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The Clinician's Guide to Short Bowel Syndrome



Carol Rees Parrish

Individuals with short bowel syndrome (SBS) are some of the most challenging patients for health care practitioners to manage. In addition to complex fluid, electrolyte and nutritional issues, clinicians must also treat ongoing medical problems and facilitate the administration of total parenteral nutrition (TPN). Appropriate attention to these issues can significantly improve the quality-of-life for a patient with SBS. This article is intended to provide the clinician with a logical, stepwise approach toward maximizing the potential of the remaining bowel of an adult patient with SBS in order to reduce or eliminate the use of TPN or intravenous (IV) therapy.

CASE STUDY

SM, a 35-year-old woman, presents for an initial visit to gastroenterology (GI) nutrition clinic with a diagnosis of short bowel syndrome. Her medical history is significant for stage III B ovarian cancer which required 4 bowel resections and a course of chemotherapy within the past four years. She had not

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received radiation therapy. Total parenteral nutrition (TPN) was initiated via a Hickman catheter 5 months earlier due to failure to thrive and multiple admissions for dehydration and electrolyte disturbances including one septic episode to date. She recently moved to the area to be closer to family with her active 5-year son and she divulged in clinic that her primary goal is to see him start kindergarten. She is 5'3", has been stable at 98 pounds (lbs), but remains 22 lbs below her usual weight of 120 lbs. Her only medication is Estrase. She is on nocturnal TPN that infuses over 14 hours. The TPN provides 1600 kilocalories, (80 grams of which is pro-

Table 1
The GI Balance Sheet—Normal Gut (37)

Gastrointestinal Water Movement

	<i>mL of water</i>
<i>Additions</i>	
Diet	2000
Saliva	1500
Stomach	2500
Pancreas/Bile	2000
Intestine	1000
<i>Subtractions</i>	
Colointestinal	8900
NET STOOL LOSS	100

tein) in a total volume of 1800 mL. SM admits that she occasionally skips her TPN infusion and that her oral intake, though not restricted, is sporadic. Now, where do we begin?

INTRODUCTION

Patients with SBS offer a unique challenge to clinicians. In addition to the fluid, electrolyte and nutritional issues, clinicians must help patients manage the very difficult sequelae that result from the physiologic changes occurring in SBS. There are many excellent reviews of SBS (1–2, Best Practice Series—see other references), but this article will focus on providing specific management principles to optimize the chronic care of the patient with SBS.

Diarrhea and steatorrhea, cardinal symptoms of SBS, occur when the fluid-substrate load exceeds the

absorptive capacity of the remaining, viable mucosa. This may result from: changes in motility; increased gastric secretions; osmotic stimulation from fatty acids, deconjugated bile salts and carbohydrates (CHO); bacterial overgrowth; lactose intolerance and/or fatty acid irritation of the colon. In addition to osmotic drag from hypertonic medications, food and fluids within the intestinal lumen also contribute to the diarrhea and malabsorption seen in these patients. See Table 1 for a review of intestinal water movement and Table 2 for electrolyte content of intestinal secretions.

DEFINITION

Many definitions exist for SBS. It can be broadly defined as an inadequate absorptive capacity due to decreased length and/or decreased functional bowel. Typically, a 70%–75% loss of small bowel will result in SBS. SBS has also been defined as a bowel length of 100–120 centimeters (cm) of small bowel (SB) without a colon, or 50 cm of SB with a colon. In truth, the real definition of SBS is inadequate functional bowel to support nutrient and fluid requirements for that individual, regardless of the length of the GI tract in the setting of normal fluid and nutrient intake.

ETIOLOGY

The etiology of SBS is multifactorial and unfortunately, the disorder can occur at any age. However the causes differ somewhat between children and adults (Table 3). Regardless of the origin, much of the clinical intervention remains the same.

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Table 2
Fluid and Electrolytes Your Patients May Be Losing (mEq/L) (11,38–40)

<i>Body Fluid</i>	<i>Na</i>	<i>K</i>	<i>HCO₃</i>	<i>H</i>	<i>Cl</i>	<i>pH</i>	<i>Volume/24 hours (L)</i>
Sweat	30–50	5	—	—	45–55		.5
Saliva	45	20	60	—	44	7	.5–1.5
Gastric	40–65	10	—	90	100–140	2	2–4
Pancreas	135–155	5	70–90	—	55–75	8	1
Bile	135–155	5	35–50	—	80–110	7	1.5
Jejunum/Ileostomy	100–120	10	50–70	—	50–60	7	1.8 liters
Diarrhea	25–50	35–60	30–45	—	20–40		
Normal stool	5	10	—	—	10		0.1

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Table 3
Etiology of Short Bowel Syndrome in Children and Adults (2,31)

Children

- Necrotizing Enterocolitis (NEC)
- Intestinal atresia
 - Volvulus
 - Hernia
 - Intussusception
- Congenital short bowel syndrome
- Trauma
- Gastroschisis
- Apple peel anomaly
- Crohn's disease
- Abdominal tumors
- Radiation enteritis
- Hirschsprung's disease

Adults

- Massive surgical resection
- Crohn's
- Malignancy
- Radiation enteritis
- Trauma
- Vascular catastrophies
 - Embolism/thrombus
- Volvulus
- Stagnulated hernias
- SB fistulas
- Surgical bypass
- Surgical error or obesity treatment
- Chronic intestinal pseudo-obstruction

ADAPTION

Adaptation is the time it takes for the gut to adjust to the damage that's been done. The bowel compensates for a reduction in surface area by increasing the length and diameter of the remaining bowel, by villous hypertrophy (increase in the number and size of crypts and villi, cell proliferation and enhanced enzyme activity), and by altering motility and hormonal responses. The ultimate effect is to maximally increase the remaining luminal absorptive capacity.

Factors Affecting Adaptation/Success of Remaining Free of TPN or IV Fluids

Many factors affect intestinal adaptation and successful transition from TPN to enteral nutrition in the SBS patient, one of the most important is the length of the

remaining bowel. It cannot be over emphasized that clinicians must make every effort to determine the extent of available bowel (e.g., by reviewing operative or radiation oncology reports or discussing the length of resection with the surgeon). The location of small bowel resection and overall health of the remaining small bowel will also affect a SBS patient's adaptation potential as well as the presence of an ileocecal (IC) valve or any portion of the colon. Another consideration is the length of time that has elapsed since the initial surgery or insult. Lastly, the age of the patient and any other GI organ involvement (e.g., pancreas, liver, stomach) may play a role in future adaptation.

Adaptation can be enhanced by stimulating the residual bowel via macronutrient (protein, fat, carbohydrate) exposure. Furthermore, nutrient complexity also increases the functional workload of the digestive mechanisms involved. As an example, monosaccharides require no digestion, thus they result in little hyperplasia as compared to polysaccharides (Wilmore-Best Practice Series). Providing more complex macronutrients to the remaining bowel is therefore an extremely important factor in the successful transition to enteral nutrition.

Adaptation may take up to 1–2 years. Factors affecting the adaptation process include:

- Stimulation by intraluminal nutrients
- Stimulation by bile and pancreatic secretions
- Trophic effects of gut hormones
- Altered intestinal blood flow
- Altered enervation

It is important to recruit as many of these factors as possible to achieve maximal adaptive potential of the mucosal surface area by way of hyperplasia, deeper crypts and increasing the absorptive capacity per cm of the remaining intestinal surface area.

GOALS OF MANAGEMENT

The primary goal in managing SBS is to maximize the utilization of the existing gut while assuring that patients are provided with adequate nutrients, water and electrolytes to maintain health and/or growth. Clinicians must focus on reducing the severity of intestinal failure while treating and preventing complications when they arise. To improve the chance for successful

weaning from TPN or IV fluids, it is essential to increase nutrient and fluid retention by slowing intestinal transit time, controlling gastric acid hypersecretion and by enhanced mixing of pancreatic enzymes and bile salts. Additionally, it is imperative to feed in the most proximal bowel location, to avoid osmotic agents, and to treat bacterial overgrowth when necessary.

REVIEW OF THE ACTIVE PLAYER— THE GI TRACT

The following section will review the primary structures and functions of the small and large intestine.

Small Bowel

Duodenum

A powerhouse for absorption, the duodenum, measuring about 25–30 cm (~10 inches) is rarely involved or resected. It is the preferred site of absorption for iron and folate. However, it is a key player as pancreatic enzymes and bile salts enter here to mix with food.

Jejunum

The jejunum ranges in length from 200–300 cm (6–10 feet) and is responsible for much nutrient absorption, in fact, >90% of nutrient absorption occurs in the first 100–150 cm of the SB. Jejunal enterohormones including cholecystokinin (CCK), secretin, gastric inhibitory peptide and vasoactive inhibitory peptide each play a specific role in absorption. It is essential to remember the important role of each of these enterohormones when managing the SBS patient. CCK stimulates pancreatic secretion and gall bladder contraction while secretin is responsible for bicarbonate secretion. Gastric inhibitory peptide inhibits gastric secretion and gastric motility, and finally, vasoactive inhibitory peptide inhibits gastrin and bicarbonate secretion. Another important role of the jejunum is drug absorption; many, but not all drugs, are absorbed in this section of small bowel (4).

Ileum

The ileum, about 300–400 cm (10–13 feet) long, has a functional length almost double that of the jejunum. In addition, the ileum's intrinsic motility is much slower than that of the jejunum (2). A phenomenon known as

the ileal break—a slowing of intestinal transit in the stomach and upper gut in response to undigested carbohydrate (CHO) and fatty acids entering the ileum—provides an opportunity for more nutrient contact time with the mucosa and therefore greater nutrient absorption (5).

If greater than 100 cm of terminal ileum is lost, the bile salt pool cannot be maintained due to the disruption of the enterohepatic circulation; hepatic synthesis will not keep pace with intestinal losses. This reduction in bile salts may result in steatorrhea and fat-soluble vitamin loss. Absorption of vitamin B₁₂ can be impaired if >60 cm of terminal ileum is resected. Glucagon-like peptide 1 and 2, peptide YY and neurotensin are important neurohormonal mediators released by the terminal ileum that impart trophic effects on the mucosa. If the ileocecal region is removed, then this mechanism is lost. GLP-2 is receiving closer scrutiny as a therapeutic agent in the treatment of SBS (6). It is worth noting that jejunal resection is better tolerated than ileal resection due to the unique characteristics of the ileum and its adaptation potential.

