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SHORT BOWEL SYNDROME: NURSING ASSESSMENT, CLINICAL MANAGEMENT, AND EVIDENCE-BASED CARE-AN UPDATED REVIEW

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ABSTRACT

Background: Short bowel syndrome (SBS) is a complex gastrointestinal disorder resulting from extensive loss of functional small intestine, leading to intestinal failure, malabsorption, fluid imbalance, electrolyte disturbances, and chronic nutritional deficiencies. Despite advances in intestinal rehabilitation and parenteral nutrition (PN), SBS continues to present significant clinical challenges that require comprehensive multidisciplinary management. Nurses play a pivotal role in early assessment, nutritional monitoring, prevention of complications, patient education, and coordination of long-term care. **Aim:** This review aimed to provide an updated evidence-based overview of short bowel syndrome, emphasizing its epidemiology, pathophysiology, intestinal adaptation, nutritional management, nursing assessment, pharmacological therapies, and multidisciplinary interventions that improve clinical outcomes and quality of life. **Methods:** A comprehensive narrative review of current scientific literature was conducted using published clinical guidelines, systematic reviews, observational studies, and landmark clinical trials addressing the diagnosis, management, and rehabilitation of patients with SBS. The review integrates contemporary evidence regarding intestinal physiology, parenteral nutrition, emerging pharmacological therapies, and nursing care strategies. **Results:** The evidence demonstrates that successful management of SBS depends on individualized multidisciplinary care focused on maintaining hydration, correcting nutritional deficiencies, promoting intestinal adaptation, minimizing dependence on long-term PN, and preventing treatment-related complications. Early nutritional intervention, optimized enteral feeding, meticulous catheter management, pharmacological therapies including antidiarrheal agents and glucagon-like peptide-2 analogues such as teduglutide, and structured intestinal rehabilitation programs significantly improve intestinal function and patient outcomes. **Conclusion:** Short bowel syndrome remains a lifelong condition requiring individualized, evidence-based management delivered through coordinated multidisciplinary care. Nurses are central to optimizing nutritional status, preventing complications, supporting intestinal rehabilitation, promoting patient self-management, and improving long-term survival and quality of life.

KEYWORDS: Short bowel syndrome; Intestinal failure; Parenteral nutrition; Home parenteral nutrition; Intestinal adaptation.

INTRODUCTION

Short bowel syndrome (SBS) represents an uncommon, yet clinically significant gastrointestinal disorder characterized by a substantial reduction in the functional absorptive capacity of the intestine following extensive intestinal loss. This condition most frequently develops after major surgical resection of the small intestine but may also arise from congenital anatomical abnormalities or pathological diseases that compromise intestinal integrity and function. The clinical course of SBS is remarkably heterogeneous among adult patients because outcomes are influenced by numerous factors, including the underlying disease process, the length and anatomical segments of the remaining intestine, preservation of the colon and ileocecal valve, intestinal adaptation, and the individual's physiological and psychosocial

characteristics. Consequently, patients demonstrate considerable variability in disease severity, nutritional status, and treatment requirements. Despite this heterogeneity, SBS is commonly associated with a constellation of gastrointestinal manifestations, including persistent diarrhea, steatorrhea, abdominal discomfort, malnutrition, electrolyte disturbances, and chronic dehydration, all of which substantially impair daily functioning and overall health-related quality of life. The etiological conditions leading to SBS are diverse and frequently necessitate extensive intestinal resection to preserve life or control severe disease. Common indications for surgical bowel removal include mesenteric ischemia, radiation-induced enteritis, Crohn's disease, traumatic intestinal injury, and selected congenital gastrointestinal malformations. The anatomical configuration remaining after bowel resection varies according to the extent and location of intestinal loss. Surgical reconstruction may result in an ileocolonic or jejunocolonic anastomosis, whereas some patients require the creation of an end-jejunostomy or ileostomy. These anatomical differences play a pivotal role in determining the absorptive potential of the remaining intestine, influencing fluid and electrolyte balance, nutritional status, and the likelihood of achieving intestinal adaptation. Variations in postoperative anatomy therefore contribute substantially to the clinical diversity observed among individuals with SBS.[1-2]

