

Clinical trial

Comparison of an elemental and polymeric enteral diet in patients with normal gastrointestinal function

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SUMMARY In a prospective controlled clinical trial, 70 patients with normal gastrointestinal function were randomised to receive either an elemental diet based on Vivonex HN or an isonitrogenous isocaloric polymeric diet based on Clinifeed 400, administered by continuous 24 hour nasogastric infusion. The two groups of patients were well matched for age, sex, diagnosis, prior starvation, duration of feeding, initial nutritional status, and metabolic status. Nitrogen losses were significantly less on the polymeric feed, despite similar intakes. Serum transferrin rose significantly (1.85 ± 0.2 to 2.30 ± 0.2 g/l, $p < 0.05$) only in the Clinifeed group, but nutritional parameters were otherwise maintained in both groups. The incidence of diarrhoea (Vivonex, 23.5%; Clinifeed, 30.6%) was not significantly different and was attributable to antibiotics in most cases. Hypokalaemia, which occurred in nearly half the patients, was equally distributed in the two groups, but hypophosphataemia occurred more often in the Vivonex group ($p < 0.05$). Liver enzyme disturbances were similar in both groups. The present findings, therefore, provide no evidence that chemically defined 'elemental' diets containing free amino acids as their nitrogen source are in any way superior to polymeric diets containing whole protein and fat when administered to patients with normal gastrointestinal function.

One of the major differences between chemically defined 'elemental diets' and standard polymeric liquid diets used for enteral feeding is that elemental diets contain partially or completely hydrolysed protein as their nitrogen source whereas polymeric diets contain undigested protein. Despite the fact that elemental diets are more hypertonic and expensive than polymeric diets, they have been widely used in the clinical setting, not only in patients with impaired gastrointestinal function, but also in those with normal gastrointestinal function, all of whom would appear to be perfectly capable of assimilating whole protein.¹ In agreement with others²⁻⁴ we have held the view that this inappropriate usage of 'elemental diets' cannot be supported, as there is very little clinical or experimental data available to substantiate the claims made for them.¹

We now report the results of a controlled

prospective clinical trial designed to compare the nutritional efficacy and side-effects of isonitrogenous isocaloric regimes of an elemental diet, Vivonex HN, and a polymeric diet, Clinifeed 400, administered to a diagnostically heterogeneous group of patients with no clinical evidence of impairment of gastrointestinal function and in whom nasogastric feeding was indicated as the sole source of nutritional intake.

Methods

PATIENTS, MATERIAL, AND DIETARY REGIMES

Included in the study were 70 patients with no clinical evidence of impaired luminal nutrient digestion or absorptive capacity and in whom it was considered that nasogastric feeding rather than parenteral feeding was indicated as the sole source of nutritional support in order to maintain or improve nutritional status. Patients were referred from all specialties throughout the hospital and were considered for enteral nutritional support only if

they were considered unlikely to be able to return to normal oral nutrition within one week. The patients did not have to satisfy criteria for the presence of impaired nutritional status.

Patients in whom fluid restriction was indicated – for example, those with cardiac, respiratory or renal failure, or inappropriate secretion of antidiuretic hormone (ADH), diabetics, and those with recent diarrhoea, vomiting or regurgitation of gastric contents were all excluded from the study. Finally, a small number of patients known to have nitrogen excretion in excess of 14.4 g/day before the study were excluded.

The trial was conducted over a 12-month period (April 1979–April 1980) and the 70 patients constituted 36% of 185 patients receiving nutritional support during the same period, 47 (24.1%) of whom received total parenteral nutrition. The remaining 78 (40%) were treated with various enteral regimes outside the trial.

Having satisfied inclusion criteria, 70 patients were randomised by the drawing of numbered envelopes containing cards allocated by random numbers to one of two diets, Vivonex HN (Eaton Laboratories Ltd; 34 patients) or Clinifed 400 (Roussel Laboratories Ltd; 36 patients).

Both regimes (Table 1) contained 4.8 g nitrogen/l and a non-protein calorie to nitrogen ratio of 200 Kcal/g nitrogen achieved by supplementing the basic diets with a glucose polymer energy source (Caloreen, Roussel Laboratories Ltd). Starter regimes were used for both groups starting with 2 l of half strength feed on day 1 and progressing to 2 l of full strength by day 4.

