Proceedings of the Nutrition Society (2003), **62**, 703–710 DOI:10.1079/PNS2003283

The Annual Meeting of the Clinical Nutrition and Metabolism Group of the Nutrition Society with the British Association for Parenteral and Enteral Nutrition, in conjunction with the 24th Congress of the European Society for Parenteral and Enteral Nutrition, was held at the Scottish Exhibition and Conference Centre, Glasgow on 4 September 2002

Symposium on 'Intestinal failure'

The medical management of intestinal failure: methods to reduce the severity

Jeremy M. D. Nightingale
Leicester Royal Infirmary, Leicester LE1 5WW, UK

A new definition of intestinal failure is of reduced intestinal absorption so that macronutrient and/or water and electrolyte supplements are needed to maintain health or growth. Severe intestinal failure is when parenteral nutrition and/or fluid are needed and mild intestinal failure is when oral supplements or dietary modification suffice. Treatment aims to reduce the severity of intestinal failure. In the peri-operative period avoiding the administration of excessive amounts of intravenous saline (9 g NaCl/l) may prevent a prolonged ileus. Patients with intermittent bowel obstruction may be managed with a liquid or low-residue diet. Patients with a distal bowel entero-cutaneous fistula may be managed with an enteral feed absorbed by the proximal small bowel while no oral intake may be needed for a proximal bowel enterocutaneous fistula. Patients undergoing high-dose chemotherapy can usually tolerate jejunal feeding. Rotating antibiotic courses may reduce small bowel bacterial overgrowth in patients with chronic intestinal pseudo-obstruction. Restricting oral hypotonic fluids, sipping a glucose–saline solution (Na concentration of 90–120 mmol/l) and taking anti-diarrhoeal or anti-secretory drugs, reduces the high output from a jejunostomy. This treatment allows most patients with a jejunostomy and > 1 m functioning jejunum remaining to manage without parenteral support. Patients with a short bowel and a colon should consume a diet high in polysaccharides, as these compounds are fermented in the colon, and low in oxalate, as 25 % of the oxalate will develop as calcium oxalate renal stones. Growth factors normally produced by the colon (e.g. glucagon-like peptide-2) to induce structural jejunal adaptation have been given in high doses to patients with a jejunostomy and do marginally increase the daily energy absorption.

Intestinal failure: Short bowel: Clinical management: Nutritional support: Water and electrolyte status

The commonly quoted definition of intestinal failure is of 'reduction in functioning gut mass below the minimum amount necessary for adequate digestion and absorption of nutrients' (Fleming & Remington, 1981). This definition has often been interpreted as referring only to patients who need parenteral nutrition. This interpretation would be similar to defining patients as having renal failure only when they needed dialysis. This definition makes no mention of water and electrolyte losses and yet this issue dominates the clinical management of most patients with intestinal failure. Unfortunately, there is no simple clinical or biochemical measurement to define and grade the severity of intestinal failure (as serum creatinine in renal failure or blood gases in respiratory failure), although xylose absorption or post-prandial plasma citrulline (Crenn *et al*. 2000) measurements have been proposed.

A new definition of intestinal failure is of reduced intestinal absorption so that macronutrient and/or water and electrolyte supplements are needed to maintain health and/or growth (Nightingale, 2001*a*). Without such treatment or

Corresponding author: Dr Jeremy Nightingale, fax +44 116 2586985, email jnight@globalnet.co.uk

compensatory mechanisms undernutrition and/or dehydration will result. This definition allows the severity of intestinal failure to be graded according to the type of nutritional support needed (Fig. 1) and a wide range of underlying diagnoses are included (Fig. 2). Acute (or temporary) intestinal failure is potentially reversible and most commonly encountered, with > 90 % of patients with severe intestinal failure being in the peri-operative period (Kennedy *et al*. 2002). Chronic intestinal failure is less common and most patients have a short bowel.

There are four aims in the management of patients with intestinal failure:

1. to provide the nutrition and/or water and electrolytes necessary to maintain health and/or growth;
2. to reduce the severity of intestinal failure;
3. to prevent and treat complications, including those related to the underlying disease, intestinal failure itself or the treatments;
4. to achieve a good quality of life.

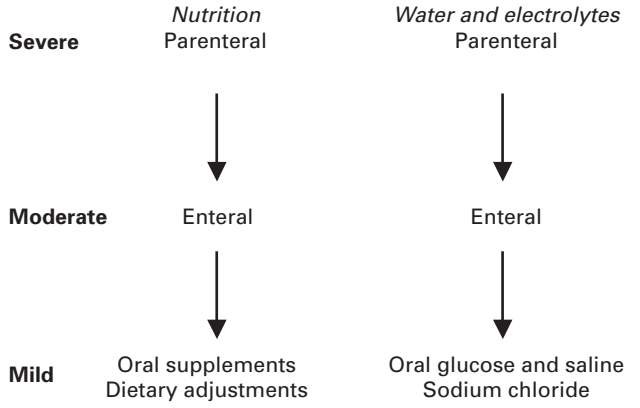

Fig. 1. Severity of intestinal failure. An aim of treatment is to reduce the severity of intestinal failure.