The nutritional consequences of SBS extend far beyond impaired digestion and absorption, producing progressive dysfunction across virtually every physiological system. Chronic malnutrition negatively affects immune competence, muscular strength, wound healing, endocrine regulation, cardiovascular stability, and overall functional capacity, ultimately resulting in a marked decline in health status and quality of life. Furthermore, inadequate nutritional intake and nutrient absorption increase susceptibility to infections, exacerbate complications associated with both medical illness and surgical interventions, prolong recovery periods, and contribute to increased mortality rates.[1,2] Fluid depletion and excessive gastrointestinal sodium losses place affected individuals at considerable risk of developing persistent hypotension as well as both acute and chronic renal impairment.[3] Additional metabolic complications include the formation of gallstones secondary to impaired gallbladder motility and bile stasis, while deficiencies of magnesium, albumin, and calcium frequently occur because of inadequate intestinal absorption. These biochemical abnormalities may produce widespread systemic manifestations involving the neuromuscular, cardiovascular, skeletal, and metabolic systems, further increasing disease burden and clinical complexity.[3,4] Moreover, many patients continue to experience severe chronic diarrhea that significantly disrupts daily activities and remains refractory despite intensive dietary modifications and conventional pharmacological therapies. For a considerable proportion of patients, oral nutritional strategies alone are insufficient to maintain adequate hydration, caloric intake, and essential nutrient requirements because the residual intestine lacks adequate absorptive capacity. Even substantial increases in oral food and fluid consumption may fail to compensate for extensive nutrient and fluid losses, necessitating prolonged dependence on parenteral nutrition (PN) and intravenous (IV) fluid supplementation to sustain metabolic homeostasis and survival. Although PN and IV therapy have transformed the prognosis of SBS by providing life-sustaining nutritional support, long-term dependence on these interventions is associated with numerous serious complications that may arise either from the underlying disease process or from the treatment itself.[5] Among the most clinically significant complications are catheter-related bloodstream infections, catheter occlusion, venous thrombosis, metabolic bone disease, progressive hepatic dysfunction, liver failure, and a substantial deterioration in patients' quality of life.[6–11] These limitations underscore the importance of developing therapeutic strategies that enhance the absorptive function of the remaining intestine, promote intestinal adaptation, decrease dependence on long-term PN and IV support, and ultimately improve clinical outcomes and long-term quality of life for individuals living with SBS.

Epidemiology

Determining the true epidemiological burden of short bowel syndrome (SBS) remains a considerable challenge because of the rarity of the disorder, variations in diagnostic definitions, and the absence of universally standardized classification criteria. These limitations have restricted the availability of reliable population-based epidemiological data and have complicated efforts to accurately quantify both the incidence and prevalence of SBS across different healthcare systems. Consequently, researchers have frequently relied on indirect indicators, particularly the prevalence of home parenteral nutrition (HPN), to estimate the number of individuals living with SBS. Although this approach provides a useful approximation, it fails to capture the complete spectrum of patients affected by the condition, particularly those who have successfully achieved intestinal adaptation or those who succumb before long-term nutritional support becomes necessary. The relationship between SBS and long-term dependence on HPN has made HPN registries an important epidemiological resource for estimating disease frequency. In the United States, one of the earliest national estimates, published in 1991, suggested that approximately 20,000 individuals were receiving HPN.[12] While this estimate represented an important milestone in understanding the burden of chronic intestinal failure at that time, its applicability to contemporary clinical practice is uncertain because of substantial advances in intestinal rehabilitation, surgical techniques, nutritional management, and supportive care over subsequent decades. Furthermore, demographic changes, improved survival rates, and evolving indications for HPN have likely altered the

epidemiological landscape, emphasizing the need for updated nationwide surveillance systems capable of accurately documenting disease prevalence.

European epidemiological investigations have demonstrated considerable variation in HPN utilization among different countries, reflecting differences in healthcare infrastructure, referral pathways, reimbursement policies, and the availability of specialized intestinal rehabilitation centers.[13,14] Reported prevalence estimates range from approximately 1.1 individuals per million adults in Poland to 12.7 per million in Denmark, illustrating more than a tenfold variation across European populations.[13,14] Intermediate prevalence estimates have been reported in several other countries, including Belgium with approximately 3 cases per million, France with 3.6 per million, the Netherlands with 3.7 per million, Spain with 5.1 per million, and the United Kingdom with 3.7 per million.[13,14] These substantial geographical differences likely reflect disparities in healthcare accessibility rather than genuine differences in disease occurrence. Regions with highly developed intestinal rehabilitation services and specialized multidisciplinary centers are more likely to identify appropriate candidates for HPN, resulting in higher documented prevalence rates. Conversely, limited access to specialized care may contribute to underdiagnosis, delayed referral, or inadequate long-term management, thereby producing artificially lower prevalence estimates. Within individual countries, regional variability further illustrates the influence of healthcare organization on reported epidemiological data. In Spain, one of the most comprehensive national HPN registries reported an overall prevalence of approximately five adults per million receiving HPN.[13] However, marked regional differences were evident, with prevalence estimates ranging from only 1.4 per million in the Canary Islands to 11.5 per million in Madrid.[13] These findings suggest that the concentration of specialized intestinal rehabilitation programs in major metropolitan centers enhances access to advanced nutritional support and facilitates referral of patients requiring complex multidisciplinary management. Consequently, epidemiological estimates based solely on HPN utilization should be interpreted with caution, as they may primarily reflect healthcare availability rather than the actual distribution of SBS within the general population.

