CARDIOVASCULAR MEDICINE

Exercise effects on cardiac size and left ventricular diastolic function: relationships to changes in fitness, fatness, blood pressure and insulin resistance

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Objectives: To determine exercise training effects on cardiac size and left ventricular (LV) diastolic function and relationships of exercise induced changes in physiological and body composition parameters with cardiac parameters.
Design: Prospective, randomised controlled trial.
Subjects: Men and women (63.6 (5.7) years, body mass index 29.5 (4.4) kg/m^2) with untreated hypertension (systolic blood pressure (BP) 130–159 or diastolic BP 85–99 mm Hg).
Main outcome measures: Cardiac size and LV diastolic function, peak oxygen uptake (V_{O_2}), muscle strength, general and abdominal fatness, and insulin resistance.
Interventions: 6 months of exercise training versus usual care.
Results: When analysed by group at six months, cardiac size and LV diastolic function did not differ between exercisers (n = 51) and controls (n = 53), whereas exercisers had significantly higher peak V_{O_2} (28 *v* 24 ml/kg/min) and strength (383 *v* 329 kg), and lower fatness (34% *v* 37%), diastolic BP (73 *v* 75 mm Hg) and insulin resistance (quantitative insulin sensitivity check index 0.35 *v* 0.34) versus controls (all $p \leqslant 0.05$). By regression analysis, among six month changes, increased peak V_{O_2} and reduced abdominal fat were associated with increased cardiac size. Increased peak V_{O_2} and reduced abdominal fat, BP and insulin resistance were associated with improved LV diastolic function. *r* Values ranged from 0.20 to 0.32 ($p \leqslant 0.05$).
Conclusions: When examined by group assignment, exercise had no effect on cardiac size or LV diastolic function. When individual variations in six month changes were examined, participants attaining the greatest increases in fitness and reductions in abdominal fatness, insulin resistance and BP showed a modest trend towards physiological hypertrophy characterised by increased cardiac size and improved LV diastolic function. These results suggest that decreased abdominal fatness may have a role in improving cardiovascular health.

Hypertension is associated with increased cardiac size and impaired left ventricular (LV) diastolic function. Although exercise training is recommended for reducing blood pressure (BP),[1 2] each workout acutely increases BP and hence may have adverse cardiac effects. Studies examining exercise effects on cardiac size and function in older people have been equivocal and limited by small sample sizes.[3–5]

Increased fatness and the associated condition of insulin resistance also have adverse cardiac effects.[6 7] Conversely, exercise reduces fatness,[8–10] which is a pathway by which exercise reduces BP and insulin resistance.[10] For this reason, exercise induced reductions in body fatness may lead to beneficial cardiac effects.

The present participants were enrolled in the Senior Hypertension and Physical Exercise (SHAPE) study, a randomised controlled trial of six months of aerobic and resistance exercise.[9 10] The present study examined changes in cardiac size and LV diastolic function and relationships between changes in fitness, fatness, BP, and insulin resistance and cardiac parameters.

METHODS

The Johns Hopkins Institutional Review Board, Baltimore, Maryland, approved the study. Informed written consent was obtained from each participant.

The details of study recruitment, procedures, intervention and participant flow are given elsewhere.[9 10] Briefly, participants were aged 55–75 years and in good health except for untreated mild hypertension. Exclusion criteria were cardiovascular diseases or other serious illnesses; smoking; diabetes; and regular exercise > 90 min/week. For BP eligibility, participants were required to have untreated systolic BP (SBP) between 130–159 or diastolic BP (DBP) between 85–99 mm Hg during two consecutive weekly visits and an average in this range over four visits.

