STATE-OF-THE-ART REVIEW ARTICLE

Echocardiography: Profiling of the Athlete's Heart

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Cardiovascular physiologic remodeling associated with athleticism may mimic many of the features of genetic and acquired heart disease. The most pervasive dilemma is distinguishing between normal and abnormal physiologic remodeling in an athlete's heart. Imaging examinations, such as magnetic resonance imaging and computed tomography, which focus predominantly on anatomy, and electrocardiography, which monitors electrical components, do not simultaneously evaluate cardiac anatomy and physiology. Despite nonlinear anatomic and electrical remodeling, the athlete's heart retains normal or supernormal myocyte function, whereas a diseased heart has various degrees of pathophysiology. Echocardiography is the only cost-effective, validated imaging modality that is widely available and capable of simultaneously quantifying variable anatomic and physiologic features. Doppler echocardiography substantially redefines the understanding of normal remodeling from preemergent and overt disease. (J Am Soc Echocardiogr 2014;27:940-8.)

Keywords: Athletic training, Adaptive cardiac remodeling, Athletic heart, Structural heart disease, Echocardiography

The athletic heart's structural and functional changes intrigue those in the medical community who care for the athlete population.[1] Intense or chronic athletic training prompts complex remodeling of the anatomy and physiology of the heart to accommodate a state of enhanced cardiovascular performance.[1] The distinction between adaptive and maladaptive remodeling is the fundamental challenge in accurately defining individualized risk in athletes.[2] Typically, an athletic heart is associated with cardiac chamber enlargement, increased left ventricular (LV) mass and modest aortic root enlargement to accommodate increased physiologic demands. Adapted morphology and physiology vary considerably among athletes and are affected by each individual's form and intensity of athletic training.[3] Cardiovascular remodeling is nonlinear, meaning that any particular feature can change unpredictably and vacillate between normal and abnormal during the remodeling process. Myocardial adaptation and performance are unique to each individual athlete and represent complex nonlinear interactions between multiple organ and physiologic systems.[4]

Although conventional pathophysiologic guided diagnostics and therapeutics have been used for decades, there are significant limitations that are particularly challenging.[5] Disease is rarely a consequence of a single effector but rather a reflection of a set of morphophysiologic processes that interact in a complex network. (i.e., a module composed of closely related features is discernible only by appreciating the behavior of the network as a whole rather than its individual components). This shortcoming accounts for many limitations of defined disease determinants and design of rational decisions. This background highlights the need to reconsider and redefine the determinants of cardiovascular risk in athletes and the logic of implementing clinical Doppler echocardiographic classification methods.

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In this report, we review the physiologic and morphologic features associated with variable athletic training in endurance and strength athletes, the incidence and associations of sudden cardiac death (SCD) in athletes, and the importance of personal and family history and physical examination in guiding diagnostic testing. We offer a detailed discussion of how echocardiography plays an essential role in distinguishing adaptive from maladaptive remodeling.

The distinction between adaptive and maladaptive remodeling requires an understanding of the evolving myocyte changes temporally. In disease states, there is an early transition from normal to abnormal myocyte function, followed by longitudinal myocyte dysfunction (manifested as a reduction in early diastolic mitral annular tissue velocity [e′]), followed by diastolic strain, strain rate, and twist dysfunction and, ultimately, global systolic strain rate (indicating fibrosis and cell death). The athletic heart begins with hypernormal function. The athlete's heart continues to be normal or hypernormal, whereas the diseased heart evolves along a cascade from early abnormal myocyte dysfunction to myocardial dysfunction. This distinction is illustrated when comparing athletic heart and hypertrophic cardiomyopathy (HCM) (Table 1).

REMODELING FEATURES

Athletic heart exhibits complex, variable physiologic states. For example, intense physical exercise enhances cardiac output six- to eightfold and increases pulmonary oxygen uptake. An athlete's heart rate can range from <40 beats/min at rest to >220 beats/min at peak exertion. The dynamic changes in LV relaxation in the athletic heart account for the increased stroke volume and cardiac output at extreme heart rates. The body's cardiovascular network is a fine-tuned, nonlinear feedback system. The contiguous architecture of the cardiovascular system (atria, ventricles, and aorta) undergoes continuous nonlinear remodeling that reflects adaptive changes in both athletic and disease states (i.e., a state of ongoing change or "flux"). A disease with a contiguous system is referred to as a "continuity disease." An example of a continuity disease is hypertension, which causes abnormal aortic pressure with negative feedback to

Abbreviations

ARVD = Arrhythmogenic right ventricular dysplasia

HCM = Hypertrophic cardiomyopathy

LV = Left ventricular

MRI = Magnetic resonance imaging

RV = Right ventricular

SCD = Sudden cardiac death

the ventricles and, subsequently, the atria, resulting in remodeling of the adjacent cardiac chambers of the contiguous cardiovascular system.

ENDURANCE VERSUS STRENGTH TRAINING

Individual athletic disciplines result in individualized variability of cardiovascular remodeling, which reflects the nature and intensity of the individual athletic activity. Endurance exercise involves sustained elevation in cardiac output with reduced peripheral vascular resistance, resulting in a continuous volume challenge for all cardiac chambers. Long-distance running, cycling, and swimming represent endurance exercise. Alternatively, strength training involves exercise activities that are characterized by cardiac output that is normal or slightly elevated and increased peripheral vascular resistance; this results in increased blood pressure and LV afterload. Weightlifting, football, and wrestling are athletic disciplines representative of strength training. Overlap sports, including soccer, basketball, and hockey, encompass significant constituents of endurance and strength exercise training. The variable hemodynamic effects play a major role in the degree and type of individualized cardiovascular remodeling.

