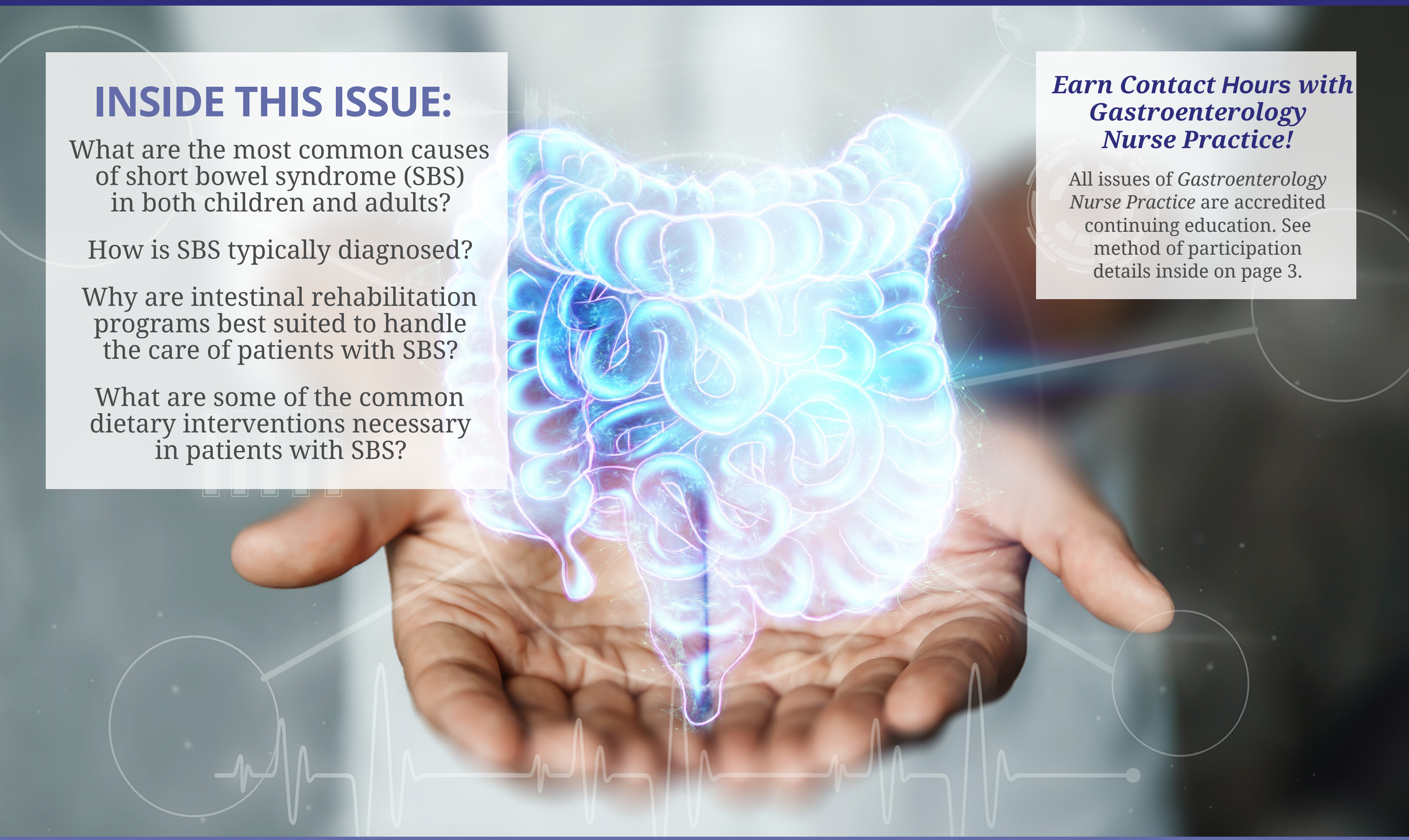
GASTROENTEROLOGY NURSE PRACTICE

DECEMBER 2022 | VOLUME 1, ISSUE 1



INSIDE THIS ISSUE:

What are the most common causes of short bowel syndrome (SBS) in both children and adults?

How is SBS typically diagnosed?

Why are intestinal rehabilitation programs best suited to handle the care of patients with SBS?

What are some of the common dietary interventions necessary in patients with SBS?

Earn Contact Hours with Gastroenterology Nurse Practice!

All issues of *Gastroenterology Nurse Practice* are accredited continuing education. See method of participation details inside on page 3.

Welcome to the first issue of *Gastroenterology Nurse Practice*!
This is the first in a series of print and online educational resources that is being developed by the Society of Gastroenterology Nurses and Associates, thanks to an unrestricted educational grant from Takeda. We hope that you find the information in this and subsequent publications of informational and educational value as you progress in your career as a gastroenterology nurse. If there are topics you'd like to learn about in the future or if you have any feedback on this education, please don't hesitate to drop us a note at info@sgna.org. We'd love to hear from you!

Educational Planning Committee:

Rosemary Pauley, APRN / Pediatric Nurse Practitioner / *Boys Town National Research Hospital, Omaha, NE*

Carol Rees Parrish, MS, RDN / GI Nutrition Support Specialist / *University of Virginia Health, Charlottesville, VA*

Kristen Roberts, PhD, RDN, LD, CNSC, FASPEN, FAND / Assistant Clinical Professor / *Division of Gastroenterology, Hepatology, and Nutrition, Department of Medical Dietetics, The Ohio State University, Columbus, OH*

Michele Rubin, MSN, APN, CNS, CGRN / Advanced Practice Nurse / *University of Chicago Medicine, Chicago, IL*

PROVIDED BY:

Society of Gastroenterology Nurses and Associates, Inc.

Support for this activity has been made possible through an educational grant from Takeda

Release date: **December 1, 2022**
Expiration date: **December 1, 2023**
Activity URL: **https://elearn.sgna.org/**

TARGET AUDIENCE

This activity has been designed to meet the educational needs of nurses, nurse practitioners, and physician assistants. Other healthcare providers may also participate.

ACTIVITY DESCRIPTION

In this issue of *Gastroenterology Nurse Practice,* we highlight the evolving understanding of the drivers of short bowel syndrome (SBS), including the unique ways the disease develops and manifests in patients of different ages, from infancy through adulthood. This issue also offers an introductory-level understanding of the basic pathophysiology of SBS. Finally, it provides an overview of the SBS diagnostic process, including common symptom combinations in patients with the condition.

LEARNING OBJECTIVES

After participating in the activity, learners should be better able to:

- List common causes and risk factors associated with the development of short bowel syndrome (SBS)
- Identify characteristics of the three anatomical types of SBS that result from surgical resection of the intestines
- Determine tools and protocols that may be used to help appropriately diagnose patients with SBS
- Discuss the role of intestinal rehabilitation programs in the management of patients with SBS

DISCLOSURE STATEMENT

According to the disclosure policy of the Society of Gastroenterology Nurses and Associates, all faculty, planning committee members, editors, managers and other individuals who are in a position to control content are required to disclose any relevant relationships with any ineligible companies related to this activity. The existence of these interests or relationships is not viewed as implying bias or decreasing the value of the presentation. All educational materials are reviewed for fair balance, scientific objectivity, and levels of evidence.

RELATIONSHIPS ARE ABBREVIATED AS FOLLOWS:

E: Educational planning committee

G: Grant/research support recipient

A: Advisor/review panel member

C: Consultant

S: Stock shareholder

SB: Speaker bureau

PE: Promotional event talks

H: Honoraria

O: Other

DISCLOSURES AS FOLLOWS:

Rosemary Pauley, APRN, has disclosed the following relevant financial relationships specific to the subject matter of the content of the activity: iHealth/C.

Carol Rees Parrish MS, RDN, has disclosed the following relevant financial relationships specific to the subject matter of the content of the activity: 9 Meters BioPharma/G,SB,C; Takeda/H,A; Napo Pharmaceuticals/A.

Kristen M. Roberts, PhD, RDN, LD, CNSC, FASPEN, FAND, has disclosed the following relevant financial relationships specific to the subject matter of the content of the activity: Elsevier Publishing/O.

Michele Rubin, MSN, APN, CNS, CGRN, has disclosed that she has no relevant financial relationships specific to the subject matter of the content of the activity.

All relevant relationships have been mitigated.

OFF-LABEL PRODUCT DISCLOSURE

This activity does not include discussion of investigational and/or off-label use of pharmaceutical products or devices.

PLANNING COMMITTEE

Cynthia M. Friis, MEd, BSN, RN, NPD-BC, Society of Gastroenterology Nurses and Associates, has disclosed that she does not have any relevant financial relationships specific to the subject matter of the content of the activity.

Scott Kober, MBA, Managing Director, Excalibur Medical Education, has disclosed that he does not have any relevant financial relationships specific to the subject matter of the content of the activity.

Kristin Harper, PhD, MPH, ELS, Medical Writer, has disclosed that she does not have any relevant financial relationships specific to the subject matter of the content of the activity.

Insights Into the Development and Presentation of Short Bowel Syndrome

Short bowel syndrome (SBS) is a rare condition, estimated to affect only 10,000 to 20,000 individuals in the United States.[1] However, it is the main cause of intestinal failure — the inability of the gut to absorb necessary water, macronutrients, micronutrients, and electrolytes sufficient to sustain life, resulting in the need for intravenous nutritional support, or parenteral nutrition (PN).[2] The consequences of SBS can be devastating for patients. Individuals with SBS are at risk for fluid and electrolyte disorders, undernutrition, and a variety of micronutrient deficiencies, not to mention various other complications such as liver failure.[3] In some cases, these complications can be life-threatening.

In the past decade, new treatments have dramatically improved prospects for patients with SBS. However, if patients are to benefit from these advances, healthcare providers must be able to recognize and respond to patients with SBS promptly and effectively.

ACCREDITATION AND CREDIT DESIGNATION

NURSES

The Society of Gastroenterology Nurses and Associates is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation (Provider No. P0166).

The Society of Gastroenterology Nurses and Associates designates this accredited continuing education for 2.0 contact hours.

METHOD OF PARTICIPATION

There are no fees to participate in the activity. Participants must review the activity information, including the learning objectives and disclosure statements, as well as the content of the activity. To receive accredited continuing education credit for your participation, please go to **https://elearn.sgna.org/products/gastroenterology-nurse-practice-short-bowel-syndrome** and complete the post-test (achieving a passing grade of 70% or greater) and program evaluation. Once the program has been successfully completed, you will be able to print your certificate.