Ileocecal Valve

The Ileocecal (IC) valve, at the junction of the ileum and cecum, controls the amount, and slows the passage of ileal contents into the colon, thereby increasing nutrient/lumen contact time in the proximal GI tract. The IC region possesses specific absorptive functions and plays a crucial role in the regulation and integration of postprandial gastrointestinal motility and secretion. Finally, the IC valve also prevents reflux of colonic bacteria into the small bowel, decreasing the risk for bacterial overgrowth.

Role of the Colon—Colon Matters!

The colon measures about 160 cm (5 feet) in length and on average recovers approximately 1–1.5 liters of electrolyte-rich fluid daily. The colon is highly adaptable and can increase its absorptive capacity 3 to 5 fold, with a maximal absorption of approximately 5–6 L per day. Notably, sodium, chloride and water are avidly absorbed here.

In a normal adult, the gastro-colic reflex occurs within 10 minutes of eating, however (chyme, food) remains in the right colon for 8 hours, 6–8 hours in the

Table 4
Clinical Sequelae of Gastric Hypersecretion (11)

- Alters degradation of "R" protein and hence release of B₁₂ for binding to intrinsic factor
- Impairs pancreatic enzyme activity
- Destroys pancreatic lipase
- Precipitates bile salts and disrupts micelle formation
- Acid damage of duodenal mucosa
- Stimulates peristalsis

transverse colon and about 4 hours in the descending colon. Stool, 70% water by weight, enters the colon at approximately 50 mL/hr.

Preservation of at least half of the colon is equivalent to retaining 50 cm of functional SB. SBS patients with remaining colon will have a qualitative and quantitative alteration in colonic flora resulting in an increased capacity to metabolize CHO. CHO and fiber fermentation result in the production of short chain fatty acids (SCFA) that are absorbed in the colon, providing up to 525–1170 kilocalories per day (7,8). Recently, Epperson demonstrated that medium chain triglycerides (MCT) share the ability of SCFA to be absorbed by the colon in patients with mean residual SB length of 143 cm (9).

WHAT THE CLINICIAN IS UP AGAINST— PHYSIOLOGIC CHANGES

Gastric Hypersecretion

After significant resection of the small bowel, gastric hypersecretion (particularly problematic when jejunal surface area is lost) must be addressed. Lasting up to 6 months or longer post-operatively, it not only increases the sheer volume of secretions entering the small bowel, but also drops the pH of the upper gut. Hypersecretion results from loss of CCK and secretin secretion/feedback, which regulates gastrin secretion. Without this control, gastrin levels remain high, signaling acid production to continue. Not only is the increased acid load caustic to the proximal small bowel, the added volume of secretion contributes to the total stool output. Finally, the higher concentration of acid being dumped into the upper gut denatures pancreatic enzymes and

Table 5
Acid Reducing and Other Anti-secretory Agents

Proton Pump Inhibitors (PPI)*

- Need >50 cm of jejunum to absorb
- Liquid form may be better utilized
- Suggest trial of Intravenous pantoprazole (if absorption is in question)
 - Nexium (esomeprazole)—40 mg bid
 - Prilosec (omeprazole)—40 mg bid
 - Prevasid (lansoprazole)—30 bid
 - Protonix (pantoprazole)—40 bid

Histamine₂-Receptor Antagonists*

- Cimetidine (Tagamet)—400 mg oral or IV qid
- Famotidine (Pepcid)—40 mg bid
- Ranitidine (Zantac)—300 mg bid

Instructions/Preparations of PPIs

TAP Pharmaceuticals, (800) 622-2011; www.prevacid.com (Lansoprazole)

- Solu-tab
 - Dissolves under the tongue if oral intake acceptable
 - For tube delivery: mix 30 mg tab with 10 mL water and flush down feeding tube
- Oral suspension—reconstitute with water (do not put down feeding tubes—will clog!)

AstraZeneca, (800) 236-9933; www.astrazeneca.com (Esomeprazole, Omeprazole)

To make liquid with capsules:

- Empty Omeprazole capsule into glass containing 20 mL water
- Add 1/2 teaspoon baking soda
- Let stand for 15 minutes
- Mixture can be made in quart quantities and lasts for weeks to months if kept refrigerated and out of the sunlight

Omeprazole (Zegerid) powder

- Empty the contents of the packet into a small cup containing 2 tablespoons of water.

Other Anti-Secretory Agents

Octreotide (a somatostatin analog)

- 50–250 mcg to TID-QID (subcutaneously)
- May be needed if insufficient jejunum to absorb a PPI (<50 cm)

Clonidine

- 0.1–0.3 mg up to TID

***Note:** If oral, take on an empty stomach at least 1 hour before food.

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Table 6
LAR Depo-Octreotide—Considerations for Clinicians

Novartis has a very good program for anyone who does not have prescription coverage, including Medicare patients. Call 1-877-LAR-HELP. Novartis will do financial screening and provide reduced pricing or free drug for patients who qualify.

The LAR must be given in a facility where staff is trained to mix and administer as it is very tricky to give. It is a suspension, which hardens immediately in the needle if you don't mix correctly.

One Physician's Practice at UVAHS:

- Start patients out on 50 mcg TID for 2 days (every 8 hours)
- Increase to 100 mcg TID for 2 days
- Followed by 150 mcg TID for 2 days
- Finally, bring them in for the first LAR injection, usually start at 20 mg monthly.
- Continue the short acting for another week until a level is achieved and then stop. Observe for about an hour after the first short acting injection as patient may have profound swings in blood pressure or other side effects such as headache or nausea. If your patient is diabetic, pay attention to glucose levels—may have hypo- or hyperglycemia. Patients can self-inject the remaining short acting at home and then return for the LAR.

compromises bile salt function. Pancreatic enzymes and bile salts function optimally at a pH of 7–8. See Table 4 for clinical effects of gastric hypersecretion.

The treatment for gastric hypersecretion is acid suppression. Both histamine₂-receptor antagonists (H₂-blockers) and proton pump inhibitors (PPI) have been used, however, PPIs suppress significantly more acid than H₂-blockers (10). PPIs come in many forms—capsules, encoated tablets, liquid and SoluTabs. It may be necessary to provide the medication in the liquid or SoluTab form to increase absorption and efficacy. In order to “measure” the success of this intervention, monitor for a decrease in stool output or check the pH of *fresh* ostomy effluent (only in patients with a jejunostomy or ileostomy). If the pH is <6 in the setting of >1 liter of effluent, the dosage of the PPI should be increased and/or given more frequently. Remember that some (or a lot) of an orally delivered PPI may not be absorbed, especially if sufficient jejunum is not available (50 cm) (11). Finally, if the clinician is still unsure of efficacy, a trial of intravenous PPI for 2–3 days may be indicated (while stopping the oral med-

ication); if successful, as evidenced by a drop in stool/ostomy output, this may suggest that enteral administration, given the current conditions, is not working. Continued monitoring of 24-hour stool and urine output is prudent when attempting to transition patients off acid suppression. If output increases significantly when acid suppressive therapy is reduced or eliminated, (with no other changes in treatment plan), then PPI therapy should be reinstated.

In patients whose output is not osmotic in nature, but driven by secretory processes (“net secretors”—typically patients who lose more water and sodium from their stoma than they take in orally/enterally), octreotide may be useful. It has been suggested that the change from a net secretory state to a net absorptive state occurs with a jejunal length of greater than 100 cm. However, although the remaining jejunum may be longer in some patients, if the remaining bowel is damaged, then the patient may still be a “net secretor.” McDoniel reported the successful use of clonidine in two cases of SBS in reducing intestinal fluid and electrolyte losses (12). Suggested dosing of anti-secretory medications can be found in Table 5. Table 6 provides consideration for clinicians on depo-octreotide.

Motility

The body maintains several mechanisms to slow the transit of nutrients through the small bowel to obtain maximum nutrient salvage. Delivery of food from the stomach into the upper gut is regulated by pathways within the ileum and colon, segments often lost in SBS. These mechanisms include maximal mixing of pancreatic enzymes and bile salts with foodstuffs along with feedback produced by intestinal hormones such as glucagon-like peptide 1 and 2, neurotensin and peptide YY (PYY) (Keller-Best Practice). These hormones delay gastric emptying and slow intestinal transit by way of the ileal brake. These processes are only available if the ileocecal region remains intact. Exposure of undigested nutrients, primarily CHO and fat in the ileum, induces the ileal brake, thereby inhibiting digestive secretory and motor functions, and consequently slowing gastric motility. Loss of this segment of ileum may contribute to gastric hypersecretion and accelerated small bowel transit. The ileocecal valve also acts

to decrease transit time by regulating the rate that ileal contents are dumped into the colon. Finally, the rate of liquid gastric emptying may be slowed in those patients with an intact colon segment. This is most likely due to elevated circulating levels of PYY as a result of unabsorbed nutrients reaching the colon (1).

Anti-motility agents are the primary treatment to slow intestinal transit (Table 7). In the clinical setting, patients should receive a trial of less potent (e.g., loperamide [Imodium]) gut slowing medications with a plan to titrate up or change to more potent, prescription agents (e.g., diphenoxylate (Lomotil), opiates such as codeine or tincture of opium, etc.) if needed. Imodium has no effect on the central nervous system unlike Lomotil, nor is it sedating or addictive like the opiates, hence most clinicians prefer Imodium. Clinicians are often too tentative with the stronger anti-motility agents because of the high doses required, resulting in cumbersome stool output and persistent need for IVF. In "refractory secretors," a trial of both Imodium and codeine phosphate together may have a greater effect than either agent alone (11).

Because endogenous intestinal output rises after meals, it is imperative that these medications be given one half to one hour before mealtimes and at bedtime to ensure they do not compete with food or fluid for mucosal contact; if receiving enteral feeding, do not hold feedings, but scheduled dosing may be even more important. Furthermore, if a patient is willing to take a scheduled dose of anti-motility medication every 6 hours (because the patient is up at night anyway), this may improve overall efficacy.

