Cardiac Cycle-Dependent Changes in Aortic Area and Distensibility Are Reduced in Older Patients With Isolated Diastolic Heart Failure and Correlate With Exercise Intolerance

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OBJECTIVES
The goal of this study was to determine if cardiac cycle-dependent changes in proximal thoracic aortic area and distensibility are associated with exercise intolerance in elderly patients with diastolic heart failure (DHF).

BACKGROUND
Aortic compliance declines substantially with age. We hypothesized that a reduction in cardiac cycle-dependent changes in thoracic aortic area and distensibility (above that which occurs with aging) could be associated with the exercise intolerance that is prominent in elderly diastolic heart failure patients.

METHODS
Thirty subjects (20 healthy individuals [10–30 years of age] and 10 > the age of 60 years with DHF) underwent a magnetic resonance imaging (MRI) study of the heart and proximal thoracic aorta followed within 48 h by maximal exercise ergometry with expired gas analysis.

RESULTS
The patients with DHF had higher resting brachial pulse and systolic blood pressure, left ventricular mass, aortic wall thickness and mean aortic flow velocity, and, compared with healthy older subjects, they had a significant reduction in MRI-assessed cardiac cycle-dependent change in aortic area and distensibility (p < 0.0001) that correlated with diminished peak exercise oxygen consumption (r = 0.79). After controlling for age and gender in a multivariate analysis, thoracic aortic distensibility was a significant predictor of peak exercise oxygen consumption (p < 0.04).

CONCLUSIONS
Older patients with isolated DHF have reduced cardiac cycle-dependent changes in proximal thoracic aortic area and distensibility (beyond that which occurs with normal aging), and this correlates with and may contribute to their severe exercise intolerance. (J Am Coll Cardiol 2001;38:796–802) © 2001 by the American College of Cardiology

In contrast to patients with heart failure (HF) due to left ventricular (LV) systolic dysfunction, data from several population-based studies indicate that many patients over the age of 65 with HF have normal LV systolic function (termed diastolic heart failure [DHF]) without significant ischemic or valvular heart disease (1–3). Although these patients suffer from chronic exercise intolerance, dyspnea on exertion and episodes of pulmonary edema requiring hospitalization (4,5), the factors that contribute to DHF in the elderly are not completely understood (6).

With aging, thoracic aortic distensibility becomes reduced (7,8). Several studies have identified a relationship between limitation of exercise capacity and increased vascular stiffness in healthy older individuals (8–10). We hypothesized that a severe exaggerated reduction in cardiac cycle-dependent change in proximal thoracic aortic area and distensibility (beyond that which occurs with aging) may contribute to the exercise intolerance in older patients with DHF. To test this hypothesis, we noninvasively assessed cardiac cycle-dependent changes in the proximal thoracic aortic area and distensibility and wall thickness, LV mass and ejection fraction and exercise capacity in healthy subjects as well as in older patients with DHF.

METHODS
Study population and design. The Institutional Review Board of the Wake Forest University School of Medicine approved the study protocol. All participants gave written informed consent. The study population consisted of 10 young healthy volunteers aged 20 years to 30 years with no medical illness and taking no medication; 10 healthy community-dwelling volunteers aged >60 years who took no medication, had no illness, had a normal physical examination, a systolic and diastolic blood pressure below 140 mm Hg and 90 mm Hg, respectively and a normal
exercise echocardiogram (HO), and 10 individuals aged >60 years with established New York Heart Association Class II or III heart failure and an LV ejection fraction ≥55% without evidence of ischemic or valvular heart disease or chronic pulmonary disease (DHF). The diagnosis of HF was based on clinical criteria that included: 1) a HF clinical score from National Health and Nutrition Examination Survey-I of ≥4 (6), and 2) those utilized by Rich, et al. (5) that included a history of acute pulmonary edema or the occurrence of at least two of the following that improved with diuretic therapy without another identifiable cause: dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, bilateral lower extremity edema or exertional fatigue. Patients were excluded from participation if they had chronic renal insufficiency (creatinine ≥2.5 mg/dl), anemia (Hgb ≤11 mg/dl) or a contraindication to magnetic resonance imaging (MRI) (implantable pacemakers or defibrillator, intracranial metal or claustrophobia). Each subject underwent an MRI study of the heart and the proximal thoracic aorta followed within 48 h by maximal cycle ergometry with expired gas analysis. Of those with DHF, seven, three, one and six subjects were chronically prescribed angiotensin-converting enzyme inhibitors, calcium antagonists, beta-adrenergic blocking agents and diuretics, respectively. All tests were performed between 0800 and 1100 with the patient in a postabsorptive baseline compensated state and all cardioactive medications withheld at least 12 h before testing.