Several studies have confirmed that patients with SBS constitute a substantial proportion of individuals requiring long-term HPN. A large European survey conducted in 1997 involving 756 patients receiving HPN demonstrated that approximately 35% of all HPN users had SBS as the underlying indication for nutritional support.[14] More recent evidence from the Spanish HPN registry reported an even greater contribution of SBS, with approximately 47% of 148 registered HPN recipients, including both adult and pediatric patients, diagnosed with SBS.[16] These observations reinforce the close association between SBS and chronic intestinal failure requiring long-term nutritional supplementation. Based on these findings, investigators have estimated the prevalence of SBS in Europe to range broadly between approximately 0.4 and 6.0 cases per million individuals.[14] Nevertheless, these calculations remain imperfect because they exclude patients who recover sufficient intestinal function to discontinue HPN, as well as individuals who experience early mortality following extensive bowel resection. Consequently, the actual prevalence of SBS almost certainly exceeds current estimates, highlighting the need for comprehensive disease-specific registries capable of capturing the full clinical spectrum of affected patients. Recognizing these limitations, efforts have been undertaken to improve epidemiological surveillance. In 2011, the American Society for Parenteral and Enteral Nutrition introduced Sustain™, LLC, an online national registry designed to systematically collect information regarding patients receiving HPN throughout the United States.[17] The establishment of this registry represents a significant advancement toward generating accurate national prevalence estimates and improving understanding of the proportion of HPN recipients diagnosed with SBS. Comprehensive participation by healthcare providers, infusion services, and specialized nutrition centers has the potential to produce high-quality epidemiological data that can guide healthcare planning, resource allocation, clinical research, and policy development for this complex patient population.

Despite improvements in supportive care, individuals with SBS continue to experience lower long-term survival than the general population because of the severity of their underlying gastrointestinal disease and associated medical complications. Reported survival rates among patients with nonmalignant SBS remain relatively favorable, with approximately 86% surviving two years and 75% surviving five years following diagnosis or intestinal resection.[2] However, survival outcomes vary substantially according to several important clinical characteristics. Patients with an end-jejunostomy, a residual small bowel length measuring less than 50 cm, or bowel resection resulting from arterial mesenteric infarction demonstrate significantly poorer long-term survival compared with other anatomical or etiological subgroups.[2] These findings emphasize that anatomical configuration and the underlying cause of intestinal loss play critical roles in determining long-term prognosis. Importantly, long-term dependence on parenteral nutrition itself has not been identified as an independent predictor of mortality among patients with SBS.[2] Instead, mortality is more commonly attributed to the severity of the primary disease responsible for intestinal resection or to associated comorbid conditions that complicate clinical management.[18,19] This distinction underscores the importance of addressing the underlying pathological process in addition to providing optimal nutritional support. Advances in catheter care, infection prevention,

liver preservation strategies, and metabolic monitoring have substantially improved the safety profile of long-term HPN, allowing many patients to maintain prolonged survival despite persistent intestinal failure.

Recent evidence has also highlighted the value of specialized multidisciplinary care in improving patient outcomes. A retrospective study evaluating children with intestinal failure between 1990 and 2009 demonstrated significant improvements in survival following implementation of an integrated multidisciplinary care model.[20] Although multiple factors likely contributed to these favorable outcomes, coordinated management by specialized pediatric surgeons, gastroenterologists, dietitians, nurses, pharmacists, and other healthcare professionals enabled earlier intervention, closer clinical monitoring, more effective nutritional management, and prompt treatment of complications.[20] These findings reinforce the growing recognition that multidisciplinary intestinal rehabilitation programs are fundamental to optimizing both survival and quality of life for patients living with SBS across all age groups.

Definition

A universally accepted definition of short bowel syndrome (SBS) has not yet been established because the condition demonstrates considerable clinical variability and cannot be accurately characterized solely by the remaining length of the small intestine. Historically, clinical practice during the late 1980s was guided by the belief that patients with less than 100 cm of residual small bowel would inevitably require permanent dependence on parenteral nutrition (PN) and intravenous (IV) fluid support to maintain adequate nutritional status and survival.[2,21,22] This anatomical criterion served as a practical benchmark for predicting intestinal failure; however, subsequent clinical experience and research have demonstrated that bowel length alone is an insufficient predictor of functional intestinal capacity. Current healthcare policies continue to incorporate anatomical measurements into clinical decision-making. For example, the U.S. Centers for Medicare and Medicaid Services (CMS) considers individuals with less than approximately 5 feet (152 cm) of remaining small intestine eligible for reimbursement of home parenteral nutrition. Nevertheless, this criterion is intended primarily for administrative purposes rather than as a comprehensive clinical definition of SBS. In recognition of the complexity of the disorder, an international panel of experts proposed a broader conceptual framework for intestinal failure associated with SBS in 2006, emphasizing that residual bowel length should not be regarded as the sole determinant of disease severity or intestinal dysfunction.[23] The degree of malabsorption experienced by patients with SBS is influenced by multiple anatomical and physiological factors in addition to intestinal length. The location of the intestinal resection is particularly important, as removal of the distal small intestine often results in greater nutritional deficiencies than resection of proximal segments because of the ileum's essential role in the absorption of bile acids, vitamin B12, and other nutrients. Furthermore, persistent disease affecting the remaining bowel, absence of the colon or ileocecal valve, and previous gastric resection can significantly compromise digestive and absorptive function, thereby increasing the severity of intestinal failure.[2,24,25] Consequently, SBS is now recognized as a multifactorial clinical syndrome in which functional intestinal capacity, rather than residual bowel length alone, determines disease manifestations, nutritional requirements, and long-term therapeutic outcomes.

