Postprandial Hypotension: Evaluation by Ambulatory Blood Pressure Monitoring

Katsuhiko Kohara, Kinji Uemura, Yasunori Takata, Takafumi Okura, Yutaka Kitami, and Kunio Hiwada

To elucidate whether postprandial hypotension (PPH) is associated with any diurnal change of blood pressure, ambulatory blood pressure monitoring was performed on 121 hospitalized essential hypertensive patients who received standardized meals. Postprandial change in blood pressure was defined as the difference between mean systolic blood pressure (SBP) 1 h before and 2 h after each meal. The postprandial decline of SBP showed age-dependent augmentation. The degree of PPH was significantly related to the level of preprandial blood pressure for each meal.

Patients were divided into the following three groups according to the mean PPH of three meals: Normal group (n = 79); mean postprandial decline of SBP < 5 mm Hg, PPH-1 group (n = 24); 5 mm Hg ≤ mean PPH < 10 mm Hg, and PPH-2 group (n = 18); PPH ≥ 10 mm Hg. There was no difference in 24-h, nighttime, or daytime blood pressure among the three groups. The prevalence of dipper and nondipper patients was not different among the three groups. However, patients in PPH-2 showed significantly greater daytime and 24-h blood pressure variability. Furthermore, there was a significant positive relationship between the morning surge of SBP and PPH after breakfast (r = 0.36, P < .001).


KEY WORDS: Postprandial hypotension, ambulatory blood pressure monitoring, nondipper, morning surge, variability.
urnal change in blood pressure in essential hypertensive patients, with special emphasis on dipper/non-dipper phenomena and morning surge. It is well demonstrated that the time and content of meals significantly influence PPH.\textsuperscript{1} Daily activity including the wakening and sleeping time could also influence diurnal change in blood pressure. To minimize these effects, the present study was performed on hospitalized essential hypertensive patients with standardized daily activity including meals.

**METHOD**

A total of 121 hospitalized essential hypertensive patients (mean age, 57 ± 14 years, 65 men and 56 women) participated in the study. They were recruited from consecutive cases who were admitted to the Second Department of Internal Medicine, Ehime University Hospital for the evaluation of hypertension from January 1995 to December 1997. Patients with autonomic nervous dysfunction, congestive heart failure, previous myocardial infarction, or history of symptomatic cerebrovascular accident were excluded from the study. Patients received a diet containing 7 g (120 mmol) NaCl/day, and their daily activities including the times of meals were controlled uniformly throughout the hospitalization period. All medications were discontinued on admission. Patients had either never been treated or had received no medication for ≥1 week at the time of the study. Informed consent to the procedures was obtained from each patient.

Total caloric intake for the patients was 30 kcal/kg ideal body weight/day. The dietary composition was 66% carbohydrate, 16% protein, and 18% fat for each meal times were 8:00 to 8:30 am for breakfast, noon to 12:30 for lunch, and 6:00 to 6:30 pm for dinner. Snacks and drinks, including coffee after meals, were prohibited.

**Twenty-Four-Hour Blood Pressure Determination**

Twenty-four-hour blood pressure was measured by cuff-oscillometric method using TM-2421 (A/D, Ltd., Tokyo, Japan). Blood pressure was measured every 30 min from 6:00 AM to 10:00 PM and every 60 min from 10:00 PM to 6:00 AM on the following day.\textsuperscript{5} Daytime blood pressure and nighttime blood pressure were obtained as the average values during the awake period between 6:00 AM and 10:00 PM and during the sleep period between 10:00 PM and 6:00 AM, respectively.\textsuperscript{5} Patients were allowed to move freely but were asked to sit quietly on the bed or chair during blood pressure measurement. The wakening time, time of falling asleep, and quality of sleep were assessed by interview in each patient. The following parameters were obtained for each patient.

**Postprandial Hypotension**

In the present study, PPH was defined as the difference between mean systolic blood pressure (SBP) 1 h before and 2 h after the meal. The values of blood pressure during the meal were excluded to avoid the influence of postural or eating-related change in blood pressure.

The patients were divided into the following three groups according to the mean postprandial change in SBP for three meals: normal group (n = 79); mean postprandial decline of SBP < 5 mm Hg, PPH-1 group (n = 24); 5 mm Hg ≤ mean PPH < 10 mm Hg, and PPH-2 group (n = 18); PPH ≥ 10 mm Hg (n = 18).

**Dipper and Nondipper Phenomena**

Patients with nocturnal fall of SBP ≥ 20%, 10% to 20%, and < 10% of daytime SBP were classified as extreme dippers, dippers, and nondippers, respectively.\textsuperscript{10} As only one patient was classified as an extreme dipper, the extreme dipper was included in dippers for further evaluation.

**Morning Surge**

In the present study, the morning surge of blood pressure was defined as the difference between the lowest SBP in the middle of the night (2:00 to 4:00 AM) and the highest SBP in the morning before breakfast (5:00 to 7:30 AM).