**MRI technique.** Each patient was imaged with a 1.5 T Horizon (General Electric Medical Systems, Milwaukee, Wisconsin) whole body imaging system utilizing a phased array cardiac surface coil placed on the chest. Multislice and frame gradient echo images with a temporal resolution of 50 ms were acquired and used to calculate LV mass and ejection fraction according to Simpson’s rule formula (11). These slices were 8 mm thick with a 2 mm gap and were positioned perpendicular to the long axis of the ventricle-spanning base to apex. They had a 256 × 192 matrix, a 38-cm field of view (FOV), a 35° flip angle, a 14 ms-repetition time (TR) and a 6.7 ms echo time (TE).

Cardiac cycle-dependent changes in the aortic lumen were assessed according to previously published techniques (12) with interleaved, velocity-encoded, phase-contrast, gradient echo images acquired perpendicular to the course of the proximal ascending thoracic aorta approximately 4 cm above the aortic valve. These scans had slices 1-cm thick with a 256 × 256 matrix, a 32-cm FOV, a 40° flip angle, an 11 ms TR, a 3.5 ms TE and a through-plane velocity encoding of 150 cm/s. After image acquisition, images were transferred to a processing workstation where the cross-sectional area of the vessel lumen was defined on the magnitude image of the reference scan by manually tracing a region of interest. Aortic distensibility was defined according to the previously published formula (12):

\[
\text{Aortic distensibility} = \frac{\text{area of aorta at end systole (mm}^2\text{)} - \text{area aorta at end diastole (mm}^2\text{)}}{\text{brachial pulse pressure (mm Hg) × area aorta at end diastole (mm}^2\text{)}}
\]

Brachial pulse pressure was measured noninvasively with a nonferromagnetic arm blood pressure cuff and recorded at the time of the phase-contrast acquisition.

To assess aortic wall thickness, double inversion recovery (13) fast spin echo images were acquired in the same slice position as that used to assess aortic distensibility. These scans had an echo train length of 32, a TR of 2 × the R-wave to R-wave interval, a 650 ms inversion time, a 42 ms TE, a 30-cm FOV and a 256 × 256 matrix (pixel sizes = 1.18 mm × 1.18 mm). Wall thickness was measured along the wall of the aorta adjacent to the border of the superior vena cava in a region absent of atherosclerotic plaque.

**Exercise testing protocol.** Exercise began at an initial workload of 12.5 W, was advanced to 25 W 2 min into exercise and then was increased by 25-W increments every 3 min thereafter (14). Data were collected during the fifth minute of the rest stage and during the last minute of each stage of exercise. Continuous expired gas analysis was performed with a Medical Graphics Cardio II System (Medigraphics, Minneapolis, Minnesota) that was calibrated before each study with gases of known volume and concentration as previously described (14). Peak oxygen consumption (VO2) was considered as the highest oxygen consumption achieved during exercise.

**Data analysis.** The tracings of the LV, aortic lumen and aortic wall were performed separately by individuals blinded to patient status. Measurements from the LV, proximal thoracic aorta and exercise testing were evaluated between the three patient groups using paired comparisons. In addition, comparisons were performed using analysis of covariance adjusting for age and gender. All p values were based on two-sided comparisons, and a value of <0.05 was considered significant. The relationship between cardiac cycle-dependent change in proximal thoracic aortic area and distensibility assessed noninvasively with MRI and peak VO2 was examined with a linear regression analysis. In all patients, associations between demographic data (age, gender, systolic and diastolic blood pressure, pulse pressure, and heart rate), MRI measures of LV ejection fraction and mass,

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**Abbreviations and Acronyms**