INDIVIDUAL MORPHOLOGY AND PHYSIOLOGY

Before an echocardiographic evaluation, there must be a comprehensive medical evaluation. The athlete's health profile, including supine and standing blood pressure, heart rate, venous and arterial examination, and cardiac auscultation, should be recorded. The athlete's medical history, personal family history, and type and intensity of athletic activity must be obtained and recorded. This background information will guide the type of testing needed. Perfunctory stress and electrocardiographic testing is usually of little value and is associated with false-positive results, which can markedly increase ancillary costs.[6] Screening stress tests are not cost effective.[7-9]

INCIDENCE AND ASSOCIATIONS OF SUDDEN CARDIAC DEATH

Approximately 50% of all athletic deaths are due to unanticipated accidents.[10] Most common forms of disease associated with SCD are attributed to structural and physiologic cardiovascular disease that predispose athletes to fatal events. HCM is the disease most commonly associated with cardiac sudden death in the United States. Arrhythmogenic right ventricular (RV) dysplasia (ARVD) is the disease most commonly associated with cardiac sudden death in Italy. Arrhythmogenic syndromes, such as ion channelopathies, are relatively uncommon causes of SCD that typically lack evidence of structural heart disease. Sports-related SCD in the general population is considerably more common than previously suspected.[11] The cardiovascular sudden death rate in college athletes is higher than previous reports in high school athletes.[12] The determinants of these finding are uncertain, but they are potentially attributable to the longer exposure of college athletes to rigorous training regimens and longer durations of training. Drug and alcohol accessibility may be responsible for the higher sudden death rate in college athletes.[12] Black college athletes are at a fivefold greater risk for cardiovascular sudden death than white athletes.[13] Male athletes' risk for SCD exceeds that for female athletes by three- to sixfold.[14]

Table 1 Distinguishing athlete's heart from HCM

Data feature	Athlete's heart	HCM	Value
Increased wall thickness	Yes	Yes	No
Atrial remodeling	Yes	Yes	No
Systolic function	Normal	Normal	No
Mechanical function (strain)	Normal	Abnormal	Yes
Diastolic physiology	Normal	Abnormal	Yes

Data from Paterick TE, Jan MF, Paterick ZR, Umland MM, Kramer C, Lake P, et al. Cardiac evaluation of collegiate student athletes: a medical and legal perspective. Am J Med 2012;125:742–752.

MORPHOPHYSIOLOGIC ECHOCARDIOGRAPHY

Echocardiography is the most logical means of defining and quantifying normal and abnormal physiology and morphology in a single examination. Combining cardiac morphology and physiology is recommended as the most definitive way to classify cardiovascular risk in most athletes.[15] Imaging modalities that do not incorporate simultaneous physiologic information provide much less useful discriminatory information.

SPECIFIC FEATURES OF ATHLETIC HEART

The Left Ventricle

The athletic heart typically has increased chamber dimensions and increased LV wall thickness. These findings often mimic the echocardiographic features of diseases affecting the left ventricle.[16-18] These unique findings of increased wall thickness and LV dilation are more common in athletes who engage in the most strenuous levels of exercise training.[19]

The unique remodeling of endurance athletes results in eccentric hypertrophy, in which there is increased wall thickness and chamber dilation. Strength-trained athletes display thickening of the LV wall with mild LV dilation, resulting in concentric hypertrophy. Combination athletes typically display a phenotype with overlapping features of endurance and strength-trained athletes.[18] LV eccentric and concentric hypertrophy, without physiologic classification, can be inappropriately misinterpreted as HCM.

Increased LV wall thickness results from chamber pressure, volume overload, or both.[20,21] The true phenotypic expression is often a combination of pressure and volume overload of the myocardium. Concentric remodeling increases the relative wall thickness without an increase in LV mass.[22] The remodeling of the left ventricle is more eccentric in endurance athletes, but athletes often maintain balanced hypertrophy.[23] In general, athletes more commonly have concentric remodeling. Extreme LV remodeling occurring in some ultra-elite athletes has raised a concern as to whether such extreme morphologic adaptation has potential adverse clinical consequences, as 10% to 45% of elite endurance athletes have LV cavity end-diastolic dimensions $>$ 60 mm. This magnitude of enlargement is identified in pathologic forms of dilated cardiomyopathy.[17] The

Table 2 LV characteristics in athletes and nonathletes

LV function	Athletes	Nonathletes
Morphology		
IVSd (mm)	8–13	6–10
LVIDd (mm)	49–65	42–59
LVM (g)	113–400	88–224
Volumes/EF		
LV EDV (mL)	130–240	67–155
LV EF (%)	45–70	>55
Tissue Doppler		
Sm (cm/sec)	6.5–14	>6
e′ (cm/sec)	7.5–16	>8
Mechanical parameter		
Strain/strain rate	Similar to nonathletes (GLS > −18%)	GLS > -18%

EDV, End-diastolic volume; *EF*, ejection fraction; *GLS*, global longitudinal strain; *IVSd*, interventricular septal thickness at diastole; LVIDd, LV internal diameter at diastole; LVM, LV mass; *Sm*, tissue Doppler imaging peak velocity at systole.
Data from Paterick TE, Jan MF, Paterick ZR, Umland MM, Kramer C, Lake P, et al. Cardiac evaluation of collegiate student athletes: a medical and legal perspective. Am J Med 2012;125:742–752.

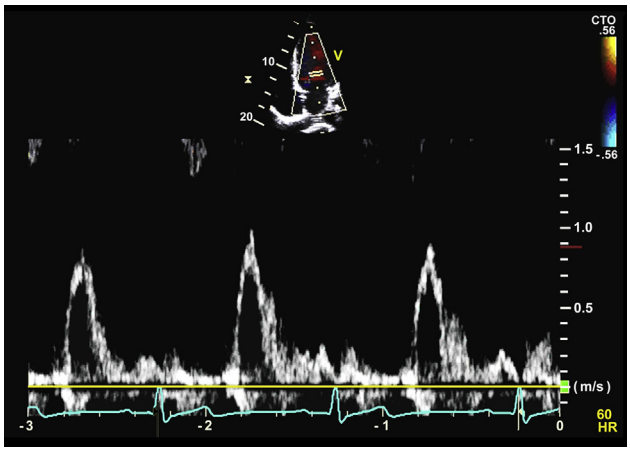

Figure 1 Pulsed-wave Doppler of mitral inflow obtained by placing the pulsed-wave Doppler gate at the very tip of the mitral valve leaflets in the apical four-chamber window: Doppler tracing reveals superior early diastolic filling as assessed by E-wave velocity and near absence of the A wave. This is characteristic of an endurance-trained athlete.

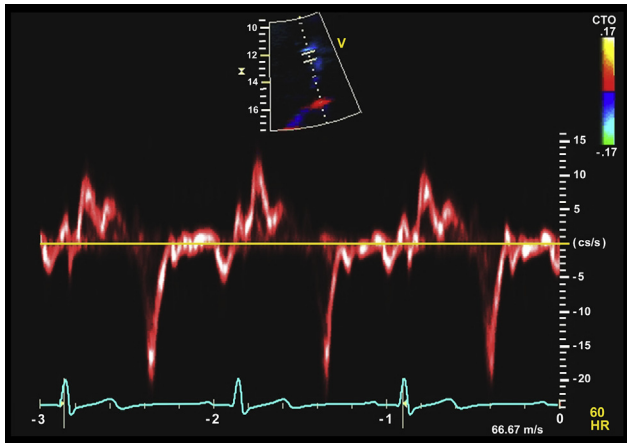

Figure 2 In the apical four-chamber view, a 2-mm pulsed-wave Doppler gate is placed at the medical annulus, obtaining the peak early diastolic, atrial, and systolic tissue velocities. Tissue Doppler reveals supernormal peak early diastolic velocity (e′) and annular systolic tissue velocities (Sm) This represents super competent diastolic function characteristic of adaptive remodeling from intense athletic training.

severe remodeling may not reverse in all elite athletes with deconditioning; chamber enlargement persists in 20% of retired elite athletes after 5 years.[24] There is no available evidence that cardiac remodeling from intense physical training results in LV disease or SCD. These measurements of LV mass and muscle distribution have little role in definitively distinguishing physiologic from maladaptive remodeling in athletes. Healthy athletes and healthy nonathletes can be distinguished morphologically and physiologically (Table 2).

