JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY
VOL. 78, NO. 14, 2021

THE PRESENT AND FUTURE

JACC STATE-OF-THE-ART REVIEW

Exercise-Induced Cardiovascular Adaptations and Approach to Exercise and Cardiovascular Disease

JACC State-of-the-Art Review

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ABSTRACT

The role of the sports cardiologist has evolved into an essential component of the medical care of athletes. In addition to the improvement in health outcomes caused by reductions in cardiovascular risk, exercise results in adaptations in cardiovascular structure and function, termed *exercise-induced cardiac remodeling*. As diagnostic modalities have evolved over the last century, we have learned much about the healthy athletic adaptation that occurs with exercise. Sports cardiologists care for those with known or previously unknown cardiovascular conditions, distinguish findings on testing as physiological adaptation or pathological changes, and provide evidence-based and "best judgment" assessment of the risks of sports participation. We review the effects of exercise on the heart, the approach to common clinical scenarios in sports cardiology, and the importance of a patient/athlete-centered, shared decision-making approach in the care provided to athletes. (J Am Coll Cardiol 2021;78:1453–1470) © 2021 Published by Elsevier on behalf of the American College of Cardiology Foundation.

The specialty of sports cardiology, recognized within the American College of Cardiology (ACC) and European Society of Cardiology, cares for athletic individuals across the spectrum of performance and age, and addresses the challenges encountered in this growing population (1). In practice, sports cardiologists perform the following: 1) evaluate symptomatology concerning for cardiovascular

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received March 22, 2021; revised manuscript received August 2, 2021, accepted August 4, 2021.

Listen to this manuscript's audio summary by Editor-in-Chief Dr. Valentin Fuster on JACC.org.

ISSN 0735-1097/$36.00
https://doi.org/10.1016/j.jacc.2021.08.003

ABBREVIATIONS AND ACRONYMS

EICR = exercise-induced cardiac remodeling

LGE = late gadolinium enhancement

disease in athletes; 2) distinguish test findings as physiological adaptation vs pathology; 3) counsel athletes with cardiovascular disease on return to play limitations and exercise; and 4) provide strategies for prevention of sudden cardiac death, including screening and emergency action planning.

In this state-of-the-art review, members of the ACC Sports and Exercise Leadership Council address the cardiovascular effects of exercise and expected findings of the athlete's heart; describe an approach to common clinical sports cardiology scenarios; and review the importance of a patient/athlete-centered, shared decision-making (SDM) approach in the care provided to athletes. The paper is designed for those comfortable with basic understanding of principles of exercise physiology. Those less comfortable with this field of expertise should consider a primer on general principles, such as Dr Paul Thompson's review series (2).

HIGHLIGHTS

- Athletes may develop physiological adaptations found on ECG and imaging that may overlap with pathological conditions.
- ECG and imaging findings must be integrated with both the clinical history and other diagnostic testing to determine expected adaptions vs pathological abnormality.
- For athletes found to have asymptomatic and symptomatic cardiovascular disease, risk stratification must be considered before return to competitive sports participation.
- In contemporary clinical practice, a patient-centered SDM approach has become the accepted paradigm of treatment option discussions with patients.

EXERCISE

Sports are classified by the level of intensity (low, medium, or high) of dynamic and static exercise required to perform that sport during competition (1,3). "Dynamic" refers to endurance-type activities requiring regular contraction of large muscle groups and characterized by the relative percentage of maximal aerobic power (maximal oxygen uptake) required to perform that activity. "Static" refers to the strength type activities requiring sustained muscle contractions performed to overcome resistance and can be characterized by the relative percentage of a maximal voluntary contraction. Although sometimes considered in isolation, most athletic activities involve combinations of dynamic and static exercise, both of which will vary with changes in the intensity of effort and metabolic demand that occur in the context of training or competition. The combination of static and dynamic components of each sport result in specific hemodynamic effects that influence the type and degree of exercise-induced cardiac remodeling (EICR) (**Figure 1**).

Physical activity is defined as any body movement resulting from the contraction of skeletal muscle that raises energy expenditure above the resting metabolic rate of ~3.5 mL O_2/min/kg, or 1 metabolic equivalent (4). Recreational exercise, as a subcategory of physical activity, is defined as any planned, structured, and repetitive action that is pursued to maintain or improve physical fitness/health. Recreational athletes or exercisers seek the physiological and/or psychological health benefits of exercise rather than competition. Many athletes that participate in endurance or ultraendurance events are recreational athletes seeking to maximize health and wellness despite the time required to train for these events. In contrast, a competitive athlete participates in organized team or individual sporting competitions, places a high premium on achievement, and performs systematic training (5). Optimal performance and competition may be of higher importance than exercising itself, supporting the etymology of the word "athlete," which comes from the Greek root "athlos" meaning achievement (6).

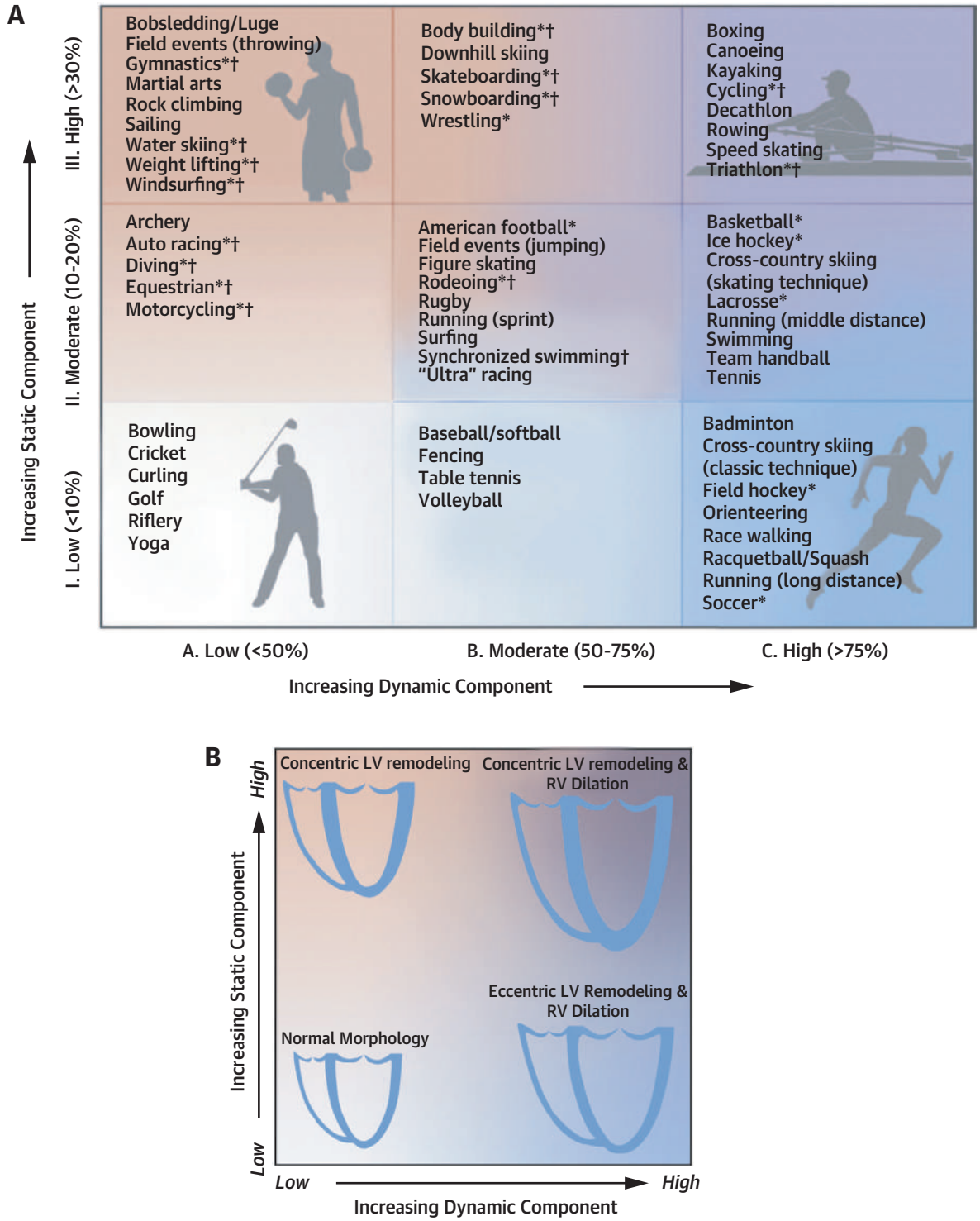
Physical activity/exercise requires a concomitant increase in pulmonary oxygen uptake, circulatory oxygen transport, and oxygen utilization at the cellular level, which is required to support the higher metabolic demands of the body. Endurance sports (ie, running, cycling, etc) require athletes to sustain high workloads at a submaximal effort over prolonged periods of time.

