recovery may be a marker of poorer outcomes.[320] These observations are derived from general populations, and the specificity of these findings in athletic populations is unclear. Details of exercise stress testing in athletes appears in Section 2.3

3. Performance-enhancing drugs and supplements have the potential to trigger ventricular arrhythmias in athletes (see

Table 4).[321] Evidence supporting this assertion is largely limited to case studies and anecdotes in part because of the illicit nature of many substances used by athletes.[322] However, it is widely accepted that androgenic agents, stimulants including agents treating attention-deficit/hyperactivity disorder, agents interfering in hemoglobin synthesis or oxygen transit, and many other experimental agents can have arrhythmogenic effects. It is critical that the use of supplements and drugs be identified so that the athlete can be counseled as to the potential health effects.

4. Atypical or complex PVCs refer to ventricular ectopic beats that are not of RVOT or fascicular origin or are multifocal, have a short-coupling interval, or occur as multiples (couplets, triplets, or nonsustained ventricular tachycardia). Typical PVCs are those arising from the RVOT (left bundle branch block pattern with inferior axis) or fascicles (right bundle branch block pattern and QRS duration < 130 ms). Higher burden is associated with a higher likelihood of underlying heart disease.[292]

   Complex ventricular arrhythmias can be a marker of underlying structural heart disease. In some cases, abnormalities may be identified with echocardiography, but it is important to recognize that CMR may be necessary to identify subtle pathology. In particular, tissue characterization with delayed enhancement imaging can be used to identify myocardial scar even in the absence of overt wall-thinning of segmental functional abnormalities. In athletes, the identification of some scar patterns (particularly mid-wall or subepicardial left ventricular scar) have been associated with potentially life-threatening ventricular arrhythmias.[308,309,313] CMR is also critical in the setting of PVCs and mitral valve regurgitation on echocardiography to evaluate for ABiMVP.[172,323]

5. The evaluation of athletes with a single typical PVC is controversial because PVCs arising from the RVOT or fascicles are common and rarely associated with structural heart disease. However, the RVOT is also the most frequent site of origin of PVCs in ACM and can be the only manifestation of underlying disease.[324] Consideration should be given to the poor specificity of typical PVCs in identifying structural heart disease. When accompanied by other ECG abnormalities or any suspicion of symptoms, further investigation is warranted.

6. It is recognized that PVCs commonly originate from some cardiac sites of which the RVOT is most common. Although RVOT arrhythmias can be associated with pathologies such as ACM, in almost all settings this will be associated with other risk markers such as symptoms, family history, multiple ectopic morphologies, or abnormalities on ECG or echocardiography. Thus, it is reasonable that evaluation be restricted to a more limited evaluation in athletes with echocardiography alone in whom none of these red flags are present.

7. Table 16 describes higher-risk features that may indicate the need for more extensive evaluation to identify or rule out underlying structural heart disease.

   There have been few studies that have quantified the additional diagnostic yield of CMR as compared with echocardiography in athletes with ventricular arrhythmias, mostly because CMR has assumed the clinical standard of care in most settings. It may be reasonable to extrapolate from studies of ECG abnormalities in athletes in which echocardiography identified an underlying cardiomyopathy in 37 of 118 (31%) athletes with abnormal T-wave inversion and CMR identified a further 24 cardiomyopathies in the remaining 94 (26%) athletes in which the echocardiogram was considered normal.[325] If the echocardiogram suggests ABiMVP, CMR is needed to evaluate for features of ABiMVP including mitral annular dysjunction and late gadolinium enhancement indicative of scar.[172] In athletes in whom there is suspicion of cardiac sarcoid, positron emission tomography may aid in diagnosis.[326] In those with higher suspicion of coronary anomaly, computed tomography angiography can be indicated.[280]

8. Cardiac imaging has been combined with high-intensity exercise to increase the diagnostic sensitivity in the differentiation between healthy athletes and athletes with complex right ventricular arrhythmias. In athletes with right ventricular pathology, exercise imaging testing using echocardiography[327] and CMR[328] has been shown to unmask right ventricular dysfunction that was subtle or absent at rest. These observations are limited to a single-center experience and require specialist exercise imaging equipment and expertise. However, where available, exercise cardiac imaging could be considered as part of the thorough diagnostic workup of athletes in whom pathology is suspected.

   Stress testing is critical to evaluate for the presence of CPVT, as both resting ECG and structural imaging are normal. Stress testing protocols for athletes are described in detail in Section 2.3. Also, some data suggest that suppression of PVCs with exercise may portend a better prognosis,[316] while emergence of or increasing PVCs during recovery can raise concern.[312]

   Evidence supporting the use of myocardial biopsy in the evaluation of athletes with ventricular arrhythmias is confined to case reports or small observational studies.[292,315] Data on the clinical utility of signal-averaged ECGs are inconclusive, and thus this modality is rarely used in clinical practice.[8]

9. The extent and location of myocardium with abnormal EP (arrhythmogenic foci or low-voltage regions) can assist in diagnosis, risk stratification, and planning of therapy. Corrado et al[315] used electroanatomical mapping to identify low-voltage regions in the RVOT that were then used to guide endocardial biopsies for the confirmation of ACM in cases (inclusive of athletes) in which other disease features were absent or subtle. Venlet et al[307] described electrophysiological characteristics that appeared unique to highly trained endurance athletes. In a cohort of consecutive patients with right ventricular tachycardia, athletes were observed to have RVOT scar from which very rapid ventricular tachycardia could be readily induced. Furthermore, these arrhythmias

could be successfully treated with epicardial ablation.[307] Heidbuchel et al[304] included an invasive EP study in their evaluation of athletes presenting with right ventricular arrhythmias and determined that ventricular tachycardia inducibility was the only test that was predictive of a subsequent major arrhythmic event. In a study by Dello Russo et al,[329] electroanatomic mapping and endomyocardial biopsy increased diagnostic yield in athletes with complex ectopy and nondefinitive noninvasive evaluation.

A high-dose isoproterenol challenge may be a useful adjunct during an EP study, as demonstrated in a single-center experience of patients presenting with PVCs or suspected ACM.[330,331] The induction of polymorphic PVCs with ≥ 1 couplet or non-RVOT ventricular tachycardia following isoproterenol infusion accurately identified patients with ACM. Although this procedure has not been specifically studied in athletic populations, it would seem a reasonable addition to a standard EP study in athletes with right ventricular arrhythmias, especially if there are any additional features raising the possibility of ACM.

10. As above, these higher-risk features indicate a higher likelihood of underlying structural or electrical disease that may be life-threatening. For athletes with many of these entities, appropriate treatment can decrease risk of death. Informed shared decision-making around treatments and return to play for athletes with these entities requires diagnosis, and thus return to play should be delayed to allow for this informed process.

6.2 Treatment of ventricular arrhythmias in the athlete

6.2.1 Treatment of benign ventricular arrhythmias in the athlete

Benign PVCs and benign idiopathic ventricular tachycardia are defined as those occurring in the absence of structural heart disease, identified electrical disease, or high-risk electrical features, as noted above. As described in Section 6.1, in athletes with ventricular arrhythmias, a comprehensive approach is required to evaluate for an underlying etiology and to appropriately risk stratify into benign or high-risk types. Treatment of benign ectopy, once benign nature is confirmed, is determined by symptoms and burden.

Recommendations for treatment of benign ventricular arrhythmias in the athlete

COR	LOE	Recommendations
1	B-R (caffeine, sleep apnea) C-EO (other risk factors)	1. In athletes with symptomatic benign ventricular arrhythmias, risk factor management, including avoidance of performance-enhancing or illicit drugs, weight loss, treatment of obstructive sleep apnea, smoking cessation, alcohol and caffeine avoidance, and hypertension management, as well as reassurance is recommended.[332–335]
1	C-EO	2. In the asymptomatic athlete with benign ventricular arrhythmias, observation is recommended.
1	C-LD	3. In the asymptomatic athlete with a high burden of benign PVCs (> 10% burden) in the absence of structural heart disease, active monitoring for PVC-induced cardiomyopathy with serial imaging and ambulatory monitoring is recommended.[336]
1	B-R	4. In athletes with symptomatic benign ventricular arrhythmias desiring treatment, catheter ablation is useful as first-line therapy, or if antiarrhythmic drugs are contraindicated or poorly tolerated.[337–344]
2a	B-R	5. In athletes with symptomatic benign ventricular arrhythmias, a trial of medical therapy with beta blockers or calcium channel blockers is reasonable.[345–347]
2a	B-R	6. In athletes with symptomatic benign ventricular arrhythmias, antiarrhythmic drug therapy with class IC agents is reasonable.[348,349]
2a	C-EO	7. In athletes with benign ventricular arrhythmias, stress testing after treatment with either ablation or medications can be useful to determine arrhythmia suppression.

Synopsis

Management of ventricular arrhythmias in the athlete is largely similar to that in the nonathlete population. In those considered low risk with benign ventricular arrhythmias, close follow-up and reassurance with treatment or change in exercise regimen may be pursued, as spontaneous reduction may occur. In those who require treatment for benign PVCs or benign idiopathic ventricular tachycardia due to ongoing symptoms, medical therapy may be poorly tolerated due to detrimental impact on exercise performance; therefore, there may be a lower threshold to consider catheter ablation as first-line therapy in athletes. An algorithm of the recommendations

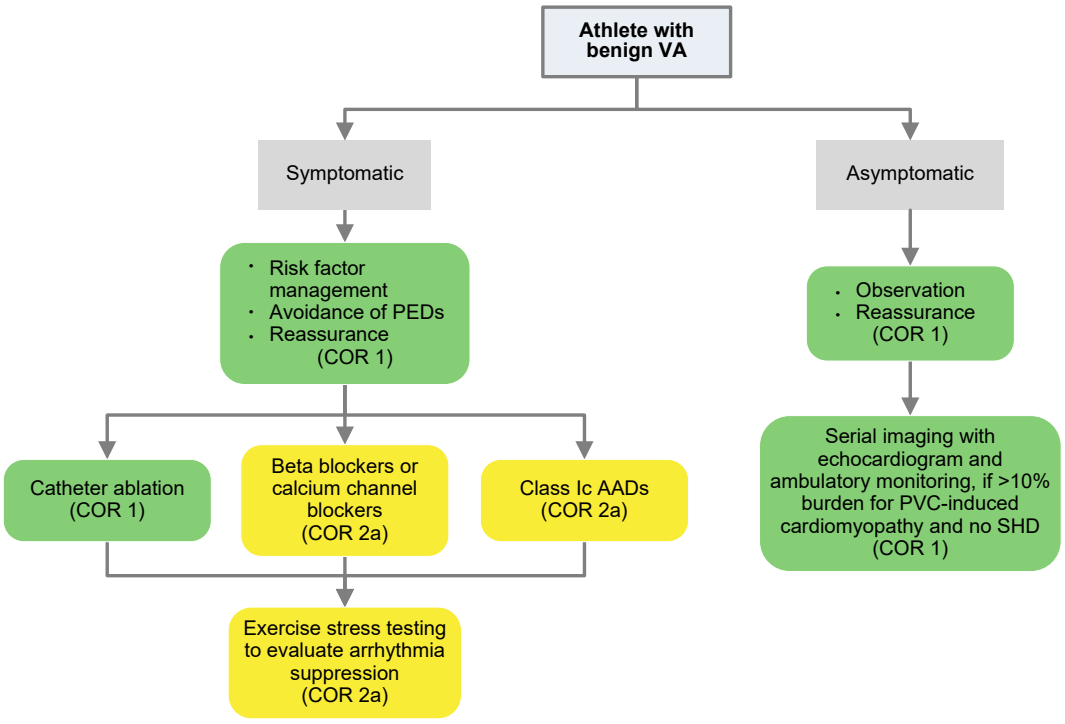

Figure 10
Algorithm for the treatment of athletes with benign ventricular arrhythmias (VAs). Colors correspond to the class of recommendation (COR) in Table 1. AAD = antiarrhythmic drug; PED = performance-enhancing drug; PVC = premature ventricular contraction; SHD = structural heart disease.

for treating benign ventricular arrhythmias in athletes is shown in Figure 10.

Recommendation-specific supportive text

1. Modifiable factors include common risk factors observed in the nonathlete population, such as hypertension, smoking, and sleep apnea, while specific risk factors likely more relevant to the athlete include use of performance-enhancing drugs and stress from poor recovery and sleep deprivation.[318,321,332,333,350,351] The use of cannabis and association with PVCs has not been described, although it is reasonable to recommend cessation to evaluate response. For caffeine and sleep apnea, data suggest that modifying these risk factors decreases PVCs. For other factors, trials of discontinuation of known predisposing factors are warranted, although data on intervention are sparse.[332,335] Reassurance on the benign nature with favorable prognosis should be provided, along with education on symptoms that may be attributed to ventricular arrhythmias that would warrant future evaluation. Reassurance of the benign nature may decrease anxiety as well as perceived symptoms.
2. In athletes who are found to have benign ventricular arrhythmias after evaluation and are asymptomatic, treatment is not indicated and should not be given.
3. Although there is no single threshold burden cutoff, observational studies have suggested that highest risk of PVC-induced cardiomyopathy is at a burden of $\geq$ 20%, although it may occur with a burden of > 10%.[311,336,337] Serial monitoring with an echocardiogram will evaluate for left ventricular dysfunction or dilation for evaluation of PVC-induced cardiomyopathy and ambulatory monitoring for changes in PVC burden. There is no accepted timeframe for monitoring, as it will depend on ongoing burden of ventricular arrhythmias and clinical symptoms; a repeat at 6 months and then every 1-2 years may be reasonable.
4. In a meta-analysis including 5 studies with only 1 randomized controlled trial including 1113 patients, those that underwent catheter ablation had a lower burden of PVCs in follow-up as compared with antiarrhythmic drug therapy.[340] The study highlighted the high heterogeneity in PVC morphologies included, mapping and ablation technology utilized, and follow-up evaluation of PVC burden. Complication rates from ablation ranged from of 0% to 5.6%, while adverse effects from antiarrhythmic drugs ranged from 9.5% to 21%.

   The location of idiopathic ventricular arrhythmias impacts the success rate. Those with ventricular arrhythmias originating from the RVOT have reported success rates approaching 80% to 95% with limited complication rates.[338,344,352] Ablation of ventricular arrhythmias originating from the left ventricular outflow tract (LVOT) are more complex, as these may require ablation in adjacent structures, such as epicardial ablation in the coronary venous vasculature.[341] Less common locations include right ventricular and left ventricular papillary muscles, with repeat procedures required in approximately 30% of cases.[343] Locations involving the fascicular system or parahisian

locations can also be successfully ablated in 70%-90%; however, the risk of AV block needs to be carefully considered.

5. Beta blockers and calcium channels blockers are effective at reducing symptoms attributed to ventricular arrhythmias and may reduce burden in approximately one-third of patients.[348] Antiarrhythmic drugs, specifically the class IC agent flecainide, have shown superior efficacy in reducing PVC burden as compared with the beta blockers.[349] Verapamil has shown to be effective in fascicular ventricular tachycardia.[353] When considering this option, it is important to discuss with the athlete that beta blockers and nondihydropyridine calcium channel blockers may lead to fatigue[354] or impact exercise performance, although a low or moderate dose may be tolerated in some athletes. For those choosing this option, follow-up to determine any adverse effects is needed. Importantly, beta blockers are prohibited by the World Anti-Doping Agency only in sports that rely on stability of the upper extremities, such as archery, golf, or shooting.[46]
6. Antiarrhythmic drugs, specifically the class IC agent flecainide, have shown superior efficacy in reducing PVC burden as compared with beta blockers. In a study including 103 participants with outflow tract PVCs (burden $\geq$ 5%) randomized to carvedilol versus flecainide, overall PVC burden decreased in both groups with superior efficacy in flecainide (20.3% to 14.6% with carvedilol versus 17.1% to 6.6% with flecainide, $p < 0.0001$).[349] In an observational prospective study including 120 patients with frequent PVCs ($\geq$ 5%), the median relative reduction of PVCs was 32.7%, 30.5%, and 81.3%, in the conservative therapy, beta blockers/calcium channel blockers, and antiarrhythmic groups, respectively.[348] Only one-third achieved complete cessation of PVCs in the antiarrhythmic drug group. Taken together, antiarrhythmic drug therapy results in a greater reduction of PVC burden often without complete cessation. Exercise stress test should be performed after initiation of class IC agents to monitor for use-dependent QRS widening. Confirmation of no over inducible ischemia is also important prior to initiation. In a study of serial testing, when an initial stress test did not preclude use of flecainide, later development of QRS widening was not found.[355]
7. As these arrhythmias are benign, symptoms, rather than exercise results, will guide return to play. However, as benign PVCs can be exercise-induced as above, stress testing as part of evaluation of efficacy of treatment may be helpful.

6.2.2 Treatment of complex ventricular arrhythmias in the athlete

Recommendations for complex ventricular arrhythmias

COR	LOE	Recommendations
1	C-EO	1. In the athlete with complex ventricular arrhythmias, management based on underlying pathology is recommended, with consideration of treatment impact on exercise performance, and shared decision-making to individualize treatment strategy.
1	A	2. In athletes who have survived sustained ventricular tachycardia in the absence of a reversible cause, an ICD is recommended, based on underlying pathology.[189,356]
1	B-NR	3. In athletes with monomorphic ventricular arrhythmias with underlying structural heart disease including inherited entities and coronary artery disease, catheter ablation for arrhythmia suppression is useful as first-line therapy or when antiarrhythmic drug therapy is contraindicated or has failed.[357–361]
1	B-NR	4. In athletes with VF triggered by monomorphic PVCs, catheter ablation is recommended.[362–366]
1	C-EO	5. In athletes with complex ventricular arrhythmias, documentation of suppression of arrhythmias with a maximal exercise stress test is recommended prior to return to play.
1	B-NR	6. In athletes with suspected PVC-induced cardiomyopathy, catheter ablation is useful as first-line therapy or when antiarrhythmic drug therapy is contraindicated or has failed regardless of symptoms.[337,344,367–369]
2a	B-NR	7. In athletes with suspected PVC-induced cardiomyopathy, medical therapy (antiarrhythmic drug therapy, as well as guideline-directed medical therapy for decreased ejection fraction) is reasonable to improve left ventricular function regardless of symptoms.[370]
2a	C-LD	8. In athletes with ventricular arrhythmias and nonfamilial and/or genotype-negative phenotype-positive ARVC (presumed exercised induced), catheter ablation and/or ICD is reasonable after appropriate risk stratification.[307]
3: Harm	B-NR	9. In athletes with ventricular arrhythmias and nonfamilial and/or genotype-negative phenotype-positive ARVC (presumed exercised induced), continuation of vigorous endurance sports is harmful.[304,305,307]

Synopsis

Athletes with complex ventricular arrhythmias, such as those with polymorphic or sustained patterns or those with underlying pathology such as cardiomyopathies, myocarditis, and inherited arrhythmia disorders, require appropriate risk assessment and directed treatment approaches; these entities have important implications on exercise recommendations. Medical therapy with beta blockers, calcium channel blockers, and antiarrhythmic drugs may be considered, although catheter ablation has been shown to be more effective than medical therapy in most cases. Device therapy recommendations, including transvenous and subcutaneous ICDs, follow recommendations similar to those for nonathletes for primary and secondary prevention. For all entities underlying complex arrhythmias, both sudden death prevention and arrhythmia suppression are critical prior to return to play, including confirmation of suppression of arrhythmia during exercise. Understanding of the impact of exercise on ventricular arrhythmias continues to evolve. An algorithm of the recommendations for the management of athletes with complex ventricular arrhythmias is shown in Figure 11.

Recommendation-specific supportive text

1. While most athletes with ventricular arrhythmias will have no underlying cardiac pathology, the management of those with structural or electrical heart disease depends on the specific etiology. Ventricular arrhythmias associated with ARVC forms of ACM, HCM, myocarditis, or genetic arrhythmias including Brugada or CPVT may localize to the right ventricular free wall or left ventricular endocardium, have a polymorphic or repetitive pattern, may exacerbate with exercise, and put the athlete at higher risk of SCD. The role of PVC burden as a predictor of risk varies based on underlying disease. For example, even > 500 PVCs/hour is considered a minor criterion for ARVC.[371] Thorough evaluation and shared decision-making involving the potential utility of EP study, catheter ablation, medical therapy, device therapy, and change in exercise regimen are needed on an individual basis.
2. The decision process for implantation of an ICD follows that for nonathletes.[8] For most underlying etiologies of sustained ventricular tachycardia (after excluding benign idiopathic ventricular tachycardia; see Table 16), clinical trial data have demonstrated a reduction in the risk of death driven by reduction in arrhythmic death in those with an ICD. In most cases, only a single lead device is required, and decision to proceed with a transvenous versus subcutaneous ICD relies on multiple factors, including patient body size, age, athlete's sport, and need for pacing or antitachycardia pacing in the setting of sustained monomorphic ventricular tachycardia. Implanting transvenous leads at a young age portends a higher risk of lead damage over the lifetime and complexity and morbidity with extraction. Sports with extreme ipsilateral arm movements (golf, tennis, swimming) may increase the risk of transvenous lead damage or dislodgement and may favor subcutaneous ICD. Detailed discussion regarding device choice and return to play with an ICD appears in Section 3.5
3. In addition to prevention of sudden death with an ICD, athletes with ventricular tachyarrhythmias may need arrhythmia suppression. In the general population, ablation has been

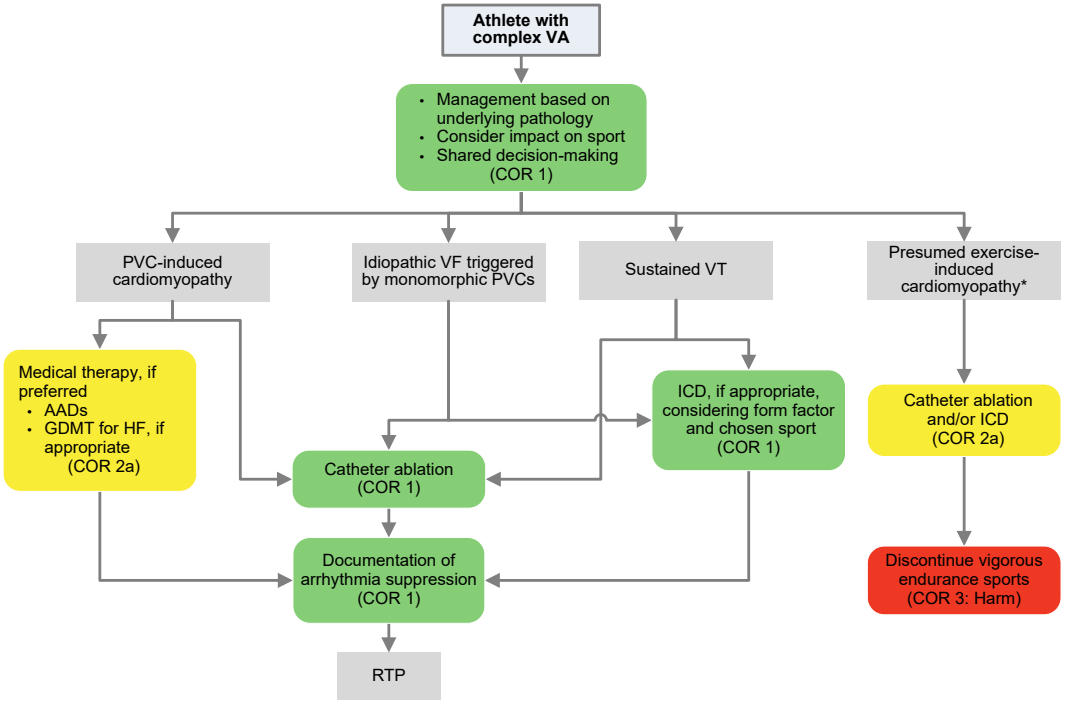

Figure 11

Algorithm for the treatment of athletes with complex ventricular arrhythmias (VAs). Colors correspond to the class of recommendation (COR) in Table 1. *Genotype-negative phenotype-positive arrhythmogenic right ventricular cardiomyopathy. AAD = antiarrhythmic drug; AVN = atrioventricular node; GDMT = guideline-directed medical therapy; HF = heart failure; ICD = implantable cardioverter-defibrillator; PVC = premature ventricular contraction; RTP = return to play; VF = ventricular fibrillation; VT = ventricular tachycardia.

demonstrated as superior to alternative strategies for suppression of ventricular arrhythmias.[358,372] The outcomes of ventricular arrhythmia ablation depend on the underlying substrate. Those with ARVC forms of ACM or myocarditis have observed lower risk of ventricular tachycardia recurrence following ablation than those with HCM or sarcoidosis.[357] Superior outcomes have also been observed with a combined endocardial and epicardial ablation approach, such as in those with ARVC/ACM and HCM, and this approach should be performed in centers with appropriate expertise.[359] Ablation alone does not improve survival in those with underlying structural or electrical heart disease but is useful for suppression of arrhythmia.[360,361] Arrhythmia suppression is one factor to be considered prior to return to play. For some entities, such as ACMs, other considerations also need to be addressed, as discussed in detail in Section 7.

