©Journal of Sports Science and Medicine (2007) **6,** 166-171
http://www.jssm.org

Review article

The role of echocardiography in the differential diagnosis between training induced myocardial hypertrophy *versus* cardiomyopathy

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Abstract
Increased myocardial mass due to regular high-volume intense exercise training (so-called athlete's heart) is not uncommon. Although directly correlated with the extent of training loads, myocardial hypertrophy is not present exclusively in well-trained or elite athletes. Athlete's heart is considered a physiological phenomenon with no known harmful consequences. However, extreme forms of myocardial hypertrophy due to endurance training resemble a structural heart disease such as hypertrophic cardiomyopathy, a condition associated with substantially increased risk of cardiac event. Endurance sports such as rowing and road cycling, rather than strength/power training, are most commonly associated with left ventricular (LV) wall thickness compatible with hypertrophic cardiomyopathy. The differentiation between physiological and maladaptive cardiac hypertrophy in athletes is undoubtedly important, since untreated cardiac abnormality often possesses a real threat of premature death due to heart failure during intense physical exertion. Luckily, the distinction from pathological hypertrophy is usually straightforward using transthoracic echocardiography, as endurance athletes, in addition to moderately and proportionally thickened LV walls with normal acoustic density, tend to possess increased LV diameter. In more uncertain cases, a detailed evaluation of myocardial function using (tissue) Doppler and contrast echocardiography is effective. When a doubt still remains, knowledge of an athlete's working capacity may be useful in evaluating whether the insidious cardiac pathology is absent. In such cases cardiopulmonary exercise testing typically resolves the dilemma: indices of aerobic capacity are markedly higher in healthy endurance athletes compared to patients. Other characteristics such as a decrease of LV mass due to training cessation are also discussed in the article.

Key words: Left ventricle, physical exercises, physiological adaptation.

Introduction

The heart of moderately increased mass and high working capacity triggered by the repetitive cardiac overload which is induced by regular exercise training without any serious valvular or other functional disorders, is called an athlete's heart (Firoozi et al., 2003; Scharhag et al., 2002; Shephard, 1996). In endurance athletes, myocardial hypertrophy usually develops due to both the dilation of its cavities and the thickening of its walls, though the latter usually predominates in relative terms (McCann et al., 2000). The extent of these changes varies depending on many factors, but left ventricular (LV) mass in athletes is usually within twice of the size of sedentary but otherwise healthy people (Hildick-Smith and Shapiro, 2001). Strength/power-trained sportsmen usually possess cardiac dimensions in proportion to their fat-free body mass, and chamber dilation is especially rare in these subjects, but other factors, for example, intake of large doses of anabolic steroids may stimulate LV mass gain in these athletes (Urhausen and Kindermann, 1992).

An agreement about the athlete's heart as a physiological phenomenon exists (Akova et al., 2005; De Castro et al., 2006, 2007; Iemitsu et al., 2003; Rodriguez Reguero et al., 1995; Sharma et al., 2000; Strom et al., 2005). In a symmetric (both approximately equal dilation and wall thickening) type of LV hypertrophy due to endurance training, the lifespan of cardiomyocytes does not shorten and thus their number does not decrease quicker (Katz and Lorell, 2000; Lips et al., 2003). The activity of myosin- and Ca^{2+}-ATPase in athletes' cardiac myocytes is increased, while it declines in patients with continuous myocardial volume or pressure overload. In athlete's heart, this is reflected in better inotropy, lusitropy, and augmented cardiac pumping capacity (Katz and Lorell, 2000), which are clearly an advantage. As an evidence of the benign nature of the athlete's heart may be longer life expectancy in former elite endurance athletes as compared with healthy untrained subjects (Sarna et al., 1997).

