Absence of left ventricular and arterial adaptations to exercise in octogenarians


Absence of left ventricular and arterial adaptations to exercise in octogenarians. J Appl Physiol 97: 1654–1659, 2004; doi:10.1152/japplphysiol.01303.2003.—Recent evidence suggests that octogenarians exhibit attenuated adaptations to training with a small increase in peak $\dot{V}_{\text{O}}$$_2$ consumption ($\dot{V}_{\text{O}_2}$) that is mediated by a modest improvement in cardiac output without an increase in arteriovenous $\text{O}_2$ content difference. This study was designed to determine whether diminished increases in peak $\dot{V}_{\text{O}_2}$ and cardiac output in the octogenarians are associated with absence of left ventricular and arterial adaptations to exercise training. We studied 22 octogenarians (81.9 ± 3.7 yr, mean ± SD) randomly assigned a group that exercised at an intensity of 82.5 ± 5% of peak heart rate for 9 mo and 14 (age 83.1 ± 4.1) assigned to a control group. Peak $\dot{V}_{\text{O}_2}$ increased 12% in the exercise group but decreased slightly (−7%) in the controls. The exercise group demonstrated significant but small decreases in the heart rate (6%, $P = 0.002$) and the rate-pressure product (9%, $P = 0.004$) during submaximal exercise at an absolute work rate. Training induced no significant changes in the left ventricular size, geometry (wall thickness-to-radius ratio), mass, and function assessed with two-dimensional echocardiography or in arterial stiffness evaluated with applanation tonometry. Data suggest that the absence of cardiac and arterial adaptations may in part account for the limited gain in aerobic capacity in response to training in the octogenarians. crate and arterial adaptations to exercise

AGING IS ASSOCIATED WITH A progressive decrease in aerobic power [maximal $\dot{V}_{\text{O}_2}$ consumption ($\dot{V}_{\text{O}_2}$max)] (1, 24). The factors that contribute to the age-related decline in $\dot{V}_{\text{O}_2}$max include J) intrinsic structural and functional deteriorations of the cardiovascular system reflected in reduced maximal heart rate, left ventricular (LV) diastolic dysfunction (19, 20), diminished LV systolic reserve (19), increased arterial stiffness (19), and impaired NO-mediated vasodilation (9); 2) physical inactivity; and 3) chronic degenerative diseases commonly associated with old age. Recent studies have reported that 60- to 72-yr-old adults can undergo physiological adaptations to exercise training characterized by a higher $\dot{V}_{\text{O}_2}$max physiological volume-overload LV hypertrophy (30), improved LV systolic and diastolic function (27, 29, 30), enhanced endothelial function (5), and reduced arterial stiffness (33, 34). Similar adaptations have also been demonstrated in master athletes (26). These observations suggest that endurance exercise training can partially compensate for age-associated cardiovascular impairments in 60- to 72-yr-old subjects. On the basis of these findings, we hypothesized that octogenarians should undergo similar adaptations to training (2, 8). However, it became clear that advanced age associated with frailty may attenuate physiological adaptations to exercise training (8). We found that octogenarians with mild to moderate frailty have a limited capacity to increase their aerobic power (~2 ml·kg−1·min−1, 14%) in response to exercise training (8) and that cardiac output rather than arteriovenous $\text{O}_2$ content difference was the primary mechanism responsible for the gain in peak $\dot{V}_{\text{O}_2}$ in these octogenarians. Therefore, the limited rise in peak $\dot{V}_{\text{O}_2}$ in the octogenarians was consequence of a small increase in cardiac output during peak effort (8). We also found that the larger cardiac output was mediated mostly by a faster heart rate with only a modest and insignificant increase in stroke volume (8). The focus of this study was to explore the basis for the attenuated increase in cardiac output during peak exercise. Specifically, we sought to determine whether the increased increases in cardiac output and stroke volume are associated with the absence of cardiovascular adaptations that modulate stroke volume, i.e., LV size, geometry, and function as well as arterial stiffness. In this context, our hypothesis was that octogenarians may not exhibit left ventricular and arterial adaptations in response to exercise training.