Maximal exercise testing followed the BP screening. Participants with ST segment depression > 1 mm, complex arrhythmias or ischaemic symptoms were excluded. Ultimately, 115 participants were randomly assigned to study groups.[8–10]

Baseline and follow-up measures

BP and heart rate

BP was measured by an automated cuff (Dinamap MPS Select; Johnson & Johnson, New Brunswick, New Jersey, USA). After five minutes of rest, BP was measured three times with one minute between readings. If they differed by

Abbreviations: A, peak late diastolic filling wave velocity; Am, peak mitral annular late diastolic velocity during atrial contraction; BP, blood pressure; DBP, diastolic blood pressure; E, peak early diastolic filling wave velocity; Em, peak mitral annular early diastolic velocity; LV, left ventricular; MRI, magnetic resonance imaging; QUICKI, quantitative insulin sensitivity check index; SBP, systolic blood pressure; V_{O_2}, oxygen uptake

more than 5 mm Hg, extra readings were obtained. The mean of three consecutive readings within 5 mm Hg of each other was the examination value. Baseline BP was the mean of all screening visits and a visit after the participant qualified for the study. Final BP was the mean of BP taken during the last month of the study and during final testing. Heart rate was determined similarly.

Fitness

Peak oxygen uptake (V_{O_2}) was measured with a Vmax 229 Metabolic System (SensorMedics, Yorba Linda, California, USA). Treadmill walking began at 4.8 km/h, at a grade of 0%, and was increased by 2.5% every three minutes. Participants exercised to volitional fatigue. A participant's maximum effort was validated by reaching 18 or higher on the Borg Rating of Perceived Exertion scale,[11] by reaching a respiratory exchange ratio of 1.1 or greater and by heart rate or V_{O_2} plateauing.

Muscle strength was assessed by one-repetition maximum (highest weight on one attempt)[12] on each of seven exercises on a multistation machine (Hoist Fitness, San Diego, California, USA). Exercises were the bench press, shoulder press, seated mid-rowing, lat pull-down, leg extension, leg curl and leg press. Muscle strength is reported as the sum of weights of these exercises.

Body composition

Anthropometric measures were body weight, height, body mass index[9 10] and body surface area.[13] Percentage body fat and lean mass were determined by dual energy x ray absorptiometry (GE Lunar Prodigy; General Electric Medical Systems, Milwaukee, Wisconsin, USA) Abdominal total, visceral and subcutaneous fat were measured by magnetic resonance imaging (MRI) with a Vision 1.5 T MRI system (Siemens Medical Systems, Iselin, New Jersey, USA). An experienced reader following procedures described elsewhere[12 14] traced and averaged 1 cm wide slices obtained at one slice below, at and above the umbilicus.

Blood chemistry analysis

Glucose and insulin were measured from fasting blood samples. Insulin resistance was assessed with the quantitative insulin sensitivity check index (QUICKI).[15] QUICKI is defined as $1/[\log (I_0)+\log (G_0)]$, where I_0 is fasting plasma insulin and G_0 is fasting plasma glucose concentration. A lower index indicates more insulin resistance.

LV mass by MRI

A stack of short axis gradient echo slices from the base to apex was acquired from the same MRI instrument that was used for abdominal measurements. Images were processed with the Cardiac Image Processing System (Cardiology Division Systems and Computing laboratory, The Johns Hopkins Medical Institutions, Baltimore, Maryland, USA).[16 17] The end diastolic endocardial margin was contoured manually. The LV cavity area in each slice was calculated, and volume was estimated by factoring the known slice thickness. This was repeated at the epicardial boundary, and the volume estimated included LV myocardium and cavity. By subtracting cavity volume and multiplying by tissue mass density, all slices were summed to yield total LV mass. Our reproducibility for LV mass[17] is intraobserver percentage error of 2.7%, correlation coefficient $r = 0.99$; and interobserver percentage error of 10.4%, $r = 0.95$. LV mass was indexed by dividing LV mass by body surface area (left ventricular mass index).

