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Relationship between Echocardiogram and Physical Parameters in Experienced Resistance Trainers: A Pilot Study

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Abstract:

Background: A paucity of research exits concerning physiological factors influencing heart structure and function in strength athletes. This pilot study investigated whether body composition and muscle performance is associated with indices of cardiac structure and function in experienced resistance trainers.

Methods: A cross-sectional study designed was employed to address the study aim. Seventeen males (median age 33.0 years) and eight females (median age 32.5 years) with backgrounds in bodybuilding and powerlifting participated in this study. Muscle performance, body composition and echocardiographic measures were performed. Mann–Whitney *U* tests were used to examine differences between males and females. Spearman's Rho partial correlation analyses (adjusting for sex) were conducted to examine relationships between physical and echocardiogram parameters.

Results: Moderate to strong positive correlations were found between fat-free mass and aortic root, right ventricular internal dimension, interventricular septum thickness, left ventricular posterior wall thickness, left atrium area, left ventricular end-diastolic volume, and left ventricular end-systolic volume (r = 0.43-0.76, p = ≤0.03). Moderate to strong positive correlations were found between leg press 1RM and aortic root, left ventricular internal dimension diastole, eft atrium area, left ventricular end-diastolic volume, and left ventricular end-systolic volume (r = 0.49-0.67, p ≤0.02).

Conclusions: Resistance trainers with greater fat-free mass and lower body strength appear to have larger cardiac structures. Changes in heart size and function are likely to result from long-term strenuous resistance training. Due to the suspected prevalence of performance enhancing drug use among powerlifters and bodybuilders, care is required to rule out pathological conditions.

Keywords: Athlete heart; bodybuilding; powerlifting; heart function; training adaptation

Introduction

Similar to skeletal muscle, cardiac muscle has been shown to undergo adaptations following chronic exercise that results in changes in size, mass, geometry and function, also known as cardiac remodelling.[1] Heart adaptations had been postulated to result in two different morphological forms referred to as the endurance and strength athlete heart.[2,3] However, the findings from recent studies provide strong evidence that changes in left ventricle mass is related to increase left ventricle volume irrespective of the type of exercise training history.[4-6] Therefore, cardiac remodeling results in a spectrum of morphological changes which is influenced by peak static and dynamic overload, the degree of hemodynamic stress exposure, and exercise history training.[7] The development of eccentric left ventricle hypertrophy (increased cavity size) occurs predominately from volume overload on the heart (i.e.

increased heart rate, stroke volume, increase venous return).[8,9] Concentric left ventricle hypertrophy (increase left ventricle wall thickness) develops from pressure overload on the heart which includes marked blood pressure elevation.[8,9] For instance, blood pressure was shown to increase to 480/350 mmHg in bodybuilders while performing the leg press.[10]

A distinctive factor that needs to be considered when explaining changes in heart structure and function following a history of exercise training is whether the hemodynamic load imposed during exercise bouts is constant or intermittent.[11] The remodeling of the heart (particularly of the ventricles) may also differ according to age, sex,[12] ethnicity, use of anabolic androgenic steroids,[13] as well as the exercise prescription (i.e. intensity, duration).[14] The two types of athletes that are commonly considered at opposite ends of the spectrum when considering exercise prescription and training-induced adaptations are strength and endurance athletes. There is evidence that shows that strength compared to endurance athletes have a higher prevalence of hypertension which causes left ventricular hypertrophy and is an independent predictor of cardiovascular risk.[15,16] The effect of anabolic steroids on the heart in strength athletes is also well-documented,[17] however there is a paucity of research investigating other factors that may influence heart changes (physiologic and pathologic) in strength athletes.

Strength and endurance athletes tend to follow training programs that include dynamic and static exercise, therefore differing degrees of concentric and eccentric left ventricle hypertrophy can result.[18,19] There are obvious differences in the physical and performance

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characteristics between strength and endurance trained athletes.[20,21] Strength athletes will generally have greater fat-free mass compared to endurance athletes which is understandable due to it being a major determinant of maximal strength.[22-24] Interestingly, fat-free mass has been strongly related to left ventricle morphology in elite athletes (strength and endurance trained),[25] endurance athletes,[26] and in the general population.[27] Fat-free mass represents the metabolically active tissue, so it is likely that an exercise-induced increase of this tissue compartment would lead to cardiac adaptations. In a study on 11 weight lifters and 45 age-matched controls a superior influence of fat-free mass on various cardiac dimensions was found compared to other body dimension indices such as body mass, body surface area and height.[28] Unfortunately to date, there has been less focus on investigating associations between body composition and the heart in strength athletes. In terms of muscle performance in strength athletes, the evidence is scarce concerning its influence on cardiac structure and function. In a prospective cohort study of more than 500,000 men and women aged 40–69 it was shown that greater handgrip strength was associated with numerous measures of cardiac structure and function that were indicative of less cardiac hypertrophy and remodelling.[29] However, it is unknown whether any relationship exists between muscle performance and the heart in strength trained athletes.