Intestinal Anatomy and Fluid and Nutrient Absorption

A comprehensive understanding of normal intestinal anatomy and the physiological mechanisms responsible for nutrient and fluid absorption is essential for appreciating the profound metabolic consequences associated with short bowel syndrome (SBS). The severity of malabsorption observed in SBS is determined not only by the quantity of intestine that has been removed but also by the specialized absorptive functions performed by different regions of the gastrointestinal tract. Because individual segments of the small intestine possess distinct structural and functional characteristics, surgical resection of specific portions results in predictable deficiencies involving nutrients, fluids, electrolytes, vitamins, and minerals. The principal clinical consequences of intestinal malabsorption in SBS include excessive fluid loss, electrolyte imbalance, hypokalemia, hypomagnesemia, hypoalbuminemia, disturbances in acid-base homeostasis, progressive weight loss, and multiple vitamin and mineral deficiencies, all of which contribute significantly to morbidity and reduced quality of life. The proximal small intestine, comprising the duodenum and jejunum, serves as the primary site for the digestion and absorption of carbohydrates and proteins. Following enzymatic digestion, carbohydrates are converted into monosaccharides and proteins into amino acids and small peptides, which are efficiently absorbed across the intestinal mucosa. Although the distal small intestine retains some capacity to absorb these macronutrients, the majority of nutrient uptake normally occurs within the proximal segments because of their extensive mucosal surface area and abundant digestive enzyme activity.[26,27] Consequently, preservation of adequate proximal intestinal length is important for maintaining sufficient caloric and protein absorption, although adaptive mechanisms within the remaining intestine may partially compensate for proximal losses over time.

Fat digestion and absorption involve considerably more complex physiological processes than those required for carbohydrates or proteins. Lipid absorption begins primarily within the proximal small intestine but depends on coordinated interactions among bile acids secreted by the liver and digestive enzymes, including pancreatic lipase and colipase, which hydrolyze dietary fats into

absorbable molecules. These processes facilitate the formation of micelles, allowing lipids to remain soluble within the aqueous intestinal environment and enabling efficient absorption by enterocytes.[26,28] Although lingual lipase secreted in the oral cavity and gastric lipase produced in the stomach contribute modestly to fat digestion, their overall role in healthy adults remains relatively limited compared with pancreatic enzymes.[29] Following intestinal absorption, bile acids are actively reabsorbed almost exclusively within the terminal ileum before being returned to the liver through the enterohepatic circulation for subsequent reuse.[30] Extensive resection involving more than 100 cm of the terminal ileum disrupts this recycling mechanism, resulting in depletion of the bile acid pool and failure to maintain the critical micellar concentration required for normal lipid absorption. Consequently, patients develop fat malabsorption, steatorrhea, and deficiencies of essential fatty acids and fat-soluble vitamins.[31] The absorption of fat-soluble vitamins, including vitamins A, D, E, and K, is closely dependent on normal lipid digestion and micelle formation. Therefore, impaired fat absorption frequently results in deficiencies of these vitamins, producing diverse clinical manifestations affecting vision, bone metabolism, immune function, blood coagulation, and antioxidant defense mechanisms. In contrast, most water-soluble vitamins and numerous essential minerals are absorbed predominantly within the proximal small intestine, where specialized transport mechanisms facilitate efficient nutrient uptake.[32,33] Vitamin B12 represents an important exception because its active absorption occurs almost exclusively within the terminal ileum following binding to intrinsic factor. Consequently, resection of this intestinal segment places patients at high risk for vitamin B12 deficiency, potentially leading to megaloblastic anemia and neurological dysfunction. Nevertheless, administration of high-dose oral vitamin B12, typically ranging between 1000 and 2000 µg daily, may permit sufficient passive absorption to maintain adequate vitamin status even in the absence of the terminal ileum.[34]

Fluid and electrolyte homeostasis depends on coordinated absorptive processes occurring throughout both the small intestine and colon. Water, sodium, potassium, chloride, and, to a lesser extent, amino acids are absorbed continuously along the intestinal tract, while the colon plays a particularly important role in reclaiming fluids and electrolytes that escape absorption in the small intestine.[35,36] Magnesium absorption occurs predominantly within the distal small intestine and colon, making patients with distal intestinal or colonic resection especially susceptible to hypomagnesemia.[32,33] Loss of these absorptive regions substantially reduces the body's ability to maintain hydration and electrolyte balance, often resulting in chronic dehydration, electrolyte depletion, metabolic disturbances, and increased dependence on intravenous fluid replacement.

