Effects of long term resistance training on left ventricular morphology

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OBJECTIVES: To assess the effects of long term (mean ± SD 10±5 years, range three to 25 years) resistance training on left ventricular (LV) dimensions and mass.

METHODS AND RESULTS: The study participants were 21 elite male powerlifters (age 33.4±5.9 years) and 10 sedentary male control subjects (age 30.9±4.2 years). Two-dimensionally guided transthoracic M-mode echocardiograms were obtained at rest to quantify LV diastolic cavity dimension, posterior wall thickness, ventricular septal wall thickness and LV mass. Long term resistance training was not associated with an alteration in LV diastolic cavity dimension (resistance trained 54.4±4.3 mm versus control 51.8±5.6 mm), ventricular septal wall thickness (resistance trained 9.7±1.0 mm versus control 10.1±0.7 mm), posterior wall thickness (resistance trained 9.6±1.5 mm versus control 9.3±1.4 mm) or LV mass (resistance trained 200.3±32.5 g versus control 186.5±39.6 g). In addition, no resistance-trained athlete was found to have an LV mean wall thickness above clinical normal limits (12 mm or less).

CONCLUSION: Contrary to common beliefs, long term resistance training as performed by elite male powerlifters does not alter LV morphology.

Key Words: *Echocardiography; Exercise; Left ventricle*

Effets d'un entraînement prolongé utilisant une résistance sur la morphologie ventriculaire gauche

OBJECTIFS : Évaluer les effets d'un entraînement prolongé (moyenne ± ET 10 ± 5 ans, fourchette de trois à 25 ans) utilisant une résistance sur les dimensions et la masse ventriculaire gauche (VG).

MÉTHODES ET RÉSULTATS : Les participants de l'étude étaient 21 athlètes haltérophiles (âgés de 33,4 ±5,9 années) et 10 sujets témoins sédentaires de sexe masculin (âgés de 30,9 ± 4,2 ans). Des échocardiogrammes temps-mouvement transthoraciques guidés à deux dimensions ont été obtenus au repos pour quantifier la dimension de la chambre VG en diastole, l'épaisseur de la cloison postérieure, l'épaisseur du septum ventriculaire et la masse VG. Un entraînement utilisant une résistance n'était pas associé à une modification de la dimension de la chambre VG en diastole (entraînement avec résistance 54,4 ± 4,3 mm par rapport au témoin 51,8 ± 5,6 mm), de l'épaisseur du septum ventriculaire (entraînement avec résistance 9,7 ± 1,0 mm par rapport au témoin 10,1 ± 0,7 mm), de l'épaisseur de la cloison postérieure (entraînement avec résistance 9,6 ± 1,5 mm par rapport au témoin 9,3 ± 1,4 mm) ou de la masse VG (entraînement avec résistance 200,3 ± 32,5 g par rapport au témoin 186,5 ± 39,6 g). De plus, aucun athlète pratiquant un entraînement avec résistance n'a démontré une épaisseur moyenne de la cloison VG au-dessus des limites cliniques normales (12 mm ou moins).

CONCLUSION : Contrairement aux idées reçues, un entraînement prolongé utilisant une résistance tel que pratiqué par des athlètes haltérophiles de sexe masculin ne modifie pas la morphologie VG.

Resistance training has gained popularity as a safe and effective exercise intervention to increase skeletal muscle mass and muscle strength, particularly in healthy individuals (1). Despite the benefits of resistance training on skeletal muscle morphology, the effects of this type of training on left ventricular (LV) morphology remain uncertain. Some studies have found that the resistance training-mediated pressure load (ie, systolic pressures greater than 250 mmHg) (2,3) may result in alterations in LV morphology, including increases in LV wall thickness, relative wall thickness and estimated LV mass (4-6). Other studies report that resistance training is not associated with a change in

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TABLE 1
Maximal weight lifted in the squat, bench press and deadlift at the 1996 Canadian National Powerlifting Championships

Exercise	Maximal weight lifted (kg)
Squat	251.8±2
Bench press	155±17.4
Deadlift	257.6±14.4

Values are mean ± SD

absolute LV dimensions or mass (7-9), leading to the suggestion that the heightened pressure load may have been too brief to alter LV morphology (10,11).

Previous echocardiographic investigations assessing the 'athlete's heart' have shown that the magnitude of the alteration in LV morphology was related to the duration of athletic conditioning (12-14). A limitation of the studies assessing the effects of resistance training on LV morphology is that the subjects were younger athletes (less than 25 years) who had been training for relatively short periods (less than five years). The effects of long term (10 years) resistance training on LV morphology has not been well studied. The purpose of this study was to evaluate the effects of long term resistance training on LV dimensions and mass in elite male resistance-trained athletes.

