Clinical Reviews
The Small Intestine and Nutrition

Nutritional Management of Short Bowel Syndrome in Adults
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Abstract
Short bowel syndrome (SBS) comprises the sequelae of nutrient, fluid, and weight loss that occurs subsequent to greatly reduced functional surface area of the small intestine. Signs and symptoms of SBS include electrolyte disturbances; deficiencies of calcium, magnesium, zinc, iron, vitamin B12, or fat-soluble vitamin deficiency; malabsorption of carbohydrates, lactose, and protein; metabolic acidosis, gastric acid hypersecretion; formation of cholesterol biliary calculi and renal oxalate calculi; and dehydration, steatorrhea, diarrhea, and weight loss. Thorough nutritional management is the key factor in achieving an optimal outcome in SBS. Total parenteral nutrition is necessary in the early stages, as is replacement of excess fluid and electrolyte losses. Nutritional management of SBS has traditionally been divided into three phases: an acute phase when total parenteral nutrition is usually begun, an adaptation phase, and a maintenance phase. Recommendations regarding the need for parenteral nutrition vary depending on the presence or absence of certain factors: the ileocecal valve, jejunum, and functional colon. Patients with residual small bowel length of 100 cm or less usually require the administration of parenteral nutrition at home with good results. The total parenteral nutrition diet should consist of a majority of calories from fat, followed by protein, and the remaining as carbohydrates. Vitamins, minerals, and trace elements should also be added accordingly. Although total parenteral nutrition is initially necessary, treatment goals should focus on early transition to enteral nutrition followed by oral feeds. Other recent advances in the medical management of SBS include pharmacologic treatment and the use of specific nutrients and growth factors to stimulate intestinal absorption and adaptation. Both animal studies and clinical trials in humans have shown much promise in supplementation with growth factors and hormones. This strategy is likely to play a greater role in the treatment of SBS in the future.

Key Words: Short bowel—Total parenteral nutrition—Supplementation—Colon—Adaptation.

PATHOPHYSIOLOGY OF SHORT BOWEL SYNDROME

Most authors agree that SBS occurs when greater than 70% to 75% of the small intestine is resected (the average normal length is approximately 600 cm).9,10,12 This reduced absorptive surface area leads to an abnormally rapid transit of intestinal contents.9,10,12,13 Prognosis for patients with SBS is related to the length of remaining bowel.14 Although almost all nutrients are usually absorbed in the first 100 to 150 cm of the small bowel, the specific region of the bowel resected determines the nutritional needs of patients because certain areas of the small intestine are more efficient at absorption than others.2,14,15

Resections of the duodenum alone are rare and do not result in SBS, although they do play a role in dumping syndrome.5 Removal of the duodenum causes poor tolerance of concentrated sugars, lactose intolerance, and mal-
The ileum is usually able to compensate for these absorptive functions if the jejunum is removed.2,5

The ileum, however, cannot compensate for the reduced levels of jejunal inhibitory enterohormones such as gastric inhibitory peptide and vasoactive peptide that result from jejunal resections.17 Gastric hypersecretion subsequent to increased production of gastrin occurs to a greater extent after jejunal resections than ileal resections. The increased gastric secretions and acidity inactivate pancreatic enzymes, reducing the efficiency of protein and lipid digestion.18 The excess gastric acid and low intraduodenal pH may damage the bowel mucosa, inactivate digestive enzymes, and stimulate peristalsis.5,13 Decreased secretion of cholecystokinin and secretin further reduces gallbladder contraction and pancreatic secretion. These factors along with secretion of a high salt load by the stomach may compound the diarrhea associated with SBS.8

Patients with a jejunostomy may initially experience problems with electrolyte and fluid balance because liquids empty rapidly, although solids empty normally.2,19,20 Patients who have 60 to 100 cm of jejunum intact tend to have large stomal outputs and absorb less than 35% of energy available.21 Extensive sodium depletion may lead to hypotension and prerenal uremia. Chronic sodium loss may be associated with low plasma volume, reduced sodium output in urine, and increased plasma aldosterone.22,23

The ileum ranges from 300 to 400 cm (equivalent to 12 ft), with the distal 100 cm being the most critical physiologically.5 Although the ileum plays a small role in the digestion and absorption of macro- and micronutrients, its slower rate of peristalsis explains the fact that distal resections of the small intestine lead to more complications than do proximal resections.5,24 Interestingly, the functional length of the ileum is almost double that of the jejunum.14 Resection of the ileum may destroy the ileal brake that tends to slow transit through the stomach and proximal small bowel; this results in decreased interaction among the absorptive surface area, luminal contents, and the digestive juices.25

Excessive loss of micronutrients such as magnesium, zinc, copper, and selenium tends to occur in SBS. Iron deficiency can result from loss of duodenal–jejunal absorptive area, or from perianastomotic ulceration as a late complication of ileocolic anastomosis. Deficiencies of watersoluble vitamins such as B12 and other group B vitamins occur when there is extensive loss of jejunum, and distal ileal loss may prevent adequate absorption of intrinsic factor-bound B12. Routine injections of B12 with long-term follow-up of B12 status may be necessary.22

Furthermore, the ileocecal valve is a physiologic sphincter that slows the emptying of chyme into the colon and prevents reflux of colonic bacteria into the small intestine.14,24 Malabsorption, absence of the ileocecal valve, and hypomotile dilated bowel segments increase the risk of small bowel bacterial overgrowth. This results in deconjugation of bile acids, mucosal inflammation, and further compromise of digestion and absorption. Malabsorption of peptides and amino acids leads to nitrogen deficiency, and resulting malnutrition will further impair pancreatic exocrine function.16,23,26,27

Short bowel syndrome is also marked by an increased prevalence of cholelithiasis subsequent to interruption of enterohepatic circulation of bile salts, changes in gallbladder emptying, and cholestasis associated with parenteral nutrition. Furthermore, steatorrhea leads to deficiency of fat-soluble vitamins with the possibility of night blindness, osteomalacia, and bleeding secondary to the loss of vitamins A, D, and K, respectively.15 Although bone disease during parenteral nutrition is common and multifactorial in etiology, long-term problems related to vitamin D deficiency in those who have achieved full enteral feeding appear to be rare, even in the absence of supplementation.28,29

ROLE OF THE COLON
Compensatory Functions of the Colon

The presence of a functioning colon is also important after extensive small bowel resection because it not only gives added bowel length but also slows intestinal transit.30 Preservation of at least half of the colon is equivalent to maintaining 50 cm of small bowel; this reduces fecal loss of nutrients and improves a patient’s ability to survive without parenteral support.15,31–33