- DHF = diastolic heart failure
- FOV = field of view
- HF = heart failure
- HO = healthy old
- LV = left ventricular or left ventricle
- MRI = magnetic resonance imaging
- TE = echo time
- TR = repetition time
- VO2 = volume of oxygen

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Table 1. Magnetic Resonance Imaging and Exercise Data

<table>
<thead>
<tr>
<th></th>
<th>Healthy Young (n = 10)</th>
<th>Healthy Old (n = 10)</th>
<th>Diastolic Heart Failure (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>24 ± 1</td>
<td>71 ± 2*</td>
<td>77 ± 2†</td>
</tr>
<tr>
<td>Men/women</td>
<td>4:6</td>
<td>3:4</td>
<td>2:8†</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>120 ± 4</td>
<td>124 ± 4</td>
<td>145 ± 6†</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>72 ± 4</td>
<td>71 ± 4</td>
<td>76 ± 3</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>48 ± 4</td>
<td>52 ± 5</td>
<td>68 ± 7†</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>71 ± 3</td>
<td>61 ± 3*</td>
<td>71 ± 3†</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172 ± 4</td>
<td>168 ± 4</td>
<td>163 ± 3*</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72 ± 6</td>
<td>73 ± 5</td>
<td>76 ± 6</td>
</tr>
<tr>
<td>LV EF</td>
<td>0.63 ± 0.02</td>
<td>0.66 ± 0.03</td>
<td>0.69 ± 0.02</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>123 ± 11</td>
<td>99 ± 8*</td>
<td>124 ± 5†</td>
</tr>
<tr>
<td>Ao wall thickness (mm)</td>
<td>2.7 ± 0.16</td>
<td>2.9 ± 11</td>
<td>3.8 ± 0.20†</td>
</tr>
<tr>
<td>Ao area change (mm²)</td>
<td>92 ± 7</td>
<td>73 ± 12</td>
<td>23 ± 4†</td>
</tr>
<tr>
<td>Ao distensibility</td>
<td>4.2 ± 0.4</td>
<td>2.2 ± 0.4*</td>
<td>0.5 ± 0.1†</td>
</tr>
<tr>
<td>(10⁻³ mm Hg⁻¹)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ao Max Vel (cm/s)</td>
<td>36 ± 6</td>
<td>26 ± 2</td>
<td>40 ± 2†</td>
</tr>
<tr>
<td>VO₂Max (ml/min)</td>
<td>2,056 ± 171</td>
<td>1,480 ± 115*</td>
<td>1,006 ± 76†</td>
</tr>
<tr>
<td>Exercise time (min)</td>
<td>19 ± 2</td>
<td>14 ± 1*</td>
<td>6 ± 1†</td>
</tr>
<tr>
<td>RER</td>
<td>1.1 ± 0.03</td>
<td>1.2 ± 0.03</td>
<td>1.6 ± 0.49</td>
</tr>
<tr>
<td>Peak exercise SBP</td>
<td>172 ± 5</td>
<td>180 ± 11</td>
<td>182 ± 12</td>
</tr>
</tbody>
</table>

*p < 0.05 difference from young normal; †p < 0.05 difference from old normal.

Ao = aorta; BP = blood pressure; EF = ejection fraction; LV = left ventricular;
Max = maximum; RER = respiratory exchange ratio; Vel = velocity; VO₂ = volume of oxygen.

Aortic distensibility and wall thickness and peak oxygen consumption were assessed with Pearson correlations and stepwise scatterplots. Multiple regression was performed to determine significant predictors of peak VO₂ and to determine the independent effects of proximal thoracic aortic distensibility on peak VO₂. The reproducibility of our technique for measuring proximal thoracic aortic distensibility and wall thickness was determined in five patients >60 years of age studied twice at 48-h intervals at the same time of day without any change in their use of prescribed or nonprescribed medications.

RESULTS

Magnetic resonance imaging examinations and exercise tests were well tolerated in all individuals. Our study population included three African American subjects, and the remainder was Caucasian. As shown in Table 1, the ages and gender distribution of the elderly patients with DHF (Table 1) were similar to that reported in large population-based studies (1–3); the group with DHF had a higher percentage of women and was slightly older compared with their healthy counterparts over age 60. For this reason, the results of MRI and exercise testing in the older subjects were adjusted for age and gender (Table 2).

