Sports Cardiology: Core Curriculum for Providing Cardiovascular Care to Competitive Athletes and Highly Active People.

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THE PRESENT AND FUTURE

STATE-OF-THE-ART REVIEW

Sports Cardiology

Core Curriculum for Providing Cardiovascular Care to Competitive Athletes and Highly Active People

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ABSTRACT

The last few decades have seen substantial growth in the populations of competitive athletes and highly active people (CAHAP). Although vigorous physical exercise is an effective way to reduce the risk of cardiovascular (CV) disease, CAHAP remain susceptible to inherited and acquired CV disease, and may be most at risk for adverse CV outcomes during intense physical activity. Traditionally, multidisciplinary teams comprising athletic trainers, physical therapists, primary care sports medicine physicians, and orthopedic surgeons have provided clinical care for CAHAP. However, there is increasing recognition that a care team including qualified CV specialists optimizes care delivery for CAHAP. In recognition of the increasing demand for CV specialists competent in the care of CAHAP, the American College of Cardiology has recently established a Sports and Exercise Council. An important primary objective of this council is to define the essential skills necessary to practice effective sports cardiology. (J Am Coll Cardiol 2017;70:1902–18)

Competitive athletes and highly active people (CAHAP) are a growing population. Although routine physical exercise is an effective way to reduce the risk of cardiovascular (CV) disease, it does not confer complete immunity (1,2), and actually increases the risk of CV events acutely, even in trained individuals (3). The complex interplay between CV disease and vigorous physical activity remains incompletely understood, but is increasingly relevant in clinical practice. People with occult CV disease are susceptible to sudden cardiac death during exercise. However, sudden death prevention represents only 1 element of caring for CAHAP. Accurate interpretation of diagnostic testing with an emphasis on differentiating pathology from physiological exercise-induced adaptation, efficient and targeted assessment of symptoms, and provision of longitudinal care including the development of exercise

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recommendations following CV disease diagnosis are all critical skills for the effective clinical care of CAHAP.

Multidisciplinary teams comprising athletic trainers, physical therapists, primary care sports medicine physicians, and orthopedic surgeons have traditionally provided clinical care for CAHAP, and this team-based approach will continue to represent the standard of care. However, the optimal CAHAP care team includes a dedicated CV specialist to provide collaboration during pre-participation cardiovascular disease screening (PPCS), evaluation and management of athletes with suspected and confirmed CV disease, and participation in the generation of policies designed to maximize safe sport participation. The practice of sports cardiology is growing at a rapid pace, yet currently, an official competency statement such as those developed by the Core Cardiovascular Training Statement and endorsed by the American Board of Internal Medicine for other CV subspecialties is lacking. This document was written in response to this unfulfilled need. The purpose of this paper is to highlight the basic fund of knowledge and the corollary skills required for the effective practice of sports cardiology. Although the present document was not designed to serve as a formal education curriculum or template for continuing medical education, it is anticipated that it will set the stage for future discussions about how to include sports cardiology into existing CV training requirements and board certification processes. This document identifies 4 fundamental clinical domains of knowledge that are critical to the care of CAHAP (**Central Illustration**). Each domain will be defined, and key skill sets and/or clinical approach algorithms will be presented. Recommendations in this document are based on available evidence, and where evidence is lacking, reflect expert opinion. Recommendations are presented in a *Medical Knowledge* and *Patient Care and Procedural Skills* format to maintain consistency with the American College of Cardiology's (ACC's) recent Lifelong Learning statement (4). It is anticipated that this document will provide a clinical framework for CV practitioners who participate in the care of CAHAP.

EXERCISE-INDUCED CARDIAC REMODELING VERSUS CV PATHOLOGY

Moderate- to high-intensity physical activity, either for recreational exercise or competitive athletics, requires an adaptive and healthy CV system. The CV system adapts to repetitive bouts of exercise through a complex series of structural and functional changes. These adaptations often manifest during clinical evaluation and may be evident during physical examination and interpretation of diagnostic testing. Accurate interpretation of clinical data derived during the assessment of CAHAP requires an understanding both of basic exercise physiology and the numerous adaptive CV changes that are common in this patient population. Essential knowledge base and skill sets for the sports cardiologist pertaining to exercise physiology and exercise-induced cardiac remodeling (EICR) are shown in **Table 1**.

FUNDAMENTAL CLINICAL EXERCISE PHYSIOLOGY. All forms of vigorous physical activity and competitive athletics involve some combination of static and dynamic exercise physiology (5). The terms *static* and *dynamic* refer to patterns of skeletal muscle activity and their consequent changes in CV structure and function. Static activity is characterized by short and forceful skeletal muscle contractions, which can be quantified as an estimated percentage of maximal voluntary contraction for involved muscle groups. During bouts of relatively pure static/strength activities, such as power weight lifting and track and field throwing events, intense skeletal muscle activation coupled with stimulation of mechanical and metabolic afferent signals lead to acute increases in systemic vascular resistance and arterial blood pressure. The primary role of the CV system during acute bouts of static activity is to *maintain* cardiac output in the face of increased left ventricular (LV) afterload. This is accomplished by increasing myocardial contractility, which facilitates preservation of end-diastolic and -systolic LV volume. In contrast, dynamic/endurance activities are characterized by repetitive, often rhythmic contraction and relaxation of large skeletal muscle groups, which require increases in oxidative metabolism. The intensity of dynamic activity can thus be quantified by measurement of oxygen uptake ($\dot{V}O_2$). The primary CV response to dynamic activity is to *increase* cardiac output to ensure adequate delivery of metabolic substrate to active muscle beds. The magnitude of cardiac output augmentation is tightly regulated by energetic signals in skeletal muscle, is proportional to the intensity of the dynamic activity, and is accomplished by increases in heart rate, augmentation of stroke volume, and reductions in systemic vascular resistance. Diseases of the CV system that impair the ability to maintain cardiac output in the face of increased LV afterload and/or to increase cardiac

