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Athlete's Heart and Cardiovascular Care of the Athlete Scientific and Clinical Update

Aaron L. Baggish, MD; Malissa J. Wood, MD

*"The physiologic capabilities of the heart are enormous, and in judging the effect of any undue exertion on it, we must not regard the murmurs of the irregularity alone, but must also consider carefully, the way in which the heart is doing its work, its strength, as shown by its ability to maintain proper arterial tension, and its recuperative power. As with other muscles, not size but quality tells in the long run."*¹

—Eugene Darling

The heart of the athlete has intrigued clinicians and scientists for more than a century. Early investigations in the late 1800s and early 1900s documented cardiac enlargement and bradyarrhythmias in individuals with above-normal exercise capacity and no attendant signs of cardiovascular disease. Since that time, scientific understanding of the association between sport participation and specific cardiac abnormalities has paralleled advances in cardiovascular diagnostic techniques. It is now well established that repetitive participation in vigorous physical exercise results in significant changes in myocardial structure and function. Recent increases in the popularity of recreational exercise and competitive athletics have led to a growing number of individuals exhibiting this phenomenon. This review provides an up-to-date summary of the science of cardiac remodeling in athletes and an overview of common clinical issues that are encountered in the cardiovascular care of the athlete.

Historical Perspective: Past to Present

Initial reports describing cardiac enlargement in athletes date back to the late 1890s. In Europe, the Swedish clinician Henschen² used the rudimentary yet elegant physical examination skills of auscultation and percussion to demonstrate increased cardiac dimensions in elite Nordic skiers. Similar observations were made during the same year by Eugene Darling¹ of Harvard University in university rowers. In the early 1900s, Paul Dudley White³ studied radial pulse rate and pattern among Boston Marathon competitors, and was the first to report marked resting sinus bradycardia in long-distance runners.⁴ Early chest radiography work confirmed the physical examination findings of Darling and Henschen by showing global cardiac enlargement in trained athletes.^{5–7} The subsequent development of ECG enabled widespread

study of electric activity in the heart of the trained athlete.^{8–14} In addition to the morphological patterns of cardiac hypertrophy, bradyarrhythmias¹⁵ and tachyarrhythmias¹⁶ were observed in healthy exercise-trained individuals. The development and rapid dissemination of 2-dimensional echocardiography led to important further advances in our understanding of the athlete's heart. Descriptions of ventricular chamber enlargement, myocardial hypertrophy, and atrial dilation have led to a more comprehensive understanding of the athlete's heart. Most recently, advanced echocardiographic techniques and magnetic resonance imaging have begun to clarify important functional adaptations that accompany previously reported structural characteristics of the athlete's heart.

The significance of cardiac enlargement in athletes has been debated since the time Darling and Henschen made their initial observations. Although both investigators speculated that their findings represented beneficial adaptations to exercise, this view was not universally accepted. It was postulated as early as 1902 that cardiac enlargement in athletes is a form of overuse pathology, and that prolonged participation in sport could lead to premature cardiovascular system collapse.¹⁷ This line of thinking underscores the concept of the athlete's heart syndrome, a term often applied to the athletic patient who presents with subjective symptoms or an abnormal cardiovascular finding. This concept has resurfaced numerous times over the last 110 years of scientific inquiry despite the fact that there is no clear evidence to substantiate its validity. Importantly, a recent study of Olympic-caliber Italian athletes (n=114) demonstrated no deterioration in left ventricular (LV) function or occurrence of cardiovascular events over an extended period (8.6±3 years) of intense training.¹⁸ Although the debate is ongoing¹⁹ and more prospective, long-term studies are needed, the modern view of the athlete's heart implicates adaptive physiology, not pre-clinical disease.

In the present era, participation in competitive sport and/or vigorous recreational exercise continues to gain in popularity in the United States and other countries. Factors such as the documented health benefits of regular physical exercise, the increasing availability of community-based athletic programs, and growing numbers of open-enrollment sport events (ie, community-based road-running races) underlie this popularity growth. In the face of the growing obesity epidemic,

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cardiovascular practitioners should be encouraged to support this trend both with individual patients and at the community level. However, the increasing interest in sport participation will likely be paralleled by increases in the number of people with features of athlete heart. Thus, the practicing cardiovascular clinician may find it increasingly important to possess a basic knowledge of this subject.

Exercise-Induced Cardiac Remodeling

Overview of Relevant Physiology

Numerous superb reviews of clinically relevant exercise physiology are available^{20–22}; thus, only key aspects relevant to cardiac remodeling are reviewed here. There is a direct relationship between exercise intensity (external work) and the body's demand for oxygen. This oxygen demand is met by increasing pulmonary oxygen uptake ($\dot{V}O_2$). The cardiovascular system is responsible for transporting oxygen-rich blood from the lungs to the skeletal muscles, a process quantified as cardiac output (liters per minute). The Fick equation ($\text{cardiac output} = \dot{V}O_2 \times (\text{arterial} - \text{venous } O_2) / \Delta$) can be used to quantify the relationship between cardiac output and $\dot{V}O_2$. In the healthy human, there is a direct and inviolate 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. Coordinated autonomic nervous system function, characterized by rapid and sustained parasympathetic withdrawal coupled with sympathetic activation, is required for this to occur. Heart rate in the athlete may range from <40 bpm at rest to >200 bpm in a young maximally exercising athlete. Heart rate increase is responsible for the majority of cardiac output augmentation during exercise. Maximal heart rate varies innately among individuals, decreases with age,²³ and does not increase with exercise training.²⁴

In contrast, stroke volume both at rest and during exercise may increase significantly with prolonged exercise training. Cardiac chamber enlargement and the accompanying ability to generate a large stroke volume are direct results of exercise training and cardiovascular hallmarks of the endurance-trained athlete. Stroke volume rises during exercise as a result of increases in ventricular end-diastolic volume and, to a lesser degree, sympathetically mediated reduction in end-systolic volume (particularly during upright exercise).²¹ Left ventricular end-diastolic volume is determined by diastolic filling, a complex process that is affected by a variety of variables, including heart rate, intrinsic myocardial relaxation, ventricular compliance, ventricular filling pressures, atrial contraction, and extracardiac mechanical factors such as pericardial and pulmonary constraints. At the present time, to what degree each of these factors contributes to stroke volume augmentation during exercise remains uncertain.

Hemodynamic conditions, specifically changes in cardiac output and peripheral vascular resistance, vary widely across sporting disciplines. Although some overlap exists, exercise activity can be segregated into 2 forms with defining hemodynamic differences. Isotonic exercise, further referred to as endurance exercise, involves sustained elevations in cardiac output, with normal or reduced peripheral vascular resistance.

Such activity represents primarily a volume challenge for the heart that affects all 4 chambers. This form of exercise underlies activities such as long-distance running, cycling, rowing, and swimming. In contrast, isometric exercise, further referred to as strength training, involves activity characterized by increased peripheral vascular resistance and normal or only slightly elevated cardiac output. This increase in peripheral vascular resistance causes transient but potentially marked systolic hypertension and LV afterload. Strength training is the dominant form of exercise in activities such as weightlifting, track and field throwing events, and American-style football. Many sports, including popular team-based activities such as soccer, lacrosse, basketball, hockey, and field hockey, involve significant elements of both endurance and strength exercise. As discussed later, sport-specific hemodynamic conditions may play an important role in cardiac remodeling.

The Left Ventricle

The impact of exercise training on LV structure has been the topic of extensive study. Early studies with ECG demonstrated a high prevalence of increased cardiac voltage suggestive of LV enlargement in trained athletes.^{9,25} Subsequent work with 2-dimensional echocardiography confirmed underlying LV hypertrophy and dilation.²⁶ Italian physician-scientists have contributed a great deal to our understanding of LV structure in athletes using data derived from their long-standing preparticipation screening program. Pelliccia et al²⁷ reported echocardiographic LV end-diastolic cavity dimensions in a large group (n=1309) of Italian elite athletes. This cohort was made up predominantly of male athletes (73%) and included individuals from 38 different sports. Left ventricular end-diastolic diameters varied widely, from 38 to 66 mm in women (mean, 48 mm) and from 43 to 70 mm in men (mean, 55 mm). Importantly, LV end-diastolic diameter was ≥ 54 mm in 45% and >60 mm in 14% of the cohort. Markedly dilated LV chambers (>60 mm) were most common in athletes with higher body mass and those participating in endurance sports (cycling, cross-country skiing, and canoeing).

Pelliccia et al²⁸ have also reported echocardiographic measurements of LV wall thicknesses among 947 elite Italian athletes. Within this cohort, a small but significant percentage of athletes (1.7%) had LV wall thicknesses ≥ 13 mm, and all of these individuals had concomitant LV cavity dilation. Sharma et al²⁹ also reported a low incidence (0.4%) of LV wall thickness >12 mm among 720 elite junior athletes and confirmed that increased LV wall thickness is associated with increased chamber size in young athletes. It must be emphasized that LV wall thickness in excess of 13 mm is a rare finding in healthy athletes. This finding should prompt consideration of pathological hypertrophy, and may serve as an indication for further diagnostic assessment. However, as reflected by the above data, a very small but significant number of healthy, highly trained individuals have wall thickness values in the 13- to 15-mm range. This finding may be particularly common among elite athletes who engage in the highest level of exercise training.³⁰ Furthermore, it has been consistently shown that the most marked LV hypertro-

phy occurs in athletes with relatively large body size, and those of Afro-Caribbean descent. As such, careful interpretation of LV hypertrophy in athletes, particularly with respect to differentiating adaptive from pathological hypertrophy, requires consideration of body size and ethnicity.

