Effects of meal composition on the postprandial blood pressure, catecholamine and insulin changes in elderly subjects

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

Departments of Geriatric Medicine, *Dietetics and †Medical Statistics, University of Newcastle upon Tyne, Newcastle upon Tyne, U.K., and ‡Department of Physiology and Pharmacology, Medical School, Queen's Medical Centre, Nottingham, U.K.

(Received 18 October 1988/6 February 1989; accepted 13 February 1989)

SUMMARY

1. The effects of four meals of similar energy, but different nutritional composition on postprandial blood pressure, heart rate, autonomic function, catecholamines, insulin and packed cell volume levels were studied in seven fit elderly subjects.

2. The high carbohydrate and high protein meals led to a significant overall fall in supine systolic and diastolic blood pressure compared either with no change or a rise after the normal (i.e. mixed) and high fat meals. Similar between-meal differences were seen with erect diastolic but not erect systolic blood pressure. No significant postural blood pressure fall occurred after any of the meals. Supine heart rate was unaffected by meal type or by time, and although erect heart rate showed a small increase during the study there was no between-meal difference.

3. Parasympathetic function was unaffected by meal type. Plasma noradrenaline rose after the high carbohydrate and mixed meals only, remaining elevated for 120 min after meal consumption. This increase was not related to the changes in blood pressure or plasma insulin levels.

4. Plasma insulin and glucose rose after the high carbohydrate and mixed meals, but were unchanged after the high protein and high fat meals. Packed cell volume showed a small decrease towards the end of the study, although there was no between-meal variation.

5. The differences in the cardiovascular changes after the different meals could not be ascribed to alterations in autonomic function, insulin release or fall in plasma volume. We propose that the postprandial changes in blood pressure are due to the nutrient composition of the meal rather than the actual energy load.

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

Abbreviations: CHO, carbohydrate; DBP, diastolic blood pressure; NA, noradrenaline; PCV, packed cell volume; SBP, systolic blood pressure; SED, standard error of the difference; SNS, sympathetic nervous system.

INTRODUCTION

Ageing alters the normal homoeostatic responses to certain cardiovascular stresses [1, 2]. This has recently been highlighted by reports showing that marked postprandial falls in blood pressure occur in both fit [3] and institutionalized [4] elderly subjects, but not in young subjects [5, 6]. The exact clinical effects of these blood pressure changes in old people are, as yet, speculative, but could be important in the pathogenesis of falls, post-meal angina or even stroke.

Several reports have documented the fall in blood pressure which occurs after meals without attempting to establish the underlying mechanisms responsible [3, 6]. Plasma insulin [7, 8], glucose [9] and gut peptides [10] could cause a fall in blood pressure by a direct vasodilatory effect on splanchnic and skeletal muscle vessels, and it has been proposed that insulin may also reduce blood volume [11]. The largest postprandial falls in blood pressure occur in subjects with autonomic dysfunction [12]. Autonomic, including baroreceptor, activity is known to become impaired with increasing age [13] and is further compromised by raised blood glucose levels [14]. Thus the postprandial cardiovascular changes may be secondary to an alteration in autonomic control. Whatever the initial cause of the reduction in blood pressure, the elderly appear to be unable to maintain an adequate homoeostatic reflex response.
From the results of previous work it is uncertain whether the postprandial fall in blood pressure seen in the elderly is due solely to the calorie load or is dependent upon the nutrient and electrolyte composition or bulk of the meal. In fact many of the studies have used non-physiological calorie loads, i.e. pure glucose [5, 15], fructose or xylose drinks [16].

This present study was undertaken both to investigate the possible physiological mechanisms whereby postprandial hypotension occurs in the elderly and to define the relative importance of different 'meal factors' in producing these changes.