4. VF can be caused by short-coupled PVCs from fascicular system, outflow tracts, moderator band, papillary muscles, or, less commonly, the ventricular myocardium, in the absence of structural heart disease or genetic arrhythmia syndromes.[362,363] Ablation can be highly successful for arrhythmia suppression in those with monomorphic and frequent PVCs, although recurrence may occur requiring repeat ablation. ICD is also generally considered for SCA prevention.[364–366,373] In those with unsuccessful ablation, polymorphic PVCs, or patient preference to avoid invasive procedures, antiarrhythmic drug therapy with quinidine may be considered, as it has demonstrated high short-term success rates based on observational studies.[366,374]
5. For athletes in whom a comprehensive shared decision-making approach has led to a decision to return to play, arrhythmia suppression should be documented prior to their return. Athletes with complex ventricular arrhythmias suppressed with either medical therapy or ablation should undergo evaluation to document suppression during exercise with an exercise stress test. Details on stress testing in athletes appear in Section 2.3 Underlying specific heart conditions also influence the decision to return to play and must be considered on an individual basis. In cases of myocarditis, it is reasonable to return to competitive sport at a minimum of 3 months after repeat imaging to demonstrate resolution along with demonstration of suppression of ventricular arrhythmias.[375,376]
6. Left ventricular dysfunction has been associated with high PVC burden (generally > 10% and usually > 20%). In prospective studies of patients with high-burden idiopathic PVCs, left ventricular function normalized in approximately 80% of patients with catheter ablation.[344,367] In some cases, such as those with an epicardial origin, recovery of ventricular function may be delayed over a year.[368]
7. In those for whom ablation is not preferred or ineffective, adequate pharmacological suppression of PVCs in those with suspected PVC-induced cardiomyopathy can lead to recovery. In a retrospective study of 20 patients with PVC-induced cardiomyopathy treated with class IC antiarrhythmic drugs, PVC burden decreased from an average 36% to 10% and mean left ventricular ejection fraction (LVEF) increased from 36% to 49% over nearly 4 years of treatment with no adverse effects.[370] As PVC-induced cardiomyopathy is often a diagnosis of exclusion, guideline-directed medical therapy for heart failure with reduced ejection must also be included in the medical regimen.
8. Athletes with high volumes of high-intensity exercise with predominately right ventricular enlargement and dysfunction with no desmosomal variant presenting with symptomatic ventricular arrhythmias are candidates for an EP study and cardiac ablation with high likelihood of success.[305] Although most arrhythmias originate in the right ventricle, epicardial ablation may be required.[307] In those with sustained ventricular tachycardia that is not treated effectively with ablation and/or those with sustained cardiac arrest, an ICD should be placed in accordance with evidence-based criteria for secondary prevention of SCD.[189,356]
9. For those diagnosed with exercise-induced ARVC or genotype negative ARVC,[306] as described above, observational data have suggested that detraining may result in resolution of ventricular arrhythmias and structural changes.[377] As studies are still limited on this diagnostic entity, shared decision-making discussions regarding management decisions for ongoing evaluation of ventricular arrhythmias, further risk assessment of SCD, and serial imaging should be pursued.

Section 7 Inherited arrhythmias and cardiomyopathies

7.1 Athletes with inherited arrhythmia syndromes

Inherited arrhythmia syndromes (IAS), including LQTS, Brugada syndrome, CPVT, and short QT syndrome (SQTS), have been identified in series of sudden deaths in athletes.[79,91] Previously, this resulted in disqualification for most IAS-positive athletes from most sports, even in those with only a positive genetic test result and no phenotypic expression.[9,378] However, in 2015, the AHA/ACC sports participation guidelines acknowledged and enabled return to play with a model of shared decision-making for IAS-positive athletes.[5,58] This was catalyzed by observational data demonstrating very low rates of IAS-associated breakthrough cardiac events after diagnosis and institution of guideline-directed and genotype/patient-tailored therapies for athletes with LQTS[26,194,379–384] and athletes with CPVT.[26,383,385] Accordingly, after expert evaluation and treatment, athletes with IAS are increasingly returning to play in a shared decision-making model under the guidance of their genetic cardiologist or sports cardiologist who has direct expertise with their specific IAS. These recommendations aim to assist physicians with treatment and management of athletes with IAS, before and after return to play.

Recommendations for athletes with inherited arrhythmia syndromes		
COR	LOE	Recommendations
1	C-LD	1. In athletes with IAS, an assessment by an expert in genetic cardiology and a shared decision-making model of care is recommended.[194,381]
1	B-NR	2. In athletes with a positive genetic test for IAS, comprehensive assessment and cardiac testing are recommended to determine the risk category.[41,386–388]
2a	B-NR	3. In athletes with LQTS or CPVT in whom beta blocker therapy leads to decreased performance in their sport and/or subsequent quality-of-life issues, left cardiac sympathetic denervation (LCSD) can be effective.[389–391]
2a	B-NR	4. In athletes with IAS who have received a clinically indicated ICD, sports participation is reasonable.[192,193,392,393]
3: Harm	C-LD	5. In athletes with IAS, ICD implantation for the sole purpose of return to play is potentially harmful and should not be done.[394]

Synopsis

Prior to return to play, it is critical that athletes undergo assessment by an expert in genetic cardiology, with appropriate cardiac testing to guide treatment and risk assessment and inform shared decision-making, as shown in Figure 12. The desire to return to play without loss of athletic performance may be a factor in treatment decisions. For athletes with IAS in whom ICDs are clinically indicated, return to play after appropriate treatment is reasonable, but as ICDs are not without risks, they should not be implanted for the sole purpose of return to play.

Recommendation-specific supportive text

1. IAS are complex conditions and can vary widely in their clinical manifestations and potential risks. Given the intricate nature of these syndromes, a thorough evaluation by a specialist in genetic cardiology is crucial to accurately diagnose the specific syndrome and determine its implications for an athlete's health. While exercise training can lengthen repolarization,[35] a prolonged QT requires evaluation. This assessment involves evaluating both genetic and clinical features including personal and family history and test findings. By conducting a comprehensive risk assessment, the expert can guide appropriate risk management strategies, to allow informed decision-making around lifestyle modifications, medication, or even restrictions on certain physical activities. As described in detail in Section 2.3, a shared decision-making model empowers the athlete to actively participate in decisions about their health care, ensuring that the chosen interventions align with their personal values, preferences, and goals. Athletes often have unique goals and aspirations related to their sport. Balancing these aspirations with the potential health risks associated with IAS requires careful consideration. A shared decision-making model allows the athlete to collaborate with the health care provider in making informed choices that optimize both their athletic pursuits and their long-term well-being.[9,194,395]
2. Athletes who have been identified as having a positive genetic test for an IAS require comprehensive clinical evaluation, with a minimum of ECG and maximum capacity stress testing (LQT/CPVT) with or without high-lead ECG (Brugada syndrome) and Holter monitoring to determine their phenotypic disease expression and guide treatment and for risk assessment before they return to play. Various clinical parameters are known to confer differing levels of risk, including specific genotype, QTc duration, disease expression, symptoms, and documentation of arrhythmias.[41,386–388,396]
3. Beta blockers can lead to side effects including fatigue and reported decreased athletic performance in some athletes.[354] In athletes with LQTS or CPVT, LCSD monotherapy has been shown to be a safe and effective therapeutic option when performed in experienced centers,[389] with the largest cohort of 64 patients demonstrating a low nonlethal recurrent event rate of 5% with no surgical complications at 2.7 years mean follow-up.[391] LCSD performed in high-volume centers has demonstrated effective reduction of arrhythmias and a lower risk of adverse events. A recent study reviewed long-term follow-up for 125 patients (mean follow-up of 12.9 $\pm$ 10.3 years) and demonstrated an overall 86% decrease in mean yearly event rate with no major complications and a low minor complication rate.[390] While rates of significant complications are low, and quality of life not adversely impacted by LCSD,[391,397] impact on athletic performance has not been reported.
4. Data from the ICD multinational sports registry show that competitive and recreational sports participation for athletes with ICDs can be safe, including for those who participate in vigorous and competitive sports; however, they should be informed they are at risk of both appropriate and inappropriate shocks.[192,392,393] In athletes with IAS with clinical evidence of high risk for SCD and benefit from ICD based on standard clinical criteria, the programming of the ICD should include consideration for the individual's age, sport, and specific disease phenotype (see Section 3).

5. Although lifesaving in those at significant risk of SCA, ICDs are not without risk. A meta-analysis[394] of 4916 young patients with IAS and ICDs showed a 20% rate of inappropriate shocks (annual rate of 4.7% per year) as well as a 22% rate of ICD-related complications (4.4% per year) and a 0.5% ICD-related mortality (0.08% per year).

7.1.1 Athletes with long QT syndrome

Recommendations for athletes with long QT syndrome

COR	LOE	Recommendations
2a	B-NR	1. In athletes with LQTS under expert assessment and supervision, return to play is reasonable in a shared decision-making model after risk assessment, education, and initiation of appropriate therapies.[26,194,379–382,398]
1	B-NR	2. In athletes with LQTS, review and/or cessation of medications known to prolong the QT interval is recommended, and whenever possible, prevention and correction of electrolyte disturbances are recommended.[399,400]
1	B-NR	3. In athletes with asymptomatic LQTS and a normal corrected QT interval (concealed variant-positive LQTS), initiation of QT-related preventative measures is recommended prior to return to play.[399,400]
2a	B-NR	4. In athletes with asymptomatic LQTS and a corrected QT interval < 470 ms, therapy with beta blockers can be useful.[387]
1	B-NR	5. In athletes with LQTS with symptoms and/or a corrected QT interval > 470 ms, guideline-directed and genotype/patient-tailored therapy with medications, LCSD, and/or device therapy should be optimized fully before return to play.[26,387,390,401]
1	B-NR	6. In athletes with LQTS on beta blocker therapy, nonselective beta blockers (especially nadolol and propranolol) are recommended, with dosing tailored to the patient's risk profile and response to therapy.[387]
2a	B-NR	7. For athletes with LQTS and severe bradycardia, other treatment configurations besides beta blockers (eg, alternative medical therapy, LCSD, and device therapy) are reasonable.[26,389–391]
2a	B-NR	8. In athletes with LQTS (including type 1), participation in swimming/diving is reasonable with appropriate precautions.[402]
1	B-NR	9. In athletes with LQTS who are unable to tolerate beta blockers or who have ongoing events on beta blockers, treatment intensification with medication, LCSD, and/or device therapy should be done and reoptimized fully prior to return to play.[26,390,401,403]

Synopsis

Despite a paucity of scientific data, athletes with LQTS have historically been recommended to avoid competitive sports. In more recent iterations of guidelines, such as the AHA/ACC sports participation guidelines, as observational data has emerged showing a low rate of events for athletes with LQTS, there has increasingly been an acceptance that in a shared decision-making model, return to play for athletes with LQTS can be enabled. The current recommendations incorporate recent observational studies demonstrating low rates of breakthrough cardiac events for athletes who are managed in a specialized clinic. Figure 12 highlights the recommended approach for athletes with LQTS. Athletes should be assessed at a specialized center and managed as part of a shared decision-making model where their personalized risk and management decisions are optimized. Athletes with LQTS breakthrough events require reassessment and optimization of therapies, which may include medications, LCSD, and devices before considering return to play.

Recommendation-specific supportive text

1. Athletes with LQTS who are managed in a specialized center may return to play as part of a shared decision-making model following an expert assessment and discussion of risk. Prior to return to play, a plan should be in place including access to an AED and consideration for purchasing their own personal AED and having an individualized EAP. The largest cohort to date, from Mayo Clinic, including 494 athletes with LQTS returning to play after risk assessment and personalized treatment plans from a specialized genetic heart disease team, showed no deaths and a low event rate of 1.16 nonlethal events per 100 athlete-years follow-up.[26] In a recent French cohort, they also showed no deaths and a low event rate of 0.0007/year after diagnosis, with no events in any competitive athlete, and no events in patients treated with beta blockers.[380] Smaller series have shown similar findings.[379,398]
2. It is important that athletes with LQTS minimize risk through a thorough physician-led review for known triggers for QT prolongation and torsade de pointes, including QT-prolonging

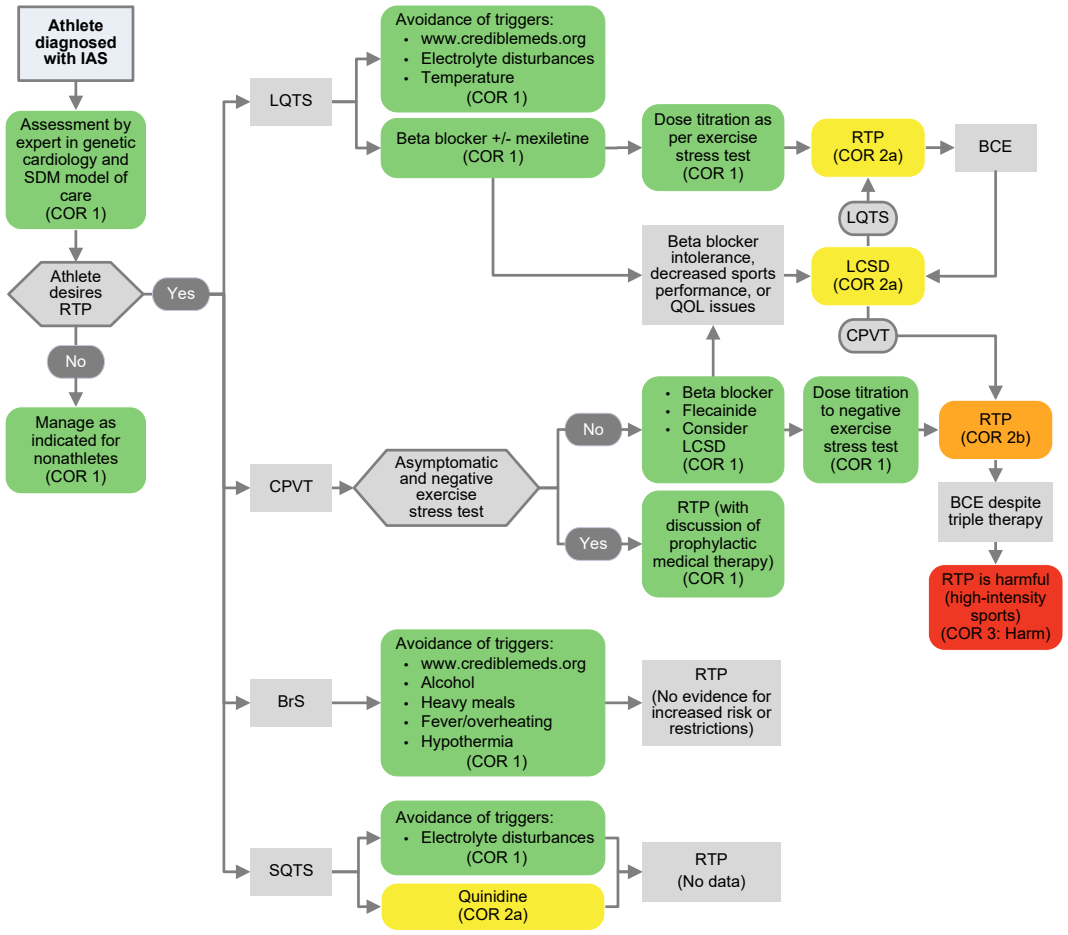

Figure 12

Algorithm for the management and treatment of inherited arrhythmias. Colors correspond to the class of recommendation (COR) in Table 1. BCE = breakthrough cardiac event; BrS = Brugada syndrome; CPVT = catecholaminergic polymorphic ventricular tachycardia; IAS = inherited arrhythmia syndrome; LCSD = left cardiac sympathetic denervation; LQTS = long QT syndrome; RTP = return to play; SQTS = short QT syndrome; QOL = quality of life.

medications (www.crediblemeds.org) and electrolyte disturbances (particularly hypokalemia and hypomagnesemia). They should avoid training-related heat exhaustion and heat stroke (particularly LQT2), take caution with regard to over-the-counter medications (including supplements), and receive counseling around avoidance of illicit substances.[396,399,400]

3. There are no current data to support exercise restrictions in athletes with LQTS who have no symptoms and a normal corrected QT interval (concealed variant-positive LQT athletes). A single-center study[383] demonstrated a low event rate of 0.3 nonlethal events per 100 patient-years for low-risk patients. Intentional nontherapy is a valid option in this lower-risk cohort, with a study of 55 asymptomatic low-risk patients with LQTS with a mean QTc 448 ms and managed with conservative preventative measures only, including avoidance of QT-prolonging medications and potential electrolyte disturbance, demonstrating no events at mean follow-up of 7.5 years.[404]
4. In athletes with QTc < 470ms, data from a large Italian cohort[387] show that the risk of life-threatening arrhythmias is very low in all genotypes for QTc < 460 ms (< 1% 5-year risk off therapy) and low for QTc 460-470 ms (< 3% for LQT1 and LQT2 and 3%-6% for LQT3 5-year risk off therapy). In these individuals, conservative preventive measures with intentional nontherapy can be considered in an individualized shared decision-making discussion.[399,400,404] The initiation of beta blockers in those with indication of higher risk of arrhythmia such as LQT2/3 genotypes or those at the upper end of the QTc range (< 460-470 ms) can confer further arrhythmic protection.[387]
5. In athletes with symptomatic LQTS or QTc prolongation > 470 ms, treatment with nonselective beta blockers, particularly nadolol or propranolol, has been shown to reduce the risk of ventricular arrhythmias.[387,405] The dose of beta blockers can be titrated according to exercise stress testing performed 3 months after commencement of therapy, with an aim to reduce mean heart rate by 15%-20%, or aiming for 0.8-1.5 mg/kg nadolol.[25,43] In patients who are confirmed genotype LQT3 (and selected individuals with LQT2), there is evidence to support the use of mexiletine either in conjunction with beta blockers or as monotherapy in athletes unable to tolerate beta blockers.[401,403] In some higher-risk athletes—

based on genotype, QTc interval, and clinical history[396] or those with documented arrhythmias requiring escalation of therapies—LCSD and ICD are further adjunctive therapies. LCSD has been shown to be a safe and effective therapeutic option in a large LQT cohort of 125 patients, with an overall 86% decrease in mean yearly event rate with no major complications and a low minor complication rate of ptosis of 2.4%.[390] Slow beta blocker dose titration may help achieve appropriate dosage with fewer side effects. Individuals should be stable on therapy for a duration of 3 months with no breakthrough arrhythmias to ensure adequacy of current therapy prior to returning to play.

6. In athletes with LQTS on beta blockers, data from a large Italian cohort including 1710 LQTS patients followed for median 7.1 years demonstrated that treatment with nonselective beta blockers, particularly nadolol, is superior to other beta blockers in preventing arrhythmias across all LQT genotypes (HR0.38).[387,405] When nadolol is not available, propranolol would be an appropriate substitute beta blocker.
7. Athletes with LQTS may be unable to tolerate beta blockers due to severe bradycardia-associated symptoms. In these athletes, therapeutic options include active nontherapy (in very-low-risk individuals), alternative medical therapy (QT-preventive therapies, or mexiletine for LQT2/3), LCSD monotherapy, device implantation, or a combination of these options. LCSD has been shown to be a safe and effective option for monotherapy.[389] The largest published LCSD monotherapy cohort of 64 patients demonstrated a low nonlethal recurrent event rate of 5% with no surgical complications at 2.7 years mean follow-up.[391] Device implantation may be considered in this cohort, with studies demonstrating some benefit from intentional atrial pacing particularly for higher-risk individuals with LQT2 (reduction in event rate from 1.01 breakthrough cardiac events per year to 0.02 breakthrough cardiac events per year),[406] while ICD implantation for higher-risk individuals unable to tolerate beta blockers, particularly those with symptomatic LQT2, can be considered.[26,383] While most LQT patients receiving a device will benefit from both pacing and defibrillation capability, rarely, pacing alone may provide sufficient protection.[406]
8. Swimming/diving in athletes with LQT1 has historically required careful consideration due to prior data describing swimming as a genotype-specific trigger with increased risk of ventricular arrhythmias and SCD.[402,407] However, the swimming-LQT1 connection is most established in those individuals who are previously undiagnosed and therefore untreated. For LQT1 athletes whose competitive sport of choice is swimming, development of a personalized management plan including purchase of a personal AED to be in the area of the athlete while swimming, avoidance of swimming alone, and a preference for swimming in pools rather than in open water are considerations for the athlete in a shared decision-making model of care.
9. LCSD has been shown to be an effective additional therapy at reducing further breakthrough cardiac events for those who have recurrent ICD shocks or cardiac events on beta blocker therapy. For athletes unable to tolerate beta blockers despite a gradual loading phase, LCSD has been effective as monotherapy in selected LQTS populations as above. In a large international cohort, patients with a QTc > 500 ms have a 50% chance of QT reduction by 60 ms, and 86% reduction in mean yearly event rate after LCSD.[390] Mexiletine has been shown to reduce the QT interval by a mean of 63 ms and reduce annual event rate to 0.7% per year in a small cohort of 34 patients with LQT3 and is an appropriate adjunctive therapy or monotherapy for athletes with LQT3. There are also limited data showing that mexiletine can shorten the QT interval for patients with LQT2.[403]

7.1.2 Catecholaminergic polymorphic ventricular tachycardia

Recommendations for athletes with catecholaminergic polymorphic ventricular tachycardia

COR	LOE	Recommendations
1	C-LD	1. In athletes with asymptomatic CPVT and a negative exercise stress test (genotype-positive phenotype-negative), return to play is recommended with discussion of prophylactic CPVT-directed medical therapy.[26,383,385,408]
1	B-NR	2. In athletes with asymptomatic CPVT and a positive exercise stress test, a CPVT-directed medical treatment program guided by normalization of the stress test is recommended before considering return to play.[26,41,383,385]
2b	C-LD	3. In athletes with previously symptomatic CPVT while not on therapy, return to play may be considered after establishing and confirming appropriate therapy.[383]
1	C-LD	4. In athletes with previously symptomatic CPVT for whom return to play is being considered, combination therapy with beta blocker and flecainide, and consideration of triple therapy with LCSD, is recommended before return to play, with a goal of optimizing therapy to normalize the exercise stress test.[41,383]
3: Harm	C-EO	5. In athletes with ongoing symptomatic CPVT despite establishment of dual or triple therapy, return to play is potentially harmful.