The notion that endurance-based exercise and strength-based exercise lead to distinctly different changes in LV structure was first proposed by Morganroth et al in 1975.³¹ That study compared M-mode echocardiographic LV measurements in wrestlers (strength training), swimmers (endurance training), and sedentary control subjects and found significant differences across these 3 groups. Specifically, athletes exposed to strength training demonstrated concentric LV hypertrophy, whereas individuals exposed to endurance training demonstrated eccentric LV enlargement. This study led to the concept of sport-specific cardiac remodeling, often referred to as the Morganroth hypothesis. Although data have been presented that refute the concept of sport-specific LV remodeling,^{32–34} the majority of cross-sectional data and recent longitudinal work support Morganroth's original findings.³⁵ In our experience, the magnitude of eccentric hypertrophy that results from endurance training is typically more pronounced than the concentric hypertrophy that accompanies strength training, although, rarely, extreme forms of both subtypes can be encountered. The interested reader is referred to a recent comprehensive review of this topic by Naylor and colleagues.³⁶

Exercise-induced adaptations in LV function have also been studied. Numerous investigators have examined resting LV systolic function in athletes using cross-sectional, sedentary control study designs.^{37–40} These studies and a large meta-analysis show that LV ejection fraction is generally normal among athletes,⁴¹ although at least 1 study of 147 cyclists participating in the Tour de France found that 17 (11%) had a calculated LV ejection fraction $\leq 52\%$.⁴² Such results suggest that healthy endurance athletes may occasionally demonstrate borderline or mildly reduced LV ejection fractions at rest. Recent advances in functional myocardial imaging, including tissue Doppler echocardiography and strain echocardiography, have also suggested that exercise training may lead to changes in LV systolic function that are not detected by assessment of a global index like LV ejection fraction.^{43–45} The importance of these findings with respect to our understanding of exercise physiology and for differentiating athletic from pathological remodeling is an area of active investigation.

Left ventricular diastolic function has also been extensively evaluated in trained athletes. Most studies of diastolic function in athletes have used conventional 2-dimensional (transmitral) and tissue Doppler echocardiography. It is now well recognized that endurance exercise training leads to enhanced early diastolic LV filling as assessed by E-wave velocity and mitral annular/LV tissue velocities.^{30,46–50} It is likely that improved LV diastolic function, particularly the ability of the LV to relax briskly at high heart rates, is an essential mechanism of stroke volume preservation during exercise. There are sparse data examining diastolic function in strength-trained athletes, but a longitudinal study suggested that the concentric LV hypertrophy associated with strength

training is accompanied by either unchanged or relative impairment of LV relaxation.³⁵

The majority of data characterizing LV structure and function in athletes come from cross-sectional studies in which participants are characterized at a single time point. This approach is popular because of its relative logistical ease, but it does not permit definitive conclusions about important issues, including the temporal nature, permanence/long-term implications, and dose-response relationships between exercise and cardiac remodeling. In contrast, longitudinal studies in which measurements are made serially and exercise exposure can be controlled, or at least accurately measured, provide an opportunity to address these areas of uncertainty. Representative prior longitudinal work is summarized in Table 1. Unfortunately, such studies constitute only a small fraction of the total body of literature. In our opinion, further longitudinal work is required to address many of the key remaining areas of uncertainty in this field.

The Right Ventricle

Exercise-induced cardiac remodeling 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. In the absence of significant shunting, both chambers must augment function to accomplish this task. Recent advances in noninvasive imaging have begun to clarify how the RV responds to the repeated challenges of exercise. An initial M-mode echocardiographic study demonstrated symmetrical RV and LV enlargement in a small ($n=12$) cohort of highly trained endurance athletes.⁵⁷ Subsequently, Henriksen et al⁵⁸ examined RV and LV cavity and wall measurements using M-mode and 2-dimensional echocardiography in 127 male elite endurance athletes. Compared with historical control subjects, endurance athletes demonstrated significantly larger RV cavities and a trend toward thicker RV free walls. In an elegant magnetic resonance imaging–based study, Scharhag et al⁵⁹ recently confirmed that RV enlargement is common among endurance athletes. Data from this study suggest that RV enlargement parallels LV enlargement, supporting the concept of balanced, biventricular enlargement. A recent magnetic resonance imaging study in professional soccer players demonstrated similar findings.⁶⁰

The impact of strength training on the RV remains unclear because the limited available data are inconsistent. Perseghin et al⁶¹ compared RV and LV structure in endurance athletes (marathon runners), strength athletes (sprinters), and sedentary control subjects and found the largest RV volumes among the strength athletes. However, there were no significant difference between the RV dimensions in strength and endurance athletes after adjustment for body surface area. Right ventricular structure in collegiate endurance-trained (rowers) and strength-trained (American-style football players) athletes was recently assessed before and after 90 days of team-based exercise training.³⁵ There was statistically significant RV dilation in the endurance athletes but no changes in RV architecture in the strength athletes. Further elucidation of how the RV responds to different forms of exercise and its contribution to exercise capacity is an important area for future work.

Table 1. Selected Studies Using a Longitudinal Study Design Documenting Significant Exercise-Induced Cardiac Remodeling (Training Studies) and Regression (Detraining Studies) in Athletes

Reference	Year	Athletes, n	Athlete Type	Exercise Exposure	Primary Findings*
Training studies					
Ehsani et al ⁵¹	1978	8	Swimmers (7 male, 1 female)	Swimming for 9 wk	Increased LVMI (104 vs 109 g/m ²)
Wieling et al ⁵²	1981	9	Freshman rowers (all male)	Rowing for 7 mo	Increased LVIDd (51.5 vs 53.3 mm)
		14	Senior rowers (all male)	Rowing for 7 mo	Increased LVIDd (56.1 vs 57.9 mm)
Abergel et al ⁴²	2004	37	Cyclists (all male)	Professional cycling for 3 y	Increased LVID (58.3±4.8 to 60.3±4.2 mm); decreased LVWT (11.8±1.2 to 10.8±1.2 mm)
Naylor et al ⁴⁶	2005	22	Rowers (17 male, 5 female)	Rowing for 6 mo	Increased LVM (236±7 vs 249±9 g; <i>P</i> <0.05); increased early diastolic function
duManoir et al ⁵³	2007	10	Rowers (all male)	Rowing training for 10 wk	Increased LVMI (88±20 vs 103±22; <i>P</i> <0.05) associated with increased LVEDV and LVWT
Baggish et al ⁹⁵	2008	40	Rowers (20 male, 20 female)	Rowing for 90 d	Increased LVM (116±18 vs 130±19 g/m ²) with increased LVEDV
		24	Football (24 male)	Football for 90 d	Increased LVM (115±14 vs 132±11 g/m ²) with increased LVWT
Baggish et al ⁴⁴	2008	20	Rowers (10 male, 10 female)	Rowing for 90 d	Increased LV radial and longitudinal strain; decreased septal circumferential strain owing to concomitant RV dilation
Weiner et al ⁵⁴	2010	15	Rowers (all male)	Rowing for 90 d	Increased LV torsion and LV early diastolic untwisting rate
Detraining studies					
Fagard et al ⁵⁵	1983	12	Cyclists (all male)	Detraining for 12 wk	Decreased IVS (12.3±0.4 vs 11.5±0.4) Decreased PWT (13.0±0.5 vs 11.8±0.6); LVIDd unchanged
Pelliccia et al ⁵⁶	2002	40	Mixed, elite (all male)	Detraining for 12 wk	Decreased LVWT by 15% (12.0±1.3 vs 10.1±0.8); decreased LVIDd by 7% (61.2±2.9 vs 57.2±3.1)

LVMI indicates left ventricular mass index; LVIDd, left ventricular end-diastolic dimension; LVWT, left ventricular wall thickness; LVEDV, left ventricular volume; IVS, interventricular septum; and PWT, posterior wall thickness.

*All findings reported have statistical significance of at least *P*<0.05.

The Aorta

The aorta experiences a significant hemodynamic load during exercise. The nature of this load is dependent on sport type, with endurance activity causing high-volume aortic flow with modest systemic hypertension and strength activity resulting in normal-volume aortic flow with potentially profound systemic hypertension. It is logical to assume that such conditions should result in variable aortic remodeling in athletic individuals, and this premise has been the topic of several studies. Babae Bigi and Aslani⁶² compared aortic dimensions in 100 elite strength-trained athletes with those in 128 age-matched control subjects. They reported significantly larger aortic dimensions at the valve annulus, sinuses of Valsalva, sinotubular junction, and proximal root in the strength-trained athletes. The largest dimensions were observed in those with the longest duration of exercise training. Similarly, D'Andrea et al⁶³ used transthoracic echocardiography to measure aortic dimensions in 615 elite athletes (370 endurance-trained athletes and 245 strength-trained athletes; 410 men; mean age, 28.4±10.2 years; range, 18 to 40 years). These authors found that the aortic root diameter was significantly larger among strength-trained athletes. Vascular remodeling may also take in place in the descending abdominal aorta.⁶⁴

In contrast to the above work, Pelliccia et al⁶⁵ recently reported aortic root dimensions in a heterogeneous group of 2317 Italian athletes and found the largest measurements in endurance-trained athletes, specifically swimmers and cyclists.

Such contradictory data make definitive conclusions about the impact of sport-specific exercise training on aortic dimensions impossible. Of note, these and other studies⁶⁶ have found that the ascending aortic root rarely exceeds the clinically accepted upper limits of normal (40 mm) in trained athletes. While we await further study, it seems reasonable to conclude that athletic training alone is not a common cause of marked aortic dilation.

The Left Atrium

Numerous authors have examined the left atrial structure in trained athletes. Hauser et al⁵⁷ presented an early echocardiographic study demonstrating that a small group of endurance athletes (n=12) had larger left atria than sedentary control subjects. A similar early study documented left atrial enlargement in older individuals with a significant history of exercise training.⁶⁷ Hoogsteen et al⁶⁸ compared atrial dimensions in young competitive cyclists (age, 17±0.2 years; n=66) with those in older, presumably more experienced cyclists (29±2.6 years; n=35) and found larger dimensions in the older athletes. Pelliccia et al⁶⁹ presented the largest data set of atrial measurements in athletes (n=1777) and demonstrated that left atrial enlargement (>40 mm in an anterior/posterior transthoracic echocardiographic view) was present in 20% of the athletes. Of note, few athletes with left atrial dilation in the Pelliccia et al series had clinical evidence of supraventricular arrhythmias.⁶⁹ D'Andrea et al⁷⁰ recently confirmed a high prevalence of left atrial enlargement in trained athletes and demonstrated an association with endurance exercise training.