COPYRIGHT

SBS: Definition, Causes, and Risk Factors

SBS develops when portions of the small intestine are missing or damaged. This can occur because of congenital issues, disease or trauma, or surgical removal due to conditions such as Crohn's disease, complications of hernia repair or Roux-en-Y gastric bypass surgery, cancer, traumatic injuries, intussusception, or thrombosis in the arteries that provide blood to the intestines.[4,5]

In children, SBS is currently defined as a need for PN for >60 days after intestinal resection or a bowel length <25% of expected.[6] In adults, no single definition of SBS has been agreed upon.[7] Older definitions of SBS concentrated on the length of the residual small intestine and the likelihood of developing intestinal failure. For example, through the 1980s, it was commonly believed that any patient with a residual small bowel of <100 cm would require long-term PN support, so this cutoff was used as an informal way to define SBS.[7] Today, the U.S. Centers for Medicare and Medicaid Services considers patients with a residual small intestine of less than 5 feet—or roughly 152 cm—eligible for home PN reimbursement, making this a working definition for SBS that some practitioners use.[7] However, although the length of a patient's remaining small intestine is an important contributor to intestinal failure, and having a clear cutoff for SBS is appealing, there is no one specific length of residual small intestine that can be used to predict a patient's degree of physiological dysfunction. In fact, not all patients with SBS develop intestinal failure. In light of these considerations, one group of experts arrived at the following consensus definition for SBS: "SBS results from surgical resection, congenital defect, or disease-associated loss of absorption and is characterized by the inability to maintain protein-energy, fluid, electrolyte, or micronutrient balances when on a conventionally accepted, normal diet."[8]

SBS CAUSES AND RISK FACTORS IN ADULTS

In adults, the most common cause of SBS is surgical complications **(see Table 1)**. One study found that, among patients who develop SBS after surgery, approximately 75% do so after a single, massive resection of the small intestine, whereas 25% do so after multiple resections.[9] Multiple resections may be necessary if complications such as intestinal obstruction or ischemia related to vascular injury arise during an abdominal operation.[5]

Mesenteric infarction, cancers, Crohn's disease, and radiation enteritis are other causes of SBS in adults.[5] Mesenteric infarction can result from arterial thrombosis and embolism, although drug abuse and coagulation disorders are becoming increasingly frequent causes as well.[5] Historically, SBS was often caused by surgical resection among adult and pediatric patients with Crohn's disease.[10] In recent decades, however, the average age of patients with SBS has increased as causes of SBS such as cancer or complications from surgeries unrelated to Crohn's disease have become more common. Whereas the average age of patients who develop SBS because of Crohn's disease is 41 years, it is 61 years for those with cases caused by cancer and 55 years for those with cases caused by surgeries unrelated to Crohn's disease or cancer.[10] The number of SBS cases caused

Table 1 *Most Common Causes of SBS in Adults*[5]

Cause	Relative frequency (%)
Surgical complications	29-50
Mesenteric infarction (arterial or venous thrombosis)	25-30
Neoplasms	20
Radiation enteritis	10
Crohn's disease	10-20
Abdominal trauma	5-10
Benign conditions	5

by Crohn's disease is likely decreasing because Crohn's disease is now often diagnosed earlier and therapeutic options for the condition have improved; as a result, the frequency of aggressive surgical resections due to this condition has declined.

SBS CAUSES AND RISK FACTORS IN INFANTS AND CHILDREN

SBS is more common in infants than in the general youth and adult population, affecting 25 per 100,000 live births. In newborns, SBS can result from a variety of causes **(see Table 2)**.[6,11] Some infants develop SBS because they are born with an abnormally short small intestine or with part of their bowel missing. However, it is more common for SBS to occur following surgery to treat necrotizing enterocolitis (NEC).[12] In fact, roughly 30% of SBS cases in children are caused by complications related to surgical treatment for NEC, and the number of patients with SBS secondary to NEC is increasing in tandem with the number of extremely premature infants who are able to survive thanks to medical advances.[13] Other common causes of SBS in children include gastroschisis (the presence of a hole outside the abdominal wall other than the belly button), intestinal atresia (a complete blockage or obstruction of the intestines), and intestinal malrotation/volvulus (i.e., twisting).[11,14]

Preterm infants are especially vulnerable to SBS. In one large U.S. study, the incidence of surgery-related SBS was 70 per 100,000 among very low birth weight infants (those weighing <1500 g at birth) and 110 per 100,000 among extremely low birth weight infants (<1000 g at birth).[13] Among these infants, NEC was responsible for 96% of cases. However, preterm infants are at risk for SBS from all common causes of the condition among newborns, primarily because they are born before their small intestines have had a chance to fully mature. Because the small intestine lengthens dramatically during the last trimester of gestation, a baby born full-term has a small intestine ~250 cm in length while a baby born at the end of the second trimester has one only ~120 cm in length.[15] Less intestinal length and area mean less absorption of nutrients and less capacity to function adequately, especially if removal of part of the small intestine becomes necessary.[6] Preterm infants with comorbidities, such as cardiac or pulmonary disease, are at particularly high risk of developing SBS.[16] Just as in adults, children can also develop SBS from trauma, complications from abdominal surgery, inflammatory bowel disease, and cancer.

Table 2 *Most Common Causes of SBS in Infants and Children*[11]

Infants:

- Necrotizing enterocolitis
- Intestinal atresia
- Gastroschisis
- Midgut volvulus
- Congenital

Children:

- Trauma
- Postoperative complication
- Cancer
- Motility disorders

SBS Diagnosis, Manifestations, and Complications

It is critical for providers to diagnose patients with SBS as soon as possible and link them to effective treatment. The consequences of a delayed or missed diagnosis can be considerable. For example, a patient with SBS who is discharged after surgery without a diagnosis may soon appear in the emergency room to be treated for dehydration. Identifying patients who need support before they go home from the hospital is the best way to optimize outcomes. When assessing patients prior to discharge, it is important to keep in mind that they may be eating meals and/or taking medications without absorbing them. Furthermore, many patients who receive IV fluids until the point of discharge appear well hydrated. However, when IV fluids are removed, some of these patients cannot maintain hydration. For this reason, running appropriate tests and mimicking the home plan prior to discharge is critical.

Healthcare providers should be alert for SBS in any patient who has undergone significant intestinal resection, experienced trauma to the intestine, or been born without parts of their intestine. Diagnosis typically starts by determining how much small intestine remains. For patients who have undergone resection, providers can often find information about the remaining length of the bowel in surgical reports, although this is not always available (see the essay entitled "Gut Intelligence: Length Matters" later in this issue for more on this topic).

The most common symptom of SBS is diarrhea.[12] Other signs and symptoms include greasy, foul-smelling stools; cramps; fatigue; weight loss; and swelling (edema) of the lower extremities.[4,12] In pediatric patients, signs and symptoms can also include failure to thrive, motility disturbances, bone density issues or fractures, and skin breakdown, especially in the diaper area.[17]

Assessment of the amount of remaining small intestine and patient symptoms can be supplemented with additional information, such as a patient's medical and family history.[9] A provider can also perform a physical exam to look for weight loss, muscle wasting, and signs of vitamin and mineral deficiencies. During the exam, the clinician can use a stethoscope to auscultate the patient's abdomen and tap different parts of the patient's body to look for bloating. Blood tests can be ordered to measure vitamin and mineral levels, as well as electrolyte and creatinine levels. A complete blood count should also be performed, with special attention to ruling out anemia and macrocytosis.[18] Fecal fat testing should be performed to determine how well the small intestine is working.[12]

Imaging can also be used to aid assessment. For example, an abdominal CAT scan can help providers identify problems such as bowel obstruction as well as assess liver health.[18] An abdominal ultrasound can be used to detect biliary sludge or gallstones, both of which are often present in patients with SBS. An upper gastrointestinal X-ray series can help providers identify strictures or fistulas, and assess the health of the small intestine's lining. It should be noted that radiographic procedures are used sparingly in pediatric patients due to the potential cumulative effects of radiation.

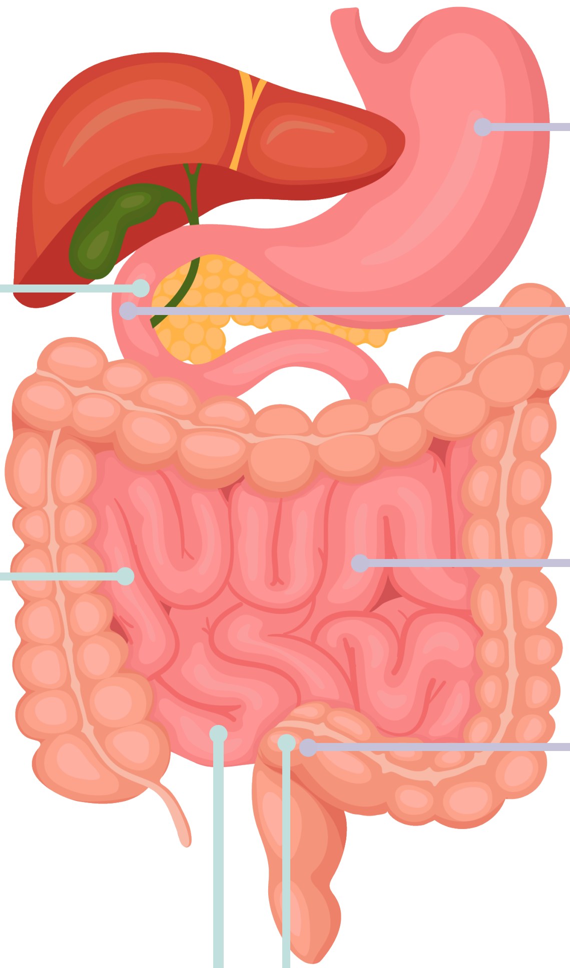
When diagnosing and treating a patient with SBS, healthcare providers should be alert for complications stemming from impaired intestinal function. These complications range from malabsorption of a single micronutrient to complete intestinal failure.[5] Other possible complications include gastric acid hypersecretion (present in 50% of patients), kidney stones, gallstones, small intestinal bacterial overgrowth (SIBO; present in 60% of patients), and liver disease.[3,12,14] SIBO, in particular, exacerbates malabsorption in the gut, as the invading bacteria consume nutrients—particularly vitamin B12—before they are able to be absorbed by the patient.[5] The bacteria also denature pancreatic enzymes and deconjugate bile acids, resulting in further lipid malabsorption.

Complications related to reliance on PN are also common in patients with SBS. These complications include conditions linked to central venous access such as infections, venous thrombosis, and loss of venous access.[3] Metabolic bone disease is also common among individuals who have depended on PN for lengthy periods. Finally, intestinal failure-associated liver disease (IFALD) is caused by long-term exposure to multiple components of PN, including the protein, fat, and micronutrients such as copper that it contains. In particular, it is critical that any infections are identified and treated before sepsis arises in patients who rely on PN.

IFALD occurs in 40-60% of infants and 20-75% of adults who require long-term PN.[14,19] A number of factors associated with greater dependence on PN also increase the risk of developing IFALD, including massive resection, gestational age (in pediatric patients), and lack of enteral (ie, feeding tube, or non-PN) feedings.[16] Recent research from Italy indicates that replacing soybean oil–based lipid emulsion therapy with fish oil–based lipid emulsion therapy can halt and even reverse IFALD in pediatric and adult patients, although it should be noted that fish oil–based liquid emulsion is not commonly available in the United States.[20] Purely soybean oil–based emulsions can also be replaced with composite lipid emulsions that rely on a variety of sources, such as soybean, fish, coconut, and olive oil; such composite emulsions are generally recommended for long-term use as they prevent essential fatty acid deficiency and, in pediatric patients, adverse effects on growth and development. Although combined liver/intestinal transplants may be necessary for some individuals with IFALD, thanks to newer and better therapies, the number of SBS patients who need this procedure is decreasing. For example, among 135 children with SBS who were treated for IFALD at the Intestinal Rehabilitation Program of Children's National Hospital, in Washington DC, between 2007-2018, 97% achieved transplant-free survival and 81% achieved enteral autonomy.[21]

It should be noted that complications of SBS are especially debilitating for infants and children.[6] In addition to the complications already described, SBS can affect growth and development more generally in young patients. Infants with SBS are at high risk for poor growth during the first 2 years of life.[22] In addition, in one study of preterm infants with SBS caused by NEC or spontaneous intestinal perforation, an increased risk of adverse developmental outcomes at 18-26 months (with age adjusted for prematurity) relative to preterm infants with NEC or spontaneous intestinal perforation who did not develop SBS was also documented.[23] Therefore, SBS itself, and not just the harmful conditions that often accompany it, poses a threat to healthy growth and development. Healthcare providers should be aware that in infants with SBS, feeding disorders often develop later in life due to the lack of oral stimulation at developmentally appropriate ages. For example, in one study of 28 patients diagnosed with SBS at age 6 months or younger, the prevalence of pediatric feeding disorder was 100% at age 1 year and 70% at age 4 years.[24] Among these patients, 21 were weaned off of PN by 48 months and 13 were weaned off enteral nutrition. Collectively, these possible complications in infants and children emphasize the importance of recognizing SBS early and linking young patients to effective treatment.

