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The Clinical Management of Short Bowel Syndrome: Steps to Avoid Parenteral Nutrition

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INTRODUCTION

Short bowel syndrome may result from congenital abnormalities including jejunal or ileal atresia, or may be acquired as in cases of massive resection due to mesenteric venous thrombosis or arterial embolism, trauma, small bowel obstruction, volvulus, necrotizing enterocolitis, inflammatory bowel disease with multiple resections, carcinoma, or radiation enteritis. There are as many as 20 000–40 000 patients that receive long-term parenteral nutrition in the United States.[1] Of these, perhaps 7,000–8,000 have short bowel syndrome.

ADAPTATION

Several factors are important for determining the prognosis of a patient following massive small bowel resection. These include age, length of remaining bowel, the presence or absence of a colon and ileocecal valve, adaptation of the remaining bowel, and the underlying pathology.[2–4] The remaining bowel may be functionally insufficient in patients with active Crohn's disease, radiation enteritis, carcinoma or pseudo-obstruction in the remaining bowel. The minimum length of functional bowel necessary is generally thought to be 100 cm in the absence of an intact colon, and 60 cm in the presence of an intact colon. However, the degree of adaptation may be highly individualized. Patients with <100 cm of residual jejunum may have a net secretory response to food.[5] Hypovolemia and dehydration will occur in the presence of massive small bowel and colonic resections. However, there are reports of infant survival without total parenteral nutrition (TPN) with as little as 30 cm or less.[6,7]

The small bowel has a significant adaptation response to resection. In animal models the intestinal surface area increases because of slightly increased length, but more importantly, significantly increased circumference. Crypt depth and villus width increase in rodents, dogs, and humans.[8–15] Therefore, the absorptive surface area increases. These effects appear to be most significant in the ileum in animal models.[11–13] Accelerated mucosal proliferation is an important part of the adaptation mechanism. These adaptations may occur over a 1- to 2-y period in humans.[15] Similar morphologic changes have been found in humans in the functioning loop of bowel following jejuno-ileal bypass.[16] Fluid and electrolyte absorption by the colon (which hypertrophies) will increase to help compensate for intestinal losses.[17] In addition, animal models suggest that ileal absorption increases within 6–8 wk following resection by an upregulation of enterocyte absorptive capacity.[18]

Massive distal (ileal) resections are less well tolerated than proximal resections because the remaining jejunum cannot adapt as efficiently as the ileum, and the enterohepatic circulation of bile salts will be interupted. In addition, vitamin B_{12} malabsorption occurs.

Massive fluid and electrolyte losses occur due to profound diarrhea during the initial postoperative week or two in patients with significant small bowel resection. Parenteral nutrition and fluids are necessary in this immediate postoperative period. Diarrhea will decrease over the following 1–3 mo, although TPN is generally still necessary. In order for enteral feeding to be tolerated, intestinal transit time must be sufficiently slow in order to permit nutrient absorption along the remaining intestine. In animal models, intestinal adaptation is more rapid and significant in the presence of luminal nutrients.[19,20]

MEDICAL MANAGEMENT

Transient gastric hypersecretion occurs for a few months to a year (at most) postoperatively. This is related to hypergastrinemia, the etiology of which is uncertain. H_2 antagonists or proton pump inhibitors may be useful for decreasing jejunostomy output and potassium losses during this period.[21–23] The rate of gastric emptying is increased. However, with proximal resections, intestinal transit usually remains normal because of the normal, slowed

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transit through the ileum. An intact ileocecal valve will normally prolong intestinal transit and permit greater nutrient contact time with intestinal epithelium. Resection of the ileocecal valve will also permit bacterial colonization of the intestine, with the possibility of bacterial overgrowth. Bacteria compete with enterocytes for nutrients (such as vitamin B_{12}), and malabsorption and diarrhea could result. In addition, fat malabsorption may be increased because bacteria may deconjugate bile salts, resulting in more significant bile salt malabsorption.[24] Antibiotic therapy with either metronidiazole or tetracycline may be necessary to treat bacterial overgrowth. D-Lactic acidosis may also rarely occur when anerobic bacteria ferment malabsorbed simple carbohydrates such as glucose and lactose.[25–27] This condition may be suggested by severe changes in mental status, including slurred speech, ataxia, opthalmoplegia, nystagmus, stupor, and coma. As with bacterial overgrowth, a course of antibiotic therapy with metroniadazole or tetracycline may be helpful. However, antibiotic use may cause vitamin K deficiency, as well as diarrhea (*Clostridium difficile* or non-*C. difficile*). The risk of D-lactic acidosis (rare) should be balanced with the need to supply soluble fiber as an additional energy source (addressed in a subsequent section).