The primary aim of this pilot study was to investigate whether body composition and muscle performance is associated with indices of cardiac structure and function in experienced resistance trainers. A secondary aim was to examine whether cardiac structure and function differed between sexes in this cohort. It was hypothesized that increased fat-free mass and muscle performance would be associated with larger diameters or thicknesses of various heart morphology. Also, it was hypothesized that more pronounced cardiac hypertrophy and greater cardiac function would be observed for males compared to females.

Methods

Experimental Approach to the Problem

A cross-sectional study design was employed to investigate the relationship between body composition and muscle performance is associated with indices of cardiac structure and function in experienced resistance trainers. This study involved muscle performance testing (upper and lower body strength and power) echocardiogram, and a whole-body dual energy x-ray absorptiometry scan. All testing was performed within a given week and conducted at the laboratories at the University of Sydney, Cumberland Campus. Body composition (in a 10-12 hour fasted state) was either assessed in a separate visit or during the visit prior to the muscle performance tests. Prior to all testing sessions participants were asked to refrain from using caffeine or pre-workout supplements 2- 3 hours prior to testings sessions. Also to avoid any strenuous physical activity 24-48 hours before the testing sessions. If a participant reported fatigue or soreness from previous exercise the testing session was rescheduled to another day to prevent negative influences on performances.

Participants

Twenty five experienced resistance trainers (males, n = 17 and females, n = 8) participated in this study. The male participants (median age 33.0 years [interquartile range (IQR) 26.0-

40]; height 177.0 [173.0–182.0] cm; body mass 88.9 [74.7-94.3] kg; resistance training experience 5.5 [3.6-12.5] years) comprised of powerlifters (n= 10) and bodybuilders (n=7). The female participants (median age 32.5 years [IQR 22.5-43.8]; height 160.4 [155.7–166.9] cm; body mass 68.0 [63.0-72.4] kg; resistance training experience 4.0 [2.3-20] years) also comprised of powerlifters (n=7) and a bodybuilder (n=1). Participants reported predominantly being involved in resistance training with none or very little aerobic training. In a sub-sample (n = 16) that completed training logs resistance training was performed 5 [4.0-6.0] times per week and 6/16 participants performed aerobic training totaling 40 [30.0-72.5] minutes per week. A food diary was completed by 15 participants which indicated that 13 participants used protein supplements and 5 participants used creatine monohydrate. Only participants that were not using any performance enhancing substances were eligible for the study. Information was given to the participants stating possible screening for substances that enhance athletic performance based on the 2013 World Anti-Doping Agency (WADA) List of Prohibited Substances via a urine sample collection and to inform the researchers prior to study commencement if they had taken any prohibited substances during the previous 12 months. An information statement explaining all procedures and study risks was provided to participants, alongside verbal explanations prior to study participation. Verbal and written consent was provided by participants prior to study commencement. The study was formally approved by the local human Ethics Committee and conducted according to the declaration of Helsinki.

Muscle performance

Muscle performance testing was conducted using Keiser A420 pneumatic resistance training equipment (Keiser Sports Health Equipment, Inc., Fresno, CA, USA). Upper body performance was assessed with the chest press while the leg press was used for the lower body. Participants were instructed how to perform the exercises and provided with information concerning the test protocols for assessing muscle strength, power and endurance. For each test, the chest press was performed before the leg press. Participants were monitored for fatigue throughout to ensure that performance would not be negatively affected. Prior to assessing muscular performance, the technique for each exercise was thoroughly explained to each participant. Muscular strength was assessed first via the one-repetition maximum (1RM) test. Participants completed a warm-up that involved a set of five repetitions at ~50% of perceived 1RM followed by 1–2 sets of 2–3 repetitions at a load corresponding to ~60–80% 1RM. The 1RM protocol involved performing trials of a single repetition of increasing load (~5–10% increments) with 3–5 min rest between attempts. This cycle was continued until the participant was unable to complete a lift, with the 1RM being the heaviest load that was successfully lifted. For one participant the tester followed a different 1RM protocol which has previously been described.[30]

Following measurement of 1RM, peak muscle power (W) was assessed at five relative intensities (40%, 50%, 60%, 70%, and 80% 1RM) for the chest press and leg press. Participants were instructed to complete the concentric portion of the repetition as rapidly as possible when verbally cued, then to slowly lower the weight. Three trials were given at each of the five loads specified, separated by a 10-15-second rest period between trials. Participants were encouraged to take longer rest periods between trials when lifting at higher relative intensities. Peak power was calculated via the Keiser machines and the highest peak power produced throughout the loads tested was used in subsequent data analysis. The final muscle performance test conducted was muscular endurance which was assessed by a maximum repetition task at 60% 1RM. The test was ceased if the exercise technique was not appropriate or if the participant requested to stop the test.