METHODS

Subjects

The subjects for this study were selected from the volunteers already randomized into exercise and control groups that our laboratory has reported recently (2). The selection of the volunteers was based only on the availability of the data that were relevant to the focus of this study, i.e., echocardiographic images for assessment of LV size and function and applanation tonometry for evaluation of arterial stiffness. The subjects’ eligibility and the randomization process (3:2 ratio, 3 exercisers to 2 controls) have been described in detail elsewhere (2). In brief, the inclusion criteria were: 1) age ≥78 yr old; 2) mild to moderate degree of frailty, mostly due to functional deterioration, defined as peak $\dot{V}_{\text{O}_2}$ between 10 and 18 ml·kg−1·min−1 and physical performance test score <32 (22); 3) sedentary state; 4) absence of severe (New York Heart Association Class III) congestive heart failure; 5) absence of current tobacco use; and 6) absence of other clinical conditions severe enough to be unsafe or prevent the octogenarians from exercising, such as a) frequent angina with mild effort, b) significant pulmonary disease, c) marked peripheral arterial disease with claudication, d) cerebrovascular disease with residual motor deficit, e) significant musculoskeletal disorders, f) severe cognitive impairment, and g) marked frailty. Preenrollment screening...
included clinical evaluation; assessment of frailty with the use of the standard procedures, including physical performance test (2); and laboratory assessment: blood chemistry, urinalysis, chest X-ray, and ECG. We studied 22 volunteers (age 81.9 ± 3.7 yr old, 11 women and 11 men) from the octogenarians already randomized into the exercise group (2), and 14 (age 83.1 ± 4.1 yr old, 8 women and 6 men) who were assigned to the control group (2). Six volunteers in the exercise group and five in the control group were taking cardiac medications. Six were treated with calcium channel blockers (3 in each group) and five with converting enzyme inhibitors (2 exercisers and 3 controls). None of the volunteers was taking β-blockers or angiotensin receptor blockers. Peak exercise data (\(\dot{V}_O_2\max\) and cardiac output) from 13 subjects have been published recently (8).

**Exercise Test and Determination of Highest Attainable (Peak) \(\dot{V}_O_2\)**

Each participant had a maximal treadmill exercise test, with use of a protocol described recently (8). After a warm-up exercise when the subjects walked flat on a treadmill for 3–4 min at a speed ranging from 0.5–1.2 miles/h, they began to exercise at the same speed as used during warm-up with the grade increasing 1 or 2% every 1 or 2 min until the subjects could no longer continue to exercise because of severe fatigue or because of development of cardiac symptoms, abnormal blood pressure (BP) responses, or ECG changes (ST segment depression > 0.3 mV or arrhythmias) that were considered unsafe for them to continue. The speed and grade of the treadmill were adjusted to keep the duration of the exercise test between 5 and 10 min. \(\dot{V}_O_2\max\) was measured continuously with open-circuit spirometry with the use of a metabolic measurement cart (MAX 1, FITCO, Farmingdale, NY). \(\dot{V}_O_2\max\) was defined by using the criteria previously described (16), i.e., an attainment of plateau of \(\dot{V}_O_2\) despite increasing exercise intensity, 2) a respiratory exchange ratio (RER) ≥ 1.1, and 3) measured \(\dot{V}_O_2\) lower than predicted for the work rate. Because many subjects were not able to attain the \(\dot{V}_O_2\max\), the highest \(\dot{V}_O_2\) recorded was designated as peak \(\dot{V}_O_2\). A repeat peak \(\dot{V}_O_2\) test was performed in 13 subjects before and after training, ~2 wk after the first treadmill test for reevaluation of aerobic exercise capacity. Peak \(\dot{V}_O_2\) data were not available in two participants in the exercise group.