The colon is capable of increasing its absorptive capacity threefold to fivefold.25 For example, the main functions of the colon are to absorb sodium and water and excrete potassium and bicarbonate.14,34 Presence of a functional colon after small bowel resection helps to slow the rate of early gastric emptying of liquids.35 In the adult, 0.5 to 1.5 L of fluid are salvaged each day via the “colonic brake.”20,35,36

Because of the efficiency with which the small intestine digests fat and protein, only small amounts of these compounds reach the colon.31–33 Carbohydrate absorption occurs via a different mechanism than that of proteins and fat. The normal small intestine absorbs only 2–20% of dietary starch so that the remaining starch becomes available for bacterial fermentation in the large intestine along with dietary fiber, sloughed epithelial cells, mucins, and intestinal enzymes.25,27,31–33,37 The colonic bacteria ferment the 60–80 g/d of unabsorbed complex carbohydrates into short-chain fatty acids (SCFAs), namely, acetate, butyrate, and propionate.25,27,31–33,37 These SCFAs enter the portal...
circulation to provide a source of calories and trophic stimuli to the colonocytes for basal energy production and multiplication.25,27,31

Healthy people with intact small and large intestines retain greater than 95% of dietary energy and lose less than 5% in feces. This is supported by the evaluation by Nordgaard et al.32,33 of the maximum amount of energy salvaged during colonic digestion in humans. Their finding that the energy absorption of the colon may exceed 4.2 MJ/d emphasizes the importance of sparing as much nondiseased bowel as possible in patients who are at risk for developing chronic bowel failure because the colon does much more than absorb water and electrolytes as originally thought. Thus, patients with short bowel and colon in continuity benefit from a high-carbohydrate, high-calorie diet because of colonic absorption of carbohydrates.32,33

Adverse Effects of an Intact Colon

The presence of a colon may involve several problems, namely, diarrhea, lactose intolerance, lactic acidosis, and formation of calcium oxalate renal stones. Colonic bacteria deconjugate the bile salts entering the colon to free bile acids that stimulate secretion, leading to the development of watery diarrhea. Massive resection of the ileum (intestinal remnant less than 50 cm) leads to loss of bile acid to an extent that exceeds production of hepatic bile salts. Decreased production of the bile acid pool causes fat malabsorption with subsequent steatorrhea. Increased diarrhea occurs when colonic bacteria hydroxylate long-chain fatty acids within the colon. Lactose from milk and other liquids is not as easily digested in patients with SBS as that from other sources such as yogurt.38,39 The lactose is unhydrolyzed, leading to hyperosmolarity in the intestinal lumen.40 Furthermore, this malabsorbed lactose in the small intestine becomes fermented in the colon to SCFAs and gases such as hydrogen and carbon dioxide (and in some cases, methane) that produce abdominal discomfort, flatulence, and osmotic diarrhea.40,41

Loss of mucosa containing border hydrolases adversely affects carbohydrate digestion. Nonabsorbed sugars produce osmotic diarrhea, and, rarely, severe metabolic acidosis through conversion of carbohydrate by lactobacilli to D-lactic acid. Certain species of Lactobacillus (e.g., L. acidophilus and L. casei) produce D-lactate.42

Additionally, colonic bacteria metabolize fatty acids to D-lactate and SCFAs. This is associated with a neurologic syndrome including headache, drowsiness, behavioral disturbance, ataxia, and blurred vision.15,42–45 Lactic acidosis may be worse if there is coexisting thiamine deficiency.46 Metabolic abnormalities include hyperchloremia, an elevated anion gap, and elevated serum and urinary D-lactate (with normal serum L-lactate).42,45

Iron, calcium, and magnesium are maximally absorbed in the proximal small bowel. After small bowel resection, the increased absorption of dietary oxalate, which—under normal circumstances—would form insoluble calcium oxalate in the bowel lumen, results in hyperoxaluria and increases the risk of forming renal oxalate stones. Twenty-five percent of adult patients with less than 200 cm jejunum anastomosed to the colon tend to develop symptomatic renal stones.47

NUTRITION SUPPORT IN SHORT BOWEL SYNDROME

Thorough nutritional management is the key factor in achieving an optimal outcome in SBS. Total parenteral nutrition (TPN) is necessary in the early stages, as is replacement of excess fluid and electrolyte losses. Nutritional management of SBS has traditionally been divided into three phases: an acute phase, an adaptation phase, and a maintenance phase.

Phase I: Acute Phase

Phase I, the acute phase, occurs during the immediate postoperative weeks and may last 1 to 3 months. This phase is marked by poor absorption of almost all nutrients, including water, electrolytes, proteins, carbohydrates, fats, vitamins, and trace elements.8 Fluid loss from the gastrointestinal tract tends to be greatest during the first few days after massive small intestinal resection; ostomy outputs may exceed 5 L/d. Aggressive fluid and electrolyte replacement therapy is necessary to reduce life-threatening dehydration, hypotension, and electrolyte imbalances. Frequent measurements of vital signs, intake and output, and central venous pressures are required because of rapid metabolic changes and possible hemodynamic instability.8,15

Almost all patients with SBS have abnormal liver function; most will experience transient hyperbilirubinemia.8 This may be caused by of the translocation of microorganisms and/or their toxins through ischemic or gangrenous intestinal mucosa into the portal vein secondary to the reduced mesenteric venous return subsequent to massive small-bowel resection.48

Cyclical parenteral nutrition (overnight feeding) may decrease the risk of hepatic complications. Additionally, treatment with oral antibiotics such as neomycin and metronidazole may reduce deleterious bacterial translocation across the gut. Because low plasma concentrations of cholecystokinin during parenteral nutrition may be associated with risk of cholestasis, prophylaxis with cholecystokinin may be helpful.49

Enteral feeding should also be initiated at this time; however, it must be emphasized that to be tolerated, intestinal transit time must be relatively slow to permit nutrient absorption along the remaining intestine.50

Total Parenteral Nutrition

The inability of the bowel to adequately absorb nutrients early in the course of SBS warrants immediate treatment
with TPN. The prognosis of SBS has greatly improved during the past 25 years as a result of TPN. Total parenteral nutrition usually may be initiated on the second or third postoperative day, when the patient’s cardiovascular and pulmonary functions have stabilized. Initially, daily electrolyte monitoring is continued until stabilization is achieved, after which monitoring may be decreased accordingly.

