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Forearm Arterial Distensibility in Systolic Hypertension

HAROLD SMULYAN, MD, FACC,* SUMAN VARDAN, MD,* AMOS GRIFFITHS, MB, FRCP,†
BRIAN GRIBBIN, MD, FRCP†

Syracuse, New York and Oxford, England

It is unclear whether the stiffened arterial tree in systolic hypertension is the cause or the effect of the disease. In this study, brachial and radial arterial pulses were sensed by external Pixie transducers and measurements of pulse wave velocity converted to volume distensibility using the Bramwell-Hill equation. Blood pressure was controlled as a variable by repeating the measurements at a variety of transmural arterial pressures. This was accomplished by encasing the forearm in a rigid plastic cylinder within which pressures were varied.

Twenty-nine patients with systolic hypertension were compared with 22 age-matched control subjects. At ambient pressures the volume distensibility of patients was lower than that of control subjects (0.10 versus 0.18% Δ volume/mm Hg, $p < 0.001$) but there was no difference in volume distensibility between the two groups at any comparable transmural pressure. Nineteen patients were treated for 1 month with a thiazide diuretic agent and the studies were then repeated. Systolic and diastolic blood pressure decreased significantly and volume distensibility increased (0.10 to 0.15% Δ volume/mm Hg, $p < 0.001$) at ambient pressures. But at comparable transmural pressures, volume distensibility was unchanged.

It is concluded that, in the forearm, increased arterial stiffness is the result and not the cause of systolic hypertension, but these data cannot exclude increased aortic stiffness as a significant factor. Thiazide diuretic drugs increase forearm arterial compliance by lowering blood pressure without a demonstrable drug effect on this arterial wall.

There is increasing evidence that isolated systolic hypertension is a clinically important disease rather than a benign consequence of aging. This perception has developed because patients with this disorder have been shown to suffer from increased cardiovascular morbidity and mortality when compared with age-matched normotensive individuals (1–3). Despite its clinical importance, little is known of either the cause of systolic hypertension or its relation to essential hypertension. Although systolic hypertension is hemodynamically variable (4–6), it is still a common belief that isolated systolic hypertension is due to a stiffened arterial tree. Evidence for this assumption is limited, because methods for measuring arterial distensibility are difficult and pertinent reports few. The best technique for this purpose is aortic input impedance, but this method requires catheterization of the central aorta to measure simultaneous pressure and flow, and is not applicable to asymptomatic ambulatory patients. Simpler and less invasive techniques have been tried (7–9) but have not been widely used, probably because they are highly indirect and involve unacceptable assumptions. Recently, Simon et al. (4) described a method for calculating arterial compliance using a previously measured systemic vascular resistance and assuming the diastolic portion of the brachial artery pulse to be a monoexponential. These investigators applied this method to the study of patients with systolic hypertension and concluded that their arterial trees were indeed stiff.

All methods for estimating arterial distensibility suffer from an additional and important drawback when they are applied to the study of patients with hypertension; that is, elevation of the arterial pressure itself induces an increase in arterial stiffness. Therefore, unless the blood pressure is controlled as a variable, the demonstrated decrease in arterial distensibility in patients with systolic hypertension could be the effect, as well as the cause, of the disease. Any method used to study the role of arterial distensibility in patients with systolic hypertension not only must compare

From the Cardiology Section and Department of Medicine, Veterans Administration Medical Center, and Upstate Medical Center of State University of New York, Syracuse, New York* and Radcliffe Infirmary, Oxford, England.† This study was supported in part by grants from the Veterans Administration, Washington, D.C. Manuscript received May 31, 1983; revised manuscript received September 13, 1983, accepted September 30, 1983.

Address for reprints: Harold Smulyan, MD, Syracuse Veterans Administration Medical Center, 800 Irving Avenue, Syracuse, New York 13210.

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age-matched individuals with and without high blood pressure, but also must do so at the same levels of blood pressure.

