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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 electrophysiologists, 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	■ 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	■ 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	■ 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	■ 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	■ 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	■ 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	■ 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	■ 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	■ 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	■ 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	■ 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

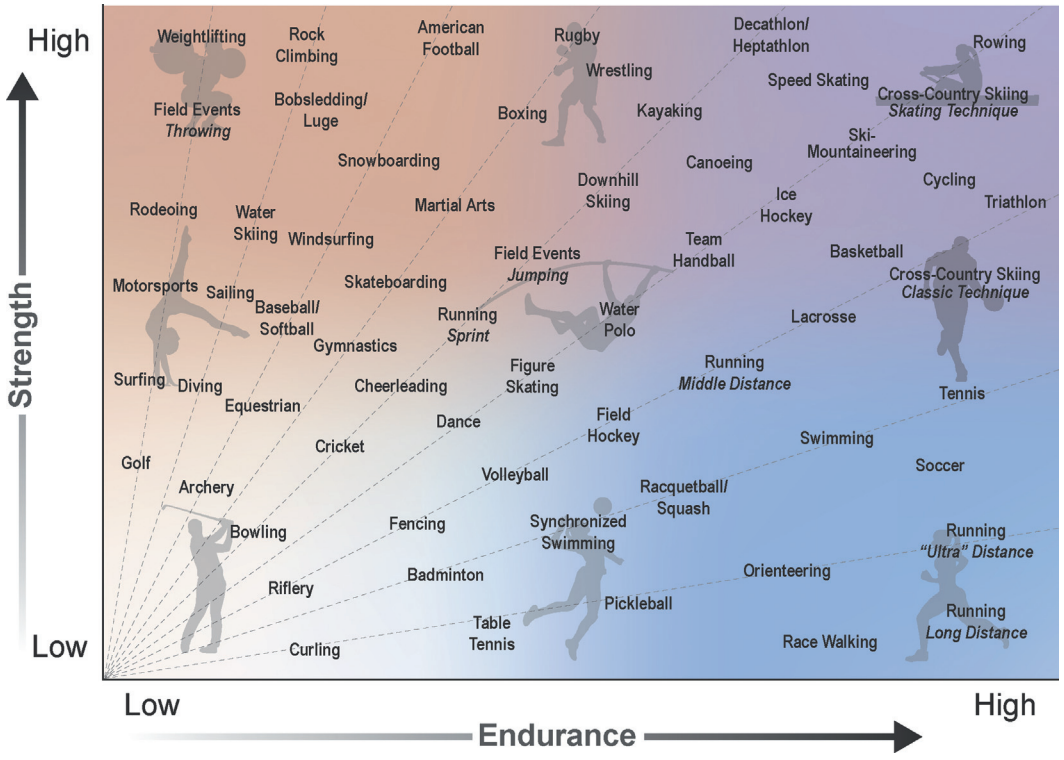
must be a call to action on disparities if we are to pursue equity in the care of all athletes.[42,47]

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 al[50] with permission and modified.

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.	■ 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.	■ 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.	■ 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 disorders[1-4]; 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 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]

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.

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.

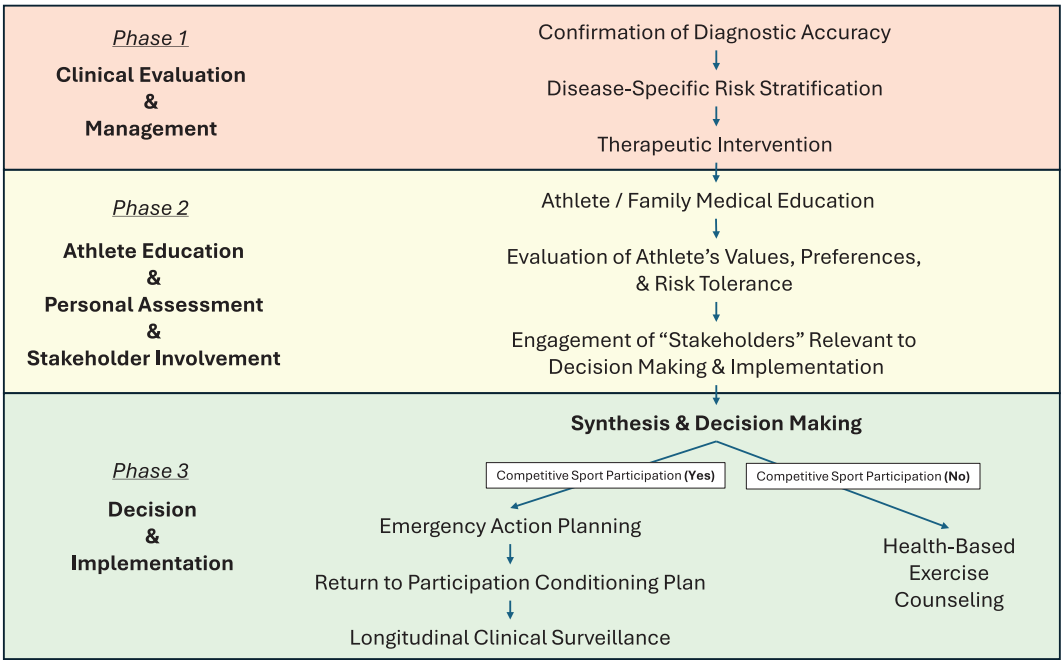
The application of SDM for competitive sports participation decisions aligns with more broadly changing cardiovascular practice patterns. Professional societies have increasingly called for the integration of SDM,[84,85] as now reflected in clinical practice guidelines, including those for HCM and CAD.[86-88] 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.

Continued on the next page

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.

Continued on the next page

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 ≈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	■ Small, moderate ASD ■ Small, moderate restrictive VSD ■ Small, moderate restrictive PDA ■ Partial anomalous pulmonary venous connection			■ Hypoxemia ■ Complex arrhythmias‡ ■ RVH on imaging	Can participate in all competitive sports after appropriate clinical evaluation*
Moderate or large	■ Qp:Qs >1.5–2.0§ ■ Typically asymmetric chamber enlargement due to shunt‖	■ Moderate, large ASD ■ Moderate, large VSD ■ Moderate, large PDA ■ PAPVC	■ CPET (or EST) is reasonable¶ ■ Intervention likely indicated	No evidence of pulmonary hypertension	■ 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	■ After TOF repair ■ Before or after intervention: valvar PS, subvalvar PS, peripheral PS	For postrepair patients, should perform CPET (or EST)¶	■ No or mild RVH on imaging ■ Normal RV function	■ 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	■ After TOF repair ■ Before or after intervention: valvar PS, subvalvar PS, peripheral PS	■ CPET (or EST)¶ ■ Ambulatory cardiac rhythm monitoring ■ Consider CMR or CCT ■ Intervention likely indicated	■ Mild or moderate RVH on imaging ■ Normal RV function	■ 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	■ Valvar AS ■ Subvalvar AS ■ Supravalvar AS ■ AV septal defect s/p repair with mild LVOT obstruction		■ No LVH ■ Normal LV size and function on imaging‖	■ 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	■ Valvar AS ■ Subvalvar AS ■ Supravalvar AS ■ AV canal defect s/p repair with moderate LVOT obstruction	■ CPET (or EST)¶ ■ Ambulatory cardiac rhythm monitoring	■ 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	■ 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	■ Valvar AS ■ Subvalvar AS ■ Supravalvar AS ■ AV septal defect s/p repair with severe LVOT obstruction (Section V)	■ CPET (or EST)¶ ■ Ambulatory cardiac rhythm monitoring ■ Intervention likely recommended		■ Diagnostic ischemic ECG changes with stress test ■ Rapidly increasing LVOT gradient ■ Complex arrhythmias at rest or stress‡ ■ Exertional symptoms ■ LV dysfunction‖ ■ Pulmonary hypertension	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). After intervention: see the row "less than severe" above.
Pulmonary regurgitation						
Less than severe	See appropriate ASE and SCMR guidelines‖	■ TOF s/p repair ■ PS s/p repair (catheter or surgical)	■ Consider CPET (or EST)¶ ■ Consider CMR/CCT ■ Consider ambulatory cardiac rhythm monitoring	■ Normal biventricular size and function‖ ■ No complex arrhythmias	■ 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‖	■ TOF s/p repair ■ PS s/p repair (catheter or surgical) ■ Isolated pulmonary regurgitation	■ CPET (or EST)¶ ■ CMR/CCT ■ Consider ambulatory cardiac rhythm monitoring	■ Mild or moderate RV dilation on imaging‖ ■ Normal biventricular function‖ ■ No complex arrhythmias at rest or stress	■ 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	■ CPET (or EST)¶ ■ Consider CMR/CCT ■ Ambulatory cardiac rhythm monitoring	■ 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‖	■ 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		■ Hypoplastic left heart syndrome, s/p Fontan procedure ■ Right dominant unbalanced AV canal, s/p Fontan procedure	■ CPET (or EST)¶ ■ CMR/CCT (with special attention to DKS, arch, and coronaries) ■ Ambulatory cardiac rhythm monitoring	■ See single ventricle (systemic LV) ■ Ensure normal coronary blood flow, no aortic obstruction	■ 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		■ Dextro-TGA s/p Mustard or Senning procedure ■ CCTGA	■ CPET (or EST)¶ ■ CMR/CCT ■ Consider evaluation for baffle leaks ■ Consider ambulatory cardiac rhythm monitoring	■ Mild or moderate RV dilation on imaging‖ ■ Normal biventricular function‖ ■ Mild tricuspid (systemic AVV) regurgitation‖ ■ No arrhythmias	■ 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 I).
Transposition of the great arteries with anatomic repair	Circulation has LV aortic flow and RV PA flow	■ D-TGA ■ Some types of DORV ■ Surgical repairs include** arterial switch, Rastelli procedure ■ Nikaidoh procedure	■ CPET (or EST)¶ ■ CMR/CCT (special attention to coronary arteries)	■ Normal biventricular size and function‖ ■ No ischemia and normal exercise capacity on stress testing ■ Normal aortic dimensions‖ ■ No more than mild aortic regurgitation‖	■ 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		■ Ebstein anomaly ■ Dysplastic tricuspid valve ■ AV canal defect s/p repair	■ Ambulatory cardiac rhythm monitoring	■ For Ebstein anomaly or dysplastic tricuspid valve: no WPW or residual WPW, normal RV and LV function	■ 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		■ 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	■ Upper and lower extremity BPs ■ CPET (or EST)¶ ■ CTA or CMR ■ Hypertension should be treated before exercise (Section XI)	■ Normotensive at rest, treated or untreated ■ No or minimal resting upper extremity/lower extremity BP gradient ■ Normal BP response to exercise ■ No aortic aneurysms	■ 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 heterogenous 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 ^{13}n-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 heterogenous.[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.

Continued on the next page

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 remodeling[7] 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, nonsustained 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.

Continued on the next page

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: ■ Outflow tract: LBBB pattern, inferior axis, transition <V_4 favors RVOT and ≥V_4 favors LVOT ■ Fascicular morphology: RBBB pattern, QRS duration typically <130 ms, inferior axis (anterior fascicle), superior axis (posterior fascicle)	Morphology consistent with: ■ 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)

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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 ≈5% per year risk of inappropriate shocks and ≈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 ≥460 ms prepuberty, ≥470 male, ≥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 ≥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 VII).
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 ≥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.

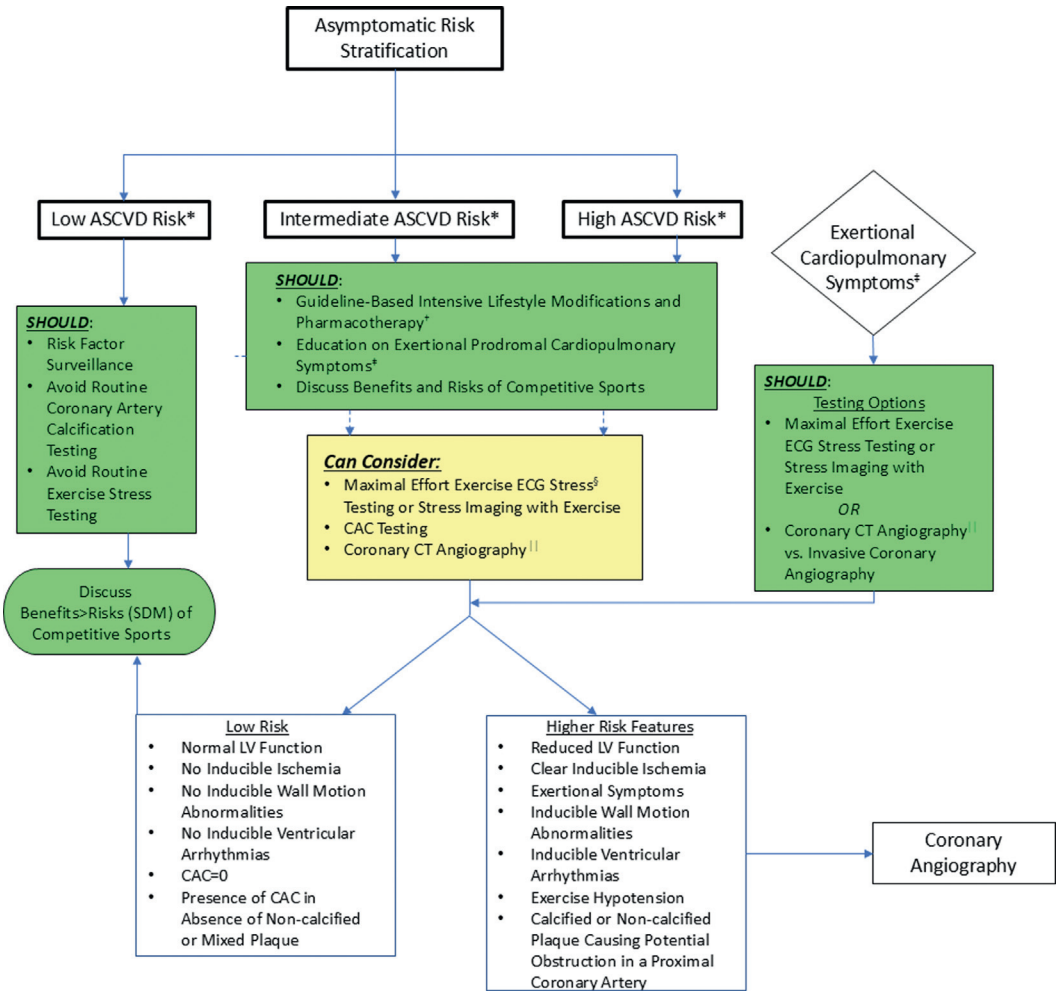
*Cardiovascular disease risk scores (% risk per decade) have not been validated in masters athlete populations. In the general population, CVD risk scores include the following: low risk, <5% atherosclerotic CVD (ASCVD) risk score pooled cohort or Astro-CHARM (Astronaut Cardiovascular Health and Risk 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, ≥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 Disease[88]: (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 Disease[186] 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.