Recommendations regarding the need for parenteral nutrition vary depending on the presence or absence of three key structures: jejunum, the ileocecal valve, and functional colon. If the duodenum and 60 cm of jejunum or ileum remain intact, a favorable prognosis is likely. Furthermore, the ileocecal valve can effectively increase the absorptive capacity of the remaining small bowel up to two times that expected for the same length of small bowel without an intact ileocecal valve (Table 1).

Patients who retain less than 50 cm of jejunum will usually need parenteral nutrition permanently even if most of the colon is functional. Patients with jejunostomy whose total small intestinal remnant is less than 100 cm will also require TPN; however, some jejunostomy patients with more than 100 cm of small intestine may still require enteral supplementation of water and minerals. Patients with less than 30 cm of bowel and a competent ileocecal valve will require indefinite home parenteral nutrition, as will patients with between 60 and 100 cm of small bowel without an ileocecal valve.

**Composition of Total Parenteral Nutrition**

The average caloric recommendation for TPN is 32 kcal/kg/d. Patients who rely exclusively on home parenteral nutrition should be given the following per day: 20% to 30% of total calories as intravenous fat to prevent essential fatty acid deficiency, 1 to 1.5 g/kg of protein per day, and 30% of total calories as carbohydrates. The method provides the additional benefits of minimizing immunosuppression of the reticuloendothelial system as well as reducing elevations in liver enzymes. Vitamins, minerals, and trace elements should also be added to the formulation to restore and maintain normal blood concentrations.

**Adverse Effects of Total Parenteral Nutrition**

Malnutrition or prolonged periods without enteral nutrition lead to decreased gut surface area, mass, and function. Lack of oral feedings induces mucosal atrophy. The high cost of home parenteral nutrition (approximately $100.00/y) compounds the substantial adverse effects of TPN. Those patients who receive only parenteral feedings without concomitant enteral nutrition frequently manifest multiple nutrient deficiencies. These include essential fatty acid deficiency, which may be more severe in patients with Crohn’s disease, as well as deficiencies in fat-soluble vitamins and minerals that tend to occur in patients with high-output stomaties. Deficiencies of zinc, copper, chromium, manganese, selenium, and molybdenum as well as a potentially lethal deficiency of thiamine have also been found in patients receiving TPN.

The survival rate for patients receiving home parenteral nutrition for nonneoplastic conditions has been found to be 70% at 3 years. Patients who receive home TPN tend to be hospitalized several times per year, most often because of catheter-related septicemia. Other causes of morbidity and mortality associated with prolonged dependence on parenteral nutrition—and lack of enteral nutrition—result from hepatic dysfunction, progressive renal insufficiency, bone demineralization, and intestinal failure (there is a 95% probability of intestinal failure in patients dependent solely on TPN for 2 or more years).

Hepatic dysfunction is caused by direct toxic effects of amino acid solutions on hepatocytes, loss of integrity of the gastrointestinal immunologic barrier with subsequent bacterial translocation into the portal venous system, enteric mucosal atrophy, intestinal bacterial overgrowth, and imbalance of carbohydrate/polypeptide/lipid in TPN formulations. Patients who receive prolonged TPN are likely to develop mild steatosis because of excess calories or lack of essential fatty acids; this tends to develop early but paradoxically resolves despite continued parenteral nutrition. A few patients develop severe hepatic complications such as steatohepatitis, intrahepatic cholestasis, periportal inflammation, fibrosis, and eventually liver failure.

**Phase II: Adaptation of the Residual Intestine**

Phase II, the adaptation phase, generally begins within 24 to 48 hours after resection and may last from 1 to 2 years. During this period, 90% to 95% of the bowel adaptation potential (including nutritional and metabolic stability) has been realized, and only 5% to 10% of additional improvement in bowel adaptation and absorption is possible. Adaptive changes also take place in the stomach, pancreas, and colon. Clinical manifestations of intestinal adaptation include weight change and stabilization of fluid and electrolyte levels.

Adaptation involves increased mucosal surface area through enterocyte hyperplasia and migration within the villus leading to villous hyperplasia and increased crypt depth. Intestinal dilatation and lengthening also occur. Distal resection tends to show a greater adaptive response than more proximal resections; that is, the ability of the ileum to adapt is greater than that of the jejunum, although the jejunum is capable of enhanced absorption.

The most beneficial role of the colon in adaptation may be its ability to salvage calories by converting unabsorbed carbohydrates entering it into absorbable SCFAs. As
<table>
<thead>
<tr>
<th>Location of resection</th>
<th>Duodenum</th>
<th>Jejunum (ileum intact)</th>
<th>Ileum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indications for resection</td>
<td>Bilroth I and II, Whipple’s procedure</td>
<td>Trauma, adhesions, Crohn’s disease, volvulus, necrotizing enterocolitis</td>
<td>Crohn’s disease</td>
</tr>
<tr>
<td>Adverse effects of resection</td>
<td>Lactose intolerance, poor tolerance of concentrated sugars, anemia, malabsorption of calcium, iron, and magnesium</td>
<td>Malabsorption of calcium, iron, and zinc; deficiency of copper; deficiency of folate</td>
<td>Malabsorption of fat and fat-soluble vitamins, malabsorption of vitamin B&lt;sub&gt;12&lt;/sub&gt;/intrinsic factor, cholelithiasis, hyperoxaluria</td>
</tr>
<tr>
<td>Length of resection/size of remnant</td>
<td>Total small intestinal remnant &gt;100 cm</td>
<td>Total small intestinal remnant &lt;100 cm or &lt;50 cm jejunum remaining (colon intact)</td>
<td>Ileal resection</td>
</tr>
<tr>
<td>Type of nutrition necessary after resection</td>
<td>Oral; parenteral supplementation of water and minerals as needed with gradual advancement to oral nutrition</td>
<td>Indefinite TPN</td>
<td>Oral</td>
</tr>
<tr>
<td>Supplements required after resection</td>
<td>Calcium, iron, magnesium</td>
<td>IV fluids, electrolytes, calcium, iron, zinc, copper, folate</td>
<td>Fat-soluble vitamins, vitamin B&lt;sub&gt;12&lt;/sub&gt;; trace elements: iron, zinc, copper, selenium, magnesium, chromium</td>
</tr>
</tbody>
</table>

IV indicates intravenous; TPN, total parenteral nutrition.
much as 500 kcal/d can be absorbed through the colon in adults.\textsuperscript{43} Interestingly, the colonic bacteria themselves are able to adapt. Bacteria are able to adapt by increasing the production of SCFAs if provided with an increased amount of substrates for colonic fermentation.\textsuperscript{24,43} This adaptation has the potential to increase energy from colonic digestion.\textsuperscript{24,43}

Luminal nutrition is essential for complete adaptation and should be initiated as early as possible. Other trophic influences include pancreaticobiliary secretions and gut hormones such as enteroglucagon, gastrin, epidermal growth factor, and insulin-like growth factor.