The relationship between cardiac output ($\dot{Q}_c$) and $\dot{V}O_2$ can be quantified by the Fick equation:

$$\dot{V}O_2 = \text{cardiac output (heart rate} \times \text{stroke volume)} \times \text{arterio} - \text{venous oxygen difference (a} - \text{v}O_2 \text{ difference)}$$

Maximal oxygen uptake ($\dot{V}O_{2max}$) is thus a physiological characteristic determined by the product of maximal cardiac output, itself the product of heart rate and left ventricular (LV) stroke volume (SV), and maximal arteriovenous oxygen content difference

FIGURE 1 **The Interplay Between Exercise Physiology and EICR**



(A) Physiological classification of common sporting disciplines representing relative contributions of static and dynamic physiology; 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; RV = right ventricular. Modified from Baggish et al (1).

(a − vO_2 difference). $\dot{V}O_{2max}$ is an important determinant of endurance exercise performance because it represents a true measure of cardiorespiratory capacity for an individual at a given level of oxygen availability (7). Prolonged exercise training facilitates higher SV, at rest and during exercise, caused by adaptive increases in LV end-diastolic volume (8). Trained athletes with enlarged hearts and corollary increases in SV maintain normal resting $\bar{Q}_c$ with heart rates as low as 30-40 beats/min. $\bar{Q}_c$ augmentation during low-intensity exercise occurs because of work-dependent increases in the heart rate and SV, but increases in $\bar{Q}_c$ during higher-intensity exercise are primarily caused by increases in heart rate.

Resting bradycardia, cardiac chamber enlargement, and the ability to generate larger SV are the cardiovascular hallmarks of the endurance athlete (9). During exercise, the cardiovascular system is exposed to the hemodynamic stresses of pressure and volume with moderate- to high-intensity dynamic exercise being the greatest stimulus for structural and functional cardiac adaptation (10). Another significant determinant of cardiac remodeling is the time that the myocardium is exposed to increased hemodynamic stress. Thus, exercise exposure can be quantified as an "exercise-dose" with principal components including the following: 1) duration; 2) frequency; and 3) intensity (11). Exercise intensity can be measured in absolute terms as the metabolic cost of an exercise session (metabolic equivalents or kilocalories) or in relative terms (eg, maximal heart rate, $\dot{V}O_{2max}$).

EICR, or athlete's heart, requires repetitive and sustained exercise exposure; both the exercise dose and nature of the sport (combination of static and dynamic exercise) influence the structural cardiac adaptations (eg, concentric LV remodeling, eccentric LV remodeling or eccentric LV remodeling) that are seen. Most studies of EICR come from cross-sectional studies of competitive athletes, and some longitudinal studies of athletes at different stages of their training cycle (12). The minimally required exercise dose to stimulate this process has been studied in relatively small numbers of healthy individuals longitudinally with high-resolution techniques such as cardiac magnetic resonance (CMR). These studies suggest that LV hypertrophy can occur after 3 months of exercise training and as little as 3-4 h/week (13,14). Moreover, it has been suggested that 3 hours of exercise/week is required to see adaptations in the resting electrocardiogram (ECG), resting heart rate, peak VO_2, and LV mass (15). The International Criteria for ECG interpretation in athletes recommend that the criteria only be applied to those exercising vigorously for at least 4-8 h/week (16). Longitudinal studies in trained athletes have shown that 90 days of intense team-based training (13.1 ± 0.9 h/week) (17) is sufficient to demonstrate EICR, and a study following cyclists over a 3-year period showed progressive LV remodeling, suggesting that EICR is an ongoing process and responsive to a sustained or repetitive exercise stimulus (18). High-intensity interval training has also been shown to elicit EICR and increase $\dot{V}O_{2max}$. High-intensity interval training is more effective in improving $\dot{V}O_{2max}$ (subsequent reductions in blood pressure and improved metabolic health) than moderate-intensity training of equal energy cost and results in larger improvements in cardiorespiratory fitness in patients with heart failure or coronary artery disease compared with sustained moderate-intensity exercise, but may result in increased musculoskeletal injuries (19,20). EICR varies considerably across individual athletes and is influenced by sport-specific physiology, prior exercise exposure, current exercise intensity and duration, host genetic factors, sex, race, and age (21).

EXERCISE AND CARDIOVASCULAR ADAPTATIONS

Identification of EICR requires a thorough understanding of the type and volume of training for the athlete to predict the type and extent of EICR specific to the athlete under evaluation (**Figure 1**) (22).

EXERCISE-RELATED ACUTE CARDIAC FINDINGS. Studies assessing ventricular function immediately following ultraendurance exercise (ranging ∼50 km to 160.93 km or 31 to 100 miles) have reported an attenuated LV and right ventricular (RV) ejection fraction, with larger reductions on the RV (23). Biventricular function immediately following an ultraendurance triathlon (a 3.8-km swim, 180-km bike, and 42.2-km run) was attenuated with larger reductions on the RV (23). This phenomenon is known as exercise-induced cardiac fatigue (24). The greater vulnerability of the right heart is likely caused by differences in wall thickness, because exercise-induced relative increases in wall stress are substantially larger for the RV vs LV (±125% vs ±4%, respectively) (25). Longer exercise duration (26) and lower training status (27) are also associated with larger reductions in cardiac function after prolonged exercise. The magnitude of LV and RV dysfunction following ultraendurance exercise (which typically range from 50 km to 160.93 km or 31 to 100 miles) is generally mild and normalizes within 24-48 hours postexercise (28). Mechanisms responsible for cardiac fatigue, which typically manifest as decreases in

global longitudinal strain and systolic twist (29), remain unclear, but several hypotheses have been proposed (30): 1) decreases in blood volume may attenuate cardiac preload (27); 2) circulating catecholamines may decrease β-adrenoreceptor sensitivity (31); and 3) oxidative stress and/or cardiomyocyte membrane damage may attenuate ventricular function (32).

