Whole body and splanchnic metabolic, circulatory, and thermal effects of oral vs. intravenous fat administration

TOMAS BRUNDIN
Department of Clinical Physiology, Karolinska Hospital, S-171 76 Stockholm, Sweden

Brundin, Tomas. Whole body and splanchnic metabolic, circulatory, and thermal effects of oral vs. intravenous fat administration. Am. J. Physiol. 274 (Endocrinol. Metab. 37): E684–E691, 1998.—Relatively few studies on the physiological effects of fat administration have been published. In the present study, whole body and splanchnic oxygen consumption, blood flow, blood temperature, glucose and insulin economy, and arterial and hepatic venous concentrations of hemoglobin, free fatty acids (FFA), and glycero were measured by indirect calorimetry and catheterization technique in seven healthy men before and during 2.5 h after oral ingestion of 850 kJ of fat and in five healthy men before and during a 2.5-h intravenous (iv) infusion of 850 kJ of fat. Oral fat increased the splanchnic blood flow by 57 ± 25%, reduced the plasma volume by 6 ± 1%, reduced the arterial concentrations of FFA and glycero by 33 ± 7% and 50 ± 16%, respectively, and increased arterial insulin concentration by 52 ± 12% despite a simultaneous reduction in splanchnic insulin release, thus suggesting a reduction of the extrasplanchnic extraction of insulin. None of these effects occurred during intravenous fat infusion, and it is suggested that intestinal hormones might elicit these effects. Body heat content, unaffected after oral fat, increased by 67 ± 20 kJ during intravenous fat infusion.

Nutrient ingestion stimulates oxidative metabolism (nutrient-induced thermogenesis), blood flow, and body temperature. Our three nutrients affect these variables differently (5). The thermic effect, i.e., the rise in whole body oxidative energy expenditure in percentage of the energy content of the nutrient given, is 30–40% for protein, 6–9% for carbohydrates, and 0–2% for fat (19). Oral protein ingestion gives a thermic effect similar to that during intravenous amino acid infusion (10, 11). Thus the intestinal absorption of even large protein meals causes no measurable metabolic costs. In contrast, the thermic effect of intravenously administered glucose is only half of that after a glucose meal (7). It is not known whether the thermic effect of intravenous fat differs from that after oral fat.

Half of the thermogenesis induced by protein meals or intravenous amino acid infusions occurs in the splanchnic tissues, probably representing the metabolic costs for hepatic processing of amino acids (10, 11). Carbohydrates (oral glucose, oral fructose, or iv glucose) stimulate the net oxygen uptake exclusively in extrasplanchnic tissues when administered after an overnight fast (5, 7, 9). The splanchnic metabolic costs for glucose absorption are counterbalanced by a simultaneous inhibition of the metabolic costs for the hepatic gluconeogenesis going on in the basal state (7). The inhibition of hepatic gluconeogenesis was illustrated by a significant reduction of the splanchnic oxygen consumption during intravenous glucose infusion (7). The absorption of a fructose meal did not cause any measurable increase in splanchnic oxygen uptake (9). After a mixed meal, 40–45% of the rise in whole body oxygen uptake occurs in the splanchnic tissues (5, 8, 18). The splanchnic proportion of the weak thermogenic response to fat administration is unknown.

Also with regard to blood flow stimulation, the nutrients act differently. Glucose-containing mixed meals or pure glucose meals stimulate both splanchnic and extrasplanchnic blood flow (5–9), but pure fructose does not (9). Both orally ingested protein and intravenously infused amino acid mixtures moderately stimulate the blood flow in the extrasplanchnic tissues and cause a considerably hypokinetic splanchnic circulation, i.e., a low regional blood flow in relation to the simultaneous regional oxygen consumption (10, 11). Oralysed glucose stimulates strongly splanchnic blood flow, whereas intravenously infused glucose does not (7). By ultrasound techniques, orally ingested fat has been found to stimulate blood flow in the superior mesenteric artery (23). To what extent human cardiac output and total splanchnic blood flow are affected by oral or intravenous administration of fat seems not to have been reported.

