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# Assessment of Arterial Compliance by Carotid Midwall Strain-Stress Relation in Hypertension

Jonathan N. Bella, Mary J. Roman, Riccardo Pini, Joseph E. Schwartz,  
Thomas G. Pickering, Richard B. Devereux

**Abstract**—To elucidate the relations between arterial hypertrophy and compliance in hypertension, we studied 205 unmedicated hypertensive patients (129 men and 76 women) and 82 normotensive adults (56 men and 26 women) from an employed population by carotid ultrasound, noninvasive applanation tonometry, and echocardiography. Carotid midwall strain and circumferential stress were calculated at end diastole and peak systole. The relations of luminal and midwall strain to the increment in circumferential stress from end diastole to peak systole ( $\Delta$ carotid stress in normal subjects) were used to calculate ratios of observed/predicted carotid luminal and midwall strain. Mean stress-corrected luminal strain ( $82 \pm 26\%$ ) and midwall strain ( $78 \pm 23\%$ ) were lower (both  $P < 0.001$ ) in hypertensive patients than in normal adults. Stress-corrected luminal strain identified 14% of hypertensive patients with low arterial compliance, while stress-corrected midwall strain was low in 18% of patients. Patients with subnormal carotid midwall strain were older ( $61 \pm 12$  versus  $54 \pm 12$  years,  $P < 0.01$ ) and had larger carotid diameters ( $6.6 \pm 0.8$  versus  $5.7 \pm 0.8$  mm,  $P = 0.002$ ) and higher brachial pulse pressures ( $71 \pm 25$  versus  $63 \pm 17$  mm Hg,  $P < 0.05$ ) than other patients. Patients with arterial hypertrophy had lower stress-corrected midwall strain than those without hypertrophy ( $70 \pm 24\%$  versus  $79 \pm 23\%$ ,  $P = 0.05$ ), whereas no difference was observed in stress-corrected luminal strain ( $P = 0.40$ ). Stress-corrected midwall strain tended to be lower in patients with discrete atherosclerotic plaques than in those without ( $74 \pm 20\%$  versus  $79 \pm 24\%$ ,  $P = 0.15$ ). Compared with patients with normal left ventricular geometry, those with concentric hypertrophy had larger carotid diameters ( $6.6 \pm 0.7$  versus  $5.8 \pm 0.9$  mm,  $P < 0.05$ ) and lower stress-corrected luminal strain ( $62 \pm 11\%$  versus  $85 \pm 25\%$ ,  $P < 0.05$ ) and midwall strain ( $59 \pm 10\%$  versus  $81 \pm 22\%$ ,  $P < 0.05$ ). Therefore, stress-corrected midwall strain identifies patients with reduced arterial compliance, increased arterial wall thickness, and abnormal left ventricular geometry better than conventional measures based on arterial lumen diameters. (*Hypertension*. 1999;33:793-799.)

**Key Words:** carotid arteries ■ hypertension, chronic ■ hypertrophy, arterial ■ hypertrophy, left ventricular ■ compliance, arterial

The chronic increase in blood pressure during sustained hypertension increases arteriolar wall thickness.<sup>1,2</sup> In addition, decreased large-artery compliance is a major determinant of increased pulse pressure.<sup>3,4</sup> Decreased arterial compliance in hypertensive patients has been attributed both to elevated distending pressure and to hypertension-induced wall thickening that keeps arterial wall stress mainly unchanged despite elevated blood pressure.<sup>5,6</sup> However, despite numerous studies of the influence of risk factors on carotid artery intimal-medial thickness (IMT),<sup>7-9</sup> the relation between arterial wall hypertrophy and compliance in hypertension has not been clearly elucidated. Recently, we evaluated the relation of carotid midwall strain to the increment in carotid stress during systole ( $\Delta$ carotid stress) as a measure of arterial compliance in normotensive adults.<sup>10</sup> In this study we examined carotid midwall mechanics in normotensive and hypertensive individuals to identify the characteristics of

hypertensive patients with reduced arterial compliance by the carotid midwall strain-stress relation.