The Impact of Sex and Ethnicity

Exercise-induced cardiac remodeling is similar in male and female athletes. However, available data suggest that female athletes exhibit quantitatively less physiological remodeling than their male counterparts.^{70–75} This appears to be true even when cardiac dimensions are corrected for the typically smaller female body size. Explanations for the sex-specific magnitude of remodeling remain elusive. Race is also an important determinant of remodeling, with black athletes tending to have thicker LV walls than white athletes. Basavarajiah and colleagues^{75a} recently studied a group of white and black athletes using echocardiographic imaging and found that nearly 20% of the black athletes had an LV wall thickness of at least 12 mm compared with 4% in white athletes. Importantly, 3% of black athletes in this cohort were found to have a wall thickness of >15 mm. Similarly, Rawlins et al^{75b} studied ethnic/race-related differences in a group of 440 black and white female athletes using ECG and echocardiography. Black female athletes demonstrated significantly higher LV wall thickness and mass compared with the white women (in black athletes: LV wall thickness = 9.2 ± 1.2 mm and LV mass = 187.2 ± 42 g; in white athletes: LV wall thickness = 8.6 ± 1.2 mm and LV mass = 172.3 ± 42 g). Such anatomic differences are likely responsible for the increased prevalence of ECG abnormalities in black athletes, including LV hypertrophy, precordial T-wave inversions, and early repolarization.⁷⁶

Common Clinical Cardiovascular Issues in the Athletic Patient

Although participation in sport and regular exercise promotes good health, athletes are not immune to cardiovascular symptoms and disease and thus represent an important population encountered in clinical practice. The athletic patient population is heterogeneous and comprises individuals spanning broad ranges of age, athletic talent, and performance goals. Athletes typically become patients in 1 of 2 ways. First, an abnormal value for one of the structural or function parameters discussed above may be detected in the asymptomatic individual. Such findings often raise concerns about potential underlying pathology, thereby prompting further assessment. Second, athletes may develop symptoms suggestive of cardiovascular disease during training or competition and thus seek or are referred for a symptom-driven evaluation.

Cardiac Enlargement: Pathology Versus Physiological Adaptation

The phenotypic overlap between exercise-induced cardiac remodeling and pathological structural heart disease is widely appreciated. Cardiac structural abnormality of unclear origin and significance may be detected during preparticipation cardiovascular disease screening, routine health examinations, or evaluation of the athlete with symptoms. Extreme cases of exercise-induced ventricular remodeling may be difficult to differentiate from mild forms of hypertrophic cardiomyopathy, familial or acquired dilated cardiomyopathy, and arrhythmogenic RV cardiomyopathy. The clinical task of differentiating marked exercise-induced remodeling from these important

forms of disease remains important, with implications including sport restriction, pharmacological therapy, and placement of an implantable cardiac defibrillator.

The overlap between features of the athlete's heart and characteristics of cardiomyopathy, specifically hypertrophic cardiomyopathy, that may affect young athletes has been coined the Maron gray zone.⁷⁷ A valuable schema for approaching the athletic patient with LV hypertrophy of unclear origin has been presented, and remains useful in clinical practice.⁷⁸ It is noteworthy that this diagnostic approach was developed at a time when noninvasive cardiovascular imaging was in its infancy and, for the most part, restricted to basic 2-dimensional echocardiography. Recent advances in cardiovascular diagnostics have proven to be useful additions to these original criteria.

Functional myocardial echocardiography, including tissue Doppler and speckle-tracking imaging, permits detailed and accurate assessment of myocardial function. Tissue Doppler imaging permits assessment of myocardial relaxation and contraction velocity. Across numerous studies, early diastolic relaxation velocity has been shown to be normal or increased in athletes with LV hypertrophy resulting from exercise-induced remodeling.^{79,80} In contrast, pathological forms of LV hypertrophy are typically associated with reduced early diastolic relaxation velocity and peak systolic tissue velocity.^{45,81–83} Tissue strain and strain rate may also provide useful insight into the origin of LV hypertrophy in athletes.^{84,85}

Cardiac magnetic resonance imaging has similarly emerged as an invaluable tool for the evaluation of the athlete with indeterminate cardiac enlargement. Cardiac magnetic resonance allows highly accurate assessment of myocardial thickness, chamber volumes, tissue composition, extracardiac anatomy, and cardiac magnetic resonance–derived reference values in athletes have been published.⁸⁶ Importantly, the use of gadolinium contrast with delayed imaging provides information about the presence and location of myocardial fibrosis. Recent data document myocardial fibrosis patterns that are relatively specific to certain cardiomyopathies and highlight the utility of cardiac magnetic resonance for the assessment of indeterminate cardiac enlargement in the athlete.^{87,88} At the present time, we use cardiac magnetic resonance in athletes with either indeterminate echocardiographic imaging or clinical features that suggest a diagnosis that may not be definitively assessed by echocardiography (ie, myocarditis). The interested reader is referred to a recently published comprehensive review on this topic.⁸⁹

At the present time, there is no single diagnostic test with adequate accuracy for differentiating adaptive from pathological cardiomyopathy. Consequently, we encourage clinicians faced with this diagnostic dilemma to begin the assessment with an integrated consideration of personal and family medical history, 12-lead ECG, and echocardiography. In many cases, this relatively basic but informative triad will provide sufficient information for informed diagnostic decision making. In cases that remain unclear after this initial evaluation, further measures, including tissue Doppler echocardiography, speckle-tracking echocardiography, magnetic resonance imaging, cardiopulmonary exercise testing, pre-

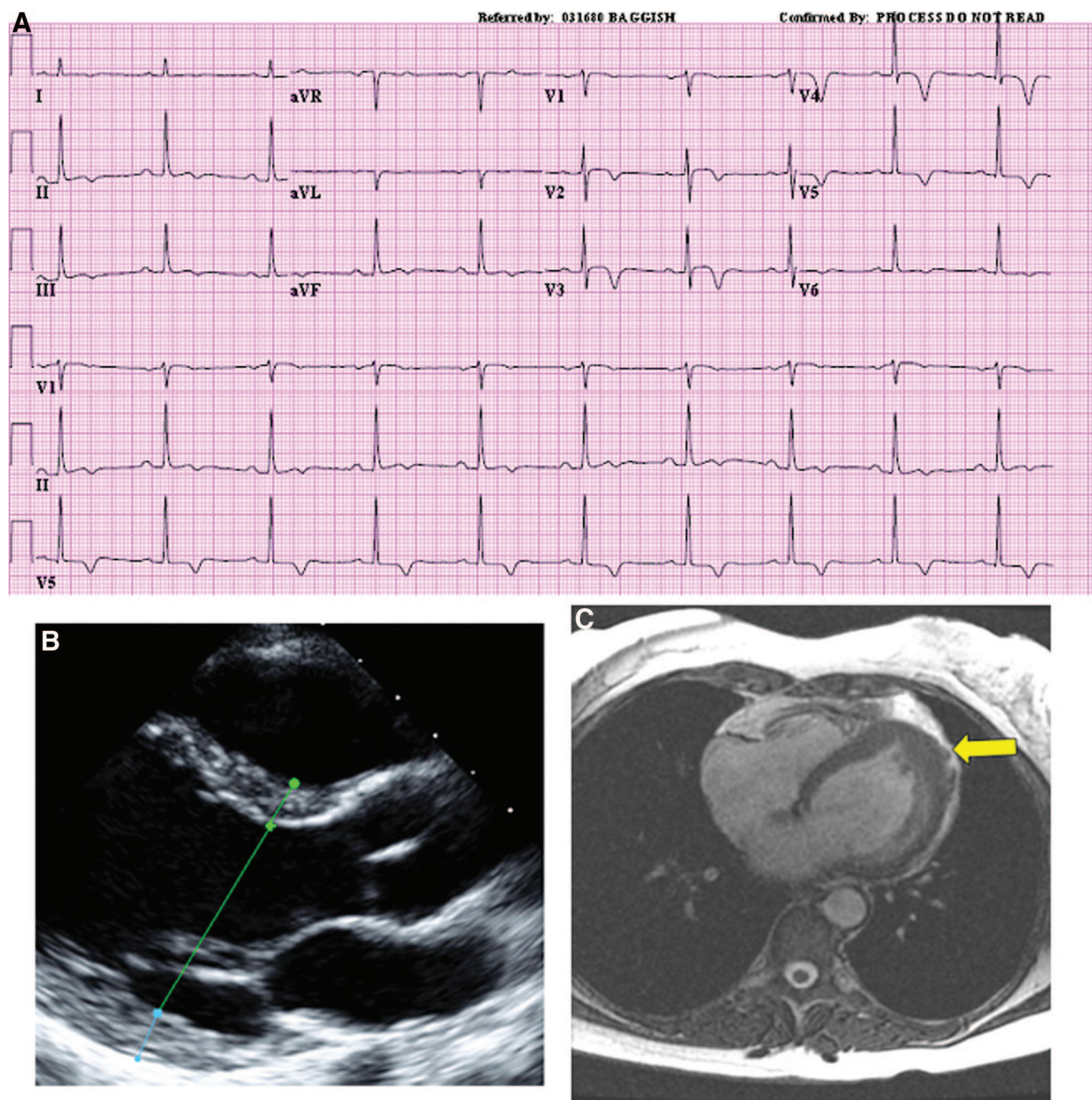


Figure 1. **A**, An electrocardiogram of a 46-year-old male triathlete who presented after long-standing palpitations and a recent episode of syncope. The findings of diffuse T-wave inversions prompted an echocardiogram (**B**), which revealed normal left ventricular (LV) dimensions and function. **C**, A subsequent magnetic resonance imaging study confirmed the presence of focal asymmetric LV hypertrophy (yellow arrow) consistent with the apical variant form of hypertrophic cardiomyopathy.

scribed detraining, and disease-specific genetic testing, may be considered. Examples illustrating the utility of advanced echocardiographic techniques (Figure 1) and magnetic resonance imaging (Figure 2) for the assessment of athletes with suspected myocardial pathology are shown.