Digestion initially occurs in the duodenum and proximal jejunum where pancreatic proteases, salivary and pancreatic amylase, lipase, bile acids, and intestinal disaccharidases aid in the digestion of macronutrients such as protein, fat, and carbohydrate. The products of digestion are nearly completely absorbed within the first 100–150 cm of jejunum.[28] In addition, minerals such as iron and calcium are absorbed in the duodenum and proximal jejunum, as are the water-soluble vitamins. Resection of <100 cm of ileum may result in cholerrhetic diarrhea and steatorrhea because bile salts are incompletely absorbed in the remaining distal ileum and enter the colon where they are toxic to colonocytes.[29] A bile salt sequesterant such as cholestyramine may be a useful adjunct in the treatment of such resections because it may prevent the transit of unabsorbed bile salts into the colon.[30] Resection of >100 cm will result in the massive loss of bile salts and cannot be compensated by increased hepatic synthesis. Micellar solubilization of dietary fat will not occur because the critical micellar concentration is not reached, owing to the decreased intraluminal bile salt concentration. Cholestyramine may worsen diarrhea by increasing malabsorption. Fat intake should therefore be restricted in patients with remaining colon[29–31]; whereas jejunostomy output appears unaffected by fat restriction.[32] However, too severe a restriction will render meals unpalatable, and caloric intake may subsequently decrease. Bile acid replacement with ox bile has been reported to improve steatorrhea in patients with and without residual colon following ileectomy.[33–35]

Many animal studies (rodent and canine) have demonstrated that intestinal adaptation following massive resection is impaired in the absence of luminal nutrients. Despite claims to the contrary, the available data from humans suggest standard diets and enteral formulas are as efficacious as so-called "specialty, defined-formula or elemental" products for the nutritional maintenance of patients with short bowel syndrome. Woolf et al.[36] randomized eight patients (in a cross-over design) with complete ileal and right colon resections (5/8 had no colon remaining, and 3/8 had only 30.4–50.8 cm of jejunum) to receive isonitrogenous, lactose-free diets that were either high fat (75% nonprotein calories as fat), or a high-carbohydrate diet where the ratio of fat to carbohydrate was reversed. There was no difference in fecal weight, water excretion, osmolality, sodium, potassium, chloride, calcium, magnesium, or zinc output, or mean total calorie, fat calorie and protein + carbohydrate calories absorbed as expressed as a percentage of intake between the two diets. Fecal fat excretion was significantly greater during the high-fat diet, but the proportion of fat absorbed was similar between the two groups. However, neither experimental diet was compared with a regular diet, and it is possible that both experimental diets were either equally better or worse than a regular diet. McIntyre et al.[37] compared a polymeric formula diet with an isonitrogenous, chemically defined diet delivered via nasogastric tube (NGT) in five patients, and orally in two others with jejunostomies (60–150 cm remaining jejunum). No significant difference in stool wet and dry weight, percentage of total calorie, nitrogen, or fat absorption was seen between groups, although there was a wide patient-to-patient variation. Stomal output of sodium, potassium, calcium, and magnesium was similar between groups. Levy et al.[38] found similar results. On the other hand, Cosnes et al.[39] found that a peptide-based diet enhanced nitrogen absorption in six patients with a jejunostomy (90–150 cm of jejunum). However, total calorie, fat, electrolyte, and mineral absorption were unaffected, as was jejunostomy output. Peptide-based enteral formulas may be generally ineffective when used for the purpose of decreasing fecal weight because the major cause of the watery diarrhea seen in patients with short bowel syndrome is carbohydrate, not nitrogen malabsorption.[40] Regardless of the diet or enteral formula used, patients may require double their predicted caloric and protein requirements based on height or body surface area in order to avoid reliance on TPN.