## Methods

### Subjects

The study group consisted of 205 asymptomatic unmedicated hypertensive patients (63% male; 34% nonwhite; age,  $55 \pm 12$  years; brachial arterial pressure,  $158 \pm 21/93 \pm 11$  mm Hg; body mass index,  $26.6 \pm 4.0$  kg/m<sup>2</sup>) and a reference group of 82 normotensive adults (68% male; 21% nonwhite; age,  $50 \pm 17$  years; brachial arterial pressure,  $122 \pm 12/73 \pm 9$  mm Hg; body mass index,  $25.1 \pm 4.0$  kg/m<sup>2</sup>) from an employed population in New York.<sup>11,12</sup> Hypertensive patients had systolic blood pressure  $>140$  mm Hg, diastolic blood pressure  $>90$  mm Hg, or both. Segmental left ventricular (LV) wall motion abnormalities and valvular regurgitation or stenosis were excluded by 2-dimensional and Doppler echocardiograms. On the basis of the Second National Health and Nutrition Examination Survey (NHANES II), subjects were classified as overweight if body mass index was  $>27.8$  kg/m<sup>2</sup> for men and  $>27.3$  kg/m<sup>2</sup> for women.<sup>13</sup>

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Subjects had no clinical evidence of coronary heart disease and gave informed consent under protocols approved in 1979 and regularly thereafter.

### Echocardiographic Methods

M-mode and 2-dimensional echocardiograms were performed by a skilled research technician and interpreted by a single investigator (M.J.R.), as previously described.<sup>14</sup> Penn convention measurements were used for LV mass,<sup>15,16</sup> and American Society of Echocardiography measurements<sup>17</sup> were used for LV internal diameter and wall thicknesses. When optimal orientation of the LV could not be obtained, correctly oriented 2-dimensional linear dimensions were made by the American Society of Echocardiography recommendations.<sup>18</sup> Brachial blood pressure was taken 3 times and averaged at the end of the echocardiogram. Gender-specific partition values for LV mass/body surface area used to classify LV geometric patterns were the same as those previously used for comparison with ambulatory blood pressures.<sup>19</sup> Patients were classified as abnormal if the LV relative wall thickness was  $>0.41^{20}$  or if the LV mass/body surface area was  $>108 \text{ g/m}^2$  in women or  $>118 \text{ g/m}^2$  in men.<sup>21</sup>

### Carotid Ultrasound

A Biosound Genesis II system (OTE Biomedica) or Acuson 128 (Acuson, Inc) equipped with a 7.0- to 7.5-MHz transducer was used to scan the common, internal, and external carotid arteries for discrete carotid plaques.<sup>8,11–14,22–24</sup> Two-dimensionally guided M-mode recordings of the distal common carotid artery  $\approx 1 \text{ cm}$  proximal to the carotid bulb with simultaneous ECG and carotid pressure waveforms were recorded on videotape and digitized with the use of a frame grabber and customized software. Electronic calipers were used to measure the internal diameter ( $D_d$ ) and far wall IMT ( $\text{IMT}_d$ ) at end diastole, recognized from the nadir of the simultaneous arterial pressure waveform or the minimal arterial diameter, as well as the diameter at peak systole ( $D_s$ ). All measurements were performed on several cycles by a single investigator (M.J.R.) and averaged.

As previously described,<sup>24</sup> a multiple regression equation predicting IMT from other potentially relevant variables was  $\text{IMT}_d = 0.00586(\text{Age}) + 0.015267(\text{Body Surface Area}) + 0.16569$ . The ratio observed/predicted  $\text{IMT}_d$  was calculated for each subject, and arterial hypertrophy was identified if this ratio was  $>2 \text{ SD}$  above the mean ratio of the reference population.

### Arterial Function Assessment

Arterial pressure waveforms were recorded noninvasively by placing a solid-state high-fidelity external pressure transducer (model SPT-301; Millar Instruments, Inc) over the right common carotid artery while recording M-mode images of the left common carotid artery.<sup>25</sup> Orientation and pressure applied to the transducer were adjusted to achieve apposition of the artery between the transducer and underlying tissue, as has been validated to yield accurate estimates of intra-arterial pulse pressure by comparison with simultaneous invasive pressure recordings.<sup>26,27</sup> The transducer is internally calibrated ( $1 \text{ mV} = 10 \text{ mm Hg}$ ) and registers absolute changes in applied pressure over a range of 300 mm Hg. Actual carotid pressures were obtained by external calibration; on the basis of the observation that mean arterial pressure is nearly identical in all capacitance vessels,<sup>28,29</sup> mean brachial artery pressure was considered to equal the planimetrically computer-derived mean blood pressure of the carotid waveform. Alternative analyses in which carotid diastolic pressure was also set equal to brachial diastolic pressure yielded similar results and are not reported separately.