Arrhythmia

Arrhythmia and conduction alterations are common in the trained athlete, and recommendations for their evaluation are available.^{90,91} Bradyarrhythmias such as sinus bradycardia, junctional bradycardia, first-degree atrioventricular block, and Mobitz type I atrioventricular block are commonly

observed.⁹² Athletic patients with these bradyarrhythmias are almost always asymptomatic, and profound bradycardia in the context of rest or sleep does not appear to portend a poor prognosis. Heightened parasympathetic activity, specifically increased efferent vagus nerve activity, is responsible for these bradyarrhythmias. However, some experimental data suggest that repeated exercise training may also lead to intrinsic sinoatrial node slowing.⁹³ In the asymptomatic athletic patient with any of these common bradyarrhythmias, reassurance and documentation of an appropriate chronotropic response to exercise are typically sufficient to exclude a pathological process.⁹⁴ Advanced forms atrioventricular

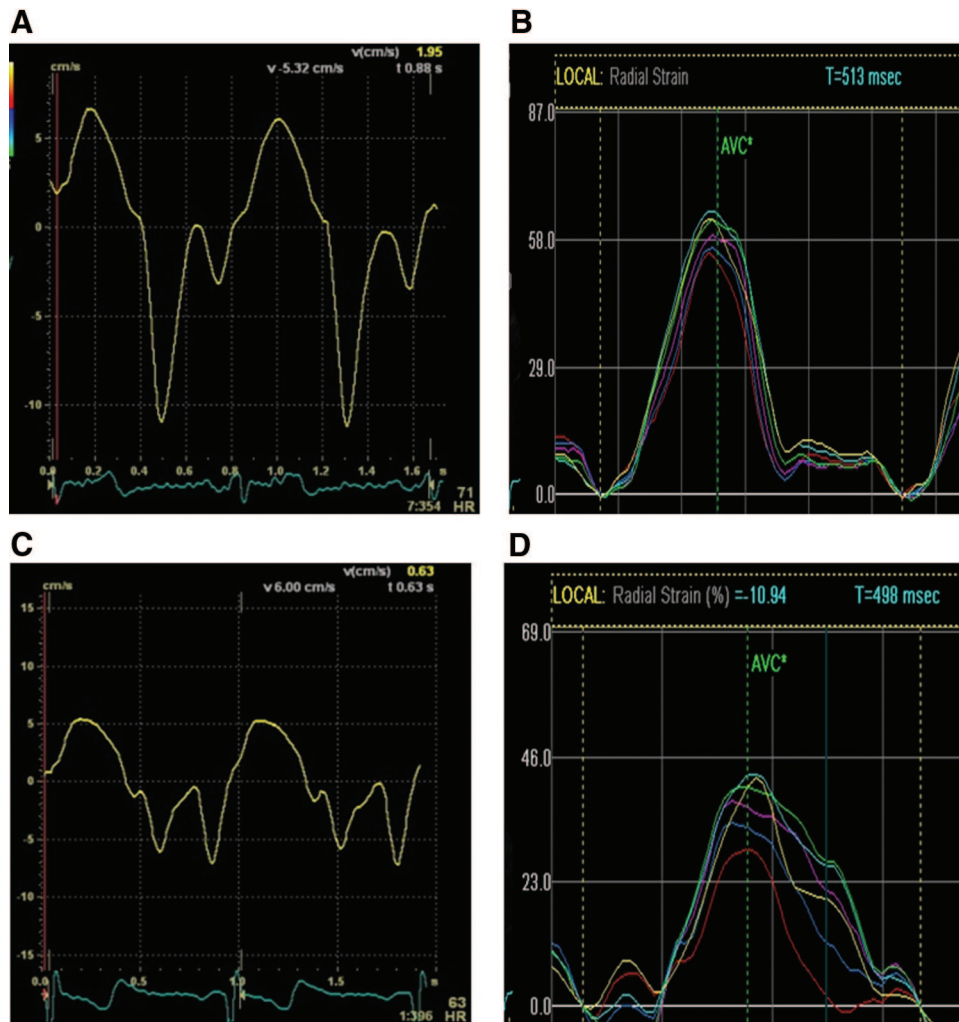


Figure 2. Echocardiographic tissue Doppler imaging (**A** and **C**) and speckle-tracking radial strain analysis (**B** and **D**) in 2 different athletic patients presenting with left ventricular hypertrophy. Normal tissue velocity (**A**) and radial strain (**B**) in a 23-year-old male elite rower with marked eccentric left ventricular (LV) hypertrophy (interventricular septal thickness=15 mm, posterior wall thickness=15 mm, LV end-diastolic dimension=64 mm by echocardiography). Pathological tissue velocities (**C**) and reduced heterogeneous radial strain (**D**) in a 20-year-old male collegiate football player with concentric LV hypertrophy (interventricular septal thickness=15 mm, posterior wall thickness=15 mm, LV end-diastolic dimension=42 mm by echocardiography) who was subsequently diagnosed with hypertrophic cardiomyopathy after minimal regression during prescribed detraining and a genetic test identifying a pathological mutation in the cardiac β -myosin heavy chain gene.

dissociation, such as second-degree Mobitz type II and third-degree heart block, are unusual in athletes and should be considered pathological.⁹⁵

Premature beats (both atrial and ventricular) and nonsustained ventricular tachycardia may be observed in trained athletes. Because athletic patients are typically of lean physical build and know their bodies well, they may be particularly sensitive to such rhythm alterations. Palpitations caused by premature beats are a common reason for athletes to seek medical attention. In such instances, it is important to exclude structural and valvular heart disease and to assess the impact of exercise on the frequency of the premature beats. In the absence of structural heart disease, and when suppressed by exercise, premature atrial and ventricular beats are usually benign and without long-term implications.^{96,97} Similarly reassuring data have been published regarding the benign nature of nonsustained ventricular tachycardia in athletes without structural heart disease.^{98,99} Prescribed detraining has

been shown to decrease the burden of such arrhythmias, and may be useful as a therapeutic intervention in the symptomatic athlete.⁹⁷

Tachyarrhythmias, specifically atrial fibrillation, may be particularly problematic in the trained athlete. Several studies suggest that atrial fibrillation is more common among currently or previously trained athletes than in their sedentary counterparts.^{100–103} This appears to be particularly true of the older athletic patient. The mechanisms producing atrial fibrillation in athletes are speculative, but a combination of exercise-induced left atrial remodeling and inflammation,^{69,104} increased sympathetic activity during exercise,¹⁰⁰ and parasympathetically mediated slow resting heart rates potentiating atrial escape⁹³ has been suggested as a potential contributor.

Initial evaluation of the athlete with supraventricular tachyarrhythmias, including atrial fibrillation, should include exclusion of metabolic derangements, especially hyperthy-

roidism, detailed nutritional and supplement intake history (ie, caffeine, other stimulants), and an assessment for structural and valvular heart disease. Careful attention to the physiological conditions during which an individual athlete develops arrhythmia, specifically differentiating fast-heart-rate conditions (ie, exercise) from slow-heart-rate conditions (ie, sleep, exercise recovery), may be useful for individualizing management. Rate-control agents such as β -blockers and calcium channel blockers do not reduce the frequency of atrial fibrillation, but may minimize symptoms in the athlete. However, these agents reduce maximal and submaximal heart rate during exercise and may lead to unfavorable, poorly tolerated reductions in exercise capacity, and β -blocker use may be prohibited during competition in certain sports. Class IC antiarrhythmic agents, including flecainide and propafenone, may be effective ways of maintaining sinus rhythm, but both increase atrioventricular conduction and generally require an additional agent to prevent an accelerated ventricular response if atrial flutter should occur. Both flecainide and propafenone can be used with the pill in the pocket approach to atrial fibrillation,¹⁰⁵ which may be attractive in some athletes. Although data documenting the success of catheter-based ablation for atrial fibrillation in athletes are sparse,^{106,107} this is an appropriate strategy for certain individuals.

Syncope

Syncope, defined as a transient loss of consciousness accompanied by loss of postural tone, is common in trained athletes. In a large cohort of Italian athletes, roughly 6% reported syncope in the prior 5 years.¹⁰⁸ In this important study, the vast majority of syncope was unrelated to exercise (86.7%) or occurred in the postexertional period (12.0%). In the small minority with true exertional syncope (1.3%), explanatory structural heart disease was common.

The majority of syncope in athletes is attributable to neurocardiogenic mechanisms. Typically, neurocardiogenic syncope in the athlete manifests in the immediate postexercise setting owing to a sudden reduction in venous return. This reduction in venous return, caused by both cessation of skeletal muscle contraction and altered sympathetic/parasympathetic balance, facilitates transient cerebral hypoperfusion. The athlete with postexertional neurocardiogenic syncope will typically report presyncopal feelings of warmth, diaphoresis, or lightheadedness, which culminate in a loss of consciousness ranging from several seconds to a minute. Syncope during intense exercise is almost never due to neurocardiogenic mechanisms and should alert the practitioner to the possibility of malignant arrhythmia, structural/valvular heart disease, or myocardial ischemia.

The approach to the athletic patient with syncope begins with a detailed history, physical examination, and 12-lead ECG.⁹¹ Care should be taken to characterize potential triggers, the timing and duration of the event, and the risk associated with future similar loss of consciousness. The resting 12-lead ECG should be inspected for abnormalities of conduction (QT prolongation, pre-excitation, right bundle-branch block with early precordial ST elevation suggestive of Brugada syndrome) and structural heart disease (left bundle-branch block, LV hypertrophy with repolarization abnormal-

ities, diffuse T-wave inversions). Transthoracic echocardiography is recommended to exclude structural and valvular heart disease in individuals with syncope, especially if anything abnormal is detected during physical examination or ECG interpretation. Syncope that occurs during exercise requires diagnostic assessment with exercise stress testing. This testing should be designed to approximate the exercise conditions in which the syncope occurred, and careful attention should be given to the exercise ECG for the detection of explanatory arrhythmias. However, exercise testing is frequently normal in individuals with coronary artery anomalies, so coronary imaging may be required in athletes with syncope and this clinical possibility.¹⁰⁹ The use of diagnostic tilt-table testing in athletes is controversial owing to concerns of false-positive testing; thus, we do not routinely use this strategy. Ambulatory monitoring with a loop or an event recorder may prove useful in patients with symptoms that are not reproduced during a laboratory-based exercise assessment.