FIGURE 3 Cardiac Risk Stratification Considerations for Masters Athletes



*Cardiovascular disease (CVD) risk scores (% risk per decade) have not been validated in masters athlete populations, where high cardiorespiratory fitness is protective of CVD development. 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, 2.5% to <7.5% (high) SCORE2; high risk, >20% ASCVD, ≥7.5% Astro-CHARM or very high SCORE2. Family history of early coronary artery disease should be considered a high-risk feature. †Includes dietary modifications, smoking cessation, aggressive blood pressure control, and guideline-directed lipid- lowering pharmacotherapy. ‡Cardiopulmonary symptoms, defined as exertional chest pain or tightness, dyspnea, palpitations, lightheadedness, syncope, or exercise intolerance. §Maximal-effort exercise stress testing should be sport-specific and is generally defined as exercise to volitional exhaustion despite vigorous encouragement (unless cardiac symptoms limit performance). ‖With borderline or obstructive lesions detected by computed tomography (CT) coronary angiography, assessment of functional significance is required by either functional stress testing with exercise or maximum-effort exercise ECG stress testing. CAC indicates coronary artery calcification; LV, left ventricular; and SDM, shared decision-making.

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 controls[244-246]; 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]

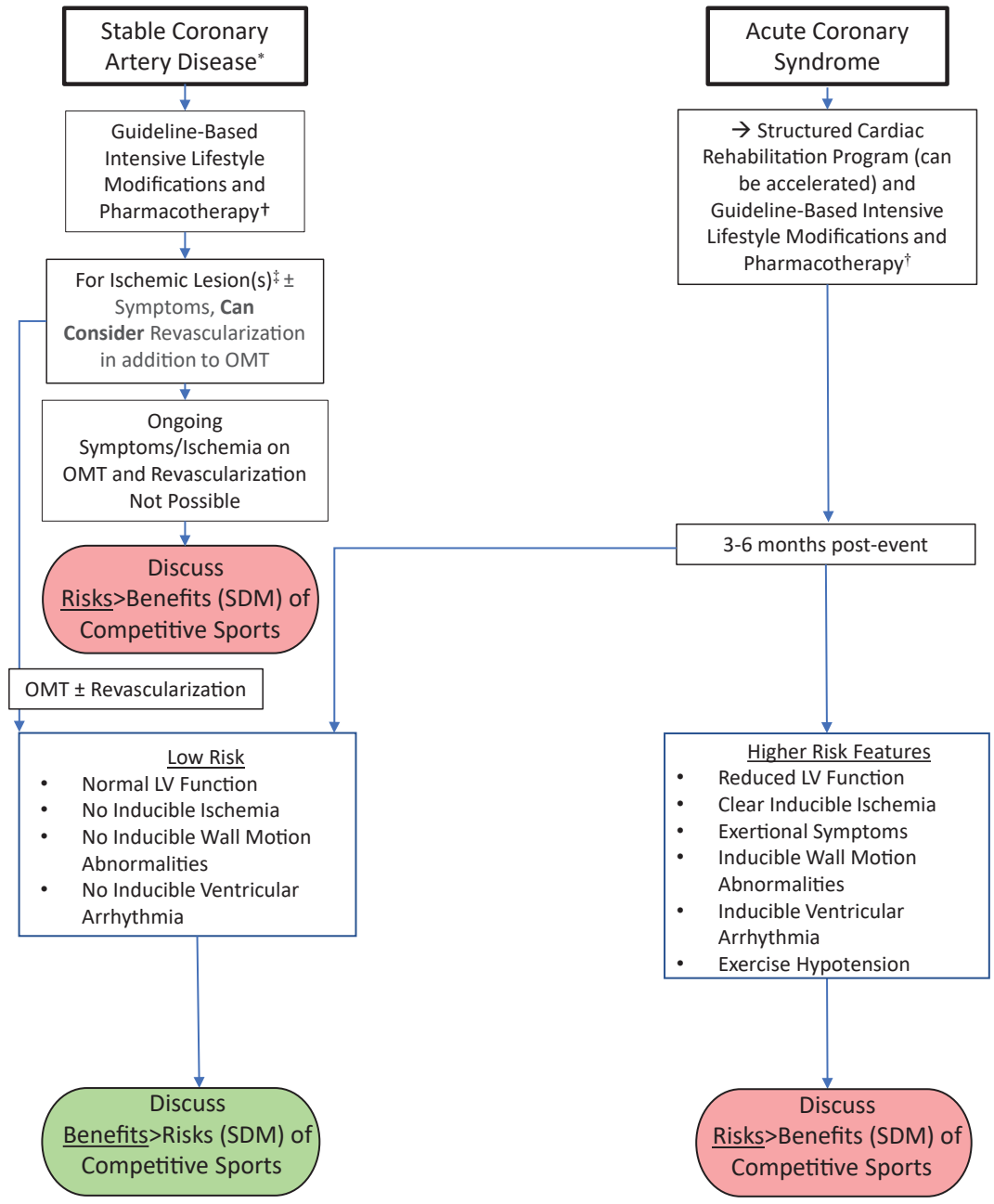
Chronic Mitral and Aortic VHD

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 Disease[88] 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.

REFERENCES

1. Brock DW, Wartman SA. When competent patients make irrational choices. *N Engl J Med*. 1990;322:1595-1599. https://doi.org/10.1056/NEJM199005313222209

2. Sandman L, Munthe C. Shared decision-making, paternalism and patient choice. *Health Care Anal*. 2010;18:60-84. https://doi.org/10.1007/s10728-008-0108-6

3. Mitchell JH, Maron BJ, Epstein SE. 16th Bethesda Conference: Cardiovascular abnormalities in the athlete: recommendations regarding eligibility for competition: October 3-5, 1984. *J Am Coll Cardiol*. 1985;6:1186-1232. https://doi.org/10.1016/s0735-1097(85)80200-x

4. Maron BJ, Mitchell JH. Revised eligibility recommendations for competitive athletes with cardiovascular abnormalities. *J Am Coll Cardiol*. 1994;24:848-850. https://doi.org/10.1016/0735-1097(94)90837-0

5. Maron BJ, Zipes DP. Introduction: eligibility recommendations for competitive athletes with cardiovascular abnormalities: general considerations. *J Am Coll Cardiol*. 2005;45:1318-1321. https://doi.org/10.1016/j.jacc.2005.02.006

6. Maron BJ, Zipes DP, Kovacs RJ, on behalf of the American Heart Association Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology. Council on Cardiovascular Disease in the Young, Council on Cardiovascular and Stroke Nursing, Council on Functional Genomics and Translational Biology, and the American College of Cardiology. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: preamble, principles, and general considerations: a scientific statement from the American Heart Association and American College of Cardiology. *J Am Coll Cardiol*. 2015;66:2343-2349. https://doi.org/10.1016/j.jacc.2015.09.032

7. Baggish AL, Battle RW, Beaver TA, Border WL, Douglas PS, Kramer CM, Martinez MW, Mercandetti JH, Phelan D, Singh TK, et al. Recommendations on the use of multimodality cardiovascular imaging in young adult competitive athletes: a report from the American Society of Echocardiography in collaboration with the Society of Cardiovascular Computed Tomography and the Society for Cardiovascular Magnetic Resonance. *J Am Soc Echocardiogr*. 2020;33:523-549. https://doi.org/10.1016/j.echo.2020.02.009

8. Lampert R, Ackerman MJ, Marino BS, Burg M, Ainsworth B, Salberg L, Tome Esteban MT, Ho CY, Abraham R, Balaji S, et al. LIVE Consortium. Vigorous exercise in patients with hypertrophic cardiomyopathy. *JAMA Cardiol*. 2023;8:595-605. https://doi.org/10.1001/jamacardio.2023.1042

9. Lampert R, Olshansky B, Heidbuchel H, Lawless C, Saarel E, Ackerman M, Calkins H, Estes NAM, Link MS, Maron BJ, et al. Safety of sports for athletes with implantable cardioverter-defibrillators: long-term results of a prospective multinational registry. *Circulation*. 2017;135:2310-2312. https://doi.org/10.1161/CIRCULATIONAHA.117.027828

10. Martinez KA, Bos JM, Baggish AL, Phelan DM, Tobert KE, Newman DB, Scherer E, Petek BJ, Ackerman MJ, Martinez MW. Return-to-play for elite

athletes with genetic heart diseases predisposing to sudden cardiac death. *J Am Coll Cardiol*. 2023;82:661–670. https://doi.org/10.1016/j.jacc.2023.05.059

11. Tobert KE, Bos JM, Garmany R, Ackerman MJ. Return-to-play for athletes with long QT syndrome or genetic heart diseases predisposing to sudden death. *J Am Coll Cardiol*. 2021;78:594–604. https://doi.org/10.1016/j.jacc.2021.04.026

12. Churchill TW, Disanto M, Singh TK, Groezinger E, Loomer G, Contursi M, DiCarli M, Michaud-Finch J, Stewart KM, Hutter AM, et al. Diagnostic yield of customized exercise provocation following routine testing. *Am J Cardiol*. 2019;123:2044–2050. https://doi.org/10.1016/j.amjcard.2019.03.027

13. Levine BD, Stray-Gundersen J. The medical care of competitive athletes: the role of the physician and individual assumption of risk. *Med Sci Sports Exerc*. 1994;26:1190–1192. https://doi.org/10.1249/00005768-199410000-00002

14. Sharma S, Drezner JA, Baggish A, Papadakis M, Wilson MG, Prutkin JM, La Gerche A, Ackerman MJ, Borjesson M, Salerno JC, et al. International recommendations for electrocardiographic interpretation in athletes. *J Am Coll Cardiol*. 2017;69:1057–1075. https://doi.org/10.1016/j.jacc.2017.01.015

15. Xu J, Haigney MC, Levine BD, Dineen EH. The tactical athlete: definitions, cardiovascular assessment, and management, and "fit for duty" standards. *Cardiol Clin*. 2023;41:93–105. https://doi.org/10.1016/j.ccl.2022.08.008

16. Baggish AL, Battle RW, Beckerman JG, Bove AA, Lampert RJ, Levine BD, Link MS, Martinez MW, Molossi SM, Salerno J, et al. Sports cardiology: core curriculum for providing cardiovascular care to competitive athletes and highly active people. *J Am Coll Cardiol*. 2017;70:1902–1918. https://doi.org/10.1016/j.jacc.2017.08.055

17. Institute of Medicine Committee on Quality of Health Care in America. *Crossing the Quality Chasm: A New Health System for the 21st Century*. National Academies Press; 2001.

18. Johnson JN, Ackerman MJ. Return to play? Athletes with congenital long QT syndrome. *Br J Sports Med*. 2013;47:28–33. https://doi.org/10.1136/bjsports-2012-091751

19. Harmon KG, Asif IM, Maleszewski JJ, Owens DS, Prutkin JM, Salerno JC, Zigman ML, Ellenbogen R, Rao AL, Ackerman MJ, et al. Incidence, cause, and comparative frequency of sudden cardiac death in National Collegiate Athletic Association athletes: a decade in review. *Circulation*. 2015;132:10–19. https://doi.org/10.1161/CIRCULATIONAHA.115.015431

20. Petek BJ, Churchill TW, Moulson N, Kliethermes SA, Baggish AL, Drezner JA, Patel MR, Ackerman MJ, Kucera KL, Siebert DM, et al. Sudden cardiac death in National Collegiate Athletic Association athletes: a 20-year study. *Circulation*. 2023;149:80–90. https://doi.org/10.1161/circulationaha.123.065908

21. Engel DJ, Schwartz A, Homma S. Athletic cardiac remodeling in US professional basketball players. *JAMA Cardiol*. 2016;1:80–87. https://doi.org/10.1001/jamacardio.2015.0252

22. Shames S, Bello NA, Schwartz A, Homma S, Patel N, Garza J, Kim JH, Goolsby M, DiFiori JP, Engel DJ. Echocardiographic characterization of female professional basketball players in the US. *JAMA Cardiol*. 2020;5:991–998. https://doi.org/10.1001/jamacardio.2020.0988

23. D'Ascenzi F, Biella F, Lemme E, Maestrini V, Di Giacinto B, Pelliccia A. Female athlete's heart: sex effects on electrical and structural remodeling. *Circ Cardiovasc Imaging*. 2020;13:e011587. https://doi.org/10.1161/CIRCIMAGING.120.011587

24. Churchill TW, Petek BJ, Wasfy MM, Guseh JS, Weiner RB, Singh TK, Schmied C, O'Malley H, Chiampas G, Baggish AL. Cardiac structure and function in elite female and male soccer players. *JAMA Cardiol*. 2021;6:316–325. https://doi.org/10.1001/jamacardio.2020.6088

25. Kim JH, Hollowed C, Liu C, Al-Badri A, Alkhoder A, Dommisse M, Gowani Z, Miller A, Nguyen P, Prabakaran G, et al. Weight gain, hypertension, and the emergence of a maladaptive cardiovascular phenotype among US football players. *JAMA Cardiol*. 2019;4:1221–1229. https://doi.org/10.1001/jamacardio.2019.3909

26. Martinez MW, Kim JH, Shah AB, Phelan D, Emery MS, Wasfy MM, Fernandez AB, Bunch TJ, Dean P, Danielian A, et al. Exercise-induced cardiovascular adaptations and approach to exercise and cardiovascular disease: JACC state-of-the-art review. *J Am Coll Cardiol*. 2021;78:1453–1470. https://doi.org/10.1016/j.jacc.2021.08.003

