Echocardiographic Assessment of Cardiac Anatomy and Function in Hypertensive Subjects

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SUMMARY Cardiovascular complications are a major source of morbidity and mortality in hypertensive patients. To assess the prevalence of anatomic and functional abnormalities of the heart in such patients, we studied 234 asymptomatic subjects with mild-to-moderate systemic hypertension by echocardiography. After adjusting the echocardiographic values for age and body surface area, we found abnormally increased ventricular septal and/or posterobasal free-wall thickness in 61% of the hypertensive subjects. We found increased left atrial, aortic root, and left ventricular internal dimension (at end-diastole) in 5–7%, and decreased mitral valve closing velocity (E-F slope) and left ventricular ejection fraction were noted in six and 15% of the subjects, respectively. Four percent of the patients had disproportionate septal thickening (i.e., ventricular septal-to-left ventricular free-wall thickness ratio \(\geq 1.3\)). In contrast to the high prevalence of cardiac abnormalities detected by echocardiography, less than 10% of the hypertensive subjects had abnormal 12-lead ECGs or abnormal chest x-rays. These findings demonstrate a high prevalence of cardiac abnormalities in a population of asymptomatic hypertensive subjects. These abnormalities can be detected by echocardiography before they are otherwise apparent.

ABNORMALITIES OF CARDIAC STRUCTURE and function occur in response to sustained hypertension. Before echocardiography, however, it was difficult to detect early cardiac complications in patients with hypertension. As a result, the first indication of cardiac involvement in many patients was the development of overt congestive heart failure.

Echocardiography, which provides a direct means of assessing both functional and anatomic abnormalities of the heart, has recently been applied to the study of hypertensive subjects. The results of these studies have suggested that increases in left atrial dimension\(^1\) and left ventricular wall thickness\(^2\) and decreases in closing velocity (E-F slope) of the anterior leaflet of the mitral valve\(^3\) occur frequently even in the absence of other signs of cardiac involvement. However, similar changes have been reported to occur in normal subjects as a result of aging.\(^4\), \(^5\)

To determine whether some or all of the abnormalities reported in hypertensive subjects could be related to age rather than blood pressure elevation, we evaluated a population of subjects with mild-to-moderate hypertension and compared the results with normal data using regression equations that accounted for both body surface area and age. We also examined the relation of the various echocardiographic measurements to severity of hypertension. Finally, we compared the relative sensitivity of the echocardiogram, the standard 12-lead ECG, and the routine chest x-ray for detecting cardiac abnormalities in hypertensive subjects.

Materials and Methods

Subjects

Two hundred sixty hypertensive subjects and 124 normotensive subjects gave informed consent and were studied by echocardiography. The hypertensive subjects were selected from the outpatient hypertension clinic at the New York Hospital — Cornell Medical Center. The following patients were excluded: 1) patients with malignant hypertension; 2) patients on antihypertensive medications other than diuretics, propranolol, clonidine, \(\alpha\)-methyl dopa or guanethidine; and 3) patients with a history of angina pectoris, myocardial infarction, atrial fibrillation or congestive heart failure (i.e., orthopnea or paroxysmal nocturnal dyspnea). Twenty-six of the 260 subjects (10%) were excluded because their echocardiograms were technically unsatisfactory for measurement of the anatomic variables included in the study. The remaining 234 hypertensive subjects included 134 on antihypertensive therapy at the time of the echocardiographic study and 100 who had had therapy discontinued for at least 3 weeks before the study. The selection and evaluation of the normotensive subjects are extensively described in a previous report.\(^4\)

Clinical characteristics of the hypertensive and normotensive subjects are shown in table 1. Blood pressures were measured in the supine position with standard sphygmomanometric methods. Mean arterial pressure was estimated from the sum of the diastolic pressure and one-third of the pulse pressure. Seventy-three of the 134 patients (54%) on antihypertensive therapy and 53 of the 100 patients (53%) not on

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such therapy had diastolic blood pressures lower than 95 mm Hg at the time of echocardiographic study. However, all hypertensive subjects had had diastolic blood pressure $\geq 95$ mm Hg on two or more measurements within 1 year before study. Only two hypertensive subjects had diastolic blood pressures greater than 130 mm Hg at the time of echocardio-graphic study. None of the subjects had evidence of a known secondary cause of hypertension by history, physical examination or routine laboratory tests.

