

SCIENTIFIC STATEMENT

Clinical Considerations for Competitive Sports Participation for Athletes With Cardiovascular Abnormalities

A Scientific Statement From the American Heart Association and American College of Cardiology

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ABSTRACT

This American Heart Association/American College of Cardiology scientific statement on clinical considerations for competitive sports participation for athletes with cardiovascular abnormalities or diseases is organized into 11 distinct sections focused on sports-specific topics or disease processes that are relevant when considering the potential risks of adverse cardiovascular events, including sudden cardiac arrest, during competitive sports participation. Task forces comprising international experts in sports cardiology and the respective topics covered were assigned to each section and prepared specific clinical considerations tables for practitioners to reference. Comprehensive literature review and an emphasis on shared decision-making were integral in the writing of all clinical considerations presented.

The purpose of this scientific statement is to provide updated clinical considerations for competitive sports participation for athletes with cardiovascular abnormalities. Prevention of sudden cardiac death (SCD) and worsening cardiac disease, historically addressed by universal competitive sports participation restriction among athletes with cardiovascular abnormalities, remains a priority. However, sports cardiology has evolved in complexity. Thus, the contemporary clinical approach to competitive athletes with cardiovascular disease (CVD) has moved away from paternalistic decision-making toward a strategy characterized by nuanced deliberation that provides options to practitioners and athletes.^{1,2} Shared decision-making (SDM) with patients is now a fundamental principle in clinical medicine and foundational in this scientific statement. The writing committee of this document sought to provide updated clinical considerations based on pragmatism and a careful review of the evidence to be a useful resource for practitioners who care and advocate for competitive athletes.

This is the fifth United States-based expert consensus article addressing this topic. The Bethesda Conference (American College of Cardiology) proceedings were

published in 1985, 1994, and 2005, and the first scientific statement sponsored by the American Heart Association and American College of Cardiology was published in 2015.³⁻⁶ This update integrates 40 years of scientific progress and advances past antiquated concepts. There have been improvements in our understanding of the “athlete’s heart,” the term used to capture the complex structural, functional, and electrical cardiac adaptations in response to habitual exercise training, which enables more accurate differentiation of athletic cardiac adaptations from pathology.⁷ Emerging outcomes data are now available for several cardiac conditions that suggest risk is not as high during competitive sports participation as previously assumed.⁸⁻¹¹ Athletes also present unique clinical challenges because of their habitual exposure to high levels of exercise intensity. There remains a critical emphasis on sport-specific effort exercise stress testing customized to provoke symptoms, rather than pharmacologic stress testing or exercise testing to arbitrary heart rate thresholds, in the clinical evaluation of the competitive athlete.¹² The contemporary paradigm of “sports eligibility” has shifted in philosophical approach from conservative medical paternalism to one that embraces clinical uncertainty and SDM.¹³ For the first time, we

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emphasize that this is not an article outlining “disqualification recommendations,” but rather a compendium of clinical considerations that should guide the SDM process for athletes who present with cardiovascular abnormalities or disease.

DEFINITIONS AND TARGETED AUDIENCE

The target population for this scientific statement is competitive athletes, defined as in previous articles as “individuals who participate in organized team or individual sports that require regular competition against others, place a high premium on achievement, and require some form of systematic and intense training.”⁶ The term “competitive sports” is intended to include all aspects of athletic training and competition that are required of competitive athletes. Similar to past articles, competitive athletes include both pediatric and adult-aged individuals, but in contrast to past articles, we diverge from the previous arbitrary generality that this scientific statement is only intended for athletes between 12 and 25 years of age.^{6,14} Some children begin competitive sports participation before 12 years of age, and professional athletes are often older than 25 years. The clinical considerations in this scientific statement apply to prepubertal athletes (<12 years of age), adolescent athletes (middle and high school; between 12 and 17 years of age), young adult athletes (college and professional; between 18 and 25 years of age), adult athletes (professional or other elite-level athletes between 25 and 35 years of age), and masters athletes (Section X; defined as ≥ 35 years of age). Sports cardiology bridges pediatric and adult cardiology, and continued engagement of pediatric cardiologists is critical as the field continues to grow. Six pediatric cardiologists participated in the task forces involved in compiling this scientific statement (see Format). The clinical considerations in this scientific statement may also be applied to athletes participating in high-level recreational sports (eg, regular marathon runners, triathletes, cyclists, CrossFit enthusiasts) who fulfill the general description of the competitive athlete. Whereas this scientific statement is not directly intended for other highly active populations, such as tactical athletes,¹⁵ this scientific statement may be a resource in the care of these individuals.

This scientific statement is intended to be a clinical reference for use by general cardiologists, sports cardiologists, interventional cardiologists and electro-

physiologists, pediatric cardiologists, internists, general pediatricians, family medicine practitioners, advanced practice professionals (eg, nurse practitioners, physician assistants), school nurses, primary care sports medicine practitioners (including team physicians), and athletic trainers. In addition, nonclinical professionals who play a role in the oversight of competitive athletes, including coaches, school or organizational administrators, and policymakers, may benefit from an understanding of the clinical considerations presented in this scientific statement. While recognizing the importance of numerous stakeholders in the care of the competitive athlete, it is anticipated that the large majority of competitive athletes with CVD will ultimately be best served by primary clinical oversight from a sports cardiologist. Although there is no uniform consensus for who qualifies as a sports cardiologist,¹⁶ this individual should be trained in cardiology and routinely see competitive athletes and highly active individuals.

FORMAT

This scientific statement is separated into 11 sections (see Structure), with changes in organization compared with 2015. Each section was written by a task force comprising international experts in sports cardiology and the respective disease or topic and chaired by 1 of the primary authors (9 American, 1 European, 1 Australian). A total of 49 international experts were invited to participate in the task forces (36 American, 11 European, 2 Australian), and all contributed to the completion of this scientific statement. Section I updates the classifications of sports and Section II updates strategies for preparticipation cardiac screening and the critical element of emergency action planning (EAP). New to this scientific statement is Section III, which details the ethics of sports eligibility and importance of SDM. Sections IV through XI update participation considerations among athletes with specific probable or confirmed CVDs. Changes in organization include myocarditis and pericarditis considered separately (Section V), and new sections specific to masters athletes (Section X) and populations and sports not previously addressed (Section XI). Important diseases and clinical entities (eg, hypertension, commotio cordis) remain in Section XI, but not as an independent section. Each disease-specific section includes a primary clinical considerations table for practitioners to reference with ease and efficiency. The terms included in the tables

TABLE 1

Specific Terminology Used in the Clinical Considerations Tables With the Intended Meaning or Interpretation in the Context of Competitive Sports Participation

Clinical consideration action item	Intended meaning
Should	Clinicians should proceed with practices that are accepted standards of medical care.
Should not	Clinicians should avoid practices that are contrary to standards of medical care.
Can	Available evidence or expert consensus opinion, or both, suggest minimal cardiac risk associated with unrestricted athletic training and competition; sport participation can proceed without the need for SDM.
Reasonable	Based on substantive available evidence and expert consensus opinion, cardiac risks during unrestricted athletic training and competition can be considered low and nonprohibitive; proceed with SDM.
Reasonable to consider	Expert consensus opinion and limited available evidence suggest that cardiac risks during unrestricted athletic training and competition are probably low and nonprohibitive; proceed with SDM.
Can consider	No or limited evidence is available. Expert consensus opinion considers cardiac risks during unrestricted athletic training and competition may be low and nonprohibitive; proceed with SDM.
Risks may outweigh benefits	SDM should integrate available evidence or expert consensus opinion, or both, that indicate at least moderately elevated cardiac risks during unrestricted athletic training and competition.
Risks likely outweigh benefits	SDM should integrate available evidence or expert consensus opinion, or both, that indicate markedly elevated cardiac risks during unrestricted athletic training and competition.

SDM indicates shared decision-making.

and the intended meanings are provided for additional reference in [Table 1](#). Key highlights are summarized in [Table 2](#).

STRUCTURE

The sections in this scientific statement include Sports Classifications ([Section I](#)); The Preparticipation Cardiac Evaluation ([Section II](#)); Ethical Aspects of Competitive Sports Eligibility ([Section III](#)); Cardiomyopathies ([Section IV](#)); Myocarditis/Pericarditis, Valvular Heart Disease, and Other Acquired Cardiovascular Conditions ([Section V](#)); Congenital Heart Disease ([Section VI](#)); Aortopathy (Including Bicuspid Aortic Valve) and Spontaneous Coronary Artery Dissection ([Section VII](#)); Arrhythmias, Devices, and ECG Abnormalities ([Section VIII](#)); Cardiac Channelopathies ([Section IX](#)); Masters Athletes ([Section X](#)); and Additional Cardiac Conditions and Considerations ([Section XI](#)). Each section provides a brief summary detailing the rationale for key clinical

considerations and a respective clinical considerations table.

RATIONALE FOR UPDATES

Ethical Imperative for SDM

[Section III](#) is devoted to the ethical aspects of sports eligibility. This addition is predicated on the ethical imperative for patient-centered care using SDM,¹⁷ which is widely advocated as the accepted model for clinical management discussions between physicians and patients. Historical dogma that athletes lack the ability to make rational and informed decisions and should not have their own values included in the process of determining sports eligibility is neither ethical nor supported by the medical literature.^{10,18} Respect for the athlete's values and preferences is essential in providing guidance about competitive sports participation after a diagnosis of CVD.¹³

New Research and Consensus Recommendations

The past decade of sports cardiology research has led to a number of findings that underscore the need for updates.^{19–26} Single-center outcomes data demonstrating the safety of competitive sports participation while under expert clinical guidance have been published for athletes with the cardiac channelopathies long QT syndrome (LQTS) or catecholaminergic polymorphic ventricular tachycardia (CPVT; [Section IX](#)).^{11,27} In a recent multicenter analysis, elite athletes with common genetic heart conditions associated with SCD (primarily hypertrophic cardiomyopathy [HCM] and LQTS) who participated in SDM continued competitive sports participation with a low incidence of breakthrough cardiac events and no deaths.¹⁰ It is increasingly evident that athletes with implantable cardioverter defibrillators (ICDs) can safely resume competitive sports participation.^{28,29} In the LIVE-HCM study (Lifestyle and Exercise in Hypertrophic Cardiomyopathy), individuals with HCM who participated in vigorous exercise, including a subgroup of competitive athletes, did not have increased adverse cardiac events compared with less active individuals with HCM.⁸ These critical data challenge the rationale for previous universal sport restriction for athletes with HCM, leading to the updated clinical considerations presented in this scientific statement ([Section IV](#)).

Improved rigor in epidemiologic research has led to changes in the understanding of the differential diagnosis of sudden cardiac arrest (SCA) and SCD in competitive athletes. Studies suggest that autopsy-negative sudden unexplained death, rather than HCM or other genetic cardiomyopathies, is the most common cause of SCD in

TABLE 2 Highlights in the 2024 American Heart Association/American College of Cardiology Scientific Statement on Competitive Sports Participation in Athletes With Cardiovascular Abnormalities

Section and task force	Highlights and key updates
Section I (task force 1): Sports Classifications	<ul style="list-style-type: none"> ■ Updated sports classification schema with removal of discrete categories of sport ■ Sports classification presented as a continuum of endurance and strength training loads ■ Sports classification also presented as levels of bodily collision and impact relevant for competitive athletes on oral anticoagulation
Section II (task force 2): The Preparticipation Cardiac Evaluation	<ul style="list-style-type: none"> ■ Acknowledgment of the limitations of both history & physical and 12-lead screening ECG ■ 12-lead ECG screening is reasonable as long as equitable access to expertise and a downstream process with appropriate resources are available
Section III (task force 3): Ethical Aspects of Competitive Sports Eligibility	<ul style="list-style-type: none"> ■ SDM is an ethical imperative to include in the clinical management of competitive athletes, including decisions for competitive sports participation ■ SDM is the foundation of these updated clinical considerations
Section IV (task force 4): Cardiomyopathies	<ul style="list-style-type: none"> ■ A uniform mandate of sports restrictions for athletes with all types of genetic cardiomyopathies should not be applied ■ Competitive sports participation may be reasonable to consider in competitive athletes with genetic cardiomyopathies
Section V (task force 5): Myocarditis/Pericarditis, Valvular Heart Disease, and Other Acquired Cardiovascular Conditions	<ul style="list-style-type: none"> ■ Resumption of competitive sports participation can be considered before 3 mo of exercise restrictions in select cases of clinical myocarditis ■ Clinical considerations are provided for SARS-CoV-2 (including vaccination) ■ Clinical considerations are provided for competitive athletes with valvular heart disease, including mitral valve prolapse
Section VI (task force 6): Congenital Heart Disease	<ul style="list-style-type: none"> ■ Clinical considerations are provided in the context of physiology and anatomy rather than specific congenital diagnoses ■ Clinical considerations are provided for coronary artery anomalies, including anomalous coronary origins and myocardial bridging
Section VII (task force 7): Aortopathy (Including Bicuspid Aortic Valve) and Spontaneous Coronary Artery Dissection	<ul style="list-style-type: none"> ■ Clinical considerations are provided for the approach to aortopathy in young competitive athletes ■ Clinical considerations for aortopathy are succinctly separated into sections on bicuspid aortic valve with aortopathy and heritable thoracic aortic disease (gene-positive and gene-negative)
Section VIII (task force 8): Arrhythmias, Devices, and ECG Abnormalities	<ul style="list-style-type: none"> ■ Clinical considerations are provided for competitive athletes who survive sudden cardiac arrest ■ Clinical considerations are provided for asymptomatic competitive athletes identified with abnormal results on screening ECG
Section IX (task force 9): Cardiac Channelopathies	<ul style="list-style-type: none"> ■ Clinical considerations are provided for the approach to competitive athletes with cardiac channelopathies ■ Competitive sports participation can be considered for competitive athletes with catecholaminergic polymorphic ventricular tachycardia who are clinically stable and under expert supervision
Section X (task force 10): Masters Athletes	<ul style="list-style-type: none"> ■ Clinical algorithm provided for masters athletes with coronary artery disease ■ Clinical considerations are provided for masters athletes with atrial fibrillation, myocardial fibrosis, dilated or aneurysmal ascending aorta, or chronic valvular heart disease
Section XI (task force 11): Additional Cardiac Conditions and Considerations	<ul style="list-style-type: none"> ■ Competitive athletes with hypertension, in the absence of hypertensive emergency, can continue with competitive sports participation ■ Clinical considerations are provided for competitive athletes diagnosed with pulmonary embolism ■ Clinical considerations are provided for competitive athletes competing at extreme altitude or recreational athletes participating in scuba ■ Clinical considerations are provided for competitive athletes without established cardiovascular disease who desire continued competitive sports participation while pregnant

SDM indicates shared decision-making.

athletes <35 years of age.^{20,30} For masters athletes, ischemic heart disease remains the most common cause.³¹

Observational data have enhanced our understanding of exercise-induced cardiac remodeling by sex, age, and sport type, reinforcing the limitations of extrapolating normative cardiac measurement values from the general population to athletes.^{26,32} Among masters athletes, observational findings of increased prevalence of coronary calcification and aortic dilation have led to

clinical uncertainty and the need for guidance in the cardiac care of this population (Section X).³³⁻³⁶

New consensus athlete recommendations affecting the care of competitive athletes have been published since the last iteration of this scientific statement. International criteria for ECG interpretation in athletes were published in 2017 (Section VIII).¹⁴ Guidelines from the European Society of Cardiology on exercise in patients with CVD were published in 2021.³⁷ Consensus recommendations from the Heart Rhythm Society for the diagnosis and

management of arrhythmias in athletes are also referenced in [Section VIII](#).³⁸

Important scientific findings brought to light during the COVID-19 pandemic are reflected in these clinical considerations.^{39,40} In particular, cardiac magnetic resonance imaging (CMR) data from athletes with SARS-CoV-2 infection suggest that resolution of myocardial inflammation may occur earlier than 3 months.⁴¹ As such, changes to the approach for myocarditis are outlined in [Section V](#).

Evolution in Clinical Sports Cardiology

Concomitant with recognition of the challenges inherent in the cardiovascular care of athletes, the growth of sports cardiology as a unique subspecialty within cardiovascular medicine has evolved and continues to accelerate internationally. However, whereas dedicated clinical centers are available to athletes in the United States and worldwide, most athletes do not have access to high-level sports cardiology expertise. Similar to other clinical arenas in adult cardiology, such as advanced heart failure and adult congenital heart disease, and in pediatric cardiology, the demand has outpaced supply. A considerable gap in quality exists for athletes with heart disease, for whom sports eligibility decisions are based solely on the 2015 scientific statement in the absence of expert-level care. Athletic populations and additional sporting disciplines not previously considered in previous scientific statements also require attention ([Section XI](#)).

Recognition of Social Disparities

The rationale for this update includes consideration of racial disparities in athlete cardiovascular health.⁴² The effects of social determinants of health and structural racism on numerous outcomes in athletes remain inadequately understood. In the preparticipation cardiac screening of athletes ([Section II](#)), it is crucial that equitable access to health care resources, as part of downstream systematic clinical processes, is available for all athletes. Because false-positive ECG findings are more frequent among self-identified Black athletes,⁴³ screening programs with appropriate downstream resources and sensitivity to issues of health equity should be provided; suboptimal screening practices have the potential to harm athletes from underrepresented racial or ethnic groups.

Sports Cardiology as Part of the Athlete Health Care Team

Sports cardiologists who fulfill the team cardiologist role in organized competitive team athletics play a vital role as part of the athlete health care team. In the team physician model, the head team physician is the final arbiter for all medical decisions for the respective team or organization. For cardiac issues pertaining to sports eligibility, the team

cardiologist is critical within this paradigm. A recent team physician consensus statement, in underscoring the primary responsibility of the head team physician to assess risk, stresses the mandate to include SDM and respect for athletes' values and preferences.⁴⁴ As such, reliance on the team cardiologist's expertise is critical to this process for cardiac issues. Whereas the team physician model applies to athletes participating at higher levels of sport, athletes competing at lower levels infrequently have a similar health care team framework in place. It is therefore necessary that athletic trainers, consulting team sports medicine physicians, and cardiology referral networks have access to an updated reference to assist and guide complex sports eligibility cases. Broad dissemination of this scientific statement will enhance consistency and quality care for all competitive athletes.

FUTURE DIRECTIONS

The clinical considerations presented in this scientific statement reflect an updated point in the continuum of our understanding of the cardiovascular risks and benefits associated with competitive sports participation in athletes with heart disease. Uncertainties remain, but important directives set the stage to advance these important and unresolved issues.

First, capture of prospective multicenter registry data, inclusive of a diverse population of athletes with CVD, who have chosen to either continue or cease competitive sports participation, is a scientific imperative. The ongoing Outcomes Registry for Cardiac Conditions in Athletes is the first long-term repository of young, competitive athletes with CVD, designed to better understand a myriad outcomes after disease diagnosis.⁴⁵ Important knowledge gaps persist regarding cardiac outcomes among masters athletes. As prospective data collection continues, studies must emphasize diversity by sex, race, and ethnicity as essential components. Inclusion of clinical trials specific for athletic populations is also needed to determine best clinical practices. For example, decisions for revascularization in stable coronary artery disease (CAD) or pulmonary vein isolation in atrial fibrillation (AF) are 2 common masters athlete clinical conundrums in which extrapolation of clinical trial data from the general population has substantial limitations.

Second, consideration of race as a sociopolitical construct must be reflected in future sports cardiology research and in clinical practice. Appreciating that disparities exist for competitive athletes mandates a commitment to capture social determinants of health in future studies. Relevant to clinical practices ([Section II](#)), contemporary ECG screening criteria lack equivalent specificity between Black and White athletes.^{43,46} There

must be a call to action on disparities if we are to pursue equity in the care of all athletes.^{42,47}

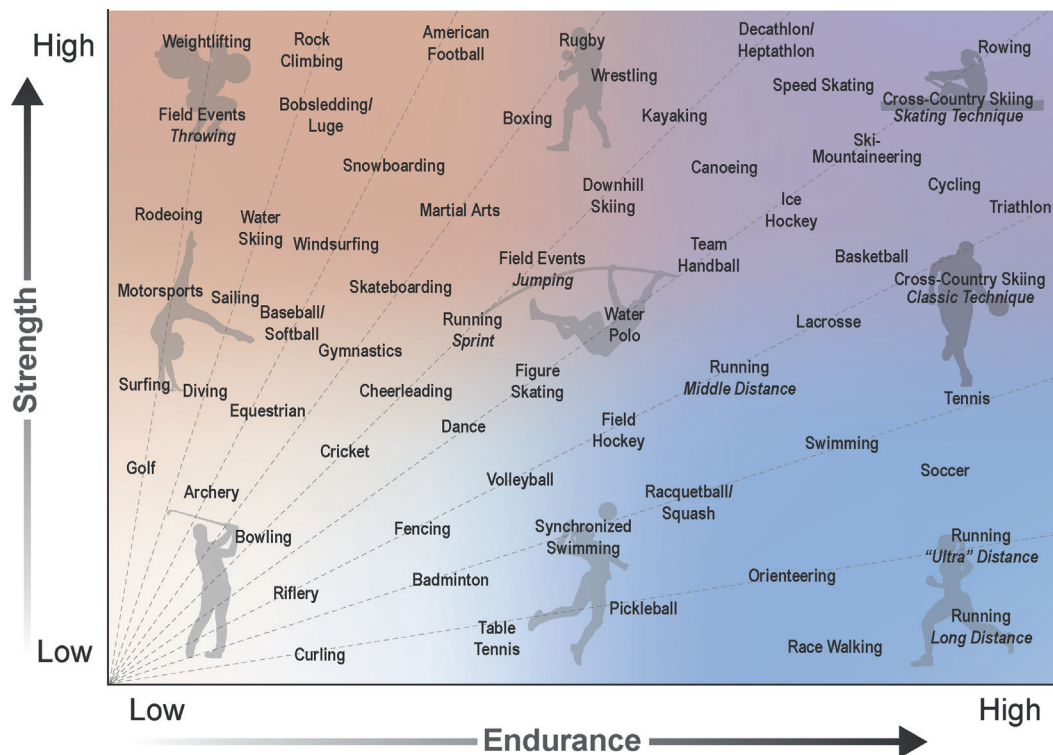
Third, SCA and SCD in athletes will never be prevented completely. In recognition of this fact, this scientific statement underscores the importance of EAP to include cardiopulmonary resuscitation education for all stakeholders in the global sporting community and the availability of automated external defibrillators (AEDs) at all sporting venues (Section II). In addition, the writing committee urges that caution must be exercised to avoid marked shifts in philosophy as a visceral response to future SCA and SCD cases. SCA and SCD among competitive athletes will continue to occur irrespective of screening and sport participation decisions, but should not be regarded as failure or ineffectiveness of an SDM approach. Prioritizing the values and preferences of athletes in a contemporary clinical paradigm achieves balance among clinical uncertainty, risk tolerance, and cardiac safety during sport, particularly in conditions

where data do not suggest prohibitive cardiac risks in the context of competitive sports participation.

