Metabolic effects of the use of protein-sparing infusions in postoperative patients

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(Received 13 March 1979; accepted 31 December 1979)

Summary

1. The mechanism of the purported protein-sparing effects of different postoperative intravenous regimens still remains controversial. We have therefore measured circulating concentrations of metabolites and hormones in blood and urine nitrogen excretion in patients receiving (a) sodium chloride solution (saline), (b) 1.5 g of glucose day\(^{-1}\) kg\(^{-1}\) body weight, (c) 1.5 g of glucose day\(^{-1}\) kg\(^{-1}\) with insulin (0.56 unit day\(^{-1}\) kg\(^{-1}\)) or (d) mixed amino acids (1.5 g day\(^{-1}\) kg\(^{-1}\)) after abdominal operation.

2. Compared with results for saline-treated patients, glucose infusion resulted in raised glucose and insulin concentrations and lowered ketone body and non-esterified fatty acid concentrations but did not influence protein catabolism.

3. Addition of insulin lowered blood glucose by approximately 1 mmol/l. Total nitrogen excretion during glucose/insulin infusion was significantly less than during saline infusion.

4. Infusion of amino acids, compared with saline infusion, resulted in raised blood glucose, alanine, serum insulin and plasma glucagon concentrations but lower concentrations of plasma non-esterified fatty acids and blood ketone bodies. Insulin concentrations, however, were similar in both amino acid- and glucose-treated groups.

5. Amino acid infusion increased urea and total nitrogen excretion but net nitrogen loss was only 1.9 mmol of nitrogen day\(^{-1}\) kg\(^{-1}\) compared with 12.7 mmol day\(^{-1}\) kg\(^{-1}\) in the saline-treated group and 11.0 mmol day\(^{-1}\) kg\(^{-1}\) in the glucose-treated group.

6. Glucose (and insulin) infusion appeared to inhibit gluconeogenesis, and amino acids to enhance it. The nitrogen-sparing effect of amino acids appears largely related to their mass and is apparently unrelated to changes in ketone bodies and insulin.

Key words: alanine, amino acids, glucagon, glucose, insulin, ketone bodies, 3-methylhistidine, nitrogen, surgery.

Introduction

Surgery is well known to increase protein catabolism (Kinney, Long, Gump & Duke, 1968). Frequently, surgical patients have low amounts of body proteins before operation (Hill, Pickford, Young, Schorah, Blackett, Burkinshaw, Warren & Morgan, 1977). Consequently, protein depletion after operation may be both common and severe. Such protein depletion is widely believed to be associated with impaired wound healing (Kobak, Benditt, Wissler & Steffee, 1947) and increased susceptibility to infection (Spanier, Pietsch, Meakins, MacLean & Shizgal, 1976). In general, oral intake of protein is initially precluded after abdominal surgery and therefore methods of limiting the catabolism of endogenous amino acids...
are desirable. There is a need for more simple therapy than total parenteral nutrition.

Blackburn, Flatt, Clowes & O'Donnell (1973) reported that infusion of an isotonic solution of amino acids via a peripheral venous cannula resulted in a marked decrease in negative nitrogen balance compared with an approximately equicaloric glucose infusion. They described this as 'protein sparing', ascribing it to the presence of relatively raised circulating ketone body and non-esterified fatty acid concentrations. They argued that these lipid fuels would then be used preferentially by tissues, thus decreasing catabolism of amino acids for energy. However, this hypothesis has been challenged by others (Felig 1976; Tweedle, Fitzpatrick, Brennan, Culebras, Wolfe, Ball & Moore 1977). Glucose infusion increases insulin secretion and inhibits gluconeogenesis and ureagenesis from amino acids. Thus, in normal subjects, as little as 0.55 mol (100 g) of glucose daily will markedly decrease nitrogen excretion (Gamble, 1946). In view of this controversy, and in search of a simple protein-sparing therapy, we have reassessed the metabolic actions of amino acids and glucose with and without additional insulin upon nitrogen balance in postoperative subjects, and have compared each therapy with results of sodium chloride infusion alone.