SUBJECTS AND METHODS

Subjects: The study group consisted of 21 resistance-trained athletes who were members of the Canadian Powerlifting Union. The study included 10 sedentary control subjects who were matched for age and body surface area. The resistance-trained athletes had been training for 10±5 years (range three to 25 years) and had qualified and competed at the 1996 Canadian National Powerlifting Championships, where they were recruited for the study. In preparation for this contest, the athletes performed 4.0±1.0 workouts/week at 98±30 mins/workout for 15±13 weeks. Table 1 shows the maximal weight the powerlifters lifted at the 1996 Canadian National Powerlifting Championships. The control subjects were not participating in regular exercise training. Ethical approval for this study was obtained from the University of Alberta Faculty of Physical Education and Recreation Ethics Committee for Human Experimentation, and informed consent was obtained before study participation.

Echocardiography: LV imaging was performed with a commercially available ultrasound instrument (Sonos 2500, Hewlett Packard, Andover, Massachusetts) with a 3.5 MHz transducer. Two-dimensionally guided M-mode echocardiogram examinations were performed, and measurements were obtained from the parasternal short axis view just apical to the mitral valve leaflets. The echocardiographic measures were performed in accordance with the American Society of Echocardiography guidelines (15) and included ventricular septal wall thickness (VST), posterior wall thickness (PWT) and diastolic cavity dimension (LVIDd). Relative wall thickness (h/R ratio) was measured at end-diastole as 2 (PWT/LVIDd) (16). LV mean wall thickness was measured as ½ PWT + VST). Estimated LV mass was determined by the corrected American Society of Echocardiography formula (17).

TABLE 2
Effects of long term resistance training on left ventricular dimensions and mass

Variable	Resistance-trained athletes	Controls
VST (mm)	9.7±1.0	10.1±0.7
VST (mm/m^2)	4.9±0.6	5.1±0.6
LVIDd (mm)	54.4±4.3	51.8±5.6
LVIDd (mm/m^2)	27.3±2.4	25.8±2.9
PWT (mm)	9.6±1.5	9.3±1.4
PWT (mm/m^2)	4.8 ±0.7	4.6±0.6
LVM (g)	200.3±32.5	186.5±39.6
LVM (g/m^2)	100.2±14.5	92.5±17.3
h/R ratio (%)	0.4±0.1	0.4±0.1

h/R Ratio relative wall thickness; LVIDd Left ventricular diastolic cavity dimension; LVM Left ventricular mass; PWT Posterior wall thickness; VST Ventricular septal wall thickness. All comparisons P>0.05. Values are mean ± SD

Statistical analysis: Echocardiographic variables between the two groups were compared with a one-way ANOVA using Statistica (Statsoft, Oklahoma City, Oklahoma) software. The alpha level was set a priori at $P<0.05$.

RESULTS

No significant difference was found between the resistance-trained and control groups for age (33.4±5.9 years versus 30.9±4.2 years, respectively), body surface area (2.0±0.1 m^2 versus 2.0±0.2 m^2, respectively), heart rate (77.8±13.1 beats/min versus 76.1±12.6 beats/min, respectively), systolic blood pressure (140.1±18.3 mmHg versus 127.8±10.5 mmHg, respectively), diastolic blood pressure (88.3±9.9 mmHg versus 84.6±5.0 mmHg, respectively) or mean arterial pressure (105.6±11.6 mmHg versus 99.0±4.7 mmHg, respectively). In addition, no significant difference was found between the resistance-trained and control groups for absolute or relative LVIDd, PWT, VST, h/R ratio or estimated LV mass (Table 2). Finally, no resistance-trained athlete was found to have a measured LV mean wall thickness above normal clinical limits (12 mm or less, Figure 1).

DISCUSSION

The major finding of this study is that, contrary to common belief (4,6), long term resistance training was not associated with differences between the resistance-trained and control groups for absolute or relative LVIDd, PWT, VST, h/R ratio or LV mass. These findings, however, are clearly consistent with a number of previous studies that found that resistance training was not associated with changes in LVIDd (8,9,18-23), VST (7,9,20,23,24), PWT (9,25,26) or estimated LV mass (7-9,23). In addition, our finding that no resistance-trained athlete had an absolute LV mean wall thickness above normal clinical limits (ie, 12 mm or less) is similar to that reported previously for younger (mean age 26.3 years) resistance-trained athletes (21). Together, these