SECTION I: SPORTS CLASSIFICATIONS

The classification of sports has been included in all previous versions of this scientific statement to characterize levels of effort and intensity and the hemodynamic consequences that are imparted by specific sports.^{3,48-50} The goal of this approach has been to create discrete categories of sports for physicians to consider when determining sports eligibility for athletes with CVD. In this updated version (Figure 1), sports are displayed on a continuum of endurance and strength physiologic components without the discrete bins present in previous iterations. This new approach reflects the fact that competitive sports participation exists on a spectrum of physiologic demands and mirrors the evolution of this scientific statement away from prescriptive limitation of

FIGURE 1 Updated Competitive Sports Participation Classification Schema Presented as a Continuum of Competitive Sports Participation Based on Relative Strength and Endurance Intensities



Sports are presented on arcs that signify the relative contributions of static and dynamic hemodynamic stress that accompany participation in the respective sport. These are not fixed exposures or classifications, and exercise intensities may vary on the basis of numerous factors, including playing position in certain team sports, event in certain individual sports, varying training intensities dependent on level of competition and individual athlete preference, time of season or year, and environmental stressors. Adapted from Mitchell et al^{3,48} and Levine et al⁵⁰ with permission and modified. Copyright © 1985, 1994, 2005, American College of Cardiology Foundation, and 2015, American Heart Association, Inc. and American College of Cardiology Foundation.

TABLE 3 Competitive Sport-Specific Risks and Participation Considerations Related to Bodily Collision, Bodily Impact, and Projectile Impact

Bodily collisions or impacts are not expected or occur at low velocity	Bodily collisions or impacts occur unpredictably at moderate velocity	Bodily impacts occur unpredictably but at high velocity	Bodily collisions occur routinely and at moderate to high velocity
Athletes receiving full anticoagulation, partial anticoagulation, or aspirin monotherapy can participate in these competitive sports.	<ul style="list-style-type: none"> After SDM, these competitive sports may be reasonable for athletes receiving full anticoagulation or partial anticoagulation. Athletes receiving aspirin monotherapy can participate in these competitive sports. 	<ul style="list-style-type: none"> Risks associated with these competitive sports generally outweigh benefits for athletes receiving full anticoagulation. Risks associated with these competitive sports may outweigh benefits for athletes receiving partial anticoagulation; SDM recommended. Athletes receiving aspirin monotherapy can participate in these competitive sports. 	<ul style="list-style-type: none"> Risks associated with these competitive sports generally outweigh benefits for athletes receiving full anticoagulation. Risks associated with these competitive sports may outweigh benefits for athletes receiving partial anticoagulation; SDM recommended. Athletes receiving aspirin monotherapy can participate in these competitive sports.
Archery Badminton Bowling Curling Cross-country skiing Dance Golf Orienteering Pickleball Race walking Riflery Rowing Distance running/track & field Sailing Swimming Table tennis Tennis Volleyball Weightlifting	Baseball/softball* Cricket* Basketball Fencing Racquetball/squash* Cheerleading Gymnastics Figure skating Soccer* Field hockey* Team handball* Diving Surfing Water skiing Windsurfing Track & field (pole vault)	Downhill skiing Cycling Triathlon (cycling portion) Motorsports Rodeoing Speed skating Skateboarding Snowboarding Bobsled/luge Rock climbing Kayaking/canoeing Equestrian	American football Boxing Ice hockey* Lacrosse Martial arts Rugby Wrestling

Colors correspond to low (green), moderate (yellow), or high (red) velocity. Collisions are defined as contact between ≥ 2 athletes occurring during competitive sport. Impacts are defined as contact between an athlete and a fixed or stationary object during competitive sport. Full anticoagulation is defined as a medical regimen including warfarin or direct thrombin inhibitors. Partial anticoagulation is defined as a medical regimen including dual antiplatelet therapy.

*Denotes competitive sports in which injury from high-velocity projectiles (ie, balls, pucks) may pose risk of serious bleeding independent of bodily collision or impact.

SDM indicates shared decision-making.

some athletes with CVD to lower-risk sport bins and toward an SDM approach, which requires individualized understanding of the demands of competitive sports participation. Each sport has been placed to approximate the typical exposure for a competitive athlete. These are not fixed classifications, and nuances exist in most sports. For example, the endurance and strength demands for American-style football players differ between nonlinemen and linemen,^{51,52} endurance demands differ between soccer goalies and midfielders,⁵³ and stroke styles and distances differ in swimming. Moreover, within a given sport, training loads will vary on the basis of athlete fitness, competitive level, position, and time of season, as well as environmental considerations, such as altitude and temperature. Athletes may also intensify training beyond what is intrinsic to their specific sports competitions, such as golfers who elect to weightlift.

Sports Physiology

Sports involve varying amounts of 2 main physiologic training demands, which form the basis of **Figure 1**. Endurance exercise involves repetitive contraction of

large muscle groups that generate movement of the body through space, from minutes to many hours, and obligate sustained increases in cardiac output to deliver blood flow and substrate to support metabolic demands. Bursts of dynamic contractions in a pattern similar to endurance exercise, but of high-power outputs lasting only ≈ 1 to 5 minutes (ie, “anaerobic” or middle distance exercise) fall in this category, and many sports require a combination of speeds and intensities throughout training or competition.

Strength exercise involves discrete contractions of individual muscle groups at an intensity that completely occludes blood flow and that generates high amounts of muscle tension and power output. This type of contraction leads to cyclical (and large) elevations in blood pressure that are proportionate to the total amount of muscle mass engaged,⁵⁴ and the relative percentage of a maximal contraction (the exercise pressor reflex).⁵⁵ However, the hemodynamic effects of strength exercise within the chest are uncertain given the concomitant rise in intrathoracic pressure conferred by the Valsalva maneuver, which balances out transmural wall stress on the left ventricle (LV) and aorta.⁵⁶ Brief, high-intensity repetitive contractions

that occur during short-duration (seconds) sprints are also considered a form of strength exercise.

Risks of Collisions and Impacts in Consideration of Anticoagulation Therapy

Risks of bleeding during competitive sports participation for athletes who are taking anticoagulant drugs were presented in the previous scientific statement.⁵⁰ In this update, these bleeding risks have been reconsidered with the goal to better facilitate clinical decision-making (Table 3). Trauma during competitive sports participation can be categorized as collisions, or contact between athletes (eg, American-style football tackle); impacts, or contact between an athlete and a fixed or stationary object (eg, a cyclist or skier hitting a tree); and projectile impacts, or contact between an athlete and a high-velocity projectile (eg, a baseball batter struck by a fastball). Risks of bleeding while on anticoagulants are determined both by the likelihood of trauma and the physics of the traumatic event. Risks of bleeding are likely higher if the participant is on full anticoagulation (eg, warfarin or direct thrombin inhibitor) versus dual antiplatelet therapy. Based on these considerations, Table 3 identifies sports with high and typically prohibitive levels of trauma risk for competitive athletes requiring any type of anticoagulation therapy; sports with intermediate risk of trauma during any type of anticoagulation therapy, thereby necessitating SDM in the consideration of competitive sports participation; and sports with low risk of trauma, in which athletes can participate regardless of anticoagulation therapy.

SECTION II: THE PREPARTICIPATION CARDIAC EVALUATION

The preparticipation evaluation (PPE) plays a critical role in the cardiac care of competitive athletes. Components of an appropriate PPE, beginning with the 14-point history and physical (H&P), and consideration of additional screening tests, particularly a 12-lead ECG, are the focus of this section (Table 4).⁵⁷

SCA and SCD in Competitive Athletes

SCA is the leading medical cause of sudden death during competitive sports participation.^{19,20,58,59} Among athletes <35 years of age, causes include various genetic, congenital, or acquired structural or electrical cardiac disorders¹⁻⁴; in masters athletes (Section X; ≥35 years of age), coronary atherosclerosis predominates.³¹ In a considerable proportion of cases (10%-42%), SCD pathogenesis is not identified by autopsy.^{31,58-61} Because of the lack of widespread, mandatory case reporting, the precise incidence of SCD in competitive athletes is uncertain, but likely varies as a function of factors including sex, self-identified race, and sport type.^{20,62} Among US collegiate

TABLE 4 Clinical Considerations for the Preparticipation Cardiac Evaluation of Competitive Athletes

Specific clinical considerations

Cardiac screening should be considered 1 component of SCA prevention that aims to identify competitive athletes with unrecognized cardiovascular disease to allow individualized and disease-specific management to prevent an adverse event.

A cardiac screening program should ensure access to high-quality primary screening and secondary evaluation, including the financial and logistical resources to ensure a systematic process for downstream clinical evaluation.

As a component of preparticipation screening, the cardiovascular medical history and physical examination should be performed as it can detect symptomatic competitive athletes with previously unrecognized disease and those with a family history suggestive of an inherited cardiovascular disorder.

The inclusion of a resting 12-lead ECG is reasonable as it improves detection of underlying cardiac conditions in asymptomatic competitive athletes compared with medical history and physical examination alone.

Effective ECG-inclusive preparticipation screening requires the involvement of clinicians with adequate training in the use of contemporary athlete-specific ECG interpretation criteria to minimize potential harm.

Cardiac imaging, exercise stress testing, and ambulatory rhythm monitoring have insufficient data to suggest incremental value for use in the primary screening of asymptomatic competitive athletes.

No approach to cardiac preparticipation screening provides absolute protection against SCA. Thus, an emergency action plan that includes training in high-quality CPR, prompt access to an AED, and a coordinated medical transport system should be developed, practiced, and used for all environments in which competitive athletes train and compete.

AED indicates automated external defibrillator; CPR, cardiopulmonary resuscitation; and SCA, sudden cardiac arrest.

athletes from 2002 through 2022, the annual incidence of SCD at any time during the day was 1:63 682, with increased risk in male compared with female athletes, Black compared with White athletes, and certain sports, such as basketball, American-style football, and soccer.²⁰ In Denmark, where reporting of SCD in athletes is mandated, the annual incidence of SCD among competitive athletes during or within 1 hour after physical exertion is 1.2:100 000.⁶³

Preparticipation Cardiac Screening: H&P

Preparticipation cardiac screening aims to detect cardiovascular conditions that increase the risk of SCA or SCD during competitive sports participation.^{60,61} Contact with health care professionals during the PPE screening also provides the opportunity for equitable health care access for all athletes.⁶⁴ A focused H&P has historically been at the core of the PPE and should be performed as part of preparticipation cardiac screening.⁶ Although the H&P has relatively low sensitivity (10%-20%) in detecting silent cardiac conditions,⁶⁶⁻⁶⁸ symptomatic athletes with previously unrecognized disease and individuals with family history suggestive of inherited cardiovascular disorders can be identified. Improved emphasis on practices and resources, including adequately trained personnel to conduct the H&P, carefully planned logistics for mass screenings, and newer digital technologies to

educate athletes about relevant cardiovascular symptoms,⁶⁹ may improve the sensitivity of the H&P.

Preparticipation Cardiac Screening: ECG

An ECG enhances detection of ion channelopathies, accessory pathways, and many cardiomyopathies, increasing the sensitivity of the PPE for detection of potentially fatal cardiac conditions to 94%.^{68,70} This increased sensitivity comes at the cost of decreased specificity and increased need for subsequent detailed cardiac evaluation, which in the case of false-positive ECG results will prove to have been unnecessary. These secondary evaluations require access to expert care to minimize unnecessary testing or procedures, unjustified sports disqualifications, financial costs, or emotional stresses. Therefore, an effective ECG-inclusive PPE necessitates the involvement of appropriately trained clinicians in the interpretation of athletic ECGs and timely access to appropriate resources for downstream secondary evaluations, including sports cardiology consultation, to minimize potential harms attributable to unnecessary or prolonged restriction from sports.¹⁴

Contemporary athletic ECG interpretation criteria have led to substantial improvements in the sensitivity and specificity of screening ECGs and have reduced interobserver variation in interpretation.^{43,71} However, contemporary criteria are still associated with substantial racial disparities, with higher false-positive rates in Black athletes.^{43,72} It is important to recognize the inability of screening ECGs to detect electrically silent cardiac conditions that contribute to SCA or SCD, such as anomalous aortic origin of the coronary arteries, aortopathies, substantial valve disease, and adrenergically mediated arrhythmias. Whereas cardiac imaging, exercise testing, ambulatory rhythm monitoring, and genetic testing are necessary during the secondary evaluation of athletes, there are insufficient data to recommend these tests as screening tools for asymptomatic competitive athletes.

Secondary Prevention: EAP

No primary prevention approach provides absolute SCA protection.⁹ Organizations that sponsor competitive sports must have an EAP that addresses immediate SCA recognition, performance of high-quality cardiopulmonary resuscitation, rapid retrieval and use of an AED, and medical transport to a designated clinical center.^{64,73,74} Core elements of a competitive sports EAP also include the designation of a primary EAP coordinator who is responsible for oversight of the EAP and ensuring that the plan is written and distributed to members of the response team and other key stakeholders. Education on SCA recognition and cardiopulmonary resuscitation and AED training should be emphasized in the core curriculum provided to members of the athlete health care team.

The EAP should be reviewed before organized competitive events, with team members aware of their respective responsibilities, location of equipment, and location of the accepting medical facility in the event of a medical emergency. Regular practice drills and review and revisions of the written EAP, at least on an annual basis, are essential.

Health Equity

Whereas the PPE is an ethically justified precaution to offer athletes, more research is needed to understand the effects of various screening strategies on long-term health outcomes, including prevention of SCA or SCD, health disparities, and health care costs.^{60,75,76} Moreover, there are complex ethical issues relating to equitable access and the potential to cause harm, which include delays in evaluation and inappropriate sports disqualifications, particularly among competitive athletes who come from geographic areas with limited access to health care resources.⁴² Equitable treatment in screening includes addressing potential cultural sensitivities, language, and financial barriers. Future work is required to refine the ethico-legal approach to risk mitigation that integrates evidence-based processes, respect for athlete autonomy, and considerations of the effects of social determinants of health.⁷⁷

SECTION III: ETHICAL ASPECTS OF COMPETITIVE SPORTS ELIGIBILITY

Competitive sports eligibility recommendations have historically endorsed a paternalistic care model in which physicians unilaterally determined eligibility for competitive athletes with CVD. This precedent began with the 1985 Bethesda Conference proceedings, which stated that “the athlete may not be able to use proper judgment in determining whether to extricate himself or herself from the competitive event.”³; Regardless of the intended effects of this phrase, it has fostered the notion that competitive athletes are incapable of meaningfully participating in their own health care decisions. In the majority of clinical situations, this is neither accurate nor ethical.^{78–80} Therefore, this scientific statement presents a considerable paradigm shift. SDM, the process by which patients and clinicians work together to define reasonable decisions,^{13,81,82} is now universally recommended for competitive athletes with CVD (Table 5).

SDM is supported by emerging sport participation outcomes data and fundamental ethical principles.⁸³ SDM serves to respect autonomy and advance beneficence by giving the competitive athlete the agency to incorporate personal preferences and values into decisions that affect their lives. SDM also enhances justice given power imbalances and socioeconomic and cultural differences

TABLE 5 Clinical Considerations for Shared Decision-Making With Competitive Athletes**Ethical aspects of sports participation for athletes with cardiovascular disease**

The use of SDM, the process by which competitive athletes and clinicians work together to define reasonable health care decisions and sports participation options that align with the individual athlete's values and preferences, is an ethical imperative and critical to ensure equitable outcomes for all competitive athletes with cardiovascular diagnoses.

SDM should be applied to competitive athletes <18 y with direct involvement and informed consent for the process and outcome provided by parents or legal guardians.

Clinicians should confirm diagnostic accuracy of the suspected cardiovascular disease and complete disease-specific risk stratification as dictated by current clinical guidelines before definitive discussion of competitive sport participation. This process should be conducted with input from subspecialists in sports cardiology and disease-specific experts to fully inform SDM with the competitive athlete.

When treatment of a specific cardiovascular disease is indicated, clinicians should engage subspecialists in sports cardiology and disease-specific experts to provide guidance on therapeutic options that take into consideration the competitive athlete's sporting discipline, field position, and personal values.

Clinicians should provide the competitive athlete with evidence-based medical information related to their diagnosis, management options, and potential risk of subsequent sport participation, while also conveying areas of medical uncertainty.

Clinicians should approach SDM as a series of meetings between 2 experts—where the clinician is the expert on the medical data and the potential clinical implications of different choices, and the patient-athlete is the expert on personal values and ambitions—in which both sides work toward an informed decision about future exercise and competitive sports participation.

Clinicians should elicit a competitive athlete's values, goals, and preferences, including risk tolerance, to help inform the SDM discussion on whether to continue, modify, or terminate competitive sports participation.

Competitive athletes and clinicians should identify and engage key stakeholders early in the SDM process regarding future competitive sports participation. Stakeholders may include but are not limited to parents, other family members, spouses, coaches, team physicians, athletic trainers, school administrators, and team/league/federation leadership.

In clinical scenarios suggestive of higher risk, based on either sport type or cardiac diagnosis, competitive athletes should be withheld from competitive sports participation until completion of diagnostic evaluation and guideline-directed risk reduction therapy.

Clinicians should document the process and initial outcome of SDM, including an assessment of the competitive athlete's understanding of the benefits and risks associated with the decision to continue or terminate competitive sports participation.

Regardless of the sport participation outcome that follows diagnosis and SDM, clinicians should formulate plans for longitudinal clinical surveillance, including periodic reassessment of clinical course, reflection on shared decisions to date, current exercise habits, and local emergency action planning.

SDM indicates shared decision-making.

between many competitive athletes and their clinicians, and potentially schools or sporting organizations. There are several additional reasons why SDM for athletic participation has compelling ethical justification. First, decisions regarding competitive sports participation have major implications that may not be obvious to clinicians without effective engagement. Second, these decisions involve balancing incommensurable benefits, goals, and values with potential risks that are often similarly challenging to quantify. Third, these decisions must reconcile

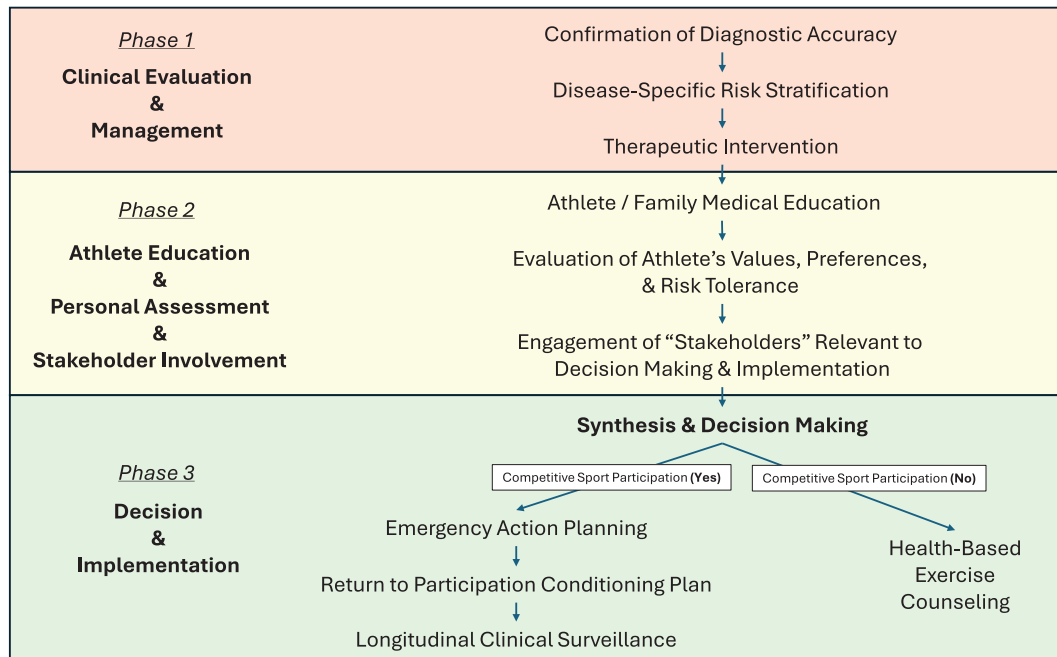
the short-term benefits of competitive sports participation with longer-term risks of adverse cardiac events and disease progression.

The application of SDM for competitive sports participation decisions aligns with more broadly changing cardiovascular practice patterns. Professional societies have increasingly called for the integration of SDM,^{84,85} as now reflected in clinical practice guidelines, including those for HCM and CAD.⁸⁶⁻⁸⁸ In addition, third-party payers recognize the importance of SDM. Medicare's National Coverage Determination for placement of both primary prevention ICD and left atrial appendage closure devices require documentation of SDM.^{89,90}

There is no legal precedent regarding potential protections and liability risks for clinicians who engage in SDM. In the 1996 ruling in Knapp versus Northwestern University,⁹¹ the courts upheld the right of a university to disqualify a competitive athlete after an individualized medical evaluation leading to a reasonable medical basis for sport exclusion. Whereas the Knapp ruling provides legal support for the "team physician medical judgement model," it does not address the role of SDM in the process of sport participation decision-making. SDM may undergo direct legal scrutiny in the future. Whereas simulation studies suggest that SDM may decrease likelihood of lawsuits after an adverse event,⁹² this prediction has not been rigorously tested in sports cardiology. It also remains unknown whether clinicians who do not use SDM can be held legally liable for adverse effects of competitive sport disqualification.