Figure 1 *(A) Absorption of Nutrients and (B) Release of Hormones in Different Sections of the Intestines[25]*

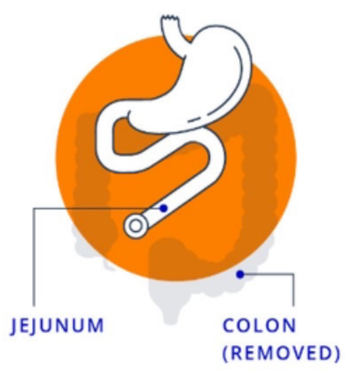
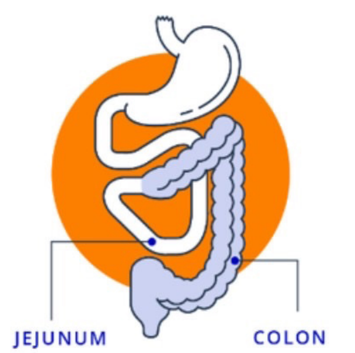
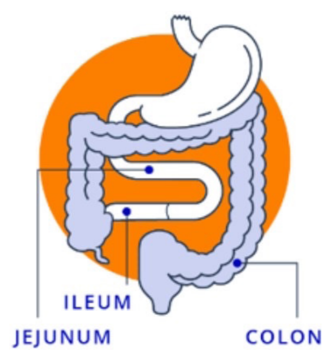


Pathophysiology of SBS

Understanding the pathophysiology of SBS may be helpful for comprehending not only the way in which the condition manifests, but also how it can be treated. The small intestine is the part of the gastrointestinal tract responsible for most digestion and nutrient absorption.[6,12] Every day, roughly 9 liters of nutrient-filled fluid passes through the small intestine, which is approximately 20 feet long in the average adult.[9] Carbohydrates, lipids, and proteins can be absorbed throughout the small intestine. However, other types of nutrients can only be absorbed in dedicated sections of the small intestine (see Figure 1).[25] The duodenum, the first section of the small intestine, is where iron and other minerals are absorbed. The jejunum, the middle section, is where carbohydrates, proteins, fat, and most vitamins are absorbed. And the ileum, the last section of the small intestine, is where bile acids and vitamin B12 are absorbed. Lactose digestion, which is especially important for infants, occurs primarily in the jejunum and proximal ileum.[6] The colon, which may also be affected in patients with SBS, is approximately 5 feet long in adults; water, electrolytes, and short-chain fatty acids are absorbed here. Colonic bacteria produce vitamin K and B vitamins that are also absorbed in the large intestine.[26,27]

In adults, three different types of SBS have been identified based on the patient's underlying physiology (see Figure 2).[3,28] Patients with an **end jejunostomy** lack an ileum and colon, and possess only a remnant of the jejunum. These patients are the most likely to develop intestinal failure; their risk of doing so is considered significant if <115 cm of their small intestine remains.[3]

Figure 2 *Characteristics of the Three Anatomical Types of SBS that Result from Surgical Resection of the Intestines*[1,5,25]

	End-jejunostomy	Jejunocolonic Anastomosis	Jujunoileal Anastomosis
			
Surgical procedure	Complete resection of ileum and colon. Jejunum preserved.	Resection of most of the ileum. Some colon preserved.	Resection of jejunum. At least 10 cm of terminal ileum and colon preserved
Ileocecal valve present?	No	No	Yes
Colon preserved?	No	Partially	Yes
Clinical features	• Dehydration immediately post-surgery with risk of electrolyte imbalance • Jejunal output increases after food and drink intake • Significant nutrient and fluid malabsorption • Vitamin B12 and magnesium deficiences • Impaired bile salt resorption	• Weight loss • Diarrhea/steatorrhea • Impaired bile salt resorption • Deficiences in fat-soluble vitamins • Fat malabsorption • Steatorhhea • Severe malnutrition within months of surgery	• Transient gastric acid hypersecretion • Malnutrition rare • Usually no need for parenteral nutrition

This type of SBS is observed most commonly in clinical practice. In one study of 152 patients receiving PN for SBS in the United Kingdom, 75% had an end jejunostomy.[29] In patients with a **jejunocolonic anastomosis**, the jejunum is joined to the colon. These individuals lack an ileum and sometimes part of the colon. They are at significant risk for developing intestinal failure if <60 cm of their small intestine remains.[3] Patients with a jejunoileal anastomosis retain their entire colon and ileocecal valve, giving them the best chance at avoiding intestinal failure.[5,25] These patients are at significant risk for developing intestinal failure if <35 cm of their small intestine remains.[3]

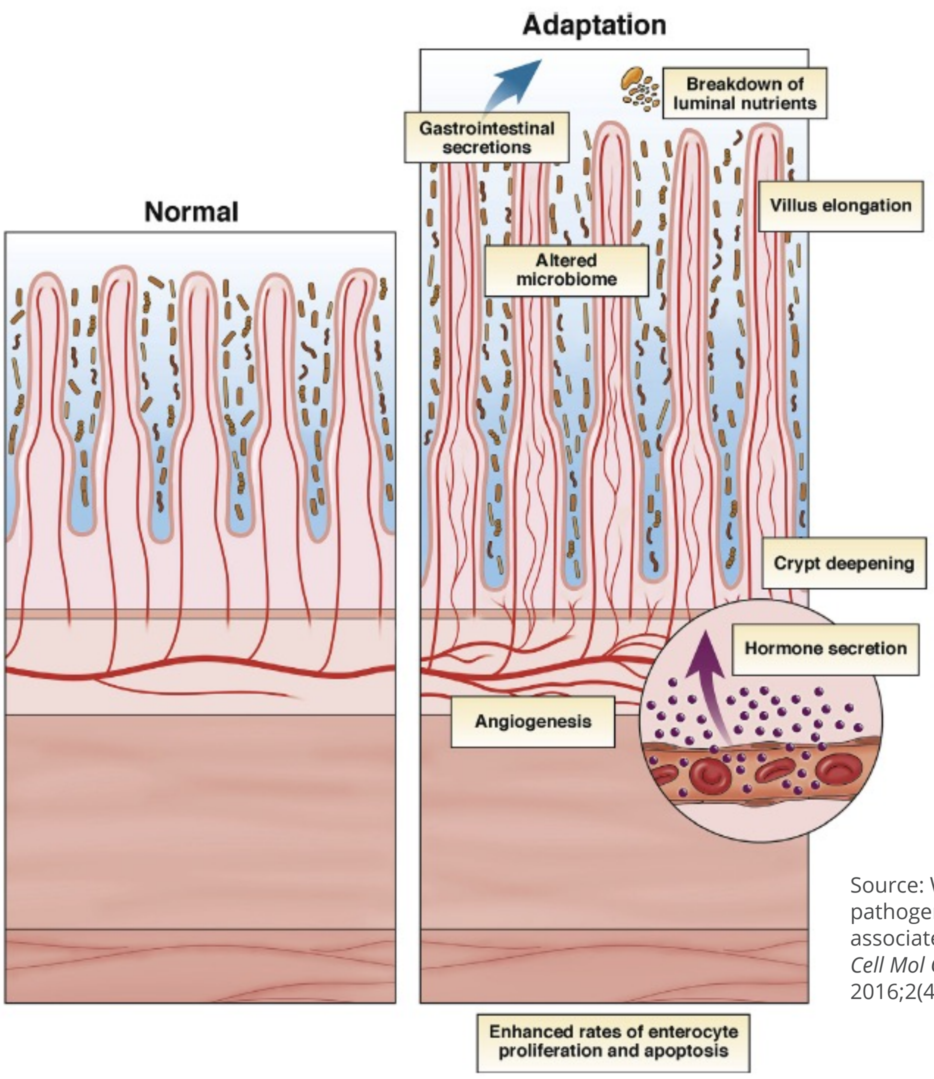
Progression of SBS Over Time

Ideally, patients with SBS move as swiftly as possible from an acute phase to a recovery phase as their remaining small intestine adapts to its new state and regains function. Next, they progress to a longer-term maintenance phase. Here, we briefly discuss each phase in this progression, including the physiologic process of intestinal adaptation.

ACUTE PHASE

Patients with SBS typically experience an acute phase that starts roughly 1 week after intestinal surgery (or damage) and can last for up to 3 weeks.[14] This phase is characterized by fluid and electrolyte loss, necessitating IV fluids and PN. H2 blockers or proton pump inhibitors may be necessary to combat gastric hypersecretion, which can last up to 6-12 months. By identifying patients at risk for intestinal failure in advance, based on the magnitude and type of their intestinal loss as well as other factors, healthcare providers can be prepared to offer the care needed in this critical phase.

Figure 3 *Characteristics of Intestinal Adaptation Following Resection*[32]



Source: Warner BW. The pathogenesis of resection-associated intestinal adaptation. *Cell Mol Gastroenterol Hepatol*. 2016;2(4):429-438.

RECOVERY PHASE

The recovery phase of SBS, which begins when the acute phase ends, can continue for years, though most adaptation tends to occur in the first 2 years post-surgery.[14] During the recovery phase, a patient's diarrhea gradually improves, and their dependence on PN may decrease. Enteral nutrition and eating and drinking by mouth are often carefully introduced, with the goal of weaning patients off of PN. Of note, in one study that followed more than 250 adult patients with SBS who did not have malignancies, 27% achieved PN independence after 2 years.[30] In fact, patients continued to achieve PN independence up to 5 years post-surgery, albeit at a low rate. Therefore, providers should provide continued support to their SBS patients throughout the recovery phase, the length of which will vary from patient to patient.

MAINTENANCE PHASE

In the maintenance phase, many pediatric patients are able to discontinue PN, and enteral nutrition may no longer be necessary.[14] However, some patients—especially adults—will continue to need nutritional support. Roughly 50% of adult patients with prolonged acute intestinal failure develop chronic intestinal failure; though they are metabolically stable, they continue to require PN.[5] With proper support, a patient with SBS will ideally remain in the maintenance phase for the rest of their life.

Successful progression through each of these three phases is driven by physiologic adaptation, which starts as soon as 48 hours after surgery and is often complete within 2 years.[14] Within the small intestine, the ileum appears to possess greater adaptive potential than the jejunum.[31] Thus, patients with an intact ileum tend to fare better than those without one. In addition, successful adaptation is especially likely in patients receiving luminal nutrients (oral or enteral) who retain a colon segment in continuity with their small intestine; in these patients, the colon takes over some of the small intestine's functions.[3]

A variety of physiologic processes are responsible for intestinal adaptation. First, the depth of a patient's colonic crypts increases and crypt cell hyperplasia occurs **(see Figure 3)**.[32] These changes increase the absorptive surface area of the remaining intestine, improving a patient's nutritional status and electrolyte balance. Hormonal adaptation also takes place. Gastrointestinal hormones such as gastrin, ghrelin, peptide YY, and glucagon-like peptides-1 and 2 (GLP-1, GLP-2) play a key role in regulating intestinal absorption, gastric emptying, and food intake. After surgery resulting in SBS, rising GLP-2 levels increase the absorptive surface of the gut, while rising peptide YY and GLP-1 levels slow gastrointestinal transit time to increase nutrient absorption. Many patients also spontaneously begin to eat more, which aids in nutrient absorption.