Postexercise increases in cardiac troponin concentrations have been observed in athletes across many sporting disciplines (23,33). Exercise-induced troponin elevations are transient and typically normalize within 48-72 hours postexercise (34). In general, it is believed that postexercise elevations of cardiac troponin are physiological rather than pathological (35). This concept has been questioned by a recent study demonstrating increased postexercise cardiac troponin-I in long-distance walkers, which was associated with increased risk for all-cause mortality and major adverse cardiovascular events (36), and another with an association to occult obstructive coronary artery disease (37). However, it is important to note that the populations included in these studies are different from the younger and more athletic cohorts included in prior studies of cardiac fatigue (30,38). The underlying mechanisms that are responsible for exercise-induced troponin elevations are unknown (39), but recent studies have shown that marathon running can compromise cardiomyocyte integrity (40), which may allow cytosolic troponin fragments to leak from the cell (41). Future studies are needed to establish which groups, if any, (age, sex, sport discipline) with exercise-induced troponin elevations represent cardiac vulnerability as a novel cardiovascular risk factor (42).

CHRONIC CARDIAC EXERCISE ADAPTATIONS VS PATHOLOGY. Systematic exercise training results in cardiac ECG, morphological, and functional adaptations. Exercise training-induced ECG findings that do not require additional investigations include sinus bradycardia, first-degree atrioventricular (AV) block, Mobitz type I (Wenckebach) second-degree AV block, ectopic atrial arrhythmia, incomplete RBBB, early repolarization, anterior T-wave inversions in those under 16 years of age, and Black athletes and sinus arrhythmia. In addition, athletes often meet criteria for LVH using accepted criteria, which should not be applied to athletes because it results in significant false-positive ECGs.

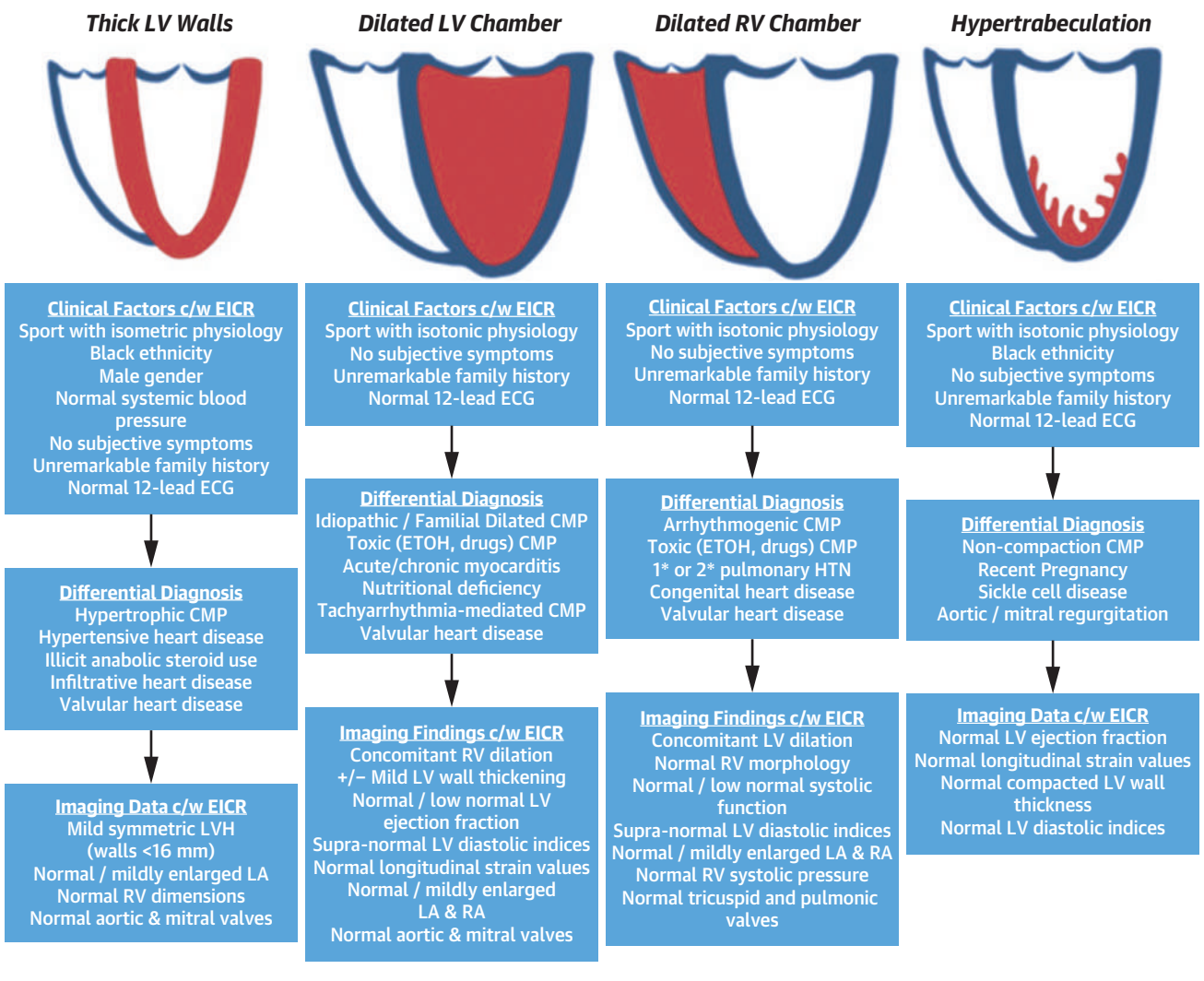
An ECG with ≤2 ventricular premature contractions is common and is considered normal in athletes. Over 2,000 ventricular premature contractions per 24 hours is associated with structural heart disease in nearly one-third of athletes (43) and warrants further investigation (44). Contemporary guidelines for interpretation of 12-lead athlete ECGs are established and should be familiar to the cardiologist asked to evaluate athletes (16).

Changes in $\dot{Q}_c$ and peripheral vascular resistance vary widely across sporting disciplines and influence degrees of chamber enlargement and cardiac wall thickening (45-47). The increased availability of sport- and sex-specific reference values of cardiac characteristics allow better discrimination of benign EICR from high-risk pathology (48,49). Dynamic (ie, endurance) exercise requires sustained elevations in $\dot{Q}_c$ with normal or reduced peripheral vascular resistance leading to 4-chamber dilation and eccentric LV remodeling (22). In contrast, static (ie, strength or resistance) training results in elevations in blood pressure accompanied by pulsatile increases in peripheral vascular resistance with resultant concentric LV remodeling (50).

Ventricular hypertrophy. In dynamic training, enlargement of all cardiac chambers is observed (51) and is proportional to the athlete's fitness and body size (52). EICR is often mild and easily attributable to exercise; in some athletes, the degree of remodeling may overlap with mild forms of cardiomyopathy, referred to as a diagnostic "gray zone" (**Figure 2**). This gray zone is of high clinical relevance given that genetically inherited structural cardiomyopathies are among the top causes of sudden cardiac arrest identified in young athletes (53-55). In these cases, it is important to clarify the pretest probability of significant cardiac disease, which is higher when the athlete is presenting with exertional symptoms or a family history of cardiomyopathy vs an incidental finding. Evaluation of gray-zone cases should be individualized based on the clinical context by a medical team experienced in cardiac imaging and assessment of athletes.