Methods

Procedure

Patients undergoing elective abdominal surgery were randomly allocated to one of four groups who were given infusions of: (1) 2–3 litres of sodium chloride solution (0.15 mol/l) daily, (2) 0.24 mol of glucose/l (43 g/l) in sodium chloride solution (0.03 mol/l), providing 1.5 g of glucose day−1 kg−1 body weight, (3) the same solution as (2) but with the addition of 16 units of soluble insulin/l (0.56 unit day−1 kg−1 body weight) or (4) 3.5% Synthamin solution (Travenol Laboratories Ltd., Thetford, Norfolk, U.K.), containing 35 g of mixed amino acids, 35 mmol of sodium/l and 30 mmol of potassium/l, supplying 1.5 g of mixed L-amino acids day−1 kg−1 body weight. Each litre of sodium chloride solution (saline) or glucose/saline contained 27 mmol (2 g) of potassium chloride. Infusions were given for 72 h at a constant rate from 09.00 hours on the first postoperative day. During operation and until 09.00 hours on the first postoperative day all patients received 2–3 litres of the glucose/saline solution, providing 0.48–0.72 mol (86–129 g) of glucose.

Patients received standard hospital diets after admission to hospital and before the operation day. After an overnight fast anaesthesia was induced with thiopentone and suxamethonium and maintained with nitrous oxide, halothane and tubocurarine. Nothing was taken by mouth during the study period.

Patients

Details of patients are given in Table 1. All patients gave informed consent for the procedures, which had been approved by the local Ethical Committee. All the patients underwent elective

### Table 1. Characteristics of patients studied after elective abdominal surgery

<table>
<thead>
<tr>
<th>Infusion</th>
<th>Saline*</th>
<th>Glucose</th>
<th>Glucose/insulin</th>
<th>Amino acids</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>n</strong></td>
<td>6</td>
<td>8</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td><strong>Age (years)</strong></td>
<td>58 ± 5</td>
<td>59 ± 2</td>
<td>67 ± 2</td>
<td>61 ± 3</td>
</tr>
<tr>
<td><strong>Sex ratio (male:female)</strong></td>
<td>2:4</td>
<td>5:3</td>
<td>2:4</td>
<td>4:2</td>
</tr>
<tr>
<td><strong>Body wt. (kg)</strong></td>
<td>65 ± 10</td>
<td>64 ± 6</td>
<td>61 ± 7</td>
<td>66 ± 5</td>
</tr>
<tr>
<td><strong>Ideal body wt.† (%)</strong></td>
<td>104 ± 6</td>
<td>103 ± 4</td>
<td>101 ± 7</td>
<td>107 ± 3</td>
</tr>
<tr>
<td><strong>Lean body wt. (kg)</strong></td>
<td>49 ± 6</td>
<td>50 ± 7</td>
<td>45 ± 5</td>
<td>49 ± 5</td>
</tr>
<tr>
<td><strong>Diagnoses</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastric ulcer (1)</td>
<td>Gastric ulcer (2)</td>
<td>Carcinoma stomach (2)</td>
<td>Duodenal ulcer (1)</td>
<td></td>
</tr>
<tr>
<td>Duodenal ulcer (1)</td>
<td>Duodenal ulcer (3)</td>
<td>Carcinoma colon (4)</td>
<td>Carcinoma colon (4)</td>
<td></td>
</tr>
<tr>
<td>Carcinoma stomach (1)</td>
<td>Carcinoma stomach (1)</td>
<td>Carcinoma pancreas (1)</td>
<td>Carcinoma colon (1)</td>
<td></td>
</tr>
<tr>
<td>Carcinoma colon (3)</td>
<td>Carcinoma colon (1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Operation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partial gastrectomy (3)</td>
<td>Partial gastrectomy (4)</td>
<td>Carcinoma colon (4)</td>
<td>Carcinoma colon (1)</td>
<td></td>
</tr>
<tr>
<td>Partial colectomy (3)</td>
<td>Partial colectomy (6)</td>
<td>Carcinoma colon (4)</td>
<td>Carcinoma colon (1)</td>
<td></td>
</tr>
<tr>
<td>Partial pancreactomy (1)</td>
<td>Partial pancreactomy (1)</td>
<td>Carcinoma colon (1)</td>
<td>Carcinoma colon (1)</td>
<td></td>
</tr>
<tr>
<td>Partial colectomy (1)</td>
<td></td>
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</tr>
</tbody>
</table>

* Previously reported (Foster et al., 1979).
† Metropolitan Life Insurance Co., 1959.
abdominal surgery; none suffered from serious hepatic, renal, cardiorespiratory or endocrine dysfunction; none was on concomitant drug therapy, and none had postoperative complications during the period of the study. Ideal body weight was calculated from the midpoint of the range quoted for subjects of medium build in the Metropolitan Life Insurance Company tables (Documenta Geigy, 1962). Lean body mass was estimated from skinfold thicknesses by the technique of Durnin & Womersley (1974). Some of the data on the saline control group have been published previously (Foster, Alberti, Binder, Hinks, Karran, Smythe, Talbot, Turnell & Ørskov, 1979).