### Arterial Compliance and Stiffness Indices

As previously described,<sup>10</sup> carotid luminal strain, the percent systolic expansion of the arterial lumen,<sup>30</sup> was calculated as  $[(D_s - D_d)/D_d] \times 100$ . Carotid pressures  $D_d$ ,  $D_s$ , and  $\text{IMT}_d$  were used to calculate several measures of regional arterial stiffness, including Peterson's elastic modulus, Young's elastic modulus,<sup>31</sup> and a pressure-independent measure ( $\beta$ ).<sup>32,33</sup> Systemic arterial compliance was

estimated by the ratio stroke volume/brachial pulse pressure.<sup>34</sup> In addition, the arterial compliance index was calculated using a method modified<sup>25</sup> from that described by Randall et al.<sup>35</sup> This index was normalized for body surface area. Cardiac output was calculated from echocardiographic diastolic and systolic LV volumes.<sup>36</sup>

### Measures of Carotid Midwall Function

As previously described in detail,<sup>10</sup> carotid midwall strain was derived with the use of a cylindrical model, adapted from Shimizu et al.,<sup>37</sup> which assumes that the volumes of the total carotid wall and of its inner and outer halves during the cardiac cycle are constant. If it is assumed that the arterial long axis remains constant, inner shell cross-sectional areas at end diastole and at end systole are equal, allowing use of end-diastolic carotid lumen diameter and wall thickness and peak systolic diameter to calculate the systolic thickness of the inner arterial wall shell as well as other midwall dimensions. From these values, previously reported equations<sup>10</sup> were used to calculate carotid midwall strain (expressed as percentage), the midwall Peterson's elastic modulus, the midwall Young's elastic modulus, and the midwall  $\beta$ .

Carotid end-systolic stress was estimated at the midwall from M-mode tracings, with the adaption of a cylindrical model<sup>37</sup> previously used for cardiac studies,<sup>38,39</sup> and the same approach was used to calculate carotid end-diastolic stress. These values were used to calculate the increment in carotid stress during the cardiac cycle ( $\Delta$ carotid stress).

Equations relating carotid luminal strain and carotid midwall strain to  $\Delta$ carotid stress in the clinically normal subjects were used to predict the expected carotid luminal and midwall strain, respectively, for observed  $\Delta$ carotid stress. The ratios of observed/predicted carotid strains were then derived to yield measures of carotid luminal and midwall strain adjusted for the imposed stress, called stress-corrected carotid strain.

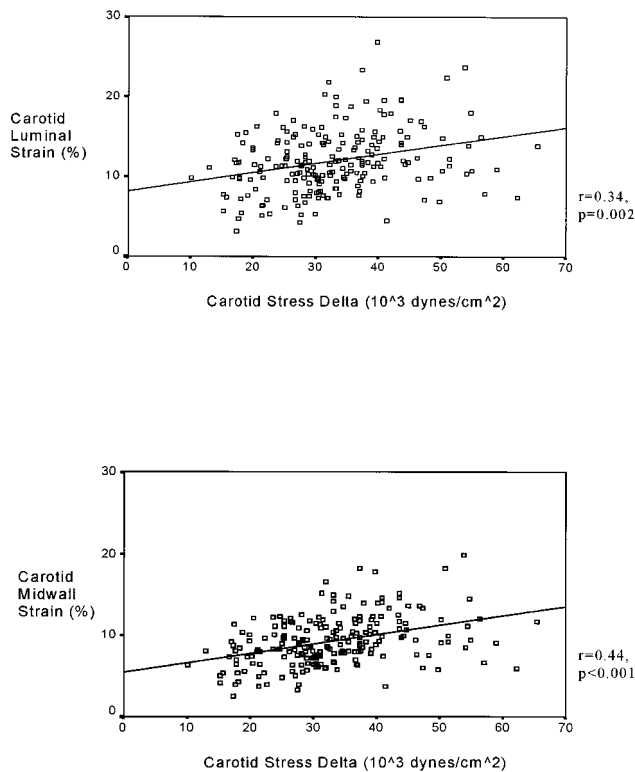
### Statistical Analysis

Data are presented as mean  $\pm$  SD. Continuous variables were compared by 1-way ANOVA, followed by the Scheffé post hoc test. Independent sample *t* tests and ANCOVAs that took into account relevant covariates were used to compare mean values between groups. Proportions were compared among groups by the  $\chi^2$  statistic. The independence of relations between continuous variables was evaluated by linear regression. The null hypothesis was rejected at 2-tailed  $P < 0.05$ .