Exercise training *per se* is not a sufficient stimulus to damage a normal heart (Oakley, 2001; Wilkins et al., 2004), but if hypertrophic cardiomyopathy (HCM) or a similar condition is present, the risk of a sudden cardiac event during intense physical exertion is high (Sharma, 2003). Being the most common inherited cardiac disease, HCM affects about one per 500 humans, the athletes and the non-athletic populations equally (Maron, 2002; 2005). The tragedy of a sudden cardiac death is rare in athletes. The cause of the majority of such highly visible events is an underlying serious cardiac pathology, which is usually asymptomatic and not diagnosed (Firoozi et al., 2003; Maron, 2003). In young athletes the lethal pathology is most frequently inherited, while in those older than 35 years it may also be acquired (coronary artery sclerosis being the most common) (Maron, 2003). Apparently, it is HCM that is the most frequent cause of the exercise-related cardiac arrest in young athletes and it is responsi-

Received: 27 November 2006 / Accepted: 15 March 2007 / Published (online): 01 June 2007

ble for approximately one third of the sudden deaths in this major group of the athletic population (Firoozi et al., 2003; Maron, 2003; 2005). Accordingly, when a serious cardiac pathology is established, the affected subject, in addition to other necessary preventive treatments, should also be considered to be barred from intense athletic training and competition to possibly reduce the likelihood of a sudden cardiovascular death.

Thus, an increased cardiac mass is not necessarily a sign of a disease or a disease itself. Instead, it may be a desirable functional adaptation to a physiological stimulus, such as regular endurance exercise training, and may enable an athlete to reach higher competitive level when young, as well as to remain independent longer when old (Barbier et al., 2006; Shephard, 1996). The aim of this paper was to review and discuss the echocardiographic differentiation criteria in diagnosis between athlete's heart and inherited cardiac disease, mainly hypertrophic cardiomyopathy.

Transthoracic echocardiography

Transthoracic two-dimensional M-mode and Doppler echocardiography has made substantial contribution and revealed itself as a useful non-invasive tool for differentiating the extreme phenotypes of physiological athlete's heart from cardiac pathology, particularly HCM, which manifests with both structural alterations and malfunctioning (Maron, 2005; Pelliccia et al., 2002; Rajiv et al., 2004; Sharma, 2003; Whyte et al., 2004). Given the limitations (e.g. expensive procedures) of other diagnostic techniques, transthoracic echocardiography remains the principal means in distinguishing between adaptive and maladaptive cardiac hypertrophy. In the majority of countries, routine echocardiographic testing is, however, not mandatory for athletic population, and is performed primarily when sportsmen are directed due to abnormal screening results (e.g. ECG). This can also happen when a physician, a coach, parents and/or athletes themselves show a desire to undertake the procedure; or when they are recruited into scientific study as volunteers.

Whereas the increase in heart mass due to exercise training usually is not so profound (in fact, not always present) as in HCM patients, a fair proportion of athletes still possesses a myocardial structure similar to that of patients with mild phenotypic expression of HCM (Maron, 2003; 2005; Whyte et al., 2004). The discrimination between benign athlete's heart and pathological cardiac hypertrophy is obviously important: in addition to the undisputed significance to the individual, the differentiation and subsequent measures are also important because the unnecessary furor drawn by a sports-related sudden cardiac death following a serious condition overlooked may serve as a deterrent from exercise for the general population. Furthermore, familial evaluation should follow the established cardiomyopathy to detect and treat affected relatives. So, what are the criteria behind this differential diagnosis and to what extent can echocardiography be useful in this dilemma?

First of all, it is important to delineate the extent and type of a cardiac hypertrophic response to athletic conditioning. Concentric type of myocardial hypertrophy is characterized as an increase in LV mass with an augmented relative wall thickness (RWT, which is the ratio of the sum of posterior wall thickness and interventricular septum thickness to the LV diameter) above arbitrary 0.42 or 0.45 value, whereas eccentric hypertrophy is an increased LV mass without augmented RWT (Haykowsky et al., 2002). It is usually both processes of chamber dilation and wall thickening that occur during long-term adaptation to serious athletic training, that's why the term 'symmetric' remodelling (De Castro et al., 2006).

Eccentric cardiac hypertrophy

Large-sample studies have shown that a significant proportion of endurance athletes exceed the 'normal upper limit' (55 mm) for LV end-diastolic internal diameter (Pelliccia et al., 1991; Spirito et al., 1994; Urhausen et al., 1997). Perhaps the most striking results were obtained by the authors who detected that more than one-half of small-body-size male 100-km runners had LV diameter in excess of 60 mm (Nagashima et al., 2003). In addition, 33 (13%) of their subjects showed LV dilation with LV diameter of 70 to 75 mm. LV diameter was over 55 mm in 55% of male rowers (Pelliccia and Maron, 1997). Among high-level athletes from different sports, LV diameter was measured to be 60 to 70 mm in 6% (Whyte et al., 2004) and in 14% (Pelliccia et al., 1991) of male Caucasians. LV systolic and diastolic function was normal or supernormal in all individual cases (Pelliccia et al., 1991). Others have reported maximum values of 67 mm in the rower (Urhausen and Kindermann, 1992), and 70 mm in the cyclist (Rost, 1997) with no signs or symptoms of pathology. These and similar situations represent examples of the extreme physiological adaptation to endurance conditioning.