METHODS

Subjects

Seven fit, ambulant, community-living volunteers [mean age 70.7 ± 1.9 (SEM) years, range 61–77 years] were drawn from local pensioners' clubs. Subjects were screened to exclude hypertension [systolic blood pressure (SBP) > 160 mmHg and/or diastolic blood pressure (DBP) > 95 mmHg (21.3/12.6 kPa)], ischaemic heart disease, cerebrovascular disease, autonomic neuropathy (by standard methods [17]), postural hypotension [SBP fall > 20 mmHg (2.7 kPa)] and diabetes mellitus. None was taking any medication at the time of the study and all were within 10% of ideal body weight [mean 68.8 ± 3.9 (SEM) kg, range 49.5–80 kg, Quetelet index 24.0 ± 1.0 (SEM) kg/m²]. All subjects gave their informed consent and the study was approved by the local Joint Ethical Committee.

Protocol

Each subject was studied on four separate occasions at least 3 days apart. On the morning of each study, subjects were allowed a light breakfast (taken at least 5 h before the study), having avoided caffeinated products, smoking and alcohol for 12 h before the study. Subjects were asked to micturate before being weighed; they then rested supine and an intravenous cannula was inserted retrogradely into a superficial hand vein and kept patent by flushing with heparinized saline (Hepsal CP Pharmaceuticals). The hand and forearm were placed in a heated thermostatically controlled box at 55°C to obtain arterialized venous blood samples. Subjects rested supine for 60 min before entering the study.

Blood pressure and heart rate were recorded in the other arm using a semi-automatic recorder (Dinamap 1100; Critikon, Tampa, FL, U.S.A.), taking the mean of three readings on each occasion. Autonomic function was assessed by the standard methods of Ewing & Clarke [17]. The heart rate variation during deep breathing was measured using a standard paper-recording electrocardiograph set at 5 cm/s with the subjects having a set respiratory rate of 6 breaths/min. Subjects timed this using a clock with a second hand, readings being taken on the third of such inspiratory/expiratory cycles. The maximum expiratory and minimum inspiratory R–R intervals were measured using a ruler during each respiratory cycle (the expiration/inspiration ratio). The heart rate response to standing unaided was also noted, measuring the shortest R–R interval at around the beat 15 and the longest R–R interval at around beat 30 (the 30:15 ratio). Erect blood pressure was taken 2 min after standing.

All subjects were familiarized with the protocol before any measurements were taken and they rested supine between measurements. At 30 and 15 min before entry into the study, haemodynamic measurements were made and at time 0 (baseline) blood was also taken for measurement of packed cell volume (PCV), plasma osmolality, blood glucose, plasma insulin, plasma noradrenaline (NA) and plasma adrenaline. Subjects then received in a double-blind, randomized cross-over fashion, one of the four different meals. These were matched as closely as possible: all consisted of chicken, vegetables and potatoes (see Table 1) so that they were isocaloric (2420 kJ) with similar sodium, potassium and calcium contents. The electrolyte content was balanced by the use of supplements given in 100 ml of water with each of the meals. Meals were consumed sitting over a 10–15 min period, the time when the subjects finished being noted. They then rested supine and the first postprandial measurements were taken 15 min after finishing the meal. Haemodynamic and autonomic function measurements were made every 15 min for the first 60 min and then 90 and 120 min. Blood samples were taken supine at 30, 60, 90 and 120 min. The hand was kept constantly in the heated compartment until the end of the study.

Table 1. Energy and electrolyte content of the four test meals

<table>
<thead>
<tr>
<th>Meal type...</th>
<th>High CHO</th>
<th>High fat</th>
<th>High protein</th>
<th>Mixed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate (% of total energy)</td>
<td>72</td>
<td>19</td>
<td>17</td>
<td>49</td>
</tr>
<tr>
<td>Fat (% of total energy)</td>
<td>7</td>
<td>66</td>
<td>30</td>
<td>27</td>
</tr>
<tr>
<td>Protein (% of total energy)</td>
<td>21</td>
<td>15</td>
<td>53</td>
<td>24</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>330</td>
<td>243</td>
<td>368</td>
<td>299</td>
</tr>
<tr>
<td>Potassium (mg)</td>
<td>1841</td>
<td>673</td>
<td>1458</td>
<td>1504</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>456</td>
<td>89</td>
<td>325</td>
<td>244</td>
</tr>
<tr>
<td>Fibre (g)</td>
<td>11</td>
<td>3</td>
<td>6</td>
<td>8</td>
</tr>
</tbody>
</table>
Assay methods