One hundred twenty-eight of the hypertensive subjects had systemic arterial blood pressure recorded (sitting) on two or three clinic visits during the 6 months before echocardiographic study (with no change in treatment status during these visits). The average of these blood pressures was used for assessment of the relation of echocardiographic measurements to severity of hypertension.

### Echocardiographic Measurements

M-mode echocardiograms were performed with the patient on his or her left side. An Aerotech transducer ultrasound receiver, a Honeywell 1856 Line Scan Recorder, a Hewlett-Packard X-Y display, and a custom-built video amplifier were used. Studies were performed with the transducer in the fourth intercostal space near the left sternal edge. If necessary, the transducer was moved laterally and/or to a different interspace so that both mitral leaflets could be visualized with the transducer perpendicular to the chest wall. To obtain consistent and reproducible recordings, the T-scan method was used.\(^8\)

Figure 1 shows illustrative echocardiograms which indicate where measurements were taken. The thickness of the ventricular septum and postero basal left ventricular wall were measured at or slightly below the tips of the mitral valve leaflets. Both thicknesses were measured in the portion of the cardiac cycle that occurs after rapid ventricular filling but before atrial systole (fig. 1A).\(^6\) Postero basal left ventricular wall was measured near the damped portion of the record (fig. 1A). Left ventricular transverse dimensions at end-diastole and at end-systole were measured in the same portion of the record and taken as the maximal and minimal distances between septum and posterior left ventricular wall, respectively (fig. 1A).\(^7\) Left atrial dimension was measured as the maximal distance between the posterior aortic root wall and the posterior left atrial wall. Measurements were taken in the damped portion of the record when the ultrasonic beam passed through the aortic valve leaflets.\(^7\) Aortic root dimension was measured in the same portion of the recording. This measurement was made from the midpoint of the line denoting the anterior aortic root wall to the midpoint of the line denoting the posterior aortic root wall at end-diastole. The mitral valve E-F slope, corresponding to the rate of early diastolic closure of the anterior leaflet of the mitral valve, was measured at a point in which the excursion of the anterior mitral leaflet was maximal and both leaflets were visualized (fig. 1B).\(^10\)

Values derived from the measurements included estimated left ventricular mass,\(^11\) percent fractional shortening of the left ventricular transverse dimension,\(^12\) and left ventricular ejection fraction, which was calculated using the cubed assumption to estimate left ventricular volume.\(^13\)

### Electrocardiographic Measurements

Two hundred seventeen of the hypertensive subjects had standard 12-lead ECGs within 1 day to 12 weeks of the echocardiographic study (median 1 day). No
subject had a change in treatment status during the time between electrocardiographic and echocardiographic study. The Romhilt-Estes scoring system was used to assess electrocardiographic evidence of left ventricular hypertrophy. A point score of four indicated probable left ventricular hypertrophy and five or more points indicated definite left ventricular hypertrophy.

Chest Roentgenographic Measurements

One hundred sixty-eight of the hypertensive subjects had routine posteroanterior chest x-rays within 1 day to 12 weeks of the echocardiographic study (median 1 day). As with the ECG, no patient had a change in treatment status during the time between the chest x-ray and the echocardiogram. A cardiothoracic ratio > 0.5 was considered evidence of left ventricular enlargement.

Statistical Analyses

Statistical analyses used included standard regression analysis and analysis of covariance. Where appropriate, the t test or the chi square test was used to assess statistical significance.