Medium-chain triacylglycerols (MCT) may also be of value as an energy supplement in patients with steatorrhea because they do not require bile salts for digestion. MCTs are a component of many enteral products, although they are also available separately. MCTs, however, do not supply essential fatty acids, are expensive and unpalatable to some patients despite modern recipe innovations, and excessive ingestion may worsen diarrhea. Typically, daily doses of >40 g are poorly tolerated. It should also be noted that in rat studies, intestinal hyperplasia following small bowel resection was greater in animals that received diets containing long-chain triacylglycerols when compared with MCT.[41]

Regardless of the diet, patients with newly acquired short bowel syndrome should probably receive at least some enteral feeding as soon as possible after resection. Usually this is administered for a postoperative period of 7–10 days. Isotonic fluids should be administered; hypotonic fluid ingestion will promote additional salt and water loss. If possible, dry solids should be consumed first, followed by isotonic fluids an hour later. However,

TABLE I.

VITAMIN AND MINERAL SUPPLEMENTS FOR PATIENTS WITH SHORT BOWEL SYNDROME*

Vitamin A	10,000–50,000 units daily
Vitamin B_{12}	300 mcg subcutaneously monthly for those w/terminal ileal resections or disease
Vitamin C	200–500 mg
Vitamin D	1600 units DHT daily; may require 25-OH- or 1,25 (OH_2)-D_3
Vitamin E	30 IU daily
Vitamin K	10 mg weekly
Calcium	See text
Magnesium	See text
Iron	As needed
Selenium	60–100 μg daily
Zinc	220–440 mg daily (sulfate form)
Bicarbonate	As needed

* The table lists rough guidelines only. Vitamin and mineral supplementation must be routinely monitored and tailored to the individual patient because relative absorption and requirements may vary.

this approach is often inpractical. When enteral feeding, bolus feedings should be avoided, at least initially.

All enteral products should be lactose-free. Lactose maldigestion is common because of the decreased intestinal surface area (and lactase-containing brush border).[42] Other foods that may have laxative effects such as caffeine and osmotically active drugs or sweeteners (e.g., sorbitol) should be eliminated from the diet.

Multivitamin and trace mineral supplementation should be routine. Fat-soluble vitamin status (A, D, E, and K) should be routinely monitored in patients who do not require TPN, to which these vitamins are routinely added. Vitamin A (10,000–50,000 U/d), vitamin D (1600 U DHT/d), and vitamin E (30 U/d) may be necessary (Table I). Patients who have concurrent renal insufficiency may be unable to metabolize 25-OH D_3 to its active form (1,25 OH_2D_3), and may require supplementation with the active compound. Both water-soluble vitamin A and D supplements still require micelles for absorption although the water-soluble vitamin E supplement does not. Toxicity from either excessive vitamin D or vitamin A intake may occur. Therefore, serum concentrations of these vitamins should be monitored during supplementation (and calcium in the case of vitamin D), and patients should be monitored for possible signs of toxicity. Vitamin A toxicity may be manifested in headache, vomiting, diplopia, alopecia, dryness of the mucus membranes, hypercalcemia, and elevations in serum hepatic aminotransferase concentrations.[43] Vitamin D toxicity is associated with hypercalcemia, and subsequent progress to renal failure if not recognized.[44] Vitamin K deficiency is rare, except generally in the patient that receives broad-spectrum antibiotics or in the patient without a colon in whom there are fewer vitamin K-producing bacteria. Vitamin K status is monitored by the prothrombin time (PT), which is affected by vitamin K-dependent coagulation factors. Requirements are approximately 1 mg/d.[45]

Water-soluble vitamin deficiencies are rare, even in patients with short bowel syndrome, but certainly may occur. Therefore, such patients should ingest one or two B-complex vitamin supplements and 200–500 mg of vitamin C daily. Excessive vitamin C is to be avoided because of the risk of hyperoxaluria related to increased endogenous production, because oxalic acid is a product of vitamin C metabolism. Toxicity from overdose of other water-soluble vitamins has not been recognized. Vitamin B_{12} should be administered at a dose of 300 μg every 3 mo (intramuscularly) to patients with ileal resections, or in those patients who have diseased ileum (Crohn's disease, radiation enteritis, etc.)