Synopsis

Exercise restrictions have historically been a mainstay of management for individuals with CPVT due to the adrenergic basis of the ventricular arrhythmias that are the hallmark of the condition. There are very limited data reviewing the safety of athletes with CPVT competing in high-intensity sports. Most data come from a single center, demonstrating no deaths for CPVT athletes who are managed in a specialized clinic; however, the nonfatal event rates are higher than for other IAS from the same center. For athletes with CPVT considering return to play, the absence of ventricular arrhythmias and normalization of the stress test are critical prior to considering return to play. Some athletes may require escalation with multiple therapies before this can be considered, as demonstrated in Figure 12.

Recommendation-specific supportive text

1. For athletes who have been diagnosed with CPVT on predictive (cascade) familial genetic testing but have normal exercise stress testing (no clinical evidence of exercise-induced arrhythmias, complete absence of PVCs including burst protocol to unmask those with incomplete penetrance), return to play is reasonable with consideration for commencement of medical therapy with beta blocker and/or flecainide as part of a shared decision-making model. Data show that repeatability for ventricular arrhythmias can be variable, and therefore repeat exercise stress tests should be performed in these patients annually.[26,41,383,385,408]
2. Data for ongoing sports participation in overt CPVT are very limited. A single-center retrospective study of 63 individuals with CPVT is the largest published cohort and demonstrated the same event rate of 1.41/100 years for athletes and nonathletes with CPVT being managed in a specialized center, with no deaths in either group.[26,383,385]
3. Athletes who have CPVT and have had symptoms including syncope or cardiac arrest prior to commencing therapy should undergo a thorough clinical assessment, risk stratification, genotype and be established on dual or triple medical therapy before considering return to play.[383]
4. When titrating dual or triple therapies in athletes with CPVT, complete normalization of the stress test is the goal (complete absence of PVCs). Bigeminal PVCs may be acceptable, but couplets or more extensive nonsustained SVTs require continued treatment intensification. There are limited data for athletes with symptomatic CPVT who wish to return to play; however, the expert consensus is that this can only be considered in individuals who are established on dual or triple therapy (beta blocker ± flecainide ± LCSD) in a shared decision-making model. LCSD is best performed by an experienced high-volume center. The individuals need to be closely monitored with regular (6-12 month) burst exercise stress testing).[41,383]
5. For athletes who demonstrate a severe CPVT phenotype that is unable to be suppressed with optimization of medical therapy, high-intensity exercise with increased heart rate and adrenergic response is not safe.[14]

7.1.3 Brugada syndrome

Recommendations for athletes with Brugada syndrome

COR	LOE	Recommendations
1	B-NR	1. In athletes with Brugada syndrome, avoidance of arrhythmia triggers including sodium-channel blocking drugs, alcohol, and heavy meals is recommended.[409]
1	C-LD (Fever) C-EO (Hyperthermia)	2. In athletes with Brugada syndrome, aggressive treatment of fever (C-LD)[410] and avoidance of hyperthermia (C-EO) are recommended, including taking precautions to prevent overheating particularly for prolonged endurance exercise in warm climates.

Synopsis

There are limited data in athletes with Brugada syndrome. As there are no data showing that exercise increases risk in these individuals, there is no evidence to support exercise restrictions for these individuals. Athletes with Brugada syndrome may be at risk with increased core body temperature, due to the known association of fever and arrhythmia, and therefore this is an important consideration for endurance athletes, particularly in warm climates. The recommendations for athletes with Brugada appear in Figure 12.

Recommendation-specific supportive text

1. Athletes who are diagnosed with Brugada syndrome need to avoid known triggers for arrhythmia including the sodium-channel blocking medications listed at www.brugadadrugs.org.[396,411] It is also recommended that athletes with Brugada syndrome avoid heavy meals,[409] marijuana,[409,412] and excessive alcohol consumption[412] due to limited case reports suggesting increased risk of arrhythmia.

2. In athletes with Brugada syndrome, fever or raised core body temperature is known to be a trigger for arrhythmia; therefore, aggressive treatment of fever and avoidance of training-related heat exhaustion and heat stroke is recommended, with particular attention for heat management in prolonged endurance exercise, particularly when being performed in warm climates. Case series as well as case report describe individuals demonstrating a spontaneous type 1 Brugada pattern when febrile and subsequently suffering arrhythmic events. In a cellular model of Brugada syndrome, the phenotype was exacerbated with increased temperatures of the cell culture from 37 to 40 degrees.[410,413–415] As arrhythmias in individuals with Brugada syndrome are not adrenergically driven, should an athlete with Brugada suffer an arrhythmia, treatments will not specifically impact sports participation.[162]

7.1.4 Short QT syndrome

Recommendations for athletes with short QT syndrome		
COR	LOE	Recommendations
1	C-EO	1. In athletes with SQTS, patient education about the importance of fluid and electrolyte balance during endurance exercise is recommended.[400]
2a	C-LD	2. In athletes with symptomatic SQTS and/or a QTc < 320 ms, treatment with quinidine can be beneficial.[416,417]

Synopsis

There are no athlete specific data for SQTS, which is a very rare condition. These recommendations are based on limited data in nonathletic SQTS cohorts and are shown in Figure 12.

Recommendation-specific supportive text

1. SQTS is very rare, and therefore there are no data for safety or risk of exercise in athletes with this condition. Avoidance of electrolyte disturbances is recommended to reduce the risk of ventricular arrhythmias.[400]
2. There are limited data in small cohorts demonstrating that quinidine may be a useful therapy in athletes with SQTS, particularly in those with symptoms, those with SQT1, or those who demonstrate a short QT interval (QTc < 320 ms).[416,417] ICDs have also been used in this population, although data are insufficient to define indications.

7.2 Athletes with inherited cardiomyopathies

Inherited cardiomyopathies including HCM, ACM (of which one form is ARVC), and DCM have been identified in series of SCD in athletic cohorts.[82,84,91] Due to a perception of elevated risk, guidelines have historically led to blanket restrictions for athletes with inherited cardiomyopathies from participating in sports despite limited evidence of risk in athletes who have been diagnosed with these entities, risk-assessed, and appropriately treated.[418–420] More recently, guidelines have begun to acknowledge that some athletes with cardiomyopathies can return to play in a shared decision-making model.[10] Recent studies in athletes with HCM who have been appropriately risk-assessed and treated have not shown evidence of risk with return to play. The recent international multicenter LIVE-HCM study[421] demonstrated no increased cardiac event rate in vigorous exercisers compared with nonvigorous exercisers with HCM, without findings demonstrating risk in multiple retrospective studies.[26,194,422,423] However, in athletes with some ACMs, studies have demonstrated that participation in sports involving high-intensity endurance training may increase risk of ventricular arrhythmias, heart failure, and SCD, particularly in *PKP2*-mediated or exercise-induced ACM/ARVC.[27,154,155,424] Studies in individuals with DCM have demonstrated certain genotypes such as *LMNA* to pose higher risk to athletes than other genotypes such as *TTN*.[425,426] For an athlete with potential DCM, careful evaluation including advanced imaging such as stress echocardiogram and CMR, is important to differentiate EICR, which can include mildly decreased ejection fraction, from DCM.

7.2.1 Treatment and management of athletes with inherited cardiomyopathies before and after return to play

Recommendations for the treatment and management of athletes with inherited cardiomyopathies before and after return to play		
COR	LOE	Recommendations
1	B-NR	1. In athletes with inherited cardiomyopathies, expert assessment by clinician(s) with genetic and sports cardiology experience and a shared decision-making model of care is recommended.[26]
1	B-NR	2. In athletes with inherited cardiomyopathies, genetic testing is recommended.[27,425–429]
1	C-LD	3. In athletes with inherited cardiomyopathies considering return to play, or returning to play after arrhythmia treatment, a maximal stress test is recommended to identify exercise-induced ventricular arrhythmias.[430,431]
3: Harm	B-NR	4. In athletes with inherited cardiomyopathies, an ICD should not be implanted solely to facilitate return to play.[192,193,383]

Synopsis

Athletes with inherited cardiomyopathies require careful assessment, risk stratification, and management in expert genetic heart disease or sports cardiology centers. These recommendations aim to assist physicians with treatment and management of athletes with inherited cardiomyopathies before and after return to play. Nonfamilial cardiomyopathies are not discussed in this section of the document. The overall recommendations are summarized in Figure 13.

Recommendation-specific supportive text

1. Athletes with inherited cardiomyopathies require specialized expertise in genetic heart disease and/or sports cardiology in order for the athlete, their family, and other stakeholders to be fully informed with regard to participation in their particular sport for an athlete with their phenotype and/or genotype. The discussion should center around any potential risks (or lack thereof) of their ongoing sports participation, as part of a shared decision-making model.[10,26,395,432]

2. In athletes who fulfill diagnostic criteria for inherited cardiomyopathies, genetic testing can guide personalized therapeutic decision-making and risk stratification. The identification of a genetic result can be used for predictive testing in family members.[433] Genetic testing can be helpful to clarify diagnosis in athletes who have borderline phenotypes, as the identification of a genetic result can assist in providing the athlete with a personalized assessment.[27,425–429,434]

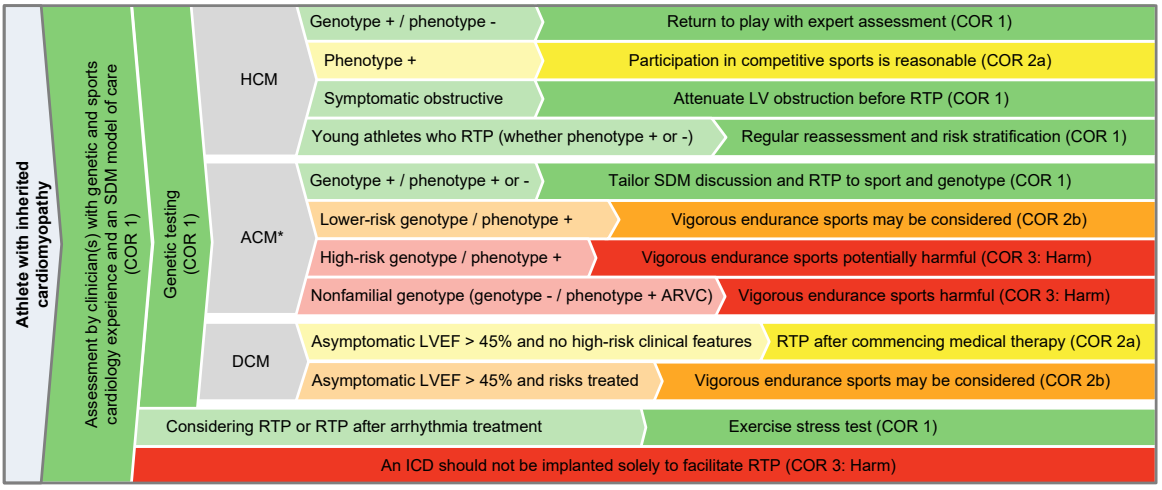

Figure 13
Recommendations for athletes with inherited cardiomyopathies returning to play. Colors correspond to the class of recommendation (COR) in Table 1. *Tailor management to genotype. ACM = arrhythmogenic cardiomyopathy; ARVC = arrhythmogenic right ventricular cardiomyopathy; DCM = dilated cardiomyopathy; GDMT = guideline-directed medical therapy; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter-defibrillator; LV = left ventricular; LVEF = left ventricular ejection fraction; RTP = return to play.

3. As described above, a key component of all series of athletes with cardiomyopathies who have returned to play, is expert assessment and treatment.[10] Stress testing in HCM is important in initial assessment, for evaluation of both LVOT obstruction and possible unrecognized symptoms of dyspnea on exertion as well as increases in ventricular arrhythmia. Nonsustained ventricular tachycardia is an important marker of risk for young and young adult individuals with HCM and for those with ACMs.[430,431] As for all athletes returning to play after arrhythmia treatment, stress test evaluates efficacy of arrhythmia suppression interventions.
4. Athletes with cardiomyopathies should receive an ICD based on standard clinical risk assessment. Data from both the ICD sports multinational registry and the Mayo Clinic show that competitive and recreational sports participation for athletes with inherited cardiomyopathies and ICDs can be safe, with no deaths, device malfunctions, or damage associated with participation in competitive athletes. However, athletes need to be informed of the risk of both inappropriate and appropriate shocks, although the risk of shocks has not been shown to be greater during competition than in leisure activities.[192,193,383,393] ICDs have risks and should never be implanted solely to facilitate return to play (see Section 7.2.1).

7.2.2 Treatment and management for athletes with hypertrophic cardiomyopathy

Recommendations for the treatment and management specific to athletes with hypertrophic cardiomyopathy

COR	LOE	Recommendations
1	B-NR	1. In athletes with genotype-positive phenotype-negative HCM, return to play in conjunction with expert assessment is recommended.[421]
1	B-NR	2. In young athletes with HCM who return to play, close follow-up with regular reassessment and ongoing risk stratification is recommended due to risk of evolution of their phenotype, including in those who are genotype-positive phenotype-negative.[435]
2a	B-NR	3. In athletes with phenotype-positive HCM, participation in competitive sports is reasonable with appropriate therapy and EAP including access to an AED.[194,384,421–423]
1	B-NR	4. In athletes with symptomatic obstructive HCM, intentional measures to attenuate the left ventricular obstruction are recommended before return to play.[436–438]

Synopsis

Until recently, athletes with a diagnosis of HCM were restricted from participating in sports, due to perceived increased risk of sudden death with sports despite limited evidence in this area.[418–420] More recently, guidelines have begun to acknowledge that some athletes with cardiomyopathies, including those with HCM, can return to play in a shared decision-making model of care.[10] There have been a number of recent publications that have begun to provide increased evidence for recommendations for physicians managing patients with HCM. The recent international multicenter LIVE-HCM study demonstrated no increased cardiac event rate in vigorous exercisers compared with nonvigorous exercisers with HCM,[421] which was also demonstrated in multiple retrospective series of athletes.[26,194,422,423] Figure 13 presents the summary of the recommendations for assessment and management of athletes with HCM.

Recommendation-specific supportive text

1. In athletes with genotype-positive phenotype-negative HCM, there are no data to support exercise restrictions. The LIVE-HCM study[421] included 126 genotype-positive phenotype-negative HCM individuals with no events reported in this group. Expert assessment to ensure full evaluation of current phenotypic expression and appropriate serial testing in follow-up should be facilitated in a timely fashion.
2. There are less data assessing the safety of high-intensity exercise in young (children/adolescent) athletes with HCM compared with adult cohorts. In addition, there is known age-related penetrance of disease, and therefore some young athletes may still be evolving their full disease phenotype at their initial assessment and require serial reassessment.[10,435] Close follow-up (at least once per year) for young (children and adolescent) athletes with HCM is important for ongoing assessment and risk stratification.
3. Recent data support a shared decision-making model of guided return to play for athletes with HCM after expert assessment, with multiple studies showing lack of harm,[26,194,421–423] and only a case series of 2 athletes suggesting harm.[84] The most important aspect of expert assessment is evaluation for risk for SCA, with ICD implantation for those meeting standard criteria indicating elevated SCA risk. Risk assessment includes evaluation of personal and family history, presence of nonsustained

ventricular tachycardia, and evaluation for CMR markers of elevated risk.[10]

The LIVE-HCM study[421] examined the impact of vigorous exercise on arrhythmic events in 1660 individuals with HCM. Among the participants who continued to exercise, there was no increased risk of arrhythmic outcomes—including death, cardiac arrest, appropriate ICD shock, or arrhythmic syncope—in the vigorous exercisers compared with those who were sedentary (event rate 4.7% vs 4.6%, respectively). In the LIVE-HCM study,[421] a post hoc analysis of a small group of younger athletes (aged 14-22 years) identified 42 athletes competing in varsity-level sports in whom there was 1 event of a resuscitated cardiac arrest (event rate 5.7/1000 person-years), 50 athletes participating in other vigorous exercise with no events (event rate 0), and 97 athletes participating in nonvigorous sports with 6 events (event rate 20.7/1000 person-years). Multiple small single-center studies of athletes with HCM who elected to continue to participate in sports have shown no evidence of increased harm in low-risk HCM athletes who continue to participate compared with those from the same center who elect to discontinue.[26,194,422,423] A recent series of elite athletes with genetic cardiovascular disease (HCM, n=40)[194] showed no adverse events, as did a series of 40 athletes with nonobstructive HCM who continued to play,[422] including 28 professional athletes with no life-threatening arrhythmias or change in structural or functional phenotype. There are limited data to support liberal adoption of high-intensity exercise in athletes with phenotype-positive HCM who are identified to have high-risk features (eg, high HCM risk score, exercise-induced syncope, documented ventricular arrhythmias). A screening cohort study reported 2 athletes diagnosed with HCM who continued to play despite advice and died suddenly,[84] leading to concern that higher-risk HCM athletes may be at higher risk of ventricular arrhythmias if they continue to participate in high-intensity sports, but clinical details of these individuals were not described. Most patients with HCM, including athletes, at high risk for sudden death will receive an ICD. Data from the ICD sports registry, which included 65 athletes with HCM, showed no ICD failures or injury.[192,193] Athletes require careful counseling and personalized risk assessment with appropriate protection from SCA as indicated, in a specialized center with a shared decision-making model, an EAP, and access to an AED. For athletes with HCM whose evaluation does not indicate ICD, consideration can be given to purchasing a personal AED. Data on risks of exercise in individuals with HCM who have undergone septal reduction therapy (myectomy or alcohol ablation) are not yet available.

The latest guidelines from the European Society of Cardiology (ESC)[15] and the AHA/ACC,[18] drawing on similar data, emphasize the importance of expert assessment (including appropriate treatment for athletes with elevated SCA risk) and shared decision-making (addressing potential and not fully understood risks) in consideration of return to play for athletes with HCM. The writing committee concurs with shifting away from universal exercise restrictions for those with HCM, with similar emphasis on expert assessment and shared decision-making. The writing committee reached consensus that with expert assessment, shared decision-making, and the implementation of appropriate therapy and EAPs, returning to play for athletes with HCM is reasonable (2a classification), whereas the ESC and AHA/ACC guidelines state it may be considered (2b classification). This decision was based on weighing the increasing evidence of low risk against the minimal evidence of high risk and the harm of restriction, as was done with other conditions discussed in this consensus statement for which evidence is similar and were given a 2a classification. While reaching consensus, the writing committee decision was not unanimous, with concerns raised that a 2a recommendation might deemphasize the mandatory need for expert assessment and long-term follow-up of these athletes, which is critical for ensuring equity of risk assessment and care for all athletes with HCM. However, the consensus of the group was that by emphasizing the need for expert assessment, this evidence-based recommendation will foster a model of process for return to play.

4. Athletes with HCM and symptoms attributed to LVOT obstruction (> 30 mmHg) have been successfully managed with therapies including medical therapy, alcohol septal ablation, and surgical myectomy to reduce the obstruction and improve symptoms to facilitate return to play.[10] In a single-center retrospective study of 58 HCM athletes,[436] 22 (38%) were found to have LVOT obstruction, of whom 5 underwent myectomy; however, only 4 (7%) reported exertional symptoms. There were no deaths; however, 1 patient with severe hypertrophy and myectomy had recurrent ICD shocks and no longer continued participating in sports. In this cohort, reduced peak VO2 did not correlate with outcomes.[436] Increased exercise LVOT gradients in asymptomatic HCM patients have been shown to be associated with decreased exercise performance.[437] Increasing data show that use of sarcomere inhibitors can improve symptoms in LVOT obstruction and reduce need for myectomy; however, there are no specific data reviewing these new medications in athletic individuals.[438]

7.2.3 Management specific to athletes with arrhythmogenic and dilated cardiomyopathies

Recommendations for management specific to athletes with arrhythmogenic and dilated cardiomyopathies

COR	LOE	Recommendations
1	B-NR	1. In athletes with genotype-positive phenotype-negative ACM, genotype-informed discussion with the athlete around the potential associations between high-intensity endurance exercise and increased likelihood of developing overt ACM phenotype and ventricular arrhythmias is recommended.[27]
1	B-NR	2. In athletes with genotype-positive but phenotype-negative ACM, sports participation should be tailored to patient's genotype and the intensity and duration of sport.[27,425–429]
1	B-NR	3. In athletes with phenotype-positive ACM, sports participation should be tailored to patient's genotype and the intensity and duration of sport.[27,425–429]
2b	C-LD	4. In athletes with phenotype-positive ACM and a lower-risk genotype, participation in vigorous endurance sports may be considered.[439]
3: Harm	B-NR	5. In athletes with phenotype-positive ACM and higher-risk genotypes, participation in vigorous endurance sports is potentially harmful.[27]
3: Harm	B-NR	6. In athletes with nonfamilial and/or genotype-negative phenotype-positive ARVC (presumed exercised induced), continuation of vigorous endurance sports is harmful.[305,328]
2a	B-NR	7. In athletes with DCM who are asymptomatic with LVEF > 45% and no high-risk clinical features, return to play following expert assessment and commencement of medical therapy is reasonable.[440–442]
2b	C-LD	8. In athletes with DCM who are asymptomatic with LVEF < 45% who have been appropriately risk assessed and risk treated, return to play may be considered.[440–442]

Synopsis

There is established evidence that exercise, particularly endurance exercise (see Section 2 and Figure 1), requires careful consideration for individuals with ACM and DCM. There is increasing evidence demonstrating that the underlying genotype is important when reviewing exercise-related risk stratification, management, and return to play. These recommendations present the current data, with Table 17 showing the gene-specific evidence currently available. Figure 13 presents the summary of the recommendations for assessment and management of athletes with ACM and DCM. Informed discussion with shared decision-making for exercise is the preferred and evidence-based approach.[53,54,432]

Recommendation-specific supportive text

1. In athletes with *PKP2* variants associated with ACM/ARVC (genotype-positive phenotype-negative), studies have shown ongoing participation in vigorous-endurance exercise to lead to more rapid progression to overt disease, increased risk of ventricular arrhythmias, and increased risk of heart failure.[27,443] In athletes with a positive genetic test for ACM pathogenic variants in genes besides *PKP2* and normal imaging and stress testing, there are limited data assessing the

Table 17 Association of vigorous (> 6 METs) endurance exercise with clinical outcomes in ACMs

Genotype of ACM	Arrhythmias/ SCD	Progression	Penetrance
PKP2	+++	+++	+++
Nonfamilial/gene-negative ARVC	+++	++	Not applicable
LMNA	+	++	++
TMEM43	++	+	+
DSP	++	+/-	+/-
DSG2	+	+	+
DSC2	+	+	?
PLN	+	-	-
FLNC	+	?	?
JUP/Naxos disease	?	?	?
RBM20	?	?	?
DES	?	?	?