ABBREVIATIONS AND ACRONYMS

ACC = American College of Cardiology

AHA = American Heart Association

ASCVD = atherosclerotic cardiovascular disease

CAHAP = competitive athletes and highly active people

CV = cardiovascular

ECG = 12-lead electrocardiography

EICR = exercise-induced cardiac remodeling

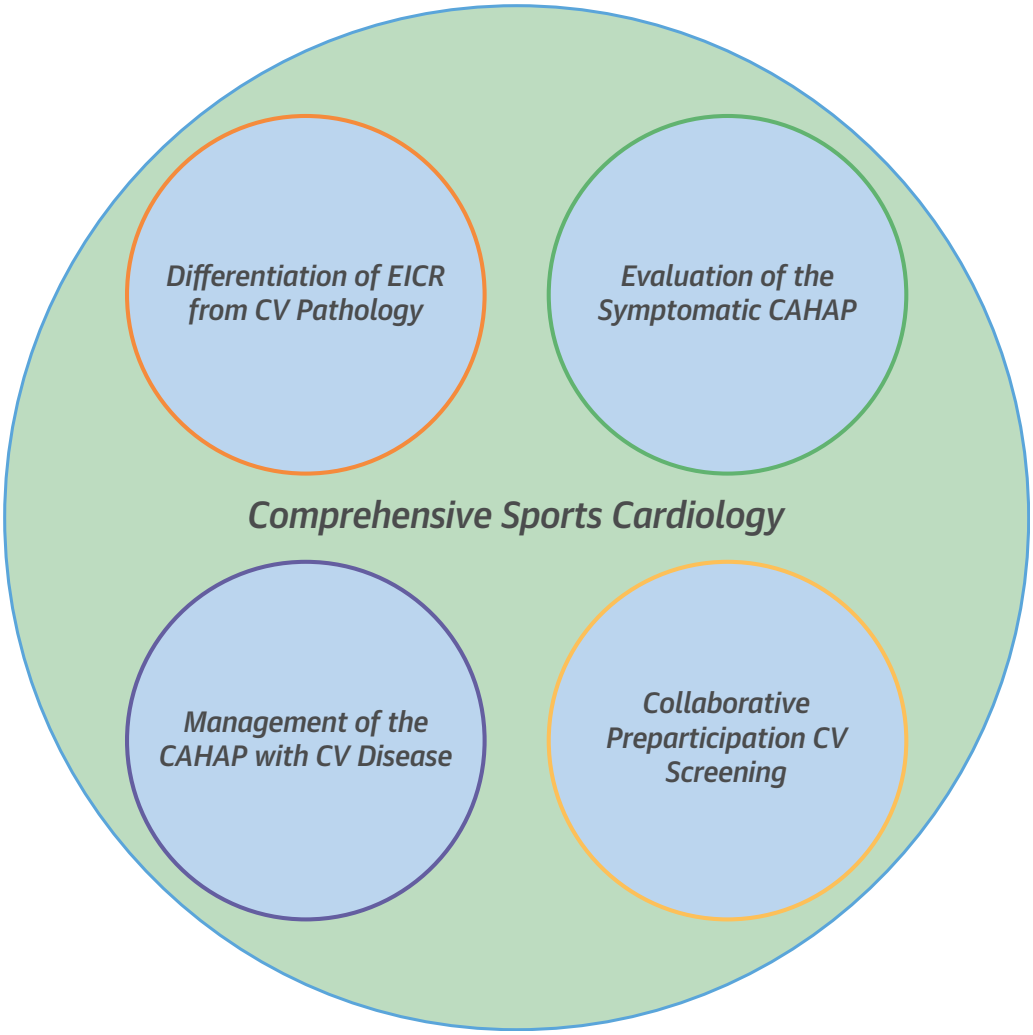
ICD = implantable cardiac defibrillator

LV = left ventricular

PPCS = pre-participation cardiovascular disease screening

RV = right ventricular

CENTRAL ILLUSTRATION **Overview of Fundamental Core Competencies in Sports Cardiology**



Baggish, A.L. et al. J Am Coll Cardiol. 2017;70(15):1902–18.

The 4 principal competency domains underlying the growing and rapidly evolving specialty of sports cardiology. Each domain requires integration of the fundamental principles of general and subspecialty clinical cardiovascular medicine with a knowledge base unique to the population of competitive athletes and highly active people (CAHAP). CV = cardiovascular; EICR = exercise-induced cardiac remodeling.

output in response to dynamic physiology often manifest as symptoms including chest pain, inappropriate exertional dyspnea, and subjective exercise intolerance.

EXERCISE-INDUCED CARDIAC REMODELING. Repetitive participation in vigorous physical exercise stimulates adaptive changes in CV structure and function. This process, referred to as EICR, varies considerably across athletic populations and individual athletes based on a number of factors, including but not limited to ethnicity, sex, genetics (underlying genome), and epigenetic factors including sporting discipline and duration of exercise exposure. In a landmark report, athletes participating in sports with predominantly dynamic physiology (swimmers and runners) were found to have larger LV chamber diameters than athletes practicing wrestling, a sport with a predominantly static physiology (6). Although the notion that EICR varies as a function of sport physiology has been challenged (7), this concept has been verified by numerous investigators (8–10). Recent reports extend the scope of sport-specific EICR beyond LV structure to include LV diastolic function (11), right ventricular

(RV) morphology (12), and atrial structure (13,14), which respond to the principal stresses of static and dynamic exercise in variable ways.

Dichotomization of sporting disciplines into either static or dynamic activity is, however, overly simplistic. Contemporary classifications of exercise physiology acknowledge that most sporting disciplines involve some element of both static and dynamic stress (5), and that corollary EICR should reflect the relative contributions of each cardinal form of hemodynamic stress (**Figure 1A**). For example, endurance sports all require the ability to markedly augment cardiac output (dynamic stress) but involve variable amounts of concomitant static stress. A recent study evaluating rowers and runners documented increases in LV volume in both groups of athletes, but thickening of LV walls only among rowers due to the pulsatile pressure challenge inherent in this sport (15). In sum, the biventricular and biatrial volume challenge inherent in dynamic physiology sports leads to chamber dilation, whereas the pressure challenge inherent in static sports leads to LV wall thickening with minimal effects on the other 3 cardiac chambers. Cardiac morphology among CAHAP may be also driven by comorbid factors, with recent studies of American style football lineman demonstrating evidence of hypertensive LV hypertrophy (16) and subclinical systolic dysfunction (17).

TABLE 1 Exercise Physiology and Exercise-Induced Cardiovascular Remodeling: Essential Skills for the Sports Cardiologist

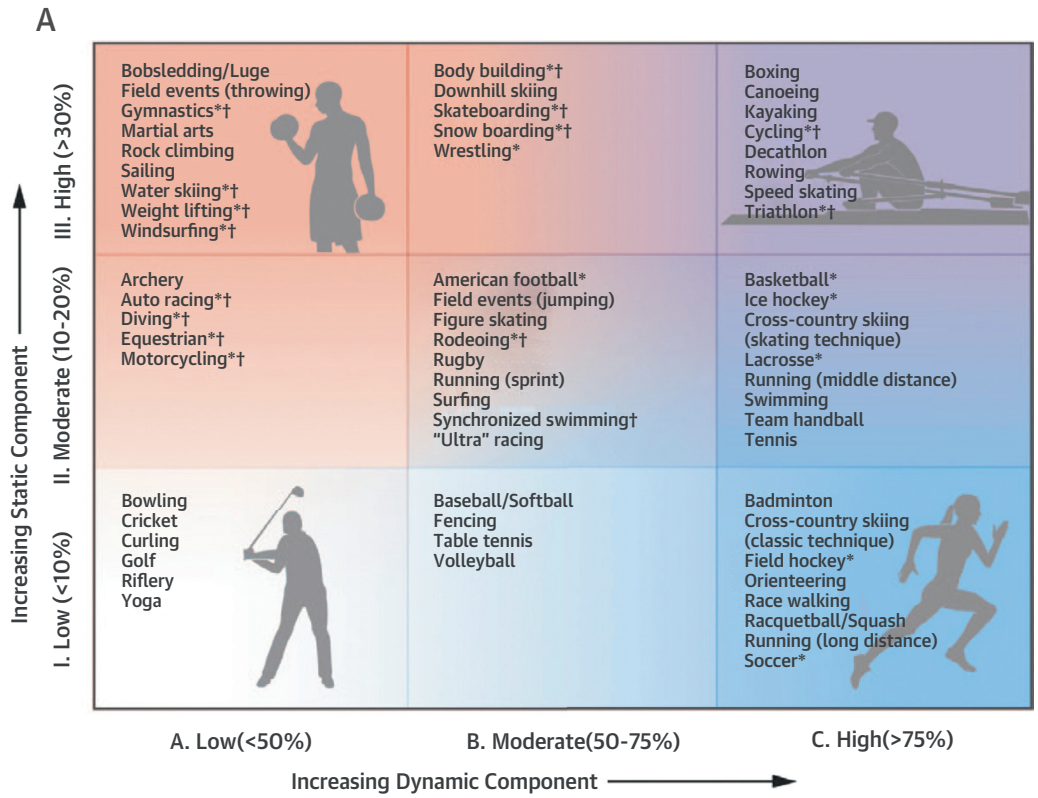
Medical Knowledge
Know the fundamental hemodynamic characteristics of static exercise.
Know the fundamental hemodynamic characteristics of dynamic exercise.
Know the current ACC/AHA classification of sports.
Know the basic structural and functional patterns of cardiovascular adaptation that accompany different forms of exercise.
Know factors that may contribute to variability of EICR.
Know the rationale for the use of athlete-specific ECG interpretation criteria.
Know benign/adaptive ECG patterns that are considered "common and training-related."
Know ECG patterns that are not related to exercise training and are potentially reflective of true underlying disease.
Know definitions of the key geometric left ventricular hypertrophy variants encountered during noninvasive imaging of CAHAP.
Know which geometric left ventricular hypertrophy variants typically associate with which specific forms of exercise.
Know the expected response of the right ventricle, right atrium, and left atrium in response to activities with dynamic physiology (endurance sports).
Know that resting biventricular systolic function, as assessed by ejection fraction, may be at or slightly below the lower limits of normal under resting conditions among CAHAP with dilated ventricles.
Know that resting diastolic function should be normal to supranormal, as assessed echocardiographically using complementary indexes (i.e., transmitral Doppler, tissue Doppler imaging, and so on) among endurance-trained CAHAP.
Know the differential diagnosis for left ventricular chamber dilation among CAHAP.
Know the differential diagnosis for right ventricular chamber dilation among CAHAP.
Know the differential diagnosis for left ventricular wall thickening among CAHAP.
Know the rationale for prescribed detraining including relevant areas of physiological uncertainty in clinical practice.
Patient Care and Procedural Skills
Skill to comprehensively evaluate physical activity and exercise patterns, including type, duration, and intensity of training and competition, to determine expected patterns of underlying EICR.
Skill to apply contemporary ECG interpretation criteria for CAHAP in varied clinical settings ranging from traditional clinic encounters to "in the field" pre-participation screening.
Skill to interpret transthoracic echocardiographic data, in partnership with cardiovascular imaging experts, among CAHAP with emphasis on recognition of imaging data consistent with adaptive exercise-induced remodeling.
Skill to prescribe physiological deconditioning in selected cases where multimodality diagnostic assessment fails to resolve "gray-zone" findings among CAHAP.

