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Keith P. George · Alan M. Batterham · Bryan Jones

Echocardiographic evidence of concentric left ventricular enlargement in female weight lifters

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Abstract In this study we investigated resting left ventricular structure and function in elite female weightlifters. Fifteen National Squad members [mean age (SD) 25 (6) years] were compared to a recreationally active control group [$n = 46$, 23 (3) years]. Subjects were matched for body mass, body surface area and fat free mass, but the controls were slightly taller ($P < 0.01$). Athletes and controls demonstrated similar resting heart rates and blood pressures. Septal wall (ST), posterior wall (PWT) and left ventricular internal dimension in diastole and systole (LVIDd and LVIDs, respectively) were measured from M-mode echocardiograms. Calculations were made for left ventricular mass (LVM), mass-volume ratio (m:V), wall-thickness-cavity dimension ratio (h:R) and systolic function. Left ventricular filling velocities were determined via Doppler echocardiography. ST [9.0 (1.1) vs 7.7 (1.0) mm] and PWT [8.7 (1.4) vs 7.5 (1.3) mm] were greater, whereas LVIDd [46.2 (2.8) vs 48.4 (3.4) mm] was smaller in the weightlifters ($P < 0.05$). After allometrically adjusting for differences in height, the weightlifters had a greater ST, PWT and LVM ($P < 0.05$) and similar LVIDd. Both m:V and h:R were increased in the weightlifters ($P < 0.05$). All functional data were within normal limits and no group differences were observed. The female weight-lifters demonstrated a concentric left ventricular enlargement that was not detrimental to left ventricular performance at rest.

Key words Left ventricular mass · Body dimensions · Scaling · Diastolic inflow

K.P. George (✉) · A.M. Batterham · B. Jones
Department of Exercise and Sport Science,
The Manchester Metropolitan University,
Crewe and Alsager Faculty, Hassall Road,
Alsager, Cheshire ST7 2HL, UK

Introduction

Grossman et al. (1975) proposed that in cardiovascular diseases any adaptation of the myocardium depends upon the pattern and quantity of the associated haemodynamic loading. In hypertensives, or people with aortic stenosis, thickening of the septum and left ventricular posterior free wall is observed, with no changes in cavity dimensions. This type of cardiac muscle remodelling is often referred to as concentric enlargement.

Grossman's theory linking specific patterns of haemodynamic loading and left ventricular enlargement was adopted as an explanation for the training-specific nature of the cardiac adaptations witnessed in elite sportsmen. Morganroth et al. (1975) reported increased left ventricular wall thicknesses in male wrestlers and shot putters. These activities, along with sports such as weightlifting, have been classified as a high static:low dynamic (Mitchell et al. 1994), requiring extreme levels of muscle power production (Garhammer 1980), and generating an intermittent, but significant, haemodynamic pressure overload (MacDougall et al. 1985).

Confirmation of a link between resistance training and specific cardiac adaptation can be found (Longhurst et al. 1980; Deligiannis et al. 1988; Roy et al. 1988; MacFarlane et al. 1991; Pelliccia et al. 1991, 1993). However, other studies (Menapace et al. 1982; Pearson et al. 1986; Brown and Thompson 1987; Fisher et al. 1989; Fleck et al. 1989, 1993; George et al. 1998) have failed to demonstrate any evidence of a concentric left ventricular enlargement in a broad range of weight- and resistance-trained athletes.

The conflicting findings may reflect differences in the use of a range of structural indices to represent concentric enlargement (i.e. wall thickening, increased left ventricular mass, increased mass-volume ratio, increased wall thickness-cavity dimension ratio), subject group selection, activity levels and the partitioning out of group differences in body size variables. Most studies are cross-sectional, comparing male athletes with sedentary

controls. When matching is not achieved for body size or composition, the most appropriate scaling process is applied. Whilst authors are aware of the need to take into account body size variation (Urhausen and Kindermann 1992), this has been achieved by a ratio-standard approach despite theoretical criticisms (Tanner 1949). The use of allometric scaling is supported in clinical (Lauer et al. 1995), non-athletic (Batterham et al. 1997) and athletic (George et al. 1998) populations.

Only limited evidence of cardiac adaptation to resistance training in females has been published. Whilst some cross-sectional studies exist (Pelliccia et al. 1996; George et al. 1995) there are no data for elite female weightlifters. Studies of females have either not scaled structural data or scaled for differences in body dimensions (usually body surface area) via a ratio-standard approach. Ignoring allometric procedures (Lauer et al. 1995; Batterham et al. 1997) may have compromised the validity of the comparisons.

The purpose of the study presented here was to investigate the cardiac structural responses to chronic conditioning in a group of elite female weightlifters compared to a healthy recreationally-active population of control subjects.