+++ Replication across multiple clinical studies from different cohorts and supported by understanding of pathophysiology and experimental data.
++ Replication across multiple clinical studies from different cohorts OR quality clinical study plus support from experimental data.
+ Single clinical study or clear pathophysiology.
+ / - Mixed findings from studies.
? No strong clinical evidence or clear pathophysiology.
- Evidence does not support deleterious effect of endurance exercise.
ACM = arrhythmogenic cardiomyopathy; SCD = sudden cardiac death.

risks of endurance or high-intensity exercise. Therefore, the risk of ongoing participation in endurance sports for these athletes incorporating genotype and type of exercise can be approached with athlete counseling and shared decision-making. Table 17 shows the role of exercise in increasing penetrance in specific ACM genotypes.

2. There are limited data to support blanket exercise restrictions for nonendurance exercise in genotype-positive phenotype-negative ACM athletes. Regular surveillance (at least once per year) assessing cardiac imaging, stress testing, and monitoring ensures that any development of an overt phenotype is identified, risk stratified, and managed appropriately.[444] Table 17 shows the role of exercise in increasing penetrance in specific ACM genotypes. Evidence of the association of participation in frequent, high-intensity exercise with disease onset (eg, penetrance) is most compelling for *PKP2*, in which such exercise is associated with higher likelihood of disease expression and worse clinical presentation.[27,154,443]
3. In athletes with clinical evidence of ACM, participation in competitive/recreational sports involving low to moderate intensity is reasonable; however, participation in high-intensity or frequent endurance exercise is not recommended, particularly for patients with *PKP2*-related ARVC, due to an association with increased risk of ventricular arrhythmias and sudden death.[27,154] In one study of these patients,[154] competitive sport was associated with two-fold increased risk of ventricular arrhythmias, death, and symptoms compared with patients who were inactive or who participated in recreational sports. In this cohort, recreational sports were not associated with increased symptoms, ventricular arrhythmias, or death. In a large postmortem ACM SCD cohort of 202 cases,[424] athletes were 16 times more likely to die during exertion than nonathletes. In this cohort, a small proportion (25%) underwent genetic testing, with pathogenic variants identified in *PKP2*, *DSP*, and *TMEM43*. In the largest cohort of patients with genetic heart disease who have returned to play, athletes with ACM had an event rate of 8.16/100 patient-years, which trended higher than seen in other genetic heart diseases at the same institution.[26] Table 17 summarizes the data on the impact of exercise on risk of sustained ventricular arrhythmias and structural progression that differs based on specific ACM genotypes. Data are most robust for patients with *PKP2* variants and for patients with nonfamilial gene-negative ARVC.
4. As demonstrated in Table 17, studies in some genotypes such as *PLN* demonstrate no signal for increased risk of progression or penetrance with endurance exercise, and therefore blanket restrictions cannot be supported.[439] Nonetheless, in a retrospective study of 185 *PLN* carriers,[429] the majority (74%) of malignant ventricular arrhythmia events occurred during exercise (2/3 during low-intensity exercise and 1/3 during moderate to high intensity exercise) as did 13/19 (68%) SCDs, highlighting the importance of nuanced discussions and shared decision-making. Disease-specific risk assessment, with ICD implantation if indicated, as well as disease-specific treatment, is critical prior to consideration of return to play.
5. In athletes with *PKP2*-mediated ACM/ARVC with evidence of disease on imaging or stress testing, participation in high-intensity or endurance exercise is not recommended due to an association with increased risk of ventricular arrhythmias and sudden death.[27,154] This has also been demonstrated in mouse models of ARVC across desmosomal genotypes.[443,445–447] Other higher-risk genotypes include *DSP*, *LMNA*, and *TMEM43*, as mentioned previously (see Table 17).[424–426,448,449]
6. Studies have shown endurance athletes with genotype-negative exercise-induced right ventricular remodeling to be associated with higher rates of ventricular arrhythmias with persistent endurance exercise[305,328] and greater clinical benefit from exercise modification.[306] While studies comparing continuation versus discontinuation are lacking, since the phenotype develops due to endurance exercise, a pillar of treatment is removal of the underlying cause (see Section 5).
7. There are no data demonstrating benefit for exercise restriction in athletes with DCM who do not have symptoms or higher-risk clinical features (LVEF < 45%, documented ventricular arrhythmias on Holter or EST, significant late gadolinium enhancement on CMR, high-risk genotypes, failure of LVEF to augment by > 10% with stress).[439–442,450,451] With borderline LVEF, it is important to differentiate DCM from EICR.[450,451]
8. There are limited data reviewing the safety of ongoing exercise for athletes with DCM with no symptoms but impaired ventricular function. Assessment of risk for SCA, with ICD implantation for those meeting standard criteria indicating elevated SCA risk, is critical. Risk assessment includes evaluation of personal and family history, presence of nonsustained ventricular tachycardia, and CMR markers of elevated risk. There are data demonstrating the presence of late gadolinium enhancement to be a predictor of SCD risk in DCM cohorts; it is therefore an important factor in risk stratification and shared decision-making.[440–442]

7.3 Moving from athlete to family: Implications of a genetic diagnosis

Most genetic heart diseases are inherited in an autosomal dominant manner with incomplete penetrance and variable expressivity.[178,433] This means family members have a 50% chance of inheriting the same cardiac condition. An athlete may represent the initial patient found with inherited disease, (proband) whether due to presentation with symptoms or to preparticipation screening. For the physician treating the athlete diagnosed with an inherited condition, appropriate medical care also involves addressing the athlete's family. As availability for genetic testing has expanded exponentially, with many more individuals undergoing genetic testing, understanding the nuances of cardiac genetic testing is important. Genetic counseling is an important adjunct to genetic testing.[452,453]

Recommendations for moving from athlete to family: Implications of a genetic diagnosis		
COR	LOE	Recommendations
1	B-NR	1. In athletes with suspected genetic heart disease, family history including 3 generations on both sides of the family is recommended.[454,455]
1	B-NR	2. In athletes with suspected genetic heart disease based on family history and/or phenotype, consultation with (or referral to) a multidisciplinary team with expertise in genetic heart disease is recommended.[456–458]
1	B-NR	3. In athletes with genetic heart disease who have a positive genetic test, variant-specific, predictive cascade testing in the appropriate family members is recommended in conjunction with genetic counseling.[459–466]
1	B-NR	4. In the absence of genetic testing or when the athlete has a negative test result for a genetic heart disease, first-degree relatives should undergo clinical screening including ECG and echocardiogram as minimum baseline investigations.[464,465,467–470]
1	B-NR	5. In athletes with a genetic variant but without phenotypic expression of the disease, counseling about return to play should be disease- and variant-specific.[456–466]
3: Harm	B-NR	6. Athletes with a genetic variant for arrhythmogenic conditions should not be restricted from play by governing bodies based on genetic results alone.[49–52]

Synopsis

Genetic testing can assist with diagnosis, risk stratification, and management decisions in an athlete.[434,471] Most genetic heart diseases are inherited in an autosomal dominant manner, which carries important implications for the extended family.[178,433] A detailed family history is important when establishing the genetic basis to disease. The yield of genetic testing for genetic heart diseases ranges from 20%-30% (Brugada syndrome) up to 75%-80% (LQTS).[175,176,178,179,181–183] The yield also increases in the presence of a family history of disease or sudden death.[452] If an athlete is positive for a pathogenic or likely pathogenic variant, predictive testing of family members and relatives can identify those who are at risk and require ongoing screening. Figure 14 summarizes

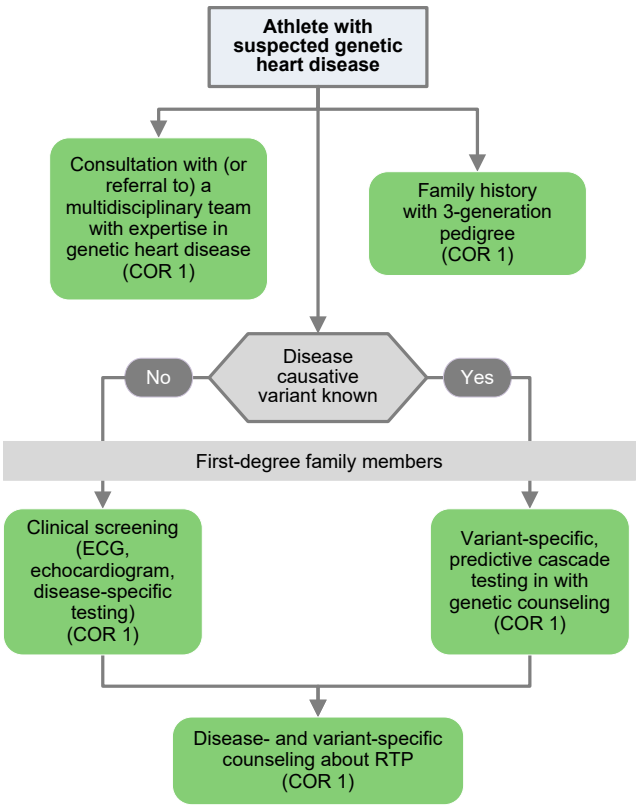

Figure 14
Flow chart demonstrating approach to families of athletes with genetic heart disease. Colors correspond to the class of recommendation (COR) in Table 1. ECG = electrocardiogram; RTP = return to play.

the approach to athletes' families following a diagnosis of an inherited cardiac condition.

Recommendation-specific supportive text

1. Documenting a careful family history in athletes who have or are suspected to have a genetic heart disease (including IAS and cardiomyopathies), extending to 3 generations of relatedness, may identify any high-risk family history (eg, family history of SCD) to aid in risk assessment of the athlete as well as in identifying individuals who are at 50% risk of having the same condition due to the autosomal dominant inherited pattern of most of these conditions, to aid in family cascade screening.[178,433,453,454]
2. Multidisciplinary teams should include both genetic counselors and clinicians with experience in the clinical evaluation and risk assessment of genetic cardiovascular disease. In athletes and their families undergoing genetic testing, there are important ethical and legal considerations, including potential insurance implications or sport restrictions imposed by governing bodies. Genetic counseling in conjunction with genetic testing ensures that the athlete and family fully understand the implications of the testing being performed.[456–458]
3. Athletes who have undergone genetic testing may have an actionable variant identified (class 4 or 5 variants, ie, likely pathogenic or pathogenic), and their first-degree family members are at a 50% chance of carrying the same variant.[452] Family members who test positive will need clinical evaluation to determine presence and severity of phenotype. Ongoing clinical screening is not necessary for family members who test negative for the family's disease-causative variant.[433,471] Genetic counseling guides the process for athlete and family.[459–466]
4. In athletes with inherited cardiac conditions, if no variant has been identified, clinical screening in first-degree relatives (parents, siblings, and children), with a minimum of ECG and echocardiogram (and including Holter monitoring, exercise stress testing, or CMR depending on the underlying condition in the family), has been shown to increase the diagnostic yield in family members who may have the same genetic heart disease.[464,465,467–470]
5. As described in Section 7.2.3 and shown in Table 17, there is significant variation among genotypes regarding the role of vigorous exertion in increasing penetrance of disease. Individuals in whom a genetic variant is found based on family cascade screening after the diagnosis in a proband, with subsequent determination of absence of phenotype, should be similarly counseled.[456–466]
6. Genetic testing in medicine has the ability to improve recognition and treatment of disease. However, the conflict between the potential of genetic testing to improve health and the potential ethical issues it raises for privacy and autonomy are increasingly recognized, in general,[472,473] and in the context of sports.[474] Particularly as the penetrance of cardiomyopathy as well as the clinical course of carriers of genetic variants for electrical disease is highly variable and the factors poorly understood, genetic results should not be used to guide decisions by governing bodies to determine eligibility. As described in Section 2.4, restriction from sports can lead to significant harm.[51,52]

Section 8 Atrial fibrillation

Managing athletes with AF is complex because they perceive themselves as healthy individuals and often want to continue sports. AF in athletes can occur in several situations. First, AF can occur in the setting of a previously undetected genetic electrical or structural disease, such as Brugada syndrome or HCM. Acquired conditions may also lead to AF, such as hypertension or myocarditis. Finally, it may be the consequence of long-term endurance training or a combination of the previous factors. Evaluation and establishing a diagnosis, prognosis, and specific treatment are mandatory.

Long-term high-intensity endurance training is a risk factor for AF with a cumulative effect. Therefore, AF is uncommon in young athletes in the absence of underlying abnormality. However, as a causative factor for AF, endurance training often goes unrecognized because the incidence of AF starts to rise when athletes are in their 50s and 60s and have already significantly declined endurance practice. AF related to endurance training is much more prevalent among men, but the reasons are not yet fully understood. This may be due to lower cumulative training in women or to differences in the presence of concomitant factors for AF between men and women, such as height or autonomic tone.

In patients involved in endurance sports, such as cycling or running, a careful discussion regarding the potential contribution of their practice to recurrences is probably necessary. To date, data on the reversibility of AF upon endurance practice cessation are based only on clinical observations and experimental models. Current management of the athlete with AF is similar to that for the nonathlete, with additional considerations detailed below.

In summary, athletes with AF should undergo a careful evaluation to uncover preexisting conditions. A detailed analysis of their lifetime and present sport practice is also necessary to establish the appropriate management.

8.1 Epidemiology and pathophysiology of atrial fibrillation in athletes

There are many putative mechanisms underpinning the risk of AF in endurance athletes, although few have current implications in the assessment and management of the athlete with AF.

8.1.1 Epidemiology of atrial fibrillation

In athletes, the prevalence and risk of AF varies based on sex, age, and type of sports participation. Although the prevalence of AF increases with age, the relative risk for athletes compared with nonathletes is greater in younger athletes.[475–478] Strong evidence from multiple studies demonstrates a relationship between AF prevalence and

participation in endurance sports. While initial studies showed this association in males, recent studies suggest that AF may also be more prevalent among female endurance athletes.[475,479,480] Overall, although the prevalence of AF increases with age, the relative risk of AF associated with athletic training is greater in younger individuals. In Scandinavian cross-country skiers of mean age 37 years,[478] 1.2% developed AF in 8 years of follow-up and AF was significantly more common in the fastest athletes and those who had completed the most races. In older skiers,[476] AF was nearly twice as common among 505 athletes of median age 68 years as compared with 1867 nonathletes (prevalence 29% versus 18%; relative risk 1.9 [1.5-2.4]). This is comparable to the findings of a recent meta-analysis of 13 studies[477] over the past 2 decades including nearly 64,000 athletes that concluded that athletes were 2.46 times more likely to have prevalent AF (95% CI 1.73 to 3.51). The odds ratio increased to 3.6 when athletes aged < 55 years were considered.[477] A potential exception to the almost universal observation of increased AF risk among athletes was a study by Boraita et al[481] in which a low prevalence of AF (0.3%) was reported among 6813 Spanish athletes. However, it should be noted that the cohort comprised very young athletes (mean age 22 years) of which 65% were male and only 28% were endurance athletes. Male gender, increasing age, endurance sport participation, and years of sports training were all associated with AF, thereby confirming a likely association between athleticism and AF even in a cohort with lowest expected prevalence.[481] Although less studied, atrial flutter is also more common among endurance athletes than among nonathletic individuals.[475]

While endurance sports alone may increase vulnerability to AF, factors underlying AF in the general population can also affect athletes. Conditions that are associated with AF in nonathletes include hypertension, valvular heart disease, and hyperthyroidism.[16] It is reasonable to assume that these conditions would also increase the prevalence of AF in athletes, although this has not been specifically investigated in athlete populations. There is speculation about a link between an exaggerated blood pressure response during exercise stress testing and AF, but the evidence supporting this association is currently limited.

There is a clear association between several cardiomyopathies and AF that is particularly relevant to younger athletes with AF. The strongest associations are with HCM, DCM, and ACM (where AF is found in 10%-25% of patients in each subtype). Similarly, AF is more common in patients with LQTS and Brugada syndrome.[482] There are no studies that have assessed the prevalence of underlying cardiac disorders in athletic populations. As described above, athletic training is associated with AF and the relative risk is greatest in young athletes. It is unclear to what extent the excess risk of AF in young athletes is due to environmental factors (athletic training) or genetic predisposition.

For young and young adult athletes, AF might be the first manifestation of an inherited cardiac condition. Three genes (*TTN*, *SCN5A*, and *KCNA5*) have been strongly associated with AF, while a further 9 genes (including *LMNA* and *KCNQ1*) have been associated with AF to a lesser extent. Studies in early-onset AF cohorts have returned a yield of pathogenic variants (predominantly in *TTN*) in approximately 1%-3% of early-onset AF patients.[483–487] The prevalence of rare variants among athletic populations with AF has not been specifically investigated. Discussions regarding the utility of clinical genetic testing in patients with AF should highlight the low expected yield.

Mechanisms underlying the increase in AF incidence in endurance athletes are hypothesized. Atrial dilation is known to be a risk factor for AF in nonathletes and is also associated with more advanced atrial disease in which reversion to and maintenance of sinus rhythm may be less likely. In athletes, atrial dilation is common and can be profound. Whereas there has been a consistent observation of greater atrial volumes in patients with AF than in patients without a history of AF, this is not as clear in athletes. In a matched cohort of athletes with and without AF, Trivedi et al[488] found no difference in atrial volumes. Similarly, Sorensen et al[489] observed considerable overlap in atrial volumes between nonathletes with AF and athletes with no history of AF. Thus, the association between atrial enlargement and AF, and the resulting prognostic implications, should be considered with care in endurance athlete populations.

The use of performance-enhancing drugs (stimulants, etc) may be an additional factor that predisposes athletes to AF. There is limited evidence linking the use of performance-enhancing drugs to AF, but this may be due to the fact that it is an extremely elusive cohort for study. In the general population, stimulants taken recreationally increase AF (see Table 4).

8.1.2 Pathophysiology of atrial fibrillation in the athlete

There are several putative mechanisms to explain the increase in AF observed in endurance athletes. Familiarity with these mechanisms may assist the clinician when discussing management options with an athlete with AF.

Some theories on the causation of AF in athletes have been extensively discussed elsewhere[490,491] and include structural remodeling of the atrium in response to repeated exposure to increased volume and pressure loads during exercise. There is some evidence in murine models that this remodeling includes changes to the sino-atrial node with downregulation of HCN channels that may contribute to more profound bradycardia and AF.[492] There is also evidence that exercise-induced AF may be mediated through increased vagal stimulation[493] and increases in proinflammatory substrate and fibrosis.[493,494]

8.2 Atrial fibrillation evaluation in athletes

Recommendations for the atrial fibrillation evaluation in athletes		
COR	LOE	Recommendations
1	C-LD	1. In athletes with symptoms or personal ECG (portable wearable) findings concerning for AF, electrocardiographic documentation is recommended to establish a diagnosis of AF.[495–497]
1	C-LD	2. In athletes with AF, a detailed history and physical examination focused on identifying reversible or modifiable factors as well as exercise history should be performed.[475,498–505]
1	C-LD	3. In athletes with AF, initial evaluation should include 12-lead ECG, transthoracic echocardiogram, and laboratory evaluation.[506–509]
2a	C-LD	4. In athletes with AF, rhythm monitoring can be useful to evaluate burden, rate of ventricular response during an episode, relationship to symptoms, and documentation of other arrhythmias.[510,511]
2a	C-LD	5. In young and young adult athletes with AF, advanced imaging such as CMR is reasonable.[507,512]
2a	B-NR	6. In young and young adult athletes with AF and clinical suspicion of channelopathy or cardiomyopathy, genetic testing is reasonable.[483–486,506,507]
2b	B-NR	7. In young and young adult athletes with AF, genetic testing may be considered.[483–486,506,507,513]
1	C-EO	8. In young and young adult athletes with AF, withholding from sports participation pending evaluation of malignant etiologies is recommended.
1	C-EO	9. In athletes with AF, recommendations regarding intensive endurance sports participation while evaluation is ongoing should take into consideration symptoms, heart rate, and pattern of AF.
2a	C-EO	10. In athletes with AF, it is reasonable for patient counseling to clarify that sports-related AF is not life-threatening and sports participation is guided by symptoms.
1	B-NR	11. Athletes with AF should be managed in a center with expertise in both AF and the care of athletes.[514,515]

Synopsis

The athlete with AF requires a comprehensive evaluation largely similar to that for nonathletes. Lone AF, or AF in patients with a structurally normal heart, should be a diagnosis of exclusion after thorough evaluation. Age may influence underlying etiological factors. Young athletes may have a higher incidence of underlying inherited arrhythmia syndromes (IAS) or cardiomyopathies, or SVT.[507,512] Despite otherwise excellent health status, Masters athletes may have typical risk factors, such as hypertension, obesity, and obstructive sleep apnea.[498,499,502] The relationship between exercise and the AF is well documented: moderate amounts of exercise carry benefit, while a sedentary lifestyle increases risk. A J-shaped curve has been demonstrated, with high-intensity endurance training also associated with increased risk of AF.[475,503,504] The role of exercise in promoting AF applies to athletes typically participating in greater than 10 or more hours per week of high-intensity endurance activity over many years. While the impact of detraining on AF recurrence is unknown, moderation of exercise and avoidance of sedentary behavior should be encouraged while evaluation is performed. An algorithm of the recommendations for the evaluation of athletes with possible AF is shown in Figure 15.

Recommendation-specific supportive text

1. Confirmation of AF can be obtained by 12-lead ECG during symptoms. If long-term monitoring is needed to establish a diagnosis, ambulatory rhythm monitoring, a smartwatch with ECG sensors, or implanted loop recorders can be utilized. Although smartwatches are evolving to detect AF with high sensitivity, it is recommended that rhythm strips are thoroughly reviewed to confirm AF diagnosis.[495–497] Further confirmation with an ECG or ambulatory patch monitor, particularly in those with low burden, should be pursued. Features of available monitor types appear in Table 14. In those with palpitations during exercise, an exercise stress test may aid in diagnosis.
2. The initial evaluation of a patient with AF involves classifying AF (paroxysmal, persistent, longstanding persistent, or permanent) and associated symptoms, determining its cause, reversible or inciting factors, and comorbidities, and obtaining exercise capacity and exercise history. Risk factors, many of which are potentially modifiable, are largely the same for athletes as for the general population and include obstructive sleep apnea, use of stimulants, alcohol, obesity, hypertension, and coronary artery disease, particularly in older athletes.[498–502] The use of

performance-enhancing drugs may be considered specifically in the athlete, although studies have not evaluated an association with AF.[321,516] Exercise history should focus on current and lifetime exercise volume. While moderate levels of physical activity are associated with AF risk reduction, high-intensity endurance training has been associated with increased risk of AF.[475,503–505] The physical exam may disclose resting bradycardia, irregular pulse in the presence of AF or a rapid, regular pulse in atrial flutter, elevated jugular venous pressure in setting of heart failure, or murmurs suggestive of valvular disease.