In the present study, we used a technique, originally described by Gribbin et al. (10), which calculates forearm arterial distensibility from measurements of pulse wave velocity. The details of this method have been modified slightly and presented previously (11). The technique was applied in a series of patients with systolic hypertension and the data compared with those of a normotensive age-matched group. Arterial pressure was controlled as a variable by repeating the measurements at a variety of transmural arterial pressures. This was accomplished by encasing the subjects' forearm under study in a plastic cylinder, within which the air pressure was varied.

Methods

Patients

Study group. Twenty-nine patients with systolic hypertension participated in this study. Systolic hypertension was defined as a systolic pressure equal to or greater than 160 mm Hg and also greater than two times the diastolic pressure minus 15 mm Hg (12). There were 18 men and 11 women in the group, whose ages ranged from 46 to 81 years (mean 67.7). By history, physical examination, routine electrocardiogram, chest X-ray film, blood count, fasting blood sugar, blood urea nitrogen and serum electrolytes, patients were free of disorders other than left ventricular enlargement and mild diabetes mellitus. Specifically excluded by these means were other causes of systolic hypertension such as aortic regurgitation, thyrotoxicosis, anemia, arterial venous fistula or a heart rate less than 50 beats/min. Nineteen patients were studied at the Radcliffe Infirmary, Oxford, England, and the remainder at the Veterans Administration Medical Center, Syracuse, New York.

Control group. For purposes of comparison, a control group of 22 asymptomatic normotensive, age-matched individuals were studied in Syracuse. The normotensive subjects were recruited from the attending medical, nursing and technical staffs, hospital volunteers, volunteers from the Nursing Home Unit which is part of the Syracuse Veterans Administration Medical Center and nearby senior citizens' housing. There were 11 men and 11 women in the control group, whose ages ranged from 50 to 85 years (mean 67.2). Although this group was asymptomatic and normotensive, undetected subclinical disorders could have been present.

Treatment group. Nineteen of the 29 patients with systolic hypertension were restudied after 1 month of therapy with a thiazide diuretic agent. Nine patients studied in Oxford received cyclopenthiazide, 0.5 mg/day, and 10 patients studied in Syracuse received hydrochlorothiazide, 50 mg/day. The technique used for blood pressure measurement was the same in the studies done before and after treatment. The results for male and female subjects as well as the Oxford and Syracuse data were analyzed separately, but no differences were detected. Therefore, all these groups are considered together.

Approval and patient consent. The methods described were carefully explained to each subject and written informed consent obtained. These studies had been previously approved by the Project Review and Human Studies Subcommittee of both institutions.

Measurement Techniques and Calculations

Pulsewave recordings. After measuring the blood pressure in both arms (cuff method) for parity, Pixie strain gauges, embedded in plastic (Endevco Corporation, San Juan Capistrano, California) were placed over the right brachial artery in the antecubital fossa and the right radial artery at the wrist. Each gauge was made one arm of a Wheatstone-bridge, the balanced bridge outputs amplified (model VR 6, Electronics for Medicine) and the radial dilation waves of both brachial and radial arteries displayed on an oscilloscope. Gauge positions were adjusted until undistorted pulse waves were obtained and the gauge locations maintained by encircling soft elastic bands. Satisfactory pulse curves were then recorded on ultraviolet sensitive paper at a speed of 250 mm/s (Honeywell Visicorder, model 1508).

Cylinder pressure recordings. With the gauges in place, the supinated arm was placed in a large plastic cylinder with the elbow and wrist extended. The cylinder was closed at the distal end and bound to the upper arm at the proximal end with a soft rubber sleeve. To minimize pressure loss, this sleeve was covered with an encircling blood pressure cuff which was inflated, when cylinder pressures were positive, to pressures just higher than those within the cylinder. Cylinder pressures were varied in 10 mm Hg increments using an industrial vacuum cleaner and an adjustable leak. The pressure within the cylinder was monitored through a small port using a mercury U-tube. Altered cylinder pressures were maintained for 30 to 60 seconds before pulse waves were recorded, and ambient pressures were re-established for several minutes between runs. It was usually possible to raise the cylinder pressure 50 mm Hg, and lower it 80 mm Hg without undue distortion of the pulse waveform. At the conclusion of each study, the distance between the metal recording extension of each gauge was measured by running a strip of adherent paper tape over the volar surface of the forearm and finding the shortest distance between the points of indentation on the skin. The position of the two gauge arms were marked on the tape and the tape was removed. The strip of tape was then placed on a flat surface and the distance between the marks measured.