Intestinal Adaptation

Following extensive intestinal resection, the gastrointestinal tract initiates a highly coordinated physiological process known as intestinal adaptation, which serves as a natural compensatory response aimed at restoring digestive and absorptive function. This adaptive process begins almost immediately after bowel resection and continues over an extended period, traditionally believed to exceed two years following surgery.[23] However, emerging evidence suggests that the intestine retains the capacity for further adaptive growth and functional enhancement several years after the initial operation when exposed to appropriate trophic stimuli.[37] The primary objective of intestinal adaptation is to maximize the absorptive efficiency of the remaining bowel, thereby reducing nutritional deficiencies and improving fluid balance. In many individuals, this compensatory process enables gradual reduction or complete discontinuation of parenteral nutrition (PN) and intravenous (IV) fluid support. Conversely, when adaptive mechanisms fail to sufficiently restore intestinal function to meet nutritional and hydration requirements, persistent intestinal failure develops, necessitating long-term nutritional support.[23] The magnitude and effectiveness of intestinal adaptation are influenced by numerous physiological and clinical factors. Among the most important determinants is the presence of luminal nutrients, as oral food intake provides continuous stimulation for mucosal growth and functional enhancement of the remaining intestine. Endogenous peptide hormones also play a critical regulatory role in promoting adaptation through stimulation of epithelial proliferation, enhancement of nutrient transport, and maintenance of mucosal integrity.[25,38] One of the most extensively studied mediators is glucagon-like peptide-2 (GLP-2), which has emerged as a central regulator of intestinal growth and repair. GLP-2 promotes expansion of the intestinal mucosa, increases absorptive surface area, reduces epithelial apoptosis, enhances intestinal blood flow, and improves barrier function, collectively contributing to greater absorptive capacity.[39–42] In addition to GLP-2, several other biologically active mediators participate in the adaptive response, including epidermal growth factor, growth hormone, cholecystokinin, gastrin, insulin, and neurotensin, each contributing through complementary mechanisms that support intestinal regeneration and functional recovery.[25]

Patient-related characteristics substantially influence the extent of adaptive remodeling following bowel resection. Younger individuals generally exhibit greater regenerative capacity and more robust intestinal adaptation than older adults because of enhanced cellular proliferation and tissue plasticity. Similarly, the absence of significant comorbid conditions allows physiological resources to be directed toward intestinal healing and regeneration, thereby improving adaptive outcomes.[25] Anatomical factors are equally important. The length of the residual intestine directly affects the available

absorptive surface, while the specific intestinal segments that remain after surgery determine the functional capabilities of the gastrointestinal tract. Evidence suggests that proximal bowel resections may stimulate a greater adaptive response in the remaining intestine than distal resections because nutrients that would normally be absorbed proximally are delivered to distal segments, providing enhanced luminal stimulation and promoting structural remodelling.[43,44] Adequate mesenteric blood flow is also essential because sufficient vascular perfusion ensures delivery of oxygen, nutrients, and regulatory hormones necessary for tissue regeneration. Furthermore, preservation of the ileocecal valve contributes to improved adaptation by slowing intestinal transit, limiting bacterial overgrowth, and increasing contact time between nutrients and the absorptive mucosa.[5,25] One of the most widely accepted concepts explaining intestinal adaptation is the "workload hypothesis," which proposes that exposure of the intestinal mucosa to enteral nutrients directly stimulates epithelial growth and functional maturation.[45] Experimental studies have consistently demonstrated that enteral nutrition promotes intestinal adaptation more effectively than exclusive parenteral nutrition. In contrast, prolonged reliance on parenteral nutrition without luminal nutrient exposure has been shown to suppress adaptive responses and contribute to mucosal atrophy.[46–49] The complexity of the enteral diet also appears to influence adaptation. Experimental models have demonstrated that diets containing more complex carbohydrates produce greater trophic effects than elemental nutrient preparations. For example, disaccharides stimulate larger increases in intestinal mucosal mass than monosaccharides in rodent studies.[50] Similarly, experimental investigations involving piglets undergoing bowel resection have shown superior weight gain and intestinal adaptation when animals receive polymeric formulas rather than elemental formulas.[51] These observations reinforce the fundamental principle of nutritional management in SBS, which advocates maximizing oral or enteral nutrition whenever clinically feasible while minimizing prolonged dependence on total parenteral nutrition.[52]

The adaptive response may also differ according to the anatomical region of the gastrointestinal tract that remains functional. The colon plays an especially important role in energy conservation following extensive small bowel resection. Studies have demonstrated that patients retaining a functional colon experience greater energy absorption, particularly when consuming diets rich in carbohydrates.[53] Carbohydrates escaping digestion and absorption within the small intestine undergo fermentation by the colonic microbiota, resulting in the production of short-chain fatty acids (SCFAs), including acetate, propionate, and butyrate. These metabolites serve as an additional energy source after absorption through the colonic epithelium and contribute significantly to nutritional compensation in individuals with severe intestinal loss.[53] Experimental research has further demonstrated that SCFAs, particularly butyrate, promote mucosal proliferation, enhance epithelial transporter expression, improve nutrient absorption, and reverse the intestinal atrophy associated with exclusive parenteral nutrition.[54–59] Similar trophic effects have been observed in neonatal piglets receiving butyrate-supplemented parenteral nutrition, supporting the role of SCFAs as important mediators of intestinal adaptation.[60] The administration of prebiotics may further augment these beneficial effects by selectively stimulating the growth of beneficial intestinal microorganisms such as bifidobacteria and lactobacilli, which ferment nondigestible carbohydrates to produce SCFAs, thereby supporting intestinal health and adaptive remodelling.[61,62] At the structural level, intestinal adaptation involves profound morphological and functional remodeling of the remaining bowel designed to maximize absorptive efficiency. Adaptive changes include hyperplasia of the intestinal epithelium, increased expression of epithelial transport proteins, enlargement of intestinal diameter, elongation of villi, deepening of crypts, and accelerated mucosal cell proliferation.[3,25,63–66] Although only minimal increases in overall bowel length occur, these structural modifications substantially expand the effective absorptive surface area and enhance the intestine's capacity to digest and absorb nutrients and fluids. Functional improvements accompany these anatomical changes through increased transporter activity, enhanced digestive enzyme expression, and improved nutrient uptake. Collectively, these adaptive mechanisms enable many patients to progressively reduce or discontinue parenteral nutritional support following intestinal resection. Nevertheless, in patients with intestinal failure secondary to SBS, these intrinsic adaptive responses remain insufficient to restore adequate absorptive capacity, resulting in persistent dependence on long-term parenteral nutrition and comprehensive multidisciplinary management to maintain nutritional status and overall health.[23]