Small Bowel Bacterial Overgrowth

Following bowel resection, patients are often at higher risk for small bowel bacterial overgrowth (SBBO). Factors that increase this risk include loss of the ileocecal valve, the presence of blind loops (ex. Bilroth II anastomosis), slowed motility, acid suppression, and underlying disease processes such as chronic pseudoobstruction (13). Symptoms can range from gas, bloating and nausea, to frank diarrhea. Although not a specific indicator, elevated serum folate levels may signal SBBO. B₁₂ deficiency can also accompany SBBO due to decomposition of the B₁₂-intrinsic factor complex.

Table 7
Antidiarrheal Medications Commonly Used in Short Bowel Syndrome (11,41)

General Guidelines

- Give 30–60 minutes before meals or snacks, but not more than every 6 hours.
- If patient gets up in the middle of the night and does not mind taking a medication, then dose every 6 hours and take advantage of a time when foods/fluids are not competing for absorptive surface area.
- Use elixir forms—no sustained release!
- Titrate up to maximal dose . . . and then some if necessary; try increasing doses every 2–4 days
- Imodium circulates through the enterohepatic circulation, hence higher doses may be needed in patients with >100 cm ileum lost.
- Increase dose until the stool consistency is adequate for patient or the patient is too sedated/unable to perform activities of daily living—whichever comes first.

Antidiarrheals

- Imodium (loperamide)
 - Initial, 2–6 mg up to QID, then up to 12–24 mg at a time in patients with disrupted entero-hepatic circulation (see above).
- Lomotil (diphenoxylate)
 - 2.5–5 mg up to QID
- Codeine
 - 15–100 mg up to QID
- Morphine
 - 2–20 mg up to QID
- Tincture of Opium*
 - 0.3–1.0 mL up to QID
- Paragoric*
 - 5–10 mL BID-QID

For Oral/enteral: It is important to remember that codeine, morphine, and methadone equivalents are not exact and different references have different approximate equivalents. 1 mL opium tincture = 25 mL Paregoric = 65 mg codeine = 10 mg morphine = 5 mg methadone = 5 mg oxycodone

Note:

- **Avoid the use of "drops" to avoid dosing errors;** both paragoric and tincture of opium are 20 drops/mL, however the syringe sizes differ*

*In general 1 mL = 20 drops for most medications, however, due to inaccuracies of droppers, this type of dosing is not recommended, especially in light of easy access to graduated syringes.

Table 8
Antibiotics for Small Bowel Bacterial Overgrowth

Guidelines and Schedules

- Enteral delivery preferred
- 7–10 day course unless specified
- Patients with intestinal failure from chronic dysmotility may need rotated dosing (e.g., scleroderma, amyloidosis, diabetic enteropathy, etc.):
- Example:
 - 1 week on, 3 off
 - Change to a different antibiotic every 6–8 weeks
 - 1–2 weeks on, 6–8 weeks off
 - 3 weeks on, 1 week off

Antibiotics typically used in the treatment of SBBO

- Augmentin—500 mg bid
- Chlortetracycline—333 mg tid
- Ciprofloxacin—500 mg bid
- Doxycycline—100 mg bid
- Metronidazole—250 mg tid
- Neomycin—500 bid for 10 days
- Rifaximin—400 mg tid for 7 days
- Tetracycline—250–500 mg qid

SBBO produces inflammatory changes to the intestinal mucosa and consequently increases gut permeability and a net loss of fluid into the lumen. In addition, SBBO can deconjugate bile salts in the upper gut, causing impaired micelle formation, which ultimately results in fat malabsorption and fat-soluble vitamin deficiency.

Although hydrogen breath tests are available to aid in the diagnosis, most clinicians empirically treat for SBBO due to the many controversies surrounding these tests (e.g., which substrate to use, interpretation in the setting of rapid transit, cost of test [and lack of insurance carriers to cover this cost] and time involved for patient). Treatment of SBBO consists of a 7–10 day course of enteral antibiotics. See Table 8 for commonly used antibiotics and suggested dosing.

Pancreatic Enzymes

Pancreatic enzyme supplementation is rarely necessary in SBS patients. However, when surgical anatomy creates a mismatch of pancreatic secretion and deliv-

Table 9
Bile Acid Binders and Replacers

Binders

- Cholestyramine 2-4 g up to QID

Note:

Intact colon and <100 cm ileum remaining
May worsen steatorrhea if >100 cm ileum due to binding of dietary lipid

Replacers

- Cholylsarcosine (a synthetic bile acid—2–4 g per meal)

ery of food into the small bowel, such as in the unfortunate patient who also has a Bilioth II or Roux-en-Y anastomosis, pancreatic enzyme supplementation may be necessary. In addition, patients with pancreatic insufficiency from chronic pancreatitis or cystic fibrosis may also require supplementation. If treatment is empiric, this intervention should be initiated AFTER acid reduction and gut slowing have been maximized to optimize the pH for enzyme function and for enzymes to have enough time to adequately mix with food. In the event the patient should require Viokase powder to mix with enteral formula, make sure the patient's pharmacy carries it or is able to obtain it.

Bile Salts and Cholerrheic Diarrhea

Bile salts, required for fat and fat-soluble vitamin absorption, are recycled daily from the ileum via the enterohepatic circulation. One hundred centimeters of ileum are needed for this pathway to remain viable to complete bile salt absorption. Steatorrhea can occur if <100 cm of ileum remains as the bile salt pool cannot be maintained since the bile salt loss in the stool exceeds its rate of synthesis within the liver. Loss of the bile salt pool leads to impaired micelle formation thereby decreasing solubilization of fat in the lumen. Steatorrhea further aggravates the loss of fat-soluble vitamins. Some medications also circulate through this pathway (ex. cyclosporin), hence higher doses may be needed to achieve efficacy (1,4).

In patients with less than 100 cm of remaining ileum, in the presence of an intact colonic segment, watery diarrhea (cholerrheic diarrhea) can occur due to
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the pro-secretory effect of bile salts on the colonic mucosa. Colonic bacteria deconjugate the bile salts resulting in mucosal inflammation, colonic secretion, reduction in sodium and water absorption, increased oxalate permeability, and decreased stool pH.

Treatment includes cholestyramine to bind bile salts (Table 9), calcium supplementation to bind oxalate (11), and possibly treatment of SBBO, which may also cause deconjugation of bile salts.

SPECIAL CONCERNS

Osteoporosis

The eventual development of bone disease plagues most patients with SBS, in fact, up to 30% of patients will have osteomalacia. Malabsorption of vitamin D and calcium are contributing factors. Persistent inflammation in those whose underlying disease is not yet quiescent (e.g., Crohn's disease) may increase osteoclastic activity adding to the problem. Chronic metabolic acidosis from the loss of bicarbonate in the stool, or in those with renal insufficiency due to repeated episodes of dehydration, ultimately diminishes the buffering capacity of the kidneys and can thereby reduce bone mineral content. Hypomagnesemia can also play a role in the development of osteoporosis. Magnesium is needed both for secretion of parathyroid hormone (PTH) as well as proper action of PTH on target organs such as the osteoblast and renal cell. A long history of steroid use may also increase the risk of bone disease via reduced osteoblast activity, increased urine calcium loss and further reductions in calcium absorption from the gut.

A baseline dual energy x-ray absorptiometry (DXA) scan should be obtained on all patients with SBS and followed every one to two years along with periodic assessment of 25-OH vitamin D levels (not 1, 25-OH vitamin D). Target levels for 25-OH vitamin D remain under debate, but should be in excess of 25-30 ng/mL. Monitoring vitamin D levels is essential even in patients routinely receiving vitamin D in TPN. Checking serum PTH will help with early identification of those patients who need more intensive intervention.

Treatment should be aimed at the provision of adequate vitamin D (with resultant decline in alkaline phos-

phatase and PTH levels), eliminating metabolic acidosis if present and replacement of magnesium if necessary (see section below on hypomagnesemia). It is worth noting that patients with malabsorption may need up to 1–2 micrograms of oral Rocaltrol® to have an effect (author's experience). Finally, there is a tendency towards metabolic acidosis in patients with SBS; bicarbonate in the form of acetate may need to be added to TPN/IV solutions or given oral/enterally to correct it. Administer sodium bicarbonate powder, liquid, tablets or wafers in doses of 8–12 g/day orally (14). Use caution with the commonly used Shohl's solution, a liquid form of sodium bicarbonate as it often contains sorbitol!!

Nephrolithiasis

Calcium oxalate stones can be an unpleasant complication in the SBS patient with an intact colon. Calcium oxalate stones have been reported in as many as 60% of patients (15, Keller-Best Practice). Patients without a colon are not at increased risk for this complication. The mechanism of stone formation is multifactorial. In the normal gut, calcium binds to oxalate forming an unabsorbable complex of calcium oxalate, which is then excreted in the stool. In the setting of steatorrhea, due in part to defective micellar solubilization of fatty acids, increased intraluminal free fatty acids are available to preferentially bind to calcium, resulting in an increased concentration of free dietary oxalate. This highly soluble oxalate is readily absorbed across the colonic mucosa where it ultimately is excreted via the kidney. Additionally, there is an increase in colonic permeability to oxalate stemming from the caustic effects of unabsorbed bile salts.

The risk of nephrolithiasis is compounded by volume depletion, metabolic acidosis and hypomagnesemia, resulting in a decrease in renal perfusion, urine output, pH and citrate excretion. Nephrolithiasis may lead to progressive symptomatic renal impairment if not identified and treated appropriately. The most important intervention in these patients is to ensure a urine output of >1200 mL/day or more, in those patients proven to be stone formers (14,16,17, Wilmore-Best Practice).

