Tropical Gastroenterology 2010;31(4):244–248

Quarterly Review

Successful management of the short bowel syndrome

K.N. Jeejeebhoy

Emeritus Professor of Medicine, University of Toronto, Canada

Correspondence:
Dr. Khursheed Jeejeebhoy
Email: khushjeejeebhoy@compuserve.com

ABSTRACT

Intestinal failure manifests as diarrhoea, fluid and electrolyte imbalance and malabsorption caused due to surgical resection of small intestine or very rarely due to nonfunctioning of large segment of bowel. Management of short bowel syndrome is quite challenging which requires better understanding of the site and extent of resected segment, pathophysiology of the remaining segment and the time of adaptation. Initial management includes control of diarrhoea with adequate fluid and electrolyte management which is critical for stabilization of the patient. Multidisciplinary approach to the patient is needed.

KEYWORDS: Short bowel syndrome, malabsorption, diarhoea, fluid and electrolyte imbalance.

Intestinal failure

Intestinal failure refers to the clinical effects of extensive small bowel resection. It includes diarrhoea, fluid and electrolyte disturbances and malabsorption associated with malnutrition.

Physiological Considerations

In order to understand the effects of the intestinal resection it is important to review some essential aspects of gastrointestinal physiology.

Gastric Emptying: The rate at which a meal enters the intestine is regulated by the rate of gastric emptying. Of importance in relation to the short bowel syndrome is the fact that intestinal contents entering the distal intestine inhibits gastric emptying.[1]

Small bowel: Small bowel motility is three times slower in the ileum than in the jejunum.[2] It is claimed that the ileocecal valve may slow transit.[3] However, the role of the ileocecal valve in slowing transit is controversial. The small bowel receives about 5-6 liters of endogenous secretions and 2-3 liters of exogenous fluids per day. It reabsorbs most of this volume in the small bowel. The amount reabsorbed in the small intestine depends upon the nature of the meal.[4] With a low carbohydrate meal (low in osmolarity) such as after eating meat and salad , most of the fluid is absorbed in the jejunum whereas with a milk and doughnut meal of high osmolarity, less is absorbed proximally and more distally. In addition the absorptive processes are different in the jejunum as compared with the ileum. These differences depend partly on the nature of the electrolyte transport processes and partly on the permeability of the intercellular junctions. However, the net absorption depends not only upon absorption but upon the extent of back diffusion of the transported material back into the intestinal lumen through "leaky" intercellular junctions. In the jejunum these junctions are very leaky and thus jejunal contents are always isotonic. Fluid absorption in this region of the bowel is very inefficient when compared with the ileum. It has been estimated that the efficiency of water absorption is 44% and 70% of the ingested load in the jejunum and ileum respectively. For sodium the corresponding estimates are 13% and 72%.[5] Hence, the ileum is important in the conservation of fluid and electrolytes.

Colon: The colon has the slowest transit varying between

24-150 hours. The intercellular junctions are the tightest in this part of the bowel and the efficiency of water and salt absorption in the colon exceeds 90%.[5] In addition, carbohydrate is fermented in the colon to short-chain fatty acids (SCFAs) which in turn have two important actions. First, SCFAs enhance salt and water absorption.[6] Second, the energy content of malabsorbed carbohydrates is salvaged by being absorbed as SCFAs. Our recent data suggest that in short bowel patients this salvage may be greater than in normals.[7] Thus, the colon becomes an important organ for fluid and electrolyte conservation and for the salvage of malabsorbed energy substrates in patients with a short bowel.