## Results

### Relation of Carotid Luminal Strain and Midwall Strain to $\Delta$ Carotid Stress

Figure 1 shows the relation in hypertensive patients of carotid luminal strain to  $\Delta$ carotid stress (top;  $r = 0.29$ ,  $P < 0.001$ ) and of carotid midwall strain to  $\Delta$ carotid stress (bottom;  $r = 0.39$ ,  $P < 0.001$ ). Stress-corrected luminal strain ( $82 \pm 26\%$ ) and midwall strain ( $78 \pm 23\%$ ) in hypertensive patients were on average subnormal (both  $P < 0.001$  versus mean values of 100% and 99% in normal adults). Similarly, after adjustment for age, conventional measures of arterial stiffness were higher in hypertensive than in normotensive individuals ( $6.1 \pm 2.5$  versus  $5.0 \pm 2.5$ ,  $P = 0.002$  for  $\beta$ ;  $636 \pm 264$  versus  $502 \pm 204 \text{ dyne/cm}^2 \text{ per millimeter} \times 10^{-6}$ ,  $P < 0.001$  for Young's modulus). The significance of these differences was greater for stress-corrected luminal strain ( $t = 5.36$ ) and stress-corrected midwall strain ( $t = 6.89$ ) than for  $\beta$  ( $t = -3.09$ ) or Young's modulus ( $t = -4.17$ ), primarily because of lower within-group coefficients of variation for stress-corrected strains ( $\approx 25\%$ ) than for traditional stiffness measures ( $\approx 40\%$ ).

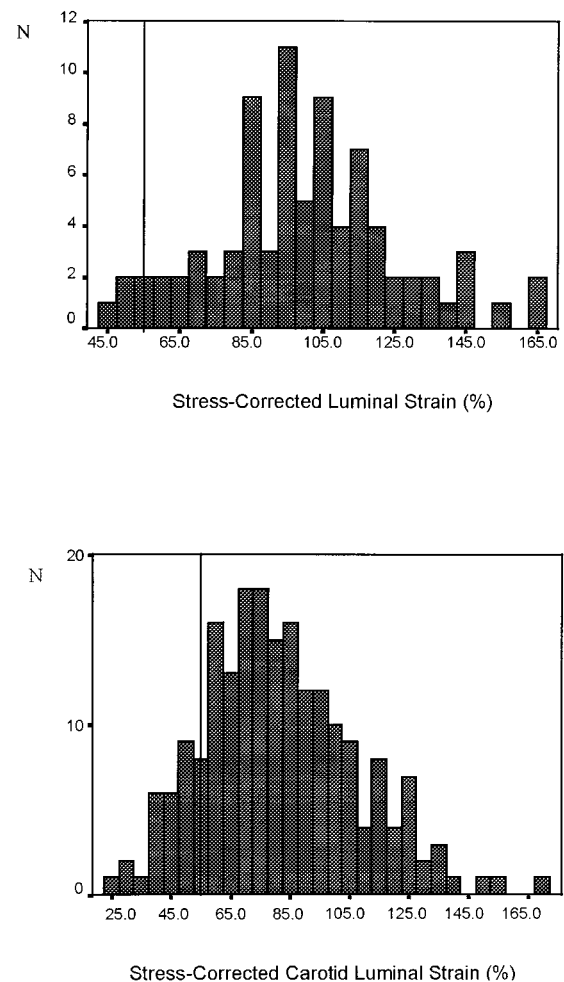


**Figure 1.** Top, Relation of carotid luminal strain (vertical axis) to  $\Delta$ carotid stress (horizontal axis) in hypertensive patients. Bottom, Relation of carotid midwall strain (vertical axis) to  $\Delta$ carotid stress (horizontal axis) in hypertensive patients.

Figure 2 compares the distribution of stress-corrected luminal strain in normal subjects and hypertensive patients. As seen in the bottom panel, the distribution of stress-corrected luminal strain was shifted to the left in hypertensive patients, with 14% below the 5th percentile of normal. Figure 3 compares the distributions of stress-corrected midwall strain in normal subjects and hypertensive patients. The distribution of stress-corrected midwall strain in the latter group was also shifted to the left, with 18% below the 5th percentile of normal.