Methods

Sixty one women gave written informed consent to participate in the study. Ethical approval was granted by the Faculty Ethics Committee. All subjects were healthy and free of known heart disease. Fifteen of the subjects [mean (SD) age, 25 (6) years] were National Weightlifting Squad members, and the athletes had been training for competitive weightlifting for 6 (3) years and members of the National squad for 3 (2) years. The athletes were tested just prior to start of the competitive season. They represented a broad range of weight categories (body mass range 46–90 kg). Performance data ranged from 105 kg for the snatch and 130 kg for the clean and jerk in a high weight category lifter (83+ kg), to 52 kg for the snatch and 65 kg for the clean and jerk for a low weight category lifter (<46 kg). The control group consisted of 46 female non-athletes [age, 23 (3) years]. None were currently or formerly competitively involved in any sport.

All anthropometric and echocardiographic variables were measured at the same visit to the laboratory. Body mass (BM, kg) and height (HT, m) were measured to derive body surface area (BSA, m^2) via the Dubois and Dubois (1916) formula. Biceps, triceps, subscapular and suprailiac skinfold thicknesses were assessed in triplicate and used to calculate percentage body fat (Durnin and Womersley 1974). Fat mass was calculated from percent body fat and BM. Fat free mass (FFM, kg) was calculated as BM minus fat mass (Table 1).

After anthropometric assessment, subjects rested in the supine position for 30 min. Electrodes were attached for a three-lead EKG system intrinsic to the HP Sonos 100 Ultrasound Imaging System (Hewlett Packard, Andover Massachusetts, USA). A single experienced electrocardiographer obtained ultrasound images of the left ventricle with the subject lying in the left lateral decubitus position. Placement of a 2.5-MHz transducer at the parasternal window allowed the production of a two-dimensional image of the left ventricle in the long axis. M-mode recordings were taken at the tip of the mitral valve leaflets. From the concomitant EKG trace septal thickness (ST), posterior wall thickness (PWT) and left ventricular end-diastolic dimension in diastole (LVIDd) were digitized at the peak of the R-wave according to Penn convention guidelines. Left ventricular end-systolic dimension (LVIDs) was measured as the minimum separation of the septal and posterior walls. Left ventricular mass (LVM) was estimated via a regression-corrected "cube-formula" (Devereux and Reichek 1977). The ratios of LVM:left ventricular end-diastolic volume (m:V; where volume was calculated from LVIDd; Ten Cate et al. 1974), and total wall thickness (ST + PWT):LVIDd (h:R) were calculated as indices of concentric enlargement. Systolic and diastolic functional data were obtained in the weightlifters and a sub-sample of 22 controls. Fractional shortening [FS (%) = $(LVIDd-LVIDs)/LVIDd*10^{-2}$] and ejection fraction [EF (%) = $(LVIDd^3-LVIDs^3)/LVIDd^3*10^{-2}$] were calculated from M-mode data. Diastolic function was assessed via pulsed-wave Doppler echocardiography. A two-dimensional sector scan of a four-chamber view from the apex position allowed the placement of the sample-volume at the level of the mitral valve parallel to mitral inflow. This facilitated the measurement of peak flow velocities ($cm \cdot s^{-1}$) for early passive (E) and late atrial contraction (A) filling of the left ventricle. The E:A ratio was then calculated. All measurements were made over a minimum of four consecutive heart cycles in held expiration by a single experienced reader who was unaware of the group allocation of the subjects. Following echocardiographic measurements a resting heart rate was attained from the EKG and blood pressure was attained via sphygmomanometry.

Data are reported as group means (SD) unless otherwise stated. Echocardiographic data are expressed in absolute units and then scaled (where appropriate) for differences in body dimensions. Via multivariate allometric scaling, common group BM, FFM, BSA and HT exponents were identified for all measured cardiac dimensions with group entered as a discrete variable. The commonality of slopes was determined between the athlete and control groups. The scaling procedures followed the format adopted for LVM (Batterham et al. 1997). The residuals were normally distributed (Kolmogrov-Smirnov one-sample test, $P > 0.05$) and no relationship was found between the absolute residuals and body size variables ($P > 0.05$). Group differences were then compared via one-way analysis of variance for all absolute and allometrically scaled data. The results of the data analysis, carried out on the Statistica software (Statsoft), were considered to be significant if $P < 0.05$.