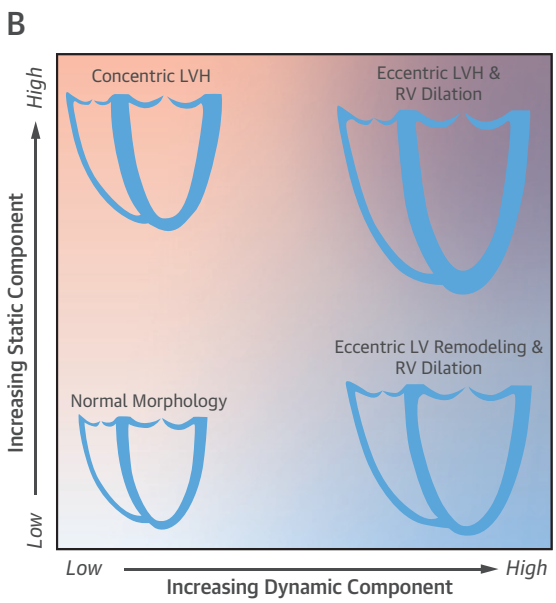
ACC = American College of Cardiology; AHA = American Heart Association; CAHAP = competitive athletes and highly active people; ECG = 12-lead electrocardiography; EICR = exercise-induced cardiac remodeling.

DIAGNOSTIC FINDINGS IN ATHLETES: 12-LEAD ELECTROCARDIOGRAPHY. CV adaptations to exercise commonly manifest on the 12-lead electrocardiography (ECG) and may overlap with patterns reflecting underlying CV disease. Criteria for ECG interpretation in athletes were first proposed in an expert consensus statement by the European Society of Cardiology (ESC) in 2005 (18), and have undergone numerous revisions (19–21). These revisions have reduced but not eliminated the ECG's propensity to generate false positive findings (22–26). Contemporary athlete ECG criteria divide findings into benign, adaptive patterns, including sinus bradycardia, early repolarization, prominent QRS voltage, and incomplete right bundle branch, and patterns that carry significant associations with CV pathology (27). To date, no ECG interpretation criteria have been correlated with definitive clinical outcomes, and large clinical trials, appropriately powered for relevant outcomes, are lacking.

DIAGNOSTIC FINDINGS IN ATHLETES: NONINVASIVE IMAGING. EICR commonly results in thickening of the LV walls and dilation of the LV chamber. LV hypertrophy, defined by wall thicknesses in the range of 11 to 13 mm, is common among CAHAP (28,29). Athletes with LV hypertrophy demonstrate either eccentric or concentric LV geometry. Eccentric LV hypertrophy, a balanced increase in wall thickness and chamber size, occurs most commonly among CAHAP who engage in sports with a combination of high dynamic and high static physiology. Physiological dilation of the RV (30) and both atrial chambers (13,14,31) often accompanies eccentric LV remodeling. In contrast, mild concentric hypertrophy, increased wall thickness with normal or even slightly reduced chamber size (8,9), may develop among CAHAP who participate in high static/low-to-moderate dynamic physiology sports, particularly when coupled with resting hypertension (16). EICR is most commonly associated with preserved LV systolic function as assessed by ejection fraction (32), and newer

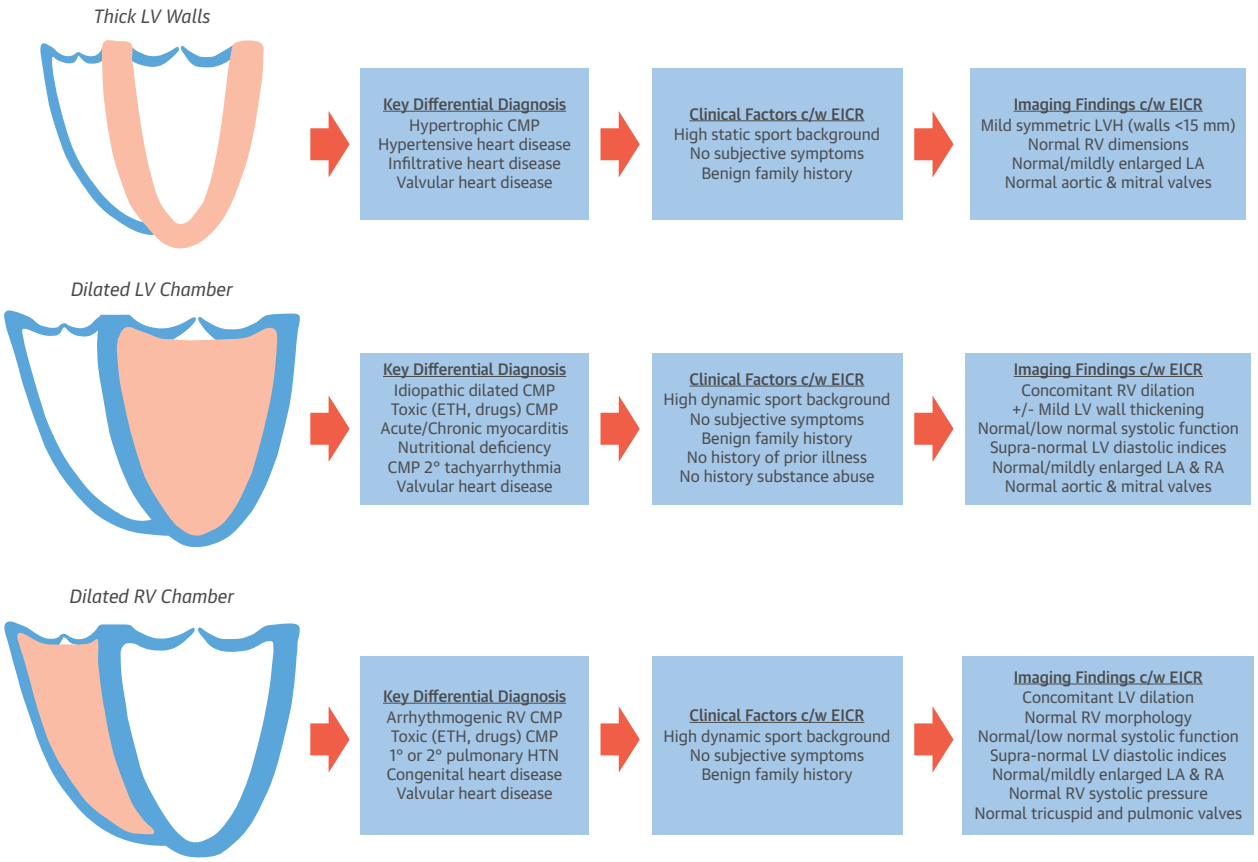
FIGURE 1 The Interplay Between Exercise Physiology and EICR





(A) Physiological classification of common sporting disciplines representing relative contributions of static and dynamic physiology (reprinted with permission from Levine et al. [5]), and **(B)** the anticipated structural cardiac adaptations that develop as a function of these underlying physiological stressors. *Danger of bodily collision. †Increased risk if syncope occurs. EICR = exercise-induced cardiac remodeling; LV = left ventricular; LVH = left ventricular hypertrophy; RV = right ventricular.