**Left Ventricular Size, Geometry, and Function**

Two-dimensional echocardiogram with a 2.5-MHz transducer (Hewlett-Packard Echocardiograph model 77020AC) was performed. Echocardiographic images were acquired and analyzed as recommended by the American Society of Echocardiography (25). LV end-diastolic and end-systolic dimensions and wall thickness were defined by conventional criteria (25). LV size and geometry were evaluated with the use of I) LV end-diastolic diameter (25), 2) LV mass, and 3) LV wall thickness-to-radius (h/r) ratio. The following were used as measures of LV systolic function: I) fractional shortening (systolic shortening) calculated with the use of the conventional equation (25) and 2) LV end-systolic wall stress, calculated with the formula described by Grossman et al. (10). End-systolic pressure was estimated as \((2 \times \text{SBP} + \text{DBP})/3\), where SBP and DBP are systolic and diastolic BP, respectively, as reported by Kelly et al. (15). Transmitral diastolic inflow velocities were recorded from the apical four-chamber view with the use of the pulsed Doppler. The following variables were measured: I) peak early diastolic flow velocity measured as the distance from the baseline to the darkest point of the first (early spectral waveform); 2) peak late diastolic flow velocity measured as the distance between the baseline and the darkest point of the second (late) spectral waveform; and 3) the ratio of the early-to-late peak left ventricular diastolic flow velocities (E/A), derived by dividing peak early flow velocity by peak late flow velocity. The E/A ratio was normalized for preload (EDD) and heart rate: E/A = (E/A)/EDD × (R-R)0.5, where E/A is normalized E/A ratio, EDD is the end-diastolic dimension, and R-R is cardiac cycle length (in s). The echocardiographic data were analyzed in a blinded fashion. The reproducibility of echocardiographic measurements has been recently reported (35).

**Assessment of Arterial Stiffness**

We used applanation tonometry of the common carotid artery with the use of the high-fidelity Milar (Milar Instruments, model TCB-500, Houston, TX) external pressure transducer to evaluate the effects of training on arterial stiffness. The arterial waveform and its harmonics obtained from this high-fidelity probe closely mimic those recorded from the central aorta (14). The subjects rested in the recumbent position for a minimum of 15 min before the applanation tonometry evaluation. The Milar probe was placed perpendicularly over the common carotid artery to display and record arterial pulse waveforms. Ten recordings of the carotid pulse wave and ECG (QRS) signals were made and stored for subsequent analysis, using the method described by Vaitkevicius et al. (36). A computer software program was used to determine the timing and the amplitude of the inflection point of the signal-averaged carotid pulse waveform using its derivatives. The augmentation index (AI) was calculated as (PiPP) × 100, where Pi is the amplitude of the inflection point and PP is amplitude of the pulse pressure with the use of the computer-generated algorithm (13, 36). BP was measured during applanation tonometry. Applanation tonometry was not performed on the subjects who had a carotid bruit.

**Exercise Training Program**

We found that, because of frailty, the participants were physically unable to engage in a vigorous exercise program needed to induce any tangible adaptations. Therefore, we designed an exercise training program that incorporated three phases, each consisting of 36 sessions of exercise to be completed in 3 mo: physical therapy exercise followed by strength training and then endurance exercise training. The first two phases also incorporated a progressive walking exercise program.

**Physical therapy exercise.** This phase was intended to improve flexibility and coordination and consisted of flexibility, stretching, and range of motion exercises as well as mild strength training. The physical therapy exercise was supplemented by a mild endurance exercise consisted of slow walking.

**Strength training.** This component of training was added to the physical therapy and walking program and designed to increase muscle strength sufficiently to make it possible for the subjects to perform a high-intensity endurance exercise. Strength training consisted of weight lifting involving lower and upper extremities as well as abdominal and back muscles, 3 days/wk. The intensity of strength training was initially equivalent to 65% of one-repetition maximal, increasing gradually under monitoring to 85–100% of the initial 1-repetition maximum. The number of repetitions increased from one set of 6 to three sets of 10–12 exercises over the training interval. During this phase, the volunteers continued the walking program at a more rapid pace, as tolerated.

**Endurance exercise training.** The endurance exercise training program included walking on a treadmill, cycling, and rowing. The intensity of endurance exercise was set at a level that elicited ~65–70% of maximal attainable \(\dot{V}_O_2\). Initially, the subjects exercised at this intensity for 10–15 min. The duration of exercise was then increased progressively under monitoring to 60 min or longer, if tolerated. After the subjects were comfortable performing these exercises, their training regimen was supplemented with interval training, consisting of several 3- to 5-min exercise bouts requiring 85–90% of the subjects’ high attainable \(\dot{V}_O_2\) interspersed with 2–3 min of rest or light exercises. The exercise intensity was adjusted to the desired level by using heart rate, measured with radiotelemetry, relative to the subjects’ peak \(\dot{V}_O_2\).
Control Group

To gain psychological benefits from exercise, the subjects randomized to a control group were given an exercise program that consisted of stretching, relaxation, and yoga type of exercises to perform in their residence 3 times/wk. The control subjects were transported to our facility on a monthly basis to perform their exercises under supervision and obtain information about their clinical status.