Methods

Free flowing venous blood samples (15 ml each) were taken at 09.00 hours daily and at 21.00 hours on the first postoperative day, after patients had rested supine for at least 1 h. Blood (1.5 ml) was immediately deproteinized in 5 ml of ice-cold 5% (v/v) perchloric acid and the supernatant separated by centrifugation for fluorimetric assay of glucose, lactate, pyruvate, alanine, glycerol and 3-hydroxybutyrate (Lloyd, Burrin, Smythe & Alberti, 1978) and spectrophotometric assay of acetoacetate (Price, Lloyd & Alberti, 1977). Plasma was rapidly separated for assay of non-esterified fatty acids by a modification of the method of Ho & Meng (1969). Serum was used for assay of insulin by a double-antibody method (Soeldner & Slone, 1965) and cortisol by a competitive protein-binding technique (Kehlet, Binder & Engbaek, 1974). For glucagon, 2.5 ml of blood was taken into tubes containing 2500 i.u. of aprotinin and 2.5 μmol of EDTA (sodium salt) and the plasma rapidly separated for subsequent assay by a wick-chromatography method (Ørskov, Thomsen & Yde, 1968). Plasma urea, electrolytes and albumin were measured by routine automated methods.

Three consecutive 24 h urine collections were made during the 72 h test infusion period with 25 ml of hydrochloric acid (5 mol/l) as preservative. Total urine nitrogen was measured by a micro-Kjeldahl technique, urea by the urease/Berthelot reaction and creatinine by the method of Bartels & Bohmer (1971). A Technicon TSM amino acid analyser was used to measure urine 3-methylhistidine excretion, with a lithium citrate buffer system.

Calculations

Total body urea production was calculated as urine urea excretion plus change in body urea (Lee, 1974), and concentrations of ketone bodies refer to the sums of the concentrations of acetoacetate and 3-hydroxybutyrate. Results are shown as means ±1 SEM. Statistical comparisons were made by paired and unpaired Student’s t-tests, logarithmic transformation being used to decrease the skew of grossly positively skewed data. The saline-treated group were regarded as controls for the glucose and amino acid infusions, and the glucose group as control for the glucose/insulin infusion. Linear correlations were sought between circulating concentrations of metabolites and hormones. The chi-squared test was used to assess the frequency of phlebitis in the amino acid group.

Results

Table 1 shows that the patient groups had similar age, weight, lean body masses and underwent operations of similar severity.

Nitrogen metabolism

Nitrogen balance in the treatment groups is compared in Table 2. There were no significant differences between the saline- and glucose-treated groups, but the glucose/insulin group had lower urea production (P < 0.01) and creatinine excretion (P < 0.05) than the saline-treated control group. Amino acid infusion significantly increased total nitrogen excretion (P < 0.02) and urea production (P < 0.01), but net nitrogen loss in this group (exclusive of cutaneous losses) was only 1.9 mmol of nitrogen day⁻¹ kg⁻¹ body weight, by far the lowest for all the groups studied. Excretion of 3-methylhistidine was similar in all four groups.

Infusion of amino acids was associated with a small rise in plasma urea concentration (P < 0.05) whereas there was a tendency for a fall in mean plasma urea during the other infusions, which was significant for the glucose/insulin group (P < 0.05) (Table 3). Plasma albumin concentration fell significantly during glucose and glucose/insulin infusion but not during saline or amino acid infusion.

Blood metabolites and hormones (Table 3)

Before operation there were only minor differences in blood metabolite concentrations between groups and all variables were within the reference range for overnight fasted subjects for this laboratory (Foster, Alberti, Hinks, Lloyd, Postle, Smythe, Turnell & Walton 1978b). After
operation, during glucose infusion and before the test infusion (09.00 hours, day 1) there were, as expected, elevations in concentration of lactate, pyruvate, glucose, glycerol, insulin, glucagon and cortisol (data not shown). Insulin values were slightly lower in the glucose and glucose/insulin groups than in the saline group, and glucagon values were lower in the saline group than in the other groups.

Results obtained 24 and 72 h after start of the test infusions are shown in Table 3.