### Characteristics of Hypertensive Patients With Reduced Arterial Compliance

Those in the subgroup of hypertensive patients with low stress-corrected midwall strain were older and had higher brachial and carotid systolic and diastolic blood pressures and higher brachial pulse pressures than the other patients (Table 1). There were no statistical differences between patient groups in gender, race, or body size. Although hypertensive patients with low stress-corrected midwall strain had slightly larger LV wall thickness, mass, and relative wall thickness than those with normal stress-corrected midwall strain, these differences did not approach statistical significance. Hypertensive patients with low stress-corrected luminal strain also had higher brachial and carotid systolic and diastolic blood pressures (Table 2). There were no significant differences with regard to age, gender, race, or body size. Patients with low stress-corrected luminal strain had slightly thicker inter-



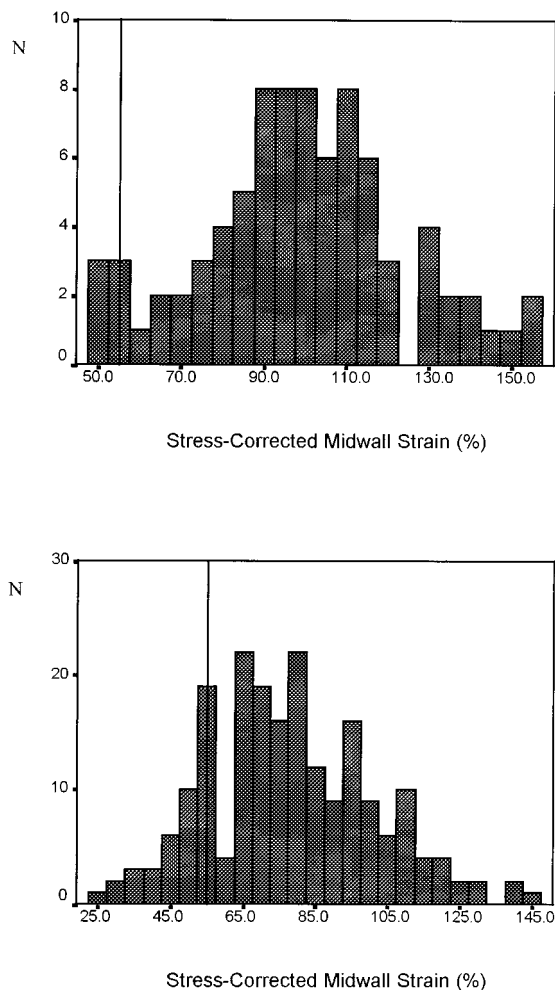
**Figure 2.** Top, Level of observed stress-corrected luminal strain in normotensive adults is normally distributed with a modal value at  $\approx 95\%$  of the predicted value. The vertical line represents the 5th percentile of the distribution. Bottom, Distribution of stress-corrected luminal strain in hypertensive patients is shifted to the left compared with normotensive individuals, with modal values of 70% to 75% of predicted and 14% below the vertical line representing the 5th percentile of normal.

ventricular septa but similar LV mass compared with the remaining patients.

### Carotid Luminal and Midwall Mechanics in Hypertensive Patients

Hypertensive patients with low stress-corrected luminal strain had larger carotid systolic and diastolic diameters than those with normal stress-corrected luminal strain ( $6.9 \pm 0.9$  versus  $6.5 \pm 0.9$ ,  $P=0.01$ ;  $6.5 \pm 0.9$  versus  $5.8 \pm 0.8$ ,  $P<0.001$ , respectively). There was no difference in arterial cross-sectional area between subgroups of patients. Patients with low stress-corrected midwall strain had larger carotid systolic and diastolic diameters than the other patients ( $7.0 \pm 0.9$  versus  $6.5 \pm 0.9$ ,  $P<0.001$ ;  $6.5 \pm 0.8$  versus  $5.7 \pm 0.8$ ,  $P=0.002$ , respectively). In contrast to the lack of difference when patients were grouped by level of stress-corrected luminal strain, those with low stress-corrected midwall strain had larger cross-sectional areas than those with normal stress-corrected midwall strain ( $20.7 \pm 6.6$  versus  $17.2 \pm 5.5$ ,  $P<0.001$ ).





