Blood pressure, heart rate and neuroendocrine responses to a high carbohydrate and a high fat meal in healthy young subjects

D. HESELTINE, J. F. POTTER*, G. HARTLEY, I. A. MACDONALD† AND O. F. W. JAMES

Department of Geriatric Medicine, University of Newcastle upon Tyne, Newcastle upon Tyne, U.K., *University Department of Medicine for the Elderly, Leicester General Hospital, Leicester, U.K., and †Department of Physiology and Pharmacology, Queens Medical Centre, Nottingham, U.K.

(Received 7 March/30 April 1990; accepted 22 June 1990)

SUMMARY

1. The responses of blood pressure, heart rate, autonomic function and plasma insulin to a high carbohydrate and a high fat meal of equivalent energy value were studied in nine young volunteers.

2. Neither meal produced a significant change in supine or erect blood pressure. The high carbohydrate meal, however, resulted in an overall rise in both supine (6 beats/min) and erect (6 beats/min; \( P<0.05 \)) heart rate, no such changes being seen after the high fat meal.

3. Plasma noradrenaline levels increased by a maximum of 126% at 90 min (0.98 to 2.22 nmol/l) after the high carbohydrate meal but were virtually unchanged after the high fat meal (\( P<0.01 \)). Parasympathetic function showed no between-meal differences. Plasma insulin and glucose levels were significantly higher after the high carbohydrate meal than after the high fat meal. No postprandial difference in packed cell volume was found between meal types.

4. We conclude that, in young subjects, the postprandial blood pressure after a high carbohydrate meal is maintained by an increase in heart rate associated with increased sympathetic nervous system activity. These changes are at variance with the blood pressure and heart rate responses seen in the elderly after a high carbohydrate meal. A high fat meal has no significant cardiovascular or neuroendocrine effects in the young or old.

Key words: autonomic function, catecholamines, insulin, postprandial blood pressure.

Abbreviations: CHO, carbohydrate; DBP, diastolic blood pressure; NA, noradrenaline; SBP, systolic blood pressure; SNS, sympathetic nervous system.

INTRODUCTION

Studies of fit and institutionalized elderly subjects have shown that blood pressure falls postprandially [1-3]. We have recently demonstrated that these blood pressure changes are related to the nutrient composition of the meal, a fall in blood pressure occurring after a high carbohydrate (CHO) meal but not after a high fat or protein meal of equivalent energy content [3]. Ageing alters the normal homeostatic response to certain cardiovascular stresses and this may reflect why some [4, 5], although not all [6], studies have shown a postprandial fall in blood pressure in the elderly, with no such changes apparently occurring in young subjects [5]. The postprandial cardiovascular and neuroendocrine responses to meals of differing nutrient content have not been closely studied in the young. Previous studies in the young age group have either not assessed the underlying mechanisms responsible for the postprandial cardiovascular changes seen [5], or non-physiological energy loads (i.e. pure glucose, xylose or protein drinks) have been used [4, 6-8].

The aim of the present study was to assess in a double-blind, randomized crossover fashion, the postprandial blood pressure changes after a high CHO and a high fat meal in young subjects, with reference to orthostatic tolerance and changes in autonomic function and insulin response.

METHODS

Subjects

Nine healthy young volunteers (aged 26.3 ± 1.3 years, mean ± SEM, range 20–34 years) drawn from the hospital
staff, took part in the study. Subjects were screened to
exclude hypertension [systolic blood pressure
(SBP) > 160 mmHg and/or diastolic blood pressure
(DBP) > 95 mmHg], diabetes mellitus, autonomic
neuropathy [9] and postural hypotension (SBP fall > 20
mmHg after 2 min standing). None was taking any medi-
cation at the time of the study and all were within 10% of
ideal body weight (71.0±3.6 kg, range 51.5-92 kg;
Quetelet index 23.2±1.8 kg/m², range 19.4-25.7).

All subjects gave their informed consent and the study
was approved by the local hospital Ethical Committee.