27. Ostby SA, Bos JM, Owen HJ, Wackel PL, Cannon BC, Ackerman MJ. Competitive sports participation in patients with catecholaminergic polymorphic ventricular tachycardia: a single center's early experience. *JACC Clin Electrophysiol*. 2016;2:253–262. https://doi.org/10.1016/j.jacep.2016.01.020

28. Lampert R, Olshansky B, Heidbuchel H, Lawless C, Saarel E, Ackerman M, Calkins H, Estes NA, Link MS, Maron BJ, et al. Safety of sports for athletes with implantable cardioverter-defibrillators: results of a prospective, multinational registry. *Circulation*. 2013;127:2021–2030. https://doi.org/10.1161/CIRCULATIONAHA.112.000447

29. Saarel EV, Law I, Berul CI, Ackerman MJ, Kanter RJ, Sanatani S, Cohen MI, Berger S, Fischbach PS, Burton DA, et al. Safety of sports for young patients with implantable cardioverter-defibrillators: long-term results of the Multinational ICD Sports Registry. *Circ Arrhythm Electrophysiol*. 2018;11:e006305. https://doi.org/10.1161/CIRCEP.118.006305

30. Egger F, Scharhag J, Kastner A, Dvorak J, Bohm P, Meyer T. FIFA Sudden Death Registry (FIFA-SDR): a prospective, observational study of sudden death in worldwide football from 2014 to 2018. *Br J Sports Med*. 2022;56:80–87. https://doi.org/10.1136/bjsports-2020-102368

31. Kim JH, Malhotra R, Chiampas G, d'Hemecourt P, Troyanos C, Cianca J, Smith RN, Wang TJ, Roberts WO, Thompson PD, et al. Race Associated Cardiac Arrest Event Registry (RACER) Study Group. Cardiac arrest during long-distance running races. *N Engl J Med*. 2012;366:130–140. https://doi.org/10.1056/NEJMoa1106468

32. Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, Flachskampf FA, Foster E, Goldstein SA, Kuznetsova T, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging*. 2015;16:233–270. https://doi.org/10.1093/ehjci/jev014

33. Merghani A, Maestrini V, Rosmini S, Cox AT, Dhutia H, Bastiaenan R, David S, Yeo TJ, Narain R, Malhotra A, et al. Prevalence of subclinical coronary artery disease in masters endurance athletes with a low atherosclerotic risk profile. *Circulation*. 2017;136:126–137. https://doi.org/10.1161/CIRCULATIONAHA.116.026964

34. De Bosscher R, Dausin C, Claus P, Bogaert J, Dymarkowski S, Goetschalckx K, Ghekiere O, Van De Heyning CM, Van Herck P, Paelinck B, et al. Lifelong endurance exercise and its relation with coronary atherosclerosis. *Eur Heart J*. 2023;44:2388–2399. https://doi.org/10.1093/eurheartj/ehad152

35. Churchill TW, Groezinger E, Kim JH, Loomer G, Guseh JS, Wasfy MM, Isselbacher EM, Lewis GD, Weiner RB, Schmied C, et al. Association of ascending aortic dilatation and long-term endurance exercise among older masters-level athletes. *JAMA Cardiol*. 2020;5:522–531. https://doi.org/10.1001/jamacardio.2020.0054

36. Tso JV, Turner CG, Liu C, Miller AB, Eapen DJ, Sperling LS, Kim JH. Exercise blood pressure changes and aortic dilatation in male masters endurance athletes. *Eur J Prev Cardiol*. 2023;30:e18–e20. https://doi.org/10.1093/eurjpc/zwac250

37. Pelliccia A, Sharma S, Gati S, Back M, Borjesson M, Caselli S, Collet JP, Corrado D, Drezner JA, Halle M, et al. 2020 ESC guidelines on sports cardiology and exercise in patients with cardiovascular disease. *Eur Heart J*. 2021;42:17–96. https://doi.org/10.1093/eurheartj/ehaa605

38. Lampert R, Chung EH, Ackerman MJ, Arroyo AR, Darden D, Deo R, Dolan J, Etheridge SP, Gray BR, Harmon KG, et al. 2024 HRS expert consensus statement on arrhythmias in the athlete: evaluation, treatment, and return to play. *Heart Rhythm*. 2024;21:e151–e252. https://doi.org/10.1016/j.hrthm.2024.05.018

39. Gluckman TJ, Bhave NM, Allen LA, Chung EH, Spatz ES, Ammirati E, Baggish AL, Bozkurt B, Cornwell WK 3rd, et al. 2022 ACC expert consensus decision pathway on cardiovascular sequelae of COVID-19 in adults: myocarditis and other myocardial involvement, post-acute sequelae of SARS-CoV-2 infection, and return to play: a report of the American College of Cardiology solution set oversight committee. *J Am Coll Cardiol*. 2022;79:1717–1756. https://doi.org/10.1016/j.jacc.2022.02.003

40. Kim JH, Levine BD, Phelan D, Emery MS, Martinez MW, Chung EH, Thompson PD, Baggish AL. Coronavirus disease 2019 and the athletic heart: emerging perspectives on pathology, risks, and return to play. *JAMA Cardiol*. 2021;6:219–227. https://doi.org/10.1001/jamacardio.2020.5890

41. Daniels CJ, Rajpal S, Greenshields JT, Rosenthal GL, Chung EH, Terrin M, Jeudy J, Mattson SE, Law IH, Borchers J, et al. Prevalence of clinical and subclinical myocarditis in competitive athletes with recent SARS-CoV-2 infection: results from the big ten COVID-19 cardiac registry. *JAMA Cardiol*. 2021;6:1078–1087. https://doi.org/10.1001/jamacardio.2021.2065

42. Krishnan S, Guseh JS, Chukumerije M, Grant AJ, Dean PN, Hsu JJ, Husaini M, Phelan DM, Shah AB,

Stewart K, et al. Racial disparities in sports cardiology: a review. *JAMA Cardiol*. 2024;9:935. https://doi.org/10.1001/jamacardio.2024.1899

43. Malhotra A, Dhutia H, Yeo TJ, Finocchiaro G, Gati S, Bulleros P, Fanton Z, Papatheodorou E, Miles C, Keteepe-Arachi T, et al. Accuracy of the 2017 international recommendations for clinicians who interpret adolescent athletes' ECGs: a cohort study of 11 168 British white and black soccer players. *Br J Sports Med*. 2020;54:739–745. https://doi.org/10.1136/bjsports-2017-098528

44. Herring SA, Putukian M, Kibler WB, LeClere L, Boyajian-O'Neill L, Day MA, Franks RR, Indelicato P, Matuszak J, Miller TL, et al. Team physician consensus statement: return to sport/return to play and the team physician: a team physician consensus statement: 2023 update. *Med Sci Sports Exerc*. 2024;56:767–775. https://doi.org/10.1249/MSS.0000000000003371

45. Moulson N, Petek BJ, Ackerman MJ, Churchill TW, Day SM, Kim JH, Kliethermes SA, Lampert R, Levine BD, Martinez MW, et al. Rationale and design of the ORCCA (Outcomes Registry for Cardiac Conditions in Athletes) study. *J Am Heart Assoc*. 2023;12:e029052. https://doi.org/10.1161/JAHA.122.029052

46. Waase MP, Mutharasan RK, Whang W, DiTullio MR, DiFiori JP, Callahan L, Mancell J, Phelan D, Schwartz A, Homma S, et al. Electrocardiographic Findings in National Basketball Association athletes. *JAMA Cardiol*. 2018;3:69–74. https://doi.org/10.1001/jamacardio.2017.4572

47. Grant A, Krishnan S, Chukumerije M, Guseh JS, Kim JH. Reckoning with race in sports cardiology: a call to action. *Br J Sports Med*. 2023;57:956–957. https://doi.org/10.1136/bjsports-2022-106553

48. Mitchell JH, Haskell WL, Raven PB. Classification of sports. *J Am Coll Cardiol*. 1994;24:864–866. https://doi.org/10.1016/0735-1097(94)90841-9

49. Thompson PD, Myerburg RJ, Levine BD, Udelson JE, Kovacs RJ, American Heart Association Electrocardiography and Arrhythmias Committee of Council on Clinical Cardiology, Council on Cardiovascular Disease in Young, Council on Cardiovascular and Stroke Nursing. Council on Functional Genomics and Translational Biology, and American College of Cardiology. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 8: coronary artery disease: a scientific statement from the American Heart Association and American College of Cardiology. *J Am Coll Cardiol*. 2015;66:2406–2411. https://doi.org/10.1016/j.jacc.2015.09.040

50. Levine BD, Baggish AL, Kovacs RJ, Link MS, Maron MS, Mitchell JH, on behalf of the American Heart Association Electrocardiography and Arrhythmias Committee of the Council on Clinical Cardiology, Council on Cardiovascular Disease in the Young, Council on Cardiovascular and Stroke Nursing. Council on Functional Genomics and Translational Biology, and the American College of Cardiology. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 1: classification of sports: dynamic, static, and impact: a scientific statement from the American Heart Association and American College of Cardiology. *J Am Coll Cardiol*. 2015;66:2350–2355. https://doi.org/10.1016/j.jacc.2015.09.033

51. Croft LB, Belanger A, Miller MA, Roberts A, Goldman ME. Comparison of National Football League linemen versus nonlinemen of left ventricular mass and left atrial size. *Am J Cardiol*. 2008;102:343–347. https://doi.org/10.1016/j.amjcard.2008.03.065

52. Kim JH, Zafonte R, Pascuale-Leon A, Nadler LM, Weisskopf M, Speizer FE, Taylor HA, Baggish AL. American-style football and cardiovascular health. *J Am Heart Assoc*. 2018;7:e008620. https://doi.org/10.1161/JAHA.118.008620

53. Wisloff U, Helgerud J, Hoff J. Strength and endurance of elite soccer players. *Med Sci Sports Exerc*. 1998;30:462–467. https://doi.org/10.1097/00005768-199803000-00019

54. MacDougall JD, Tuxen D, Sale DG, Moroz JR, Sutton JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol*. 1985;58:785–790. https://doi.org/10.1152/jappl.1985.58.3.785

55. Mitchell JH, Victor RG. Neural control of the cardiovascular system: insights from muscle sympathetic nerve recordings in humans. *Med Sci Sports Exerc*. 1996;28:S60–S69. https://doi.org/10.1097/00005768-199610000-00036

56. Haykowsky M, Taylor D, Teo K, Quinney A, Humen D. Left ventricular wall stress during leg-press exercise performed with a brief Valsalva maneuver. *Chest*. 2001;119:150–154. https://doi.org/10.1378/chest.119.1.150

57. Maron BJ, Thompson PD, Ackerman MJ, Balady G, Berger S, Cohen D, Dimeff R, Douglas PS, Glover DW, Hutter AM Jr, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2007;115:1643–1655. https://doi.org/10.1161/circulationaha.107.181423

58. Maron BJ, Haas TS, Ahluwalia A, Murphy CJ, Garberich RF. Demographics and epidemiology of sudden deaths in young competitive athletes: from the United States National Registry. *Am J Med*. 2016;129:1170–1177. https://doi.org/10.1016/j.amjmed.2016.02.031

59. Peterson DF, Kucera K, Thomas LC, Maleszewski J, Siebert D, Lopez-Anderson M, Zigman M, Schattenkerk J, Harmon KG, Drezner JA. Aetiology and incidence of sudden cardiac arrest and death in young competitive athletes in the USA: a 4-year prospective study. *Br J Sports Med*. 2021;55:1196–1203. https://doi.org/10.1136/bjsports-2020-102666

60. MacDonald J, Schaefer M, Stumph J. The preparticipation physical evaluation. *Am Fam Physician*. 2021;103:539–546.

61. Maron BJ, Friedman RA, Kligfield P, Levine BD, Viskin S, Chaitman BR, Okin PM, Saul JP, Salberg L, Van Hare GF, et al, on behalf of the American Heart Association Council on Clinical Cardiology, Advocacy Coordinating Committee. Council on Cardiovascular Disease in the Young, Council on Cardiovascular Surgery and Anesthesia, Council on Epidemiology and Prevention, Council on Functional Genomics and Translational Biology, Council on Quality of Care and Outcomes Research, and American College of Cardiology. Assessment of the 12-lead ECG as a screening test for detection of cardiovascular disease in healthy general populations of young people (12–25 years of age): a scientific statement from the American Heart Association and the American College of Cardiology. *J Am Coll Cardiol*. 2014;64:1479–1514. https://doi.org/10.1016/j.jacc.2014.05.006

62. Edwards JJ, Compton C, Chatrath N, Petek BJ, Baggish A, Borjesson M, Chung E, Corrado D, Drezner JA, Gati S, et al. International criteria for reporting study quality for sudden cardiac arrest/death tool. *J Am Heart Assoc*. 2024;13:e033723. https://doi.org/10.1161/JAHA.123.033723

63. Holst AG, Winkel BG, Theilade J, Kristensen IB, Thomsen JL, Ottesen GL, Svendsen JH, Haunso S, Prescott E, Tfelt-Hansen J. Incidence and etiology of sports-related sudden cardiac death in Denmark: implications for preparticipation screening. *Heart Rhythm*. 2010;7:1365–1371. https://doi.org/10.1016/j.hrthm.2010.05.021

64. Hainline B, Drezner JA, Baggish A, Harmon KG, Emery MS, Myerburg RJ, Sanchez E, Molossi S, Parsons JT, Thompson PD. Interassociation consensus statement on cardiovascular care of college student-athletes. *J Am Coll Cardiol*. 2016;67:2981–2995. https://doi.org/10.1016/j.jacc.2016.03.527

65. Deleted in proof.

66. Dhutia H, Malhotra A, Finocchiaro G, Parpia S, Bhatia R, D'Silva A, Gati S, Mellor G, Narain R, Chandra N, et al. Diagnostic yield and financial implications of a nationwide electrocardiographic screening programme to detect cardiac disease in the young. *Europace*. 2021;23:1295–1301. https://doi.org/10.1093/europace/euab021

67. Harmon KG, Zigman M, Drezner JA. The effectiveness of screening history, physical exam, and ECG to detect potentially lethal cardiac disorders in athletes: a systematic review/meta-analysis. *J Electrocardiol*. 2015;48:329–338. https://doi.org/10.1016/j.jelectrocard.2015.02.001

68. Malhotra A, Dhutia H, Finocchiaro G, Gati S, Beasley I, Clift P, Cowie C, Kenny A, Mayet J, Oxborough D, et al. Outcomes of cardiac screening in adolescent soccer players. *N Engl J Med*. 2018;379:524–534. https://doi.org/10.1056/NEJMoa1714719

69. Parizher G, Phelan D, Ayers C, Goodwin R, Levine B. A video enhanced, electronic modality for pre-participation examination (VPPE) of young athletes. *J Am Coll Cardiol*. 2021;77. https://doi.org/10.1016/s0735-1097(21)04558-7, 3203–3203.

