Nutrition in gastrointestinal disease

By R. I. RUSSELL, *Gastroenterology Unit, Royal Infirmary, Glasgow, G4 0SF*

The metabolic consequences of severe disease have been extensively investigated over some years. More recently, the metabolic and nutritional effects of severe gastrointestinal disease have been studied, and it is now realized that a wide range of gastrointestinal diseases may be accompanied by such problems. Recovery from major gastrointestinal illness may be speeded by nutritional support and indeed survival may depend upon supplying adequate metabolic aid.

Nutritional aspects of gastrointestinal disease

Most of the original work in relation to metabolic or catabolic responses has been described in relation to trauma, severe infection or surgery, but the findings regarding metabolic responses are also relevant to patients with severe gastrointestinal disease.

Catabolic responses and normal nutritional reserves. Catabolic or metabolic response of the body to severe or chronic gastrointestinal disease may be as marked as that following severe trauma or infection. Much information is now available about these metabolic responses. The catabolism of body protein exceeds anabolism and dietary intake is unable to correct this imbalance (Moore, 1959). Negative nitrogen balance can be partially if not completely reversed by the administration of a high protein, high energy diet (Troell & Wretlind, 1961).

Under conditions of severe or prolonged stress, injury or disease, metabolic alterations in the body occur. Features of this metabolic response are a negative N and potassium balance, retention of sodium and water, and accumulation of acid. In severe cases a continued catabolic response leads to muscle wasting, hypoproteinaemic oedema, an increased susceptibility to infection and failure to repair damaged tissues. The degree of the response is determined by the severity of the illness.

The normal nutritional reserves present in man are shown in Table 1. Reserves of carbohydrate are 8–12 h, fat 20–25 d and protein 10–15 d. In severe or chronic gastrointestinal disease the nutritional reserves are likely to have been reduced to some extent due to the severity and chronicity of the disease prior to the patient coming under hospital care.

Table 1. *Nutritional reserves in normal man*

	Quantity (kg)	Duration
Carbohydrate	0·15–0·20	8–12 h
Fat	10–15	20–25 d
Protein	4–6	10–15 d

https://doi.org/10.1079/PNS19780011 Published online by Cambridge University Press

Gastrointestinal diseases which may give rise to nutritional and metabolic problems. A wide range of gastrointestinal conditions may be associated with significant nutritional deficiency (Table 2). These deficiency states occur much more widely than was formerly believed and may not be clinically apparent in many patients for some time. Their occurrence depends largely on the severity and duration of the disease. The early recognition of such problems and their correction will improve the prognosis of many patients with severe gastrointestinal diseases.

One gastrointestinal disease which is particularly liable to give rise to severe nutritional deficiency is Crohn's disease. This condition is increasing in incidence and many of the patients have severe malabsorption, diarrhoea associated with malabsorption of fat, water, electrolytes or bile acids, chronic fistulae, local infections. They may require surgery for obstructive lesions, often necessitating the removal of part of the intestine, which may exacerbate the malabsorption and diarrhoea.

Table 2. *Gastrointestinal causes of nutritional deficiency*

1. Severe diarrhoeal states
   - Crohn's disease
   - Ulcerative colitis
   - Gastrointestinal infections
   - Tumours of colon and rectum
   - Chronic pancreatic insufficiency
   - Gastrointestinal fistulae
2. Severe malabsorption states
   - Coeliac disease
   - Crohn's disease
   - Massive small intestinal resection
3. Prolonged vomiting
   - Pyloric stenosis
   - Infections
   - Tumours of upper gastrointestinal tract
4. Prolonged poor oral intake
   - Anorexia nervosa
   - Tumours
   - Dysphagia
5. Gastrointestinal surgery
6. Paediatric conditions
   - Gut atresia
   - Tracheo-oesophageal fistula

Assessment of nutritional status

Before formulating a programme for the nutritional and metabolic support of the patients with severe gastrointestinal disease, it is necessary to ascertain the presence and the degree of the nutritional deficiency.

