Is White Coat Hypertension Innocent?
Structure and Function of the Heart in the Elderly

Iwao Kuwajima, Yasuko Suzuki, Akiko Fujisawa, Kizuku Kuramoto

To evaluate the morphological and functional characteristics of the heart in elderly patients with white coat hypertension, we performed an echocardiographic study in 67 elderly individuals older than 60 years: 17 patients with white coat hypertension, 34 patients with true hypertension, and 16 normotensive control subjects. White coat hypertension was defined as a mean 24-hour ambulatory systolic blood pressure of less than 140 mm Hg associated with office hypertension. Cardiac responses to an isometric handgrip exercise test were used to evaluate left ventricular functional reserve. Left atrial dimension and left ventricular mass index were significantly greater in the white coat hypertension group than in the normotension group (P<.05) but were similar to values in the true hypertension group. Left ventricular diastolic function, expressed by peak late-early filling ratio of diastolic mitral flow, showed increasing impairment in the order of the normotension, white coat hypertension, and true hypertension groups (analysis of variance, P<.05); the ratio in the white coat hypertension group tended to be higher than that in the normotension group (unpaired t test, P=.054). The relation between fractional shortening and end-systolic stress did not shift downward after handgrip exercise in the white coat hypertension group, indicating that functional reserve in the left ventricle was maintained. Thus, patients with white coat hypertension had a moderately increased left atrial dimension and left ventricular mass in association with a tendency for disturbed diastolic function, although systolic functional reserve remained the same. These findings suggest that white coat hypertension in the elderly may not be innocent. (Hypertension. 1993;22:826-831.)

KEY WORDS • hypertension, white coat • hypertrophy, left ventricular • blood pressure monitoring, ambulatory • aged

Since the development of devices for invasive and noninvasive ambulatory blood pressure (BP) monitoring, much information has been obtained concerning BP variability. One of the results of this new technology is that white coat hypertension has been identified, and its prevalence and mechanisms have been studied by many investigators. For the clinician, perhaps one of the most important applications of ambulatory recording techniques is the detection of white coat hypertension, which can be defined as an elevated clinic BP in association with a normal 24-hour or daytime ambulatory BP. The prevalence of white coat hypertension has been reported to range from 21% to 56% among hypertensive patients depending on the cutoff point used. Despite the wide prevalence and important clinical implications of white coat hypertension, only a few studies are available with respect to the prognosis or severity of target-organ damage in such patients. Accordingly, it remains unclear whether or not white coat hypertension should be treated like sustained hypertension.

Recent reports indicate that white coat hypertension is common not only in the younger population but also among the elderly. A high prevalence in the elderly is understandable because of the high variability of BP in that age group due to diminished baroreceptor reflex function caused by arterial sclerotic change. The purpose of this study was to compare the morphological and functional characteristics of the heart in elderly individuals with white coat hypertension and sustained hypertension, as well as normotensive elderly control subjects, in order to determine whether white coat hypertension causes target-organ damage.

Methods

Fifty-one patients with essential hypertension and 16 normotensive individuals older than 60 years were recruited for the study. Office hypertension was defined as a systolic BP of 160 mm Hg or more, a diastolic BP of 90 mm Hg or more, or both. Office BP measurements were recorded on two separate visits, excluding the patient's first visit. All BP measurements in the office were performed by physicians. The hypertensive patients either had never been treated or had been withdrawn from treatment at least 4 weeks before the present study. Patients with diabetes mellitus, autonomic neuropathy, or cerebrovascular disease that might affect the circadian BP pattern were excluded from the study on the basis of physical examination and routine laboratory tests. Patients with congestive heart failure, valvular heart disease, or ischemic heart disease were also excluded by cardiac examinations including a treadmill exercise test. Only subjects with high-quality echocar-
Ambulatory Blood Pressure Monitoring was obtained by the oscillometric method. BP and pulse rate data for the whole day were divided into daytime (6 AM to 9 PM) and nighttime (9:30 PM to 5:30 AM) for analysis. Subjects who had a consecutive measurement deficit of more than four points in the ambulatory BP record were excluded from the study.