70. Finocchiaro G, Papadakis M, Dhutia H, Zaidi A, Malhotra A, Fabi E, Cappelletto C, Brook J, Papatheodorou E, Ensam B, et al. Electrocardiographic differentiation between 'benign T-wave inversion' and arrhythmogenic right ventricular cardiomyopathy. *Europace*. 2019;21:332–338. https://doi.org/10.1093/europace/euy179

71. Drezner JA, Sharma S, Baggish A, Papadakis M, Wilson MG, Prutkin JM, Gerche AL, Ackerman MJ, Borjesson M, Salerno JC, et al. International criteria for electrocardiographic interpretation in athletes: consensus statement. *Br J Sports Med*. 2017;51:704–731. https://doi.org/10.1136/bjsports-2016-097331

72. Pambo P, Adu-Adadey M, Agbodzakey H, Scharhag J. Electrocardiographic and echocardiographic findings in elite Ghanaian male soccer players. *Clin J Sport Med*. 2021;31:e373–e379. https://doi.org/10.1097/JSM.0000000000000801

73. Pelto HF, Drezner JA. Design and implementation of an emergency action plan for sudden cardiac arrest in sport. *J Cardiovasc Transl Res*. 2020;13:331–338. https://doi.org/10.1007/s12265-020-09988-1

74. Drezner JA, Courson RW, Roberts WO, Mosesso VN, Link MS, Maron BJ. Inter-association task force recommendations on emergency preparedness and management of sudden cardiac arrest in high school and college athletic programs: a consensus statement. *J Athl Train*. 2007;42:143–158.

75. Drezner JA, O'Connor FG, Harmon KG, Fields KB, Asplund CA, Asif IM, Price DE, Dimeff RJ, Bernhardt DT, Roberts WO. AMSSM position statement on cardiovascular preparticipation screening in athletes: current evidence, knowledge gaps, recommendations and future directions. *Br J Sports Med*. 2017;51:153–167. https://doi.org/10.1136/bjsports-2016-096781

76. Petek BJ, Baggish AL. Pre-participation cardiovascular screening in young competitive athletes. *Curr Emerg Hosp Med Rep*. 2020;8:77–89. https://doi.org/10.1007/s40138-020-00214-5

77. Magavern EF, Finocchiaro G, Sharma S, Papadakis M, Borry P. Time out: ethical reflections on medical disqualification of athletes in the context of mandated pre-participation cardiac screening. *Br J Sports Med*. 2018;52:1207–1210. https://doi.org/10.1136/bjsports-2017-097524

78. Churchill TW, O'Kelly AC, Montembeau SC, Kim JH, Guseh JS, Wasfy MM, Dickert NW, Baggish AL. Risk tolerance and eligibility decision-making strategies among young competitive athletes: novel insights into the emerging practice of shared decision-making. *Eur J Prev Cardiol*. 2024;31:e1–e3. https://doi.org/10.1093/eurjpc/zwad250

79. Shapero K, Gier C, Briske K, Spatz ES, Wasfy M, Baggish AL, Pierce S, Ackerman MJ, Lampert R. Experiences of athletes with arrhythmogenic cardiac conditions in returning to play. *Heart Rhythm O2*. 2022;3:133–140. https://doi.org/10.1016/j.hroo.2022.01.009

80. Baugh CM, Kroshus E, Meehan WP 3rd, McGuire TG, Hatfield LA. Accuracy of US college football players' estimates of their risk of concussion or injury. *JAMA Netw Open*. 2020;3:e2031509. https://doi.org/10.1001/jamanetworkopen.2020.31509

81. Baggish AL, Ackerman MJ, Putukian M, Lampert R. Shared decision-making for athletes with cardiovascular disease: practical considerations. *Curr Sports Med Rep*. 2019;18:76–81. https://doi.org/10.1249/JSR.0000000000000575

82. Baggish AL, Ackerman MJ, Lampert R. Competitive sport participation among athletes with heart disease: a call for a paradigm shift in decision-making. *Circulation*. 2017;136:1569–1571. https://doi.org/10.1161/circulationaha.117.029639

83. Martinez MW, Ackerman MJ, Annas GJ, Baggish AL, Day SM, Harmon KG, Kim JH, Levine BD, Putukian M, Lampert R. Sports participation by athletes with cardiovascular disease. *J Am Coll Cardiol*. 2024;83:865–868. https://doi.org/10.1016/j.jacc.2023.12.021

84. Allen LA, Stevenson LW, Grady KL, Goldstein NE, Matlock DD, Arnold RM, Cook NR, Felker GM, Francis GS, Hauptman PJ, et al. on behalf of the American Heart Association Council on Quality of Care and Outcomes Research, Council on Cardiovascular Nursing, Council on Clinical Cardiology, Council on Cardiovascular Radiology and Intervention, and Council on Cardiovascular Surgery and Anesthesia. Decision-making in advanced heart failure: a scientific statement from the American Heart Association. *Circulation*. 2012;125:1928–1952. https://doi.org/10.1161/cir.0b013e31824f2173

85. Birtcher KK, Allen LA, Anderson JL, Bonaca MP, Gluckman TJ, Hussain A, Kosiborod M, Mehta LS, Virani SS. 2022 ACC expert consensus decision pathway for integrating atherosclerotic cardiovascular disease and multimorbidity treatment: a framework for pragmatic, patient-centered care: a report of the American College of Cardiology solution set oversight committee. *J Am Coll Cardiol*. 2023;81:292–317. https://doi.org/10.1016/j.jacc.2022.08.754

86. Ommen SR, Ho CY, Asif IM, Balaji S, Burke MA, Day SM, Dearani JA, Epps-Anderson KC, Evanovich L, Ferrari VA, et al. 2024 AHA/ACC/AMSSM/HRS/PACES/SCMR guideline for the management of hypertrophic cardiomyopathy: a report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2024;83:2324–2405.

87. Ommen SR, Mital S, Burke MA, Day SM, Deswal A, Elliott P, Evanovich LL, Hung J, Joglar JA, Kantor P, et al. 2020 AHA/ACC guideline for the diagnosis and treatment of patients with hypertrophic cardiomyopathy: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2020;76:e159–e240. https://doi.org/10.1016/j.jacc.2020.08.045

88. Virani SS, Newby LK, Arnold SV, Bittner V, Brewer LC, Demeter SH, Dixon DL, Fearon WF, Hess B, Johnson HM, et al, Peer Review Committee Members. 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA guideline for the management of patients with chronic coronary disease: a report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2023;82:833–955. https://doi.org/10.1016/j.jacc.2023.04.003

89. Centers for Medicare & Medicaid Services. National coverage determination (NCD): implantable automatic defibrillators (20.4). Published July 31, 2023. Accessed March 17, 2024. https://www.cms.gov/medicare-coverage-database/details/ncd-details.aspx?NCDId=110

90. Centers for Medicare & Medicaid Services. National coverage determination (NCD): decision memo: percutaneous left atrial appendage (LAA) closure therapy. Accessed March 18, 2024. https://www.cms.gov/medicare-coverage-database/view/ncacal-decision-memo.aspx?proposed=N&NCAId=281

91. *Knapp v* Northwestern University, 942 F. Supp. 1191 (N.D. Ill. 1996).

92. Schoenfeld EM, Kanzaria HK, Quigley DD, Marie PS, Nayyar N, Sabbagh SH, Gress KL, Probst MA. Patient preferences regarding shared decision-making in the emergency department: findings from a multisite survey. *Acad Emerg Med*. 2018;25:1118–1128. https://doi.org/10.1111/acem.13499

93. Basu J, Finocchiaro G, Jayakumar S, Schonfeld J, MacLachlan H, Miles C, Parry-Williams G, Tome M, Papadakis M, Sharma S. Impact of exercise on outcomes and phenotypic expression in athletes with nonobstructive hypertrophic cardiomyopathy. *J Am Coll Cardiol*. 2022;80:1498–1500. https://doi.org/10.1016/j.jacc.2022.08.715

94. Pelliccia A, Lemme E, Maestrini V, Di Paolo FM, Pisicchio C, Di Gioia G, Caselli S. Does sport participation worsen the clinical course of hypertrophic cardiomyopathy? Clinical outcome of hypertrophic cardiomyopathy in athletes. *Circulation*. 2018;137:531–533. https://doi.org/10.1161/CIRCULATIONAHA.117.031725

95. Turkowski KL, Bos JM, Ackerman NC, Rohatgi RK, Ackerman MJ. Return-to-play for athletes with genetic heart diseases. *Circulation*. 2018;137:1086–1088. https://doi.org/10.1161/CIRCULATIONAHA.117.031306

96. Halliday BP, Cleland JGF, Goldberger JJ, Prasad SK. Personalizing risk stratification for sudden death in dilated cardiomyopathy: the past, present, and future. *Circulation*. 2017;136:215–231. https://doi.org/10.1161/CIRCULATIONAHA.116.027134

97. Arbelo E, Protonotarios A, Gimeno JR, Arbustini E, Barriales-Villa R, Basso C, Bezzina CR, Biagini E, Blom NA, de Boer RA, et al, ESC Scientific Document Group. 2023 ESC guidelines for the management of cardiomyopathies. *Eur Heart J*. 2023;44:3503–3626. https://doi.org/10.1093/eurheartj/ehad194

98. Kumar S, Baldinger SH, Gandjbakhch E, Maury P, Sellal JM, Androulakis AF, Waintraub X, Charron P, Rollin A, Richard P, et al. Long-term arrhythmic and nonarrhythmic outcomes of lamin A/C mutation carriers. *J Am Coll Cardiol*. 2016;68:2299–2307. https://doi.org/10.1016/j.jacc.2016.08.058

99. Smith ED, Lakdawala NK, Papoutsidakis N, Aubert G, Mazzanti A, McCanta AC, Agarwal PP, Arscott P, Dellefave-Castillo LM, Vorovich EE, et al. Desmoplakin cardiomyopathy, a fibrotic and inflammatory form of cardiomyopathy distinct from typical dilated or arrhythmogenic right ventricular cardiomyopathy. *Circulation*. 2020;141:1872–1884. https://doi.org/10.1161/CIRCULATIONAHA.119.044934

100. Valdes-Mas R, Gutierrez-Fernandez A, Gomez J, Coto E, Astudillo A, Puente DA, Reguero JR, Alvarez V, Moris C, Leon D, et al. Mutations in filamin C cause a new form of familial hypertrophic cardiomyopathy. *Nat Commun*. 2014;5:5326. https://doi.org/10.1038/ncomms6326

101. Pasotti M, Klersy C, Pilotto A, Marziliano N, Rapezzi C, Serio A, Mannarino S, Gambarin F, Favalli V, Grasso M, et al. Long-term outcome and risk stratification in dilated cardiolaminopathies. *J Am Coll Cardiol*. 2008;52:1250–1260. https://doi.org/10.1016/j.jacc.2008.06.044

102. Skjolsvik ET, Hasselberg NE, Dejgaard LA, Lie OH, Andersen K, Holm T, Edvardsen T, Haugaa KH. Exercise is associated with impaired left ventricular systolic function in patients with lamin A/C genotype. *J Am Heart Assoc*. 2020;9:e012937. https://doi.org/10.1161/JAHA.119.012937

103. Corrado D, van Tintelen PJ, McKenna WJ, Hauer RNW, Anastastakis A, Asimaki A, Basso C, Bauce B, Brunckhorst C, Bucciarelli-Ducci C, et al. Arrhythmogenic right ventricular cardiomyopathy: evaluation of the current diagnostic criteria and differential diagnosis. *Eur Heart J*. 2020;41:1414–1429. https://doi.org/10.1093/eurheartj/ehz669

104. Ruwald AC, Marcus F, Estes NA 3rd, Link M, McNitt S, Polonsky B, Calkins H, Towbin JA, Moss AJ, Zareba W. Association of competitive and recreational sport participation with cardiac events in patients with arrhythmogenic right ventricular cardiomyopathy: results from the North American multidisciplinary study of arrhythmogenic right ventricular cardiomyopathy. *Eur Heart J*. 2015;36:1735–1743. https://doi.org/10.1093/eurheartj/ehv110

105. James CA, Bhonsale A, Tichnell C, Murray B, Russell SD, Tandri H, Tedford RJ, Judge DP, Calkins H. Exercise increases age-related penetrance and arrhythmic risk in arrhythmogenic right ventricular dysplasia/cardiomyopathy-associated desmosomal mutation carriers. *J Am Coll Cardiol*. 2013;62:1290–1297. https://doi.org/10.1016/j.jacc.2013.06.033

106. Saberniak J, Hasselberg NE, Borgquist R, Platonov PG, Sarvari SI, Smith HJ, Ribe M, Holst AG, Edvardsen T, Haugaa KH. Vigorous physical activity impairs myocardial function in patients with arrhythmogenic right ventricular cardiomyopathy and in mutation positive family members. *Eur J Heart Fail*. 2014;16:1337–1344. https://doi.org/10.1002/ejhf.181

107. Sawant AC, Te Riele AS, Tichnell C, Murray B, Bhonsale A, Tandri H, Judge DP, Calkins H, James CA. Safety of American Heart Association-recommended minimum exercise for desmosomal mutation carriers. *Heart Rhythm*. 2016;13:199–207. https://doi.org/10.1016/j.hrthm.2015.08.035