Results

Effects of Body Surface Area and Age on Echocardiographic Measurements of Hypertensive Subjects

To assess the effect of body surface area on echocardiographic measurements, each hypertensive subject was placed into 1 of 5 decades by age. For each age decade the echocardiographic measurements were plotted vs the previously derived appropriate root function of body surface area. The slopes from these plots were compared with those of similar plots.
derived from normotensive subjects. No significant difference was found between these slopes from hypertensive and normotensive subjects for any of the echocardiographic measurements, including left ventricular transverse dimension at end-diastole and at end-systole, ventricular septal thickness, left ventricular free-wall thickness, left ventricular mass, left atrial and aortic root dimensions, and mitral valve E-F slope. As in normotensive subjects, ejection fraction and percent fractional shortening were the only echocardiographic measurements in the hypertensive subjects which were independent of body surface area.

To assess the effect of age on the echocardiographic measurements of hypertensive subjects, the measurements were each adjusted to a body surface area of 1.8 m² using regression equations derived in our laboratory. These adjusted values were subdivided into 5 decades as before, and the values were averaged. The adjusted values for ventricular septal thickness are plotted vs age in figure 2. As in the study of normal subjects, ventricular septal thickness, free-wall thickness, left atrial dimension and aortic root dimension increased, while mitral valve E-F slope decreased with increasing age. Left ventricular transverse dimension (at end-systole and at end-diastole), ejection fraction and percent fractional shortening showed no significant changes with age. The magnitude of change in each echocardiographic measurement with age is summarized in figure 3. Blood pressure data for each of the five age groups is given in table 2. The changes in echocardiographic measurements with age remained evident after adjustment (using covariate analysis) for differences in blood pressures among the five age groups.

**Figure 2.** Effect of age on ventricular septal thickness in hypertensive and normotensive subjects. Each echocardiographic value was adjusted by regression analysis to a body surface area of 1.8 m². Numbers in parentheses indicate numbers of subjects. In this and all subsequent figures the symbol \( \bar{x} \) = mean \( \pm \) sd.

**Figure 3.** Effect of age on echocardiographic measurements in hypertensive subjects. Mean echocardiographic values for each age decade is compared with that of hypertensive subjects in the 21-30 age group after adjustment of echocardiographic values to a body surface area of 1.8 m². *For mitral valve E-F slope \( n = 227 \).

**Table 2. Blood Pressure Data of Hypertensive Subjects in Each Age Group**

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Number of subjects</th>
<th>Blood pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Systolic*</td>
</tr>
<tr>
<td>21-30</td>
<td>29</td>
<td>137 ± 12</td>
</tr>
<tr>
<td>31-40</td>
<td>29</td>
<td>138 ± 20</td>
</tr>
<tr>
<td>41-50</td>
<td>64</td>
<td>143 ± 14</td>
</tr>
<tr>
<td>51-60</td>
<td>88</td>
<td>150 ± 19†</td>
</tr>
<tr>
<td>61-70</td>
<td>24</td>
<td>167 ± 19‡</td>
</tr>
</tbody>
</table>

*Mean ± sd.
†Mean systolic blood pressure was significantly higher than that of each of the three younger age groups (\( p < 0.01 \)).
‡Mean systolic blood pressure was significantly higher than that of each of the four younger age groups (\( p < 0.001 \)).
§Mean diastolic blood pressure was significantly lower than those of the subjects aged 41 to 60 (\( p < 0.02 \) but was similar to subjects in the other age groups (\( p > 0.05 \)).
Figure 4. Distribution of absolute echocardiographic measurements of ventricular septum, left ventricular free wall, mitral valve E-F slope, and derived left ventricular mass in 234 hypertensive subjects. On this and all subsequent figures: open circles = patients on no medication, closed circles = patients on antihypertensive medication other than propranolol, and closed triangles = patients on propranolol with or without other antihypertensive medication. LV = left ventricular. *For mitral valve E-F slope n = 227.