Awareness of the possible presence of nutritional deficiency in any patient with gastrointestinal disease is clearly an important factor. A simple clinical assessment is the measurement of body-weight. If possible a comparison with the patient's previous weight will provide a measure of severity of the catabolic process. Clinical assessment may also include the analysis of basal energy expenditure, assessment of skeletal muscle compartment by measuring triceps skin fold thickness and mid arm muscle circumference, the measurement of the creatine/height index, serum albumin, serum transferrin, nitrogen balance and measurement of cell mediated

https://doi.org/10.1079/PNS19780011 Published online by Cambridge University Press

immunity (Blackburn, Bistrian, Maini, Schlamm & Smith, 1977). Many of these methods still require to be fully evaluated and may not be routinely available.

Methods of providing nutritional and metabolic support in gastrointestinal disease

After the identification of a nutritional and metabolic problem in any patient with gastrointestinal disease and the assessment of its severity, a decision must be taken as to the form of the nutritional support which should be given.

There are four ways of providing nutritional and metabolic support in such patients. (1) Supplementary feeding. (2) Nasogastric feeding with a liquidized diet. (3) Elemental diets. (4) Intravenous nutrition.

For the provision of any form of adequate nutritional support, there is a requirement for trained medical and nursing staff, together with the co-operation of a committed team of dietitians, biochemists and pharmacists. The development of a metabolic team with adequate backup facilities will certainly achieve better results.

No rules or regulations can be laid down regarding the use of any one of these four methods in the management of any particular gastrointestinal problem. Each patient is an individual whose gastrointestinal disease may differ markedly in duration, severity and effects on the gastrointestinal tract and on its nutritional disturbances.

Supplementary feeding. Supplementary feeding with simple and palatable fluids may be sufficient in a number of patients. Clearly this method will be inadequate for the management of severe or chronic gastrointestinal problems with marked diarrhoea, malabsorption or vomiting, but in minor nutritional problems and in early gastrointestinal disease this method is simple and effective. Suitable supplementary foods include preparations containing Caloreen (Berlyne, Bruis, Booth, Mallick & Simons, 1969). Caloreen is a mixture of dextrins with an average glucose chain-length of 6 units. It is palatable, blends easily with most foods and readily dissolves in water. Preparations containing Caloreen with coffee and fruit juices can be made up.

Nasogastric feeding. Some patients may be improved by the use of nasogastric feeding with suitable liquidized foods. These patients, who are unable to ingest the required quantities of nutrients, should have a gastrointestinal tract of normal digestive and absorptive capacity. The diets introduced by means of nasogastric tube are cheap and easy to prepare. Examples of such diet preparations are shown in Table 3. Combinations of Complan, Caloreen, vitamins or Caloreen, Albumaid, vitamins and egg yolk are suitable for such preparations. The use of these feeds is relatively free from side effects although some diarrhoea may occur. They should be given in small quantities at regular intervals.

Elemental diets. An elemental or chemically-defined diet is a preformulated diet containing an elemental or nearly elemental protein source, either amino acids alone or amino acids and small peptides. Simple fats, glucose, minerals and vitamins are also present in varying amounts according to the preparation. They require little or no digestion and have minimal residue (Russell, 1975). Their

https://doi.org/10.1079/PNS19780011 Published online by Cambridge University Press

Table 3. *Nasogastric feeding: Examples of diet preparations*

300 g Complan	600 g Caloreen
300 g Caloreen	60 g Albumaid
9 g Methylcellulose	8 g Metabolic mineral mixture
Vitamin syrup 2 or 3 l with water	Vitamin syrup 2 or 3 l with water
14·4 g N	10 g N
2500 kcal (10500kJ)	240 kcal (10080kJ)
64 meq Na	40 meq Na
84 meq K	30 meq K

development is still at an early stage and as nutritional support sources they are expensive compared with nasogastric feeding or supplementary feeding regimes.

The nutritional value of elemental diets is satisfactory for normal requirements, although additional requirements may be necessary in many chronic gastrointestinal diseases. They are totally absorbed in the upper small intestine, leaving only endogenous residue to enter the large bowel. No digestion or micelle formation is required for their absorption; they contain no indigestible bulk or fibrous material, and are thus low residue in nature.

There are two principal elemental diets marketed in the UK, namely Vivonex and Flexical. In addition, the products Aminutrin and Calonutrin together form a chemically defined diet. There are marked differences in the composition of the two principal elemental diets Vivonex and Flexical as is shown in Table 4. These differences in the composition in the various elemental diets and their significance has recently been reviewed. (Young, Heuler, Russell & Weser, 1975; Russell, 1978).