SBS Prognosis

The aim of SBS care is to maximize patients' health and quality of life, with the ultimate goal of helping them achieve PN independence in those able to wean from PN through intestinal rehabilitation. In general, it has been found that adult patients with >180 cm of small intestine generally do not require PN.[9] Those with >90 cm often require PN for at least 1 year. Those with <60 cm of small intestine are likely to require PN for their entire lives.

However, as mentioned previously, factors other than small intestine length shape a patient's odds of achieving PN independence. For example, patients with an end jejunostomy are much less likely to achieve PN independence than patients with a jejunoileal anastomosis.[3] Research has shown that important factors in determining the degree of intestinal dysfunction immediately after bowel resection include the following:[33]

- Length of the remaining intestine, relative to age or body size
- Loss of the ileum and ileocecal valve
- Loss of all or part of the colon
- Patient age
- Continuity of the intestines

Typically, the greater the degree of intestinal dysfunction, the more challenging intestinal rehabilitation will be.

In adults, approximately 50% of patients with SBS achieve reversal of intestinal failure (ie, significant intestinal adaptation) within 2 years.[33] In children, the numbers appear to be similar. In a study of 272 children with SBS who received high quality care from one of the 14 programs that make up the Pediatric Intestinal Failure Consortium, 47% achieved intestinal adaptation.[16] It should be noted, however, that this figure is from a study in the early 2000s, and there is some evidence that outcomes have improved since then.

Intestinal Rehabilitation Programs

Intestinal rehabilitation programs, or IRPs, are multidisciplinary groups of healthcare providers that exist solely to deliver high-quality, evidence-based care to patients with SBS. They exist in major cities throughout the United States **(see Table 3)**.[34]

As soon as possible after an SBS diagnosis, patients should be referred to an IRP.[35] Because these programs are experienced with SBS, they have consistent and effective protocols and guidelines in place, are more aggressive about promoting intestinal rehabilitation, and are more likely to be able to anticipate, detect, and manage potential SBS complications.[13] In addition, patients are able to receive seamless, integrated management at a single location. For these reasons, SBS patients who receive care from IRPs experience superior outcomes compared to those who do not.[36]

IRPs typically include gastroenterologists, surgeons, nurses, nurse specialists, dietitians, social workers, pharmacists, and psychologists.[13,15,16] IRPs that care for infants with SBS typically also include neonatologists and specialists with expertise in pediatrics. For patients, having a nurse to coordinate their day-to-day management is essential for optimizing care and outcomes.[9]

For children with SBS, one critical task that the multidisciplinary care team must play is helping them develop healthy eating behaviors. Children with SBS

Table 3 *Intestinal Rehabilitation and Transplant Programs in the United States*[34]

PEDIATRIC PROGRAMS

State	Program
Alabama	Children's of Alabama Intestinal Rehabilitation Program
California	Children's Hospital of Los Angeles Intestinal Rehabilitation Program
	Children's Hospital of Orange County Intestinal Rehabilitation Program
	Rady Children's Hospital San Diego Intestinal Rehabilitation Program
	UCSF Benioff Children's Intestinal Rehabilitation Program
Colorado	Children's Hospital Colorado Intestinal Rehabilitation Program
District of Columbia	Children's National Intestinal Rehabilitation Program
Florida	Broward Health Intestinal Rehabilitation Program
Indiana	Riley Children's Health Intestinal Rehabilitation Program
Maryland	All Children's Hospital Johns Hopkins Intestinal Rehabilitation Program
Michigan	Mott Children's Hospital Intestinal Rehabilitation Program
Minnesota	Masonic Children's Hospital Intestinal Rehabilitation Program
Missouri	Children's Mercy Kansas City
	SSM Health Cardinal Glennon Intestinal Rehabilitation Program
New York	Columbia Intestinal Rehabilitation Program
	John R. Oishei Children's Hospital Intestinal Rehabilitation Program
Ohio	Nationwide Children's Intestinal Rehabilitation Center
Pennsylvania	Children's Hospital of Philadelphia Intestinal Rehabilitation Program
Tennessee	Monroe Carell Jr. Children's Hospital Intestinal Rehabilitation Program
Texas	Children's Health North Texas Intestinal Rehabilitation Program
	Texas Children's Intestinal Rehabilitation Clinic

ADULT AND PEDIATRIC PROGRAMS

State	Program
California	University of California Los Angeles (Ronald Reagan UCLA Medical Center)
	Lucile Salter Packard Children's Hospital at Stanford
District of Columbia	Medstar-Georgetown University Hospital
Florida	Jackson Memorial Hospital Miami
Illinois	University of Illinois Hospital
	Ann & Robert H. Lurie Children's Hospital of Chicago
Indiana	Indiana University Hospital
Massachusetts	Boston Children's Hospital
Michigan	Henry Ford Hospital
Nebraska	Nebraska Medical Center
New York	Mount Sinai Healthcare
	New York Presbyterian Hospital/ Columbia University Medical Center
North Carolina	Duke University Hospital
Ohio	Cleveland Clinic
	Cincinnati Children's Intestinal Care Center
Pennsylvania	University of Pittsburgh Medical Center (UPMC)
	University of Pittsburgh Medical Center Children's Hospital of Pittsburgh
Washington	Seattle Children's Hospital

who use PN often develop oral aversions or eating disorders.[15] This is particularly true of infants who are not introduced to oral feeding within the first days, weeks, or even months of their lives. Children who rely on PN for nutrition may also display oral-motor, sensory, and developmental problems that make the transition to oral feeding challenging.

For adults and children with SBS, psychosocial care and links to resources such as caregiving assistance are also essential. Research shows that quality of life suffers significantly among children with SBS as well as their families.[35] Young patients often suffer from body image issues, restrictions on their daily lives, academic difficulties, and social challenges. For example, children who depend on PN may not be able to enjoy evening activities with friends or attend sleepovers. Their families may not be able to travel to see relatives or go on vacations, and their parents may have difficulty sleeping because they are attending to PN. Adults with SBS must deal with similar challenges. Therefore, finding ways to provide patients with non-medical support is an important aspect of SBS care.

Currently, IRPs are more common for pediatric patients than adults, although many programs provide care to patients of all ages.[13,36] For patients who are unable to receive care from an established IRP, whether because of location or other restrictions, it may be possible for their providers to consult with an IRP to ensure that they receive the best possible care. Some patients with limited access to IRPs also can make use of these specialty programs when a critical problem arises, such as IFALD or PN failure, or schedule an annual check-up to guide their care at home. Whatever a patient's situation, some contact with a multidisciplinary IRP team is beneficial.

Talking With Patients About SBS

A diagnosis of SBS, whether it is for a patient, a son/daughter, or another loved one, is frightening. Patients or their caregivers may be reassured to hear that not all individuals with SBS develop intestinal failure, and of those who do, many eventually achieve independence from PN. In one study of 268 adult patients with SBS who did not have progressive neoplastic disease, the prevalence of PN dependence fell from 74% at 1 year to 64% at 2 years and 48% at 5 years.[30] In recent studies of pediatric patients, 42-84% have eventually achieved PN independence (the wide range is likely because of the varying lengths of the studies, which ranged from 12 to 50 months).[13] Even among patients with SBS who do not achieve full PN independence, the proportion of calories derived from PN often decreases over time.

It is also important to give patients with SBS a realistic idea of the path ahead following their diagnosis. This path often includes years of intestinal rehabilitation. Moreover, home-based PN is time-consuming (it is typically administered overnight over a period of 10-12 hours) and intrusive.[35] Thus, support and education should begin when a patient is hospitalized and continue through the transition to home care.[13] Many patients and caregivers struggle with the strain that PN puts on finances, relationships, and the patient's ability to function on a day-to-day basis. Given these challenges, emphasizing the importance of psychosocial care may be helpful. It may also be useful to provide patients and caregivers with a list of resources and groups where they can find peer support. For example, the Short Bowel Syndrome Community maintains a Facebook group where patients and their families can connect.

Finally, providers can make a big difference in their patients and caregivers' experience at diagnosis by simply listening to them and answering their questions. Cultivating a human connection during this difficult time can feel healing for those affected and help pave the way for a relationship of mutual trust and respect.

Conclusion

SBS is a serious condition that can affect virtually every facet of a patient's life. Fortunately, the outlook for patients with SBS is better than ever. Particularly in preterm infants, it is important that healthcare providers are able to quickly diagnose and treat SBS. With early intervention and proper support, many patients will be able to achieve PN independence—or at least minimize their use of PN—and enjoy a good quality of life. Continued improvement in SBS outcomes is gratifying for both patients and the clinicians who care for them.

References

1. Strengthen your SBS knowledge. Available at www.shortbowelsyndrome.com/what-is-sbs#Definition. Accessed August 16, 2022.

2. Allan P, Lal S. Intestinal failure: a review. *F1000Res*. 2018;7:85.

3. Billiauws L, Maggiori L, Joly F, Panis Y. Medical and surgical management of short bowel syndrome. *J Visc Surg.* 2018;155(4):283-291.

4. Mayo Clinic. Short bowel symdrome. Available at www.mayoclinic.org/diseases-conditions/short-bowel-syndrome/symptoms-causes/syc-20355091#:~:text=Short%20bowel%20syndrome%20is%20a,into%20your%20body%20during%20digestion. Accessed August 16, 2022.

5. Massironi S, Cavalcoli F, Rausa E, Invernizzi P, Braga M, Vecchi M. Understanding short bowel syndrome: Current status and future perspectives. *Dig Liver Dis.* 2020;52(3):253-261.

6. Chandra R, Kesavan A. Current treatment paradigms in pediatric short bowel syndrome. *Clin J Gastroenterol.* 2018;11(2):103-112.

7. Kelly DG, Tappenden KA, Winkler MF. Short bowel syndrome: highlights of patient management, quality of life, and survival. *J Parenter Enteral Nutr.* 2014;38(4):427-437.

8. O'Keefe SJ, Buchman AL, Fishbein TM, Jeejeebhoy KN, Jeppesen PB, Shaffer J. Short bowel syndrome and intestinal failure: consensus definitions and overview. *Clin Gastroenterol Hepatol.* 2006;4(1):6-10.

9. Guillen B, Atherton N. Short bowel syndrome. StatPearls. Available at www.ncbi.nlm.nih.gov/books/NBK536935/. Accessed August 16, 2022.

10. Brandt CF, Hvistendahl M, Naimi RM, et al. Home parenteral nutrition in adult patients with chronic intestinal failure: the evolution over 4 decades in a tertiary referral center. *J Parenter Enteral Nutr.* 2017;41(7):1178-1187.

11. Thompson JS. Short bowel syndrome and malabsorption - causes and prevention. *Viszeralmedizin*. 2014;30(3):174-178.

12. National Institute of Diabetes and Digestive and Kidney Diseases. Short bowel syndrome. Available at www.niddk.nih.gov/health-information/digestive-diseases/short-bowel-syndrome. Accessed August 16, 2022.

13. Mutanen A, Wales PW. Etiology and prognosis of pediatric short bowel syndrome. *Semin Pediatr Surg*. 2018;27(4):209-217.

14. Amin SC, Pappas C, Iyengar H, Maheshwari A. Short bowel syndrome in the NICU. *Clin Perinatol.* 2013;40(1):53-68.

15. Goulet O, Abi Nader E, Pigneur B, Lambe C. Short bowel syndrome as the leading cause of intestinal failure in early life: some insights into the management. *Pediatr Gastroenterol Hepatol Nutr.* 2019;22(4):303-329.

