The Influence of Low-, Normal-, and High-Carbohydrate Meals on Blood Pressure in Elderly Patients With Postprandial Hypotension

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**Background.** Postprandial hypotension (PPH) is a common and serious disorder of blood pressure (BP) regulation in elderly people. It has been suggested that primarily the carbohydrate (CH) content of a meal induces the BP decrease. Therefore, we examined the relationship between the CH content of meals and postprandial BP responses in elderly patients diagnosed with PPH.

**Methods.** Twelve geriatric patients (aged 75 to 91 years; 6 men) who were previously diagnosed with PPH received standardized liquid meals with low- (25 g), normal- (65 g), and high- (125 g) CH content in random order on three separate days. Systolic BP (SBP), diastolic BP, and heart rate were measured every 5 minutes from 20 minutes before until 75 minutes after each meal. Postprandial symptoms were recorded every 15 minutes.

**Results.** The maximum decrease in SBP was significantly smaller after the low-CH meal (28 mm Hg) than after the normal- (39 mm Hg) and high-CH meals (40 mm Hg) (p < .050 between groups). In addition, the duration of PPH was significantly shorter (p < .010), and postprandial symptoms were less frequent and less severe after the low-CH meal.

**Conclusions.** Reducing the CH amount in meals induces significantly smaller decreases in SBP, shorter duration of PPH, and reduction of PPH-related symptoms. Therefore, limiting the CH content of an elderly patient’s meal can be a clinically effective nonpharmacological treatment for PPH in elderly patients and can reduce the risk of developing symptomatic PPH.

**POSTPRANDIAL** reductions in blood pressure (BP) are very common in elderly people and are associated with serious morbidity such as dizziness, falls, and, syncope (1–7).

Postprandial hypotension (PPH) is generally defined as a decrease in systolic blood pressure (SBP) ≥20 mm Hg within 75 minutes of the start of a meal (1). PPH is distinct from orthostatic hypotension (OH) and might occur more often than OH and infrequently together with OH in elderly patients (8). Studies of elderly people living in long-term health care facilities have shown that nearly all elderly subjects experience some decline in SBP after eating a meal, and 24% to 36% of these subjects have a postprandial decline in SBP ≥20 mm Hg (3,9).

The pathophysiology of PPH in elderly people is not fully understood, but several hypotheses have been suggested (1). Factors that might contribute to the occurrence of PPH are impaired cardiovascular compensation, reduced baroreflex sensitivity, impaired activation of the sympathetic nervous system, reduced compensatory peripheral vasoconstriction, and humoral factors such as gut peptides (1,6,10–12). Finally, oral carbohydrate- or glucose-mediated factors are involved (1).

Jansen and colleagues have demonstrated that the postprandial BP response depends on the composition and particularly the carbohydrate (CH) content of the meal ingested (1,13). Oral glucose and the subsequent insulin release primarily cause the BP effects of CHs. Other CHs, such as oral fructose, oral xylose, and intravenous glucose, do not or only minimally affect BP, just like fat and proteins (13–16).

Lowering the CH content in meals by adjusting the size and frequency of meals has been suggested as an important and clinically relevant nonpharmacological treatment of PPH (1). However, studies demonstrating the effect of adjusting the CH amount in meals on postprandial BP responses in elderly patients with PPH have not been reported to date. Therefore, the present study was designed to examine the effect of different amounts of CH in meals on postprandial BP responses in elderly patients with PPH to determine the value of the advice to reduce the CH amount in meals in the treatment of PPH.

**Methods**

**Subjects**

Twelve geriatric patients with previously diagnosed PPH were recruited from the (outpatient) clinic of the Department of Geriatric Medicine of the University Medical Center Nijmegen, the Netherlands. Preset inclusion criteria were age ≥70 years, a diagnosis of PPH, and the possibility of complete medication withdrawal for at least 12 hours. Preset exclusion criteria were acute diseases, insulin-dependent diabetes mellitus, chronic atrial fibrillation, fixed pacemaker, problems with oral food ingestion, and moderate or severe cognitive impairments that would prevent the patient...
POSTPRANDIAL RESPONSES ON CARBOHYDRATES

Table 1. Baseline Clinical Characteristics of 12 Geriatric Patients Diagnosed With Postprandial Hypotension