While methods for reducing the severity of intestinal failure include the judicial use of surgery, the present article only addresses medical or dietary methods for reducing the severity of intestinal failure, with particular reference to patients with a short bowel.

Methods for reducing the severity of acute intestinal failure

Patients with acute intestinal failure as the result of a distal entero-cutaneous fistula can often be managed with an enteral feed (often a peptide feed) rather than parenteral nutrition, nil by mouth with or without octreotide (Carlson, 2001). Intermittent small bowel obstruction can be managed with a low residue or liquid diet.

In a study of patients undergoing a colonic resection for cancer two groups each of ten patients were randomized to receive either a 'normal' (more than 3 litres water and 154 mmol Na/24 h) or a 'restricted' (< 2 litres water and 77 mmol Na/24 h) peri-operative fluid management (Lobo *et al*. 2002). On the second post-operative day patients in the 'normal' group had gained 3 kg weight. Measurements of gut function (solid and liquid gastric emptying, time to pass flatus and stool, and time before eating solid food) were significantly slower in the 'normal' group ($P < 0{\cdot}03$ in all cases). Complications were more frequent and hospital stays longer in those having the normal regimen. This study demonstrated that the administration of large amounts of fluid, especially saline (9 g NaCl/l), to peri-operative patients could cause and prolong the period of acute intestinal failure (ileus).

Patients undergoing high-dose chemotherapy, particularly as part of bone marrow transplantation, have traditionally been given parenteral nutrition. However, there is increasing evidence that enteral feeding may suffice (and be safer) if the nausea or vomiting is controlled. Jejunal feeding may be given via a percutaneous endoscopic gastrostomy with a jejunal tube placed through it (Steward *et al*. 2001).

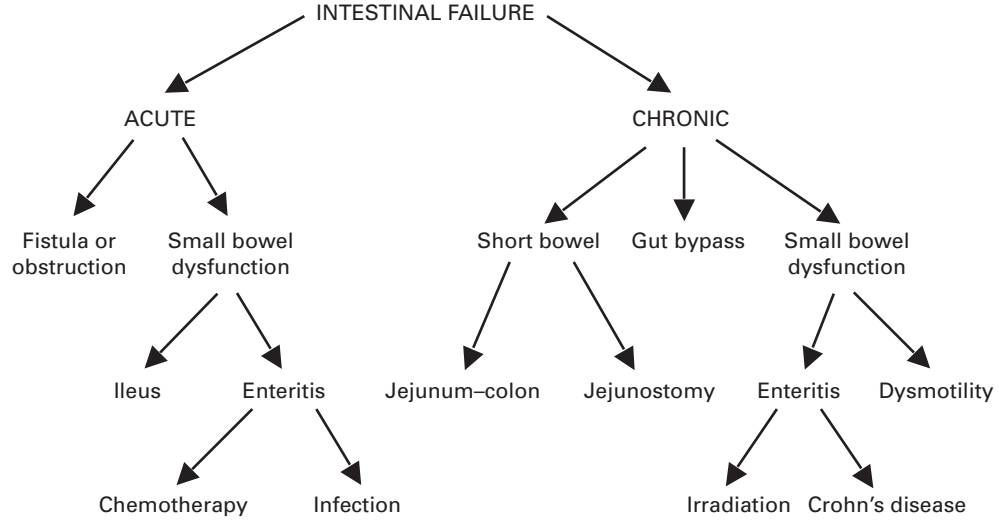

Fig. 2. Classification of intestinal failure.

https://doi.org/10.1079/PNS2003283 Published online by Cambridge University Press

Methods for reducing the severity of chronic intestinal failure: intestinal dysmotility

These patients are relatively uncommon and have the symptoms and signs of intestinal obstruction without a mechanical blockage. This outcome can be due to an intestinal myopathy (e.g. systemic sclerosis or visceral myopathy) or neuropathy (e.g. diabetes or visceral neuropathy) or a combination (e.g. amyloidosis). Treatment addresses the main problems of abdominal pain, vomiting, diarrhoea (constipation in the early stages) and undernutrition. Abdominal pain may be treated with antispasmodics, transcutaneous or sublingual opiates, or octreotide. Vomiting may be helped by prokinetic agents (metoclopramide, domperidone, cisapride, ondansetron or erythromycin) or by antibiotics. Diarrhoea may be helped by drugs that delay gastrointestinal transit (loperamide or codeine phosphate) or by antibiotics (Powell-Tuck *et al*. 2001).

Undernutrition, in addition to vomiting and diarrhoea, may be considerably helped by using oral antibiotics (rotating every 6–8 weeks or short courses of 1–2 weeks) to treat small-bowel bacterial overgrowth. Traditionally, metronidazole, tetracycline and cephalosporins have been given, although an amoxycillin–claevulinic acid combination or ciprofloxacin may be more effective.

Methods for reducing the severity of chronic intestinal failure: short bowel

Two types of patients with a short bowel are shown in Fig. 3, those with a jejunostomy and those with a jejuno–colic anastomosis.