An important gray-zone example is the evaluation of the athlete with increased LV wall thickness and possible hypertrophic cardiomyopathy (HCM). In this example, LV wall thickness ≥15 mm in an athlete should, in nearly all instances, be considered pathological. LV wall thickness in the range of 13-14 mm constitutes the diagnostic gray zone because of the overlap between mild HCM and physiologic LV remodeling (56). Physiological LV hypertrophy is more common in men, in Black athletes, and in sports with a moderate-to-high static component, including team sports and some endurance sports such as cycling and rowing (45,57-59). Pathological LV

FIGURE 2 The Gray Zone Imaging Findings



Overview of the clinical assessment of the 4 cardinal "gray zone" imaging findings in competitive athletes for use in differentiating EICR from pathological cardiomyopathy (CMP). ECG = electrocardiogram; ETOH = ethyl alcohol; HTN = hypertension; LA = left atrium; RA = right atrium; other abbreviations as in Figure 1. Reprinted from Baggish et al (22).

hypertrophy caused by HTN and steroid use can be mistaken for physiological hypertrophy.

Imaging must be integrated with the athlete's exercise history to determine if findings match what could be expected based on sport and competition level, ethnicity, sex, and body size effects of sustained exercise on cardiac remodeling (47). Exercise testing to quantify exercise capacity can corroborate the reported exercise history and, if unexpectedly low, may indicate a cardiomyopathy. However, athletes with cardiomyopathy who remain physically active may maintain normal to supranormal exercise capacity, and therefore, these results do not provide reassurance against pathology (60). Structural abnormalities of the mitral valve with resting or exercise-induced LVOT obstruction favor the diagnosis of HCM. Abnormal ECG findings escalate suspicion for cardiomyopathy, because overt ECG manifestations of an HCM phenotype can precede development of hypertrophy (56,61).

If concern for HCM persists after the initial evaluation, CMR should be performed to assess for segmental hypertrophy and for associated fibrosis with late gadolinium enhancement (LGE). CMR is superior to echocardiography for visualization of the

apex, anterior, and anterolateral walls with more precise LV wall measurements (62,63). Mild wall thickness asymmetry has been described in healthy active individuals (64), but more marked asymmetry should raise suspicion for HCM. Imaging should be scrutinized for supportive EICR findings such as balanced enlargement of all 4 cardiac chambers, enhanced SV, and enhanced LV diastolic function. Importantly, these EICR findings are more likely to be present in athletes in high dynamic/endurance sports (58) and may persist in HCM individuals who remain highly active (60). The age of the athlete should be considered during assessment as well because the expression of the phenotype occurs over time; this strengthens the argument for repeat imaging for some athletes. The presence of LGE has been associated with and favors a pathological process. However, RV insertion point fibrosis in master athletes (65) and younger asymptomatic athletes with COVID-19 with normal ECGs/examinations and without arrhythmias call into question the significance of this pattern (66).

If a thorough evaluation fails to produce a definitive diagnosis for gray-zone LV hypertrophy, prescribed detraining for hypertrophy regression may be considered. This strategy should always be used with caution because of the lack of normative data for the rate and completeness of expected LVH regression in healthy athletes, although atrophy at a rate of ~1%/week has been observed during strict bedrest (67,68). Importantly, detraining-mediated regression in some athletes with HCM has been reported; therefore, a reduction in LVH in the absence of exercise may not be specific to EICR (69). Finally, detraining requires complete cessation of exercise, which may be unappealing to the athlete.

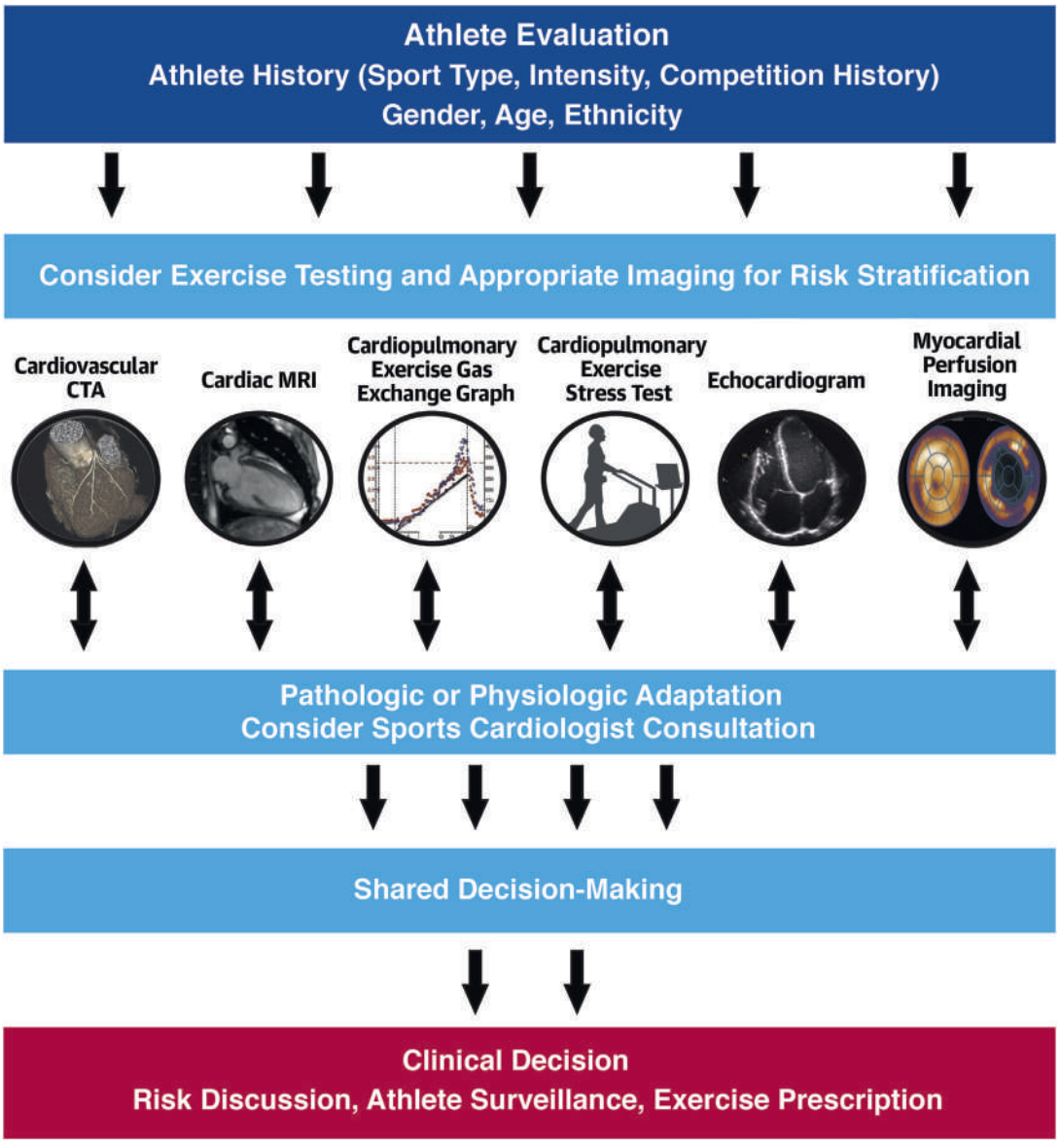
VENTRICULAR ENLARGEMENT. Ventricular dilation is common among athletes engaged in sports with a moderate-to-high dynamic component (58,70). This may be accompanied by a mildly reduced LV ejection fraction (45%-50%) in elite endurance athletes (71), which can complicate differentiation from dilated cardiomyopathy. The presence and/or magnitude of LV dilation does not distinguish between adaptive and pathological remodeling because athletes may develop marked physiological LV dilation without a true upper limit of normal. Therefore, the assessment should not focus only on chamber size. Physiological LV dilation should be accompanied by balanced dilation of all cardiac chambers coupled with normal to supranormal LV diastolic function. Isolated LV dilation, reduced diastolic function, abnormal global longitudinal strain (worse than −16%) (22), regional wall motion abnormalities, and fibrosis by LGE on CMR are all supportive of cardiomyopathy. Stress echocardiography is a potentially valuable tool in this setting with expected ejection fraction increases >11% or to >63% at peak exercise favoring EICR (72). Endurance athletes can have persistent LV dilation even after cessation of exercise, limiting the use of detraining as a diagnostic tool in those with suspected cardiomyopathy (73).