Pulse wave velocity measurements. The linearity of the Pixie strain gauges was tested by applying known forces to the gauge arm. The gauges were found to be linear from

zero force to gauge fracture. The frequency response of the gauges showed an average of 5% loss of signal amplitude at frequencies up to 20 cycles/s. Because the characteristic wave velocity of the pressure pulse represents the velocity of travel of its higher frequency components, and the pulse curve upstroke contains high frequency information, points on the brachial and radial upstrokes at 10% of the pulse amplitude were arbitrarily chosen to mark the pulse arrival time. The time interval between these two points was measured, using the inscribed time lines as a reference. This interval was measured on 8 beats at each cylinder pressure and the mean of these values designated as the pulse transmission time. Using the distance between the strain gauges the pulse wave velocity (PWV) between the brachial artery and radial artery loci was calculated. Wave velocities were then converted to volume distensibility (VD) using a modification of the Bramwell-Hill equation (13):

$$VD = (3.57/PWV)^2.$$

Pulse wave velocities measured in this way have been compared, on the same pulses, with the characteristic wave velocities of the higher harmonics calculated by Fourier analysis. Seventy-three such comparisons had a regression coefficient of + 0.97 and a Y intercept near zero (11).

Arterial blood pressure data. *Arteriosonde measurements.* Arterial blood pressure was measured with an external cuff on the opposite arm using the Arteriosonde device (model 1225, Roche Medical Electronics) in the 22 normotensive subjects and 19 of the patients with systolic hypertension. These pressures were measured during the recording of the arterial pulses at each cylinder pressure. Mean arterial pressure was calculated as one-third of the pulse pressure plus the diastolic pressure, and transmural arterial pressure was calculated by adding or subtracting the cylinder pressure from the mean arterial pressure. Fifty-seven comparisons of mean blood pressure using the Arteriosonde device in one arm with those simultaneously obtained by electronically damped intraarterial pulses in the other arm were obtained in 20 different patients with systolic hypertension. The intraarterial mean pressures ranged from 85 to 126 mm Hg. There was no significant difference between the mean values obtained by the two methods (105.5 mm Hg intraarterial versus 103.8 mm Hg Arteriosonde). The regression coefficient for the relation was + 0.91 with a Y intercept of 7.4 mm Hg.

Intraarterial measurements. In the 10 other patients with systolic hypertension, arterial pressures were measured intraarterially, simultaneously with the recording of radial dilation waves in the opposite arm. Because there was no systematic difference bewteen the mean blood pressure measured by the two methods, the data on all of the patients with systolic hypertension were considered together.

Hemodynamic data. In the 10 patients with systolic hypertension whose blood pressures were recorded intraarterially, intravenous and intraarterial cannulas were positioned under 1% lidocaine local anesthesia. The arterial line was connected via pressure monitoring tubing (Pharmaseal P124) to a strain gauge (Statham model P123 Db) whose phasic and mean outputs were amplified and recorded by an Electronics for Medicine (Simultrace recorder model DR6) recorder. The mean arterial blood pressure was obtained by electronic damping. The mean systolic ejection time was averaged from 5 beats by measuring the time interval from the upstroke to the dicrotic notch of the brachial artery pressure curves recorded at a paper speed of 100 mm/s. Cardiac output was computed from standard dye-dilution curves, using 5 mg of indocyanine green as the indicator. From these variables and the height and weight of the patients, the body surface area, cardiac index, stroke volume, mean systolic ejection rate, mean systolic ejection rate index and systemic vascular resistance were calculated by applying standard formulas.