Patients with a jejunostomy have a high-output stoma, which is apparent immediately after surgery, especially when the patient starts to take food and drink. There are two types of patient with a short bowel and a high-output stoma. Net 'secretors' generally have < 1 m jejunum and have an intestinal output that is greater than the oral intake, so that they are in negative intestinal water and Na balance, and thus need parenteral support (Fig. 4). Net 'absorbers', on the other hand, have 1–2 m jejunum remaining and have an intestinal output that is less than the oral intake, so that they achieve positive water and Na balance and can be managed with oral therapy (Nightingale *et al*. 1990).

Patients with a jejuno–colic anastomosis are often well after surgery except for diarrhoea, which is worse with food. They may feel well after the resection but present later with severe weight loss due to malabsorption.

Assessment of a patient with a high-output stoma

A patient with a high-output stoma (usually from the small bowel) is likely to feel thirsty. The patient may have suddenly lost > 2 kg in weight and may have a low urine output. The urea and creatinine levels may be high if the patient is very dehydrated. A random urine Na concentration of < 10 mmol/l suggests Na depletion. Hypomagnesaemia (see p. 706) is common. Measurement of the residual bowel length is useful and can be performed using an opisometer in a small-bowel Ba study if the length was not measured at the time of surgery (Nightingale *et al*. 1991*a*; Carbonnel *et al*. 1996).

Abdominal sepsis and partial small-bowel obstruction can give rise to a high-output stoma and may be excluded clinically with the help of radiology (computerized tomography scans and contrast studies). Occasionally, infective enteritis (e.g. by *Clostridium difficile*), recurrent disease or an internal entero-enteral fistula may cause a high-output stoma. Patients who have suddenly stopped corticosteroids (e.g. after a colectomy for ulcerative colitis) can have acute adrenal insufficiency, which includes an increase in stomal output.

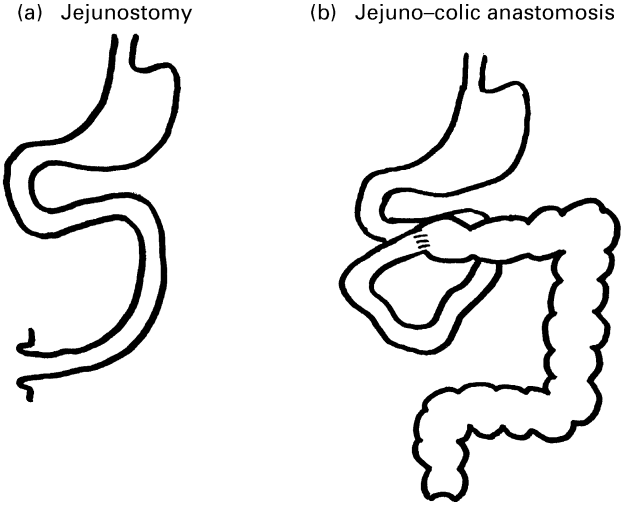

Fig. 3. Types of patient with a short bowel: (a) a jejunostomy; (b) a jejuno–colic anastomosis.

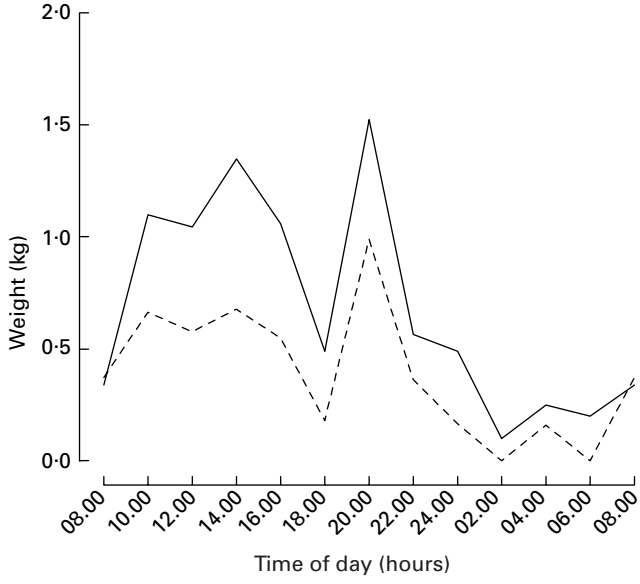

Fig. 4. Oral intake (---) and stomal output (▬) measured every 2 h in a patient who has 0·3 m residual jejunum. Note the large net secretory response to food and drink. (Redrawn from Nightingale *et al.* 1990.)

https://doi.org/10.1079/PNS2003283 Published online by Cambridge University Press

Physiological considerations for a patient with a high-output stoma

Each day 2 litres food and drink are consumed. This intake is diluted by 0·5 litres saliva, 2·0 litres gastric juice and 1·5 litres pancreatico-biliary secretions, so that 6 litres chyme pass the duodeno–jejunal flexure each day (Nightingale & Spiller, 2001). These secretions constitute most of the stomal output from a net 'secretor', and there is little output at night (Fig. 4; Nightingale *et al*. 1990). The concentration of Na in small bowel fluid is about 100 (80–140) mmol/l.