Statistics

Baseline values in the two groups (exercise and controls) were compared with the use of the t-tests. Paired t-test was used to compare initial and final data. After adjustment for the baseline values, the between-group differences in the final values were examined with the use of the group t-test. Wilcoxon rank tests were used if conditions for t-test analysis were not met (i.e., absence of normal distribution or equal variance). Least-square univariate regression analyses were performed to examine relationships between physiological variables. Data are expressed as means ± SD.

RESULTS

Exercise Training

All volunteers in the exercise group except one completed 108 sessions of exercise. The duration of the entire program was 13.0 ± 2.3 mo, during which the participants exercised 2.6 ± 0.5 days/wk. The intensity of exercise was 82.0 ± 8% of peak heart rate in the third phase of the training program. The duration of exercise sessions averaged 69.4 ± 3.7 min excluding warm-up, cool-down, and rest periods.

Adaptations at Rest

Resting heart rate, systolic BP, diastolic BP, body mass index, and weight did not change in both groups (Table 1). There were no significant differences in these variables between the two groups.

Left Ventricular Size, Geometry, and Function

There were no differences in LV end-diastolic diameter, end-systolic diameter, systolic shortening, LV end-systolic wall stress, E/A, or E/ESD between the two groups on initial evaluation (Table 2). The initial h/r ratio was larger in the control group. Training had no significant effect on LV size, geometry (the h/r ratio), mass, or function (systolic shortening, end-systolic wall stress, or the E/A ratio). There were also no changes in the controls. There were no between-group differences in the interval changes (i.e., from initial to final) in any of these variables. We found a significant inverse correlation between LV end-systolic wall stress and LV systolic shortening (r = −0.72, P = 0.003) of the pooled data (n = 44) including both groups (exercise and control) and conditions (initial and final). Furthermore, there was a significant inverse correlation between the interval changes (from initial to final) in LV end-systolic wall stress and fractional shortening in the exercise group (r = −0.63, n = 16, P = 0.008) but not in the controls (r = 0.32, P = 0.27). The absolute values for the E/A ratio or those normalized for preload and heart rate did not change with training (E/A0, initial 0.21 ± 0.09 vs. final 0.21 ± 0.08, P = 0.92).

Arterial Stiffness

AI at baseline was similar in the two groups and did not change during final evaluations in either group (Table 2). There were also no between-group differences in the changes in the AI. Systolic BP, measured during applanation tonometry, was 139.6 ± 18.2 mmHg before and 137.4 ± 14.1 mmHg after training (P = 0.755). We found a significant correlation between the changes in systolic BP and AI (ΔSBP vs. ΔAI, r = 0.724, P = 0.002), in the exercise group, showing that the subjects who had a large decrease in AI were likely to have a greater reduction in systolic BP in responses to training. In contrast, there was no significant correlation between the changes in systolic BP and AI in the control group (r = 0.32, P = 0.441). There were no changes in AI normalized for systolic BP in either group.

Adaptive Responses to Submaximal Exercise

During submaximal exercise at a similar absolute exercise intensity, the exercise group exhibited a small but significant decrease in heart rate (6%, 6 beats/min) and rate-pressure product (9%) in the trained state (Table 3). There were signif-
Table 3. Adaptations during submaximal exercise

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Initial</th>
<th>Final</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂, l/min</td>
<td>Exercise</td>
<td>0.82±0.23</td>
<td>0.80±0.22</td>
<td>0.24</td>
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<tr>
<td>HR</td>
<td>Exercise</td>
<td>106±15</td>
<td>100±15</td>
<td>0.002</td>
</tr>
<tr>
<td>SBP</td>
<td>Exercise</td>
<td>189±22</td>
<td>183±24</td>
<td>0.24</td>
</tr>
<tr>
<td>DBP</td>
<td>Exercise</td>
<td>87±12</td>
<td>86±11</td>
<td>0.42</td>
</tr>
<tr>
<td>RPP (×103)</td>
<td>Exercise</td>
<td>20.10±4.60</td>
<td>18.29±4.60</td>
<td>0.004</td>
</tr>
<tr>
<td>Controls</td>
<td>16.8±4.2</td>
<td>17.2±3.1</td>
<td>0.62</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD. RPP, rate-pressure product.