Figure 5. Distribution of absolute echocardiographic measurements of left ventricular dimensions at end-diastole and end-systole, and left atrial and aortic root dimensions in 234 hypertensive subjects. LVTD_D = left ventricular transverse dimension at end-diastole; LVTD_S = left ventricular transverse dimension at end-systole.
Prevalence of Anatomic and Functional Echocardiographic Abnormalities in Hypertensive Subjects

Figures 4, 5 and 6 show the distribution of the absolute echocardiographic measurements of the hypertensive subjects. Since the frequencies of echocardiographic abnormalities in subjects who remained on therapy, including the subgroup on propranolol, was similar to that of subjects who had therapy discontinued, we considered their data together. The effect of hypertension on the various echocardiographic measurements was assessed by calculating the ratio of the actual measurement to the predicted value for each hypertensive subject. The predicted value was calculated using our previously derived regression equations* which account for the effects of both age and body surface area. Figures 7 and 8 show the distribution of these ratios for each of the echocardiographic measurements of the hypertensive subjects. Table 3 summarizes the percentage of echocardiographic abnormalities that fall above (or below for ejection fraction, percent fractional shortening and mitral valve E-F slope) the 95% prediction interval derived from normotensive subjects.

<table>
<thead>
<tr>
<th>Echocardiographic measurement</th>
<th>Percent of patients*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular septal thickness</td>
<td>50</td>
</tr>
<tr>
<td>Left ventricular free-wall thickness</td>
<td>61</td>
</tr>
<tr>
<td>Disproportionate septal thickening</td>
<td>4</td>
</tr>
<tr>
<td>Left ventricular mass</td>
<td>51</td>
</tr>
<tr>
<td>Left ventricular transverse dimension at end-diastole</td>
<td>5</td>
</tr>
<tr>
<td>Left ventricular transverse dimension at end-systole</td>
<td>12</td>
</tr>
<tr>
<td>Left atrial dimension</td>
<td>5</td>
</tr>
<tr>
<td>Aortic root dimension</td>
<td>7</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>15</td>
</tr>
<tr>
<td>Percent fractional shortening</td>
<td>13</td>
</tr>
<tr>
<td>Mitral valve E-F slope†</td>
<td>6</td>
</tr>
</tbody>
</table>

*Echocardiographic values were considered abnormal if they were above (or below for ejection fraction, percent fractional shortening and mitral valve E-F slope) the 95% prediction interval derived from normotensive subjects.
†Disproportionate septal thickening = ratio of ventricular septal thickness to free-wall thickness ≥ 1.3.
‡The number of subjects who had mitral valve E-F slope measured was 227.
FIGURE 7. Distribution of echocardiographic ventricular septal thickness, left ventricular free-wall thickness, left ventricular mass, and mitral valve E-F slope in 234 hypertensive subjects. Each value is plotted as a percentage of the predicted value determined from normal data. Shaded areas represent the 95% prediction interval derived from normal data. *n = 227 for mitral valve E-F slope.

FIGURE 8. Distribution of echocardiographic left ventricular transverse dimension at end-diastole (LVTD₀) and at end-systole (LVTDₛ), and left atrial and aortic root dimensions in 234 hypertensive subjects. Each value is plotted as a percentage of the predicted value determined from normal data. Shaded areas represent the 95% prediction interval derived from normal data.
Left ventricular enlargement was detected by chest x-ray in nine of 168 hypertensive subjects (5%) who had recent chest x-rays. Six of the nine subjects (67%) had increased left ventricular wall thickness and increased left ventricular mass by echocardiogram. Left ventricular enlargement was detected by chest x-ray in only six of 81 hypertensive subjects (7%) who had left ventricular enlargement (at end-diastole) or increased left ventricular mass by echocardiogram, or both.