LV Diastolic Function. LV diastolic function must be integrated with LV systolic function to comprehensively appraise the athletic heart. Diastolic dysfunction typically precedes systolic dysfunction. Most important, athletic hearts do not accrue diastolic or systolic dysfunction. Trained athletes have enhanced early diastolic LV filling, depicted by increased E-wave velocity and near absence of the A wave, in addition to supernormal medial annular tissue Doppler velocities[25,26] (Figures 1 and 2, Video 1; available at www.onlinejase.com). The proficient diastolic function of endurance-trained athletes allows the left ventricle to relax briskly during extremes in heart rate, allowing the preservation of stroke volume. Cardiac diastolic function is a critical factor in distinguishing adaptive remodeling from disease remodeling.[27,28] This unique ability of diastolic functional parameters to distinguish between health and disease was eloquently shown by abnormal results on tissue Doppler echocardiography in gene mutation–positive patients with HCM independent of phenotypic expression.[29]

LV Systolic Function. LV systolic function is consistently in the normal range among highly trained athletes.[30] Elite cyclists can have LV ejection fractions lower than normal.[31] Our experience with highly conditioned professional basketball players commonly reveals global LV ejection fractions ranging from 45% to 50%, but supernormal tissue Doppler Sm and normal systolic strain and strain rate measurements.

Systolic strain and strain rate imaging of the left ventricle has been used to distinguish maladaptive from physiologic remodeling of the left ventricle. The absence of reductions in global systolic-longitudinal strain and strain rate supports the use of strain rate imaging to assess the physiologic increase in wall thickness associated with athletic training.[32] Simultaneous measurement of systolic and diastolic strain and strain rate in athletes has not been sufficiently documented at this time. Similarly, the effect of endurance training on deformation mechanics, torsion, and the untwisting rate as components of exercise-induced cardiac remodeling in young athletes needs further investigation.[33] LV systolic torsion and peak early diastolic untwisting rate may be important components of exercise-induced cardiac remodeling.[34] Endurance-trained athletes develop biventricular dilation with enhanced diastolic function, whereas strength-trained athletes develop concentric increases in LV wall thickness with diminished diastolic function.[35] Speckle-tracking echocardiography measures LV systolic and diastolic functions in individuals with structural alterations of the heart from intense physical training. LV untwisting increased with exercise more than LV lengthening or expansion.[36] An exercise-induced increase in LV untwisting rate may enhance early

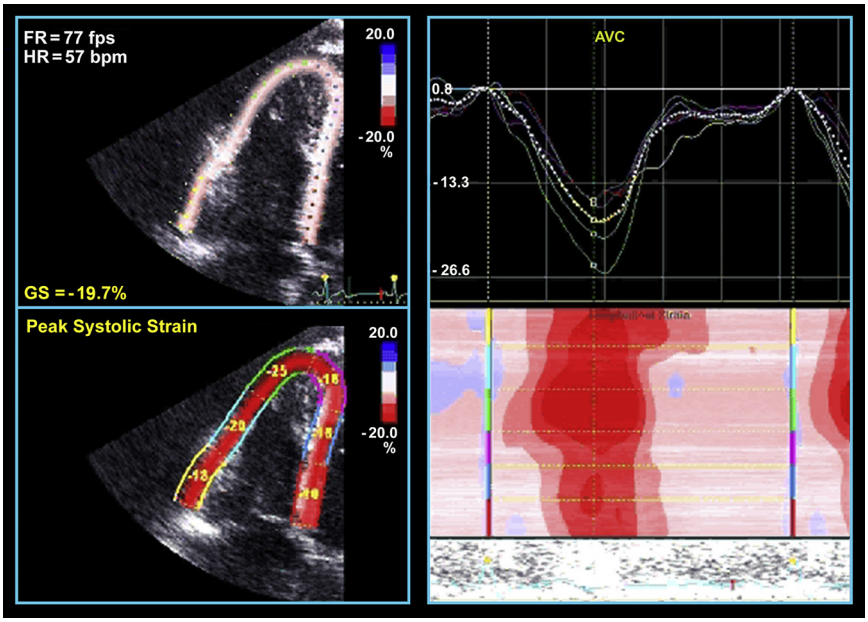

Figure 3 Strain analysis revealing normal peak systolic strain. *GS*, Global strain.

LV diastolic suction, facilitating early LV filling without an increase in left atrial pressure.[37]

Patients with HCM have reductions in longitudinal strain and in the untwisting rate in diastole. The evolving understanding of LV deformation myocardial mechanics is providing incremental information in distinguishing athletic remodeling from disease states such as HCM.

The Right Ventricle

RV remodeling in athletic training is an expectation, because the right ventricle must accept and eject a large volume of blood comparable with that of the remodeled left ventricle. Both the left and right ventricles must augment systolic function to handle the increased blood volume. Endurance-trained athletes have enlarged RV cavities and increased RV wall thickness compared with sedentary controls.[38] RV enlargement parallels LV enlargement in intensely trained athletes.[39] The RV morphologic features in strength-trained athletes do not significantly vary from those in sedentary controls.[40] The morphologic and functional parameters that appear to distinguish endurance- and strength-trained athletes are RV inflow tract diameter, RV end-diastolic area, and tricuspid inflow velocity deceleration time.[40] As expected, in systems that vacillate between normal and abnormal, RV remodeling is heterogeneous across gender, race, and sports discipline.