There are important physiological features specific to the jejunum (not present in the ileum). The intercellular junctions in the jejunum are leaky and thus jejunal contents can only be iso-osmolar with plasma. However, there is an active pump that transports glucose and Na together into the cells. Transit through the jejunum is faster than that through the ileum or colon. The jejunum does not absorb bile salts or vitamin B_{12}.

Treatments for a patient with a high-output stoma

To establish hydration the patient is kept nil by mouth and intravenous saline (9 g NaCl/l; with or without Mg) is given to restore hydration and reduce thirst. In addition, the patient's stomal losses, especially that of Na (100 mmol/l), must be replaced. The management that will be outlined is important when a patient resumes an oral intake, even if they continue to need parenteral nutrition and saline, and will reduce the volume of stomal output and the amount of parenteral support needed (Nightingale, 2001*c*).

If water (no Na) or any hypotonic solution is taken orally, Na will diffuse into the intestinal lumen to give a lumen concentration of approximately 100 mmol/l. This Na is not reabsorbed and is, therefore, lost via the stoma. The administration of hypotonic fluid is detrimental, as it increases Na losses (Newton *et al*. 1985; Rodrigues *et al*. 1988; Nightingale *et al*. 1992*b*).

The World Health Organization cholera solution (Avery & Snyder, 1990; 20 g glucose, 3·5 g NaCl and 2·5 g $NaHCO_3$ or 2·9 g sodium citrate in 1 litre tap water) sipped during the day, cold with or without flavouring, can reduce Na losses. While this World Health Organization solution (without K) has a Na concentration of 90 mmol/l and is commonly given, balance studies suggest that a Na concentration of 120 mmol/l gives optimum Na absorption (Rodrigues *et al*. 1988; Nightingale *et al*. 1992*b*) and may still be palatable (Fig. 5).

Drug therapy. As stomal output is mainly in response to an oral intake, drug therapies to reduce output need to be given in the daytime before food. Anti-diarrhoeal drugs (loperamide and/or codeine phosphate) may be given 0·5 h before food to slow gastrointestinal transit and allow more time for absorption. Loperamide 2–8 mg is used in preference to codeine phosphate, as it is not sedating, is not addictive and does not impair pancreatic function (Tytgat *et al*. 1977). Loperamide circulates through the enterohepatic circulation, which is disrupted in patients with a short bowel so that high doses may be needed. Occasionally, it is beneficial to add codeine phosphate (30–60 mg) to loperamide treatment. These drugs are given to all types of patient with a short bowel.

Anti-secretory drugs, however, are generally only effective when the stomal output exceeds the oral intake (i.e. in net 'secretors'). An oral proton pump inhibitor (omeprazole 40 mg daily; Nightingale *et al*. 1991*b*; Jeppesen *et al*. 1998), high-dose histamine receptor type 2 antagonists (e.g. ranitidine 300 mg before breakfast and supper) and octreotide (a somatostatin analogue) may be helpful (Table 1). Octreotide is usually needed if there is insufficient bowel to

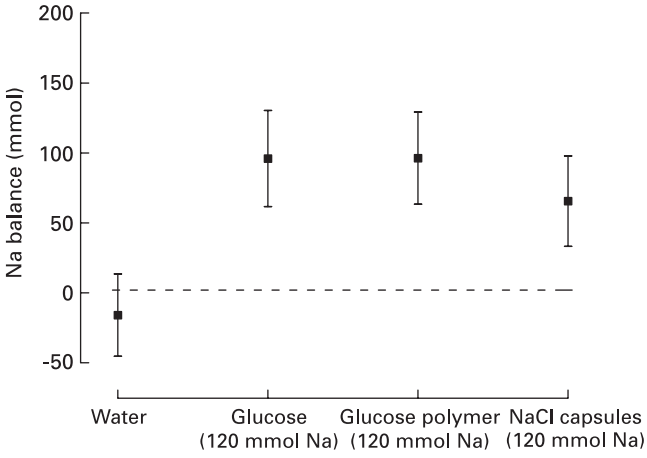

Fig. 5. Daily (mean of 2 d) intestinal sodium balance in six patients with a jejunostomy undergoing different treatments to reduce sodium losses. Values are means with their standard errors represented by vertical bars. (Redrawn from Nightingale *et al*. 1992*b*.)

Table 1. Trials of octreotide in patients with a jejunostomy

Reference	*n*	(Secretors)	Dose	Period of trial (d)	Stomal output	
					Control	Octreotide
Rodrigues *et al*. (1989)*	4	4	50 µg sc	6 h	0·9 kg/6 h†	0·4 kg/6 h†
Nightingale *et al*. (1989)	6	6	50 µg bd iv	2	5·0 kg/24 h‡	2·8 kg/24 h‡
Ladefoged *et al*. (1989)	5	4	50 µg bd sc	2	–	1·4 kg reduction‡
Lemann *et al*. (1993)	7	7	100 µg tds sc	10 h	1·6 kg/10 h†	1·0 kg/10 h†
O'Keefe *et al*. (1994)	10	10	100 µg tds sc	3	8·1 l/34 h†	4·8 l/34 h†

sc, Subcutaneous; iv, intravenous; bd, twice daily; tds, three times daily.
*Liquid meal; other studies involved normal meals.
†Means.
‡Medians.

https://doi.org/10.1079/PNS2003283 Published online by Cambridge University Press

absorb a proton pump inhibitor (approximately < 0·5 m jejunum).

