Recommendations on the Use of Multimodality Cardiovascular Imaging in Young Adult Competitive Athletes: A Report from the American Society of Echocardiography in Collaboration with the Society of Cardiovascular Computed Tomography and the Society for Cardiovascular Magnetic Resonance

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Keywords: Athlete, Athlete's heart, Pre-participation screening, Echocardiography, Cardiac computed tomography, Cardiac magnetic resonance

In addition to the collaborating societies listed in the title, this document is endorsed by the following American Society of Echocardiography International Alliance Partners: Argentine Federation of Cardiology, Argentine Society of Cardiology, Asian-Pacific Association of Echocardiography, Australasian Sonographers Association, Brazilian Department of Cardiovascular Imaging, Canadian Society of Echocardiography, Cardiovascular and Thoracic Society of Southern Africa, Cardiovascular Imaging Society of the Interamerican Society of Cardiology, Chinese Society of Cardiothoracic and Vascular Anesthesiology, Chinese Society of Echocardiography, Cuban Society of Cardiology Echocardiography Section, Indian Academy of Echocardiography, Indian Association of Cardiovascular Thoracic Anaesthesiologists, Iranian Society of Echocardiography, Israel Working Group on Echocardiography, Japanese Society of Echocardiography, Korean Society of Echocardiography, Mexican Society of Echocardiography and Cardiovascular Imaging, National Association of Cardiologists of Mexico, Philippine Society of Echocardiography, Saudi Arabian Society of Echocardiography, Thai Society of Echocardiography, and Vietnamese Society of Echocardiography.

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The following authors reported no actual or potential conflicts of interest in relation to this document: Aaron L. Baggish, MD (Chair), Robert W. Battle, MD, Timothy A. Beaver, MD, FASE, William L. Border, MBChB, MH, FASE, Matthew W. Martinez, MD, Jennifer H. Mercandetti, BS, RDCS (AE/PE), ACS, FASE, Dermot Phelan, MD, PhD, FASE, Tamanna K. Singh, MD, Rory B. Weiner, MD, FASE. The following authors reported relationships with one or more commercial interests: Pamela S. Douglas, MD, FASE owns stock in UpToDate/Kluwer and is DSMB for REAL TIMI 63B; Christopher M. Kramer, MD received grant support from Regeneron and is a consultant for Cytokinetics; Eric Williamson, MD is an unpaid consultant for Siemens Medical and is the recipient of an investigator-initiated research grant from GE Healthcare.

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0894-7317/$36.00

https://doi.org/10.1016/j.echo.2020.02.009

Abbreviations
CA = Competitive athlete(s)
CMR = Cardiac magnetic resonance imaging
CTA = Computed tomography angiography
CVD = Cardiovascular disease
EICR = Exercise-induced cardiac remodeling
LA = Left atrium/left atrial
LV = Left ventricle/left ventricular
RA = Right atrium/right atrial
RV = Right ventricle/right ventricular
SCD = Sudden cardiac death
TTE = Transthoracic echocardiography/echocardiogram

GENERAL CONSIDERATIONS

Competitive athletes (CA) are a rapidly growing population worldwide. Habitual vigorous exercise, a defining characteristic of this population, is a potent stimulus for adaptive structural and functional cardiac remodeling and is an effective way to reduce the risk of cardiovascular disease (CVD). Manifestations of CVD in CA are highly variable ranging from subtle findings during pre-participation cardiovascular screening (PPCS) to collapse or cardiac arrest during exercise. In general, young CA are most commonly affected by congenital and genetic conditions while older CA most commonly harbor acquired CVD. Cardiac imaging using transthoracic echocardiography (TTE), cardiac computed tomography angiography (CTA), and cardiac magnetic resonance imaging (CMR) plays a fundamental role in the care of CA. This document was created to provide clinical imaging specialists with a comprehensive guide for the performance of multimodality imaging in CA.

I. INTRODUCTION

The interpretation of imaging data obtained in the care of competitive athletes (CA) requires a comprehensive understanding of exercise-induced cardiac remodeling (EICR) and an ability to distinguish the characteristics of this process from findings suggestive of pathology. Accordingly, cardiovascular specialists are best positioned to provide effective care for CA if they possess the ability to integrate and interpret multimodality diagnostic imaging as required on an individual case-by-case basis. This document was designed to provide a framework for the use of multimodality imaging in the assessment of CA. Throughout this document, emphasis will be placed on the use of appropriately selected multimodality imaging as required to diagnose, exclude, and manage clinically relevant CVD in CA.

The definition of a CA, as endorsed by the American College of Cardiology and American Heart Association, has remained unchanged for more than thirty years. As described at the initial 36th Bethesda Conference Proceedings in 1985,[1] and each subsequent update,[2,3] a competitive athlete is defined as an individual "who participates in an organized team or individual sport that requires regular competition against others as a central component, places a high premium on excellence and achievement, and requires some form of systematic (and usually intense) training". Accordingly, CA adhere to a distinctive lifestyle characterized by routine individualized exercise training geared toward preparation for competition.[4] This document will focus on the assessment of young CA as defined by the age range beginning with the cessation of puberty and ending at age 35. However, key issues relevant to imaging in pediatric and aging or "masters level" CA will also be addressed.

A. The CA in Cardiovascular Practice

CA represent one of the healthiest segments of the general population and are often mistakenly viewed as being "immune" from CVD. However, vigorous physical activity, particularly in CA with underlying cardiovascular disease (CVD), transiently increases the risk of adverse events, including sudden cardiac death (SCD).[5,6] The true incidence of SCD in CA 35 years or younger is unknown, with estimates ranging from 0.11:100,000 athlete-years in high school students in Minnesota,[7] to 1.9:100,000 athlete-years in National Collegiate Athletic Association athletes.[8] Available data suggest that SCD among CA occurs more commonly in men than women, more commonly in CA of African-American descent compared to Caucasians, and in CA participating in all sports with basketball and American-style football representing relatively high-risk disciplines.[9-11] There are numerous cardiovascular causes of SCD in CA, including genetic and acquired diseases of the heart muscle, valves, electrical system, and coronary arteries (Table 1).[12] Historically, hypertrophic cardiomyopathy (HCM) was considered the most common cause of SCD in CA based on pioneering data from The United States (US) National Registry of Sudden Death in Athletes, in which one-third of deaths in CA were attributable to this cause.[9] More recently, several independent publications suggested that hypertrophic cardiomyopathy accounts for a lower proportion of SCD than previously reported, with the majority of SCD occurring in CA with structurally normal hearts on autopsy.[11,13] Additional important causes of SCD in young CA include other forms of genetic heart muscle disease (e.g., arrhythmogenic, dilated, and noncompaction cardiomyopathy), acquired heart muscle disease (e.g., myocarditis, toxic cardiomyopathy attributable to illicit performance enhancing drugs), genetic channelopathies (e.g., long QT

Table 1 Common causes of sudden cardiac death and exertional symptoms in competitive athletes

Disorders of the myocardium/cardiac structure
Hypertrophic cardiomyopathy
Arrhythmogenic right ventricular cardiomyopathy
Familial/idiopathic dilated cardiomyopathy
Left ventricular noncompaction cardiomyopathy
Toxic cardiomyopathy (alcohol, illicit anabolic steroids, etc.)
Acute and subacute myocarditis
Simple and complex congenital heart disease
Disorders of the cardiac electrical system
Ventricular pre-excitation/wolff-parkinson white syndrome
Congenital long QT syndrome
Catecholaminergic polymorphic ventricular tachycardia
Idiopathic ventricular tachycardia
Commotio cordis
Disorders of coronary circulation
Congenital anomalies of coronary arterial origin and course
Acquired atherosclerotic disease
Disorders of the heart valves
Bicuspid aortic valve (with ≥ moderate stenosis ± aortopathy)
Pulmonic stenosis (with ≥ moderate stenosis)
Mitral valve prolapse (with corollary arrhythmogenicity)
Disorders of the aorta
Bicuspid aortic valve aortopathy
Idiopathic aortopathy/thoracic aortic aneurysm
Marfan syndrome
Loeys-Dietz syndrome
Turner syndrome
Ehlers-Danlos vascular type (IV)

syndrome, catecholaminergic polymorphic ventricular tachycardia), and congenital or genetic diseases of the heart valves, coronary anatomy, and aorta.

While SCD may be the initial presentation of CVD in CA, the majority of CA present for clinical evaluation after an abnormality is detected during pre-participation cardiovascular screening (PPCS) or in the context of symptoms that occur during training or competition. Effective assessment of the CA with suspected CVD begins with a comprehensive medical history, physical examination, and in most cases a resting 12-lead electrocardiogram (ECG). This process, as tailored to meet the individual clinical needs of the CA and as delineated elsewhere in detail,[4] usually requires a team comprising medical professionals with complementary expertise in sports medicine, CV sub-specialties, and non-CV internal medicine specialists (Figure 1). The appropriate use of multimodality imaging plays a critical role in the diagnosis, risk stratification, and exclusion of CVD in CA, most of whom will require noninvasive imaging during their evaluation. As delineated throughout the remainder of this document, effective clinical imaging of CA is contingent on appropriate test selection, high quality test performance, and accurate test interpretation. Appropriate test selection requires a familiarity with the strengths and weaknesses of each available imaging modality for a given indication or suspected abnormality, while test interpretation is contingent upon a thorough understanding of EICR as the basis for differentiating adaptation from pathology.

II. INTEGRATED MULTIMODALITY IMAGING

The primary goal of imaging in the clinical assessment of the CA is to diagnose or exclude cardiovascular conditions that are associated with adverse outcomes and/or symptoms that impede athletic performance or quality of life.[14] Imaging also plays an important role in risk stratification and surveillance following disease diagnosis. Transthoracic echocardiography (TTE), cardiac computed tomography angiography (CTA), and cardiac magnetic resonance imaging (CMR) each play important and complementary roles in the assessment and management of CA.[15] Imaging protocols for use in CA, with several important exceptions addressed in detail below, should not vary substantially from those recommended in other populations.[16-18] In the vast majority of clinical situations, TTE should be considered first-line imaging followed by either CMR or CTA as dictated by the specific clinical question, institutional preferences, and provider expertise. While CA are not specifically addressed in the Appropriate Use Criteria (AUC) for echocardiography[19] and multimodality imaging,[20] application of these criteria to CA is generally appropriate with deviation considered on a case-by-case basis. The strengths, weaknesses, and key clinical applications in CA are subsequently reviewed here (Table 2).

A. Transthoracic Echocardiography (TTE)

Two-dimensional and Doppler TTE play integral roles in the evaluation of CA with suspected or confirmed CVD. TTE has the capacity to characterize myocardial structure and systolic and diastolic function, valve morphology and function, and proximal coronary anatomy with sufficient accuracy and detail to confirm or exclude the presence of clinically relevant CVD in the majority of CA. The vast majority of studies characterizing cardiovascular adaptations in athletes have utilized TTE and thus the majority of normative cardiac data defining the scope of EICR in CA have been derived by TTE. Core strengths of TTE include its unparalleled accessibility, portability, low cost, and freedom from ionizing radiation. TTE should be considered the first-line imaging modality in CA with suspected or confirmed CVD. Abbreviated TTE protocols for the assessment of CA, including the time required to perform TTE in "out of office" athletic facilities, have been developed.[21] Nonetheless, several potential limitations of TTE are relevant during the imaging assessment of CA. First, accurate and definitive determination of proximal coronary artery anatomy, while possible more than 90% of the time in CA,[22] cannot always be accomplished with TTE. Second, some portions of the ascending aorta may be inaccessible by TTE.[23] Third, acoustic shadowing caused by the left thoracic ribs may prevent full-thickness circumferential imaging of the left ventricle in short-axis views, thereby obviating complete determination of ventricular morphology. Fourth, difficulty differentiating trabecular tissue on the right ventricular aspect of the interventricular septum and obtaining true cross-sectional images of the left ventricular (LV) apex may result in the inaccurate measurement of these key structures. Finally, TTE is not capable of reliably identifying or quantifying myocardial fibrosis, edema, and inflammation, features that play important diagnostic and prognostic roles in several key diseases of the myocardium.[24,25]

B. Cardiac Magnetic Resonance Imaging (CMR)

CMR is the contemporary gold standard for defining myocardial structure and myocardial tissue architecture and is increasingly applied both for the study and clinical management of CA. CMR allows detailed assessment of myocardial function, valve morphology

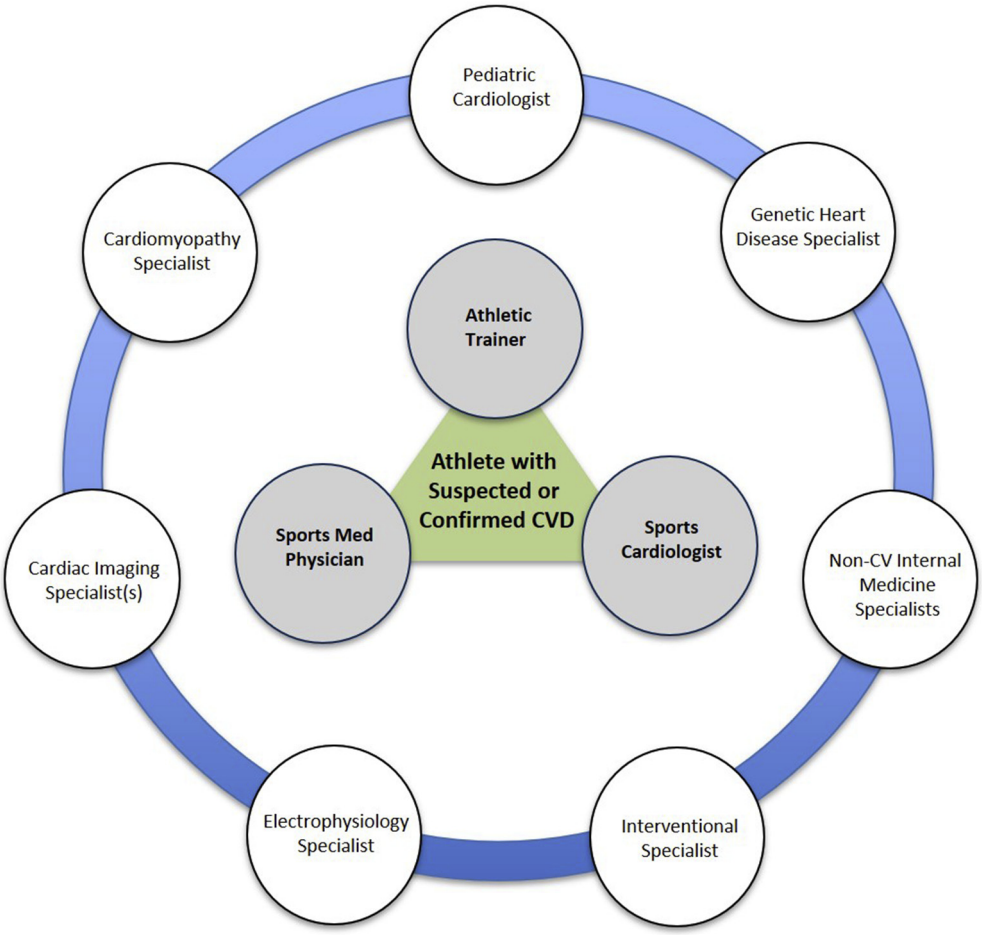

Figure 1 Overview of the clinical team approach to the assessment and management of competitive athletes with cardiovascular disease. Emphasis is placed on the role of cardiovascular specialists, including multimodality imaging experts, and non-cardiovascular internal medicine sub-specialists, in the care of competitive athletes with suspected or confirmed cardiovascular disease.

and function, coronary artery origin and proximal course, and the anatomy of the great vessels. In the assessment of CA, CMR plays a valuable role when there is clinical suspicion or definitive determination of myocardial pathology based on the initial evaluation, including data derived from TTE. CMR is the imaging modality of choice for detailed and accurate characterization of right ventricular structure and function. The CMR examination should delineate the presence, severity, and symmetry of ventricular hypertrophy and/or dilation and assess ventricular tissue architecture using qualitative and quantitative assessments of myocardial fibrosis, both focal and diffuse, edema, and fat. CMR is also capable of delineating proximal coronary artery anatomy and great vessel structure and should be considered as an option for those purposes. Limitations of CMR include high cost, incompatibility with unsafe implanted metallic devices (although not typically an issue in CA), limited accessibility, the need for a priori determination of renal function to establish the safety of contrast administration, and claustrophobia.