Iron supplements are often unnecessary because iron is absorbed primarily in the duodenum, but serum ferritin should be monitored. Zinc supplements are routinely necessary. Zinc is lost in a concentration of 17 mg/L of small bowel output.[46] Usually one or two 220-mg zinc sulfate tablets will be sufficient. Calcium supplements may also be necessary, especially because low-lactose or lactose-free diets will often be low in calcium as well. However, low plasma calcium concentration may be related to hypoalbuminemia, hypomagnesemia, or vitamin D deficiency. Alternatively, serum calcium concentration may be maintained in the normal range despite deficiency because increased parathyroid hormone secretion will lead to increased bone resorption. Bone disease may also occur because of the metabolic acidosis that results from bicarbonate losses from the small bowel.[47] BiCitra or sodium bicarbonate are of value to maintain normal acid/base status. Magnesium deficiency is difficult to correct using the enteral route because of the cathartic effect of magnesium supplements, and enteric-coated preparations are very poorly absorbed. A recent report suggests oral administration of the injectable formulation of magnesium sulfate may be preferred, although expensive.[48] Selenium deficiency may occur because of chronic stool losses.[49] Plasma selenium concentration can be monitored and replacement supplements used. Daily intake should be 60–100 μg. Deficiency may be associated with myositis, pseudoalbinism, macrocytosis, and cardiomyopathy.[50,51] Copper deficiency is very rare, but copper status should also be monitored. Deficiency has been associated with anemia, neutropenia, testicular atrophy, neuropathy, osteoporosis, retinal degeneration, and cardiomyopathy.[52,53]

Patients who have short bowel syndrome, but have an intact colon, should be placed on a low oxalate diet (Table II). It has been generally thought that oxalate is absorbed in the colon, but not in the small intestine. Normally, dietary calcium binds oxalate (and bile acids), rendering it unavailable for colonic absorption. When steatorrhea occurs, dietary calcium preferably binds unabsorbed fatty acids, rather than oxalate. This allows the oxalate to enter the colon where it is absorbed. This absorption may be enhanced because of colonic damage caused by unabsorbed bile salts.[54] The systemically absorbed oxalate is then filtered in the kidney, where it is free to bind calcium and form calcium oxalate kidney stones. Foods such as chocolate, tea, cola, spinach, celery, and carrots should be avoided. Oral calcium supplements may also be useful.[55] In the absence of a colon, calcium oxalate nephrolithiasis is unlikely to occur, and these patients do not require dietary oxalate restriction.

Other dietary interventions may also be of value in patients with short bowel syndrome who have an intact colon. The role of the colon has often been thought to be limited to fluid and electrolyte absorption. However, the colon may play a significant role in energy absorption. Soluble fiber (e.g., pectin, not wheat bran or soy) and starch that is malabsorbed in the small intestine is metabolized to short chain acids (SCFA) by colonic bacteria. These SCFA, most notably butyrate, are the prefered fuel for the human colonocyte.[56] In fact, the colon can absorb up to 1000 kcal daily via SCFA absorption.[52,57] Although these studies seem to refute the previous data that high-carbohydrate diets are not superior to standard or high-fat diets, at least for patients with an intact colon, it may simply be that only high-carbohydrate diets that contain significant amounts of soluble and some insoluble non-starch polysaccharides are efficacious.

Preliminary data suggest that oral calcium supplementation (2.4–3.6 g elemental Ca) may significantly reduce the amount of diarrhea in jejunol-ileal bypass patients.[58] It therefore may be helpful in patients with short bowel syndrome, although definitive evidence is lacking.

Oral rehydration solutions (ORS) are of use in prevention of excessive fluid losses in patients with short bowel syndrome.[59,60] Long used in cash-starved third-world countries, and routinely by pediatricians in their treatment of children with acute diarrhea in the United States, internists and gastroenterologists rarely resort to such relatively simple inexpensive therapy when intravenous solutions and emergency rooms are so accessible. Although ORS may be more expensive than intravenous dextrose, there is no cost for administration (such as an emergency room or home nursing visit with intravenous catheter insertion for fluid administration). Loperamide hydrochloride (4–16 mg total daily), diphenoxylate, codeine (30–60 mg up to three times a day), or tincture of opium (10 drops two or three times a day) may be required to control diarrhea or ostomy output (Table III).

The long-acting somatostatin analogue, octreotide, has been used to decrease output of jejuno- or ileostomies.[61–63] However, despite the statistically significant results, there is little clinically apparent significance, because the patients studied still had substantial intravenous fluid requirements in addition to their TPN. There are also concerns that octreotide use may result in nutrient malabsorption and gallstone formation (for which short bowel patients are already at increased risk). Therefore, octreotide should be used only in patients with high output ileostomies as a tempo-

TABLE II.