Image data from one subject from each group are displayed in Figure 1. Cardiac cycle-dependent change in aortic area and distensibility was reduced in older compared with younger healthy subjects and were markedly reduced in patients with DHF compared with healthy older subjects (Fig. 2). When adjusted for age and gender, aortic wall thickness, mean velocity in the thoracic aorta, resting pulse and systolic blood pressure were significantly different between HO patients and patients with DHF (Table 2).

Patients with DHF had severely reduced exercise time and peak VO₂ compared with healthy subjects (Table 1); this difference between HO patients and patients with DHF persisted after adjusting for age and gender (Table 2). As shown in Figure 3, peak VO₂ correlated significantly with aortic distensibility (r = 0.79) and aortic area change (r = 0.82); it did not correlate with peak exercise pulse pressure (r = 0.008, p > 0.9). As shown in Table 3, there were several univariate predictors of peak VO₂, but, after age and gender, only proximal thoracic aortic distensibility had a significant (p < 0.04) association with peak VO₂ when considered in a multivariate analysis. In multiple regression models after controlling for age and gender, proximal thoracic aortic distensibility assessed noninvasively with MRI was the single most important factor (r = 0.84, p < 0.001) accounting for the severely reduced oxygen consumption in elderly patients with DHF.

In the five older healthy individuals who underwent the reproducibility study, aortic wall thickness and distensibility varied by 0.09 mm ± 0.05 mm and 0.3 × 10⁻³ ± 1 × 10⁻⁴ mm Hg⁻¹, respectively on two separate days. Heart rate, systolic and diastolic blood pressure varied by 6 ± 8 beats/min, 6 ± 6 mm Hg and 6 ± 5 mm Hg, respectively.

DISCUSSION

Previous studies have shown that measures of vascular stiffness obtained using ultrasound assessments and pulse wave velocity increase with advancing age and are associated with reduced exercise capacity (8–10). Our data generated from young and older healthy individuals corroborate these findings (Table 1, Figs. 1 to 3). We hypothesized that the severe exercise intolerance experienced by older patients

Table 2. Magnetic Resonance Imaging and Exercise Data

<table>
<thead>
<tr>
<th></th>
<th>Healthy Old (n = 10)</th>
<th>Diastolic Heart Failure (n = 10)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>126 ± 5</td>
<td>144 ± 6</td>
<td>0.04</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>70 ± 4</td>
<td>75 ± 4</td>
<td>0.36</td>
</tr>
<tr>
<td>Pulse pressure (mm Hg)</td>
<td>55 ± 6</td>
<td>69 ± 7</td>
<td>0.17</td>
</tr>
<tr>
<td>LV EF</td>
<td>0.69 ± 0.02</td>
<td>0.66 ± 0.02</td>
<td>0.49</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>103 ± 7</td>
<td>123 ± 7</td>
<td>0.07</td>
</tr>
<tr>
<td>Ao wall thickness (mm)</td>
<td>3.0 ± 0.2</td>
<td>3.8 ± 0.2</td>
<td>0.01</td>
</tr>
<tr>
<td>Ao Δ area (mm²)</td>
<td>74 ± 7</td>
<td>34 ± 8</td>
<td>0.003</td>
</tr>
<tr>
<td>Ao distensibility</td>
<td>2.1 ± 0.3</td>
<td>0.7 ± 0.3</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Ao = aorta; BP = blood pressure; EF = ejection fraction; LV = left ventricular; Max = maximum; RER = respiratory exchange ratio; Vel = velocity; VO₂ = volume of oxygen; Δ = change.
with DHF may be related to an exaggerated reduction (i.e., beyond that which occurs with the aging process) in proximal thoracic aortic distensibility. We evaluated the proximal thoracic aorta because stiffness in this region can increase LV afterload and adversely affect LV diastolic relaxation (15). This study demonstrates that proximal thoracic aortic distensibility, measured noninvasively with MRI using an approximation of aortic pulse pressure from a nonferromagnetic cuff pulse pressure reading in the brachial artery, is reduced and is associated with the severe exercise intolerance experienced by older patients with DHF (Tables 2 and 3, Fig. 3). This reduction in distensibility is apparent as a diminution in the change of the aortic cross-sectional area that occurs during the cardiac cycle. Importantly, in a multivariate model incorporating assessment of LV volumes and mass, thoracic aortic distensibility assessed with MRI correlated significantly with peak exercise capacity (Table 3).