C. Cardiac Computed Tomography Angiography (CTA)

Literature defining the role of CTA in the care of CA is limited. However, by virtue of several inherent strengths, CTA plays an important role in several specific clinical situations. CTA utilizes ionizing radiation over relatively short image acquisition times to provide 3-dimensional tomographic images characterized by superb spatial resolution. Recent advances in scanner technology and gating sequences now enable CTA to provide an assessment of ventricular function although these acquisitions require relatively high radiation doses. However, radiation exposure, a potential limitation of this imaging modality, must still be considered in the application of CTA among CA. CTA should be considered when precise definition of proximal coronary anatomy or characterization of great vessel morphology is indicated. All CA with indeterminate, suspected, or confirmed anomalous coronary artery anatomy following TTE should undergo either CTA or CMR, as dictated by institutional preferences and local provider expertise, to identify or exclude the high-risk features of disease that dictate subsequent management. CTA for coronary artery calcium scoring and noninvasive coronary angiography have emerged as valuable tools for the diagnosis and management of suspected atherosclerotic disease. In addition, CA with suspected or confirmed dilation of the aortic root or ascending aorta should undergo at least one comprehensive tomographic assessment (CTA or CMR as dictated by institutional preferences and local provider expertise) of the aorta as delineated elsewhere.

III. EXERCISE-INDUCED CARDIAC REMODELING (EICR)

EICR is the process by which the heart and vasculature change with respect to structure and function in response to repetitive exercise exposure.[26] Evidence substantiating the presence of EICR dates back to the late 1800's with seminal reports of cardiac enlargement in cross country skiers,[27] and rowers.[28] A large number of cross-sectional noninvasive imaging studies, spanning the last 5 decades, chronicle features of the "athlete's heart", a term that persists in the literature without a consensus definition. The last decade has seen a number of important longitudinal prospective studies that establish causal relationships between exercise training and cardiac remodeling. The clinical imager responsible for test interpretation of CA must possess a fundamental knowledge of the scope of EICR in order to differentiate normal adaptation from occult pathology.

A. Basic Exercise Physiology

EICR is stimulated by the hemodynamic and neurohumoral conditions that exist during exercise and a basic knowledge of applied exercise physiology is necessary to understand this process. There is a direct relationship between exercise intensity and the body's demand for oxygen. Increasing oxygen demand is met by increasing oxygen uptake ($\dot{V}O_2$). Maximal oxygen consumption (Peak $\dot{V}O_2$) is defined as the amount of oxygen uptake that occurs at an individual's maximal volitional intensity or effort of exercise. The cardiovascular system is responsible for transporting oxygen-rich blood from the lungs to the skeletal muscles, a process quantified as cardiac output. In the healthy person, there is a direct relationship between $\dot{V}O_2$ and cardiac output. Cardiac output, the product of stroke volume and heart rate, may increase 5- to 6-fold during maximal exercise effort.[29] Increases in heart rate, and – to a lesser degree – stroke volume, are responsible for the majority of cardiac output augmentation during an acute bout of exercise in both CA and sedentary people. However, differences in exercise capacity between CA and untrained people are not explained by heart rate as peak heart rate is determined by age, sex, and genetics rather than exercise habits.[30,31] In contrast, cardiac chamber enlargement and the accompanying ability to generate a large stroke volume are direct results of exercise training and are the cardiovascular hallmarks of the endurance-trained athlete.

Hemodynamic conditions, specifically changes in cardiac output and peripheral vascular resistance, vary significantly across sporting disciplines (Figure 2).[32] Although there is considerable overlap, exercise physiology relevant to CA can be subdivided into 2 distinct categories. Isotonic (endurance) exercise involves sustained elevations in cardiac output, with normal or reduced peripheral vascular resistance. This form of exercise underlies activities such as long-distance running, rowing, cycling, and swimming. Such activity represents a *volume challenge* for the heart, which stimulates dilation of all 4 cardiac chambers. In contrast, isometric (strength) exercise is characterized by marked and typically pulsatile increases in peripheral vascular resistance with normal or only slightly elevated cardiac output. Strength training is the dominant form of exercise physiology during activities such as weightlifting, track and field throwing events, and American-style football. The surges in systemic blood pressure that accompany isometric exercise represent a *pressure challenge* for the left ventricle with minimal hemodynamic impact on other cardiac chambers. It should be noted that physiologic dichotomization of sporting disciplines into isotonic and isometric activities is overly simplistic as it relates to defining the cardiac stressors of exercise. Contemporary descriptions of exercise physiology acknowledge the fact that all athletic disciplines involve some element of each form of stress, with popular team-based activities (e.g., soccer, lacrosse, basketball, and hockey) and some endurance sporting disciplines (e.g., rowing and cycling) involving significant elements of both isotonic and isometric physiology.[32]

Table 2 Comparative assessment of multimodality imaging in the care of competitive athletes

Test attribute	TTE	CT	CMR
Cost	+++	++	+
Accessibility	+++	++	+
Portability	+++	+	+
Normative data in CA	+++	+	++
Ability to characterize LV morphology	++	+++	+++
Ability to characterize RV morphology	+	++	+++
Ability to characterize ventricular tissue composition	+	+	+++
Ability to define proximal coronary anatomy	+	+++	++
Ability to characterize LV systolic function	+++	++	++
Ability to characterize LV diastolic function	+++	+	+
Ability to characterize aortic morphology	++	+++	+++
Ability to characterize valve function and morphology	+++	++	++

+++, Excellent; ++, good; +, fair.

B. Determinants of EICR

The variable hemodynamic attributes described above are an important determinant of the magnitude and geometry of EICR, and not all athletes' hearts remodel in identical fashion (Figure 3). A landmark cross-sectional echocardiographic study published in 1975 showed that CA participating in sports with predominantly isotonic physiology (swimmers and runners) were found to have larger LV chamber diameters than athletes practicing wrestling, a sport with largely isometric physiology.[33] Similar findings derived from longitudinal studies designed to establish a causal relationship between exercise training and cardiac morphology have subsequently been reported.[34-36] In general, the biventricular and biatrial volume challenge inherent in isotonic or endurance-based sporting activity stimulates dilation of all 4 cardiac chambers. In contrast, isometric or strength-based sporting activity may lead to mild thickening of the LV walls without chamber dilation. Limited available data suggest that the left atrium and right heart are largely unaffected by isometric training.[34] The degree of EICR that accompanies pure isometric training, unless coupled with resting hypertension,[37] or illicit use of anabolic steroids,[38] is typically less than the remodeling that occurs as a result of exercise involving some element of isotonic training. To what degree cardiac adaptations differ across the specific endurance disciplines and individual training regimens with respect to the amount of superimposed isometric stress remains largely unexplored and represents an area of important ongoing work.[39]

Factors including sex, ethnicity, duration of prior exercise exposure, and genetics are additional determinants of EICR. Female athletes typically exhibit quantitatively less physiologic remodeling

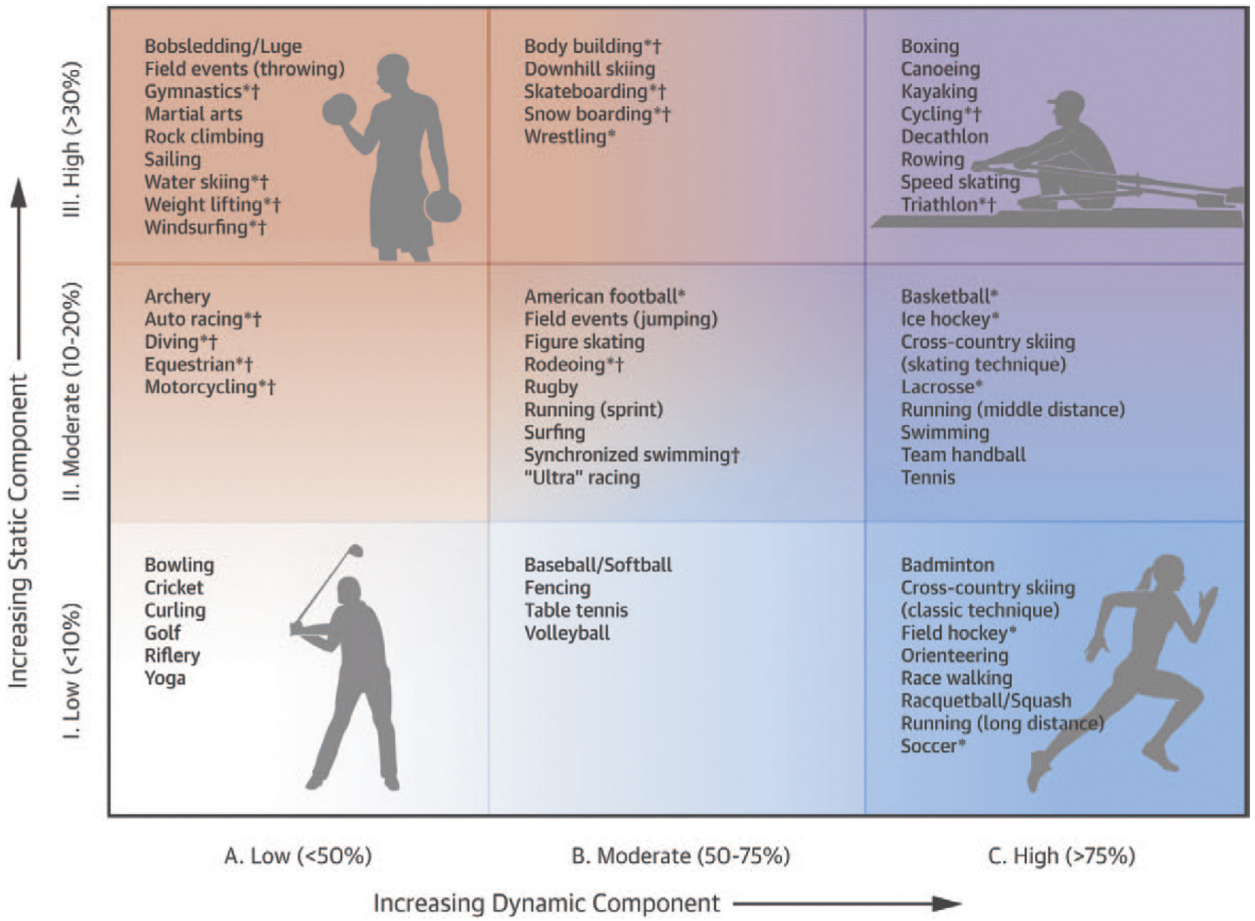

Figure 2 Physiologic classification of common sporting disciplines based on relative contributions of isotonic and isometric physiology. Isotonic sport physiology imparts a volume load on the heart leading to biventricular and biatrial dilation, while isometric sport physiology imparts a pressure load on the left ventricle and central arterial system. The relative contributions of these cardinal hemodynamic stresses can be used to predict adaptive EICR among young competitive athletes. *Indicates sports that impart increased risk of bodily collision. †Indicates sports that may impart increased risk of adverse events if syncope occurs.

than their male counterparts. In a cross-sectional study of 600 elite female Italian athletes compared with data from previously studied male athletes ($n = 738$), female athletes showed significantly smaller LV cavity dimension (11% less; $P < .001$) and wall thickness (23% less; $P < .001$) and were far less likely to have absolute measurements that exceeded normal cut-points.[40] Other studies have produced similar findings.[41,42] Although there are conflicting data in the literature, not all of the difference in absolute cardiac dimensions between men and women is eliminated when cardiac dimensions are corrected for the typically smaller female body size.[43-45] Mechanistic explanations for a sex-specific basis for EICR, including the role of sex hormone profiles, remain speculative. Ethnicity is also a determinant of EICR with CA of Afro-Caribbean descent (i.e. black athletes) tending to have thicker LV walls than Caucasians.[46,47] At present however, data defining the role of ethnicity as a determinant of EICR remain sparse.

The influence of exercise duration has recently been shown to play a role in the process of cardiac adaptation. A study of 12 sedentary middle-aged subjects advancing through an incremental training protocol to prepare for a competitive marathon run showed a biphasic increase in LV mass with initial concentric hypertrophy, increased LV wall thickening during 0-6 months, and subsequent eccentric hypertrophy attributable to LV dilation (6-12 months).[48] A separate study of college-aged elite male rowers over a 39-month period of

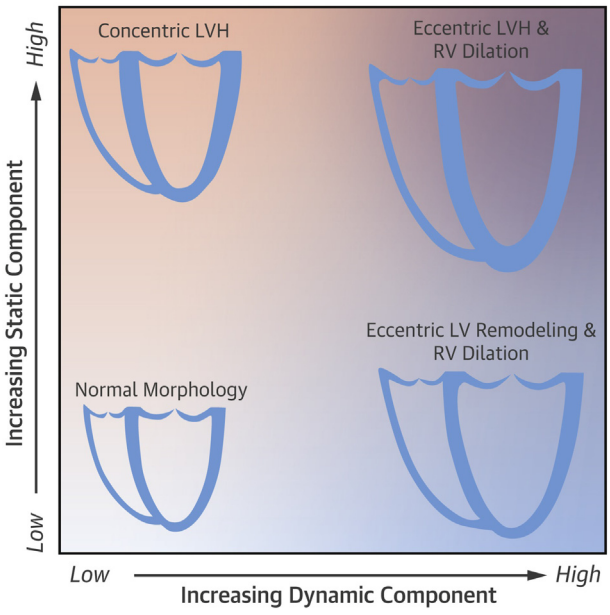

Figure 3 Anticipated exercise-induced cardiac remodeling based on relative component contributions of isotonic and isometric stress.

