Clinical research

Left ventricular structure and diastolic function with human ageing
Relation to habitual exercise and arterial stiffness

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Aim We sought to determine if attenuation of the age-associated increase in arterial stiffness by habitual aerobic-endurance exercise would have corresponding effects on left ventricular (LV) structure and diastolic function.

Methods and results We performed a cross-sectional study on 138 young, middle-aged, and older men who were either sedentary, recreationally active, or endurance exercise-trained. Ageing was associated with increased large artery stiffness (aortic pulse wave velocity) and habitual aerobic-endurance exercise was associated with decreased large artery stiffness (lower aortic pulse wave velocity; all $P<0.05$). Ageing was associated with increased mean LV wall thickness, chamber diameter, mass, concentric remodelling, and a decline in LV diastolic function (all $P<0.05$). Habitual aerobic-endurance exercise was independently associated with increased LV wall thickness, chamber diameter, and mass (echocardiography; $P=0.05$ or better). The largest LV mass was seen in older endurance trained men, suggesting an additive effect of exercise training and ageing on the LV. Indices of LV diastolic function declined with age, irrespective of habitual physical activity status. Aortic pulse wave velocity was an independent predictor of concentric LV remodelling in the pooled sample, but did not predict other properties of LV structure and diastolic function. In general, habitual aerobic-endurance exercise status was not uniformly associated with favourable modulation of age-associated changes in LV structure and diastolic function.

Conclusion We conclude that in contrast to its ability to favourably modulate the stiffness of large elastic arteries, regular aerobic-endurance exercise does not consistently modulate the changes in LV structure and diastolic function that occur with physiological ageing in men.

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KEYWORDS
Arterial stiffness; Left ventricle; Endurance training; Ultrasound; Pulse wave velocity

With physiological (healthy) ageing, the left ventricle undergoes tonic structural and functional change that includes an increase in wall thickness and chamber diameter, concentric remodelling, increased mass, and reduced diastolic function.1,2 Lakatta has recently...
emphasized the importance of understanding the age-associated changes to the cardiovascular system, as they provide an important precursor to cardiovascular disease.\textsuperscript{3–5} However, the factors that may favourably or unfavourably modulate age-associated changes in left ventricular (LV) structure and diastolic function remain uncertain. A change in arterial function, particularly an increase in large artery stiffness in the central (cardiothoracic) circulation, is thought to serve as a key physiological stimulus for age-associated changes in the LV.\textsuperscript{1,2} Based on this hypothesis, attenuation of the age-associated increase in arterial stiffness might have favourable effects on some of the age-associated changes in LV structure and diastolic function.

We have recently demonstrated that the increase in central large artery stiffness with age is attenuated in healthy men who habitually perform aerobic-endurance exercise, compared to their sedentary peers.\textsuperscript{6} Given the effect of regular exercise on arterial stiffness, and the proposed mechanistic link between age-associated changes in arterial stiffness and LV structure/diastolic function,\textsuperscript{1,2} we reasoned that habitual aerobic exercise status might modulate age-associated changes in the left ventricle in healthy men. In addition, we hypothesized that there would be an association between arterial stiffness and LV structure and diastolic function.

Although information exists on LV structure and diastolic function with ageing or with endurance exercise training,\textsuperscript{7–12} no data are available on the potential interactive effects of ageing and habitual exercise status on LV structure and diastolic function. This requires the study of subjects over a broad, continuous age-range with differing habitual physical activity status. In addition, although the concept is widely accepted, no data exist on the relation between large artery stiffness and LV structure/diastolic function in this population. Consequently, it is not known if habitual exercise modulates age-associated changes in LV structure/diastolic function, or if there is a relation of any such effects to corresponding changes in arterial stiffness.

To provide insight into the possible modulation of LV structure and diastolic function by habitual aerobic exercise status with advancing age, we studied groups of young, middle-aged, and older men who were either sedentary, moderately active, or endurance exercise-trained. To examine the possible association between LV structure/diastolic function and central large artery stiffness, we determined the relation between these LV characteristics and aortic pulse wave velocity (PWV).