Management of the athlete with syncope is dictated by cause. Individuals with significant structural or valvular heart disease should be managed with appropriate sport restriction, medication, electrophysiological study with or without ablation, implantable defibrillator placement, or surgery on the basis of their specific pathology. Neurocardiogenic syncope can often be avoided by mandating an active cool-down period after vigorous exertion and by paying attention to hydration and supplemental salt intake. In athletes with recurrent neurocardiogenic syncope despite these first-line treatments, postural training,¹¹⁰ or pharmacological therapy with either selective serotonin uptake inhibitors or midodrine, may be a reasonable next step, but should be prescribed only after careful consideration of rules delineating banned substances in athletes.^{111,112}

Performance Enhancement Agents

Advances in the science of human performance, increased societal and financial pressures to perform, and increased access to illicit drugs has led to the growing use of performance-enhancing agents. The World Anti-Doping Agency has developed a comprehensive list of medications and substances that are banned in athletes.¹¹² Importantly, some commonly prescribed cardiovascular medications, including but not limited to β -blockers, adrenergic agonists (both β - and α -agonists), diuretics, and stimulants, are prohibited universally or in specific sport disciplines. Clinicians working with athletes are encouraged to develop a comprehensive understanding of World Anti-Doping Agency guidelines to avoid prescribing a banned agent.

The most widely used form of performance-enhancing agents is androgenic anabolic steroids. Although developed in the 1940s and 1950s, steroid use became common among professional and recreational athletes in the early 1980s. Numerous small studies have documented deleterious cardiovascular effects of steroids such as dyslipidemia,¹¹³ exaggerated exercise blood pressure response,¹¹⁴ and myocardial dysfunction (Table 2). Definitive data documenting the impacts of steroid use on cardiovascular health, particularly in older athletic patients with prolonged drug exposure, remain uncertain and constitute an important area of future work.

Table 2. Summary of Recent Studies Examining the Impact of Long-Term Term Androgenic Anabolic Steroid Use on Myocardial Function in Athletes

Reference	Year	Athletes, n	Study Design	Athlete Description	Reported Prior Steroid Exposure*	Findings Compared With Control Subjects
Nottin et al ¹¹⁵	2006	6	Cross-sectional comparison with AAS-free weightlifters	Recreational weightlifters	>2 y	No difference in systolic function; decreased diastolic function (transmitral and tissue velocities)
D'Andrea et al ¹¹⁶	2007	20	Cross-sectional comparison with AAS-free weightlifters	Top-level competitive body builders	524±91 mg/wk (31±6 wk/y over 8.9±3.8 y)	Decreased systolic function (strain, strain rate); decreased diastolic function (transmitral and tissue velocities)
Krieg et al ¹¹⁷	2007	14	Cross-sectional comparison with AAS-free weightlifters	Recreational weightlifters	817±619 mg/wk (27±11 wk/y over 8.4±4.8 y)	No difference in systolic function; decreased diastolic function (transmitral and tissue velocities)
Baggish et al ¹¹⁸	2010	12	Cross-sectional comparison with AAS-free weightlifters	Recreational weightlifters	675 [513 950] mg/wk (468 [169 520] lifetime-wk)	Decreased systolic function (LVEF, strain); decreased diastolic function (transmitral and tissue velocities)

AAS indicates androgenic anabolic steroid; LVEF, left ventricular ejection fraction.

*Numbers represent testosterone equivalences dose. Values are mean±SD when appropriate. Values in brackets are interquartile ranges.

Nonsteroidal muscle mass growth stimulators, including injectable insulin, human growth hormone, and creatine, have also become popular. The cardiovascular effects of these agents in athletes remain unknown. There has also been increasing interest in the use of stimulants among competitive athletes, including numerous available prescription medications (ie, methylphenidate) and over-the-counter preparations (high-dose caffeine). Although the long-term toxicity of stimulant use remains controversial, these stimulants likely precipitate or exacerbate common complaints in the athlete, such as palpitations. Finally, erythropoietic stimulants are popular among endurance athletes because of the potential performance enhancement afforded by augmented red cell mass. Complications of erythropoietic stimulant use are related largely to the impact of excessive plasma red cell mass, such as microvascular infarction. To date, there is a dearth of study examining the impact of performance-enhancing agents in athletes. While we await further data, we encourage clinicians faced with the care of athletic patients to inquire about the use of performance-enhancing agents as a routine component of the medical history.

Sudden Cardiac Death

Sudden death in young athletic individuals is a rare but tragic event. Studies examining sudden death in athletes report a wide range of prevalence. United States data from a single-state registry suggest a sudden death prevalence of 1:200 000 per year,²⁰ whereas data from the Italian preparticipation screening program suggest a significantly higher rate.^{119,120} The variability in sudden death prevalence statistics data may be attributed to multiple factors, such as geographic variability in the prevalence of causal diseases, characteristics of the populations studied, and case ascertainment techniques. Although there appears to be some regional difference in the relative contribution causal diseases, hypertrophic cardiomyopathy is the most common cause of sudden cardiac death in the young in the United States. Sudden death has been documented in most types of competitive sports, but may be more common during participation in physically intense sports, such as basketball, soccer, and American-style foot-

ball. In addition to sport type, sex and ethnicity appear to contribute to sudden death risk, with male participants and individuals of Afro-Caribbean descent more likely to succumb to sport-related sudden death.

Most cases of sudden, sport-related death in young athletes are attributable to underlying cardiovascular pathology (Table 3). Both the American Heart Association/American College of Cardiology¹²¹ and the European Society of Cardiology⁹⁰ have established sport eligibility criteria for individuals diagnosed with these conditions. Guidelines endorsed by these 2 groups are largely similar aside from a few notable situations, including the management of athletes with asymp-

Table 3. Common Cardiovascular Conditions Associated With Sudden Death in Athletes

Disorders of the myocardium
Hypertrophic cardiomyopathy
Arrhythmogenic right ventricular cardiomyopathy
Familial/idiopathic dilated cardiomyopathy
Acute and subacute myocarditis
Disorders of myocardial electric activity and conduction
Congenital and acquired long-QT syndrome
Short-QT syndrome
Wolff-Parkinson-White syndrome
Brugada syndrome
Catecholaminergic polymorphic ventricular tachycardia
Commotio cordis
Disorders of the coronary circulation
Congenital anomalies of coronary arterial origin and course
Acquired atherosclerotic disease
Disorders of the heart valves
Bicuspid aortic valve disease associated with any of the following:
Significant aortic root dilation
Marfan syndrome
Moderate or greater stenosis or regurgitation
Mitral valve prolapse
Pulmonic stenosis

tomatic Wolff-Parkinson-White syndrome and the approach for athletes with genotype-positive/phenotype-negative myocardial or electric heart disease.¹²²

The tragic nature of sudden death in young, previously asymptomatic athletes has led to considerable efforts aimed at prevention. The logic that the detection and management of cardiovascular disease before sport participation may reduce the incidence of sudden cardiac death has led to recommendations for preparticipation screening. The American Heart Association/American College of Cardiology¹⁰⁵ and the European Society of Cardiology^{123,124} have published consensus committee-based recommendations for preparticipation athlete screening. Both governing bodies recommend a focused medical history and physical examination. The European Society of Cardiology recommends the addition of a 12-lead ECG. This addition of a 12-lead ECG to medical history and physical remains an area of intense debate. Observational data from the Italian national experience and recent prospective trial data from the United States suggest that ECG may improve the sensitivity of preparticipation cardiovascular screening.^{125,126} However, a number of issues, ie, the financial and manpower costs of the mandated ECG, the high rate of false-positive ECG findings, the cost of follow-up testing for those with abnormal results, the logistics of ECG acquisition and interpretation, and considerations about future insurability for athletes with detected disease, represent considerable obstacles to implementing a mandatory 12-lead ECG as part of preparticipation screening in the United States. Although additional observational data from organizations or nations using ECG are welcomed, this issue will almost certainly remain controversial until a prospective, randomized, multinational trial is conducted to provide a definitive answer. In the absence of such data, priority should be placed on widespread dissemination and implication of current history and physical examination recommendations with consideration of ECG only in localities with sufficient resources and expertise for this technique.

Future Directions

Although our understanding of the athlete's heart has progressed considerably since Darling and Henschen first observed cardiac enlargement in athletes in 1899, a number of unanswered questions remain. From a physiological perspective, we believe that several issues deserve future study. First, the relationship between exercise dose (intensity, frequency, duration) and both hypertrophy and subsequent regression during detraining remains inadequately characterized. Second, variability in the magnitude of remodeling within seemingly homogenous groups, likely a function of both genetic and environmental factors, remains poorly understood. Third, the exact functional aspects of exercise-induced remodeling that facilitate preserved or enhanced stroke volume during high-level exercise remain to be elucidated. Carefully conducted longitudinal study will be the best way to address these areas of scientific uncertainty.

Additionally, several clinical issues require further attention. First, a strategy for differentiating adaptive from pathological cardiomyopathy that integrates basic clinical factors with modern diagnostic tests (imaging, cardiopulmonary

exercise testing, and genetic assessment) should be developed and validated for clinical use. Second, the widely applicable consensus committee guidelines for the management of cardiovascular disease in athletes require timely updates as dictated by diagnostic and therapeutic advances. Third, prospective, longitudinal study examining the impact of preparticipation ECG screening should be conducted to end the ongoing debate about how best to reduce the incidence of sport-related sudden cardiac death. As the general population's interest in exercise and competitive sport continues to rise, the time has come to address these important scientific and clinical issues.

Disclosures

None.