In athletes with substantially dilated LV, the differentiation from dilated cardiomyopathy is relatively simple, and the latter condition can be eliminated simply by reporting well-tolerated athletic activity. Uncertainty may be completely dispelled if increased systolic function in response to physical exertion is observed (Firoozi et al., 2003).

Concentric cardiac hypertrophy

The increase in RWT is rather frequently observed in response to aerobic conditioning (Palazzuoli et al., 2002; Venckunas et al., 2005, 2006), making this cardiac hypertrophic index higher in endurance athletes such as distance runners (normally about 0.40) than in healthy non-athletes (about 0.35) (Urhausen et al., 1997). However, when an individual possesses a truly concentric myocardial hypertrophy (RWT in excess of 0.45), the investigator should carry out more thorough evaluation rather than stop short at taking the measurements of heart structure (Pelliccia and Maron, 1997; Urhausen et al., 1997).

LV posterior wall thickness higher than 13-14 mm (values compatible with HCM), especially without chamber dilation, is rare in athletes and should not be regarded as an adaptation due to athletic training (Douglas et al., 1997; Fagard, 2003; Henriksen et al., 1996; Maron, 2005; Pelliccia et al., 1991; Pelliccia and Maron, 1997; Ur-

hausen et al., 1997; Whyte et al., 2004). Sportswomen usually possess LV wall thicknesses below 11 mm (Maron, 2005). Maximum values reported in male (elite) athletes were 16 mm in rowers and cyclists (Pelliccia et al., 1991), and 19 mm in a road cyclist (Rodriguez Reguero et al., 1995). Thus, a higher percentage of professional male road cyclists, skiers, rowers, paddlers, and probably other athletes from sports where large muscle mass is involved into ('more isometric') training, are subjected to the differential diagnosis for HCM. Here the importance of proper differentiation comes into play in order to limit the rate of false positive (as well as false negative) diagnosis of HCM and subsequent unwarrantable disqualification from physical activity on the one hand, and prevent tragic events of a sudden cardiac death in a field on the other hand.

In athletes with LV wall thickness ≥13 mm, the following main echocardiographic criteria (Table 1) are suggested for making a distinction between the adaptive *vs.* pathological nature of cardiac hypertrophy:

Cardiac chambers are enlarged in athletes (Fagard et al., 1989; George et al., 1991; Henriksen et al., 1996; Rost, 1997; Venckunas et al., 2006), while in HCM the heart is hypertrophied asymmetrically with the LV chamber rarely increased in size (D'Andrea et al., 2006; Firoozi et al., 2003; Maron, 2002; 2003): its end-diastolic diameter is often below 50 mm in HCM, but frequently above 55 mm in elite male endurance athletes (Maron, 2005; Pelliccia et al., 1991). Marked left atrial enlargement is inherent to the HCM-affected heart (Firoozi et al., 2003; Maron, 2005), while in the athletic heart all cardiac chambers are enlarged proportionally (Barbier et al., 2006; George et al., 1991; Hauser et al., 1985; Pelliccia et al., 2005; Rost, 1997). Mitral valve systolic anterior motion is never present in athlete's heart (Maron, 2005).

Cardiac acoustic density, in contrast to cases of HCM, is normal in healthy athletes (Giorgi et al., 2000; Hildick-Smith and Shapiro, 2001; Lattanzi et al., 1992). In HCM, ultrasonic myocardial reflectivity is postulated to be due to increased collagen content and/or profound muscle fiber disarray (Frenneaux, 2004; Lattanzi et al., 1992). Incidentally, acoustic properties were also shown to be unchanged in patients with myocardial hypertrophy due to hypertension (Gigli et al., 1993), though the correct diagnosis in these individuals is usually easily performed by serial blood pressure measurements. In addition, strain rate analysis could be used to discern myocardial wall thickening due to athletic conditioning from cardiac hypertrophy due to hypertension (Saghir et al., 2007).