Table 1 Heart dimensions for weightlifters and controls. Values are given as the mean (SD). (*LVIDd* Left ventricular internal dimension in diastole, *ST* septal thickness, *PWT* posterior wall thickness, *LVM* left ventricular mass, *m:V* ratio of LVM to left ventricular end-diastolic volume, *h:R* ratio of wall thickness to cavity dimension ratio)

	Controls	Weightlifters	*P* values
LVIDd (mm)	48.4 (3.4)	46.2 (2.8)	0.03
ST (mm)	7.7 (1.0)	9.0 (1.1)	<0.01
PWT (mm)	7.5 (1.3)	8.7 (1.4)	<0.01
LVM (g)	137.0 (35.0)	157.5 (41.5)	0.07
m:V	1.15 (0.23)	1.50 (0.21)	<0.01
h:R	0.31 (0.05)	0.38 (0.03)	<0.01

Results

The groups were evenly matched for body dimensions [BM, 59.8 (6.7) kg and 61.5 (11.5) kg; BSA, 1.66 (0.10) m^2 and 1.64 (0.18) m^2; FFM, 46.2 (4.1) kg and 47.8 (7.8) kg for the controls and weightlifters, respectively] and composition [body fat, 23 (2)% and 22 (3)% in the controls and weightlifters, respectively]. The only exception was that the controls were slightly taller [1.66 (0.05) m vs 1.59 (0.08) m, $P < 0.01$].

Cardiac structural data mean values were within normal ranges (Table 1). The internal dimension of the

left ventricle was slightly greater in the controls. Conversely, the ST and PWT were greater in the weightlifters. LVM was 13% greater in the weightlifters compared to controls, but this difference was not statistically significant. The combination of elevated wall thicknesses and smaller internal dimensions in the weightlifters increased both the m:V and h:R ratios ($P < 0.05$). Allometric power function exponents were calculated (Table 2). Exponents corresponding to the dimensionality theory lie within the 95% confidence limits for each variable. The allometric scaling provided appropriate common group exponents for the comparison of group cardiac structural data after normalization for differences in HT. After normalization for HT, differences in wall thicknesses were still observed (weightlifter values represented 123–124% of the control group values – calculation of the antilog of the group beta score). Since the controls were taller, the mean values for LVIDd, adjusted for differences in HT, were not different between groups (weightlifter values represented 99% of the control group values). The combination of similar internal dimensions and greater ST and PWT resulted in a difference in LVM (133%) after the adjustment for HT.

Resting heart rate and blood pressures were not significantly different between groups. Despite the evidence of a concentric hypertrophy in the weightlifters, no alteration in either systolic or diastolic cardiac function was noted (Table 3). As with the structural data, the mean scores for all functional indices were within normal limits.

Discussion

Despite a high level of training and competition the majority of body dimension variables were not significantly different between the groups. The weightlifters were only slightly heavier and slightly less fat, resulting in similar estimates of FFM. The weightlifters represented a broad range of weight categories and there was a tendency for percent fat to increase as BM increased. Thus the groups were well matched in terms of the body dimensional variables that have been used to scale cardiac dimensions in resistance-trained athletes (George et al. 1991). The fact that HT was different between groups could be accounted for by the allometric scaling process. However, HT seems neglected as a scalar variable in studies of athletes, although it is used in clinical studies (Lauer et al. 1995). Batterham et al. (1997) and George et al. (1998) suggested that HT may be unsuitable as a scalar in studies of athletes because of its large residual error scores and the fact that it does not take into account differences in body composition.

The structural data presented in Table 1 suggest that the athletes exhibit a concentric left ventricular enlargement. Wall thickness was elevated in the weightlifters and the increase in LVM approached statistical significance. The scaling out of the influence of HT resulted in a group difference in LVM. Both the m:V and h:R values suggest an increase in muscle mass over and above any changes in cavity size, which represents concentric enlargement. The larger absolute LVIDd observed in the controls was not present after allometric scaling for HT.

The mean wall thickness (ST: 9.0 vs 6.9, PWT 8.7 vs 8.0) and LVM (157.5 vs 134.0) of the weightlifters are greater than those reported for resistance-trained athletes (George et al. 1995). George et al. (1995) included sprinters, jumpers and throwers within their resistance-trained group who may have been involved in a degree of aerobic-based training. The mean LVIDd of the athletes in the study by George et al. (1995) is higher (48.6 vs 46.2) than that of the current weightlifters and thus supports the contention that they were not purely resistance trained. Pelliccia et al. (1996) did not include

Table 2 Allometric power function exponents (±95% confidence intervals) and log-linear multiple *R* scores in brackets. (*BM* Body mass, *FFM* fat free mass, *BSA* body surface area, *HT* height)

	BM	FFM	BSA	HT
LVIDd (mm)	0.22 (0.12) [0.50]	0.32 (0.14) [0.58]	0.44 (0.20) [0.55]	0.97 (0.43) [0.57]
ST (mm)	0.38 (0.23) [0.59]	0.52 (0.26) [0.62]	0.73 (0.40) [0.61]	1.42 (0.84) [0.60]
PWT (mm)	0.53 (0.31) [0.54]	0.64 (0.35) [0.54]	0.96 (0.52) [0.55]	1.63 (1.11) [0.50]
LVM (g)	1.06 (0.42) [0.58]	1.42 (0.47) [0.65]	2.04 (0.70) [0.64]	4.01 (1.50) [0.60]