Cardiac size and function by echocardiography

Standard views were obtained with a Sonos 5500 or 2500 system (Agilent Technologies, Palo Alto, California, USA). Measurements were taken on an Image-Vue analysis system (Nova Microsonics, Bellevue, Washington, USA) or an EnConcert system (Philips Medical Systems, Andover, Massachusetts, USA). Two dimensional, M mode and Doppler examinations were performed with a 2.5 MHz transducer. Three measurements of each parameter were made and averaged. Dimensions were measured at end systole and diastole. Relative wall thickness at end diastole was defined as twice the posterior wall thickness divided by the LV internal radius. LV diastolic function was assessed by analysis of mitral inflow patterns, isovolumic relaxation and tissue Doppler imaging measurements of mitral annular motion during diastole. Mitral inflow measurements included early (E) and late (A) diastolic filling wave velocities recorded at the leaflet tips. LV isovolumic relaxation time was defined as the interval between aortic valve closure click and onset of transmitral flow. Propagation velocity was determined from colour M mode images. The cursor was aligned within the main direction of mitral inflow from the apical four chamber view. The slope of the first aliasing contour was measured from the mitral valve plane to a point 4 cm distally into the LV cavity. Tissue Doppler imaging measures of diastolic function from a fixed 5 mm sample at the lateral aspect of the mitral annulus were early (Em) and late (Am) mitral annular velocities and the Em:Am and E:Em ratios.

Exercise intervention

Following American College of Sports Medicine guidelines[1] the program consisted of 78 supervised sessions (three days for 26 weeks). Each session began with a warm up, followed by resistance and then aerobic training. Resistance training consisted of two sets of 10–15 repetitions for each exercise, at 50% of one-repetition maximum. The seven exercises performed were the same as those for testing. Aerobic exercise lasted 45 minutes on the participant's choice of a treadmill, stationary cycle or stair stepper. The target heart rate range was set at 60–90% of the maximum heart rate during baseline treadmill testing and was monitored with heart rate monitors (Polar, Lake Success, New York, USA). As fitness improved, the aerobic workload was increased to maintain heart rate at target levels. For resistance exercise, the weight lifted was increased when the participant could complete 15 repetitions without difficulty.

Control group, diet and physical activity, and BP monitoring

Because activity and diet are usual care recommendations for hypertension,[18] participants were given the National Institute of Aging Guidelines for Exercise (http://www.niapublications.org/exercisebook/exercisebook.asp) and the American Heart Association Step I Diet (http://www.americanheart.org). All participants reported twice monthly for BP checks. If SBP was > 159 or DBP > 99 mm Hg, they were assessed weekly, and withdrawn if BP was above range for four weeks.

Statistical analysis

Data were analysed with JMP 5.1 software (SAS Institute, Cary, North Carolina, USA). Between-group differences at baseline were examined by t tests. The group comparisons in response to treatment applied analysis of covariance with general linear models. For each outcome variable, the six month value was the dependent variable and the baseline value and group assignment were the independent variables. To ascertain determinants of individual differences in response to treatment, data for both groups were combined and Pearson correlations were calculated between six month changes in physiological and fatness variables with cardiac parameters. Where more than one bivariate relationship achieved significance, stepwise regression models were

Table 1 Baseline and six month blood pressure, heart rate, fitness and body composition of participants randomly assigned to exercise or control