**Figure 3.** Top, Level of observed stress-corrected midwall strain in normotensive adults is normally distributed with a modal value at  $\approx 95\%$  of the predicted value. The vertical line represents the 5th percentile of the distribution. Bottom, Distribution of stress-corrected midwall strain in hypertensive patients is shifted to the left compared with normotensive individuals, with modal values of 70% to 75% of predicted and 18% below the vertical line representing the 5th percentile of normal.

According to a previously derived regression equation,<sup>26</sup> arterial hypertrophy was present in 15% of hypertensive patients. These patients had lower carotid midwall strain and stress-corrected midwall strain than those without arterial hypertrophy, whereas no difference was observed in luminal or stress-corrected luminal strain (Table 3). Discrete atherosclerotic plaques were detected in 27% of hypertensive patients. Both stress-corrected carotid luminal and midwall strain were lower in those with plaque compared with those without, but this did not approach statistical significance.

After adjustment for age, hypertensive patients with low stress-corrected luminal strain had higher  $\beta$  ( $9.8 \pm 3.1$  versus  $5.5 \pm 1.9$ ) and Young's modulus ( $1032 \pm 378$  versus  $568 \pm 160$  dyne/cm<sup>2</sup> per millimeter  $\times 10^{-6}$ ) (both  $P < 0.001$ ) than the other hypertensive patients. Similarly, after adjustment for age, those with low stress-corrected midwall strain had higher  $\beta$  ( $9.6 \pm 3.0$  versus  $5.4 \pm 1.8$ ) and Young's modulus ( $1002 \pm 372$  versus  $565 \pm 161$  dyne/cm<sup>2</sup> per millimeter  $\times 10^{-6}$ )

**TABLE 1. Clinical and Echocardiographic Characteristics of Hypertensive Patients With Low or Normal Stress-Corrected Carotid Midwall Strain**

Variable	Stress-Corrected Midwall Strain	
	Low (n=33)	Normal (n=172)
Age, y	$61 \pm 12$ †	$54 \pm 12$
% Male	63	63
% White	74	65
Body mass index, kg/m <sup>2</sup>	$26.2 \pm 3.2$	$26.6 \pm 4.1$
Brachial SBP, mm Hg	$170.3 \pm 26.8$ ‡	$155.0 \pm 19.5$
Carotid SBP, mm Hg	$157.4 \pm 21.5$ ‡	$145.6 \pm 16.6$
Brachial DBP, mm Hg	$99.4 \pm 12.6$ †	$92.3 \pm 10.2$
Carotid DBP, mm Hg	$97.9 \pm 15.5$ †	$91.0 \pm 13.6$
Brachial PP, mm Hg	$70.9 \pm 25.2$ *	$62.8 \pm 17.1$
Carotid PP, mm Hg	$60.0 \pm 17.7$	$55.1 \pm 17.7$
IVST <sub>d</sub> , cm	$1.0 \pm 0.1$	$0.9 \pm 0.1$
LVID <sub>d</sub> , cm	$5.0 \pm 0.6$	$5.1 \pm 0.5$
PWT <sub>d</sub> , cm	$1.0 \pm 0.1$	$0.9 \pm 0.1$
LV mass, g	$187.3 \pm 53.0$	$177.6 \pm 47.0$
LV mass/height, g/m <sup>2.7</sup>	$44.5 \pm 8.9$	$42.0 \pm 10.0$
RWT	$0.39 \pm 0.06$	$0.37 \pm 0.06$

SBP indicates systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; IVST, interventricular septal thickness; LVID, LV internal dimension; PWT, posterior wall thickness; BSA, body surface area; and RWT, relative wall thickness. Subscript d indicates diastole.

\* $P < 0.05$ ; † $P < 0.01$ ; ‡ $P < 0.001$ .

(both  $P < 0.001$ ) than the other hypertensive patients. As expected, after adjustment for age, midwall measures of arterial stiffness were higher in hypertensive patients with low stress-corrected midwall strain than in the other hypertensive patients ( $9.3 \pm 3.4$  versus  $5.0 \pm 1.8$  for midwall  $\beta$ ;

**TABLE 2. Clinical and Echocardiographic Characteristics of Hypertensive Patients With Low and Normal Stress-Corrected Luminal Strain**