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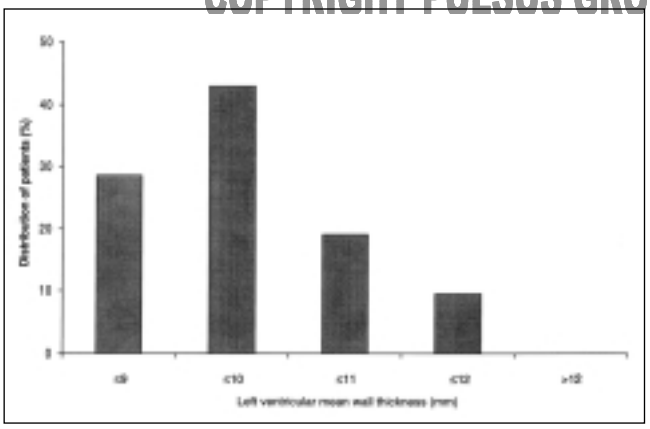

Figure 1) *Distribution of left ventricular mean wall thickness secondary to long term resistance training*

findings suggest that long term resistance training does not alter LV dimensions and mass.

The echocardiographic findings in our subjects are similar to some previous reports on resistance-trained athletes (20-24), but they are lower than those found in others (4,5, 19,22,27-31). Several reasons may explain the disparity among the studies. Previous echocardiographic studies assessing the effects of resistance training on LV morphology have commonly used bodybuilders, olympic weightlifters or powerlifters as study subjects. Although all of these athletes perform resistance exercises to increase muscle strength and/or muscle mass, their acute stroke volume and cardiac output responses during this form of training may be considerably different. For example, bodybuilders have been shown to have a higher stroke volume and cardiac output response than powerlifters when performing resistance exercise (32). The heightened pressure and volume load associated with bodybuilding may be a greater stimulus to induce LV hypertrophy compared with power lifting. This was shown by Pelliccia et al (21), who reported that the magnitude of the alteration in LV morphology may be related to the underlying type of resistance training performed, because bodybuilders were found to have larger LV dimensions and mass than powerlifters or olympic weightlifters. Thus, the disparity between our findings and those of others may be related to the different types of resistance athletes studied.

Submaximal or maximal resistance exercise has been shown to be associated with a transient, abrupt increase in systolic blood pressure (270 to 480 mmHg) (2,3). A widely held belief in sport cardiology has been that the increased pressure load can be a potent stimulus to alter the size of the LV (33). However, MacDougall et al (3) and Lentini et al (34) have shown that the transient abrupt elevation in systolic pressure during resistance training was secondary to the increased intrathoracic pressure associated with performing a brief Valsalva manoeuvre. Because the heart and lungs are intimately related within the thorax (35), positive swings in intrathoracic pressure are transmitted directly to the arterial vasculature as increases in systolic pressure; however, the pressure that the heart was 'exposed' to (ie, LV transmural pressure = LV pressure – intrathoracic pressure) was not elevated above resting values (36,37). An alternative explanation for our findings is that our resistance-trained athletes may have performed a brief Valsalva maneuver during training that may have diminished the stimulus for LV hypertrophy (34).

Other studies have reported that three months of resistance training was associated with a rapid increase (10.9%) in LV wall thickness in younger individuals (age range 16 to 27 years) (38,39). Our resistance-trained athletes may have had a rapid increase in LV wall thickness soon after initiation of training. However, a limitation of this hypothesis is that to see a similar relative increase in LV wall thickness, our athletes' baseline measurements would had to have been approximately 8.5 mm. Because LV wall thickness is independently related to body surface area (13), it is unlikely for athletes with extremely large body surface areas (2.0 m^2 or more), such as the subjects in our study, to have this small measurement at the outset. However, further studies are required to assess the effects of short versus long term resistance training on LV dimensions and mass.

A possible explanation for the increases in LV mass is the effects of anabolic androgenic steroids, which appear to be quite popular with resistance-trained athletes for enhancing sport performance (40,41). Supraphysiological doses of anabolic steroids have been shown to increase maximal muscular strength (42), muscle mass (42), LV wall thickness (43-47), h/R ratio (47), estimated LV mass (31,46,48) and sometimes extreme LV wall thickening (ie, mean wall thickness of 18.3 mm, range 14.7 to 20.9) (49). While it is understandable that the use of anabolic steroids is not usually reported, the possibility that the previously reported increased LV wall thickness and estimated LV mass may have been confounded by the effects of unreported use of anabolic steroid in some of the resistance-trained athletes cannot be ruled out. In the present study, our resistance-trained athletes were not asked about anabolic steroid use (although they were subject to random drug testing according to competition rules). However, if the athletes had been using anabolic steroids, our results would suggest that these drugs do not alter LV morphology. This is a finding that is inconsistent with the known effects of anabolic steroids as reported in previous studies.

CONCLUSION

Long term resistance training was not associated with changes in LVIDd, VST, PWT, h/R ratio or estimated LV mass. In addition, no resistance-trained athlete was found to have a mean LV wall thickness above normal clinical limits. These findings suggest that long term resistance training is an insufficient stimulus to alter LV morphology.

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