Athletes engaged in endurance or mixed sporting disciplines may also develop enlargement of the RV and the right atrium. The greater the duration and intensity of dynamic exercise, the larger the $\dot{Q}_c$ and hemodynamic stimulus for dimensional increase of RV/right atrial chambers. Athletes often exceed dimension limits used for the diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC); thus standard "cutoff values" should not be employed. To differentiate adaptive RV enlargement from pathology, there must be confirmation of normal RV architecture in combination with normal RV systolic function, which can be assessed using multiple parameters including RV fractional area change, s′ velocity, tricuspid annular plane systolic excursion, RVEF using 3-dimensional imaging, and RV strain. RV dilation attributed to EICR should be accompanied by concomitant 4-chamber enlargement; an enlarged RV in isolation is atypical and requires a complete workup. Excluding ARVC is critical, because evidence indicates that endurance exercise in those with ARVC sequence variants leads to progression of the disease phenotype (74). CMR may be useful to establish chamber size and functional assessment. CMR can better assess for focal wall motion abnormalities, saccular aneurysms, and focal RV thinning. LGE identification in the RV is challenging but has been associated with increased risk in ARVC (75).

Athletes with gray-zone findings and absent pathology should be followed longitudinally with surveillance imaging given the dynamic nature of phenotypic disease expression in genetically inherited cardiomyopathies. Involvement of experts in sports cardiology and athletic cardiac imaging in challenging gray-zone cases is highly encouraged.

Increased LV and RV trabeculations are common in athletes and should not be confused with pathology, particularly in the setting of a normal ECG and normal systolic and diastolic function (76). Underlying mechanisms and explanations for the high prevalence of hypertrabeculation in athletes are speculative; however, the volume load of chronic athletic participation may serve as a stimulus, similar to pregnancy (77). Subsequently, isolated hypertrabeculation, in the absence of any additional clinical or imaging risk

CENTRAL ILLUSTRATION **Athlete Assessment: Clinical Evaluation, Shared Decision-Making, and Athlete-Centered Decision**



Martinez, M.W. et al. J Am Coll Cardiol. 2021;78(14):1453–1470.

Illustration of the athlete assessment with cardiovascular disease and arrival at a patient centered decision. **Circles from left to right** respectively illustrate: cardiovascular CTA, cardiac MRI, cardiopulmonary exercise gas exchange graph, cardiopulmonary exercise stress test, echocardiogram, myocardial perfusion imaging.

factors, has not been demonstrated as an established marker of increased cardiac risk (78).

CARDIOVASCULAR RISK DURING EXERCISE IN ATHLETES. For the sports cardiologist, common clinical scenarios in practice involve guidance of exercise and sports participation for athletes diagnosed with cardiovascular risk factors and/or disease (Central Illustration). The approach to these challenging clinical cases is nuanced and individualized, but a general framework is often consistent among experts. Many general or specialized cardiologists will initiate a work-up and collaboratively work with a sports cardiology expert team to define risk

assessment and a management plan including exercise prescription. Overall, it is imperative that the guidance an athlete receives should remain in line with a patient-centered approach, inclusive of accurate risk assessment followed by SDM with the athlete and key stakeholders.

Atherosclerosis. In master athletes, traditional atherosclerotic risk factors may be absent and a traditional Framingham risk score may incompletely assess cardiovascular risk (79). Patients with clinically concealed and asymptomatic atherosclerotic cardiovascular disease (ASCVD) are typically recognized by the presence of coronary artery calcification (CAC) or noncalcified plaque detected by coronary computed tomography angiography and absence of ischemia on provocative testing.

Approach to the asymptomatic athlete with elevated CAC. The presence/extent of CAC is a marker of ASCVD used to detect atherosclerosis and predict prognosis (80). There exists controversy as to whether high levels of physical activity and endurance training can accelerate ASCVD as measured by CAC (81,82). Among athletes and nonathletes, a 0 calcium score carries the best prognostic risk for future events, and a lower CAC score has a better prognosis than an elevated CAC (83). Existing data indicate that higher CAC burden in older athletes can often be explained by traditional CAD risk factors but may harbor increased risk even in the absence of risk factors (82). Importantly, data suggest that those with elevated CAC who exercise more often carry better outcomes compared with those with similarly high CAC who exercise less frequently (83). Despite limited evidence, the 2010 ACC/American Heart Association (AHA) guidelines for the assessment of cardiovascular risk in asymptomatic adults recommends stress myocardial perfusion imaging for advanced cardiovascular risk assessment in asymptomatic adults with CAC score >400 with concomitant diabetes or with a strong family history of ASCVD (Class IIb) (84). The extent of CAC is associated with increased prevalence of inducible ischemia by myocardial perfusion imaging in nonathletes with an incidence of 1.3%, 11.3%, and 35.2% for CAC scores of <100, 100 to 400, and >400, respectively (85). In nondiabetic patients, the CAC score threshold at which the prevalence of ischemia increases substantially is still >400, although no data are available for athletes (86). Importantly, asymptomatic athletes with high CAC and ischemia do NOT all require invasive catheterization. All ischemia is not significant ischemia, and a maximum exercise test with borderline or mild ischemia represents a different risk than symptomatic patients with profound ischemia at a low workload (87). Stable ischemic patients should be treated aggressively (see below) with invasive risk stratification (ie, coronary angiography ± IFR/FFR) considered for patients/athletes with multivessel disease and/or those with potentially flow-limiting lesions with objective evidence of extensive inducible ischemia on functional testing, especially if present at workloads typically encountered during training or competition. Alternatively, noninvasive options should be considered if the ischemia is mild/equivocal and medical therapy can be optimized. Each approach requires an a priori discussion, using a shared decision-making about the pros and cons of percutaneous revascularization.

Guideline recommendations indicate those with a CAC >400 undergo symptom-limited, and preferably sport-specific maximal exercise stress testing (88). We also advocate aggressive lipid treatment to achieve very low, low-density lipoprotein cholesterol values with strong consideration to achieve targets below <55 mg/dL as opposed to <70 mg/dL (89-91) to facilitate regression. In those with CAC ≥100 or ≥75th percentile, statin therapy should be discussed with the athlete, emphasizing the limitations of current data with respect to athletes and potential side effects. Athletes should be reminded that initiation of statin therapy will likely lead to an increase in CAC (92), and that such a change is normal; therefore, repeated CAC testing is discouraged. In addition, a focus on dietary habits, symptom awareness, and healthy training practices is paramount.