### Discussion

Previous studies of normotensive subjects without clinically apparent heart disease have shown changes in echocardiographic measurements with increasing age and body surface area. The changes associated with increasing age include increased aortic root and left atrial size, increased left ventricular wall thickness, and decreased mitral valve E-F slope. In the present study echocardiographic measurements of hypertensive subjects showed changes with increasing age and body surface area similar in direction and degree to those seen in normotensive subjects. The magnitude of these changes suggests that the effects of both age and body surface area should be accounted for in any attempt to assess the effects of hypertension on echocardiographic measurements. This was accomplished in the present study by using the ratio of actual to predicted values. These predicted values were based on previously derived regression equations from normal subjects.

Using this approach, structural abnormalities of the heart were detected in more than 60% of our population of subjects with mild-to-moderate hypertension. The most frequent abnormalities were ventricular septal and left ventricular free-wall thickening and increased left ventricular mass. Similarly, Schlant et al. found increased left ventricular mass by echocardiography in 36 of 73 (49%) of their hypertensive subjects. The mean values and scatter of the data of left atrial, aortic root, end-diastolic and end-systolic left ventricular dimensions for our hypertensive population were similar to those found for the normotensive population. If published criteria for left atrial enlargement (even those adjusted for body surface area) were used for our hypertensive population, the prevalence of left atrial enlargement would have been three times as high as that found after accounting for both age and body surface area (i.e., 15% rather than 5%). Thus, left atrial enlargement was uncommon in our hypertensive subjects, whether or not they had left ventricular hypertrophy, and did not appear to be an early indicator of cardiac involvement in hypertensive disease, as we previously thought. Dunn et al. found larger echocardiographic left atrial dimensions in hypertensive subjects than in normal subjects. However, the hypertensive subjects in their study were older than the normal subjects. Thus, the larger left atrial dimensions might at least partially be explained by changes which normally occur with age, rather than by the effects of hypertension.

In our study, modest but statistically significant

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**Table 4. Correlation Coefficients of Blood Pressure vs Echocardiographic Measurements in 128 Hypertensive Subjects**

<table>
<thead>
<tr>
<th></th>
<th>Systolic blood pressure</th>
<th>Diastolic blood pressure</th>
<th>Mean arterial pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ventricular septal thickness</strong></td>
<td>0.209*</td>
<td>0.346†</td>
<td>0.314‡</td>
</tr>
<tr>
<td><strong>Left ventricular free-wall thickness</strong></td>
<td>0.291†</td>
<td>0.468‡</td>
<td>0.430‡</td>
</tr>
<tr>
<td><strong>Left ventricular mass</strong></td>
<td>0.231†</td>
<td>0.319‡</td>
<td>0.311‡</td>
</tr>
<tr>
<td><strong>Left ventricular transverse dimension at end-diastole</strong></td>
<td>0.022</td>
<td>-0.026</td>
<td>-0.003</td>
</tr>
<tr>
<td><strong>Left ventricular transverse dimension at end-systole</strong></td>
<td>-0.083</td>
<td>0.010</td>
<td>-0.040</td>
</tr>
<tr>
<td><strong>Left atrial dimension</strong></td>
<td>0.204*</td>
<td>0.166</td>
<td>0.208*</td>
</tr>
<tr>
<td><strong>Aortic root dimension</strong></td>
<td>-0.082</td>
<td>0.010</td>
<td>-0.040</td>
</tr>
<tr>
<td><strong>Ejection fraction</strong></td>
<td>0.120</td>
<td>-0.029</td>
<td>0.050</td>
</tr>
<tr>
<td><strong>Percent fractional shortening</strong></td>
<td>0.136</td>
<td>-0.032</td>
<td>0.058</td>
</tr>
<tr>
<td><strong>Mitrval valve E-F slope</strong></td>
<td>-0.001</td>
<td>-0.064</td>
<td>-0.037</td>
</tr>
</tbody>
</table>

* *p < 0.05.
† p < 0.01.
‡ p < 0.001.