**Effects of Parenteral versus Enteral Nutrition in Phase II**

If the total amount of small intestine remaining is 100 cm or less, TPN is essential for survival. In such cases it is unrealistic to assume that adaptation will occur to an extent that will allow the patient to rely completely on enteral nutrition.\textsuperscript{5} When transition from TPN to enteral nutrition is possible, enteral feedings are preferred because they help to maintain a stable body weight and prevent large fluctuations in fluid balance.\textsuperscript{67} Some researchers consider enteral nutrition to be the most important stimulus for adaptation.\textsuperscript{14,68} Total parenteral nutrition should be gradually reduced, as enteral nutrition is gradually increased.\textsuperscript{67} For example, attempts to wean from TPN may be accomplished by first decreasing the volume administered to 1 L/d and then by reducing the frequency of administration to every other day, then every third day, etc.\textsuperscript{14}

Because parenteral nutrition without concomitant enteral feeding may suppress the appetite and lead to mucosal hypoplasia, luminal nutrients play an important role in adaptation.\textsuperscript{24,43} In fact, villous atrophy caused by TPN or surgical bypass has been reversed in some cases by administration of luminal nutrients.\textsuperscript{14} The presence of nutrients in the lumen seems to promote an adaptive hyperplasia that results in increased length and weight of the intestines. Migration and proliferation of cells from the intestinal crypts leads to an increase in the cell population of both the mucosa and the villi. Absorption per unit of intestinal length also increases. Enterocyte enzymes may also be stimulated commensurate to the composition of the luminal feeding given.\textsuperscript{14}

Enteral nutrients (e.g., glutamine, soluble fibers, and SCFAs) act directly via contact with intestinal epithelium; they also act indirectly to stimulate the secretion of intestinal trophic hormones (e.g., growth hormone, epidermal growth factor, enteroglucagon, insulin-like growth factor I).\textsuperscript{69} Oral replacement fluids might be used initially, followed by a dilute, defined-formula diet.\textsuperscript{5,70} Patients with very short bowel (<60 cm) and those who do not tolerate oral feeds should be maintained on a defined-formula diet. This may be accomplished by constant infusion, starting with a diluted diet infused at approximately 25 mL/h.\textsuperscript{5} Both the concentration and rate should be increased commensurate with the patient’s tolerance until a concentration of 100 to 125 mL/h is reached.\textsuperscript{5}

With resections greater than 75%, complete adaptation may take 6 months to a year; therefore, it is important to refrain from excessive enteral or oral nutrition before the patient is truly ready for such support.\textsuperscript{5} Continuous-drip feedings may provide maximal stimulation and hasten recovery time. Small feedings of isotonic liquids and solid foods should be initiated with improvement of the patient’s condition.\textsuperscript{5} Total parenteral nutrition may occasionally be necessary even after adaptation has occurred; therefore, the patient’s nutritional status should be monitored closely throughout life.\textsuperscript{5,70}

Total intestinal adaptation is considered to be achieved when patients are able to discontinue parenteral feeding. Even if TPN is used to supply the majority of calories and nutrients, enteral feeding should be initiated as soon as possible to expedite adaptation of the remaining bowel. Provided that the intestinal mucosa is relatively healthy, the lower limit for the length of small bowel necessary to be weaned from TPN ranges from 50 to 70 cm of small bowel if the colon is left intact, or 100 to 150 cm if the resection includes a colectomy.\textsuperscript{1,10,27,71–73}

Gouttebel et al. studied the parenteral nutrition requirements in SBS with varying lengths of small intestine and the length of therapy with TPN. As the patient’s oral intake was increased, the frequency of TPN was reduced to every other day for 1 week, three times a week the next, and twice a week during the third week. Total parenteral nutrition was resumed if one or more of the following occurred: (1) the patient lost equal to or more than 1 kg/wk; (2) diarrhea exceeded 600 g/d; or (3) laboratory abnormalities developed.\textsuperscript{14,52,66}

**Enteral Nutrition**

Early continuous feedings with high-viscosity enteral diets tend to reduce duration of TPN therapy, allowing earlier removal of central venous catheters that may be prone to infection.\textsuperscript{14,74} Originally, elemental diets were favored for patients with SBS. Some studies favor peptide-based formulas because they are more effectively absorbed and have lower osmolarity than polymeric formulas.\textsuperscript{12} When the patient’s fluid and electrolyte balance has stabilized, bowel sounds have returned, and there is less than 2 L/d of diarrhea, an elemental diet may be initiated.\textsuperscript{7}

Elemental formulations contain monoglycerides, free amino acids, dipeptides and tripeptides, and occasionally, medium-chain triglycerides. Elemental diets are very efficiently absorbed within the first 100 cm of jejunum. They may also facilitate adaptation by preserving intestinal mass in the proximal small bowel.\textsuperscript{12} Conversely, elemental diets tend to be unpalatable, are hyperosmolar, and may increase
diarrhea if administered at full strength. In most cases, elemental diets must be given through a nasogastric tube.

Polymeric diets are easily taken orally, are less hyperosmolar, and are much less expensive than elemental formulations. For these reasons, most clinicians tend to prescribe polymeric diets initially, providing elemental diets only to those patients who are unable to tolerate polymeric formulas. Some studies show that polymeric diets provide greater stimulation of the mucosa compared with elemental diets. Furthermore, low-residue, polymeric, chemically defined, or elemental diets offer the theoretical advantage of being easily absorbed in patients with SBS. However, some researchers argue that there is no significant difference in caloric absorption, stomal output, or electrolyte losses among elemental, polymeric, and normal diets in patients with SBS. When gastrointestinal fluid losses increase, it may be assumed that enteral feedings are no longer tolerated.

Combination of Enteral Nutrition and Trophic Factors

The addition of trophic factors (e.g., growth hormone) to enteral formulations may enhance intestinal compensation and intestinal adaptation. This can reduce or even eliminate the need for TPN, thus improving nutritional status.