Table 4. *Composition of Vivonex and Flexical*

	Vivonex	Flexical
Protein	Pure L-amino acids	Hydrolyzed peptides + amino acids
% energy contribution	8·50	9·00
Carbohydrate	Glucose oligosaccharides	Sucrose oligosaccharides
% energy contribution	90·20	61·00
Fat	Safflower oil	Soya-bean oil Medium chain triglycerides Soya-bean lecithin
% energy contribution	1·30	30·00

Essentially elemental diets may be given in gastrointestinal diseases to supply nutritional requirements, or specific dietary formulations of individual elemental diets may be used in the treatment of specific gastrointestinal symptoms in particular diseases. Elemental diets may be given as a supplement to normal food

https://doi.org/10.1079/PNS19780011 Published online by Cambridge University Press

or as the sole source of nutrition. In some situations they may follow on from initial treatment with intravenous feeding.

In patients with chronic gastrointestinal fistulae, electrolyte imbalance, intraperitoneal sepsis and severe nutritional deficiency may develop over a period of time. The low residue nature and high nutritional value of elemental diets may be helpful in the management of these problems (Bury, Stevens & Randall, 1969). They have also been shown to be of value in recto-vaginal, gastrointestinal-cutaneous fistulae, and in the treatment of fistulae associated with Crohn's disease (Grundy, 1971). They are not helpful, however, in the management of high output, upper gastrointestinal fistulae.

Elemental diets have a part to play in the management of some maldigestion and malabsorption states. As they require little or no digestion in the upper alimentary tract they may be helpful in the maldigestion associated with chronic pancreatic insufficiency (Voitk, Brown, Echave, McArdle, Gurd & Thomson, 1973), or in cystic fibrosis. After massive small intestinal resection, patients may develop severe nutritional deficiency with marked diarrhoea and weight loss. Nutritional support is generally necessary in these patients. Elemental diets may be valuable (possibly after an initial course of intravenous nutrition) and have an added advantage in that they may also speed intestinal adaptation. Clinical improvement in patients with the short bowel syndrome has also been reported (Voitk, Echave, Brown & Gurd, 1973).

In severe Crohn's disease with malabsorption elemental diet therapy may be helpful. In such patients fistulae may also be a problem and one factor in the causation of the severe diarrhoea in these patients is bile acid malabsorption leading to cholerheic diarrhoea. It has been demonstrated that the elemental diet Vivonex improves diarrhoea of this type and causes a statistically significant reduction in faecal bile acid excretion (Nelson, Carmichael, Russell & Atherton, 1977). Vivonex has also been shown to give good longterm nutritional support in patients with Crohn's disease (Goode, Hawkins, Fegetter & Johnson, 1976). Elemental diets may also have a place in the preparation of patients for colonic surgery and in the management of patients who have undergone gastroinstestinal surgery.

Elemental diets may be given orally, either alone or as a supplement to normal diet, or be administered by means of an intragastric or intrajejunal tube. The route of administration depends upon the indications for the use of the diet in any individual patient. Palatability is a problem, although it can be improved by the use of flavourings, combination with 'Rise and Shine', the use of half-strength solutions and a liberal intake of water. Careful monitoring of patients on elemental diet therapy is necessary because of potential untoward effects such as nausea, vomiting, diarrhoea, the hyperosmolar syndrome, dumping and skin rashes.

Intravenous nutrition. Intravenous nutrition may be considered as an alternative to oral feeding when the latter is unable to support the patient in adequate nutritional and metabolic balance. There are a number of gastrointestinal conditions in which intravenous nutrition will be of immense value in short term

https://doi.org/10.1079/PNS19780011 Published online by Cambridge University Press

regimes and also in long term support programmes. Intravenous nutrition is not only concerned with supplying energy sources and N, but is also concerned with the maintenance of electrolyte equilibrium and serum oncotic pressure, and the replacement of fluid.