On days 1–3, nitrogen losses were estimated from urinary nitrogen according to the method described by Lee and Hartley⁵ and validated for patients with normal gastrointestinal function.⁶ This method

includes a standard correction factor for faecal and skin losses, which could not be estimated in our patients because of practical problems, such as faecal collection in the unconscious and the logistic problem of assaying large numbers of stool collections in a busy clinical chemistry department.

Patients with nitrogen losses equal to or less than 9.6 g nitrogen/day, calculated as above, were prescribed 2 l full strength diet supplying 9.6 g nitrogen and 1920 Kcal on day 4 and thereafter, unless further daily nitrogen balance studies indicated greater losses. In this case, patients were transferred to 3 l full strength feed, the regime also instituted in those patients in whom there were nitrogen losses of between 9.6 and 14.4 g nitrogen/day as detected during the first four days of treatment. By using this flexible approach, nitrogen input could be adjusted to match, or just exceed, calculated nitrogen losses.

Feeding was continued until no longer clinically indicated or until the patient was ready to start oral feeding.

TECHNIQUE OF ADMINISTRATION

The enteric diets were administered by continuous 24 hour gravity controlled infusion using a standard giving set and a 1 mm internal diameter nasogastric tube (Clinifeeding Systems I and II, Roussel Laboratories Ltd) and standard 500 ml glass Winchester (DHSS 4168).⁷ Feeds were prepared 24 hours in advance in the hospital diet kitchen using sterile water and refrigerated at 4°C before use.

NUTRITIONAL STATUS

Nutritional status of the patients was assessed before randomisation and at weekly intervals thereafter. Anthropometric assessment included measurement of triceps skinfold thickness, midarm circumference, and midarm muscle circumference.⁸ Biochemical assessment included management of serum albumin,⁹ transferrin,⁹ thyoid binding pre-albumin (TBPA),⁹ potassium, zinc, and phosphate. Haematological parameters assessed were lymphocyte count, serum B₁₂, serum folate, and red cell folate. Delayed hypersensitivity was assessed as an indication of cellular immunity by using the recall antigens PPD (Evans Medical Ltd), *Candida albicans* (Bencard Ltd), and streptodornase-streptokinase (Lederle Laboratories). These tests were judged to be impaired when more than one of the antigens failed to induce induration of greater than 5 mm 48 hours after intradermal injection.¹⁰

Twenty-four hour urinary excretion of urea and creatinine was measured daily. Urinary urea nitrogen excretion reflects both muscle mass and the degree of catabolism,¹¹ whereas urine creatinine is

Table 1 *Composition of diets*

Constituent per litre	Vivonex HN	Clinifed 400
Free amino acids (g/l)	31 (4.8 g nitrogen)	—
Whole protein (g/l)	—	30 (4.8 g nitrogen)
Total carbohydrate (g/l)	240†	160†
Lactose (g/l)	—	19.5
Fat (g/l)	0.63	26.8
Potassium (mmol/l)	13	25
Zinc (mg/l)	3.0	6.0
Phosphate (mg/l)	190	460
Folic acid (µg/l)	24	300
Non-protein calories*		
g α-amino nitrogen	200:1	200:1
Osmolality (mosm/kg)	690	395
Volume administered (l)	2–3	2–3

* 4.18 MJ = 1000 Kcal.

† Includes added Caloreen.

related more closely to muscle mass alone.¹² The ratio of urinary urea nitrogen:urinary creatinine excretion was, therefore, used to obtain a clearer assessment of metabolic status independent of differences in muscle mass.

STATISTICS

Advice concerning the statistical analysis of the trial data was sought from the Department of Medical Statistics, Charing Cross Hospital, London. Paired and unpaired Student's *t* tests and χ^2 tests were performed as appropriate.

Results

CLINICAL DIAGNOSIS (Table 2)

The patients in both the Vivonex and Clinifeed groups were well matched for age, sex, race, and duration of prior starvation (Table 2). The two groups were also comparable with respect to underlying clinical diagnosis. Neurological patients (including those with head injuries and intracranial haemorrhage) and those with neurological disorders affecting either conscious level or swallowing reflexes comprised the majority of each group (Vivonex, 59%; Clinifeed, 66.6%). The numbers in each group with surgical diagnoses were similar, although there were more postoperative cases in the Vivonex group. The medical categories included patients with anorexia nervosa, meningitis, tuberculosis, and respiratory failure requiring ventilatory support. Sepsis, defined as infection giving rise to fever, tachycardia, and leucocytosis on

more than one day, either as the primary or secondary diagnosis, was distributed evenly between the two groups. The number of patients starved for longer than one week was also similar in each group.