Protocol

The methods used in this study have previously been
described in detail elsewhere [3]. In brief, no dietary
restrictions were imposed before the study. On the
morning of each phase subjects were allowed a light
breakfast before 08.00 hours but avoided all caffeinated
products, smoking and alcohol for 12 h before the study.
Subjects arrived in the laboratory at 11.00 hours rested
supine and an intravenous cannula was then inserted
retrogradely on a superficial hand vein, the hand and
forearm then being placed in an electrically heated,
thermostatically controlled box at 55°C in order to obtain
arterialized venous blood samples. Heart rate was
recorded by using a standard paper-recording electro-
cardiograph and blood pressure was measured with a
semi-automatic recorder (Dinamap 1100; Critikon,
Tampa, FL, U.S.A.) standardized against a mercury
sphygmomanometer, taking the mean of three readings.
Erect blood pressure recordings were taken after 2 min of
standing unaided.

Autonomic function was assessed before entry into the
study using the standard methods of Ewing & Clarke [9].
Heart rate variation (R-R interval) was recorded by a
standard paper-recording electrocardiograph set at 5
cm/s during deep breathing timed at six breaths/min
(taking the mean of two maximum expiratory/minimum
inspiratory R-R ratios). The minimum R-R interval
around the fifteenth beat and the maximum R-R interval
around the thirtieth beat on standing were also recorded
(the 30:15 R-R ratio).

All subjects were familiarized with the protocol before
any measurements were taken and rested supine between
measurements. At 30 and 15 min before entering into the
study, supine and erect haemodynamic and
autonomic function assessments were made and at zero
time (baseline) blood was taken for measurement of
packed cell volume, osmolality, blood glucose, plasma
insulin, noradrenaline (NA) and adrenaline levels.
Samples for plasma NA, adrenaline and insulin were
taken in seven of the subjects. Each subject then received
in a double-blind, random-order, crossover fashion one of
the two different meals. Meals were, as previously
described [3], matched as closely as possible: all consisted
of chicken, vegetables and potatoes so that they were of
equivalent energy value (2.4 MJ) and had a similar sodium
and potassium content. The meals were consumed sitting
over a 15 min period, the time being noted when the
subjects finished their meal. They then rested supine, and
the first postprandial blood pressure measurements were
taken 15 min after finishing the meal. Haemodynamic and
autonomic function measurements were made every 15
min up to 1 h and again at 90 min. Blood samples were
taken supine at 30, 60 and 90 min postprandially.

Assay methods

Catecholamine samples were taken into chilled tubes
containing ethylene glycol bis(aminoethyl ether)tetra-
acetate and glutathione, spun at 4°C and then stored at
-80°C. Duplicate samples were assayed by h.p.l.c. with
electrochemical detection, using the modified method of
Macdonald & Lake [10]. Sensitivity for NA was 0.05
nmol/l and for adrenaline 0.08 nmol/l, with interassay
coefficients of variation of 8.4% for NA and 13.9% for
adrenaline. Plasma insulin was measured by the method
of Soeldner & Slone [11] with an interassay coefficient of
variation of 7%. Packed cell volume was measured with a
Coulter Counter and had a coefficient of variation of 1%.
Blood glucose was measured by the glucose oxidase
method (Camlab) and plasma osmolality by depression of
freezing point using an automatic micro-osmometer
(Camlab).

Statistical analysis

Results are presented as means±SEM and 95% confidence
intervals are also given where indicated. The a
priori parameters of interest were the overall and
maximum differences between the meals [12]. The overall
differences between the responses to the meals were
analysed by calculating the area under the curve for that
variable (by the trapezoid method) divided by the
duration of the time zone. This gave an overall summary
statistic for each subject for the whole study period for
that meal and these area/time values for each subject
during the two meals were corrected for baseline values.
Wilcoxon's rank sum tests were used to assess the dif-
fferences between meals. Changes with time were assessed
by two-factor analysis of variance with the Bonferroni
correction factor for multiple comparisons [13].

RESULTS

Blood pressure and heart rate

All subjects completely consumed the meals and none
experienced any adverse symptoms. Baseline blood
pressure and heart rate values for both meals are given in
Table 1. Erect SBP and DBP at zero time were signifi-
cantly higher than the supine values (P<0.05 for both
SBP and DBP). There was no significant difference in
supine SBP over the 90 min between the high CHO and
high fat meals (high CHO meal +2 mmHg (95% confi-
dence intervals -3 to +7 mmHg), high fat meal +1
mmHg (-2 to +4 mmHg), using area under curve
summary data) and no significant change with time (see
Fig. 1). Similar results were obtained for diastolic blood
Table 1. Baseline supine and erect blood pressures and heart rates for the high CHO and high fat meals in the nine young subjects