Fludrocortisone may be effective in reducing stomal output if some terminal ileum remains, as it can directly increase ileal Na absorption (Levitan & Goulston, 1967; Goulston *et al*. 1963; Kramer & Levitan, 1972).

Magnesium. Most patients with a jejunostomy are in precarious Mg balance mainly as a result of Na depletion and secondary hyperaldosteronism (Fig. 6). Initially, salt and water depletion (and thus secondary hyperaldosteronism) are treated, then an oral Mg compound (usually three MgO (4 mmol) capsules at night) is given. If these measures fail, 1-α-hydroxycholecalciferol may be given (Selby *et al*. 1984) in gradually-increasing doses, ensuring that hypercalcaemia does not occur. Regular Mg infusions, usually with saline, may be necessary.

Potassium. K problems are unusual, and net loss through the stoma occurs only when < 0·50 m jejunum remains (Nightingale *et al*. 1990). A low serum K level may be consequent upon secondary hyperaldosteronism (Nightingale, 2001*b*) resulting from Na depletion and, thus, large urinary K losses. It may also occur secondary to Mg depletion. Thus, K does not usually need to be given, but Na depletion and hypomagnesaemia should be corrected.

Dietary advice, oral or enteral nutritional support for patients with a short bowel

These patients malabsorb 30–60 % of the oral or enteral nutrition given and this 'malabsorption factor' needs to be taken into account and more energy than normal consumed. To keep up with these losses an enteral feed may be given at night to utilize the gut at a time when it is usually inactive.

Jejunostomy. A patient with a jejunostomy needs a diet or feed that is isosmolar (300 mOsm/kg) and has a Na concentration of about 100 mmol/l. To achieve this requirement large molecules of low osmolality are given (polypeptides, polysaccharides and long-chain triacylglycerols) and extra NaCl is added to the feed.

Jejunum–colon. The presence of a colon is an advantage as water and electrolytes can be absorbed against a concentration gradient, it ferments carbohydrate, slows gastrointestinal transit and promotes adaptive changes.

A diet high in polysaccharides, although bulky, is encouraged as the carbohydrate is fermented in the colon to produce short-chain fatty acids, which when absorbed provide a source of energy (Nordgaard *et al*. 1994; Table 2). A diet rich in polysaccharides needs to be of a considerable size (after the 'malabsorption factor' has also been taken into account). However, as these patients are often fastidious eaters who quickly feel satiated, such a diet is rarely practical. A diet rich in mono- and oligosaccharides can occasionally cause D(−)-lactic acidosis. Lactic acid produced by man is the L(+)-isomer; however, abnormal bacterial or fungal colonization of the colon may form the D(−)-isomer, which after absorption cannot be metabolized and can cause ataxia, blurred vision, ophthalmoplegia and nystagmus. D(−)-lactic acidosis is suspected when these symptoms occur and a

Table 2. Energy absorption in patients with a short bowel given three diets, each for 3 d (Nordgaard *et al*. 1994)

Diet composition (% energy)		Mean energy absorption (%)	
CHO	Fat	Jejunostomy (*n* 6)	Jejunum–colon (*n* 8)
20	60	48	49
40	40	52	61
60	20	55	69***

CHO, carbohydrate.
Mean value was significantly higher than that for the low-carbohydrate diet for the jejunum–colon group: ***$P < 0·001$.

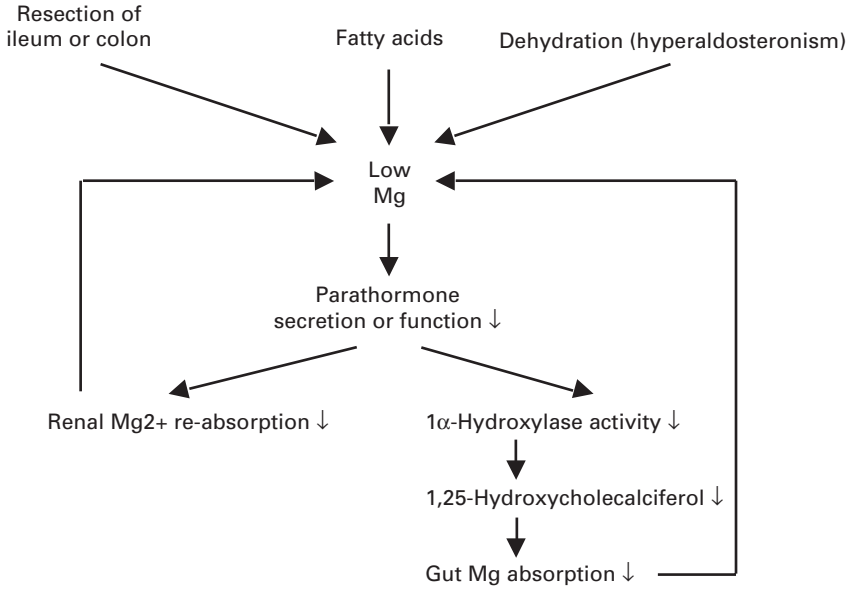

Fig. 6. Diagram showing the reasons for hypomagnesaemia in patients with a jejunostomy. (↓), Decreased.

https://doi.org/10.1079/PNS2003283 Published online by Cambridge University Press

patient has a metabolic acidosis with a large anion gap. Treatment consists of giving broad-spectrum antibiotics (neomycin or vancomycin) and thiamine, and changing the diet to one that is high in polysaccharides but low in mono- and oligosaccharides (Editorial, 1990).