FIGURE 2 **Approach to "Gray Zone" Cardiac Finding Among CAHAP**



Common cardiac findings encountered during noninvasive imaging of competitive athletes and highly active people (CAHAP) include thickened LV walls, dilated LV chambers, and dilated RV chambers. The determination of whether these findings represent benign EICR or myocardial pathology unrelated to exercise requires consideration of a focused differential diagnosis coupled with an understanding of expected sport-specific patterns of physiological cardiac remodeling and integration of key metrics derived from clinical assessment and noninvasive imaging. CMP = cardiomyopathy; c/w = consistent with; ETH = ethanol; HTN = hypertension; LA = left atrium; RA = right atrium; other abbreviations as in **Figure 1**.

complementary indexes of systolic function (33). However, healthy athletes with physiological LV dilation may demonstrate resting LV ejection fractions at or slightly below the lower limits of normal (34), as it is stroke volume, not ejection fraction, that is regulated by resting metabolic demands. LV diastolic function in CAHAP should be normal or enhanced, and any evidence of diastolic dysfunction should raise a high index of suspicion for pathology (35).

Quantitative overlap between physiological hypertrophy and dilation with mild forms of heart muscle disease has traditionally been referred to as the "gray zone" (36). First described as an LV phenomenon, this concept now extends to the RV, as both enlarge in response to endurance-based exercise training (37). Accurate diagnostic assessment of athletes with gray zone cardiac findings has important clinical implications and represents 1 of the most fundamental sports cardiology skills. This process requires the clinician to define an expected adaptive remodeling pattern after consideration of sport discipline (**Figure 1B**), and to consider key modifying factors including sex, ethnicity, and duration/intensity of exercise exposure.

EICR leads to 3 distinct gray zone findings, each with a specific set of differential diagnoses (**Figure 2**). EICR-induced LV and RV dilation may yield chamber dimensions that approximate those found in overt forms of disease, and thus, chamber dimension size cut points are of limited value. Physiological LV and RV dilation are typically accompanied by concomitant enlargement of the other ventricle, bi-atrial dilation, and normal to low-normal resting systolic function that augments during exercise. Isolated LV or RV

TABLE 2 Clinical Assessment of Symptomatic CAHAP: Essential Skills for the Sports Cardiologist

Medical Knowledge
Know the differential diagnosis, including cardiovascular and noncardiovascular causes, of chest pain in CAHAP.
Know common atypical presentations of exertional ischemic chest pain associated with atherosclerotic coronary artery disease among CAHAP.
Know common atypical presentations of exertional ischemic chest pain associated with congenital coronary anomalies among CAHAP.
Know how to optimize conventional approaches to exercise testing by avoiding test termination at "diagnostic thresholds" and the inclusion of noninvasive imaging to maximize diagnostic utility among CAHAP.
Know how and when to further customize exercise testing for CAHAP including the use of individualized exercise protocols that permit manipulation of variables, such as exercise intensity, duration, modality, and climate.
Know how and when to extend conventional exercise testing approaches to include adjunct testing such as metabolic gas exchange, pulse oximetry, direct measurement of cardiac output, and invasive hemodynamic measurements.
Know common causes of collapse, including syncopal and nonsyncopal etiologies, during exercise.
Know common presentations of nonsyncope causes of collapse during exercise.
Know how to ascertain a comprehensive medical history of exercise-associated syncope with emphasis on identifying features of both neurally mediated and alternative "high-risk" causes of syncope.
Know the physiology responsible for post-exertional neurally mediated syncope.
Know common causes of palpitations among CAHAP.
Know clinical features suggestive of benign versus higher-risk causes of palpitations among CAHAP.
Know the cardiovascular and noncardiovascular causes of impaired exercise capacity or decrements in exercise performance among CAHAP.
Patient Care and Procedural Skills
Skill to perform a comprehensive personal and family medical history, including characterization of exercise and physical activity habits (training, competition, and deconditioning) and the use to performance-enhancing agents during the evaluation of symptomatic CAHAP.
Skill to determine the appropriate evaluation of CAHAP presenting with chest pain during or unrelated to exercise.
Skill to oversee conventional assessments of exercise testing including the use of continuous ECG and standard forms of noninvasive imaging.
Skill to develop and implement customized approaches to exercise testing with an emphasis on reproducing the physiological conditions required to reproduce presenting symptoms among individual CAHAP.
Skill to determine the appropriate evaluation of CAHAP presenting with collapse during or unrelated to exercise to include selection of diagnostic imaging and ambulatory rhythm monitoring.
Skill to determine the appropriate evaluation of CAHAP presenting with palpitations during or unrelated to exercise to include selection of diagnostic imaging and ambulatory rhythm monitoring.
Skill to engage a collaborative and often multidisciplinary approach during the assessment of the CAHAP with impaired exercise capacity or decrements in exercise performance.

Abbreviations as in Table 1.

dilation with any structural or functional abnormalities should be considered pathology until otherwise proven. LV wall thickening among athletes is typically of mild to moderate magnitude, rarely exceeding 13 to 15 mm; values in excess of 15 mm necessitate an individualized, often multimodality evaluation for explanatory cardiomyopathy.

Prescribed exercise detraining coupled with serial noninvasive imaging has been proposed as a diagnostic option among CAHAP with gray zone hypertrophy and an inconclusive initial assessment (38). The use of prescribed detraining is based on the notion that EICR will regress following stimulus removal, whereas hypertrophy and/or dilation attributable to disease will not. Although "physiological myocardial atrophy" occurs during bed rest at a rate of ~1%/week (39), this principle has not been rigorously tested in CAHAP. These limited data suggest that EICR does appear to be a dynamic phenotype characterized by substantial reversibility (40–42), but the time course and magnitude of this process remain uncertain. Furthermore, there are currently no data defining the effect of exercise abstinence among patients with established cardiomyopathic conditions. Until such critical areas of uncertainty are resolved, prescribed detraining should be used cautiously and with an understanding and acknowledgment of its inherent limitations.

EVALUATION OF SYMPTOMATIC CAHAP

Sports cardiologists must be capable of evaluating CAHAP that present with symptoms suggestive of underlying CV disease. This process requires integration of basic principles of general cardiology and exercise physiology, with a comprehensive understanding of issues unique to this population including atypical presentations of disease. The following sections address common chief complaints among CAHAP and the essential knowledge base and skill sets required for comprehensive assessment (Table 2).

CHEST PAIN. Chest pain, both at rest and during exercise, is a common reason for CAHAP to seek medical attention. Noncardiac causes for chest pain, including gastrointestinal, pulmonary, and musculoskeletal processes, are common across CAHAP of all ages and account for the vast majority of diagnoses (43). Among young CAHAP (age <35 years), underlying CV disease accounts for the vast minority (~6%) of chest pain, but when present, is often attributable to potentially life-threatening diseases including hypertrophic cardiomyopathy, anomalous coronary arteries, and congenital valvular disease. CAHAP with congenital coronary anomalies often present with atypical patterns, including exertional breathlessness in the absence of chest pain, a complaint that is often misdiagnosed as drug-refractory "exertional asthma," or sporadic chest discomfort that is intermittently present at only very high exercise intensity. In contrast, atherosclerotic cardiovascular disease (ASCVD) predominates as a cardiac cause for exertional chest pain in CAHAP >35 years of age. CAHAP with ASCVD may present with typical angina, but often only experience a variant knows as "warm-up angina": chest discomfort that occurs during the first few

minutes of exercise and then abates with continued effort (44). This presentation is common among older athletes, a population in which no level of fitness is completely protective against ASCVD, and should raise a high index of suspicion for disease. In CAHAP of all ages, symptoms accompanying exertional chest discomfort, including palpitations, near-syncope or syncope, or decline in exercise performance should prompt a high index of suspicion for CV disease.

Evaluation of chest discomfort in CAHAP begins with a detailed patient history including ascertainment of familial disease patterns (genetic cardiomyopathies and premature ASCVD) and the use of illicit drugs or performance-enhancing agents. Additional diagnostic steps include resting ECG, measurement of a lipid profile, and comprehensive exercise testing according to contemporary guidelines (45). Exercise testing in CAHAP should not be terminated at a predefined target heart rate but rather continued to maximal volitional exercise capacity. This approach permits characterization of the CV response to exercise across the entire intensity spectrum, thereby maximizing the likelihood of symptom reproduction, which is necessary to exclude or confirm underlying pathology. Conventional exercise protocols may not be sufficient to reproduce chest discomfort in CAHAP, and customized testing designed to simulate conditions under which symptoms develop is often required. Test customization should consider manipulation of variables including exercise intensity, duration, modality, and climate. Cycle and rowing ergometers are often required to optimally simulate training and competitive environments, and standard treadmills present in most hospitals and practices may facilitate adequate sprint intervals in elite athletes. Thus, comprehensive exercise assessment in CAHAP may be best accomplished in referral centers that house advanced exercise testing laboratory with staff trained in the art of individualizing exercise protocols. The inclusion of adjunct testing, including metabolic gas exchange, pulse oximetry, cardiac imaging, measurement of cardiac output, and assessment of invasive hemodynamic parameters, may improve the diagnostic yield of exercise testing and should be considered on a case-by-case basis.