To avoid nephrolithiasis, the patient should be advised to avoid excess fat and high oxalate-contain-

Table 10
Foods and Beverages High in Oxalates (11,46)

Fruits: Raw apricots, blackberries, cherries, currants, figs, raw gooseberries, concord grapes, raw orange, raw pear, plums, rhubarb, strawberries, tangerines, prunes, lemons, limes, orange peels

Vegetables: Artichoke, baked beans, green and wax beans, beets, beet greens, raw red cabbage, celery, Swiss chard, escarole, chives, collards, eggplant, endive, leeks, mustard greens, Dandelion greens, okra, green peppers, rutabagas, spinach, kale, summer squash, sweet potatoes, parsley, tomatoes, tomato soup or juice, and vegetable soup, white corn, legumes

Nuts: Almonds, cashews, peanuts, peanut butter, pecans, and nut butters

Beverages: Chocolate/chocolate containing beverages, cocoa, colas, Ovaltine, tea, instant coffee

Starches: Grits, wheat germ, whole wheat bread, french fries, bran cereal

Other: Grits, tofu, soy products, black olives, chocolate, pepper (>1 tsp per day), vegetable soup with above vegetables

Alcohol: Draft beer

ing foods (Table 10). A lower fat diet (but not low fat, i.e., <60–80 grams per day), will decrease saponification of calcium and fatty acids in the intestinal lumen, leaving more calcium to bind to oxalate. In concert, provide more enteral calcium so more is available for binding with oxalate, decreasing oxalate availability for absorption (14, Keller-Best Practice). Oral calcium supplements of 800–1200 mg/day, in divided doses, not exceeding more than 500 mg, is typically used. Another strategy includes the use of cholestyramine, 4 grams three times daily, to bind oxalate found in the gut lumen (11).

***d*-lactic acidosis**

d-lactic acidosis is a rare complication seen in patients with SBS with an intact colonic segment; loss of the ileocecal valve is another predisposing factor (11,14). Its cause can be traced to malabsorption of CHO, especially of refined sugars. Colonic bacteria ferment CHO and refined sugars to produce SCFA and lactate. These fermentation by-products lower the colonic pH. Over time, growth of some of the normal flora is inhibited in this more acidic environment, promoting acid-resistant

anaerobes such as lactobacillus (e.g., Bifidobacterium, *L. acidophilus*, *L. casei* and eubacterium). These organisms have the capacity to produce *d*-lactic acid, an acid not metabolized by humans due to the lack of *d*-lactate dehydrogenase. The colon then absorbs the *d*-lactate resulting in a metabolic acidosis. Symptoms of *d*-lactic acidosis include mental status changes, ataxia, blurred vision, ophthalmoplegia and nystagmus. Patients present with a large anion gap, severe metabolic acidosis and may have symptoms that mimic Wernicke's encephalopathy.

Patients at high risk for *d*-lactic acidosis should be monitored for elevated serum and urinary *d*-lactate (when measuring serum lactate; L-lactate is what is measured and this will be reported as normal)—confirmed by elevation >3 mmol/L; (normal is <0.5 mmol/L), hyperchloremia and an elevated anion gap. Treatment includes antibiotics (metronidazole, neomycin, vancomycin for 10–14 days) thiamine supplementation (*d*-lactic acidosis may be worse in the setting of thiamine deficiency), and avoidance of refined CHO.

BACK TO OUR PATIENT—FIRST STEP—DATA GATHERING

The key to caring for patients with SBS is to address one thing at a time. This avoids over-treatment and keeps the nutrition regimen as simple as possible. A stepwise approach from the initial assessment is presented below.

1. Start a patient demographic sheet.

Key information should be compiled onto one form and remain permanently in front of the patient's chart for quick reference. Consider outlining essential information about the patient such as intact gallbladder, length of remaining bowel, etc., in a table or chart format (Table 11).

2. Defining Anatomy—Do you know your patients anatomy—REALLY?

Given the complexity of these patients and the often long-standing relationship that may ensue, it is essential to determine not only the length of bowel resected but also the length of bowel remaining. Often patients present to clinic or to the hospital with only a vague

Table 11
Demographic Checklist For Short Gut Patients—
Quick Reference

<i>Parameter</i>	<i>Results</i>
Etiology of SBS	
Date of surgery/ segment resected	
Duodenum intact	
Jejunum remaining	
Ileum remaining	
Colon remaining	
Ileocecal valve	
Cholecystectomy	
Venting g-port	
Fistulas/JP drains	
History of kidney stones	
Status of key organs	
• Stomach	
• Pancreas	
• Liver	
• Kidneys	
SBFT	
• Date	
IV/TPN	
• Central line	
– Type	
Enteral Access	
• Type	
Other drains	
• Type	

recollection of their anatomy. Discussions with their surgeon may be useful to determine what segments of bowel remain and which have been resected. Operative reports and a small bowel follow through (SBFT) should be reviewed, but oftentimes their results may not always reflect the patient's true anatomy. A **directed** or **timed** SBFT may be more useful as a timed SBFT allows the radiologist to watch the contrast going through the small bowel rather than shooting a plain film every 5–15 minutes—it gives a better idea of gross anatomy and timing of transit.

3. Past Medical and Surgical History

Identify any other underlying processes that may play a role in your ability to manage your patient. Specifically note the presence or absence of a gall bladder, history of kidney stones, progressive renal failure, diabetes mellitus or underlying Crohn's disease. Repeat renal stones or progressive renal dysfunction is a red flag that hydration needs attention (see the section on I and O below). Too many patients with SBS unnecessarily "live on the edge," of adequate hydration.

4. I and O.

- All patients with SBS should have a baseline 24-hour urine and stool output collection. This can be done in the hospital or at home. Monitoring urine and/or stool output should continue 1–2 times per week while interventions take place until the patient is stable. See Table 12 for normal ostomy output depending on the location in the bowel. This will give the clinician and patient objective information as to whether the interventions are, or are not, making a difference. For patients who are functioning reasonably well, urine and stool collection can be done on their CURRENT regimen and then changes can be made accordingly.
- For patients who are failing to thrive—start over! Stop all non-essential enteral medications, nutrients and fluids—this will remove any osmotic contribution. Next, obtain a 24-hour stool output while the patient is NPO. This will help differentiate patients who are "net secretors" (>800 mL stool or ostomy output while taking nothing by mouth/24 hours) from those whose stool output is driven primarily by osmotic agents, food and fluids. If a hospitalized patient is going for a procedure, the clinician may be able to get this information during the overnight fasting period. Remember to AVOID stool collections in patients going for procedures in which gastrograffin or a bowel cleansing (e.g., Colyte, etc.) prep is used!

Table 12
Typical ostomy/stool output with each type of resection (11)

- Colostomy = 200–600 mL/day
- Jejunostomy = up to 6 liters/day; must limit oral fluids and give IV or a vicious cycle will result—excess oral fluid in—increases stoma output
- Ileostomy = Initial = 1200 mL; down to ~600 mL/day

- The collection can be made more accurate by having the patient use a stool hat for both stool and urine output (measured and recorded separately). See Appendix I for a simple data collection form that can be given to patients. Any other losses from venting g-ports, fistulas or surgical drains should also be documented. In the hospital setting, assume ostomy output is > than that recorded on the nursing I & O sheets if leakage is a common problem or the wound and ostomy nurse (enterostomal therapist) is frequenting the patients room because the appliance will not stay on.
- All enteral, IV and/or TPN regimens must be documented including infusion time and volume of each therapy.
- A 72-hour *quantitative* fecal fat assay may be required to document medical necessity for TPN (18). A quantitative fecal fat is more accurate than a

qualitative fecal fat. The former determines *the amount* of malabsorbed fat whereas the latter only gives a (+) or (–) determination IF there is fat in the stool. Fecal fat collections done at home are typically more reliable than those obtained in the hospital setting—where it is not uncommon for patients to be started on a 72-hour fecal fat collection only to be made NPO for a procedure. Too often, hospitalized patients are deemed negative for fat in their stool when they never had the opportunity to put any in their gut! For results to be accurate, patients must consume fat (and enough of it), either by mouth or feeding tube, in order for them to malabsorb it. See Table 13 for guidelines for a 72-hour fecal fat collection and Appendix II for a sample 100 gram fat diet for patients to use during the collection.

5. Record ALL current medications including dosing, timing, and route of delivery

This is the downfall of many a patient. It is not enough to ask if patients have been on gut slowing agents. It is important to know the exact medication prescribed, including the dosage, timing and frequency. All of these factors are key in patients with SBS. For example, gut-slowing medications should always be given one half to 1 hour *BEFORE* meals and bedtime. This gives the medication time to slow peristalsis and increase the nutrient contact time. This also prevents the medication from potentially being washed out with food and fluids. Check for, and avoid using, sustained-release medications in this patient population.

6. Typical diet intake

Conduct a detailed 24 hour or longer recall of all foods AND fluids consumed. Investigate if dietary restrictions have been imposed and determine what diet, if any, the patient follows. It may be useful to keep a set of measuring cups and spoons in clinic to act as a guide for patients to better determine the size of the portions they **actually** consume. Specifically ask about the consumption of any highly osmotic beverages such as Boost, Ensure, sodas, etc., or protein powders, herbals and vitamins. Consider having outpatients complete a basic diet history questionnaire while they are waiting to be seen in clinic (see Appendix III).

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Table 13
Important Considerations When Ordering a 72 Hour Quantitative Fecal Fat

1. Order a 100 g fat, regular diet.
2. If hospitalized, ensure that the nutrition services department has such a diet and that because grams of fat are specified, it is not interpreted as a “low fat” diet.
3. Inform the patient what constitutes fat and encourage them to eat it.
4. Do not make the patient NPO at midnight for a test—clear their in-patient calendar for the test (or get the procedures done, then start).
5. Record ALL fat ingested during collection period—start a calorie count for “grams of fat *eaten*” (not calories) to run concurrent with the 72 fecal fat collection. Enlist a dietitian to oversee all of this.
6. Inform the nursing staff that the diet records are as important as the stool collection.
7. Have the patient or family keep the records if they are reliable.
8. Consider nocturnal enteral infusion of a high fat formula if the collection is absolutely necessary to document need for potential home TPN.
9. The cost of a 72-hour fecal fat
 - Cost of nursing time
 - Send out in most hospitals—turn around time ~7–10 business days; > if it is over a holiday
 - Unpleasantness factor for patient (↑↑)

(continued from page 82)

Table 14
Assessing and Maximizing Hydration

- Maintain urine output >1200 mL (have patients measure their UOP periodically).
- Serial weights: if patient loses >0.5 lb/day or 1 kg/wk—add, or increase IV fluids.
- A rise in BUN/creatinine ratio is a late indicator in ostomates; use other indicators such as blood pressure, signs of tachycardia or drop in urine output.
- Patients with end jejunostomies: drink/sip higher sodium, relatively isotonic beverages—avoid hypotonic fluids such as water, tea, coffee, juices and alcohol.
- Patients with colon intact can handle a wider range of fluids, but avoidance of very hypertonic fluids is still beneficial.
- Try oral rehydration therapy.
- Try “isotonic” beverage recipes (see Tables 20, 21, 23).
- Avoid nephrolithiasis
 - Avoid foods high in oxalate
 - Consider avoiding large doses of vitamin C supplementation—theoretical-no data to support (converts to oxalate endogenously)
 - Try calcium citrate supplements to bind oxalate and alkalize urine

7. Specific nutrient issues

Vitamin and mineral levels need to be monitored regularly in patients with SBS. Even those patients on TPN should have levels checked. Baseline levels should be checked at the first clinic visit or 3 months out from the initial surgery or insult. Patients with SBS are most at risk for deficiency of fat-soluble vitamins, A, D, E and K as well as vitamin B₁₂. Loss of ileum, gastric hypersecretion and its effects on R protein and/or bacterial overgrowth can all contribute to vitamin B₁₂ deficiency.