References

1. Darling EA. The effects of training: a study of the Harvard University crews. *Boston Med Surg J*. 1899;161:229–233.
2. Henschen S. Skidlauf und skidwettlauf: eine medizinische sportstudie. *Mitt Med Klin Upsala*. 1899;2.
3. White PD. The pulse after a marathon race. *JAMA*. 1918;71:1047–1048.
4. White PD. Bradycardia in athletes, especially long distance runners. *JAMA*. 1942;120:642.
5. Roskamm H, Reindell H, Musshoff K, Koenig K. Relations between heart size and physical efficiency in male and female athletes in comparison with normal male and female subjects. *Arch Kreislaufforsch*. 1961;35:67–102.
6. Reindell H, Roskamm H, Steim H. The heart and blood circulation in athletes. *Med Welt*. 1960;31:1557–1563.
7. Bulychev VV, Khmelevskii VA, Rutman I. Roentgenological and instrumental examination of the heart in athletes. *Klin Med*. 1965;43:108–114.
8. Hunt EA. Electrocardiographic study of 20 champion swimmers before and after 110-yard sprint swimming competition. *Can Med Assoc J*. 1963;88:1251–1253.
9. Arstila M, Koivikko A. Electrocardiographic and vectorcardiographic signs of left and right ventricular hypertrophy in endurance athletes. *J Sports Med Phys Fitness*. 1966;6:166–175.
10. Chignon JC, Distel R, Arnaud P. Morphologic variations of horizontal vectorcardiograms in athletes [in French]. *J Physiol (Paris)*. 1967;59:375.
11. Gott PH, Roselle HA, Crampton RS. The athletic heart syndrome: five-year cardiac evaluation of a champion athlete. *Arch Intern Med*. 1968;122:340–344.
12. Chignon JC, Distel R, Courtois B, Leclercq J, Andrivet R. Orientation of the analysis of electrical tracings regarding athletes. *J Sports Med Phys Fitness*. 1969;9:241–244.
13. Van Ganse W, Versee L, Eysenbosch W, Vuylsteek K. The electrocardiogram of athletes: comparison with untrained subjects. *Br Heart J*. 1970;32:160–164.
14. Hanne-Paparo N, Wendkos MH, Brunner D. T wave abnormalities in the electrocardiograms of top-ranking athletes without demonstrable organic heart disease. *Am Heart J*. 1971;81:743–747.
15. Schamroth L, Jokl E. Marked sinus and A-V nodal bradycardia with interference-dissociation in an athlete. *J Sports Med Phys Fitness*. 1969;9:128–129.
16. Fleischmann P, Kellermann JJ. Persistent irregular tachycardia in a successful athlete without impairment of performance. *Isr J Med Sci*. 1969;5:950–952.
17. Moritz F. Über orthodiographische untersuchungen am herzen. *Med Wochenschr*. 1902;49.
18. Pelliccia A, Kinoshita N, Pisicchio C, Quattrini F, Dipaolo FM, Ciardo R, Di Giacinto B, Guerra E, De Blasis E, Casasco M, Culasso F, Maron BJ. Long-term clinical consequences of intense, uninterrupted endurance training in Olympic athletes. *J Am Coll Cardiol*. 2010;55:1619–1625.
19. Harper RW, Mottram PM. Exercise-induced right ventricular dysplasia/cardiomyopathy: an emerging condition distinct from arrhythmogenic right ventricular dysplasia/cardiomyopathy. *Heart Lung Circ*. 2009;18:233–235.

20. Thompson PD. Exercise prescription and proscripton for patients with coronary artery disease. *Circulation*. 2005;112:2354–2363.
21. Rowell LB. *Human Circulation: Regulation During Physical Stress*. New York, NY: Oxford University Press; 1986.
22. Levine BD. *Exercise and Sports Cardiology*. New York, NY: McGraw Hill; 2001.
23. Jose AD, Collison D. The normal range and determinants of the intrinsic heart rate in man. *Cardiovasc Res*. 1970;4:160–167.
24. Uusitalo AL, Uusitalo AJ, Rusko HK. Exhaustive endurance training for 6–9 weeks did not induce changes in intrinsic heart rate and cardiac autonomic modulation in female athletes. *Int J Sports Med*. 1998;19: 532–540.
25. Venerando A, Rulli V. Frequency morphology and meaning of the electrocardiographic anomalies found in Olympic marathon runners and walkers. *J Sports Med Phys Fitness*. 1964;50:135–141.
26. Roeske WR, O'Rourke RA, Klein A, Leopold G, Karliner JS. Noninvasive evaluation of ventricular hypertrophy in professional athletes. *Circulation*. 1976;53:286–291.
27. Pelliccia A, Culasso F, Di Paolo FM, Maron BJ. Physiologic left ventricular cavity dilatation in elite athletes. *Ann Intern Med*. 1999;130: 23–31.
28. 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.
29. Sharma S, Maron BJ, Whyte G, Firoozi S, Elliott PM, McKenna WJ. Physiologic limits of left ventricular hypertrophy in elite junior athletes: relevance to differential diagnosis of athlete's heart and hypertrophic cardiomyopathy. *J Am Coll Cardiol*. 2002;40:1431–1436.
30. Baggish AL, Yared K, Weiner RB, Wang F, Demes R, Picard MH, Hagerman F, Wood MJ. Differences in cardiac parameters among elite rowers and subelite rowers. *Med Sci Sports Exerc*. 2010;42:1215–1220.
31. Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. *Ann Intern Med*. 1975;82: 521–524.
32. Fisman EZ, Pelliccia A, Motro M, Auerbach I, Frank AG, Tenenbaum A. Effect of intensive resistance training on isotonic exercise Doppler indexes of left ventricular systolic function. *Am J Cardiol*. 2002;89: 887–891.
33. Roy A, Doyon M, Dumesnil JG, Jobin J, Landry F. Endurance vs. strength training: comparison of cardiac structures using normal predicted values. *J Appl Physiol*. 1988;64:2552–2557.
34. 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;101:336–344.
35. Baggish AL, Wang F, Weiner RB, Elinoff JM, Tournoux F, Boland A, Picard MH, Hutter AM Jr, Wood MJ. Training-specific changes in cardiac structure and function: a prospective and longitudinal assessment of competitive athletes. *J Appl Physiol*. 2008;104: 1121–1128.
36. Naylor LH, George K, O'Driscoll G, Green DJ. The athlete's heart: a contemporary appraisal of the "Morganroth hypothesis." *Sports Med*. 2008;38:69–90.
37. Fagard R, Aubert A, Staessen J, Eynde EV, Vanhees L, Amery A. Cardiac structure and function in cyclists and runners: comparative echocardiographic study. *Br Heart J*. 1984;52:124–129.
38. Bar-Shlomo BZ, Druck MN, Morch JE, Jablonsky G, Hilton JD, Feiglin DH, McLaughlin PR. Left ventricular function in trained and untrained healthy subjects. *Circulation*. 1982;65:484–488.
39. Bekaert I, Pannier JL, Van de Weghe C, Van Durme JP, Clement DL, Pannier R. Non-invasive evaluation of cardiac function in professional cyclists. *Br Heart J*. 1981;45:213–218.
40. Douglas PS, O'Toole ML, Hiller WD, Reichel N. Left ventricular structure and function by echocardiography in ultraendurance athletes. *Am J Cardiol*. 1986;58:805–809.
41. 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;40:528–533.
42. Abergel E, Chatellier G, Hagege AA, Oblak A, Linhart A, Ducardonnet A, Menard J. Serial left ventricular adaptations in world-class professional cyclists: implications for disease screening and follow-up. *J Am Coll Cardiol*. 2004;44:144–149.
43. Richard V, Lafitte S, Reant P, Serri K, Lafitte M, Brette S, Kerouani A, Chalabi H, Dos Santos P, Douard H, Roudaut R. An ultrasound speckle tracking (two-dimensional strain) analysis of myocardial deformation in professional soccer players compared with healthy subjects and hypertrophic cardiomyopathy. *Am J Cardiol*. 2007;100:128–132.
44. Baggish AL, Yared KL, Wang F, Weiner RB, Hutter Jr AM, Picard MH, Wood MJ. The impact of endurance exercise training on left ventricular systolic mechanics. *Am J Physiol Heart Circ Physiol*. 2008;295: H1109–H1116.
45. Cardim N, Oliveira AG, Longo S, Ferreira T, Pereira A, Reis RP, Correia JM. Doppler tissue imaging: regional myocardial function in hypertrophic cardiomyopathy and in athlete's heart. *J Am Soc Echocardiogr*. 2003;16:223–232.
46. Naylor LH, Arnold LF, Deague JA, Playford D, Maurogiovanni A, O'Driscoll G, Green DJ. Reduced ventricular flow propagation velocity in elite athletes is augmented with the resumption of exercise training. *J Physiol*. 2005;563:957–963.
47. Caso P, D'Andrea A, Galderisi M, Liccardo B, Severino S, De Simone L, Izzo A, D'Andrea L, Mininni N. Pulsed Doppler tissue imaging in endurance athletes: relation between left ventricular preload and myocardial regional diastolic function. *Am J Cardiol*. 2000;85:1131–1136.
48. Tumuklu MM, Ildizli M, Ceyhan K, Cinar CS. Alterations in left ventricular structure and diastolic function in professional football players: assessment by tissue Doppler imaging and left ventricular flow propagation velocity. *Echocardiography*. 2007;24:140–148.
49. Prasad A, Popovic ZB, Arbab-Zadeh A, Fu Q, Palmer D, Dijk E, Greenberg NL, Garcia MJ, Thomas JD, Levine BD. The effects of aging and physical activity on Doppler measures of diastolic function. *Am J Cardiol*. 2007;99:1629–1636.
50. D'Andrea A, Cocchia R, Riegler L, Scarafie R, Salerno G, Gravino R, Golia E, Pezzullo E, Citro R, Limongelli G, Pacileo G, Cuomo S, Caso P, Russo MG, Bossone E, Calabro R. Left ventricular myocardial velocities and deformation indexes in top-level athletes. *J Am Soc Echocardiogr*. 2010;23:1281–1288.
51. Ehsani AA, Hagberg JM, Hickson RC. Rapid changes in left ventricular dimensions and mass in response to physical conditioning and deconditioning. *Am J Cardiol*. 1978;42:52–56.
52. Wieling W, Borghols EA, Hollander AP, Danner SA, Dunning AJ. Echocardiographic dimensions and maximal oxygen uptake in oarsmen during training. *Br Heart J*. 1981;46:190–195.
53. duManoir GR, Haykowsky MJ, Syrotoik DG, Taylor DA, Bell GJ. The effect of high-intensity rowing and combined strength and endurance training on left ventricular systolic function and morphology. *Int J Sports Med*. 2007;28:488–494.
54. Weiner RB, Hutter AM Jr, Wang F, Kim J, Weyman AE, Wood MJ, Picard MH, Baggish AL. The impact of endurance exercise training on left ventricular torsion. *J Am Coll Cardiol Cardiovasc Imaging*. 2010; 3:1001–1009.
55. Fagard R, Aubert A, Lysens R, Staessen J, Vanhees L, Amery A. Noninvasive assessment of seasonal variations in cardiac structure and function in cyclists. *Circulation*. 1983;67:896–901.
56. 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;105:944–949.
57. 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; 109:1038–1044.
58. Henriksen E, Landelius J, Wesslen L, Arnell H, Nystrom-Rosander C, Kangro T, Jonason T, Rolf C, Lidell C, Hammarstrom E, Ringqvist I, Friman G. Echocardiographic right and left ventricular measurements in male elite endurance athletes. *Eur Heart J*. 1996;17:1121–1128.
59. Scharhag J, Schneider G, Urhausen A, Rochette V, Kramann B, Kindermann W. Athlete's heart: right and left ventricular mass and function in male endurance athletes and untrained individuals determined by magnetic resonance imaging. *J Am Coll Cardiol*. 2002;40:1856–1863.
60. 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;159:911–918.
61. Perseghin G, De Cobelli F, Esposito A, Lattuada G, Terruzzi I, La Torre A, Belloni E, Canu T, Scifo P, Del Maschio A, Luzi L, Alberti G. Effect of the sporting discipline on the right and left ventricular morphology and function of elite male track runners: a magnetic resonance imaging and phosphorus 31 spectroscopy study. *Am Heart J*. 2007;154:937–942.
62. Babae Bigi MA, Aslani A. Aortic root size and prevalence of aortic regurgitation in elite strength trained athletes. *Am J Cardiol*. 2007;100: 528–530.