Trophic Factors in Adaptation

Growth Hormone

Benefits of Exogenous Growth Hormone

Administration of exogenous growth hormone improves fluid and electrolyte absorption and nutrient transport in the human gut. It also improves nitrogen balance in stressed postoperative patients receiving parenteral support. It may stimulate structural adaptation and improve nutrient absorption. Exogenous administration of growth hormone also increases amino acid transport in the jejunum and ileum in humans by increasing the number of functional carriers in the brush-border.

Disadvantages of Growth Hormone

Common side effects of high-dose administration of growth hormone include fluid retention, arthralgias, and carpal tunnel syndrome. These are usually reversible a few days after cessation of growth hormone therapy. Weaning patients from TPN offers major cost–benefit advantages when the cost of TPN is compared with that of growth hormone. The cost of growth hormone, however, is approximately $7,000 for a 3-week treatment period for a 70-kg person. Clearly, the cost–benefits and indications for use must be studied further.

Glutamine

Glutamine is a nonessential amino acid that acts in humans to maintain intestinal permeability. Glutamine is the most abundant amino acid in the plasma and comprises nearly 20% of the total circulating amino acid pool. Nearly all tissue are capable of glutamine synthesis, but most is synthesized and stored in muscle, where its concentration is 30 times that of plasma. Glutamine is released from muscle stores after small bowel resection. It accelerates hyperplasia after resection and enhances intestinal sodium absorption. Glutamine preserves mucosal integrity, enhances various T-cell functions after colorectal resection, and improves nitrogen balance (this is especially important in catabolic patients). Glutamine also regulates protein synthesis, is the main substrate for renal production of ammonia, and is an essential precursor for nucleotide synthesis.

Although some studies have found minimal influence of glutamine on growth, serum proteins, and small-intestinal histology, others have found evidence to the contrary. For example, studies in a variety of animals show that the intestinal tract’s uptake of glutamine exceeds that of any other amino acid. In fact, because it serves as a major fuel source for both the enterocytes and the colonocytes, it seems that glutamine is necessary for the maintenance of intestinal structure, in both normal and stressed states. Glutamine may improve specific nutrient absorption as well as fluid and electrolyte absorption via stimulation of electrogenic sodium transport. By providing a metabolic source of fuel, glutamine may enhance the normal hypertrophy that occurs after small bowel resection.

Glutamine added to standard amino acid solutions may prevent villous atrophy because of parenteral nutrition and lack of enteral nutrition. In critically ill patients who are unable to tolerate enteral nutrition, parenterally administered glutamine may reduce the degree of gastrointestinal atrophy associated with TPN.

Recent studies have focused on the effects of enteral glutamine on bowel structure and function. In humans, approximately 50% of enterally administered glutamine is metabolized by the intestinal mucosa; the amount reaching the peripheral circulation depends directly on the concentration delivered. The enteral administration of glutamine has been shown to prevent bowel permeability, decrease bacterial translocation, and improve survival in experiments involving sepsis originating in the gut. Enteral glutamine has also been shown to support crypt cell proliferation, accelerate small bowel healing, and improve outcome after abdominal radiation. In contrast to intravenously administered glutamine, luminal glutamine enhances glucose absorption.

Combination of Growth Hormone and Glutamine

Byrne et al. combined these three modalities and found that a high dose of growth hormone (0.14 mg/kg/d), intravenous or oral glutamine, and a high carbohydrate/low fat diet increased nutrient and fluid absorption at the end of 3 to
4 weeks of treatment. Nearly half of their patients were able to discontinue parenteral nutrition at follow-up 1 year later.

In contrast, Scolapio et al. evaluated the effect of high-dose growth hormone and glutamine in eight patients who were treated continuously with a high carbohydrate diet in a controlled, randomized study and found no alterations in assimilation of macronutrients, stool volumes, or morphology of the small-bowel mucosa. Additionally, Szkudlarek et al. reported that combined high-dose growth hormone and glutamine administered for 4 weeks did not improve intestinal absorption 5 days after treatment was terminated in short-bowel patients taking their usual diet.

Combination of Glutamine and Insulin-like Growth Factor 1

Insulin-like growth factor 1, which is regulated by growth hormone, has been shown to increase the weight and length of the small and large intestine, and to enhance bowel hyperplasia and hypertrophy after extensive jejunoileal resection. In animal studies, the combined administration of glutamine and insulin-like growth factor 1 was investigated. Together, they enhanced mucosal structure and absorptive capacity in the rat small-bowel allograft. They also significantly enhanced protein deposition in the resected bowel to a greater extent than that obtained with glutamine or insulin-like growth factor 1 alone.

Epidermal Growth Factor

Other trophic factors have been shown to have a beneficial effect on the small bowel after enterectomy. Epidermal growth factor administered as a parenteral infusion to rabbits after 80% enterectomy significantly increased active transport of amino acids and glucose into brush border membrane vesicles of the remnant. Epidermal growth factor further improved the harmful effects of octreotide, which is commonly used in patients with SBS to control fluid losses.

Epidermal growth factor given parenterally has been shown to increase the rate of crypt production and maintain the weight of both the small intestine and colon in animals. Intraluminal epidermal growth factor, infused into the ileum via a catheter, has been shown to increase ornithine decarboxylase activity, which is considered to be a marker of cell division. In contrast, oral administration of epidermal growth factor in animals showed no effect; this may be because of its digestion within the acid environment of the stomach or a basolateral receptor site.

Other Hormones

Other hormones that may be involved in the adaptive process include enteroglucagon, prostaglandin E, insulin, testosterone, and neurotensin. Clinical roles of these hormones are not well known at present, although enteroglucagon was secreted by a tumor in a patient with gastrointestinal mucosal hypertrophy. Patients with SBS with ileal resections and a preserved colon had elevated fasting plasma concentrations of glucagon-like peptide (glucagon-like peptide 1 and glucagon-like peptide 2); these may contribute to the positive effects of a preserved colon in terms of intestinal motility and functional adaptation.

Intestinal hormones may stimulate the production of polyamines within the enterocyte; this is the most critical step for hyperplasia after resection. Polyamines are trophic compounds that stimulate enterocyte proliferation. The enzyme ornithine decarboxylase has been found to be the rate-limiting enzyme in polyamine synthesis.