Practical Application of SDM

Practical considerations for the application of SDM are summarized in **Figure 2**. SDM begins with clinical evaluation and management. This phase of the process involves confirmation of diagnostic accuracy, comprehensive risk stratification, and implementation of therapeutic options that reduce the risk of future adverse events or disease progression. It is reasonable, on a case-by-case basis, to withhold or modify competitive sports participation during this phase. The second phase involves competitive athlete and family medical education regarding the known and unknown risks and benefits of future competitive sports participation. This education should occur in parallel with discussions that elicit the competitive athlete's core values, personal preferences, and risk tolerances. It is appropriate during this phase to include stakeholders relevant to the competitive athlete, including family members, coaching staff, athletic trainers, team physicians, and organization administrators. Active stakeholder participation ensures procedural transparency, facilitates input from various perspectives, and increases the likelihood that a reasonable consensus

FIGURE 2 A Stepwise Approach to the Implementation of Shared Decision-Making Regarding Participation in Competitive Sports Among Athletes with Cardiovascular Disease

decision can be made and ultimately implemented. The final phase of SDM is decision-making and implementation. For competitive athletes who are <18 years of age, final adjudication rests with both parents or legal guardians, who should be in agreement with the decision. Among competitive athletes who elect to terminate competitive sports participation after SDM, counseling about the transition to noncompetitive lifelong exercise should be performed. For competitive athletes who elect to continue competitive sports participation, development of a return-to-sports conditioning plan, EAP, and long-term clinical surveillance program represent clinical imperatives.

SECTION IV: CARDIOMYOPATHIES

This section provides competitive sports participation considerations for athletes with genetic cardiomyopathies (Table 6). Competitive athletes who have positive genetic test results for a specific cardiomyopathy, but no disease phenotype (ie, genotype-positive, phenotype-negative), are also addressed. SDM, risk stratification, guideline-directed treatments, and longitudinal assessments are central principles of clinical management and guidance for competitive sports participation.

Hypertrophic Cardiomyopathy

HCM is primarily defined by unexplained LV hypertrophy with an inherited basis and is often linked to a known or suspected sarcomeric genetic variant. The phenotypic spectrum, clinical course, and SCA risk in patients with HCM is variable.⁸⁷ Most identifiable causal genetic variants are in genes encoding cardiac sarcomere proteins, but ≈50% of patients are genotype-negative. Given the low arrhythmic risk associated with genotype-positive, phenotype-negative status,⁸ these individuals can participate in competitive sports. Recent data suggest lower cardiac risks associated with competitive sports participation than has been hypothesized in previous sports eligibility articles. These include series of athletes with HCM participating in competitive sports without substantial breakthrough cardiac events,^{10,93} retrospective comparisons between athletes with HCM who continued competitive sports participation versus those who medically retired,^{94,95} and a prospective study that showed that individuals with HCM who engaged in vigorous exercise, including a subset of competitive athletes, were not more likely to experience malignant ventricular arrhythmias (VAs) compared with those who exercised moderately or were less active.⁸ Therefore, it is reasonable to consider competitive sports participation for competitive athletes with HCM.

TABLE 6 Clinical Considerations for Competitive Athletes With Cardiomyopathies**General considerations**

A uniform approach of restriction or disqualification from competitive sports participation should not be applied to competitive athletes with cardiomyopathies. Rather, SDM should be used to determine participation in competitive sports.

Regardless of the initial decision to continue or terminate competitive sports participation, competitive athletes with cardiomyopathy should undergo longitudinal clinical reassessment and risk stratification to assess for disease progression, or stability, and to readdress the key components of SDM for competitive sports participation or exercise guidance.

In competitive athletes with cardiomyopathy, guideline-directed treatments should be initiated and optimized before participation in competitive sports.

Among the subgroup of athletes with cardiomyopathy who fulfill contemporary clinical guideline criteria for an ICD, a postimplantation recovery period (see Section VIII) should be completed before consideration of resumption of competitive sports participation.

In competitive athletes diagnosed with a cardiomyopathy, an ICD should not be implanted for the sole purpose of competitive sports participation.

Athletes with a cardiomyopathy who choose to discontinue competitive sports participation should be counseled about the established importance of recreational physical activity to optimize health and longevity.

Hypertrophic cardiomyopathy

Specific clinical considerations

Competitive athletes with positive genetic test results for a pathogenic or likely pathogenic HCM variant (typically identified by variant-specific, cascade family testing) without a clinical diagnosis of HCM can participate in competitive sports.

It is reasonable to consider competitive sports participation for competitive athletes with HCM after comprehensive expert assessment with SDM in which benefits and potential risks, including SCD, are discussed.

Dilated cardiomyopathy

Specific clinical considerations

Competitive sports participation is reasonable for competitive athletes with positive genetic test results for a pathogenic or likely pathogenic DCM variant (typically identified by variant-specific, cascade family testing) without a clinical diagnosis of DCM.

It is reasonable to consider competitive sports participation for competitive athletes with DCM after comprehensive expert assessment with SDM in which benefits and potential risks, including SCD, are discussed.

Arrhythmogenic cardiomyopathy

Specific clinical considerations

For competitive athletes with positive genetic test results for a pathogenic or likely pathogenic ACM variant (typically identified by variant-specific, cascade family testing), but without a clinical diagnosis of ACM, competitive sports participation is reasonable to consider after expert assessment with SDM. For competitive athletes who continue competitive sports participation, close surveillance to detect early signs of phenotypic conversion is warranted.

Competitive athletes with a clinical diagnosis of *PKP2* ACM (ie, ACM attributable to a pathogenic or likely pathogenic variant in *PKP2*) should be advised that the risks of ventricular arrhythmias, structural disease progression, and SCD with continued endurance or higher-intensity competitive sports participation likely outweigh benefits.

Competitive athletes with non-*PKP2* ACM can consider competitive sports participation after comprehensive expert assessment with SDM in which benefits and potential risks, including SCD, are discussed. Although evidence of disease acceleration or increased arrhythmic risk is not established for non-*PKP2* ACM, these considerations should be individualized given the level of uncertainty.

Left ventricular hypertrabeculation

Specific clinical considerations

Competitive athletes with LVHT in the absence of symptoms, family history of cardiomyopathy, abnormal ECG, impaired LV systolic function inconsistent with exercise-induced cardiac remodeling, or complex ventricular arrhythmias can participate in competitive sports.

All considerations for DCM apply to competitive athletes with superimposed LVHT and reduced LV systolic function that is inconsistent with exercise-induced cardiac remodeling.

All considerations for HCM apply to competitive athletes with superimposed LVHT and LV hypertrophy that is inconsistent with exercise-induced cardiac remodeling.

ACM indicates arrhythmogenic cardiomyopathy; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; ICD, implantable cardioverter defibrillator; LV, left ventricular; LVHT, left ventricular hypertrabeculation; SCD, sudden cardiac death; and SDM, shared decision-making.

Dilated Cardiomyopathy

DCM is defined by LV or biventricular dilation and systolic dysfunction. The phenotypic spectrum and arrhythmogenic potential is broad and varies, attributable in part to the underlying genotype. Genes in virtually all cellular compartments of the cardiac myocyte have been implicated in DCM, but ≈60% of patients with DCM are genotype-negative. SCA risk in DCM increases with lower ejection fraction (EF), symptoms, and higher scar burden.^{96,97} VAs are more common in some genetic

subtypes, such as lamin A/C (*LMNA*), desmoplakin (*DSP*), and filamin C (*FLNC*) genes.⁹⁸⁻¹⁰¹ However, it is unclear whether competitive sports participation heightens this risk. It is reasonable for genotype-positive, phenotype-negative athletes to participate in competitive sports, but there is uncertainty whether vigorous exercise could trigger phenotypic conversion with specific genotypes, such as *LMNA*.¹⁰²

It is reasonable to consider competitive sports participation for athletes with DCM, but the effect of vigorous

exercise on the progression of DCM and risk of SCA is unknown. Preliminary evidence suggests that higher cumulative lifetime exercise exposure is associated with lower LVEF in *LMNA*-associated DCM.^{101,102} Thus, for *LMNA* variants or other genes associated with VA, close surveillance is warranted. Any athlete with EF <40% or symptoms who continues with competitive sports participation should be aware that their risks may be higher than for asymptomatic athletes with DCM and mildly reduced EF.

Arrhythmogenic Cardiomyopathy

Arrhythmogenic cardiomyopathy (ACM) is characterized by dysfunction of the right, left, or both ventricles, and a high frequency of VA out of proportion to chamber dilation or systolic dysfunction.¹⁰³ Most identifiable variants are in genes encoding cardiac desmosome proteins, with the plakophilin-2 gene (*PKP2*) the most common, but ≈50% of patients with ACM are genotype-negative. Genotype is an important SCA risk factor for athletes with ACM. With competitive sports participation, particularly endurance-based sports, there is heightened SCA risk and disease acceleration caused by *PKP2*-mediated ACM.¹⁰⁴⁻¹⁰⁷ Evidence for increased SCA risk or disease acceleration is not well established for other genotypes or for genotype-negative ACM.

In the clinical evaluation of athletes with genotype-positive ACM, avoidance of misdiagnosis of an ACM phenotype (dilation of the right ventricle [RV] as a consequence of exercise-induced cardiac remodeling) is crucial, and requires careful assessments and clinical interpretations. For genotype-positive, phenotype-negative athletes, there is no evidence for heightened SCA risk associated with competitive sports participation.¹⁰⁷ However, vigorous exercise is a risk factor for phenotypic conversion for *PKP2*-mediated ACM, and possibly transmembrane protein 43 gene (*TMEM43*) ACM.¹⁰⁵⁻¹⁰⁸ Therefore, whereas competitive sports participation is reasonable to consider, close surveillance with imaging assessments every 6 to 12 months to detect early signs of phenotypic conversion is warranted.

Athletes with *PKP2*-mediated ACM should be advised that competitive endurance sports will likely increase their risk of VA, ICD shocks, and structural disease progression.^{105,106,109} These risks of higher-intensity competitive sports participation likely outweigh benefits.

In athletes with non-*PKP2* genotype-positive ACM, or genotype-negative ACM, evidence for either increased SCA risk or disease acceleration with competitive sports participation is not established. Competitive sports participation can be considered in these individuals, but should be carefully individualized given uncertainty and need for more data.¹¹⁰⁻¹¹²

Left Ventricular Hypertrabeculation

Left ventricular hypertrabeculation (LVHT), the preferred term over LV noncompaction, is no longer considered a distinct cardiomyopathy.¹¹³ It is characterized by prominent LV trabeculae and deep intertrabecular recesses.¹¹⁴⁻¹¹⁶ In its pathologic form, LVHT occurs coincident with HCM or DCM associated with LV hypertrophy or wall thinning, VA, and thromboembolism. In the absence of pathologic forms, adverse cardiac events in competitive athletes with LVHT have not been demonstrated. There is no evidence that SCA risk in individuals with DCM or HCM is heightened by coexistent LVHT.¹¹⁶ Asymptomatic athletes with LVHT and no pathologic features of cardiomyopathy should be considered unaffected and can continue with competitive sports participation. Athletes with LVHT and cardiomyopathic features, VA, or symptoms should follow considerations for athletes with DCM or HCM.

SECTION V: MYOCARDITIS/PERICARDITIS, VALVULAR HEART DISEASE, AND OTHER ACQUIRED CARDIOVASCULAR CONDITIONS

In this section, consideration of competitive sports participation in cases of myocarditis or pericarditis is detailed, along with valvular heart disease (VHD), particularly mitral valve prolapse (MVP), and other acquired cardiac conditions (Table 7).

Myocarditis

Myocarditis is an inflammatory condition with heterogeneous underlying pathogeneses that often follows a benign clinical trajectory. However, SCA or SCD can occur,^{19,20} as vigorous exercise in the setting of active myocardial inflammation can provoke malignant VA.¹¹⁷⁻¹²¹ With appropriate pretest clinical probability,³⁹ initial diagnostics include ECG, cardiac biomarkers, and echocardiography as part of the clinical evaluation. The inclusion of CMR, with T2-weighted imaging, parametric mapping, and late gadolinium enhancement, has also emerged as standard of care in the initial assessment and clinical follow-up of myocarditis.¹²² Late gadolinium enhancement indicates inflammation or necrosis,^{123,124} early fibrosis,^{125,126} and a potential arrhythmia substrate by promoting adverse LV remodeling.^{122,127} Impaired LVEF also predicts adverse outcomes.¹²⁸ Based solely on expert opinion in the absence of data, previous recommendations proposed competitive sports participation cessation for 3 to 6 months after myocarditis.^{37,129} Recent data from competitive athletes with myocardial inflammation after SARS-CoV-2 infection suggest that resolution of inflammation, as initially detected by CMR, can occur 4 to 6 weeks after diagnosis.⁴¹ Thus, an earlier return to

TABLE 7 Clinical Considerations for Competitive Athletes With Myocarditis, Pericarditis, Valvular Heart Disease, or Other Acquired Cardiac Conditions**Myocarditis**

Specific clinical considerations

Competitive athletes with suspected myocarditis by symptom presentation, abnormal 12-lead ECG findings, or biomarkers indicating inflammation and nonischemic myocardial injury should undergo CMR imaging to evaluate for myocardial scar and edema.

Independent of LV function, competitive athletes with myocarditis should not participate in competitive sports until both symptoms and active inflammation or edema (T2 or elevated troponin levels, or both) have resolved.

It is reasonable for competitive athletes with myocarditis and reduced LV function at the time of diagnosis to resume competitive sports participation if all the following criteria are met:

- There has been a period of ≥ 3 mo without symptoms.
- LV systolic function has returned to normal range.
- There is resolution of inflammation or edema by CMR imaging (T2 signal) or serum biomarkers (inflammation).
- Clinically relevant arrhythmias, including frequent or complex repetitive forms of ventricular arrhythmias, are absent on ambulatory ECG monitoring and exercise testing.

Resumption of competitive sports participation for competitive athletes with myocarditis and preserved LV function can be considered 4 to 6 wk after complete resolution of symptoms if all the following criteria are met:

- There is resolution of inflammation or edema by CMR (T2 signal) or serum biomarkers (inflammation).
- Clinically relevant arrhythmias, including frequent or complex repetitive forms of ventricular arrhythmias, are absent on ambulatory ECG monitoring and exercise testing.

It is reasonable for asymptomatic competitive athletes with persistent LGE suggestive of previous myocarditis to continue competitive sports participation if all the following criteria are met:

- LV systolic function is within the normal range.
- Clinically relevant arrhythmias, including frequent or complex repetitive forms of ventricular arrhythmias, are absent on ambulatory ECG monitoring and exercise testing.

In competitive athletes with resolved myocarditis and persistent LGE, it is reasonable to continue longitudinal clinical surveillance.

For competitive athletes who have recovered from myocarditis but continue to exhibit LV dysfunction or ventricular arrhythmias, refer to [Section IV](#) and [Section VIII](#), respectively.

Acute viral infections, including SARS-CoV-2 (COVID-19)

Specific clinical considerations

For competitive athletes who have recovered from acute respiratory viral infections, including with SARS-CoV-2, with noncardiopulmonary symptoms,* cardiac testing or screening should not be performed before participation in competitive sports.

For competitive athletes who have recovered from acute SARS-CoV-2 infection with persistent cardiopulmonary symptoms,† similar to other upper respiratory tract viral infections, a clinical cardiac evaluation‡ should be performed to exclude myocardial involvement before participation in competitive sports.

Long COVID§

Specific clinical considerations

Competitive athletes with presumed long COVID and cardiopulmonary symptoms† should have an initial clinical cardiac evaluation‡ to exclude myocardial involvement before participation in competitive sports.

Whereas an immediate return to competitive sports participation may be limited because of symptoms, supervised exercise training should be a part of the treatment plan for long COVID recovery.

SARS-CoV-2 vaccination

After SARS-CoV-2 vaccination, flu-like side effects are common; thus a clinical cardiac evaluation‡ should not be performed before participation in competitive sports.

Competitive athletes who complain of acute cardiopulmonary symptoms† within 1 wk of SARS-CoV-2 vaccination should have a clinical cardiac evaluation‡ to exclude the presence of vaccine-associated myocarditis.||

Incidental myocardial fibrosis

Specific clinical considerations

Isolated LGE on CMR at the RV insertion points may be observed in competitive athletes and does not require further evaluation.

Asymptomatic competitive athletes with myocardial fibrosis, indicated by LGE on CMR, and in a pattern other than RV insertion point LGE, should undergo appropriate risk stratification and longitudinal assessment. SDM should guide the determination of competitive sports participation.

Pericarditis

Specific clinical considerations

Competitive athletes with acute pericarditis should be managed according to the standard of care, which includes nonsteroidal anti-inflammatory medications for 2 to 4 wk and 3 mo of colchicine. Steroids generally should be avoided to reduce the risk of recurrent pericarditis.

Competitive athletes with active pericarditis should not participate in competitive sports because of the increased risk of exacerbating an inflammatory response.

Competitive athletes with acute pericarditis and a complicated presentation¶ comprise a subset of patients at greater risk for recurrence and progression to pericardial constriction and should have longitudinal clinical surveillance.

It is reasonable for competitive athletes to resume competitive sports participation when there are no signs and symptoms of active disease (no chest pain, pericardial effusion, or elevation of serum markers of inflammation).

Competitive athletes who have recovered from pericarditis should resume competitive sports participation in a gradual fashion with longitudinal monitoring for symptoms of recurrent pericarditis.

In competitive athletes with chronic recurrent pericarditis or pericardial constriction, the risks may outweigh the benefits of competitive sports participation.

TABLE 7 Continued**Valvular heart disease**

General considerations

Competitive athletes presenting with symptomatic VHD should be managed with established guidelines from the AHA and ACC.

Competitive athletes with VHD should continue longitudinal clinical surveillance to monitor disease progression.

If clinically available, CPET can be a valuable tool for confirming the presence of VHD symptoms and determining the appropriate timing for intervention with longitudinal assessments.

Competitive athletes exhibiting mild severity of left-sided VHD, including AS, AR, mitral stenosis, and MR, can participate in competitive sports.

Aortic stenosis

Specific clinical considerations

Moderate AS represents a continuum of risk, and a comprehensive evaluation including careful assessment of symptoms and exercise testing to the level of activity achieved in competitive sports participation should be considered. Competitive sports participation is reasonable with SDM in asymptomatic competitive athletes with moderate AS and normal stress testing results.

Competitive athletes with severe or nonsevere symptomatic AS should not participate in competitive sports and valvular intervention should be considered with SDM.

The risks likely outweigh the benefits of competitive sports participation for competitive athletes with asymptomatic, severe AS, with the exception of lower-intensity strength and endurance sports (see [Section I](#)), and valvular intervention should be considered with SDM.

Mitral stenosis

Specific clinical considerations

Competitive sports participation is reasonable for asymptomatic competitive athletes with moderate mitral stenosis and normal exercise tolerance.

In competitive athletes with asymptomatic severe mitral stenosis, the risks, which include the provocation of atrial arrhythmias, may outweigh the benefits of competitive sports participation.

In competitive athletes with any severity of mitral stenosis and a history of atrial fibrillation who must receive anticoagulant therapy, the risks of some competitive sports involving collisions or impacts likely outweigh benefits (see [Section I](#)).

Mitral and aortic regurgitation

Specific clinical considerations

Asymptomatic competitive athletes with moderate regurgitant lesions can participate in competitive sports if all the following criteria are met:

- Normal LV systolic function
- Cardiac ventricular dimensions are normal or exhibit ventricular dilation that is most consistent with exercise-induced cardiac remodeling
- Echocardiography shows no evidence of pulmonary hypertension
- Normal exercise tolerance

Competitive sports participation is reasonable for asymptomatic competitive athletes with severe AR (which may be better tolerated during exercise as compared with severe MR) or severe MR if all the following criteria are met:

- Normal LV systolic function
- Cardiac ventricular dimensions are normal or exhibit ventricular dilation that is most consistent with exercise-induced cardiac remodeling
- Echocardiography shows no evidence of pulmonary hypertension
- Normal exercise tolerance

The risks may outweigh the benefits of competitive sports participation for asymptomatic competitive athletes with severe AR or MR if any of the following criteria are present:

- Reduced LV systolic function without normal augmentation with exercise stress
- Increased ventricular dimensions inconsistent with exercise-induced cardiac remodeling
- Echocardiography shows evidence of pulmonary hypertension
- Presence of exercise-induced ventricular arrhythmias

Cardiac valve surgery

Specific clinical considerations

Competitive sports participation is reasonable for competitive athletes with bioprosthetic valves and normal LV systolic function. Postsurgical evaluation should include a maximal-effort exercise stress test. Resumption of competitive sports participation should be individualized after complete sternal healing and participation in postsurgical exercise rehabilitation.

In competitive athletes with mechanical valves or interventions requiring indefinite oral anticoagulation, the risks likely outweigh the benefits for some competitive sports involving collisions or impacts (see [Section I](#)).

Athletes with mitral valve prolapse

Specific clinical considerations

Asymptomatic competitive athletes without any high-risk features by history, ECG, or echocardiography[#] related to MVP do not require further evaluation and can participate in competitive sports.

Asymptomatic competitive athletes with MVP who have high-risk features[#] should undergo additional risk stratification with exercise testing, ambulatory rhythm monitoring, and CMR.

Competitive athletes with arrhythmic MVP (ie, documented ventricular arrhythmias with or without symptoms [exertional palpitations or syncope]) should undergo comprehensive expert assessment for treatment of arrhythmias and risk stratification for an implantable cardioverter defibrillator. Competitive sports participation can be considered with SDM for competitive athletes with arrhythmic MVP who are clinically stable after appropriate arrhythmia suppression and rhythm stability.

In competitive athletes with MVP and secondary MR, refer to MR considerations above.

Competitive sports participation for competitive athletes with asymptomatic MVP and inferolateral LV LGE without complex ventricular arrhythmias is reasonable with longitudinal clinical surveillance for disease progression or ventricular arrhythmias.

TABLE 7 Continued**Kawasaki disease**

Coronary artery involvement: ectasia, small aneurysm**

Specific clinical considerations

Competitive sports participation is reasonable for competitive athletes with ectasias or small aneurysms after comprehensive evaluation as per published guidelines.

Competitive athletes with coronary artery involvement should have longitudinal clinical surveillance.