Effects of intestinal resection

Motility: Gastric motility is enhanced by small bowel resection.[8] While proximal resection does not increase the rate of intestinal transit, ileal resection significantly accelerates intestinal transit.[8,9] In this situation, the colon aids in slowing intestinal transit so that in patients with a short bowel without a colon, an unabsorbable marker fed by mouth was completely excreted in a few hours.[10]

Absorption of fluid and electrolytes: The effect of intestinal resection depends upon the extent and site of resection. Proximal resection results in no bowel disturbance because the ileum and colon absorb the increased fluid and electrolyte load efficiently. The remaining ileum continues to absorb bile salts and thus there is little effluent reaching the colon to impede salt and water resorption. In contrast, when the ileum is resected, the colon receives a much larger load of fluid and electrolytes and also receives bile salts which reduce its ability to absorb salt and water, resulting in diarrhoea. In addition, if the colon is resected the ability to maintain fluid and electrolyte homeostasis is severely impaired.[11]

Absorption of Nutrients: Absorption of nutrients occurs throughout the small bowel and the removal of the jejunum alone results in the ileum taking over most of the lost function. In this situation there is no malabsorption.[12] In contrast, even a loss of a 100 cm of ileum causes steatorrhoea.[13] The degree of malabsorption increases with the length of resection and the variety of nutrients malabsorbed increases.[14,15] Balance studies of energy absorption showed that the absorption of fat and carbohydrate were equally reduced to between 50 and 75 per cent of intake.[16] However, nitrogen absorption was reduced to a lesser extent namely to 81 per cent of intake. In the study of Ladefoged et al,[15] the degree of calcium, magnesium, zinc and phosphorus absorption were reduced but did not correlate with the remaining length of bowel. Our studies showed similar reduction in absorption. The data taken as a whole suggest that it is easier to meet needs for energy and nitrogen by increasing oral intake than the needs for electrolytes and divalent ions. A review of the literature prior to the availability of parenteral nutrition shows that resections up to 33 per cent result in no malnutrition and those up to 50 per cent could be tolerated without special aids but those in excess of 75 per cent require nutritional support to avoid severe malnutrition.[17-27]

Nutritional treatment

Based on the considerations discussed above, the approach to a patient with intestinal resection depends upon the extent of the resection, the presence of continuing intestinal disease that reduces the functional length of the intestine, the site of resected bowel and time for adaptation. The progress of the patient with time will lead to modifications of therapy. However, there are several therapeutic avenues applicable to all patients. First these general approaches are considered and then the specific applications are discussed.

General therapeutic approaches

Initially an assessment should be made to determine whether the patient has had a resection which is unlikely to cause serious malabsorption, such as a jejunal resection leaving an intact ileum and colon. Such patients need observation and are likely to recover full bowel function without the need for nutritional or other therapeutic support. Others who have had a resection of less than 100 cm of terminal ileum will only require the use of a bile salt binder, cholestyramine 4-12 g per day to control bile salt-induced diarrhoea. The remaining patients with a greater length of resection should be treated as follows:-

Initial treatment after resection

Control of diarrhoea-Diarrhoea is due to a combination of increased secretions, increased motility and osmotic stimulation of water secretion due to malabsorption of luminal contents. Soon after resection, diarrhoea is controlled by keeping the patient nil per orally (NPO) to reduce any osmotic component. Gastic hypersecretion can be controlled by the continuous infusion of a proton pump inhibitor. In addition, loperamide

can be used to slow gastric and intestinal transit. If loperamide does not work then codeine or phenoxylate may be tried. However, these agents do not influence the small intestine and act by slowing gastric and colonic motility.

Intravenous fluids

In the immediate postoperative period all patients will require intravenous fluids and electrolytes to replace losses. Sodium and potassium chloride as well as magnesium are the most important ions to be replaced and plasma levels of these ions should be monitored frequently. Fluid is infused according to measured losses and to maintain a urine output of about 2 litres per day. The infusion is tapered as oral intake is increased.