	Exercise (n = 51)		Control (n = 53)		
Variable	**Baseline**	**6 Months**	**Baseline***	**6 Months**	**p Value†**
Resting haemodynamic function					
Systolic blood pressure (mm Hg)	140.3 (138.2 to 142.4)	135.0 (131.3 to 138.8)	141.7 (139.7 to 143.8)	137.3 (134.4 to 140.2)	0.66
Diastolic blood pressure (mm Hg)	76.8 (74.8 to 78.9)	73.1 (71.0 to 75.2)	76.4 (73.9 to 78.9)	74.8 (72.3 to 77.4)	0.02
Heart rate (beats/min)	69.8 (67.7 to 71.9)	65.9 (64.0 to 67.8)	71.9 (69.3 to 74.4)	69.7 (67.5 to 71.9)	0.01
Aerobic and strength fitness					
Peak oxygen uptake (ml/kg/min)	24.4 (22.9 to 25.9)	28.4 (26.7 to 30.1)	24.2 (22.8 to 25.7)	24.1 (22.6 to 25.6)	<0.001
Upper body muscle strength (kg)	173.2 (154.5 to 192.0)	199.9 (178.5 to 221.4)	175.9 (155.9 to 195.9)	174.9 (154.4 to 195.3)	<0.001
Lower body muscle strength (kg)	155.2 (141.0 to 169.4)	183.0 (168.3 to 197.8)	151.8 (138.8 to 164.7)	154 (140.6 to 167.4)	<0.001
Total muscle strength (kg)	328.4 (296.2 to 360.6)	383.0 (347.4 to 418.6)	327.7 (295.3 to 360.0)	328.9 (296.0 to 361.8)	<0.001
Body composition					
Body mass index (kg/m^2)	29.4 (28.3 to 30.4)	28.5 (27.5 to 29.6)	29.7 (28.3 to 31.0)	29.5 (28.2 to 30.8)	<0.001
Weight (kg)	83.2 (79.1 to 87.3)	80.9 (76.7 to 85.2)	84.9 (79.6 to 90.2)	84.4 (79.1 to 89.6)	0.002
Abdominal total fat (cm^2)‡	432.6 (399.5 to 465.6)	377.3 (341.5 to 413.1)	449.6 (404.2 to 495.0)	443.1 (400.0 to 485.9)	<0.001
Abdominal visceral fat (cm^2)‡	146.5 (127.3 to 165.7)	119.4 (101.4 to 137.4)	142.7 (123.7 to 161.6)	138.9 (120.1 to 157.6)	<0.001
Abdominal subcutaneous fat (cm^2)‡	285.1 (255.4 to 314.9)	257.0 (226.8 to 287.1)	305.7 (268.9 to 342.4)	302.9 (267.7 to 338.1)	<0.001
Total body fat (%)§	37.9 (35.4 to 40.4)	34.4 (31.7 to 37.1)	37.7 (35.0 to 40.5)	37.5 (34.8 to 40.3)	<0.001
Lean body mass (%)§	58.5 (56.1 to 61.5)	62 (59.4 to 64.6)	58.9 (56.2 to 61.5)	59.1 (56.4 to 61.7)	<0.001
Glucose and insulin					
Glucose (mmol/l)	5.59 (5.4 to 7.8)	5.67 (5.5 to 5.84)	5.61 (5.4 to 5.8)	5.75 (5.6 to 5.9)	0.33
Insulin (pmol/l)	59.7 (51 to 68)	68.1 (57 to 79)	55.6 (48 to 63)	68.1 (58 to 78)	0.14
QUICKI	0.35 (0.34 to 0.36)	0.35 (0.33 to 0.35)	0.35 (0.34 to 0.36)	0.34 (0.33 to 0.35)	0.04

Data are mean (95% confidence interval).
*There were no exercise versus control group differences for any baseline measures (p values for these comparisons not shown); †p values shown are for the comparison of exercisers versus controls at six months; ‡measured by magnetic resonance imaging; §measured by dual-energy *x* ray absorptiometry.
QUICKI, quantitative insulin sensitivity check index.

constructed to assess independent contributions of physiological and fatness variables to cardiac changes.

RESULTS

Participant characteristics

Eight patients dropped out (four in each group for personal reasons) and three withdrew (one in each group for raised BP, and one exerciser for an unrelated illness). Complete data are available for 104 participants: 51 exercisers (25 men; 26 women) and 53 controls (26 men; 27 women); 91 (87%) were non-Hispanic white, 11 (11%) African-American, 1 (1%) Asian-American and 1 (1%) Hispanic. Age among exercisers (means (SD) 63.0 (5.3) years) did not differ significantly from that of controls (64.1 (6.1) years). There were no

Table 2 Baseline and six month cardiac structure and function of randomised participants in the SHAPE study