63. D'Andrea A, Cocchia R, Riegler L, Scarafie R, Salerno G, Gravino R, Vriz O, Citro R, Limongelli G, Di Salvo G, Cuomo S, Caso P, Russo MG, Calabro R, Bossone E. Aortic root dimensions in elite athletes. *Am J Cardiol*. 2010;105:1629–1634.
64. Gabriel H, Kindermann W. Ultrasound of the abdomen in endurance athletes. *Eur J Appl Physiol Occup Physiol*. 1996;73:191–193.
65. Pelliccia A, Di Paolo FM, De Blasiis E, Quattrini FM, Pisicchio C, Guerra E, Culasso F, Maron BJ. Prevalence and clinical significance of aortic root dilation in highly trained competitive athletes. *Circulation*. 2010;122:698–706.
66. Kinoshita N, Mimura J, Obayashi C, Katsukawa F, Onishi S, Yamazaki H. Aortic root dilatation among young competitive athletes: echocardiographic screening of 1929 athletes between 15 and 34 years of age. *Am Heart J*. 2000;139:723–728.
67. Hoglund C. Enlarged left atrial dimension in former endurance athletes: an echocardiographic study. *Int J Sports Med*. 1986;7:133–136.
68. Hoogsteen J, Hoogeveen A, Schaffers H, Wijn PF, van der Wall EE. Left atrial and ventricular dimensions in highly trained cyclists. *Int J Cardiovasc Imaging*. 2003;19:211–217.
69. Pelliccia A, Maron BJ, Di Paolo FM, Biffi A, Quattrini FM, Pisicchio C, Roselli A, Caselli S, Culasso F. Prevalence and clinical significance of left atrial remodeling in competitive athletes. *J Am Coll Cardiol*. 2005;46:690–696.
70. D'Andrea A, Riegler L, Cocchia R, Scarafie R, Salerno G, Gravino R, Golia E, Vriz O, Citro R, Limongelli G, Calabro P, Di Salvo G, Caso P, Russo MG, Bossone E, Calabro R. Left atrial volume index in highly trained athletes. *Am Heart J*. 2010;159:1155–1161.
71. 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;276:211–215.
72. George KP, Wolfe LA, Burggraf GW, Norman R. Electrocardiographic and echocardiographic characteristics of female athletes. *Med Sci Sports Exerc*. 1995;27:1362–1370.
73. Sun B, Ma JZ, Yong YH, Lv YY. The upper limit of physiological cardiac hypertrophy in elite male and female athletes in China. *Eur J Appl Physiol*. 2007;101:457–463.
74. 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;25:380–383.
75. Haykowsky M, Chan S, Bhambhani Y, Syrotuik D, Quinney H, Bell G. Effects of combined endurance and strength training on left ventricular morphology in male and female rowers. *Can J Cardiol*. 1998;14:387–391.
- 75a. Basavarajiah S, Boraita A, Whyte G, Wilson M, Carby L, Shah A, Sharma S. Ethnic differences in left ventricular remodeling in highly-trained athletes: relevance to differentiating physiologic left ventricular hypertrophy from hypertrophic cardiomyopathy. *J Am Coll Cardiol*. 2008;51:2256–2262.
- 75b. Rawlins J, Carre F, Kervio G, Papadakis M, Chandra N, Edwards C, Whyte GP, Sharma S. Ethnic differences in physiological cardiac adaptation to intense physical exercise in highly trained female athletes. *Circulation*. 2010;121:1078–1085.
76. Magalski A, Maron BJ, Main ML, McCoy M, Florez A, Reid KJ, Epps HW, Bates J, Browne JE. Relation of race to electrocardiographic patterns in elite American football players. *J Am Coll Cardiol*. 2008;51:2250–2255.
77. Maron BJ. Sudden death in young athletes. *N Engl J Med*. 2003;349:1064–1075.
78. Maron BJ, Pelliccia A, Spirito P. Cardiac disease in young trained athletes: insights into methods for distinguishing athlete's heart from structural heart disease, with particular emphasis on hypertrophic cardiomyopathy. *Circulation*. 1995;91:1596–1601.
79. Pela G, Bruschi G, Montagna L, Manara M, Manca C. Left and right ventricular adaptation assessed by Doppler tissue echocardiography in athletes. *J Am Soc Echocardiogr*. 2004;17:205–211.
80. Zoncu S, Pelliccia A, Mercurio G. Assessment of regional systolic and diastolic wall motion velocities in highly trained athletes by pulsed wave Doppler tissue imaging. *J Am Soc Echocardiogr*. 2002;15:900–905.
81. Matsumura Y, Elliott PM, Virdee MS, Sorajja P, Doi Y, McKenna WJ. Left ventricular diastolic function assessed using Doppler tissue imaging in patients with hypertrophic cardiomyopathy: relation to symptoms and exercise capacity. *Heart*. 2002;87:247–251.
82. McMahon CJ, Nagueh SF, Pignatelli RH, Denfield SW, Dreyer WJ, Price JF, Clunie S, Bezold LI, Hays AL, Towbin JA, Eidem BW. Characterization of left ventricular diastolic function by tissue Doppler imaging and clinical status in children with hypertrophic cardiomyopathy. *Circulation*. 2004;109:1756–1762.
83. Nagueh SF, Bachinski LL, Meyer D, Hill R, Zoghbi WA, Tam JW, Quinones MA, Roberts R, Marian AJ. 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;104:128–130.
84. Kato TS, Noda A, Izawa H, Yamada A, Obata K, Nagata K, Iwase M, Murohara T, Yokota M. Discrimination of nonobstructive hypertrophic cardiomyopathy from hypertensive left ventricular hypertrophy on the basis of strain rate imaging by tissue Doppler ultrasonography. *Circulation*. 2004;110:3808–3814.
85. Galderisi M, Lomoriello VS, Santoro A, Esposito R, Olibet M, Raia R, Dario Di Minno MN, Guerra G, Mele D, Lombardi G. Differences of myocardial systolic deformation and correlates of diastolic function in competitive rowers and young hypertensives: a speckle-tracking echocardiography study. *J Am Soc Echocardiogr*. 2010;23:1190–1198.
86. Prakken NH, Velthuis BK, Teske AJ, Mosterd A, Mali WP, Cramer MJ. Cardiac MRI reference values for athletes and nonathletes corrected for body surface area, training hours/week and sex. *Eur J Cardiovasc Prev Rehabil*. 2010;17:198–203.
87. Maron MS, Appelbaum E, Harrigan CJ, Buros J, Gibson CM, Hanna C, Lesser JR, Udelson JE, Manning WJ, Maron BJ. Clinical profile and significance of delayed enhancement in hypertrophic cardiomyopathy. *Circ Heart Fail*. 2008;1:184–191.
88. Rubinstein R, Glockner JF, Ommen SR, Araoz PA, Ackerman MJ, Sorajja P, Bos JM, Tajik AJ, Valeti US, Nishimura RA, Gersh BJ. Characteristics and clinical significance of late gadolinium enhancement by contrast-enhanced magnetic resonance imaging in patients with hypertrophic cardiomyopathy. *Circ Heart Fail*. 2010;3:51–58.
89. Prakken NH, Velthuis BK, Cramer MJ, Mosterd A. Advances in cardiac imaging: the role of magnetic resonance imaging and computed tomography in identifying athletes at risk. *Br J Sports Med*. 2009;43:677–684.
90. Pelliccia A, Fagard R, Bjornstad HH, Anastassakis A, Arbustini E, Assanelli D, Biffi A, Borjesson M, Carre F, Corrado D, Delise P, Dorwarth U, Hirth A, Heidebuchel H, Hoffmann E, Mellwig KP, Panhuyzen-Goedkoop N, Pisani A, Solberg EE, van-Buuren F, Vanhees L, Blomstrom-Lundqvist C, Deligiannis A, Dugmore D, Glikson M, Hoff PI, Hoffmann A, Hoffmann E, Horstkotte D, Nordrehaug JE, Oudhof J, McKenna WJ, Penco M, Priori S, Reybrouck T, Senden J, Spataro A, Thiene G. Recommendations for competitive sports participation in athletes with cardiovascular disease: a consensus document from the Study Group of Sports Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*. 2005;26:1422–1445.
91. Zipes DP, Ackerman MJ, Estes NA 3rd, Grant AO, Myerburg RJ, Van Hare G. Task Force 7: arrhythmias. *J Am Coll Cardiol*. 2005;45:1354–1363.
92. Ector H, Bourgeois J, Verlinden M, Hermans L, Vanden Eynde E, Fagard R, De Geest H. Bradycardia, ventricular pauses, syncope, and sports. *Lancet*. 1984;2:591–594.
93. Stein R, Medeiros CM, Rosito GA, Zimmerman LI, Ribeiro JP. Intrinsic sinus and atrioventricular node electrophysiologic adaptations in endurance athletes. *J Am Coll Cardiol*. 2002;39:1033–1038.
94. Zeppilli P, Fenici R, Sassara M, Pirrami MM, Caselli G. Wenckebach second-degree A-V block in top-ranking athletes: an old problem revisited. *Am Heart J*. 1980;100:281–294.
95. Barold S, Padeletti L. Mobitz type II second-degree atrioventricular block in athletes: true or false? *Br J Sports Med*. September 18, 2008. DOI: 10.1136/bjsm.2008.047365. <http://bjsm.bmj.com/ezp-prod1.hul.harvard.edu/content/early/2011/04/21/bjsm.2008.047365.full?sid=fda404f7-e58e-4441-a7d9-9fe4e13d7c98>. Accessed November 5, 2010.
96. Biffi A, Pelliccia A, Verdile L, Fernando F, Spataro A, Caselli S, Santini M, Maron BJ. Long-term clinical significance of frequent and complex ventricular tachyarrhythmias in trained athletes. *J Am Coll Cardiol*. 2002;40:446–452.
97. Biffi A, Maron BJ, Verdile L, Fernando F, Spataro A, Marcello G, Ciardo R, Ammirati F, Colivicchi F, Pelliccia A. Impact of physical deconditioning on ventricular tachyarrhythmias in trained athletes. *J Am Coll Cardiol*. 2004;44:1053–1058.
98. Baldesberger S, Bauersfeld U, Candinas R, Seifert B, Zuber M, Ritter M, Jenni R, Oechslin E, Luthi P, Scharf C, Marti B, Attenhofer Jost CH.