OXALATE CONTENT OF FOODS FOR LOW OXALATE DIET, RESTRICTED TO 40–50 MG DAILY

Food group	Little or no oxalate <2 mg/serving Eat as desired	Moderate oxalate content 2–10 mg/serving Limit: 2 (1/2 cup) servings day	High oxalate >10 mg/serving Avoid completely
Beverages	Apple or pineapple juice Bottled beer Colas (12 oz limit/d) Distilled alcohol Grapefruit juice Lemonade or limeaid (no peel) Wine, red, rose Tap water Milk, yogurt	Coffee, any kind (8 oz) Cranberry juice (4 oz) Grape juice (4 oz) Orange juice (4 oz) Tomato juice (4 oz) Nescafe powder	Draft beer, stout, Juices containing berries Ovaltine and other beverage mixes Tea, cocoa
Meat group	Eggs Cheese, cheddar Lean lamb, beef, or pork Poultry Seafood	Sardines	Baked beans canned in tomato sauce Peanut butter Soybean curd (tofu)
Fruits and vegetables	Avocado Brussels sprouts Cauliflower Cabbage Mushrooms Onions Peas, green Potatoes (Irish) Radishes	Asparagus Broccoli Carrots Corn Cucumber, peeled Green peas, canned Lettuce, iceberg Lima beans Parsnips Tomato, 1 small Turnips	Beans: green, wax, dried Beets: tops, root, greens Celery Swiss chard Chives Collards Dandelion greens Eggplant Escarole Kale Leeks Mustard greens Okra
	Bananas Cherries, Bing Grapefruits Grapes, Thompson seedless Mangoes Melons Cantaloupe Casaba Honeydew Watermelon Nectarines Peaches, Hiley Plums, green/golden	Apples Apricots Black currants Cherries, red sour Orange Peaches, Alberta Pears Pineapples Plums, Damson Prunes, Italian	Blackberries Blueberries Concord grapes Red currants Dewberries Fruit cocktail Gooseberries Lemon peel Lime peel Raspberries Rhubarb Strawberries Tangerines
Breads and other starches	Cornflakes Macaroni Noodles Oatmeal Rice Spaghetti White bread	Cornbread Sponge cake Spaghetti, canned in tomato sauce	Fruit cake Grits, white corn Soybean crackers Wheat germ
Miscellaneous	Mayonnaise Salad dressing Vegetable oils Jelly or preserves (made w/allowed fruits) Salt, pepper (1 tsp/d) Soups made w/allowed ingredients Sugar	Chicken noodle soup, dehydrated	Nuts Peanuts Pecans Chocolate, cocoa Pepper (in excess of 1 tsp/d) Vegetable soup Tomato soup

From Denise M. Ney, R.D., *The Low Oxylate Diet Book for the Prevention of Oxylate Stones,* University of California, San Diego, California, 1981 and *UCLA Manual of Clinical Dietetics,* University of California, Los Angeles, 1986.

rary solution until other methods for the prevention of dehydration resulting from excess fluid loss are successful.

Recent data suggest that a combination of growth hormone, glutamine, and fiber enhances adaptation of the remnant bowel following resection.[64,65] In the initial study by Byrne et al.,[65] significantly improved nitrogen, sodium, water, and caloric absorption were observed after 4 wk of treatment in six patients compared with baseline in an open-labeled study. The patients averaged 44 y of age and had a mean length of approximately 45 cm of jejunum and ileum remaining. All had intact colon. In the second, larger study from the same group, 10 patients with a similar age (and possibly including, it seems, most or all of the same patients) were found to exhibit increased calorie, protein, carbohydrate, water, and sodium absorption. Stool output also decreased. However, closer inspection of the report indicates only 6/10 patients actually required TPN at the beginning of the study, and all patients had an intact colon. This suggests that conventional therapy as discussed previously may have been equally effective for these patients; there was no control group for either study. Soluble fiber and ORS were also used in the study. Only 1/10 patients was at or above their ideal body weight (IBW), with 5/10 under 85% of their IBW, despite over 6 y of home TPN. This suggests that the patients may have been inappropriately managed before entering the study, and protein malnutrition could have resulted in increased malabsorption. Therefore, despite seemingly dramatic and exciting results, these studies should be interpreted with caution until more data, and data from randomized, controlled trials are available. Recent data presented from a placebo-controlled trial at the Mayo Clinic indicates three-week therapy with growth hormone, oral glutamine, and a high complex carbohydrate diet lead to modest improvements in sodium and potassium absorption, but no change in fat or nitrogen absorption, no decrease in stool volume, no improvement in O-xylose absorption, and no improvement in small bowel morphology.[75]