Catecholamine samples were taken into chilled tubes containing ethyleneglycolbis(aminooxyethleyether)tetraacetate 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 [18]. Sensitivity for NA was 0.05 nmol/l and for adrenaline 0.08 nmol/l, with an interassay coefficient of variation of 8.4% for NA and 13.9% for adrenaline. Plasma insulin was measured by the method of Soeldner & Slone [19] with an interassay coefficient of variation of 7%. PCV was measured with a Coulter counter and had a coefficient of variation of 1%. Blood acetate and glutathione, spun at 4°C and then stored at 80°C. Duplicate samples were assayed by h.p.l.c. with 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 differences between the responses to the meals were analysed in two ways. First, the profiles for each subject within each pre-defined supine and erect time zone were summarized by calculating the area under the curve for each variable (by the trapezium method) divided by the duration of the time zone. This gives an overall summary statistic for each subject covering the whole study period for that meal. The differences of these mean values from baseline were compared by two-way analysis of variance. Secondly, the maximum changes within the 120 min study period were compared by analysis of covariance, using the baseline value as a covariate. Where an overall difference was detected, differences between diets were assessed by t-tests using the standard error of the difference (SED) as calculated from the residual mean square from the analysis of variance. Pearson correlation coefficients were also calculated.

RESULTS

Table 2. Baseline supine and erect blood pressures and heart rate for each of the four meals (high CHO, high fat, high protein and mixed, i.e. normal)

<table>
<thead>
<tr>
<th>Meal type ...</th>
<th>CHO</th>
<th>High fat</th>
<th>High protein</th>
<th>Mixed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>123 ± 4</td>
<td>122 ± 4</td>
<td>122 ± 4</td>
<td>126 ± 4</td>
</tr>
<tr>
<td></td>
<td>(111-136)</td>
<td>(106-138)</td>
<td>(109-138)</td>
<td>(112-140)</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>71 ± 3</td>
<td>74 ± 3</td>
<td>69 ± 4</td>
<td>69 ± 4</td>
</tr>
<tr>
<td></td>
<td>(60-78)</td>
<td>(62-88)</td>
<td>(52-84)</td>
<td>(64-82)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>58 ± 1</td>
<td>57 ± 1</td>
<td>39 ± 1</td>
<td>60 ± 2</td>
</tr>
<tr>
<td>(beats/min)</td>
<td>(56-62)</td>
<td>(51-62)</td>
<td>(54-65)</td>
<td>(49-68)</td>
</tr>
<tr>
<td>Erect</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>122 ± 4</td>
<td>121 ± 5</td>
<td>124 ± 2</td>
<td>125 ± 2</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>77 ± 5</td>
<td>75 ± 5</td>
<td>73 ± 4</td>
<td>77 ± 4</td>
</tr>
<tr>
<td></td>
<td>(60-88)</td>
<td>(61-94)</td>
<td>(56-86)</td>
<td>(66-90)</td>
</tr>
<tr>
<td>Heart rate</td>
<td>63 ± 1</td>
<td>61 ± 2</td>
<td>65 ± 2</td>
<td>64 ± 2</td>
</tr>
<tr>
<td>(beats/min)</td>
<td>(58-67)</td>
<td>(57-72)</td>
<td>(58-70)</td>
<td>(58-76)</td>
</tr>
</tbody>
</table>

Table 2. Baseline supine and erect blood pressures and heart rate for each of the four meals (high CHO, high fat, high protein and mixed, i.e. normal)

Data are presented as mean ± SEM (n = 7). Ranges are given in parentheses. Baseline supine and erect blood pressures were similar, although overall erect heart rates were greater than supine (P<0.01). To convert mmHg to kPa, divide by 7.5.
showing a small rise with time, did not differ between meals.