16. Cohran VC, Prozialeck JD, Cole CR. Redefining short bowel syndrome in the 21st century. *Pediatr Res.* 2017;81(4):540-549.

17. Short bowel syndrome in children. Available at www.shortbowelsyndrome.com/sbs-in-children. Accessed August 16, 2022.

18. National Organization for Rare Disorders. Short bowel syndrome. Rare Disease Database. Available at rarediseases.org/rare-diseases/short-bowel-syndrome/. Accessed August 16, 2022.

19. Huijbers A, Wanten GJA. Hepatobiliary complications of intestinal failure and home parenteral nutrition. In: *Corrigan ML, Roberts K, Steiger E, eds. Adult Short Bowel Syndrome*. Academic Press; 2019:129-145.

20. Rochling FA. Intravenous lipid emulsions in the prevention and treatment of liver disease in intestinal failure. *Nutrients.* 2021;13(3).

21. Torres C, Badalyan V, Mohan P. Twelve-year outcomes of intestinal failure-associated liver disease in children with short-bowel syndrome: 97% transplant-free survival and 81% enteral autonomy. *J Parenter Enteral Nutr.* 2022;46(1):197-206.

22. McLaughlin CM, Channabasappa N, Pace J, Nguyen H, Piper HG. Growth trajectory in children with short bowel syndrome during the first 2 years of life. *J Pediatr Gastroenterol Nutr.* 2018;66(3):484-488.

23. Bell M, Cole CR, Hansen NI, Duncan AF, Hintz SR, Adams-Chapman I. Neurodevelopmental and growth outcomes of extremely preterm infants with short bowel syndrome. *J Pediatr.* 2021;230:76-83.e75.

24. Christian VJ, Van Hoorn M, Walia CLS, Silverman A, Goday PS. Pediatric feeding disorder in children with short bowel syndrome. *J Pediatr Gastroenterol Nutr.* 2021;72(3):442-445.

25. Tappenden KA. Pathophysiology of short bowel syndrome: considerations of resected and residual anatomy. *J Parenter Enteral Nutr.* 2014;38(1 Suppl):14s-22s.

26. Azzouz L, Sharma S. Physiology, large intestine. StatPearls. Available at www.ncbi.nlm.nih.gov/books/NBK507857/. Accessed September 23, 2022.

27. von Engelhardt W, Rönnau K, Rechkemmer G, Sakata T. Absorption of short-chain fatty acids and their role in the hindgut of monogastric animals. *Anim Feed Science Technol.* 1989;23:43-53.

28. Billiauws L, Joly F. Emerging treatments for short bowel syndrome in adult patients. *Expert Rev Gastroenterol Hepatol.* 2019;13(3):241-246.

29. Bond A, Taylor M, Abraham A, et al. Examining the pathophysiology of short bowel syndrome and glucagon-like peptide 2 analogue suitability in chronic intestinal failure: experience from a national intestinal failure unit. *Eur J Clin Nutr.* 2019;73(5):751-756.

30. Amiot A, Messing B, Corcos O, Panis Y, Joly F. Determinants of home parenteral nutrition dependence and survival of 268 patients with non-malignant short bowel syndrome. *Clin Nutr.* 2013;32(3):368-374.

31. Kong W, Wang J, Ying R, et al. A potential anatomic subtype of short bowel syndrome: a matched case-control study. *BMC Gastroenterol.* 2016;16:12.

32. Warner BW. The pathogenesis of resection-associated intestinal adaptation. *Cell Mol Gastroenterol Hepatol.* 2016;2(4):429-438.

33. DiBaise J. Management of the short bowel syndrome in adults. UpToDate. Available at www.uptodate.com/contents/management-of-the-short-bowel-syndrome-in-adults. Accessed August 16, 2022.

34. Intestinal rehabilitation and transplantation centers. Available at www.transplantunwrapped.org/centers. Accessed August 30, 2022.

35. Pederiva F, Khalil B, Morabito A, Wood SJ. Impact of short bowel syndrome on quality of life and family: the patient's perspective. *Eur J Pediatr Surg.* 2019;29(2):196-202.

36. Merritt RJ, Cohran V, Raphael BP, et al. Intestinal rehabilitation programs in the management of pediatric intestinal failure and short bowel syndrome. *J Pediatr Gastroenterol Nutr.* 2017;65(5):588-596.

AUTHOR PROFILE:

Carol Rees Parrish, MS, RDN, is a GI nutrition support specialist at University of Virginia Health in Charlottesville, VA

Gut Intelligence: *Length Matters*

When someone has lung surgery, there is typically a detailed report that includes what part of the lung was operated on, how much of the affected lobe was removed, how much healthy lung remains, and other important information. One might expect a similar protocol in other surgical situations where key organs are removed, but that's not always the case. For example, in patients who have significant sections of their intestinal tract removed, a common post-op report will say, "*Removed some small bowel.*" That's it.

Since there are very different sections of the gastrointestinal (GI) tract, each with very specific functions, architecture, and adaptation potential, one can imagine how hard this truncated post-op report makes it for specialists who now have to develop a treatment plan to bring back the functions of those sections that have been removed.[1,2] It is nearly impossible to best serve our patients without knowing what sections were removed and being well educated about the role of those (and surviving) sections.

For example, because the terminal ileum is responsible for food-bound B^{12} absorption, if that section is surgically removed, the patient will require intramuscular B^{12} for the remainder of their life. The terminal ileum is also responsible for the recycling of 95% of our bile salts by way of enterohepatic circulation, which also must be accounted for during treatment planning, as if 100 cm (3 feet) or more of the terminal ileum has been removed, bile salts cannot be reabsorbed and will be lost in the stool. This creates further fat malabsorption, and in those patients with remaining colon, bile salt malabsorption (BAM) can occur. BAM is the process by which excess bile salts enter the colon — a place they are not supposed to be — and cause significant irritation to the colonic mucosa. This then causes the mucosa to self-secrete fluid into the colon lumen in an effort to protect itself. Water absorption is also hindered, all of which causes propulsive contractions that shorten colonic transit time. The patient then experiences all of the miserable symptoms associated with BAM: watery diarrhea, bloating, fecal urgency, and fecal incontinence. That doesn't make for a happy individual.

GI clinicians must also use good common sense in some instances when the information they receive from other specialists appears incomplete or suspicious. As many of us know, the three main sections of small bowel are the duodenum (~25 cm [10 inches] long), the

FREE RESOURCES FOR MORE INFORMATION

*For both patients **and** clinicians:*
Parrish CR. The adult patients guide to managing short bowel syndrome, 5th edition, 2021. Available at www.shortbowelsyndrome.com/sign-up. Accessed August 8, 2022.

For clinicians only:
SBSCurbside.org -- website where clinicians can ask questions and get help with their SBS patients. Also includes many useful SBS resource materials.

jejunum (~240 cm [8 feet] long), and the ileum (~335cm [11 feet] long). The colon checks in at ~152 cm (5 feet) long.

Last week, I was contacted to consult on a patient with what was being called an "end ileostomy" for high stool output. Throughout this patient's chart, that was the term being used – "end ileostomy." Following surgery, the patient's ostomy output was >4 liters over 24 hours. Following the usual interventions with focused medication adjustments and diet alterations, the patient did not respond as expected. I emailed the surgeon to verify the length of the remaining small bowel, and she responded that there was "*120 cm of proximal (small bowel) left.*" This set off warning bells on my end as I typed my response: "*Then she cannot have an end ileostomy. If only 120 cm of proximal small bowel is left and the normal jejununal (which precedes the ileum) length is approximately 240 cm, she can only have jejunum left. Hence, we are dealing with an end jejunostomy, and short bowel syndrome (SBS) to boot, which explains why her ostomy output is so high still!*" My conclusions were not based on any sort of clinical superpower but rather basic math and knowledge of the anatomy of each SB segment. I added some further information about the clinical definition of SBS to explain my thought process, which the surgeon said she appreciated (she also agreed that the patient would need long-term total parenteral nutrition). Sadly, this was far from the first time that vague post-operative reports have caused unnecessary confusion, delay in treatment, and potential patient harm.

Another important note: it is crucial to recognize when a bowel is too short to absorb enough and sustain a patient's nutrition and hydration, meaning the patient will need total parenteral nutrition (TPN), at least for a very long time and sometimes for life. That means that, no matter how many septic episodes a patient has on TPN, or how many times the clinician (or patient) wants them to "just try eating on their own," it will not go well. It would be like taking a patient with end-stage renal disease off hemodialysis and hoping their kidney function will magically start working. This is the kind of thing that lawsuits are made from, so be vigilant!

Anyone taking care of a patient with SBS should know that the following anatomies will require TPN for life:[3]

1. End jejunostomy or ileostomy with <115 cm functional SB
2. Jejuno-colonic anastomosis with <60 cm functional SB [no ileocecal valve]
3. Jejuno-ileo-colonic anastomosis with <35 cm functional SB [ileocecal valve/colon intact]

These are not exclusive criteria, as those with longer lengths/sections than the above criteria that have impaired working bowel (i.e., due to radiation, active Crohn's disease, etc.) may also require lengthy or lifelong TPN.

So can how can providers determine true bowel lengths if operative reports are unreliable? One option is an abdominal CT scan. A SB follow through provides a radiologic image of the small intestine that can also be useful to help provide a gross estimate of SB length as well as transit time (how fast food or fluid moves through the intestinal tract).

I suggest having regular discussions with surgeons that you work with about the importance of recording what segment(s) were removed and, even more importantly, which sections and how much of the SB remains. The same is true for the colon—did they remove the right, transverse, or left colon? Hopefully, with enough reminders, something more useful in the medical record than "*removed some small bowel*" will become a more routine part of their practice.

References

1. Massironi S, Cavalcoli F, Rausa E, et al. Understanding short bowel syndrome: Current status and future perspectives. *Dig Liver Dis.* 2020;52(3):253-261.
2. DiBaise JK. Managing the adult patient with short bowel syndrome. *Gastroenterol Hepatol* (N Y). 2017;13(10):600-608.
3. Carbonnel F, Cosnes J, Chevret S, et al. The role of anatomic factors in nutritional autonomy after extensive small bowel resection. *JPEN J Parenter Enteral Nutr.* 1996;20:275-280.

Small Intestinal Bacterial Overgrowth in Pediatric Short Bowel Syndrome

AUTHOR PROFILE:

Rosemary Pauley, APRN, is a pediatric gastroenterology nurse practitioner at Boys Town National Research Hospital in Omaha, NE

Small bowel bacterial overgrowth (SIBO) is a condition in which non-native bacteria and/or native bacteria are present in increased numbers. The condition can result in excessive fermentation, inflammation, or malabsorption. In the healthy gut, enteric bacteria colonize the alimentary tract soon after birth. After the first year of life, the composition of the intestinal microflora remains relatively constant.[1] While much remains unknown about what is considered "normal" intestinal flora—particularly in the developing infant and child—what is currently understood is that the diverse presence of intestinal microbes in varying numbers does not necessarily constitute an active disease state. Several mechanisms prevent the excessive colonization of the intestine by micro-organisms and subsequent development of pathologic SIBO.

Pediatric patients with short bowel syndrome (SBS), meanwhile, may be prone to the pathologic effects of SIBO due to several factors that include, but are not limited to, the child's genetics, diet, presence or absence of the ileocecal valve, the total amount and location of the removed intestinal tract, dysmotility, and other comorbid conditions and medications. After intestinal resection, an adaptation response ensues with enhancement of the surface area through deepening of the crypts, lengthening of the villi, and bowel dilatation. Neuroendocrine factors in patients with SBS work to slow bowel transit, thereby enhancing absorption. However, this may contribute to the development of microbial overgrowth.[2]

Pediatric SIBO is particularly problematic not only due to side effects such as diarrhea, gas, and bloating, but also from its negative impact on nutritional status, overall growth, and bowel adaptation. SIBO also may cause an inflammatory response that damages the absorptive surface, resulting in abdominal distention/cramping, bloody stools, malnutrition (fat/protein losses and decreases in fat-soluble vitamins) and, in severe cases, large joint arthritis and neurological symptoms such as encephalopathy and seizures.