108. Paulin FL, Hodgkinson KA, MacLaughlan S, Stuckless SN, Templeton C, Shah S, Bremner H, Roberts JD, Young TL, Parfrey PS, et al. Exercise and arrhythmic risk in TMEM43 p.S358L arrhythmogenic right ventricular cardiomyopathy. *Heart Rhythm*. 2020;17:1159–1166. https://doi.org/10.1016/j.hrthm.2020.02.028

109. Lie OH, Dejgaard LA, Saberniak J, Rootwelt C, Stokke MK, Edvardsen T, Haugaa KH. Harmful effects of exercise intensity and exercise duration in patients with arrhythmogenic cardiomyopathy. *JACC Clin Electrophysiol*. 2018;4:744–753. https://doi.org/10.1016/j.jacep.2018.01.010

110. La Gerche A, Claessen G, Dymarkowski S, Voigt JU, De Buck F, Vanhees L, Droogne W, Van Cleemput J, Claus P, Heidbuchel H. Exercise-induced right ventricular dysfunction is associated with ventricular arrhythmias in endurance athletes. *Eur Heart J*. 2015;36:1998–2010. https://doi.org/10.1093/eurheartj/ehv202

111. La Gerche A, Robberecht C, Kuiperi C, Nuyens D, Willems R, de Ravel T, Matthijs G, Heidbuchel H. Lower than expected desmosomal gene mutation prevalence in endurance athletes with complex ventricular arrhythmias of right ventricular origin. *Heart*. 2010;96:1268–1274. https://doi.org/10.1136/hrt.2009.189621

112. Sawant AC, Bhonsale A, te Riele AS, Tichnell C, Murray B, Russell SD, Tandri H, Tedford RJ, Judge DP, Calkins H, et al. Exercise has a disproportionate role in the pathogenesis of arrhythmogenic right ventricular dysplasia/cardiomyopathy in patients without desmosomal mutations. *J Am Heart Assoc*. 2014;3:e001471. https://doi.org/10.1161/JAHA.114.001471

113. Faber JW, D'Silva A, Christoffels VM, Jensen B. Lack of morphometric evidence for ventricular compaction in humans. *J Cardiol*. 2021;78:397–405. https://doi.org/10.1016/j.jjcc.2021.03.006

114. Gati S, Chandra N, Bennett RL, Reed M, Kervio G, Panoulas VF, Ghani S, Sheikh N, Zaidi A, Wilson M, et al. Increased left ventricular trabeculation in highly trained athletes: do we need more stringent criteria for the diagnosis of left ventricular non-compaction in athletes? *Heart*. 2013;99:401–408. https://doi.org/10.1136/heartjnl-2012-303418

115. Gati S, Papadakis M, Papamichael ND, Zaidi A, Sheikh N, Reed M, Sharma R, Thilaganathan B, Sharma S. Reversible de novo left ventricular trabeculations in pregnant women: implications for the diagnosis of left ventricular noncompaction in low-risk populations. *Circulation*. 2014;130:475–483. https://doi.org/10.1161/circulationaha.114.008554

116. de la Chica JA, Gomez-Talavera S, Garcia-Ruiz JM, Garcia-Lunar I, Oliva B, Fernandez-Alvira JM, Lopez-Melgar B, Sanchez-Gonzalez J, de la Pompa JL, Mendiguren JM, et al. Association between left ventricular noncompaction and vigorous physical activity. *J Am Coll Cardiol*. 2020;76:1723–1733. https://doi.org/10.1016/j.jacc.2020.08.030

117. Richardson P, McKenna W, Bristow M, Maisch B, Mautner B, O'Connell J, Olsen E, Thiene G, Goodwin J, Gyarfas I, et al. Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the definition and classification of cardiomyopathies. *Circulation*. 1996;93:841–842. https://doi.org/10.1161/01.cir.93.5.841

118. Caforio ALP, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB, Fu M, Heliö T, Heymans S, Jahns R, et al. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases. *Eur Heart J*. 2013;34:2636–2648. https://doi.org/10.1093/eurheartj/eht210

119. Caforio ALP, Calabrese F, Angelini A, Tona F, Vinci A, Bottaro S, Ramondo A, Carturan E, Iliceto S, Thiene G, et al. A prospective study of biopsy-proven myocarditis: prognostic relevance of clinical and aetiopathogenetic features at diagnosis. *Eur Heart J*. 2007;28:1326–1333. https://doi.org/10.1093/eurheartj/ehm076

120. Basso C, Calabrese F, Corrado D, Thiene G. Postmortem diagnosis in sudden cardiac death victims: macroscopic, microscopic and molecular findings. *Cardiovasc Res*. 2001;50:290–300. https://doi.org/10.1016/s0008-6363(01)00261-9

121. Felker GM, Hu W, Hare JM, Hruban RH, Baughman KL, Kasper EK. The spectrum of dilated cardiomyopathy: the Johns Hopkins experience with 1,278 patients. *Medicine (Baltimore)*. 1999;78:270–283. https://doi.org/10.1097/00005792-199907000-00005

122. Georgiopoulos G, Figliozzi S, Sanguineti F, Aquaro GD, Di Bella G, Stamatelopoulos K, Chiribiri A, Garot J, Masci PG, Ismail TF. Prognostic impact of late gadolinium enhancement by cardiovascular magnetic resonance in myocarditis. *Circ Cardiovasc Imaging*. 2021;14:e011492. https://doi.org/10.1161/circimaging.120.011492

123. Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U, Kindermann I, Gutberlet M, Cooper LT, Liu P, et al. Cardiovascular magnetic resonance in nonischemic myocardial inflammation: expert recommendations. *J Am Coll Cardiol*. 2018;72:3158–3176. https://doi.org/10.1016/j.jacc.2018.09.072

124. Kotanidis CP, Bazmpani M-A, Haidich A-B, Karvounis C, Antoniades C, Karamitsos TD. Diagnostic accuracy of cardiovascular magnetic resonance in acute myocarditis: a systematic review and meta-analysis. *JACC Cardiovasc Imaging*. 2018;11:1583–1590. https://doi.org/10.1016/j.jcmg.2017.12.008

125. Friedrich MG, Sechtem U, Schulz-Menger J, Holmvang G, Alakija P, Cooper LT, White JA, Abdel-Aty H, Gutberlet M, Prasad S, et al. Cardiovascular magnetic resonance in myocarditis: a JACC white paper. *J Am Coll Cardiol*. 2009;53:1475–1487. https://doi.org/10.1016/j.jacc.2009.02.007

126. Mahrholdt H, Goedecke C, Wagner A, Meinhardt G, Athanasiadis A, Vogelsberg H, Fritz P, Klingel K, Kandolf R, Sechtem U. Cardiovascular magnetic resonance assessment of human myocarditis: a comparison to histology and molecular pathology. *Circulation*. 2004;109:1250–1258. https://doi.org/10.1161/01.cir.0000118493.13323.81

127. Gulati A, Jabbour A, Ismail TF, Guha K, Khwaja J, Raza S, Morarji K, Brown TD, Ismail NA, Dweck MR. Association of fibrosis with mortality and sudden cardiac death in patients with nonischemic dilated cardiomyopathy. *JAMA*. 2013;309:896–908. https://doi.org/10.1001/jama.2013.1363

128. Ammirati E, Cipriani M, Moro C, Raineri C, Pini D, Sormani P, Mantovani R, Varrenti M, Pedrotti P, Conca C, et al, Registro Lombardo delle Miocarditi. Clinical presentation and outcome in a contemporary cohort of patients with acute myocarditis: multicenter Lombardy registry. *Circulation*. 2018;138:1088–1099. https://doi.org/10.1161/CIRCULATIONAHA.118.035319

129. Maron BJ, Udelson JE, Bonow RO, Nishimura RA, Ackerman MJ, Estes NAM 3rd, Cooper LT Jr, Link MS, Maron MS, American Heart Association Electrocardiography and Arrhythmias Committee of Council on Clinical Cardiology, Council on Cardiovascular Disease in Young, Council on Cardiovascular and Stroke Nursing. Council on Functional Genomics and Translational Biology, and American College of Cardiology. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 3: hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy and other cardiomyopathies, and myocarditis: a scientific statement from the American Heart Association and American College of Cardiology. *J Am Coll Cardiol*. 2015;66:2362–2371. https://doi.org/10.1016/j.jacc.2015.09.035

130. Pelliccia A, Solberg EE, Papadakis M, Adami PE, Biffi A, Caselli S, La Gerche A, Niebauer J, Pressler A, Schmied CM, et al. Recommendations for participation in competitive and leisure time sport in athletes with cardiomyopathies, myocarditis, and pericarditis: position statement of the Sport Cardiology Section of the European Association of Preventive Cardiology (EAPC). *Eur Heart J*. 2019;40:19–33. https://doi.org/10.1093/eurheartj/ehy730

131. Grant JK, Shah NP. The impact of physical activity on pericarditis. *Curr Cardiol Rep*. 2021;23:150. https://doi.org/10.1007/s11886-021-01578-0

132. Shah NP, Verma BR, Ala CK, Khayata M, Phelan D, Imazio M, Klein AL. Exercise is good for the heart but not for the inflamed pericardium? *JACC Cardiovasc Imaging*. 2019;12:1880–1881. https://doi.org/10.1016/j.jcmg.2019.01.022

133. Sivalokanathan S, Chokshi N. Pericarditis in Athletes: Approach to Exercise Restriction. Published September 7, 2022. Accessed February 1, 2024. https://www.acc.org/Latest-in-Cardiology/Articles/2022/09/06/11/53/Pericarditis-in-Athletes

134. Moulson N, Petek BJ, Drezner JA, Harmon KG, Kliethermes SA, Patel MR, Baggish AL, Outcomes Registry for Cardiac Conditions in Athletes Investigators. SARS-CoV-2 cardiac involvement in young competitive athletes. *Circulation*. 2021;144:256–266. https://doi.org/10.1161/CIRCULATIONAHA.121.054824

135. Thaweethai T, Jolley SE, Karlson EW, Levitan EB, Levy B, McComsey GA, McCorkell L, Nadkarni GN, Parthasarathy S, Singh U, et al, RECOVER Consortium.

Development of a definition of postacute sequelae of SARS-CoV-2 infection. *JAMA*. 2023;329:1934–1946. https://doi.org/10.1001/jama.2023.8823

136. Grigoratos C, Pantano A, Meschisi M, Gaeta R, Ait-Ali L, Barison A, Todiere G, Festa P, Sinagra G, Aquaro GD. Clinical importance of late gadolinium enhancement at right ventricular insertion points in otherwise normal hearts. *Int J Cardiovasc Imaging*. 2020;36:913–920. https://doi.org/10.1007/s10554-020-01783-y

137. La Gerche A, Burns AT, Mooney DJ, Inder WJ, Taylor AJ, Bogaert J, Macisaac AI, Heidbuchel H, Prior DL. Exercise-induced right ventricular dysfunction and structural remodelling in endurance athletes. *Eur Heart J*. 2012;33:998–1006. https://doi.org/10.1093/eurheartj/ehr397

138. Clark DE, Parikh A, Dendy JM, Diamond AB, George-Durrett K, Fish FA, Slaughter JC, Fitch W, Hughes SG, Soslow JH. COVID-19 Myocardial Pathology Evaluation in Athletes With Cardiac Magnetic Resonance (COMPETE CMR). *Circulation*. 2021;143:609–612. https://doi.org/10.1161/CIRCULATIONAHA.120.052573

139. Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP 3rd, Gentile F, Jneid H, Krieger EV, Mack M, McLeod C, et al. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *Circulation*. 2021;143:e72–e227. https://doi.org/10.1161/CIR.0000000000000923

140. Zoghbi WA, Adams D, Bonow RO, Enriquez-Sarano M, Foster E, Grayburn PA, Hahn RT, Han Y, Hung J, Lang RM, et al. Recommendations for noninvasive evaluation of native valvular regurgitation: a report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance. *J Am Soc Echocardiogr*. 2017;30:303–371. https://doi.org/10.1016/j.echo.2017.01.007

141. Baumgartner H, Hung J, Bermejo J, Chambers JB, Evangelista A, Griffin BP, Iung B, Otto CM, Pellikka PA, Quinones M, American Society of Echocardiography. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. *J Am Soc Echocardiogr*. 2009;22:1–23. https://doi.org/10.1016/j.echo.2008.11.029. quiz 101.