NUTRITIONAL STATUS (Table 3)

Patients in the two treatment groups had similar nutritional status on entry to the study, anthropometric data indicating only a moderate degree of muscle mass depletion as indicated by midarm muscle circumference values, but considerable depletion of body fat reserves as indicated by triceps skinfold thickness values.¹³ Moreover, there was no significant difference in muscle mass as inferred from daily urinary creatinine excretion results¹² (Vivonex, 8.2 ± 0.8 mmol/d; Clinifeed, 9.0 ± 1.0 mmol/d). It was not possible to measure the patients' height because of the nature of their illnesses and therefore the 'creatinine height index' could not be calculated. For similar reasons, weights have not been included, as so few patients were physically capable of being weighed and thus data would have been unrepresentative of the group as a whole.

The metabolic status of each complete group initially, as determined by the ratio of urinary urea nitrogen to urinary creatinine, did not differ significantly (Vivonex, 1.0 ± 0.08 ; Clinifeed, 1.28 ± 0.25) on entry to the study.

DURATION OF FEEDING

The mean duration of feeding (Vivonex, 14.3 ± 1.5 days; Clinifeed, 15.2 ± 2.0 days) was similar, as was

Table 2 Comparability of two treatment groups

	Vivonex (n=34)	Clinifeed (n=36)
Age (mean \pm SEM) (yr)	55.1 \pm 3.1	56.1 \pm 3.1
Sex		
Male	20	23
Female	14	13
Race		
Caucasian	31	34
Non-Caucasian	3	2
Diagnostic category		
Neurosurgical	11	12
Neurological	9	12
Surgical		
Preop	3	7
Postop	7	2
Medical	4	3
Numbers of patients starved seven days before trial	16 (47%)	17 (47%)
Mean duration of prior starvation (days) (mean \pm SEM)	18.6 \pm 4.5	20.0 \pm 3.9
Presence of infection	15	16

Table 3 Nutritional status on entry to study

	Vivonex group	Clinifeed group
Triceps skin fold (TSF)		
Male (7.5–11.3 mm)*	7.5 \pm 0.7	7.2 \pm 0.7
Female (9.9–14.9 mm)*	10.9 \pm 1.5	11.3 \pm 1.2
Midarm circumference (MAC)		
Male (17.6–26.3 cm)*	26.3 \pm 0.9	24.4 \pm 1.0
Female (17.1–25.7 cm)*	22.5 \pm 2.0	24.7 \pm 1.5
Midarm muscle circumference (MAMC)†		
Male (15.2–22.8 cm)*	24.2 \pm 0.7	22.6 \pm 0.6
Female (13.9–20.9 cm)*	20.6 \pm 0.9	21.1 \pm 1.2
Albumin g/l (35.0)‡	35.5 \pm 1.0	35.8 \pm 1.0
Transferrin g/l (2.0)‡	1.97 \pm 0.12	1.95 \pm 0.1
Lymphocyte count $\times 10^6$ /l (1500)‡	1450 \pm 120	1600 \pm 120
Impaired skin tests (% patients)	78%	83.3%

* Values in parentheses represent 60–90% of adult reference values.⁸

† MAMC = MAC – ($\pi \times$ TSF).

‡ Lower limit of normal.

(All values are means \pm SEM.)

the number of days on which the higher nitrogen regime was administered (Vivonex, 4.1 ± 1.1 days; Clinifeed, 4.5 ± 1.6 days).

There was, however, a wide range of feed duration in each group (Vivonex, one to 35 days; Clinifeed, two to 60 days) but 27 patients on Vivonex and 25 on Clinifeed were fed for longer than one week.

The reasons for termination of feeding are shown in Table 4. Twice as many patients on Clinifeed made successful return to normal nutrition ($p < 0.02$). If, however, the number of patients successfully completing a course of elective preoperative feeding is included with those returning to normal nutrition, the difference between the two treatment groups is no longer significant.