Physiologic endurance conditioning results in adaptive remodeling of RV structure and function (Video 2; available at www.onlinejase.com). This remodeling may make the distinction from ARVD diagnostically challenging.[41] The echocardiographic features of ARVD reflect the pathologic process of adipose and fibrous infiltration of the RV myocardium, most frequently affecting the RV outflow tract, apex, and inferior basal wall.[42] The distinction between remodeling and RV disease is further distorted by the concern that RV adaptive remodeling associated with prolonged, intense physical training can result in RV pathology. This had led to consideration of possible "exercise-induced RV cardiomyopathy."[43] The potential adverse effects of intense exercise resulting in decreased contractility and altered myocardial substrate–induced arrhythmias[44] beg the question of needing to distinguish between disease states such as ARVD and advanced adaptive remodeling. ARVD has ominous potential, being implicated in 4% to 21% of all cases of SCD.[45] The criterion standard for the diagnosis of ARVD remains poorly defined. Presently, resting measures of RV function such as RV fractional area change, strain, and strain rate are not reflective of RV function reserve, and therefore, low resting RV function as defined by RV fractional area change, S′, strain, and strain rate at rest does not implicate subclinical myocardial damage. RV function may be below normal at rest in highly trained athletes, but there is robust RV functional reserve after provocation with exercise, suggesting that the increased RV and right atrial sizes reflect physiologic remodeling rather than maladaptive changes.[46] Thus, RV contractility, strain, and strain rate at rest do not accurately reflect RV function in highly trained athletes, and exercise testing may be essential to assess RV function. Our need to investigate the impact of high-level training on RV function is related to contradictory data showing that highly trained athletes with RV dilation performing endurance and strength exercises have global strain and strain rate that are preserved or enhanced and that correlate with enhanced S′ and RV fractional area change (Figure 3).[41,47,48] These conflicting data may reflect the heterogeneous response of exercise across variable sports disciplines, volume loading, afterload, and duration of training. Further investigations have identified enhanced RV diastolic function at rest and augmentation of these parameters with intense training. Further investigation of RV diastolic indices, including tissue velocities, strain, and strain rate, at rest and after exercise across a large spectrum of athletes is needed to help distinguish adaptive from maladaptive RV remodeling.

Enhanced contraction of RV free wall segments in athletes compared with regional and global deformation parameters in patients with ARVD may allow the differentiation of physiologic remodeling from disease.[49] Additionally, as stated previously, RV diastolic function, global strain, and response to exercise are key variables separating RV physiologic adaptation from pathology.[50]

Although differences in RV geometric remodeling are observed in endurance- and strength-trained athletes, few reproducible differences in RV cavity deformation or relaxation exist, suggesting that despite remodeling in athletes, RV systolic and diastolic function

Table 3 Relative aortic size predicting rupture

Aortic size index (cm/m^2)	Risk (%)
<2.75	4
2.75–4.25	8
>4.25	20–25

Data from Davies *et al*.[53]

remains normal.[40] It is premature to define "cutoff" values distinguishing remodeling due to athletic training from pathology due to disease. The nonlinear nature of remodeling with heterogeneous phenotypic expression among similarly trained athletes makes this a difficult task. The available data for RV morphologic and functional parameters in relationship to body surface area in athletes are scant. Recent work showed that RV dimensions are greater in athletes than nonathletes. This work provided absolute values for RV morphology and functional parameters in 102 elite endurance-trained athletes and defined the parameters in relationship to body surface area.[41] This is a starting point mandating the need for large, cooperative studies attempting to define normal ranges for athletes across all the various athletic groups.

The Aorta

The remodeling of the aortic root in highly trained athletes is commonly documented. Increased demand resulting in increased stroke volume often results in a larger aortic conduit. Endurance activity results in high-volume aortic flow with modest systolic blood pressure elevation, and strength training results in normal volume aortic flow with profound elevation of systolic blood pressure. There is evidence supporting greater aortic root diameter among strength-trained athletes[51] and refuting data showing the largest aortic dimensions in endurance-trained athletes.[52] The best available measurement in the literature on aortic morphology takes into account aortic dimension and body surface area.[53] The aortic size index is calculated by measuring aortic diameter (centimeters) and dividing by body surface area (square meters). Aortic size index measurements in the athletic population are not well defined. The finding of aortic indexed enlargement should prompt comprehensive evaluation. See Table 3 for guidelines to assess aortic dimensions.

The Atria

A remodeled left atrium is completely normal in the athletic population. Intense athletic training results in increased stroke volume, with subsequent remodeling of all cardiac chambers, including the left and right atria.[54] A more recent study confirmed left atrial remodeling in endurance-trained athletes.[55] The atrial enlargement in trained athletes occurs with preservation of normal LV, RV, left atrial, and right atrial pressures.

Left atrial remodeling in athletes is an expected physiologic adaptive response to exercise training. Left atrial indexed volumes are significantly higher in athletes compared with healthy controls. Doppler tissue imaging is a useful tool to identify improved myocardial diastolic properties in athletes, and speckle-tracking echocardiography elucidates the role of the left atrium in athletic heart remodeling.[56] Left atrial strain and strain rate by two-dimensional speckle-tracking are indices of left atrial function that facilitate distinction of physiologic and pathologic remodeling. Left atrial myocardial deformation is significantly impaired in patients with HCM compared with athletes and healthy controls.[57]

EXERCISE TESTING

Screening stress testing is not useful in asymptomatic patients. LV systolic function response to exercise is more robust in healthy athletes compared with patients with HCM.[58] Maximum oxygen uptake is increased in athletes as a result of upregulation of cardiopulmonary delivery of oxygen and skeletal muscle use of oxygen. The major difference between athletes and nonathletes is the ability to increase cardiac output. This is attributable to enhanced stroke volume.[59] Doppler tissue imaging measures have demonstrated augmentation of systolic and diastolic functions in elite athletes.[60]

SEX

Athletic cardiac remodeling exhibits no gender bias; it occurs in both sexes and is of similar but not equivalent magnitude in male and female athletes. The degree of individualized physiologic remodeling is less pronounced in women matched to men, even when corrections are made to take account of slighter lower body surface areas in women.[61,62]

RACE

Race is an important element of cardiac remodeling, with individual differences in wall thickness between white and black athletes. This distinction transcends sex, with black female athletes having greater wall thickness and LV mass than white female athletes.[63] Black athletes show a more prominent pattern of LV remodeling compared with whites, characterized by greater increases in LV wall thickness, mass, and relative wall thickness but similar cavity size and volume.[64] Individual differences in the degree of wall thickening suggest an indefinite etiologic factor associated with the black race that augments physiologic wall thickening in response to the volume burden associated with intense athletic training. Athletes of all races are expected to have preservation of normal or hypernormal cardiac myocyte function despite morphologic changes.