What is "Normal" Gut Flora?

The gastrointestinal tract is populated by 10 times as many microbial organisms vs. cells in the human body. A typical gastrointestinal tract contains at least 500 different bacterial species.[3] Most microbial species in the gut remain unculturable. In addition, the types of microbial species and their concentrations differ along the alimentary tract. For example, the stomach and proximal small bowel normally contain relatively few bacteria due to the presence of gastric acid and the effects of peristalsis. Lactobacilli, enterococci, gram-positive aerobes, and facultative anaerobes predominate in the mid-to-distal jejunum.[4] The concentration of coliforms is low in the mid-to-distal jejunum while *Bacteroides*, the predominant organism in the colon, are rarely found in the proximal small bowel. The microbiology of the terminal ileum represents an intermediary zone between the aerobic flora found in stomach and proximal small bowel and the dense population of anaerobic organisms found in the colon. The concentration of anaerobic organisms is much higher in the colon than surrounding areas and includes enterobacteria and other coliforms. In the colon, the predominant anaerobic organisms are *Bacteroides*, *Lactobacillus*, *Clostridium*, and *Bifidobacteria*, although many species coexist.[5]

The normal complement of intestinal bacteria has several functions as illustrated in studies of animals reared in a germ-free environment. The small intestinal villi of these animals are thin and consistently regular, with relatively shortened crypts. The enterocytes are cuboidal rather than columnar. In addition, the number and size of Peyer's patches, the degree of leukocyte infiltration in the lamina propria, and the rate of mucosal regeneration are all reduced, which affects intestinal function.[6] Functionally, the small intestine of germ-free animals have demonstrated reduced digestive enzyme activity, decreased local cytokine production, alterations in the gut-associated lymphoid tissues, and a depressed migrating motor complex.[7]

In the human intestines, microorganisms perform several important physiologic functions, including the following:

- In the small intestine, bacterial-enterocyte crosstalk via binding sites and toll-like receptors differentiate commensal and pathogenic bacteria as well as food antigens. In this way, nonpathogenic bacteria protect the host by mounting an immune response to disease, causing bacteria and preventing an immune reaction to protein antigens in our diet (ie, oral tolerance)[8]

- Colonic bacteria salvage unabsorbed dietary carbohydrates by fermenting them to form fatty acids. These can be absorbed through the colonic mucosa and used as an energy source.

- Enteric bacteria produce nutrients and vitamins such as folate and vitamin K

- Numerous resident bacteria may also produce chemicals or bacteriocins that kill or inhibit proliferation of other surrounding bacterial species

- Bacterial metabolism of several medications within the intestinal lumen is essential for the action of some drugs. An example is the splitting off of the active molecule of sulfasalazine.

What is "Abnormal" Gut Flora?

SIBO is usually associated with abnormally high microbial/bacterial populations in the small intestine. The gold standard for the diagnosis of SIBO in pediatric patients has traditionally been the demonstration of excessive microbial concentrations in a jejunal aspirate, which can be performed during endoscopy or by fluoroscopy with jejunal intubation. Clinically significant SIBO is diagnosed when these counts exceed 105 organisms/mL (normal jejunal concentrations are ≤104 organisms/mL).[9]

However, more recent definitions of SIBO focus less on the actual numbers or types of organisms since these values can be highly variable between individuals. The definition of SIBO should ideally reflect the functional or pathological effects of the organisms in the remaining bowel. If, for example, the organisms are associated with the production of toxins that may have a systemic untoward effect, a medical intervention is typically needed. Additionally, some enteric bacteria degrade protein and urea to produce elevated levels of ammonia, which is associated with the development of encephalopathy in patients with hepatic impairment. SIBO may also be associated with the development of D-lactic acidosis, which can cause seizures. Gram-negative organisms can elaborate endotoxins, which can activate multiple inflammatory cytokines that interfere with the function of hepatocyte membrane transporters and lead to jaundice and liver injury.[10,11]

The presence of excess numbers of bacteria in the small bowel does not necessarily mean that these bacteria are doing any harm, a point that is often misunderstood. As noted earlier in this essay, a certain number of commensal bacteria is important, but it is the inflammation that occurs as a result of bacterial overgrowth that leads to the development of SIBO-related symptoms. The nonspecific inflammation that occurs with overgrowth is likely due to more invasive strains of bacteria, which results in a variety of epithelial changes. Extensive biopsies of the small bowel may give the best indication of whether the micro-organisms present in a given patient are actually harmful.[12] On light microscopy, the histologic appearance of the small intestine may not be significantly altered; however, in some cases, subtotal villus atrophy and increased cellularity in the lamina propria can occur.[13] Focal areas of ulceration and erosions also may be seen.

Impaired absorption of nutrients in patients with SIBO results from either maldigestion in the intestinal lumen or from malabsorption at the level of the intestinal microvillus membrane due to enterocyte damage. Facultative anaerobes may injure the intestinal surface by direct adherence and production of enterotoxins. Aerobic bacteria produce enzymes and metabolic products that are also capable of inducing epithelial cell injury.

Fat malabsorption may result from bacterial deconjugation of bile acids and the toxic effect of bile acids within the intestinal mucosa.[14] At a physiologic pH, bile acids are fully ionized, preventing their absorption in the proximal small intestine. Deconjugated bile salts resulting from SIBO are reabsorbed by the jejunum, which may lead to insufficient concentrations for normal fat absorption. Bacterial deconjugation also leads to the production of lithocholic acid, which may be toxic to intestinal epithelium, resulting in impaired absorption of fat and other nutrients.

Carbohydrate malabsorption results from the intraluminal breakdown of sugars, and enterocyte damage from the bacteria themselves may diminish enterocyte disaccharidase activity and reduce the transport of monosaccharides.[15] Similarly, protein malabsorption can occur as a result of decreased mucosal uptake of amino acids and the intraluminal degradation of protein precursors by some microorganisms. Cobalamin (vitamin B12) malabsorption is also commonly associated with SIBO.

What are the Intrinsic Human Mechanisms Present to Prevent SIBO?

Several host defense mechanisms prevent excessive colonization by bacteria in a child with SBS. Antegrade peristalsis prevents attachment of ingested microorganisms and, therefore, patients with SBS receiving anti-motility drugs should be monitored for the effects of overgrowth. Gastric and bile acids destroy many microorganisms before they leave the stomach. The use of acid suppression should be carefully considered in patients with SBS as this may negatively impact the human intestinal microbiome.[15]

One method to prevent bacterial overgrowth is via the production of the intestinal mucus layer, which traps bacteria to be excreted in the stool. An intact ileocecal valve as well as the antegrade motility pattern of the ileum itself inhibits retrograde translocation of bacteria from the colon to the small bowel. If the valve is absent, the chances of developing SIBO may increase. In these instances, clinicians should use caution when prescribing anti-motility drugs. The largest fraction of immunoglobulins secreted in the human body comes from the secretory IgA originating in the gastrointestinal tract. Pathogenic bacteria may be able to dampen the local immune system and disrupt normal gastrointestinal function, although the specific harmful effects are not well understood.

What About Treatment?

SIBO must first be appropriately diagnosed and treated if problematic. Obtaining appropriate, non-contaminated jejunal aspirates and then transporting and culturing them in the appropriate fashion is difficult. Interpretation of the standard lactulose or glucose breath testing is also complicated because of rapid intestinal transit, which can cause false-positive results. As mentioned earlier in this essay, the most accurate way to identify problematic overgrowth is through intestinal biopsy. However, many pediatric patients with SBS are treated empirically when typical symptoms occur, especially if they have intestinal dysmotility or dilated small bowel loops.

SIBO usually responds to treatment with antibiotics, although repeat or cyclical dosing is often required. Dietary changes, including the reduction of easily fermentable foods (primarily sugary carbohydrates) and an increase in fatty foods to slow bowel motility, may help in minor cases. Administration of anti-inflammatory drugs such as glucocorticoids and salicylate products may be necessary in severe cases when bacterial invasion of the mucosa has resulted in the development of a significant inflammatory lesion. Children with SBS often withhold their stools, which may exacerbate intestinal stasis and promote bacterial overgrowth. If intestinal stasis is suspected to contribute to bacterial overgrowth, promotility agents may be beneficial. When the bacterial overgrowth is related to small bowel dilation, surgical procedures to taper the bowel, with or without bowel lengthening, may be helpful.

SIBO: The Real-World Perspective

So what does SIBO look like in the clinic? Let's look at a brief example.

Christina is a 5-year-old girl we have managed since birth. She had a history of SBS as a result of necrotizing enterocolitis, with just 35 cm of jejunum anastomosed to the transverse colon. Christina had been on total parenteral nutrition for the first 4 years of her life but was weaned to total enteral nutrition after a bowel lengthening procedure was performed due to SIBO. The lengthening procedure was ordered after multiple attempts to advance Christina's feeding rate failed, causing abdominal distention, nausea/vomiting, and diarrhea.

A year after the lengthening procedure and switch to enteral nutrition, Christina was doing well, maintaining normal growth and averaging three mushy bowel movements per day. She was receiving continuous enteral nutrition via an extensively hydrolyzed formula delivered via G-button and had just started attending preschool. During one of her routine follow-up visits, Christina's mother told us that her daughter's stools appeared looser in recent weeks, with some bloody streaks and accompanying crampy pains. Her weight had decreased by approximately 2 pounds as her appetite was slightly diminished. Christina was eating a greater variety of foods at preschool, especially carbohydrates, and seemed to be enjoying an expanded palate. We ordered stool fecal occult blood and lacroferrin tests, which confirmed the presence of blood in the stools and a moderate amount of overall inflammation. We advised Christine's mother to send food with her daughter to preschool so that she could reinstitute a diet higher in fat and protein content and lower in carbohydrates. We also prescribed a short course of oral sulfasalazine to get Christina's inflammation under control.

One month later, Christina's condition had stabilized. She had regained weight and her stools were back to normal. To maintain this delicate homeostasis, Christina's mother agreed to continue packing a separate lunch for her daughter and even sent along additional high-protein snacks for some of the other kids in Christina's class to enjoy.

Conclusion

SIBO is a condition in which non-native bacteria and/or native bacteria are present, resulting in excessive fermentation, inflammation, and/or malabsorption. SIBO has been associated with the production of toxins that may have both local and systemic effects. In the pediatric patient, SIBO may not only cause symptoms of abdominal cramping, bloating, and diarrhea but can also have significant malabsorptive effects that impact overall patient growth.