Table 3 Selected studies providing normative data for left ventricular end-diastolic internal dimension in competitive athletes

Author[ref.]	Imaging tech.	Athlete type	Age (y)	W/M, (*n*)	Women, mean ± SD (max.)	Men, mean ± SD (max.)
Spirito[53]	TTE	C (Italian, national)	22 ± N/A	209/738	–	53 ± 5 (66)*
Finocchiaro[54]	TTE	C (British, regional/ national)	22 ± 6	600/644	49 ± 4 (–)	54 ± 5 (–)
Pelliccia[40]	TTE	C (Italian, national)	21 ± 5	600/738	49 ± 4 (11)	54 ± 4 (66)
Weiner[21]	TTE	C (American, university)	19 ± 3	197/300	47 ± 7 (55)	54 ± 6 (62)
Pelliccia[55]	TTE	C (Italian, national)	24 ± N/A	352/957	48 (66)	55 (70)
Pluim[56]	TTE	S (meta-analysis)	18-40	–/544	–	52 ± 1 (–)
Pluim[56]	TTE	E (meta-analysis)	18-40	–/413	–	54 ± 1 (–)
Engel[57]	TTE	C (American, pro-basketball)	26 ± 4	–/526	–	57 ± 0.2 (71)
Krysztofiak[58]	TTE	C (Polish, pediatric)	12 ± 5	327/464	–	44 ± 1 (60)*
Makan[59]	TTE	C (British, pediatric)	16 ± 1	236/664	–	51 ± 4 (60)*
Weiner[37]	TTE	C (American, university, football)	19 ± 1	–/113	–	53 ± 4 (60)
Prakken[60]	CMR†	E (Dutch, national)	26 ± 6	33/46	55 ± 4 (63)	60 ± 4 (68)
Luijkx[61]	CMR‡	E (Dutch, national)	27 ± 5	24/57	55 ± 3.9 (63)	60 ± 4 (67)

C, Combined endurance and strength training physiology; *CMR*, Cardiac magnetic resonance imaging; *E*, endurance training physiology; *M*, Men; *Max.*, Maximal values recorded; *S*, strength training physiology; *TTE*, transthoracic echocardiography; *W*, women.
Data reflect measurements of the left ventricular end-diastolic major dimension (mm) obtained from a parasternal long-axis transthoracic echocardiographic view unless otherwise specified.
*Denotes cardiac data inclusive of both men and women.
†Data reflect maximum end-diastolic dimension as measured from a 4-chamber view.
‡Data reflect maximal end-diastolic dimension as measured from a short-axis view.

high-intensity and high-volume team-based training showed a phasic remodeling response with distinct acute adaptations, including increases in LV chamber size, early diastolic function, and systolic twist followed by a chronic phase of adaptation characterized by increasing wall thickness and regression in LV twist.[49] Finally, genetics appear to be a determinant of EICR. Studies examining polymorphisms within genes coding for proteins of the renin-angiotensin-aldosterone axis found that the angiotensin converting enzyme-deletion/deletion (DD) polymorphism was associated with more LV hypertrophy than the insertion/insertion (II) polymorphism during 10 weeks of exercise training in military recruits.[50] Specific polymorphisms of the angiotensinogen gene have also been associated with LV remodeling.[51] In addition, familial hypertension, a complex polygenic trait, was shown to be associated with both the magnitude and geometry of exercise-induced concentric LV remodeling in youthful normotensive endurance-trained athletes.[52] The integration of prior exercise exposure and genetics into the interpretation of clinical imaging will require further studies clarifying their impact on myocardial structure and function.

C. Left Ventricular Adaptations

Dilation of the LV is common and should be considered as a normal finding in endurance CA (Table 3). LV end-diastolic dimensions, as measured by TTE, in a large group ($n = 1,309$) of Italian elite CA representing 38 different sports varied from 38 to 66 mm in women (mean = 48 mm) and from 43 to 70 mm in men (mean = 55 mm).[55] LV end-diastolic diameter was ≥55 mm in 45% and ≥60 mm in 14% of this cohort. Thus, use of the recommended upper limits of normality of 55-58 mm[62] would render approximately 40% of male CA in this study as abnormal. In a US-based study of approximately 500 university CA, approximately 25% exceeded sex-specific recommended limits for LV end-diastolic diameter.[21] However, it is noteworthy that the majority of CA in these two studies had LV chamber dimensions within normal limits, indicating that not all CA demonstrate LV dilation. Thus, the use of "cut-off" values for LV end-diastolic diameter or volume, to either establish or exclude the presence of pathologic cardiomyopathy, is not recommended.

Mild thickening of LV walls, either with or without concomitant LV chamber dilation, may develop in CA (Table 4). Balanced LV wall thickening and chamber dilation (i.e., eccentric LV hypertrophy as defined by increased LV mass and a relative wall thickness <0.42) is common among CAs who engage in endurance sports with concomitantly highly levels of isotonic and isometric loads such as rowing and cycling (Figure 4). In contrast, mild isolated LV wall thickening (i.e., concentric LVH as defined by increased LV mass and a relative wall thickness ≥0.42) may be seen in athletes who participate in strength-based activities with no significant isotonic component, including weight lifting and American-style football (Figure 5).[33,34] Functional implications of concentric LVH stimulated by EICR, the same variant of hypertrophy that accompanies pathologic forms of ventricular pressure overload such as hypertension and aortic stenosis, remain incompletely understood. Longitudinal studies of male American-style football players showed that the development of concentric LVH was associated with relative impairments both of early diastolic relaxation velocity,[34] and systolic function,[67] raising uncertainty about the adaptive nature of this form of EICR. LV wall thickening attributable to EICR, regardless of whether it occurs in an eccentric or concentric morphology, rarely leads to measurements that exceed 12-13 mm in Caucasian CA. In 947 elite Italian CA, only a small number

Table 4 Selected studies providing normative data for left ventricular wall thickness in competitive athletes

Author[ref.]	Imaging tech.	Athlete type	Age (y)	W/M, (*n*)	Women, mean ± SD (max.)	Men, mean ± SD (max.)
Spirito[53]	TTE	C (Italian, national)	22	209/738	–	10 ± 1 (16)*
Finocchiaro[54]	TTE	C (British, regional/national)	22 ± 6	600/644	8.4 ± 1.2 (–)	9.6 ± 1.2 (–)
Pelliccia[40]	TTE	C (Italian, national)	21 ± 5	600/738	7.8 ± 0.9 (11)	9.4 ± 0.9 (13)
Weiner[21]	TTE	C (American, university)	19 ± 3	197/300	9.2 ± 1.4 (12)	10.5 ± 1.5 (14)
D'Andrea[35]	TTE	S (Italian, "highly trained")	29 ± 10	120/160	–	11.3 ± 2.4 (–)*
D'Andrea[35]	TTE	E (Italian, "highly trained")	28 ± 10	160/210	–	9.7 ± 3.1 (–)*
Pluim[56]	TTE	S (meta-analysis)	18-40	–/413	–	10.3 ± 0.3 (–)
Pluim[56]	TTE	E (meta-analysis)	18-40	–/526	–	11.0 ± 0.8 (–)
Engel[57]	TTE	C (American, pro-basketball)	26 ± 4	–/791	–	11.0 ± 0.1 (15)
Krysztofiak[58]	TTE	C (Polish, pediatric)	12 ± 5	327/464	–	8.0 ± 0.2 (12)*
Makan[59]	TTE	C (British, pediatric)	16 ± 1	236/664	–	9.6 ± 1.3 (14)*
Lee[63]	CMR	C (British, military recruits)	20 ± 2	–/309	–	10.7 ± 1.4 (14.1)
Baggish[64]	TTE	E (American, olympic, rowing)	25 ± 3	–/20	–	12.7 ± 1.5 (15)
Weiner[37]	TTE	C (American, university, football)	19 ± 1	–/113	–	10.6 ± 1.0 (13.9)
Prakken[60]	CMR†	E (Dutch, national)	26 ± 6	33/46	11 ± 1.4 (14)	9.0 ± 1.3 (12)
Luijkx[61]	CMR‡	E (Dutch, national)	27 ± 5	24/57	11 ± 1.5 (14)	9.3 ± 1.3 (12)
Scharf[65]	CMR§	E (German, national, triathlon)	28 ± 4	–/26	–	9.8 ± 1.0 (11.6)
Scharf[66]	CMR§	C (German, professional, soccer)	27 ± 4	–/29	–	9.4 ± 0.9 (11.6)

C, Combined endurance and strength training physiology; *CMR*, cardiac magnetic resonance imaging; *E*, endurance training physiology; *M*, Men; *Max.*, Maximal values recorded; *S*, strength training physiology; *TTE*, transthoracic echocardiography; *W*, women.
Data reflect measurements of the posterolateral left ventricular wall (mm) as obtained from a parasternal long-axis transthoracic echocardiographic view unless otherwise specified.
*Denotes cardiac data inclusive of both men and women.
†Data reflect maximum end-diastolic septal thickness as measured from a 4-chamber view.
‡Data reflect maximal end-diastolic septal thickness as measured from a short-axis view.
§Data reported reflect average of 6 segment thickness measurements (anterior, anterolateral, anteroseptal, inferior, inferolateral, inferoseptal) as measured from a short-axis view.

(1.7%) had LV wall thicknesses ≥13 mm.[68] Similarly, a low prevalence (0.4%) of LV wall thickness >12 mm was observed in 720 elite British junior CA,[69] and in nearly 500 collegiate American CA, not a single participant had LV wall thickness >14 mm.[21] While uncommon, EICR may lead to LV wall thicknesses of 13-15 mm in CA participating in high isotonic/high isometric sporting disciplines or in CA with large body size or Afro-Caribbean descent.[70-72] In contrast, an LV wall thickness >15 mm should raise suspicion for pathology and should stimulate further testing to exclude or confirm an explanatory cardiomyopathy.

EICR leads to preservation or enhancement of LV diastolic function with normal noninvasive estimates of left atrial pressure.[72-74] Endurance-trained CA with eccentric LV hypertrophy typically demonstrate supranormal indices of diastolic function under resting conditions (Figure 6). In contrast, strength trained athletes with concentric LV hypertrophy may demonstrate mildly impaired TTE indices of diastolic function (Figure 5). Abnormal transmitral filling profiles and reductions in early diastolic tissue velocities should raise suspicion for pathology in young CA and also older CA who do not show typical age-related diastolic changes.[75] LV ejection fraction in CA is generally in the normal range despite variable amounts of LV chamber dilation and wall thickening.[76,77] However, trained endurance CA with substantial LV dilation, when imaged under resting conditions, may demonstrate an LV ejection fraction at or slightly below the lower limits of normal.[78] This reflects the fact that stroke volume, not ejection fraction, is physiologically regulated with large ventricles ejecting a lower fraction of end-diastolic volume than smaller ventricles under resting conditions. However, studies using pulsed-wave Doppler, tissue Doppler, and speckle-tracking echocardiography have shown that endurance CA typically demonstrate preserved or enhanced systolic function.[64,72,79-81]

D. Right Ventricular Adaptations

EICR is not confined to the LV. Endurance exercise requires both the LV and right ventricle (RV) to accept and eject relatively large quantities of blood. For the comparatively thin-walled RV, remodeling typically takes the form of mild to moderate RV dilation without significant hypertrophy. In an echocardiographic study of 102 endurance CA, RV chamber dimensions were larger than "normal" values in over one-half of the athletes and 28% had an RV outflow tract dimension that met the proposed major size criteria for the diagnosis of arrhythmogenic RV cardiomyopathy (ARVC).[82] In a similar study of Italian CA, 15.6% exceeded a basal RV dimension of 40 mm.[36] CMR has similarly demonstrated that RV enlargement is common among endurance athletes.[83] Differentiating exercise-induced RV changes from the diagnosis of ARVC is one of the most important clinical challenges faced by the clinical imager. RV dilation in the endurance-trained CA should be associated with concomitant LV remodeling (dilation), and the finding of isolated RV enlargement should raise suspicion of a pathologic process. Furthermore,

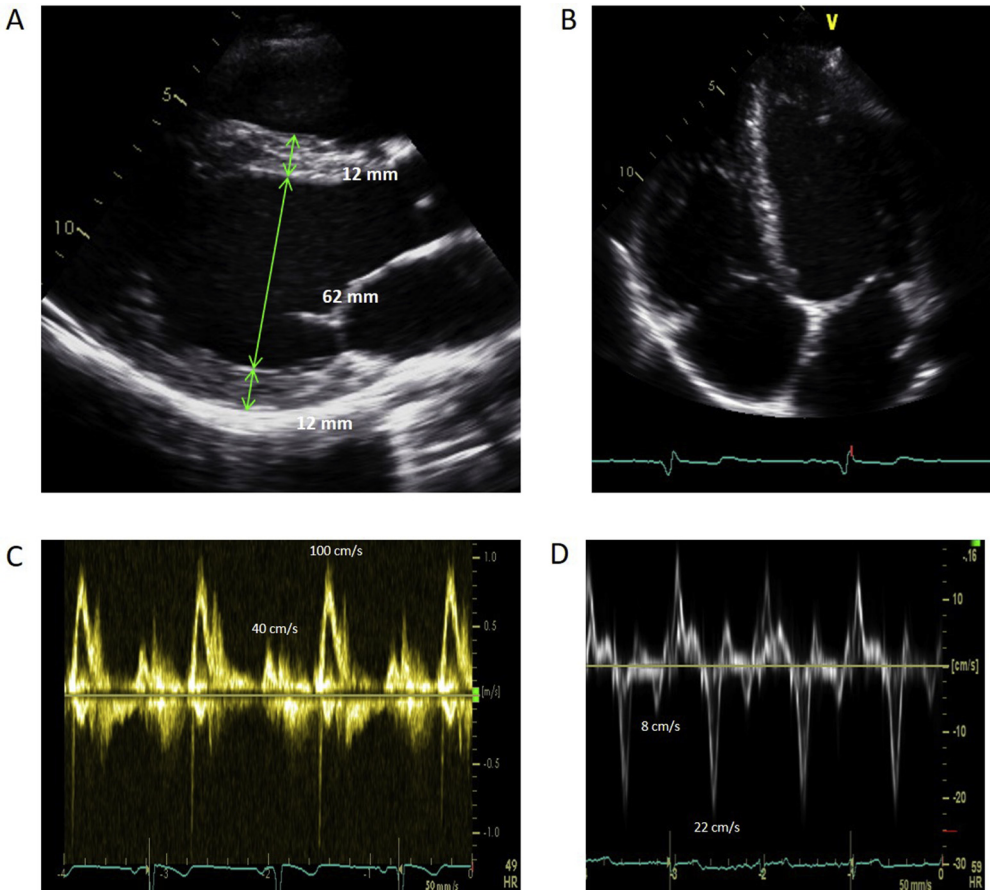

Figure 4 Representative transthoracic echocardiographic imaging from a healthy competitive endurance-sport athlete. **(A)** Parasternal long-axis view demonstrating eccentric left ventricular hypertrophy as manifested by simultaneous left ventricular wall thickening and chamber dilation. **(B)** Apical 4-chamber view demonstrating comparable left and right ventricular end-diastolic areas. **(C)** Transmitral pulsed-wave Doppler showing E/A ratio >2.0. **(D)** Tissue Doppler of the lateral mitral annulus showing e' prominence with early diastolic relaxation velocities in excess of 20 cm/s.

physiologic RV dilation in endurance CA is a global process that is not associated with focal defects such as aneurysmal dilation or segmental dysfunction. Strict "cut-off" values for RV size are not helpful in distinguishing EICR from pathologic cardiomyopathy. Knowledge of the diagnostic criteria for arrhythmogenic RV cardiomyopathy, which integrate family history, electrocardiography, and cardiac imaging, is essential.[84] Mild reductions in RV systolic function under resting conditions are often observed in trained endurance CA.[60] The impact of strength training on the RV has not been as well studied but one echocardiographic study of endurance and strength CA found that endurance athletes had larger RV dimensions than strength-trained CA but both groups had similar RV function, as assessed by systolic strain and indices of diastolic function.[85]