The rationale for the subgroup analysis was to gain further insight into the adaptive responses in those octogenarians who were able to increase their aerobic power in response to training, which may be more relevant to the focus of this study. We found that the responses in this subgroup were quite similar to those in the entire exercise group. There were no changes in LV size (end-diastolic diameter, before: 49.6±6.1 mm, after: 50.4±6.3 mm, P = 0.50), geometry (h/r, before: 0.41±0.06, after: 0.40±0.08, P = 0.65), LV mass (before: 175.0±48 g, after: 182.0±62 g, P = 0.58), LV systolic shortening (before: 35.6±9%, after: 38.1±6%, P = 0.21), LV diastolic filling (E/Ao, before: 0.22±0.18, after: 0.22±0.1, P = 0.95), or arterial stiffness (AI, before: 20.1±9.3%, after: 18.4±9.5% P = 0.65). There were no significant correlations between changes in arterial stiffness and LV mass (r = 0.03, P = 0.93), geometry (r = 0.36, P = 0.34), wall stress (r = 0.33, P = 0.39), or systolic shortening (r = 0.23, P = 0.56).

DISCUSSION

The data suggest that frail octogenarians have a limited capacity to adapt to exercise training characterized by a modest increase in aerobic power, a small reduction in heart rate during submaximal exercise, and the lack of tangible cardiac and arterial adaptations. The diminished adaptive responses to training are evidenced by a small increase in peak VO₂, an absence of LV physiological hypertrophy, improvement in LV function, a reduction in arterial stiffness, or resting bradycardia. Endurance exercise training of similar relative intensity has been reported to induce a larger increase in VO₂max (20–25%) in the 60- to 72-yr-old subjects (28, 31, 32). The diminished adaptations in the autonomic nervous system in response to training in these octogenarians are reflected in the absence of resting bradycardia, a roughly 60% smaller decrease in heart rate (6 vs. 15%), and a modest decrease in the rate-pressure product during submaximal exercise compared with that reported in the 60- to 72-year-old subjects (7). The small decrease in myocardial O₂ demand, as reflected in the rate-pressure product, suggests that, in the octogenarians who have coronary artery disease, the improvement in myocardial ischemia in response to training is likely to be attenuated.

The reasons for diminished adaptations to training are not clear. One possibility is an inadequate training stimulus because our subjects could not train more vigorously and frequently. However, the training intensity relative to peak heart rate in these octogenarians was high and similar to that used in older adults (28). Nevertheless, because of the very low peak VO₂ values, the exercise intensity in absolute terms was much lower than the training stimulus used in 60- to 72-yr-old adults (28). The low energy level and the residual fatigue made it difficult for the octogenarians to exercise more frequently. Another possible reason is the presence of concurrent cardio-

Table 4. Adaptations during peak exercise

<table>
<thead>
<tr>
<th>Variable</th>
<th>Group</th>
<th>Initial</th>
<th>Final</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂, l/min</td>
<td>Exercise</td>
<td>1.14±0.3</td>
<td>1.23±0.34</td>
<td>0.002</td>
</tr>
<tr>
<td>VO₂, ml·kg⁻¹·min⁻¹</td>
<td>Exercise</td>
<td>17.0±3.0</td>
<td>19.0±4.1</td>
<td>0.001</td>
</tr>
<tr>
<td>RER</td>
<td>Exercise</td>
<td>1.08±0.07</td>
<td>1.15±0.10</td>
<td>0.006</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>Exercise</td>
<td>137.7±6.0</td>
<td>142.6±20.7</td>
<td>0.15</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>Exercise</td>
<td>201.5±24.6</td>
<td>214.4±26.9</td>
<td>0.60</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>Exercise</td>
<td>87.9±13.7</td>
<td>86.6±15.7</td>
<td>0.57</td>
</tr>
</tbody>
</table>

Values are means ± SD. RER, respiratory exchange ratio; VO₂, peak oxygen consumption.
vascular disorders in our participants. The presence of either coronary artery disease or hypertension, however, does not generally prevent attainment of cardiovascular adaptations to exercise training exercise (4, 6, 23, 35). Another possibility is that advanced age per se could have resulted in a partial loss of the ability to adapt to training. In particular, a marked reduction in cardiovascular reserve due to advanced age may limit the adaptations to training.