The maximum changes in blood pressure and heart rate seen during the study period with each of the four meals are illustrated in Table 3. There was a significant between-meal effect on maximum supine SBP fall \((P<0.001)\), being greater after the high CHO \([-13\; \text{mmHg} (1.7\; \text{kPa})\), \(-19\) to \(-7\; \text{mmHg}\)] than the high fat diet \([0\; \text{mmHg}, \; -6\; \text{to}\; +6\; \text{mmHg}, \; P<0.01]\) or mixed meals \([-7\; \text{mmHg} (0.9\; \text{kPa}), \; -13\; \text{to}\; -1\; \text{mmHg}, \; P<0.05]\), but not the high protein meal \([-9\; \text{mmHg} (1.2\; \text{kPa}), \; -15\; \text{to}\; -3\; \text{mmHg}\)]. There was no statistical difference in the maximum fall in DBP or the heart rate changes between meals. The maximum drop in erect DBP, however, did differ between meals \((P<0.02)\), being greater after the high CHO meal \([-11\; \text{mmHg} (1.5\; \text{kPa}), \; -16\; \text{to}\; -6\; \text{mmHg}\)] than either the high fat \([-4\; \text{mmHg} (0.5\; \text{kPa}), \; -9\; \text{to}\; +1\; \text{mmHg}, \; P<0.05]\) or high protein \([-5\; \text{mmHg} (0.7\; \text{kPa}), \; -10\; \text{to}\; 0\; \text{mmHg,}\; P<0.05]\) but not the mixed \([-8\; \text{mmHg} (1.1\; \text{kPa}), \; -13\; \text{to}\; -3\; \text{mmHg}\)] meal. Erect SBP and heart rate did not differ between meals and there was no difference in the maximum rises in supine or erect blood pressure or heart rate between meals.

No subject showed a postural SBP fall of more than 20 mmHg (2.7 kPa) after any meal. This resulted from both erect and supine blood pressures falling to a similar extent such that SBP and DBP were maintained on standing.

Maximum haemodynamic changes occurred within 45-60 min of finishing the meal in all cases.

**Parasympathetic and sympathetic function**

Parasympathetic function was assessed by two standard methods. All subjects had baseline values within the normal age standardized range [20], with a coefficient of variation for baseline values for the four meals of 3.3% for the expiration/inspiration ratio and 7.9% for the 30:15 ratio (Table 4). There was no difference between meals over the study period for each of the two methods used. Baseline supine plasma NA levels for all four meals were similar (Fig. 3). However, there was a significant difference in the NA response to the four meals \((P<0.01)\): the high CHO and mixed meals resulted in similar responses with a maximum increase of 74% and 72%, respectively. Plasma NA levels were little changed after the high fat or high protein meals, with a significant difference between the response to the high CHO and mixed meals compared with the high fat or high protein meals \((P<0.05)\). After 120 min plasma NA levels were still rising after the CHO meal, but reached a peak at 90 min with the mixed meal, although NA levels were still higher than baseline values at the end of the study \((P<0.05)\).
Table 3. Maximum changes in blood pressure and heart rate during the 120 min study period after each of the four meals (high CHO, high fat, high protein and mixed, i.e. normal) for the seven elderly subjects

Values given are means with SED as calculated from the analysis of covariance. Where an overall difference was found, differences between the diets were calculated using t-tests with the SED: *P< 0.05, **P< 0.01. Abbreviation: NS, not significant. To convert mmHg to kPa, divide by 7.5.