E. Atrial Adaptations

Initial echocardiographic studies showed that left atrial (LA) enlargement is common in CA, particularly endurance CA.[86,87] A recent meta-analysis of LA size in more than 7000 athletes and 1000 controls provided quantitative data about the magnitude of LA dilation in CA.[88] Compared with sedentary controls, LA diameter was found to be 4.6 mm greater in endurance CA, 3.5 mm greater in combined strength- and endurance-trained CA, and 2.9 mm greater in purely strength-trained CA. Recent studies have also begun to examine the right atrium (RA) in CA and have documented similar increases in RA size in endurance athletes; a clinically useful normative data set for RA size in CA has been published.[89] Studies of atrial function in athletes, assessed with speckle-tracking echocardiography, are beginning to emerge and have yielded conflicting results. One study showed that LA global peak longitudinal strain and peak atrial contraction strain significantly decreased after training in CA.[90] In contrast, another study did not find differences in atrial strain when comparing athletes and sedentary controls.[91]

F. Aortic Adaptations

The impact of exercise training and competition athletics on the aortic root and ascending aorta remains incompletely understood. However, available data support several clinically relevant concepts. First, CA often demonstrate evidence of mild physiologic remodeling manifesting as slightly larger aortas compared to sedentary counterparts. Meta-analytic data from a large pooled cohort reported larger aortic diameters (by 3.2 mm at the sinuses of Valsalva and by 1.6 mm at the aortic annulus) in young CA compared to sedentary controls. Second, despite the mild degree of dilation reported in this study, aortic measurements in young CA rarely exceed normal

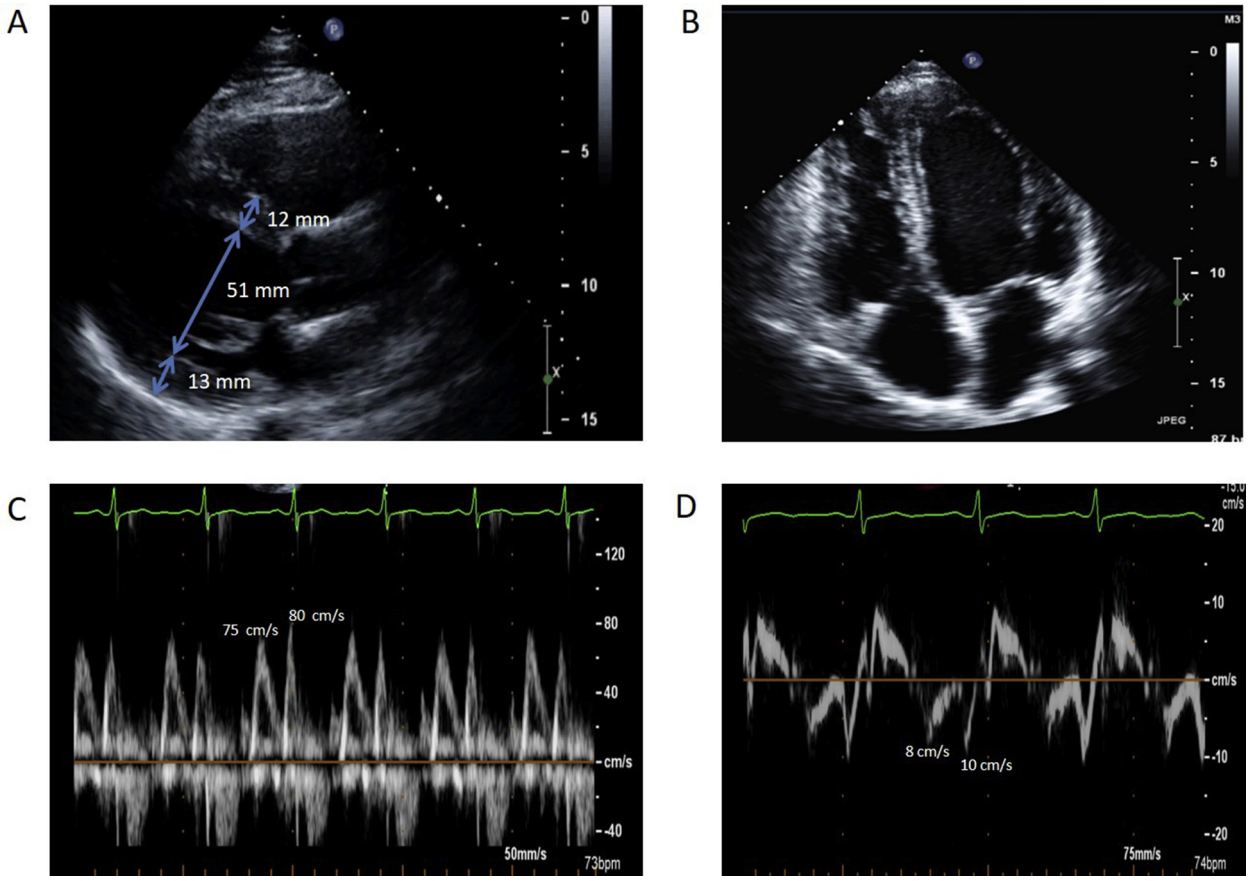

Figure 5 Representative transthoracic echocardiographic imaging from a healthy asymptomatic competitive strength-sport athlete. **(A)** Parasternal long-axis view demonstrating concentric left ventricular hypertrophy as manifested by left ventricular wall thickening in the absence of chamber dilation. **(B)** Apical 4-chamber view demonstrating left greater than right ventricular end-diastolic areas. **(C)** Trans-mitral pulsed-wave Doppler imaging showing E/A ratio ~1.0. **(D)** Tissue Doppler imaging of the lateral mitral annulus showing mild reductions in early diastolic relaxation velocities (e' ~ 8 cm/s) and a' prominence.

ranges for the general population in the absence of underlying aortopathy, with aortic root sizes of greater than 40 mm in men or 34 mm in women having a prevalence of 0.5-1.8%.[92,93] CA with aortic measurements that exceed these values at the level of the valve, aortic sinuses, sinotubular junction, or ascending aorta merit consideration for comprehensive diagnostic testing to evaluate for potential aortopathy. Third, available data are inconclusive as to whether strength-versus endurance-based training have differential effects on ascending aortic size.[93-96] However, a recent study of former professional American-style football players, who exhibit predominantly strength-based physiology, document the highest prevalence of overt aortic dilation reported to date.[97] Fourth, aortic dimensions in CA, as is the case in the non-athletic population, are collinear with body size, particularly height, with one important caveat.[60] Recent data derived from a study of professional basketball players from the National Basketball Association demonstrated a plateauing of the relationship between aortic dimensions and body size at the extremes of height with even the tallest male athletes (some in the range of 7 feet) rarely exceeding an aortic root size of 40 mm.[57] Finally, it is noteworthy that little is known about the impact of lifelong or extended duration competitive athletics on aortic size. Future work geared toward delineating normal aortic dimensions and the possibility of an athletic aortopathy in masters CA is of paramount importance.

The generation of normative aortic data sets and the clinical interpretation of aortic measurements in CA is complicated by variability in measurement technique. Using TTE, the aortic root should be measured from the parasternal long-axis view at ventricular end-diastole using the "leading edge-to-leading edge" technique as delineated by current ASE guidelines.[23] Measurement during different phases of the cardiac cycle or with the use of different anatomic landmarks (e.g., inner edge to inner edge) will result in different values. We suggest that clinical imaging laboratories that evaluate CA indicate their chosen measurement technique in the formal clinical report to facilitate comparison with other modalities and accurate longitudinal follow-up. Gated CTA and CMR are important complements to TTE for the assessment of the proximal aorta as both modalities afford an opportunity to assess multiple aortic segments, including those that cannot be reliably visualized by echocardiography. Standardized technique for the measurement of aortic dimensions using CTA and MR imaging have not been delineated and published guidelines vary in their approach across key factors including suggested imaging planes, anatomic landmarks, and timing during the cardiac cycle. At present, we advocate for careful CTA or MR imaging, processing, and measurement with clear delineation of technique (i.e. inner-edge to inner-edge versus leading-edge to leading-edge) aimed at maximizing short-axis end-diastolic views at multiple aortic levels including the aortic annulus, aortic sinuses (averaging the three sinus-to-sinus measurements and paying careful attention to marked asymmetry), sinotubular junction, mid ascending aorta, high ascending aorta just below the innominate origin, aortic arch, and descending aorta.[23] Factors

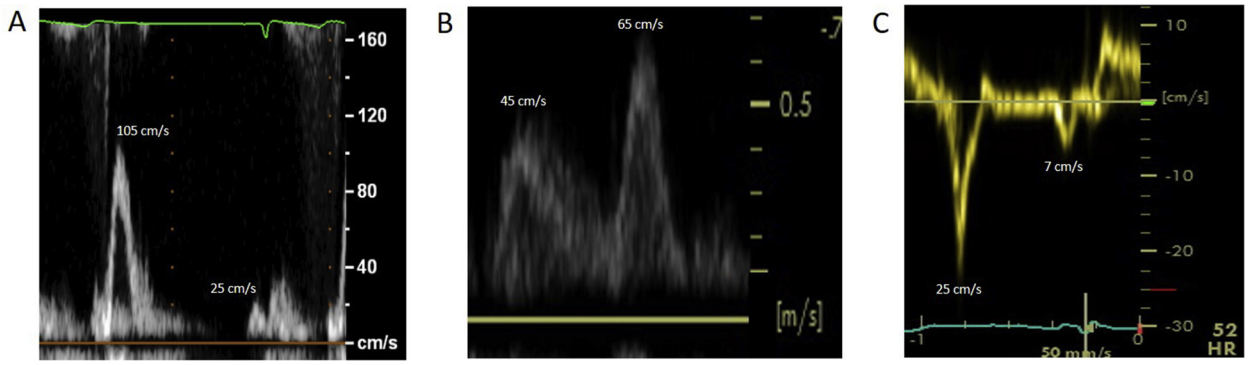

Figure 6 Transthoracic echocardiographic imaging demonstrating complementary indices of supra-normal left ventricular diastolic function in a healthy endurance-sport athlete. **(A)** Trans-mitral pulsed-wave Doppler imaging showing E-wave prominence with an E/A ratio >2.0. **(B)** Pulmonary vein pulsed-wave Doppler showing D-wave predominance due to rapid relaxation and brisk early diastolic left ventricular filling. **(C)** Tissue Doppler of the lateral mitral annulus showing e' prominence with early diastolic relaxation velocities in excess of 20 cm/s.

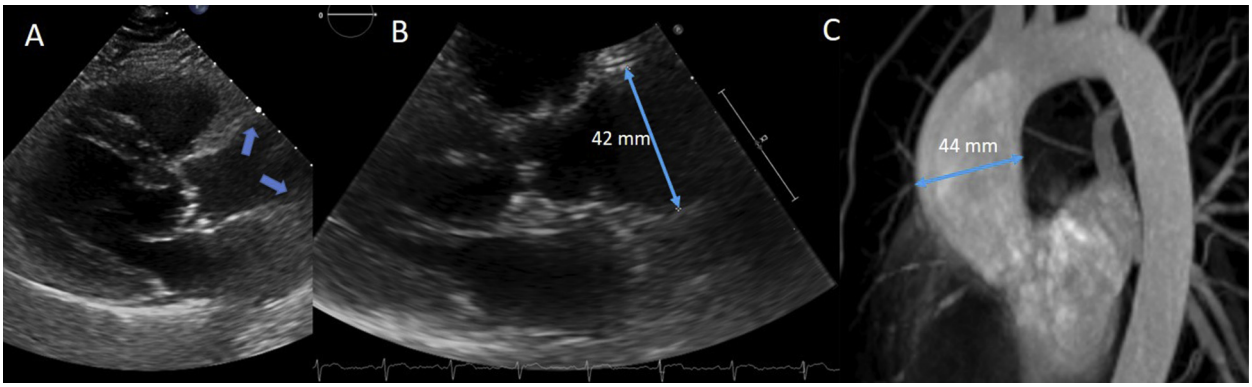

Figure 7 Representative multimodality aortic imaging in a competitive athlete with an ascending aortopathy. **(A)** Transthoracic echocardiographic parasternal long-axis view demonstrating possible proximal ascending aortic dilation (blue arrows). **(B)** Transthoracic echocardiographic parasternal long-axis view obtained one rib space higher demonstrating fusiform dilation of the ascending aorta. **(C)** Cardiac MR angiography imaging of the thoracic aorta demonstrating aneurysmal dilation confined to the ascending aorta.

known to influence aortic size, including age, sex, and height, should be considered in the clinical interpretations of aortic dimensions with the understanding that currently available adult nomograms, and pediatric z-score data, were not designed for use in CA.[98,99] Future work will be needed to develop similar tools in CA. Representative multimodality aortic imaging in a CA is shown in Figure 7.

Key Points

1. Clinical imaging specialists performing and/or interpreting imaging studies in CA should possess a basic knowledge of fundamental exercise physiology and EICR.
2. The magnitude (i.e., absolute wall thickness and chamber dimensions/volumes) and geometry (eccentric vs. concentric) of LV adaptation in CA is defined by the complex interplay between numerous factors, including sport type, sex, ethnicity, and duration of prior exercise exposure.
3. When due to EICR, RV dilation, a common adaptation in CA engaging in endurance sports, should be accompanied by LV eccentric remodeling/hypertrophy and biatrial dilation.
4. Mild aortic sinus or ascending aortic dilation may occur in young CA but absolute aortic measurements of ≥40 mm (men) and ≥34 mm (women) are uncommon. A finding of aortic sinus or ascending aortic dimensions in excess of these sex-specific cut-points should prompt clinical consideration of aortic pathology and subsequent imaging with either gated CTA or CMR.