Body composition

A whole-body dual energy x-ray absorptiometry scanner, (Lunar Prodigy, GE Medical Systems, Madison, WI) was used to measure body composition under standardised conditions (early morning, overnight fasted, bladder/bowel voided, and standardised body positioning on the scanning bed). Following the scan, in-built analysis software (version 13.60.033; enCORE 2011, GE Healthcare, Madison, WI) allowed the calculation of total, fat and fat-free mass. To eliminate differences in fat-free mass associated with height fat-free mass index (FFM index) was calculated. This was derived as FFM (kg) divided by height (m) squared (kg/m^2).[31]

Echocardiogram

Echocardiograms were performed on resting participants in a steep left lateral decubitus position by a single qualified, experienced and accredited sonographer (for consistency) using a Philips iU22 (Philips Ultrasound, Bothell, WA, USA) ultrasound machine, 1 - 5 MHz sector transducer, with simultaneous electrocardiogram recording (ECG). Echocardiography was conducted according to the Guidelines for Performing a Comprehensive Transthoracic Echocardiographic Examination in Adults proposed by the American Society of Echocardiography.[32] Aortic root (AoR), left atrium (LA) anterior to posterior linear measurement, right ventricular internal diameter (RVID), interventricular septum thickness end systole (IVSs), left ventricular posterior wall thickness end systole (LVPWs), left ventricular internal dimension diastole (LVIDd), left ventricular internal dimension systole (LVIDs) and left atrial area were measured. Left ventricular end-diastolic volume (LVEDvol), left ventricular end-systolic volume (LVESvol), (FS) fractional shortening, and (EF) ejection fraction were calculated. Mitral inflow velocities were evaluated by pulsed-wave Doppler echocardiography with measures including peak E (early diastolic) and A (atrial contraction) flow velocities, E wave deceleration time, E/A velocity ratio, and isovolumic relaxation time (IVRT).

Statistical Analyses

Statistical analyses were performed using SPSS version 24.0 for Windows (IBM Corp. Armonk, NY USA). Data were inspected visually and statistically for normality using the Kolmogorov–Smirnov test. Given the small subject sample size and inconsistent normal data distribution non-parametric tests were used for all analyses. Data were presented as median with interquartile range (IQR). Muscle strength (1RM) and peak power (W) were expressed in absolute terms and relative to fat-free mass (1RM or W divided by fat-free mass in kg, respectively). Differences between males and females for all variables of interest was analysed using the Mann–Whitney *U* test. Spearman's Rho partial correlation analyses (adjusting for sex) were performed to examine relationships between body composition, muscle performance and echocardiogram parameters. Strength of correlations were qualitatively assessed using the following criteria: trivial ($r < 0.1$), small ($r > 0.1$ to 0.3), moderate (r > 0.3 to 0.4), strong (r > 0.5 to 0.7), very strong (r > 0.7 to 0.9), nearly perfect (r > 0.9), and perfect (r = 1.0).[33] Significance was set at p <0.05.

Results

Results for the echocardiogram parameters between male and female participants are presented in Table 1. Males compared to females were found to have greater aortic root (p<0.01), ascending aorta dimension parameters (p ≤0.02), interventricular septum thickness end systole (p=0.02), left ventricular internal dimension diastole (p<0.01) and left ventricular internal dimension systole (p<0.01) with no statistical differences between sexes for left ventricular posterior wall thickness end systole, left atrium and right ventricular internal diameter. Males were found to have greater left ventricular end-diastolic volume (p<0.01) and left ventricular end-systolic volume (p<0.01), with no statistical differences between sexes for fractional shortening, mitral inflow E and A wave velocities, E wave deceleration, E/A velocity ratio, E', E/E', isovolumic relaxation time, left atrial area and ejection fraction. The median values for the majority of echocardiogram parameters for males and females were within the normative values for healthy adults. The exceptions being slightly greater left ventricular internal dimension diastole for males (54.0 mm versus norm: 36.8-52.7 mm), slower mitral inflow A wave velocity for males (RT: 44.0 versus norms: 48.0-69.0), slower mitral E wave deceleration for both sexes (males: 225.0 ms and females: 213.0 ms versus norms: 157.0-211.0 ms), and higher E/A velocity ratio also for males (1.9 versus norms: 1.0—1.6).

The body composition and muscle performance characteristics of participants are presented in Table 2. Males compared to females had greater body mass, fat-free mass, fat-free mass index and lower relative fat mass $p<0.01$). Absolute and relative chest press 1RM (N and N/fat-free mass [kg]), peak power (W and W/fat-free mass [kg]), and endurance was greater for males compared to females ($p\leq0.02$). Absolute leg press 1RM (N) and peak power (W), was also greater for males compared to females ($p<0.01$). There were no differences between sexes for relative leg press 1RM (N/fat-free mass [kg]), peak power (W/fat-free mass [kg]), and absolute muscle endurance.

The relationships between echocardiogram parameters and body composition is presented in Table 3. Moderate to strong positive correlations were found between fat-free mass and aortic root, ascending and descending aorta dimensions, right ventricular internal diameter (Figure 1a), interventricular septum thickness end systole, left ventricular posterior wall thickness end systole, left ventricular end-diastolic volume and left ventricular end-systolic volume ($r = 0.43$-0.64, $p = \leq0.03$). A very strong positive correlation was found between fat-free mass and left atrial area ($r = 0.76$, $p = <0.01$) (Figure 1b). Free-free mass index was found to have very similar relationships with the echocardiogram parameters as fat-free mass. Strong positive correlations were found between body mass and ascending and descending aorta dimensions, interventricular septum thickness end systole, left ventricular posterior wall thickness end systole, left atrial area, left ventricular end-diastolic volume and left ventricular end-systolic volume ($r = 0.50$-0.66, $p = <0.01$). A moderate positive correlation was found between fat mass and left ventricular internal dimension diastole ($r = 0.45$, $p = 0.03$). There were no other significant correlations were found between any other echocardiographic parameter and body composition.