• Sodium (Na)/Water

One of the most problematic minerals for SBS patients is sodium (Na). Excessive Na depletion may lead to hypotension and prerenal azotemia. Chronic losses are associated with low plasma volume, reduced sodium output in urine and results in increased plasma aldosterone. The secretion of aldosterone by the adrenal cortex is regulated by two mechanisms: first, the concentration of sodium ions may be a factor since increased rates of aldosterone secretion are found when dietary sodium is severely limited; second, by reduced blood

flow to the kidney. Aldosterone acts directly on the kidney to decrease the rate of sodium (with accompanying retention of water). Hyperaldosteronism is not uncommon in this patient population. Sodium losses are greater in ileostomy effluent compared to jejunostomy effluent (120 versus 90 mEq/L, respectively), however, net jejunostomy Na losses are often greater given the higher volume of stool lost each day (11).

Identifying dehydration may be difficult in this population because low muscle mass is a common finding, and BUN and creatinine may remain low until the late stages of dehydration; only in severe dehydration will serum creatinine and blood urea nitrogen (BUN) begin to rise. Rather, look for rapid weight loss (>0.5–1.0 kg/day), changes in blood pressure, tachycardia, and drop in urine output. Urinary electrolytes are more useful than serum electrolytes in these patients as serum concentrations are often maintained because of normal homeostatic mechanisms that preserve these levels until the late stages of dehydration. To detect sodium depletion, check random urine Na (make sure patient is not receiving diuretics). A urine sodium of <5–10 mmol/L, is indicative of maximal sodium concentration and hence, Na depletion. The goal should be to keep urine sodium >20 mmol/L. Advise patients to add salty meals and snacks to their diet.

Some patients with jejunostomies suffer from excessive thirst tricking them into quenching it with water or other hypotonic fluids. The leaky epithelium in the jejunum causes significant sodium loss, further increasing stomal output, chronic dehydration, thirst and fluid intake, precipitating a vicious cycle difficult to halt. See Table 14 for tips on assessing and maximizing hydration.

• Magnesium (Mg)

Hypomagnesemia is also a common problem in patients with SBS. Sodium and water depletion cause secondary hyperaldosteronism, which increases urinary magnesium losses, hence these need to be addressed first. Patients with end jejunostomies often have high magnesium losses and often suffer from recalcitrant hypomagnesemia. Magnesium deficiency may precipitate a serum calcium deficiency due to impaired release of parathyroid hormone. Hypomagnesemia reduces secretion and function of PTH resulting in poor renal conservation of calcium along with insufficient 1,25-hydroxy-

vitamin D production, resulting in decreased calcium and magnesium absorption in the gut. Normal urinary magnesium excretion is important in the prevention of hypocitraturia and calcium oxalate renal stone formation. Citrate excretion and urine pH are decreased by metabolic acidosis (usually from GI bicarbonate loss) as well as hypomagnesemia. Hypocitraturia can be corrected by oral citrate supplementation such as citrical liquitabs, twice daily. Some suggest that treatment should be aimed at normalizing urinary magnesium and correction of metabolic acidosis (19).

If oral magnesium is to be tried, prescribe it at night when transit may be slowest and the patient is NPO to avoid competition with other medications/foods (20). Desai reported that an oral sipping solution made of injectible (IV) magnesium, a highly purified form, may improve absorption in patients who have lost large segments of small bowel (21); palatability can be improved by mixing it with a flavored oral rehydration solution.

Finally, a trial of an active vitamin D analog, such as calcitriol or Rocaltrol, may need to be given (20). See Table 15 for guidelines to treat hypomagnesemia.

8. Medication Absorption—May need a “malabsorption factor” just like with nutrients...

Medication absorption, like fluids and nutrients, may be impaired in patients with SBS. Consider that some patients have to consume 200%–400% of their basal metabolic rate to meet their nutrient needs and remain free from TPN. It stands to reason that medication delivery may need to be escalated considerably as well (examples: thyroxine, warfarin, digoxin, etc.) (11). Also consider that medication absorption depends on: the surface area and health of the remaining intestine, morphologic and physiologic factors (such as intact ileum for bile salts—required for some medications to be absorbed such as cyclosporin), the pH of the intestine and transit time (4,22).

Because many medications are absorbed in the jejunum, patients with a significant jejunal resection are at particular risk of medication malabsorption. Imodium and cyclosporin circulate through the enterohepatic circulation, hence higher doses may be needed in patients with <100 cm of remaining ileum (1).

Doses that would appear excessive in the typical patient may be required to achieve efficacy in the

Table 15
Treatment of Hypomagnesemia (11,19–21,43)

- Correct water and sodium depletion (and thus secondary hyperaldosteronism).
- Use an oral magnesium preparation (e.g., 12 mMol magnesium oxide (20) at night when transit may be slowest and the patient is NPO to avoid competition with other medications/foods.
- Reduce lipid in diet as a high fat diet binds magnesium increasing stool losses.
- Trial calcitriol or Rocaltrol (active vitamin D analog).
- Give intravenous magnesium over as many hours as possible → >8 hours minimum; continuous is best, to decrease renal wasting.
- Try adding Injectible magnesium to ORT for sipping
 - Magnesium sulfate—5mEq/mL Abbott Pharmaceuticals, Inc.
- Replete magnesium until normal urinary magnesium achieved.

Adapted and used with permission from Nightingale JM (ed). *Intestinal Failure*. Greenwich Medical Media Limited. London, England. 2001:188.

patient with short gut. To determine if patients are benefiting from the medication you have prescribed, determine if they are responding according to your expectations for that particular drug, such as a decrease in ostomy/stool output, etc. If they are not, then absorption must be questioned. If available, a trial of a sublingual, liquid or IV version may be considered. If the pharmacy that the patient uses does not have readily available alternative forms of medications, it may be necessary to utilize other specialized pharmacies, request a special order, or the clinician may need to adjust the treatment plan. Additionally, insurance companies may require written justification for a particular formulation (liquid versus tablet) of a medication—just saying the patient has SBS may not be enough.

One final consideration is to make sure the local pharmacy where the patient gets their prescriptions filled carries, or can obtain, the medication the patient is to be discharged on (e.g., tincture of opium and Viokase powder).

9. Finally—Meeting the patient's needs and expectations

No plan, however carefully constructed, will be successful without the cooperation of the patient. It is essential to establish a good relationship and uncover

the patient's chief complaints and goals. This may vary drastically between patients. Common goals are the need to gain weight, improve sleep, improve ostomy management and to reduce the hassles of TPN, namely septic episodes and prolonged infusion time.

NUTRITION INTERVENTION

Oral Diet (23,24)

- *High fat vs low fat or high CHO vs low CHO?*

It is now accepted that patients with SBS should consume whole food diets, or if enterally fed, use a polymeric formula for maximum intestinal stimulation and adaptation (Wilmore-Best Practice). Although a high fat diet can contribute to nutrient loss by aggravating steatorrhea, in order to potentially gain freedom from TPN, patients with SBS may need to eat 200%–400% over their needs to surmount the malabsorption that occurs (25). It is also suggested that diets should differ somewhat in patients with a colonic segment versus those without. A higher CHO, lower fat diet is preferable in patients with colon in continuity as compared to a higher fat, lower CHO diet in patients with jejunostomies, to reduce total fluid losses (8,26). Review the patient's usual diet intake and modify fat in the diet only as needed, while instituting the other interventions, rather than imposing a set 40-g/day limit. Remember that fat is an important calorie source! The author suggests maximizing medication delivery before imposing strict dietary guidelines—no diet is a good diet if not eaten. Be aware, however, that in patients on higher fat diets, the divalent minerals (calcium, magnesium, zinc and copper) may need to be increased. Oral dietary guidelines can be found in Table 16 with a sample meal plan in Table 17.

- *Avoid sweets*

A diet high in simple CHO will pull water into the lumen of the GI tract precipitating net fluid/nutrient loss. Avoiding simple sugars such as sweets, fruit juices and sodas are one of the few beneficial dietary restrictions in this patient population.

- *Chew foods well*

Because the first step in the process of digestion of food takes place in the mouth with mastication of food, theoretically, it is even more important in patients with

SBS to aid in maximizing surface area for pancreatic enzyme and bile salt attack.

- *Liberal use of salt*

Remember that jejunostomies and ileostomies lose ~90 mmol sodium/Liter of ostomy output at the duodeno-jejunal flexure and up to 140 mmol/L in the terminal ileum (11). Do not restrict salt and encourage use of the salt shaker and salty foods.

- *Fiber*

Use of a moderate fat, higher complex CHO diet that contains fiber is best in the patient with an intact colon. The caloric capture of SCFA from fiber fermentation by colonic bacteria provides up to 500–1000 kcal/day (7,8). Avoid Metamucil or other bulk-forming agents in patients with jejunostomies or ileostomies. A colon is necessary for fiber-containing, bulk forming agents to work. In high output states, it may draw more fluid into the SB and potentially drag nutrients out with it.

- *Lactose (27,28)*

SBS may precipitate lactose intolerance. However, Marteau demonstrated that a diet providing 20-g/d lactose (with no more than 4 g/d as milk), was well tolerated in patients with short-bowel syndrome and concluded that a lactose-free diet is not particularly beneficial in these subjects (29). Symptoms of lactose intolerance are dose-dependent, hence, if intake of lactose is spread out over the day, symptoms may be alleviated. Avoiding lactose-containing foods may further aggravate the premature development of osteoporosis so commonly seen in this patient population. See Table 18 for lactose content of selected foods/fluids.