Figure 1. Short-axis magnetic resonance images of the left ventricle (LV) acquired at the midpapillary muscle level at end systole (top row) and the magnitude component of the phase-contrast gradient echo cardiography images of the ascending thoracic aorta (bottom row) from a participant in each of the three patient groups. The myocardium is gray, and the blood pool within the cavity is white in the images of the LV. Increased LV mass relative to cavity size in the patient with diastolic heart failure is displayed in the top right panel. The blackened silhouettes on the images of the aorta represent the difference in aortic area between end diastole and end systole. Cardiac cycle-dependent change in aortic area decreased with advancing age and, importantly, were most reduced in older participants with diastolic heart failure (bottom right).

Figure 2. Measurement of aortic distensibility for participants in each of the patient subgroups. Each symbol represents data from one patient.
Reduced aortic distensibility and exercise intolerance. Although our data identify a significant relationship between severely reduced proximal thoracic aortic distensibility measured with MRI and peak VO₂ in elderly patients with DHF, they do not prove a causal relationship or delineate the precise mechanism by which reduced distensibility limits exercise capacity. Several mechanisms exist by which reduced aortic distensibility could influence exercise capacity in DHF patients. First, the failure of the stiff rigid aorta to augment the efflux of blood to the periphery after ventricular systole could increase LV afterload, accelerate the return of reflected waves from the periphery back to the aortic root (16) and reduce exercise-associated cardiac output and muscle perfusion (4,17). For this reason, muscle fatigue may occur at lower levels of exercise due to an early onset of anaerobic metabolism and metabolic acidosis (18,19). Second, chronic exposure to increased LV afterload can induce LV wall thickening and hypertrophy by increasing myocyte size (20), the number of cardiac fibroblasts present (21) or myocardial collagen content (9). As the duration of diastole shortens during exercise, incomplete filling from a stiff hypertrophied LV can cause an elevation of left atrial pressure, which may produce dyspnea through an increase in either pulmonary interstitial fluid (22) or physiologic dead space (23). Third, the combination of a stiff aorta and LV may reduce exercise capacity to a greater extent than the impact from either alone (10). Increased afterload from reduced distensibility can reduce LV ejection by inhibiting LV relaxation (24); to compensate, the cardiovascular system operates at a higher total volume and left atrial pressure (i.e., utilization of preload reserve), and, thus, the margin to tolerate further elevations in left atrial pressure that occur with exercise is reduced (4,10).

Aortic wall thickness. Our data indicate that aortic wall thickness is increased in patients with DHF compared with young and older healthy individuals (Tables 1 and 2). Reduced aortic distensibility could occur through either a passive (wall composition) (9,25) or active (increase in smooth muscle tone) process (9,26) involving the aortic

![Figure 3. Relation between peak volume of oxygen (VO₂) (horizontal axis, A and B) and proximal thoracic aortic distensibility (vertical axis, A) and cardiac cycle-dependent changes in aortic area (vertical axis, B) for the 30 participants. Each symbol represents the data from one participant. The regression lines (solid lines) and equations are shown. Solid diamond = healthy young; solid square = healthy old; solid triangle = diastolic heart failure.]