The relationships between the echocardiogram parameters and muscle performance is presented in Table 4. A strong positive correlation was found between chest press peak power and left atrium ($r = 0.54$, $p < 0.01$) (Table 4). Moderate to strong negative correlations were found between chest press relative peak power and aortic root ($r = -0.54$, $p < 0.01$) and left atrial area ($r = -0.47$, $p = 0.02$). Therefore, suggesting that participants with greater chest press relative peak power had lower aortic root diameter and left atrial area. A moderate negative correlation was found between chest press endurance and left ventricular end-systolic volume ($r = -0.44$, $p = 0.03$), suggesting participants with greater endurance had lower left ventricular end-systolic volume. Moderate to strong positive correlations were found between leg press 1RM and aortic root, ascending aorta sinus of Valsalva; left ventricular internal dimension diastole, left atrial area, left ventricular end-diastolic volume (Figure 2) and left ventricular end-systolic volume ($r = 0.49$-0.67, $p \leq 0.02$). A moderate positive correlation was found between leg press relative peak power and left atrial area ($r = 0.45$, $p = 0.04$). No other significant correlations found between any other echocardiographic parameter and muscle performance.

Strong positive correlations were found between fat-free mass and chest press 1RM ($r = 0.60$, $p < 0.01$) and leg press 1RM ($r = 0.63$, $p < 0.01$). A strong positive correlation was also found between fat-free mass and leg press peak power ($r = 0.68$, $p < 0.01$) but for chest press peak power no significant correlation was found ($r = 0.36$, $p = 0.08$). No significant relationships were found between fat-free mass and muscle endurance for the chest press and leg press ($r \leq 0.23$, $p > 0.05$).

Discussion

The primary aim of this study was to examine whether body composition and muscle performance is associated with indices of cardiac structure and function in experienced resistance trainers. In agreement with the original hypothesis numerous relationships were found between body composition and echocardiogram parameters. The results suggested that participants with greater fat-free mass had larger diameters or thicknesses of various heart echocardiographic measurements. In particular, greater fat-free mass and increased left atrium area and left ventricle end systolic and diastolic volumes. Body mass showed similar associations with the echocardiogram parameters to fat-free mass. There were numerous relationships found between muscle performance and echocardiogram parameters, again in agreement with the original hypothesis. For the upper body, results suggested that participants with higher relative peak power had smaller aortic root diameters and left atrium areas. Additionally, better upper body muscle endurance (increased number of repetitions performed) seemed to be present in participants with lower left ventricular end-systolic volume. For the lower body, participants with greater absolute strength appeared to have a larger aorta root, left ventricular internal dimension diastole, left atrial area, and left ventricular end-diastolic and systolic volumes. There were differences found between sexes for a number of echocardiogram parameters. Males had a larger aortic root diameter, interventricular septum thickness and left ventricular internal dimension. Additionally, males compared to females had greater left ventricle end diastolic and systolic volumes. The results of this pilot study showed that various echocardiogram parameters were correlated with body composition and muscle performance in experienced resistance trainers. However, since this was an observational study causality cannot be established and caution is warranted when interpreting the findings due to the small sample size.

Associations between fat-free mass and left ventricle morphology have been shown to vary between approximately r = 0.5-0.7 in endurance athletes, weight-lifters, and the general population,[26-28] which was very similar to the present study. Fat-free mass was positively associated with a greater number of echocardiogram parameters compared to body mass. It therefore appears that fat-free mass has a superior influence on cardiac structure as concluded by George et al.[28] Resistance training is known to increase fat-free mass which consists of skeletal muscle, internal organs, bone, water, and connective tissue.[34] However, skeletal muscle represents a large proportion of fat-free mass and changes in fat-free mass following a resistance training intervention is predominantly due to increases in skeletal muscle mass.[34] Fat-free mass consists of a greater amount of highly metabolically active tissue which requires greater cardiovascular work to meet the metabolic demands.[35] The increased cardiovascular work would likely provide a stimulus to induce cardiac adaptations. This may explain the stronger associations observed for fat-free mass and echocardiogram parameters. Although, as identified by Pressler et al.[25] in elite athletes there are exercise-induced cardiac adaptations beyond the sole influence of fat-free mass.