<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>
<th>120</th>
</tr>
</thead>
</table>
| R-R ratio interval changes between expiration/inspiration
  High CHO
  (0.01) | 1.13 | 1.14 | 1.11 | 1.11 | 1.10 | 1.09 | 1.13 | 1.13 |
  High fat
  (0.03) | 1.18 | 1.13 | 1.12 | 1.12 | 1.11 | 1.11 | 1.13 | 1.09 |
  High protein
  (0.02) | 1.15 | 1.12 | 1.13 | 1.12 | 1.12 | 1.12 | 1.11 | 1.12 |
  Mixed
  (0.01) | 1.14 | 1.11 | 1.11 | 1.12 | 1.15 | 1.14 | 1.15 | 1.15 |
| R-R ratio interval change for the 30:15 ratio
  High CHO
  (0.03) | 1.06 | 1.04 | 1.01 | 1.05 | 1.01 | 1.00 | 1.07 |
  High fat
  (0.04) | 1.11 | 0.97 | 1.04 | 1.03 | 1.04 | 1.06 | 1.05 |
  High protein
  (0.03) | 1.11 | 1.07 | 1.09 | 1.05 | 1.08 | 1.07 | 1.07 |
  Mixed
  (0.03) | 1.08 | 1.01 | 1.04 | 1.08 | 1.07 | 1.12 | 1.10 |

Plasma adrenaline levels showed no change with meal composition or time.

Changes in plasma insulin, plasma glucose and PCV

Plasma insulin changes with the four meals are shown in Fig. 4. Baseline values were similar, although peak levels were higher and occurred later, at 60 min, with the high CHO and mixed meals than with the high protein and fat meals. There was a significant difference in overall insulin response between meals (P<0.01): the high CHO meal resulted in a greater increase than either the mixed (P<0.02), high protein or high fat (P<0.01) meals. Although the patterns of the insulin and NA response curves to each meal were similar, there was no significant correlation between the actual levels or the changes in insulin and NA. Baseline blood glucose levels were again similar for all four meals and reached maximum levels at 30 min after the meal (Table 5). As expected, the high CHO meal produced the highest blood glucose levels but mean levels were only minimally raised after the high protein meal; there was a significant between-meal effect
DISCUSSION

As far as we are aware this is the first study to assess the effects of differing meal composition on the postprandial cardiovascular changes in old people. After the high CHO and high protein meals there were similar reductions in both supine and erect blood pressure, the normal (i.e. mixed) meal led to a less pronounced fall, whereas after the high fat meal there was either no change or a small overall rise in blood pressure. None of the meals appeared to impair orthostatic control of blood pressure and no subject developed a significant postural blood pressure drop. Despite the fall in blood pressure with the high CHO and high protein meals, there were no changes in supine heart rate and although erect heart rate increased (especially after the high CHO intake) there was no overall between-meal effect on heart rate. The postprandial fall in blood pressure appears therefore to be dependent on the nutritional composition of meal and not the actual calorie load.

Previous studies in the elderly have shown that blood pressure decreases after a normal meal in both fit and institutionalized subjects when compared with a control period when no food is taken [3, 21]; thus the changes in blood pressure in the present study are not likely to be due solely to repeated measurements or failure to achieve a stable baseline blood pressure. This fall is dependent on age, the largest changes occurring in the elderly [5, 6] and on the energy load being taken orally and not intravenously [15]. The blood pressure fall may result from a decrease in plasma volume or total peripheral resistance without a full compensatory increase in cardiac output. Gundersen & Christensen [11] suggested that insulin decreased plasma volume by causing a loss of intravascular water and albumin, although others have failed to confirm these findings [8]. Plasma volume changes were not measured directly, but PCV fell rather than increased after the high CHO, high protein and mixed meals (and remained unchanged after the high fat meal). It is unlikely therefore that plasma volume decreased. We did not measure total peripheral resistance but this has been shown to fall after meals [22, 23]. Lipsitz et al. [4] also noted that the elderly failed to increase heart rate with a fall in blood pressure after a meal, although this has not been shown in all postprandial studies [5]. This lack of a compensatory increase in heart rate may result from an age-related impairment of baroreceptor function [13], which is further complicated by the elevated blood glucose levels [14]. Ageing per se also decreases myocardial inotropic [24] and chronotropic responses to β-adrenergic stimulation or stress [25].