EFFECT OF NUTRITIONAL SUPPORT ON NUTRITIONAL STATUS

The effect of enteral nutrition on nutritional status and nitrogen balance was compared only in those patients fed for more than seven days with nitrogen losses not exceeding 14.4 g nitrogen per day as shown in Tables 5 and 6. There were no significant changes in anthropometric parameters *within* either group except for pre-albumin (thyroid binding pre-albumin) which rose significantly in both groups ($p < 0.001$) and transferrin which increased only in the Clinifeed group ($p < 0.05$). The rise in thyroid

Table 4 End points of trial

	Vivonex group	Clinifeed group
1 Returned to normal oral feeding	11	22
2 Elective preoperative feeding terminated by operation	5	3
3 Transfer to other hospitals	2	3
4 Complication of enteral feeding necessitating cessation of feeding	4	3
5 Deterioration in clinical condition	6	2
6 Death	6	3

binding pre-albumin occurred independently of nitrogen balance and changes in albumin and transferrin, this reflecting the known sensitivity of this test to refeeding.⁹

No statistically significant rises in lymphocyte counts occurred in either treatment group. There was a high incidence of negative skin tests at the start of the study in those tested (Vivonex, 25/32 (78%); Clinifeed, 30/36 (83.3%)) with only small numbers of patients in each group showing an improvement in responsiveness to recall antigens by the end of the study (Vivonex, 9/24 (37.4%); Clinifeed, 5/24 (21%)). There was no significant difference between the two treatment groups with respect to the absolute values as shown in Table 5, or to the numbers of patients with improved, maintained, or deteriorating parameters.

Table 5 Nutritional status of patients fed more than seven days and with nitrogen losses 14.4 g/day before and after study

	Vivonex group (n=25)		Clinifeed group (n=21)	
	Before	After	Before	After
Triceps skin fold (TSF)				
Male (7.5–11.3 mm)*	7.4±1.0	7.4±0.9	7.0±0.8	6.3±0.8
Female (9.9–14.9 mm)*	11.4±1.4	11.0±1.2	10.9±2.0	10.0±1.6
Midarm circumference (MAC)				
Male (17.6–26.3 cm)*	26.5±1.2	26.3±1.0	24.6±0.3	24.6±0.7
Female (17.1–25.7 cm)*	24.9±1.1	24.9±1.1	24.9±2.0	24.7±0.7
Midarm muscle circumference (MAMC)†				
Male (15.2–22.8 cm)*	22.7±1.8	23.8±0.8	22.4±0.8	22.5±0.6
Female (13.9±20.9 cm)*	21.3±0.9	21.4±0.9	21.5±1.6	21.5±1.2
Albumin g/l (35.0)‡	35.0±1.1	32.1±1.6	35.5±1.3	35.5±1.1
Transferrin g/l (2.0)‡	1.80±1.0	1.95±0.1	1.85±0.2§	2.30±0.2§
Serum pre-albumin mg/l (290)‡	140±20	214±20	153±10¶	244±20¶
Lymphocyte count $\times 10^6/l$ (1500)‡	1450±150	1850±250	1650±200	1900±250
Impaired skin tests (% patients)	72%	44%	81%	67%

* Values in parentheses represent 60–90% of adult reference values.⁸

† MAMC = MAC – ($\pi \times$ TSF).

‡ Lower limit of normal.

§ $p < 0.05$.

|| $p < 0.001$.

¶ $p < 0.001$.

(All values are means \pm SEM.)

Table 6 Nitrogen balance data of patients fed more than seven days and with nitrogen losses less than 14.4 g/day

	Vivonex group (n=25)	Clinifeed group (n=21)	Significance of difference
Intake of nitrogen (g)	10.4±0.5	10.2±0.4	NS
Output of nitrogen ³ (g)	9.7±0.7	7.7±0.5	p<0.05
Nitrogen balance (g)	+0.64±0.8	+2.6±0.6	NS
Mean duration of feeding (days)	16.0±1.6	20.3±2.8	NS
Patients in positive balance (no.)	15 (60%)	17 (81%)	NS
Urinary creatinine excretion (mmol/24 h)	7.5±0.7	7.3±1.0	NS
Urinary urea nitrogen (g): creatinine excretion (mmol)			
Days 1-3	0.95±0.08	0.96±0.1	NS
Day 3 onward	1.23±0.12	0.83±0.07	p<0.01
Patients infected (no.)	10 (40%)	12 (57%)	NS

(All values are means ± SEM.)

As shown in Table 6, significantly lower nitrogen excretion was found in the Clinifeed-fed patients, despite the fact that nitrogen and energy intake as well as initial assessment of muscle mass and the incidence of sepsis were similar in both groups. Despite the trial protocol – namely, to maintain nitrogen balance by appropriate adjustment of intake – only 60% of the Vivonex-fed patients and 81% of the Clinifeed-fed patients were maintained in overall positive balance throughout the study. In both groups, reduction of prescribed intakes because of intercurrent investigative and therapeutic manoeuvres requiring prior fasting, poor nasogastric tube compliance with delays in replacement, and failure to regulate infusion rates correctly combined to impede achievement of positive nitrogen balance.