<table>
<thead>
<tr>
<th></th>
<th>High CHO meal</th>
<th>High fat meal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Supine</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>120 ± 3</td>
<td>117 ± 3</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>69 ± 2</td>
<td>68 ± 3</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>56 ± 3</td>
<td>60 ± 3</td>
</tr>
<tr>
<td><strong>Erect</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>127 ± 3</td>
<td>127 ± 2</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>78 ± 2</td>
<td>78 ± 2</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>66 ± 4</td>
<td>70 ± 4</td>
</tr>
</tbody>
</table>

Pressure ([high CHO meal - 2 mmHg (−5 to +1 mmHg), high fat meal - 2 mmHg (−5 to +1 mmHg)]: However, supine heart rate over the 90 min period increased by a significantly greater amount after the high CHO meal than after the high fat meal [high CHO meal +6 beats/min (4 to 8 beats/min), high fat meal +2 beats/min (−1 to +5 beats/min), P<0.02]. The increase in heart rate was seen at 30 min and lasted up to the end of the study after the high CHO meal, the maximum increase being +9 beats/min (+7 to +11 beats/min) for the high CHO meal compared with +3 beats/min (+1 to +5, P<0.02) for the high fat meal.

No difference in erect SBP was found over the 90 min study period between the high CHO meal (0 mmHg, −3 to +3 mmHg) and the high fat meal (0 mmHg, −5 to +5 mmHg; Fig. 2) or in DBP between the high CHO meal (−3 mmHg, −6 to 0 mmHg) and the high fat meal (−2 mmHg, −6 to +2 mmHg). Overall, erect SBP and DBP remained higher than supine values after the high fat meal (P<0.05). A similar postural difference was found for DBP, but not SBP, after the high CHO meal (P<0.05). No subject developed any orthostatic symptoms or a postural fall in blood pressure of >15 mmHg. Mean heart rate values were higher on standing than supine at baseline for both meals (P<0.05) and this difference was maintained throughout both study periods (P<0.05). Erect heart rate showed no between meal differences, although there was a significant increase after the high CHO meal (+6 beats/min, +3 to +9 beats/min; P<0.05) over the 90 min.

**Autonomic function**

Parasympathetic function was assessed by the standard method of measuring the expiratory/inspiratory R–R ratio and the lying to standing 30:15 R–R ratio [9]. The coefficients of variation for baseline values were 3.5% and 8.2%, respectively, in these young subjects and all baseline values were within normal age-standardized ranges [9, 14]. There were no overall significant differences in any of these parasympathetic autonomic tests between meal types (Table 2).

Baseline plasma NA levels were similar for both phases of the study. The increases in plasma NA levels were, however, significantly greater after the high CHO meal than after the high fat meal (P<0.01; see Fig. 3). Plasma NA levels remained elevated above the baseline value for up to 90 min after the high CHO meal. There were no significant differences in plasma adrenaline after either meal and no significant change from baseline values.

**Changes in plasma insulin level**

Plasma insulin levels are shown in Table 3. Baseline values were similar for the high CHO and high fat meals,
although there was a greater overall rise after the high CHO meal than after the high fat meal \( (P<0.01) \). Plasma levels were still elevated at 90 min after both meals, although remaining higher after the high CHO meal.

Changes in blood glucose concentration, plasma osmolality and packed cell volume

As expected, the overall increase in blood glucose was greater after the high CHO meal \( (P<0.05) \), with levels remaining elevated above baseline values up to 90 min (see Table 3). Blood glucose levels were only elevated at 30 min after the high fat meal. Plasma osmolality values were similar after the high CHO and fat meals. Packed cell volume tended to fall postprandially, with no overall differences between the two meals.

DISCUSSION

This study has demonstrated that postprandial blood pressure is unchanged in young subjects after a high CHO and a high fat meal. This is at variance with the results in fit elderly subjects where we have previously shown that a high CHO meal \([3]\) reduces supine SBP, supine DBP and erect SBP, although it does not compromise orthostasis. However, in these young subjects, supine and erect heart rate increased after the high CHO meal but were unaffected by the high fat meal, demonstrating a specific nutrient effect on heart rate in the young. No such increase in heart rate was seen in the elderly after either meal type \([3]\).