Variable	Stress-Corrected Luminal Strain	
	Low (n=30)	Normal (n=175)
Age, y	$59 \pm 12$	$55 \pm 12$
% Male	65	63
% White	71	65
Body mass index, kg/m <sup>2</sup>	$25.6 \pm 3.0$	$26.7 \pm 4.1$
Brachial SBP, mm Hg	$168.3 \pm 26.8$ †	$155.6 \pm 20.0$
Carotid SBP, mm Hg	$154.1 \pm 21.0$ *	$146.4 \pm 17.1$
Brachial DBP, mm Hg	$99.4 \pm 12.9$ †	$92.4 \pm 10.2$
Carotid DBP, mm Hg	$98.6 \pm 15.6$ †	$91.0 \pm 13.5$
Brachial PP, mm Hg	$68.8 \pm 24.9$	$63.3 \pm 17.6$
Carotid PP, mm Hg	$56.7 \pm 14.2$	$55.8 \pm 18.3$
IVST <sub>d</sub> , cm	$1.0 \pm 0.1$ *	$0.9 \pm 0.1$
LVID <sub>d</sub> , cm	$5.0 \pm 0.6$	$5.0 \pm 0.5$
PWT <sub>d</sub> , cm	$1.0 \pm 0.1$	$0.9 \pm 0.1$
LV mass, g	$188.7 \pm 53.0$	$177.5 \pm 47.1$
LV mass/height, g/m <sup>2.7</sup>	$43.9 \pm 8.9$	$41.5 \pm 10.0$
RWT	$0.39 \pm 0.06$	$0.37 \pm 0.06$

Abbreviations are as defined in Table 1.

\* $P < 0.05$ ; † $P < 0.01$ .

**TABLE 3. Carotid Luminal and Midwall Mechanics in Relation to Clinical Characteristics of Hypertensive Patients**

Variable	Gender		Age		Body Habitus		Arterial Hypertrophy		Arterial Plaque	
	Male	Female	<55 y	>55 y	Normal	Overweight	Absent	Present	Absent	Present
Luminal strain, %	11.9±3.9	12.0±4.1	12.2±3.9	11.7±4.1	12.1±4.1	11.6±3.8	12.1±3.9	10.9±4.6	12.1±4.2	11.5±3.3
midwall strain, %	9.2±3.0	9.3±3.0	9.6±3.0	8.9±3.0	9.4±3.1	9.0±2.9	9.5±2.9	7.9±3.1†	9.4±3.2	8.8±2.5
Stress-corrected luminal strain, %	81±26	83±27	84±25	79±27	82±26	81±25	82±25	78±31	83±27	79±22
Stress-corrected midwall strain, %	78±23	79±23	82±22	75±23*	78±24	77±22	79±23	70±24*	79±24	74±20

\* $P<0.05$ ; † $P<0.01$ .

2921±1044 versus 1665±564 dyne/cm<sup>2</sup> per millimeter  $\times 10^{-6}$  for midwall Young's modulus) (both  $P<0.001$ ).

### Carotid Midwall Mechanics and LV Hypertrophy

Compared with hypertensive patients with normal LV geometry and concentric remodeling, those with concentric hypertrophy had lower luminal strain and midwall strain as well as stress-corrected luminal strain and midwall strain (Table 4). The patients with eccentric LV hypertrophy had lower luminal and midwall strain as well as stress-corrected luminal and midwall strain, but these differences did not attain statistical significance.  $\Delta$ Carotid stress did not differ among the LV geometric patterns.

### Discussion

Measurement of arterial compliance may provide evidence of vascular changes that precede the development of overt atherosclerotic disease.<sup>40</sup> Therefore, noninvasive assessment of arterial compliance may aid in early detection and subsequent prevention of atherosclerotic disease. Risk factors for vascular disease, including older age, high blood pressure and cholesterol, diabetes, and LV hypertrophy, are known to be associated with decreased arterial compliance.<sup>24,41–48</sup>

Several methods of noninvasive assessment of arterial compliance rely on the relation between systolic lengthening of arterial lumen diameter in relation to the corresponding change in blood pressure.<sup>32–34</sup> However, conventional methods do not examine the average strain of the arterial wall, approximated by the behavior of the midwall of the artery, nor do they take into account the average imposed stress. On the basis of research on the left ventricle that demonstrated that shifting the examination of LV mechanics to the midwall improves understanding of ventricular function in individuals with abnormal cardiac geometry,<sup>49,50</sup> we recently used carotid ultrasound and the

highly skill-dependent technique of carotid applanation tonometry to evaluate in apparently normal adults a measure of arterial compliance based on carotid midwall strain and its relation to the increment in carotid stress during systole ( $\Delta$ carotid stress).<sup>10</sup> In that study, carotid midwall strain was unrelated to gender, positively related to  $\Delta$ carotid stress, and negatively related to age, overweight, and standard measures of arterial stiffness. In addition, stress correction strengthened the negative relation of carotid midwall strain with age, suggesting that assessment of carotid midwall mechanics may enhance noninvasive assessment of the compliance of conduit arteries.