Competitive athletes requiring aspirin monotherapy can participate in competitive sports.

Coronary artery involvement: medium, large/giant aneurysms**

Specific clinical considerations

Competitive athletes with medium or large/giant aneurysms should undergo comprehensive evaluation for inducible myocardial ischemia or ventricular arrhythmias with provocative exercise stress before participation in competitive sports.††

Competitive sports participation is reasonable for competitive athletes with medium or large/giant aneurysms and no inducible myocardial ischemia and ventricular arrhythmias with provocative exercise stress.††

In competitive athletes with medium or large/giant aneurysms who have inducible myocardial ischemia or ventricular arrhythmias, the risks likely outweigh benefits of competitive sports participation.

For competitive athletes who are on dual antiplatelet‡‡ or full anticoagulation therapy, the risks likely outweigh the benefits in some competitive sports participation (see Section I).

Infiltrative cardiomyopathies (Anderson Fabry, sarcoidosis, amyloidosis, hemochromatosis)

Specific clinical considerations

Asymptomatic competitive athletes with a family history or who are gene-positive/phenotype-negative for select infiltrative cardiomyopathies can participate in competitive sports.

In competitive athletes with clinical expression and high-risk features (heart failure symptoms, ventricular arrhythmias, decreased LV systolic function) of an infiltrative cardiomyopathy, the risks likely outweigh the benefits of competitive sports participation.

Competitive athletes with clinical expression of an infiltrative cardiomyopathy, but who are treated and clinically stable with normal LV systolic function, can consider competitive sports participation with SDM and longitudinal clinical surveillance.

*Including but not limited to cough, congestion, headache, fever, myalgias, arthralgias, anosmia, dysgeusia, and gastrointestinal symptoms.

†Chest pain/tightness, dyspnea, palpitations, presyncope/syncope.

‡Evaluation generally includes 12-lead ECG, transthoracic echocardiogram, and high-sensitivity troponin assay. Abnormal findings or persistent cardiopulmonary symptoms require CMR with consideration of exercise testing as additional testing.

§Long COVID is defined as symptoms that persist for >12 weeks after SARS-CoV-2 infection.

||Adolescent and young adult male individuals have been shown to be a particular higher-risk demographic category.

¶Defined as temperature >38 °C (>100.4 °F), subacute course, large pericardial effusion, cardiac tamponade, biomarker evidence of myocardial injury (myopericarditis), or resistance to nonsteroidal anti-inflammatory drugs.

#High-risk features of MVP include, but are not limited to, T-wave inversions in the inferior leads, the presence of a prolonged QT interval, mitral annular disjunction, and a family history of sudden cardiac death. Other markers indicative of higher risk include severe MR, substantial LV dysfunction, and documented ventricular arrhythmias, which include frequent premature ventricular contractions with right bundle branch block morphology.

**Coronary artery: ectasia, z score ≥ 2 and < 2.5 ; small aneurysm, z score ≥ 2.5 and < 5 ; medium aneurysm, z score ≥ 5 and < 10 , with absolute luminal dimension < 8 mm; large/giant aneurysm, z score ≥ 10 , with absolute luminal dimension ≥ 8 mm.

††Studies should not be restricted solely to exercise stress testing and should include advanced imaging.

‡‡Dual antiplatelet therapy refers to the preferential use of aspirin and the P2Y12 inhibitor clopidogrel.

ACC indicates American College of Cardiology; AHA, American Heart Association; AR, aortic regurgitation; AS, aortic stenosis; CMR, cardiac magnetic resonance imaging; CPET, cardiopulmonary exercise testing; LGE, late gadolinium enhancement; LV, left ventricular; MR, mitral regurgitation; MVP, mitral valve prolapse; RV, right ventricular; SDM, shared decision-making; and VHD, valvular heart disease.

competitive sports participation can be considered with resolution of myocardial inflammation and absence of cardiopulmonary symptoms, VA induced by exercise, and LV dysfunction.

Pericarditis

Clinical pericarditis includes pleuritic chest pain, characteristic ECG findings, and often a pericardial friction rub or effusion.^{37,129,130} Assessment with ECG, biomarkers (to exclude myopericarditis), and imaging is essential.^{37,130} Competitive athletes should avoid competitive sports participation during the acute phase to prevent inflammatory progression.^{37,129-132} Long-term concerns include exacerbation, recurrence, and evolution to pericardial constriction. To avoid adverse outcomes, return to

competitive sports participation should be gradual and only after complete symptom resolution and normalization of inflammatory biomarkers.^{37,129,130,133}

Viral Infections (Including SARS-CoV-2)

Return to competitive sports participation after substantial upper respiratory viral infection was outlined in a previous American College of Cardiology expert consensus article focused on SARS-CoV-2 and COVID-19.³⁹ In the absence of cardiopulmonary symptoms, cardiac testing is unnecessary.^{39,134} Long COVID, with cardiopulmonary symptoms persisting >12 weeks,¹³⁵ requires evaluation to exclude myocardial injury. With SARS-CoV-2 vaccination, flu-like symptoms immediately after treatment do not require cardiac assessment, but acute

cardiopulmonary symptoms ≤ 1 week after vaccination warrant evaluation for vaccine-associated myocarditis.

Myocardial Fibrosis

Clinically relevant myocardial fibrosis, suggested by late gadolinium enhancement on CMR, occurs in various disease states. However, RV insertion point late gadolinium enhancement, a finding without known risk of adverse cardiovascular events,¹³⁶ can be observed in masters athletes (Section X).¹³⁷ Recent data from competitive athletes with COVID-19 suggest this finding may also be incidentally observed among young athletes and does not require further clinical investigations.¹³⁸

Valvular Heart Disease (Including MVP)

Management of symptomatic competitive athletes with VHD and estimation of severity (stenosis or regurgitation) should comply with current guidelines.¹³⁹⁻¹⁴¹ Bicuspid aortic valves (BAVs) are discussed in Section VII. For competitive athletes with most forms of mild or moderate VHD, competitive sports participation can continue. However, it is reasonable for competitive athletes with moderate aortic stenosis to undergo risk stratification with exercise testing. With asymptomatic severe regurgitant VHD (mitral regurgitation and aortic regurgitation), hemodynamic volume overload leads to LV dilation, which can be difficult to differentiate from exercise-induced cardiac remodeling and potentially lead to overestimation of the degree of pathologic chamber enlargement in the presence of concomitant athletic heart remodeling.^{7,26} In a seminal study of 1309 Italian athletes without VHD, 14% presented with LV end-diastolic dimensions ≥ 60 mm.¹⁴² Therefore, the use of VHD cardiac dimension or volume cut points derived from the general population should be considered cautiously and applied to clinical management considerations and competitive sports participation guidance for competitive athletes.¹³⁹⁻¹⁴¹ However, asymptomatic competitive athletes with severe aortic regurgitation and extreme degrees of LV dilation (LV end-systolic diameter >50 mm or LV end-diastolic diameter >70 mm) likely warrant further clinical evaluation. Substantial LV dilation that is consistent with exercise-induced cardiac remodeling should coincide with other phenotypic features of athletic cardiac remodeling, such as symmetric enlargement of the RV and atria and supranormal LV lusitropy.⁷ Severe aortic regurgitation may have less effect on exercise tolerance compared with severe mitral regurgitation because sinus tachycardia decreases diastolic filling time and reduces AR. With severe aortic stenosis, risks of ischemia-induced SCA are likely increased with

competitive sports participation.¹⁴³ Acute SCA risk associated with severe mitral stenosis is less evident, but provocation of symptomatic atrial arrhythmias is a concern.

MVP is present in $\approx 2\%$ of the general population.¹⁴⁴ Although most MVP phenotypes are benign,¹⁴⁴ arrhythmic MVP variants are associated with SCA or SCD.^{145,146} High-risk MVP features (Table 7) can be identified by history, ECG, and echocardiography, and, if present, further risk stratification should proceed with exercise testing, ambulatory rhythm monitoring, and CMR.¹⁴⁷⁻¹⁵⁰ Competitive athletes with untreated malignant MVP and VA are at higher risk for SCA, which likely outweighs the benefits of competitive sports participation.

Kawasaki Disease

Kawasaki disease may result in coronary aneurysms predisposing to myocardial ischemia or infarction and SCD.¹⁵¹ With medium and large aneurysms, risk stratification is necessary before participation in competitive sports. Competitive athletes with coronary aneurysms who require aspirin monotherapy can participate in competitive sports, but with full anticoagulation or dual antiplatelet therapy, the risks likely outweigh benefits for some competitive sports participation with collisions or impacts (see Section I).

Infiltrative Cardiomyopathies

Infiltrative cardiomyopathies include amyloidosis, Anderson-Fabry disease, sarcoidosis, hemochromatosis, and other less common diseases. Data concerning competitive sports participation for athletes with infiltrative cardiomyopathies are limited. Expert consensus favors that gene-positive but asymptomatic and phenotype-negative competitive athletes or those with positive family histories can participate in competitive sports. With clinical signs or symptoms, the risks of acute adverse cardiac outcomes generally outweigh the benefits of competitive sports participation. Athletes under expert treatment and surveillance with clinically stable disease can consider competitive sports participation.

SECTION VI: CONGENITAL HEART DISEASE

Exercise-related SCA or SCD among patients with congenital heart disease is not common.¹⁵²⁻¹⁵⁴ However, limited data are available delineating risks of competitive sports participation for people with congenital heart disease.¹⁵⁵ Therefore, SDM is required when counseling competitive athletes with congenital heart disease. With

TABLE 8 Congenital Heart Disease Anatomy and Physiology and Competitive Sports Participation Considerations

Anatomy and physiology	Definition	Diagnoses	Additional clinical evaluations*	Reassuring clinical findings	Concerning clinical findings†	Competitive sports participation considerations in absence of concerning clinical findings‡
Left-to-right shunt						
Small	No chamber enlargement, or only enlargement consistent with expected exercise-induced cardiac remodeling	<ul style="list-style-type: none"> Small, moderate ASD Small, moderate restrictive VSD Small, moderate restrictive PDA Partial anomalous pulmonary venous connection 			<ul style="list-style-type: none"> Hypoxemia Complex arrhythmias‡ RVH on imaging 	Can participate in all competitive sports after appropriate clinical evaluation*
Moderate or large	<ul style="list-style-type: none"> Qp:Qs >1.5–2.0§ Typically asymmetric chamber enlargement due to shunt 	<ul style="list-style-type: none"> Moderate, large ASD Moderate, large VSD Moderate, large PDA PAPVC 	<ul style="list-style-type: none"> CPET (or EST) is reasonable¶ Intervention likely indicated 	No evidence of pulmonary hypertension	<ul style="list-style-type: none"> Hypoxemia Complex arrhythmias‡ RVH on imaging Severely dilated RV with RV dysfunction (ASD, PAPVC) Severely dilated LV with LV dysfunction (VSD/PDA) 	It is reasonable to consider competitive sports participation before and after intervention, as tolerated, following an SDM model with the athlete/family.*
RVOT/PA obstruction						
Less than severe	Peak echo Doppler gradient <64 mm Hg or estimated RV systolic pressure <1/2 systemic pressures	<ul style="list-style-type: none"> After TOF repair Before or after intervention: valvar PS, subvalvar PS, peripheral PS 	For postrepair patients, should perform CPET (or EST)¶	<ul style="list-style-type: none"> No or mild RVH on imaging Normal RV function 	<ul style="list-style-type: none"> Hypoxemia Complex arrhythmias‡ Severe RVH on imaging Substantial RV dysfunction 	Can participate in competitive sports after appropriate clinical evaluation*
Severe	Peak echo Doppler gradient >64 mm Hg or RV systolic pressure >1/2 systemic pressures	<ul style="list-style-type: none"> After TOF repair Before or after intervention: valvar PS, subvalvar PS, peripheral PS 	<ul style="list-style-type: none"> CPET (or EST)¶ Ambulatory cardiac rhythm monitoring Consider CMR or CCT Intervention likely indicated 	<ul style="list-style-type: none"> Mild or moderate RVH on imaging Normal RV function 	<ul style="list-style-type: none"> Hypoxemia at rest or with exercise (<85% on pulse oximeter) Complex arrhythmias at rest or stress‡ Severe RVH on imaging Substantial RV dysfunction 	Before intervention: risks may outweigh benefits for competitive sports participation. Clinicians should engage in SDM with patient/family. After intervention: See row above "less than severe."
Fixed LVOT obstruction						
Mild	Mean echo Doppler gradient <25 mm Hg or peak echo Doppler gradient <40 mm Hg	<ul style="list-style-type: none"> Valvar AS Subvalvar AS Supravalvar AS AV septal defect s/p repair with mild LVOT obstruction 		<ul style="list-style-type: none"> No LVH Normal LV size and function on imaging 	<ul style="list-style-type: none"> Greater than mild LVH inconsistent with exercise-induced cardiac remodeling LV dysfunction Complex arrhythmias‡ 	Can participate in competitive sports after appropriate clinical evaluation*
Moderate	Mean echo Doppler gradient 25–40 mm Hg or peak echo Doppler gradient 40–64 mm Hg	<ul style="list-style-type: none"> Valvar AS Subvalvar AS Supravalvar AS AV canal defect s/p repair with moderate LVOT obstruction 	<ul style="list-style-type: none"> CPET (or EST)¶ Ambulatory cardiac rhythm monitoring 	<ul style="list-style-type: none"> Asymptomatic Normal ECG No or mild LVH on imaging Normal LV size and function Normal BP response to exercise No ischemia and normal exercise capacity on exercise testing 	<ul style="list-style-type: none"> Severe LVH on imaging LV dysfunction Diagnostic ischemia: ECG changes with stress testing Rapidly increasing LVOT gradient Complex arrhythmias at rest or stress‡ Exertional symptoms 	It is reasonable to consider competitive sports participation following an SDM model with the athlete/family.*

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TABLE 8 Continued

Anatomy and physiology	Definition	Diagnoses	Additional clinical evaluations*	Reassuring clinical findings	Concerning clinical findings†	Competitive sports participation considerations in absence of concerning clinical findings‡
Severe	Mean echo Doppler gradient >40 mm Hg or peak echo Doppler gradient >64 mm Hg	<ul style="list-style-type: none"> Valvar AS Subvalvar AS Supravalvar AS AV septal defect s/p repair with severe LVOT obstruction (Section V) 	<ul style="list-style-type: none"> CPET (or EST)¶ Ambulatory cardiac rhythm monitoring Intervention likely recommended 		<ul style="list-style-type: none"> Diagnostic ischemic ECG changes with stress test Rapidly increasing LVOT gradient Complex arrhythmias at rest or stress‡ Exertional symptoms LV dysfunction Pulmonary hypertension 	<p>Before intervention: risks likely outweigh benefits of competitive sports participation for competitive athletes with severe fixed LVOT obstruction (with the exception of lower-intensity strength and endurance sports).</p> <p>After intervention: see the row "less than severe" above.</p>
Pulmonary regurgitation						
Less than severe	See appropriate ASE and SCMR guidelines	<ul style="list-style-type: none"> TOF s/p repair PS s/p repair (catheter or surgical) 	<ul style="list-style-type: none"> Consider CPET (or EST)¶ Consider CMR/CCT Consider ambulatory cardiac rhythm monitoring 	<ul style="list-style-type: none"> Normal biventricular size and function No complex arrhythmias 	<ul style="list-style-type: none"> Complex arrhythmias at rest or stress‡ Moderate to severe RV dilation Greater than mild RV dysfunction 	Can participate in competitive sports after appropriate clinical evaluation*
Severe	See appropriate ASE and SCMR guidelines	<ul style="list-style-type: none"> TOF s/p repair PS s/p repair (catheter or surgical) Isolated pulmonary regurgitation 	<ul style="list-style-type: none"> CPET (or EST)¶ CMR/CCT Consider ambulatory cardiac rhythm monitoring 	<ul style="list-style-type: none"> Mild or moderate RV dilation on imaging Normal biventricular function No complex arrhythmias at rest or stress 	<ul style="list-style-type: none"> Complex arrhythmias at rest or stress‡ Greater than mild RV dysfunction 	It is reasonable to consider competitive sports following an SDM model with the athlete/family.*
Single ventricle physiology						
Systemic LV		Tricuspid atresia, double inlet left ventricle, unbalanced atrioventricular canal, s/p Fontan procedure	<ul style="list-style-type: none"> CPET (or EST)¶ Consider CMR/CCT Ambulatory cardiac rhythm monitoring 	<ul style="list-style-type: none"> Normal or near normal ventricular function Minimal or no arrhythmias at rest, none with exercise No evidence of substantial hypoxemia with exercise (<85% on pulse oximeter) No more than mild valvular regurgitation 	<ul style="list-style-type: none"> Complex arrhythmias at rest or stress‡ Severe ventricular dysfunction Substantial hypoxemia with exercise (<85% on pulse oximeter) RV-dependent coronary circulation (pulmonary atresia with intact septum and no antegrade pulmonary blood flow) Systemic anticoagulation 	Limited data are available. Clinicians may consider competitive sports participation on an individualized basis after SDM with patient/family.# Additional consideration of bleeding risk (if anticoagulated) is necessary based on sport type (Section I).
Systemic RV		<ul style="list-style-type: none"> Hypoplastic left heart syndrome, s/p Fontan procedure Right dominant unbalanced AV canal, s/p Fontan procedure 	<ul style="list-style-type: none"> CPET (or EST)¶ CMR/CCT (with special attention to DKS, arch, and coronaries) Ambulatory cardiac rhythm monitoring 	<ul style="list-style-type: none"> See single ventricle (systemic LV) Ensure normal coronary blood flow, no aortic obstruction 	<ul style="list-style-type: none"> Complex arrhythmias at rest or stress‡ Severe ventricular dysfunction Substantial hypoxemia with exercise (<85% on pulse oximeter) Systemic anticoagulation 	Limited data are available. Clinicians may consider competitive sports participation on an individualized basis after SDM with patient/family.# Additional consideration of bleeding risk (if anticoagulated) is necessary based on sport type (Section I).

Continued on the next page

TABLE 8 Continued

Anatomy and physiology	Definition	Diagnoses	Additional clinical evaluations*	Reassuring clinical findings	Concerning clinical findings†	Competitive sports participation considerations in absence of concerning clinical findings‡
Biventricular circulation		<ul style="list-style-type: none"> ■ Dextro-TGA s/p Mustard or Senning procedure ■ CCTGA 	<ul style="list-style-type: none"> ■ CPET (or EST)¶ ■ CMR/CCT ■ Consider evaluation for baffle leaks ■ Consider ambulatory cardiac rhythm monitoring 	<ul style="list-style-type: none"> ■ Mild or moderate RV dilation on imaging ■ Normal biventricular function ■ Mild tricuspid (systemic AVV) regurgitation ■ No arrhythmias 	<ul style="list-style-type: none"> ■ Complex arrhythmias at rest or stress‡ ■ Severe ventricular dysfunction ■ Moderate or greater AVV regurgitation 	Limited data are available. Clinicians may consider competitive sports participation on an individualized basis after SDM with patient/family.# Additional consideration of bleeding risk (if anticoagulated) is necessary based on sport type (Section 1).
Transposition of the great arteries with anatomic repair	Circulation has LV aortic flow and RV PA flow	<ul style="list-style-type: none"> ■ D-TGA ■ Some types of DORV ■ Surgical repairs include** arterial switch, Rastelli procedure ■ Nikaidoh procedure 	<ul style="list-style-type: none"> ■ CPET (or EST)¶ ■ CMR/CCT (special attention to coronary arteries) 	<ul style="list-style-type: none"> ■ Normal biventricular size and function ■ No ischemia and normal exercise capacity on stress testing ■ Normal aortic dimensions ■ No more than mild aortic regurgitation 	<ul style="list-style-type: none"> ■ Severe neoaortic root dilation ■ Severe aortic regurgitation ■ Moderate or greater ventricular dysfunction ■ Coronary artery obstruction ■ Ischemic symptoms; if other concerns for coronary stenosis, proceed with stress imaging ■ Exercise-induced arrhythmias, especially those related to surgical coronary obstructions‡ 	It is reasonable to consider competitive sports participation after SDM with the athlete/family.*††
Congenital AV valve regurgitation		<ul style="list-style-type: none"> ■ Ebstein anomaly ■ Dysplastic tricuspid valve ■ AV canal defect s/p repair 	<ul style="list-style-type: none"> ■ Ambulatory cardiac rhythm monitoring 	<ul style="list-style-type: none"> ■ For Ebstein anomaly or dysplastic tricuspid valve: no WPW or residual WPW, normal RV and LV function 	<ul style="list-style-type: none"> ■ Ebstein anomaly or dysplastic tricuspid valve: RV dysfunction, complex arrhythmias at rest or stress‡ ■ AV canal defect s/p repair: substantial LVOT obstruction (see above), complex arrhythmias‡ 	It is reasonable to consider competitive sports participation after SDM model with the athlete/family.*††

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combined congenital heart disease conditions (eg, repaired tetralogy of Fallot with a residual patch margin ventricular septal defect with severe RV outflow tract obstruction), clinicians should consider the most severe component of disease first when counseling on competitive sports participation. In this section, competitive sport considerations are based on general congenital heart disease anatomy and physiology (Table 8). In addition, considerations for coronary artery anomalies, including myocardial bridging, are detailed (Table 9).

Simple and Moderate-Complexity Congenital Heart Disease

Competitive athletes with left-right shunt lesions that are of minimal hemodynamic consequence can continue competitive sports participation. Large left-right shunt lesions may lead to considerable volume overload and pulmonary hypertension; thus longitudinal surveillance

with SDM is necessary in consideration of the appropriate timing of intervention. Exercise-induced cardiac remodeling can be mistaken for shunt-mediated dilation; therefore, chamber dilation and shunt fraction, in conjunction with absence or lack of other features of exercise-induced cardiac remodeling, must be consistent to ensure that an intervention is appropriate.