The mechanisms by which the nutrients stimulate blood flow in different vascular beds are incompletely understood. After glucose-containing meals, both the whole body and splanchnic circulatory stimulation is most marked during the first postprandial hour (4, 5–9). However, in patients with tetraplegia due to complete cervical spinal cord lesions, oral glucose caused only a relatively weak stimulation of cardiac output and no significant increase in splanchnic blood flow (1). Nonselective β-adrenergic receptor inhibition markedly reduces the glucose-induced rise in splanchnic blood flow (6). The findings suggest that efferent nervous activity (possibly activation of sympathoadrenal system) and a permissive action by intestinal hormones are involved in the mechanisms behind the glucose-induced circulatory changes (1, 5, 6).

The present study was undertaken to compare the whole body and splanchnic thermogenic and circulatory responses to orally vs. intravenously administered fat in healthy subjects.

METHODS

Twelve healthy male volunteers participated in the study. Seven subjects (age 28 ± 2 yr, height 1.85 ± 0.02 m, and weight 77 ± 4 kg) were studied before and during 2.5 h after oral ingestion of 23 g of vegetable oil, corresponding to 850 kJ
of fat energy. The results were compared with those from a group of five healthy men (age 29 ± 3 yr, height 1.81 ± 0.01 m, and weight 80 ± 3 kg) studied before and during a 2.5-h intravenous infusion of 850 kJ of fat energy in the form of 85% soy oil, 10% glycerol, and 5% egg phospholipids (Emulsan, 200 mg/ml, Leras Oy, Abo, Finland). The subjects reported to the laboratory in the morning after an overnight fast of 12–14 h. They were studied in the supine position, comfortably dressed in a cotton shirt and shorts and covered by a thin blanket. The room temperature was 20–22°C. Respiratory gas exchange and energy expenditure were measured for 8-min periods by means of continuous breath-by-breath analysis (MedGraphics System CPX/D, Medical Graphics, St. Paul, MN) using a noseclip-and-mouthpiece technique with which the subjects were previously familiar.

Table 1. Pulmonary O₂ uptake, respiratory exchange ratio, whole body energy expenditure, arterial-pulmonary arterial O₂ difference, cardiac output, heart rate, stroke volume, splanchnic O₂ uptake, arterial-hepatic venous O₂ difference, splanchnic blood flow, and arterial and hepatic venous hemoglobin concentration

<table>
<thead>
<tr>
<th>Minutes</th>
<th>Pulmonary O₂ uptake, ml/min</th>
<th>Respiratory exchange ratio</th>
<th>Wholebody energy expenditure, W</th>
<th>Cardiac output, l/min</th>
<th>Stroke volume, ml</th>
<th>Splanchnic blood flow, l/min</th>
<th>Arterial hemoglobin concentration, g/l</th>
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<tr>
<td>Oral</td>
<td>242 ± 11</td>
<td>0.82 ± 0.03</td>
<td>82 ± 3</td>
<td>54 ± 2</td>
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<td>1.06 ± 0.09</td>
<td>140 ± 3</td>
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<td>iv</td>
<td>223 ± 9</td>
<td>0.82 ± 0.03</td>
<td>75 ± 2</td>
<td>49 ± 2</td>
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<td>1.07 ± 0.09</td>
<td>146 ± 6</td>
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Values are means ± SE for 7 men before and after oral ingestion of 850 kJ of fat (oral) and in 5 men before and during intravenous infusion of 850 kJ of fat (iv). Statistically significant difference from basal (paired t-test): *P < 0.05; †P < 0.01; ‡P < 0.001.
Two venous catheters were inserted under local anesthesia and fluoroscopic control. One thermistor-equipped catheter (Edslab 93A-131–7F, Edwards Laboratories, Santa Ana, CA) was introduced from an antecubital vein into the pulmonary artery and another from the right femoral vein into a right-sided hepatic vein. A thin catheter was inserted percutaneously into a brachial artery.