MULTIMODALITY IMAGING IN ATHLETE'S HEART

Echocardiography provides a robust understanding of cardiac pathophysiology. High temporal resolution enables the assessment of myocardial function at rest and during exercise regionally and globally. Echocardiography is the best imaging modality because it allows the simultaneous evaluation of cardiac morphology and diastolic function. This allows individualization of the global morphophysiologic state.

Cardiac magnetic resonance imaging (MRI) has excellent spatial resolution and image quality that is not influenced by body habitus, permitting precise measurements of cardiac chamber size, myocardial mass, and systolic function. This has particular benefit for the irregularly shaped right ventricle.[65] Cardiac MRI has incremental value in distinguishing athletic heart and HCM in selected cases because of its superiority in identifying segmental increase in LV wall thickness in the anterolateral free wall and apex.[66] In the severe phenotypic expression of ARVD, all imaging modalities are likely to identify the disease.[67] An important role of cardiac MRI in the athletic population lies in its ability to detect early and subtle cases of ARVD that are otherwise insensitive to diagnosis. Cardiac MRI can uniquely detect regional and diastolic dysfunction, which may represent early manifestations of ARVD.[68] Cardiac MRI also allows the detection of LV involvement in

ARVD[69] and is a useful technique for the noninvasive identification of anomalous coronary arteries and their anatomic course.[70]

Radionuclide ventriculography attempts to provide measures of ventricular systolic function that do not rely on geometric assumptions and has been used to assess function with exercise. This imaging modality is limited by cost and radiation exposure and has little value given the alternative modalities for measuring systolic function.

Positron emission tomography imaging has been used to evaluate perfusion reserve in athletes. Supine bicycle exercise with positron emission tomographic scanning has demonstrated that increased myocardial mass does not impede blood supply in athletes. Positron emission tomography through tissue characterization in the future may allow the identification of subtle changes in myocardial and interstitial composition, ascertaining whether the morphologic changes observed in athletes are physiologic or pathologic.[71]

Cardiac computed tomography allows the evaluation of coronary artery disease and possesses superb spatial resolution for anatomic definition. Cardiac computed tomography has been limited to use in older athletes to exclude coronary artery disease. The amount of radiation exposure limits its use to assess cardiac function.

PARTING THOUGHTS

The accuracy of Doppler echocardiography is time tested and validated. Doppler echocardiography allows one to sensitively and specifically distinguish adaptive from maladaptive remodeling. It is clinically effective and cost effective. Echocardiography is available throughout the world and allows the simultaneous assessment of morphology and physiology.

CASE EXAMPLES

The first example is of a National Basketball Association player who presented for a second opinion because he was disqualified from sports participation. His disqualification was based on a diagnosis of LV noncompaction cardiomyopathy. The diagnostic predicament was tackled with an integrated consideration of history, 12-lead electrocardiography, and echocardiography. The personal and family histories were unremarkable. The 12-lead electrocardiogram revealed increased voltage and early repolarization. These electrocardiographic changes were compatible with an athletically trained black athlete. The echocardiogram was intriguing (Figures 1–3, Videos 1–6; available at www.onlinejase.com). The morphologic changes were obvious, with remodeling and enlargement of all cardiac chambers, increased LV wall thickness, and evidence of marked apical trabeculations and chords and a bilayered myocardium. The calculated LV ejection fraction was 55%. In this case, the morphologic changes are marked and give pause for the physician trying to separate a healthy heart from a disease state.

This patient had cardiac remodeling with increased wall thickness (12 mm) and enlargement of all cardiac chambers. Tissue Doppler echocardiography revealed supernormal early diastolic relaxation ($e' > 17$ cm/sec) and supernormal peak systolic tissue velocity ($Sm > 10$ cm/sec). Additionally, global longitudinal strain and strain rate were normal. The patient achieved 22 metabolic equivalents on the Bruce protocol.

Integrating the patient's morphology, physiology, myocardial mechanics, and exercise testing results led to a conclusion of athletic heart superimposed on LV noncompaction morphology with normal LV systolic function, the latter being a morphologic finding and not representative of a disease state. The patient was cleared to play professional basketball with a recommendation for follow-up in 1 year.

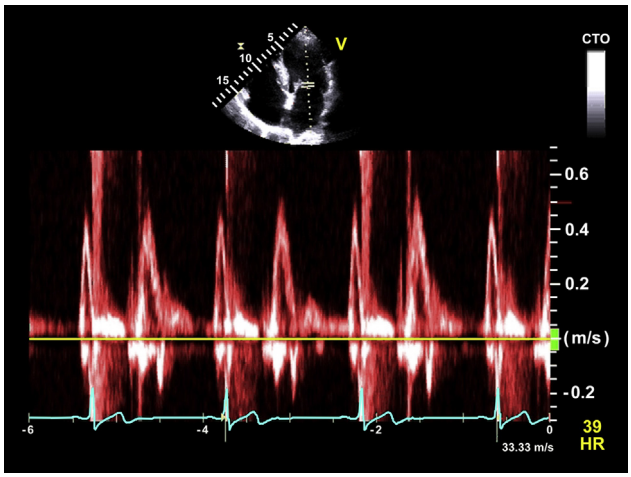

Figure 4 Pulsed-wave Doppler of mitral inflow. The heart rate was 39 beats/min, and there was a normal period of diastasis. Despite intense athletic training, this 74-year-old man had less proficient diastolic filling and a dominant A wave.

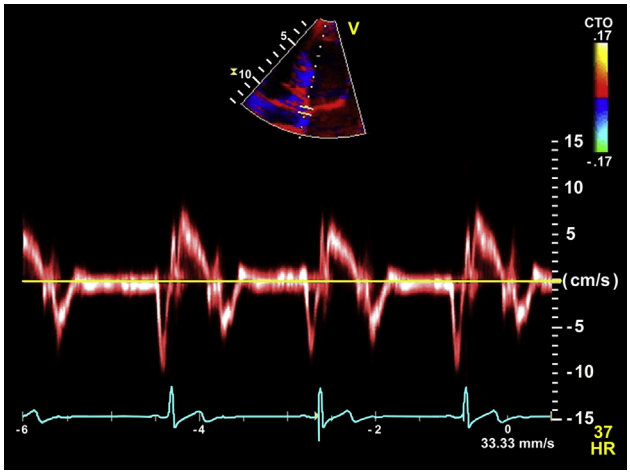

Figure 5 Medial annular tissue Doppler with a grade 2/4 diastolic pattern but normal LV filling pressure ($E/e' = 7$). The remodeled left atrium has preserved LV filling pressure, consistent with athletic heart.