For competitive athletes with RV outflow tract obstruction, severe obstruction requires surgical or catheter-based intervention before participation in competitive sports, and these athletes should be reassessed longitudinally to evaluate the degree of residual obstruction and symptoms.¹⁵⁶ Whereas competitive athletes with moderate LV outflow tract obstruction require additional risk stratification, those with severe LV outflow tract obstruction may require surgical or catheter-based intervention before participation in competitive

TABLE 8 Continued

Anatomy and physiology	Definition	Diagnoses	Additional clinical evaluations*	Reassuring clinical findings	Concerning clinical findings†	Competitive sports participation considerations in absence of concerning clinical findings‡
Aortic arch obstruction		<ul style="list-style-type: none"> ■ Coarctation of the aorta, s/p surgical repair ■ Coarctation of the aorta, s/p stent angioplasty ■ Coarctation of the aorta, unrepaired ■ Interrupted aortic arch, s/p repair ■ Can be seen in combination with VSD or DORV or other congenital repairs 	<ul style="list-style-type: none"> ■ Upper and lower extremity BPs ■ CPET (or EST)¶ ■ CTA or CMR ■ Hypertension should be treated before exercise (Section XI) 	<ul style="list-style-type: none"> ■ Normotensive at rest, treated or untreated ■ No or minimal resting upper extremity/lower extremity BP gradient ■ Normal BP response to exercise ■ No aortic aneurysms 	<ul style="list-style-type: none"> ■ Resting hypertension or hypertensive response to exercise ■ Aortic aneurysm (Section VII), either ascending or at the site of surgical repair ■ LV hypertrophy inconsistent with exercise-induced cardiac remodeling ■ Substantial residual coarctation ■ Complex arrhythmias rest or stress‡ 	It is reasonable to consider competitive sports participation after SDM with the athlete/family.#

*All competitive athletes with diagnosed congenital heart abnormalities should have 12-lead ECG, echocardiography, oxygen saturation, BP, symptom assessment, and testing listed above assessed within 1 y of initial competitive sports participation, depending on lesion (diagnosis). Patients with moderate or greater complexity or those who have inconsistent diagnostic findings should undergo specific congenital heart disease or adult congenital heart disease cardiology evaluation before initiation of competitive sports participation. See also the 2018 American Heart Association/American College of Cardiology guidelines for the management of adults with congenital heart disease.¹⁵⁵

†Sports participation considerations assume no concerning clinical findings (including no concerning symptoms with exercise). If concerning clinical findings are present, further clinical assessment is necessary before considering competitive sports participation in an SDM process. Certain findings may suggest a different diagnosis or risk classification or merit consideration for intervention before participation in competitive sports.

#For complex arrhythmias, refer to Section VIII. Tachyarrhythmias and bradyarrhythmias in congenital heart disease may portend increased risk and may require additional monitoring and treatment.

§As assessed on CMR or hemodynamic catheterization.

¶For imaging definitions of the severity of valve or ventricular dysfunction (also must contextualize with the effects of exercise-induced cardiac remodeling on ventricular function in athletes), refer to the most recent available guidelines or recommendation statements.^{7,32,140,155}

¶¶Oxygen saturation, BP, and symptoms should be assessed with maximum-effort exercise stress testing using CPET (preferred) or EST if CPET is unavailable. Pharmacologic stress testing is not advised for preparticipation competitive sports evaluation.

*Competitive sports participation in these groups is not well-studied and requires careful assessment of anatomy and physiology at rest and with exercise, specifically focusing on the physiology required for the intended sport. Pharmacologic stress testing is not advised for preparticipation competitive sports evaluation.

**Patients with double-switch TGA not addressed here; see text for further discussion.

††If hemodynamically significant residual or postintervention lesions are found, refer to the appropriate sections.

AS indicates aortic stenosis; ASD, atrial septal defect; ASE, American Society of Echocardiography; AVV, atrioventricular valve; BP, blood pressure; CCT, cardiac computed tomography; CCTGA, congenitally corrected transposition of the great arteries; CMR, cardiac magnetic resonance imaging; CPET, cardiopulmonary exercise test; CTA, computed tomography angiography; DKS, Damus-Kaye-Stansel; DORV, double outlet right ventricle; EST, exercise stress test; LV, left ventricle; LVH, left ventricular hypertrophy; LVOT, left ventricular outflow tract; PA, pulmonary artery; PAPVC, partial anomalous pulmonary venous connection; PDA, patent ductus arteriosus; PS, pulmonary stenosis; RV, right ventricle; RVH, right ventricular hypertrophy; RVOT, right ventricular outflow tract; s/p, status post; SCMR, Society for Cardiac Magnetic Resonance; SDM, shared decision-making; TGA, transposition of the great arteries; TOF, tetralogy of Fallot; VSD, ventricular septal defect; and WPW, Wolff-Parkinson-White syndrome.

sports.^{157,158} With aortic arch obstruction (coarctation), patients may be at risk for cardiovascular events even after successful repair; thus these competitive athletes should continue with longitudinal surveillance inclusive of blood pressure control, imaging, and exercise testing.¹⁵⁹

Competitive athletes with repaired tetralogy of Fallot may experience pulmonary valve dysfunction with subsequent RV hypertrophy or severe pulmonary regurgitation, atrial arrhythmias and VAs, or ventricular dysfunction, placing them at increased risk of SCA with exertion. Longitudinal surveillance and risk stratification with imaging, exercise testing, and ambulatory rhythm monitoring is required for competitive athletes with tetralogy of Fallot.¹⁵⁶

High-Complexity Congenital Heart Disease

Limited data are available assessing the safety of competitive sports participation for individuals with repaired and palliated complex congenital heart disease.

Individuals who desire competitive sports participation require pediatric or adult congenital cardiology involvement and close longitudinal surveillance. Individuals with dextro-transposition of the great arteries with arterial switch are at risk for neo-aortic root dilation or regurgitation, ventricular dysfunction, and coronary obstruction. Imaging of the coronary arteries and aorta and exercise testing are necessary before competitive sports participation. Patients with a systemic RV with biventricular circulation are at risk for tricuspid regurgitation, complex tachyarrhythmias, complete heart block, and ventricular dysfunction. After an atrial switch, patients are at increased risk for atrial arrhythmias, VAs, and baffle obstruction. However, this primarily occurs among older patients.¹⁶⁰

Patients who have undergone a Fontan procedure represent a heterogeneous group that may include competitive athletes.¹⁶¹⁻¹⁶³ Limited data are available on the safety of competitive sports participation. Patients

TABLE 9 Clinical Considerations for Competitive Athletes With Coronary Artery Anomalies**Anomalous aortic origin of a coronary artery**

Right AAOCA (interarterial)

Specific clinical considerations

Competitive athletes with interarterial right AAOCA should be assessed for symptoms suggestive of myocardial ischemia and inducible myocardial ischemia with provocative stress testing.*†‡§

It is reasonable to temporarily withhold or limit competitive sports participation during the initial clinical evaluation of interarterial right AAOCA.

Competitive athletes with interarterial right AAOCA should be considered for surgical intervention if there is evidence of inducible myocardial ischemia by testing or symptoms suggestive of ischemia.§

For competitive athletes with interarterial right AAOCA and no symptoms suggestive of myocardial ischemia and no evidence of inducible myocardial ischemia or complex ventricular arrhythmias, competitive sports participation is reasonable with SDM and longitudinal clinical surveillance.

For competitive athletes with interarterial right AAOCA who undergo surgical repair, resumption of competitive sports participation can proceed after complete sternal healing and testing showing no evidence of myocardial ischemia and no complex ventricular arrhythmias.*

Left AAOCA (interarterial)

Specific clinical considerations

Competitive athletes with interarterial left AAOCA should be considered for surgical intervention of this high-risk anatomic variant regardless of the initial clinical presentation or the results of an ischemia assessment. Competitive athletes should not participate in competitive sports if left unrepaired.

At the time of diagnosis, it is reasonable to assess competitive athletes with interarterial left AAOCA for the presence of myocardial fibrosis or scar to inform perioperative management and long-term prognosis.

For competitive athletes with interarterial left AAOCA who undergo surgical repair, resumption of competitive sports participation can proceed after complete sternal healing and testing showing no evidence of myocardial ischemia and no complex ventricular arrhythmias.*

Left AAOCA (intraseptal, noncoronary sinus)

Specific clinical considerations

Competitive athletes with generally benign left AAOCA variants should be assessed for symptoms suggestive of myocardial ischemia and inducible myocardial ischemia with provocative stress testing.*†‡§

Competitive athletes with generally benign left AAOCA variants can participate in competitive sports in the absence of symptoms suggestive of myocardial ischemia, no inducible myocardial ischemia, and no complex ventricular arrhythmias.

For competitive athletes with noninterarterial left AAOCA variants who undergo surgical repair, resumption of competitive sports participation can proceed after complete sternal healing and testing showing no evidence of myocardial ischemia and no complex ventricular arrhythmias. If medical management is used, competitive sports participation can be considered with resolution of symptoms and normal provocative stress testing.*

Other congenital coronary artery anomalies

Competitive athletes with other benign coronary artery anomaly subtypes that are not associated with myocardial ischemia (including left AAOCA with a prepulmonic or retroaortic course, anomalous circumflex from the right aortic sinus or from the right coronary artery with retroaortic course) can participate in competitive sports.

Anomalous origin of a coronary artery from the pulmonary artery

Specific clinical considerations

With the exception of lower-intensity strength and endurance sports (see [Section I, Figure 1](#)), competitive athletes should not participate in competitive sports until surgical repair of anomalous origin of a coronary artery from the pulmonary artery, which is indicated regardless of symptoms, results of ischemic evaluation, LV function, or the presence of ischemic mitral regurgitation.

For competitive athletes with anomalous origin of a coronary artery from the pulmonary artery who undergo surgical repair, resumption of competitive sports participation should be individualized after complete sternal healing and testing showing no evidence of myocardial ischemia, no complex ventricular arrhythmias, and normal LV systolic function. Additional considerations include:

- Residual postoperative mitral regurgitation: refer to [Section V](#)
- Delayed enhancement on cardiac magnetic resonance imaging (suggestive of previous infarction) and no more than mild LV dysfunction (not consistent with exercise-induced cardiac remodeling): competitive sports participation can be considered with SDM

Coronary artery atresia (congenital or acquired chronic total occlusion)

Specific clinical considerations

Competitive athletes with coronary artery atresia should be assessed for symptoms suggestive of myocardial ischemia and inducible myocardial ischemia with provocative stress testing.*†‡§

For competitive athletes with coronary artery atresia who undergo surgical revascularization, resumption of competitive sports participation can proceed after complete sternal healing and testing showing no evidence of myocardial ischemia and no complex ventricular arrhythmias. If medical management is used, competitive sports participation can be considered with resolution of symptoms and normal provocative stress testing.*

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with unrepaired shunt lesions and right-left shunting at rest (Eisenmenger physiology) have fixed elevated pulmonary vascular resistance, in which the risks of competitive sports participation outweigh benefits.¹⁶⁴

Coronary Artery Anomalies

Coronary artery anomalies are a leading cause of SCA or SCD in competitive athletes, most commonly from anomalous aortic origin of the left coronary artery with an

TABLE 9 Continued**Myocardial bridging**

Specific clinical considerations

Asymptomatic competitive athletes with an incidental diagnosis of myocardial bridging can participate in competitive sports.

Symptomatic competitive athletes with myocardial bridging should undergo assessment for inducible myocardial ischemia.*†§

For competitive athletes with myocardial bridging who have inducible myocardial ischemia or symptoms suggestive of myocardial ischemia,§ treatment options, either medical or surgical, should be considered before participation in competitive sports. The risks likely outweigh the benefits of competitive sports participation for competitive athletes with myocardial bridging and evidence of persistent or residual myocardial ischemia.§

For competitive athletes with myocardial bridging who undergo surgical repair, resumption of competitive sports participation can proceed after complete sternal healing and testing showing no evidence of myocardial ischemia and no complex ventricular arrhythmias.* If medical management is used, competitive sports participation can be considered with resolution of symptoms and normal provocative stress testing.*

*May include maximum-effort stress electrocardiography, cardiopulmonary exercise stress testing, stress echocardiography, stress nuclear perfusion imaging, or stress magnetic resonance perfusion imaging. Vasodilator stress testing is not recommended.

†The usefulness of invasive fractional flow reserve or instantaneous wave-free ratio testing with provocative stress is not yet determined; limited data suggest it may be useful in certain cases when overseen by experienced operators.

‡Cardiac magnetic resonance imaging, single-photon emission computed tomography, and ¹³n-ammonia positron emission tomography may be considered.

§Concerning cardiovascular symptoms include exertional syncope not explained by a vasovagal event or dehydration, exertional angina or chest pain, dizziness, palpitations, or syncope with accompanying diagnostic biomarker or test abnormalities.

AAOCA indicates anomalous aortic origin of a coronary artery; LV, left ventricular; and SDM, shared decision-making.

interarterial course.^{20,59,152} Affected individuals may be asymptomatic before an SCA or SCD event. For non-SCA presentations of anomalous aortic origin of the left coronary artery with an interarterial course, risk stratification includes assessment for inducible myocardial ischemia and fibrosis for perioperative management and long-term prognosis. Given this high-risk anatomy, surgical intervention should be performed before competitive sports participation. For competitive athletes with interarterial anomalous right coronary artery, competitive sports participation is reasonable if there are no exertional cardiopulmonary symptoms or inducible myocardial ischemia. Surgical intervention should be considered before participation in competitive sports if there is evidence of ischemia or concerning symptoms.¹⁶⁵⁻¹⁶⁷ Proposed high-risk coronary phenotypes include intramural course, high take-off, or slit-like proximal orifice in interarterial anomalous aortic origin of the left coronary artery or longer intramural length in interarterial anomalous right coronary artery.^{20,168} Whereas many patients with these features may not experience symptoms or SCA or SCD, there are ongoing research efforts to determine which phenotypes correlate with clinical outcomes.^{168,169} Other anomalous aortic origin of the left coronary artery subtypes, such as intraseptal and noncoronary sinus origin, are generally well-tolerated, thus competitive sports participation can likely continue. Rarely, they can present with SCA or myocardial ischemia; therefore, risk stratification should be performed.^{169,170}

Most patients with anomalous origin of a coronary artery from the pulmonary artery present with heart failure >1 year of age and undergo surgical repair. Presentations in later childhood or adulthood are rare.^{171,172} After repair, competitive sports participation is reasonable after complete sternal healing and with a

normal ischemic evaluation, normal LV function, and no inducible VA.^{173,174}

Myocardial bridging is generally incidental and clinically insignificant and requires no further risk stratification before competitive sports participation. However, if there are exertional cardiopulmonary symptoms, assessment of inducible ischemia is reasonable. In patients with inducible ischemia or with previous SCA deemed related to myocardial bridging, medical or surgical intervention is required. Risks of SCA likely outweigh the benefits of competitive sports participation in those individuals with persistent ischemia after medical therapy or surgery.^{169,175-178}

SECTION VII: AORTOPATHY (INCLUDING BICUSPID AORTIC VALVE) AND SPONTANEOUS CORONARY ARTERY DISSECTION

Acute aortic syndromes (AAS) that include aortic dissection or rupture represent a rare cause of SCD in competitive athletes and occur most frequently in heritable aortopathies.^{20,59,179} Optimal counseling of competitive athletes with an aortopathy includes SDM in considering the risk of AAS,¹⁸⁰ which varies on the basis of the underlying aortic condition, aortic size, and possibly sport type. Whether competitive sports participation accelerates disease progression is uncertain as data are limited and heterogeneous.¹⁸¹⁻¹⁸⁴ Evaluation requires diverse expertise that includes aortic, genetic, and sports cardiology specialists.

In this section, the approach to competitive athletes with aortopathy, BAV, heritable thoracic aortic disease (HTAD) or unexplained thoracic aortopathy, or spontaneous coronary artery dissection (SCAD) are detailed (Table 10). Masters athletes with aortic enlargement are discussed in Section X.

TABLE 10 Clinical Considerations for Competitive Athletes With Aortopathy, Bicuspid Aortic Valve, or Spontaneous Coronary Artery Dissection**Approach to the athlete with thoracic aortic dilation or disease**

General considerations

A comprehensive tomographic imaging evaluation of the entire thoracic aorta (and branch vessels, as indicated in certain HTAD) should be performed at least once in the evaluation of competitive athletes with thoracic aortic disease to assess for all sites of dilation as well as to screen for associated conditions (ie, coarctation of the aorta, branch vessel disease).

Clinical decision-making for competitive athletes with thoracic aortic disease should incorporate careful aortic measurements using standardized, guideline-recommended imaging techniques and normative values.

When comparing aortic measurements over time, images should be compared side by side rather than relying on previous imaging reports.

After initial diagnosis, the imaging follow-up intervals should be individualized to the specific aortic condition and the degree of aortic dilation. An initial 6- to 12-mo interval is recommended per established guidelines, with subsequent follow-up based on aortic size, underlying diagnosis, clinical features, and stability over time.

Multigenerational family history and genetic evaluation should be performed to evaluate for HTAD, which may influence risk stratification and management in competitive athletes with unexplained thoracic aortic dilation and any one of the following:

- Aortic dilation with z score ≥ 3
- Family history of thoracic aortic disease or dissection, cerebral or peripheral aneurysm, or unexplained sudden death at a relatively young age
- Other clinical features suggesting a heritable connective tissue disorder or other genetic pathogenesis

Unexplained aortic dilation is defined by a z score ≥ 3 and comprehensive multigenerational family history, imaging screening of parents, and genetic evaluation yields no substantial findings. However, competitive athletes with overt features of a connective tissue disorder may harbor a de novo genetic variant, and further evaluation with expert consultation should be considered.

Competitive athletes with aortic dilation, HTAD, or previous aortic repair should be counseled on the physiologic effects of different types of exercise (see [Section I](#)) while highlighting the benefits of regular recreational aerobic exercise.

Aortic size thresholds may require adjustment for male or female competitive athletes who are significantly taller or shorter than average (as well as in Turner syndrome). Normalizing for body size can be considered but data regarding size-adjusted normative aortic dimensions and risk are limited in competitive athletes.

Normative values for aortic dimensions differ between female and male athletes in line with differences in body size, with female athletes typically having smaller aortic diameters.

It is rare for young competitive male athletes with a tricuspid aortic valve to have aortic diameter >42 mm or for young competitive female athletes to have aortic diameter >40 mm. Unexplained aortic dilation of this magnitude may represent an aortopathy rather than a normal variant.

Existing data outlining the normal ranges of aortic size in competitive athletes predominantly focus on the population <25 y. Data are limited on athletes between 25 and 35 y with longer athletic careers, who may exhibit slightly higher normal ranges. Refer to [Section X](#) for aortic dilation in masters athletes (≥ 35 y).

BAV: aortopathy

Specific clinical considerations

The evaluation of competitive athletes with BAV should include assessment of additional features that may increase the risk for aortic dissection, including family history of aortic dissection, rapid aortic growth (≥ 3 mm/y), untreated substantial aortic coarctation, or features suggesting an underlying HTAD.

Competitive athletes with BAV (refer to BAV: valvular disease, below) and normal aortic dimensions can participate in competitive sports.

Competitive sports participation for competitive athletes with BAV and mild to moderate thoracic aortic dilation (40–44 mm) and no additional risk factors for aortic dissection (as defined above) can be considered with SDM. Risk stratification should consider the degree of dilation relative to age, sex, and body size.

For competitive athletes with BAV and moderate to severe aortic dilation (≥ 45 mm), the risks likely outweigh the benefits of competitive sports participation. However, competitive sports participation can be considered in select cases with SDM, consultation with experts in aortic disease or sports cardiology, and longitudinal clinical surveillance.

Competitive athletes with BAV and thoracic aortic aneurysm meeting surgical thresholds should not participate in competitive sports until surgical intervention (see [Surgical repair, below](#)).

BAV: valvular disease

For competitive athletes with BAV and AR or AS, refer to considerations for AR and AS in [Section V \(Table 7\)](#).

HTAD (syndromic and nonsyndromic, gene-positive and gene-negative)

Specific clinical considerations

HTAD comprises various conditions with highly variable risks of aortic dissection and extra-aortic and branch vessel complications. Competitive athletes with HTAD should be evaluated by experts in aortic disease or sports cardiology with condition-specific risk stratification.

Competitive sports participation for competitive athletes with HTADs whose aorta and branch vessels are normal in size can be considered with SDM, which should include the underlying condition and sport type. For such competitive athletes, the risks of competitive sports participation involving high-intensity strength physiology likely outweigh the benefits (see [Section I, Figure 1](#)).

The risks likely outweigh the benefits of competitive sports participation for competitive athletes with HTAD and aortic dilation or branch vessel disease, depending on the underlying condition and sport type.

Competitive athletes with HTAD and aortic diameters meeting surgical thresholds should not participate in competitive sports.

Unexplained thoracic aortic dilation without a known familial or heritable aortopathy

Specific clinical considerations

Competitive athletes with a tricuspid aortic valve and unexplained thoracic aortic dilation ≤ 42 mm can generally participate in competitive sports. Risk stratification should consider the degree of dilation relative to body size.

TABLE 10 Continued**Approach to the athlete with thoracic aortic dilation or disease**

Competitive sports participation for competitive athletes with unexplained mild to moderate thoracic aortic dilation (≥ 43 – 44 mm) can be considered with SDM, consultation with experts in aortic disease or sports cardiology, and longitudinal clinical surveillance.

The risks may outweigh the benefits of competitive sports participation for competitive athletes with unexplained moderate to severe thoracic aortic dilation (≥ 45 mm). Competitive sports participation can be considered in select cases with SDM, consultation with experts in aortic disease or sports cardiology, and longitudinal clinical surveillance.

Competitive athletes with unexplained thoracic aortic dilation meeting surgical thresholds should not participate in competitive sports until surgical intervention (see Surgical repair, below).

After aortic dissection and after surgical repair

Specific clinical considerations

Competitive athletes with previous aortic dissection should not participate in competitive sports.

Competitive sports participation for competitive athletes with BAV and aortopathy who have undergone ascending thoracic aortic aneurysm repair is reasonable after complete sternal healing and with SDM, consultation with experts in aortic disease or sports cardiology, and longitudinal clinical surveillance.

With the exception of lower-intensity strength and endurance sports (see Section I, Figure 1), competitive athletes with HTAD who have undergone aortic aneurysm repair should not participate in competitive sports.

The risks of competitive sports participation are uncertain for competitive athletes who have undergone aneurysm resection for unexplained thoracic aortic dilatation. Competitive sports participation can be considered in select cases after complete sternal healing and with SDM, consultation with experts in aortic disease or sports cardiology, and longitudinal clinical surveillance.