Methods

Subjects

We recruited and screened 160 men. Of these, 138 men were considered to be healthy and had satisfactory ultrasound images. The men were categorized as young (18–37 years), middle-aged (38–57), or older (58–77). Subjects were sedentary (no regular aerobic exercise), recreationally active (light to moderate aerobic exercise, 2–4 times/week, not competitively active), or endurance trained (vigorous aerobic-endurance exercise ≥5 times/week and competitively active) for at least 2 years prior to enrolment in the study. The endurance-trained subjects had been training for 9±1 (young), 19±3 (middle-aged), and 24±4 (older) years. All subjects were normotensive (blood pressure <140/90 mmHg), non-obese (body mass index <30 kg/m\textsuperscript{2}), were non-smokers, and were not taking any medications. In addition, subjects of >40 years passed a physical examination and an electrocardiography monitored maximal treadmill exercise test. Subject characteristics are reported in Table 1.

Left ventricular structure and mass

Ultrasound echocardiography (Toshiba SSH-140 interfaced with a 3.5 MHz transducer) images of the LV were obtained from the parasternal and apical windows with the subject in the left lateral decubitus position. Examinations were recorded on super-VHS videotape and measurements were made using digitization software integral to the ultrasound system (mean of five consecutive cardiac cycles). Measurement of systolic and diastolic chamber dimensions and wall thickness were measured from M-mode images following established guidelines.\textsuperscript{13} The ratio of LV posterior wall thickness to chamber radius (h/r ratio), an index of LV geometry, was calculated from M-mode diastolic dimensions. Left ventricular mass was calculated using a validated formula.\textsuperscript{14}

Diastolic function

Transmitral blood flow from the left atria to the LV was measured using pulsed-Doppler to determine early (E) and late (A) diastolic peak filling velocity and the ratio of E and A velocity (E/A ratio). The sample volume was carefully positioned in the LV inflow tract at the tips of the mitral valve when maximally opened. Mitral deceleration time was measured as the time from the peak to the end of the Doppler E-wave. Isovolumic relaxation time (IVRT) was measured as the time between the closing artifact of the aortic valve and the earliest detection of trans-mitral blood flow. Pulsed Doppler waveforms from the pulmonary vein were analysed for flow velocity during LV systole, diastole, and flow reversal during left atrial contraction. Subjects were screened against abnormal diastolic blood flow patterns, such as a pseudonormal LV filling pattern, using the following criteria: pulmonary vein systolic flow velocity lower than pulmonary vein diastolic flow velocity, pulmonary vein peak velocity during flow reversal greater than 0.35 m/s, a slow filling pattern revealed by performing the Valsalva manoeuvre. This procedure was standardized for all subjects and the sonographer was blinded to the subjects’ exercise status and age.

Arterial function

Because we were interested in the relation between the left ventricle and the vascular system, we chose to use an index of arterial stiffness that was specific to the stiffness of the aorta. According to recent consensus,\textsuperscript{15} aortic PWV, a measure of the speed at which the pressure wave travels through the aorta, is the best available technique to assess aortic stiffness non-invasively. Aortic PWV was determined by measuring the time delay and distance between a Doppler flow velocity signal in the arch of the aorta and in the common femoral artery. The time delay was measured by determining the time between the ‘foot’ (that is, the start of the sharp systolic upstroke) of the aortic and femoral Doppler flow signals. Two cutaneous Doppler flow meters were used to register the Doppler flow wave at each site on a physiological recorder.\textsuperscript{16,17} Resting brachial artery blood pressure was measured with subjects in the supine position with
a semi-automated device (Dinamap XL, Johnson and Johnson) in accordance with American Society of Hypertension guidelines.18

Scaling data to body composition

Left ventricular structure is subject to the influence of body mass and composition and appropriate scaling methods need to be employed. Recently, the use of fat free mass (FFM) has been shown to be more appropriate than the use of height, body mass, or body surface area.19–23 Body mass was measured to the nearest 0.1 kg using a beam-balance scale and fat mass and fat free mass were determined by dual energy x-ray absorptiometry (Lunar Radiation Corp, model DPX-IQ). For the purpose of comparison to other studies using more conventional scaling procedures, LV mass and mean wall thickness were also scaled to body surface area;24 data are reported in Table 2.

Treadmill exercise

To obtain measurements of aerobic fitness, subjects performed incremental treadmill exercise with a modified Balke protocol, as described previously.25 Maximal oxygen consumption was measured with on-line, computer assisted, open circuit spirometry. Heart rate and ratings of perceived exertion (Borg scale)26 were measured throughout the protocol.