Exogenous gastrin appears to have its effects limited to the gastric and duodenal mucosa. In patients with short bowel with and without a colon, high serum gastrin concentrations may cause gastric acid hypersecretion, whereas high cholecystokinin values may account for satiety. Low peptide YY values in patients with short bowel without a colon may be partly responsible for the high volume of jejunosotomy output. High peptide YY values in those with a retained colon may slow gastric emptying and so contribute to the “colonic brake.”

Other compounds, including prostaglandins, cholecystokinin, and secretin, were not found to adequately maintain the mucosa in the setting of TPN.

Intrinsic Causes of Adaptation

Although the presence of various growth factors, luminal secretions, and nutrients are important in achieving a maximal response, they may not be essential for adaptation to occur. Enterocyte mass in the intestinal remnant increases but then reaches a plateau as adaptation is completed. Because more extensive resection stimulates greater adaptation, it is possible that adaptation may be triggered by the loss of an inhibiting effect of the mucosa. Pancreatic and biliary secretions also facilitate adaptation locally within their site of administration and by being diverted to the distal small bowel where they facilitate distal intestinal hyperplasia.

ORAL NUTRITION

Frequent oral feedings in the form of six small solid meals per day should be offered to patients during the adaptation phase. With significant adaptation, it may be possible for some patients to be given an unrestricted diet. However, in patients with inadequate absorption of nutrients during the day, a nasogastric tube can be used at bedtime and infused with a liquid supplement of 1,000 kcal overnight, thus taking advantage of the intestine’s absorptive capacity. Because most patients with SBS absorb approximately two thirds of their usual energy intake, they must increase their dietary energy intake by approximately half to maintain weight. An economical way to increase caloric intake is to supplement the oral diet with short and long-acting carbohydrates.
medium-chain triglycerides (e.g., 30 mL of coconut oil), which can be absorbed directly by the proximal small bowel.\(^8\)

It is important to maintain an adequate intake of protein, vitamin B\(_{12}\), folate, calcium, magnesium, iron, sodium, vitamin C, and potassium.\(^7\) If colostomy has been performed, clear liquids should be administered initially. As the patient’s condition improves, a bland/low residue diet should be given to reduce stomal discharge and irritation. To facilitate recovery, the diet should also be high in protein and calories. Regular mealtimes and “constipating foods” like boiled milk, rice, or peanut butter can help to normalize evacuation times. It is important to then progress to a high-fiber diet.\(^7\)

A low-oxalate, low-fat diet along with high fluid intake is recommended for patients with SBS who experience hyperoxaluria to reduce susceptibility to forming oxalate stones. Foods such as chocolate, tea, cola, spinach, celery, carrots, beets, rhubarb, and peanuts are high oxalate foods and should be restricted.\(^7,24\)

**Phase III: Maintenance**

By phase III, the maintenance phase, the absorptive capacity of the gut is at a maximum. Although some patients still require parenteral nutrition, others do well on diet alone. Attempts should be made to compensate for continued malabsorption by increasing the quantity of small meals and supplementation with vitamins and minerals.\(^15\) At this point, the patient has either adapted maximally so that nutritional and metabolic homeostasis can be achieved entirely by oral feeding, or the patient is committed to receiving supplemental or complete nutritional support for life, either by ambulatory home TPN and/or specialized enteral or oral feedings.\(^8\)

**DIETARY SUPPLEMENTATION**

In general, nutritional therapy for SBS is based on the presence or absence of the colon. If the colon is present, the diet should be high in protein, fat, and complex carbohydrates. Patients who lack a colon should receive a diet low in complex carbohydrates.

**Carbohydrates**

As discussed herein, carbohydrates and proteins are absorbed in both the small and large intestine. Decreasing length of small bowel correlates with increasing malabsorption; this allows colonic bacteria within the large bowel to degrade carbohydrates and protein to SCFAs.\(^33,37,96\) A diet high in complex carbohydrates further enhances this process. Because these SCFAs are easily absorbed across the colonic mucosa, the result is salvage of carbohydrate energy that would otherwise be lost in feces.\(^21,33,37,38\) This can provide up to 540 kcal per metabolizing episode.\(^53\) In short-bowel patients with a functional colon, loss of fecal energy was reduced by 30% to 40% when diets were rich in carbohydrates than when they were low in carbohydrate and high fat.\(^96\) Disaccharides such as saccharose, maltose, or lactulose are stronger stimulators of adaptive responses than monosaccharides.\(^2,97\)

**Fiber**

Insoluble fibers or bulk fibers (e.g., wheat, bran) undergo minimal digestion in the gastrointestinal tract, mainly serving to increase stool bulk and colonic transport. Because insoluble fibers increase excretion of nitrogen, calcium, zinc, and iron as well as increase fecal fat, caution against overuse is necessary because they may harm rather than help patients with SBS.

Soluble fibers (pectin, guar gum) that are fermented in the colon to produce SCFAs may be of benefit because they delay gastric emptying by increasing intragastric viscosity.\(^24\) In SBS, pectin serves as a precursor for SCFAs; SCFAs increase O\(_2\) uptake in the colon, therefore maintaining gut integrity. Mucosal growth is stimulated by early refeeding (free fatty acids, sugars, and proteins).\(^7\)

Most fruits and vegetables provide are not used efficiently in patients with SBS because they provide few calories. In contrast, foods with small amounts of finely divided fiber (e.g., potatoes, bread, tomato juice) may serve as substrates for colonic epithelial cells. Small amounts of fiber are likely to absorb small amounts of water, and small quantities of some fibers may be salvaged in the colon with other carbohydrates. If the patient is able to maintain nutritional stability with oral foods, small amounts of fibrous foods may add to the variety of foods in the diet.\(^5\)

**Dietary Fat**

Dietary fat is largely absorbed in the small intestine. Fat restriction is not necessary in patients who lack a colon. For patients who do have a colon intact, study results regarding fat content of feedings have been mixed. In a crossover study comparing high-fat (60% of calories as fat) and low-fat (20% of calories as fat) diets, there were no differences in electrolyte loss and stool volumes between patients on the two diets. In addition, some clinicians argue that restricting fat from the diet may actually be harmful in that it results in decreased palatability of food with subsequent loss of appetite and caloric deficiency.\(^98,99\)

Patients with a colon who consume a high-fat diet may experience increased diarrhea as a result of increased secretion of water and sodium secondary to maldigestion of fatty acids. These patients may benefit from a diet high in complex carbohydrates (50–60% of calories) and lower in fat (30% of calories).\(^24,25\) In fact, a low-fat diet to minimize diarrhea and steatorrhea is recommended to prevent excessive colonic absorption.\(^53\) Both diarrhea and steatorrhea
may be decreased if dietary fat is reduced from 90–100 g to 20–40 g per day; body cell mass may be increased.100