SYNCOPE. Collapse during exercise due to alteration or complete loss of consciousness is a medical emergency that requires immediate assessment (46). Causes of collapse include cardiac and neurally mediated syncope, heat stroke, hyponatremia, "exercise associated collapse," and seizure. Victims of heat stroke, hyponatremia, and exercise-associated collapse typically retain some level of consciousness throughout their event. In contrast, athletes with cardiac syncope, neurally mediated syncope, or seizure typically experience a period of complete unconsciousness. Patients with cerebral hypoxia due to cardiac arrest often have myoclonic jerking that can be confused with seizure activity and frequently experience incontinence. Seizures of neurological origin may occur at times other than exercise and convulsions often precede loss of consciousness.

Syncope is defined as a transient loss of consciousness and postural tone resulting from global cerebral hypoperfusion with spontaneous and complete recovery and no neurological sequelae (47). Syncope in young CAHAP is most commonly neurally mediated, but may be caused by alternative, potentially life threatening conditions including transient tachyarrhythmias, bradyarrhythmias, and LV outflow obstruction (48). Neurally mediated syncope is common in the immediate post-exercise period and during times unrelated to exercise, but is extremely rare during exertion. Neurally mediated syncope is typically preceded by premonitory symptoms including a generalized feeling of warmth, flushing, and lightheadedness culminating in a loss of consciousness. Affected CAHAP often lower themselves to the ground or fall slowly due to the relatively gradual onset of symptoms rendering bodily injury uncommon. The ensuing period of unconsciousness is typically brief with minimal residual confusion in the absence of concomitant hypoglycemia, which may affect CAHAP with insulin-dependent diabetes, but is often followed by persistent vagal symptoms including flushing, warmth, fatigue, and nausea. In contrast, syncope caused by an acute arrhythmia is typically characterized by an abrupt loss of consciousness followed by dramatic collapse and injury (34).

The diagnostic evaluation of syncope begins with careful acquisition of medical history and when available, review of video capture of the event, with an emphasis on defining the circumstances and subjective recall of the event. In situations where the history is nondiagnostic, ECG and noninvasive cardiac imaging are useful adjuncts that should be used to exclude underlying electrical, valvular, or myocardial disease. Exercise testing designed to trigger culprit arrhythmias and provocative testing to bring out the ECG features of the long QT syndrome (LQTS) may be warranted in selected individuals. Prolonged ambulatory monitoring, including the use of implantable loop recorders, should be considered for those with suspected arrhythmic syncope and an initial nondiagnostic evaluation. It is imperative to capture a recording of cardiac rhythm during a syncopal event before excluding an arrhythmic cause of syncope. Because relative orthostatic intolerance is

characteristic of CAHAP and is a direct consequence of the cardiovascular adaptation (49,50), tilt-table testing should not be used to establish a diagnosis of neurally mediated syncope in this population as a positive test is nonspecific.

PALPITATIONS. Palpitations, defined as the sensation of the heart beating too strong, too fast, or irregularly, are a symptom, not a diagnosis. During times of excitement, fear, and psychological stress, palpitations typically reflect sinus tachycardia and are common among CAHAP due to their heightened sense of body awareness. In contrast, palpitations that occur in the absence of such triggers or during exercise are more likely to be caused by arrhythmias. Palpitations characterized by sudden heart rate acceleration or deceleration in the absence of an explanatory exercise pattern should raise the index of suspicion of pathological arrhythmia. CAHAP presenting with palpitations require evaluation to exclude possible life-threatening arrhythmias. Medical history is often unrevealing, but should be geared to differentiating exertional palpitations from those unrelated to exercise and clarification of potentially explanatory factors, including stimulant use, suboptimal sleep hygiene, and psychosocial stress. A resting ECG should be performed to evaluate for preexcitation, to check for disorders of repolarization including LQTS and Brugada syndrome, and to screen for underlying structural heart disease. CAHAP that present with palpitations during exercise should undergo exercise testing with adherence to the principles delineated in the previous text, and cardiac imaging. When these initial diagnostic steps fail to reproduce palpitations, ambulatory rhythm monitoring should be employed with a goal of rhythm capture during symptoms. There are numerous devices designed to facilitate ambulatory rhythm detection, including conventional 24-h Holter monitors, wearable adhesive rhythm monitors, patient triggered devices designed to be used with smart phone ECG, longer-term external loop monitors, and long-term implantable monitors. Device selection must be made on an individualized basis and may involve the use of several devices in a sequential fashion until a diagnosis is secured (38). The evaluation of CAHAP with palpitations often reveals benign but explanatory rhythm disturbances including premature atrial and ventricular beats, which are best addressed by counseling and reassurance and infrequently, pharmacological suppression.

IMPAIRED EXERCISE CAPACITY. CAHAP with exercise intolerance present with subjective complaints, often comparing themselves to more capable peers or with objective evidence of declining capacity based on competition results or data derived from training sessions. Exercise intolerance may occur in isolation or in conjunction with other symptoms including dyspnea, chest pain, dizziness, palpitations, and generalized fatigue. Comprehensive evaluation of CAHAP with exercise intolerance requires a collaborative approach that includes athletic trainers, coaches, internists, primary care sports medicine providers, and clinicians representing other medical subspecialties. The evaluation begins with a careful medical history focused on exercise training routine, potential changes in lifestyle unrelated to exercise, prior performance records, and data derived from wearable physiology and fitness monitors. Physical examination should assess for CV disease but also signs of alternative pathology including overtraining, autoimmune diseases, iron deficiency, viral illnesses, asthma, laryngeal dysfunction, and endocrine disorders. Subsequent diagnostic testing should aim to exclude CV conditions, including chronotropic incompetence, reduced LV function, coronary artery disease, atrial fibrillation, aortic stenosis, and HCM, followed by a broader assessment aimed at other organ systems, systemic diseases, and psychiatric pathology including depression and performance anxiety. Objective determination of exercise capacity, most accurately obtained by measurement of peak oxygen consumption coupled to some meaningful metric of external work (i.e., treadmill speed/incline, cycle ergometer power, and so on), should be routine among athletes presenting with exercise intolerance. These metrics can be compared to population-based or athlete-specific normative data and used to assess both disease trajectory and response to therapeutic intervention. Among athletes older than age 35 years, normal aging is a consideration during differential diagnosis. However, it must be emphasized that aging does not cause sudden declines in exercise capacity and should be considered a diagnosis of exclusion.

MANAGEMENT OF CAHAP WITH ESTABLISHED CV DIAGNOSES

Longitudinal care of CAHAP with established CV disease is among the most important responsibilities of the sports cardiologist. In all cases, health and safety should be prioritized over athletic goals, but in most cases, simultaneous attention to both outcomes facilitates optimal care. Essential knowledge base and skill sets pertaining to the clinical management of CAHAP with established CV disease are shown in **Table 3**.

COMPETITIVE SPORTS ELIGIBILITY CRITERIA. Comprehensive recommendations for sport participation

eligibility for competitive athletes with established CV disease have been published (51). U.S.-based recommendations, first proposed by the 36th Bethesda Conference in 1985, have been updated approximately every decade. These recommendations were designed specifically for use among competitive athletes, a group defined by their participation in organized team or individual sports that require regular competition, place a high premium on excellence and achievement, and require some form of systematic and usually intense training. This invaluable resource represents the starting point for all considerations about sport participation following CV disease diagnosis. The most recent update, cosponsored by the American Heart Association (AHA) and ACC and published in 2015 (52), uses the standard ACC recommendation classification system and includes a large number of Class IIa and IIb recommendations with wording such as "may be reasonable" or "may be considered," providing sports cardiologists and their patients an opportunity to engage in shared decision making in situations with uncertain risk and limited outcomes data. This approach represents an important departure from prior versions of this document, which gave a dichotomous yes/no recommendation for sports eligibility. The following considerations have been developed to complement the eligibility recommendations by providing a disease management framework that extends beyond the issue of competition eligibility.