Oral Feeding

The next consideration is to determine the nature of oral feeds. In patients who have more than 60-80 cm of bowel left, re-feeding should be progressive with a view ultimately to feeding a normal oral diet. By contrast in patients who have little small bowel left, the initial target should be small volume isotonic feeds containing a glucose-electrolyte content similar to the oral rehydration solution. The composition of this solution should be glucose 3.4% with sodium 85-90 mM/l, potassium 12 mM/l, bicarbonate 9 mM/l and chloride 80-90 mM/l. Such a solution avoids osmotic stimulation of secretion and yet stimulates the bowel to absorb, thus promoting adaptation. For those having intermediate lengths of bowel progressive feeding should be attempted with the following plan. The same carbohydrate- electrolyte feeds as above should be started. A mixture of a similar composition has been shown to be well absorbed by patients with massive resection who have previously been dependent on intravenous fluids.[28] The diet should be lactose-free since lactase levels in such patients are reduced.[29] Vitamin B_{12} absorption should be measured and if subnormal injections of 200 micrograms per month should be started. While it is popular to try defined formula diets in these patients, studies by McIntyre et al[30] have shown that they are not absorbed better than a solid diet.

Early observations had suggested that low fat diet with medium-chain triglyceride (MCT) and containing a high carbohydrate content was better for patients with a short bowel.[31-33] The theory behind these suggestions was the observation that malabsorbed long-chain fatty acids (LCT) can cause colonic water secretion resulting in higher fecal output with steatorrhea, and consequently greater loss of divalent ions. However, such studies were not controlled and MCT can also cause osmotic diarrhoea. Using a controlled cross over design in two studies[10,16] we showed that a high-fat diet was comparable to a high-carbohydrate diet in regard to total fluid, energy, nitrogen, sodium, potassium and divalent ion absorption.

We therefore recommend a low lactose diet containing high calories from both fat and carbohydrate and a high nitrogen intake. We aim to increase intake gradually to about 60 kcals/kg body weight to provide sufficient absorbed calories despite malabsorption. The rationale for this approach is discussed by Woolf et al.[10] In essence these patients malabsorb 50% of their intake and by doubling the intake we showed that they absorbed sufficient calories for their daily needs. Supplements of potassium, magnesium and zinc are given while monitoring serum levels. We recommend the gluconate and glucoheptonate form of zinc and magnesium because the sulfate form results in the ingestion of an unabsorbable anion (sulfate) which will increase diarrhea.

Parenteral Nutrition

In patients with less than 60 cm of remaining small bowel and in those with a combined small bowel and colon resection parenteral nutrition is lifesaving. It is started in such patients within a few days of the resection and initially 32 kcals/kg of a mixed energy substrate and 1 g/kg amino acids is infused with sodium 150-200 mM, potassium 60-100 mM, calcium 9-11 mM, magnesium 7-15 mM and zinc 70-100 micromoles per day. Among trace elements zinc is the most important as we have found large losses in patients with a high endogenous output of intestinal fluids. We recommend adding a basal amount of 5 mg/day to which is added 12 mg/L of diarrhea + fistula + stomal losses. Oral feeds are simultaneously started and attempts are made to reduce parenteral feeding as oral feeds are increased. It will become apparent whether the patients needs parenteral feeding on a long- term basis. If that is the case then the patient should be started on a program of home parenteral nutrition. We have found that as the bowel adapts over months and even years the patient requires less parenteral feeding and ultimately in about 30 per cent of our patients HPN can be replaced by 2 liters of oral rehydration solution, high calorie diet and supplements of potassium, magnesium, calcium, fat soluble vitamins and zinc. They are monitored regularly until the weight is stable and they are electrolyte balance.

Hypomagnesemia is particularly a serious problem in these patients. Ingestion of magnesium salts orally enhances diarrhea and therefore it often becomes difficult to use magnesium supplements orally. The author has successfully used magnesium glucoheptonate (Magnesium Rougier) for this purpose. This preparation is available as a palatable liquid which is added to the gastrolyte supplement in quantities of 30 mM/d. If this approach is not successful then magnesium sulfate is injected intramuscularly in doses of 12 mM 1-3 times a week to supplement the oral intake.