**Saline infusion.** During saline infusion there were marked declines in glucose, alanine and insulin concentrations and rises in concentrations of the lipid metabolites, particularly ketone bodies. Cortisol concentrations at 09.00 hours on days 3 and 4 were higher than the pre-operative value (P < 0.05) and at 21.00 hours on day 1 (12 h after onset of the test period) the mean cortisol concentration (523 ± 111 mmol/l) was higher than expected, implying loss of diurnal variation. Plasma glucagon concentration rose to a peak of 132 ± 12 ng/l at 21.00 hours on day 1 but declined subsequently to the pre-operative value by day 4.

**Glucose infusion.** Patients given an infusion of glucose alone had significantly higher concentrations of blood glucose and serum insulin and lower concentrations of ketone bodies and glycerol than the saline-treated control group at nearly all sampling times. Particularly notable were the ketone body concentrations, which rose to 2.94 mmol/l in the saline-treated group by the fourth postoperative day (day 4) compared with only 0.32 mmol/l in the glucose-treated group (P < 0.001). Glucagon concentrations were not significantly different during the test infusions. Lactate and pyruvate concentrations were also slightly higher in the glucose-treated group.

**Glucose and insulin.** Infusion of insulin with glucose resulted in a slightly lower blood glucose concentration than in the infusion group receiving glucose alone, but values were still higher than in the saline-treated group. Over the whole of the infusion period, the mean blood glucose (5.6 ± 0.3 mmol/l) was lower than that during infusion of glucose alone (6.7 ± 0.2 mmol/l, P < 0.01), and the overall mean insulin concentration (average of all values during the test period) was significantly higher (19 ± 3 munits/l, and 10 ± 1 munits/l, P < 0.01). Insulin values were on average fivefold those in the saline-treated control group.

Surprisingly, there was a small increase in blood ketone body concentrations in the insulin-treated group, significant on day 3 (0.44 ± 0.14 mmol/l, compared with 0.14 ± 0.03 mmol/l, P < 0.05), but values before operation were also higher in the insulin-treated group. Plasma glucagon was also significantly higher in this group on day 3 (124 ± 14 ng/l) than in the glucose group (81 ± 13 ng/l, P < 0.05).

**Amino acids.** Amino acid infusion was associated with higher glucose and insulin concentrations and lower glycerol, non-esterified fatty acid and ketone body infusions than saline infusion. Insulin concentrations were similar to those observed during glucose infusion. Despite this, blood ketone body concentrations were significantly higher during amino acid than during glucose infusion, although the final value in the amino acid group on day 4 was still only 17% of that found after saline infusion. Conversely, plasma non-esterified fatty acid concentration fell during amino acid therapy and the overall mean concentration (0.33 ± 0.04 mmol/l) was less than during glucose infusion (0.51 ± 0.04 mmol/l, P < 0.05). Two notable findings during amino acid infusion were
**Table 3. Blood concentrations of metabolites and hormones 24 and 72 h after start of test infusions (48 and 96 h postoperatively)**

Numbers of subjects are shown in parentheses. Test infusions were commenced at 09.00 hours on the day after operation. In the previous 24 h patients received 86–129 g of glucose. Significance: *P* < 0.05; **P** < 0.01, compared with saline-treated control. †Glucose/insulin group significantly different from glucose group: *P* < 0.05.