In athletic populations the type of exercise training will influence cardiac adaptations.[8,9,19] The participants in the present study engaged in resistance training focused either on bodybuilding or powerlifting. The focus of training for bodybuilding is improving aesthetic appearance (i.e. muscle mass, symmetry, and definition),[36] whereas training for powerlifting is focused on increasing the maximal load that can be lifted for three main exercises.[37] Despite differences in training goals, anecdotally bodybuilders and powerlifters may engage in similar exercise practices throughout a training cycle (e.g. muscle hypertrophy phase for powerlifters and strength phase for bodybuilders). The present study is possibly the first to explore the relationships between muscle performance and echocardiogram parameters in a resistance trained cohort. Since cardiac dimensions were strongly associated with leg press 1RM and leg press peak power it suggests that resistance trainers with greater lower body strength and power may have a larger left side of the heart. Although, it is possible that these associations were reflecting the influence of fat-free mass, due to being a major determinant of maximal strength.[22-24] This hypothesis is supported by fat-free mass being strongly associated with leg press 1RM ($r = 0.63$) and leg press peak power ($r = 0.68$). However, a strong association was found between fat-free mass and chest press 1RM ($r = 0.60$) but no cardiac dimensions were associated with chest press 1RM. It is also interesting to note that relative chest press peak power was associated with left atrium diameter ($r = 0.54$) but there was no association between fat-free mass and chest press peak power. Therefore, it could be assumed that the conflicting results may reflect the somewhat small sample recruited in the study and or perhaps reflect differences in training programs used by powerlifters and bodybuilders including, frequency, intensity, volume (sets and repetitions) and recovery between sets.

Since fat-free mass was considered an important factor influencing cardiac dimensions for both muscle strength and peak power, these muscle performance variables were expressed relative to fat-free mass in an attempt to differentiate this influencing component of body composition. However, there were only two significant associations found both of which involved chest press relative peak power. These negative associations seem to suggest that resistance trainers with greater relative upper body power have smaller aortic root diameters and left atrium areas. As shown in the present study body mass and fat-free mass are associated with larger cardiac dimensions, therefore it could be argued that participants with greater relative upper body strength weighed less and had lower fat-free mass. Although, this conclusion would not be consistent with results for the lower body where no associations were found for leg press relative peak power and echocardiogram parameters. Another difficult finding to explain is chest press muscle endurance being associated with a larger left ventricle end-systolic volume. As has already been stated, possibly resistance training factors may explain the chest press relative muscle peak power and muscle endurance relationships found with the echocardiogram parameters.

Sex differences between males and females for echocardiogram parameters is commonly related to the influence of sex hormones, which is testosterone in men compared to estradiol and progesterone in women.[38] In the present study there were noticeable differences between sexes for various echocardiogram parameters (as well as for muscle performance) which justified the decision to use partial correlations (adjusting for sex) to explore relationships. For most cardiac morphology parameters differences between sexes for echocardiogram parameters (e.g. left ventricle dimensions, left atrial volume) is related to smaller body size.[39] In comparison, cardiac function parameters such as stroke volume and ejection fraction is lower in females after making adjustments for body size.[39] For this study's cohort, resistance training did not seem to affect the commonly reported sex-difference in echocardiogram parameters. However, it should be noted that there can be sex-specific peculiarities of echocardiogram parameters attributed to hemodynamic factors, body size and hormonal factors.[39]

Strength compared to endurance athletes have a higher prevalence of hypertension which causes left ventricular hypertrophy and is an independent predictor of cardiovascular risk.[15,16] However, for the participants in this study the median resting blood pressure was 110/68 mmHg, which is considered to be normal.[40] Therefore, it is likely that any cardiac remodelling present would be physiological (through training) and not pathological.[8,9]

Overall the echocardiogram parameters were within the reference range for the participants, although there were a few exceptions. Males had a slightly higher left ventricle internal dimension although it did not reach the threshold for a mildly dilated left ventricle proposed by The American Society of Echocardiography criteria (men: 60 to 63 mm).[41] The other parameters outside of the reference ranges were also found in males and included the higher mitral inflow A wave velocity, a higher E/A velocity ratio and the mitral inflow E wave deceleration being slower. These findings are not uncommon in athletes due to greater left ventricle dimensions and is thought to be due to athletes being less dependent on the atrial contraction for filling.[42]

Changes in heart size and function are likely to result from long-term strenuous resistance training. Powerlifters and bodybuilders that have greater fat-free mass and high lower body strength are likely to present with a larger absolute heart size. Besides these changes being attributed to strict training and dietary regimens, use of anabolic androgenic steroids (AAS) can also play a major role. As mentioned previously use of performance enhancing drugs such as AAS is suspected to be prevalent among athletes such as powerlifters and bodybuilders.[17] It has been documented that high-dosage of AAS intake is associated with increase cardiac hypertrophy as well as reduced heart function and development of heart disease.[17,43] Therefore, resistance trainers that show echocardiogram measurements exceeding the normative reference ranges require further investigation to rule out pathological conditions and potentially cardiovascular disease risk follow-ups on a regular basis.

Although participants reported not using performance enhancing drugs over the previous 12 months it is possible that usage of drugs prior to this time frame may have influenced heart morphology and function. It should however be emphasised that approximately 80% of participants had a consistent history of competing in drug-tested events prior to enrolment in the study. Also, it has been suggested that fat-free mass index could be used as a screening tool for identification of possible anabolic androgenic steroid abuse.[44] The threshold for non-steroid users appears to be 25.0 kg/m^2 and for the present study this was only exceeded by one participant. Therefore, if any participants were taking AAS it appears this would likely be low. This study was observational so it is inevitable that other unmeasurable exposures or factors may have caused the results found. While there were novel and interesting findings concerning the associations between muscle performances, further information regarding training history of participants may have assisted with explaining inconsistencies between certain parameters. For example, to understand why there were moderate to strong associations found between echocardiogram parameters and 1RM leg press but no significant associations with 1RM chest press. The sample size of the study was small which increases the risk of Type 2 errors (false negative). However, the findings from this study do provide novel information in relation to the potential influence of muscle performance on the heart in resistance trainers which should be further examined in larger sample sized studies in the future.