Unabsorbed non-esterified fatty acids resulting from triacylglycerol digestion cause problems within the colon, as they reduce colonic water and Na absorption, increase the colonic transit rate, are toxic to bacteria (so reducing the amount of carbohydrate fermented) and bind divalent cations (Ca and Mg), thus increasing their loss in the stools. A low-fat diet is theoretically ideal as it will reduce diarrhoea and malodorous steatorrhoea, but fat yields twice as much energy as comparable weights of carbohydrate and makes food palatable, thus cannot be excessively restricted. Medium-chain triacylglycerols are an alternative source of energy that can be absorbed in the colon (Jeppesen & Mortensen, 1998).

Calcium oxalate renal stones occur in 25 % of patients with a retained colon (Nightingale *et al*. 1992*a*) because of increased colonic absorption of dietary oxalate. This situation results in increased oxalate excretion in the urine where it may precipitate. The increased colonic absorption of oxalate is partly the result of free unabsorbed fatty acids preferentially binding to Ca, which allows oxalate to become soluble and hence absorbed. Other mechanisms include: unabsorbed bile salts directly increasing colonic permeability to oxalate; *Oxalobacter formigenes*, a species of bacteria that normally metabolize oxalate within the colon, may be absent or present in small numbers; urinary citrate, which prevents initial nucleation of calcium oxalate, may be present in a reduced concentration. The formation of calcium oxalate stones is prevented by advice about a low-oxalate diet (avoid rhubarb, spinach, beetroot, peanuts and excessive amounts of tea), reducing or avoiding excess fat in the diet, taking oral Ca supplements or a Ca-containing organic marine hydrocolloid and/or cholestyramine (to bind bile salts; Tomson, 2001).

In patients with a colon the absorption of nutrients and minerals may continue to improve for 2 years (Gouttebel *et al*. 1989).

New treatments to increase energy absorption

Most new treatments have been aimed at increasing nutrient absorption.

Increasing fat absorption. Cholylsarcosine, a synthetic conjugated bile acid resistant to bacterial deconjugation and dehydroxylation that does not itself cause diarrhoea, has been given to patients with and without a retained functioning colon (Gruy-Kapral *et al*. 1999; Heydorn *et al*. 1999). Taking 4 g three times daily increased fat absorption from 1·4 MJ/d to 2·2 MJ/d without affecting carbohydrate or protein absorption (Heydorn *et al*. 1999); however, this increase in energy absorption was not enough to change the severity of intestinal failure.

Growth factors. The aim of growth factors is to cause hypertrophy of the jejunal mucosa. The initial enthusiasm for combined treatment with growth hormone, glutamine and fibre has waned (Scolapio *et al*. 1997; Szkudlarek *et al*. 2000). Synthetic glucagon-like peptide 2 at a dose of 400 µg twice daily was given subcutaneously to eight patients with 0·30–1·7 m small bowel remaining (six with Crohn's disease, four with home parenteral nutrition) for 35 d. Balance studies (3 d) using identical diets showed increases in mean daily energy absorption of 0·44 MJ (106 kcal; P = 0·09), mean daily wet weight absorption of 0·42 kg (P = 0·04) and solid gastric emptying time for 50 % of the meal of 30 min (P = 0·002; Jeppesen *et al*. 2001). Other growth factors include epidermal growth factor, which has been used for children with necrotising enterocolitis or microvillus atrophy (Walker-Smith *et al*. 1985; Sullivan *et al*. 1991), and aminoguanidine, an inhibitor of polyamine breakdown that has been used in animal studies (Rokkas *et al*. 1990).

Slow gastrointestinal transit. Studies attempting to induce functional adaptation by giving peptide YY analogues have yet to be performed.

Summary

A new definition of intestinal failure that includes reference to water and electrolytes has been given and the severity of intestinal failure graded according to the route by which nutrients and fluid are given. An aim in the management of patients with intestinal failure is to reduce its severity. Acute intestinal failure in the peri-operative period may be prevented or its duration reduced by avoiding the administration of excessive intravenous saline. A patient with a high-output small bowel stoma should restrict oral hypotonic fluids; serum Mg and random urinary Na concentrations should be monitored. The severities of intestinal failure can be reduced in terms of water and electrolyte requirements by careful fluid balance management and the judicial use of drugs. The severity of intestinal failure in terms of macronutrient requirements is rarely markedly altered by dietary or enteral feeding adjustments or the use of growth factors.

References

Avery ME & Snyder JD (1990) Oral therapy for acute diarrhoea. The underused simple solution. *New England Journal of Medicine* **323**, 891–894.