	Exercise (n = 51)		Control (n = 53)		
Variable	**Baseline**	**6 Months**	**Baseline**	**6 Months**	**p Value***
LV size by MRI					
LV mass (g)	125.2 (116.8 to 133.5)	125.7 (116.7 to 134.8)	130.1 (119.5 to 140.1)	129.5 (119.3 to 139.7)	0.99
LVMI (g/m^2)	63.6 (60.1 to 67)	65.1 (61.3 to 68.8)	65.1 (60.9 to 69.4)	65.0 (61.3 to 68.7)	0.63
LV size by echocardiography					
LVEDD (cm)	4.7 (4.6 to 4.8)	4.6 (4.5 to 4.8)	4.5 (4.4 to 4.6)	4.6 (4.4 to 4.7)	0.21
LVESD (cm)	3.2 (3 to 3.3)	3.0 (2.9 to 3.2)	3.1 (2.9 to 3.2)	3.0 (2.9 to 3.2)	0.52
VST (mm)	1.0 (1.0 to 1.1)	1.0 (1.0 to 1.1)	1.1 (1.0 to 1.1)	1.1 (1.0 to 1.1)	0.48
PWT (mm)	1.0 (1.0 to 1.1)	1.0 (1.0 to 1.1)	1.1 (1.0 to 1.1)	1.0 (1.0 to 1.1)	0.88
RWT (mm)	0.44 (0.42 to 0.46)	0.45 (0.43 to 0.47)	0.47 (0.45 to 0.49)*	0.46 (0.44 to 0.47)	0.93
LA size (cm)	3.7 (3.6 to 3.8)	3.7 (3.5 to 3.8)	3.8 (3.7 to 3.9)	3.7 (3.6 to 3.9)	0.68
LV function by Doppler echocardiography					
E (cm/s)	74.9 (71.2 to 78.5)	73.5 (69.5 to 77.4)	73.8 (70.2 to 77.4)	72.3 (68.6 to 76.9)	0.88
A (cm/s)	81.4 (77.1 to 85.7)	83.3 (73.2 to 93.5)	83.4 (79.4 to 87.4)	83.0 (78.9 to 87.1)	0.77
E:A ratio	0.93 (0.88 to 0.99)	0.94 (0.87 to 1.01)	0.91 (0.85 to 0.96)	0.89 (0.84 to 0.95)	0.25
DT (ms)	213.1 (204.3 to 222.0)	233.3 (219.8 to 246.9)	225.2 (209.9 to 240.6)	232.2 (212.2 to 252.1)	0.58
IRT (ms)	93.6 (89.6 to 97.5)	93.1 (88.8 to 97.5)	90.8 (86.2 to 95.4)	92.7 (87.3 to 98.1)	0.71
LV function by tissue Doppler and colour M mode propagation					
Em (cm/s)	10.1 (9.4 to 10.6)	9.9 (9.2 to 10.5)	10.6 (9.9 to 11.4)	9.8 (9.2 to 10.4)	0.25
Am (cm/s)	13.4 (12.6 to 14.1)	12.4 (11.7 to 13.1)	13.2 (12.4 to 14.1)	12.8 (11.9 to 13.7)	0.35
Em:Am ratio	0.84 (0.77 to 0.90)	0.81 (0.75 to 0.87)	0.87 (0.80 to 0.95)	0.79 (0.73 to 0.85)	0.18
Vp (cm/s)	56.5 (50.1 to 62.8)	54.3 (48.1 to 60.5)	56.5 (51.1 to 62.8)	53.6 (48.1 to 59.1)	0.97
E:Vp ratio	1.5 (1.3 to 1.6)	1.5 (1.3 to 1.7)	1.4 (1.3 to 1.6)	1.5 (1.3 to 1.6)	0.98
E:Em ratio	7.8 (7.2 to 8.5)	7.7 (7.1 to 8.3)	7.3 (6.8 to 7.8)	7.7 (7.1 to 8.3)	0.6

Data are mean (95% confidence interval).
*p Values shown are for the comparison of exercisers versus controls at six months; †p<0.01 for comparison of exercisers versus controls at baseline. No other measures were different between groups at baseline.
A, peak late diastolic filling wave velocity; Am, peak mitral annular late diastolic velocity during atrial contraction; DT, deceleration time; E, peak early diastolic filling wave velocity; Em, peak mitral annular early diastolic velocity; IRT, isovolumic relaxation time; LA, left atrial; LV, left ventricular; LVEDD, left ventricular end diastolic dimension; LVESD, left ventricular end systolic dimension; LVMI, left ventricular mass index; PWT, posterior wall thickness; RWT, relative wall thickness; Vp, propagation of mitral inflow; VST, ventricular septal thickness.

significant group differences in physiological or fatness parameters at baseline (table 1). Participants not completing the study had baseline values similar to those completing the study (data not shown).