The second example, which shows the distinction between athletic training–induced cardiac remodeling and structural heart disease, is of a 74-year-old white man referred for evaluation of a cardiomyopathy and the need for an implantable cardioverter-defibrillator. The patient had an episode of fatigue on a 40-mile bike ride. A previous cardiac catheterization test revealed normal coronary arteries. The personal history revealed that the patient rode his bike an average of 40 mi/d and recently had ridden from the west coast to the east coast of the United States in 2 months. The patient's family history was unremarkable. Twelve-lead electrocardiography revealed marked sinus bradycardia with heart rates ranging from 30 to 39 beats/min, increased voltage, and early repolarization.

Echocardiography revealed obvious chamber enlargement (Figures 4–6, Videos 7–11; available at www.onlinejase.com). LV and RV systolic function appeared depressed visually. No significant valvular abnormalities were identified. Diastolic function revealed that e' and E/e' were normal, as was peak annular systolic tissue velocity. Calculated LV ejection fraction and RV fractional area change

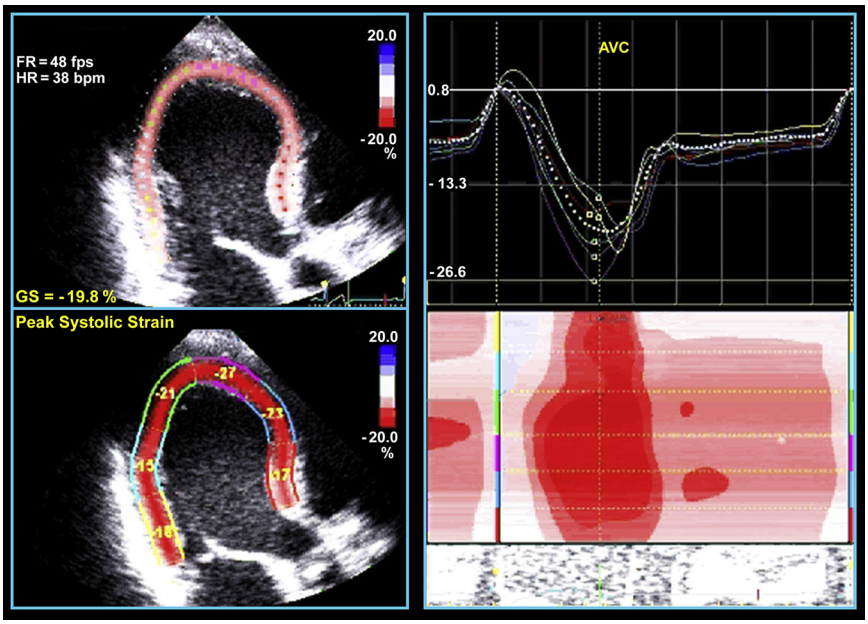

Figure 6 Strain analysis revealing normal mechanical function with peak systolic strain equal to −19.8%. *GS*, Global strain.

were normal, as were the results of strain analysis. This 74-year-old man reached a workload of 19 metabolic equivalents on exercise testing. Echocardiographic analysis demonstrated extreme cardiac remodeling from chronic, intense physical training. The left ventricle raised concern for dilated cardiomyopathy, and the RV dimensions raised concern for RV cardiomyopathy. The analysis of systole, diastole, LV and RV mechanics, and stress testing identified physiologic remodeling of the left and right ventricles.

These two examples embody the degree and magnitude of cardiac remodeling associated with intense physical training. Although morphologic changes associated with intense exercise training can create a diagnostic conundrum when attempting to distinguish athletic remodeling from pathologic structural heart disease, functional myocardial imaging generally permits precise assessment of function and separation of health from disease.

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SUPPLEMENTARY DATA

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REFERENCES

1. Pluim BM, Zwinderman AH, van der Laarse A, van der Wall EE. The athlete's heart. A meta-analysis of cardiac structure and function. Circulation 2000;101:336-44.
2. D'Andrea A, Limongelli G, Caso P, Sarubbi B, Della Pietra A, Brancaccio P, et al. Association between left ventricular structure and cardiac performance during effort in two morphological forms of athlete's heart. Int J Cardiol 2002;86:177-84.
3. Fagard RH. Impact of different sports and training on cardiac structure and function. Cardiol Clin 1997;15:397-412.
4. Mickelson JK, Byrd BF 3rd, Bouchard A, Botvinick EH, Schiller NB. Left ventricular dimensions and mechanics in distance runners. Am Heart J 1986;112:1251-6.
5. Loscalzo J, Barabasi AL. Systems biology and the future of medicine. Wiley Interdiscip Rev Syst Biol Med 2011;3:619-27.
6. Baggish AL, Hutter AM Jr., Wang F, Yared K, Weiner RB, Kupperman E, et al. Cardiovascular screening in college athletes with and without electrocardiography: a cross-sectional study. Ann Intern Med 2010;152:269-75.
7. Galper BZ, Moran A, Coxson PG, Pletcher MJ, Heidenreich P, Lazar LD, et al. Using stress testing to guide primary prevention of coronary heart disease among intermediate-risk patients: a cost-effectiveness analysis. Circulation 2012;125:260-70.
8. Uberoi A, Stein R, Perez MV, Freeman J, Wheeler M, Dewey F, et al. Interpretation of the electrocardiogram of young athletes. Circulation 2011;124:746-57.
9. Wellens HJ, Gorgels AP. How important is the electrocardiogram in protecting and guiding the athlete? Circulation 2011;124:669-71.
10. Harmon KG, Asif IM, Klossner D, Drezner JA. Incidence of sudden cardiac death in National Collegiate Athletic Association athletes. Circulation 2011;123:1594-600.
11. Marijon E, Tafflet M, Celermajer DS, Dumas F, Perier MC, Mustafic H, et al. Sports-related sudden death in the general population. Circulation 2011;124:672-81.
12. Maron BJ, Haas TS, Ahluwalia A, Rutten-Ramos SC. Incidence of cardiovascular sudden deaths in Minnesota high school athletes. Heart Rhythm 2013;10:374-7.
13. Maron BJ, Carney KP, Lever HM, Lewis JF, Barac I, Casey SA, et al. Relationship of race to sudden cardiac death in competitive athletes with hypertrophic cardiomyopathy. J Am Coll Cardiol 2003;41:974-80.
14. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation 2009;119:1085-92.
15. D'Andrea A, D'Andrea L, Caso P, Scherillo M, Zeppilli P, Calabrò R. The usefulness of Doppler myocardial imaging in the study of the athlete's heart and in the differential diagnosis between physiological and pathological ventricular hypertrophy. Echocardiography 2006;23:149-57.