Carbonnel F, Cosnes J, Chevret S, Beaugerie L, Ngo Y, Malafosse M, Parc R, Le Quintrec Y & Gendre JP (1996) The role of anatomic factors in nutritional autonomy after extensive small bowel resection. *Journal of Parenteral and Enteral Nutrition* **20**, 275–280.

Carlson G (2001) Surgical causes and management. In *Intestinal Failure*, pp. 39–49 [JMD Nightingale, editor]. London: Greenwich Medical Media Limited.

Crenn P, Coudray-Lucas C, Thuillier F, Cynober L & Messing B (2000) Postabsorptive plasma citrulline concentration is a marker of absorptive enterocyte mass and intestinal failure in humans. *Gastroenterology* **119**, 1496–1505.

Editorial (1990) The colon, the rumen, and D-lactic acidosis. *Lancet* **336**, 599–600.

Fleming CR & Remington M (1981) Intestinal failure. In *Nutrition and the Surgical Patient*, pp. 219–235 [GL Hill, editor]. New York: Churchill Livingstone.

Goulston K, Harrison DD & Skyring AP (1963) Effect of mineralocorticoids on the sodium/potassium ratio of human ileostomy fluid. *Lancet* **ii**, 541–543.

Gouttebel MC, Saint Aubert B, Colette C, Astre C, Monnier LH & Joyeux H (1989) Intestinal adaptation in patients with short

https://doi.org/10.1079/PNS2003283 Published online by Cambridge University Press