DISEASES OF THE HEART MUSCLE. Hypertrophic cardiomyopathy, the most common form of inherited cardiomyopathy, was among the first genetic heart diseases linked with sudden death during exercise (53). Diagnostic criteria and clinical management strategies for CAHAP with cardiomyopathy, including medication use, indications for implantable defibrillator placement, and collaboration with a cardiomyopathy specialist, should not differ substantially from those designed and routinely applied to more sedentary members of the general population (54).

CAHAP with cardiomyopathy may be at increased risk of malignant tachyarrhythmias and progression of underlying disease. Recent data suggest that routine vigorous exercise may increase the risks of congestive heart failure and future arrhythmias among CAHAP with arrhythmogenic RV cardiomyopathy, a form of cardiomyopathy that can present across the age spectrum (55). Recommendation statements thus restrict vigorous exercise among those with diagnosed or even probable/possible arrhythmogenic RV dysplasia. For CAHAP with other cardiomyopathies, the absolute risk of arrhythmia and disease progression remains poorly understood and difficult to quantify, and consensus recommendations regarding

TABLE 3 Clinical Management of CAHAP With Established Cardiovascular Disease: Essential Skills for the Sports Cardiologist

Medical Knowledge
Know the rationale and suggested use of the AHA/ACC Eligibility and Disqualification Recommendations for Competitive Athletes.
Know and understand an approach for determining eligibility for CAHAP with cardiovascular diagnoses for which Class IIa and IIb eligibility recommendations have recently been proposed.
Know current physical activity guidelines proposed for use in the general population with an emphasis differentiating the role of exercise for health promotion versus athletic performance.
Know common genetic and acquired forms of heart muscle disease that are associated with a risk of adverse events during exercise.
Know contemporary guidelines for diagnosis, risk stratification, and management of genetic and acquired forms of heart muscle disease.
Know common genetic and acquired forms of electrical heart disease that are associated with risk of adverse events during exercise.
Know contemporary guidelines for diagnosis, risk stratification, and management of genetic and acquired forms of electrical heart disease.
Know current indications for ICD implantation in the primary and secondary prevention settings.
Know emerging data surrounding the safety of competitive sports eligibility among CAHAP with conditions including but not limited to implantable cardiovascular devices and long QT syndrome among CAHAP.
Know epidemiological data establishing atherosclerotic coronary artery disease as a common cause of exertional symptoms and cardiac arrest among CAHAP older than age 35 years.
Know the pros and cons of medical therapy versus revascularization among CAHAP with atherosclerotic coronary artery disease.
Know the effect on exercise physiology and exercise safety of commonly prescribed medications for the management of atherosclerotic coronary artery disease.
Know the importance of hypertension as a causal factor in the development of cardiovascular disease among CAHAP.
Know the impact of antihypertensive agents on exercise physiology.
Know the association between atrial arrhythmias and long-term exposure to endurance sporting activity.
Know how and when to opt for a rhythm versus a rate control strategy, including the use of pharmacological and catheter-based sinus maintenance options, among CAHAP with atrial arrhythmias.
Know the risk-benefit profiles of various anticoagulation strategies (including the uses of aspirin, warfarin, novel oral anticoagulants, and dual antiplatelet therapy) among CAHAP.
Patient Care and Procedural Skills
Skill to apply recommendations provided by the AHA/ACC Eligibility and Disqualification Recommendations for Competitive Athletes following diagnosis of definitive or suspected cardiovascular disease.
Skill to implement shared decision-making strategies for eligibility decisions when risk of participation is either uncertain or significantly modifiable by medical, catheter-based, or surgical therapy.
Skill to differentiate benign adaptive EICR from various forms of heart muscle disease that are associated with risk of adverse events during exercise.
Skill to prescribe exercise designed to optimize long-term health among CAHAP disqualified from competitive sports due to underlying structural or electrical heart disease.
Skills to assist with strategies to optimize safety of sport participation among CAHAP with cardiovascular conditions, including but not limited to implantable cardiac devices and long QT syndrome, who elect to participate in competitive and recreational sports.
Skills to direct clinical management of CAHAP with atherosclerotic coronary artery disease, including recommendations regarding selection and duration of medications, decision making regarding revascularization, and application of cardiac rehabilitation following acute coronary syndromes.
Skills to diagnose hypertension at rest and during exercise.
Skills to implement lifestyle modifications and medical therapy for CAHAP with hypertension that have minimal effects on exercise capacity and sport safety.
Skills to diagnose and manage, in partnership with an electrophysiologist, paroxysmal atrial arrhythmias among CAHAP. This will include determination of rhythm versus rate control strategies, selection and sequencing of medical and catheter based techniques, and guideline-dictated use of anticoagulation with consideration of its effect on sport and exercise safety.

ICD = implantable cardiac defibrillator; other abbreviations as in Table 1.

sport participation are thus conservative. However, relegation of CAHAP with cardiomyopathy to a sedentary lifestyle may have significant adverse implications, including weight gain and reduced emotional well-being (56). Sports cardiologists therefore play a critical role in the management of CAHAP with cardiomyopathy by working to define and continually refine a treatment strategy, including exercise recommendations that minimize risk while capitalizing on the health benefits of routine low- to moderate-intensity exercise (57).

Myocarditis, an acquired inflammatory heart muscle disease, is associated with increased risk of arrhythmia during sport participation, and limited animal data suggest that exercise during the acute phases of myocarditis may exacerbate cardiac dysfunction (58). Consequently, all athletic participation should be restricted in the setting of suspected or confirmed myocarditis. In most cases, myocarditis is a transient and self-limited condition, and full resumption of exercise, following documentation of disease resolution with some combination of biochemical testing, exercise testing, noninvasive imaging, and ambulatory rhythm monitoring, is usually appropriate. The time course for resolution of myocarditis and the prognostic implications of residual myocardial scarring remain incompletely characterized. The majority of CAHAP will demonstrate complete resolution by 3 to 6 months, but prolonged or recurrent symptoms and laboratory/imaging evidence of ongoing inflammation do occur and may prolong the time required for safe resumption of training and competition.

ELECTRICAL HEART DISEASE. Abnormalities of the cardiac ion channels may increase the risk of malignant, potentially fatal arrhythmia during sport participation. CAHAP with suspected or confirmed channelopathy should be evaluated at specialized centers that possess expertise in both genetic electrical heart disease and sports cardiology. This multidisciplinary approach facilitates accurate characterization of individual patient risk, customization of treatment that is adherent to current recommendations, and effective counseling regarding longitudinal approaches to competitive athletics and recreational exercise. For CAHAP with channelopathy, precautionary management steps include avoidance of certain prescription and over the counter medications; avoidance of dehydration, fever, and heat stroke; and establishment of an emergency action plan emphasizing external defibrillator access.

Exercise among patients with LQTS, particularly LQTS type 1, has been associated with an increased risk of arrhythmia (59). However, recent observational data describe no deaths in athletes among competitive LQTS athletes who elect to continue sport participation in conjunction with appropriate disease-specific treatment (60). Current guidelines are therefore more permissive regarding sports among LQTS athletes by permitting consideration of continued participation after evaluation at specialized centers and treatment, including the use of beta-blockers or surgical sympathetic denervation. In all cases, the sports cardiologist's recommendation to endorse unrestricted sport participation in athletes with established LQTS, or alternative diagnoses with similarly attendant risk, requires comprehensive shared decision making.

Wolf-Parkinson-White syndrome, as well as benign arrhythmias including re-entry mechanism supraventricular arrhythmias (e.g., atrioventricular nodal supraventricular tachycardia), automaticity mediated supraventricular arrhythmias (e.g., atrial tachycardia), and relatively benign idiopathic ventricular tachycardias (e.g., outflow tract tachycardias) are common among CAHAP. These conditions may be particularly amenable to catheter-based therapies that offer a potential cure. It is the responsibility of the sports cardiologist, in conjunction with a CV electrophysiologist, to ensure definitive diagnosis, risk stratification, and therapy. Evaluation may include maximal effort-limited exercise testing and an invasive electrophysiology study that can be used to determine optimal management strategies, including observation, drug therapy, or catheter ablation (61). In most cases, effective treatment will sufficiently reduce or eliminate risk to permit unrestricted participation in competitive athletics.