142. Pelliccia A, Culasso F, Di Paolo FM, Maron BJ. Physiologic left ventricular cavity dilatation in elite athletes. *Ann Intern Med*. 1999;130:23–31. https://doi.org/10.7326/0003-4819-130-1-199901050-00005

143. Maron BJ. Sudden death in young athletes. *N Engl J Med*. 2003;349:1064–1075. https://doi.org/10.1056/NEJMra022783

144. Freed LA, Levy D, Levine RA, Larson MG, Evans JC, Fuller DL, Lehman B, Benjamin EJ. Prevalence and clinical outcome of mitral-valve prolapse. *N Engl J Med*. 1999;341:1–7. https://doi.org/10.1056/NEJM199907013410101

145. Basso C, Perazzolo Marra M, Rizzo S, De Lazzari M, Giorgi B, Cipriani A, Frigo AC, Rigato I, Migliore F, Pilichou K, et al. Arrhythmic mitral valve prolapse and sudden cardiac death. *Circulation*. 2015;132:556–566. https://doi.org/10.1161/CIRCULATIONAHA.115.016291

146. Essayagh B, Sabbag A, Antoine C, Benfari G, Yang L-T, Maalouf J, Asirvatham S, Michelena H, Enriquez-Sarano M. Presentation and outcome of arrhythmic mitral valve prolapse. *J Am Coll Cardiol*. 2020;76:637–649. https://doi.org/10.1016/j.jacc.2020.06.029

147. Dejgaard LA, Skjølsvik ET, Lie OH, Ribe M, Stokke MK, Hegbom F, Scheirlynck ES, Gjertsen E, Andresen K, Helle-Valle TM, et al. The mitral annulus disjunction arrhythmic syndrome. *J Am Coll Cardiol*. 2018;72:1600–1609. https://doi.org/10.1016/j.jacc.2018.07.070

148. Perazzolo Marra M, Basso C, De Lazzari M, Rizzo S, Cipriani A, Giorgi B, Lacognata C, Rigato I, Migliore F, Pilichou K, et al. Morphofunctional abnormalities of mitral annulus and arrhythmic mitral valve prolapse. *Circ Cardiovasc Imaging*. 2016;9:e005030. https://doi.org/10.1161/CIRCIMAGING.116.005030

149. Ermakov S, Gulhar R, Lim L, Bibby D, Fang Q, Nah G, Abraham TP, Schiller NB, Delling FN. Left ventricular mechanical dispersion predicts arrhythmic risk in mitral valve prolapse. *Heart*. 2019;105:1063–1069. https://doi.org/10.1136/heartjnl-2018-314269

150. Basso C, Iliceto S, Thiene G, Perazzolo Marra M. Mitral valve prolapse, ventricular arrhythmias, and sudden death. *Circulation*. 2019;140:952–964. https://doi.org/10.1161/CIRCULATIONAHA.118.034075

151. Kato H, Sugimura T, Akagi T, Sato N, Hashino K, Maeno Y, Kazue T, Eto G, Yamakawa R. Long-term consequences of Kawasaki disease: a 10-to 21-year follow-up study of 594 patients. *Circulation*. 1996;94:1379–1385. https://doi.org/10.1161/01.cir.94.6.1379

152. Lynge TH, Jeppesen AG, Winkel BG, Glinge C, Schmidt MR, Søndergaard L, Risgaard B, Tfelt-Hansen J. Nationwide study of sudden cardiac death in people with congenital heart defects aged 0 to 35 years. *Circ Arrhythm Electrophysiol*. 2018;11:e005757. https://doi.org/10.1161/CIRCEP.117.005757

153. Finocchiaro G, Radaelli D, D'Errico S, Papadakis M, Behr ER, Sharma S, Westaby J, Sheppard MN. Sudden cardiac death among adolescents in the United Kingdom. *J Am Coll Cardiol*. 2023;81:1007–1017. https://doi.org/10.1016/j.jacc.2023.01.041

154. Finocchiaro G, Papadakis M, Robertus JL, Dhutia H, Steriotis AK, Tome M, Mellor G, Merghani A, Malhotra A, Behr E, et al. Etiology of sudden death in sports: insights from a United Kingdom regional registry. *J Am Coll Cardiol*. 2016;67:2108–2115. https://doi.org/10.1016/j.jacc.2016.02.062

155. Stout KK, Daniels CJ, Aboulhosn JA, Bozkurt B, Broberg CS, Colman JM, Crumb SR, Dearani JA, Fuller S, Gurvitz M, et al. 2018 AHA/ACC guideline for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2019;73:e81–e192. https://doi.org/10.1016/j.jacc.2018.08.1029

156. Geva T, Mulder B, Gauvreau K, Babu-Narayan SV, Wald RM, Hickey K, Powell AJ, Gatzoulis MA, Valente AM. Preoperative predictors of death and sustained ventricular tachycardia after pulmonary valve replacement in patients with repaired tetralogy of Fallot enrolled in the INDICATOR cohort. *Circulation*. 2018;138:2106–2115. https://doi.org/10.1161/circulationaha.118.034740

157. Saiki H, Sugimoto M, Senzaki H. Exercise-induced cardiopulmonary arrest in a child with aortic stenosis. *Cardiol Young*. 2016;26:1013–1016. https://doi.org/10.1017/s1047951116000330

158. Santana S, Gidding SS, Xie S, Jiang T, Kharouf R, Robinson BW. Correlation of echocardiogram and exercise test data in children with aortic stenosis. *Pediatr Cardiol*. 2019;40:1516–1522. https://doi.org/10.1007/s00246-019-02177-1

159. Meijs TA, Minderhoud SCS, Muller SA, de Winter RJ, Mulder BJM, van Melle JP, Hoendermis ES, van Dijk APJ, Zuithoff NPA, Krings GJ, et al. Cardiovascular morbidity and mortality in adult patients with repaired aortic coarctation. *J Am Heart Assoc*. 2021;10:e023199. https://doi.org/10.1161/JAHA.121.023199

160. Broberg CS, van Dissel A, Minnier J, Aboulhosn J, Kauling RM, Ginde S, Krieger EV, Rodriguez F 3rd, Gupta T, Shah S, et al. Long-term outcomes after atrial switch operation for transposition of the great arteries. *J Am Coll Cardiol*. 2022;80:951–963. https://doi.org/10.1016/j.jacc.2022.06.020

161. Tran DL, Celermajer DS, Ayer J, Grigg L, Clendenning C, Hornung T, Justo R, Davis GM, d'Udekem Y, Cordina R. The "super-Fontan" phenotype: characterizing factors associated with high physical performance. *Front Cardiovasc Med*. 2021;8:764273. https://doi.org/10.3389/fcvm.2021.764273

162. Moon J, Lancaster T, Sood V, Si MS, Ohye RG, Romano JC. Long-term impact of anatomic subtype in hypoplastic left heart syndrome after Fontan completion. *J Thorac Cardiovasc Surg*. 2023;168:193–201.e3. https://doi.org/10.1016/j.jtcvs.2023.11.008

163. Elias P, Poh CL, du Plessis K, Zannino D, Rice K, Radford DJ, Bullock A, Wheaton GR, Celermajer DS, d'Udekem Y. Long-term outcomes of single-ventricle palliation for pulmonary atresia with intact ventricular septum: Fontan survivors remain at risk of late myocardial ischaemia and death. *Eur J Cardiothorac Surg*. 2018;53:1230–1236. https://doi.org/10.1093/ejcts/ezy038

164. Arvanitaki A, Gatzoulis MA, Opotowsky AR, Khairy P, Dimopoulos K, Diller GP, Giannakoulas G, Brida M, Griselli M, Grünig E, et al. Eisenmenger syndrome: JACC state-of-the-art review. *J Am Coll Cardiol*. 2022;79:1183–1198. https://doi.org/10.1016/j.jacc.2022.01.022

165. Gaudino M, Di Franco A, Arbustini E, Bacha E, Bates ER, Cameron DE, Cao D, David TE, De Paulis R, El-Hamamsy I, et al. Management of adults with anomalous aortic origin of the coronary arteries: state-of-the-art review. *Ann Thorac Surg*. 2023;116:1124–1141. https://doi.org/10.1016/j.athoracsur.2023.09.025

166. Stephens EH, Jegatheeswaran A, Brothers JA, Ghobrial J, Karamlou T, Francois CJ, Krishnamurthy R, Dearani JA, Binsalamah Z, Molossi S, et al. Anomalous aortic origin of a coronary artery. *Ann Thorac Surg*. 2024;117:1074–1086. https://doi.org/10.1016/j.athoracsur.2024.01.016

167. Molossi S, Doan T, Sachdeva S. Anomalous coronary arteries: a state-of-the-art approach. *Card Electrophysiol Clin*. 2024;16:51–69. https://doi.org/10.1016/j.ccep.2023.09.007

168. Jegatheeswaran A, Devlin PJ, McCrindle BW, Williams WG, Jacobs ML, Blackstone EH, DeCampli WM, Caldarone CA, Gaynor JW, Kirklin JK, et al. Features associated with myocardial ischemia in anomalous aortic origin of a coronary artery: a Congenital Heart Surgeons Society study. *J Thorac*

Cardiovasc Surg. 2019;158:822–834.e3. https://doi.org/10.1016/j.jtcvs.2019.02.122

169. Doan TT, Sachdeva S, Bonilla-Ramirez C, Reaves-O'Neal DL, Masand P, Mery CM, Binsalamah Z, Heinle JH, Molossi S. Ischemia in anomalous aortic origin of a right coronary artery: large pediatric cohort medium-term outcomes. *Circ Cardiovasc Interv*. 2023;16:e012631. https://doi.org/10.1161/CIRCINTERVENTIONS.122.012631

170. Doan TT, Wilkes JK, Reaves O, Neal DL, Bonilla-Ramirez C, Sachdeva S, Masand P, Mery CM, Binsalamah Z, Heinle JS, Molossi S. Clinical presentation and medium-term outcomes of children with anomalous aortic origin of the left coronary artery: high-risk features beyond interarterial course. *Circ Cardiovasc Interv*. 2023;16:e012635. https://doi.org/10.1161/circinterventions.122.012635

171. Thomas AS, Chan A, Alsoufi B, Vinocur JM, Kochilas L. Long-term outcomes of children operated on for anomalous left coronary artery from the pulmonary artery. *Ann Thorac Surg*. 2022;113:1223–1230. https://doi.org/10.1016/j.athoracsur.2021.07.053

172. Triglia LT, Guariento A, Zanotto L, Zanotto L, Cattapan C, Hu R, Zhang H, Herbst C, Hörer J, Sarris G, et al. Anomalous left coronary artery from pulmonary artery repair: outcomes from the European Congenital Heart Surgeons Association database. *J Card Surg*. 2021;36:1910–1916. https://doi.org/10.1111/jocs.15448

173. Schmitt B, Bauer S, Kutty S, Nordmeyer S, Nasseri B, Berger F, Alexi-Meskishvili V. Myocardial perfusion, scarring, and function in anomalous left coronary artery from the pulmonary artery syndrome: a long-term analysis using magnetic resonance imaging. *Ann Thorac Surg*. 2014;98:1425–1436. https://doi.org/10.1016/j.athoracsur.2014.05.031

174. Fratz S, Hager A, Schreiber C, Schwaiger M, Hess J, Stern HC. Long-term myocardial scarring after operation for anomalous left coronary artery from the pulmonary artery. *Ann Thorac Surg*. 2011;92:1761–1765. https://doi.org/10.1016/j.athoracsur.2011.06.021

175. Sternheim D, Power DA, Samtani R, Kini A, Fuster V, Sharma S. Myocardial bridging: diagnosis, functional assessment, and management: JACC state-of-the-art review. *J Am Coll Cardiol*. 2021;78:2196–2212. https://doi.org/10.1016/j.jacc.2021.09.859

176. Hemmati P, Schaff HV, Dearani JA, Daly RC, Lahr BD, Lerman A. Clinical outcomes of surgical unroofing of myocardial bridging in symptomatic patients. *Ann Thorac Surg*. 2020;109:452–457. https://doi.org/10.1016/j.athoracsur.2019.07.005

177. Maeda K, Schnittger I, Murphy DJ, Tremmel JA, Boyd JH, Peng L, Okada K, Pargaonkar VS, Hanley FL, Mitchell RS, et al. Surgical unroofing of hemodynamically significant myocardial bridges in a pediatric population. *J Thorac Cardiovasc Surg*. 2018;156:1618–1626. https://doi.org/10.1016/j.jtcvs.2018.01.081

178. Doan TT, Bonilla-Ramirez C, Eilers L, Reaves-O'Neal D, Sachdeva S, Dolgner SJ, Masand PM, Gowda S, Qureshi AM, Binsalamah Z, et al. Myocardial bridges in a pediatric population: outcomes following a standardized approach. *J Thorac Cardiovasc Surg*. 2024;168:1203–1212. https://doi.org/10.1016/j.jtcvs.2023.12.024

179. Harris KM, Tung M, Haas TS, Maron BJ. Underrecognition of aortic and aortic valve disease and the risk for sudden death in competitive athletes. *J Am Coll Cardiol*. 2015;65:860–862. https://doi.org/10.1016/j.jacc.2014.09.094

180. Kim JH, Dickert NW. Athletes with cardiovascular disease and competitive sports participation eligibility: progress and challenges ahead. *JAMA Cardiol*. 2022;7:663–664. https://doi.org/10.1001/jamacardio.2022.0806

181. Pelliccia A, Di Paolo FM, De Blasiis E, Quattrini FM, Pisicchio C, Guerra E, Culasso F, Maron BJ. Prevalence and clinical significance of aortic root dilation in highly trained competitive athletes. *Circulation*. 2010;122:698–706. https://doi.org/10.1161/CIRCULATIONAHA.109.901074

182. Gati S, Malhotra A, Sedgwick C, Papamichael N, Dhutia H, Sharma R, Child AH, Papadakis M, Sharma S. Prevalence and progression of aortic root dilatation in highly trained young athletes. *Heart*. 2019;105:920–925. https://doi.org/10.1136/heartjnl-2018-314288

183. Thijssen CGE, Bons LR, Gokalp AL, Van Kimmenade RRJ, Mokhles MM, Pelliccia A, Takkenberg JJM, Roos-Hesselink JW. Exercise and sports participation in patients with thoracic aortic disease: a review. *Expert Rev Cardiovasc Ther*. 2019;17:251–266. https://doi.org/10.1080/14779072.2019.1585807

184. Tso JV, Turner CG, Liu C, Prabakaran G, Jackson M, Galante A, Gilson CR, Clark C, Williams BR 3rd, Quyyumi AA, et al. Longitudinal aortic root dilatation in collegiate American-style football athletes. *J Am Heart Assoc*. 2023;12:e030314. https://doi.org/10.1161/JAHA.122.030314

185. Boraita A, Heras ME, Morales F, Marina-Breysse M, Canda A, Rabadan M, Barriopedro MI, Varela A, de la Rosa A, Tunon J. Reference values of aortic root in male and female white elite athletes according to sport. *Circ Cardiovasc Imaging*. 2016;9:e005292. https://doi.org/10.1161/CIRCIMAGING.116.005292

186. Isselbacher EM, Preventza O, Black JH 3rd, Augoustides JG, Beck AW, Bolen MA, Braverman AC, Bray BE, Brown-Zimmerman MM, Chen EP, et al. 2022 ACC/AHA guideline for the diagnosis and management of aortic disease: a report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2022;80:e223–e393. https://doi.org/10.1016/j.jacc.2022.08.004

187. Devereux RB, de Simone G, Arnett DK, Best LG, Boerwinkle E, Howard BV, Kitzman D, Lee ET, Mosley TH Jr, Weder A, et al. Normal limits in relation to age, body size and gender of two-dimensional echocardiographic aortic root dimensions in persons >/=15 years of age. *Am J Cardiol*. 2012;110:1189–1194. https://doi.org/10.1016/j.amjcard.2012.05.063