MEDICATION DELIVERY IN SHORT BOWEL SYNDROME

Patients with short bowel syndrome not only have nutrient malabsorption, but may have medication malabsorption as well. Therefore, significantly greater than conventional doses may be required. The clinician may also be forced to explore alternative routes of medication administration in the patient with severe malabsorption and rapid intestinal transit. These may include topical, buccal, intravenous, or aerosol. Oral doses may be effective, but often only with substantially increased doses (e.g., warfarin).[66,67] However, one must also be aware that patients may be very sensitive to small doses of warfarin in the presence of vitamin K deficiency that may occur because of fat malabsorption and bacterial overgrowth. If a patient requires TPN or tube feeding, compatibility issues of nutrients and medications must be considered.

SURGICAL MANAGEMENT

Numerous attempts at bowel lengthening or other means to slow intestinal transit and thereby increase absorptive time (including the use of aperistaltic segments, colonic interposition, and recirculating small intestine loops) have been made for patients with short remaining small bowel remnants.[68,69] None have been considered routinely successful. Success has been reported for interposition of a reversed bowel loop in a preliminary study for patients with approximately 50 cm of remnant bowel.[70]

More recently, small bowel transplantation has been performed, primarily for patients dependent on long-term TPN. Small bowel transplantation has greater similarity to bone marrow transplantation than to solid organ transplants. There is a significant risk of rejection, graft-versus-host disease, as well as a lymphoproliferative disease that appear related to over immunosuppression.[71,72] These complex immunological problems do not appear to be easily controlled with currently available therapies. One-, 2-, and 4-y survival rates are 62%, 48%, and 37%, respectively, in the University of Pittsburgh experience (71 patients at the time of publication), where by far the greatest experience has been generated.[72] A recent report from transplant centers throughout the world that included 170 patients reported 1- and 3-y survival of 57% and 50%, respectively, for isolated small intestine transplants when cyclosporin was used.[72] Survival was increased to 83% at 1 y and 47% at 3 y when tacrolimus was used as the primary immunosuppressive agent.[71] Survival was substantially less for those with a combined small bowel-liver transplant or patients undergoing multivisceral transplantation. However, 78% of survivors were able to discontinue TPN.

The survival rate for patients on home TPN varies depending on age and the underlying disease. For patients with Crohn's disease and congenital bowel defects, the 3-y survival is approximately 90%, approximately 70% for those with ischemic bowel diseases, chronic adhesive obstructions, and motility disorders, but only 30% for patients with malignancies and 20% for those with AIDS.[73] These figures are significantly better than for comparable patients who have undergone small intestine transplantation. However, the longer term complications of TPN are more recently becoming known. Despite this, the current indication for small intestinal transplantation should be only for patients with short bowel syndrome and TPN-associated hepatic failure, in whom an orthotopic liver transplant is necessary. Small bowel transplantation is hazardous and extraordinarily expensive. It is not a true therapeutic option for patients who are stable, and satisfactorily maintained on home TPN. It is extremely rare for patients who require long-term home TPN to lose venous access, although thoracotomy for line placement may become necessary in extremely rare instances.[74] Because in the absence of hepatic failure, small intestinal transplantation must be considered an elective procedure and the few number of patients that would actually require such a procedure, it is unlikely that clinical experience will develop as rapidly as with other organ transplants. The costs of the transplant as well as for immunosuppressive medication and treatment of transplant-related medical issues are extremely expensive and have not been fully described. While the charges for home TPN may range as high as $100,000–$150,000 yearly in the United States (unpublished observations), in a managed care environment it is the actual costs, not the charges, that become important. One day's supply of TPN

TABLE III.

TREATMENT OF DIARRHEA IN THE SHORT BOWEL PATIENT*

Oral rehydration solutions (Rehydralyte®, Pedialyte®, Ricealyte®, WHO glucose-electrolyte formula)	As needed
Loperamide hydrochloride	4–16 mg daily
Diphenoxylate	4–16 mg daily
Codein	Up to 30–60 mg three times per day
Tincture of opium	10 drops two to three times per day

* Suggested in order of use or need.

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