IV. DIFFERENTIATING EICR FROM PATHOLOGY

EICR commonly leads to imaging findings that overlap with the common forms of heart muscle disease that are associated with adverse events in CA. Accordingly, differentiating EICR from heart muscle pathology is among the most important tasks for the clinical imager.[100] This process requires a comprehensive understanding of both the EICR fundamentals discussed above and the imaging features of the key disease processes. The term "gray zone" has been used to describe the clinical overlap between EICR and pathology, and clinical frameworks that integrate findings from noninvasive imaging have been proposed. There are four fundamental variants of the "gray zone": 1) LV wall

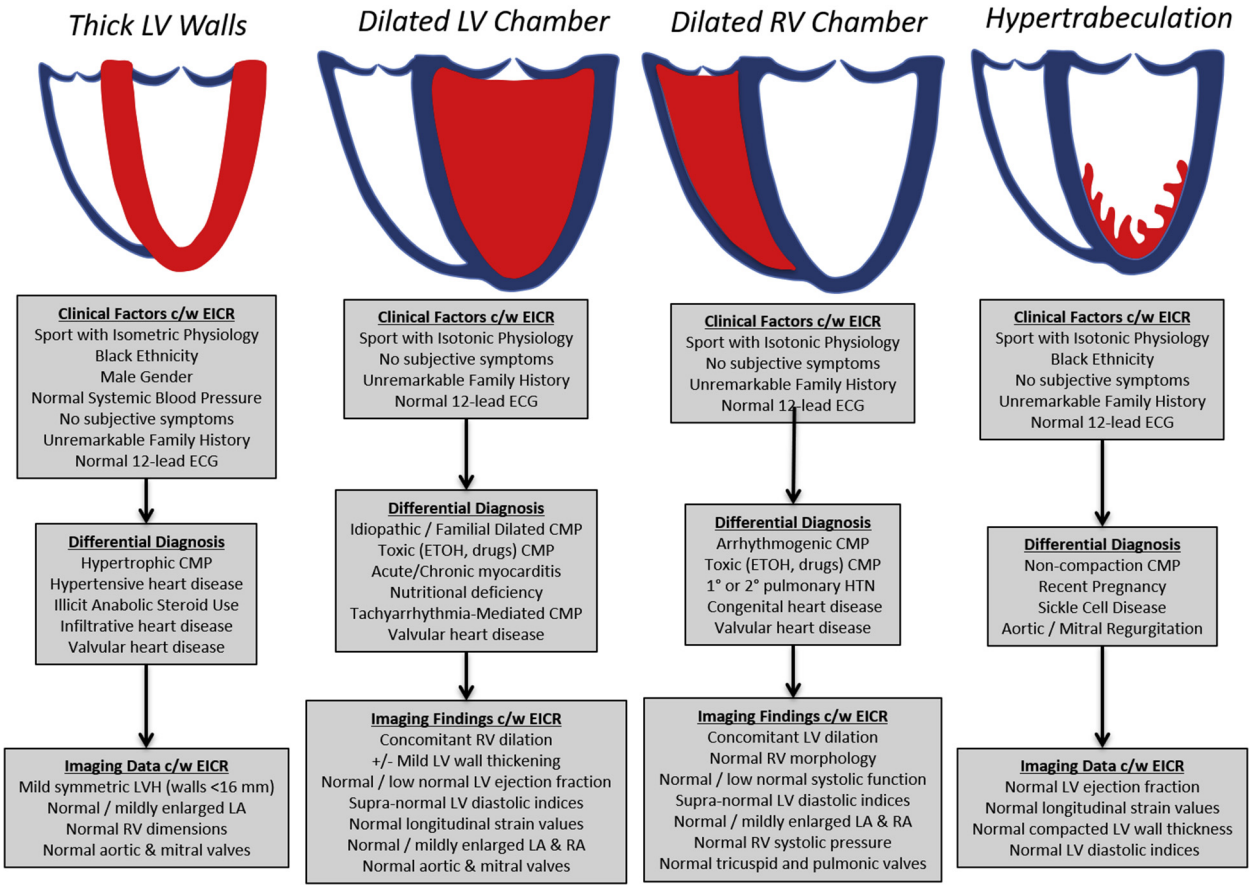

Figure 8 Overview of the clinical assessment of the 4 cardinal "gray zone" imaging findings in competitive athletes for use in differentiating exercise-induced cardiac remodeling (EICR) from pathologic cardiomyopathy (CMP).

thickening; 2) LV chamber dilation; 3) RV dilation; and 4) LV hypertrabeculation, with each leading to distinct differential diagnoses (Figure 8). It must be emphasized that the effective use of clinical imaging data requires integration with other aspects of the clinical presentation, including the presence or absence of symptoms, a family history of genetic heart disease, the 12-lead ECG, and maximal effort exercise testing.

A. Left Ventricular Wall Thickening

Mild to moderate LV wall thickening is common among CA and several forms of LV pathology, including hypertrophic cardiomyopathy, hypertensive heart disease, infiltrative cardiomyopathy, and LV hypertrophy secondary to aortic or mitral valvular heart disease. LV wall thickening attributable to EICR is typically mild, with values in the range of 11-13 mm in Caucasian CA and values up to 15 mm in Black CA. The assessment of LV wall thickening begins by consideration of whether the CA participates in a sporting discipline and/or training regimen with potentially explanatory physiology (i.e., some element of isometric stress). LV wall thickening in endurance CA should be accompanied by concomitant LV dilation,[101] normal or accentuated LV diastolic function, symmetric LV hypertrophy with only mild segmental variation,[63] preserved or low normal systolic function as assessed by LV ejection fraction, and preserved myocardial LV systolic strain.[102] In contrast, isolated symmetric or focal LV hypertrophy with impairment of diastolic function and/or myocardial strain and/or the presence of other anatomic abnormalities including mitral valve leaflet elongation, anomalous papillary muscle insertion, and myocardial crypts or recesses should raise suspicion of myocardial pathology.[103-106] While abnormal indices of diastolic function are atypical in healthy CA, the presence of normal diastolic function does not exclude a diagnosis of hypertrophic cardiomyopathy. The LV apex must be carefully imaged in multiple planes to assess for apical variant hypertrophic cardiomyopathy, using an ultrasound enhancing agent as necessary to delineate the endocardial border during TTE. During TTE, care must be taken to avoid the inclusion of RV septal trabeculations and the posterolateral chordal apparatus to avoid over-measurement of the septal and posterior LV wall, respectively (Figure 9, Figure 10, Supplemental Videos 1 and 2, available at www.onlinejase.com). All CA with LV wall thickening of unclear etiology (i.e. LV wall thickening that cannot be definitively attributed to EICR) by TTE should undergo CMR. CMR evaluation should include careful and comprehensive assessment of LV wall thickness, chamber volume and tissue characterization by late gadolinium enhancement (LGE) and mapping techniques.[107] Limited data suggest that LV hypertrophy among CA develops as a function of myocyte enlargement with minimal fibrosis.[108] In contrast, the presence of LGE and/or interstitial fibrosis is highly suggestive of hypertrophic cardiomyopathy or alternative phenocopy myopathies. In 45 patients with HCM and 734 controls (75% athletic), the LV end-diastolic volume/mass index was significantly lower in HCM than in athletes.[61] In a recent CMR study comparing HCM patients to CA

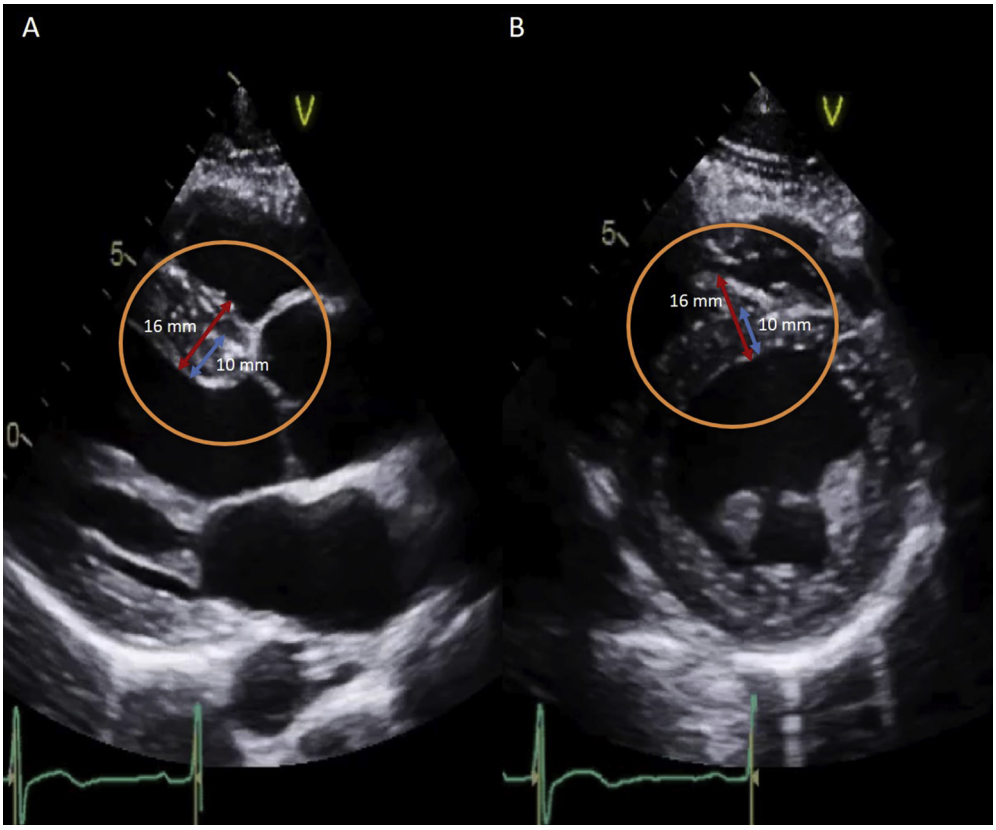

Figure 9 Selected transthoracic imaging from a competitive athlete referred to a tertiary care cardiomyopathy center with asymmetric septal hypertrophy and a presumptive diagnosis of hypertrophic cardiomyopathy due to inaccurate measurement technique. **(A)** Parasternal long-axis view demonstrating measurement of the interventricular septum with (red line) and without inclusion of right ventricular trabecular tissue (blue line). **(B)** Parasternal short-axis view confirming clear delineation of the contractile portion of the true interventricular septum with adjacent RV trabeculations and corollary accurate (blue line) and inaccurate (red line) measurements of septal thickness. Supplemental Videos 1 and 2 demonstrate clear differentiation of the contractile interventricular septum from the akinetic trabecular tissue.

and sedentary controls, markers of interstitial expansion, commonly referred to as extracellular volume by T1 Mapping, were higher in HCM patients than in CA and controls and demonstrated excellent discriminatory capacity across the 3 groups.[109] Patchy late gadolinium enhancement, particularly within focal areas of hypertrophy and/or RV insertion sites, can be seen 1/2 to 2/3 of patients with HCM while isolated RV inferior insertion point fibrosis is only infrequently observed in endurance-trained CA.[110]

Speckle-tracking strain echocardiography has shown promise as a tool to complement routine two-dimensional (2D) imaging in the assessment of ventricular morphology among CA. Global systolic longitudinal LV strain values in CA, the most extensively studied metric, overlap with healthy non-athletic populations (range -16-22%) and values higher (i.e., less negative) than -15% should raise consideration of pathology, particularly in the context of other suggestive findings such as marked ventricular hypertrophy or dilation. At present, the accuracy of TTE-derived myocardial mechanics for differentiating EICR from pathologic myocardium has yet to be firmly established.

Key Points

1. LV wall thickening attributable to EICR is typically mild, with values in the range of 11-13 mm in Caucasian CA, and up to 15 mm in Black CA and Caucasian CA with large body habitus. Values in excess of these cut-points should raise suspicion of pathologic LV remodeling.
2. TTE measurements of wall thickness taken from parasternal long-axis views must be made carefully to avoid inclusion of RV septal trabeculations and posterolateral chordal tissue.
3. LV wall thickening with concomitant reductions in pulsed-wave Doppler and/or tissue Doppler indices of diastolic function and/or reductions in LV longitudinal systolic strain should raise suspicion of pathologic LV remodeling.
4. LV wall thickening of unclear etiology or incomplete visualization of all LV wall segments during TTE should prompt additional imaging with CMR.

B. Left Ventricular Dilation

LV dilation is also common among CA, with several distinct pathology overlaps, including idiopathic and familial dilated cardiomyopathy, toxic cardiomyopathy associated with alcohol over-consumption, tachycardia-mediated cardiomyopathy, acute or chronic myocarditis, and regurgitant aortic or mitral valve disease. The magnitude of LV dilation cannot be used in isolation to differentiate EICR from pathology as athletes often demonstrate marked physiologic dilation. However, physiologic dilation in CA should always be accompanied

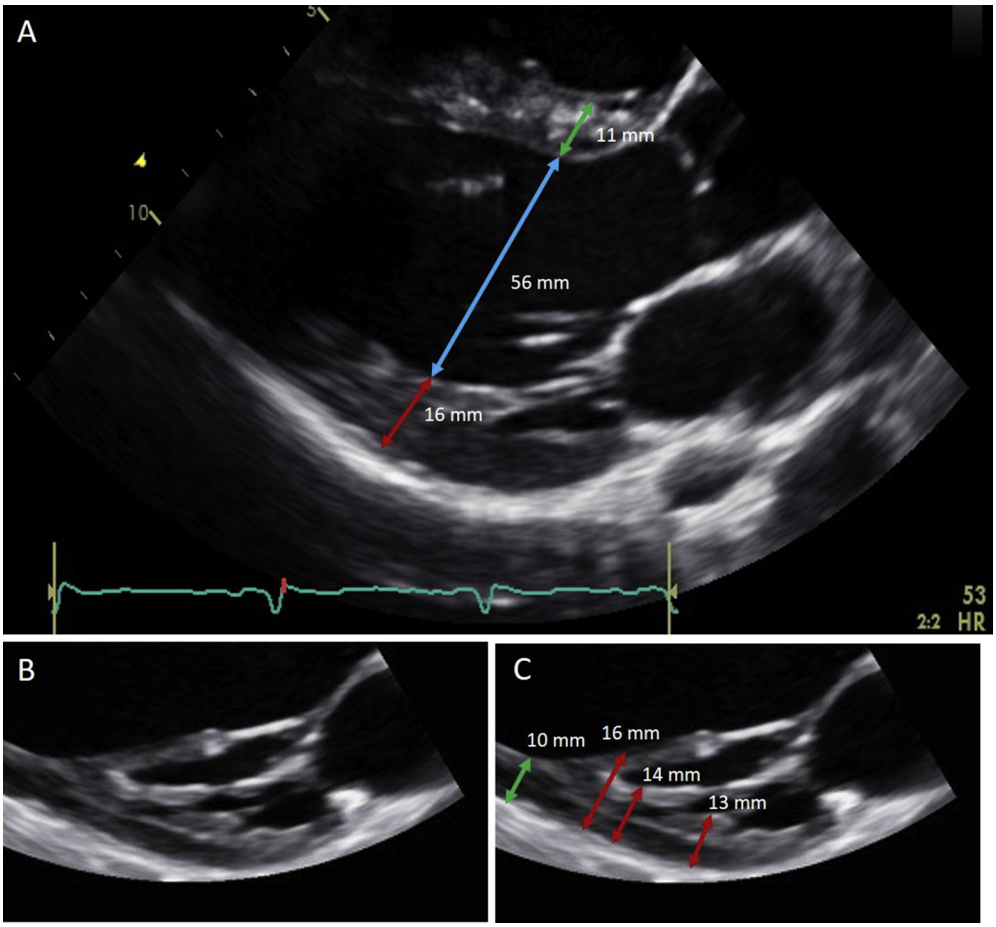

Figure 10 Selected transthoracic imaging from a competitive athlete referred to a tertiary care cardiomyopathy center with asymmetric posterior wall left ventricular hypertrophy and a presumptive diagnosis of hypertrophic cardiomyopathy. **(A)** Parasternal long-axis view demonstrating asymmetric thickening of the posterior wall due to inclusion of posterolateral papillary muscle and chordal tissue (red line) accompanied by a normal left ventricular end-diastolic chamber dimension (blue line) and normal accurately measured interventricular septal thickness (green line). **(B)** Zoomed-in parasternal long-axis view of posterolateral papillary muscle and chordal tissue showing the anatomic complexity that introduces increased likelihood of measurement inaccuracy. **(C)** Common locations of inaccurate measurement leading to over-estimation of posterior wall thickening (red lines) compared to an accurate measurement of posterior wall thickness done by excluding papillary muscle and chordal tissue (green line).

by concomitant dilation of other cardiac chambers with preserved RV systolic function, normal or enhanced diastolic function, and preserved LV systolic strain indices. Endurance-trained CA with corollary EICR may demonstrate mild reductions in LV ejection fraction (45-50%) with preserved resting stroke volume.[78,111] In these cases, impaired LV diastolic function, the presence of regional wall motion abnormalities, the absence of mild concomitant LV wall thickening, less-negative global strain, and impaired contractile reserve during stress TTE or CMR,[112] favor pathology over EICR. Assessment of LA size in the context of LV dilation or hypertrophy, as measured by volumetric assessment or linear dimensions, is of limited value in differentiating left heart disease from EICR as LA enlargement is extremely common among healthy CA.