References

1. Rutayisire E, Huang K, Liu Y, Tao F. The mode of delivery affects the diversity and colonization pattern of the gut microbiota during the first year of infants' life: a systematic review. *BMC Gastroenterol.* 2016;16(1):86.
2. Kalafatakis K, Triantafyllou K. Contribution of neurotensin in the immune and neuroendocrine modulation of normal and abnormal enteric function. *Regul Pept.* 2011;170(1-3):7-17.
3. Sender R, Fuchs S, Milo R. Revised estimates for the number of human and bacteria cells in the body. *PLoS Biol.* 2016;14(8):e1002533.
4. Walker MM, Talley NJ. Review article: bacteria and pathogenesis of disease in the upper gastrointestinal tract--beyond the era of Helicobacter pylori. *Aliment Pharmacol Ther.* 2014;39(8):767-79.
5. Evaldson G, Heimdahl A, Kager L, Nord CE. The normal human anaerobic microflora. *Scand J Infect Dis Suppl.* 1982;35:9-15.
6. Kennedy EA, King KY, Baldridge MT. Mouse Microbiota Models: Comparing germ-free mice and antibiotics treatment as tools for modifying gut bacteria. *Front Physiol.* 2018 Oct 31;9:1534.
7. Tlaskalová-Hogenová H, Stepánková R, Hudcovic T, et al. Commensal bacteria (normal microflora), mucosal immunity and chronic inflammatory and autoimmune diseases. *Immunol Lett.* 2004;93(2-3):97-108.
8. Weng M, Walker WA. The role of gut microbiota in programming the immune phenotype. *J Dev Orig Health Dis.* 2013;4(3):203-14.
9. Khoshini R, Dai SC, Lezcano S, Pimentel M. A systematic review of diagnostic tests for small intestinal bacterial overgrowth. *Dig Dis Sci.* 2008;53(6):1443-54.
10. Avelar Rodriguez D, Ryan PM, Toro Monjaraz EM, Ramirez Mayans JA, Quigley EM. Small intestinal bacterial overgrowth in children: A state-of-the-art review. *Front Pediatr.* 2019;7:363.
11. Bushyhead D, Quigley EM. Small intestinal bacterial overgrowth. *Gastroenterol Clin North Am.* 2021;50(2):463-474.
12. Wang F, Gerhardt BK, Iwansky SN, et al. Glucocorticoids improve enteral feeding tolerance in pediatric short bowel syndrome with chronic intestinal inflammation. *J Pediatr Gastroenterol Nutr.* 2021;73(1):17-22.
13. Harrison E, Hayes S, Howell L, Lal S. All that glitters is not always gold. *BMJ Case Rep.* 2014;2014:bcr2013201963.
14. Saltzman JR, Russell RM. Nutritional consequences of intestinal bacterial overgrowth. *Compr Ther.* 1994;20(9):523-30.
15. Levy EI, Hoang DM, Vandenplas Y. The effects of proton pump inhibitors on the microbiome in young children. *Acta Paediatr.* 2020;109(8):1531-1538.

AUTHOR PROFILE:

Michele Rubin, MSN, APN, CNS, CGRN, is an advanced practice nurse at the University of Chicago Medicine in Chicago, IL

Psychosocial Challenges in Patients with Short Bowel Syndrome and Crohn's Disease

Everyone copes with physical illness differently. Some people react in a highly emotional manner, whether through fear, sadness, depression, or another psychological response. Others may integrate a healthy acceptance and develop the resilience needed to manage their physical illness.

As many of us know, the psychosocial challenges associated with short bowel syndrome (SBS) can be overwhelming for both patients and caregivers. Patients with SBS have to deal with many of the uncomfortable and unpleasant symptoms associated with the disease such as frequent diarrhea, cramping, dehydration, fatigue, loss of appetite, enteral or parenteral nutrition, and ileostomy issues. These can all cause psychological distress and physical changes that impact our patients' daily life. The most frequent psychosocial issues that I have seen in adult SBS patients with Crohn's disease—the most common subset of SBS patients in our practice—involve depression, anxiety, denial, and post-traumatic stress.

Depression is defined as persistently depressed, sad mood or loss of interest. Specific symptoms associated with depression include trouble sleeping, changes in appetite, hopelessness, guilt, difficulty concentrating, and suicidal thoughts. **Anxiety**, meanwhile, is defined as an emotional state characterized by tension, worried thoughts, and physical changes. Symptoms associated with anxiety include nervousness, irritability, GI upset, and excess sweating. Some patients with extreme anxiety will avoid social situations in which symptoms might occur, spend excessive time researching information online, send an abundance of emails to the medical team with questions, and ruminate about short- and long-term "what ifs."[1]

In our patients with SBS and Crohn's disease, depression and anxiety are common. These conditions can be caused by a combination of several factors, including the following:

- **A reaction to the progression of the disease state itself** — Many patients with moderate-to-severe Crohn's disease undergo multiple bowel surgeries, with each surgery shortening the length of the short bowel itself and increasing the likelihood of an eventual SBS diagnosis. These patients often require regular visits to the ER and hospital admissions for replenishment of nutrients through IV fluids and oral or intravenous supplementation, as well as intestinal blockages due to strictures.

- **Disruptive symptoms** — These commonly include recurrent diarrhea, frequent bathroom breaks, abdominal pain and cramping, dehydration, malnutrition, and extreme fatigue. Among patients who require an ileostomy, symptoms such as the potential for leaks of stool, skin irritation issues, and noisy stoma with gas passage can be even more psychologically paralyzing.
- **Loss of enjoyment from eating** — For many people, food and the socialization that comes with eating are a primary form of daily pleasure. However, for our patients with SBS and Crohn's disease, food and nutrition are a common cause of stress due to the need for frequent IV fluids, oral rehydration drinks, enteral or parenteral nutrition, and decreased overall appetite. There is also often fear of eating certain foods that may cause diarrhea, bowel cramping, or leakage of stool.[2,3] Some patients may stop socializing with friends and family in settings where food is involved to avoid uncomfortable situations and potential embarrassment.
- **Guilt about the burden their condition places on others** — We all need support from our friends and family from time to time, but for people with SBS, there are frequent periods where the emergency call goes out to the support team for rides to medical appointments or the emergency room, help with daily activities, and other common needs. Putting this burden on loved ones can lead to embarrassment and anxiety, not to mention the impact of a loss of personal independence.[4]
- **Denial about their diagnosis** — Some patients simply don't want to accept that they have SBS or acknowledge the issues that are currently burdening them. I have had patients who refuse the use the term "short bowel syndrome" ("If I say it or hear it, that makes it real"), refuse to ask family or friends for help ("I can do it myself"), or stop taking medications if they don't feel like they are getting better or suffering side effects ("It's not working").[3,4]
- **Post-Traumatic Stress Symptoms (PTSS)** — As it relates to healthcare, PTSS involves situations where a patient has flashbacks to experiences that were upsetting or painful. Examples can include excessive time spent in pain, not being heard or properly acknowledged by providers, bad experiences with needle sticks for blood draws, or prolonged diarrhea or other unpleasant side effects.[5]

Table 1 *Tools to Use in Patients with Suspected Psychological Issues*

Depression

Patient Health Questionnaire-9 (PHQ-9)

Generalized anxiety

Generalized Anxiety Disorder-7 (GAD-7), PROMIS Anxiety Short Form

Helpful clinical interview questions:

"How have you been handling things emotionally?"

"Sometimes, people with SBS experience issues with (depression/anxiety/other psychological issue). Has this been a problem for you?"

"Have you been able to do the things you enjoy or function normally in your daily routines?"

When assessing psychosocial issues with your patients (whether these are patients with SBS, Crohn's disease, or other issues), there are several validated questionnaires that can be used. There are also guides available with a list of helpful interview questions to engage your patient and ease them into a discussion of psychological issues or concerns **(see Table 1)**. My current personal favorite resources to help explain the association between gut and brain health include two recent articles by Keefer et al that can be found in this article's references.[8,9]

Here are some other tips I have found helpful to engage my patients about psychosocial issues and their potential impact on quality of life:

- **Optimize body language** – Maintain eye contact and lean into the patient to show them that you are listening
- **Validate their thoughts** – Use phrases like, "I hear how much this impacting your life," or "I heard you mention you were having issues with (depression/anxiety/another psychological issue)"[9]
- **Normalize their experience** – "This is a common occurrence for many patients with SBS who I see"[9]
- **Educate on the gut-brain response** – The association between gut and brain health has been demonstrated in multiple studies.[7] Stress and anxiety can elicit cramping, diarrhea, constipation, and other issues. Patients should be educated on this link and, if necessary, referred to a psychologist who specializes in helping patients with gastroenterology issues.[8]
- **Use the "Teach Back Method" when teaching or discussing key issues** – When you feel you have explained yourself as thoroughly as possible, ask the patient to tell you what they heard. You may be surprised by what they have to say!
- **Provide additional resources** – Some patients I see come in with incomplete or erroneous information, which can clearly lead to anxiety. Sometimes, the best things we can do for our patients with psychological concerns is provide them with high-quality education that alleviates their stress and anxiety. Be clear about the plan of care, explain why you are recommending specific therapies, and be proactive to anticipate potential quality of life issues.

Coping with and managing SBS is very challenging. Not only does it require an experienced and specialized interdisciplinary team, but also a team that works and communicates well. In this era, it may also be important to have the ability to conduct telehealth visits to maintain a relationship with patients who live in remote areas. For these patients, communicating with their local primary care provider and/or helping them find a local psychologist to talk through their issues may also be helpful.

One more piece of advice – don't discount the power of peer communication. Patient support groups can be invaluable for our patients with SBS who are struggling with psychological issues **(see Table 2 for a list of relevant groups).** The best advice, help, and understanding often comes from those people who know and have struggled with the same experiences. A referral to a psychologist or psychiatrist is certainly often warranted in some cases, but I find that many patients with SBS find strength from their peer network.

Table 2 *SBS Patient Support Groups and Other Resources*

Group	Website
Rome GastroPsych	https://romegipsych.org
Short Bowel Syndrome Foundation	http://shortbowelfoundation.org
Crohn's and Colitis Foundation	https://crohnscolitisfoundation.org
United Ostomy Associations of America, Inc.	https://www.ostomy.org
GI on Demand	https://GIondemand.com
Oley Foundation	https://oley.org
Short Gut Family Support Group	https://www.facebook.com/groups/shortgutsupport/
Association for Behavioral & Cognitive Therapies	https://www.abct.org/
National Eating Disorders Association	https://www.nationaleatingdisorders.org/

References

1. Barberio B, Zamani M, Black CJ, Savarino EV, Ford AC. Prevalence of symptoms of anxiety and depression in patients with inflammatory bowel disease: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol*. 2021;6(5):359-370.

2. Ireton-Jones C, Shingleton K, Kakani S, Eating – An unappreciated complexity of nutrition care of people with short bowel syndrome (SBS). ASPEN Nutrition Science & Practice Conference. March 2022, Seattle, WA.

3. Robelin K, Senada P, Ghoz H, et al. Prevalence and clinician recognition of avoidant/restrictive food intake disorder in patients with inflammatory bowel disease. *Gastroenterol Hepatol* (N Y). 2021;17(11):510-514.

4. Stroebe M, Schut H. The dual process model of coping with bereavement: Rationale and description. *Death Studies.* 1999;23(3):197-224.

5. Taft TH, Quinton S, Jedel S, Simons M, Mutlu EA, Hanauer SB. Posttraumatic stress in patients with inflammatory bowel disease: Prevalence and relationships to patient-reported outcomes. *Inflamm Bowel Dis.* 2022;28(5):710-719.

6. Shmidt E, Suárez-Fariñas M, Mallette M, et al. A longitudinal study of sexual function in women with newly diagnosed inflammatory bowel disease. *Inflamm Bowel Dis.* 2019;25(7):1262-1270.

7. Shmidt E, Suárez-Fariñas M, Mallette M, et al. Erectile dysfunction is highly prevalent in men with newly diagnosed inflammatory bowel disease. *Inflamm Bowel Dis.* 2019;25(8):1408-1416.

8. Keefer L, Ballou SK, Drossman DA, Ringstrom G, Elsenbruch S, Ljótsson B. A Rome working team report on brain-gut behavior therapies for disorders of gut-brain interaction. *Gastroenterology.* 2022;162(1):300-315.