Conclusions

Findings from this pilot study show that body composition is associated with echocardiogram parameters in experienced resistance trainers, with fat-free mass appearing to have the greatest influence. This confirms findings from previous studies regarding the relationship between body composition and the heart in both athletic and general healthy population cohorts. Muscle performance was also shown to influence heart structure and function which has not been previously investigated within experienced resistance trainers. Caution is warranted when interpreting the current study's findings due to the small sample size and causality cannot be established since this was an observational study. Since training (e.g. resistance and aerobic training history) and dietary practices (e.g. types of diets and supplementation) were not extensively examined in this study, these behaviors as well as possible anabolic androgenic steroids use may have confounded the results. Therefore, this pilot study should be used to develop future research with larger sample sizes to substantiate the current study's findings.

Disclosure Statement

No potential conflicts of interest were reported by the authors.

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Table 1. Echocardiographic characteristics with referenced normative values.

Echocardiogram Parameters	Normative values (not sex specific)	Males (n = 17)	Females (n = 8)	P value
AoR (mm)	21.0 – 43.0	30.0 (27.0-37.0)	24.0 (19.0-26.0)	<0.01
AAo Ann (mm)	20.0-31.0	2.4 (2.1-2.7)[a]	2.0 (1.8-2.0)[c]	<0.01
AAo SoV (mm)	29.0-45.0	3.3 (2.9-3.6)[a]	2.7 (2.5-2.8)[c]	<0.01
AAo STJ(mm)	22.0-36.0	2.9 (2.5-3.3)[a]	2.5 (2.1-2.5)[c]	0.02
DAo (mm)	20.0-30.0	2.8 (2.5-3.2)[a]	2.6 (2.1-3.0)[c]	0.31
LA (mm)	26.7–41.0	36.0 (32.0-37.0)	33.0 (31.0-37.0)	0.37
RVID (mm)	19.7–37.5	22.0 (18.0-23.0)	21.0 (18.0-26.0)	0.98
IVSs (mm)	6.0–11.3	10.0 (9.0-11.0)	8.0 (7.0-9.0)	0.02
LVPWs (mm)	6.5–11.4	10.0 (9.0-11.0)	9.0 (7.0-10.0)	0.08
LVIDd (mm)	36.8–52.7	54.0 (51.0-58.0)	45.0 (41.0-48.0)	<0.01
LVIDs (mm)	22.3–37.7	35.0 (32.0-37.0)	30.0 (28.0-31.0)	<0.01
FS (%)	>26	34.9 (30.3-37.3)[a]	36.0 (29.8-38.6)	0.79
MVEwave (mm/s)	65.0–87.0	83.0 (73.5-91.7)	82.0 (77.3-95.0)	0.89
MVAwave (mm/s)	48.0-69.0	44.0 (34.0-52.5)	52.5 (48.0-58.0)	0.10
Ewave Decel. (ms)	157.0–211.0	225.0 (180.0-250.0)	213.0 (191.8-251.8)	0.93
E/A ratio	1.0-1.6	1.9 (1.6-2.3)	1.6 (1.4-1.9)	0.10
E' (m/s)	4.6-11.3	10.4 (9.7-11.8)[b]	10.9 (9.0-11.9)	0.57
E/E'	4.0-11.6	8.1 (6.8-8.9)[b]	8.0 (6.6-9.1)[d]	0.60
IVRT (ms)	89.0 ± 21.0	77.0 (74.0-87.3)[a]	74.0 (67.0-77.0)[c]	0.20
LA area (cm^2)	11.5–21.9	19.9 (15.3-22.9)[a]	18.7 (14.6-21.3)	0.70
LVEDvol (ml)	58.5–146.3	139.0 (121.5-161.5)	92.0 (81.5-111.0)	0.01
LVESvol (ml)	18.9–56.6	54.0 (45.0-70.00	38.0 (23.8-46.0)	<0.01
LV EF (%)	55.2–73.3	58.0 (57.0-60.8)[a]	65.0 (56.5-67.8)	0.12

[a] participant n =16; [b] participant n =15; [c] participant n =7; [d] participant n =6

AoR = aortic root; AAo Ann = ascending aorta annulus; AAo SoV = ascending aorta Sinus of Valsalva; AAo STJ = ascending aorta Sinotubular junction; DAo = descending aorta; LA = left atrium; RVID = right ventricular internal diameter; IVSs = interventricular septum thickness end systole; LVPWs = left ventricular posterior wall thickness end systole; LVIDd = left ventricular internal dimension diastole; LVIDs = left ventricular internal dimension systole; FS = fractional shortening; MVEwave = mitral inflow velocities E (early diastolic); MVAwave = mitral inflow velocities A (atrial contraction); Ewave Decel. = E (early diastolic) wave deceleration time; E/A velocity ratio = early diastolic/ atrial contraction velocity ratio; E' = Early diastolic mitral annular motion; E/E' = ratio of E to E'; IVRT = isovolumic relaxation time; LA area = left atrial area; LVEDvol = left ventricular end-diastolic volume; LVESvol = left ventricular end-systolic volume; LV EF = left ventricle ejection fraction.