Assumptions in Pulse Wave Velocity Method

The pulse wave velocity measurement for estimation of arterial distensibility involves several assumptions. The Bramwell-Hill equation, which is derived from the Moens-Korteweg equation, applies only to thin-walled nonviscous tubes. However, in vitro measurements of mean pulse wave velocity have satisfactorily predicted arterial distensibility measured by direct methods (14–18). These data showed considerable scatter, but the mean values for pulse wave velocity accurately reflected the direct distensibility measurements. The calculated volume distensibility cannot distinguish between active or static components of arterial wall behavior. Our method further assumes that changes in cylinder pressure are faithfully transmitted to the arterial wall. This issue for both positive and negative pressures has been studied previously (19,20) and found to be satisfactory. Undetected occlusive atherosclerosis in the forearm arteries is uncommon, but was excluded by ensuring the equality of blood pressures in both arms. The scatter of the results (see later) is due to the effect of these assumptions and to the variations in arterial diameter from person to person, unmeasured in the present study, which also influence pulse wave velocity. Differences in blood viscosity from patient to patient probably had little effect on the pulse wave velocity measurement, because anemia or polycythemia was excluded. Another minor variable is the velocity of blood flow, which adds to the pulse wave velocity but is small when compared with the rate of pressure pulse travel.

Results

For each patient, the relation between volume distensibility and the transmural pressure is curvilinear (Fig. 1). The relation between log volume distensibility and transmural pressure was linear in some but not in all patients.

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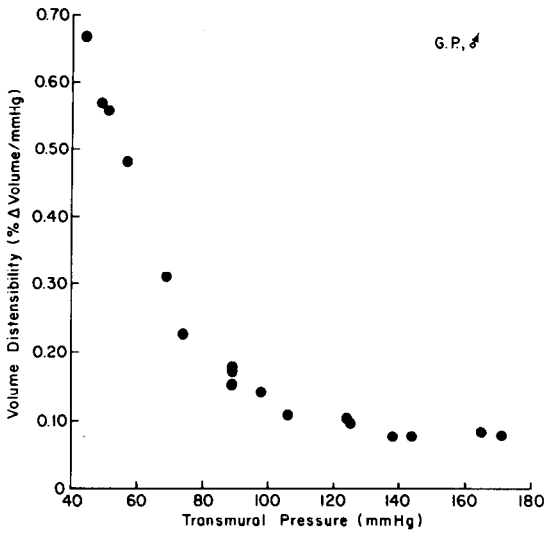


Figure 1. Relation between calculated volume distensibility and arterial transmural pressure in a single patient (G.P.).

In most patients the plot of these variables was linear above and below ambient transmural pressure, but showed a bend in the line at that point. For this reason, the data were best represented by separate linear regression lines (method of least squares) for the upper and lower portion of the plot, using the ambient transmural pressure as the separation point (Fig. 2). With these regressions, it was possible to calculate volume distensibility for any transmural pressure common to both hypertensive and normotensive patients.

Comparison of normotensive and systolic hypertensive groups (Table 1). The two groups were of comparable age (normotensive 67.2, hypertensive 67.7 years). The systolic, diastolic, mean and pulse pressures were, as defined, significantly greater in the systolic hypertensive group. The volume distensibility of the group with systolic hypertension

Figure 2. Relation between log volume distensibility and arterial transmural pressure in a single patient (J.Z.) Least square regression lines calculated separately for values above and blow ambient mean blood pressure (B.P.).