Vitamin supplementation needs comment. These patients can absorb water soluble vitamins but have difficulty absorbing fat soluble vitamins. They require large doses of vitamin A, D and E to maintain normal levels. Also pills often pass out whole in these patients, hence liquid preparations have to be used. The author recommends the measurement of these vitamin levels and supplementation with aqueous preparations of vitamin A and E (Aqasol A and E) and 1, 25 dihydroxy-vitamin D in doses which normalize the plasma levels. Normalization may not be possible with oral vitamins in some individuals especially vitamin E levels.

In others, an oral diet with intravenous fluid and electrolytes becomes necessary and in the remainder full parenteral nutrition is given.

Special considerations

Somatostatin analogue: Long acting somatostatin analogue has become available and can be given. All studies have shown a reduction in the volume of output and an increase in sodium or chloride absorption.[34-36] However, the reduction did not seem to be sufficient to avoid parenteral nutrition in patients who required it.[35]

Jejunal resection with intact ileum and colon

Patients in this category can be fed orally immediately and rarely have any problems.

Ileal resection of less than 100 cm with colon largely intact

Patients in this category have so called choleraic diarrhea, and are best helped by the administration of 4 g of cholestyramine three times a day to bind bile salts left unabsorbed by the resected ileum. Vitamin B_{12} absorption should be measured and if low should be injected intramuscularly in doses of 100 to 200 μg per month.

Ileal resection of more than 100 to 200 cm with colon largely intact.

These patients have little difficulty in maintaining nutrition with an oral diet, but may have fatty acid diarrhea. For such a patient fat restriction is mandatory. With the larger resection the bile salt pool is depleted and cholestyramine is no longer beneficial. Parenteral vitamin B_{12} replacement is required.

Resection in excess of 200 cm of small bowel and lesser resection with associated colectomy

Patients of this class require the graduated adaptation program indicated previously under general considerations.

Resection leaving less than 60 cm small bowel or only duodenum: Massive bowel resection.

Patients in this category need home parenteral nutrition indefinitely. However many patients even in this category may show a surprising degree of adaptation and require less parenteral nutrition and benefit from orally absorbed nutrients. The indication to reduce parenteral nutrition is weight gain beyond the desired limit and the fact that reduced infusion does not cause electrolyte imbalance and dehydration.

References

1. Hellstrom PM, Naslund E. Interactions between gastric emptying and satiety, with special reference to glucagon-like peptide-1. *Physiology & Behavior*. 2001,**74**:735–41
2. Summers RW, Kent TH,Osborne JW. Effects of drugs, ileal obstruction and irradiation on rat gastrointestinal propulsion. *Gastroenterology*. 1970;**59**:731–9.
3. Ricotta J, Zuidema GD, Gadacz TR, Sadri D. Construction of an ileocecal valve and its role in massive resection of the small intestine. *Surg Gynecol Obstet*. 1981;**152**:310–4.
4. Fordtran JS, Locklear TW. Ionic constituents and osmolality of gastric and small-intestinal fluids after eating. *Am J Dig Dis*. 1966;**11**:503–21.
5. Powell, D.W. Intestinal water and electrolyte transport. In Physiology of the Gastrointestinal Tract. *2nd ed. Edited by L.R. Johnson. New York, Raven Press*, 1987:1267–306