16. Roeske WR, O'Rourke RA, Klein A, Leopold G, Karliner JS. Noninvasive evaluation of ventricular hypertrophy in professional athletes. Circulation 1976;53:286-91.
17. Pelliccia A, Culasso F, Di Paolo FM, Maron BJ. Physiologic left ventricular cavity dilatation in elite athletes. Ann Int Med 1999;130:23-31.
18. Pelliccia A, Maron BJ, Spataro A, Proschan MA, Spirito P. The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. N Engl J Med 1991;324:295-301.
19. Baggish AL, Yared K, Weiner RB, Wang F, Demes R, Picard MH, et al. Differences in cardiac parameters among elite rowers and subelite rowers. Med Sci Sports Exerc 2010;42:1215-20.
20. Grossman W, Jones D, McLaurin LP. Wall stress and patterns of hypertrophy in the human left ventricle. J Clin Invest 1975;56:56-64.
21. Katz AM. Cardiomyopathy of overload. A major determinant of prognosis in congestive heart failure. N Engl J Med 1990;322:100-10.
22. Milani RV, Lavie CJ, Mehra MR, Ventura HO, Kurtz JD, Messerli FH. Left ventricular geometry and survival in patients with normal left ventricular ejection fraction. Am J Cardiol 2006;97:959-63.
23. Douglas PS, O'Toole ML, Katz SE, Ginsburg GS, Hiller WD, Laird RH. Left ventricular hypertrophy in athletes. Am J Cardiol 1997;80:1384-8.
24. Pelliccia A, Maron BJ, De Luca R, Di Paolo FM, Spataro A, Culasso F. Remodeling of left ventricular hypertrophy in elite athletes after long-term deconditioning. Circulation 2002;105:944-9.
25. D'Andrea A, Cocchia R, Riegler L, Scarafile R, Salerno G, Gravino R, et al. Left ventricular myocardial velocities and deformation indexes in top-level athletes. J Am Soc Echocardiogr 2010;23:1281-8.
26. Claessens PJ, Claessens CW, Claessens MM, Claessens MC, Claessens JE. Supernormal left ventricular diastolic function in triathletes. Tex Heart Inst J 2001;28:102-10.
27. Caso P, D'Andrea A, Galderisi M, Liccardo B, Severino S, De Simone L, et al. Pulsed Doppler tissue imaging in endurance athletes: relation between left ventricular preload and myocardial regional diastolic function. Am J Cardiol 2000;85:1131-6.
28. Möckel M, Störk T. Diastolic function in various forms of left ventricular hypertrophy: contribution of active Doppler stress echo. Int J Sports Med 1996;17(Suppl 3):S184-90.
29. Nagueh SF, Bachinski LL, Meyer D, Hill R, Zoghbi WA, Tam JW, et al. Tissue Doppler imaging consistently detects myocardial abnormalities in patients with hypertrophic cardiomyopathy and provides a novel means for an early diagnosis before and independently of hypertrophy. Circulation 2001;104:128-30.
30. Gilbert CA, Nutter DO, Felner JM, Perkins JV, Heymsfield SB, Schlant RC. Echocardiographic study of cardiac dimensions and function in the endurance-trained athlete. Am J Cardiol 1977;40:528-33.
31. Abergel E, Chatellier G, Hagege AA, Oblak A, Linhart A, Ducardonnet A, et al. Serial left ventricular adaptations in world-class professional cyclists: implications for disease screening and follow-up. J Am Coll Cardiol 2004;44:144-9.
32. Saghir M, Areces M, Makan M. Strain rate imaging differentiates hypertensive cardiac hypertrophy from physiologic cardiac hypertrophy (athlete's heart). J Am Soc Echocardiogr 2007;20:151-7.
33. Sengupta PP, Tajik AJ, Chandrasekaran K, Khandheria BK. Twist mechanics of the left ventricle: principles and application. JACC Cardiovasc Imaging 2008;1:366-76.
34. Weiner RB, Hutter AM Jr., Wang F, Kim J, Weyman AE, Wood MJ, et al. The impact of endurance exercise training on left ventricular torsion. JACC Cardiovasc Imaging 2010;3:1001-9.
35. Baggish AL, Wang F, Weiner RB, Elinoff JM, Tournoux F, Boland A, et al. Training-specific changes in cardiac structure and function: a prospective and longitudinal assessment of competitive athletes. J Appl Physiol 1985;2008:1121-8.
36. Simsek Z, Hakan Tas M, Degirmenci H, Gokhan Yazıcı A, Ipek E, Duman H, et al. Speckle tracking echocardiographic analysis of left ventricular systolic and diastolic functions of young elite athletes with eccentric and concentric type of cardiac remodeling. Echocardiography 2013;30:1202-8.
37. Notomi Y, Martin-Miklovic MG, Oryszak SJ, Shiota T, Deserranno D, Popovic ZB, et al. Enhanced ventricular untwisting during exercise: a mechanistic manifestation of elastic recoil described by Doppler tissue imaging. Circulation 2006;113:2524-33.
38. Hauser AM, Dressendorfer RH, Vos M, Hashimoto T, Gordon S, Timmis GC. Symmetric cardiac enlargement in highly trained endurance athletes: a two-dimensional echocardiographic study. Am Heart J 1985;109:1038-44.
39. Scharf M, Brem MH, Wilhelm M, Schoepf UJ, Uder M, Lell MM. Cardiac magnetic resonance assessment of left and right ventricular morphologic and functional adaptations in professional soccer players. Am Heart J 2010;159:911-8.
40. Pagourelias ED, Kouidi E, Efthimiadis GK, Deligiannis A, Geleris P, Vassilikos V. Right atrial and ventricular adaptations to training in male Caucasian athletes: an echocardiographic study. J Am Soc Echocardiogr 2013;26:1344-52.
41. Oxborough D, Sharma S, Shave R, Whyte G, Birch K, Artis N, et al. The right ventricle of the endurance athlete: the relationship between morphology and deformation. J Am Soc Echocardiogr 2012;25:263-71.
42. Marcus FI, Fontaine GH, Guiraudon G, Frank R, Laurenceau JL, Malergue C, et al. Right ventricular dysplasia: a report of 24 adult cases. Circulation 1982;65:384-98.
43. Heidbüchel H, Hoogsteen J, Fagard R, Vanhees L, Ector H, Willems R, et al. High prevalence of right ventricular involvement in endurance athletes with ventricular arrhythmias. Role of an electrophysiologic study in risk stratification. Eur Heart J 2003;24:1473-80.
44. Aziz EF, Kukin M, Javed F, Musat D, Nader A, Pratap B, et al. Right ventricular dysfunction is a strong predictor of developing atrial fibrillation in acutely decompensated heart failure patients, ACAP-HF data analysis. J Card Fail 2010;16:827-34.
45. Maron BJ, Thompson PD, Ackerman MJ, Balady G, Berger S, Cohen D, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. Circulation 2007;115. 1643–455.
46. La Gerche A, Burns AT, D'Hooge J, Macisaac AI, Heidbüchel H, Prior DL. Exercise strain rate imaging demonstrates normal right ventricular contractile reserve and clarifies ambiguous resting measures in endurance athletes. J Am Soc Echocardiogr 2012;25:253-62.
47. D'Andrea A, Caso P, Bossone E, Scarafile R, Riegler L, Di Salvo G, et al. Right ventricular myocardial involvement in either physiological or pathological left ventricular hypertrophy: an ultrasound speckle-tracking two-dimensional strain analysis. Eur J Echocardiogr 2010;11:492-500.
48. D'Andrea A, Riegler L, Golia E, Cocchia R, Scarafile R, Salerno G, et al. Range of right heart measurements in top-level athletes: the training impact. Int J Cardiol 2013;164:48-57.
49. Bauce B, Frigo G, Benini G, Michieli P, Basso C, Folino AF, et al. Differences and similarities between arrhythmogenic right ventricular cardiomyopathy and athlete's heart adaptations. Br J Sports Med 2010;44:148-54.
50. Pagourelias ED, Efthimiadis GK, Parcharidou DG, Gossios TD, Kamperidis V, Karoulas T, et al. Prognostic value of right ventricular diastolic function indices in hypertrophic cardiomyopathy. Eur J Echocardiogr 2011;12:809-17.
51. D'Andrea A, Cocchia R, Riegler L, Scarafile R, Salerno G, Gravino R, et al. Aortic root dimensions in elite athletes. Am J Cardiol 2010;105:1629-34.
52. Pelliccia A, Di Paolo FM, De Blasiis E, Quattrini FM, Pisicchio C, Guerra E, et al. Prevalence and clinical significance of aortic root dilation in highly trained competitive athletes. Circulation 2010;122:698-706.
53. Davies RR, Gallo A, Coady MA, Tellides G, Botta DM, Burke B, et al. Novel measurement of relative aortic size predicts rupture of thoracic aortic aneurysms. Ann Thorac Surg 2006;81:169-77.
54. Nistri S, Galderisi M, Ballo P, Olivotto I, D'Andrea A, Pagliani L, et al. Determinants of echocardiographic left atrial volume: implications for normalcy. Eur J Echocardiogr 2011;12:826-33.

