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.)

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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