Immediately after the catheterization procedure, arterial blood was drawn for zero-point analysis of indocyanine dye (Cardio-Green, Hynson, Westcott & Dunning Products, Beckton-Dickinson, Cockeysville, MD), which was then infused into the right atrium via the side hole of the pulmonary arterial catheter. The infusion was continued for 40–60 min to achieve steady-state plasma concentrations before the baseline blood flow measurements were performed. During this 45- to 60-min period of indocyanine infusion and recovery after the catheterization, recording of the blood temperatures was started and continued during the entire study period.

The baseline measurements were performed twice with a 10-min interval. After that, the fat was given, either by oral ingestion of oil, followed by 100 ml of water at 36.5°C (duration of intake 1–3 min), or by infusion of Emulsan (0.67 ml/min, during 2.5 h) into the right atrium via the side hole of the pulmonary arterial catheter.

Blood samples were drawn at timed intervals from the catheters for analyses of oxygen content, hematoglobin, indocyanine green, glucose, insulin, C-peptide, free fatty acids (FFA), and glycerol concentrations.

The splanchnic blood flow was estimated by the continuous indocyanine infusion technique (3, 24). The blood temperatures were recorded continuously from the thermistor-equipped catheters at a sampling frequency of 1 Hz as described earlier (8). The blood thermometer is capable of detecting small temperature variations, the absolute measuring accuracy being ±0.001°C and the sensitivity, 0.0003°C. All subjects were informed of the nature, purpose, and possible risks of the study before giving their voluntary consent to participate. The study protocol was reviewed and approved by the institutional ethics committee.

Analyses. Plasma concentrations of indocyanine green were estimated by HPLC technique (12). Blood contents of oxygen and immediately after catheterization. Plasma concentrations of insulin and C-peptide were determined fluorometrically according to Wieland (28). FFA were determined fluorometrically according to Wieland (28).

Calculations. The whole body energy expenditure was calculated from the respiratory gas exchange (20, 29). Cardiac output, regional splanchnic oxygen uptake, and extrasplanchnic arteriovenous oxygen difference were calculated according to the Fick principle (14). The means of the values obtained at -10, -5, and 0 min were used as basal values when changes were calculated from the basal state.

The values for oxygen uptake and blood flow in extrasplanchnic tissues were calculated from the individual differences in whole body and splanchnic oxygen uptake and blood flow, respectively. The plasma volume was calculated from the central hematocrit measured in arterial blood and the individual blood volumes according to body dimensions (27). Changes in the whole body heat content were calculated from the individual body weights and changes in mixed venous blood temperature, using the traditional normal value for whole body specific heat, 3.474 kJ·°C⁻¹·kg⁻¹ (22). The splanchnic (hepatic) extraction of insulin (%) was calculated from the splanchnic release of insulin and C-peptide according to the formula \((\frac{I_A \times Q + C_{PHV-A} \times Q}{Q} - I_{HV} \times Q)\) \(I_A \times Q + C_{PHV-A} \times Q\), where \(I_A\) is the arterial plasma concentration of insulin (pM), \(Q\) is the splanchnic plasma flow (l/min), \(I_{HV}\) is the hepatic venous plasma concentration of insulin (pM), \(C_{PHV-A}\) is the arterial plasma concentration of C-peptide (pM), and \(C_{HV}\) is the hepatic venous plasma concentration of C-peptide (pM). The sample frequency did not allow for a proper calculation of the extrasplanchnic insulin extraction.

Statistics. Significant changes from basal within a group were calculated by the Student’s paired t-test. When differences were compared between the groups, data were first analyzed by repeated-measures ANOVA, and differences were calculated by post hoc testing (25). Data are presented throughout as means ± SE.

RESULTS

Pulmonary oxygen consumption, respiratory exchange ratio, and whole body energy expenditure. In the basal state, before fat administration, the pulmonary oxygen uptake, respiratory exchange ratio, and energy expenditure were within the basal ranges calculated from body dimensions and age (15) and were similar in the intravenous and oral groups (Table 1). During the first 90 min of fat load, the pulmonary oxygen uptake showed a small but significant increase (Fig. 1). The respiratory exchange ratio fell significantly, particularly in the intravenous group (Fig. 2). Reduced by the fall in respiratory exchange ratio, the whole body energy expenditure changed little (Fig. 3). Thus the calculated thermic effect of fat was nonsignificant except for the last measurement at 150 min of fat load.