Role of Parenteral Nutrition

Parenteral nutrition (PN) constitutes the cornerstone of long-term nutritional management for patients with short bowel syndrome (SBS) who fail to achieve sufficient intestinal adaptation to maintain adequate hydration, electrolyte balance, and nutritional status through oral or enteral intake alone. The principal objective of PN is to provide essential macronutrients, micronutrients, fluids, and electrolytes directly into the systemic circulation, thereby bypassing the compromised gastrointestinal tract. This life-sustaining therapy is particularly important for individuals with severe intestinal failure, in whom the remaining bowel lacks sufficient absorptive capacity to support normal metabolic requirements. Following extensive bowel resection, PN is generally initiated during the early postoperative period after stabilization of fluid and electrolyte disturbances. In contrast, patients with less extensive intestinal resections or those who retain a functional colon in continuity may

achieve adequate intestinal adaptation without requiring long-term PN, although some individuals develop progressive malnutrition and intestinal insufficiency months after surgery, necessitating delayed initiation of nutritional support.[67] The likelihood of achieving independence from PN depends largely on the extent of intestinal adaptation and the anatomical characteristics of the remaining bowel. A prospective study involving patients with nonmalignant SBS demonstrated that approximately 55% of patients successfully discontinued PN within five years of intestinal resection.[2] However, individuals who remained dependent on PN beyond two years, a condition defined as permanent intestinal failure, had only a 6% probability of eventually achieving nutritional independence.[2] Multivariate analyses have identified several important predictors of persistent PN dependence, including a residual small bowel length of less than 100 cm and the presence of an end-jejunostomy or jejunocolic anastomosis, both of which significantly reduce the absorptive capacity of the remaining intestine.[2] In addition to anatomical factors, biochemical markers have emerged as valuable tools for assessing intestinal function. Plasma citrulline, a nonessential amino acid synthesized primarily by enterocytes, reflects functional intestinal mass, and concentrations below 20 µmol/L have been strongly associated with continued PN dependence two years after bowel resection.[68]

Despite its indispensable therapeutic role, prolonged PN is associated with numerous complications that require vigilant monitoring and comprehensive multidisciplinary management. Central venous catheter-related bloodstream infections remain among the most serious adverse events, while vascular complications such as catheter occlusion and venous thrombosis may compromise long-term vascular access. Hepatic complications, particularly cholestasis and progressive liver dysfunction, also represent significant concerns during extended PN therapy.[6–9] Strategies aimed at minimizing these complications include meticulous catheter care, early introduction and maintenance of enteral nutrition whenever feasible, avoidance of excessive caloric administration, limitation of intravenous lipid emulsions, and gradual reduction or discontinuation of PN as intestinal adaptation progresses. Importantly, although PN-related complications contribute substantially to morbidity, mortality among patients receiving long-term PN is more frequently attributable to the underlying disease process or associated comorbidities rather than PN itself. Studies indicate that deaths directly related to PN complications account for approximately 9% to 33% of total mortality, with 50% to 71% of these deaths resulting from central venous catheter infections.[2,18,19,69] Long-term dependence on home parenteral nutrition (HPN) also imposes considerable financial and psychosocial burdens on patients and healthcare systems. In the United States, reimbursement policies often cover only a portion of HPN-related expenses and require fulfillment of specific eligibility criteria, leaving many patients responsible for substantial out-of-pocket costs.[70–72] Effective long-term management therefore requires continuous clinical surveillance to optimize nutritional status while preventing complications. Routine follow-up includes regular assessment of body weight, serum biochemical parameters, blood glucose concentrations, electrolyte status, liver function, and urine output, with maintenance of a daily urine volume of at least one liter serving as an important indicator of adequate hydration.[73,74] Oral fluid management also plays a critical supportive role because beverage composition significantly influences intestinal fluid absorption. Hypertonic fluids, including fruit juices and many commercial sports drinks, increase intestinal osmotic load and may aggravate diarrhea, whereas plain water and other sodium-free hypotonic fluids are inefficient for maintaining hydration in patients with SBS.[74–78] Instead, consumption of small, frequent volumes of slightly hypo-osmolar oral rehydration solutions containing appropriate sodium concentrations is recommended to maximize fluid absorption, improve hydration status, and reduce dependence on intravenous fluid supplementation.[74]