Absence of physiological LV hypertrophy and an improvement in LV diastolic filling in our subjects suggest that exercise training did not induce significant cardiac adaptations in these octogenarians. Both cross-sectional studies in master athletes and longitudinal training studies in 60- to 72-yr-old men have documented increases in LV end-diastolic volume and diastolic filling as well as eccentric LV remodeling, characterized by a normal LV h/r ratio, in the trained state (26, 31, 32). However, the lack of improvement in LV systolic function at rest in response to exercise training is consistent with previous studies in younger subjects showing that training does not generally have a significant effect on resting LV systolic performance (29, 31). The significant inverse correlation between fractional shortening and end-systolic wall stress suggests that LV systolic function is dependent on LV systolic load.

The findings of this study suggest that exercise training is also unlikely to induce significant arterial adaptations in the frail elderly, as evidenced by the absence of significant decreases in the pulse pressure and the AI. The significant relationship between the changes in the AI and systolic BP in the exercise group suggests that systolic BP is in part influenced by arterial stiffness even though the overall effects of training on these variables may not be significant. The absence of a decrease in arterial stiffness in our subjects may reflect a limited capacity to adapt to training, an inadequate endurance training stimulus, or the conflicting effects of the two training modes used in this study. It appears that strength and endurance exercise training may have opposing effects on the arterial stiffness in older adults. Unlike endurance training, which reduces arterial stiffness (12, 34), strength training may actually increase it (1, 21). Therefore, it is possible that the increase in arterial stiffness induced by strength training could have concealed the expected decrease in stiffness by endurance training, resulting in no discernable change.

We have recently reported that the cardiovascular adaptations to exercise training are limited in octogenarians and are characterized by a modest increase in aerobic power mediated by a small increase in cardiac output with no change in the arteriovenous O2 content difference during peak effort (8). The findings of the present study may provide an explanation for the limited gain in peak VO2 in advanced age. Because adaptations in skeletal muscle probably play only a minor role in the increase in aerobic power in the octogenarians, the pivotal factor responsible for the increase in peak VO2 seems to be an increase in cardiac output, as our laboratory reported recently (8). Under these circumstances, the absence of physiological volume-overload LV hypertrophy, improvement in LV diastolic filling, and reduction of arterial stiffness together can limit the capacity of the left ventricle to generate a greater stroke volume, resulting in small increases in peak cardiac output and VO2 in response to training (8). Although it is possible that increased blood volume may have contributed to the increase in peak VO2 even in the absence of physiological eccentric LVH or enhanced diastolic function (3), the increase in blood volume population is generally small in this age group.

A lower RER in the initial exercise tests suggests that a portion of the increase in aerobic capacity after training was due to the inability of our subjects to attain true VO2 max before training. This provides further evidence in support of blunted adaptive responses to training because the increase in aerobic capacity attributable to physiological adaptations may even be smaller than what we observed in these frail octogenarians.

Limitations of this study are as follows: 1) The small number of subjects may be responsible in part for the absence of the statistical significance. Although this is a valid concern, the differences that we noted were small. 2) We cannot exclude the possibility that a greater training stimulus could have induced adaptations similar to those in 60- to 72-yr-old adults. However, the training regimen that was employed in these octogenarians was the maximum that they could have possibly tolerated. 3) We did not evaluate LV systolic or diastolic function during peak exercise or in response to a β-adrenergic agonist (30) that might have shown adaptations that are detectable only in response to stress. 4) It is possible that the E/A ratio used as a surrogate for LV diastolic filling is insensitive to detect changes in LV diastolic function attributable to training. More reliable and nuanced measures of LV diastolic function and chamber stiffness such as the model-based image processing of the transmitral Doppler E wave, i.e., parameterized diastolic filling formalism (17, 18), might have demonstrated adaptive changes in LV diastolic function.

In conclusion, the results of this study suggest that octogenarians with mild to moderate frailty can adapt to exercise training with a modest increase in aerobic capacity despite the lack of significant left ventricular and arterial adaptations. This implies that these adaptations may not be necessary for the octogenarians to improve their aerobic power in response to exercise training. However, our data suggest that absence of adaptive increases in LV size and function and an improvement in arterial stiffness can considerably limit the increase in aerobic power in response to training in the octogenarians.

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