Arteriovenous arterial oxygen difference, cardiac output, heart rate, and stroke volume. In the basal state, all these variables were well within normal resting ranges (Table 1). The arteriovenous arterial oxygen difference, cardiac output, and stroke volume were essentially unchanged during the fat loads. Heart rate increased significantly from 45 min after...
oral fat ingestion (Table 1) but not during intravenous fat infusion.

Hemoglobin concentration and plasma volume. The hemoglobin concentration was in the basal state 140 ± 3 and 146 ± 5 g/l in the oral and intravenous groups, respectively. After oral fat, the arterial and hepatic venous hemoglobin concentrations increased significantly (Table 1), reflecting a reduction of the plasma volume (Fig. 4), corresponding to an increment in lymph production of ~200 ml between 30 and 90 min after the fat meal. The plasma volume returned to its basal magnitude at 150 min after the meal. Intravenous fat infusion did not affect the hemoglobin concentration, neither in arterial nor in hepatic venous blood (Table 1).

Splanchnic oxygen uptake. In the basal state, the splanchnic tissues accounted for 22 ± 6 and 23 ± 2% (not significant) of the simultaneous whole body oxygen consumption in the oral and intravenous groups, respectively (Table 1). During the 2.5 h of fat load, the splanchnic oxygen consumption tended to rise, but the average 2.5-h postprandial splanchnic oxygen consumption did not differ significantly from the basal level in any group (Table 1).

Splanchnic blood flow and arterial-hepatic venous oxygen difference. In the basal state, the splanchnic blood flow accounted for 20 ± 1 and 24 ± 1% of cardiac output in the oral and intravenous groups, respectively (Table 1). After oral fat, the splanchnic blood flow increased markedly, reaching a maximal value at 90 min after the meal (Fig. 5). Simultaneously, the arterial-hepatic venous oxygen difference fell considerably in the oral group (Table 1) and was still significantly below its basal level at 150 min after the meal. Intravenous infusion of fat affected neither the splanchnic blood flow nor the arterial-hepatic venous oxygen difference (Table 1).

Glucose. In the basal state, the arterial and hepatic venous blood glucose concentrations were well within normal basal limits in both groups (Table 2). The basal splanchnic release of glucose was 1.06 ± 0.17 and 0.82 ± 0.12 mmol/min in the oral and intravenous groups, respectively. At 60 min after oral fat, the glucose concentration showed a small but significant
reduction from its basal levels both in arterial and hepatic venous blood. The splanchnic glucose release was essentially unchanged in both the oral and intravenous groups (Table 2).

Insulin. In the basal state, the plasma concentrations and splanchnic release of insulin were well within the basal ranges in both groups (Table 2). After oral fat ingestion, the arterial plasma insulin concentration increased significantly (Fig. 6). The average postprandial arterial plasma insulin concentration was 36 ± 6% higher than the basal level. Simultaneously, the splanchnic release of insulin tended to be lower than that in the basal state (Fig. 7). During intravenous fat infusion, the arterial concentration and splanchnic release of insulin were essentially unchanged (Table 2). C-peptide and splanchnic (hepatic) extraction of insulin. Arterial and hepatic venous plasma concentrations of C-peptide were within the normal ranges in the basal state and showed only small changes during the fat load period. In the oral group, the splanchnic release of C-peptide, 79 ± 10 pmol/min in the basal state, did not change significantly after the fat meal. The splanchnic (hepatic) extraction of insulin, 65 ± 6% in the basal state, tended to fall (40 ± 10% at 120 min after fat meal), but the change did not attain statistical significance.