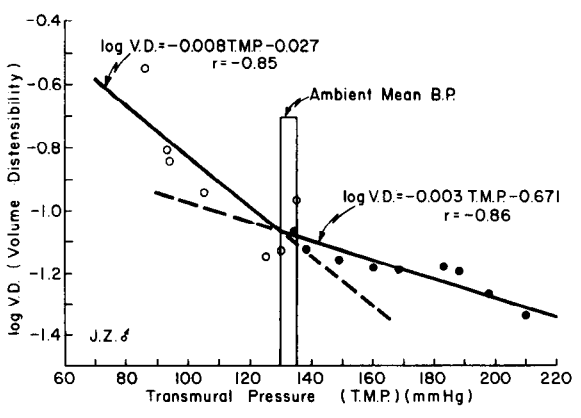


Table 1. Systolic Hypertension and Age-Matched Normal Subjects

	Normotensive	Hypertensive	p* value
Number	22	29	
Age (yr)			
mean	67.2	67.7	NS
SE	10.2	8.4	
Blood pressure (mm Hg)			
Systolic			
mean	125.2	179.4	<0.001
SE	2.7	3.1	
Diastolic			
mean	74.9	88.3	<0.001
SE	1.65	1.7	
Mean			
mean	91.7	120.9	<0.001
SE	1.3	1.7	
Pulse			
mean	50.3	91.0	<0.001
SE	3.3	3.6	
Volume distensibility (% Δ volume/mm Hg)			
90 mm Hg			
mean	0.183	0.174	NS
SE	0.0088	0.0119	
120 mm Hg			
mean	0.099	0.107	NS
SE	0.0041	0.0061	
Ambient BP			
mean	0.179	0.108	<0.001
SE	0.0083	0.0070	

*Student's *t* test. BP = blood pressure; NS = not significant; p = probability; SE = standard error.

was significantly lower than that of the normotensive group at ambient blood pressure. At 90 mm Hg (the approximate mean pressure of the normotensive group) the volume distensibility of normal and hypertensive groups are not significantly different. At 120 mm Hg (the approximate mean ambient blood pressure in the systolic hypertension group) there was again no significant difference between the volume distensibility in the two groups.

Because the normotensive group had a lower ambient pressure than the hypertensive group, the extremes of transmural pressures investigated were lower than those of the hypertensive group (Fig. 3). The volume distensibility for the ambient mean pressure was lower for the hypertensive than for the normal patients. However, with the curves for the two groups superimposed, the volume distensibility at any equivalent transmural pressure was the same.

The data on cardiac output, cardiac index, stroke volume, stroke volume index, mean systolic ejection rate or mean systolic ejection rate index are part of a larger series of patients whose hemodynamic data are reported elsewhere (6). In the 10 patients studied, these variables were not significantly correlated to the volume distensibility at ambient pressures, to volume distensibility at a variety of trans-

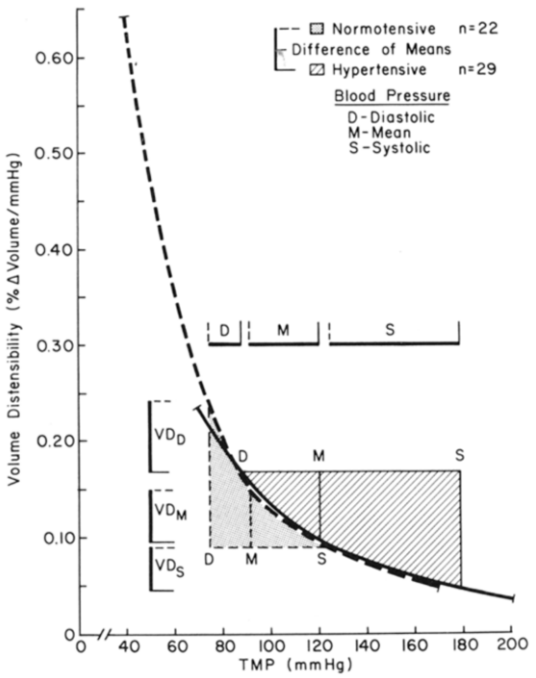


Figure 3. Relation between calculated volume distensibility and arterial transmural pressure for the hypertensive and control groups. **Dark horizontal lines** indicate differences between the groups in systolic, mean and diastolic pressures. **Dark vertical lines** indicate differences between the groups in volume distensibility due to the differences in respective blood pressures. Abbreviations as before.

Table 2. Systolic Hypertension—Effects of Treatment

	Untreated	Treated	p* value
Number	19	19	
Age (yr)	66.7	66.7	
Blood Pressure (mm Hg)			
Systolic			
mean	180.6	153.5	<0.001
SE	3.9	4.6	
Diastolic			
mean	88.0	72.2	<0.001
SE	2.3	2.7	
Mean			
mean	122.2	100.4	<0.001
SE	2.0	2.8	
Pulse			
mean	92.6	81.3	<0.005
SE	4.8	4.5	
Volume Distensibility (% Δ volume/mm Hg)			
90 mm Hg			
mean	0.176	0.173	NS
SE	0.0164	0.0106	
120 mm Hg			
mean	0.105	0.096	NS
SE	0.0089	0.0057	
Ambient BP			
mean	0.107	0.142	<0.001
SE	0.0103	0.0090	