Adherence to exercise training

Exercisers completed 69 (8) sessions (88% compliance). Because of missed sessions, 11 participants exercised for an extra month to achieve at least 62 sessions (80%). In separate analyses (not shown), the responses to training from these participants did not differ from responses from the other exercisers. Only one exerciser fell below 80% compliance (34 sessions; 44%). Exercise heart rate was in the prescribed ranges 98% of the time, averaging 135.5 (10.4) beats/min during 2587 (55) seconds/session of aerobic exercise.

Exercise training effects

At six months, SBP was not significantly different between groups, whereas DBP and heart rate were lower among exercisers (each $p < 0.01$) (table 1). At six months, peak Vo_2, muscle strength and percentage lean body mass were significantly higher among exercisers than among controls. Each measure of fatness and insulin resistance was significantly decreased and lean mass was significantly increased among exercisers compared with controls.

The only baseline group difference between cardiac parameters (table 2) was a slightly lower relative wall thickness ($p < 0.01$) among exercisers. No significant differences between groups were found in any cardiac measure at six months.

Determinants of change in cardiac size and diastolic function

Although there were no training effects on cardiac parameters, change varied between participants. The change in left atrial size correlated positively with change in peak Vo_2 ($r = 0.22$, $p = 0.03$). The change in LV mass index correlated negatively with change in abdominal total ($r = -0.24$, $p = 0.02$), visceral ($r = -0.20$, $p = 0.04$) and subcutaneous fat ($r = -0.20$, $p = 0.05$). In a stepwise model, abdominal fat change was the only independent correlate of LV mass index change, accounting for 6% ($p = 0.02$) of the variance. No other measures of change in cardiac size correlated with changes in physiological and fatness parameters.

Determinants of change in LV diastolic function

Table 3 shows parameters of diastolic function that had at least one significant bivariate correlation with a physiological or fatness parameter. The peak E change correlated negatively with the changes in abdominal total, subcutaneous and visceral fat, and insulin and positively with changes in SBP and QUICKI. The peak A change correlated negatively with change in peak Vo_2. The E:A change correlated positively with change in peak Vo_2 and negatively with change in abdominal total and visceral fat and heart rate. The change in colour M mode flow propagation of mitral inflow correlated negatively with the change in abdominal total fat. The Em change correlated positively with changes in abdominal total, subcutaneous and visceral fat. The Em:Am change correlated negatively with changes in these same measures of abdominal fat and DBP and correlated positively with the change in QUICKI. The E:Em change correlated positively with the change in SBP.

In the stepwise regression models, an increase in E was accounted for by reductions in insulin, accounting for 7% of the variance ($p < 0.01$), increased SBP (7%, $p < 0.01$) and decreased abdominal total fat (5%, $p = 0.02$). An increase in E:A was accounted for by an increase in peak Vo_2 (6%, $p < 0.01$). Increases in Em and Em:Am were each accounted for by decreased abdominal total fat, which accounted for 10% of the variance ($p < 0.01$) for each parameter. Decreased E:Em was accounted for by decreased SBP (7%, $p = 0.01$). These associations were unchanged when adjusted for sex.

DISCUSSION

This is the first randomised controlled trial to examine cardiac size and LV diastolic function in older people with mild hypertension participating in an exercise programme recommended for hypertension.[1] When analysed by group assignment, there were no training effects on any measured cardiac parameter. This is also the first study in adults in which cardiac parameters, fitness, body composition, BP and insulin resistance were studied simultaneously. When responses were examined individually, reductions in abdominal fatness were associated, albeit modestly, with improvements in LV diastolic function.

Exercise effects on cardiac size and LV diastolic function

Aerobic training imposes primarily a volume load on the heart, whereas resistive exercise imposes primarily a pressure load. These haemodynamic responses have raised concerns that the increases of preload or afterload during a workout may be detrimental to people with hypertension. Despite evidence of a beneficial training effect on physiological and fatness parameters, we found no evidence of adverse effects of exercise on cardiac size or LV diastolic function.