### Table 2. Arterial concentrations, arterial-hepatic venous differences, and splanchnic release and uptake of glucose, insulin, glycerol, and FFA

<table>
<thead>
<tr>
<th>Minutes</th>
<th>–10</th>
<th>0</th>
<th>15</th>
<th>30</th>
<th>60</th>
<th>90</th>
<th>120</th>
<th>150</th>
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<tr>
<td><strong>Arterial glucose concentration, mM</strong></td>
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<td>Oral</td>
<td>4.58 ± 0.05</td>
<td>4.58 ± 0.04</td>
<td>4.52 ± 0.05</td>
<td>4.43 ± 0.05</td>
<td>4.35 ± 0.09*</td>
<td>4.51 ± 0.08</td>
<td>4.61 ± 0.09</td>
<td>4.65 ± 0.07</td>
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<td>iv</td>
<td>4.75 ± 0.09</td>
<td>4.79 ± 0.08</td>
<td>4.82 ± 0.06</td>
<td>4.85 ± 0.05</td>
<td>4.81 ± 0.07</td>
<td>4.81 ± 0.05</td>
<td>4.81 ± 0.06</td>
<td>4.78 ± 0.04</td>
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<td><strong>Hepatic venous-arterial glucose difference, mM</strong></td>
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<tr>
<td>Oral</td>
<td>1.06 ± 0.22</td>
<td>1.13 ± 0.30</td>
<td>0.87 ± 0.19</td>
<td>0.76 ± 0.19</td>
<td>0.87 ± 0.22</td>
<td>0.85 ± 0.33</td>
<td>0.70 ± 0.20</td>
<td>0.62 ± 0.16*</td>
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<td>iv</td>
<td>0.65 ± 0.05</td>
<td>0.67 ± 0.07</td>
<td>0.64 ± 0.09</td>
<td>0.58 ± 0.06</td>
<td>0.59 ± 0.07</td>
<td>0.70 ± 0.12</td>
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<td><strong>Splanchnic release of glucose, pmol/min</strong></td>
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<tr>
<td>Oral</td>
<td>1.04 ± 0.15</td>
<td>1.09 ± 0.22</td>
<td>1.06 ± 0.22</td>
<td>1.03 ± 0.17</td>
<td>1.36 ± 0.33</td>
<td>1.59 ± 0.62</td>
<td>1.11 ± 0.28</td>
<td>0.97 ± 0.23</td>
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<tr>
<td>iv</td>
<td>0.83 ± 0.13</td>
<td>0.81 ± 0.12</td>
<td>0.78 ± 0.11</td>
<td>0.73 ± 0.09</td>
<td>0.72 ± 0.09</td>
<td>0.81 ± 0.12</td>
<td>0.72 ± 0.08</td>
<td>0.77 ± 0.12</td>
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| Values are means ± SE for same procedures and subjects as in Table 1. FFA, free fatty acids. Statistically significant difference from basal (paired t-test): *P < 0.05; †P < 0.01; ‡P < 0.001.
Glycerol. In response to oral fat ingestion, the arterial and hepatic venous concentrations of glycerol fell significantly, whereas the net splanchnic glycerol uptake was unchanged (Table 2). During intravenous infusion of fat and glycerol, the blood concentrations and the splanchnic uptake of glycerol increased (Table 2).

FFA. After oral fat ingestion, the arterial concentrations of FFA decreased significantly during 30–90 min after the meal (Fig. 8). Simultaneously, the hepatic venous plasma concentration of FFA was unchanged (Table 2). During intravenous fat infusion, the arterial and hepatic venous plasma concentrations of FFA increased significantly, whereas the net splanchnic uptake remained unchanged (Table 2).

Blood temperature. Oral fat ingestion was not accompanied by any significant changes of the blood temperature. Intravenous fat infusion increased the mixed venous blood temperature by 0.23°C ± 0.06°C, corresponding to an increase in whole body heat content of 67 ± 20 kJ (P < 0.05). The hepatic venous-arterial temperature difference did not change significantly.

DISCUSSION

Oral ingestion of 850 kJ of fat was found to elicit several circulatory and metabolic processes that seem not to have been described earlier. The splanchnic blood flow increased immediately and reached levels almost twice those in the basal state. The central hemoglobin concentration rose, indicating a loss of ~0.2 liters of the plasma volume into the extravascular space. Provided that all this fluid represented an increased lymph production, it would constitute ~12% of our daily lymph flow. The arterial concentrations of FFA and glycerol fell considerably, indicating an immediate inhibition of lipolysis from extrasplanchnic fat stores. The simultaneously unchanged hepatic venous concentration of FFA indicates no net inhibition of the lipolysis in the splanchnic fat stores. In this context, it should be noted that the splanchnic turnover of FFA could not be calculated from the variables measured in this study.