Spontaneous coronary artery dissection

Specific clinical considerations

Competitive athletes should participate in cardiac rehabilitation after recovery from a SCAD event.

Competitive athletes with SCAD should be counseled about the benefits of regular aerobic exercise after SCAD.

In competitive athletes with a previous SCAD event, the risks likely outweigh the benefits of competitive sports participation.

AR indicates aortic regurgitation; AS, aortic stenosis; BAV, bicuspid aortic valve; HTAD, heritable thoracic aortic disease; SCAD, spontaneous coronary artery dissection; and SDM, shared decision-making.

General Approach to Competitive Athletes With Thoracic Aortic Dilation or Disease**Exercise Physiology and Normative Data**

Understanding of sport-specific exercise hemodynamics is critical in considering the risk of AAS. Although limited data exist defining potential risks across different sports and physiologies,⁵⁶ physiologic stresses from endurance and strength-training impart different degrees of loading stress on the aorta, which must be considered during SDM with a competitive athlete with aortopathy.

Normative data defining aortic size among competitive athletes are limited, but uniformly demonstrate that substantial aortic enlargement (>42 mm [male athletes] and >40 mm [female athletes]) is rare among young competitive athletes,^{181,182,184,185} regardless of body size.²¹ The presence of marked enlargement requires assessment for an underlying aortopathy or connective tissue disorder.¹⁸⁶

Imaging Best Practices

Meticulous aortic measurements using guideline-recommended techniques and side-by-side comparison of previous images, rather than reliance on reports,^{7,186} is mandatory. The frequency of longitudinal surveillance imaging should be individualized on the basis of considerations of the aortic condition, degree of dilation, and sport type.

Among different imaging metrics,¹⁸⁶ the most robust outcomes data are based on absolute aortic dimensions.

Adhering to existing guidelines that recommend using absolute values to define an aneurysm and surgical thresholds, this framework is used for these clinical considerations (Table 10). Sex-specific criteria for aortic size are not included, although differences in aortic dimensions between men and women exist,^{187,188} primarily because of variations in body size. Z score-based criteria are not included given the lack of robust outcomes data associated with this metric. However, body size cannot be ignored, given its correlation with aortic size, and should be considered in the evaluation of competitive athletes with concern for aortopathy.

BAV With Aortopathy

Registry data indicate that the majority of AAS do not occur during exercise, although it is unknown how these data translate to competitive athletes.¹⁸⁹ In the general population, risk of AAS is significantly higher with aortic root or ascending aorta ≥ 50 mm compared with <45 mm.¹⁹⁰ BAV is commonly associated with aortopathy,¹⁹¹ yet the risk of dissection is low in competitive athletes, particularly without aneurysmal dilation.

With BAV, aortic regurgitation (as detailed in Section V) is generally well-tolerated and low risk for SCD. Understanding sport-specific exercise-induced cardiac remodeling⁷ is essential to differentiate from valve-related pathologic cardiac remodeling. BAV-associated severe aortic stenosis is high risk for exercise-related adverse events and SCA.

Heritable Thoracic Aortic Disease

Competitive athletes with HTAD (which can coexist with BAV), by contrast, are at higher risk versus the non-HTAD population, although considerable heterogeneity in risk exists by specific disease. Because individuals with HTAD have a more diffuse arteriopathy and are at higher aortic risk at relatively smaller aortic diameters (and may have other systemic features that inform risk), the risks likely outweigh the benefits of competitive sports participation.

Unexplained Thoracic Aneurysms

Among athletes with tricuspid aortic valves and without HTAD, data suggest up to 1% to 2% of competitive athletes have an aorta ≥ 40 mm, but rarely > 42 mm.^{21,181,185} Acknowledging these data and contemporary guidelines that define ≥ 45 mm as an ascending thoracic aortic aneurysm, in this scientific statement, 43 to 44 mm is considered mild to moderate aortic dilation, with corresponding increase in risk from baseline, and ≥ 45 mm unexplained aortic aneurysms are considered higher risk for AAS.¹⁸⁶

Post-Dissection and Surgical Repair

Competitive athletes with previous aortic dissection should not participate in competitive sports. Among competitive athletes who have undergone elective ascending aortic aneurysm repair, residual risk likely varies on the basis of the underlying cause of the aortic disease.

Spontaneous Coronary Artery Dissection

Given the association between SCAD and exercise and the substantial rate of recurrence (10%-20%),¹⁹²⁻¹⁹⁴ return to competitive sports participation after SCAD poses high risk. The benefits from cardiac rehabilitation after SCAD, however, are clear,¹⁹⁴ and all competitive athletes should participate in rehabilitation after SCAD.

SECTION VIII: ARRHYTHMIAS, DEVICES, AND ECG ABNORMALITIES

This section outlines clinical considerations for competitive athletes with arrhythmias (Table 11). Refer to the 2024 Heart Rhythm Society Expert Consensus Statement on Arrhythmias in the Athlete for further discussion on the diagnosis and management of arrhythmias in competitive athletes.³⁸

Ventricular Arrhythmias

VAs consist of premature ventricular contractions, non-sustained ventricular tachycardia and sustained ventricular tachycardia, and ventricular fibrillation. The most common VA observed in athletes are premature ventricular contractions, with a prevalence similar to sedentary

counterparts.¹⁹⁵ Although the majority of premature ventricular contractions are benign, further evaluation guided by morphology, clinical, and electrical features is warranted.¹⁹⁶ Athletes with VA should undergo ECG, ambulatory rhythm monitoring, exercise stress testing, and echocardiography as part of an initial evaluation.¹⁹⁷ After risk stratification, VA can be classified as follows (Table 12): benign VA without structural heart disease or high-risk VA with or without structural heart disease. For high-risk VA, return to competitive sports participation should be based on the underlying diagnosis (eg, cardiomyopathy, genetic arrhythmia syndrome, myocarditis), documented efficacy of arrhythmia treatment, and an SDM approach. For athletes who survive SCA, competitive sports participation is reasonable with SDM in the context of the underlying pathogenesis, implemented treatment, and documented rhythm stability.

Implantable Cardiac Defibrillators

The safety of competitive sports participation for athletes with an ICD has been demonstrated in several prospective observational studies.^{9,28,198} Appropriate and inappropriate shocks may occur, but adverse event rates are low, with no deaths, arrests, or injury reported.^{9,28,198} Additional considerations include risks associated with the underlying pathogenesis, estimated risk of rhythm instability, and the potential risk of device damage on the basis of sport type (see Section I). These discussions necessitate SDM, careful EAP, and longitudinal care with a cardiologist experienced with athletes and ICD.

AF and Supraventricular Tachycardia

Competitive athletes with AF require a comprehensive evaluation. Masters athletes with AF are detailed in Section X. In young competitive athletes with AF, clinical tests should exclude the presence of an underlying inherited arrhythmia syndrome, cardiomyopathy, or other supraventricular tachycardia. This assessment should include ECG, echocardiography, and CMR if indicated. Performance-enhancing agents should be queried, although their effect on AF risk remains unclear.¹⁹⁹ Competitive sports participation generally depends on symptom severity and effects on exercise tolerance or performance. AF and most supraventricular tachycardias are not considered SCA risks. Ablation may be considered as first-line therapy for both symptomatic AF and supraventricular tachycardia given the positive outcomes and low risk of complications.²⁰⁰⁻²⁰²

Wolff-Parkinson-White Syndrome

For competitive athletes with Wolff-Parkinson-White (WPW) syndrome (with ECG preexcitation and symptoms), consultation with an electrophysiologist is required for consideration of ablation. WPW pattern

TABLE 11 Clinical Considerations for Competitive Athletes With Arrhythmias, Implantable Cardioverter Defibrillators or Pacemakers, Syncope, and Asymptomatic Abnormal Electrocardiographic Interpretation**General considerations**

Competitive athletes with incidentally detected or likely low-risk arrhythmias (isolated PVCs, AF, or SVT) can continue competitive sports participation during the subsequent clinical evaluation.

For competitive athletes with potentially high-risk arrhythmias as indicated by complex morphology PVCs, VT, family history of SCD or arrhythmogenic disorders, or symptoms of exertional palpitations, syncope, or exercise intolerance, the risks likely outweigh the benefits of competitive sports participation during the subsequent clinical evaluation.

For competitive athletes who undergo pharmacologic suppression or catheter-based intervention for the treatment of potentially high-risk arrhythmias, maximal-effort exercise testing and ambulatory rhythm monitoring should be performed to document therapeutic efficacy. For all catheter-based procedures, exercise training should not resume until vascular access site healing occurs, generally 7 to 14 d after the procedure (for AF and pulmonary vein isolation, see [Section X](#)).

Ventricular arrhythmias**Benign PVCs**

Specific clinical considerations

Competitive athletes with benign PVCs and an unremarkable clinical evaluation can participate in competitive sports.

Competitive athletes with benign symptomatic PVCs who undergo catheter ablation can return to competitive sports participation after vascular access site healing.

Higher-risk ventricular arrhythmias: includes complex PVCs and monomorphic VT

Specific clinical considerations

The risks may outweigh the benefits of competitive sports participation for competitive athletes with ventricular arrhythmias and high-risk features that include an underlying cardiomyopathy, genetic or arrhythmic syndromes, or myocarditis. However, competitive sports participation can be considered with SDM and based on the underlying diagnosis, treatment, efficacy of arrhythmia suppression, and longitudinal clinical surveillance.

Malignant VT/VF and previous sudden cardiac arrest

Specific clinical considerations

Resumption of competitive sports participation for competitive athletes with a reversible cause of malignant VT or ventricular VF, such as resolved myocarditis, successful ablation of monomorphic PVCs that induced VF, or electrolyte abnormalities, is reasonable after confirmation of successful treatment or resolution of the underlying disease process.

Resumption of competitive sports participation for competitive athletes who have survived sudden cardiac arrest is reasonable with SDM, which takes into consideration the underlying diagnosis, appropriate therapeutic interventions, and comprehensive confirmation of rhythm stability with maximum-effort, sport-specific exercise testing and extended duration ambulatory rhythm monitoring.

Implantable cardioverter defibrillator

Specific clinical considerations

Competitive sports participation is reasonable for competitive athletes who have received an ICD for primary or secondary prevention with SDM, which takes into consideration the underlying diagnosis, comprehensive confirmation of rhythm stability, and the possibility of both appropriate and inappropriate device therapies.

For competitive athletes who receive a new ICD, competitive sports participation should be restricted for 4 to 8 wk (or 2 wk after generator replacement) as determined by sporting discipline to allow for postprocedural recovery.

Competitive sports participation for competitive athletes with an ICD who participate in collision or impact competitive sports (see [Section I](#)) can be considered with SDM that addresses the potential risk of ICD system damage or malfunction.

SVT and atrial fibrillation

Specific clinical considerations

Young competitive athletes with AF should undergo a comprehensive clinical evaluation for potential explanatory underlying causes with a 12-lead ECG, echocardiography, and cardiac magnetic resonance imaging (if indicated).

Anticoagulation for young competitive athletes with AF should be guided by standard risk algorithms.

In competitive athletes who require anticoagulant therapy for AF, the risks of bleeding likely outweigh the benefits for some competitive sports involving collisions or impacts (see [Section I](#)).

Young competitive athletes with AF or SVT with no underlying structural heart disease can participate in competitive sports as neither arrhythmia has been shown to be associated with SCD. Treatments with rhythm maintenance strategies, which include atrioventricular nodal agents, antiarrhythmic drugs, or ablation, should be guided by SDM taking into account the arrhythmia symptom burden, antidoping regulations based on sport and competitive rules (and potential need for therapeutic use exemption), and the effects of treatment on quality of life, which may include the effects on competitive sports performance.

Wolff-Parkinson-White pattern

Specific clinical considerations

Competitive athletes with WPW pattern on ECG should undergo a cardiac evaluation including physical examination, personal and family history, and echocardiogram.

Competitive athletes with WPW syndrome, which includes symptoms suggestive of arrhythmias or documented arrhythmias, should be evaluated by an electrophysiologist to discuss treatment options to reduce the risk of life-threatening events.

Asymptomatic competitive athletes with WPW pattern should be seen in consultation with sports cardiology and electrophysiology to discuss the options of noninvasive (exercise stress testing) or invasive risk stratification (electrophysiology study with or without catheter ablation) with SDM, which includes consideration of the age of the competitive athlete and the risks, benefits, and limitations of each modality.

Competitive athletes with asymptomatic WPW pattern should not be restricted from competitive sports participation during the clinical evaluation process.

After WPW ablation, competitive sports participation can resume after vascular access site healing. Resumption of competitive sports participation should also take into consideration risk assessment of the pathway and success of the procedure.

TABLE 11 Continued**Athletes with syncope**

Specific clinical considerations

Competitive sports participation for competitive athletes with exertional syncope, who have had a complete evaluation as recommended by other guidelines and without concerning findings, is reasonable. Long-term ambulatory rhythm monitoring (ie, implantable loop recorder) during competitive sports participation is also reasonable if clinical uncertainty remains.

Competitive athletes with nonexertional syncope, whose history, physical examination, and resting 12-lead ECG results support a diagnosis of neurally mediated syncope or postexertional collapse, can return to competitive sports participation without further evaluation.

Athletes with abnormal ECG results

Specific clinical considerations

ECGs obtained on competitive athletes should be interpreted using current consensus recommendations. The clinical evaluation of an abnormal ECG result should be tailored to address the specific cardiovascular pathology suggested by the ECG abnormality.

Competitive sports participation during the clinical evaluation of an abnormal screening ECG obtained on an asymptomatic competitive athlete should depend on the perceived level of risk, which considers other historical features (family history), physical examination findings, the specific ECG abnormality or abnormalities present, and the evidence supporting the SCD risk associated with the specific cardiovascular disease suggested by the ECG abnormality.

Competitive athletes with an abnormal ECG in whom the clinical evaluation does not show evidence of cardiac disease should be followed longitudinally.

Asymptomatic competitive athletes with substantial ECG abnormalities (including specific T-wave inversion patterns [anterolateral, inferolateral, anterior, and lateral], diffuse ST-segment depressions, pathologic Q waves, and complete left bundle branch block) but normal initial clinical evaluation results should be followed with longitudinal clinical surveillance (includes imaging).

Athletes with bradycardia and pacemakers

Specific clinical considerations

Asymptomatic sinus bradycardia (≥ 30 beats/min while awake) and atrioventricular node slowing (first-degree and Type 1 second-degree atrioventricular block) are normal adaptations to exercise and do not require clinical evaluation. Symptomatic bradycardia or marked sinus bradycardia (< 30 beats/min while awake) and distal conduction disease beyond isolated right bundle branch block or isolated hemiblock require clinical evaluation before participation in competitive sports.

Competitive sports participation for competitive athletes with a pacemaker, who are not pacemaker-dependent, is reasonable.

For competitive athletes who are pacemaker-dependent, participation in collision or impact competitive sports (see Section I) can be considered with SDM, which includes the underlying condition and the absence of data on risk.

AF indicates atrial fibrillation; ICD, implantable cardioverter defibrillator; PVC, premature ventricular contraction; SCD, sudden cardiac death; SDM, shared decision-making; SVT, supraventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia; and WPW, Wolff-Parkinson-White.

requires a complete history, physical, and imaging with echocardiography. A careful history is required for adolescent or younger competitive athletes, in whom symptom ascertainment may be unreliable. A slight increased risk of SCD has been described in children with WPW and a short refractory period (< 250 ms) as compared with adults with WPW.²⁰³⁻²⁰⁶ Increasing data, particularly in young individuals, show that noninvasive exercise testing lacks sensitivity and may not exclude the risk of rapidly conducted AF or SCA. Electrophysiologic study to identify high-risk pathways, and ablation of those pathways, may be beneficial.^{203,204,207-209} However, invasive versus noninvasive risk stratification for asymptomatic WPW pattern remains controversial among competitive athletes and requires SDM, particularly given the difference in SCD risk as a function of age. For athletes with WPW pattern, competitive sports participation does not require restriction during the time period of the initial clinical evaluation, as clinical events are rare and not demonstrated to be more frequent during exercise.^{203,210}

Syncope

Competitive athletes with syncope require a comprehensive history and physical, which includes interviewing witnesses and reviewing video, if available. Most cases are

benign, including neurally mediated, previously referred to as vasovagal, and after exertional syncope.²¹¹ Syncope that occurs during exercise and with high-risk features requires a complete evaluation for structural or electrical heart disease (including imaging, ambulatory rhythm monitoring, and maximal-effort exercise testing). High-risk features include unheralded collapse, abrupt palpitations, and exertional angina or excessive dyspnea. High-risk syncope warrants restriction from competitive sports participation until the clinical evaluation is completed.²¹²

Abnormal ECG Results

The International Criteria for ECG interpretation provide contemporary expert consensus recommendations on athletic ECG interpretation.¹⁴ For asymptomatic competitive athletes with abnormal ECG findings, temporary cessation of competitive sports participation during the subsequent evaluation should be considered, based on the specific abnormality or abnormalities identified and additional findings obtained by history or physical examination.

Bradycardia and Pacemakers

Sinus bradycardia, first-degree atrioventricular block, and Mobitz I (Wenckebach) block are normal in competitive

TABLE 12 Distinguishing Between Low- and High-Risk Ventricular Arrhythmias

Low-risk features	High-risk features
Clinical characteristics	
Asymptomatic	Presyncope, syncope, dyspnea, or sudden-onset exercise intolerance
Infrequent palpitations	Sustained or frequent rapid palpitations
No history suggestive of inherited heart disease	Family history of collapse, syncope, sudden cardiac death, or cardiomyopathy
Electrophysiologic characteristics	
Morphology consistent with: <ul style="list-style-type: none"> ■ Outflow tract: LBBB pattern, inferior axis, transition $<V_4$ favors RVOT and $\geq V_4$ favors LVOT ■ Fascicular morphology: RBBB pattern, QRS duration typically <130 ms, inferior axis (anterior fascicle), superior axis (posterior fascicle) 	Morphology consistent with: <ul style="list-style-type: none"> ■ RV free wall or moderator band: LBBB with intermediate or superior axis ■ LV cardiomyopathy: RBBB with wide QRS (>130 ms) ■ Papillary muscles: RBBB, QRS >130 ms, mid-precordial transition, inferior axis (anterolateral papillary muscle), superior axis (posteromedial papillary muscle)
Monomorphic PVCs or short runs of NSVT at subphysiologic maximum heart rate	Polymorphic, repetitive, short coupling interval (<360 ms)
Normal ECG	Abnormal ECG (other than PVCs) includes low voltages
Low-burden PVCs	High-burden PVCs (>8000 in 24 h)
Exercise testing findings	
Suppression of PVCs with exercise	Nonsuppression, new emergence of PVCs with exercise, or increased burden of PVCs during exercise
No symptoms and normal hemodynamics	Symptoms of sudden-onset exercise intolerance associated with emergence of arrhythmias
Echocardiographic findings	
Normal cardiac structure and function for an athlete (includes exercise-induced cardiac remodeling)	Pathologic wall thickness or ventricular dilation, wall segmental abnormalities; reduced LV systolic function
Clear augmentation of biventricular function with exercise	Reduced contractile reserve (LV or RV)
Cardiac magnetic resonance imaging findings	
Normal cardiac structure and function for an athlete (includes exercise-induced cardiac remodeling)	Pathologic wall thickness or ventricular dilation, wall segmental abnormalities
No evidence of postcontrast enhancement	Late gadolinium enhancement, particularly mid-wall or epicardial enhancement
Invasive electrophysiologic characteristics	
Focal arrhythmogenic site	Multiple inducible arrhythmias
Catecholamine triggering of focal site	Catecholamine triggering of rapid polymorphic arrhythmias
Normal electroanatomic mapping	Low-voltage regions (noting a tendency to epicardial pathology in endurance athletes)

Adapted with permission from Lampert et al.³⁸ Copyright © 2024 Heart Rhythm Society.

LBBB indicates right bundle branch block; LV, left ventricle; LVOT, left ventricular outflow tract; NSVT, nonsustained ventricular tachycardia; PVC, premature ventricular contraction; RBBB, right bundle branch block; RV, right ventricle; and RVOT, right ventricular outflow tract.

athletes. Athletes with marked conduction abnormalities require further clinical evaluation. There are no data on competitive athletes after pacemaker implantation. Consideration of competitive sports participation should take into account pacemaker dependency and the possibility of system damage through collisions or impacts (Section I).

SECTION IX: CARDIAC CHANNELOPATHIES

In this section, LQTS, CPVT, and Brugada syndrome are detailed (Table 13). Because of the complexity of these conditions, comprehensive assessment, treatment, and follow-up by a specialist with expertise in genetic heart disease is critical when considering competitive sports participation, and is associated with a low event rate.^{10,78,82,213} Recent data have demonstrated low rates of breakthrough cardiac events in competitive athletes with cardiac channelopathies after diagnosis and initiation of appropriate genotype- and phenotype-specific management plans.^{10,11,18,27,198,214,215} For all cardiac channelopathies, ICD implant for the sole purpose of competitive sports participation should not be performed because of a $\approx 5\%$ per year risk of inappropriate shocks and $\approx 4\%$ per year risk of ICD-related complications.²¹⁶

Long QT Syndrome

Competitive sports participation is reasonable for asymptomatic competitive athletes who are genotype-positive with a normal corrected QT interval at rest (ie, gene-positive, phenotype-negative, now referred to as concealed variant positive LQTS, with resting QTc <460 ms).^{11,38} This includes individuals with phenotypic penetrance confined to maladaptive QT reactivity during exercise or recovery in the setting of exercise testing. Overall, recent data show no deaths and a low event rate (0.3 nonlethal events per 100 patient-years of follow-up) among competitive athletes with concealed variant positive LQTS.¹⁹⁸ Nonetheless, these individuals should avoid exposures to known risk for QT prolongation and may be considered for prophylactic β -blocker therapy.^{217,218}

It is reasonable for competitive athletes with LQTS (symptomatic or asymptomatic, with resting QTc ≥ 460 ms before puberty, ≥ 470 ms in male patients, ≥ 480 ms in female patients) under specialized care to participate in competitive sports with SDM. Management includes nonselective long-acting β -blockers (eg, nadolol or propranolol), having an EAP with access to an AED, and may include mexiletine (especially LQT3). In some higher-risk competitive athletes, or for those who require therapy escalation, adjunctive therapies with left cardiac sympathetic denervation or ICD may be considered.^{11,198,219} Data from the largest LQTS single-center cohort of 494 athletes showed no deaths and a low event rate (1.16 nonlethal

TABLE 13 Clinical Considerations for Competitive Athletes With Cardiac Channelopathies**General considerations**

Competitive athletes with a cardiac channelopathy (including LQTS, CPVT, and BrS) should be assessed by a pediatric or adult cardiologist with expertise in cardiac channelopathies and with SDM.