In this study of hypertensive patients, carotid midwall strain was positively related to  $\Delta$ carotid stress, albeit slightly less closely than in normotensive individuals.<sup>10</sup> The likely explanation for this is that hypertensive patients respond to elevated blood pressure heterogeneously, with proportionate increases in normally compliant connective tissue and muscular elements in some patients but disproportionate increases in noncompliant connective tissue in others.<sup>49</sup>

We have previously demonstrated that increased carotid wall thickness is associated with decreased carotid distensibility in hypertensive patients compared with age-matched normotensive subjects.<sup>8</sup> In our initial report,<sup>25</sup> when differences in wall thickness were taken into account using Young's elastic modulus, carotid artery stiffness was not statistically greater in hypertensive patients than in normotensive subjects. In contrast, the present study of substantially larger hypertensive and normotensive populations reveals statistically higher arterial stiffness by conventional indices in hypertensive patients, a difference that became more striking when the new measures of arterial mechanics were used. Moreover, the present study demonstrates that the carotid arterial wall is stiffer in hypertensive patients than in normo-

**TABLE 4. Carotid Midwall Mechanics and LV Geometric Patterns**

	Hypertensive Patients				
	Normal (n=82)	Normal Geometry (n=129)	Concentric Remodeling (n=42)	Eccentric LV Hypertrophy (n=21)	Concentric LV Hypertrophy (n=13)
Luminal strain, %	14.6±3.9	12.4±3.7	12.3±4.8	10.3±3.4	8.8±1.7*
Midwall strain, %	11.7±3.2	9.6±2.9	9.4±3.5	8.1±2.5	6.8±1.3*
$\Delta$ Stress, 10 <sup>3</sup> dyne/cm <sup>2</sup>	32.8±10.6	32.9±10.6	33.7±8.9	31.9±11.1	30.9±7.8
Stress-corrected luminal strain, %	100±26	85±25	83±31	71±22	62±11*
Stress-corrected midwall strain, %	100±24	81±22	78±27	69±19	59±10*

\* $P<0.05$ , compared with patients with either normal LV geometry or concentric remodeling by ANOVA with Scheffé post hoc test.

tensive subjects when compared at a given circumferential  $\Delta$ stress (assessed by stress-corrected strains), when arterial wall thickness is taken into account (Young's modulus and midwall Young's modulus), and when the curvilinear arterial pressure-diameter relation is taken into account ( $\beta$  and midwall  $\beta$ ).

In our study,  $\Delta$ carotid stress was statistically indistinguishable in normotensive adults and hypertensive patients. Because carotid luminal strain was reduced (by 19% on average) in hypertensive patients, use of stress-corrected luminal strain identified 14% of patients with low arterial compliance. A slightly larger proportion of hypertensive patients (18%) had low arterial compliance by stress-corrected midwall strain. These patients were older and had thicker arterial walls and higher blood pressures than the hypertensive patients with normal stress-corrected midwall strain. In addition, all conventional measures of arterial stiffness were higher in hypertensive patients with low stress-corrected midwall strain.

In a previous study,<sup>47</sup> we demonstrated that hypertensive patients with concentric LV hypertrophy have a greater increase in arterial wall thickness, cross-sectional area, and conventional measures of arterial stiffness at the operating level of distending pressure than hypertensive patients with other LV geometric patterns. In the present study, those with concentric LV hypertrophy had reduced stress-corrected luminal and midwall strain. Stress-corrected luminal and midwall strain also tended to be more subnormal in those with eccentric LV hypertrophy than in those with normal LV geometry. An intriguing result is that LV hypertrophy is related to stress-corrected midwall strain but not to conventional measures of arterial stiffness, implying that the association between LV hypertrophy and arterial dysfunction is better assessed by use of carotid midwall mechanics.

## Conclusion

Stress-corrected midwall strain may identify hypertensive patients with reduced arterial compliance, increased arterial wall thickness, and abnormal LV geometry better than conventional arterial function measures based on arterial lumen diameters.

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