The colon absorbs short-chain (C₂-C₆) fatty acids, but not the long-chain (C₁₄-C₁₈) fatty acids usually found in dietary fat. Hence, the amount of energy absorbed via colonic digestion is commensurate with the dietary carbohydrate–fat ratio.101 Fecal fat excretion may thus be further reduced in patients with SBS by replacing long-chain fatty acids in the diet with medium-chain fatty acids. In contrast to SCFAs, which are easily absorbed by the colon, long-chain fatty acids that are malabsorbed by the small intestine are not subsequently absorbed in the large intestine to any extent.13,86,87 The water-soluble medium-chain fatty acids are absorbed directly in the large intestine in a manner similar to that of SCFAs, even in patients with virtually no absorption of long-chain fat.14,37 In fact, medium-chain triglycerides in otherwise isocaloric diets may even increase the amount of total calories absorbed.38,70

Byrne et al. studied the efficacy of a progressive-step diet in intestinal adaptation. Administration of pectin was started at the beginning of the special oral diet (step 1), followed by medium-chain triglycerides (MCTs) and complex, nonfermentable sugars (step 2); coconut oil (47% MCTs) and simple sugars (step 3); and long-chain triglycerides and lactose (step 4).1,80,81 Total parenteral nutrition was interrupted at step 3 or 4 if any of three events occurs: (1) the energy content of the diet reached 150% of the patient’s resting energy expenditure; (2) serum albumin and weight were stable or increasing; and (3) the frequency, amount, and consistency of stools remained unchanged. Follow-up showed that patients responded well to this protocol; indeed, they were able to return to their previous professional activities. Thus, enteral formulas were not critical for gastrointestinal adaptation.1,66,80,81

Manipulation of the dietary fat–carbohydrate ratio tends to be less efficacious in patients with SBS without a functioning colon; likewise, the use of MCTs has not proven effective in overall energy absorption if the large bowel is not present.102,103 For example, dietary replacement of 50% long-chain triglycerides with MCTs in small bowel–resected patients increased fecal volume significantly but had no beneficial effect on the amount of calcium and magnesium absorbed.104

Presence of the colon alleviates progressive short-bowel failure by taking over the functions of water and sodium absorption as well as extracting energy from carbohydrates and MCTs.13,102,103 Recent research has shown that high-fat diets may be comparable to high-carbohydrate diets in terms of calories absorbed, blood chemicals, stool or ostomy output, urine output, and electrolyte excretions.98,99 However, enteral intake of fat should approach 50% to 100% greater than expected goals to compensate for malabsorbed nutrients.98,99

**Protein and Fluids**

Despite reduced protein absorption, most patients with SBS who maintain a stable weight with oral nutrition alone do not require protein supplementation because they have a positive nitrogen balance.50,98,99 If protein deficiency is suspected, patients may be given 80 to 100 g of dietary protein daily.98,99

Fluids should be ingested between meals rather than with meals to reduce the likelihood of dumping.5,7 Gastric emptying of liquids is fastest when the stomach is full.19 Although hyperosmolar diets should be given initially to reduce gastrointestinal fluid losses, hyperosmolar liquids such as fruit juices and carbonated beverages should be avoided.19,24 Beverages containing caffeine or alcohol should be restricted or consumed in very small quantities.7

Rehydration can be achieved by oral fluid restriction, intravenous fluids, and the World Health Organization rehydration solution.53 The World Health Organization rehydration solution contains 90 mmol/L of sodium, which is the same concentration found in jejuno-stomy fluids.19,46,47,59,105

If urine output is less than 1 L/d, patients are at risk of developing renal dysfunction and should be given intravenous fluids.21 Postoperative oral rehydration should consist of glucose–electrolyte sipping solutions containing at least 90 mmol/L of sodium to aid in the absorption of this electrolyte.59 Most commercially available rehydration solutions lack optimal amounts of salt, glucose, or both. To compensate for this discrepancy, 2.5 to 5.0 mL of salt should be added to the sodium concentration to reach the desired 90 to 120 mmol.24

Critically ill patients receiving prolonged TPN may develop iatrogenic volume overload because they are also receiving intravenous antibiotics and other medications in aqueous solution. Sources of fluid that must be monitored in these patients include (1) the oxidation of substrates, which yields approximately 50 mL of water daily in a typical TPN regimen; (2) muscle catabolism, which yields up to 500 mL water daily during severe catabolism; and (3) the TPN fluid itself.5,46,47,59 Weight gain and hyponatremia, especially in patients with compromised hepatic or cardiac function, may be caused by excess sodium from TPN, medications, or exogenous albumin.5,46,47,59

The small intestine receives and processes approximately 8 L of fluid per day, including dietary ingestion and endogenous secretions.8,14 Normally, the small bowel absorbs approximately 80% of this fluid load. Of the remaining amount, the colon usually absorbs approximately 1 to 2 L of fluid with a maximal absorptive capacity of approximately 6 L of fluid per day.8 Because the ileum and colon have a large capacity for absorbing excess fluid and electrolytes, proximal small-bowel resections rarely result in diarrhea. However, extensive or complete ileal or colonic resections may lead to malabsorption and diarrhea, along with dehydration, hypovolemia, and electrolyte derangements.8
VITAMINS AND MINERALS

Both fat-soluble and water-soluble vitamins should be accounted for in the SBS diet. Serum 25 (OH) vitamin D levels can be normalized by giving 50,000 IU of oral vitamin D every other day until normalization of the plasma levels occurs. Patients with osteoporosis should also receive 1,500 mg of elemental calcium per day along with one multivitamin.

Magnesium supplements are extremely important in preventing formation of calcium oxalate kidney stones in patients with steatorrhea. The decline in urinary magnesium occurs before that of serum magnesium and is a more reliable indicator of magnesium deficiency. Furthermore, parenterally administered magnesium tends to decrease the likelihood of formation of calcium oxalate stones. Thus, attempts at replacing magnesium in patients with gut failure should be aimed at normalizing urinary magnesium levels. Magnesium salts given enterally increase diarrhea. Soluble magnesium salts given as part of an oral rehydration solution may be better tolerated and absorbed than other magnesium complexes.