- Sinus node disease and arrhythmias in the long-term follow-up of former professional cyclists. *Eur Heart J*. 2008;29:71–78.
99. Biffi A, Maron BJ, Di Giacinto B, Porcaccia P, Verdile L, Fernando F, Spataro A, Culasso F, Casasco M, Pelliccia A. Relation between training-induced left ventricular hypertrophy and risk for ventricular tachyarrhythmias in elite athletes. *Am J Cardiol*. 2008;101:1792–1795.
 100. Sorokin AV, Araujo CG, Zweibel S, Thompson PD. Atrial fibrillation in endurance-trained athletes. *Br J Sports Med*. 2011;45:185–188.
 101. Mont L, Sambola A, Brugada J, Vacca M, Marrugat J, Elosua R, Pare C, Azqueta M, Sanz G. Long-lasting sport practice and lone atrial fibrillation. *Eur Heart J*. 2002;23:477–482.
 102. Molina L, Mont L, Marrugat J, Berrueto A, Brugada J, Bruguera J, Rebato C, Elosua R. Long-term endurance sport practice increases the incidence of lone atrial fibrillation in men: a follow-up study. *Europace*. 2008;10:618–623.
 103. Karjalainen J, Kujala UM, Kaprio J, Sarna S, Viitasalo M. Lone atrial fibrillation in vigorously exercising middle aged men: case-control study. *BMJ*. 1998;316:1784–1785.
 104. Basavarajiah S, Mekan J, Naghavi SH, Whyte G, Gati S, Sharma S. Physiological upper limits of left atrial diameter in highly trained adolescent athletes. *J Am Coll Cardiol*. 2006;47:2341–2342.
 105. Alboni P, Botto GL, Baldi N, Luzi M, Russo V, Gianfranchi L, Marchi P, Calzolari M, Solano A, Baroffio R, Gaggioli G. Outpatient treatment of recent-onset atrial fibrillation with the “pill-in-the-pocket” approach. *N Engl J Med*. 2004;351:2384–2391.
 106. Furlanello F, Lupo P, Pittalis M, Foresti S, Vitali-Serdoz L, Francia P, De Ambroggi G, Ferrero P, Nardi S, Inama G, De Ambroggi L, Cappato R. Radiofrequency catheter ablation of atrial fibrillation in athletes referred for disabling symptoms preventing usual training schedule and sport competition. *J Cardiovasc Electrophysiol*. 2008;19:457–462.
 107. Calvo N, Mont L, Tamborero D, Berrueto A, Viola G, Guasch E, Nadal M, Andreu D, Vidal B, Sitges M, Brugada J. Efficacy of circumferential pulmonary vein ablation of atrial fibrillation in endurance athletes. *Europace*. 2010;12:30–36.
 108. Colivicchi F, Ammirati F, Santini M. Epidemiology and prognostic implications of syncope in young competing athletes. *Eur Heart J*. 2004;25:1749–1753.
 109. Basso C, Maron BJ, Corrado D, Thiene G. Clinical profile of congenital coronary artery anomalies with origin from the wrong aortic sinus leading to sudden death in young competitive athletes. *J Am Coll Cardiol*. 2000;35:1493–1501.
 110. Di Girolamo E, Di Iorio C, Leonzio L, Sabatini P, Barsotti A. Usefulness of a tilt training program for the prevention of refractory neurocardiogenic syncope in adolescents: a controlled study. *Circulation*. 1999;100:1798–1801.
 111. Grubb BP. Clinical practice: neurocardiogenic syncope. *N Engl J Med*. 2005;352:1004–1010.
 112. WADA. The prohibited list. 2010. <http://www.wada-ama.org>. Accessed January 3, 2011.
 113. Kiraly CL. Androgenic-anabolic steroid effects on serum and skin surface lipids, on red cells, and on liver enzymes. *Int J Sports Med*. 1988;9:249–252.
 114. Riebe D, Fernhall B, Thompson PD. The blood pressure response to exercise in anabolic steroid users. *Med Sci Sports Exerc*. 1992;24:633–637.
 115. Nottin S, Nguyen LD, Terbah M, Obert P. Cardiovascular effects of androgenic anabolic steroids in male bodybuilders determined by tissue Doppler imaging. *Am J Cardiol*. 2006;97:912–915.
 116. D'Andrea A, Caso P, Salerno G, Scarafile R, De Corato G, Mita C, Di Salvo G, Severino S, Cuomo S, Liccardo B, Esposito N, Calabro R. Left ventricular early myocardial dysfunction after chronic misuse of anabolic androgenic steroids: a Doppler myocardial and strain imaging analysis. *Br J Sports Med*. 2007;41:149–155.
 117. Krieg A, Scharhag J, Albers T, Kindermann W, Urhausen A. Cardiac tissue Doppler in steroid users. *Int J Sports Med*. 2007;28:638–643.
 118. Baggish AL, Weiner RB, Kanayama G, Hudson JI, Picard MH, Hutter AM Jr, Pope HG Jr. Long-term anabolic-androgenic steroid use is associated with left ventricular dysfunction. *Circ Heart Fail*. 2010;3:472–476.
 119. Maron BJ, Gohman TE, Aeppli D. Prevalence of sudden cardiac death during competitive sports activities in Minnesota high school athletes. *J Am Coll Cardiol*. 1998;32:1881–1884.
 120. Corrado D, Basso C, Thiene G. Arrhythmogenic right ventricular cardiomyopathy: diagnosis, prognosis, and treatment. *Heart*. 2000;83:588–595.
 121. Maron BJ, Zipes DP. Introduction: eligibility recommendations for competitive athletes with cardiovascular abnormalities: general considerations. *J Am Coll Cardiol*. 2005;45:1318–1321.
 122. Pelliccia A, Zipes DP, Maron BJ. Bethesda Conference #36 and the European Society of Cardiology consensus recommendations revisited: a comparison of U.S. and European criteria for eligibility and disqualification of competitive athletes with cardiovascular abnormalities. *J Am Coll Cardiol*. 2008;52:1990–1996.
 123. Maron BJ, Thompson PD, Ackerman MJ, Balady G, Berger S, Cohen D, Dimeff R, Douglas PS, Glover DW, Hutter AM Jr, Krauss MD, Maron MS, Mitten MJ, Roberts WO, Puffer JC. 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;115:1643–1455.
 124. Corrado D, Pelliccia A, Bjornstad HH, Vanhees L, Biffi A, Borjesson M, Panhuyzen-Goedkoop N, Deligiannis A, Solberg E, Dugmore D, Mellwig KP, Assanelli D, Delise P, van-Buuren F, Anastasakis A, Heidbuchel H, Hoffmann E, Fagard R, Priori SG, Basso C, Arbustini E, Blomstrom-Lundqvist C, McKenna WJ, Thiene G. Cardiovascular preparticipation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol: consensus statement of the Study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the Working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*. 2005;26:516–524.
 125. Corrado D, Basso C, Pavei A, Michieli P, Schiavon M, Thiene G. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. *JAMA*. 2006;296:1593–1601.
 126. Baggish AL, Hutter AM Jr, Wang F, Yared K, Weiner RB, Kupperman E, Picard MH, Wood MJ. Cardiovascular screening in college athletes with and without electrocardiography: a cross-sectional study. *Ann Intern Med*. 2010;152:269–275.

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