The arterial insulin concentration rose to postprandial levels significantly higher than in the basal state, although the postprandial splanchnic release of insulin was below its basal level. These findings may suggest that the extrasplanchnic extraction of insulin was considerably reduced after fat ingestion. In addition, the postprandial splanchnic release of C-peptide showed no significant differences from the fasting values.

The circulatory and metabolic effects occurred promptly after the fat was ingested, long before any significant amounts of fat could have been absorbed. This indicates that the marked effects were due to a hormonal or neural response, elicited by the deposition of fat onto the mucous membranes of the gastrointestinal tract. It is well known that the duodenal mucosa is capable of producing several hormones, at least in response to glucose deposition in the duodenum (see Ref. 13). Further studies are needed to estimate which of the possible duodenal hormones (or nerves) accounted for the marked splanchnic vasodilation, the increased lymph production, the apparent inhibition of extrasplanchnic insulin extraction, and the inhibition
of extrasplanchnic lipolysis, possibly secondary to the increased insulin concentration.

The splanchnic release of insulin tended to be slightly (not significantly) above its basal level at 30 min after the meal, but the average postprandial release was below the basal level. Nevertheless, the arterial concentration of insulin increased significantly after fat ingestion. However, the sample frequency used in the present study did not allow for a proper calculation of the extrasplanchnic insulin extraction. Therefore no quantitative calculation of insulin kinetics were performed. The splanchnic (hepatic) insulin extraction tended to fall at 2 h after the fat meal but did not change significantly. It might be argued that an early increment of the splanchnic insulin release would have been overlooked if it occurred before the first postprandial blood samples were drawn (at 15 min after meal). On the other hand, if so, the blood glucose concentration would have been expected to vary more than it did.

The physiological importance of the processes induced by oral fat ingestion cannot be judged from the present data. It is, however, conceivable that the increased blood flow and increased lymph production after fat intake may facilitate the absorption of nutrients, especially fat chylomicrons. The inhibition of extrasplanchnic lipolysis, possibly caused by the elevated insulin concentration in arterial plasma, may contribute to reduce the postprandial blood concentrations of FFA and glycerol.

By ultrasound technique, increased blood flow has been described in the superior mesenteric artery after fat ingestion (23). By use of the same technique, high-fat mixed meals were found to stimulate the mesenteric artery blood flow to a greater extent than did low-fat mixed meals (26).

Compared with orally ingested fat, intravenously infused fat and glycerol caused different physiological effects. The significant reduction of the respiratory exchange ratio that occurred tended to be somewhat more marked during intravenous than after oral fat, indicating that the intravenously infused fat stimulated whole body fat oxidation more promptly. This effect may reflect the more rapid appearance of fatty acids in the blood during intravenous infusion.

The present finding of a very low and nonsignificant thermic effect of fat is well in line with earlier findings (see Ref. 19).

Intravenous fat infusion significantly increased the whole body heat content. All the extra heat produced during the 2.5-h fat infusion was accumulated as stored heat. However, the splanchnic heat production was not stimulated significantly. No thermal effects occurred in response to oral fat ingestion.

It is concluded that oral fat ingestion stimulates markedly splanchnic blood flow, reduces the plasma volume (suggesting an increased lymph production), reduces the arterial concentrations of FFA and glycerol, and increases the arterial plasma concentration of insulin by reducing the extrasplanchnic insulin extraction. Intravenous fat infusion did not affect these variables but reduced the respiratory exchange ratio and increased the body heat content. It is suggested that intestinal hormones contribute to the effects observed after oral fat ingestion.

This study was supported by a grant from the Swedish Medical Research Council Project 3108.

Address for reprint requests: T. Brundin, Dept. of Clinical Physiology, Karolinska Hospital, S-171 76 Stockholm, Sweden.

Received 6 October 1997; accepted in final form 19 December 1997.

REFERENCES