Statistical analysis

Two-way analysis of variance (ANOVA) was used to determine the main effects for age group and exercise status group and the interaction between age-group and exercise status. To confirm the interaction between age and exercise status group with age as a continuous variable, a multiple regression model was used. The model included age, two dummy variables for exercise training status (sedentary versus active; sedentary versus endurance trained), and their interactions. Analysis of covariance (ANCOVA) was used to test for differences between exercise status groups and age-groups for PWV, with mean arterial blood pressure as a covariate. To examine the relation between aortic PWV and LV structure/diastolic function, scatterplots were created to visually check the linearity of the bivariate relations, and one-tailed Pearson correlation analysis was used. Where significant relations were found, linear regression was used to evaluate the ability of aortic PWV and age to predict LV structure and diastolic function. Where both aortic PWV and age were significant predictors, part (semi-partial) correlation coefficients were used to determine the independent (unique) contribution of aortic PWV and age in the variance of LV structure/diastolic function. All data are reported as mean±standard error.

Results

Subject characteristics

Age was associated with shorter stature, greater body fat, reduced fat free mass, higher diastolic blood pressure, and lower maximal aerobic capacity (Table 1). At any age, habitual exercise status was associated with lower body mass, lower body fat, lower body surface area, lower resting heart rate, and higher maximal

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Selected subject characteristics</th>
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<tbody>
<tr>
<td></td>
<td>Sedentary</td>
</tr>
<tr>
<td>Young</td>
<td>Middle-age</td>
</tr>
<tr>
<td>n</td>
<td>16</td>
</tr>
<tr>
<td>Age, years</td>
<td>29±2</td>
</tr>
<tr>
<td>Height, cm</td>
<td>181±2</td>
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<tr>
<td>Body mass, kg</td>
<td>89±5</td>
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<tr>
<td>Body fat, %</td>
<td>23±3</td>
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<tr>
<td>Fat free mass, kg</td>
<td>68±3</td>
</tr>
<tr>
<td>Body surface area, m²</td>
<td>2.09±0.04</td>
</tr>
<tr>
<td>Systolic BP, mmHg</td>
<td>119±5</td>
</tr>
<tr>
<td>Diastolic BP, mmHg</td>
<td>72±1</td>
</tr>
<tr>
<td>Resting heart rate, b min⁻¹</td>
<td>58±2</td>
</tr>
<tr>
<td>VO₂max, ml kg⁻¹ min⁻¹</td>
<td>41±2</td>
</tr>
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</table>

*p<0.05 main effect of age. **p<0.05 main effect of habitual exercise status from ANOVA.
aerobic capacity, with the greatest effect being seen in the endurance trained men (Table 1).

LV structure
ANOVA showed that LV wall thickness \((P<0.0001; \text{Fig. 1A})\) and diastolic chamber dimension \((P=0.02; \text{Fig. 1B})\) increased with age. Habitual exercise status was independently associated with greater wall thickness \((P=0.05)\) and chamber dimension \((P=0.001)\). However, there were no significant interactions between age and habitual exercise status. The same statistical outcomes were found when LV wall thickness was normalized for the more conventional scalar variable, body surface area (Table 2). The age-associated increase in LV wall thickness was greater than the increase in chamber radius resulting in a progressive increase in \(h/r\) ratio with advancing age \((P<0.0001)\). There were no significant differences in \(h/r\) ratio among men of different habitual exercise status \((P=0.30; \text{Fig. 1C})\) and no significant age-exercise status interaction for the \(h/r\) ratio. The outcome of each ANOVA was confirmed using regression analysis of each LV structure variable with age treated as a continuous variable.

LV mass
ANOVA showed that there was a moderate increase in LV mass normalized for FFM with age \((P=0.02)\) and with increased habitual exercise \((P<0.0001; \text{Fig. 1D})\), tending to be largest in the endurance-trained men. However, there was no significant interaction between age and habitual exercise status \((P=0.30)\). When corrected to body surface area, LV mass was greater with habitual exercise status but not with age (Table 2). The outcome of the ANOVA was confirmed with the regression model with age entered as a continuous variable. The regression model showed that age \((P=0.001)\) and the dummy variable for sedentary vs. endurance trained physical activity status predicted LV mass normalized for FFM \((P=0.0001)\), but sedentary vs. moderately active physical activity status did not \((P=0.62)\). The part correlations from this analysis were 0.25, 0.37 and 0.04, respectively.