Key Points

1. Initial characterization of LV chamber volume should be performed using TTE. Among CA with suspected pathologic dilation, CMR should be performed to confirm dilation and to characterize structure and function of the LV and other chambers.
2. The magnitude of LV and LA dilation in CA cannot be used in isolation to differentiate EICR from pathology. Integration of clinical history, myocardial function, and additional diagnostic testing are generally required for this purpose.
3. Low normal to mildly reduced (45-55%) LV ejection fractions in asymptomatic CA with LV dilation are common and can be considered physiologic when accompanied by normal indices of diastolic function and concomitant dilation of the RV and both atria.

C. Right Ventricular Dilation

RV dilation attributable to EICR occurs in endurance-trained CA and must be differentiated from pathologic conditions including arrhythmogenic RV cardiomyopathy, toxic cardiomyopathies, primary and secondary pulmonary hypertension, and congenital heart diseases

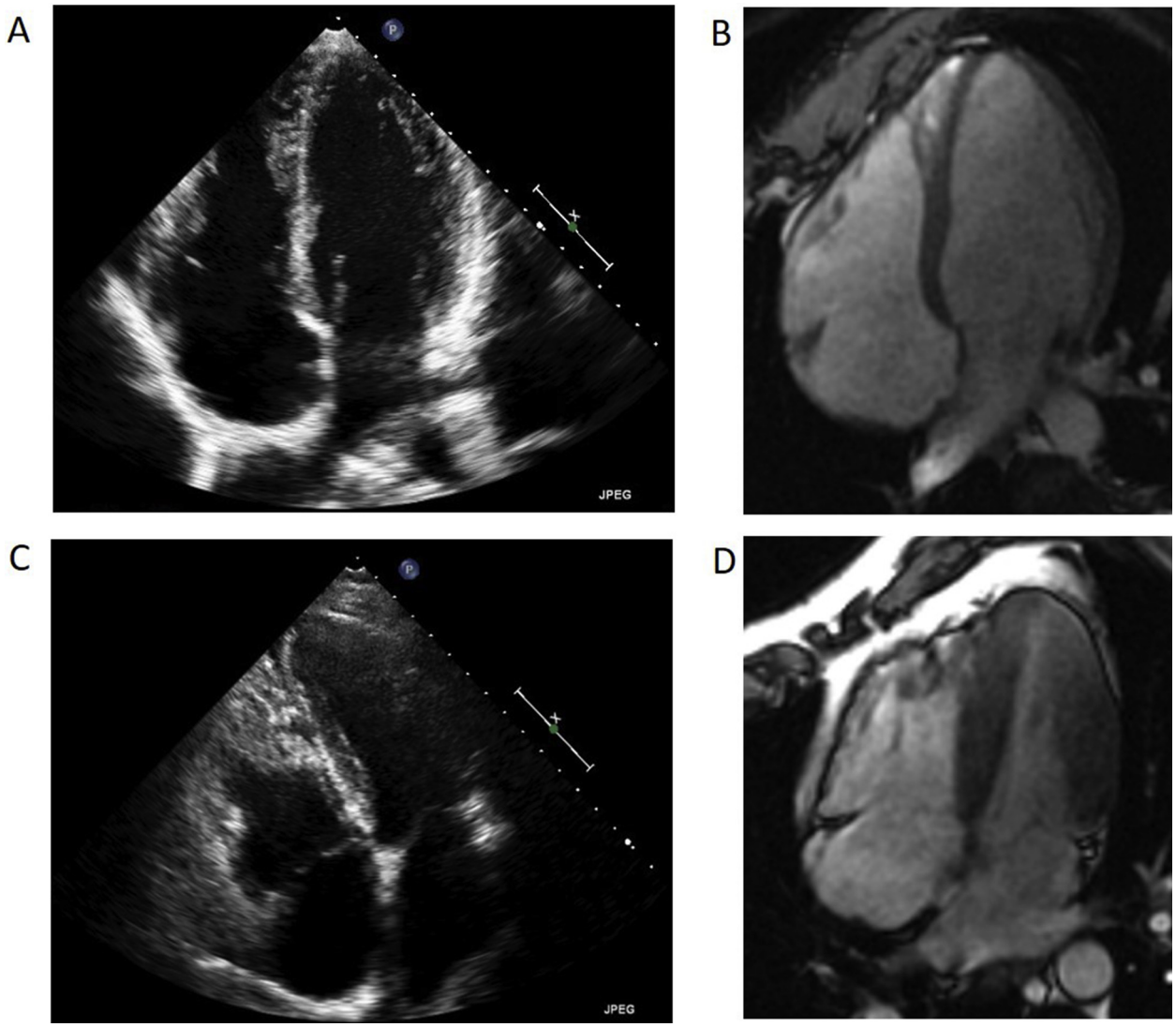

Figure 11 Representative multimodality imaging comparing CA with physiologic RV dilation **(A & B)** and a CA found to have an abnormal ECG during pre-participation ultimately diagnosed with gene-positive arrhythmogenic right ventricular cardiomyopathy **(C & D)**. **(A)** Apical 4-chamber transthoracic echocardiographic view demonstrating comparable left and right ventricular end-diastolic areas and normal biventricular systolic function (Supplemental Video 3, available at www.onlinejase.com). **(B)** Steady-state free precession 4-chamber cardiac MR image demonstrating mild biventricular dilation and a smooth minimally trabeculated right ventricular free wall. **(C)** Apical 4-chamber transthoracic echocardiographic view with incomplete visualization of the RV but suggestion of RV hypokinesis (Supplemental Video 4, available at www.onlinejase.com). **(D)** Steady-state free precession 4-chamber cardiac MR image at end-systole demonstrating isolated right ventricular dilation with sacculation and focal aneurysmal dilation of the mid to distal RV free wall.

with chronic right heart volume overload. Physiologic RV dilation should be accompanied by concomitant LV dilation, preserved or only slightly reduced systolic function without focal wall motion defects, and the absence of anatomic RV abnormalities including sacculations, aneurysms, and focal thinning (Supplemental Video 3, available at www.onlinejase.com). Chamber size in isolation is of limited value in differentiating EICR from pathology. While TTE is capable of establishing RV dilation, it has important limitations for characterizing the relatively complex anatomy of the RV (Supplemental Video 4, available at www.onlinejase.com). Therefore, CMR is required for most athletes with RV dilation of unclear etiology as it provides superior diagnostic accuracy for identifying morphological abnormalities of the RV. As with the LV, mild reductions in RV systolic function in CA should be accompanied by significant contractile reserve during stress imaging.[113] Speckle-tracking strain echocardiography has recently emerged as a promising tool to differentiate RV EICR from pathology.[114-116] However, the limited available data and lack of consensus regarding normal ranges represent current limitations to its implementation in clinical practice. Representative multimodality imaging comparing a CA with physiologic RV dilation with a CA diagnosed with arrhythmogenic right ventricular cardiomyopathy is shown in Figure 11.

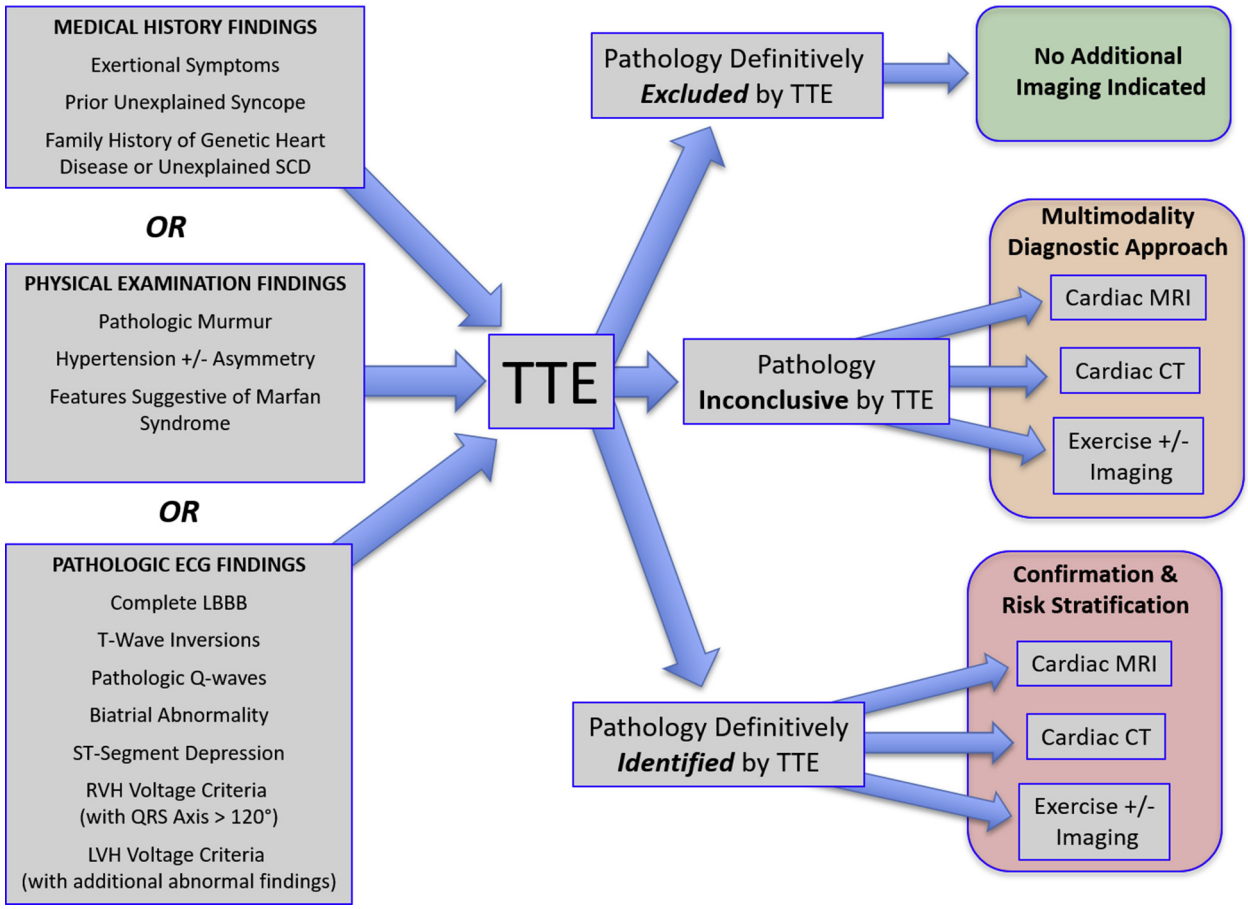

Figure 12 Clinical algorithm for the application of multimodality imaging in competitive athletes following pre-participation cardiovascular screening.

Key Points

1. RV dilation, in conjunction with LV dilation, is common among endurance-trained CA. Physiologic dilation of the RV should occur without structural (i.e. aneurysms and/or focal wall thinning) or functional (i.e. focal hypokinesis) RV abnormalities. Clinical cut-points for RV dilation cannot be used in isolation to differentiate EICR from pathologic RV remodeling.
2. TTE has important limitations with respect to delineating the magnitude and etiology of RV dilation in CA. CMR should be performed in all CA with RV dilation of unclear etiology.

D. Hypertrabeculation

Hypertrabeculation of the LV and or RV apex with normal LV wall thickness is common among CA.[117] Although explanatory mechanisms remain speculative, a chronic increase in cardiac preload in the setting of isotonic physiology has been suggested as a stimulus for trabecular enlargement. Physiologic hypertrabeculation, a benign component of EICR, must be differentiated from noncompaction cardiomyopathy which should be considered among athletes with malignant ventricular arrhythmias or a family history of sudden death. This distinction relies on the ability to image and quantify the two-layer structure (i.e., compacted and noncompacted) of the LV, with a ratio of noncompacted to compacted myocardial layer thickness >2.0 (at end-systole by TTE),[118] or >2.3 (end-diastole by CMR),[119] suggested as diagnostic cut-points for pathologic noncompaction. However, use of these criteria may lead to over-diagnosis of disease as evidenced by a recent screening study in which approximately 15% of normal individuals met at least 1 diagnostic criterion for LV noncompaction.[119,120] Physiologic hypertrabeculation is typically accompanied by preserved systolic function and normal or enhanced diastolic function. The finding of hypertrabeculation, particular when associated with a very thin compacted layer (<5 mm), marked impairment of LV systolic function, LGE on CMR, and other clinical features suggestive of disease require a comprehensive multifaceted evaluation for true noncompaction cardiomyopathy.

Key Points

1. Physiologic hypertrabeculation of the LV apex is common among symptomatic CA. Most frequently observed in Black and endurance-sport CA, physiologic hypertrabeculation should be accompanied by normal LV wall thickness and normal indices of LV systolic and diastolic function.
2. TTE, often requiring the addition of an IV ultrasound enhancing agent for image optimization, represents first line imaging for characterization LV hypertrabeculation among CA. Incomplete characterization by TTE of the LV apex among CA with suspected noncompaction cardiomyopathy should prompt CMR imaging.