9. Keefer L, Bedell A, Norton C, Hart AL. How should pain, fatigue, and emotional wellness be incorporated into treatment goals for optimal management of inflammatory bowel disease? *Gastroenterology.* 2022;162(5):1439-1451.

Nutritional Interventions in Short Bowel Syndrome: A Complex Concept

AUTHOR PROFILE:

Kristen M. Roberts, PhD, RDN, LD, CNSC, FASPEN, FAND, is an associate clinical professor in the School of Health and Rehabilitation Sciences at The Ohio State University in Columbus, OH
Columbus, OH

While many—though not all, patients with short bowel syndrome (SBS)—require nutritional support to maintain their nutritional status and reduce hospital admissions, determining the appropriate type and duration of support relies on an astute healthcare team that is well-versed in SBS management.[1]

Nutritional support comes in several forms, including the use of enteral nutrition (EN), parenteral nutrition (PN), and intravenous fluids (IVFs) when oral diet alone is insufficient. While SBS is fortunately uncommon, its rarity means that few providers have true clinical expertise managing the condition. From my lens as a dietitian with a specific interest in nutrition support among patients with gastrointestinal (GI) disorders, I find that many patients with GI issues, and especially those with SBS, are inappropriately educated on proper dietary parameters. Too often, patients report to me that their healthcare team encouraged consumption of high-calorie, high-protein shakes and/or calorically dense desserts since "all calories are good calories." Unfortunately, patients receiving this advice often present to our emergency department with dehydration and electrolyte abnormalities. It is typically not the SBS patient to blame for these visits and subsequent hospital admissions, but rather the healthcare professional who provided inappropriate education to a patient at high risk for nutrition support and dehydration.

Let's look at some of the dietary interventions with which clinicians need to familiarize themselves to minimize the need for emergency care among patients with SBS.

Dietary interventions are designed based on the anatomical configuration of the gastrointestinal tract **(see Table 1).**[2] When determining a dietary prescription, the registered dietitian nutritionist (RDN) determines the appropriate mix of carbohydrate, protein, and fat necessary to support the patient's particular anatomy. Although there are small differences in the contribution of each of these macronutrients, understanding carbohydrate intake is the bedrock to better understanding the importance of specific dietary interventions.

Carbohydrate is an umbrella term that incorporates fibers, complex carbohydrates, sugar alcohols, and simple sugars. Each of these carbohydrates have various physiological functions in the gastrointestinal tract.[3] **Table 2** includes a more detailed description of the implications of each type of carbohydrate consumed in the diet and their specific relevance for patients with SBS.

Complex carbohydrates are commonly found in foods such as pasta, rice, and potatoes, form the basis of many meals, and can slow intestinal transit time and encourage bowel adaptation, thereby reducing the likelihood of needing nutrition support.

Intake of *simple sugars*, such as those found in soda, fruit juice, sports drinks, sugary desserts, and ice cream, should be minimized due to their poor nutritional quality and contributions to chronic disease.[4] Specifically in patients with SBS, simple sugars create an osmotic load that drags water into the intestine, resulting in malabsorption due to the shortened length of intestine.

Table 1 *Dietary interventions based on the anatomical configuration of the gastrointestinal tract in SBS*[2]

SBS anatomy defined	Dietary Interventions
Type I: Ostomy in place, no colon in continuity	• Carbohydrate 40-50% of kilocalories • Protein 20-30% of kilocalories • High-fat diet (30-40% is acceptable) • Oral rehydration solution as tolerated • Lactose as tolerated • If intestinal length is <100 cm without high output despite pharmacological therapy, food is for pleasure only; consider restricting fluids to 1-1.5 liters per day
Type II: Small bowel resection and partial colon in continuity	• Carbohydrate 50-60% of kilocalories • Protein 20-30% of kilocalories • Fat-restriction 20-30% of kilocalories • Simple sugar restriction to reduce the risk of D-lactic acidosis and malabsorption • Oral rehydration solution as tolerated • Oxalate restriction and/or calcium supplementation in those with hyperoxaluria • Inclusion of soluble, fermentable fibers as tolerated for short-chain fatty acid production
Tyle III: Small bowel resection with entire colonic preservation	• Carbohydrate 50-60% of kilocalories • Protein 20-30% of kilocalories • Fat-restriction 20-30% of kilocalories • Simple sugar restriction to reduce the risk of malabsorption • Oral rehydration solution usually not necessary since colon reabsorbs dietary fluids efficiently • Oxalate restriction and/or calcium supplementation in those with hyperoxaluria • Inclusion of soluble, fermentable fibers as tolerated for short-chain fatty acid production
General for all subtypes	• Small, frequent meals may improve digestion and absorption • Avoidance of fluids with meals may improve intestinal transit time • Avoidance of hypertonic (ie, sports drinks, soda, juice) and hypotonic beverages (ie, water, tea, coffee) • Minimize insoluble fiber when stool volume is high and watery • Complex carbohydrates should be included in all meals and snacks

Table 2 *Defining the physiological role of carbohydrates in the gastrointestinal tract* [4]

Carbohydrate Subclass	Example food sources	Suggested benefits	Possible risks
Complex carbohydrates	• White rice/pasta/bread • Bagels, rolls, biscuits • Crackers, saltines, hot cereals • Bananas, firm • Potatoes	• May slow intestinal transit time • May enhance intestinal adaptation	• Excessive intake of complex carbohydrates can contribute to poorly controlled diabetes or hepatic steatosis in a non-SBS patient population
Simple carbohydrates (mono- and di-saccharides)	• Table sugar, soda, sports drinks, cake, candy, cookies	• Caloric source • Enhances the taste and palatability of some foods	• Hyperosmolar foods and beverages cause an osmotic pull in the gastrointestinal tract, leading to gas, diarrhea, and dumping • Feeding of the pathogenic microbiota in the gastrointestinal tract can contribute to small intestinal bacterial overgrowth
Sugar alcohols	• Sugar-free candy or desserts, sugar-free chewing gum, apples, cauliflower, mushrooms	• Reduces or replaces the simple sugar content of foods, which can be beneficial for blood sugar control in a non-SBS cohort of patients with diabetes mellitus	• Increases gas production • Doses exceeding 10 g/day are more likely to lead to diarrhea
Soluble and fermentable fibers	• Oats/oatmeal (viscous), guar gum (viscous), wheat dextrin, inulin	• Thickens stool • Decreases intestinal transit time • Promotes short-chain fatty acid production in those with colon in continuity	• Increases gas production
Insoluble, non-fermentable fibers	• Cellulose and lignins found in the skins and seeds on fruits, vegetables, and whole grains	• Increases fecal weight	• Can lead to an increase in watery stools, especially in those without a colon in continuity • Can contribute to abdominal pain, cramping, and obstructions in those with intestinal strictures and adhesions

Furthermore, small intestinal bacterial overgrowth (SIBO) is a known risk factor associated with the loss of the ileocecal valve or presence of strictures/adhesions in patients with SBS.[5] Simple sugars and sugar alcohols are the optimal food source for pathogenic bacteria, and their intake can lead to an increase in bacterial overgrowth, ultimately exacerbating SIBO. In extreme cases of SIBO, liver pathology, micronutrient deficiency, and weight loss can occur, increasing the likelihood that a patient will require nutritional support.[5]

The dietary role of *fibers* is an area of active research due to their diverse functions in human physiology and noted health benefits. In school, many of us learned that fibers are either classified as soluble or insoluble, but we now understand that this is an over-simplified message. Today, fibers are more distinctly classified by their solubility, viscosity, and fermentability. In SBS, the use of fiber is based on anatomy and patient tolerance. In type II and type III subtypes of SBS, the colon is in continuity. The colon is a lifesaving organ due to its ability to effectively absorb fluids and ferment fibers, unleashing not only their prebiotic effects but also allowing of salvage up to 1000 kcals/day as the result of short chain fatty acid production. Therefore, intake of soluble and fermentable fibers is encouraged in patients with a colon who are able to tolerate an increased fiber load. Unfortunately, insoluble and non-fermentable fibers may contribute to an increase in fecal output, especially in patients with an ostomy, and lead to fluid and electrolyte losses. Therefore, in patients with type I SBS, fiber is only introduced in the absence of contraindications in a fluid stable patient.

Now that we have a basic grounding in the role of specific macronutrients, let's circle back to that recommendation of hyperosmolar, high calorie, high protein shakes packed with excessive doses of simple sugars that I often see suggested for severely malnourished patients. In some instances, there may even be sugar alcohols within the formulation. Can you then see how "all calories **are not** good calories" in patients with SBS? Both high doses of simple sugars and even moderate levels of sugar alcohols increase the risk of malabsorption in patients with SBS. Additionally, because most of these beverages come in 240 mL (8 fluid ounce) bottles, if consumed consecutively, they can exacerbate intestinal transit time, leading to a higher dependence on intravenous nutritional support. Conversely, consumption of fluids in smaller doses (ie, 120 mL/4 fluid ounces, sipping over time) may improve tolerance; iso-osmolar beverages such as oral rehydration solutions (closer to 300-340 mOsm/L) are also an appropriate choice for patients with SBS.

Let's see how these concepts come together in clinical practice.

Christine is a recent patient whose history I reviewed. She had type 1 SBS and was severely malnourished. Her baseline urine output was 800 mL/d and ileostomy output was 2.2 L/d. She was eating 6 small meals each day, drank water, and reported a diet that involved some of her favorite foods such as white bread, canned vegetables, white rice, tender meats, and occasional bananas. Her healthcare team—which did not include a dietitian—determined that Christine was not consuming enough daily kilocalories or consuming adequate fluid. They therefore recommended that she drink a commercial protein shake three times daily that contained 26 g of sugar per 8 ounces. Can you guess what happened next? Yes, while her kilocalories increased initially, Christine presented to the emergency room a few days later with an acute kidney injury and another 2 pounds of lost weight.

So what should have been done differently? Instead of introducing protein shakes, it would have been more appropriate to initiate or adjust her pharmacotherapy—likely to involve antidiarrheals—in addition to substituting oral rehydration solution for water. The oral supplement was likely exacerbating Christine's ostomy output, further depleting her total body water.

Appropriate dietary management of the patient with SBS starts by understanding their specific anatomy. Matching the optimal dietary intervention to that anatomical configuration allows the clinician to appropriately monitor absorption and determine the need for dependence on PN/IVFs. This in turn should reduce hospital emergency room visits and admissions, improving overall patient outcomes and putting less stress on the healthcare system.

References

1. Pironi L. Definitions of intestinal failure and the short bowel syndrome. *Best Pract Res Clin Gastroenterol.* 2016;30(2):173-85.
2. Pogatschnik C, Steiger E. Therapeutic diet modifications in short bowel syndrome. In: Corrigan M (Ed.). Adult Short Bowel Syndrome: Nutritional, Medical, and Surgical Management. Elsevier. 2018:55-65.
3. Gu J, Roberts KM. Probiotics and prebiotics. In Corrigan M (Ed.). Adult Short Bowel Syndrome: Nutritional, Medical, and Surgical Management. Elsevier. 2018:67-80.
4. U.S. Department of Agriculture and U.S. Department of Health and Human Services. Dietary Guidelines for Americans, 2020-2025. 9th Edition. December 2020. Available at DietaryGuidelines.gov. Accessed August 15, 2022.
5. DiBaise J. Nutritional consequences of small intestinal bacterial overgrowth. *Practical Gastroenterology.* 2008;69:1-28.

Society of Gastroenterology Nurses and Associates, Inc.

330 N Wabash Avenue - Suite 2000
Chicago IL 60611
Phone: (312) 321-5165