Diastolic function
ANOVA showed that E/A ratio declined with advancing age \((P<0.0001)\). There was a significant effect of habitual exercise \((P=0.003; \text{Fig. 1E})\), with E/A ratio tending to be highest in the endurance-trained men at all ages. However, there was no significant interaction between age and habitual exercise status \((P=0.57)\). Isovolumic relaxation time was progressively longer with advancing age \((P<0.0001; \text{Table 3})\). Although IVRT was longer with habitual physical activity \((P=0.004)\), it is possible that this was a function of lower heart rate in more active men. When IVRT was expressed as a percentage of the R-R interval from the ECG (Table 3), it rose with age \((P=0.007)\) and there was a significant effect of habitual physical activity status \((P=0.002)\), tending to be lowest in endurance-trained men. There was no significant interaction between age and habitual physical activity with respect to IVRT \((P=0.52)\) or IVRT as a percentage of R-R \((0.40)\). Similarly, mitral deceleration time increased with age \((P=0.0001)\). Although there was no significant effect of habitual physical activity status \((P=0.67)\), mitral deceleration time tended to be lower in endurance trained middle aged and older men. Pulmonary venous flow velocity was greater with advancing age during LV systole and left atrial contraction (flow reversal), and was reduced during LV diastole (all \(P<0.0001; \text{Table 3})\). There was no effect of habitual exercise status \((P=0.20–0.92)\) and no interaction between age and habitual exercise status \((0.25–0.90)\). The outcome of the ANOVA for each diastolic function variable was consistent with the regression analysis where age was treated as a continuous variable.

Arterial function
ANOVA showed that aortic PWV increased with age \((P<0.0001)\) and was also influenced by habitual exercise status \((P<0.0001; \text{Fig. 2})\). The difference in aortic PWV between the young and older adults was smaller in the endurance-trained \((14\%)\) compared with the recreationally active and sedentary men \((24\%\) and \(27\%)\), respectively. These age and exercise status differences in aortic PWV were not affected after normalizing aortic PWV for mean arterial pressure (ANCOVA).

Relations between LV structure and function and arterial stiffness
Aortic PWV correlated with LV wall thickness \((r=0.28; P<0.0001)\), \(h/r\) ratio \((r=0.41; P<0.0001 \text{Fig. 3})\), and E/A

### Table 2 Conventional ultrasound measurements of left ventricular structure

<table>
<thead>
<tr>
<th></th>
<th>Sedentary</th>
<th>Recreationally-active</th>
<th>Endurance-trained</th>
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<tbody>
<tr>
<td></td>
<td>Young</td>
<td>Middle-age</td>
<td>Older</td>
</tr>
<tr>
<td>(n)</td>
<td>16</td>
<td>24</td>
<td>11</td>
</tr>
<tr>
<td>LV mass/BSA(^{a})</td>
<td>79±3</td>
<td>85±2</td>
<td>85±5</td>
</tr>
<tr>
<td>Mean LV wall thickness/ (\text{BSA}^{a,b})</td>
<td>0.85±0.02</td>
<td>0.92±0.02</td>
<td>0.95±0.02</td>
</tr>
</tbody>
</table>

Mean±standard error. LV=left ventricle; BSA=body surface area.
\(^{a}\)\(P<0.05\) main effect of age.
\(^{b}\)\(P<0.05\) main effect of habitual exercise status from ANOVA.
ratio \( (r=-0.36; \ P<0.0001) \). A linear regression model that included both age and aortic PWV revealed that age \( (P<0.0001) \) but not aortic PWV \( (P=0.84) \) predicted LV wall thickness; both age \( (P=0.002) \) and aortic PWV \( (P=0.008) \) predicted h/r ratio; and age \( (P<0.0001) \) but not aortic PWV \( (P=0.16) \) predicted E/A ratio. Part correlation showed that the relation between h/r ratio and age was 0.25 and the relation between h/r ratio and aortic PWV was 0.21. Aortic PWV did not correlate with LV mass normalized for FFM \( (r=0.12; \ P=0.16) \) or with LV chamber diameter \( (r=0.10; \ P=0.11) \).

**Discussion**

The key new findings of this study include the following. First, in general, the age-associated changes in left ventricular structure and diastolic function that occur in healthy sedentary men are also observed in men performing habitual aerobic exercise. This is observed in both middle- and older age ranges, and in both recreationally active and vigorously endurance exercise-trained men. Indeed, a novel finding of this study is that there appears to be an additive influence of ageing and endurance training on LV wall thickness and mass. Second, large artery stiffness is an independent predictor of a concentric LV remodelling in healthy men varying in age and habitual aerobic exercise behaviour after accounting for the influence of age.