The results of previous studies on postprandial blood pressure and heart rate changes in the young have been conflicting due to differences in the type and form of energy loading ingested (e.g. a pure glucose drink or a mixed meal) and in the timing of the haemodynamic measurements. Young \textit{et al.} \([6]\) and Jansen \& Hoefnagels \([15]\) reported no change in mean arterial pressure or heart rate after a glucose drink \((1.7 \text{ and } 1.2 \text{ MJ, respectively})\). Robertson \textit{et al.} \([16]\) and Westenend \textit{et al.} \([17]\) demonstrated a significant, although small, increase in heart rate after a mixed meal \((1.9 \text{ and } 1.7 \text{ MJ, respectively})\). Robertson \textit{et al.} \([16]\) also observing a rise in mean arterial pressure. Meals of higher energy content than those used in this study \((>3 \text{ MJ})\) have resulted in an increase in heart rate and SBP but a fall in DBP \([18, 19]\).

### Table 2. R–R interval values for the lying/standing (30:15 ratio) and for expiration/inspiration (E/I ratio) after the high CHO and high fat meals

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>0</th>
<th>15</th>
<th>30</th>
<th>45</th>
<th>60</th>
<th>90</th>
</tr>
</thead>
<tbody>
<tr>
<td>R–R interval for the 30:15 ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High CHO meal</td>
<td>1.17 ± 0.08</td>
<td>1.19 ± 0.06</td>
<td>1.18 ± 0.07</td>
<td>1.17 ± 0.06</td>
<td>1.16 ± 0.06</td>
<td>1.18 ± 0.1</td>
</tr>
<tr>
<td>High fat meal</td>
<td>1.19 ± 0.07</td>
<td>1.25 ± 0.07</td>
<td>1.21 ± 0.09</td>
<td>1.18 ± 0.05</td>
<td>1.17 ± 0.09</td>
<td>1.19 ± 0.04</td>
</tr>
<tr>
<td>R–R interval for the E/I ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High CHO meal</td>
<td>1.45 ± 0.05</td>
<td>1.38 ± 0.03</td>
<td>1.36 ± 0.07</td>
<td>1.38 ± 0.08</td>
<td>1.38 ± 0.08</td>
<td>1.40 ± 0.04</td>
</tr>
<tr>
<td>High fat meal</td>
<td>1.46 ± 0.04</td>
<td>1.47 ± 0.10</td>
<td>1.40 ± 0.09</td>
<td>1.38 ± 0.09</td>
<td>1.41 ± 0.13</td>
<td>1.44 ± 0.06</td>
</tr>
</tbody>
</table>
Table 3. Plasma insulin level, packed cell volume, plasma osmolality and blood glucose concentration in young subjects after the high CHO and high fat meals

Values are means ± SEM. Statistical significance: *P<0.05 compared with baseline values. Abbreviation: NS, not significant.

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>0</th>
<th>30</th>
<th>60</th>
<th>90</th>
<th>P value for the difference between meals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma insulin level (m-units/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High CHO meal</td>
<td>3.5 ± 1.2</td>
<td>43.8 ± 8.3*</td>
<td>29.5 ± 2.6*</td>
<td>20.7 ± 3.9*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>High fat meal</td>
<td>3.9 ± 0.8</td>
<td>25.8 ± 5.4*</td>
<td>12.1 ± 1.5*</td>
<td>9.5 ± 1.0*</td>
<td></td>
</tr>
<tr>
<td>Packed cell volume</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High CHO meal</td>
<td>0.403 ± 0.016</td>
<td>0.400 ± 0.016</td>
<td>0.400 ± 0.016</td>
<td>0.400 ± 0.013</td>
<td>NS</td>
</tr>
<tr>
<td>High fat meal</td>
<td>0.414 ± 0.015</td>
<td>0.411 ± 0.017</td>
<td>0.411 ± 0.016</td>
<td>0.408 ± 0.017</td>
<td></td>
</tr>
<tr>
<td>Plasma osmolality (mosmol/kg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High CHO meal</td>
<td>289 ± 2</td>
<td>289 ± 1</td>
<td>289 ± 2</td>
<td>289 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>High fat meal</td>
<td>288 ± 2</td>
<td>289 ± 2</td>
<td>289 ± 2</td>
<td>290 ± 2</td>
<td></td>
</tr>
<tr>
<td>Blood glucose concn. (mmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High CHO meal</td>
<td>4.8 ± 0.2</td>
<td>7.6 ± 0.2*</td>
<td>6.1 ± 0.4*</td>
<td>5.6 ± 0.2*</td>
<td>0.05</td>
</tr>
<tr>
<td>High fat meal</td>
<td>4.9 ± 0.2</td>
<td>6.1 ± 0.2*</td>
<td>5.0 ± 0.2</td>
<td>5.1 ± 0.2</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 3. Supine postprandial plasma NA concentrations after the high CHO (•) and high fat (○) meals. Values are means ± SEM. Statistical significance: *P<0.05, **P<0.01 compared with baseline values.