INTRACARDIAC DEVICE CONSIDERATIONS. CAHAP should receive an implantable cardiac-defibrillator (ICD) based on standard recommendations for primary and secondary prevention. ICD placement in CAHAP that do not meet routine ICD implantation criteria simply to facilitate "safer" future sport participation is inappropriate. Historically, sport participation among CAHAP with an ICD was restricted based on hypothetical risks, including potential failure of the ICD to terminate an arrhythmia during exercise, injury or discomfort due to an appropriate shock, and damage to the ICD system, thereby rendering it ineffective. However, recent prospective observational data suggest that young competitive athletes may be able to participate safely in competitive sports with minimal risk of life-threatening events, although ICD shocks did occur during exercise (62,63). Consequently, current

recommendations permit consideration of sports participation among athletes with ICDs, which requires a thoughtful, shared decision-making process involving the patient, care providers, and other relevant stake holders, including parents, coaches, athletic trainers, and sport organization leadership. In cases where a consensus decision is made that return to play is desirable and permissible, customized maximal effort exercise testing to evaluate for inducible arrhythmias, careful individualized device programming, and development of a remote ICD monitoring plan should be conducted prior to participation.

ATHEROSCLEROTIC CV DISEASE. Routine exercise has favorable effects on traditional CV risk factors and reduces incident ASCVD, but does not confer complete immunity. Recent survey data from community-based competitive master athletes demonstrate a high prevalence of ASCVD risk factors and indicate that ASCVD is among the most common forms of acquired heart disease in this population (64). In addition, autopsy and clinical data from observational sudden death and cardiac arrest studies routinely identify ASCVD as the most common cause of sudden death in aging CAHAP (65). The pathophysiology of ASCVD in CAHAP remains incompletely understood and constitutes an area of active investigation (66). ASCVD may be driven by traditional risk factors or by less obvious mediators, including macronutrient dietary choices, psychological stress, and perhaps high volumes of exercise itself.

Management of CAHAP with ASCVD, including those with stable disease and those who manifest with acute coronary syndromes, should adhere to consensus recommendations for the general population. Sports cardiologists may elect for more aggressive and complete revascularization strategies in CAHAP than in sedentary patients based on data documenting asymptomatic exertional ischemia caused by obstructive ASCVD as an important cause of sudden death (2). CAHAP with ASCVD should be prescribed high-dose statins and antiplatelet agents as dictated by presentation and coronary anatomy. Statin-related muscle side effects during exercise are relatively frequent among CAHAP. However, statin therapy following an acute coronary syndrome, even if it requires reductions in exercise training and competition, should be prioritized for 1 to 2 years, as this initial period of drug exposure appears to have the greatest benefit (67). The role for novel lipid-lowering agents in CAHAP who are intolerant of statins has not yet been defined, but may be considered. All CAHAP presenting with an acute coronary syndrome should be strongly encouraged to participate in a formal cardiac rehabilitation program with close oversight from a sports cardiologist, who can tailor an exercise regimen that may necessarily exceed intensity levels commonly used in more sedentary patients.

HYPERTENSION. Hypertension is common among CAHAP in clinical practice. Many hypertensive CAHAP, by virtue of how healthy they appear, are underdiagnosed and undertreated. Bilateral brachial artery blood pressures should be assessed in CAHAP at each clinical encounter, and blood pressure should be measured at least once in the leg to exclude coarctation of the aorta. Diagnostic criteria and corollary management strategies proposed by the most recent Joint National Committee document (68) are appropriate for use in CAHAP with several caveats. CAHAP with borderline blood pressure should be encouraged to perform home monitoring, using a commercial sphygmomanometer that has been calibrated against a professional grade aneroid cuff, to definitively diagnose or exclude hypertension. Most CAHAP meet or exceed levels of physical activity that have been associated with reductions in systolic and diastolic blood pressure (69), rendering escalation of exercise volume of limited value. Pharmacotherapy should prioritize vasodilators (i.e., angiotensin-converting enzyme inhibitors, dihydropyridine calcium-channel blockers) with minimal effect on exercise physiology over drugs with negative chronotropic effects and diuretic agents, which may affect chronotropic function and predispose to dehydration, respectively. Maximal effort exercise testing is of considerable value in the assessment of suspected hypertension and the management of those with established disease. Exaggerated exercise systolic blood pressures are predictive of future hypertension (70,71), and may indicate suboptimal medical therapy in CAHAP taking antihypertensive drugs (72). It is, however, important for the sports cardiologist to recognize that blood pressure may reach extremely high levels during high-intensity exercise training and competition (73): transient peak systolic blood pressures up to 250 mm Hg or even higher are expected and permissible in CAHAP without resting hypertension and underlying CV disease.

ATRIAL FIBRILLATION. Atrial fibrillation is the most common arrhythmia in the general population and may be even more prevalent among aging CAHAP. A compelling body of published data suggests that aging CAHAP, particularly those that engage in endurance sports, are at increased risk for atrial fibrillation and flutter compared with age- and sex-matched normally active people (74). As reviewed elsewhere,

TABLE 4 PPCS: Essential Skills for the Sports Cardiologist

Medical Knowledge
Know current AHA/ACA and other relevant professional organization recommendations for PPCS practices.
Know that there are insufficient data to define the effect of PPCS on sudden cardiac death.
Know the rational and potential limitations of PPCS confined to a focused medical history and physical examination.
Know the importance of using tools to standardize medical history and physical examination during PPCS.
Know the controversial role of adjunct testing, including the use of ECG and noninvasive imaging during PPCS.
Know the current variability in PPCS practices in the United States as dictated by competition level, provider preferences, and local resources.
Know the importance of establishing local resources in determining when and how to implement a PPCS program.
Know how to identify and engage relevant stakeholders that play critical roles in the design and implementation of PPCS programs.
Know potential roles of the sports cardiologist during the design, implementation, and refinement of PPCS programs.
Know how to identify clinical practice resources required to optimally support local, regional, or national PPCS initiatives.
Know how to rapidly and effectively handle abnormal findings documented during PPCS.
Know how and when to partner with specialized sports cardiology centers during the evaluation of CAHAP with abnormal findings documented during PPCS.
Patient Care and Procedural Skills
Skills to direct the planning of a new or revision of an established PPCS program.
Skills to identify key stakeholders, including but not limited to school administrators and athletic directors, coaches, athletic trainers, team physicians, and parents, during the planning and implementation of a PPCS program.
Skills to conduct and/or oversee all aspects of PPCS, including performance and documentation of a focused medical history and physical examination, performance and interpretation of ECG using contemporary athlete criteria, and direction of assessments for CAHAP deemed to have abnormal findings during PPCS.
Skills to document the yield of a PPCS program, including characterization of both true and false positive findings.
Skills to work in a team-based, collaborative approach during all aspects of the design, implementation, and refinement of a PPCS program.

PPCS = pre-participation cardiovascular screening; other abbreviations as in Table 1.

the pathogenesis of atrial fibrillation in CAHAP is complex, multifactorial, and incompletely understood (75), with casual mediators including atrial remodeling, enhanced vagal nerve activity resulting in marked resting bradycardia, inflammation, exercise-induced surges in left atrial pressure, genetics, and alcohol consumption. The first management step is to determine triggers for arrhythmia. CAHAP that develop atrial arrhythmias during exercise may be managed using low doses of negative chronotropic agents, including beta-blockers or calcium-channel blockers, with little or no effect on exercise capacity. However, this approach may be ineffective and may actually increase arrhythmia burden among CAHAP that develop atrial arrhythmias during sleep or at rest. The choice between rate control and rhythm control is generally clear in this population, as atrial fibrillation and flutter are usually poorly tolerated. In those that choose a rhythm control strategy, options including antiarrhythmic medications and ablation should be considered in the context of a shared decision making process, as there are inherent risks and benefits to both approaches. Experience with catheter-based therapy for atrial fibrillation continues to mount and appears to be effective for many CAHAP. Current U.S. and European guidelines (both Class IIa recommendations) allow for ablation as an initial approach for rhythm control. Anticoagulation to reduce the risk of thromboembolic disease should be considered in all CAHAP with atrial fibrillation and flutter (76). Many CAHAP have lone atrial fibrillation or sufficiently low thromboembolic risk profiles to avoid anticoagulation. Warfarin or a novel oral anticoagulant should be used based on algorithms proposed for use in the general population. Athletes taking these agents should be counseled about the risks of bleeding in the setting of contact sports or sports with an inherent risk of trauma such as cycling or martial arts. Following catheter ablation, anticoagulation should be continued for some period of time, although data defining the optimal duration of therapy are lacking.