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**Relation of Echocardiographic Measurements to Severity of Hypertension**

One hundred twenty-eight of the hypertensive subjects had systemic arterial blood pressure recorded on two or three clinic visits during the 6 months before echocardiographic study (with no change in treatment status during these clinic visits). There was only a modest (but statistically significant) correlation between the averaged mean arterial pressures recorded during these visits and the ratios of actual to predicted values for ventricular septal thickness, left ventricular free-wall thickness, left atrial dimension and left ventricular mass (table 4). No significant correlations were found between mean arterial blood pressure and aortic root dimension, left ventricular transverse dimension (at end-systole or end-diastole), left ventricular ejection fraction or mitral valve E-F slope.

**Comparison of the Echocardiogram with the 12-Lead Electrocardiogram and the Chest X-ray**

The echocardiogram was compared with both the standard 12-lead ECG and the routine chest x-ray for detecting cardiac abnormalities in hypertensive subjects. Probable or definite left ventricular hypertrophy was detected by ECG in seven of the 217 hypertensive subjects (3%) who had recent ECGs. Six of these seven subjects (86%) had increased left ventricular mass by echocardiogram and the seventh had an echocardiographic left ventricular mass at the upper limit of normal (as well as increased left ventricular free-wall thickness). Probable or definite left ventricular hypertrophy by ECG was detected in only six of 108 hypertensive subjects (6%) who had increased left ventricular mass by echocardiogram.
correlations were found between systemic arterial blood pressure and left ventricular wall thicknesses, left ventricular mass and left atrial dimension. Schlant et al. found poorer correlations between left ventricular wall thicknesses and diastolic pressure than those found in the present study. However, their series was smaller than ours. In addition, they correlated left ventricular wall thicknesses with a single diastolic pressure measurement, while two or more measurements from separate clinic visits were used in the present study.

Disproportionate septal hypertrophy has been reported in 47%, 10%, 19%, 10%, and less than 1% of subjects with hypertension. We found this abnormality in 4% of our hypertensive subjects (nine of 234). Exclusion of subjects with angina pectoris, evidence of myocardial infarction and severe hypertension from our study population may have lowered the prevalence of disproportionate septal thickening. However, it would appear that this abnormality is uncommon in subjects with mild-to-moderate hypertension.

We did not study the relatives of patients who had disproportionate septal thickening and, therefore, do not know whether this abnormality represents genetically transmitted hypertrophic cardiomyopathy (i.e., asymmetric septal hypertrophy), is an atypical form of left ventricular wall thickening secondary to hypertension, or neither. Of note, a recent necropsy study of hypertensive subjects from our laboratory showed that the disproportionate septal thickening present in two patients with hypertension did not have the features characteristic of genetic hypertrophic cardiomyopathy.

Left ventricular systolic function (as assessed by ejection fraction) was reduced in 35 of the 234 hypertensive subjects (15%) in the present study, and in two subjects ejection fraction was considerably reduced (23% and 35%). This was found despite our exclusion of subjects with severe hypertension. Two other echocardiographic studies, which included subjects with more severe hypertension, showed that mean left ventricular ejection fraction diminished as the severity of hypertension increased. 

Left ventricular diastolic function (as measured by mitral valve E-F slope) was below the lower limit of normal in only 14 of the 227 hypertensive subjects (6%), but overall was shifted toward low-normal values so that the mean E-F slope of the hypertensive population was significantly below that of the normotensive population.

The echocardiogram appears to be superior to the routine chest x-ray and the standard 12-lead ECG for detecting cardiac abnormalities in hypertensive subjects. Thus, while the echocardiogram identified an increase in estimated left ventricular mass in over 50% of the patients, the chest x-ray and the ECG identified left ventricular enlargement or hypertrophy in less than 10%. Echocardiograms of sufficient quality to make all measurements could not be obtained in some subjects. More important, ECGs and chest x-rays may convey different information than echocardiograms, and therefore, echocardiograms should be used in conjunction with them, rather than instead of them.