Both age and habitual aerobic exercise are associated with increased LV mean wall thickness, chamber diameter, and mass. Our results show that the highest mean values for these LV characteristics were observed in the older endurance trained men, suggesting an additive effect of ageing and exercise behaviour. Consistent with this idea, the part correlation between LV mass and the endurance trained dummy variable was stronger than the part correlation between LV mass and age and LV mass and the recreationally active dummy variable. Although habitual aerobic exercise generally does not attenuate age-associated changes in the left ventricle, some selective effects are apparent. Among middle-aged and older men, mean h/r ratio was lower, compared with...
sedentary men, suggesting that habitual endurance exercise may have a beneficial effect on ventricular aging by eliciting a more appropriate LV remodeling. The E/A ratio was higher in the endurance-trained men compared with the sedentary men. The E/A ratio is affected by heart rate and a lower resting heart rate in endurance-trained men may account for at least some of this difference. Ageing prolonged IVRT in all habitual physical activity groups. Although crude, when IVRT was expressed relative to the ECG R-R interval in an attempt to remove the influence of heart rate, diastolic function appeared to be mildly elevated in more active men. The mitral deceleration time and pulmonary venous data support the finding of reduced diastolic function with age in all habitual physical activity groups, but did not show an affect of habitual physical activity. Taken together, the diastolic function data consistently indicate that diastolic function declines with age, irrespective of participation in regular physical activity. Regular endurance exercise training may mildly preserve diastolic function with aging, but this may be explained largely by a lower heart rate in trained men, rather than by better LV compliance.

Given that regular aerobic-endurance exercise modulates the influence of human ageing on arterial

Table 3 Indices of left ventricular diastolic function

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Middle-age</th>
<th>Older</th>
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<tbody>
<tr>
<td>IVRT, msec,a,b</td>
<td>94±4</td>
<td>108±3</td>
<td>116±5</td>
</tr>
<tr>
<td>IVRT as a percentage of R-R interval, %a,b</td>
<td>9.8±0.6</td>
<td>11.0±0.4</td>
<td>11.8±1.0</td>
</tr>
<tr>
<td>Mitral deceleration time, msec,c</td>
<td>195±10</td>
<td>213±15</td>
<td>236±11</td>
</tr>
<tr>
<td>Pulmonary venous flow velocity systole, cm.s⁻¹a</td>
<td>0.39±0.02</td>
<td>0.46±0.02</td>
<td>0.47±0.03</td>
</tr>
<tr>
<td>Pulmonary venous flow velocity diastole, cm.s⁻¹a</td>
<td>0.48±0.02</td>
<td>0.39±0.02</td>
<td>0.39±0.02</td>
</tr>
<tr>
<td>Pulmonary venous flow reversal velocity, cm.s⁻¹a</td>
<td>0.15±0.007</td>
<td>0.18±0.007</td>
<td>0.19±0.008</td>
</tr>
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Mean±standard error. IVRT= isovolumic relaxation time. R-R=interval between R-waves of electrocardiogram.

Table 3 (continued)

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Middle-age</th>
<th>Older</th>
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<tbody>
<tr>
<td>a P&lt;0.05 main effect of age.</td>
<td></td>
<td></td>
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<tr>
<td>b P&lt;0.05 main effect of habitual exercise status from ANOVA.</td>
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Fig. 2 (A) Aortic pulse wave velocity increased with age (P<0.0001) and was also related to habitual exercise status (P<0.0001).

Fig. 3 Scatter plot of the relation of aortic pulse wave velocity and h/r ratio. The regression line shows the relation adjusted for age.
vascular function,\textsuperscript{6,17,27} and the presumed cause and effect relation between changes in central arterial stiffness and the left ventricle, we postulated that regular aerobic-endurance exercise would modulate age-associated changes in LV structure, mass and/or diastolic function. Instead we found that many of the age-associated changes in LV structure were not attenuated in endurance exercise-trained men, despite some attenuation of the age-associated increase in aortic stiffness. That there are two likely explanations for the finding that endurance training did not attenuate the age-associated changes to the left ventricle. First, ventricular ageing is affected by many different stimuli, and the modulation of just one of these stimuli (aortic stiffness) may not be sufficient to alter LV ageing. Second, the extent to which aortic stiffness is attenuated by habitual aerobic exercise may not be sufficient to prevent ventricular ageing.