References for normative values = AoR [45]; LA, RVID, IVSs, LVPWs, LVIDd, LVIDd, LVIDs, FS, LA area, LVEDvol, VVESvol, LV ejection fraction [46]; FS [47]; MVEwave, MVAwave, Ewave Decel., E/A ratio [48]; IVRT [49]; E', E/E' [48]; AAo Ann, AAo SoV, AAo STJ, DAo [50].

Table 2. Body composition and muscle performance characteristics.

Parameter	Males (n = 17)	Females (n = 8)	P value
Age (y)	33.0 (26.0-40.0)	32.5 (22.5-43.8)	0.71
Height (cm)	177.0 (173.0-182.0)	160.4 (155.7-166.9)	<0.01
Body mass (kg)	88.9 (74.7-94.3)	68.0 (63.0-72.4)	0.01
Fat mass (kg)	15.5 (8.4-22.8)	19.7 (14.1-26.6)	0.11
FFM (kg)	70.0 (65.7-77.8)	48.9 (42.2-52.7)	<0.01
FFM index (kg/m^2)	22.9 (21.1-24.1)	19.1 (17.2-19.4)	<0.01
Body fat (%)	19.5 (10.6-25.2)	29.6 (26.8-39.6)	<0.01
CP 1RM (N)	1000.0 (900.0-1128.0)	485.0 (452.5-585.5)	<0.01
CP 1RM/FFM (N/kg)	13.8 (12.7-13.8)	10.9 (9.4-11.8)	<0.01
CP peak power (W)	849.0 (720.5-959.0)	371.0 (360.0-478.3)	<0.01
CP peak power/FFM (W/kg)	11.6 (10.6-13.7)	8.4 (7.2-9.4)	<0.01
CP endurance (repetitions)	21.0 (18.5-23.0)	28.5 (21.3-30.80	0.03
LP 1RM (N)	3725.0 (2868.8-4045.0)[a]	2250.0 (2050.0-2875.0)	0.01
LP 1RM/FFM (N/kg)	48.5 (41.9-56.4)[a]	49.5 (43.0-52.8)	0.75
LP peak power (W)	2427.0 (2037.8-2932.3)[a]	1413.5 (1235.8-1649.3)	<0.01
LP peak power/FFM (W/kg)	34.1 (30.8-37.3)[a]	28.0 (26.6-34.8)	0.24
LP endurance (repetitions)	32.5 (26.8-42.5)[a]	40.0 (35.8-53.3)	0.21

[a] participant n =16; CP = chest press; LP = leg press; 1RM = one-repetition maximum; FFM = fat-free mass

Table 3. Relationship between echocardiographic parameters and body composition in experienced resistance trainers.

Echocardiogram Parameters	Body mass	Fat mass	Fat-free mass	Fat-free mass index	Body fat (%)
AoR	0.38	0.26	0.43*	0.39	0.15
AAo Ann	0.43*	0.36	0.37	0.39	0.33
AAo SoV	0.58†	0.38	0.55*	0.52*	0.31
AAo STJ	0.50*	0.15	0.46*	0.48*	0.17
DAo	0.51*	0.21	0.45*	0.47*	0.23
LA	0.46	0.23	0.29	0.31	0.13
RVID	0.35	-0.08	0.60†	0.57†	-0.14
IVSs	0.58†	0.27	0.57†	0.44*	0.21
LVPWs	0.59†	0.23	0.55†	0.46*	0.15
LVIDd	0.32	0.45*	0.10	0.26	0.38
LVIDs	0.08	0.01	0.14	0.08	-0.06
FS	0.21	0.18	0.14	0.07	0.19
MVEwave	0.36	0.43	0.09	0.01	0.36
MVAwave	0.39	0.39	0.16	0.21	0.35
Ewave Decel.	-0.15	-0.29	-0.11	-0.12	-0.21
E/A ratio	-0.25	-0.21	-0.17	-0.23	-0.21
E'	-0.15	0.06	-0.18	-0.42	0.10
E/E'	0.33	0.21	0.21	0.35	0.14
IVRT	-0.23	-0.14	-0.07	-0.02	-0.04
LA area	0.65†	0.30	0.76†	0.59†	0.14
LVEDvol	0.66†	0.34	0.64†	0.65†	0.22
LVESvol	0.55†	0.37	0.47*	0.45*	0.24
LV EF	0.08	0.04	0.15	0.17	0.04

*Correlation is significant at $p < 0.05$; †Correlation is significant at $p < 0.01$.

AoR = aortic root; AAo Ann = ascending aorta annulus; AAo SoV = ascending aorta Sinus of Valsalva; AAo STJ = ascending aorta Sinotubular junction; DAo = descending aorta; LA = left atrium; RVID = right ventricular internal diameter; IVSs = interventricular septum thickness end systole; LVPWs = left ventricular posterior wall thickness end systole; LVIDd = left ventricular internal dimension diastole; LVIDs = left ventricular internal dimension systole; FS = fractional shortening; MVEwave = mitral inflow velocities E (early diastolic); MVAwave = mitral inflow velocities A (atrial contraction); Ewave Decel. = E (early diastolic) wave deceleration time; E/A velocity ratio = early diastolic/ atrial contraction velocity ratio; E' = Early diastolic mitral annular motion; E/E' = ratio of E to E'; IVRT =

isovolumic relaxation time; LA area = left atrial area; LVEDvol = left ventricular end-diastolic volume; LVESvol = left ventricular end-systolic volume; LV EF = left ventricle ejection fraction.