These findings are consistent with those of Schlant et al. and Pisarczyk and Ross.

In summary, echocardiography identifies anatomic and functional cardiac abnormalities in a large percentage of asymptomatic hypertensive subjects before abnormalities are detected by ECG or chest x-ray. This conclusion is strengthened by correcting the echocardiographic values for effects of body surface area and age. Whether echocardiography should be used for routine screening of such subjects will depend partly on the prognostic significance of these abnormalities.

Acknowledgments

The authors gratefully acknowledge the excellent technical assistance of Joyce McKay and Cora Barn in obtaining the echocardiograms. The assistance of Pamela Peters and Rose Aceto in the logistics of evaluating the large number of patients is greatly appreciated. We also acknowledge Erica Brittain for help with the statistical analyses.

References

Carotid Baroreflex Function in Young Men with Borderline Blood Pressure Elevation

DWAIN L. ECKBERG, M.D.

SUMMARY Carotid baroreflex function was assessed in 10 normotensive young men and 20 age-matched subjects with borderline hypertension (successive blood pressures above and below 140/90 mm Hg) by measuring sinus node responses to brief neck suction. Subjects with borderline hypertension were divided into two equal groups according to their average systolic arterial pressures. Baroreflex responses were reset to function at higher pressure levels than normal in subjects with mild borderline hypertension, but reflex sensitivity was normal. Responses were also reset in subjects with more severe borderline hypertension, but reflex sensitivity was subnormal. The results suggest that a gradation of baroreflex responsiveness exists among patients classified as having borderline hypertension: Subnormal responsiveness was found in those subjects whose resting average systolic arterial pressure was ≥ 140 mm Hg.

SOON AFTER THE CAROTID ARTERIAL BAROREFLEX was discovered, Koch and Mies1 and Volhard2 suggested that defective baroreflex buffering of blood pressure might cause essential hypertension. Despite numerous subsequent attempts to delineate hypertensive mechanisms, the role of the arterial baroreflex in the pathogenesis of hypertension remains an enigma. The validity of the theory of Koch and Mies and Volhard has been questioned,3 but the theory has not been discredited altogether. The study of Bristow and co-workers4 supports their postulate: In their study arterial baroreceptor-cardiac reflex responses were found to be strikingly depressed in patients with moderate (average mean arterial pressure 123 mm Hg), sustained hypertension. In these patients, baroreceptor reflex malfunction might have contributed to the development of hypertension, or it might have been a consequence of hypertension.

If a defective baroreceptor reflex mechanism causes hypertension, subnormal baroreflex responses should be found in patients with mild degrees of blood pressure elevation. Data on this issue are conflicting. Takeshita and associates5 found subnormal baroreflex responses in young men whose average blood pressure was 160/82 mm Hg, but Julius6 found normal responses in borderline hypertensive patients whose blood pressures were reported to be lower than those studied by Takeshita and co-workers. I have attempted to clarify this issue by using new techniques to measure baroreflex responses of asymptomatic young men whose blood pressures oscillate above and below 140/90 mm Hg.

Methods

Seven intensities of neck suction were delivered briefly to stretch carotid baroreceptors of young men with normal blood pressures and young men with borderline hypertension (defined as blood pressures above and below 140/90 mm Hg on successive examinations). Sinus node responses were measured.

Hypertensive and Normal Volunteers

Volunteers, ages 19–25 years, were recruited from 900 university students whose blood pressures were measured during registration. Blood pressures of each volunteer were measured two to four (average 2.85) times with subjects in the sitting position, after 10 minutes of rest. Weight, height and skin fold thickness, measured in the midline midway between the chin and the upper margin of the thyroid cartilage were also measured.
Echocardiographic assessment of cardiac anatomy and function in hypertensive subjects.
D D Savage, J I Drayer, W L Henry, E C Mathews, Jr, J H Ware, J M Gardin, E R Cohen, S E Epstein and J H Laragh

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