Hypertensive patients were divided into two groups according to 24-hour systolic BP: 17 patients with an average 24-hour systolic BP of less than 140 mm Hg were classified as the white coat hypertension group, and 34 patients with a 24-hour systolic BP of 140 mm Hg or higher were classified as the true hypertension group. These criteria were based on the 90th percentile values for a normal elderly population as determined in our previous study.

Echocardiography was done within 2 weeks of ambulatory BP monitoring in all patients. The sector-guided M-mode echocardiograms were recorded using a 2.5-MHz transducer (Hewlett-Packard Co, Andover, Mass) in the left lateral decubitus position via the left parasternal window. Two blinded investigators coded echocardiographic recordings and made measurements in random order without knowledge of BP or other findings.

Left atrial dimension, septal and posterior wall thicknesses, and left ventricular (LV) internal dimension at systole and diastole were measured according to the recommendations of the American Society of Echocardiography. Wall thickness was defined as the sum of the septal wall thickness (IVST) and posterior wall thickness (PWT) at end diastole. Relative wall thickness, which indicates the degree of concentric hypertrophy, was expressed as (Wall Thickness) / (LV Internal Dimension).

The relation between LV fractional shortening and the end-systolic LV meridional wall stress (ESS) was determined to evaluate LV contractility during a stress test. ESS was calculated from the echocardiographic dimension and the BP obtained at the time of echocardiography using the formula of Reichek et al.11 A pulsed-wave Doppler unit at a frequency of 2.5 MHz was used for the assessment of LV diastolic function. Transmural flow
TABLE 2. Echocardiographic Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>NC (n=16)</th>
<th>WC (n=17)</th>
<th>TH (n=34)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP, mm Hg</td>
<td>129.5±13.8</td>
<td>145.7±19.1</td>
<td>160.7±18.6</td>
<td>.009</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>63.0±11.1</td>
<td>75.4±15.3</td>
<td>78.6±13.7</td>
<td>.013</td>
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<tr>
<td>Heart rate, bpm</td>
<td>67.3±11.0</td>
<td>69.8±6.9</td>
<td>68.0±10.0</td>
<td>NS</td>
</tr>
<tr>
<td>LAD, mm</td>
<td>33.1±5.1</td>
<td>36.4±4.6</td>
<td>38.5±4.8</td>
<td>.048</td>
</tr>
<tr>
<td>LVDd, mm</td>
<td>42.6±4.9</td>
<td>44.2±5.9</td>
<td>43.1±4.7</td>
<td>.630</td>
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<tr>
<td>LVDs, mm</td>
<td>28.1±14.8</td>
<td>26.4±6.8</td>
<td>24.7±4.6</td>
<td>NS</td>
</tr>
<tr>
<td>WT, mm</td>
<td>17.4±20</td>
<td>19.6±4.5</td>
<td>22.3±4.8</td>
<td>NS</td>
</tr>
<tr>
<td>Relative WT, %</td>
<td>41.6±7.5</td>
<td>45.5±13.7</td>
<td>53.1±16.0</td>
<td>NS</td>
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<tr>
<td>LVMI, g/m²</td>
<td>90.7±16.3</td>
<td>119.3±39.8</td>
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<td>.012</td>
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<tr>
<td>LVFS, %</td>
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<td>40.9±8.9</td>
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<td>NS</td>
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<tr>
<td>ESS, dyne/cm²</td>
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<td>69.8±43.9</td>
<td>60.3±26.0</td>
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<tr>
<td>E, cm/s</td>
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<td>39.6±8.0</td>
<td>40.3±11.0</td>
<td>NS</td>
</tr>
<tr>
<td>A, cm/s</td>
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<td>57.6±10.4</td>
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<td>NS</td>
</tr>
<tr>
<td>A/E ratio</td>
<td>1.31±0.23</td>
<td>1.50±0.35</td>
<td>1.65±0.44</td>
<td>NS</td>
</tr>
</tbody>
</table>

NC indicates normotensive control group; WC, white coat hypertension group; TH, true hypertension group; SBP, systolic blood pressure; DBP, diastolic blood pressure; bpm, beats per minute; LAD, left atrial dimension; LVDd, left ventricular end-diastolic dimension; WT, wall thickness; LVMI, left ventricular mass index; LVFS, left ventricular fractional shortening; ESS, left ventricular meridional end-systolic wall stress; E, early diastolic flow velocity; and A, peak flow velocity during atrial contraction.