Urinary urea nitrogen:urinary creatinine ratios from the third day onwards revealed that the metabolic status with regard to urea nitrogen excretion was significantly different in the two groups. Thus, more urea nitrogen was lost per mmol creatinine excreted during Vivonex therapy than during Clinifeed therapy (p<0.01), despite similar nitrogen intake and initial metabolic status.

GASTROINTESTINAL SIDE EFFECTS

The incidence of gastrointestinal side effects was evenly distributed between the two treatment groups, whether fed for less than seven days or more. Comparing the complete treatment groups, eight (23.5%) on Vivonex and 11 (30.6%) on Clinifeed developed diarrhoea, although none of these was withdrawn for this reason. All but one patient developing diarrhoea on Vivonex and two

patients on Clinifeed were receiving concurrent antibiotic therapy. In one patient, laxatives were also being administered. Diarrhoea resolved within 24 hours of stopping treatment with antibiotics, but, when it was not possible to stop such treatment on clinical grounds, codeine phosphate 30–60 mg thrice daily was sufficient to stop diarrhoea until such time as the antibiotics could be withdrawn.

Vomiting or regurgitation of gastric contents occurred in seven (20.6%) of the Vivonex group and eight (22.2%) of the Clinifeed group. In the Vivonex group, two comatose patients with head injuries and one with Guillain-Barré syndrome were withdrawn because of persistent regurgitation and, in one case, pulmonary aspiration. In half the cases of vomiting, a sudden bolus of feed, intercurrent urinary tract infection, a contaminated feed, or poor maintenance of the subdiaphragmatic position of the nasogastric tube could be implicated. A quantitative assessment of vomitus was possible in only a minority of patients.

METABOLIC SIDE EFFECTS

In the Clinifeed group an undiagnosed diabetic who became hyperglycaemic and ketotic was withdrawn. Two neurosurgical patients who developed severe hyponatraemia attributable to the inappropriate ADH syndrome and one patient who developed hypercalcaemia related to underlying neoplasia, all fed with Clinifeed, were also withdrawn. Potassium fell to below normal limits in 53% of patients on Vivonex and 47% on Clinifeed. Serum zinc levels fell below normal lower limits in only two patients on Vivonex and three on Clinifeed. No significant changes occurred in serum magnesium levels. Serum phosphate fell significantly during feeding with Vivonex (1.11±0.7 to 0.98±0.08 mmol/l, p<0.05) by comparison with a rise in serum phosphate during Clinifeed administration (1.03±1.06 to 1.28±0.09, p<0.05), and levels below normal occurred more frequently on Vivonex (p<0.05).

Red cell folate, serum B₁₂, and serum iron did not change significantly in either group. Serum folate, however, rose significantly in the Clinifeed group (5.2±1.3 to 7.2±1.4 µg/l, p<0.05) but no such trend was detected in the Vivonex group (3.6±0.8 to 3.6±0.5, NS).

LIVER ENZYME CHANGES

A rise of one or more of the three liver enzymes, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (AP), above the upper limit of normal occurred in 35% of the Vivonex group and 41.7% of the Clinifeed group. In approximately one-third of those affected, an associated factor such as concurrent sepsis or

antituberculous therapy could be implicated. Maximum levels reached during the study were as follows, with the upper limit of normal in parentheses: ALT, 141 (30) IU/l; AST, 170 (40) IU/l; AP, 156 (80) IU/l.

In none of the patients could a rise in serum bilirubin be attributed to enteral feeding alone. By the end of the study, the majority of abnormal results had returned to within normal limits and it was not necessary to withdraw any patient because of liver dysfunction.

Discussion

The results of this controlled study show that no major clinical differences existed between isonitrogenous isocaloric regimes of the 'elemental diet' Vivonex HN and the polymeric diet Clinifed 400, when used as the sole means of nutritional support in a diagnostically heterogeneous group of patients with normal digestion and absorptive capacity. Both treatment groups were well matched and had moderate, but similar, degrees of nutritional depletion on entry to the study. In those fed for more than one week, the overall effect of nutritional support was to maintain rather than to improve skeletal muscle mass and fat reserves, which agrees well with the results of others.^{2,3} This reflects both the high incidence of sepsis and immobility among our patients, as well as the number of patients not nutritionally depleted on entry to the study, but fed prophylactically.