188. Saura D, Dulgheru R, Caballero L, Bernard A, Kou S, Gonjilashvili N, Athanassopoulos GD, Barone D, Baroni M, Cardim N, et al. Two-dimensional transthoracic echocardiographic normal reference ranges for proximal aorta dimensions: results from the EACVI NORRE study. *Eur Heart J Cardiovasc Imaging*. 2017;18:167–179. https://doi.org/10.1093/ehjci/jew053

189. Chaddha A, Kline-Rogers E, Braverman AC, Erickson SR, Jackson EA, Franklin BA, Woznicki EM, Jabara JT, Montgomery DG, Eagle KA. Survivors of aortic dissection: activity, mental health, and sexual function. *Clin Cardiol*. 2015;38:652–659. https://doi.org/10.1002/clc.22418

190. Solomon MD, Leong T, Sung SH, Lee C, Allen JG, Huh J, LaPunzina P, Lee H, Mason D, Melikian V, et al. Association of thoracic aortic aneurysm size with long-term patient outcomes: the KP-TAA study. *JAMA Cardiol*. 2022;7:1160–1169. https://doi.org/10.1001/jamacardio.2022.3305

191. Boraita A, Morales-Acuna F, Marina-Breysse M, Heras ME, Canda A, Fuentes ME, Chacon A, Diaz-Gonzalez L, Rabadan M, Parra Laca B, et al. Bicuspid aortic valve behaviour in elite athletes. *Eur Heart J Cardiovasc Imaging*. 2019;20:772–780. https://doi.org/10.1093/ehjci/jez001

192. Tweet MS, Hayes SN, Pitta SR, Simari RD, Lerman A, Lennon RJ, Gersh BJ, Khambatta S, Best PJ, Rihal CS, et al. Clinical features, management, and prognosis of spontaneous coronary artery dissection. *Circulation*. 2012;126:579–588. https://doi.org/10.1161/CIRCULATIONAHA.112.105718

193. Fahmy P, Prakash R, Starovoytov A, Boone R, Saw J. Pre-disposing and precipitating factors in men with spontaneous coronary artery dissection. *JACC Cardiovasc Interv*. 2016;9:866–868. https://doi.org/10.1016/j.jcin.2016.02.024

194. Hayes SN, Kim ESH, Saw J, Adlam D, Arslanian-Engoren C, Economy KE, Ganesh SK, Gulati R, Lindsay ME, Mieres JH, et al, on behalf of the American Heart Association Council on Peripheral Vascular Disease; Council on Clinical Cardiology; Council on Cardiovascular and Stroke Nursing; Council on Genomic and Precision Medicine; and Stroke Council. Spontaneous coronary artery dissection: current state of the science: a scientific statement from the American Heart Association. *Circulation*. 2018;137:e523–e557. https://doi.org/10.1161/CIR.0000000000000564

195. Verdile L, Maron BJ, Pelliccia A, Spataro A, Santini M, Biffi A. Clinical significance of exercise-induced ventricular tachyarrhythmias in trained athletes without cardiovascular abnormalities. *Heart Rhythm*. 2015;12:78–85. https://doi.org/10.1016/j.hrthm.2014.09.009

196. Biffi A, Pelliccia A, Verdile L, Fernando F, Spataro A, Caselli S, Santini M, Maron BJ. Long-term clinical significance of frequent and complex ventricular tachyarrhythmias in trained athletes. *J Am Coll Cardiol*. 2002;40:446–452. https://doi.org/10.1016/s0735-1097(02)01977-0

197. Steriotis AK, Nava A, Rigato I, Mazzotti E, Daliento L, Thiene G, Basso C, Corrado D, Bauce B. Noninvasive cardiac screening in young athletes with ventricular arrhythmias. *Am J Cardiol*. 2013;111:557–562. https://doi.org/10.1016/j.amjcard.2012.10.044

198. Tobert KE, Bos JM, Cannon BC, Ackerman MJ. Outcomes of athletes with genetic heart diseases and implantable cardioverter-defibrillators who chose to return to play. *Mayo Clin Proc*. 2022;97:2028–2039. https://doi.org/10.1016/j.mayocp.2022.03.024

199. La Gerche A, Brosnan MJ. Cardiovascular effects of performance-enhancing drugs. *Circulation*. 2017;135:89–99. https://doi.org/10.1161/CIRCULATIONAHA.116.022535

200. Andrade JG, Wells GA, Deyell MW, Bennett M, Essebag V, Champagne J, Roux JF, Yung D, Skanes A, Khaykin Y, et al, EARLY-AF Investigators. Cryoablation or drug therapy for initial treatment of atrial fibrillation. *N Engl J Med*. 2021;384:305–315. https://doi.org/10.1056/NEJMoa2029980

201. Wazni OM, Dandamudi G, Sood N, Hoyt R, Tyler J, Durrani S, Niebauer M, Makati K, Halperin B, Gauri A, et al. Cryoballoon ablation as initial therapy for atrial

fibrillation. *N Engl J Med*. 2021;384:316–324. https://doi.org/10.1056/nejmoa2029554

202. Hsu JC, Darden D, Du C, Marine JE, Nichols S, Marcus GM, Natale A, Noseworthy PA, Selzman KA, Varosy P, et al. Initial findings from the national cardiovascular data registry of atrial fibrillation ablation procedures. *J Am Coll Cardiol*. 2023;81:867–878. https://doi.org/10.1016/j.jacc.2022.11.060

203. Etheridge SP, Escudero CA, Blaufox AD, Law IH, Dechert-Crooks BE, Stephenson EA, Dubin AM, Ceresnak SR, Motonaga KS, Skinner JR, et al. Life-threatening event risk in children with Wolff-Parkinson-White syndrome: a multicenter international study. *JACC Clin Electrophysiol*. 2018;4:433–444. https://doi.org/10.1016/j.jacep.2017.10.009

204. Obeyesekere MN, Leong-Sit P, Massel D, Manlucu J, Modi S, Krahn AD, Skanes AC, Yee R, Gula LJ, Klein GJ. Risk of arrhythmia and sudden death in patients with asymptomatic preexcitation: a meta-analysis. *Circulation*. 2012;125:2308–2315. https://doi.org/10.1161/CIRCULATIONAHA.111.055350

205. Janson CM, Millenson ME, Okunowo O, Dai D, Christmyer Z, Tan RB, Ramesh Iyer V, Shah MJ, O'Byrne ML. Incidence of life-threatening events in children with Wolff-Parkinson-White syndrome: Analysis of a large claims database. *Heart Rhythm*. 2022;19:642–647. https://doi.org/10.1016/j.hrthm.2021.12.009

206. Al-Khatib SM, Arshad A, Balk EM, Das SR, Hsu JC, Joglar JA, Page RL. Risk stratification for arrhythmic events in patients with asymptomatic pre-excitation: a systematic review for the 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *J Am Coll Cardiol*. 2016;67:1624–1638. https://doi.org/10.1016/j.jacc.2015.09.018

207. Pappone C, Santinelli V, Rosanio S, Vicedomini G, Nardi S, Pappone A, Tortoriello V, Manguso F, Mazzone P, Gulletta S, et al. Usefulness of invasive electrophysiologic testing to stratify the risk of arrhythmic events in asymptomatic patients with Wolff-Parkinson-White pattern: results from a large prospective long-term follow-up study. *J Am Coll Cardiol*. 2003;41:239–244. https://doi.org/10.1016/s0735-1097(02)02706-7

208. Pappone C, Vicedomini G, Manguso F, Saviano M, Baldi M, Pappone A, Ciaccio C, Giannelli L, Ionescu B, Petretta A, et al. Wolff-Parkinson-White syndrome in the era of catheter ablation: insights from a registry study of 2169 patients. *Circulation*. 2014;130:811–819. https://doi.org/10.1161/circulationaha.114.011154

209. Pappone C, Vicedomini G, Manguso F, Baldi M, Pappone A, Petretta A, Vitale R, Saviano M, Ciaccio C, Giannelli L, et al. Risk of malignant arrhythmias in initially symptomatic patients with Wolff-Parkinson-White syndrome: results of a prospective long-term electrophysiological follow-up study. *Circulation*. 2012;125:661–668. https://doi.org/10.1161/CIRCULATIONAHA.111.065722

210. Olen MM, Baysa SJ, Rossi A, Kanter RJ, Fishberger SB. Wolff-Parkinson-White syndrome: a stepwise deterioration to sudden death. *Circulation*. 2016;133:105–106. https://doi.org/10.1161/CIRCULATIONAHA.115.019703

211. O'Connor FG, Levine BD, Childress MA, Asplundh CA, Oriscello RG. Practical management: a systematic approach to the evaluation of exercise-related syncope in athletes. *Clin J Sport Med*. 2009;19:429–434. https://doi.org/10.1097/JSM.0b013e3181b732c3

212. Shen W-K, Sheldon RS, Benditt DG, Cohen MI, Forman DE, Goldberger ZD, Grubb BP, Hamdan MH, Krahn AD, Link MS, et al. 2017 ACC/AHA/HRS guideline for the evaluation and management of patients with syncope: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society [published correction appears in Circulation. 2017;136:e271–e272]. *J Am Coll Cardiol*. 2017;70:e39–e110. https://doi.org/10.1016/j.jacc.2017.03.003

213. Semsarian C, Gray B, Haugaa KH, Lampert R, Sharma S, Kovacic JC. Athletic activity for patients with hypertrophic cardiomyopathy and other inherited cardiovascular diseases: JACC focus seminar 3/4. *J Am Coll Cardiol*. 2022;80:1268–1283. https://doi.org/10.1016/j.jacc.2022.07.013

214. Aziz PF, Sweeten T, Vogel RL, Bonney WJ, Henderson J, Patel AR, Shah MJ. Sports participation in genotype positive children with long QT syndrome. *JACC Clin Electrophysiol*. 2015;1:62–70. https://doi.org/10.1016/j.jacep.2015.03.006

215. Johnson JN, Ackerman MJ. Competitive sports participation in athletes with congenital long QT syndrome. *JAMA*. 2012;308:764–765. https://doi.org/10.1001/jama.2012.9334

216. Olde Nordkamp LR, Postema PG, Knops RE, van Dijk N, Limpens J, Wilde AA, de Groot JR. Implantable cardioverter-defibrillator harm in young patients with inherited arrhythmia syndromes: a systematic review and meta-analysis of inappropriate shocks and complications. *Heart Rhythm*. 2016;13:443–454. https://doi.org/10.1016/j.hrthm.2015.09.010

217. Scherr J, Schuster T, Pressler A, Roeh A, Christle J, Wolfarth B, Halle M. Repolarization perturbation and hypomagnesemia after extreme exercise. *Med Sci Sports Exerc*. 2012;44:1637–1643. https://doi.org/10.1249/MSS.0b013e318258aaf4

218. Amin AS, Herfst LJ, Delisle BP, Klemens CA, Rook MB, Bezzina CR, Underkofler HA, Holzem KM, Ruijter JM, Tan HL, et al. Fever-induced QTc prolongation and ventricular arrhythmias in individuals with type 2 congenital long QT syndrome. *J Clin Invest*. 2008;118:2552–2561. https://doi.org/10.1172/JCI35337

219. Niaz T, Bos JM, Sorensen KB, Moir C, Ackerman MJ. Left cardiac sympathetic denervation monotherapy in patients with congenital long QT syndrome. *Circ Arrhythm Electrophysiol*. 2020;13:e008830. https://doi.org/10.1161/CIRCEP.120.008830

220. Ackerman MJ, Tester DJ, Porter CJ, Edwards WD. Molecular diagnosis of the inherited long-QT syndrome in a woman who died after near-drowning. *N Engl J Med*. 1999;341:1121–1125. https://doi.org/10.1056/NEJM199910073411504

221. Ackerman MJ, Tester DJ, Porter CJ. Swimming, a gene-specific arrhythmogenic trigger for inherited long QT syndrome. *Mayo Clin Proc*. 1999;74:1088–1094. https://doi.org/10.4065/74.11.1088

222. Roston TM, Kallas D, Davies B, Franciosi S, De Souza AM, Laksman ZW, Sanatani S, Krahn AD. Burst exercise testing can unmask arrhythmias in patients with incompletely penetrant catecholaminergic polymorphic ventricular tachycardia. *JACC Clin Electrophysiol*. 2021;7:437–441. https://doi.org/10.1016/j.jacep.2021.02.013

223. Peltenburg PJ, Pultoo SNJ, Tobert KE, Bos JM, Lieve KVV, Tanck M, Clur SB, Blom NA, Ackerman MJ, Wilde AAM, et al. Repeatability of ventricular arrhythmia characteristics on the exercise-stress test in RYR2-mediated catecholaminergic polymorphic ventricular tachycardia. *Europace*. 2023;25:619–626. https://doi.org/10.1093/europace/euac177

224. Stroker E, de Asmundis C, Chierchia GB, Brugada P. Exercise-related Brugada pattern and monomorphic ventricular tachycardia in a patient with Brugada syndrome: interplay between body temperature, haemodynamics and vagal activity. *Eur Heart J*. 2016;37:655. https://doi.org/10.1093/eurheartj/ehv263

225. Lacunza J, San Roman I, Moreno S, Garcia-Molina E, Gimeno J, Valdes M. Heat stroke, an unusual trigger of Brugada electrocardiogram. *Am J Emerg Med*. 2009;27:634.e1–634.e3. https://doi.org/10.1016/j.ajem.2008.09.036

226. Piercy KL, Troiano RP, Ballard RM, Carlson SA, Fulton JE, Galuska DA, George SM, Olson RD. The physical activity guidelines for Americans. *JAMA*. 2018;320:2020–2028. https://doi.org/10.1001/jama.2018.14854

227. Radford NB, DeFina LF, Leonard D, Barlow CE, Willis BL, Gibbons LW, Gilchrist SC, Khera A, Levine BD. Cardiorespiratory fitness, coronary artery calcium, and cardiovascular disease events in a cohort of generally healthy middle-age men: results from the Cooper Center Longitudinal Study. *Circulation*. 2018;137:1888–1895. https://doi.org/10.1161/CIRCULATIONAHA.117.032708