3. All patients with documented AF should have an ECG during AF episode and in sinus rhythm to evaluate for potential underlying etiologies including supraventricular arrhythmias such as typical atrial flutter and presence of delta wave suggestive of an accessory pathway (AP). Moreover, an ECG may suggest an IAS, including Brugada pattern or LQTS, as both have been associated with AF, particularly in children and young adults.[506,507] An echocardiogram will evaluate for structural heart disease including CHD and cardiomyopathy, cardiac function, and atrial size.[509] It is important to take into consideration the athlete's sport and training history to distinguish the athlete's heart from pathological remodeling on imaging, as athletes have been shown to have an increased incidence of left atrial enlargement.[508,517] Initial laboratory evaluation should include complete blood count, serum electrolytes, renal and hepatic function, and thyroid function.
4. For those with frequent symptoms, ambulatory monitors with a single patch electrode can diagnose and quantify arrhythmia burden for up to 30 days. Extended monitoring (> 30 days) with an implantable cardiac monitor can be used for long-term monitoring of AF burden in those with infrequent episodes.[510,511] The use of smartwatches for detection of AF and monitoring of AF may be a long-term monitoring option if rhythm strips can be reliably obtained by the user and reviewed by the provider.[495–497] See Table 14, which describes features of some available monitoring systems.
5. AF is rare in general in the young and is also rare in young and young adult athletes. Young athletes may have a higher incidence of underlying IAS or cardiomyopathies, or SVT, than older athletes.[507,512] Thus, advanced imaging with CMR, computed cardiac tomography, and/or positron emission tomography is reasonable for evaluation of underlying structural cardiac abnormalities. CMR is discussed in further detail in Section 5.
6. Young adult athletes with AF who have a strong family history or imaging or electrocardiographic findings concerning for a cardiomyopathy or channelopathies should undergo genetic testing.[506,507] Several genes associated with cardiomyopathies and channelopathies associated with familial AF have been identified in the general AF population.[513,518,519] Three genes (*TTN*, *SCN5A*, and *KCNA5*) have been strongly associated with AF, while a further 9 genes (including *LMNA* and *KCNQ1*) have been associated with AF to a lesser extent. Studies in early-onset AF cohorts have returned a yield of pathogenic variants (predominantly in *TTN*) in approximately 1%-3% of early-onset AF patients.[483–486,518,519] As these entities may be associated with SCA, if there is clinical suspicion for cardiomyopathy or channelopathy, referral for genetic counseling and testing, as discussed in more detail in Section 7, is useful.
7. Whether all young and young adult athletes should be referred for genetic counseling, even in the absence of other clinically suspicious findings, is less clear-cut. As above, data suggest a small prevalence of genes associated with cardiomyopathies and channelopathies, which may be associated with SCA; some of these have preventive measures to decrease that risk, others have potential increase in penetrance with exercise (see Section 7). Studies are ongoing to determine clinical utility of genetic testing in all early-onset AF.
8. As some underlying etiologies of AF may carry risk of SCA,[506,507] exclusion of underlying cardiomyopathy, myocarditis, or electrolyte or thyroid abnormalities should be evaluated and managed prior to intensive sport participation, particularly in the young or young adult athlete.
9. For adult athletes with AF in whom episodes are frequent with rapid ventricular rates and causing severe symptoms, intensive sports participation should be discontinued until AF is managed effectively. For athletes without high suspicion of an underlying life-threatening etiology, and whose symptoms are not life-threatening, restriction while evaluation is undergoing is not indicated.
10. With AF that occurs during exercise that is asymptomatic to minimally symptomatic with controlled ventricular rates and without preexcitation, exercise may continue if tolerated. If symptoms are severe or worsen with exercise, AF conducts with rapid ventricular rate, or if there is preexcitation, then it is recommended to stop exercise until rate and/or rhythm is controlled. For tactical athletes such as military pilots, who are required to maintain physical fitness similar to that of athletes, governing bodies will determine participation, balancing potential risks with individualized approaches. In one small series of 27 active-duty pilots,[520] of whom 44% completed deployments flying low-performance aircraft, half of these were treated with ablation. Long-term follow-up is not described.
11. Patients with AF have better outcomes including decreased stroke and death when managed by specialists.[515] A recent study from a single center[514] has shown reduced readmission rates, as well as reduced initial length of stay and costs, when AF patients were managed as part of a dedicated AF center. Poor outcomes associated with variations in care are well documented.[521] Similarly, specialists in the care of athletes provide additional expertise.[7] Not all centers may have access to all specialists, and geographic and insurance considerations may not always allow consultations with individual/s with these expertise, but when possible, such consultations should be sought.

8.3 Treatment of atrial fibrillation in athletes

8.3.1 Risk factor modification in athletes with atrial fibrillation

Recommendations for risk factor modification in athletes with atrial fibrillation

COR	LOE	Recommendations
1	B-R	1. In athletes with AF, risk factor management, including weight loss, treatment of obstructive sleep apnea, alcohol avoidance, and hypertension management is recommended.[522,523]
2b	C-LD	2. In athletes with AF who engage in long-term, high-intensity endurance training, exercise detraining with modification to low to moderate levels of exercise may be considered.[477,524–528]

Synopsis

Management of AF in the athlete is largely based on extrapolation of clinical trial data from nonathletes, observational studies, and expert opinion. Standard risk factors should be discussed and modified, as in nonathletes. Data on detraining are sparse but may be effective in some and can be discussed as an option. An algorithm of the recommendations for treatment of AF in athletes including risk factor management, prevention of thromboembolism, and rhythm control in athletes with AF is shown in Figure 15.

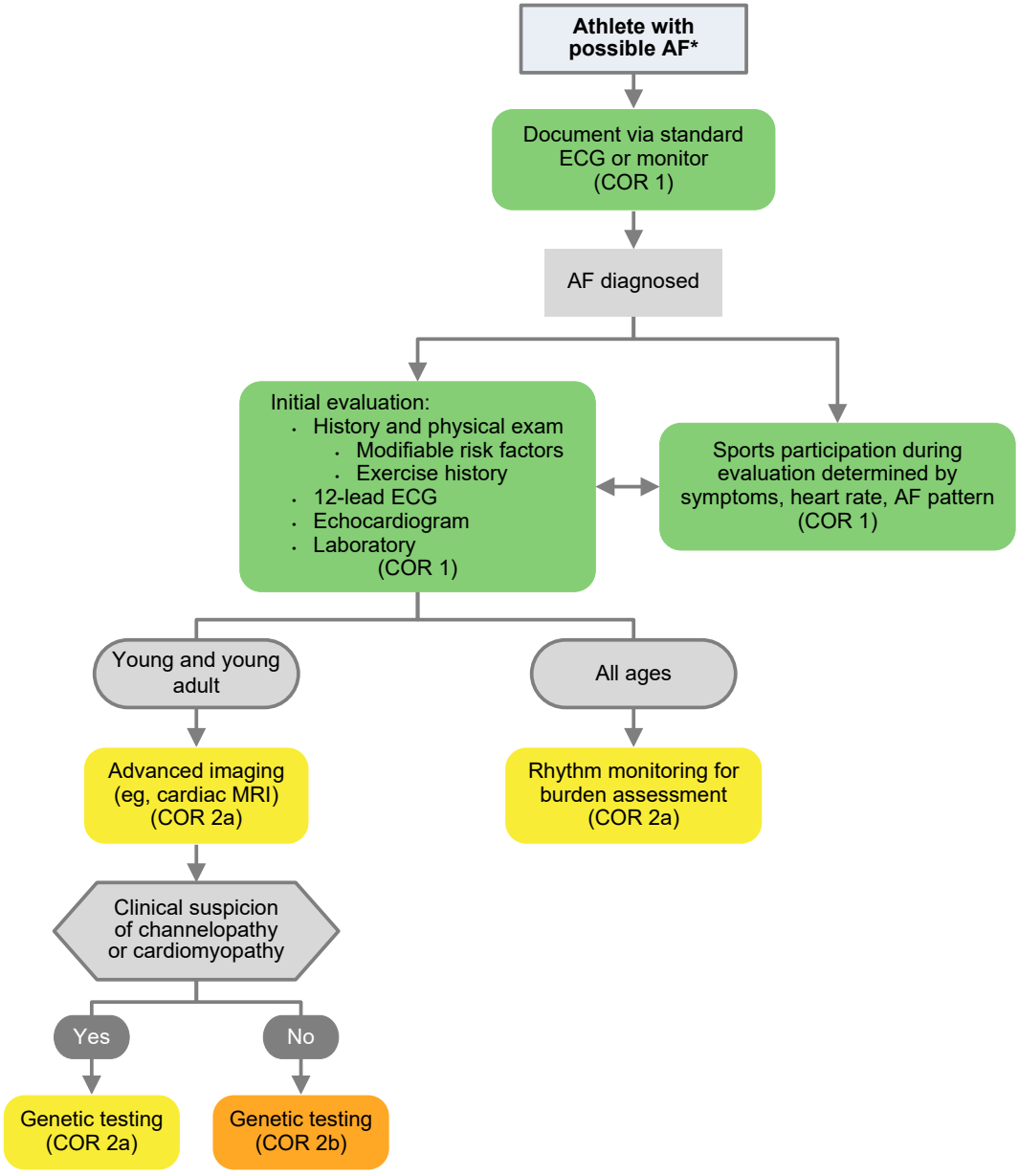

Figure 15

Algorithm for the evaluation of athletes suspected of having AF. Colors correspond to the class of recommendation (COR) in Table 1. *Based on symptoms or wearable device data. AF = atrial fibrillation; ECG = electrocardiogram; MRI = magnetic resonance imaging; SMD = shared decision-making;

Recommendation-specific supportive text

1. Modifiable risk factors that are commonly observed in the nonathlete population should not be overlooked in the athlete population. These include hypertension, obesity, obstructive sleep apnea, smoking, and use of alcohol and, for some individuals, caffeine.[522,523,529] These also include use of performance-enhancing drugs. While the association of performance-enhancing drugs with AF risk remains unclear,[321] anabolic steroids have been associated with atrial electrical mechanical delay in bodybuilders, and testosterone replacement in nonathletic populations is associated with an increase in AF.[516,530]
2. The association between volume of exercise and development of AF is well established as a U-shaped relationship based on observational data, particularly in middle-aged men, in which both low and high volumes of exercise, specifically endurance exercise, increases the risk of AF.[477,526–528] This relationship has not been demonstrated in nonendurance sports. The increased risk of AF with endurance exercise applies to a select group of athletes participating in high levels of endurance exercise, such as greater than 10 or more hours per week of high-intensity exercise.[491]

While the association of long duration of vigorous endurance athletics with the development of AF is well established, whether decrease in exercise once AF develops will decrease AF recurrence has not yet been demonstrated. Although studies are ongoing, there currently are no published randomized controlled data evaluating the impact of exercise detraining on AF burden in endurance athletes. Complete cessation of exercise may not be necessary given the protective benefits of moderate exercise on AF and the detrimental effects of exercise cessation on psychological mindset and quality of life.[525] Not all athletes may wish to pursue exercise cessation, but discussion of this option will inform shared decision-making.

8.3.2 Prevention of thromboembolism in athletes with atrial fibrillation

Recommendations for prevention of thromboembolism in athletes with atrial fibrillation

COR	LOE	Recommendations
1	B-NR	1. For athletes with nonvalvular AF, stroke risk assessment using a validated risk score, such as CHA_2DS_2-VASc, or other disease-specific factors is recommended.[531–533]
1	A	2. For athletes with nonvalvular AF with an estimated annual thromboembolic risk of ≥ 2% per year (CHA_2DS_2-VASc score of ≥ 2 in men or ≥ 3 in women, or other disease-specific factors), oral anticoagulation is recommended [534–538]
1	C-EO	3. In athletes with AF on anticoagulation who are participating in sports with a risk of trauma, a shared decision-making discussion about continued participation is recommended.
2b	B-NR	4. In athletes with AF, left atrial appendage occlusion (LAAO) may be considered based on anticoagulation indication and bleeding risk, taking into account patient preference to avoid long-term anticoagulation, in a shared decision-making context.[539,540]
2b	C-EO	5. In athletes meeting anticoagulation criteria who wish to temporarily participate in sports with a high risk of bleeding, temporarily withholding anticoagulation may be considered with a shared decision-making discussion.

Synopsis

The risk of stroke is lower in athletes with AF than in nonathletes with AF, based on 2 observational cohort studies that have made similar observations regarding the risk of stroke in athletes with and without AF as compared with matched nonathlete populations.[476,478] The risk of stroke in athletes with AF was 27%-40% lower than in nonathletes with AF.[476,478] Whether these data can support differing anticoagulation regimens has not been tested. For athletes participating in collision sports and other sports with risk of bleeding, anticoagulation may increase risk, and shared decision-making about continued participation and form of stroke prevention is needed. An algorithm of the recommendations for treatment of AF in athletes including risk factor management, prevention of thromboembolism, and rhythm control in athletes with AF is shown in Figure 15.

Recommendation-specific supportive text

1. Similar to the approach in the nonathlete population, consideration of anticoagulant therapy should be based on risk of thromboembolism assessed by the CHA_2DS_2-VASc score (or other emerging scores, not yet equally validated in most populations), as available data suggest that athletes with risk factors remain at risk of stroke.[16,531–533] While some studies suggest athletes are at lower risk than nonathletes with AF,[476,478,534] data are not sufficient to support different anticoagulation regimens. For athletes with AF in the setting of underlying specific disease entities, anticoagulation decisions should follow recommendations for that entity. For example, athletes with AF in the setting of HCM should receive anticoagulation regardless of CHA_2DS_2-VASc score.[10]
2. In those with elevated stroke risk, direct oral anticoagulation is preferred over warfarin based on large, randomized trials.[536–538] Although the CHA_2DS_2-VASc score has not been validated in the athletic population, stroke risk is still considered to be elevated in small studies even in those with low CHA_2DS_2-VASc score.[534] Furthermore, female sex is considered a risk modifier and age dependent; female sex is added to the score for age > 65 years or $\geq$ 2 non-sex-related stroke risk factors. Recommendations should not be influenced by whether AF pattern is paroxysmal, persistent, or permanent, although there remain insufficient data for anticoagulation for those with overall low burden or short episodes of AF, such as those with < 6 hours of AF.[541–543] As above, for those with AF in the setting of specific disease entities with distinct risk assessment, anticoagulation should follow those guidelines.

   Targeting intermittent anticoagulation around an episode of AF, termed "pill-in-the-pocket" oral anticoagulation, has shown feasibility in pilot studies in those with a low risk of stroke and has been suggested as an alternative approach by some.[544,545] However, the small sample sizes were not powered to evaluate long-term stroke outcomes, and thus data do not support or refute this approach.[544,546] Therefore, pill-in-the-pocket strategy should be based on a shared decision-making process with consideration of overall stroke risk based on comorbidities and need for long-term AF monitoring, especially in those who are asymptomatic. As above, athletes with underlying disease-entities should follow disease-specific anticoagulation recommendations.
3. Athletes who are on anticoagulation for stroke risk with AF have a higher risk of major bleeding if they participate in contact sports (such as football, rugby, wrestling, martial arts, or boxing) or in sports with low but potential risk of traumatic injury (such as bicycling or skiing). No data exist on intermittent withholding of anticoagulation to allow participation in those at high risk of stroke, and in most cases, anticoagulation should not be withheld to allow participation. Data are also lacking on to what degree anticoagulation increases risk of serious bleeding in the setting of injury. A shared decision-making process considering patient preferences and values, overall stroke risk, and type of sport should guide the decision on sport and anticoagulation management.
4. Clinical trials have demonstrated that percutaneous LAAO provides stroke risk reduction similar to that of warfarin, although there remains an upfront procedural risk.[540,547] LAAO has not been studied in the athlete population. Given the potentially lower stroke risk based on a low CHA_2DS_2-VASc score, oral anticoagulation will remain the preferred strategy for stroke prevention in most athletes. In patients with an elevated stroke risk who are poor candidates for long-term anticoagulation, LAAO may be considered after documentation of shared decision-making.[548]
5. There are minimal data addressing the safety of brief periods of withholding anticoagulation. In one study of perioperative direct oral anticoagulation management, withholding anticoagulation 1-2 days prior to surgery was associated with an arterial thromboembolism rate of 0.16%-0.6%.[549] Intermittent withholding of anticoagulation in athletes participating in contact/collision sports with risk of bleeding in the context of drug pharmacokinetics has been suggested in the setting of venous thromboembolic disease. However, data are not available to support the safety of this approach.[550]

8.3.3 Rate and rhythm control in athletes with atrial fibrillation

Recommendations for rate and rhythm control in athletes with atrial fibrillation		
COR	LOE	Recommendations
1	A (QoL) B-NR (Exec. perf.)	1. For symptomatic athletes, maintenance of sinus rhythm is recommended to improve quality of life (A) and exercise performance (B-NR).[551–553]
1	B-R	2. In athletes with symptomatic AF, catheter ablation is recommended as first-line therapy, or if antiarrhythmic drugs are contraindicated or poorly tolerated.[552,554–562]
1	A	3. In athletes undergoing catheter ablation for AF, in whom typical atrial flutter has been previously documented or induced during an EP study, cavotricuspid isthmus ablation is recommended.[563–567]
1	C-EO	4. In athletes with AF and rapid ventricular rate, control of ventricular rate using a beta blocker or nondihydropyridine calcium channel blocker is recommended after consideration of impact on exercise performance and resting heart rate.
1	B-R	5. In athletes with symptomatic paroxysmal AF undergoing ablation without a documented arrhythmia trigger, a pulmonary vein isolation (PVI)-only approach is recommended.[568–570]
2a	A	6. In athletes with infrequent, symptomatic paroxysmal AF, antiarrhythmic drug therapy (with flecainide or propafenone with the addition of a beta blocker or nondihydropyridine calcium channel blocker) is reasonable as a "pill-in-the-pocket" approach if drug therapy is preferred.[571–573]
2a	B-NR	7. In young and young adult athletes with AF, an EP study is reasonable to evaluate an AP or predisposing arrhythmias such as atrial flutter or paroxysmal SVT, for ablation either as a stand-alone procedure or as part of a planned PVI.[512,574–576]
2b	C-LD	8. In athletes with AF, antiarrhythmic drug therapy with flecainide or propafenone with the addition of a beta blocker or nondihydropyridine calcium channel blocker may be considered as a daily medication if drug therapy is preferred.[571,577]
3: Harm	B-R	9. In athletes with AF, catheter ablation to restore sinus rhythm with the sole intent of eliminating the need for long-term anticoagulation should not be performed.[578,579]

Synopsis

Rate and rhythm control strategies should be individualized based on a shared decision-making after discussion of symptoms and risks of medications and catheter ablation and consideration of the athlete's sport and preference. Detraining has limited data in its role in AF treatment and may be challenging to implement over extended periods of time in the athlete. Rhythm control is the preferred strategy in athletes with symptomatic AF. Given the superiority of catheter ablation as compared with antiarrhythmic drug therapy in reducing AF recurrence in the nonathlete population and the likely higher intolerance to medical therapy in the athletic population, as well as data showing equivalent efficacy in athletes, catheter ablation can be recommended as first-line therapy. An algorithm of the recommendations for treatment of AF in athletes including risk factor management, prevention of thromboembolism, and rhythm control in athletes with AF is shown in Figure 16.

Recommendation-specific supportive text

1. AF has been shown to negatively impact quality of life and exercise performance in the general population. Although no studies have specifically evaluated these outcomes in athletes, this remains particularly relevant to the athlete with AF. Rhythm control, primarily with catheter ablation, has shown to improve quality of life and exercise performance as evaluated by CPET in the nonathlete population.[551–553]
2. Clinical trial data have demonstrated the superiority of catheter ablation with PVI as compared with antiarrhythmic drug therapy in prevention of AF recurrence and improvement in quality of life with low risk of adverse events.[552–557] Several observational studies have shown no difference in AF recurrence after ablation in athletes as compared with nonathletes.[558–561] In one small series,[580] quality of life and training time increased after ablation. An early rhythm control strategy with ablation is useful as first-line therapy[581] or if the athlete does not tolerate antiarrhythmic drugs. The 2023 ACC/AHA/ACCP/HRS guideline on AF[16] describes that catheter ablation as a first-line therapy for all patients with AF who prefer this approach is useful (class I), and for athletes specifically, as reasonable (2a). Given that AF ablation is shown to be as effective in athletes as nonathletes, this writing committee has determined that catheter ablation is recommended in
Based on consensus, intensive exercise should be restricted for at least 2 weeks post-procedure to avoid vascular complications at line insertion sites in the groin. Resumption of exercise should begin with moderate levels

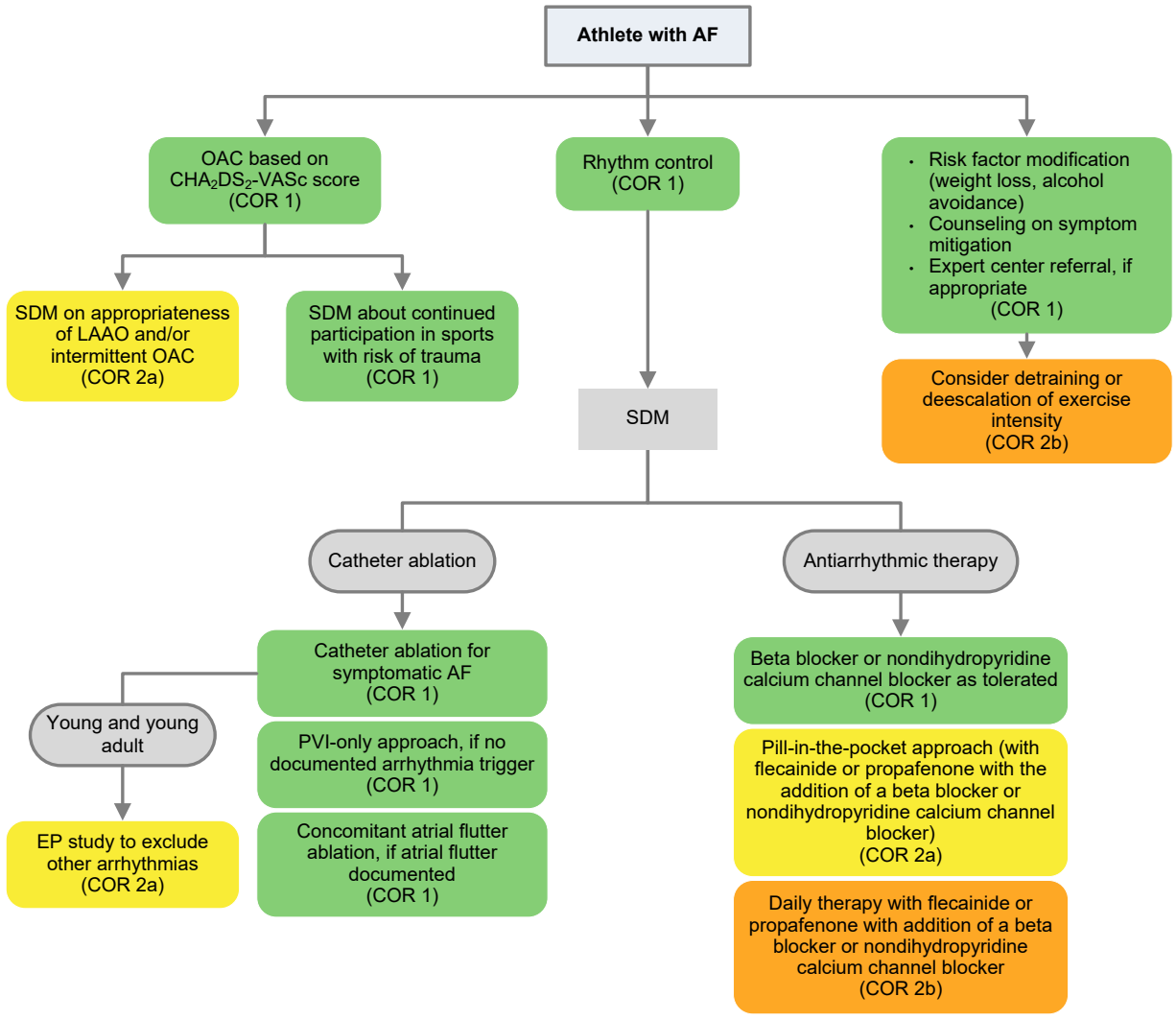

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Figure 16

Algorithm for the evaluation and management of athletes suspected of having atrial fibrillation (AF). Colors correspond to the class of recommendation (COR) in Table 1. AF = atrial fibrillation; ECG = electrocardiogram; EP = electrophysiology; LAAO = left atrial appendage occlusion; OAC = oral anticoagulation; MRI = magnetic resonance imaging; PVI = pulmonary vein isolation; SDM = shared decision-making.

of activity and increased over the following weeks to regular intense activity, assuming no post-procedural complications or symptomatic AF recurrence. While post-ablation inflammation may increase the risk of AF for the 2-3 months after the procedure,[582] whether vigorous exercise during this period increases the short- or long-term risk of recurrence after ablation has not been evaluated.