**Statistical Analysis**

All values are expressed as mean ± SD if not specified. The difference among groups was evaluated by analysis of variance followed by Duncan’s multiple range test. The difference in the prevalence of dipper/nondipper was analyzed by $\chi^2$ test. A probability of < .05 was defined as significant.

**RESULTS**

**Postprandial Hypotension Analyzed by Ambulatory Blood Pressure Monitoring**

Figure 1 depicts the diurnal change in blood pressure in patients with age ≥ 60 years. A decline in blood pressure in response to the meal was observed for every meal. In all patients, the mean postprandial changes in SBP were −2.8 ± 16.5 mm Hg for breakfast, 1.0 ± 13.7 mm Hg for lunch, and −2.8 ± 15.0 mm Hg for dinner. The postprandial decline of SBP showed age-dependent augmentation; mean PPH of age < 60, 60 to 64, 65 to 70, and ≥ 70 were 1.3 ± 9.7, −1.6 ± 7.8, −4.3 ± 10.9, and −7.0 ± 12.8 mm Hg, respectively (F[3,117] = 4.04, P < .01).

Figure 2 depicts the significant negative correlation between preprandial SBP and postprandial change in SBP after lunch. Significant negative relationships were also observed for breakfast (r = −0.504, P < .0001) and dinner (r = −0.423, P < .0001), indicating that the higher the preprandial blood pressure, the larger the postprandial decline of blood pressure.
Postprandial Hypotension and Diurnal Parameters of Blood Pressure Table 1 summarizes the demographic features and diurnal parameters of blood pressure in the three groups. There was no difference in daytime and nighttime hemodynamic variables, as well as in the prevalence of dippers/nondippers among the three groups. However, blood pressure variability expressed as the standard deviation of SBP was significantly higher in the PPH-2 group in the daytime.

Morning Surge and Postprandial Hypotension Figure 3 illustrates the change in SBP in the morning. In patients with PPH, the wakening-related increase in blood pressure before breakfast was prominent. Figure 4 depicts a significant relationship between the magnitude of morning surge of SBP and the degree of PPH after breakfast.

DISCUSSION

The major findings in the present study were: 1) the degree of PPH significantly increased with age; 2) the degree of PPH showed a significant relationship with the preprandial level of blood pressure; 3) there was no relationship between PPH and nondipper phenomena; 4) PPH contributed significantly to the variability of blood pressure in the daytime; and 5)
the degree of PPH after breakfast was significantly related to the magnitude of the morning surge of blood pressure.

Postprandial hypertension is a frequently observed phenomenon associated with several clinical symptoms including dizziness, falls, syncope, TIA, and angina pectoris. Because, it is a rather common phenomenon in elderly essential hypertensive patients, physicians should be aware of PPH to help patients to avoid meal-related falls and cardiovascular events. Recently, Aronow and Ahn reported in their prospective study that elderly nursing residents with PPH showed a higher incidence of falls, syncope, new coronary events, new stroke, and total mortality. In the present study, using ABPM, we confirmed the earlier observation that PPH is age-dependent, and PPH correlates with preprandial blood pressure.

Ambulatory blood pressure monitoring provides useful information. The JNC VI recommends the use of ABPM in situations including the evaluation of syncope. In the present study, none of the patients had any symptoms suggesting PPH, such as dizziness, lightheadedness or syncope. However, ABPM revealed a significant decline of SBP after every meal, especially in the elderly patients. Although PPH has been shown to be much more common than orthostatic hypotension, only a few studies have ever evaluated PPH with ABPM. One of the reasons is that timing of meals as well as the nutrient composition of meals may influence the detection of PPH. In the present study, ABPM measurement was performed in hospitalized patients with standardized meals provided at regular times, which could allow interindividual comparisons.

FIGURE 3. Change in systolic blood pressure in hospitalized essential hypertensive patients with (circle) and without (open circle) > 10 mm Hg decline of systolic blood pressure after breakfast. The dotted area indicates breakfast. Patients with postprandial hypertension showed a larger increase in awakening-related surge in systolic blood pressure before breakfast. Values of mean ± SEM.