Whereas the group analysis showed no changes in cardiac size and function, analysis of individual responses showed a

Table 3 Pearson correlations of changes in measures of left ventricular diastolic function with changes in physiological and fatness measures

Variable	Peak E	Peak A	E:A	Vp	Em	Em:Am	E:Em
Peak oxygen uptake	0.01	−0.31**	0.27**	−0.07	0.12	0.13	−0.10
Muscle strength	−0.01	−0.01	0.05	−0.07	0.19	0.19	0.05
Body weight	−0.10	0.05	−0.16	−0.06	−0.07	−0.09	−0.01
Body mass index	−0.14	0.04	−0.18	−0.08	−0.07	−0.09	−0.04
Percentage body fat	0.02	0.02	−0.13	0.01	−0.16	−0.18	−0.06
Abdominal total fat	−0.23*	0.03	−0.22*	−0.20*	−0.33**	−0.30**	0.08
Abdominal subcutaneous fat	−0.21*	0.03	−0.25*	−0.19	−0.32**	−0.28**	0.08
Abdominal visceral fat	−0.16	0.01	−0.10	−0.16	−0.22*	−0.23*	−0.02
Systolic blood pressure	0.24*	0.03	0.07	0.07	−0.15	−0.17	0.27**
Diastolic blood pressure	0.11	−0.06	−0.01	−0.01	−0.17	−0.24*	0.07
Resting heart rate	−0.03	0.07	−0.21*	0.02	−0.14	−0.16	−0.08
Insulin	−0.27**	−0.19	−0.02	−0.14	−0.18	−0.19	−0.03
QUICKI	0.21*	0.13	0.09	0.08	0.19	0.21*	0.01

*p<0.05; **p<0.01.
QUICKI, quantitative insulin sensitivity check index.

modest increase in left atrial size among participants attaining the greatest increases in peak VO_2 and a modest increase in LV mass index among participants attaining the greatest decreases in abdominal fat. Increases in cardiac size resulting from exercise differs from those resulting from hypertension in that hypertension is accompanied by wall thickening and impaired diastolic filling.[19] In this regard, we found that improvements in physiological and fatness parameters correlated with modest improvements in diastolic function based on several methods including standard Doppler E, A and E:A ratio, and newer parameters reflecting LV relaxation by colour M mode and Em and Em:Am by tissue Doppler imaging. Tissue Doppler imaging measurements of mitral annular velocities represent subendocardial changes in strain and are sensitive to subtle changes in LV function.[20] Although modest, these results suggest a physiological hypertrophy among people having the greatest increases in fitness and reductions in fatness. The apparent paradoxical finding of an association of decreased fatness with increased LV mass index as opposed to the increases in LV mass index seen in sedentary obese people reflects the fact that decreased fatness resulted from exercise training that imposed multiple bouts of increased myocardial loading over six months.

Obesity and cardiac size and function

Obesity is a determinant of LV diastolic dysfunction.[21] Pascual *et al*[22] found that increased fatness adversely affected LV diastolic but not systolic function. Possible mechanisms for diastolic dysfunction in obesity are increases in blood volume leading to hypertension, increased LV wall stress and LV hypertrophy.[23] Most studies of cardiac function and fatness have used body mass index, an indicator of general fatness. A new observation herein, albeit modest, is that a decrease in abdominal fatness was associated with improved LV diastolic function. Diastolic function can be impaired by hyperglycaemia,[24] insulin resistance and hyperinsulinaemia.[25–27] In the parent study in which the present participants are enrolled,[9] exercise-induced decreases in insulin resistance were associated with decreases in abdominal fatness.[9] In the present study, decreased fasting insulin correlated with increased E, whereas improved insulin resistance was associated with increased E and Em:Am. Although the finding that decreased SBP was associated with increased mitral E velocity is not easy to explain, more plausible are the associations of decreased SBP with decreased E:Em, an indicator of diastolic filling pressure, and decreased DBP with increased Em:Am. Not surprisingly, participants with the greatest increases in fitness tended to have improved LV diastolic function. An alternate explanation is that abdominal obesity is not causally related to LV diastolic function and may instead be paralleling increased fitness and decreased BP, each of which improve LV diastolic function. In the parent study,[10] decreased abdominal fat was associated with decreased BP and increased fitness.