3. Empiric cavotricuspid isthmus (CTI) ablation at the time of PVI has not been shown to improve freedom from long-term atrial arrhythmia recurrence,[563–565] and thus empiric CTI ablation without documentation of typical atrial flutter is not recommended.[567,583] However, if typical atrial flutter has been documented or induced, CTI ablation is recommended, as atrial flutter may precede and induce AF. In those with only typical atrial flutter without AF, CTI ablation is recommended as first-line therapy.[564,584,585]
4. In athletes with rapid ventricular rate during AF, rate control is recommended with or without a rhythm control strategy. Beta blockers and nondihydropyridine calcium channel blockers may lead to fatigue or impact exercise performance,[354] although a low or moderate dose may be tolerated in some athletes. Importantly, beta blockers are prohibited by the World Anti-Doping Agency only in sports that rely on stability of the upper extremities, such as archery, golf, or shooting.[46]
5. Given the unclear benefit of empiric adjunctive lesions beyond PVI particularly in paroxysmal AF, such as left atrial linear ablation, posterior wall isolation, left atrial appendage isolation, superior vena cava isolation, or complex fractionated electrogram ablation, further empiric ablation does not provide benefit beyond PVI alone and should be avoided given the possible excess risk of the procedure.[569,570,586] In addition to procedural complications, extensive catheter ablation may result in stiff left atrial syndrome impacting exercise performance.[568,587]
6. In the absence of structural heart disease or coronary artery disease, class IC agents may be the preferred antiarrhythmic drugs in the athlete with AF due to minimal side effects and

when used as the pill-in-the-pocket approach. Although serious adverse events are rare, initiation of pill-in-the-pocket strategy should be performed in a monitored setting.[572] Importantly, class IC agents should be coadministered with an AV nodal blocker. While data are lacking in athletes, based on the potential for proarrhythmias with class IC agents (see the supportive text for recommendation 8 below), the general absence of stress test to evaluate for QRS widening with exercise in pill-in-the-pocket users, and the drug pharmacokinetics, sport participation should be avoided for 48 hours to allow drug clearance, to minimize the risk of 1:1 atrial flutter and ventricular proarrhythmia.

7. Rapid atrial activation during an SVT may trigger AF particularly in young patients. Inducible SVT during an EP study, such as AV nodal reentrant tachycardia or AV reentrant tachycardia, has been observed in up to 39% of young patients with AF with ablation resulting in decreased AF recurrence.[512,574–576] The 2023 ACC/AHA/ACCP/HRS guideline on AF[16] states that in patients with early-onset AF, an EP study and targeted ablation of any SVTs found "may be reasonable" (2b). This writing group has determined that given the high prevalence of other supraventricular arrhythmias, and the high rate of arrhythmia-free success with a targeted ablation in the young and young adults, this approach is reasonable (2a) either as a standalone procedure or as part of planned PVI.
8. Like the general population, athletes who have frequent episodes of AF who prefer antiarrhythmic drug therapy will require daily medication as compared with a pill-in-the-pocket approach. In the absence of structural or coronary heart disease, flecainide or propafenone along with an AV nodal blocker may be considered an initial strategy, although these may be poorly tolerated due to the negative impact on exercise performance and should be used with care in athletes in light of their use-dependence and risks of proarrhythmia and development of atrial flutter with 1:1 conduction.[588,589] Exercise stress test should be performed after initiation of class IC agents to monitor for use-dependent QRS widening. If palpitations occur, activity should be stopped and rhythm diagnosis made. Dronedarone is another option, although efficacy may be less.[590,591] Class III agents such as sotalol or dofetilide may be considered; however, these should be used with care in athletes at risk of dehydration due to transient renal failure leading to drug accumulation and proarrhythmic drug effects.[16] Sotalol may be poorly tolerated due to the beta blocker effect of decreased exercise performance. Disopyramide may be considered in athletes with vagal-mediated AF given the anticholinergic effect of the medication. Amiodarone should be considered as relatively contraindicated or used as last resort due to the significant toxicities.
9. AF catheter ablation to restore sinus rhythm should not be performed with the sole intent of eliminating the need for anticoagulation. The indication for anticoagulation should be based on the stroke risk assessment by the CHA_2DS_2-VASc score. There are currently no data supporting the use of a rhythm control strategy with catheter ablation to remove the need for anticoagulation.[583] As most studies have not shown ablation to reduce stroke risk, and given the increased stroke risk in those who discontinue anticoagulation after ablation,[578,579] current guidelines[16] recommend continuation of anticoagulation after ablation if CHA_2DS_2-VASc score is ≥ 2. A shared decision-making discussion based on competing risks of stroke along with consideration for long-term monitoring of AF is needed to guide anticoagulation strategies. A low threshold for anticoagulation continuation should remain in those at high risk of stroke.

Section 9 Wolff-Parkinson-White pattern and syndrome

The following terms are used to differentiate between WPW pattern and syndrome in this document:

- **WPW pattern:** The term "WPW pattern" refers to patients with preexcitation manifest on an ECG in the absence of symptoms.
- **WPW syndrome:** The term "WPW syndrome" refers to patients with both preexcitation manifest on an ECG and symptomatic arrhythmias involving the AP.

As described in Section 2, the use of ECG screening in preparticipation evaluation of athletes is controversial, with some professional societies in favor, and others less so. Regardless, the use of ECG screening is increasing, with one survey of NCAA team physicians describing use of ECG screening in 38% of schools and in 50% of Division 1 programs.[592] Some regions in the United States and in other countries have mandated ECG screening for the preparticipation exam in high school sports. These ECGs detect potentially lethal conditions in 0.3% of athletes, with the most common finding, WPW pattern, representing almost half of the abnormalities found.[129] WPW syndrome accounts for 1% of SCD in the athletic population based on a long-term registry,[116] but the exact incidence is not known because the autopsy detail necessary to diagnose WPW syndrome as a cause of sudden death is difficult. It is likely that a proportion of sudden death secondary to WPW syndrome is attributed to "autopsy negative" SCD, which is recognized as a common etiology of SCD in contemporary studies in young and athletic individuals.[82,92,593] As described in detail below, incidence of WPW pattern, and death due to WPW syndrome, does not differ in athletes and nonathletes. However, as athletes represent the population of young, asymptomatic individuals most likely to undergo ECG and thus be found with WPW pattern, management of WPW pattern is uniquely relevant to this group.

9.1 Epidemiology and natural history

The prevalence of WPW pattern is 0.1%, as documented in the Copenhagen Baby Heart Study looking at ECGs for over 17,000 neonates.[594] It is usually an isolated abnormality in an otherwise normal heart in a healthy person. In a minority it occurs as part of structural, congenital, or genetic cardiac

conditions, notably Ebstein malformation of the tricuspid valve, cardiac rhabdomyomas, and HCM often associated with glycogen storage and regulation disorders.[595–599]

Both epidemiological and EP study–based data have demonstrated that the incidence of life-threatening events (LTEs) is higher in individuals with a WPW pattern on ECG and symptoms (ie, WPW syndrome). The clinical presentation and natural history of individuals with the WPW pattern on ECG (ie, without symptoms) are highly variable and may differ with age, AP location, and AP conduction properties.[595,600–603] Most individuals with preexcitation (the WPW pattern) never experience an arrhythmia and are found to harbor the abnormality incidentally, for instance, on an ECG performed during preparticipation athletic screening. Rarely, patients with WPW syndrome present with a cardiomyopathy due to the dyssynchronous ventricular contraction associated with a preexcited sinus rhythm; this is most often seen in children.[604–606] Finally, syncope or aborted SCD may be the first manifestation of WPW syndrome.[596,607] Although most patients with WPW syndrome resuscitated from SCD have had prior symptoms, a cardiac arrest may be the sentinel event, particularly in children and adolescents.[607] The overall incidence of SCD in WPW syndrome is most often quoted as 1 event per 1000 person-years, but risk has been observed to be higher in children, with one meta-analysis reporting a rate of 1.93 events per 1000 person-years in asymptomatic children compared with 0.86 events per 1000 person-years in asymptomatic adults.[608] Of note, geographical differences have been cited in the reported risk of WPW syndrome–related SCD.[608] In Italian studies,[609–613] the risk is estimated at 2.16 per 1000 person-years, which is higher than the 0.36 per 1000 person-years (95% CI, 0.05–0.94) reported in non-Italian studies.[614]

Previous guidelines have advocated a more aggressive approach to the management of WPW pattern in competitive athletes and patients with high-risk occupations.[9,615] Theoretically, during exercise, fluctuations in autonomic tone and exercise-related adrenergic stimulation may increase vulnerability to atrioventricular reciprocating tachycardia (AVRT), which in turn may increase the risk of AVRT disorganizing to AF, or even preexcited AF independent of AVRT, culminating in VF and SCD. Studies have shown that exercise-induced adrenergic activation increases AP conduction.[616,617]

However, while WPW pattern may be found more commonly in athletes due to ECG screening in this population, epidemiological studies do not suggest that SCD from just having the WPW pattern is more common in the athlete versus the nonathlete, and training does not alter the conduction properties of the AP.[618] In a series of children with WPW syndrome who had experienced an LTE,[607] this event occurred most often at rest or with noncompetitive activity. Activities of daily life may also precipitate an arrhythmia, and therefore SCD is not limited to the athlete and LTEs are not limited to athletic activities.[607,619]

Given the lack of evidence showing a difference in the risk of WPW-related SCD in athletes versus nonathletes, while the recommendations in Sections 9.1 and 9.2 refer to the athlete population, they may be considered for all patients with WPW pattern and a structurally normal heart, irrespective of athletic participation.

Based on data showing that the risks of pediatric patients and adults differ, with the incidence of LTEs in children with a WPW pattern being much higher than in adults, the strengths of recommendations in this section have been adjusted for individuals < 18 years (prepubertal and adolescent) and ≥ 18 years of age (young adult and adult).[4,608,614,620]

9.2 Evaluation of athletes with Wolff-Parkinson-White pattern or syndrome

Recommendations for the evaluation of athletes with Wolff-Parkinson-White pattern or syndrome

COR	LOE	Recommendations
1	C-LD	1. In athletes with a WPW pattern on ECG, cardiac evaluation including physical examination, family history, and echocardiography at diagnosis is recommended whether symptoms are present or absent.[595,621]
1	C-LD	2. In athletes with a WPW pattern and no symptoms, return to play during evaluation and pending treatment is recommended.[607]
1	C-EO	3. In athletes with a WPW pattern, regardless of symptoms, shared decision-making among patients, families, and their electrophysiologist is recommended regarding catheter ablation and/or EP studies, which includes a discussion of procedural risks, benefits, AP recurrences after ablation, and limitations of risk stratification tools.
1	C-LD	4. In athletes with a right anteroseptal WPW pattern on ECG, pharmacologic testing with intravenous adenosine and/or an EP study should be performed to rule out a fasciculoventricular pathway before attempting catheter ablation, as fasciculoventricular pathways do not participate in the AVRT circuit and are not associated with rapid antegrade conduction.[622,623]
1	C-LD	5. In athletes with a WPW pattern who either choose not to have an ablation or have an unsuccessful ablation, periodic cardiology follow-up is recommended regardless of symptoms.[624]
1	C-LD	6. In athletes with WPW pattern or syndrome who have had a catheter ablation, periodic cardiology follow-up is recommended for at least 1 year post-procedure to evaluate for AP recurrence.[625]

Synopsis

The lifetime risk of mortality related to WPW pattern appears to be numerically low. However, the risk seems to be "front loaded" in the earlier decades of life,[4,607,608,620] and SCD can be the sentinel event in a child with WPW pattern. Several decades of research has shown that risk stratification in WPW pattern is imperfect. The greatest concern is the ability of the AP to conduct rapidly to the ventricles, which has been traditionally measured by noninvasive parameters such as persistent preexcitation or intermittent loss of preexcitation on ambulatory monitoring, serial ECGs, and exercise stress testing, and invasively with EP studies to assess the shortest and average intervals between preexcited beats during AF (shortest preexcited R-R interval [SPERRI]) and accessory pathway effective refractory period (APERP) (Table 18). Recent data have shown that the noninvasive and even invasive parameters previously thought to reassure the clinician that an AP had a "low" or "high" risk for rapid ventricular conduction may not accurately risk stratify all WPW pattern patients.[607,626,627] Table 18 describes the evolution of recommendations for evaluation and treatment of WPW pattern in prior documents based on the evolving data as above.

Recommendation-specific supportive text

1. WPW has been associated with CHD, most commonly Ebstein malformation of the tricuspid valve and congenitally corrected transposition of the great arteries,[595] hypertrophic and infiltrative cardiomyopathy typically associated with *PRKAG2* gene variants,[598] cardiac rhabdomyomas,[599] tachycardia-induced cardiomyopathy, and dyssynchrony-related cardiomyopathy.[604] Thus, a complete evaluation and an echocardiogram are indicated in every patient with WPW pattern.
2. As above, in series of WPW patients experiencing LTEs,[607] these events occurred most often at rest or with noncompetitive activity.[607,619] Thus, there is no evidence that return to play during evaluation would increase risk.

Table 18 WPW pattern on ECG: Differences in the class of recommendation between 4 previously published expert consensus statements and this 2024 expert consensus statement

Recommendation	2003 ACC* (adults)	2015 ACC† (adults)	2012 PACES‡	2019 ESC§	2020 ESC˙	2024 HRS¶
Exercise stress test for risk stratification of asymptomatic patients with WPW pattern on ECG	n/a	1, B-NR	2a, B/C	2b, B	n/a	n/a
EP study for risk stratification in asymptomatic patients with WPW pattern on ECG	n/a	2a, B-NR	2a, B/C	1, B	1, B	1, B-NR
Catheter ablation in patients with WPW pattern on ECG with symptoms and/or arrhythmias	1, B	1, B-NR	n/a	1, B	1, C	1, B-NR
Catheter ablation in asymptomatic patients with WPW pattern on ECG and high-risk markers	2a, B	2a, B-NR	2a, B/C	1, B	n/a	1, B-NR
Catheter ablation in asymptomatic patients with WPW pattern on ECG without high-risk markers	2a, B	2a, B-NR	2b, C	2b, C	n/a	2a, C-LD
Catheter ablation in asymptomatic patients with WPW pattern on ECG and ventricular dysfunction secondary to LV dyssynchrony	n/a	n/a	2b, C	2b, C	n/a	1, C-LD

ECG = electrocardiogram; EP = electrophysiology; LV = left ventricular; n/a = not applicable (there is no equivalent recommendation); WPW = Wolff-Parkinson-White.

*2003 ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmia—executive summary.[640]

†2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular arrhythmia.[641]

‡2012 PACES/HRS expert consensus statement on the management of the asymptomatic young patient with a Wolff-Parkinson-White (WPW, ventricular preexcitation) electrocardiographic pattern.[4]

§2019 ESC guidelines for the management of patients with supraventricular tachycardia.[642]

˙2020 ESC guidelines on sport cardiology and exercise in patients with cardiovascular disease.[9]

¶2024 HRS expert consensus statement on the management of arrhythmias in the athlete: Evaluation, treatment, and return to play

3. Although the reported risk of SCD in WPW is numerically low, data suggest that the incidence of LTEs in children with asymptomatic WPW pattern may be much higher than in adults and is higher than in children without.[4,607,608,614,620] Given the limitations of current risk stratification strategies, it may be difficult to identify who is at risk for SCD. Therefore, the pediatric EP community has adopted a low threshold for offering catheter ablation as a treatment strategy to all children with a WPW pattern.[628] This may result in overtreatment of asymptomatic patients who are actually not at risk for SCD. Although ECG algorithms have improved over time,[629] localization of APs is imperfect, and the EP study can clarify both anatomic location and arrhythmia risk. Contemporary studies have shown high efficacy and low complications with catheter ablation,[624,630–634] and to date there have been no reported cases of permanent AV block with cryoablation of APs near the conduction system. However, no invasive procedure is without risk. In addition, studies have shown that 5%-12% of APs recur during follow-up.[624,630,631,634] The patient and family should be engaged in a shared decision-making process in which both conservative and more aggressive options are presented and discussed on a case-by-case basis.[202,635] Specifically, they should be informed of the small risk of life-threatening arrhythmias developing in the absence of treatment, along with the success rate and complications associated with catheter ablation of the AP as well as the infrequent risks associated with a standalone EP study.
4. Fasciculoventricular APs are rare variants of preexcitation. These pathways demonstrate minimum preexcitation on the ECG and are uncommon but may also be underrecognized. Fasciculoventricular ECGs may be misinterpreted as WPW pattern with right anteroseptal APs; therefore, it is important to differentiate the two entities. Typically, fasciculoventricular pathway ECG characteristics are (1) a shorter QRS duration (< 130 ms), (2) a not-so-short PR interval (110–120 ms), (3) a flat or negative delta wave in V1, (4) a narrow delta/R wave in V2, (5) S wave amplitude < 20 mm in V1, and (6) notching in the descending limb of the S wave in V1.[636,637] These pathways are completely infranodal structures, so there is no risk of rapid antegrade conduction during atrial tachyarrhythmias. However, fasciculoventricular pathways may be associated with other tachycardia circuits as a bystander, and recognizing their bystander role is crucial in order not to attempt unnecessary, and potentially harmful, ablation close to the His bundle region. This diagnosis during an EP study can be made by (1) multisite atrial pacing that results in no effect on the preexcitation degree in a fasciculoventricular pathway, (2) constant and positive HV interval with incremental and programmed atrial stimulation, (3) preexcited junctional beats, and 4) AV block or increase in PR interval without change in QRS morphology in response to intravenous adenosine.[622,623,638] Patients with a fasciculoventricular pathway associated with a *PRKAG2* variant have distinct clinical, ECG, echocardiographic, and electrophysiologic profiles and should be correctly identified because of their ominous long-term prognosis. Patients without a variant have an excellent arrhythmia-free prognosis.[639]
5. If after a shared decision-making discussion, the patient with WPW pattern and/or their family/caregiver elect not to proceed with an EP study and/or ablation, or if there is failure to eliminate the AP with ablation, routine cardiology follow-up is indicated to monitor for the development of symptoms.[624] Whether alternative treatment is needed after a failed ablation will depend on clinical and electrophysiological characteristics.
6. In patients with WPW pattern who have had a catheter ablation, periodic cardiology follow-up is recommended for at least 1 year after the ablation procedure to evaluate for AP recurrence. Follow-up is recommended for symptoms suggestive of SVT and to monitor the ECG for reappearance of WPW pattern.[624] Recurrence of anterograde and/or retrograde AP conduction after catheter ablation occurs in 5%-12% of cases.[624,630,631,634] Although this recurrence is more likely within the first 36 hours of the ablation procedure,[625] it can occur $\geq$ 1 year after the ablation procedure.

9.3 Treatment of athletes with Wolff-Parkinson-White

Recommendations for the treatment of athletes with Wolff-Parkinson-White		
COR	LOE	Recommendations
1	B-NR	1. In athletes with a WPW pattern on ECG and symptomatic or documented arrhythmias, catheter ablation of AP(s) is recommended to manage the symptoms caused by the arrhythmia and reduce the risk of LTEs.[600,611,625,630,631]
1	C-LD	2. In athletes with a WPW pattern on ECG and left ventricular dysfunction attributed to left ventricular dyssynchrony, catheter ablation of AP(s) is recommended to improve left ventricular remodeling and left ventricular function regardless of anterograde characteristics of the AP(s).[604,606,621]
1	B-NR	3. In young athletes (aged < 18 years) with a persistent WPW pattern on the ECG, an EP study is recommended to identify high-risk* AP(s) properties.[607,611,628]
2a	C-LD	4. In young athletes (aged < 18 years) with an intermittent WPW pattern on the ECG, an EP study is reasonable to identify high-risk* AP(s) properties.[627,628]
2a	B-NR	5. In young adult and adult athletes (aged ≥ 18 years) with a persistent WPW pattern on the ECG, an EP study is reasonable to identify high-risk* AP(s) properties.[611,613]
2b	B-NR	6. In young adult and adult athletes (aged ≥ 18 years) with an intermittent WPW pattern on the ECG, an EP study may be considered to identify high-risk* AP(s) properties.[611,613,643]
1	B-NR	7. In athletes with a WPW pattern on ECG and ≥ 1 high-risk properties* present during the EP study, catheter ablation of AP(s) is recommended to prevent LTEs.[607,628,644]
2a	C-LD	8. In athletes with a WPW pattern on ECG and without high-risk properties* identified on EP testing, catheter ablation of AP(s) is reasonable.[628,645]
2a	C-LD	9. In athletes with a WPW pattern on ECG undergoing ablation, cryoablation is reasonable in those with anteroseptal and midseptal AP(s) to reduce the risk of permanent injury to the conduction system.[634,646–649]
3: Harm	C-LD	10. In athletes with a WPW pattern on ECG found to be due to fasciculoventricular pathway, catheter ablation should not be performed due to potential harm to the conduction system.[622,623]

*High-risk markers: spontaneous or inducible SVT, APERP ≤ 250 ms, SPERRI ≤ 250 ms, multiple APs.

Synopsis

Catheter ablation of an AP, when performed by an experienced operator, is associated with a high cure rate (>90%) and low risk (<1%) of major complications.[624,630–632,634,650,651] Significant complications associated with catheter ablation of APs include but are not limited to AV block, cardiac perforation, stroke, and coronary artery injury.[624,630–634] With the use of cryoablation, there are currently no reported cases of permanent AV node injury requiring a pacemaker.[647–649] In the last 15 years, catheter ablation of APs is routinely performed with zero or minimal fluoroscopy.[652,653] Given that both noninvasive and even invasive testing do not confer absolute certainty about risk, the threshold for catheter ablation in WPW as a strategy for preventing SCD is lower in children including those with a "low-risk" AP (Figure 16). AP recurrences occur despite accurate mapping and initially successful elimination of APs. Although the complication rate is low, an ablation procedure cannot be guaranteed to be without risk. Specific recommendations, even when supported by substantial data, do not replace the need for clinical judgment and patient-specific decision-making, which should involve shared decision-making among the patient, their family, and the electrophysiologist performing the procedure. An algorithm of the recommendations for the treatment of athletes with WPW is shown in Figure 17.