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>80 ± 1</td>
<td>Systolic blood pressure†</td>
<td>159 ± 9</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>6 / 6</td>
<td>Diastolic blood pressure†</td>
<td>83 ± 3</td>
</tr>
<tr>
<td>Quetelet index, kg/m²</td>
<td>22.3 ± 1.9</td>
<td>Heart rate, hpm†</td>
<td>70 ± 3</td>
</tr>
<tr>
<td>Medical history</td>
<td></td>
<td>Number of medications†</td>
<td>4 ± 1</td>
</tr>
<tr>
<td>Cardiovascular problems</td>
<td>6</td>
<td>Cardiovascular medication</td>
<td>6</td>
</tr>
<tr>
<td>Hypertension</td>
<td>4</td>
<td>ACE-inhibitors</td>
<td>1</td>
</tr>
<tr>
<td>Orthostatic hypotension</td>
<td>6</td>
<td>Beta-blockers</td>
<td>1</td>
</tr>
<tr>
<td>Heart failure</td>
<td>2</td>
<td>Diuretics</td>
<td>2</td>
</tr>
<tr>
<td>Respiratory problems</td>
<td>2</td>
<td>Glycosides</td>
<td>1</td>
</tr>
<tr>
<td>COPD</td>
<td>2</td>
<td>Anticoagulants</td>
<td>5</td>
</tr>
<tr>
<td>Neurological problems</td>
<td>2</td>
<td>Respiratory medication</td>
<td>1</td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td>2</td>
<td>Corticosteroids</td>
<td>1</td>
</tr>
<tr>
<td>Dementia</td>
<td>—</td>
<td>Levodopa/benseraside</td>
<td>1</td>
</tr>
<tr>
<td>Digestive problems</td>
<td>3</td>
<td>Laxatives</td>
<td>3</td>
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<tr>
<td>Constipation</td>
<td>3</td>
<td>Antacids</td>
<td>2</td>
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<tr>
<td>Peptic ulcer disease</td>
<td>2</td>
<td>Psychiatric medication</td>
<td>1</td>
</tr>
<tr>
<td>Psychiatric problems</td>
<td>10</td>
<td>Benzodiazepines</td>
<td>1</td>
</tr>
<tr>
<td>Anxiety</td>
<td>1</td>
<td>Antidepressant drugs</td>
<td>4</td>
</tr>
<tr>
<td>Depression/mood disorders</td>
<td>5</td>
<td>Antipsychotic drugs</td>
<td>1</td>
</tr>
<tr>
<td>Agitation</td>
<td>1</td>
<td>Other agents</td>
<td></td>
</tr>
<tr>
<td>Memory disturbances</td>
<td>3</td>
<td>Analgesics</td>
<td>3</td>
</tr>
<tr>
<td>Urinary problems</td>
<td>3</td>
<td>Vitamin B12</td>
<td>3</td>
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<tr>
<td>Prostate problems</td>
<td>2</td>
<td>Eye drops</td>
<td>2</td>
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<tr>
<td>Urinary incontinence</td>
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<td>Antiemetics</td>
<td>2</td>
</tr>
<tr>
<td>Mobility problems</td>
<td>4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Notes: COPD = chronic obstructive pulmonary disease; ACE = angiotensin-converting enzyme.
†Value is mean ± SEM.

SBP, diastolic BP (DBP), and heart rate (HR) were measured every 5 minutes (Dinamap 1846 SX; Critikon Inc., Tampa, FL) from 20 minutes before until 75 minutes after the start of the meal. Postprandial symptoms and changes in the patients’ baseline condition in relation to BP changes were recorded every 15 minutes during the test. A standardized list with symptoms related to PPH according to the literature was used, and patients were observed continuously by the researcher. Symptoms were scored on a four-point scale: absent, mild, moderate, or severe.

Statistical Analysis

Statistical analysis was performed with SPSS for Windows 8.0 (SPSS Inc, Chicago, IL). A p value <.050 was taken as the level of significance. The results are expressed as mean and standard error of the mean.

Baseline values of SBP, DBP, and HR were defined as the values measured just before the meal ingestion after 20 minutes of rest in a sitting position. Differences in baseline values or maximum changes in BP or HR between the tests were tested by paired t tests. Two-way repeated measures analysis of variance was applied to examine the overall effects of time, CH amount in meals, and the time-by-CH amount interaction on BP changes versus baseline over the three tests. The correlation between BP responses and OH, hypertension, weight, and age were determined by Pearson’s correlation test and stepwise linear regression analysis.