In competitive athletes diagnosed with a cardiac channelopathy, an ICD should not be implanted for the sole purpose of competitive sports participation.

Long QT syndrome

Specific clinical considerations

It is reasonable for competitive athletes with positive genetic test results for LQTS but who have a resting QTc <460 ms (ie, concealed variant positive LQTS) to participate in competitive sports.

In competitive athletes with LQTS (asymptomatic [QTc \geq 460 ms prepuberty, \geq 470 male, \geq 480 female] or previously symptomatic) but who are under expert assessment and supervision, competitive sports participation is reasonable with SDM after risk assessment, education, and implementation of guideline-directed therapies.

In competitive athletes with LQTS (including LQT1), competitive swimming and diving can be considered with appropriate precautions.*

Catecholaminergic polymorphic ventricular tachycardia

Specific clinical considerations

In an asymptomatic competitive athlete with positive genetic test results for CPVT but no exercise-induced ventricular ectopy on exercise stress testing (ie, genotype-positive and phenotype-negative), competitive sports participation is reasonable with discussion about prophylactic CPVT-directed medical therapy.

In competitive athletes with asymptomatic CPVT who have a positive stress test with evidence of exercise-induced ventricular ectopy, competitive sports participation can be considered with SDM and after optimization of therapies and normalization of the stress test.†

In competitive athletes with previously symptomatic CPVT for whom competitive sports participation are being considered, combination therapy with β -blocker and flecainide, and possibly the addition of LCSD, is required before resumption of competitive sports participation. Such CPVT therapies should be optimized with normalization of the stress test before participation in competitive sports.†

Brugada syndrome

Specific clinical considerations

It is reasonable for competitive athletes with BrS to participate in competitive sports after expert assessment and management.

*Precautions include swimming with supervision by an individual who is trained in cardiopulmonary resuscitation, with preference for pools rather than open water, and access to an automatic external defibrillator.

†Normalization of the stress test ideally encompasses absence of ectopy; bigeminal premature ventricular contractions may be acceptable but ventricular couplets or more extensive nonsustained ventricular tachycardia require continued treatment intensification.

BrS includes Brugada syndrome; CPVT, catecholaminergic polymorphic ventricular tachycardia; ICD, implantable cardioverter defibrillator; LCSD, left cardiac sympathetic denervation; LQTS, long QT syndrome; and SDM, shared decision-making.

events per 100 athlete-years of follow-up).^{10,11} In competitive athletes with symptoms or who require therapy escalation, there must be clinical stability with no breakthrough arrhythmias for at least 3 months before resumption of competitive sports participation. There is limited evidence that swimming is a genotype-specific trigger for VA in patients with LQT1. Previous reported cases were mostly among those previously undiagnosed and untreated.^{220,221} Additional considerations for

competitive swimmers and divers with LQT1 who wish to compete include AED availability and avoidance of training alone or in open water.

Catecholaminergic Polymorphic Ventricular Tachycardia

All competitive athletes with genotype-positive CPVT should undergo maximum-effort exercise stress testing that includes burst efforts in the protocol.²²² With absence of exercise-induced ventricular ectopy or arrhythmias, or family history of SCD, competitive sports participation can continue with discussion of prophylactic therapies, including nonselective β -blockers and flecainide.²⁷ Longitudinal and serial exercise stress testing is recommended 1 or 2 times per year. With conversion to inducible ventricular ectopy or arrhythmias, CPVT-directed therapies should be initiated or further optimized.²²³

For asymptomatic competitive athletes with CPVT with exercise-induced ventricular ectopy or arrhythmias, prophylactic therapies including β -blockers and flecainide should be initiated. With adequate suppression, competitive sports participation can be considered with SDM.^{11,27} Adequate suppression ideally involves absence of exercise-induced ventricular ectopy; in some cases, bigeminal premature ventricular contractions may be acceptable, but couplets or nonsustained ventricular tachycardia require continued treatment intensification and ongoing exclusion from competitive sports participation during treatment optimization.

Symptomatic competitive athletes with CPVT are a higher-risk group with limited data. Expert consensus is that these athletes require dual medical therapy with β -blockers and flecainide and may also require left cardiac sympathetic denervation before consideration of competitive sports participation in an SDM model.^{11,27} These individuals need to be monitored closely and reassessed longitudinally with burst exercise stress testing every 6 to 12 months.^{222,223}

Brugada Syndrome

There are no data to support competitive sports participation restrictions with Brugada syndrome. These individuals should avoid known arrhythmic triggers, including heat exhaustion and exercise during febrile illnesses, and hydration during exercise should be prioritized.^{224,225}

SECTION X: MASTERS ATHLETES

Masters athletes are defined as people \geq 35 years of age who place a high premium on competitive sports participation. As moderate levels of habitual exercise are encouraged for all,²²⁶ these clinical considerations are not applicable to

TABLE 14 Clinical Considerations for Masters Athletes With Coronary Artery Disease, Atrial Fibrillation, Myocardial Fibrosis, Thoracic Aortic Dilation or Aneurysm, and Chronic Valvular Heart Disease**Coronary artery disease**

General considerations

High cardiorespiratory fitness and regular exercise reduce the overall risk of cardiovascular disease and death among healthy individuals and those with established cardiovascular risk factors or clinical coronary heart disease. However, vigorous exercise is associated with a transient increase in acute cardiac events in those with underlying cardiovascular disease.

Cardiovascular risk scores derived from the general population have not been validated in masters athletes. These scores, which do not include habitual physical activity levels, may overestimate risk when applied to masters athletes.

Subclinical CAD (includes CAC)

Specific clinical considerations

Although CAC may be observed commonly in masters athletes, its presence likely portends lower cardiovascular risk compared with sedentary individuals in the general population with similar levels of CAC.

Low-risk* masters athletes should not undergo routine cardiac risk stratification testing, including imaging for CAC.

Presumed intermediate* and high-risk* masters athletes should be counseled on appropriate guideline-based lifestyle modifications (ie, smoking cessation, diet, alcohol), treated according to guideline-based medical therapy, and counseled on symptoms that may indicate underlying ischemic heart disease.

Clinicians should consider further risk stratification with options including CAC, maximal-effort exercise stress testing, functional stress imaging (with maximal-effort exercise), or imaging (coronary CT angiography), for presumed intermediate* and high-risk* masters athletes.

Chronic stable CAD†

Specific clinical considerations

The benefits of competitive sports participation likely outweigh risks for asymptomatic masters athletes with chronic stable CAD and if all of the following criteria are met:

- Normal LV systolic function
- Absence of inducible ventricular arrhythmias
- No regional wall motion abnormalities
- No ischemic ECG changes or exertional symptoms during maximal-effort exercise stress testing

The risks likely outweigh the benefits of competitive sports participation for masters athletes with chronic stable CAD and any of the following criteria:

- Reduced LV systolic function inconsistent with exercise-induced cardiac remodeling (in this population generally <45% in the absence of LV dilation)
- The presence of inducible complex ventricular arrhythmias
- Regional wall motion abnormalities
- Ischemia as manifest by ECG changes or exertional symptoms during maximal-effort exercise stress testing
- The presence (including magnitude) of ischemic scar identified by CMR associated with any of the above risk factors

Among masters athletes with any of the above risk factors, limited participation in competitive sports can be considered with SDM using individualized intensity thresholds.

Revascularization, in addition to aggressive guideline-based lifestyle modifications and optimal medical therapy, can be considered with SDM for masters athletes with evidence of obstructive CAD associated with ischemia±symptoms (Figure 2).

Routine surveillance stress testing for asymptomatic masters athletes with stable CAD, who have incorporated appropriate lifestyle modifications and are compliant with guideline-based medical therapy, should not be performed.

Acute coronary syndrome

Specific clinical considerations

After an acute coronary syndrome, participation in a structured cardiac rehabilitation exercise training program (time and intensity can be accelerated on an individual basis) should be completed before participation in competitive sports.

It is reasonable for masters athletes, who have completed structured cardiac rehabilitation and are on optimal medical therapy, to resume competitive sports participation 3 to 6 mo after an acute coronary syndrome and if all of the following criteria met:

- Normal LV systolic function
- Absence of inducible complex ventricular arrhythmias
- No regional wall motion abnormalities
- No ischemic ECG changes or exertional symptoms during maximal-effort exercise stress testing

After an acute coronary syndrome and completion of a structured cardiac rehabilitation program, the risks likely outweigh the benefits of competitive sports participation for masters athletes with any of the following criteria:

- Reduced LV systolic function inconsistent with exercise-induced cardiac remodeling (in this population generally <45% in the absence of LV dilation or regional wall motion abnormalities)
- The presence of inducible complex ventricular arrhythmias
- Regional wall motion abnormalities
- Ischemia as manifest by ECG changes or exertional symptoms during maximal-effort exercise stress testing
- The presence (including magnitude) of ischemic scar identified by CMR associated with any of the above risk factors

Among masters athletes with any of the above risk factors, limited participation in competitive sports can be considered with SDM using individualized intensity thresholds.

Continued on the next page

TABLE 14 Continued**Atrial fibrillation**

Specific clinical considerations

Masters athletes with AF should be managed according to established guidelines. Risk factors (including hypertension, sleep apnea, and alcohol consumption) should be addressed and individuals counseled on the higher relative risk of AF associated with long-term and higher-intensity competitive endurance sports.

Masters athletes with AF can participate in competitive sports, as symptom-tolerated, during the clinical evaluation process.

Management options for symptomatic AF should be individualized with SDM to guide an AF rhythm control strategy of either PVI or antiarrhythmic drug therapy.

After PVI, the determination of return to competitive sports participation after the first 7 to 14 d postprocedure (needed for vascular access site healing) should proceed with SDM, acknowledging the uncertain, possible increased risk of AF recurrence associated with vigorous exercise and postprocedural left atrial inflammation (may be present up to 2 to 3 mo after PVI).

Myocardial fibrosis

Specific clinical considerations

Masters athletes with myocardial fibrosis identified by CMR should have appropriate risk stratification and treatments based on the underlying and specific disease process.

Asymptomatic masters athletes with incidentally discovered myocardial fibrosis at the insertion points of the RV into the intraventricular septum, and no evidence of underlying substantial pulmonary hypertension or pathologic RV dysfunction, can continue competitive sports participation.

Thoracic aortic dilation or aneurysm

Specific clinical considerations

All masters athletes with unexplained thoracic aortic dilation should undergo evaluation for an underlying aortopathy or heritable thoracic aortic disease (see [Section VI](#)).

All masters athletes with thoracic aortic dilation or aneurysms should undergo surveillance for hypertension and subsequent treatment as indicated.

All masters athletes with thoracic aortic dilation or aneurysms should have longitudinal surveillance imaging assessing for rate of aortic growth and absolute aortic dimensions.

Competitive endurance sports are reasonable for masters endurance athletes with unexplained thoracic aortic dilation of the aortic root or ascending aorta <45 mm.

Competitive endurance sports can be considered with SDM for masters endurance athletes with unexplained thoracic aortic aneurysms of the aortic root or ascending aorta between 45 and 49 mm.

Competitive strength sports for masters athletes with unexplained thoracic aortic dilation of the aortic root or ascending aorta or an unexplained aortic root or ascending thoracic aortic aneurysm <50 mm can be considered with SDM that includes potential alterations to strength training intensity and technique.

Masters athletes with unexplained thoracic aortic aneurysms of the aortic root or ascending aorta \geq 50 mm should not participate in competitive sports and surgical intervention should be considered with SDM.†

Resumption of competitive sports participation for masters athletes who have undergone surgical repair of an unexplained thoracic aortic aneurysm can be considered after complete sternal healing and with SDM.

Chronic mitral and aortic VHD

Specific clinical considerations

Masters athletes with asymptomatic VHD should not proceed to surgical intervention for the sole purpose of improving athletic performance.

For masters athletes with chronic VHD, including AR, AS, MR, or mitral stenosis, refer to these valvular lesions in [Section V](#).

After valve surgery and complete sternal healing, competitive sports participation for masters athletes with a well-functioning aortic or mitral bioprosthetic valve (or mitral valve repair) is reasonable. If there is residual valve disease present (stenosis or regurgitation), refer to [Section V](#). For masters athletes who are temporarily placed on anticoagulation therapy after valve surgery, the risks of some competitive sports involving collisions or impacts likely outweigh benefits (see [Section I](#)).

Antithrombotic therapy

Specific clinical considerations

For masters athletes who require dual antiplatelet therapy for any reason, such as after coronary revascularization or transcatheter valve replacement, or full oral anticoagulation for any reason, such as AF or aortic or mitral mechanical prosthetic valves, therapy should be based on established clinical guidelines. The risks of some competitive sports involving collisions or impacts likely outweigh benefits (see [Section I](#)).

Masters athletes who are initiated on oral anticoagulation and who choose to participate in competitive sports with higher risks of collisions or impacts (see [Section I](#)), temporary discontinuation of oral anticoagulation can be considered with SDM.

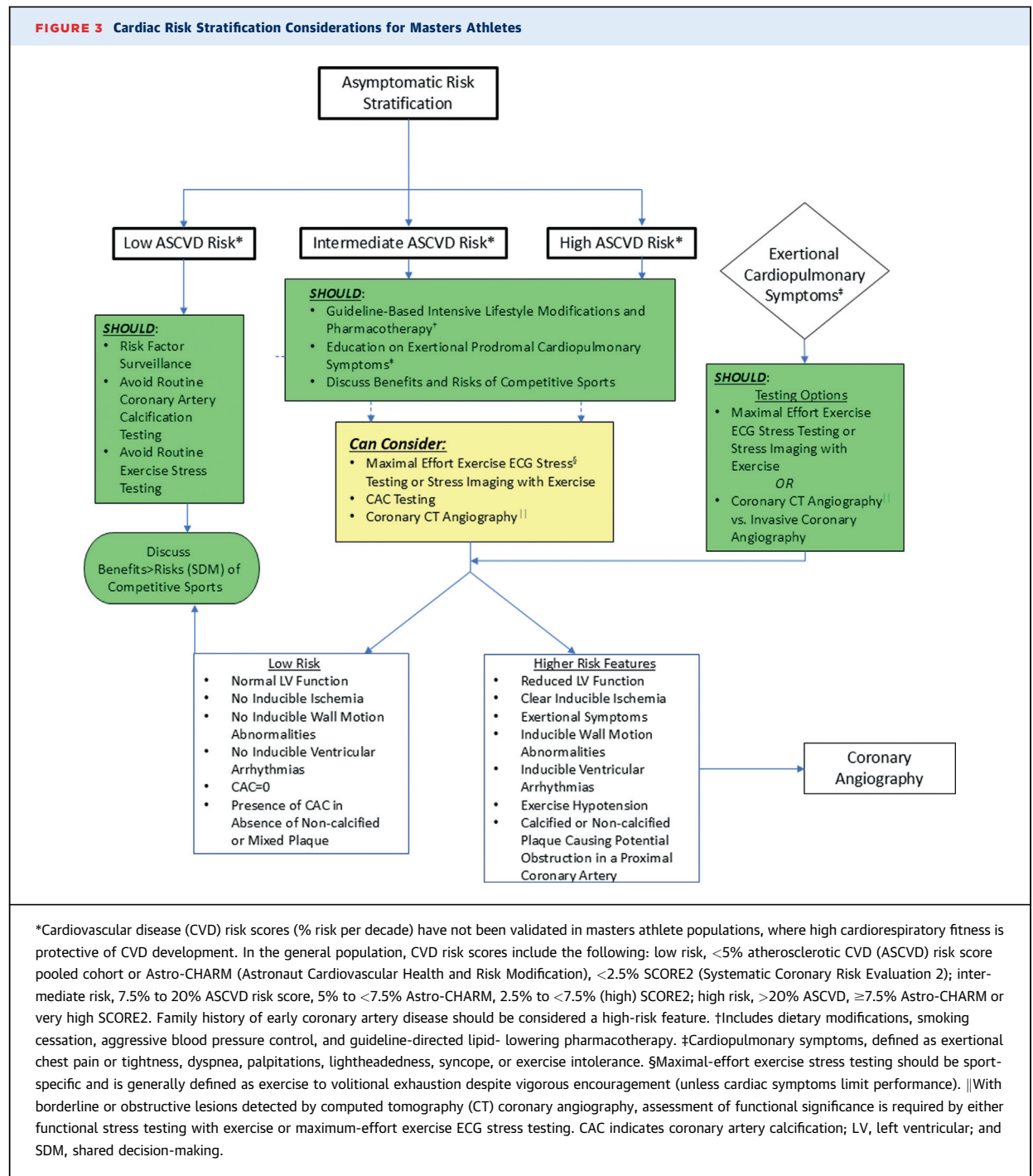
Masters athletes who require antiplatelet monotherapy can participate in all competitive sports.

*Cardiovascular disease risk scores (% risk per decade) have not been validated in masters athlete populations. In the general population, CVD risk scores include the following: low risk, <5% atherosclerotic CVD (ASCVD) risk score pooled cohort or Astro-CHARM (Astronaut Cardiovascular Health and Risk Modification), <2.5% SCORE2 (Systematic Coronary Risk Evaluation 2); intermediate risk, 7.5% to 20% ASCVD risk score, 5% to <7.5% Astro-CHARM, or 2.5% to <7.5% (high) SCORE2; high risk, >20% ASCVD, \geq 7.5% Astro-CHARM or very high SCORE2. Family history of early coronary artery disease is considered a high-risk feature.

†Chronic stable coronary artery disease (or chronic coronary syndrome) definition: stable coronary artery disease defined as per 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA Guideline for the Management of Patients With Chronic Coronary Disease⁹⁸; (1) after stable discharge of acute coronary syndrome or revascularization of stable disease, (2) established ischemic cardiomyopathy, (3) stable anginal symptoms on medical management, (4) coronary vasospasm/endothelial dysfunction, or (5) abnormal cardiovascular screening test suggesting coronary artery disease.

‡See also the 2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease¹⁸⁶ and integrate within the SDM process.

AF indicates atrial fibrillation; AR, aortic regurgitation; AS, aortic stenosis; CAC, coronary artery calcifications; CAD, coronary artery disease; CMR, cardiac magnetic resonance imaging; LV, left ventricular; MR, mitral regurgitation; PVI, pulmonary vein isolation; RV, right ventricle; SDM, shared decision-making; and VHD, valvular heart disease.



older individuals engaged in recreational physical activity. CAD, AF, myocardial fibrosis, aortic dilation or aneurysm, and chronic VHD are detailed (Table 14).

Coronary Artery Disease

Regular vigorous exercise decreases cardiovascular risk,²²⁷ but there is also transient increased risk of SCA

during vigorous exercise.²²⁸ Underlying CAD leading to plaque rupture or demand ischemia is the most common cause of SCA among masters athletes.^{31,228,229} Consideration of cardiac risk stratification for asymptomatic masters athletes is reasonable (Figure 3) with traditional risk scores,²³⁰⁻²³³ although scores have not been validated among masters athletes.

Low-risk, asymptomatic masters athletes should not undergo additional risk stratification, including coronary artery calcification assessment. Emerging data suggest coronary artery calcification may be commonly observed among long-term, mostly male, masters endurance athletes without traditional risk factors.^{33,34,234-237} However, underlying mechanisms are uncertain,²³⁸ and high cardiorespiratory fitness reduces cardiovascular risk compared with equivalent coronary artery calcification scores in more sedentary individuals.^{227,234}

Presumed intermediate or high-risk masters athletes require lifestyle modifications and guideline-based medical therapy. With SDM, further risk stratification should be considered, acknowledging the potential for false-positive stress ECG findings and other test limitations. Exercise testing may identify ischemia secondary to stable CAD, but poorly predicts exertional cardiac events caused by acute plaque rupture. If ischemia is present, besides counseling risk factor modification and optimal medical therapy, coronary angiography and potentially revascularization can be considered carefully with SDM. Pharmacologic vasodilation (fractional flow reserve) recorded during invasive angiography, which is based on changes in flow dynamics across a fixed lesion at rest, may not be equivalent to the substantial myocardial oxygen demand that occurs during maximum-effort exercise and lack of supply as a consequence of stable coronary disease. Thus, caution is advised in the interpretation of fractional flow reserve in masters athletes.²³⁹

For masters athletes with chronic stable CAD,⁸⁸ or who have recovered after acute coronary syndrome, resumption of competitive sports participation is similar (Table 14 and Figure 4) to previous recommendations.⁴⁹ Masters athletes with acute coronary syndrome should be prescribed standard lipid-lowering therapy and complete structured cardiac rehabilitation.^{37,241} Although the time frame for optimal plaque stabilization is uncertain,^{242,243} 3 to 6 months after acute coronary syndrome is reasonable for resumption of competitive sports participation. For masters athletes with stable CAD or after acute coronary syndrome with higher-risk features, risks of a recurrent cardiac event during competitive sports participation likely outweigh benefits and should be discussed during SDM.

Atrial Fibrillation

Long-term masters endurance athletes have a higher relative risk of AF compared with sedentary controls²⁴⁴⁻²⁴⁶; however, outcomes are better compared with sedentary individuals with AF.²⁴⁶⁻²⁴⁸ In considering competitive sports participation, AF does not increase risk of SCA, thus training can continue as tolerated throughout

the clinical evaluation. Stroke prevention strategies for masters athletes with AF should follow contemporary guidelines and other issues related to AF, including treatment options for competitive athletes, which are further detailed in the 2024 Heart Rhythm Society expert consensus statement on arrhythmias in the athlete.³⁸

Myocardial Fibrosis

Incidentally discovered myocardial fibrosis at the insertion points of the RV into the intraventricular septum can be observed in masters athletes,¹³⁷ possibly as a consequence of hemodynamic stresses from competitive sports participation.²⁴⁹ This phenotype is not known to represent clinically significant pathology.²⁵⁰ Myocardial fibrosis identified in other locations requires appropriate diagnostic evaluations.