Results

Clinical Background Data and Office Blood Pressure

Age, body surface area, and serum creatinine did not differ among the three groups (Table 1). Office BP was higher in the hypertensive groups than in the normotensive group, but there was no significant difference between the two hypertensive groups with regard to both systolic and diastolic BP. Hypertension was defined as a systolic BP greater than or equal to 160 mm Hg or a diastolic BP of greater than or equal to 90 mm Hg at enrollment. However, no hypertensive patient whose systolic BP was less than 160 mm Hg was observed in the present study. Consequently, all patients satisfied the systolic criteria of greater than or equal to 160 mm Hg.

Ambulatory Blood Pressure

The whole-day systolic and diastolic BP values were significantly higher in the hypertensive groups than in the normotensive group (Table 1). In addition, both systolic and diastolic BPs were significantly higher in the true hypertension group than in the white coat hypertension group. Daytime systolic BP was significantly higher in the true hypertension group than in the white coat hypertension or normotension groups, and a difference was also observed between the white coat hypertension and normotension groups. Nighttime systolic and diastolic BPs were significantly higher in the
true hypertension group than in the white coat hypertension or normotension groups, but there was no significant difference in systolic BP between the white coat hypertension and normotension groups.

**Structural Characteristics of the Heart**

Left atrial dimension was significantly larger in the two hypertensive groups than in the normotension group (Table 2, P<.05), although no difference was seen between the two hypertensive groups. Both LVDD and LVDs were similar in all three groups. Wall thickness in the true hypertension group was greater than in the normotension group but was similar to that in the white coat hypertension group. LV mass index was significantly greater in the white coat hypertension and true hypertension groups than in the normotension group (Fig 1, P<.05), but there was no difference between the two hypertensive groups.

**Left Ventricular Function at Rest**

Fractional shortening, an index of LV systolic function, was similar in all three groups (Table 2). As shown in Fig 2, the A/E ratio, which represents LV diastolic function, tended to be higher in the white coat hypertension group than the normotension group (P=.054) and tended to be lower than in the true hypertension group (P=.062), although the differences did not reach statistical significance.

**Left Ventricular Function After Handgrip Exercise**

Table 3 shows the changes in hemodynamic and echocardiographic parameters during handgrip exercise. The increase in BP and heart rate were comparable among the three groups. The increase in fractional shortening in the white coat hypertension group was comparable to that in the normotension group but was higher than that in the true hypertension group. Fig 3 summarizes the relation between ESS and LV systolic functional reserve against increased afterload. In contrast, the white coat hypertension and normotension groups showed a shift to the right in accordance with the increase in ESS and without any reduction of fractional shortening, suggesting that LV systolic function was better preserved in these groups.

**Discussion**

The present study demonstrated that white coat hypertension in the elderly was associated with greater
LV mass than in subjects with normotension but was less than in patients with true hypertension. Moreover, it was associated with a tendency for disturbed LV diastolic function, although LV systolic function was preserved.

Criteria and Prevalence of White Coat Hypertension

Although it has long been known that BP temporarily increases when a patient enters the doctor's office, little attention was given to this phenomenon until it was reported as "white coat hypertension" by Pickering et al. They defined white coat hypertension as a 24-hour BP within the normal range despite an office hypertension and noted such hypertension in 21% of 292 hypertensive patients. Julius et al reported that white coat hypertension was present in 7.1% of surveyed individuals in Tecumseh, Mich, when it was defined as a normal home BP despite an office BP greater than 140/90 mm Hg. Floras et al considered that white coat hypertension existed when the difference between office and ambulatory BP values was more than 10 mm Hg and found it in 54.2% of their hypertensive patients. These differences in prevalence may depend on a variety of factors, such as the nature of the population studied and the way in which white coat hypertension is defined.