Recommendation-specific supportive text

1. Children and young adults with WPW syndrome typically present with arrhythmia symptoms, but the prevalence of documented SVT may be lower and is age related.[4,600] Both epidemiological and EP study–based data demonstrate that symptoms are a predictor of higher risk for LTEs. In the pivotal study by Klein et al,[596] VF during AF with rapid conduction over the AP occurred with a higher prevalence in patients with a prior history of documented arrhythmias (AVRT and/or AF compared with those who were asymptomatic and without documented arrhythmias). In a 5-year prospective study,[610] malignant presentation with rapid preexcited AF was reported in 7% and cardiac arrest in 1.4% of previously symptomatic patients with WPW syndrome, suggesting that a subset of symptomatic patients may eventually experience more catastrophic arrhythmic events, including syncope, hemodynamic collapse, and/or cardiac arrest. In a prospective study of 2169 patients,[611] VF occurred in 1.5% of 1001 patients with WPW syndrome who did not

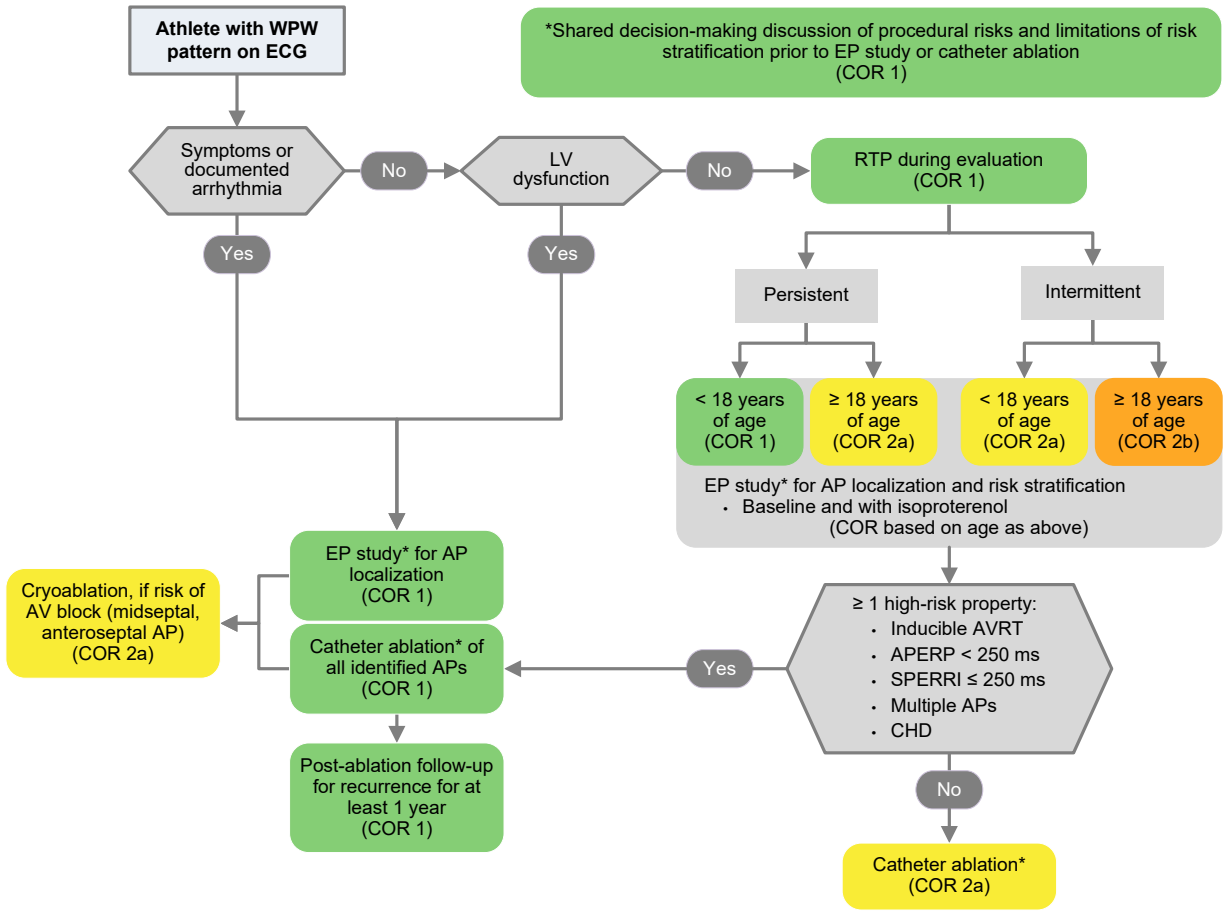

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Figure 17

Algorithm for the treatment of Wolff-Parkinson-White (WPW) pattern in athletes. Colors correspond to the class of recommendation (COR) in Table 1. AP = accessory pathway; APERP = accessory pathway effective refractory period; AV = atrioventricular; AVRT = atrioventricular reciprocating tachycardia; CHD = congenital heart disease; ECG = electrocardiogram; EP = electrophysiology; LV = left ventricular; SPERRI = shortest preexcited R-R interval.

undergo catheter ablation, whereas no patients with a successful catheter ablation developed malignant arrhythmias over an 8-year follow-up period, making a strong case for catheter ablation as a beneficial treatment to prevent AVRT and SCD in WPW syndrome. In accordance with prior scientific guidelines,[640–642,654] a class 1 indication for catheter ablation in patients with WPW syndrome remains unchanged in this document. Studies have established that AVRT triggering AF is an independent predictor of the risk for SCD in WPW syndrome.[596,609,610] The majority of initially symptomatic patients with WPW syndrome may remain asymptomatic after the first episode of AVRT or experience an SVT recurrence without SCD, but it is not possible to predict the natural history of each individual patient. In the current era, the established efficacy and safety of catheter ablation in permanently eliminating APs with a low risk of serious complications and ability to perform procedures with zero or minimum radiation exposure[630,631,633,634] justifies catheter ablation as the first-line therapy for reducing risk of SCD as well as AVRT in patients with WPW. While data are lacking, following ablation, a waiting period of 1-2 weeks to allow healing of incision sites is generally observed.

2. Ventricular preexcitation-associated cardiomyopathy is defined as left ventricular dysfunction in the absence of sustained tachyarrhythmias and is different from tachycardia-induced cardiomyopathy, as left ventricular dysfunction is attributed to chronic electromechanical dyssynchrony from alterations in cardiac contraction.[655] Preexcitation-induced cardiomyopathy occurs predominantly in the setting of right-sided pathways.[621] Abnormal early activation of the right ventricle from right-sided pathways leads to late activation of the interventricular septum and left ventricular myocardium.[656] Although the "typical" age of presentation is unknown, preexcitation-induced dysfunction can occur in infancy,[657] may be severe enough to necessitate mechanical support,[658] and affects those with intermittent as well as persistent preexcitation.[659] In children this usually responds favorably to loss of preexcitation by spontaneous resolution or catheter ablation, which allows for mechanical resynchronization, reverse remodeling, and improvements in ventricular function.[606]
3. Increasingly, children (aged < 18 years) with WPW pattern are identified when preexcitation is incidentally detected on ECG performed for sports screening, medication administration, or other noncardiac reasons. The lifetime

risk of SCD in those with WPW pattern is low, yet it is "front loaded" in the young.[660] A meta-analysis[608] evaluating sudden death among patients with an asymptomatic WPW pattern identified an LTE rate of 1.93 per 1000 person-years in children and 0.85 per 1000 patient-years (< 0.1% per year) in adults. In the prospective WPW registry study,[611] among the 15 patients who had VF over the 8-year follow-up period, 13 (86%) were children (median age 11 years). Determining which patients with a WPW pattern are at highest risk for LTEs by history alone remains a challenge. Very young children are less reliable communicators of symptoms than adults. Even older children may not report or recognize symptoms and can have symptoms that are not typical of an arrhythmia.[612] Present data confirm that asymptomatic WPW pattern is not without risk, and malignant arrhythmias correlate better with EP study–derived risk stratification than the presence or absence of symptoms.[607,610] In a contemporary study of pediatric WPW syndrome,[607] the LTE was the sentinel symptom in 65%. Given the potential for a more malignant course of WPW in children irrespective of symptoms, benefit of directly proceeding to an EP study regardless of the outcome of noninvasive testing likely outweighs the risk. This is a change that has occurred since the published recommendations from the 2012 consensus document[4] based on contemporary data.[607,608,626,627,661] Data affirm that SPERRI < 250 ms, anterograde APERP ≤ 240 ms, and the presence of multiple AP and Ebstein malformation of the tricuspid valve are useful markers of high-risk APs[607,610,612] but remain imperfect predictors, and young patients may experience LTEs from WPW syndrome without prior symptoms or markers of high risk on EP study.

4. It has been postulated that patients with intermittent preexcitation generally have poor antegrade conduction characteristics and therefore a lower risk of SCD.[4] However, there are no large studies regarding intermittently conducting APs with regard to long-term risk of developing VF. There are recent reports of intermittent preexcitation in patients with fast anterograde AP conduction during an EP study and also presentation with an LTE.[662–666] Orczykowski et al[667] investigated 1007 patients with APs and reported that 7% of the 56 patients who experienced SCA had intermittent preexcitation on resting ECG. Other studies[643,664,665] found that APs with intermittent conduction were capable of very fast anterograde conduction, especially when tested during isoproterenol infusion at an EP study. It is possible that fast anterograde conduction observed in cases of intermittent preexcitation mostly reflects the high catecholamine sensitivity of some APs. Aside from catecholamine sensitivity, repetitive retrograde penetration of the AP (linking) can contribute to the presence of intermittent preexcitation despite short AP anterograde refractory period, and this mechanism might act synergistically with catecholamine sensitivity.[668] Therefore, in patients with intermittent WPW pattern on the ECG, an EP study with isoproterenol is reasonable to identify high-risk APs properties.
5. As outlined above, the prognosis and risk of the WPW pattern is lower in adults compared with children, which is presumably driven by survival bias with a lower residual risk. Moderate-quality data support a differential risk, with the greatest risk in symptomatic patients with persistent WPW pattern, followed by asymptomatic WPW pattern, followed by intermittent preexcitation.[596,614,643] The lifetime risk of sudden death in WPW is low, emphasizing the importance of shared decision-making given the competing risks of undergoing diagnostic EP studies, that frequently lead to ablation, compared to the natural history rate of approximate 0.86 events per 1000 person-years.[608] Age plays a role in risk and shared decision-making, with male sex, previous syncope, and age < 30 years representing risk factors for adverse outcomes.[596,607,669–671] If an EP study is pursued, consideration should be given to the fact that EP study–derived risk stratification is imperfect, and a low threshold for ablation should be considered.
6. As outlined above, clear evidence of abrupt loss of preexcitation with exercise stress testing or during ambulatory monitoring predicts a longer AP refractory period and a longer SPERRI.[661] This associates with lower risk of rapidly conducted AF and associated degeneration to VF. The residual risk is undoubtedly very low but not zero, based largely on case reports and more robust data in pediatric series.[626,627,664] Though generally a reason to convey low risk and not proceed with an EP study and ablation, shared decision-making is important, and proceeding with invasive risk stratification and possible ablation is reasonable.
7. For patients with a WPW pattern on ECG, high-risk APs are associated with younger age, the presence of multiple APs, CHD, SPERRI ≤ 250 ms, anterograde APERP ≤ 240 ms, or AVRT precipitating preexcited AF or atrial flutter.[4,596,610,641,644,654,672] Based on the seminal study by Klein et al,[596] SPERRI ≤ 250 ms during an EP study has become the "gold standard" for invasive risk assessment in patients with a WPW pattern on the ECG. Studies have shown SPERRI ≤ 250 ms to be more sensitive at detecting high-risk AP than the antegrade AP effective refractory period in children and adults.[596,673] Bromberg et al[669] evaluated symptomatic children with WPW ≤18 years of age and found that all patients with clinical VF in whom AF was inducible at EP study had a SPERRI ≤ 220 ms (sensitivity 100%) and concluded that SPERRI ≤ 220 ms was more sensitive than clinical history for identifying those at risk for SCD. The sensitivity of SPERRI in AF is 88%–100% for identifying adult patients with WPW at risk for VF, but because of the low incidence of cardiac arrest in these patients, the positive predictive value of a SPERRI in adults is low.[596,608,674] The negative predictive value of the SPERRI ≥ 250 ms is well established.[608,675] Studies have shown that catheter ablation of APs markedly reduces the frequency of arrhythmic events and may prevent SCD in patients with ≥ 1 high-risk markers.[610,612,644] It is important to note that the

use of isoproterenol markedly increases the prevalence of positive assignment of patients to a high-risk category.[611,673,676,677] Conversely, the effects of anesthetic agents may have similar capacity to affect the AP properties that are used to determine indications for catheter ablation.[607,645] It appears logical that sympathetic stimulation in a patient with AF and manifest preexcitation may enhance AP conduction to become more rapid and degenerate to VF and enhance AV nodal and retrograde AP conduction rendering baseline noninducible AVRT to become inducible. Isoproterenol use in the EP laboratory may therefore be considered a means to counterbalance the effects of sedation and anesthesia, but to what extent it represents physiologic stress or exertion is unknown.[673,674,678]

8. Studies have shown that while the direct measurement of AP characteristics are a better measure of risk than symptoms or noninvasive testing, these measures are imperfect, especially in children.[607,645] In a large international multicenter case-control registry study of 912 children,[607] although survivors of an LTE had significantly shorter APERP, SPERRI, and shortest preexcited paced cycle length (SPPCL) values than WPW syndrome controls without LTEs, over one-third had a SPERRI $\geq$ 250 ms and would have been classified as low risk even though they had experienced a high-risk clinical event. One explanation for this may be that most pediatric EP studies are performed under general anesthesia, which may hinder the ability to measure pathway conduction accurately.[645] Considering the concerns with accurate invasive risk stratification, the majority of pediatric electrophysiologists surveyed ablate most APs, even in patients with asymptomatic WPW pattern, regardless of risk assessment measures. An exception to this may be the low-risk septal pathways, where risk of heart block is low but not zero, potentially modifying the risk-benefit ratio.[628]
9. Radiofrequency ablation in the septal arrhythmia substrates has low but irreversible risk of AV block leading to permanent pacemaker implantation. However, with the advent of transcatheter cryoablation, anteroseptal and midseptal APs can be ablated without permanent damage to the conduction system. Currently, there are no reports in the literature of high-grade AV requiring a permanent pacemaker after cryoablation of an AP. Several studies have previously reported a lower acute success rate and a higher recurrence rate with cryoablation of septal APs when compared with radiofrequency ablation.[646] The higher recurrence rate may be attributed to the limitations in achieving effective lesions associated with the tip size of the cryoablation catheter as well as defining the cryothermal application time and temperature profile[647,648] for successful AP elimination. With over 15 years of worldwide experience in cryoablation of arrhythmias in children, techniques continue to be refined so that the success rate of cryoablation of anteroseptal and midseptal pathways is comparable to radiofrequency ablation (85%-95% success rate, 12%-18% recurrence rate)[647,648] and the safety profile significantly superior to radiofrequency ablation.[631,634,679,680]
10. Fasciculoventricular APs arise from the His bundle and the fascicles and usually connect to the right ventricle, in which case the ECG features mimic a right anteroseptal AP. They have only anterograde conduction with decremental properties and are not substrates for reentrant tachycardia or sudden death and should not be ablated. If SVT is present, it is important to recognize that the fasciculoventricular pathway is a bystander and not integral to the SVT circuit. The diagnosis is critical to avoid unnecessary and potentially harmful ablation to the AV node and His bundle region.[637,638]

Section 10 Bradycardia and pacemakers

10.1 Athletes with bradycardia

Sinus and AV nodal slowing are expected adaptations to athletic training, particularly in high vagal states such as sleep or at rest. These changes are dose dependent, with one study showing a significant difference in sinus bradycardia (< 3 hours/week vs 3-6 hours/week) and first-degree AV-block (< 3 hours/week vs > 10 hours/week) based on current sport exposure and, similarly, a significant difference in sinus bradycardia (0-1000 hours vs 2001-3000 hours) and first-degree AV-block (0-1000 hours vs > 4000 hours) in lifetime sport exposure.[681] These adaptations are likely due to both changes in autonomic balance and ion-channel effects on the sinus and AV nodes.[682,683]

Recommendations for athletes with bradycardia

COR	LOE	Recommendations
1	B-NR	1. For athletes with significant distal conduction disease, including left bundle branch block, bifascicular block, or complete heart block at any level, evaluation prior to return to play is recommended.[684,685]
2a	C-EO	2. In athletes with significant sinus and/or AV node disease that does not correct with light exercise, further evaluation during return to play is reasonable.
3: No benefit	B-NR	3. In athletes with asymptomatic sinus node slowing or first-degree heart block/second-degree Mobitz type I AV block (Wenckebach) at rest, further evaluation is not recommended because these are expected adaptations to training.[686–688]

Synopsis

More profound bradycardias should first be evaluated with response to light exercise, and, if they persist, with ambulatory monitoring and/or exercise stress testing. If they remain persistent, further testing to exclude the possibility of cardiomyopathy may be indicated. Mild distal abnormalities including isolated right bundle branch block or left anterior fascicular block can be seen in athletes, but more significant distal disease, or heart block at any level, requires further evaluation. Findings should be correlated with history to ensure that the patient is asymptomatic and that there is no family history of cardiomyopathy or conduction system disease. Cardioneuroablation, first described as a means of targeting ganglionated plexi for vasovagal syncope, has gained recent interest as a treatment for hypervagotonic sinus node dysfunction.[689,690] However, much more study is needed to understand this procedure's role in the treatment of symptomatic bradycardia.

Recommendation-specific supportive text

1. Mild distal abnormalities including left anterior fascicular block and right bundle branch block in isolation (eg, no symptoms or underlying structural heart disease) can be seen in athletes.[6,691] More significant distal abnormalities including left bundle branch block (present in < 1:1000 athletes), bifascicular block, or Mobitz II second-degree AV block or complete heart block often reflect underlying myocardial and/or electrical pathology.[684,685] These include cardiomyopathies, infiltrative disease such as sarcoidosis, amyloidosis, or Chagas disease, or channelopathies including LQTS with functional 2:1 AV block. Congenital complete heart block can be due to genetic heart disease including genes coding for transcription factors such as TBX, NKX2.5, and GATA4,[692,693] and family history should include presence of AV block or pacemakers in other family members. Evaluation includes imaging, cardiac monitoring, and exercise stress testing.
2. Profound sinus bradycardia (heart rates < 30 bpm while awake) or AV nodal slowing with PR interval > 400 ms that does not correct with light exercise may require evaluation with ambulatory monitoring and/or stress test. If it remains persistent, the abnormality can reflect underlying pathology including myocardial or electrical disease (including Lyme), and evaluation with imaging and cardiac monitoring are reasonable. Clinical sick sinus syndrome has been reported as more common in adult current and prior competitive endurance skiiers[527] and cyclists,[694] in dose-dependent fashion based on numbers of races and finishing times.
3. Bradycardias are an expected adaptation to intense exercise, due to changes in sympathovagal balance[682,683] and to modulation of ion channels in the sinus node.[682,683] Thus, sinus bradycardia, nonsinus atrial rhythms, junctional isorhythmic or escape rhythms, first-degree AV block, and second-degree AV block type 1 (Wenckebach) are all expected findings in a trained athlete. These findings are most evident during sleep and can also be seen at rest.

10.2 Athletes with a pacemaker

Recommendations for athletes with a pacemaker

COR	LOE	Recommendations
1	C-LD	1. In athletes with a pacemaker and without exercise-limiting underlying conditions, return to play is recommended.[192,193,207,393]
2b	C-EO	2. For athletes who are completely pacemaker-dependent, collision sports may be considered after a shared decision-making discussion of the potential risks and the absence of data on safety.
1	C-LD	3. For athletes undergoing permanent pacemaker implantation, consideration for cardiac physiological pacing to reduce symptoms from right ventricular pacing and the risk of pacing-induced cardiomyopathy should be based on characteristics including ejection fraction, pacing burden, and QRS morphology.[695–702]
2a	C-EO	4. For athletes undergoing pacemaker implantation who will be returning to play, a waiting period of 4-6 weeks after a new transvenous implant, or 2 weeks after leadless implant or generator replacement, is reasonable.
1	C-LD	5. For athletes with a pacemaker and AV block, programming rate-adaptive AV delay and post-ventricular atrial refractory period shortening should be performed to prevent pacemaker Wenckebach or 2:1 conduction at high sinus rates.[703]
1	B-NR	6. For athletes with a pacemaker and sinus node dysfunction, programming should be optimized to avoid right ventricular pacing.[704,705]
1	C-EO	7. For a young or young adult athlete undergoing pacemaker implant, it should be confirmed that the device programming options allow age-appropriate heart rates.
1	C-LD	8. For athletes with sinus node dysfunction and a permanent pacemaker, an exercise stress test with the appropriate modality based on the type of sport and the environment in which symptoms are elicited should be performed for programming of rate-response and other exercise-related parameters.[706]

Synopsis

For athletes who have received a pacemaker, return to play should be based on the presence of other exercise-limiting conditions. For a pacemaker-dependent athlete who wishes to return to collision sports, data on safety are lacking. Cardiac physiologic pacing (CPP), which includes cardiac resynchronization therapy (CRT), His bundle pacing, and left bundle branch area pacing, may be particularly important for athletes and should be the chosen mode as indicated for the general population. Choice of device and programming to maximize heart rate as needed for sport performance should address rate-responsive parameters, and these should be maximized using exercise stress testing, along with selecting ventricular pacing avoidance algorithms to minimize ventricular pacing burden. Figure 18 is a summary of the recommendations for athletes with permanent pacemakers. The role for a leadless pacemaker is not yet well defined. Lead issues are obviated with this device, but neither the ability to perform adequate pacing to meet the needs of an athlete nor lifelong management for a young person with this device has been demonstrated.[707,708] For those with heart block, maintaining 1:1 AV synchrony is critical to maintain heart rate and cardiac output. Reports of currently available dual-chamber leadless pacemakers do not describe complete AV synchrony.[709,710]

Recommendation-specific supportive text

1. There are no prospective studies of athletes with permanent pacemakers. Data can be extrapolated from studies of sports participation for athletes with ICDs, which have not shown levels of lead malfunction higher than that in unselected populations.[192,193,207,393] The decision should be made in shared-decision-making fashion, taking into account underlying diagnosis and physiology.
2. While studies of athletes with ICDs have not shown higher-than-expected levels of lead malfunction, few athletes participated in collision sports. Lead malfunction rates in collision sports are not known.[192,193,207,393] Trauma-induced lead fracture and pacemaker failure are rare events and have been reported with blunt trauma to the chest, weightlifting, hyperextension injuries to the lead-bearing upper extremity, clavicular crush, clavicular fractures, and sudden deceleration, suggesting that similar sports-related injuries may result in lead and pacemaker malfunction.[711–719]
3. Although no studies have specifically assessed right ventricular pacing versus CPP in athletes who require a pacemaker, the importance of minimizing pacing to prevent pacing-induced cardiomyopathy is well established in the general pacing population.[17,695,697,698,700,701,720] A recent meta-analysis showed that His bundle pacing and biventricular pacing, compared with right ventricular pacing, were superior to right ventricular pacing in patients with normal or mildly reduced left ventricular function in terms of heart failure and all cause death.[696] Multiple small studies also showed that the various CPP approaches can preserve left ventricular function in those who need significant pacing.[17,696,699]

   The benefit of CPP (CRT, His, or left bundle branch area pacing) has so far been mainly studied in CRT candidates with ejection fraction $\leq$ 35%.[721–724] The

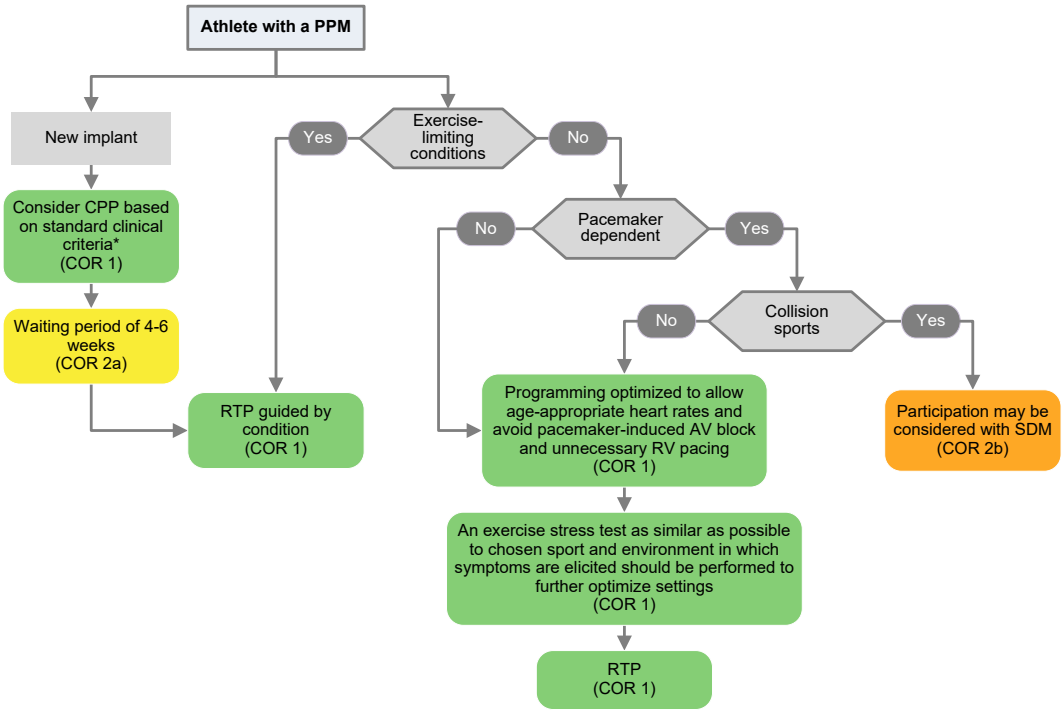

Figure 18

Algorithm for return to play in athletes with permanent pacemakers. Colors correspond to the class of recommendation (COR) in Table 1. **See 2023 HRS/APHRS/LAHRS guideline on cardiac physiologic pacing for the avoidance and mitigation of heart failure.*[17] AV = atrioventricular; CPP = cardiac physiological pacing; PPM = permanent pacemaker; RTP = return to play; RV = right ventricular; SDM = shared decision-making.