Aortic Enlargement or Thoracic Aortic Root and Ascending Aorta Aneurysms

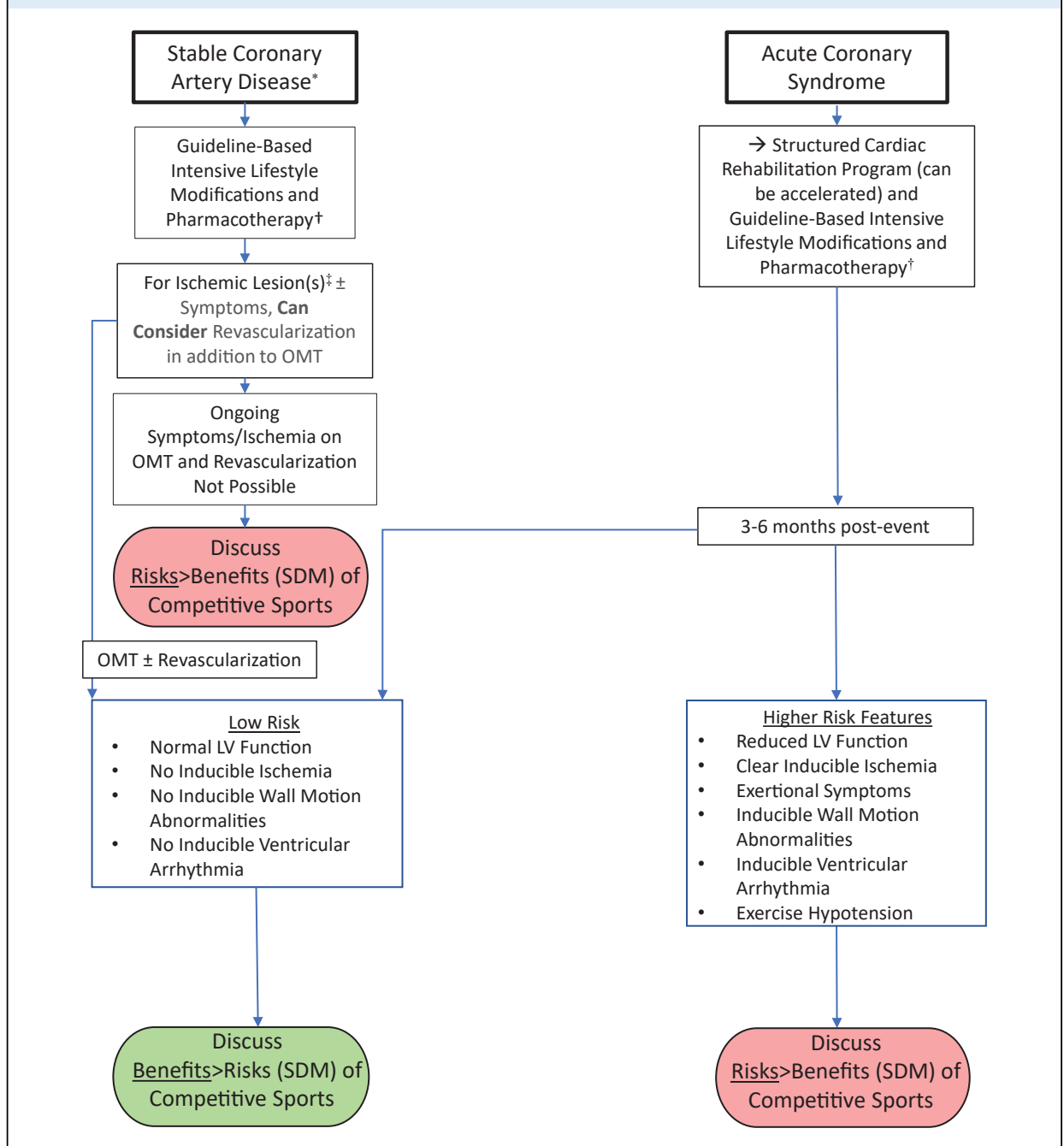
Unexplained thoracic aortic dilation <45 mm is common among masters endurance athletes.^{35,36} For unexplained thoracic aortic aneurysms ≥ 45 to 49 mm,¹⁸⁶ despite a paucity of data, competitive sports participation can be considered with SDM. With strength sports, concerns of static afterload stress on the aorta may lead to consideration of reductions to moderate-intensity strength training. With aneurysms ≥ 50 mm, risk of aortic dissection with competitive sports participation is likely high, thus competitive sports participation should be avoided. Competitive sports participation can be considered (see section VII) after surgical repair on the basis of limited data from athletes with BAV aortopathy.²⁵¹

Chronic Mitral and Aortic VHD

Considerations for masters athletes with mitral or aortic VHD is similar to those for younger competitive athletes with VHD (Section V). For considerations related to athletes with BAV, refer to Section VII.

Antithrombotic Therapy

It is reasonable for patients on antiplatelet monotherapy to participate in competitive sports.²⁵² Bleeding risks may be increased for masters athletes treated with dual antiplatelet therapy who participate in sports with risks of collision or impact (see Section I). The risks likely outweigh the benefits for some competitive sports participation (see Section I) for masters athletes who require full anticoagulation. Consideration of risk mitigation, such as temporary discontinuation of anticoagulation, can be considered with SDM for some masters athletes who desire continued competitive sports participation, although data establishing the safety of this approach are lacking.

FIGURE 4 Management and Competitive Sports Participation Considerations for Masters Athletes Diagnosed with Clinical Coronary Artery Disease

*Stable coronary artery disease, defined per 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA Guideline for the Management of Chronic Coronary Disease⁸⁸ as follows: (1) after hospital discharge of stable acute coronary syndrome or revascularization of stable disease, (2) established ischemic cardiomyopathy, (3) stable anginal symptoms on medical management, (4) coronary vasospasm or endothelial dysfunction, or (5) abnormal cardiovascular screening test results.

[†]Includes dietary modifications, smoking cessation, aggressive blood pressure control, and guideline-directed lipid-lowering pharmacotherapy. [‡]Obstructive coronary disease, defined as vessel stenoses at least >50% and associated with ischemia. Caution is advised in the interpretation of borderline instantaneous wave-free ratio or fractional flow reserve measurements, as these measures may not be equivalent to the physiologic stress of maximal-effort exercise. LV indicates left ventricular; OMT, optimal medical therapy; and SDM, shared decision-making.

SECTION XI: ADDITIONAL CARDIAC CONDITIONS AND CONSIDERATIONS

This section covers a spectrum of cardiovascular conditions and clinical scenarios that may affect competitive athletes, not all contributing to SCA or SCD risk. Hypertension, commotio cordis, pulmonary embolus (PE), performance-enhancing drugs and supplements, extreme exercise environments, and pregnancy are detailed (Table 15).

Hypertension

Hypertension is an established cardiovascular risk factor and hypertensive emergency is an acute clinical crisis.²⁵³ Hypertension is prevalent among certain populations of competitive athletes, particularly in strength-based sports, and may lead to early maladaptive cardiac remodeling.²⁵ There is insufficient evidence to support the use of exercise blood pressure measurements in isolation to establish the diagnosis of hypertension or to guide the clinical management of competitive athletes with established hypertension.²⁵⁴ Competitive athletes with confirmed hypertension should be assessed for drug and supplement use and lifestyle factors, with carefully selected antihypertensive medications initiated, if indicated. Competitive athletes with hypertensive emergency should not continue with competitive sports participation until adequate blood pressure control is achieved.

Commotio Cordis

Commotio cordis, the clinical entity of SCA triggered by blunt and generally projectile chest impact,²⁵⁵ has been described in numerous sports and nonsport activities.²⁵⁶⁻²⁵⁸ Survival hinges on immediate SCA recognition, cardiopulmonary resuscitation, and early defibrillation. All individuals who interact with competitive athletes regularly must be educated on this condition. Survivors of presumed commotio cordis must first have a comprehensive cardiac assessment to rule out underlying structural heart disease or inherited arrhythmias. Risk may be reduced by using softer projectiles and National Operating Committee on Standards for Athletic Equipment-approved chest protectors.^{259,260} Survivors of commotio cordis can resume competitive sports participation, although the psychologic effects and degree of recovery are important factors to consider with SDM. Although unlikely, it is uncertain whether previous commotio cordis increases the risk of recurrence.

Pulmonary Embolism

Competitive athletes may have increased thromboembolic risk, with contributing factors including long-distance travel, dehydration, surgery, or trauma.²⁶¹ Acute pulmonary embolism may be life-threatening, and

competitive athletes with symptoms suggestive of pulmonary embolism or deep venous thrombosis require urgent evaluation. A confirmed diagnosis warrants therapeutic anticoagulation per contemporary guidelines.²⁶² In consideration of competitive sports participation, it is essential to first ensure resolution of cardiopulmonary symptoms and absence of pulmonary hypertension and RV dysfunction. The risks likely outweigh benefits for some competitive sports participation (see Section I) in athletes with pulmonary embolism who are receiving full anticoagulation therapy.²⁶³ In exceptional cases, tailored anticoagulation plans that allow for temporary discontinuation during high-risk (trauma) competitive sports participation can be considered, although data establishing the safety of this approach are lacking.

Performance-Enhancing Drugs and Supplements

Performance-enhancing drug or supplement use is common among competitive athletes. The cardiovascular effects and toxicities associated with commonly used performance-enhancing drugs or supplements, particularly anabolic-androgenic steroids, have been described, and potentially include SCA.^{199,264,265} Stimulant use, particularly caffeine present in dietary and workout beverages and powders, should be queried and considered in competitive athletes with hypertension, symptomatic palpitations, or documented ectopy or arrhythmias.²⁶⁶ Competitive athletes must be educated on the risks of performance-enhancing drugs and supplements.

Extreme Exercise Environments

Competitive sports participation at high altitude (>2500 meters) is generally well-tolerated by competitive athletes, but those with CAD, pulmonary hypertension, or congenital heart disease may face increased cardiac risk while at altitude.²⁶⁷ Acclimatization may help reduce cardiac risks, but at-risk competitive athletes should undergo a clinical assessment before engaging in high-altitude activities.

With free diving and recreational scuba, individuals with CVD confront additional risk because of increased hydrostatic pressure, which can be pronounced at relatively shallow depths, and the cold-induced diving reflex, which can lead to SCA related to arrhythmogenic effects of complex autonomic activation, termed “autonomic conflict.”²⁶⁸⁻²⁷⁰; Although routine cardiac screening, particularly for patent foramen ovale, is unnecessary, risk assessment and education on diving-related health risks are critical, particularly for those with known cardiovascular conditions. Cardiovascular events that occur underwater are unique because of the higher risk of adverse outcomes for the diver and the additional risks that extend to other dive team members. As such, SDM with divers should incorporate these additional considerations.

TABLE 15

Clinical Considerations for Competitive Athletes With Hypertension, Commotio Cordis, or Pulmonary Embolus, With Additional Clinical Considerations for Competitive Athletes and Performance-Enhancing Drugs or Supplements, Extreme Exercise Environments (Altitude and Scuba), and Pregnancy**Hypertension**

Assessment of resting blood pressure using an appropriately sized cuff should be performed during the preparticipation evaluation for competitive athletes.

Competitive athletes with prehypertension (SBP 120–129 mm Hg and DBP <80 mm Hg) can participate in competitive sports. Such competitive athletes should be counseled on lifestyle modifications (eg, nutrition, sleep, supplements, stimulants, alcohol consumption) and receive follow-up care with their primary care health care professional or sports medicine team.

Competitive sports participation is reasonable for competitive athletes with stage 1 hypertension (SBP 130–139 mm Hg or DBP 80–89 mm Hg) or stage 2 hypertension (SBP ≥140 mm Hg or DBP ≥90 mm Hg). Such competitive athletes should be counseled on lifestyle modifications (eg, alcohol consumption, supplement/stimulant use, nutrition, sleep) and receive close follow-up care for longitudinal blood pressure monitoring with their primary care health care professional or sports medicine team. Initiation of antihypertensive medication is reasonable if hypertension persists despite lifestyle modification.

For competitive athletes diagnosed with stage 2 hypertension, it is reasonable to obtain a 12-lead ECG and echocardiogram to assess for evidence of adverse cardiac remodeling.

Competitive athletes diagnosed with hypertensive emergency, as defined by SBP >180 mm Hg or DBP >120 mm Hg in conjunction with evidence of new or worsening end-organ damage, should be managed according to contemporary guidelines and should not participate in competitive sports until adequate blood pressure control is achieved.

Before prescribing antihypertensive medication to competitive athletes, clinicians should cross-reference the lists of prohibited drugs by the appropriate governing bodies of their sport and attempt to use agents that are not contained on these lists. In rare situations where a banned substance has no equivalent alternative and is deemed medically necessary, a therapeutic use exemption should be pursued at the time of prescription and approved before subsequent competition.

Commotio cordis

To ensure prompt recognition and to optimize resuscitation of commotio cordis, competitive athletes, coaches, staff, officials, and sporting event medical personnel should be educated about commotio cordis.

A comprehensive evaluation for underlying cardiac pathology that predisposes to malignant ventricular arrhythmias should be performed in competitive athletes who survive presumed commotio cordis.

It is reasonable to use age-appropriate sport safety projectiles (ie, baseballs, softballs, lacrosse balls) and National Operating Committee on Standards for Athletic Equipment-approved chest protectors to reduce the risk of commotio cordis.

If no underlying explanatory cardiac abnormality is identified, competitive athletes who survive commotio cordis can resume competitive sports participation with SDM.

Pulmonary embolism

Competitive athletes who present with signs or symptoms suggestive of either DVT or PE should not participate in competitive sports until they undergo prompt and comprehensive diagnostic evaluation for these conditions.

Competitive athletes with a DVT or PE should be treated with therapeutic anticoagulation as dictated by contemporary guidelines.

The risks of bleeding likely outweigh benefits in some competitive sports for competitive athletes diagnosed with a DVT or PE and who are taking full anticoagulation therapy (see Section I).

Competitive athletes should be evaluated longitudinally for symptoms of persistent breathlessness, pulmonary hypertension, or RV dysfunction after diagnosis of PE.

In rare circumstances, anticoagulation strategies that involve transient cessation of therapy to reduce bleeding risk during some competitive sports with collision or impact risk (see Section I) can be considered, which includes weighing the risks of decreased therapeutic efficacy and recurrent clotting events.

Performance-enhancing drugs and supplements

Competitive athletes should receive formal education and counseling on the potential dangers, including but not limited to the cardiovascular complications, of substance misuse, which include recreational substances and supplements, performance-enhancing drugs, and illicit use of prescription drugs.

Prescription medications that are considered banned substances by doping control organizations (eg, β-adrenergic blockers/agonists, bronchodilators, stimulants) may be subject to exceptions in cases of medical necessity when alternative comparable therapeutic options are lacking. Medical need should be determined by the treating clinician on a case-by-case basis and authorized before training and competition by the procedures defined by the appropriate international and national antidoping regulatory authorities.

Extreme exercise environments

High-altitude travel, training, and competition are generally well-tolerated by competitive athletes with most forms of established asymptomatic cardiovascular disease. However, competitive athletes with pulmonary arterial hypertension (mean pulmonary artery pressure >25 mm Hg, pulmonary vascular resistance >3 WU) or congenital defects with right-to-left shunting may be at elevated risk of adverse events and should be evaluated clinically before consideration of high-altitude travel, training, and competition.

Routine screening for PFO or ASD should not be performed for recreational scuba and free divers.

The presence of a PFO is not a contraindication to recreational scuba diving. Divers found to have a PFO should be counseled on routine measures to reduce the risk of embolic complications of decompression illness.

It is reasonable to perform a cardiac ischemic risk assessment for scuba and free divers with established atherosclerotic cardiovascular disease who may be at increased risk for cardiovascular complications while diving.

Recreational diving for scuba and free divers with established cardiovascular disease can be considered with SDM, which includes the additional risks of a cardiovascular event occurring while underwater, thus increasing risks to other members of the dive team.

Continued on the next page

TABLE 15 Continued**Pregnancy**

Competitive athletes without established cardiovascular disease desiring to continue competitive sports participation during pregnancy should be offered a formal preconception consultation with maternal fetal medicine to discuss the risks and benefits of continuation of competitive sports participation while pregnant, as well as guidance regarding nutritional needs, fetal surveillance, and injury prevention.

Pregnant competitive athletes who continue competitive sports participation should be cared for by a clinical team with expertise in the care of pregnant athletes.

Competitive sports participation for pregnant competitive athletes is reasonable, as tolerated, if there are no acquired conditions related to pregnancy that represent a contraindication to exercise.

Competitive sports that carry an inherent risk of collisions or impacts (see [Section I](#)) or additional high-risk physiology (ie, hypoxia, profound dehydration) should not be continued during pregnancy.

The clinical team should provide pregnant competitive athletes with individualized guidance regarding return to competitive sports participation during the postpartum period, which represents a time when the athlete may be most vulnerable to injury in the setting of hormonal shifts. Special consideration should also be given to nutrition and hydration needs during breastfeeding.

ASD indicates atrial septal defect; DBP, diastolic blood pressure; DVT, deep venous thrombosis; PE, pulmonary embolism; PFO, patent foramen ovale; SBP, systolic blood pressure; and SDM, shared decision-making.

Pregnancy

Pregnancy induces substantial hemodynamic adaptations that result in shifting physiologic and metabolic landscapes for competitive athletes.^{271,272} Competitive athletes planning competitive sports participation while pregnant should be offered preconception consultation with specialists who can guide the risk/benefit assessment and counsel the athlete on aspects such as nutritional needs and fetal surveillance.²⁷² For pregnant

competitive athletes, the risks of competitive sports participation to the fetus are uncertain. Consensus opinion among obstetricians is that pregnant athletes should avoid maximal exertion, avoid exposure to extreme heat and altitude, take precautions to avoid injury and abdominal impact, and ensure proper hydration and nutrition.^{271,273} Future research is needed to identify safe exercise thresholds for pregnant competitive athletes.

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DISCLOSURES

APPENDIX Writing Group Disclosures

Writing group member	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Expert witness	Ownership interest	Consultant/advisory board	Other
Jonathan H. Kim	Emory University School of Medicine	None	None	None	None	None	None	None
Aaron L. Baggish	Centre Hospitalier Universitaire Vaudois (Switzerland)	National Football League Players Association (Football Players Health Study at Harvard University) [†] ; American Heart Association (Outcomes Registry for Cardiac Conditions in Athletes [ORCCA]) [†]	None	None	None	None	United States Soccer Federation [†] ; United States Olympic Paralympic Organization*	None
Benjamin D. Levine	University of Texas, Southwestern Medical Center, Texas Health, Presbyterian Hospital, Dallas Institute for Exercise and Environmental Medicine	None	None	None	None	None	None	None
Michael J. Ackerman	Mayo Clinic	None	None	None	None	AliveCor*; Anumana*; ARMGO Pharma [†] ; Pfizer [†] ; Thryv Therapeutics*; Prolaio*	Abbott*; BioMarin*; Boston Scientific*; Bristol Myers Squibb [†] ; Daiichi Sankyo*; Illumina*; Invitae*; Medtronic*; Tenaya Therapeutics [†] ; UpToDate [†] ; Pfizer [†] ; Solid Biosciences [†]	None
Sharlene M. Day	University of Pennsylvania	NHLBI [†] ; Lexicon Pharmaceuticals [†] ; Bristol Myers Squibb [†]	None	None	None	None	Lexicon Pharmaceuticals [†] ; Cytokinetics*	None
Elizabeth H. Dineen	Mayo Clinic	Miami Heart Research Institute (joined a research project as an investigator and the research is partially funded by Miami Heart Research Institute; the grant is <\$100 000 and it is for a project studying coronary calcification in athletes; none of the money is going to Dr Dineen or any of the other investigators personally)*	None	None	None	None	None	None
J. Sawalla Guseh II	Massachusetts General Hospital, Harvard Medical School	None	None	None	None	None	New England Patriots Organization [†]	None
Andre La Gerche	Victor Chang Cardiac Research Institute (Australia)	None	None	None	None	None	None	None
Rachel Lampert	Yale University School of Medicine	None	None	None	None	None	None	None
Matthew W. Martinez	Morristown Medical Center; Atlantic Health System	None	None	Bristol Myers Squibb [†]	None	None	Cytokinetics*; Bristol Myers Squibb [†]	None
Michael Papadakis	St George's, University of London (United Kingdom)	Charity Cardiac Risk in the Young (received research grants through the university over a number of years) [†]	None	None	None	None	Bristol Myers Squibb*	None
Dermot M. Phelan	Atrium Health, Sanger Heart and Vascular Institute	None	None	None	None	None	None	None
Keri M. Shafer	Boston Children's Hospital	None	None	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives \$5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns \$5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

[†]Significant.

APPENDIX Reviewer Disclosures

Reviewer	Employment	Research grant	Other research support	Speakers' bureau/honoraria	Expert witness	Ownership interest	Consultant/advisory board	Other
Travis C. Batts	United States Air Force, Lackland AFB, Texas	None	Philips Ultrasound USA (nonpromotional educational speaker) [†] ; Novo Nordisk (nonpromotional educational speaker and promotional) [†]	None	None	None	None	None
Robert O. Bonow	Northwestern University Feinberg School of Medicine	None	None	None	None	None	None	None
Alfred Danielian	Mountain View Hospital	None	None	None	None	None	None	None
Eddie Davenport	United States Air Force	None	None	None	None	None	None	None
Eli M. Friedman	Baptist Health Heart and Vascular Care, Miami Cardiac and Vascular Institute	None	None	None	None	None	None	None
Mustafa Husaini	Washington University School of Medicine	None	None	None	None	None	None	None
Elizabeth V. Saarel	St Luke's Health System	None	None	None	None	None	None	None
Victoria L. Vetter	Children's Hospital of Philadelphia, Perelman School of Medicine at the University of Pennsylvania	None	None	None	None	None	None	None
Kim A. Williams	University of Louisville	None	None	None	None	None	None	None

This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives \$5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns \$5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.