55. D'Andrea A, Riegler L, Cocchia R, Scarafile R, Salerno G, Gravino R, et al. Left atrial volume index in highly trained athletes. Am Heart J 2010;159:1155-61.
56. D'Ascenzi F, Cameli M, Zacà V, Lisi M, Santoro A, Causarano A, et al. Supernormal diastolic function and role of left atrial myocardial deformation analysis by 2D speckle tracking echocardiography in elite soccer players. Echocardiography 2011;28:320-6.
57. Gabrielli L, Enríquez A, Córdova S, Yáñez F, Godoy I, Corbalán R. Assessment of left atrial function in hypertrophic cardiomyopathy and athlete's heart: a left atrial myocardial deformation study. Echocardiography 2012;29:943-9.
58. Firoozi S, Sharma S, McKenna WJ. Risk of competitive sport in young athletes with heart disease. Heart 2003;89:710-4.
59. Vella CA, Robergs RA. A review of the stroke volume response to upright exercise in healthy subjects. Br J Sports Med 2005;39:190-5. Erratum in: Br J Sports Med2005;39:688.
60. D'Andrea A, Caso P, Galderisi M, Di Maggio D, Cicala S, D'Andrea L, et al. Assessment of myocardial response to physical exercise in endurance competitive athletes by pulsed Doppler tissue imaging. Am J Cardiol 2001;87:1226-30. A8.
61. Pelliccia A, Maron BJ, Culasso F, Spataro A, Caselli G. Athlete's heart in women. Echocardiographic characterization of highly trained elite female athletes. JAMA 1996;276:211-5.
62. Whyte GP, George K, Nevill A, Shave R, Sharma S, McKenna WJ. Left ventricular morphology and function in female athletes: a meta-analysis. Int J Sports Med 2004;25:380-3.
63. Rawlins J, Carre F, Kervio G, Papadakis M, Chandra N, Edwards C, et al. Ethnic differences in physiological cardiac adaptation to intense physical exercise in highly trained female athletes. Circulation 2010;121:1078-85.
64. Di Paolo FM, Schmied C, Zerguini YA, Junge A, Quattrini F, Culasso F, et al. The athlete's heart in adolescent Africans: an electrocardiographic and echocardiographic study. J Am Coll Cardiol 2012;59:1029-36.
65. Angelini P, Vidovich MI, Lawless CE, Elayda MA, Lopez JA, Wolf D, et al. Preventing sudden cardiac death in athletes: in search of evidence-based, cost-effective screening. Tex Heart Inst J 2013;40:148-55.
66. Rickers C, Wilke NM, Jerosch-Herold M, Casey SA, Panse P, Panse N, et al. Utility of cardiac magnetic resonance imaging in the diagnosis of hypertrophic cardiomyopathy. Circulation 2005;112:855-61.
67. Sen-Chowdhry S, Prasad SK, Syrris P, Wage R, Ward D, Merrifield R, et al. Cardiovascular magnetic resonance in arrhythmogenic right ventricular cardiomyopathy revisited: comparison with task force criteria and genotype. J Am Coll Cardiol 2006;48:2132-40.
68. Bomma C, Dalal D, Tandri H, Prakasa K, Nasir K, Roguin A, et al. Regional differences in systolic and diastolic function in arrhythmogenic right ventricular dysplasia/cardiomyopathy using magnetic resonance imaging. Am J Cardiol 2005;95:1507-11.
69. Corrado D, Basso C, Thiene G, McKenna WJ, Davies MJ, Fontaliran F, et al. Spectrum of clinicopathologic manifestations of arrhythmogenic right ventricular cardiomyopathy/dysplasia: a multicenter study. J Am Coll Cardiol 1997;30:1512-20.
70. McConnell MV, Ganz P, Selwyn AP, Li W, Edelman RR, Manning WJ. Identification of anomalous coronary arteries and their anatomic course by magnetic resonance coronary angiography. Circulation 1995;92:3158-62.
71. La Gerche A, Taylor AJ, Prior DL. Athlete's heart: the potential for multimodality imaging to address the critical remaining questions. J Am Coll Cardiol Img 2009;2:350-63.