Intravenous feeding may be total or supplementary. Thus, in a patient in which bowel rest may be regarded as important (as in severe Crohn's disease), then intravenous nutrition, if indicated, will be the sole source of nutrition. If, however, bowel rest is not a requirement and the important aspect of a patient is the recovery of his nutritional state and metabolic balance as quickly as possible then intravenous nutrition may be given in conjunction with supplementary oral feeding. A great deal depends upon the indications for nutritional support and the state of the gastrointestinal tract of the patient.

Intravenous feeding may be helpful in patients with severe Crohn's disease to give nutritional support or bowel rest, or both. In some instances, a disease remission may be achieved. Intermittent courses may be helpful especially in young patients with Crohn's disease, in whom body-weight and height may be improved. Patients with ulcerative colitis, chronic pancreatic disease and gastrointestinal fistulae may also respond to this form of therapy, and in the initial stages after massive small intestinal resection, intravenous nutrition may be essential in maintaining the patient in a good clinical state before starting an elemental diet.

In a few cases with severe coeliac disease, and in patients with severe vomiting and gastrointestinal tumours, intravenous nutrition may be helpful in the short term in improving the patient.

In planning an intravenous nutrition regime, it is important to appreciate the daily requirements of energy and nutrients for patients, while bearing in mind that in patients with disease processes as in many chronic gastrointestinal problems, increased requirements may be necessary. The requirements of water, energy and nitrogen sources, carbohydrate, fat, electrolytes, trace elements and vitamins are shown in Table 5.

Table 5. *Recommended daily nutritional requirements*

	Requirement (per kg body-wt)
Water	30–35 ml
Nitrogen	80–95 mg
Total energy	30–35 kcal (126–147 kJ)
Carbohydrate	2 g
Fat	2–3 g
Sodium	1·0–1·5 mmol
Potassium	0·7–0·9 mmol
Calcium	0·11 mmol
Magnesium	0·04 mmol
Iron	1·00 mmol
Zinc	0·30 mmol
Copper	0·07 mmol
Vitamin B	0·50 mg
Vitamin C	0·50–1·0 mg

https://doi.org/10.1079/PNS19780011 Published online by Cambridge University Press

Fluid requirements are important. The current fluid balance status of the patient must be known and abnormal fluid losses carefully recorded. The clinical condition of the patient also requires to be taken into account. It is generally accepted that approximately 200–250 kcal (840–1050 kJ) are required for each g N given, and that the N provision should be about 95 mg N or 0·7 g amino acids/kg (Lee, 1974; Wretlind, 1974). Potassium (5 mmol) and magnesium (1 mmol) is required for each g N, and there should be adequate electrolyte and vitamin supplementation. The energy sources should be infused concurrently and at least 20% of the energy should be in the form of carbohydrates. N source solutions which are available include both protein hydrolysates and synthetic amino acid preparations. The pure amino acid preparations show the best results on N balance studies but are most expensive. The cheaper peptide hydrolysates may be suitable for most requirements in gastrointestinal disease. The most important sources of energy available are carbohydrate and fat. The principal carbohydrate source of energy is glucose. The rate of glucose intake, however, should not be greater than 0·5 g/kg per h but minimum intake of 100 g carbohydrate/d is sufficient to avoid ketosis and increased protein catabolism. Blood glucose levels must be carefully monitored and insulin may be required. Fructose can be substituted for glucose in many situations provided that the infusion rate does not exceed 0·5 g/kg per h. With fructose there is an increase in the lactic acid level in the liver and a risk of lactic acidosis if given too rapidly. Ethanol is also a valuable source of energy but the richest source of energy available are fat solutions. There are two principal types of fat solutions available; one derived from cotton seed oil (Lypiphysan) and one from soya-bean oil (Intralipid). The principal advantage of fat emulsions is that a large amount of energy can be given in a small volume of isotonic fluid. Linoleic acid (0·1 g/kg) is required to prevent the symptoms of essential fatty acid deficiency in patients on intravenous nutrition, and this is obtained in 15 g of soya-bean oil.

Intravenous nutrients are best administered directly into a large vein such as the superior vena cava or subclavian vein. After insertion of the catheter the position must be checked radiologically. Full aseptic precautions must be adopted. The success of any intravenous nutrition regime is dependent upon daily care of the needle site, daily changing of infusion sets and changing of the catheter at least once every 2 weeks, or more frequently if infection occurs. Antibiotics should be started immediately if infection develops.