bowel syndrome. Measurement by calcium absorption. *Digestive Diseases and Sciences* **34**, 709–715.
Gruy-Kapral C, Little KH, Fortran JS, Meziere TL, Hagey LR & Hofmann AF (1999) Conjugated bile acid replacement therapy for short-bowel syndrome. *Gastroenterology* **116**, 15–21.
Heydorn S, Jeppesen PB & Mortensen PB (1999) Bile acid replacement therapy with cholylsarcosine for short-bowel syndrome. *Scandinavian Journal of Gastroenterology* **34**, 818–823.
Jeppesen PB, Hartmann B, Thulesen J, Graff J, Lohmann J, Hansen BS, Tofteng F, Poulsen SS, Madsen JL, Holst JJ & Mortensen PB (2001) Glucagon-like peptide 2 improves nutrient absorption and nutritional status in short bowel patients with no colon. *Gastroenterology* **120**, 806–815.
Jeppesen PB & Mortensen PB (1998) The influence of a preserved colon on the absorption of medium chain fat in patients with small bowel resection. *Gut* **43**, 478–483.
Jeppesen PB, Staun M, Tjellesen L & Mortensen PB (1998) Effect of intravenous ranitidine and omeprazole on intestinal absorption of water, sodium, macronutrients in patients with intestinal resection. *Gut* **43**, 763–769.
Kennedy JF, Baker ML & Nightingale JMD (2002) Appropriate parenteral nutrition – the role of the hospital nutrition support team. *Clinical Nutrition* **21**, Suppl. 1, 35.
Kramer P & Levitan R (1972) Effect of 9 α-fluorohydrocortisone on the ileal excreta of ileostomized subjects. *Gastroenterology* **62**, 235–241.
Ladefoged K, Christensen KC, Hegnhoj J & Jarnum S (1989) Effect of a long acting somatostatin analogue SMS 201–995 on jejunostomy effluents in patients with severe short bowel syndrome. *Gut* **30**, 943–949.
Lemann M, de Montigny S, Mahé S, Thuillier F, Huneau JF, Tomé D, Rambaud J-C & Messing B (1993) Effect of octreotide on water and electrolytes losses, nutrient absorption and transit in short bowel syndrome. *European Journal of Gastroenterology and Hepatology* **5**, 817–822.
Levitan R & Goulston K (1967) Water and electrolyte content of human ileostomy fluid after d-aldosterone administration. *Gastroenterology* **52**, 510–512.
Lobo DN, Bostock KA, Neal KR, Perkins AC, Rowlands BJ & Allison SP (2002) Effect of salt and water balance on recovery of gastrointestinal function after elective colonic resection: a randomized controlled trial. *Lancet* **359**, 1812–1818.
Newton CR, Gonvers JJ, McIntyre PB, Preston DM & Lennard-Jones JE (1985) Effect of different drinks on fluid and electrolyte losses from a jejunostomy. *Journal of the Royal Society of Medicine* **78**, 27–34.
Nightingale JMD (editor) (2001*a*) Introduction. Definition and classification of intestinal failure. In *Intestinal Failure*, pp. xix–xx. London: Greenwich Medical Media Limited.
Nightingale JMD (editor) (2001*b*) The short bowel. In *Intestinal Failure*, pp. 177–198. London: Greenwich Medical Media Limited.
Nightingale JMD (editor) (2001*c*) Management of a high output jejunostomy. In *Intestinal Failure*, pp. 375–392. London: Greenwich Medical Media Limited.
Nightingale JMD, Bartram CI & Lennard-Jones JE (1991*a*) Length of residual small bowel after partial resection: Correlation between radiographic and surgical measurements. *Gastrointestinal Radiology* **16**, 305–306.
Nightingale JMD, Lennard-Jones JE, Gertner DJ, Wood SR & Bartram CI (1992*a*) Colonic preservation reduces the need for parenteral therapy, increases the incidence of renal stones but does not change the high prevalence of gallstones in patients with a short bowel. *Gut* **33**, 1493–1497.
Nightingale JMD, Lennard-Jones JE, Walker ER & Farthing MG (1990) Jejunal efflux in short bowel syndrome. *Lancet* **336**, 765–768.
Nightingale JMD, Lennard-Jones JE, Walker ER & Farthing MJG (1992*b*) Oral salt supplements to compensate for jejunostomy losses: comparison of sodium chloride capsules, glucose electrolyte solution and glucose polymer electrolyte solution (Maxijul). *Gut* **33**, 759–761.
Nightingale JMD & Spiller RC (2001) Normal intestinal anatomy and physiology. In *Intestinal Failure*, pp. 15–36 [JMD Nightingale, editor]. London: Greenwich Medical Media Limited.
Nightingale JMD, Walker ER, Burnham WR, Farthing MJG & Lennard-Jones JE (1989) Octreotide (a somatostatin analogue) improves the quality of life in some patients with a short intestine. *Alimentary Pharmacology and Therapeutics* **3**, 367–373.
Nightingale JMD, Walker ER, Farthing MJG & Lennard-Jones JE (1991*b*) Effect of Omeprazole on intestinal output in the short bowel syndrome. *Alimentary Pharmacology and Therapeutics* **5**, 405–412.
Nordgaard I, Hansen BS & Mortensen PB (1994) Colon as a digestive organ in patients with short bowel. *Lancet* **343**, 373–376.
O'Keefe SJD, Peterson ME & Fleming R (1994) Octreotide as an adjunct to home parenteral nutrition in the management of permanent end-jejunostomy syndrome. *Journal of Parenteral and Enteral Nutrition* **18**, 26–34.
Powell-Tuck J, Martin J, Domizio P & Wingate D (2001) Small bowel dysfunction. In *Intestinal Failure*, pp.123–139 [JMD Nightingale, editor]. London: Greenwich Medical Media Limited.
Rodrigues CA, Lennard-Jones JE, Thompson DG & Farthing MJG (1988) What is the ideal sodium concentration of oral rehydration solutions for short bowel patients? *Clinical Science* **74**, Suppl. 18, 69.
Rodrigues CA, Lennard-Jones JE, Walker ER, Thompson DG & Farthing MJG (1989) The effects of octreotide, soy polysaccharide, codeine and loperamide on nutrient, fluid and electrolyte absorption in the short bowel syndrome. *Alimentary Pharmacology and Therapeutics* **3**, 159–169.
Rokkas T, Vaja S, Murphy GM & Dowling RH (1990) Aminoguanidine blocks intestinal diamine oxidase (DAO) activity and enhances the intestinal adaptive response to resection in the rat. *Digestion* **46**, Suppl. 2, 447–457.
Scolapio JS, Camilleri M, Fleming CR, Oenning LV, Burton DD, Sebo TJ, Batts KP & Kelly DG (1997) Effect of growth hormone, glutamine, and diet on adaptation in short-bowel syndrome: a randomized, controlled study. *Gastroenterology* **113**, 1074–1081.
Selby PL, Peacock M & Bambach CP (1984) Hypomagnesaemia after small bowel resection: treatment with 1 alpha-hydroxylated vitamin D metabolites. *British Journal of Surgery* **71**, 334–337.
Steward W, Hunter A, O'Byrne K & Snowden J (2001) Chemotherapy and haemopoietic stem cell transplantation. In *Intestinal Failure*, pp. 65–86 [JMD Nightingale, editor]. London: Greenwich Medical Media Limited.
Sullivan PB, Brueton MJ, Tabara ZB, Goodlad RA, Lee CY & Wright NA (1991) Epidermal growth factor in necrotising enteritis. *Lancet* **338**, 53–54.
Szkudlarek J, Jeppesen PB & Mortensen PB (2000) Effect of high dose growth hormone with glutamine and no change in diet on intestinal absorption in short bowel patients: a randomised, double blind, crossover, placebo controlled study. *Gut* **47**, 199–205.

Tomson CRV (2001) Nephrocalcinosis and nephrolithiasis. In *Intestinal Failure*, pp. 227–242 [JMD Nightingale, editor]. London: Greenwich Medical Media Limited.

Tytgat GN, Huibregtse K, Dagevos J & van den Ende A (1977) Effect of loperamide on fecal output and composition in well-established ileostomy and ileorectal anastomosis. *Digestive Diseases* **22**, 669–676.

Walker-Smith JA, Phillips AD, Walford N, Gregory H, Fitzgerald JD, MacCullagh K & Wright NA (1985) Intravenous epidermal growth factor/urogastrone increases small-intestinal cell proliferation in congenital microvillous atrophy. *Lancet* **ii**, 1239–1240.

https://doi.org/10.1079/PNS2003283 Published online by Cambridge University Press