- *Oxalate (30)*

For those patients with retained colon and in particular <100 cm of ileum remaining, malabsorption of fat occurs with preferential binding to calcium freeing oxalate to be absorbed in the colon. Increased oxalate absorption results in an increase in oxalate excretion via the kidney. Add marginal hydration, and the stage is set for nephrolithiasis. Metabolic acidosis due to excessive bicarbonate loss in the stool adds further insult by aiding stone precipitation. See Table 10 for a list of high oxalate foods to avoid in those patients who have a history of kidney stones.

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Table 16
Diet Guidelines for Short Bowel Syndrome

General Guidelines

- Patients with jejunostomies/ ileostomies (higher fat): approximately 20–30% CHO, 20–30% protein, 50–60% fat
- Patients with intact colon (higher CHO): approximately 50–60% CHO, 20–30% protein, 20–30% fat
- Avoid concentrated sweets and fluids
- Chew foods well
- Add salty meals and snacks if no colon
- Eat smaller meals, more often
 - Decrease total nutrient load over the day and space out over time
- Trial of oral rehydration solutions
- Limit fluids with meals; drink isotonic beverages
- Separate solids and liquids at meals as much as possible (solids before liquids)
 - Solids slow emptying
 - Too much liquid creates a column effect (imagine the swelling of a stream when it rains and the increased flow generated)
- Use MCT containing beverages if necessary vs MCT oil (45)
- Lactose restriction if necessary (may try lactaid)
- Avoid high oxalate foods in those patients with kidney stones
- Liquid or chewable vitamin/mineral supplements if necessary
- Limit or avoid enteral stimulants such as alcohol and caffeine

Good Choices

Avoid

Starches/breads

- Breads, pita bread, rolls
- Bagels, English muffins
- Plain waffles or pancakes
- Corn bread, plain muffins
- Banana or zucchini bread
- Tortillas—whole wheat or white flour, corn—toasted
- Pasta, macaroni, noodles
- Rice, brown rice, wild rice

Donuts, sweet rolls, pastries, pop-tarts

Cereals

- *Unsweetened* cereals (wet or eaten dry as a snack)
- Cheerios, cornflakes, rice krispies, Rice Chex, Spoonfuls, Special K, Kix, puffed rice or wheat
- Hot cereals: cream of rice or wheat, grits, oatmeal

Sugary cereals, high fiber cereals (>1–2 grams fiber/serving), bran cereals
Flavored hot cereals

Vegetables

- Canned or cooked vegetables
- Potatoes, sweet potatoes, yams
- Small amounts of lettuce (1/2 cup)

Creamed vegetables, legumes such as lima, kidney, pinto beans, etc.

Fruits

- Bananas, melons, unsweetened canned fruits (applesauce, pears, peaches, mandarin oranges, apricots, cherries, plums, etc.)

Dried fruits, fruit canned in syrup
Fruit juice, fruit drinks, watch out for high fructose corn syrup in drinks (Ex. Capri-sun, canned fruits in syrup, etc.)

Meats/fish/poultry

- Meats, fish, shellfish, poultry, tuna fish, ham

Heavily fried meats, fish, poultry

Dairy/Soy

- Cheese, cottage cheese, plain yogurt or yogurt sweetened with artificial sweeteners, cream cheese
- Plain soy milk

Highly sweetened yogurts or kefir, chocolate or other flavored milks, cream, half and half, Go-Gurts, flavored soy milks

Table 16 (continued)

Good Choices

Eggs

- Poached, hard or soft cooked, omelet, scrambled

Nut butters

- Peanut, almond, cashew

Beverages

- Oral Rehydration solution
- Soups, broth—4 oz per day
- Lactaid milk

Snacks

- Crackers—saltines, soda, etc.
- Pretzels, matzo
- Corn or potato chips
- Bagel snack crackers

Desserts

- Animal crackers, graham crackers, angel food cake, vanilla wafers, shortbread, plain pound cake, cake donuts—*no icing*, marshmallows

Miscellaneous

- Salt, pepper, herbs, spices, dill pickles, Splenda, Equal, Sweet 'n Low, etc.

Avoid

Eggs prepared with ingredients not allowed

Nutella, peanut butter with jam/jelly mixed in it

> 4 oz coffee, tea, ice tea, flavored coffees or teas, hot cocoa, Ovaltine, Quick, fruit juices or fruit drinks (watch out for *high fructose corn syrup* in drinks.), koolaid, tang, regular sodas (all kinds), alcohol, water, sugar free beverages, supplements such as Boost or Ensure, etc.

Iced Cakes, cookies, Little Debbie Cakes, pie, ice cream, sherbet, candies, donuts, sweetened gelatin, etc.

Sugar, sorbitol containing sweets, maple or other syrups, jams, jellies, chocolate syrup, honey, molasses

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- *Medium Chain Triglycerides—leave home without them*

Medium chain triglycerides (MCT) are often recommended for use in patients with SBS as they are absorbed directly across the mucosa into the bloodstream and taken directly to the liver. However, MCT are less effective than long chain fat in inducing gut adaptation (31). Although the rationale is appealing, using MCT as part of a daily diet plan is at best just that, an appealing rationale. Most patients would just as soon leave the MCT oil on the shelf (author's experience). Expensive, unpalatable and somewhat of a hassle to administer, too much MCT can overwhelm the mucosal receptors and actually increase steatorrhea. A less expensive way to incorporate MCT is by using commercial products that contain MCT as part of the fat source (Table 19).

- *Oral Rehydration Therapy (ORT)*

Absorption of sodium (and hence water—as water

always moves with sodium) occurs by 3 different mechanisms across the GI tract brush border:

1. Passive absorption; probably through the intracellular junctions of the mucosal cells.
2. Active absorption of sodium, mediated by the sodium-potassium pump.
3. Glucose-coupled transport of sodium (most active in the jejunum):
 - The coupling between glucose and sodium is obligatory—the “glucose carrier” will not translocate sodium in the absence of glucose and vice-versa.
 - The carrier permits one sodium ion to enter the intestinal epithelial cell along with each glucose molecule.
 - Coupled transport is uni-directional.

The beauty of ORT is that it can be absorbed even in the setting of significant diarrhea (32). However, it is not a panacea, and in some SBS patients, it too can increase stool output. Drinking ORT is a grand idea, but in reality, patients do not relish it. Even though your patient

NUTRITION ISSUES IN GASTROENTEROLOGY, SERIES #31

Table 17
Sample Menu (2400 calories, 50% carbohydrate, 20% protein, 30% fat)

<p>Breakfast</p> <p>1 cup oatmeal 2 oz lactose-free milk 1 egg 1 English muffin or 2 slices toast 2 teaspoons margarine 1–2 teaspoons diet jelly 4 oz coffee</p> <p>Morning Snack</p> <p>1/2 bagel 1 ounce cheese 1 teaspoon margarine 1/2 banana 4 oz water</p> <p>Lunch</p> <p>3 oz baked ham 1/2 cup rice 1/2 cup carrots 2 small dinner rolls 2 teaspoons margarine 4 oz water or diet soda</p>	<p>Afternoon Snack</p> <p>1/2 bagel 1 oz cheese 1/2 banana 1 oz pretzels 4 oz water</p> <p>Dinner</p> <p>3 oz roasted chicken 1 large baked potato 2 dinner rolls 2 teaspoons margarine 4 oz water or diet soda</p> <p>Evening Snack</p> <p>1 roast beef sandwich: (2 slices whole wheat bread, 1 oz meat, 1 teaspoon mayonnaise, 1 teaspoon mustard) 4 oz water</p> <p>Additional Fluid over the course of the day in-between meals</p> <p>1.5–2.0 liters (or more as needed)</p>
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Adapted from Byrne TA, Veglia L, Camelio M, et al. Beyond the prescription: optimizing the diet of patients with short bowel syndrome. *Nutr Clin Pract.* 2000; 15:309, with permission from the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). A.S.P.E.N. does not endorse the use of this material in any form other than its entirety.

Table 18
Lactose Content of Common Dairy Products (44)

Product	Serving Size	Lactose Content (g)
Milk (whole, lowfat, or nonfat)	1 cup	11
Buttermilk	1 cup	10
Acidophilus milk	1 cup	11
Yogurt, whole milk	1 cup	10–12
Yogurt, low fat	1 cup	5–19
Cheese (such as cheddar, American, Swiss, parmesan)	1 oz	0.4–3
Cottage cheese	1/2 cup	3–4
Ice Cream	1/2 cup	5–6
Sherbet, orange	1/2 cup	2
Whipped cream	1/2 cup	3
Sour cream	1/2 cup	4
Cream cheese	1 oz	1
Half & Half/cream	2 tbsp	1
Butter/margarine	1 tbsp	Trace

may need 2–3 liters, start with the goal of 1 liter per day. If the patient will drink (or sip ideally), this over the course of the day, and is willing to maintain doing this, increase the volume as needed. If not, then abandon ORT as a therapy. Of note, fructose does not participate in the same coupling mechanism as glucose, hence it is not a useful CHO source in ORT. See Table 20 and 21 for commercial products and home recipes to try (if regular ORT is not successful). These improvised recipes, even if not the exact same chemical make-up of ORT, may be more palatable. This may increase the chance that the patient will comply with the recommended volume and can serve as a more appropriate substitute for the alternatives often chosen by patients (water, soda, juice, Boost, Ensure, etc.). Finally, one more option is to try giving ORT as a nocturnal infusion, first via an NGT and if successful, place a PEG for infusion. This may allow some patients to leave their intravenous fluids behind (33).

• *Isotonic Fluids*

As clinicians we are taught to use isotonic fluids in this patient population, however, aside from oral rehydration solutions, what does that mean and how does this translate into drinkable beverages for patients? Table 22 provides the osmolality of common beverages consumed by patients. Although they have not been clinically tried, Table 23 provides some “isotonic recipes” that can be tried with patients and theoretically, given their near isotonicity, might be less problematic than the sodas, fruit juices and gallons of water, etc., that our patients succumb to out of frustration for something to quench their often insatiable thirst.