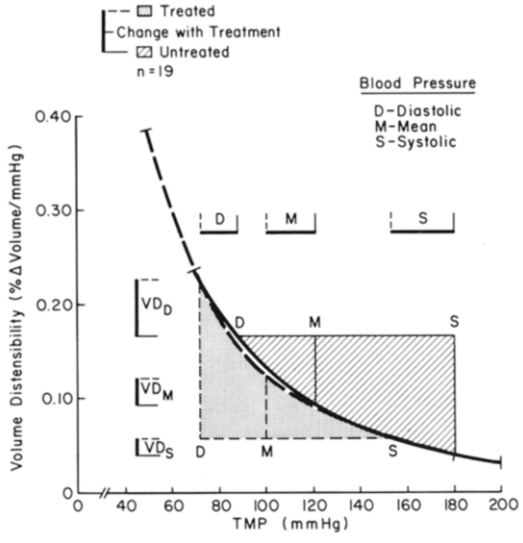
*Student's *t* test for paired values. Abbreviations as in Table 1.

mural pressures, or to the slopes of the log volume distensibility-transmural pressure relation.

Effects of thiazide diuretic therapy (Table 2). As expected, 1 month of thiazide diuretic therapy reduced the systolic, diastolic, mean and pulse pressures significantly. The reduction in mean blood pressure was associated with an increase in volume distensibility at ambient blood pressure (Table 2). However, when the volume distensibilities for these patients were compared before and after treatment at the same transmural pressure, there was no significant effect of therapy (Fig. 4). Figure 4 also shows the significant reduction in blood pressure associated with treatment, the significant increase in volume distensibility associated with the decrease in ambient mean blood pressure and the overlap of the curves. This overlap indicates no significant change in volume distensibility as a result of therapy at any of the comparable transmural pressures. Figure 4 also shows the reduction in volume distensibility associated with the decrease of the systolic, mean or diastolic pressure individually. Because of the shape of the curve relating volume distensibility and transmural pressure, the largest increase in volume distensibility is associated with the decrease in diastolic pressure and the smallest increase in volume distensibility is associated with the decrease in systolic blood pressure. The increase in volume distensibility associated with the decrease in mean blood pressure is intermediate between that of the systolic and diastolic pressures.

Cardiac output, stroke volume or mean systolic ejection rate did not correlate significantly with any of the measures of arterial distensibility, before or after treatment.

Figure 4. Relation between calculated volume distensibility and arterial transmural pressure in hypertensive patients before and after thiazide diuretic treatment. **Dark horizontal lines** indicate the reduction in systolic, mean and diastolic pressures. **Dark vertical lines** indicate the increase in volume distensibility associated with the change in respective blood pressures.



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Discussion

Arterial distensibility in systolic hypertension. Elevation of the systolic pressure is due to increased stroke volume, increased rate of systolic ejection, increased arterial tree stiffness or combinations of these factors. It has been assumed, without much experimental evidence, that increased arterial stiffness is the major cause of the disorder. Koch-Weser (12) pointed out that if arterial stiffening were the only factor responsible for systolic hypertension, then in addition to systolic pressure elevation, the diastolic pressure should be low. This opinion has been confirmed experimentally (21). Because diastolic blood pressure is usually normal or slightly elevated when measured by the cuff technique, Koch-Weser believed that factors other than arterial stiffening were operative in this disease. However, previous data from our laboratory (22) have shown that the diastolic pressures obtained by the cuff technique are falsely elevated when compared with simultaneous intraarterial pressures in patients with isolated systolic hypertension. Furthermore, the intraarterial diastolic pressures were often lower than normal, making it possible that decreased arterial distensibility was a causative factor. To complicate matters, our previous studies (6) have also indicated that in most patients the systemic vascular resistance was elevated when it was considered as a function of the cardiac output, but in some cases the stroke volume was also increased. All of these efforts to indict or exclude arterial stiffness as a cause of isolated systolic hypertension have not studied the arterial wall itself.