Table 4. Relationship between echocardiographic parameters and muscle performance in experienced resistance trainers.

Echocardiogram	Chest press					Leg press				
		1RM/F		PP/FF			1RM/F		PP/FF	
Parameters	1RM	FM	PP	M	Endur.	1RM	FM	PP	M	Endur.
AoR	0.15	-0.40	-0.19	-0.54†	0.02	0.49*	0.28	0.21	0.15	0.17
AAo Ann	0.15	-0.08	-0.06	-0.35	0.12	0.37	0.09	<0.01	-0.21	0.20
AAo SoV	0.34	-0.09	0.14	-0.29	0.02	0.58*	0.18	0.15	-0.14	0.13
AAo STJ	0.32	0.01	0.25	-0.14	0.15	0.41	0.01	-0.04	-0.40	0.39
DAo	0.31	0.01	0.15	-0.21	0.18	0.36	0.02	0.04	-0.26	0.18
LA	0.27	0.12	0.54†	0.34	-0.07	0.19	-0.09	0.35	0.14	-0.03
RVID	0.26	-0.18	0.27	-0.22	-0.05	0.31	-0.09	0.17	-0.19	0.41
IVSs	0.12	-0.25	0.04	-0.35	-0.30	0.38	0.09	0.27	0.06	0.16
LVPWs	0.23	-0.10	0.22	-0.22	-0.15	0.29	0.01	0.27	0.04	0.21
LVIDd	0.27	0.15	0.19	0.07	-0.39	0.52*	0.30	0.26	0.04	-0.24
LVIDs	-0.03	-0.15	-0.20	-0.27	-0.03	0.00	-0.20	0.06	-0.11	0.24
FS	-0.16	-0.24	0.05	-0.02	-0.10	0.33	0.24	0.00	-0.08	-0.04
MVEwave	0.30	0.14	0.10	0.11	-0.28	0.27	0.33	0.22	0.14	-0.22
MVAwave	0.22	0.32	0.18	0.11	0.20	0.02	0.10	0.16	0.15	-0.10
Ewave Decel.	-0.12	-0.19	-0.10	0.19	0.20	-0.05	-0.08	-0.13	-0.05	-0.01
E/A ratio	-0.11	-0.26	-0.19	-0.08	-0.30	0.07	0.03	-0.08	-0.11	0.03
E'	-0.33	-0.30	-0.13	0.07	0.01	-0.25	-0.22	-0.20	-0.11	-0.01
E/E'	0.42	0.37	0.08	0.01	-0.10	0.27	0.33	0.33	0.26	-0.15
IVRT	-0.09	-0.22	-0.02	-0.05	-0.33	0.20	0.06	-0.24	-0.28	-0.04
LA area	0.23	-0.23	0.11	-0.47*	-0.25	0.55†	0.10	0.45*	-0.03	0.29
LVEDvol	0.31	-0.08	0.28	-0.23	-0.38	0.67†	0.29	0.26	-0.15	0.13
LVESvol	0.32	0.07	0.23	-0.17	-0.44*	0.56†	0.24	0.25	-0.13	0.15
LV EF	-0.10	-0.34	-0.16	-0.33	0.31	0.12	0.11	0.13	0.15	-0.09

*Correlation is significant at $p < 0.05$; †Correlation is significant at $p < 0.01$.

AoR = aortic root; AAo Ann = ascending aorta annulus; AAo SoV = ascending aorta Sinus of Valsalva; AAo STJ = ascending aorta Sinotubular junction; DAo = descending aorta; LA = left atrium; RVID = right ventricular internal diameter; IVSs = interventricular septum thickness end systole; LVPWs = left ventricular posterior wall thickness end systole; LVIDd = left ventricular internal dimension diastole; LVIDs = left ventricular internal dimension systole; FS = fractional shortening; MVEwave = mitral inflow velocities E (early diastolic);

MVAwave = mitral inflow velocities A (atrial contraction); Ewave Decel. = E (early diastolic) wave deceleration time; E/A velocity ratio = early diastolic/ atrial contraction velocity ratio; E' = Early diastolic mitral annular motion; E/E' = ratio of E to E'; IVRT = isovolumic relaxation time; LA area = left atrial area; LVEDvol = left ventricular end-diastolic volume; LVESvol = left ventricular end-systolic volume; LV EF = left ventricle ejection fraction.

Figure 1. Relationship between partial residuals for fat-free mass and partial residuals for right ventricular internal diameter (RVID), and partial residuals for left atrium area (LA). Relationships were both significant at $p < 0.01$.

Figure 2. Relationship between partial residuals for leg press one-repetition maximum (1RM) and partial residuals for left ventricular end-diastolic volume (LVEDvol). Relationship was significant at $p < 0.01$.