Interventricular septum thickness is usually above 16 mm in patients with HCM (Firoozi et al., 2003). In the absence of cardiac pathology, the interventricular septum to LV posterior wall thickness ratio is usually less than 1.3 (Douglas et al., 1997; Hildick-Smith and Shapiro 2001). When interventricular septum motions, shape, thickness, and its ratio to LV posterior wall thickness together with global ejection fraction are normal, the diagnosis of HCM can almost certainly be denied (Douglas et al., 1997; Urhausen et al., 1997; Whyte et al., 2004).

Left ventricular diastolic function assessed by pulsed or tissue Doppler is preserved or even improved (especially during exercise but sometimes at rest as well) in athletes free of HCM or other heart diseases, be hypertrophy present or absent (Cardim et al., 2003; D'Andrea et al., 2006; Fagard et al., 1989; Hildick-Smith and Shapiro, 2001; Libonati, 2000; Maron, 2005; Pelliccia et al., 1991; Pelliccia and Maron, 1997; Rajiv et al., 2004; Urhausen et al., 1997). Diastolic LV function is often impaired in patients and their resting E/A ratio, still a most frequently used index of diastolic function, may be ~1 or lower.

In addition, coronary vascular architecture and flow reserve as possible criteria of the nature of the cardiac enlargement have also been suggested (Hildick-Smith and Shapiro, 2001; Indermuhle et al., 2006), and contrast echocardiography (Indermuhle et al., 2006), as well as myocardial Doppler imaging (D'Andrea et al., 2006; Rajiv et al., 2004) and three-dimensional echocardiography (De Castro et al., 2006, 2007) have recently emerged as valuable tools for the differentiation discussed.

Besides and beyond resting echocardiography

Still, the differentiation between pathological and physiological nature of the hypertrophy athletes can sometimes be problematic without a more complex examination and integrated approach (Sharma et al., 2000). One of the reasons is that exercise training with its famous beneficial effects on the organism, including the cardiovascular system, may camouflage the disease (Konhilas et al., 2006). That is also why the following potentially useful

Table 1. Typical echocardiographic findings in physiological (athlete's heart) and pathological (hypertrophic cardiomyopathy) cardiac hypertrophy.

Echocardiographic index	Athlete's heart	Hypertrophic cardiomyopathy
Left ventricular chamber size (diameter)	Normal / slightly enlarged	Normal / decreased
Mitral valve systolic anterior motion	Absent	Present
Acoustic density of the myocardial wall	Normal	Increased
Interventricular septum thickness	< 16 mm	> 16 mm
Interventricular septum thickness ratio to posterior wall thickness	< 1.3	> 1.3
Interventricular septum motion and shape	Regular	Irregular
Left ventricular diastolic function	Normal / improved	Decreased
Ejection fraction	Normal	Depressed
Left atrial volume	Increased moderately in proportion to other cardiac chambers	Enlarged markedly

differentiation criteria should be regarded with care:

Resting *heart rate* is usually lower than 60 bpm in endurance athletes (Fagard, 2003; Firoozi et al., 2003; Oakley, 2001; Palazzuoli et al., 2002; Venckunas et al., 2006), while sedentary people, including HCM patients, do not tend to possess bradycardia.

Physical activity habits. The amount, the intensity, as well as the type of physical training must be carefully evaluated. The pronounced LV hypertrophy in a recreationally active and thus moderately fit subject should raise a suspicion and prompt a more detailed examination of possible pathological causes (Urhausen and Kindermann, 1992). On the other hand, well-tolerated vigorous exercise training suggests no cardiac anomalies.

Left ventricular systolic function response to exercise. During physical exertion, LV ejection fraction increases substantially in healthy athletes, in contrast to patients with HCM, and during all-out exercise sportsmen reach stroke volumes proportional to their LV mass, (Firoozi et al., 2003; Urhausen and Kindermann, 1992). Also, LV diastolic diameter was demonstrated to increase during veloergometric exercise in marathon runners but not healthy non-athletes (Fagard et al., 1989).