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NUTRITION ISSUES IN GASTROENTEROLOGY, SERIES #31

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Table 19
Products Containing Medium Chain Triglycerides (MCT) (45)

Product	Description	Serving Size	g MCT/ serving	Kcal/ serving	Cost/serving	Manufacturer
MCT Oils						
MCT Oil	Oil	1–3 Tablespoons	6–18	50–150	\$0.76–2.28	Mead-Johnson www.meadjohnson.com 800/361-6323
MCT Oil	Oil	1–3 Tablespoons	6–18	50–150	\$1.04–3.14	CVS Pharmacies www.cvs.com
MCT Fuel	Oil	1–3 Tablespoons	6–18	50–150	\$0.42–1.28	Twin Labs http://www.iherb.com/mct1.html
MCT Power (Tropical Punch Flavor)	Oil	1–3 Tablespoons	6–18	50–150	\$0.53–1.59	Universal Nutrition 800/872-0101 www.universalnutrition.com
MCT Oil Containing Products[^]						
Lipisorb	Liquid nutritional	240 mL	11	240	\$3.50	CVS Pharmacies www.cvs.com
Nutren 1.5, unflavored	Liquid nutritional	250 mL	11	375	\$1.50	Nestle www.nestleclinicalnutrition.com 800/776-5446
Nutren 2.0	Liquid nutritional	250 mL	20	500	\$5.98	Nestle www.nestleclinicalnutrition.com 800/776-5446
Promote	Liquid nutritional	240 mL	5.0	240	\$1.85	Ross www.ross.com 800/258-7677

[^]Can increase calorie content and palatability with sherbet or sorbets

ENTERAL NUTRITION (EN)

The most important intervention to improve success with EN is to feed in the most proximal location possible, e.g., gastric delivery. When choosing a formula for EN, look for one that is isotonic or near isotonic (300 mOsm), polymeric, and contains some MCT. Initially, infuse EN at slow rates over time (e.g., 12–24 hours initially) to decrease nutrient load per centimeter of bowel for maximal saturation of intestinal transporters. If intolerance (steatorrhea or excessive ostomy stool losses) occurs, consider adding pancreatic enzymes. A semi-elemental formula may also be helpful, but avoid elemental formulas as they are hypertonic and have not demonstrated a physiologic advantage (34–36). Pancreatic enzymes and semi-elemental formulas are rarely used in combination unless the semi-elemental formula contains a fairly high percentage of MCT. A

fiber-containing formula, in patients with any remaining colon, should be used to increase caloric “salvage” via bacterial fermentation of the fiber into SCFA.

TOTAL PARENTERAL NUTRITION/INTRAVENOUS FLUIDS

Some patients may require TPN and/or IV fluids permanently. When preparing the hospitalized patient for discharge on TPN, mimic the anticipated home regimen for at least 48 hours prior to discharge. For example, if the patient will be receiving only TPN at home, ensure the patient can maintain hydration status without additional IVF while in the hospital. Some patients may require both TPN and IVF. “IV chasers” may be given before or after TPN if the patient needs more fluid than a home TPN bag can hold (4 liters).

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Table 20
Commercial and "Pseudo" Oral Rehydration Solutions

COMMERCIAL PRODUCTS					"Pseudo-ORS" RECIPES			
<i>Solution</i>	<i>mOsm/kg</i>	<i>Glucose/CHO (g/L)</i>	<i>Sodium (mEq/L)</i>	<i>Potassium (mEq/L)</i>	<i>Recipe</i>	<i>Glucose/CHO (g/L)</i>	<i>Sodium (mEq/L)</i>	<i>Potassium (mEq/L)</i>
WHO packet* (Jianas Brothers)	330	20	90	20	Gatorade Base • 2 cups Gatorade • 2 cups water • 1/2 teaspoon salt	28	82	1.5
Rehydralyte (Ross)	310	25	75	20	All Sport Base • 1 1/2 cps All Sport • 3 cups water • 1/2 teaspoon salt	25	78	1.6
Parent's Choice (Wal-Mart Brand)	~250	20	45	20	Sugar and Salt Water • 1 quart water • 1/2 teaspoon salt • 6 teaspoons sugar • (optional/Crystal Light to taste—(especially lemonade or orange-pineapple flavors)	24	76	0
Ceralyte 70** (Cera)	220	40	70	20	Grape or Cranberry Juice • 1/2 cups juice • 3 1/2 cups water • 1/2 teaspoon salt	28/27	76/76	6.4/ 0.9
Ceralyte 90** (Cera)	260	40	90	20	Apple Juice • 1 cup apple juice • 3 cups water • 1/2 teaspoon salt	28	76	5.58
Liquilyte (Gerber)	250	25	45	20	Enlive Base • 8 oz Enlive • 3 1/4 cups water • 3/4 teaspoon salt	65	80	40
Pedialyte (Ross)	250	25	45	20	Resource Fruit Beverage Base • 8 oz Resource fruit beverage • 3 cups water • 1/2 teaspoon salt	35	76	0.4
EquaLyte (Ross)	305	25	78	22	Boost Breeze Base • 8 oz. Boost Breeze • 3 cups water • 1/2 teaspoon salt	31	75	5.9

*Jianas Brothers, Kansas City, MO 816/421-2880; jianasp@aol.com

**Cera Products, Inc., Columbia, MD 888/237-2598; www.ceralyte.com

Table 21
"Homemade" Oral Rehydration Solutions

WHO

- 1/2 teaspoon salt
- 1/2 teaspoon potassium chloride
- 8 teaspoon sugar
- 1/2 teaspoon sodium bicarbonate (baking soda)
- 1 liter Water (4 1/2 cups)

Combine and stir until well mixed and dissolved.

Washington University's Formula

- 1/2 teaspoon salt
- 1/2 teaspoon sodium citrate
- 3 tablespoons + 1 teaspoon powdered polycose (Ross)
- 1 liter Water (4 1/2 cups)
- Crystal Light to taste (especially lemonade or orange-pineapple flavors)

Combine and stir until well mixed and dissolved.

Homemade Cereal Based

- 1/2 cup dry, precooked baby rice cereal
- 2 cups water
- 1/2 teaspoon salt

Combine ingredients and mix until well dissolved and smooth. Refrigerate. Solution should be thick, but pourable and drinkable.

Homemade Recipe

- 1 liter Water (4 1/2 cups)
- 1 cup orange juice
- 8 teaspoons sugar
- 1/2 teaspoon baking soda
- 1/2 teaspoon salt

Combine and stir until well mixed and dissolved.

WHO Recipe for Pediatrics

- 2 tablespoons sugar or honey
- 1/2 teaspoon salt
- 1/2 teaspoon baking soda
- 1 liter Water (4 1/2 cups)

Combine and stir until well mixed and dissolved.

Some of the quality of life issues that should be addressed are:

How many hours will your patient's TPN run at home? Home IV pumps can run as high as 300 mL/hour. In the hospital setting this rate may seem alarming, but IV rates of 250–350 mL/hour are not uncommon at home. The total number of hours a

Table 22
Osmolality of Selected Liquids

<i>Beverage</i>	<i>(mOsm/kg)</i>	<i>Beverage</i>	<i>(mOsm/kg)</i>
Milk	275	Prune juice	1265
Malted milk	940	Grape juice	863
Ice cream	1905	Apple juice	683
Eggnog	695	Orange juice	614
Fruit yogurt	871	Tomato juice	595
Sherbet	1225	Punch with sugar	448
Popsicles	720	Sugar free punch	29
Ensure/Boost	590/640	Mineral water	74
Ensure Plus/Boost Plus	680/720	Broth	445
		Gatorade	330
Boost Breeze	920	Polycose	900
Enlive	840	Flavored gelatin	735
Resource fruit beverage	750	Sodas	~610
Enteral formulas	250–710	D ₁₀	505

Used with permission from the University of Virginia Health System Nutrition Support Traineeship Syllabus (42)

patient runs should be based on their preference, the number of times they are getting up at night to urinate or rarely, cardiac status. Most patients infuse their TPN over 10–12 hours overnight.

Discuss with the patient whether or not nocturnal versus daytime infusion is preferred. Frequent urination at night interferes with sleep and is a common complaint of patients on nocturnal TPN. Some patients may also be interested in IV backpacks to allow daytime infusion with increased mobility. TPN and IVF will need to be continually reevaluated and adjusted as needed. Over time, the daily volume and/or infusion time of the TPN may be able to be decreased. Some patients might even be able to maintain nutrition and hydration status without daily TPN infusion—it is not uncommon for patients with SBS to skip their TPN infusion for 1–2 days per week. A thorough review of home TPN is available elsewhere (17). See Table 24 for Nutrition Support options.

MONITORING

Implementing the suggestions in this article is just the tip of the iceberg. The real work with SBS patients requires ongoing monitoring which is essential for their successful management. Too often, interventions are

Table 23
Isotonic Beverage Recipes and their Tonicity (mOsm/L)*

Dairy Containing Recipes

1 ounce Eggnog	310	1 ounce Ensure Plus	324
11 ounces Whole Milk		8 ounces 2% Milk	
14 ounces Pedialyte	322	14 oz Pedialyte	333
1 ounce Ice Cream		1 oz Sherbet	
1 ounce Gingerale		1 oz gingerale	
1 ounce Sorbet or Sherbet	330	18 oz Osmolite	331
16 ounces 2% Milk		1 oz Sherbet	
1 ounce Ensure	310	11 oz milk	324
8 ounces 2% Milk		1 oz fruit yogurt	

Lactose Free Recipes

15 ounces Pedialyte	311	6 ounces Pedialyte	299
1 ounce Sorbet or Sherbet		2 ounces Chicken Broth	
12 ounces Pedialyte	317	8 ounces Pedialyte	324
2 ounces Orange Juice		1 ounce Boost Breeze	
2 ounces Gatorade			
13 ounces Pedialyte	318	8 oz Pedialyte	315
2 ounces Gingerale		1 oz Enlive	
1 ounce Grape Juice			
13 ounces Pedialyte	321	8 oz sugar free punch	328
2 ounces Gingerale		2 oz Sherbet	
1 ounce Apple Juice			

*Sip between meals

Prepared by Dave Stebbins, RDE & Carol Parrish, RD, MS

initiated, but since the patient does not know what to expect (and therefore does not know what questions to ask), further improvements in the plan are not identified. Assuring that treatments and interventions are working, and if they are not, devising a new plan—this is the cornerstone of effective management for the SBS patient.