<table>
<thead>
<tr>
<th>Variable</th>
<th>Univariate Predictor</th>
<th>Multivariate Predictor</th>
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<tbody>
<tr>
<td></td>
<td>Simple Correlation</td>
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</tr>
<tr>
<td>Gender</td>
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<td>0.001</td>
</tr>
<tr>
<td>Age</td>
<td>0.709</td>
<td>0.001</td>
</tr>
<tr>
<td>Aortic distensibility</td>
<td>0.793</td>
<td>0.001</td>
</tr>
<tr>
<td>Aortic wall thickness</td>
<td>0.410</td>
<td>0.024</td>
</tr>
<tr>
<td>Myocardial mass</td>
<td>0.299</td>
<td>0.108</td>
</tr>
<tr>
<td>LV mass/EDV</td>
<td>0.410</td>
<td>0.024</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>0.297</td>
<td>0.110</td>
</tr>
</tbody>
</table>

EDV = end diastolic volume; LV = left ventricular.
wall. Since we did not characterize the composition of the aortic wall in our study, we are unable to determine the mechanism that accounted for increased wall thickness in our patients. Further MRI studies incorporating fat saturation or $T_2$ weighting for characterizing aortic wall composition may be useful in this regard (13).

**MRI methodology.** To observe the aortic lumen for calculations of cardiac cycle-dependent changes in aortic area, we used PC-MRI assessments of the flow lumen within the vessel. This technique, implemented by other investigators (12), incorporates a relatively low velocity encoding (150 cm/s) in order to account for slower velocities at the vessel margin. In the future, gated black blood sequences may become available that could yield improved area determinations. To assess aortic wall thickness, the pixel sizes utilized were 1.18 mm $\times$ 1.18 mm. Although at a relatively low spatial resolution, our results indicated a significant increase in wall thickness for patients with DHF, higher spatial resolution may be achieved with future hardware modifications that incorporate a smaller FOV (<25 cm), a higher matrix size (512 $\times$ 512) or a transesophageal coil.

**Study limitations.** Our study has potential limitations. First, to estimate aortic pulse pressure for calculating aortic distensibility, we used a noninvasive brachial cuff pulse pressure measurement instead of a direct assessment of the aortic pulse pressure via catheter. While practical for serial measurements in the elderly (27,28), this methodology fails to consider amplification of pulse pressure from the aorta to the brachial artery that may occur in patients with DHF. For this reason, our calculation of aortic distensibility should be considered an approximation. Future studies that measure distensibility could incorporate noninvasive techniques that more accurately assess aortic pulse pressure (29). It is important to note that the differences we measured in distensibility are also apparent in our direct measurements of the change in aortic cross-sectional area that occurred during the cardiac cycle. As we demonstrate in Figure 3, the relationship between exercise capacity and cardiac cycle-dependent changes in aortic area and exercise capacity is the same as with our approximations of aortic distensibility. Second, most of our patients with DHF were taking medications that have been shown to improve aortic distensibility. Although these were withheld 12 h before testing, the differences we observed in LV hypertrophy and aortic distensibility may have been greater if our patients with DHF had not consumed these medications chronically. Third, although the proportion of women to men in our DHF group mirrors that of population-based studies (1–3), the differences we observed in LV hypertrophy and aortic distensibility may have been greater if our patients with DHF had not consumed these medications chronically. Third, although the proportion of women to men in our DHF group mirrors that of population-based studies (1–3), our healthy older subjects were normal volunteers, with no evidence of medical or cardiovascular disease by detailed history and physical and exercise echocardiography. Thus, this atypical finding may be due not only to the small numbers in each of the healthy groups, but also to a group of successful aging subjects. Indeed, the blood pressure ranges found in our healthy older subjects is similar to the reported from the healthy subgroup 1 in the Framingham Study (31). Because the intergroup differences and the relationship to exercise capacity were seen in the analysis of cycle-dependent aortic area change, our principle findings are not likely due primarily to blood pressure differences or lack thereof. Finally, although it is not possible to definitively exclude the influence of atherosclerosis on our results, by our case definition, none of the participants had evidence of coronary heart disease by history, physical exam and exercise electrocardiogram/echocardiogram. Our patients had “isolated” DHF with no evidence of ischemic or valvular heart disease or pulmonary disease (3,9,15). Data from the Cardiovascular Health Study indicate that this represents 42% of elderly patients with HF and normal LV ejection fraction (3). Our data may be less applicable to patients with multifactorial causes of DHF.

**Conclusions.** Our results indicate that proximal thoracic aortic distensibility assessed with MRI is substantially reduced in older patients with isolated DHF, and it is significantly related to their exercise intolerance. These data are relevant because aortic distensibility may be modifiable.

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**REFERENCES**