Table 3 Resting cardiovascular and left ventricular functional data. (*E* Early diastolic filling, *A* atrial or late diastolic filling)

	Functional Component	Controls ($n = 22$)	Weightlifters ($n = 15$)	*P* Values for group comparison
	Resting Heart Rate (beats · min^{-1})	65 ± 7	68 ± 12	0.50
	Systolic Blood Pressure (mmHg)	116 ± 8	118 ± 8	0.30
	Diastolic Blood Pressure (mmHg)	72 ± 8	76 ± 5	0.11
Systolic	Fractional Shortening (%)	37.3 ± 3.9	38.9 ± 1.7	0.15
	Ejection Fraction (%)	75.1 ± 3.9	76.7 ± 1.8	0.17
Diastolic	Peak E Velocity (cm · s^{-1})	74.2 ± 12.8	77.4 ± 10.8	0.48
	Peak A velocity (cm · s^{-1})	36.0 ± 6.3	39.7 ± 12.2	0.33
	E:A ratio	2.11 ± 0.49	2.06 ± 0.46	0.78

any weightlifters in their large sample of Italian athletes. Their study included 13 female judo players whose maximal left ventricular wall thickness [8.2 (0.8)] was slightly lower than that of the weightlifters in the present study. A maximal wall thickness of 9.3 mm, reported in a sample of Italian female cyclists (Pelliccia et al. 1996), were similar to that of the weightlifters in the current study. Sub-samples of athletes were not compared to controls in the study of Pelliccia et al. (1996), and scaling normally occurred via a ratio-standard procedure using BSA, which would be inappropriate bearing in mind the limitations of the ratio-standard process and the fact that the BSA-LVM exponent calculated from this study (2.04) is different from 1.0.

In studies of male resistance-trained athletes the choice of echocardiographic variable used to represent a concentric enlargement has differed, the type of athlete has been varied and the scaling process may have been inappropriate. Some evidence of a concentric enlargement of the left ventricle has been published (Morganroth et al. 1975; Longhurst et al 1980; Deligiannis et al. 1988; MacFarlane et al. 1991; Pelliccia et al. 1993). However, these studies have been criticized due to a combination of the scalar variable (BSA) and process (ratio-standard) chosen. Those who scaled by FFM have suggested that any increase in LVM in resistance-trained male athletes only mirrors increases in body dimensions (Menapace et al. 1982; Fisher et al. 1989; Fleck et al. 1989, 1993; George et al. 1998). The consensus of opinion (George et al. 1991; Urhausen and Kindermann 1992; Perrault and Turcotte 1994) supports the contention that in men, resistance or weight training does not induce a true concentric enlargement of the left ventricle. The lack of adaptation of the left ventricle to resistance training in women (George et al. 1995) was speculated to be based on the low levels of circulating testosterone. Testosterone is a known anabolic agent for skeletal and cardiac muscle growth or maintenance in animals (Schaible et al. 1984). Women demonstrate both a lower circulating testosterone level and smaller increase in skeletal muscularity than men in response to resistance exercise. However, the blood pressure response to resistance exercise in men and women is qualitatively and quantitatively similar. Thus, in response to chronic resistance- or weight-training exercise, the stimulus for cardiac adaptation (haemodynamic overloading) is present but one of the stimuli for muscle growth (testosterone) is not.

The allometric power function exponents calculated in the present study (Table 2) followed the pattern predicted by the dimensionality theory (Schmidt-Nielsen 1984; Gutgesell and Rembold 1990). The exponents for LVM related to BM (1.06) and FFM (1.42) included the values suggested by Batterham et al. (1997) for non-athletes within their 95% confidence intervals. Confirmation of the dimensionality theory has also been witnessed in male athletes and controls (George et al. 1998). In body-dimension-matched subject groups scaling may not be necessary. However, matching of HT, BM and BSA may not be enough in studies of athletes. Care must be taken to match for FFM. Batterham et al. (1997) suggested that FFM is the most accurate body-dimensional scalar since it takes into account differences in body composition.

In the present study normal resting systolic function was demonstrated in the weightlifters and is similar to that noted in other studies of female athletes (e.g. George et al. 1995). The impact of elite-level training upon diastolic function in female athletes has received little attention. The diastolic functional data were normal in weightlifters despite elevated m:V and h:R ratios. This is similar to findings in male athletes (Fagard et al. 1987; Missault et al. 1993). In contrast to the impact of concentric enlargement due to pathological disease states (i.e. hypertrophic cardiomyopathy; Hanrath et al. 1980), a physiologically induced concentric adaptation of the left ventricle has no detrimental effect upon mitral inflow.

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