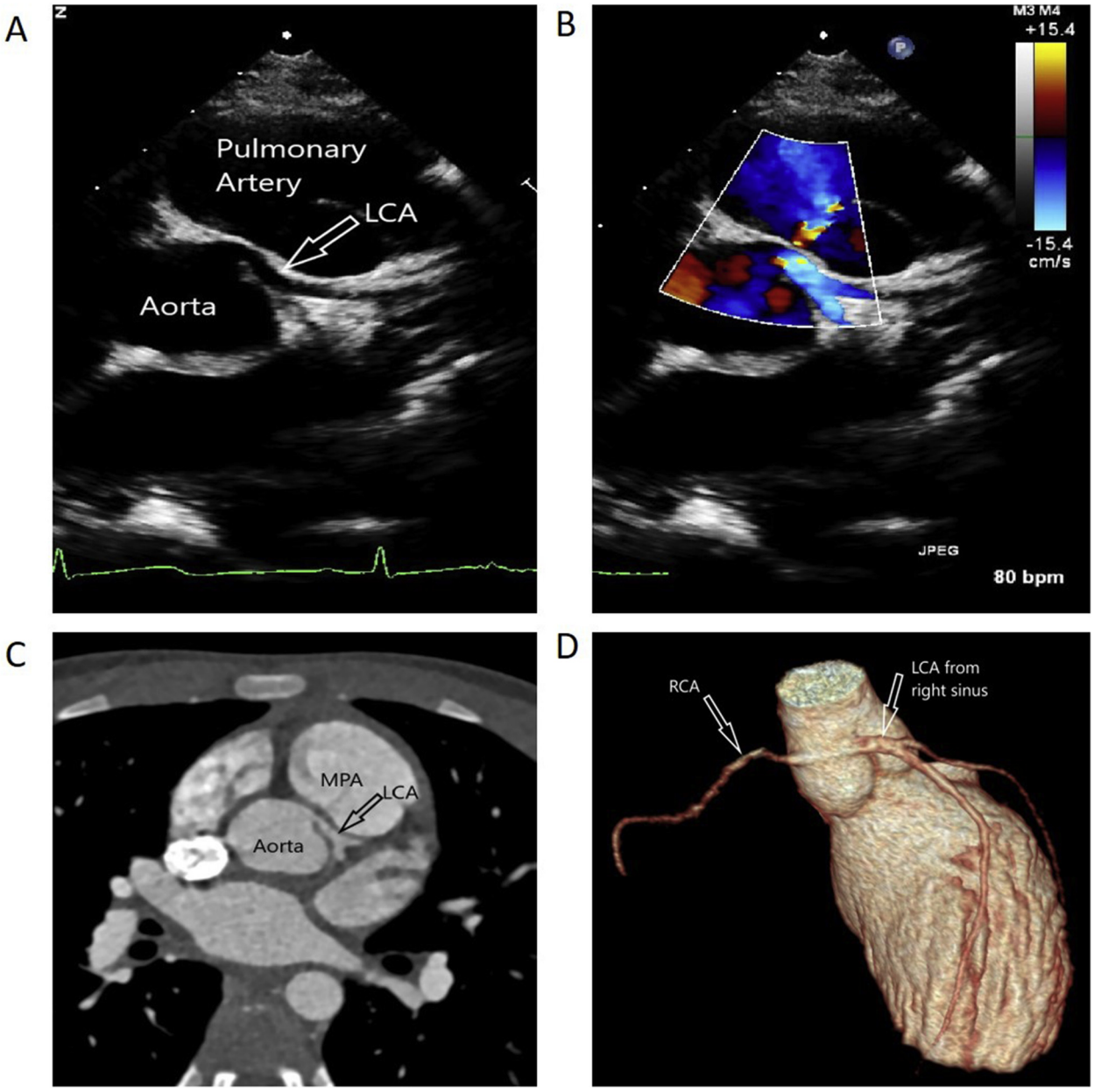

Figure 13 Representative multimodality imaging from a pediatric CA presenting with chest pain and near syncope found to have an underlying high-risk anomalous coronary artery. **(A)** Transthoracic echocardiographic parasternal short-axis view demonstrating an anterior left coronary origin feeding a long left main coronary artery coursing between the aorta and the pulmonary artery. **(B)** Color Doppler imaging demonstrating diastolic flow in the proximal left coronary artery. **(C)** Cardiac CT imaging and 3-dimensional reconstruction **(D)**, confirming origin and proximal course of high-risk anomalous left coronary artery.

V. PRE-PARTICIPATION CARDIOVASCULAR SCREENING

A. Contemporary Standard of Care

Pre-participation cardiovascular screening of CA is designed to detect unrecognized cardiovascular conditions that are associated with increased risk of sudden cardiac arrest during training and/or competition. A detailed discussion of PPCS is beyond the scope of this document, but several key points deserve mention. Current recommendations for PPCS of CA in the United States, as proposed by the American College of Cardiology/American Heart Association, suggest a basic universal screening strategy limited to a focused cardiovascular history and physical examination.[121] To date, there are no data defining the impact of this strategy on mortality in CA, and limitations regarding its accuracy, as manifested by high rates of false-positive and false-negative results, have been described.[122-124] Consequently, several organizing bodies, including the European Society of Cardiology,[125] the International Olympic Committee,[126] and the Fédération Internationale de Football Association,[127] recommend the inclusion of a 12-lead ECG based on its ability to detect clinically silent diseases of the heart muscle and cardiac conduction system. Opponents of ECG-inclusive screening cite potential issues including cost increase, inability to detect important conditions

including congenital anomalous coronary artery anatomy, unacceptable rates of false-positive findings, and lack of widespread expertise in the interpretation of the CA ECG.[128] The sensitivity and specificity of ECG-inclusive screening depends on the criteria used to define what is normal and what is not,[129-131] and on the specific population being evaluated.[132] Criteria designed for ECG interpretation in athletes, first proposed in a formal fashion in 2005,[133] have since undergone numerous revisions,[134-136] with corollary improvements in diagnostic accuracy.[137-139] Accordingly, current United States-based consensus documents now acknowledge the potential value of the ECG during PPCS but provide important stipulations for this approach, including financial resources and clinical expertise.[140]

B. Imaging During PPCS

At present, the use of noninvasive imaging is not recommended as a first-line strategy during PPCS by any professional societies. Nonetheless, some organizations, including large universities, national teams, professional teams, and charitable groups have elected to incorporate some form of imaging into routine PPCS. The impact of this strategy has not been rigorously assessed to date and we caution against its use outside of carefully controlled settings that are resourced with expertise in sports cardiology and the comprehensive noninvasive-imaging resources required to evaluate positive or equivocal findings. Proponents of an imaging-inclusive PPCS strategy, most often the use of TTE, cite potential advantages, including enhanced sensitivity to detect asymptomatic cardiovascular disorders and a reduction in the rate of temporary disqualifications that require future imaging for definitive clarification.[141] Opponents of an imaging-inclusive PPCS strategy justify this stance based on the potential for false-positive findings generated by inconclusive imaging and false-negative findings for certain conditions that may not be readily detectable by some forms of imaging, and the additional requirements of money, clinical expertise, and time. "Limited" TTE during PPCS, a term used to describe abbreviated examinations designed to detect specific high-risk conditions such as hypertrophic cardiomyopathy, have been proposed.[142] To date, the diagnostic accuracy of this approach and its impact on outcomes has not been adequately studied to justify definitive endorsement. The use of advanced imaging, including CMR and CTA as components of PPCS, are similarly discouraged due to issues including high cost, unnecessary radiation exposure for CTA, need for administration of intravenous contrast, and little additional diagnostic yield. Finally, stress echocardiography is not recommended for use during PPCS due to the exceedingly low prevalence of atherosclerotic coronary disease in young CA and its limited diagnostic yield in patients with anomalous coronary anatomy.

Despite its limited role in the setting of PPCS, noninvasive imaging is often required as part of the downstream testing following PPCS (Figure 12). Common abnormal findings that merit consideration of imaging include historical issues such as unexplained prior syncope or a family history of sudden cardiac death in a first-degree relative, subjective report by a CA of exertional symptoms including chest discomfort or inappropriately labored breathing, and "training-unrelated" ECG finding as proposed by current International recommendations.[136] Athletes who demonstrate such abnormalities during PPCS should undergo a targeted diagnostic assessment that is capable of excluding the relevant forms of suggested pathology. In the majority of cases, this evaluation will include one or more imaging tests to document valve morphology, valve function, myocardial structure, myocardial function, and proximal coronary artery anatomy. Given its cost and unparalleled accessibility, TTE should be considered the first-line imaging modality in most athletes following PPCS. Limited data suggest that on-site access to TTE during PPCS is feasible and may obviate the need for further off-site testing.[21] Regardless of where the TTE is conducted, yield following PPCS will be optimized if the referring clinicians communicate with the imaging team prior to testing to confirm the clinical question and corollary differential diagnosis that arose during PPCS. Regardless, post-PPCS imaging should routinely include careful quantitative assessment of biventricular structure and function with emphasis on the characterization of LV wall thickness and symmetry, right ventricular morphology and function, valvular morphology and function, ascending aortic geometry and dimensions, and right and left coronary origin and proximal course. In all cases, the clinical report should indicate any inconclusive TTE data such as the inability to visualize all ventricular wall segments or complete proximal coronary anatomy to facilitate the determination of the need for additional imaging. The use of CMR, CTA, and exercise testing often complements TTE in cases of diagnostic uncertainty or during risk stratification following a definitive diagnosis.

Key Points

1. Routine PPCS of young CA should include a focused personal and medical history and physical examination. The addition of a 12-lead ECG may be considered in situations with adequate financial resources and clinical expertise.
2. The use of noninvasive imaging including comprehensive and limited TTE, CTA, and CMR is not recommended as a first-line strategy during PPCS.
3. PPCS programs should ensure timely access to clinical centers with sports cardiology and clinical imaging expertise to facilitate the comprehensive multimodality imaging required to evaluate findings detected during PPCS.

VI. THE SYMPTOMATIC COMPETITIVE ATHLETE

A. Exertional Chest Discomfort

Chest discomfort is a common reason why CA require clinical evaluation. Cardiac and non-cardiac etiologies both account for chest discomfort, with the latter being responsible for the vast majority of causal diagnoses.[141] Although CV etiologies account for only ~5% of chest discomfort diagnoses, their presence is typically associated with adverse outcomes. The evaluation of exertional chest discomfort in a CA begins with a detailed medical history, physical examination, and resting 12-lead electrocardiogram. These basic steps often identify causal musculoskeletal issues and other non-cardiac etiologies, thereby obviating the need for further cardiovascular diagnostics. When a cardiac etiology is either suggested or not sufficiently excluded, exercise testing and noninvasive imaging represent the next steps in the evaluation. A comprehensive discussion of exercise testing for CA is beyond the scope of this document but several key issues deserve mention. First, exercise

testing should be conducted using a protocol and modality that best approximate the physiology responsible for the presenting symptoms and should be designed to reproduce it.[142] Second, exercise testing, both as a stand-alone test and when coupled with imaging, should never be terminated at a predetermined heart rate (e.g., 85% maximal age-sex predicted) as is common in some laboratories, but instead should be terminated by exhaustion or other high-risk findings, as some patients will manifest symptoms and corollary ischemia only at very high workloads. Third, the need for adjunctive imaging during diagnostic exercise testing should be determined based on current guidelines.[143] Finally, inducible ischemia in CA often resolves very rapidly during exercise recovery, thereby necessitating rapid imaging when post-exercise echocardiography is used to assess for dynamic wall motion defects to avoid false-negative results.

This writing group recommends TTE as the initial noninvasive imaging test for CA presenting with possible or probable cardiac chest pain. In this context, the TTE examination should focus on delineating or excluding the key cardiac causes of exertional chest discomfort, including anomalous coronary anatomy with high-risk features, hypertrophic cardiomyopathy with or without obstructive physiology, and congenital or acquired aortic valve or pulmonic valve stenosis. However, a complete echocardiographic assessment should be performed in all cases and additional imaging is often of considerable value. In CA with suspected or confirmed myocardial pathology such as hypertrophic cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy, CMR is indicated after TTE in most cases as it affords superior diagnostic and prognostic accuracy. In athletes with suspected anomalous coronary circulation by TTE or in whom normal proximal anatomy cannot be confirmed by TTE, CTA or CMR imaging is indicated as dictated by institutional preferences. The primary goals with tomographic imaging include confirmation of coronary origin and proximal course and the delineation of other high-risk features including ostial anatomy, intramural involvement, proximal aneurysmal disease reflecting prior vasculitis, and distal vessel caliber. Representative multimodality imaging in an athlete found to have a high-risk anomalous coronary artery is shown in Figure 13 and Supplemental Videos 5 and 6, available at www.onlinejase.com.

Key Points

1. TTE should be performed as the initial noninvasive imaging test in CA presenting with possible or probable cardiac chest pain.
2. CA presenting with possible or probable cardiac chest pain should undergo maximal effort-limited exercise testing with or without adjunctive imaging as dictated by current guidelines. Exercise stress echocardiography with immediate post-exercise imaging should be applied with caution as rapid heart rate recovery in CA often renders imaging non-diagnostic for the evaluation of ischemia.
3. TTE imaging in CA with possible or probable cardiac chest pain should include careful and definitive delineation of both the left and right coronary origins and proximal course to exclude anomalous coronary circulation. Failure of TTE to confirm normal coronary anatomy requires additional imaging with CMR or gated CTA as dictated by patient, provider, and institutional preferences.

B. Syncope

Syncope, a transient loss of consciousness followed by spontaneous and complete recovery, is common among CA.[144,145] In one study of CA undergoing PPCS, 474 out of 7,568 (6.2%) CA reported at least one episode of syncope during the preceding five years.[146] The vast majority of syncope in CA is attributable to neural mechanisms that carry a benign prognosis. Neurally-mediated syncope manifests as classic "vasovagal" episodes that are unrelated to exercise, or post-exertional syncope in which fainting that typically occurs within seconds to minutes of abrupt exercise termination. In contrast, syncope that occurs during exercise, often characterized by abrupt loss of consciousness with dramatic collapse and musculoskeletal injury, should be attributed to underlying CV etiology and should prompt a thorough evaluation. Common cardiovascular causes of syncope include obstructive valve and outflow tract pathology and arrhythmia arising from inducible ischemia, myocardial scar, or primary disorders of electrical conduction. A comprehensive medical history, physical examination, and resting 12-lead electrocardiogram should be performed for all CA who present following syncope. In the majority of cases, these initial diagnostic steps will be sufficient to confirm neurally-mediated physiology thereby obviating the need for additional evaluation. When one or more aspects of this initial assessment is inconclusive or suggestive of underlying CVD, further evaluation including exercise testing and noninvasive imaging is warranted. This writing group recommends TTE as the initial imaging test in CA with prior syncope and a possible or probable cardiac etiology. The TTE assessment should be geared toward the evaluation of key causes of syncope including obstructive valvular and ventricular outflow pathology, cardiomyopathies with arrhythmic and/or ischemic propensity, and high-risk anomalous coronary anatomy. The use of alternative forms of imaging should be reserved for further evaluation and risk stratification of pathology as suggested or incompletely excluded by echocardiography.

Key Points

1. Neurally-mediated syncope in the post-exercise period or in situations unrelated to exercise is common among CA and does not require evaluation with any noninvasive imaging modality.
2. CA presenting with syncope of unclear etiology, particularly syncope during exercise, should undergo comprehensive multimodality imaging beginning with TTE and extending, on a case-by-case basis, to CTA or CMR to exclude structural and valvular heart disease as part of a comprehensive evaluation.
3. CA presenting with syncope of unclear etiology should undergo maximal effort-limited exercise testing with or without adjunctive imaging as dictated by current guidelines.

C. Palpitations and Arrhythmias

The evaluation of the CA with palpitations or subjective arrhythmia begins with a comprehensive medical history, physical examination, and resting 12-lead ECG. Palpitations under resting conditions often arise from benign premature atrial and/or ventricular beats, are common among bradycardic CA, and are typically benign. In

contrast, palpitations that emerge or intensify during exercise should be considered pathologic until proven otherwise thereby prompting a comprehensive evaluation including non-invasive imaging. All CA with suspected arrhythmia should undergo electrocardiographic capture of the arrhythmia with provocative exercise testing, ambulatory rhythm monitoring or implantable monitoring, and in limited cases drug infusion and invasive electrophysiology study. The primary objective of ambulatory rhythm monitoring is to differentiate malignant ventricular arrhythmias from the more benign variants that arise from the atria or atrioventricular junction. The writing panel suggests a comprehensive TTE examination for CA presenting with palpitations or subjective arrhythmias that occur or intensify during exercise. The role of imaging in athletes with suspected arrhythmia is to diagnose or exclude an underlying causal cardiac abnormality, particularly in the setting of confirmed or suspected ventricular arrhythmias, as arrhythmias may arise from abnormal myocardial substrate including genetic, congenital or acquired cardiomyopathy, edema and inflammation in the setting of acute myocarditis, or scar from prior myocardial injury. Alternatively, arrhythmias may be caused by subendocardial ischemia from obstructive atherosclerotic disease, congenital coronary anomalies, or pressure/volume overload in the context of valvular pathology. Finally, arrhythmias in CA may be attributable to primary disorders of electrical conduction, including ventricular pre-excitation and genetic channelopathies. Ventricular pre-excitation on the 12-lead electrocardiogram, either detected during PPCS in an asymptomatic CA or in a CA presenting with arrhythmic symptoms, should prompt imaging to exclude associated cardiac conditions including Ebstein's anomaly,[147] PRKAG2 gene-mediated hypertrophic cardiomyopathy,[148] and complex forms of congenital heart disease. The primary channelopathies/inherited arrhythmia syndromes, including long QT syndromes, catecholaminergic polymorphic ventricular tachycardia, Brugada syndrome, and idiopathic ventricular tachycardia are not associated with underlying myocardial, valvular, or coronary abnormalities that can be detected with contemporary imaging. Nonetheless, this writing group recommends a comprehensive TTE to exclude alternative diagnoses,[149] when history or 12-lead ECG suggests one of these entities. CTA and CMR imaging in CA with suspected arrhythmia should be considered second line and be performed as dictated by suspected or confirmed pathology following TTE. Specifically, CMR should be performed following a normal TTE examination among CA with documented complex ventricular arrhythmia to evaluate for underlying myocardial scar.