RESULTS

Subject Characteristics

The clinical characteristics of the 12 patients with PPH are presented in Table 1.

Postprandial Changes in SBP, DBP, and HR

Figure 1 shows the group-averaged changes in SBP, DBP, and HR versus baseline after the low-, normal-, and high-CH meals.

After each meal, SBP decreased significantly over time (p < .050). The maximum reduction in SBP was significantly smaller after the low-CH meal (−28 ± 5 mm Hg) compared with the normal- (−39 ± 7 mm Hg) and high-CH meals (−40 ± 5 mm Hg) (p = .043, p = .005 between groups). The SBP responses after the normal- and high-CH meals were similar.

DBP decreased significantly after each meal (p < .050), but a significant difference between the DBP responses after the three different CH meals was present only between the low- and high-CH meals (p = .009).

The HR changes over time were not significant after any of the three meals. Significant differences between the HR responses after the low-, normal-, and high-CH meals were not present either, although HR tended to increase more after the normal-CH meal than after the low-CH meal (p = .065).

Group averages of the maximum individual changes in SBP and group averages of the simultaneous individual changes in DBP and HR are presented in Table 2. Individual DBPs differed significantly, at the maximum individual de-
creases in SBP, between the low- and high-CH meals (p < 0.044). In the low-CH meal, HR differed significantly at the time of maximum SBP decrease compared with the normal-CH amount (p = 0.044).

Duration of Postprandial Hypotension

The total period during which PPH was present was significantly shorter after the low-CH meal (18 ± 6 minutes) than after the normal-CH meal (37 ± 7 minutes) and the high-CH meal (43 ± 6 minutes) (p = 0.003 and p = 0.002 between groups) (Table 2).

Presentation of Symptoms

The postprandial symptoms varied in frequency and severity after the three meals with different CH amounts (Figure 2). After the low-CH meal, five patients had no symptoms, and seven patients felt somewhat sleepy. After the normal-CH meal, only one patient had no symptoms or complaints, whereas seven patients felt sleepy, and some patients noted restlessness, dizziness, or nausea. After the high-CH meal, all but one patient felt very sleepy, and two patients felt dizzy and restless. In addition, even more severe symptoms such as vision changes (n = 2), reduced consciousness (n = 3), and chest pain (n = 1) were noted. These symptoms were related to the hypotensive periods with SBP declines ≥ 20 mm Hg in the patients, although two patients felt sleepy at the end of the test after their SBP had recovered. The chest pain in one patient resolved as the BP returned to normal.

Correlation Between Blood Pressure and Patient Characteristics

Baseline SBP was significantly correlated with the duration of PPH in the low- (r = 0.627, p = 0.029) and normal-CH meals (r = 0.715, p = 0.009) but not in the high-CH meal (r = 0.420, p = 0.174). Significant correlation was also found between hypertension and the duration of PPH in the normal-CH meal (r = 0.702, p = 0.011). No significant correlation was found between OH and the duration of PPH or with declines in SBP, DBP, and HR in all meal sizes.

Discussion

The main finding of this study is that low-CH meals induce significantly smaller SBP declines, significantly shorter periods with SBP declines ≥ 20 mm Hg, and less frequent and less severe symptoms compared with normal- and high-CH meals in elderly patients with PPH.

Although the physiology of postprandial BP responses has not been fully understood, it has been suggested that the

Table 2. Maximum Individual Changes in SBP and Simultaneous Changes in DBP, HR, and Duration of PPH

<table>
<thead>
<tr>
<th>CH Amount</th>
<th>Δ SBP (mm Hg)</th>
<th>Δ DBP (mm Hg)</th>
<th>Δ HR (bpm)</th>
<th>Duration of PPH (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low CH</td>
<td>-28 ± 5*</td>
<td>-13 ± 2†</td>
<td>-2 ± 2‡</td>
<td>18 ± 6‡</td>
</tr>
<tr>
<td>Normal CH</td>
<td>-39 ± 7</td>
<td>-17 ± 2</td>
<td>3 ± 2</td>
<td>37 ± 7</td>
</tr>
<tr>
<td>High CH</td>
<td>-40 ± 5</td>
<td>-19 ± 4</td>
<td>1 ± 2</td>
<td>43 ± 6</td>
</tr>
</tbody>
</table>

* p = 0.043; † p = 0.044; ‡ p = 0.003 vs normal-CH meal; § p = 0.005; ¶ p = 0.044; ¶ p = 0.002 vs high-CH meal.