Approach to the patient with clinical or symptomatic ASCVD. The risk of exercise-related major adverse cardiovascular events is greater in individuals with diagnosed ASCVD than in those with subclinical ASCVD. This risk increases with exertion, extent of coronary artery disease, degree of LV dysfunction, presence and extent of ischemia, and the presence of electrical instability (3).

Athletes with a history of stable angina, percutaneous coronary intervention (PCI), or coronary artery bypass graft (CABG) surgery should all undergo supervised, exercise-based cardiac rehabilitation. Cardiac rehabilitation participation carries long-term health benefits and includes a focus on risk factor modification/reduction. Athletes recovering from PCI or CABG should refrain from competition and strenuous training to allow myocardial healing and atherosclerotic lesion stabilization, although the defined length of healing time is unknown and is influenced by the indication for revascularization (acute coronary syndromes vs stable angina). Data suggests stabilization and perhaps plaque regression after aggressive lipid reduction with statins or PCSK9

TABLE 1 Valvular Heart Disease Risk Considerations for Sport and Exercise

Valve disease with increased risk from competitive sports
1. Symptomatic athletes 2. Asymptomatic athletes with a. Severe aortic stenosis. b. Severe aortic regurgitation with LV systolic dysfunction or LV end-systolic diameter out of proportion to their athleticism. 3. Severe mitral stenosis. 4. MR and LV systolic dysfunction at rest (LV ejection fraction <60% or LV end-systolic diameter >40 mm) or pulmonary hypertension. 5. Aortic valvular heart disease and aortic dimensions of 45 mm or greater. 6. Athletes requiring oral anticoagulation. 7. Malignant mitral valve prolapse (prior arrhythmogenic syncope, ventricular tachyarrhythmias, or family history of unexplained cardiac arrest).
Valve disease with intermediate risk
1. Moderate aortic stenosis (including BAV) with normal LV systolic function, acceptable exercise capacity, and blood pressure response without tachyarrhythmias. 2. Severe MR without arrhythmias, normal LV systolic function, and LV enlargement consistent with athletic training. 3. Athletes with bioprosthetic mitral or aortic valves, without oral anticoagulants, with normal LV function, and without valve dysfunction. 4. Athletes who have undergone successful mitral valve repair without residual severe valve disease and have normal LV systolic function. 5. Athletes with aortic valvular heart disease (including BAV) with aortic dimensions of 42 mm or greater.
Valve disease with low risk from competitive sports
1. Asymptomatic mild or moderate valvular heart disease with presumed normal LV systolic function, normal pulmonary artery pressures, and normal exercise tolerance. 2. Severe AR with preserved LV function and a normal exercise tolerance. 3. Athletes with MS who have undergone successful percutaneous mitral balloon valvotomy or surgical commissurotomy based on the residual severity of MS or MR and pulmonary pressures at rest and with exercise. 4. Bicuspid aortic valves with mild to moderate valve disease and aortic size under 42 mm. 5. Mitral valve prolapse with preserved LV systolic function and without severe regurgitation or prior arrhythmogenic syncope, ventricular tachyarrhythmias, or family history of unexplained cardiac arrest.

BAV = bicuspid aortic valve; LV = left ventricular; MR = mitral regurgitation; MS = mitral stenosis.

inhibitors with a range of 6-24 months. Recommendations have included a minimum of 6 months of aggressive therapy to achieve and maintain a low-density lipoprotein cholesterol value <55 mg/dL, then slow return to exercise (90,91,93). Athletes with ASCVD should develop a strong commitment to aggressive nutrition lifestyle modifications (94). A risk stratification maximal effort exercise test and ambulatory rhythm monitoring may be appropriate in those with prior symptomatic ASCVD to estimate the risk of return to competitive sport.

Valvular heart disease and exercise considerations. There are no prospective clinical trials examining the effects of vigorous and intense exercise on disease progression or risk of competitive sports in those with valvular heart disease. Current recommendations regarding sports eligibility with underlying valvular heart disease are based on expert opinion and long-term cohort analyses of nonathletic individuals with these conditions (95,96). Opinions are based upon the presence of symptoms, etiology and severity of the valvular disease, cardiac structure and function, risk of arrhythmias, and pulmonary artery pressures (97). Patients with mild valvular heart disease, whether regurgitant or stenotic, tolerate exercise well without increased risk. Symptomatic athletes attributed to severe valvular lesions are not eligible for competition and should be referred for valve replacement or repair before the determination of sports eligibility (98). Bicuspid aortic valve (BAV) is common (0.9%-2%) with a slight male predominance, and can be a sporadic or autosomal dominant inheritance with variable penetrance (99). TTE surveillance for both valvular and aortic pathology is needed along with first-degree relatives. As a general framework, we provide categories of risk for athletes with valve disease based on the AHA/ACC scientific statement (100) (**Table 1**).

Clinicians evaluating individuals with valvular heart disease should integrate the history, physical examination, and ECG with echocardiographic findings. Exercise stress testing may help assess symptom status, functional capacity, hemodynamic response to exercise, exercise-induced arrhythmias, and ischemia. Maximal effort exercise stress testing should be conducted to the level of effort achieved during competition and training (88). To determine valve disease severity, an understanding of the specific valve disease, coupled with EICR, is mandatory because of overlap between maladaptive and adaptive structural changes.

Aortic enlargement. Thoracic aortic dissection is extremely rare in athletes but, if it occurs, can be fatal. Exercise exposes the aorta to hemodynamic stress, which in vulnerable individuals may exceed its rupture threshold and result in dissection or death. Identification of those at risk and the provision of guidance on appropriate activity levels is vital.

Thoracic aortic dilation is generally asymptomatic and identified as an incidental finding. Widespread screening for aortopathy is impractical, but aortic imaging should be obtained if there is a positive family history of aortopathy or dissection, phenotypic characteristics of a connective tissue disorder, or auscultative findings suggestive of a BAV or mitral valve prolapse.

Normal ranges of aortic size are dependent on age, sex, and body size (101). Although Z-scores are well-established in the pediatric population, such indexing is not available for adult athletes. Although there is a close relationship between body size and aortic dimensions, there is a plateau in this relationship at the extremes of height such that aortic size is rarely above 40 mm in young athletes (102).

The impact of an athletic lifestyle on aortic size is increasingly appreciated. The aorta likely adapts with changes in size and compliance. A meta-analysis of aortic diameters in young athletes (<35 years of age) compared with sedentary control subjects reported that aortic root measurements were 3.2 mm greater in athletes (103). Despite this, it remains rare (<2%) for a young athlete's aorta to exceed 40 mm in men or 34 mm in women (103). Greater dimensions warrant further evaluation for additional high-risk features. A number of studies evaluating the long-term effect of exercise on aortic size have reported a higher rate of aortic dilation in master athletes compared with control subjects (104,105). Whether this translates to increased risk of dissection is unclear, but individuals should undergo surveillance and management in line with recommendations for the general population.

Once a dilated aorta is identified, it is important to define the etiology including the presence of an inherited aortopathy, such as Marfan syndrome or BAV, who may be at higher risk of dissection and require prophylactic surgery at lower thresholds than the general population (**Table 1**). Thoracic aortic aneurysm surveillance imaging should be performed using similar imaging every 6 months to 3 years based on etiology and aortic size.