228. Franklin BA, Thompson PD, Al-Zaiti SS, Albert CM, Hivert M-F, Levine BD, Lobelo F, Madan K, Sharrief AZ, Eijsvogels TMH. on behalf of the American Heart Association Physical Activity Committee of the Council on Lifestyle and Cardiometabolic Health; Council on Cardiovascular and Stroke Nursing; Council on Clinical Cardiology; and Stroke Council. Exercise-related acute cardiovascular events and potential deleterious adaptations following long-term exercise training: placing the risks into perspective: an update: a scientific statement from the American Heart Association. *Circulation*. 2020;141:e705–e736. https://doi.org/10.1161/CIR.0000000000000749

229. Harris KM, Creswell LL, Haas TS, Thomas T, Tung M, Isaacson E, Garberich RF, Maron BJ. Death and cardiac arrest in U.S. triathlon participants, 1985 to 2016: a case series. *Ann Intern Med*. 2017;167:529–535. https://doi.org/10.7326/M17-0847

230. Khera A, Budoff MJ, O'Donnell CJ, Ayers CA, Locke J, de Lemos JA, Massaro JM, McClelland RL, Taylor A, Levine BD. Astronaut Cardiovascular Health and Risk Modification (Astro-CHARM) coronary calcium atherosclerotic cardiovascular disease risk calculator. *Circulation*. 2018;138:1819–1827. https://doi.org/10.1161/CIRCULATIONAHA.118.033505

231. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, Himmelfarb CD, Khera A, Lloyd-Jones D, McEvoy JW, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2019;74:e177–e232. https://doi.org/10.1016/j.jacc.2019.03.010

232. SCORE2 Working Group and ESC Cardiovascular Risk Collaboration. SCORE2 risk prediction algorithms: new models to estimate 10-year risk of cardiovascular disease in Europe. *Eur Heart J*. 2021;42:2439–2454. https://doi.org/10.1093/eurheartj/ehab309

233. SCORE2 Working Group and ESC Cardiovascular Risk Collaboration. SCORE2-OP risk prediction algorithms: estimating incident cardiovascular event risk in older persons in four geographical risk regions. *Eur Heart J*. 2021;42:2455–2467. https://doi.org/10.1093/eurheartj/ehab312

234. DeFina LF, Radford NB, Barlow CE, Willis BL, Leonard D, Haskell WL, Farrell SW, Pavlovic A, Abel K, Berry JD, et al. Association of all-cause and cardiovascular mortality with high levels of physical activity and concurrent coronary artery calcification. *JAMA Cardiol*. 2019;4:174–181. https://doi.org/10.1001/jamacardio.2018.4628

235. Aengevaeren VL, Mosterd A, Braber TL, Prakken NHJ, Doevendans PA, Grobbee DE, Thompson PD, Eijsvogels TMH, Velthuis BK. Relationship between lifelong exercise volume and coronary atherosclerosis in athletes. *Circulation*. 2017;136:138–148. https://doi.org/10.1161/CIRCULATIONAHA.117.027834

236. Mohlenkamp S, Lehmann N, Breuckmann F, Brocker-Preuss M, Nassenstein K, Halle M, Budde T, Mann K, Barkhausen J, Heusch G, et al, on behalf of the Marathon Study Investigators and the Heinz Nixdorf Recall Study Investigators. Running: the risk of coronary events: prevalence and prognostic relevance of coronary atherosclerosis in marathon runners. *Eur Heart J*. 2008;29:1903–1910. https://doi.org/10.1093/eurheartj/ehn163

237. Aengevaeren VL, Mosterd A, Bakker EA, Braber TL, Nathoe HM, Sharma S, Thompson PD, Velthuis BK, Eijsvogels TMH. Exercise volume versus intensity and the progression of coronary atherosclerosis in middle-aged and older athletes: findings from the MARC-2 study. *Circulation*. 2023;147:993–1003. https://doi.org/10.1161/CIRCULATIONAHA.122.061173

238. Aengevaeren VL, Mosterd A, Sharma S, Prakken NHJ, Mohlenkamp S, Thompson PD, Velthuis BK, Eijsvogels TMH. Exercise and coronary atherosclerosis: observations, explanations, relevance, and clinical management. *Circulation*. 2020;141:1338–1350. https://doi.org/10.1161/CIRCULATIONAHA.119.044467

239. Gordon JB, Ganz P, Nabel EG, Fish RD, Zebede J, Mudge GH, Alexander RW, Selwyn AP. Atherosclerosis influences the vasomotor response of epicardial coronary arteries to exercise. *J Clin Invest*. 1989;83:1946–1952. https://doi.org/10.1172/JCI114103

240. Deleted in proof.

241. Dibben G, Faulkner J, Oldridge N, Rees K, Thompson DR, Zwisler AD, Taylor RS. Exercise-based cardiac rehabilitation for coronary heart disease. *Cochrane Database Syst Rev*. 2021;2021:CD001800. https://doi.org/10.1002/14651858.CD001800.pub4

242. Zhao XQ, Dong L, Hatsukami T, Phan BA, Chu B, Moore A, Lane T, Neradilek MB, Polissar N, Monick D, et al. MR imaging of carotid plaque composition during lipid-lowering therapy: a prospective assessment of effect and time course. *JACC Cardiovasc Imaging*. 2011;4:977–986. https://doi.org/10.1016/j.jcmg.2011.06.013

243. Noyes AM, Thompson PD. A systematic review of the time course of atherosclerotic plaque regression. *Atherosclerosis*. 2014;234:75–84. https://doi.org/10.1016/j.atherosclerosis.2014.02.007

244. Drca N, Larsson SC, Grannas D, Jensen-Urstad M. Elite female endurance athletes are at increased risk of atrial fibrillation compared to the general population: a matched cohort study. *Br J Sports Med*. 2023;57:1175–1179. https://doi.org/10.1136/bjsports-2022-106035

245. Morseth B, Graff-Iversen S, Jacobsen BK, Jorgensen L, Nyrnes A, Thelle DS, Vestergaard P, Lochen ML. Physical activity, resting heart rate, and atrial fibrillation: the Tromso Study. *Eur Heart J*. 2016;37:2307–2313. https://doi.org/10.1093/eurheartj/ehw059

246. Svedberg N, Sundstrom J, James S, Hallmarker U, Hambraeus K, Andersen K. Long-term incidence of atrial fibrillation and stroke among cross-country skiers. *Circulation*. 2019;140:910–920. https://doi.org/10.1161/circulationaha.118.039461

247. Johansen KR, Ranhoff AH, Sorensen E, Nes BM, Heitmann KA, Apelland T, Bucher Sandbakk S, Wilsgaard T, Lochen ML, Thelle DS, et al. NEXAF Initiative. Risk of atrial fibrillation and stroke among older men exposed to prolonged endurance sport practice: a 10-year follow-up: the Birkebeiner Ageing Study and the Tromso Study. *Open Heart*. 2022;9:e002154. https://doi.org/10.1136/openhrt-2022-002154

248. Edward JA, Cornwell WK 3rd. Impact of exercise on cerebrovascular physiology and risk of stroke. *Stroke*. 2022;53:2404–2410. https://doi.org/10.1161/STROKEAHA.121.037343

249. van de Schoor FR, Aengevaeren VL, Hopman MT, Oxborough DL, George KP, Thompson PD, Eijsvogels TM. Myocardial fibrosis in athletes. *Mayo Clin Proc*. 2016;91:1617–1631. https://doi.org/10.1016/j.mayocp.2016.07.012

250. Thompson PD, Eijsvogels TMH, Kim JH. Can the heart get an overuse sports injury? *NEJM Evid*. 2023;2:EVIDra2200175. https://doi.org/10.1056/EVIDra2200175

251. Lai AF, Braverman AC. Endurance exercise following ascending thoracic aortic aneurysm resection in bicuspid aortic valve aortopathy. *JAMA Cardiol*. 2022;7:772–773. https://doi.org/10.1001/jamacardio.2022.1410

252. Tso J, Hollowed C, Liu C, Alkhoder A, Dommisse M, Gowani Z, Miller A, Nguyen G, Nguyen P, Prabakaran G, et al. Nonsteroidal anti-inflammatory drugs and cardiovascular risk in American football. *Med Sci Sports Exerc*. 2020;52:2522–2528. https://doi.org/10.1249/MSS.0000000000002404

253. Whelton PK, Carey RM, Aronow WS, Casey DE Jr, Collins KJ, Dennison Himmelfarb C, DePalma SM, Gidding S, Jamerson KA, Jones DW, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2018;71:e127–e248. https://doi.org/10.1016/j.jacc.2017.11.006

254. Sharman JE, LaGerche A. Exercise blood pressure: clinical relevance and correct measurement. *J Hum Hypertens*. 2015;29:351–358. https://doi.org/10.1038/jhh.2014.84

255. Link MS. Commotio cordis: ventricular fibrillation triggered by chest impact-induced abnormalities in repolarization. *Circ Arrhythm Electrophysiol*. 2012;5:425–432. https://doi.org/10.1161/CIRCEP.111.962712

256. Maron BJ, Estes NA. 3rd. Commotio cordis. *N Engl J Med*. 2010;362:917–927. https://doi.org/10.1056/NEJMra0910111

257. Lee RN, Sampaio Rodrigues T, Gan JT, Han HC, Mansour R, Sanders P, Farouque O, Lim HS. Commotio cordis in non-sport-related events: a systematic review. *JACC Clin Electrophysiol*. 2023;9:1321–1329. https://doi.org/10.1016/j.jacep.2023.01.010

258. Rosenblatt AG, Link MS. Yes, commotio cordis does occur outside of sports. *JACC Clin Electrophysiol*. 2023;9:1330–1332. https://doi.org/10.1016/j.jacep.2023.03.015

259. Link MS, Maron BJ, Wang PJ, Pandian NG, VanderBrink BA, Estes NA. 3rd. Reduced risk of sudden death from chest wall blows (commotio cordis) with safety baseballs. *Pediatrics*. 2002;109:873–877. https://doi.org/10.1542/peds.109.5.873

260. National Operating Committee on Standards for Athletic Equipment. Standard Test Method and Performance Specification Used in Evaluating the Performance Characteristics of Protectors for Commotio Cordis. *National Operating Committee on Standards for Athletic Equipment*. 2023. https://nocsae.org/standard/standard-test-method-and-performance-specification-used-in-evaluating-the-performance-characteristics-of-chest-protectors-for-commotio-cordis-2

261. Meyering C, Howard T. Hypercoagulability in athletes. *Curr Sports Med Rep*. 2004;3:77–83. https://doi.org/10.1249/00149619-200404000-00005

262. Ortel TL, Neumann I, Ageno W, Beyth R, Clark NP, Cuker A, Hutten BA, Jaff MR, Manja V, Schulman S, et al. American Society of Hematology 2020 guidelines for management of venous thromboembolism: treatment of deep vein thrombosis and pulmonary embolism. *Blood Adv*. 2020;4:4693–4738. https://doi.org/10.1182/bloodadvances.2020001830

263. Nazha B, Pandya B, Spyropoulos AC, Kessler CM. Treatment of venous thromboembolism in elite athletes: a suggested approach to individualized anticoagulation. *Semin Thromb Hemost*. 2018;44:813–822. https://doi.org/10.1055/s-0038-1673690

264. Adami PE, Koutlianos N, Baggish A, Bermon S, Cavarretta E, Deligiannis A, Furlanello F, Kouidi E, Marques-Vidal P, Niebauer J, et al. Cardiovascular effects of doping substances, commonly prescribed medications and ergogenic aids in relation to sports: a position statement of the sport cardiology and exercise nucleus of the European Association of Preventive Cardiology. *Eur J Prev Cardiol*. 2022;29:559–575. https://doi.org/10.1093/eurjpc/zwab198

265. Baggish AL, Weiner RB, Kanayama G, Hudson JI, Lu MT, Hoffmann U, Pope HG Jr. Cardiovascular toxicity of illicit anabolic-androgenic steroid use. *Circulation*. 2017;135:1991–2002. https://doi.org/10.1161/CIRCULATIONAHA.116.026945

266. Martinez KA, Bains S, Neves R, Giudicessi JR, Bos JM, Ackerman MJ. Sudden cardiac arrest occurring in temporal proximity to consumption of energy drinks. *Heart Rhythm*. 2024;21:1083–1088. https://doi.org/10.1016/j.hrthm.2024.02.018

267. Burtscher M, Ponchia A. The risk of cardiovascular events during leisure time activities at altitude. *Prog Cardiovasc Dis*. 2010;52:507–511. https://doi.org/10.1016/j.pcad.2010.02.008

268. Shattock MJ, Tipton MJ. 'Autonomic conflict': a different way to die during cold water immersion? *J Physiol*. 2012;590:3219–3230. https://doi.org/10.1113/jphysiol.2012.229864

269. Kauling RM, Rienks R, Cuypers J, Jorstad HT, Roos-Hesselink JW. SCUBA diving in adult congenital heart disease. *J Cardiovasc Dev Dis*. 2023;10:20. https://doi.org/10.3390/jcdd10010020

270. Tso JV, Powers JM, Kim JH. Cardiovascular considerations for scuba divers. *Heart*. 2022;108:1084–1089. https://doi.org/10.1136/heartjnl-2021-319601

271. Physical activity and exercise during pregnancy and the postpartum period: ACOG committee opinion, number 804. *Obstet Gynecol*. 2020;135:e178–e188. https://doi.org/10.1097/AOG.0000000000003772

272. Bo K, Artal R, Barakat R, Brown W, Davies GA, Dooley M, Evenson KR, Haakstad LA, Henriksson-Larsen K, Kayser B, et al. Exercise and pregnancy in recreational and elite athletes: 2016 evidence summary from the IOC expert group meeting, Lausanne: part 1: exercise in women planning pregnancy and those who are pregnant. *Br J Sports Med*. 2016;50:571–589. https://doi.org/10.1136/bjsports-2016-096218

273. Pivarnik JM, Szymanski LM, Conway MR. The elite athlete and strenuous exercise in pregnancy. *Clin Obstet Gynecol*. 2016;59:613–619. https://doi.org/10.1097/grf.0000000000000222

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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.