Biventricular Versus Right Ventricular Pacing in Heart Failure Patients With Atrioventricular Block (BLOCK HF) trial[724] was one of the few studies with patients with reduced left ventricular function (≤ 50%) and expected high amount of ventricular pacing. This study showed that those randomized to CRT had improved outcomes. More recently, left bundle branch area pacing has been shown to reduce QRS duration and improve ventricular function, which might preserve ventricular function or improve those with mild dysfunction.[702]

While there are no athlete-specific data, maintaining cardiac output is likely to be of importance to maximize exercise performance. In the absence of data specific to athletes, the management of athletes requiring pacing, including those who are expected to have < 40% pacing burden, should adhere to the latest pacing guidelines as appropriate.[17]

4. Avoidance of reaching or lifting for 4-6 weeks after implantation of transvenous leads has been traditionally recommended to avoid lead dislodgment before lead endothelization is complete. Several small studies of resistive range-of-motion exercise[211] and early removal of arm restriction[212,213] have not shown arm/shoulder movement to increase dislodgment or other complications, and an ongoing randomized trial of lenient versus strict arm restriction after device implant is ongoing.[214] However, at this time, as randomized data are not available, and the published small studies did not investigate vigorous exercise/arm motion in athletes, continuation of the traditional advisory is recommended. Two weeks should be allowed for healing of the skin incision.
5. At sinus rates beyond the maximum tracking rate, standard upper rate behavior leads to pacemaker Wenckebach and 2:1 conduction, which decreases maximal cardiorespiratory performance in the young, when it occurs.[703] Rate-adaptive AV delay and post-ventricular atrial refractory period shortening prevent this undesirable upper rate behavior.[725] These issues can be corrected in athletes by the programming of these parameters guided by exercise stress testing.[726]
6. Deleterious effects of right ventricular pacing include adverse remodeling, increased heart failure, and AF.[727,728] Programming of algorithms designed to decrease the frequency of right ventricular pacing have been shown to decrease incidence of heart failure and AF.[704,705] Particularly if physiological pacing is not employed, these algorithms should be utilized.[729] Data on these programming options are not available in athletes. However, it is likely that the findings would be similar.
7. Pacemakers vary in available programmable options. For an athlete to achieve maximal performance, maximizing cardiac output requires that heart rate needs to reach age-predicted maximum, with appropriate AV synchrony. To achieve this, for the young/young adult athlete with sinus node dysfunction, the maximum sensor rate programable must exceed age-predicted maximum heart rate. For the athlete with AV block, refractory periods and rate-adaptive AV delay must allow 1:1 conduction at age-predicted maximum heart rate.
8. Chronotropic incompetence may be the result of natural aging, medications for treatment of tachyarrhythmias or other heart disease, or congenital anomalies or surgery to repair these defects. All current pacemakers include sensors to detect a signal indicating the need for a faster heart rate to meet metabolic demand. Sensors can be based on parameters resulting from exercise (tertiary sensors) such as accelerometers, the least physiological type. Secondary sensors detect metabolic demand, such as minute ventilation. Primary sensors detect parameters underlying cardiac function with exercise, such as closed-loop stimulation. Sensed stimuli can be mechanical (accelerometer or piezoelectric) or intrathoracic impedance-based (for minute ventilation). Other sensors in the lead itself measure intracardiac impedance reflecting contractility. Advantages and disadvantages of these systems have been outlined,[730,731] but few data have addressed the relative efficacy in the general population and there are no data in athletes. For athletes, there are hypothesized pros and cons of each type of sensor. Accelerometers underestimate the degree of activity in many activities, particularly those in which the shoulder has little movement such as biking. Minute ventilation is proportional to effort, but onset is delayed. Blended sensors may be better in the general population.[732] Closed-loop systems have theoretical advantages but have not been tested in chronotropic incompetence or in athletes.[733] Given the absence of data in athletes, it is not currently possible to make athlete-specific recommendations regarding choice of sensor. However, regardless of sensor choice, programming should be individualized. While data in athletes are not available, in the general population, one randomized study has shown that use of exercise stress testing to program rate-response settings individually improved maximal heart and exercise capacity.[706] Patients with a pacemaker who report decreased endurance, exercise stress testing, and particularly CPET may help clarify both the components of those symptoms and their overall aerobic capacity. Principles for exercise stress testing in athletes appears in detail in Section 2.

Section 11 Gaps and future directions

The care of an athlete with arrhythmia integrates multiple aspects of cardiology and sports medicine and should incorporate relevant considerations regarding impacts of type and sport and intensity of exercise. The writing committee in this first Arrhythmias in the Athlete document has made recommendations based on data in the athlete when available, data in nonathletes where relevant, and expert consensus, unanimous or nearly for all recommendations, in areas where

guidance is clearly needed but data are lacking. Identified gaps and important avenues for future research to further inform shared decision-making around management of arrhythmias in the athlete are listed in Table 19.

Table 19 Identified gaps and needs for future studies

Knowledge gap	Future needs and directions
Impact of race and ethnicity on arrhythmias in the athlete	Recognizing populations at risk, but also recognizing the dangers of categorizing findings based on race (such as the "Black athlete ECG"), is critical to providing care to all athletes equitably.
Women athletes	The majority of studies of athletes with arrhythmias have analyzed primarily male athletes. Improved inclusion and access for women in studies is needed.
Role of performance-enhancing drugs	More data are needed on the role of performance-enhancing drugs in exacerbating arrhythmias.
Role of wearables and artificial intelligence	The roles of wearables and artificial intelligence in medicine are growing exponentially, but their roles in monitoring different aspects of health and screening for future cardiovascular disease need further study.
Post-procedure management	Data are lacking on optimal waiting periods to return to play after procedures, to allow incisional healing and avoid lead dislodgment.
SCA prevention	Understanding the epidemiology of and how to best screen athletes for underlying cardiac disease that may predispose to SCA are areas of ongoing research.
SCA treatment: EAP	Continued advocacy work for EAP and access to AEDs is needed, recognizing that EAPs may be defined differently depending on local resources and socioeconomics.
SCA treatment: Home/personal AEDs	There are many cardiac conditions in which the risk of SCA is not high enough to warrant implantation of an ICD, but the risk is not zero. While obtaining a home AED is recommended, data are needed on the medical and psychological outcomes of this intervention for children living at home and young adults on their own.
SCA treatment: ICDs	Data in moderate-sized studies have demonstrated low risk in patients participating in sports with transvenous ICDs performing most sports. Safety of sports, particularly related to system malfunction, for other modalities such as subcutaneous, extravascular, and epicardial/abdominal ICDs is not yet defined. Data are lacking on risk of collision sports to the lead, in any system.
Syncope workup	While cardiac causes of syncope are rare, they can be life-threatening. Defining the appropriate workup that eliminates nonuseful testing without losing sensitivity for-life-threatening entities is an important avenue of future research.
AF anticoagulation overall strategy	Some data suggest that athletes with AF may have lower stroke risk than nonathletes with similar CHA_2DS_2-VASc scores. However, data are not currently sufficient to support different anticoagulation strategies in athletes. Data on the impact of anticoagulation on rates of serious bleeding in the setting of injury in sports with low but potential risk of injury, such as bicycling and skiing, are also lacking.
AF safety of brief cessation of anticoagulation for activities with high risk of trauma	Data suggest thromboembolic risk in anticoagulation cessation in other circumstances, such as perioperative, is low but not zero. Safety of brief cessation in generally healthy athletes has not been investigated
AF rhythm management: Medications	While anecdotally and theoretically antiarrhythmic medications could increase proarrhythmia in athletes, the safety and tolerability of antiarrhythmic medications in athletes have not been described.
AF rhythm management: Ablation	Whether athletes would benefit from ablation strategies beyond PVI is unknown. Anecdotal reports suggest that overablation could be harmful by causing pulmonary vein stenosis or stiff left atrial syndrome and/or by affecting pulmonary vein function.
AF rhythm management: The ideal time to refrain from vigorous exercise after ablation	While inflammation at the time of AF ablation creates an immediate increased risk of recurrence during the 2-3 months post-ablation, whether vigorous exercise would alter the immediate or long-term risk of recurrence is not known.
Ventricular arrhythmia management	While documentation of arrhythmia suppression after pharmacological or ablative therapy on a stress test prior to return to play seems clinically relevant, data are needed to evaluate the predictive value of stress testing in this context.
HCM	Data are emerging that show arrhythmic risk is lower than hypothesized for patients with HCM. Data are lacking on how vigorous exercise may impact long-term progression of the underlying myopathy, with theoretical considerations supporting both beneficial and deleterious effects.
ACM	For many cardiomyopathies, data are few at best, or completely lacking on both the arrhythmic risk of exercise and the impact of exercise on penetrance and progression of disease. Even for those entities in which excessive exercise has been shown to be detrimental, safe thresholds have not been defined.
Channelopathies	Whether there is a role for extended monitoring (such as implantable loop recorders or wearable devices) has not been defined.
Rhythm monitoring for inherited cardiomyopathies and channelopathies	Whether there is a role for extended monitoring (such as implantable loop recorders or wearable devices) has not been defined.
Channelopathies: Left cardiac sympathetic denervation	While complication rate is not high and quality of life is not adversely impacted by left cardiac sympathetic denervation,[391,397] whether sympathetic denervation impacts athletic performance has not been reported.
PVCs	While prior data show that frequent PVCs in the absence of underlying heart disease do not carry risk with sports, studies restricted athletes with highest burden from return to play. More data are needed on return to play with very frequent PVCs.

(*continued*)

Table 19 Continued

Knowledge gap	Future needs and directions
Myocarditis	Data are lacking on how myocarditis may impact future arrhythmia risk. Outcomes in athletes who return to play with residual scar on CMR, or residual PVCs or premature atrial contractions, is needed.
WPW risk assessment for young adults	As described in detail above, data have emerged that show that for those with WPW who are under 18 years of age, noninvasive and even invasive risk stratification lack sensitivity and thus ablation is recommended. For those above age 30 years, risk is low and can likely be estimated noninvasively. However, data are few on individuals in the young adult range, 18-25 years old, a group of importance since WPW may be identified during preparticipation screening as entering college.
Education/training	Formal education and training in the care of arrhythmias in athletes needs to be regularly available at national scientific meetings, in board review content, and during fellowship training, if possible.
Pacemakers	Role of leadless pacing, both single and dual chamber, has not yet been studied, especially with regard to form factor, programming, and battery life.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2024.05.018.

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Appendix 1 Writing committee member disclosure of relationships with industry and other entities

Writing committee member	Employment	Honoraria/ speaking/ consulting	Speakers' bureau	Research*	Fellowship support*	Ownership/ partnership/ principal/majority stockholder	Stock or stock options	Intellectual property/ royalties	Other
Rachel Lampert, MD, FHRS (Chair)	Yale University School of Medicine, New Haven, Connecticut	1: Medtronic†	None	0: Boston Scientific 0: MediLynx 0: Medtronic	None	None	None	None	None
Eugene H. Chung, MD, MPH, MSci, FHRS (Vice-Chair)	Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts	None	None	None	None	None	None	None	None
Michael J. Ackerman, MD, PhD	Mayo Clinic, Rochester, Minnesota	0: Abbott 3: ARMGO Pharma 1: BioMarin 0: Boston Scientific 2: Bristol-Myers Squibb 0: Daiichi Sankyo 1: Illumina 0: InVitae 0: Medtronic 2: Tenaya Therapeutics 1: UpToDate	None	None	None	0: AliveCor 0: Anumana	None	5: Pfizer 0: Thryv Therapeutics	None
Alonso Rafael Arroyo, MD	Fundación Cardiotorácica-Clínica Centro, Barranquillas, Colombia	None	None	None	None	None	None	None	None
Douglas Darden, MD	Kansas City Heart Rhythm Institute, Kansas City, Kansas	None	None	None	None	None	None	None	None

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Rajat Deo, MD	University of Pennsylvania, Philadelphia, Pennsylvania	0: Boehringer Ingelheim 1: Medtronic	None	0: iRhythm Technologies	None	None	None	None	None
Joe Dolan		None	None	None	None	None	None	None	None
Susan P. Etheridge, MD, FAHA, FHRS, FACC, CEP-S	University of Utah, Salt Lake City, Utah	1: UpToDate	None	None	None	None	None	None	0: Sudden Arrhythmia Death Foundation (Vice President, Board)
Belinda R. Gray, MBBS, PhD, FHRS, CCDS	University of Sydney, Camperdown, New South Wales, Australia	1: Bristol-Myers Squibb	None	None	5: Heart Foundation Future Leader Fellowship	None	None	None	None
Kimberly G. Harmon, MD	University of Washington Medicine, Seattle, Washington	None	None	5: AMSSM 5: Football Research (NFL)	None	None	3: 98point6	None	None
Cynthia A. James, PhD, CGC	Johns Hopkins University, Baltimore, Maryland	0: LEXEO Therapeutics 0: Pfizer 0: StrideBio	None	0: Boston Scientific 3: Lexeo 3: StrideBio	None	None	None	None	0: NSGC (Board Member)
Jonathan H. Kim, MD, MSc, FACC	Emory Healthcare, Atlanta, Georgia	None	None	0: Atlanta Track Club Foundation 0: NIH/NHLBI	None	None	None	None	1: Atlanta Falcons-NFL (Salary from employment)
Andrew D. Krahn, MD, FHRS	University of British Columbia, Vancouver, British Columbia, Canada	0: Medtronic	None	None	None	None	None	None	None
Andre La Gerche, MBBS, PhD	Baker Heart & Diabetes Institute, Melbourne, Victoria, Australia	None	None	None	None	None	None	None	None
Mark S. Link, MD, FHRS	UT Southwestern Medical Center, Dallas, Texas	None	None	None	None	None	None	None	None

(*continued*)

Appendix 1 Continued

Writing committee member	Employment	Honoraria/ speaking/ consulting	Speakers' bureau	Research*	Fellowship support*	Ownership/ partnership/ principal/majority stockholder	Stock or stock options	Intellectual property/ royalties	Other
Ciorsti MacIntyre, MD	Mayo Clinic, Rochester, Minnesota	0: Abbott Medical 0: Medtronic	None	None	None	None	None	None	None
Lluis Mont, MD, PhD, FEHRA	Hospital Clínic, Universitat de Barcelona., Barcelona, Spain	1: Abbott Medical 1: Biosense Webster 1: Boston Scientific 1: Medtronic	None	2: Abbott Medical 2: Biosense Webster 2: Boston Scientific 1: Medtronic	1: Abbott 1: Biosense Webster 1: Boston Scientific 1: Medtronic	None	1: ADAS 3D 1: Corify Health Care S.L.	None	None
Jack C. Salerno, MD, FHRS	University of Washington School of Medicine, Seattle, Washington	2: Philips	None	None	None	None	None	None	None
Maully J. Shah, MBBS, FHRS, CCDS, CEPS-P	Childrens Hospital of Philadelphia, Philadelphia, Pennsylvania	0: IBHRE 1: Medtronic 0: Tenaya Therapeutics	None	0: Medtronic	None	None	None	None	1: JACC

Number value: **0** = $0; **1** = ≤ $10,000; **2** = > $10,000 to ≤ $25,000; **3** = > $25,000 to ≤ $50,000; **4** = > $50,000 to ≤ $100,000; **5** = > $100,000.

This table is a comprehensive list of the relationships with industry and other entities (RWI)—regardless of relevance to the document topic—disclosed by each writing committee member for the 12 months prior to the initial meeting of the writing committee and up through the completion of the document. The table does not necessarily reflect the RWI of the writing committee members at the time of publication. Please refer to the HRS Code of Ethics and Professionalism for definitions of disclosure categories or additional information about the HRS policy on the disclosure of relationships with industry and other entities. To mitigate potential bias and conflict of interest, the recommendations and supportive text were written by writing committee members who were free of relevant RWI.

AMSSM = American Medical Society for Sports Medicine; IBHRE = International Board of Heart Rhythm Examiners; JACC = Journal of the American College or Cardiology; NIH = ■■■; NHLBI = ■■■; NSGC = National Society of Genetic Counselors.

*Research and fellowship support are classed as programmatic support. Sources of programmatic support are disclosed but are not regarded as a relevant relationship with industry for writing group members or reviewers.

†This RWI was ended in March 2022, 8 months prior to writing committee selection and 10 months prior to the start of document development.

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Appendix 2 Reviewer disclosure of relationships with industry and other entities

Peer Reviewer	Employment	Honoraria/ speaking/ consulting	Speakers' bureau	Research*	Fellowship support*	Ownership/ partnership/ principal/ majority stockholder	Stock or stock options	Intellectual property/ royalties	Other
Sana M. Al-Khatib, MD, MHS, FHRS	Duke University School of Medicine, Durham, North Carolina	None	None	1: Boston Scientific 1: Medtronic	None	None	None	None	3: AHA (Senior Associate Editor for Circulation)
Mark E. Alexander, MD, FHRS, CEPS-P	Boston Children's Hospital, Boston, Massachusetts	1: Best Doctors	None	None	None	None	None	1: Wolters Kluwer	None
Irfan Asif, MD	University of Alabama at Birmingham, Birmingham, Alabama	None	None	5: HRSA	None	None	None	None	0: ADFM (Board Member) 0: AMSSM (CRN Leadership Chair)
Hein Heidbuchel, MD, PhD	University of Leuven, Leuven, Belgium	0: Abbott 0: Bayer Healthcare Pharmaceuticals 0: Biotronik 0: BMS/Pfizer Alliance 0: Boehringer Ingelheim 0: Medscape 0: Springer Healthcare Ltd 1: Daiichi Sankyo	None	0: Abbott 0: Bayer Healthcare Pharmaceuticals 0: Biotronik 0: BMS/Pfizer Alliance 0: Boehringer Ingelheim 0: Boston Scientific 0: Daiichi Sankyo 0: Medtronic	None	None	None	None	None
Tee Joo Yeo, MBBS	National University Heart Centre Singapore, Singapore	None	None	None	None	None	None	None	None
Miguel A. Leal, MD, FHRS	Emory School of Medicine, Atlanta, Georgia	1: Sanofi	None	1: Medtronic	None	None	None	None	None
Matthew W. Martinez, MD	Morristown Medical Center, Morristown, New Jersey	1: Bristol Myers Squibb Foundation Diverse Clinical Investigator Career Development 1: Cytokinetics	None	None	None	None	None	None	2: MLS (Independent Contractor) 2: NBA Players Association (Independent Contractor)

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Appendix 2 Continued

Peer Reviewer	Employment	Honoraria/ speaking/ consulting	Speakers' bureau	Research*	Fellowship support*	Ownership/ partnership/ principal/ majority stockholder	Stock or stock options	Intellectual property/ royalties	Other
Kristen K. Patton, MD, FHRS	University of Washington, Seattle, Washington	1: Great Wall International Congress of Cardiology	None	None	None	None	None	None	0: ACGME RC Internal Medicine 0: AHA Clinical Cardiology Council 0: JAMA Cardiology (Associate Editor) 0: U.S. Food & Drug Administration
Jordan M. Prutkin, MD, MHS, FHRS	University of Washington, Seattle, Washington	None	None	None	None	None	None	4: UpToDate	None
Elizabeth V. Saarel, MD, FHRS, CEPS-P	St. Luke's Health System, Meridian, Idaho	None	None	None	None	None	None	None	None
Richard Soto-Becerra, MD	Instituto Nacional Cardiovascular (INCOR), Lima, Peru	None	None	None	None	None	None	None	None

Number value: **0** = $0; **1** = ≤ $10,000; **2** = > $10,000 to ≤ $25,000; **3** = > $25,000 to ≤ $50,000; **4** = > $50,000 to ≤ $100,000; **5** = > $100,000.

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ACC = American College of Cardiology; ACGME = Accreditation Council for Graduate Medical Education; ADFM = Association of Departments of Family Medicine; AHA = American Heart Association; AMSSM = American Medical Society for Sports Medicine; HRCRS = Heart Rhythm Clinical Research Solutions; HRSA = Health Resources and Services Administration; JAMA = Journal of the American Medical Association; MLS = Major League Soccer; NBA = National Basketball Association; NCRD = National Cardiovascular Data Registry; NIH = National Institutes of Health.

*Research and fellowship support are classed as programmatic support. Sources of programmatic support are disclosed but are not regarded as a relevant relationship with industry for writing group members or reviewers.

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