Although Pickering et al noted that white coat hypertension was more likely to occur in younger, female, nonobese patients with recently diagnosed hypertension, it has since been found to be common in older patients, as noted by Sokolow and colleagues and our recent study. It is conceivable that an increase in BP variability in elderly hypertensive patients due to arterial sclerosis makes it more likely for a rise in office BP to occur. Thus, it was not unexpected to obtain a prevalence of 33.3% for white coat hypertension among elderly hypertensive patients in the present study. Recently, we monitored the BP of 47 hypertensive patients at 5-minute intervals 30 minutes before a patient saw a doctor until 10 minutes after the patient left the office. We noted that white coat hypertension was seen in 59.6% of the patients if a difference in BP greater than or equal to 10 mm Hg was adopted for both in-office and out-of-office readings. In elderly patients, it seems reasonable to define white coat hypertension as a systolic BP less than 140 mm Hg combined with office hypertension, because systolic BP rises with advancing age even in the normotensive population.

Although white coat hypertension is known to be common in hypertensive patients, its prognosis and the risk of cardiovascular morbidity have remained undetermined. Some investigators have indicated that patients with white coat hypertension may be at less risk compared with those with sustained hypertension. White and colleagues showed that at the same level of office BP, patients with white coat hypertension had less LV hypertrophy and better LV function than those with sustained hypertension. They also reported that the left atrial size and LV mass were significantly less in patients with white coat hypertension than in those with daytime hypertension and were not statistically different from the value in normotensive subjects. Their study was partially in agreement with our results in that they noted cardiac morphological changes in white coat hypertension to be less than those in true hypertension. However, our data showed that these changes were more severe than in normotensive control subjects. The reason for this discrepancy was the difference in the definition of white coat hypertension, although the different ages of the subjects might also have some influence. In the present study, white coat hypertension was defined as an ambulatory systolic BP of less than 140 mm Hg associated with an office BP greater than or equal to 160 mm Hg. Perloff and colleagues also showed that patients whose ambulatory BP was lower than their office BP were at less risk than those in whom it was the same or higher. Thus, both of these other studies are in agreement with our present study demonstrating that white coat hypertension causes less LV hypertrophy and less impairment of diastolic function than true hypertension.

However, the present study clearly demonstrated that patients with white coat hypertension had morphological and functional cardiac impairment when they were compared with normotensive control subjects. Recently, many studies of hypertensive hearts have demonstrated that diastolic dysfunction may precede abnormalities in systolic ventricular performance. Several investigators described that as many as one third of the patients who develop signs and symptoms of congestive heart failure have normal systolic function associated with disturbed diastolic function, although patients with overt symptoms of congestive heart failure were excluded in the present study. It has been known that the impairment of diastolic function is influenced by the age of the patients as well as myocardial hypertrophy. White et al reported that LV filling rate was more dependent on age and BP than left atrial or LV size, and it was impossible to differentiate the effects of hypertension versus aging on diastolic function. However, in the present study the ages of the patients in the three groups were similar, and the effect of aging on LV diastolic function was considered to be similar.
How does white coat hypertension cause LV overload? Some evidence that sporadic elevations of BP may not be innocuous has been obtained from experimental and epidemiologic studies. Julius et al noted that repeated neurogenic pressor episodes elicited by hind-quarter compression in dogs over 9 weeks caused concentric LV hypertrophy that was detectable 3 weeks before sustained hypertension developed. Another line of epidemiologic evidence comes from the study in Tecumseh, in which subjects with white coat hypertension at 32 years of age showed consistently elevated office BP readings in earlier examinations (at 5, 8, 21, and 23 years of age), suggesting that the emotional response to BP measurement is quite reproducible over the long term. Furthermore, Devereux et al have noted that BP during exercise is better correlated with LV mass index than BP at rest. In the present study, the daytime systolic BP was significantly higher in the white coat hypertension group than in the normotension group, although the nighttime systolic BP was similar in both groups. This higher daytime but not nighttime BP in the white coat hypertension group suggests that the temporary increase in BP associated with emotional or physical activity during the daytime might contribute to the development of morphological and functional changes in this group.

**Study Limitations**

For the clear demonstration of the functional contribution of the sporadic elevation in BP to the progression of LV hypertrophy, neurohumoral factors such as catecholamine or plasma renin activity must be determined. Any elevation of these parameters during the daytime in the white coat hypertension group would support a causal relation between office hypertension and target-organ damage.

In summary, the present study suggests that white coat hypertension may not be innocent. A future longitudinal study would be necessary to reveal more clearly how common white coat hypertension is and if it may not be innocent. A future longitudinal study would be necessary to reveal more clearly how common white coat hypertension is and if it may not be innocent.

**References**


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