An antecedent diet high in CHO increases insulin release after a glucose load [20], which could possibly potentiate the postprandial cardiovascular changes. As both energy content and proportion of dietary CHO change with age [21, 22], this may explain some of the age-related differences seen.

The age-related differences in postprandial blood pressure changes after a high CHO intake may be due to alterations in postprandial heart rate response, the elderly showing little [5] or no [1, 15] increase in heart rate despite the fall in blood pressure. Whether this is secondary to age-related changes in baroreceptor sensitivity or, in view of the increase in plasma NA level, a decrease in chronotropic response to β-adrenergic stimulation in the elderly, is unclear. Appenzeller & Goss [23] have shown that glucose impairs baroreceptor sensitivity, but Jansen & Hoefnagels [24] have recently disputed these findings, in the elderly at least. The high CHO meal did not impair the normal orthostatic increase in heart rate in the young in this study, however.

Autonomic function was within the normal age-standardized range for all subjects and no difference in parasympathetic function was seen between meals or with time. The findings are similar to those reported by us in the elderly [3]. The increase in heart rate seen in the young, but not in the elderly, after a high CHO meal is unlikely to be due to decreased vagal tone, although this cannot be discounted.

Sympathetic nervous system (SNS) activity, as gauged by changes in plasma NA levels, showed a 126% increase after the high CHO meal, although it was virtually unchanged after the high fat meal. We have previously reported similar such between-meal differences for the elderly [3], as have Welle et al. [8] studying younger subjects using pure glucose and fat drinks of a lower energy content (1.67 MJ). The reasons for this rise in the plasma NA levels after a CHO, but not a fat, load in both young and old subjects are still unclear. There is no evidence to suggest that glucose or insulin impair plasma NA clearance [6]. If the rise is due to increased SNS activity, whether this is a generalized effect or related to changes in specific organs, i.e. heart, skeletal muscle and splanchnic bed, is also uncertain. Increased SNS activity could be a direct effect of insulin [25], adenosine [26] or an increase in baroreceptor activity secondary to a decrease in total peripheral resistance after food ingestion [27]. It is unlikely to be due to a fall in plasma volume, as packed cell volume fell rather than increased after both meal types.

The underlying pathophysiological mechanisms for the postprandial rise in heart rate and the neuroendocrine changes seen in the young are unclear. The role of vasoactive gut peptides cannot be excluded, although Mathias et al. [28] and Hoeldtke et al. [29] were unable to identify
any such hormone. The high CHO meal could result in increased splanchnic blood flow [30] with a fall in total peripheral vascular resistance [27] mediated by vaso-active gut peptides, insulin or adenosine [31]. Yi et al. [27] found no evidence of decreased limb blood flow post-prandially, suggesting a lack of a compensatory change in regional blood flow to offset the increase in splanchnic blood flow in young subjects. These changes in the young must therefore be fully compensated for by an increase in heart rate as demonstrated in this study (and cardiac output [32]) as a result of increased SNS activity, or perhaps decreased vagal tone. In view of the marked increase in SNS activity a greater rise in heart rate might have been expected after the high CHO meal. Insulin-induced antagonism of the peripheral effects of NA may explain these findings [33].

In conclusion, our data show that postprandial blood pressure is unchanged in young subjects after both high CHO and high fat meals. The high CHO meal resulted in an increase in heart rate and SNS activity, features not seen after the high fat meal. This chronotropic response to the high CHO meal in the young may explain why postprandial hypotension is not seen in these subjects. The specific postprandial age-related differences that account for these findings are still unexplained.

REFERENCES