Exercise guidance is individualized based on risk and the athletes' training regimen and sport. Despite increased SV, endurance exercise only imparts modest increases in mean arterial pressure (MAP), whereas resistance training can result in dramatic changes in MAP. However, transmural pressure across the aorta may not be large when combined with a Valsalva maneuver (106). For the strength athlete, the magnitude of these changes depends on the intensity of work, muscle group size, weightlifting techniques, and the level of fatigue (106). Given these nuances, single cutoff weight restrictions should not be used, but rather, individualized and qualitative limitations should be incorporated into SDM discussions.

Sport participation risk depends on multiple factors including etiology, family history of dissection, aortic size, risk of bodily collision, and sport-specific hemodynamic factors. Current guidance is expert opinion; thus, decisions should be individualized and undertaken in conjunction with experts in sports cardiology/aortopathy using an SDM framework (107).

Hypertension. Exercise should be included as first-line therapy for hypertension (108). Endurance training leads to reductions in arterial stiffness and may attenuate age-related arterial stiffening (109). In some strength-based sports such as American-style football, there is a deleterious impact on arterial stiffness. Resting hypertension remains a commonly encountered risk factor even among competitive and recreational athletes across the age spectrum (110). In adults, normal blood pressure (BP) is defined as <120/80 mm Hg. Competitive sports are permissible with a diagnosis of essential hypertension in most cases, but may be restricted in those with uncontrolled severe hypertension until control is obtained (111).

Athletes require specific considerations in the clinical approach to elevated BP measurements. Athletes may have nonstandardized BP assessments during periods of high psychological stress (ie, preseason physicals), which can lead to falsely elevated measurements. Acquisition of serial BP measurements in the training room or serial ambulatory BP measurements for recreational athletes are necessary to confirm the diagnosis of hypertension. AHA standards recommend that BP should be averaged over 3 measurements in each arm, separated by at least 1 minute (112). Appropriately sized cuffs are of paramount importance (ie, cuff bladder encircling at least 80% of the arm), particularly for large athletes in whom increased arm circumference (undersized cuffs) may lead to falsely elevated measurements. Young athletes with hypertension may escape detection because of the lack of clinical follow-up. Finally, sport type should be considered because certain groups, such as American-style football athletes, are known to harbor increased risk of hypertension (110,113).

Athletes with hypertension should be evaluated with a focused history and physical examination to assess for secondary causes. This should include measuring BP in both arms and legs to exclude an aortic coarctation, assessment for signs/symptoms

of obstructive sleep apnea, evaluation for nonsteroidal anti-inflammatories/stimulants use (including over-the-counter supplements), alcohol or oral contraceptive use, and abuse of performance-enhancing drugs (ie, anabolic steroids). For master athletes, a complete ASCVD risk assessment, including a fasting lipid profile, serum chemistries, and social and family history, is essential (114). The diagnostic evaluation should include a baseline ECG (108) and TTE for assessment of LV hypertrophy, chamber geometry, diastolic function, aortic size, and coarctation (younger athletes) with consideration of a renal ultrasound.

After lifestyle modifications, guideline-based pharmacotherapy is dependent on cardiac risk factors including diabetes mellitus, coronary artery disease/myocardial infarction, or chronic kidney disease (108). Angiotensin-receptor blockers, angiotensin-converting enzyme inhibitors, or dihydropyridine calcium-channel blockers are considered first-line therapy based on the clinical profile of the athlete. Sport-specific factors should be considered such as fluid/electrolyte shifts during vigorous training that can be exacerbated by diuretic agents, and competitive athletes may require therapeutic exemptions for many guideline-based therapies (111).

ATRIAL FIBRILLATION. Atrial fibrillation (AF) is the most prevalent sustained clinical arrhythmia worldwide (115). AF risk factor reduction occurs with moderate physical activity, yet paradoxically, higher levels of prolonged vigorous activity attenuate the benefits of moderate activity with higher risk of developing AF compared with control subjects (116,117). Mechanisms underlying the association between AF and endurance exercise include: atrial dilatation/fibrosis, vagal tone augmenting dispersion of atrial refractoriness and pulmonary vein triggers, autonomic nervous system imbalance related to exercise-related adrenergic stimulation, and augmented atrial pressures with increased exercise-related filling pressures (118).

Risk factors in athletes with AF are the same as the general population, including sleep-disordered breathing, and a history should question excessive daytime sleepiness, use of stimulant use, or use of PEDs (119). In a study of master athletes with more than 20 years of endurance athletics experience, 64% had at least 1 cardiovascular risk factor and 12.0% had hypertension (120).

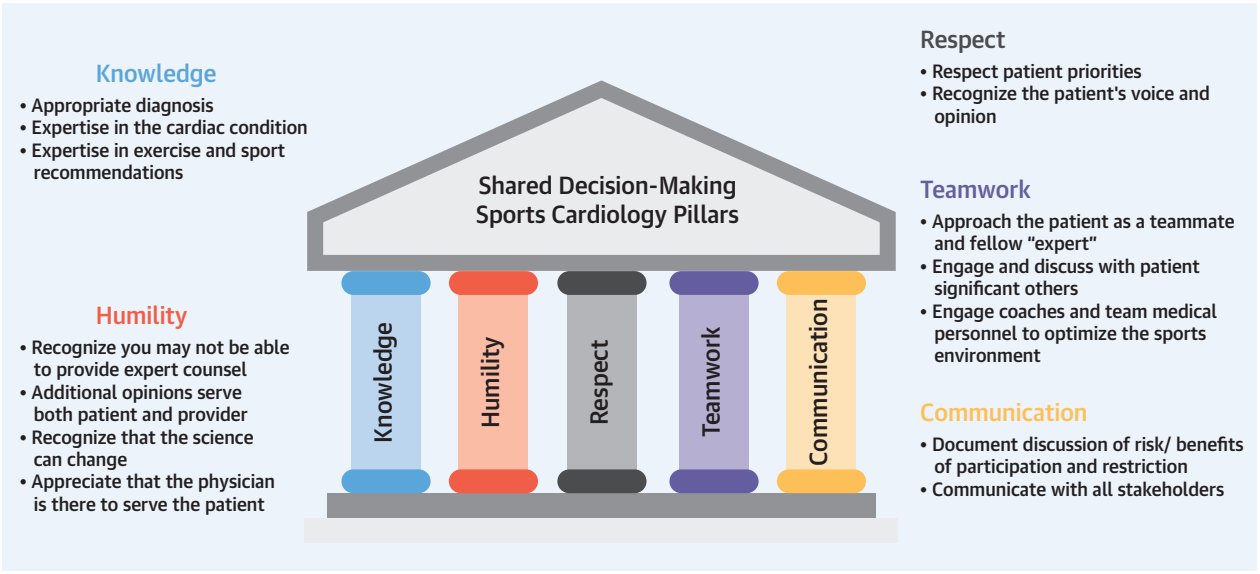
Athletes with AF require imaging to assess cardiac structure focused on increased chamber sizes, LV wall thickness/mass, and atrial morphology. In a meta-analysis of 54 studies comprised of 7,189 athletes, the mean left atrium (LA) diameter was 4.1 mm greater in athletes (4.6 mm in endurance athletes) compared with sedentary control subjects, and the LA volume index was also 7.0 mL/m^2 greater in athletes (121). LA enlargement may persist after athletes have stopped sport participation, based on a study of 62 professional cyclists after 30 years of detraining in which the average atrial volume remained 5 mL/m^2 larger compared with nonathletes (122). At present, no large comprehensive studies indicate that detraining mitigates AF risk among endurance athletes.