Periodic measurements of 24-hour urine and stool output should be conducted to determine the success or failure of the current management plan. Patients with urine output <1 liter per day are at risk for nephrolithiasis and will need nocturnal enteral or IV fluids (14). Serial weights are also a good indicator of hydration status. Decreases in weight will often occur more rapidly than an increase in blood urea nitrogen or creatinine. A sudden increase in stool or ostomy output requires evaluation

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Table 24
Potential Combinations of Nutrition Support in Patients with Short Bowel Syndrome

- If EN used:
 - Feed gastrically (vs post-pyloric) to maximize coordinated delivery to small bowel and include as much small bowel surface area as possible.
 - Run EN continuously or nocturnally vs bolusing to decrease nutrient load over time for total saturation of gut transporters
 - Use standard, polymeric formula, with fiber if colon present
- Possible combination treatments:
 - Oral diet during day, nocturnal EN
 - Daytime EN/ nocturnal ORT
 - Oral day /nocturnal IV
 - Oral, TPN
 - EN and TPN
 - EN, IVF
 - IVF's – decrease total volume down to 1 L per day, then decrease 1 day at a time (qod)—if pt losses >1 kg/week, stool losses exceed 600 mL or unacceptable electrolyte abnormalities arise, restart or increase IV fluids (2)

Note: in preparing patients for home, make sure all IV fluids are consolidated or accounted for at least 2 days prior to discharge to mimic the home plan to ensure success.

Table 25
Trouble-Shooting Sudden Increase in Ostomy/Stool Output

1. Partial small bowel obstruction
2. Abdominal sepsis
3. Recurrent disease such as Crohn's
4. Entero-entero fistula
5. Infectious enteritis
6. Check for *C. Difficile* (even in patients without a colon)
7. Adrenal insufficiency due to sudden discontinuation of corticosteroids (Ex. Patient with ulcerative colitis s/p colectomy)
8. Evaluate for new onset hyperthyroidism
9. Inadvertent use of lactulose, Kayaxalate, Neutra-Phos, Shohl's solution or other diarrheagenic medication
10. Sudden discontinuation of an important medication in the patient's treatment plan (they run out of their prescription, etc.)

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Table 26
Summary of Clinical Guidelines to Short Bowel Syndrome (42)

1. Start a patient demographic sheet for the front of the chart
 - Anatomy—(based on?), presence of gall bladder, etc., IV or TPN therapies
 - Timed small bowel follow through—date?
2. I & O
 - Baseline 24-hour stool/ostomy and urine output (Fasted or on usual intake?)
 - Recheck weekly to assess efficacy of interventions
3. Medication Issues
 - Make sure patients pharmacy carries the drug (esp. tincture of opium, Viokase powder)
 - Note doses, form of medication and timing
 - Hypersecretion
 - PPI (liquid vs capsule vs IV)
 - Clinical pearl: check pH of fresh effluent from stoma of jejunostomy or ileostomy for gross determination of PPI adequacy (look for pH >6)
 - Increase dose of PPI, use liquid/solutab or try IV to see if stool / ostomy volume decreases
 - Gut slowing
 - Review all medications (if liquid, check for sorbitol); hold any *non-essential* meds during initial gut slowing phase to avoid osmotic effects
 - Use gut-slowing—titrate to pt; if at first you don't succeed, try bigger and bigger guns
 - Give 1/2 hour before meals and bedtime to maximize efficacy—if pt routinely gets up at night and is willing to do so, change to every 6 hour dosing
 - Increase until the stool consistency is adequate for pt or the pt is too sleepy/cannot perform activities of daily living—whichever comes first
 - Net Secretors
 - Octreotide
 - Bile salt loss
 - Bile acid sequestrants such as cholestyramine (in patients with <100 cm of terminal ileum remaining)
 - Bile acid replacer such as cholesarcosine
 - Bacterial overgrowth
 - Enteral antibiotics
 - Pancreatic enzymes if needed
 - 1/2–1 teaspoon per can of standard or semi-elemental tube feeding delivered
 - 2–6 tablets with meals AND snacks
 - Viokase tablets or powder (Not sustained release!)
4. Hydration
 - Ensure patient can achieve a urine output of ≥ 1200 mL (1500 mL or > if colonic segment intact)
 - Try adding oral rehydration therapy, via a PEG if necessary, and if still unsuccessful, add IV fluids.

(see Table 25 for possible causes). Periodic assessment of vitamin and mineral indices as discussed are also necessary. In addition, regular review of the patient's medication list is imperative to check for the addition of new medications (especially over-the-counter, herbals, vitamins, etc). Finally, it is important to consistently assess the patient's overall functional status and quality of life.

CONCLUSION

Caring for a patient with SBS requires a tremendous amount of patience, persistence and attention to detail. Close follow up after the initial surgery or insult, a step-wise approach to care and continuous reformulation of the plan is imperative in order to achieve success. The purpose of this article is to provide the clinician with a template to follow in order to help the short gut patient achieve maximal gut absorption, which in turn, will bring patients closer to maximizing their overall quality of life. Summary guidelines and additional resources can be found in Tables 26 and 27. ■

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Table 27
Resources*

- **Oley Foundation**
<http://www.oley.org/>
- **Oxalosis & Hyperoxaluria Foundation**
<http://www.ohf.org/>
- **American Gastroenterological Association medical position statement: short bowel syndrome and intestinal transplantation**
http://www.guideline.gov/summary/summary.aspx?ss=15&doc_id=3795&nbr=3021
- **National Institutes of Health—Office of Dietary Supplements**
http://ods.od.nih.gov/Health_Information/Vitamin_and_Mineral_Supplement_Fact_Sheets.aspx
- **Short Bowel Syndrome: Etiology, Pathophysiology and Management**
http://www.clevelandclinicmeded.com/selected_topics/shortbowel/summary/article.htm
- **Wound Ostomy & Continence Nurses Society**
<http://www.wocn.org/>
- **United Ostomy Association**
<http://www.uoa.org/>
- **Intestinal Transplant Registry**
www.intestinaltransplant.org
- **NPS Pharmaceuticals' Short Bowel web site**
<http://www.shortbowel.com/>

*The October 2005 Practical Gastroenterology will feature Centers with Expertise in Short Bowel Syndrome

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Appendix II: UVAHS Digestive Health Center 100 Gram Fat Diet and Instructions for 72-Hour Fecal Fat Collection

Instructions:

1. Equipment needed:
 - "Hat" or specimen pan.
 - Storage Container (Can or Jug will be provided).
 - If the amount of stool exceeds the container given, you may use any container (preferably plastic or metal) with a screw on lid for the rest of the collection.
 - Stool does not need to be kept in the refrigerator or on ice.
2. After 3 days of stool is collected, take it to the UVAHS Digestive Health Clinic.
3. 100 gram fat diet instructions:
 - Start diet and follow for 4 days.
 - On the second morning, start stool collection and collect *all* stool for 3 days.
 - It is important that you eat about 100 grams of fat each day.
 - Also eat normal portions of other foods you would normally eat.
 - Please write down everything you eat and drink for the day, starting from the time you wake up in the morning until the time you go to sleep on the record sheet provided.
 - Be sure to include any sauces, etc., mayonnaise, butter or margarine added to your foods.
 - Do your best to guess the amount you have eaten.

<i>Food Item</i>	<i>Serving Size</i>	<i>Grams Fat</i>
Avocado	1/8	5
Margarine, Butter, Lard, oil	1 teaspoon	5
Diet Margarine	1 Tablespoon	5
Mayonnaise	1 teaspoon	5
Lite Mayonnaise	1 Tablespoon	5
Almonds	6 whole	5
Cashews	4 whole	5
Pecans	3 whole	5
Peanuts	20	5
Walnuts	1/4 cup	20
Peanut Butter or almond butter	2 Tablespoons	15
Vegetable/Cooking Oil (corn, soybean, sunflower, corn, olive, etc.)	1 teaspoon	5
Olives	10 small	5
Salad Dressing, mayonnaise type	2 teaspoons	5
Salad Dressing, oil varieties	1 Tablespoon	5
Bacon	1 slice	5
Sausage	2 links or 1 ounce	10
Sour Cream	2 Tablespoons	5
Heavy Whipping Cream	1 Tablespoon	5
Cream Cheese	1 Tablespoon	5
Coffee Creamer, liquid	2 Tablespoons	5
Coffee Creamer, dry	1 Tablespoon	5
Cream, Half & Half (12%)	3 Tablespoons	5
2% Milk or Whole Milk, white or chocolate	8 ounces or 1 cup	10
Ice cream	1/2 cup	10
Yogurt, plain or flavored (low fat)	8 ounces or 1 cup	5
Corn Chips	15	10

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Appendix III: Nutrition

Date: _____ Age: _____

Why would you like to see a dietitian today? _____

Height _____ Current Weight _____ Usual Weight _____

Current Medications (Please Include Vitamin, Mineral, or Herbal Supplements)

How would you describe your appetite?		Poor/Good/Very Good	Decreased/Usual/Ravenous
YES	NO	Have you experienced any recent weight loss? If so, how much? _____	
YES	NO	Have you experienced any nausea and/or vomiting recently?	
YES	NO	Do you have any trouble chewing or swallowing?	
YES	NO	Do you have dentures?	
YES	NO	Have you experienced any taste changes recently?	
YES	NO	Do you ever have the desire to eat any non-food items such as coal, dirt, clay, starch, or large amounts of ice?	
YES	NO	Do you have any food allergies or intolerances? If so please list _____	
YES	NO	Do you use alcohol?	
YES	NO	Do you follow any special diet on a regular basis? If so, please describe _____	
YES	NO	Do you use any oral nutritional drink supplements (Such as Carnation Instant Breakfast, Boost, Ensure, etc.)	
YES	NO	Do you have problems with constipation?	
YES	NO	Do you have problems with diarrhea?	

Please list below what foods you usually eat on a daily basis (For example, what do you "typically" eat for breakfast?)

BREAKFAST: _____

MORNING SNACK: _____

LUNCH: _____

AFTERNOON SNACK: _____

DINNER: _____

EVENING SNACK: _____