PRE-PARTICIPATION CARDIOVASCULAR SCREENING

RATIONALE FOR PPCS. Pre-participation screening for occult CV disease is recommended by numerous professional organizations. The rationale for PPCS is that the detection of heart diseases associated with risk for sudden death provides an opportunity to reduced adverse events through disease-specific therapy and/or sport restriction. Recently, a more comprehensive rationale for PPCS that includes education, substance abuse screening, detection of other medical issues, and introduction to medical care providers for young competitive athletes has been proposed (77). It must be emphasized that data confirming the efficacy of PPCS as an effective strategy for reducing the incidence of sport-related cardiac arrest and sudden death are lacking. Contemporary clinical practice guidelines and expert recommendations dictating PPCS practices are, therefore, based largely on indirect evidence and expert opinion. Essential knowledge base and skill sets for the sports cardiologist to the PPCS are shown in Table 4.

PPCS RECOMMENDATIONS. Current AHA/ACC recommendations regarding PPCS endorse a strategy confined to a focused medical history and physical examination (78). This approach is justified by its reliance on minimal care provider expertise and low cost, factors expected to promote widespread dissemination with minimal resource allocation. Standardization of the medical history and physical

examination has been identified as a key aspect of high-quality PPCS. The Pre-Participation Physical Evaluation (PPE)-4, commonly referred to as the PPE Monograph, created in collaborative fashion by multiple professional organizations and endorsed by the ACC and the AHA (79), was designed for this purpose. The PPE-4 is a comprehensive guide to PPCS that represents the current standard of care and should be strongly considered for use by sports cardiologists who engage in PPCS.

The efficacy of PPCS confined to medical history and physical examination remains uncertain, but may be associated with low levels of sensitivity and specificity for CV disease detection. This concern has led some organizations to recommend the inclusion of additional diagnostic testing, including the use of ECG (18), based on its proposed ability to improve the overall accuracy of PPCS. As discussed in the previous text, studies examining the performance of ECG during PPCS consistently demonstrate its ability to detect more cases of occult disease than medical history and physical examination, but also its propensity to increase the rate of false positive results. Current U.S. recommendations discourage mandatory widespread ECG-inclusive screening for athletes and nonathletic youthful populations citing concerns about the financial and medical consequences of this approach. However, current U.S. recommendations cautiously acknowledge the potential benefit of ECG-inclusive PPCS in local settings with the necessary financial resources and clinical infrastructure.

Although several professional sporting organizations, including the National Basketball Association and the Federation Internationale de Football Association, utilize PPCS protocols inclusive of transthoracic echocardiography, this approach has not been endorsed by most professional medical organizations. Some groups that perform ECG-inclusive PPCS provide on-site echocardiography with use limited to the evaluation of CAHAP with ECGs suggestive of structural heart disease (80). However, it must be emphasized that performance and interpretation of echocardiography requires considerable clinical expertise, and onsite echocardiography should be performed only in settings with adequate quality control.

ROLE OF THE SPORTS CARDIOLOGIST IN PPCS. Sports cardiologists may play an invaluable role in the PPCS process and should be involved in its planning, implementation, and refinement. Sporting organizations' official team physicians, the individuals ultimately responsible for determining sport eligibility among CAHAP with CV disease (81), are strongly encouraged to partner with a sports cardiologist or sports cardiology program. The pre-implementation planning phase of PPCS should allow ample time for discussion aimed at determining the optimal local strategy. Sports cardiologists who participate in PPCS should be: 1) versed in current professional society recommendations; 2) aware of areas of controversy surrounding different strategies; 3) familiar with all aspects of the school or sponsoring organization including financial and personnel resources; 4) collaborative with key stakeholders including team physicians, athletic trainers, coaches, parent groups, and athletic directors; and 5) capable of the evaluation of athletes found to have abnormal findings. Sports cardiologists who support PPCS initiatives must ensure that their clinical practice environment is capable of handling a rapid influx of patients and supporting the downstream testing required following PPCS.

ADDITIONAL CONSIDERATIONS

PEDIATRIC CAHAP. Adolescent and young adults represent a significant segment of the CAHAP population. Differentiation of EICR from pathology among pediatric CAHAP is complicated by factors including rapid growth and development of the heart and blood vessels, conventional reliance on body surface area-indexed measurements, and incompletely developed motor coordination and/or ability to follow the relatively complex directions required for advanced exercise and imaging testing. Management of pediatric CAHAP requires expertise in navigating the triangular relationship between the physician, the patient, and the parents with consideration of guardianship, legal responsibility, and informed consent. Sports cardiologists not trained in pediatrics should collaborate with pediatric CV medicine providers during the care of all young CAHAP that have not completed physiological maturation.

MEDICAL LEGAL CONSIDERATIONS. Sports cardiologists considering the eligibility of an athlete with CV disease should consider the relevant legal issues (82). Current guidelines state that "a physician's general legal duty is to conform to accepted, customary, or reasonable medical practice providing sports participation recommendations consistent with an athlete's medical best interests" (83). Several relevant court cases have addressed the complexity of this issue with varying legal rulings, including the support of a university's right to exclude a student athlete from participation (Knapp v Northwestern) and the support of a professional athlete's right to make an informed decision (Mobley v Madison Square

Garden). For schools and professional teams that choose to allow an athlete to make an informed decision, many issues remain unknown including best documentation practices and the role of legal waivers. Recent changes in the AHA/ACC eligibility recommendations (53) from dichotomous yes/no recommendations to less definitive approaches, in which sport participation may be "reasonable" or "considered," opens new legal questions regarding an athlete's right to return to play that have yet to be tested in a rigorous legal framework.

PUBLIC POLICY RELATED TO THE SAFETY OF SPORTS AND EXERCISE. The sports cardiology community must remain committed to working with state legislators and community leaders to formulate policies dedicated to the health and safety of CAHAP. The ACC's Sports and Exercise Cardiology Council, the AHA, and other medical societies have authored clinical documents and recommendations to guide lawmakers and stakeholders in developing public policies aimed at improving athlete safety and reducing sudden cardiac arrest (83,84). Legislators are increasingly interested in PPCS and laws stipulating specific approaches to PPCS have been implemented. Sports cardiologists are urged to remain apprised of such initiatives, to play an active role in their planning and dissemination, and to educate legislators that no PPCS approach will eliminate exercise-related sudden cardiac arrest. Comprehensive emergency action planning focusing on access to timely bystander cardiopulmonary resuscitation and early defibrillation is required to ensure efficient and structured response to sudden cardiac arrest. Sports cardiologists are encouraged to work with organizations that sponsor, house, or oversee competitive sports and organized exercise programs to develop, rehearse, and refine emergency action plans that are specific to the athletic venue and type of exercise performed.

PERFORMANCE-ENHANCING AGENTS AND THERAPEUTIC USE EXEMPTIONS. The use of unauthorized medications, illicit drugs, and performance-enhancing banned substances is a major problem in contemporary organized athletics (85). Sports cardiologists should develop a familiarity with banned substances including anabolic steroids, stimulants, and red cell-boosting agents (86,87), and should routinely question patients about their use and counsel them about health risks and uncertain safety profiles. Banned substance lists developed by national/international governing bodies include commonly prescribed medications, such as beta-adrenergic blockers, beta-adrenergic agonists, glucocorticoids, diuretic agents, and pharmaceutical grade stimulants including methylphenidate. When use of these agents is deemed a medical necessity and appropriate alternatives are lacking, therapeutic use exemptions may be obtained. Sports cardiologists should familiarize themselves with this process.

FUTURE DIRECTIONS

The emerging field of sports cardiology is in rapid evolution. Optimal care of CAHAP requires the integration of fundamental general cardiology principles with the unique knowledge base and skillset that are specific to this patient population. As the number of practicing sports cardiologists continues to rise, it is of paramount importance that high-quality standards of clinical care and targeted research initiatives continue to develop in parallel. Future efforts to ensure the provision of effective sports cardiology care must include educational initiatives geared toward CV trainees and established CV practitioners. In parallel, the scientific community must assume responsibility for the oversight of outcomes-based research designed to address the many remaining areas of uncertainty.

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