As the largest falls in postprandial blood pressure occur in subjects with an autonomic neuropathy, and autonomic activity is known to decrease with age, it is possible that the postprandial fall in blood pressure in this study was due to impairment of autonomic control. We found only small changes in cardiac parasympathetic activity with no between-meal differences. Similarly, no differences were seen in the postural change in blood pressure after the four meals, although this is only a crude guide to sympathetic impairment. Plasma NA levels, a better indicator than postural change in blood pressure of postprandial sympathetic nervous system (SNS) activity, were markedly increased after the high CHO and mixed meals but little changed after the high fat and high protein meals. This rise in plasma NA after the high CHO and mixed meals was similar to that reported in other postprandial studies in elderly subjects. Welle et al. [26] also

---

\( P < 0.05 \). At the end of the study blood glucose had returned to basal levels with all except the high CHO meal. Osmolality showed the largest rise after the high CHO meal but there was no significant overall meal effect. PCV showed a small fall with both the mixed and high CHO meals; the greatest changes occurred between 90 and 120 min after the meals, but again no overall differences between meals was found.
noted a rise in NA after glucose but not after a high fat or high protein intake. The increase in NA may thus be related to changes in glucose or insulin, as NA levels were only minimally elevated after the high protein and high fat meals in which changes in plasma glucose and insulin were small. It is improbable, however, that the rise in NA is due solely to SNS stimulation resulting from the fall in blood pressure, as NA levels were still rising at 120 min after the high CHO meal when the blood pressure had returned to baseline values. Furthermore, plasma NA levels were unchanged after the high protein meal despite a similar fall in blood pressure to that seen after the high CHO meal.

Insulin can stimulate the SNS during euglycaemia [27], as well as antagonizing the actions of NA [28]. Both insulin and NA levels increased after the high CHO and mixed meals, but there was no relationship between the changes in the two variables. Jansen et al. [5] noted, in elderly normotensive subjects, a fall in mean arterial pressure after glucose, but not fructose, although both calorie loads produced similar NA responses. They also found a greater increase in plasma insulin after glucose than after fructose, so it seems unlikely the rise in NA is due primarily to an insulin-mediated stimulation of the SNS. Finally, the elevated NA levels could be due to diminished clearance, secondary to the elevated blood glucose levels (insulin does not appear to alter NA clearance rates [27]), although this has not been formally studied and is unlikely as splanchnic blood flow will be increased postprandially.

The variation in insulin response after the four meal types reflects the differences in the postprandial blood glucose levels. As the fall in blood pressure after the high CHO and high protein meals was similar despite marked difference in insulin levels, it seems unlikely that the postprandial changes are due directly to insulin. The findings of Jansen & Hoefnagels [15] support this view. They gave similar energy loads (in the form of glucose) orally and intravenously, but only after the oral load did blood pressure fall, despite similar insulin levels being produced in both studies. We cannot exclude the role of vasoactive gut-associated peptides, such as neuropeptin, released due to the direct effect of nutrients on the gut, as a cause for the postprandial cardiovascular changes seen. Hoeldtke et al [29] found that a somatostatin analogue blocked the postprandial hypotensive effect, but were unable to identify any gut hormone responsible. However, their findings could be explained by a direct pressor effect of somatostatin analogue. One possible hypothesis for the postprandial fall in blood pressure in the elderly is as a result of an increase in splanchnic blood flow and a fall in total peripheral resistance induced by certain gut peptides or by insulin. This fall is not fully compensated for by an increase in heart rate, perhaps due to a resetting of baroreceptor activity (either due to elevated insulin and/or glucose levels). However, it is unlikely that there is marked baroreceptor impairment as heart rate still increased on standing after the meals. This failure to increase heart rate despite the fall in blood pressure and increase in NA levels may be compounded by an insulin-induced antagonism of the action of NA, especially its chronotropic action on the heart [30]. The high CHO and high protein meals that caused the greatest postprandial falls in blood pressure could have different hypotensive mechanisms, i.e. the high protein meal may lead to a more marked increase in splanchnic blood flow [31], whereas the high CHO meal with the subsequent greater increases in NA, insulin and glucose levels may alter baroreceptor activity or lead to an insulin-mediated impairment of NA-induced vasoconstriction.