Vitamin A can be replaced by a water-soluble aqueous preparation of 5,000 IU/0.1 cc dropper, and vitamin E tablets can be replaced with a solution of 4,600 IU/5 cc dropper. Vitamin K can be replaced by a 5-mg oral supplement per day and titrated to the frequency required to maintain a normal prothrombin time.

Of the water-soluble vitamins, vitamin B12 is the one that may need to be replaced in those patients with greater than 60 cm of ileal resection. Normal stores may last for 2 to 4 years, but some patients may have had inadequate vitamin B12 use before surgery. Because the enterohepatic circulation accounts for 10 µg/d, total malabsorption would require 300 µg/mo for replacement. Therefore, a monthly injection of 1,000 µg of vitamin B12 is adequate replacement therapy. Other water-soluble vitamins usually can be maintained with a daily oral multivitamin.

Zinc may be lost in stool, nasogastric aspirates, and fistulous drainage. In these cases, intravenous replacement usually is necessary to restore the zinc balance. In less severe cases, a multivitamin with zinc will provide approximately 22 mg of elemental zinc (compared with the recommended daily allowance of 15 mg). Loss of zinc is high in patients with large-volume gastrointestinal output. These patients require large doses of oral zinc supplements because of limited zinc absorption. Serum zinc may also be low because of low serum albumin, the major zinc-binding protein; this may not necessarily reflect zinc deficiency.

Other supplements that may be needed include potassium, manganese, selenium, and glucose. It is rare for SBS patients who are maintained on an oral regimen to need a potassium supplement. If low serum potassium occurs, the possibility of chronic sodium depletion with secondary hyperaldosteronism or magnesium deficiency should be considered. The water-soluble vitamins and minerals (vitamin B-complex and C, calcium, iron, magnesium) are absorbed in the proximal small intestine, whereas magnesium diffuses passively throughout the entire small bowel.

PHARMACOLOGIC THERAPY

Several drugs may be useful in the treatment of SBS, depending on the amount and location of the loss of absorptive capacity. Exogenous supplementation of pancreatic enzymes may facilitate digestion of complex nutrients. For example, pancrelipase improves fat and protein absorption after jejunal resection.

Because small-bowel resections tend to increase the incidence of peptic ulcer disease, H2 receptor blockers and proton pump inhibitors can be used to regulate gastric acid production and reduce the extent of secretory diarrhea. Antacids or sucralfate can also be given by mouth or via a nasogastric tube. Clonidine may help to decrease gastric secretion as well as intestinal motility. Metronidazole can be used to treat bacterial overgrowth and jaundice in cases of prolonged TPN. Loperamide and codeine slow intestinal transit via their actions on opiate receptors. These drugs can be used both in the early and in the maintenance phase after small-bowel resection (4–16 mg of loperamide/d). Codeine, loperamide, and diphenoxylate hydrochloride can be used as antidiarrheal agents. Patients with minor ileal resections (<100 cm) and watery diarrhea induced by bile salts can be treated with cholestyramine, which will bind the bile salts.

Almost all patients with SBS tend to develop gallstones, for which cholecystectomy is indicated within 2 years after massive intestinal resection. Even after cholecystectomy, gallstones tend to form in the common bile duct and elsewhere in the biliary tree. For nephrolithiasis that continues after diet modification, additional therapy should include cholestyramine (4 g three times a day) to bind intraluminal oxalate, calcium supplements (600–1,000 mg/d), which increase calcium oxalate–insoluble complexes, citrate to correct low urinary citrate levels, and parenteral magnesium to normalize urinary magnesium.
Long-acting somatostatin and its analogs such as octreotide may increase the absorption of water, sodium, and energy as well as reduce diarrhea by prolonging intestinal transit time. Octreotide can also be useful as an antidiarrheal drug in patients with a high-output jejunostomy or ileostomy and secretory diarrhea; however, there is no significant effect in patients requiring permanent parenteral nutrition. Furthermore, octreotide is expensive. Because of the risk of tachyphylaxis with prolonged use, octreotide may only be useful during the adaptation phase. Octreotide may actually hinder adaptation by inhibiting intestinal regeneration. Also, octreotide may cause an increased incidence of headache, nausea, abdominal pain, and cholelithiasis.

As previously mentioned, Byrne et al. showed that use of bowel growth–enhancing agents along with an optimal diet can decrease dependence on TPN absorption in individuals with less than 50 cm of jejunum–ileum. They further showed that pharmacologic therapy is predictably able to allow patients with an intestinal length–body weight ratio of approximately 0.5 cm/kg or greater to be free of TPN. In patients dependent on TPN with a ratio of jejunal–ileal length-to-body weight more than 0.5 cm/kg, the need for TPN probably can be eliminated by treatment with growth hormone, glutamine, and a diet containing modified carbohydrate–fat ratios. The strong tendency for patients with SBS to develop metabolic acidosis may be offset by administration of sodium bicarbonate powder, tablets, liquid, or wafers in doses of 8 to 12 g/d for a minimum of 6 months, and as long as 18 to 24 months. Anemia and hypoalbuminemia may be treated with intermittent infusions of human serum albumin and packed erythrocytes.

CONCLUSION

Short bowel syndrome is a consequence of significant intestinal resection. It is marked by malnutrition caused by electrolyte and fluid imbalance, weight loss, and diarrhea. The prognosis is closely related to the length of remaining bowel; therefore, it is critical that surgeons measure the amount of intestine remaining in addition to the amount resected if at all possible. Total parenteral nutrition is usually begun during the acute phase of medical management of SBS. Recommendations regarding the need for parenteral nutrition vary depending on the presence or absence of certain factors: the ileocecal valve, jejunum, and functional colon. Patients with residual small-bowel length of 100 cm (normal, 600 cm) or less usually require parenteral nutrition for survival. Before the discovery of TPN in 1968 by Dudrick et al., patients with short bowel often died as a result of fluid and nutritional deficiency. During the past 3 decades, advances have made it possible to administer parenteral nutrition in the home environment with good results. The TPN diet should consist of a majority of calories from fat, followed by protein, and the remaining as carbohydrates. Vitamins, minerals, and trace elements should also be added accordingly.

Although TPN is initially necessary, treatment goals should focus on early transition to enteral nutrition followed by oral feeds. Other recent advances in the medical management of SBS include pharmacologic treatment and the use of specific nutrients and growth factors to stimulate intestinal absorption and adaptation. Both animal studies and clinical trials in humans have shown much promise in supplementation with growth factors and hormones. This strategy is likely to play a greater role in the treatment of SBS in the future.

REFERENCES