<table>
<thead>
<tr>
<th>Infusion</th>
<th>Pre-operation (overnight fast)</th>
<th>48 h postoperatively</th>
<th>96 h postoperatively</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mmol/l)</td>
<td>5.1 ± 0.3 ± 5.0 ± 4.8 ±</td>
<td>5.1 ± 0.6 ± 4.1 ± 4.4 ±</td>
<td>0.4 ± 0.7 ± 4.8 ± 5.7 ±</td>
</tr>
<tr>
<td>Lactate (mmol/l)</td>
<td>0.3 ± 0.1 ± 0.3 ± 0.2 ±</td>
<td>0.3 ± 0.2 ± 0.8 ± 0.2 ±</td>
<td>0.7 ± 0.3 ± 0.5 ± 0.1 ±</td>
</tr>
<tr>
<td>Pyruvate (mmol/l)</td>
<td>0.6 ± 0.7 ± 0.7 ± 0.6 ±</td>
<td>0.6 ± 0.8 ± 0.9 ± 0.6 ±</td>
<td>0.6 ± 0.6 ± 0.7 ± 0.6 ±</td>
</tr>
<tr>
<td>Alanine (mmol/l)</td>
<td>0.06 ± 0.08 ± 0.07 ± 0.05 ±</td>
<td>0.06 ± 0.08 ± 0.08 ± 0.06 ±</td>
<td>0.05 ± 0.04 ± 0.07 ± 0.01 ±</td>
</tr>
<tr>
<td>Glycol (mmol/l)</td>
<td>0.37 ± 0.26 ± 0.30 ± 0.28 ±</td>
<td>0.18 ± 0.21 ± 0.18 ± 0.26 ±</td>
<td>0.15 ± 0.16 ± 0.16 ± 0.28 ±</td>
</tr>
<tr>
<td>Glycerol (mmol/l)</td>
<td>0.05 ± 0.02* ± 0.02 ± 0.03 ±</td>
<td>0.02 ± 0.02 ± 0.02 ± 0.03* ±</td>
<td>0.01 ± 0.01 ± 0.01 ± 0.03* ±</td>
</tr>
<tr>
<td>Non-esterified fatty acid (mmol/l)</td>
<td>0.053 ± 0.065 ± 0.069 ± 0.073 ±</td>
<td>0.062 ± 0.059 ± 0.081 ± 0.059 ±</td>
<td>0.091 ± 0.058 ± 0.063 ± 0.058 ±</td>
</tr>
<tr>
<td>Ketone bodies (mmol/l)</td>
<td>0.08 ± 0.08 ± 0.38 ± 0.25 ±</td>
<td>0.80 ± 0.12 ± 0.18 ± 0.41 ±</td>
<td>0.12 ± 0.05* ± 0.11 ± 0.006* ±</td>
</tr>
<tr>
<td>Insulin (munits/l)</td>
<td>0.01 ± 0.04 ± 0.20* ± 0.07* ±</td>
<td>0.28 ± 0.04 ± 0.07 ± 0.09 ±</td>
<td>0.41 ± 0.10** ± 0.16 ± 0.09** ±</td>
</tr>
<tr>
<td>Cortisol (nmol/l)</td>
<td>6 ± 8 ± 5 ± 6 ±</td>
<td>6 ± 10 ± 27 ± 11 ±</td>
<td>2 ± 9 ± 10 ± 6 ±</td>
</tr>
<tr>
<td>Glucagon (ng/l)</td>
<td>380 ± 548 ± 465 ± 456 ±</td>
<td>481 ± 752 ± 830 ± 728 ±</td>
<td>662 ± 729 ± 700 ± 701 ±</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>40 ± 40 ± 41 ± 35 ±</td>
<td>98 ± 123 ± 118 ± 218 ±</td>
<td>68 ± 62 ± 74 ± 129 ±</td>
</tr>
<tr>
<td>Albumin (g/l)</td>
<td>4.9 ± 5.9 ± 6.2 ± 5.4 ±</td>
<td>4.8 ± 4.4 ± 4.6 ± 6.8 ±</td>
<td>4.8 ± 4.4 ± 4.6 ± 6.8 ±</td>
</tr>
<tr>
<td>Urea (mmol/l)</td>
<td>0.9 ± 1.4 ± 0.9 ± 0.2 ±</td>
<td>0.3 ± 0.6 ± 0.5 ± 1.0** ±</td>
<td></td>
</tr>
</tbody>
</table>
al., 1977) suggests that a more prolonged study would not have changed the essential character of our results. Compared with saline infusion, glucose infusion was associated with lower concentrations of lipid metabolites and a tendency for a decrease in nitrogen excretion. In normal men the nitrogen-sparing effect of glucose appears more marked (Gamble, 1946). Possibly the physiological inhibition of protein breakdown and gluconeogenesis by glucose and insulin is impaired after operation, due to increased concentrations of the catabolic and gluconeogenic hormones, cortisol, glucagon and catecholamines.

In the early postoperative phase, plasma glucagon was significantly elevated from basal values, suggesting failure of the normal suppression of glucagon release by hyperglycaemia. The subsequent falls in glucagon concentration during both glucose and glucose/insulin infusions were similar to that during saline alone.