Treatment is founded upon improving symptoms and lowering risk of AF-related comorbidities. Anticoagulation to prevent thromboembolism is based upon the same guidelines as nonathletes with consideration for the risk of bleeding from collisions or falls. Creative strategies for withholding anticoagulants have been used to allow participation in some situations (123). Enhanced vagal tone-induced bradycardia is often cited as a reason to avoid AV nodal blocking agents; however, athletes often tolerate beta-blockade better than expected, primarily because of low resting sympathetic tone. Class IC antiarrhythmic agents, such as flecainide, are often preferred and reduce the risk of AF recurrence compared with beta-blockers alone with a better safety profile than class IA drugs (124). Caution must be applied during exercise because of increased risk of proarrhythmia from use-dependence (125).

Data comparing medical treatment vs invasive therapies for symptomatic AF in athletes are sparse. Early catheter ablation may improve symptoms, reduce recurrence, and avoid pro-arrhythmia. If ablation is favored, limited pulmonary vein isolation may be used to avoid compromise of atrial function and increased chamber stiffness. Adequate rest and exercise pause is important; consider 4-6 weeks following an ablation. Exercise too early may limit the efficacy and durability of the procedure. Many AF triggers recur with return to endurance athletics; thus, it is critical to inform the athlete that more than 1 procedure may be required to remain free of recurrent arrhythmia (126).

IMPLANTED DEVICES. Athletes with implantable cardioverter-defibrillators (ICDs) have historically

FIGURE 3 **SDM Sports Cardiology Pillars**



An illustration of the 5 pillars that make up a prescribed method of shared decision-making (SDM) that includes the required points to guide the patient-physician discussion.

been considered at higher risk of complications during vigorous physical activity because of concern regarding device efficacy during sport, lead dislodgement, or damage to the ICD. For example, the 2005 U.S. sports eligibility expert consensus recommendations concluded "for athletes with ICDs, all moderate and high intensity sports are contraindicated" (127).

A multinational, prospective, observational ICD registry has led to better understanding of device efficacy in patients participating in sports more vigorous than IA activities (128,129). A total of 440 athletic patients across multiple sporting disciplines, after a median follow-up of 44 months, revealed no cases of ICD failure during/after sports participation (129). In total, 10% received appropriate shocks during competition or practice, and only ARVC was associated with receiving appropriate shocks. Importantly, freedom from lead malfunction was 95% at 5 years. These data demonstrated that patients with ICDs can engage in vigorous/competitive sports without increased risk of device malfunction or tachyarrhythmia/death. Therefore, the 2015 expert consensus recommendations were updated (Class IIb recommendation), indicating that athletes with ICDs may participate in most sports if they have been reasonably free from ventricular tachyarrhythmia (130). ICD implantation was discouraged (Class III) for the purpose of sport participation.

For athletes with permanent pacemakers, sports participation depends on the underlying structural heart disease, the degree of pacemaker dependence, and risk of damage to the device (130). Those who are completely pacemaker-dependent are advised to avoid contact sports when collisions may cause device dysfunction. For athletes who are not pacemaker-dependent who participate in sports with risk of collision, protective equipment for the generator is advised.

SDM AND EXERCISE CONSIDERATIONS. In contemporary clinical practice, a patient-centered and SDM approach has become the accepted paradigm of treatment option discussions with patients (131,132). SDM requires the practitioner to counsel and explain the risks and benefits of clinical management options to patients and guide them in understanding how to consider all options in combination with the patient's personal preferences, morals, and values. This approach was first described by Levine and Stray-Gundersen (133), who emphasized the obligation of the physician to discuss risk stratification/acceptance and alternative strategies for individuals with cardiovascular disease. Of paramount importance, SDM shifts away from the former paternalistic and binary

process for potential sports disqualification (100), and instead involves equal participation from the athlete and other key stakeholders in the shared risk decisions to either proceed with sports participation or retire from athletics.

Clinical scenarios may arise in sports cardiology that necessitate an SDM approach in the context of exercise and sports eligibility. For example, new pathological cardiac diagnoses or interventions in the management of cardiac disease all require consideration of changes in exercise or training habits for the competitive athlete or highly active individual. It is noteworthy, however, that sports eligibility SDM involves risk discussions focused on lifestyle (or profession) and not clinical treatment options, which adds a layer of complexity compared with other clinical SDM scenarios.

Updated sport consensus and HCM guidelines acknowledge the clinical uncertainty and allow for individualized SDM. Guideline changes were driven by observational data from athletes that demonstrated cardiac risks of competitive sport participation with several conditions were lower than previously believed (56,134,135), and the ethical imperative to include patients in the determination of their medical care has become increasingly emphasized. The paucity of randomized controlled trials and lack of robust data that define the safety of exercise and sports participation in various cardiovascular conditions emphasize the critical need to engage the athlete in these discussions.

SDM should not be a "rubber-stamp" allowance to continue with sports participation for all athletes. Risk stratification of the athlete with consultation by experts in sports cardiology and other disciplines is mandatory. Along with the risk of sudden cardiac death, the focus of SDM must include the potential negative psychological, social, and overall health impacts of sports restriction (136).

For a successful SDM discussion, 5 pillars (**Figure 3**) should be used by the provider:

1. **Knowledge.** The provider must be knowledgeable in the field of sports cardiology, adjudicate appropriate risk stratification in specific clinical scenarios, and reasonably estimate the risk of ongoing physical activity for the specific cardiac condition. The provider must also appreciate the negative implications of exercise restriction.
2. **Humility.** Different clinical scenarios and disease processes require specific expertise and skill sets. As such, sports cardiologists must recognize when to involve separate opinions and other expert colleagues in the discussion of sports eligibility for specific diagnoses.
3. **Respect.** The provider must respect the patient's values, goals, and priorities.
4. **Teamwork.** In respecting the values of the athlete, the provider must also avoid approaching the SDM process in a hierarchical manner. Other key stakeholders, including family members, coaches, trainers, and sports medicine/primary care providers, are members of the athlete health care team and should be involved in all discussions.
5. **Communication.** The provider must be able to appropriately communicate and document the risks and benefits of participation and restriction to the athlete as well as the outcome of all SDM meetings. Ensuring that all parties agree and understand decisions that arise from detailed encounters are critical to the process.

CONCLUSIONS

In this state-of-the-art review, we describe the expected findings of the "athlete's heart" and specific clinical scenarios germane to sports cardiology, which reflect the varied considerations of a sports cardiologist. Finally, we emphasize a patient-/athlete-centered SDM approach in the care provided to athletes and highly active individuals.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr Martinez has served as a consultant for Major League Soccer and Bristol Myers Squibb. Dr Kim has received research funding through the National Institutes of Health; and has received compensation as team cardiologist for the Atlanta Falcons. Dr Bunch has received research grants from Boehringer Ingelheim, Boston Scientific, and Altathera (no personal compensation). Dr Baggish has received support from the National Institute of Health/National Heart, Lung, and Blood Institute, the National Football League Players Association, and the American Heart Association; and receives compensation for his role as team cardiologist from the U.S. Olympic Committee/U.S. Olympic Training Centers, U.S. Soccer, U.S. Rowing, the New England Patriots, the Boston Bruins, the New England Revolution, and Harvard University. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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KEY WORDS athletes, prevention, sports cardiology

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