Social and Quality-of-Life Issues of Long-Term Parenteral Nutrition

Long-term dependence on home parenteral nutrition (HPN) has profound implications for the physical, psychological, and social well-being of individuals with short bowel syndrome (SBS). Numerous investigations have demonstrated that patients receiving prolonged HPN experience significantly lower quality of life than the general population when evaluated using generic health-related quality-of-life instruments.[11,79] Nevertheless, these broad assessment tools often fail to adequately capture the unique challenges associated with living with a technologically demanding therapy while simultaneously coping with the complex clinical manifestations of SBS. Until relatively recently, no validated disease-specific instruments existed to comprehensively evaluate the quality of life of individuals dependent on HPN. To address this limitation, Baxter and colleagues developed a specialized HPN-specific quality-of-life assessment tool capable of identifying patient-centered concerns, facilitating individualized clinical management, and recognizing areas requiring targeted intervention.[80] The implementation of such disease-specific instruments in clinical research has enhanced the ability to evaluate the impact of emerging therapeutic interventions on patient-reported outcomes. For example, individuals with SBS who experienced a reduction in parenteral nutrition requirements while receiving teduglutide demonstrated improvements in overall quality-of-life scores, although statistically significant differences between placebo-treated and actively treated groups were not consistently observed across all quality-of-life domains.[81] Importantly, patients consistently report that decreasing the duration of daily parenteral nutrition infusions provides greater freedom to participate in routine activities and enhances overall life satisfaction.[82]

Beyond the physiological consequences of SBS, patients receiving HPN must adapt to the continuous demands of sophisticated medical technology that substantially alters everyday living. Administration of HPN is a technically complex, time-intensive process requiring meticulous preparation, strict adherence to aseptic procedures, and careful coordination of daily routines around infusion schedules and nursing care.[83] These requirements frequently interfere with employment, family responsibilities, leisure activities, and social interactions, contributing to lifestyle disruption and social isolation. The persistent need to organize daily activities according to infusion schedules often reduces personal independence and may negatively influence emotional well-being, resulting in frustration, anxiety, and diminished self-esteem.[82,84] Furthermore, successful HPN administration requires adequate manual dexterity, substantial storage space for medical supplies, and continuous management of infusion equipment, all of which place additional practical burdens on patients and their caregivers. The presence of a permanent central venous catheter introduces further physical and psychosocial challenges. External infusion catheters frequently influence clothing choices, restrict participation in recreational activities such as swimming and contact sports, and complicate routine personal hygiene practices including bathing and showering.[83] Continuous vigilance is necessary to maintain catheter integrity and prevent bloodstream infections through meticulous aseptic technique.[85] Consequently, many patients develop persistent concerns regarding catheter malfunction, accidental dislodgement during sleep, physical exercise, sexual activity, or interactions with young children, as well as fears surrounding equipment failure and the possibility of life-threatening complications.[86] Difficulty adapting to these long-term lifestyle restrictions may contribute to feelings of helplessness, emotional distress, sadness, and psychological exhaustion, further compromising overall quality of life.[87–89]

The burden of SBS and HPN extends across multiple dimensions of daily living. Technology-related concerns commonly include dependence on others, continuous reliance on infusion equipment, disruption of travel and social activities, adherence to demanding therapeutic schedules, limitations in personal care, and the physical burden of carrying infusion pumps and nutritional solutions. Physical symptoms frequently encompass chronic diarrhea, generalized weakness, fatigue, disturbed sleep resulting from nocturnal infusions and frequent urination, altered body image, sexual dysfunction, peripheral edema, muscle cramps, bone pain, hair loss, depression, and persistent sensations of hunger or thirst.[11,77,82,91,94,98,128] Emotional concerns are equally significant and often include anxiety regarding future surgical procedures, fear of catheter-related infections, concerns about progressive liver dysfunction, loss of reliable venous access, catheter displacement, infusion pump malfunction, inability to maintain employment, financial hardship, and potential loss of health insurance coverage.[11,77,82,91,94,98,128] Collectively, these multidimensional challenges substantially influence psychological health and overall functional capacity. Parenteral nutrition is generally delivered using an ambulatory programmable infusion pump carried in a portable backpack containing both the infusion device and nutrient solution. Patients frequently identify the prolonged duration of daily infusions together with the physical weight of the infusion equipment as major contributors to treatment burden.[82] Fatigue commonly develops because overnight infusions may interrupt normal sleep patterns and increase nocturnal urination, thereby reducing sleep quality and daytime energy levels.[82,84,90–95] Some individuals also report irritation related to sleeping while connected to infusion equipment, particularly older devices that generate audible operational noise within the home environment.[85,89,96] Interestingly, pilot investigations comparing sleep quality during HPN infusion with periods without infusion found no major objective deterioration in sleep architecture attributable to HPN therapy.[97] Modern infusion pumps have largely eliminated noise-related disturbances through quieter operation, although some patients accustomed to earlier equipment may experience anxiety when silent pumps appear to function without audible confirmation.