Control for blood pressure differences. Any method that purports to study arterial distensibility in hypertension must control for differences in the blood pressure. As shown in Figure 1, any increase in intraarterial pressure is associated with a decrease in arterial distensibility regardless of the intrinsic properties of the arterial wall. Therefore, it would be unusual if any study on arterial distensibility in patients with hypertension did not demonstrate increased arterial stiffness. Most previous attempts to study arterial distensibility considered blood pressure as a variable (4,7–9), but did not control it, and therefore could not determine whether the decreased arterial distensibility was the cause or the effect of the disease. Only Gribbin et al. (10), using the pulse wave velocity method in patients with essential hypertension, have shown no abnormality in forearm arterial distensibility when the effects of hypertension on the artery wall were controlled.

Simon et al. (4), whose technique indicated decreased arterial distensibility in patients with systolic hypertension when compared with that in age-matched control subjects, suggested that control of the blood pressure as a variable was not of concern because their experimental and control groups had similar diastolic pressures. Because their patients with isolated systolic hypertension had, by definition, a higher systolic pressure, the mean pressure must have also been higher than that of the control subjects. Because the mean pressure is most representative of the entire pulse, it seems unlikely that high blood pressure of any form can be dismissed as a cause of the arterial stiffness.

Forearm versus elastic and transitional arteries. The method used in this study also demonstrated decreased arterial distensibility in patients with isolated systolic hypertension, but this was true only at ambient pressures. When volume distensibility was measured at identical pressures, no differences could be found in arterial stiffness. Our method offers data only on the forearm arteries, which might be comparable with other muscular arteries of the body, but cannot be applied to elastic arteries, such as the thoracic aorta, or transitional arteries, such as the abdominal aorta, or carotid arteries. Stiffening of the elastic arteries, such as the aorta, may be more important to the development of systolic hypertension than changes in the muscular brachial artery which is usually spared the atherosclerotic process. Therefore, this study cannot entirely exclude arterial stiffness, especially aortic stiffness, as a factor in isolated systolic hypertension.

Scatter of data. The data generated by the present method also shows considerable scatter. This is true of most other arterial distensibility studies, but makes our negative results susceptible to a type 2 statistical error. However, statistically significant differences in wall distensibility were demonstrated at ambient blood pressures. This fact suggests that any intrinsic difference in forearm arterial distensibility between patients with systolic hypertension and control subjects, if present at all, must be small compared with the difference produced only by the hypertension.

The hemodynamic explanation for systolic hypertension remains unclear. This study makes decreased distensibility of muscular arteries an unlikely possibility, but does not exclude aortic stiffness as a factor. Our previous hemodynamic studies in such patients (6) showed a spectrum of values, some with elevated systemic vascular resistance, and others with high cardiac output and stroke volume. This suggests either that the mechanism for systolic hypertension differs from patient to patient or that the essential measurement has not yet been made.

Effects of therapy on arterial distensibility. Difficulties in assessing the effects of therapy on arterial distensibility in hypertension are similar to those we described for the comparison of arterial distensibility between normotensive and hypertensive persons. Again our study showed an increase in arterial distensibility with successful antihypertensive therapy. This increased distensibility was due, however, to the decline in blood pressure, rather than to a change in the properties of the forearm arterial wall.

Previous studies have shown that arterial distensibility and the velocity of wave travel change at any instant during the cardiac cycle because of instantaneous changes in in-

traarterial pressure (23). In the absence of hysteresis, one could then envision arterial distensibility in patients with isolated systolic hypertension moving up and down along the curve relating volume distensibility and transmural pressure during each heart beat. Although treatment does not change the position of the curve, the location of the oscillation of each arterial pulse will have moved along the same curve to the left and upward. Our study shows a greater increase in arterial distensibility from the reduced diastolic pressure than from the decrease in systolic pressure. Because antihypertensive therapy increases arterial distensibility, it seems likely that therapy also reduces arterial impedance to left ventricular ejection. However, the magnitude of work reduction for the left ventricle by this method is not readily calculable from our data.

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