6. BinderHJ, Mehta P. Short-chain fatty acids stimulate active sodium and chloride absorption in vitro in the rat distal colon. *Gastroenterology*. 1989;**96**:989–96.
7. Royall D, Wolever TM, Jeejeebhoy KN. Clinical significance of colonic fermentation. *Am J Gastroenterol*. 1990;**85**:1307–12.
8. Nylander G. Gastric evacuation and propulsive intestinal motility following resection of the small intestine in the rat. *Acta Chir Scand*. 1967;**133**:131–8.
9. ReynellPC, Spray GH. Small intestinal function in the rat after massive resections. *Gastroenterology*. 1956;**31**:361–8.
10. Woolf GM, Miller C, Kurian R, Jeejeebhoy KN. Diet for patients with a short bowel: High fat or high carbohydrate? *Gastroenterology*.1983;**84**:823–8.
11. Cummings JH, James WP, Wiggins HS. Role of the colon in ileal-resection diarrhoea. *Lancet*. 1973;**I**:344–7.
12. Booth CC, Alldis D, Read AE. Studies on the site of fat absorption. 2 Fat balances after resection of varying amounts of small intestine in man. *Gut*. 1961;**2**:168–74.
13. Hoffman AF, Poley JR. Role of bile acid malabsorption in the pathogenesis of diarrhea and steatorrhea in patients with ileal resection. I. Response to cholestyramine or replacement of dietary long-chain triglycerides by medium chain triglycerides. *Gastroenterology*. 1972;**62**:918–34.
14. Hylander E, Ladefoged K, Jarnum S. Nitrogen absorption following small-intestinal resection. *Scand J Gastroenterol*. 1980;**15**:853–8.
15. Ladefoged K, Nicolaidou P, Jarnum S. Calcium, phosphorus, magnesium, zinc and nitrogen balance in patients with severe short bowel syndrome. *Am J Clin Nutr*. 1980;**33**:2137–44.
16. Woolf GM, Miller C, Kurian R, Jeejeebhoy KN. Nutritional absorption in short bowel syndrome: evaluation of fluid, calorie, and divalent cation requirements. *Dig Dis Sci*. 1987;**32**:8–15.
17. Haymond HE. Massive resection of the small intestine: analysis of 257 collected cases. *Surg Gynecol Obstet*. 1953;**61**:693–705.
18. McClenahan JE, Fisher B. Physiologic effects of massive small intestinal resection and colectomy. *Am J Surg*. 1950;**79**:684–8.
19. Trafford HS. The outlook after massive resection of small intestine, with report of two cases. *Br J Surg*. 1956;**44**:10–3.
20. West ES, Montague JR, Judy FR. Digestion and absorption in man with 3 feet of small intestine. *Am J Dig Dis*. 1938;**5**:690–2.
21. Pilling GP, Cresson SL. Massive resection of the small intestine in the neonatal period: report of two successful cases and review of the literature. *Pediatrics*. 1957;**19**:940–8.
22. Martin JR, Pattee CJ, Gardner C, Marien B. Massive resection of small intestine. *Can Med Assoc J*. 1953;**69**:429–33.
23. Kinney JM, Goldwyn RM, Barr JS Jr, Moore FD. Loss of the entire jejunum and ileum, and the ascending colon. Management of a patient. *JAMA*. 1962;**179**:529–32.
24. Walker-Smith J. Total loss of mid-gut. *Med J Aust*. 1967;**1**:857–60.
25. Clayton BE, Cotton DA. A study of malabsorption after resection of the entire jejunum and the proximal half of the ileum. *Gut*. 1961;**2**:18–22.
26. Anderson CM. Long-term survival with six inches of small intestine. *Br Med J*. 1965;**5432**:419–22.
27. Meyer HW. Sixteen-year survival following extensive resection of small and large intestine for thrombosis of the superior mesenteric artery. *Surgery*. 1962;**51**:755–9.
28. Griffin GE, Fagan EF, Hodgson AJ, Chadwick VS. Enteral therapy in the management of massive gut resection complicated by chronic fluid and electrolyte depletion. *Dig Dis Sci*. 1982;**27**:902–8.
29. Richards AJ, Condon JR, Mallinson CN Lactose intolerance following extensive small intestinal resection. *Br J Surg*. 1971;**58**:493–4.
30. McIntyre PB, Fitchew M, Lennard-Jones JE. Patients with a high jejunostomy do not need a special diet. *Gastroenterology*. 1986;**91**:25–33.
31. Andersson H, Isaksson B, Sjogren B. Fat-reduced diet in the symptomatic treatment of small bowel disease: Metabolic studies