Figure 1. Changes in systolic blood pressure (upper panel), diastolic blood pressure (middle panel), and heart rate (lower panel) in 12 elderly patients with postprandial hypotension after ingestion of standardized liquid meals with low- (●), normal- (○), and high- (▼) carbohydrate contents. Data are presented as mean (SEM).

Figure 2. Meal-induced symptoms in 12 geriatric patients with postprandial hypotension after ingestion of standardized liquid meals with low- (●), normal- (○), and high- (▼) carbohydrate contents. Data columns present the number of patients with symptoms.
type of food plays an important role in PPH (1). The amount of CH, and in particular oral glucose, primarily causes the BP effect of meals (12,13,15,19). In healthy subjects, these hemodynamic changes result in increases in HR, plasma norepinephrine, cardiac output, and peripheral vascular resistance to compensate for splanchnic blood pooling and to prevent significant BP reductions (6). PPH can occur if compensatory mechanisms fail (1). After meals, hypertensive elderly persons have a smaller increase in HR than normotensive elderly persons (13), whereas in frail elderly persons PPH is associated with the absence of an acceleration of cardiovascular responses (2). The significant drops in BP with only small compensatory increases of HR seen in this study could be due to the frailty of our subjects, hypertension, or impairments of the autonomic nervous system. Further studies should consider whether the hemodynamic changes are explicable on the basis of the amount of CH or on different rates of gastric emptying based on the calorie delivery in the small intestine (20).

Because any decline in SBP ≥20 mm Hg can be dangerous due to symptoms such as syncope, dizziness, or falls, the prevention of postprandial hypotensive periods is very important (1). Unfortunately, there are only a few studies on the treatment of PPH (1). Pharmacological interventions in the treatment of PPH are restricted. Octreotide, a somatostatin analog, has been shown to be effective, but it is expensive and must be given parenterally (21). Well-considered treatment of hypertension could also reduce the risk for PPH (1,18). Although caffeine is often recommended as treatment for PPH, available data do not support its use (7,22). Because of the limited usefulness and possibilities of pharmacological treatment of PPH, nonpharmacological interventions become more prominent. Several nonpharmacological empirical treatments for PPH have been suggested, such as maintaining the patient’s intravascular volume or having the patient lie down after a meal for 1 to 2 hours. On the other hand, it has been demonstrated that walking restores the postprandial BP; this effect disappears immediately after a patient stops walking (1,23). One of the most important suggested measures in the treatment of PPH seems to be adjusting the size or composition of a meal.

Three studies investigated the reduction of meal size or reducing the CH amount in meals in young healthy adults and adults with primary autonomic failure (24–26). They found that in healthy adults, a high-CH meal induced larger and more prolonged blood flow responses than a small meal. In addition, there were substantial peripheral and central cardiovascular changes after food ingestion that suggested a relationship between meal size and cardiovascular responses (24,26). Changing three large meals into six small meals with an identical daily caloric intake reduced PPH significantly in elderly subjects for clinical application. This study indicates that reducing the amount of CH in a meal can be an easy, inexpensive, and successful intervention in the overall nonpharmacological treatment to reduce postprandial declines in SBP and can improve hypotensive symptoms and shorten the period at risk in elderly patients with PPH.

Several limitations of this study need to be pointed out. First, we performed our tests in the sitting position. Although orthostatic changes in BP during prolonged sitting might contribute to the postprandial decreases in BP, we found it more physiologic to give patients their meals in a sitting position. Six of our patients were diagnosed with OH before inclusion in this study. OH was not correlated with the duration of PPH or with declines in SBP, DBP, and HR. Second, some of the patients’ complaints during the test were very general. For example, some patients complained of nausea, which could have been due to the sweet taste of the test meal. However, all symptoms were related to declines in SBP. Tiredness at the end of the test could be due to the length of the measurements. Only two subjects complained about fatigue after their SBP had already recovered.

In conclusion, lowering the CH content in meals results in significantly smaller SBP declines, significantly shorter periods with SBP declines ≥20 mm Hg, and less frequent and less severe symptoms than after normal- and high-CH meals in elderly patients with PPH. Further research is warranted to establish if limiting the CH content in regular meals by reducing the size and increasing the frequency of meals can be an easy, inexpensive, and successful intervention in the overall nonpharmacological management of PPH in elderly patients.

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References


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