Objective measurements of *exercise competence*. The ratio of working capacity to myocardial size is of utmost importance: special attention should be paid to athletes if their LV mass increases without improvement or even with decline in competitive results and ergometric/aerobic capacity (Pelliccia and Maron, 1997; Rost, 1997; Urhausen and Kindermann, 1992; Urhausen et al., 1997).

Maximal oxygen uptake is usually less than 45 $ml \cdot kg^{-1} \cdot min^{-1}$ and less than 100% of predicted in the subjects with HCM, even if they are engaged in regular exercise training, while healthy endurance athletes typically possess values above 50 $ml \cdot kg^{-1} \cdot min^{-1}$ (Hildick-Smith and Shapiro, 2001; Maron, 2005; Sharma et al., 2000b).

Healthy high-level endurance athletes possess much higher *anaerobic threshold* than patients (Anastasakis et al., 2005; Sharma et al., 2000), as well as higher *peak oxygen pulse*, which is usually well above 20 $ml \cdot beat^{-1}$ (Sharma et al., 2000b).

In exceedingly rare cases, any type of echocardiography and cardiopulmonary exercise testing still may fail to give the ultimate solution. For instance, although adolescent athletes develop moderate LV hypertrophy with wall thickness not exceeding 14 mm (Makan et al., 2005), diagnostics in these subjects may be particularly complicated, because at an early age HCM may be in its early phenotypic stage, with only mildly affected myocardial structure and function. Similarly, the differentiation between dilated cardiomyopathy and athlete's heart in veteran (master) athletes may be more complicated (Kasikcioglu et al., 2006). Master athletes compose a relatively smaller and less investigated group of athletic population and they may sometimes need specific detailed examination to identify the cause of the marked chamber dilation. Even 'mild' cardiomyopathy phenotype is believed to predispose a subject to a serious life threat (Frenneaux, 2004), especially if agonistic sporting activity was continued. In such cases, endomyocardial biopsy for subsequent histological/immunological analysis could be applied, and genetic testing for the mutations in cardiomyopathy-causing genes (Maron et al., 2004; Rajiv et al., 2004) would seem to be useful. Unfortunately, these procedures are not easily implemented in practice. That is why in such a situation the interruption of training process can be more relevant – the physiological hypertrophy of athlete's heart reverses in response to detraining (Hildick-Smith and Shapiro, 2001; Pelliccia et al, 1991; 2002) with LV mass declining substantially within weeks (Mujika and Padilla, 2001), while the hypertrophy due to HCM or similar hereditary pathology is reluctant to deconditioning (Maron, 2005). However, in some, if not the majority, of the athletes the attempts to cease the usual training may fail due to high athletic pursuits.

Conclusion

With ever advancing equipment, increasing body of data obtained from both patients and athletes, and improving knowledge of differentiation techniques, transthoracic echocardiography remains a diagnostic tool-of-choice in distinguishing between cardiomyopathy and athlete's heart. The review paper has revealed that conventional echocardiographic criteria (left ventricular chamber diameter, wall thickness, ejection fraction, E/A ratio, etc.) may be successfully used to distinguish between pathological and physiological cardiac hypertrophy. In addition, parameters of Doppler tissue imaging, contrast and three-dimensional echocardiography are valuable techniques in more problematic diagnostic cases. However, when a thorough echocardiographic examination leaves an uncertainty concerning the nature of cardiac hypertrophy, the evaluation of the aerobic capacity and myocardial size changes in response to detraining may be useful in a decision-making.

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Key points

- Transthoracic echocardiography is still the most common relevant differentiation technique applied to distinguish athlete's heart from the cardiomyopathy.
- Conventional echocardiographic criteria such as left ventricular chamber size and diastolic function parameters are to be regarded first when making differential diagnosis between substantially increased wall thickness in athlete's heart (i.e. physiological adaptation) versus a disease (usually hypertrophic cardiomyopathy).
- When conventional echocardiographic parameters fail to diagnose the nature of myocardial hypertrophy, other differentiation criteria such as aerobic fitness, cardiac performance in response to physical exertion, and changes in echocardiographic parameters due to detraining, must be taken into consideration.
- Tissue Doppler, contrast and three-dimensional imaging are state-of-the-art echocardiographic techniques which have recently appeared in the differential diagnostics.

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