Addition of insulin with glucose resulted in less change than we had expected, despite the reported beneficial effects of insulin on nitrogen balance after more severe injury (Hinton, Allison, Littlejohn & Lloyd, 1971). There was a decrease in urea production with glucose/insulin therapy compared with saline infusion (Table 2), presumably due to the anabolic effects of insulin. The absence of an effect of insulin on 3-methylhistidine excretion, i.e. protein breakdown, is surprising, but may reflect the relatively low concentration of insulin achieved. Because of our desire to avoid hypoglycaemia and not to alter the amount of glucose given, relatively small amounts of insulin were used (0.56 unit day⁻¹ kg⁻¹). Postoperative patients are classically resistant to many of the actions of insulin (Wright, Henderson & Johnston, 1974). Larger doses of glucose and insulin may therefore have had more effect.

Amino acid infusion, compared with saline infusion, was associated not only with a rise in blood glucose but also in blood alanine, plasma glucagon and serum insulin and urea production. Compared with glucose infusion, insulin and glycerol concentrations were similar but non-esterified fatty acids were lower and ketone body concentrations higher during amino acid therapy.

These findings may be explained by the expected stimulatory effects of amino acids upon insulin and particularly glucagon secretion (Rocha, Faloona, Muller & Unger, 1972; Raptis, Dollinger, Schroder, Schleyer, Rothenbuchner & Pfeiffer, 1973). The fall in the insulin/glucagon ratio favours both gluconeogenesis and ureagenesis from the infused amino acids (Exton, Mallette, Jefferson, Wong, Friedman, Miller & Park, 1970; Unger, 1971) and ketogenesis from non-esterified fatty acids (McGarry, Wright & Foster, 1975).

Blackburn et al. (1973) reported that addition of glucose to the amino acid infusion increased nitrogen loss compared with the effect of amino acids alone. These authors, however, used a smaller amount of amino acids with glucose than without and it is now clear that the nitrogen-sparing effect of amino acids is related to the amount infused (Freeman, Stegink, Meyer, Thompson & Denbesten, 1975). When equal amounts of amino acids were infused with and without glucose after operation, nitrogen balance was unchanged (Greenberg et al., 1976). Our studies show that the use of glucose infusions is misleading in interpreting the metabolic effects of amino acids. We have shown that amino acid infusions improve nitrogen balance, despite lowered ketone body concentration and despite increased glucagon-activated hepatic extraction of amino acids. The mechanism is probably related to increased rates of synthesis into protein, as protein catabolism shown by 3-methylhistidine excretion was not different between groups. Others have confirmed this by measuring synthesis of protein from ¹⁴C-labelled leucine during amino acid infusion (O'Keefe, Moldawer, Tercice, Bilmazes, Young & Blackburn, 1978). Moreover, body cell mass has been reported to be better preserved during amino acid than during glucose infusion (Spanier, Carmody, Milne & Shizgal, 1975).

In our patients plasma albumin fell significantly during infusions of glucose and glucose/insulin, but not during amino acid infusions. This supports the reported improvement of albumin synthesis during amino acid infusion (Skillman, Rosenoer, Smith & Fang, 1976). Thus 'protein sparing' by amino acid infusions is probably due to increased protein synthesis rather than decreased breakdown, and 'protein sparing' is a misnomer. Amino acid infusion stimulated insulin secretion and this may be a factor in the improved nitrogen balance. Also some amino acids stimulate protein synthesis, for example, leucine in skeletal muscle (Fulks, Li & Goldberg, 1975), but the effects of different amino acids in other organs may also be important.

Ketone body infusion into starving subjects is known to decrease blood alanine concentration and urine nitrogen excretion (Sherwin, Hendler & Felig, 1975). The inverse correlations noted between alanine and ketone bodies may support a weak protein-sparing effect of ketone bodies.
Routine use of total parenteral nutrition after operation has not been observed to decrease morbidity (Abel, Fischer, Buckley, Barnett & Austin, 1976; Holter, Rosen & Fischer, 1976; Foster, Alberti, Allen, Jenkins, MacIver, Smart & Karran, 1978a) and pre-operative nutritional support of malnourished patients may be more appropriate. Therefore at present there appears little justification for the routine use of isotonic amino acid infusions, which are less effective than total parenteral nutrition in maintaining nitrogen balance after operation.

Use of 5% glucose or glucose/saline infusions is likely to remain the mainstay of fluid and electrolyte care in the postoperative period, when the majority of patients can be expected to return rapidly to oral feeding. Such solutions are relatively cheap, non-toxic and have a small nitrogen-sparing effect. There seems no justification for the routine addition of insulin.

Acknowledgments

K.J.F. was supported by a grant from Travencol Laboratories Ltd. Financial support from the British Diabetic Association is also gratefully acknowledged.

References


Postoperative protein-sparing therapy