Serum transferrin levels have been advocated as a means of assessing nutritional repletion with respect to protein synthesis.^{9,14} It is of interest, therefore, that a significant improvement in transferrin levels occurred only in the group of patients receiving the whole protein- and fat-containing diet, Clinifed, a finding which is in keeping with the significantly lower urinary nitrogen excretion found in this treatment group compared with those receiving Vivonex, although the difference in nitrogen balance between the two groups did not achieve statistical significance. As similar inputs of nitrogen and energy were achieved in both treatment groups, the above findings do suggest that the nitrogen sparing effect of the whole protein-containing diet is superior to that of the elemental diet. Others have also found that positive nitrogen balance can be more readily achieved with polymeric diets but in those studies patients receiving elemental diets such as Vivonex did not achieve similar levels of intake as those on polymeric diets, mainly because of poor tolerance of the elemental regimes.³ Although much emphasis has been placed on the type of nitrogen source in enteral feeding, the role of fat may be of

greater importance, as recent studies have demonstrated that fat appears to have a specific effect on repletion of lean body mass during parenteral nutrition of patients with moderate metabolic stress.¹⁵ It is, therefore, of interest to note that in this study the Clinifed-based diet contained 25.1% of its non-protein energy in the form of fat compared with only 0.6% for the Vivonex-based diet. Indeed, the essential fatty acid content of Vivonex has recently been noted to be insufficient to prevent essential fatty acid deficiency during prolonged enteral nutrition.¹⁶

In the present study, both feeds were generally well tolerated, there being no significant difference in the incidence of either diarrhoea or vomiting, although three of 34 (9%) Vivonex-fed patients were withdrawn because of the latter complication. As reported by others,^{7,17} antibiotics appeared to be the primary cause of diarrhoea in our patients, all of whom were fed continuously over a 24 hour period. There was no evidence to implicate either the hypertonicity of the elemental diet (690 mOsm/kg) or the lactose content (19.5 g/l) of the polymeric diet as major factors in the aetiology of diarrhoea.

Despite the study protocol, which was designed to maintain nitrogen balance, many individual patients in each group remained in negative nitrogen balance throughout the study. This finding cannot be attributed entirely to the intrinsic properties of the feeds themselves, but represents more the practical difficulties encountered during enteral nutrition, such as persistent vomiting, or intercurrent investigations, or procedures requiring a 'nothing by mouth' regime. Moreover, the degree of negative nitrogen balance has probably been underestimated in those patients with diarrhoea, as the extra faecal nitrogen losses incurred were not assessed.

Metabolic disturbances occurred surprisingly frequently during the present study and generally reflected differences in formulation of the two diets. Hypokalaemia requiring potassium supplementation was found in nearly half the patients overall and was evenly distributed between the two treatment groups despite the higher potassium content of Clinifed. The lower phosphate content of Vivonex, however, was reflected in the significantly greater incidence of hypophosphataemia in this group, and the failure of serum folate to rise from low normal values could be similarly attributed to the lower folate content. Liver enzyme rises occurred frequently in both groups. These changes, however, were not invariable as suggested by others,¹⁸ nor did we detect any rise above normal or serum bilirubin attributable to enteral feeding alone. The changes in liver enzymes were usually minor, returning to normal by the end of the study,

whether normal or abnormal on entry to the study.

In view of the diagnostic heterogeneity of the patients in this study, it was not surprising that many of the rises in liver enzymes could be attributed to factors other than enteral feeding. While a number of factors have been noted to cause either cholestasis¹⁹ or fatty infiltration¹⁸ during parenteral feeding, no clear pattern has evolved to explain the changes associated with enteral feeding.²⁰ Neither the free amino acid nitrogen source of Vivonex, the whole protein source of Clinifeed, nor the relative differences in carbohydrate and fat contents can be implicated on the basis of results of the present study.

In conclusion, the two diets chosen for this study differed in a number of ways apart from their nitrogen sources. It is, therefore, not possible to draw firm comparisons between the efficacy of the nitrogen sources alone but the available evidence suggests that there is no justification for the use of expensive elemental diets such as Vivonex when gastrointestinal function is normal. The much cheaper polymeric diets such as Clinifeed would seem to be the more appropriate nutritional sources in such patients. Moreover, certain aspects of our data suggest that protein repletion occurred more effectively on the polymeric diet than the elemental diet. Further studies are required to assess the relative efficacies of elemental diets containing small peptides as the predominant nitrogen source, and also the role of polymeric diets when gastrointestinal function is moderately impaired.

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