Key Points

1. CA presenting with palpitations or subjective arrhythmias that occur or intensify during exercise should undergo TTE to diagnose or exclude underlying structural disease. Additional imaging with CMR may be appropriate on a case-by-case basis.
2. CA presenting with palpitations or subjective arrhythmias that occur or intensify during exercise should undergo maximal effort-limited exercise testing with or without adjunctive imaging as dictated by current guidelines.
3. All CA undergoing evaluation for ventricular pre-excitation, both asymptomatic and symptomatic, should undergo TTE to exclude concomitant Ebstein's anomaly, PRKAG2 gene-mediated hypertrophic cardiomyopathy, and complex forms of congenital heart disease.

D. Inappropriate Exertional Dyspnea

Perceived breathlessness during exertion is common among CA and may be a manifestation of underlying CVD.[150,151] The neurophysiology of dyspnea is not well understood but the sensation can be generated by neural feedback mechanisms from the cardiovascular, respiratory, and musculoskeletal systems.[152] A careful history focusing on the duration and intensity of exercise is often adequate to determine if the corollary sensation of breathlessness is appropriate or inappropriate for that individual athlete. Subjective breathlessness is typically appropriate as CA approach their physiological upper limits of exercise capacity as often occurs during training escalation or after prolonged deconditioning. In contrast, dyspnea at previously tolerable exercise intensities and new-onset dyspnea without change in training regimen or competition should be considered inappropriate and thus trigger further evaluation. Associated symptoms including stridor, wheezing, chest tightness, chest pain, palpitations, and presyncope/syncope are useful in dictating the subsequent evaluation. Non-cardiac etiologies, including reactive airway disease with or without exercise-induced bronchospasm, paradoxical vocal fold movement disorder, upper respiratory infection, allergic and non-allergic rhinitis, and dysfunctional breathing account for the majority of inappropriate exertional dyspnea in young CA.[153] CA presenting with history and physical examination consistent with one of these diagnoses do not require any form of cardiac imaging prior to empiric treatment. However, failure to respond adequately to medical therapy, particularly in CA with suspected exercise-induced bronchospasm, should raise consideration of alternative, possibly cardiac etiologies.

Most forms of underlying CV disease can cause inappropriate exertional dyspnea. This writing group recommends a comprehensive TTE in CA presenting with inappropriate exertional dyspnea, and in CA with previously diagnosed non-cardiac cause of inappropriate exertional dyspnea who do not demonstrate adequate response to therapy. Exercise testing, with or without concomitant imaging, should also be performed in both situations. Assessment of gas exchange, in the form of both pulmonary function testing and cardiopulmonary exercise testing, is often valuable in patients with inappropriate exertional dyspnea. The role of tomographic imaging in CA with inappropriate exertional dyspnea should be considered second line and be performed as dictated by suspected or confirmed pathology following TTE.

Key Points

1. Inappropriate exertional dyspnea may occur in the context of numerous cardiovascular diseases. Comprehensive TTE should be performed in CA presenting with inappropriate exertional dyspnea, and in CA with a previously diagnosed non-cardiac cause of inappropriate exertional dyspnea who do not demonstrate adequate response to therapy.
2. Patients presenting with Inappropriate exertional dyspnea should undergo maximal effort-limited exercise testing with or without adjunctive imaging as dictated by current guidelines.

E. Athletic Performance Decrement

CA presenting for clinical evaluation in the context of performance decrement pose a difficult diagnostic dilemma with a broad differential diagnosis. A comprehensive history and physical should include

assessment of dietary intake to ensure adequate energy balance, sleep patterns, mood disturbances, and evaluation for signs and symptoms of common organic pathologies including endocrine disorders (such as thyroid or adrenal disorders and diabetes), infectious etiologies (such as glandular fever or hepatitis), inflammatory diseases, electrolyte deficiencies, and anemia. Training in unfamiliar conditions (altitude, heat or cold) or a significant increase in training load or change in training patterns should be considered. Overtraining syndrome is common and affects ~60% of elite runners and 33% of non-elite runners, but should be considered as a diagnosis of exclusion.[154] Evaluation is best performed by an experienced sports cardiologist or in combination with a sports medicine physician, and may ultimately require involvement of multiple other specialties. Testing following medical history and physical examination should be predicated on clinical suspicion. It is reasonable but generally of low yield to obtain a 12-lead ECG on all CA with unexplained performance decrement. The writing committee advocates for the use of TTE in CA with some historical, physical examination, or electrocardiographic finding suggestive of myocardial, coronary, or valvular pathology or in cases where no conclusive explanation is determined during initial evaluation. The use of CTA or MRI imaging should be reserved for individual situations as dictated by suspected or confirmed pathology.

Key Points

1. TTE should be performed for CA presenting with athletic performance decrement when medical history, physical examination, 12-lead electrocardiography, or routine bloodwork yield one or more findings suggestive of myocardial, coronary, or valvular pathology or when no clear explanation can be ascertained from the initial evaluation.
2. The use of exercise testing and CTA/MRI imaging should be considered on a case-by-case basis among CA presenting with athletic performance as dictated by suspected or confirmed pathology.

VII. ADDITIONAL CONSIDERATIONS

A. Masters Level CA

CA span the entire age range and masters level CA, competitive sports participants older than 35 or 40 years as dictated by specific sporting disciplines, are a rapidly growing population worldwide. Masters level CA, often presenting with the same cardinal manifestations of disease discussed above, are less likely to have congenital or genetic heart disease and more likely to be experiencing symptoms attributable to acquired diseases of the heart muscle, electrical system, and coronary arteries. The most common cardiac causes of morbidity and mortality in masters level CA are atherosclerotic coronary disease, atrial tachyarrhythmias (particularly atrial fibrillation), degenerative aortic and mitral valve disease, and hypertensive heart disease. A detailed discussion of each condition has been previously published and is beyond the scope of this document.[4] However, several practical considerations for the cardiovascular imager are noteworthy.

Atherosclerotic coronary disease is the most common cause of cardiac arrest during exercise in masters level CA.[155] The diagnosis of atherosclerotic coronary disease in this population requires a high index of suspicion in the setting of exertional symptoms and is often but not always accompanied by the presence of traditional atherosclerotic risk factors including hypertension, dyslipidemia, family history of disease, and prior/ongoing tobacco use. Exercise stress testing and coronary imaging with invasive or CT coronary angiography are key components of this diagnostic evaluation. This writing group recommends CTA following exercise testing in patients judged to be at low or intermediate risk of obstructive disease. In higher-risk CA, traditional coronary angiography in lieu of CTA may be an appropriate next step, as dictated both by patient and provider preference. The utility of coronary artery calcium (CAC) scoring in masters level CA remains controversial. Several studies document a higher CAC burden in this population than can be explained by traditional risk factors and a tendency for CA to have calcified versus non-calcified plaque.[156,157] While emerging data suggest that CAC in CA may carry a relatively benign prognosis,[158,159] the full prognostic implications of CAC and its relationship with obstructive forms of disease responsible for symptoms and attendant risk in CA have not been firmly established. Consequently, this writing group does not recommend the use of CAC scoring in isolation in asymptomatic or symptomatic masters level CA.

Atrial tachyarrhythmias, including atrial fibrillation and atrial flutter, are common among masters level CA and their incidence appears to be associated with lifelong duration and intensity of sport participation.[160] Although the diagnosis of these arrhythmias does not require noninvasive imaging, the writing group recommends comprehensive TTE for all CA with these arrhythmias to exclude concomitant tachycardia-mediated cardiomyopathy and to exclude valvular etiologies of arrhythmia. The use of tomographic imaging in masters level CA with atrial arrhythmias should be confined to use for anatomic mapping once an invasive strategy utilizing catheter-based ablation has been established.

Hypertension is common, often under-diagnosed, and routinely undertreated in masters level CA. While routine vigorous physical exercise has some beneficial effects on blood pressure, its impact is typically inadequate to prevent or treat hypertension in predisposed CA. Hypertension in CA may be diagnosed by serial resting measurements, ideally obtained during ambulatory monitoring, and is frequently suggested by hypertensive blood pressure response to exercise testing. This writing committee recommends comprehensive TTE in CA with newly established hypertension to assess for acquired hypertensive LV hypertrophy, a phenotype that can be differentiated from EICR based on assessment of diastolic function, as delineated by current clinical guidelines.[161]

B. Pediatric CA

Children and adolescents represent a significant segment of the overall CA population. Many of the key cardiac conditions associated with an increased risk of sudden death during sport become clinically apparent during this early life period. To date, there are sparse data documenting EICR in pre- and early-adolescent CA. Available data suggest that EICR, including LV hypertrophy and chamber dilation, may occur but at a lesser magnitude than that typical of adult CA.[162] Independent of sport however, pediatric CA are in a period characterized by rapid growth of the heart and blood vessels, thereby making it difficult to differentiate normal growth-related changes from changes due to EICR or from emerging pathology. Challenges in differentiating normal physiology from pathology are further compounded by standard pediatric-practice use of body surface area-normalized cardiovascular measurements and by the relative dearth of age-specific

normative data in pediatric CA. In a recent study presenting normative echocardiographic data in Polish pediatric CA ($n = 791$, age 13 ± 5 years), mean values of cardiac dimensions were higher compared to general pediatric population estimates but in most cases, the upper limit of normality in the athletes was confined within the upper limit of the general population.[58] One cross-sectional study of 140 pediatric athletes utilizing TTE and deformation imaging revealed geometric and functional differences across five sporting disciplines. Swimmers, the only endurance discipline studied, had larger LV chambers and more eccentric hypertrophy than other team and individual sporting disciplines.[163]

This writing group recommends that the use of imaging in pediatric CA be pursued in a manner analogous to that delineated above for adult athletes, under the guidance of pediatric imaging experts. In addition, the writing group recommends the use of standard z-scores during interpretation with the acknowledgement that some pediatric CA, particularly those that are nearest chronologically to adulthood and those that engage in high levels of endurance sports, may demonstrate physiologic remodeling resulting in abnormal measurements. Such cases may require a collaborative approach involving input from pediatric cardiologists and imagers with expertise in the care of athletes with input from adult sports cardiologists and imaging specialists. The development of more data delineating the extent of physiologic remodeling that can occur during early life and its corollary normative data for pediatric CA represent important areas of future work.

C. Congenital Heart Disease

Dramatic advances in the management of patients with congenital heart disease, with increasing recognition of the health benefits of routine exercise, have led to an increase in the number of CA with established congenital cardiac conditions. However, the safety of competitive sport participation in people with congenital heart defects has not been rigorously studied. Almost certainly, the overall risk-benefit balance is determined by several key factors, including the type of congenital malformation, severity of the malformation, prior and ongoing therapeutic interventions, and the volume and intensity of sport participation. Current competitive sport eligibility recommendations for people with congenital heart disease provide a useful template for use in clinical care, with data derived from noninvasive imaging factoring prominently in recommendations.[164] It should, however, be noted that these recommendations reflect the absence of rigorous supportive data and represent expert opinion. At present, there remains wide variation in the implementation of these guidelines in clinical practice and individualized recommendations for patients, including CA, is common.[165] There are several multimodality imaging guidelines geared toward specific congenital heart lesions that are relevant when tailoring the imaging approach to the CA with congenital heart disease.[166,167] While exercise stress echocardiography has recently been used to examine myocardial reserve in children with congenital heart disease, its role in the risk assessment of CA remains uncertain.[168-170]

This writing committee identifies competitive sport participation in young people with congenital heart disease, including but not limited to the role of noninvasive testing, as an important area of active research. In the interim, we recommend comprehensive assessment of all CA diagnosed with congenital heart disease that is more than minor in severity to include functional assessment with maximal effort-limited exercise testing, multimodality imaging as dictated by lesion type and severity, a shared decision-making approach to eligibility recommendations, and close surveillance as determined on a patient-by-patient basis.

VIII. CONCLUSIONS AND FUTURE DIRECTIONS

This document provides an overview of the fundamental role of noninvasive cardiovascular imaging in the care of young CA. The principles contained in this document permit clinicians to apply and interpret noninvasive imaging with accuracy and cost-effectiveness. A carefully constructed multimodality imaging strategy has the potential to diagnose and risk-stratify athletes with CVD and to exclude the presence of disease to permit safe unrestricted competition. Optimal use of multimodality imaging in CA requires both an understanding of EICR and the strengths and weaknesses of available imaging techniques. TTE should be considered the first-line imaging modality in CA with suspected pathology. CTA, CMR, and exercise with imaging provide complementary data and their use should be considered in cases of suspected or confirmed pathology. Differentiating EICR from mild forms of pathology remains challenging in clinical practice and corollary data in specific groups, including children and people with congenital heart disease, are sparse. Further acquisition of normative data, particularly data derived from CMR and CTA, may further address these important contemporary clinical challenges.

Reviewers: This document was reviewed by members of the ASE Guidelines and Standards Committee, ASE Board of Directors, ASE Executive Committee, and designated representatives from the Society of Cardiovascular Computed Tomography and Society for Cardiovascular Magnetic Resonance. Reviewers included Azin Alizadehasl, MD, FASE, Alicia Armour, MA, BS, RDCS, FASE, Scott D. Choyce, RDCS, RVT, RDMS, FASE, Maros Ferencik, MD, PhD, MCR, Craig Fleishman, MD, FASE, Benjamin Freed, MD, FASE, Edward Gill, MD, FASE, Suzanne Golz, MS, RDCS, FASE, David Harrild, MD, FASE, Renuka Jain, MD, FASE, Pei-Ni Jone, MD, FASE, William Katz, MD, FASE, Stephen H. Little, MD, FASE, Jonathan R. Lindner, MD, FASE, Viviana Maestrini, MD, PhD, Yasdet Maldonado, MD, Anuj Mediratta, MD, FASE, David Orsinelli, MD, FASE, Alan S. Pearlman, MD, FASE, Andrew Pellett, PhD, RDCS, FASE, Geoffrey A. Rose, MD, FASE, David H. Wiener, MD, FASE, Megan Yamat, RDCS, RCS, FASE.

SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.echo.2020.02.009.

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