Strengths and limitations

Among the strengths of this study are that participants had untreated hypertension, the non-completion rate was 10%, adherence to exercise was excellent, and cardiac parameters and body composition were assessed by contemporary imaging techniques. Limitations are that participants had mild hypertension and relatively normal values for cardiac parameters. Thus, the potential for remodelling and improved diastolic function was limited and these results cannot be extrapolated to patients with more severe hypertension. Furthermore, because the sample size determination was based on BP changes in the parent study,[10] the lack of significant change in cardiac parameters may be due to limited statistical power.

Conclusions and clinical implications

Exercise for six months improved fitness, body composition, BP and insulin resistance. Group assignment analysis shows that there were no effects on cardiac size and LV diastolic function. When analysed by changes in physiological and fatness parameters, participants attaining the greatest increases in fitness and reductions in abdominal fatness showed a trend of increased cardiac size. Participants attaining the greatest increases in fitness and reductions in abdominal fatness, insulin resistance and BP showed a trend of improved LV diastolic function. An increase in cardiac size accompanied by improved LV diastolic function suggests a physiological rather than a pathological hypertrophy. Although modest, the association of decreased abdominal fatness with improved LV diastolic function suggests a pathway by which exercise improves cardiovascular health.

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IMAGES IN CARDIOLOGY

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The ''mobile-phonocardiogram'', a new tool in the arrhythmia clinic

A 20 year old man presented to our clinic complaining of palpitations. He had been under cardiological follow up since childhood for a symptom-free ventricular septal defect. He took no medication and was fit and well. He played sport twice per week, did not smoke or drink excess alcohol. He had felt palpitations occasionally since age 15 but they had recently become more frequent, especially at rest after playing sport. On one of these occasions he had recorded his own cardiophonogram using a mobile phone handset pressed against his chest. Physical examination demonstrated a grade 4/6 ejection systolic murmur. His resting ECG was normal. He achieved stage 5 of the Bruce protocol; this did not induce symptoms. A two dimensional echocardiogram demonstrated a ventricular septal defect with a 1:1.2 shunt. Three dimensional echocardiography revealed three 3 × 3 mm defects just below the tricuspid valve. The right heart was not dilated. A 24 hour Holter monitor showed normal sinus rhythm throughout with occasional supraventricular ectopics.

We analysed the phonocardiogram using commercially available audio editing software. His heart rate was clearly discernible as a regular 76 beats per minute. On examination of his phonocardiogram the first heart sound and his murmur are clearly visible, despite small amounts of interference. His diastolic interval was constant, ruling out tachy- or bradyarrhythmias.

Mobile telephones have become ubiquitous in developed societies over the last 10 years. Increasingly, the devices have a functionality which has been attempted to be put to use for medical uses outside of their communication role—for example, ambulatory ECG monitoring. Our patient used his mobile phone to perform and record part of the routine cardiac examination while he was experiencing symptoms, which was later used as an invaluable aid to his diagnosis.

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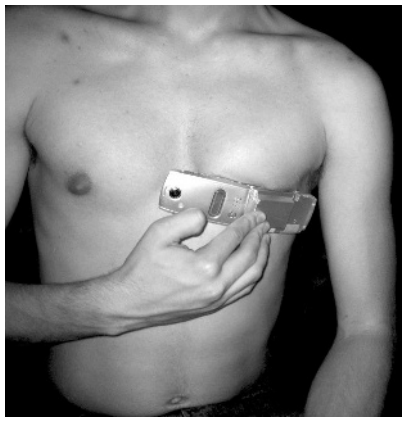
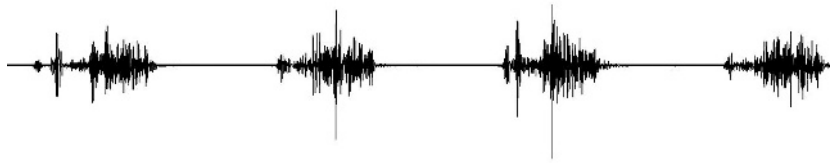


Patient demonstrating method used in self-recording heart sounds during palpitations.



The audio waveform derived from the mobile phone recording during palpitations.