TABLE 1. DEMOGRAPHIC FEATURES AND DIURNAL CHANGE IN BLOOD PRESSURE IN SUBGROUPS

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>PPH-1</th>
<th>PPH-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. (M/F)</td>
<td>79 (44/35)</td>
<td>24 (14/10)</td>
<td>18 (7/11)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>54 ± 14</td>
<td>60 ± 9*</td>
<td>68 ± 8*</td>
</tr>
<tr>
<td>Body mass index (g/m²)</td>
<td>24.6 ± 3.4</td>
<td>25.9 ± 3.4</td>
<td>23.6 ± 3.7*</td>
</tr>
<tr>
<td>Duration of hypertension (years)</td>
<td>9.1 ± 8.9</td>
<td>5.4 ± 4.4</td>
<td>11.3 ± 8.8</td>
</tr>
<tr>
<td>Daytime blood pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>141 ± 15</td>
<td>142 ± 18</td>
<td>146 ± 17</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>84 ± 12</td>
<td>85 ± 11</td>
<td>83 ± 12</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>71 ± 8</td>
<td>68 ± 8</td>
<td>68 ± 8</td>
</tr>
<tr>
<td>Nighttime blood pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>131 ± 16</td>
<td>133 ± 18</td>
<td>139 ± 16</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>77 ± 12</td>
<td>79 ± 12</td>
<td>76 ± 15</td>
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<tr>
<td>Heart rate (beats/min)</td>
<td>60 ± 8</td>
<td>59 ± 9</td>
<td>61 ± 7</td>
</tr>
<tr>
<td>Night/day ratio of SBP</td>
<td>0.93 ± 0.06</td>
<td>0.94 ± 0.05</td>
<td>0.95 ± 0.07</td>
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<tr>
<td>Dipper/nondipper</td>
<td>41/38</td>
<td>11/13</td>
<td>8/10</td>
</tr>
<tr>
<td>Standard deviation of SBP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daytime (mm Hg)</td>
<td>11.5 ± 3.5</td>
<td>12.0 ± 2.5</td>
<td>15.1 ± 3.5*</td>
</tr>
<tr>
<td>Nighttime (mm Hg)</td>
<td>10.4 ± 3.9</td>
<td>11.7 ± 3.3</td>
<td>12.0 ± 4.4</td>
</tr>
<tr>
<td>24-h (mm Hg)</td>
<td>12.7 ± 3.1</td>
<td>12.8 ± 2.4</td>
<td>15.2 ± 3.4*</td>
</tr>
</tbody>
</table>

Values are mean ± SD. SBP, systolic blood pressure; DBP, diastolic blood pressure.

*P < .05 v normal group.
ment of blood pressure does not always catch the nadir of the blood pressure reduction. The body position and physical activity of having a meal also affect blood pressure. To eliminate these possibilities, PPH was defined as the difference between mean SBP 1 h before and 2 h after the meal, and SBP obtained during the meal was not used to determine PPH.

Nondipper hypertensive patients have been shown to be associated with advanced end-organ damage, sodium sensitivity, and higher risk for future cardiovascular events. In a previous study, we demonstrated that autonomic nervous dysfunction is involved in the pathogenesis of nondipper phenomenon in essential hypertensive patients. Although autonomic nervous dysfunction has also been proposed as one of the underlying mechanisms of PPH, we failed to find a direct relationship between PPH and nondipper phenomena in the present study.

The morning surge of blood pressure is one of the mechanisms that could explain the higher incidence of cardiovascular events in the morning. The abrupt increase in endogenous catecholamine release after awakening plays an important role in awakening-related increase in blood pressure. In the present study, we first demonstrated a significant relationship between morning surge of blood pressure and PPH after breakfast. Although the underlying mechanisms of PPH are not fully understood, many mechanisms have been reported. Among them, baroreflex dysfunction could link the morning surge and PPH. Food ingestion induces splanchnic blood pooling, which could be normalized by compensation with the baroreflex. Wakening-related increase in catecholamine release could be buffered by the baroreflex. It is also reported that baroreflex sensitivity was not different between dippers and nondippers. Accordingly, impairment of baroreflex function could induce both PPH and morning surge, with little effect on nondipper phenomena.

Recently, blood pressure variability has been shown to be related to increased morbidity. In the present study, we showed that PPH significantly increase a daytime as well as a 24-h variability of blood pressure. Dipper phenomenon also significantly increased 24-h blood pressure variability independently of PPH (data not shown), in accordance with the report of Imai et al. These findings further support the involvement of the baroreflex dysfunction in PPH, as baroreflex dysfunction has been shown to increase blood pressure variability.

Pickering et al demonstrated that nighttime administration of doxazosin was effective in preventing a morning rise in blood pressure. As early morning administration of a dihydropyridine calcium antagonist has also been shown to be effective in preventing morning surge of blood pressure, attention should be paid to the risk of profound PPH induced by interaction with an antihypertensive drug.

The study limitation is that we did not repeat the blood pressure measurements in the same subjects. Although, we observed the significant association between PPH after breakfast and PPH after dinner (data not shown), the reproducibility of the diurnal change in blood pressure including PPH needs to be evaluated.

In summary, we showed that PPH increased the variability of blood pressure. Although there was no relationship between nondipper phenomenon and

![Image of Figure 4: Postprandial change in systolic blood pressure](image-url)
PPH, we found that the morning surge of blood pressure was significantly related to PPH after breakfast. The awakening-related abrupt increase in blood pressure followed by the meal-related decline may further precipitate cardiovascular events in the morning.

REFERENCES