In conclusion, we have found that isocaloric high CHO and to a lesser extent high protein or normal mixed meals result in a postprandial fall in blood pressure in fit elderly subjects, whereas a high fat meal causes no such

<table>
<thead>
<tr>
<th>Time (min)…</th>
<th>0</th>
<th>30</th>
<th>60</th>
<th>90</th>
<th>120</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PCV</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>0.394 ± 0.010</td>
<td>0.399 ± 0.010</td>
<td>0.394 ± 0.010</td>
<td>0.388 ± 0.011</td>
<td>0.382 ± 0.011</td>
</tr>
<tr>
<td>High fat</td>
<td>0.391 ± 0.011</td>
<td>0.394 ± 0.010</td>
<td>0.394 ± 0.010</td>
<td>0.390 ± 0.011</td>
<td>0.391 ± 0.013</td>
</tr>
<tr>
<td>High protein</td>
<td>0.399 ± 0.008</td>
<td>0.403 ± 0.011</td>
<td>0.399 ± 0.009</td>
<td>0.394 ± 0.009</td>
<td>0.389 ± 0.011</td>
</tr>
<tr>
<td>High CHO</td>
<td>0.390 ± 0.011</td>
<td>0.386 ± 0.009</td>
<td>0.383 ± 0.008</td>
<td>0.377 ± 0.011</td>
<td>0.378 ± 0.008</td>
</tr>
<tr>
<td><strong>Plasma osmolality (mosmol/kg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>293 ± 3</td>
<td>294 ± 2</td>
<td>296 ± 3</td>
<td>294 ± 1</td>
<td>293 ± 2</td>
</tr>
<tr>
<td>High fat</td>
<td>292 ± 3</td>
<td>295 ± 2</td>
<td>294 ± 2</td>
<td>293 ± 2</td>
<td>293 ± 2</td>
</tr>
<tr>
<td>High protein</td>
<td>295 ± 3</td>
<td>295 ± 2</td>
<td>297 ± 2</td>
<td>295 ± 2</td>
<td>295 ± 2</td>
</tr>
<tr>
<td>High CHO</td>
<td>291 ± 3</td>
<td>296 ± 2</td>
<td>296 ± 2</td>
<td>294 ± 2</td>
<td>293 ± 2</td>
</tr>
<tr>
<td><strong>Blood glucose (mmol/l)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mixed</td>
<td>5.2 ± 0.5</td>
<td>7.6 ± 0.6</td>
<td>7.2 ± 0.8</td>
<td>6.4 ± 0.6</td>
<td>5.9 ± 0.5</td>
</tr>
<tr>
<td>High fat</td>
<td>4.9 ± 0.4</td>
<td>6.2 ± 0.4</td>
<td>6.2 ± 0.6</td>
<td>5.4 ± 0.6</td>
<td>5.2 ± 0.4</td>
</tr>
<tr>
<td>High protein</td>
<td>5.4 ± 0.4</td>
<td>6.3 ± 0.5</td>
<td>5.8 ± 0.6</td>
<td>5.1 ± 0.3</td>
<td>5.0 ± 0.3</td>
</tr>
<tr>
<td>High CHO</td>
<td>5.2 ± 0.3</td>
<td>8.8 ± 0.5</td>
<td>8.5 ± 1.1</td>
<td>7.5 ± 1.0</td>
<td>6.6 ± 0.5</td>
</tr>
</tbody>
</table>
decrease. The mechanisms for these changes are unclear, but do not appear to be due to alterations in autonomic function, insulin release or a fall in plasma volume. In these healthy elderly subjects postprandial orthostatic control of blood pressure was unaffected. However, such blood pressure changes in the ‘frail’ elderly and in those with autonomic impairment may be clinically important as a cause of dizziness, falls and post-meal angina, and may perhaps explain some of the diurnal variation in cerebral thrombosis [32].

ACKNOWLEDGMENTS

We thank Miss P. A. Craik for typing the manuscript, and the dietitians at Freeman Hospital for their help in preparing the meals.

REFERENCES