Some complications of intravenous nutrition may occur. These include insertion complications such as local infection, pneumothorax and septic thrombophlebitis, and septicaemia must also always be guarded against and treated promptly if it develops. Metabolic complications may also occur. These include the hyperosmolar syndrome and metabolic acidosis. The latter may occur with fructose solutions (Woods & Alberti, 1972), but has also been seen with the use of some L-amino acid preparations. Other complications include essential fatty acid deficiency (Richardson & Sgoutas, 1975), trace element deficiency such as hypophosphataemia (Dudrick, MacFadyen, van Buren, Ruberg & Maynard, 1972),

https://doi.org/10.1079/PNS19780011 Published online by Cambridge University Press

and deficiences of zinc and copper. Hepatic complications such as the cholestatic syndrome and fatty infiltration, may also occur.

Conclusions

Nutritional deficiency and the metabolic complications of severe disease are common in gastrointestinal diseases of many types and must be considered positively and identified rapidly. Gastrointestinal diseases which may be accompanied by such problems are Crohn's disease, ulcerative colitis, coeliac disease, pancreatic disease and gastrointestinal tumours.

With the early recognition of nutritional deficiency and metabolic problems, and its management, the patient with severe gastrointestinal disease will have a much improved prognosis. Their symptoms may improve and he is more able to withstand continuing disease, infection and surgery when required. When such problems are present the most appropriate method of improving nutrition for that particular patient and for the symptoms of that patient should be selected. Ease of administration and cost effectiveness should also be considered. Whichever form of nutritional support is selected for any individual patient, careful and continuous monitoring is required and the side effects of the particular form of therapy looked for, treated if present, and avoided if possible.

REFERENCES

Blackburn, G. L., Bistrian, B. R., Maini, B. S., Schlamm, H. T. & Smith, M. F. (1977). *J. parent. enter. Nutr.* **1**, 11.
Berlyne, G. M., Bruis, R. A. L., Booth, A. M., Mallick, N. P. & Simons, P. J. (1969). *Lancet* **i**, 689.
Bury, K. D., Stephens, R. V. & Randall, H. T. (1969). *Am. J. Surg.* **121**, 174.
Dudrick, S. J., MacFadyen, D. V., van Buren, C. T., Ruberg, R. L. & Maynard, A. T. (1972). *Ann. Surg.* **176**, 259.
Goode, A., Hawkins, S. T., Fegetter, J. G. W. & Johnston, I. D. A. (1976). *Lancet* **i**, 122.
Grundy, D. J. (1971). *Br. med. J.* **2**, 531.
Lee, H. A. (1974). *Br. J. Hosp. Med.* **11**, 719.
Moore, F. D. (1959). *Metabolic care of the surgical patient.* Philadelphia: W. B. Saunders Company.
Nelson, L. M., Carmichael, H. A., Russell, R. I. & Atherton, S. T. (1977). *Gut* **18**, 786.
Richardson, T. J. & Sgoutas, D. (1975). *Am. J. clin. Nutr.* **28**, 258.
Russell, R. I. (1975). *Gut* **16**, 68.
Russell, R. I. (1978). In *Elemental Diets in Practical Nutritional Support* [S. J. Kalin and K. G. W. W. Alberti, editors]. (In the Press).
Troell, L. & Wretlind, K. A. J. (1961). *Acta chir. scand.* **122**, 15.
Voitk, A., Brown, R. A., Echave, V., McArdle, A. H., Gurd, F. N. & Thompson, A. G. (1973). *Am. J. Surg.* **125**, 223.
Voitk, A. J., Echave, V., Brown, R. A. & Gurd, F. N. (1973). *Gastroenterology* **65**, 419.
Woods, H. F. & Alberti, K. G. W. W. (1972). *Lancet* **ii**, 1254.
Wretlind, A. (1974). In *Scientific Foundations of Surgery* (2nd edn.) p. 626 [G. Wells, J. Kyle and E. Gumphy, editors]. London: Heinemann.
Young, E. A., Heuler, N., Russell, P. & Weser, E. (1975). *Gastroenterology* **69**, 1339.

Printed in Great Britain

https://doi.org/10.1079/PNS19780011 Published online by Cambridge University Press