We also expected that in our pooled cross-sectional study sample of 138 healthy men, aortic stiffness would be strongly related to LV structure and diastolic function. Aortic stiffness showed moderate relations with wall thickness, concentric remodelling, and diastolic function. However, although aortic stiffness independently predicted a concentric LV remodelling, it did not predict wall thickness or diastolic function independently of age. This supports the concept that arterial stiffening is only one factor that contributes to vascular ageing. Indeed, our findings are in agreement with the conclusions of Rajkumar et al.\textsuperscript{28} that therapeutic modulation of aortic stiffness, by itself, may not improve LV diastolic function. Other putative mechanisms that may contribute to LV remodelling and reduced diastolic function with physiological ageing include increased peripheral resistance,\textsuperscript{29} extracellular matrix proliferation and collagen cross-linking,\textsuperscript{30} and/or altered excitation–calcium release-contraction-relaxation coupling in myocardial cells together with impaired sequestration of calcium by the sarcoplasmatic reticulum.\textsuperscript{1} In addition, the progressive increase in LV mass with age that appears to occur irrespective of physical activity status may lead to LV stiffening which may, in turn, explain the deterioration of LV diastolic function.\textsuperscript{31}

Our findings may have important biomedical implications for the ageing human. Increased LV wall thickness, LV mass, LV remodelling, and reduced diastolic function with physiological ageing have been associated with increased risk of cardiovascular morbidity and mortality.\textsuperscript{32–35} In addition, diastolic heart failure is increasingly prevalent amongst older men, although little is known about its natural history and management.\textsuperscript{36} Lakatta has suggested that aerobic exercise is potentially important in the management of the age-associated changes in LV mass and function.\textsuperscript{1,2} Our data indicate that habitual endurance exercise, whilst positively influencing vascular function, did not uniformly affect the age-associated increase in LV mass and h/r ratio. Previous studies have shown that E/A ratio was higher in a small cohort of master athletes compared to sedentary older men and women.\textsuperscript{37} Our data suggest that diastolic function declines with age irrespective of habitual exercise status and better E/A ratio in athletes may be due partly to differences in heart rate.

A limitation of this study was our ability to comprehensively characterize the load on the left ventricle imposed by the arterial system. Although we employed a well-established measure of central large artery stiffness, other factors may have contributed to the workload of the LV. For example, non-invasive measurement of aortic input impedance, a more comprehensive measurement of afterload, was not, at the time of data collection, possible in humans. The absence of a direct measure of aortic input impedance may have resulted in an underestimation of the proposed physiological links between central arterial function and LV structure and function. A second limitation is that our measurements of diastolic function are somewhat crude. However, the E/A ratio has previously been used to establish age- and exercise training-associated changes in diastolic function.\textsuperscript{10,11,32,37,38} Nevertheless, our results may have been different using more sensitive measures such as tissue Doppler, which was not available at the start of this multi-year study. The complexity of diastolic function means that E/A ratio and IVRT may have been affected by many factors including heart rate, pre-load, after-load, and the inotropic and lusitropic state of the myocardium and it was not possible to determine the influence of each of these variables. Our study population consisted of men who were normotensive. Ageing is typically associated with an increase in arterial systolic blood pressure and a widening pulse pressure. Although aortic PWV increased, pulse pressure did not widen in our older men, probably due to the selection of normotensive men. Indeed, our strict selection criteria may explain the lack of association of aortic PWV with LV structure and diastolic function when others have reported a strong association. However, most studies to date have been conducted on clinical populations, in particular hypertensive patients, where pulse wave velocity may have more profound effects on LV structure and diastolic function.\textsuperscript{39–43} Our results, therefore, should only be applied to healthy men with normal blood pressure and the ability of exercise to favourably affect LV structure and function may be much greater in clinical populations. Finally, we used the guidelines of the American Society of Echocardiography to calculate LV mass but this may have over-estimated LV mass and Penn convention measurements may have been more precise. However, this error is systematic and we do not think that it affected our conclusions.

In conclusion, the present results indicate that habitual aerobic-endurance exercise attenuated age-associated increases in arterial stiffness in healthy men, without uniformly affecting changes in LV structure, mass, and diastolic function. Indeed, only a concentric LV remodelling was predicted by aortic stiffness independently of age. Although some modest benefits of habitual aerobic exercise are conferred on tonic (resting) LV structure and diastolic function, the most important effect of habitual exercise training may be an enhanced LV functional response during peak aerobic exercise. As such, combating the negative effects of ageing on tonic...
LV structure and diastolic function may require a combination of therapies, of which aerobic exercise training should be considered to be one.

Acknowledgements

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