The experience of eating undergoes a fundamental transformation for individuals living with SBS. Daily life often revolves around maintaining hydration, consuming frequent meals, and coping with repeated bowel movements. Many patients describe the necessity of "eating to survive," referring to the obligation to consume food despite anticipating abdominal pain, diarrhea, dehydration, weight loss, and progressive malnutrition.[98] Within this context, parenteral nutrition is commonly perceived as a life-sustaining intervention that provides nutritional security and relieves the constant pressure of meeting caloric requirements exclusively through oral intake. Consequently, patients are able to redefine eating as a means of supporting intestinal adaptation, maintaining health, and enjoying social interaction rather than merely ensuring survival. Participation in family meals, holiday celebrations, and the consumption of small quantities of preferred foods provide important psychological benefits, fostering pleasure, social connectedness, and improved quality of life while nutritional requirements continue to be fulfilled through parenteral support.[98] Effective clinical management therefore extends beyond correction of nutritional deficiencies to encompass comprehensive assessment of patient-defined quality of life, individual treatment goals, and personal expectations. Healthcare professionals should recognize that perceptions of well-being vary considerably among patients and should tailor interventions accordingly. Nutritional counseling, optimized pharmacological management of diarrhea and gastrointestinal symptoms, and individualized education regarding lifestyle adaptation may substantially improve

social participation and overall treatment satisfaction. Equally important is providing psychological support that enables patients to cope with the physical, emotional, and social consequences of chronic HPN dependence, thereby facilitating restoration of normal daily functioning.[82] Many individuals with chronic illness demonstrate remarkable resilience by redefining normalcy, adapting life priorities, establishing sustainable routines, and focusing on health rather than disability.[99] Patients frequently report greater psychological adjustment when they perceive HPN as a beneficial therapy that preserves independence rather than as a burden that restricts their lives.

Social support further enhances adaptation and long-term well-being. Interaction with individuals experiencing similar medical challenges provides emotional reassurance, practical guidance, and opportunities to share coping strategies. Participation in organizations such as The Oley Foundation has been associated with improved quality of life by promoting patient education, peer support, advocacy, and access to reliable information regarding long-term nutrition therapy.[100] Consequently, comprehensive multidisciplinary care should address physical symptoms, emotional distress, nutritional concerns, lifestyle modification, patient expectations, and opportunities to reduce or discontinue parenteral nutrition whenever clinically feasible, thereby maximizing both functional independence and health-related quality of life.

Current Management Options

The management of short bowel syndrome (SBS) requires a comprehensive, individualized, and multidisciplinary approach that aims to restore nutritional balance, optimize intestinal adaptation, prevent complications, and improve long-term quality of life. Therapeutic interventions are implemented in a stepwise manner according to the patient's clinical status and the stage of recovery following intestinal resection. During the immediate postoperative period, aggressive intravenous fluid resuscitation is essential to restore hemodynamic stability and correct severe fluid and electrolyte disturbances. In the early postoperative phase, total parenteral nutrition (PN) is initiated to provide adequate caloric intake, protein, vitamins, minerals, and fluids while minimizing metabolic stress on the recovering intestine. Simultaneously, clinicians focus on controlling diarrhea, correcting nutritional deficiencies, and gradually introducing oral fluids and enteral feeding to stimulate intestinal adaptation. As recovery progresses, many patients transition toward oral nutrition supplemented with oral rehydration solutions, whereas individuals with persistent intestinal failure continue to require long-term PN alongside ongoing nutritional monitoring, symptom management, and evaluation for advanced therapeutic interventions. Nutritional therapy remains the cornerstone of SBS management. Long-term PN and intravenous fluid administration are frequently scheduled during nighttime hours to improve patient mobility during the day, enhance nitrogen retention, and encourage increased oral food consumption by reducing daytime satiety.[101] Patients are encouraged to consume frequent meals and maintain hyperphagia to maximize nutrient absorption by the remaining intestine. Dietary modification also plays a critical role, with low-osmolarity and low-sugar diets recommended to reduce intestinal fluid losses and minimize dehydration.[67] Appropriate oral rehydration solutions containing balanced electrolyte concentrations are preferred over plain water or hyperosmolar beverages to optimize fluid absorption. Long-term supplementation with essential vitamins and minerals, including fat-soluble vitamins A, D, E, and K as well as vitamin B12, is necessary to prevent micronutrient deficiencies resulting from impaired intestinal absorption.[102]

Continuous clinical assessment for complications associated with SBS is equally important. Patients should be routinely evaluated for steatorrhea and small intestinal bacterial overgrowth, both of which contribute significantly to malabsorption and chronic diarrhea.[74] Bacterial overgrowth occurs most frequently in patients who retain the colon but have lost the ileocecal valve, allowing migration of colonic microorganisms into the small intestine.[74,103] Common manifestations include oily diarrhea, abdominal bloating, nausea, excessive gas production, and worsening malabsorption. Prompt recognition and appropriate antimicrobial therapy can substantially improve gastrointestinal symptoms and reduce fluid and nutrient losses. Medical treatment primarily targets symptom control while promoting intestinal adaptation. Antidiarrheal medications, particularly loperamide and diphenoxylate combined with atropine, remain first-line pharmacological agents for reducing intestinal motility and prolonging transit time, thereby improving fluid and nutrient absorption.[104,105] These medications are generally administered before meals and at bedtime, with individualized dosing according to clinical response. Although they effectively reduce stool output, they do not correct the underlying absorptive deficiency associated with SBS, emphasizing the need for therapies that directly enhance intestinal function and rehabilitation. Surgical intervention may be considered in carefully selected patients when conservative management alone is insufficient. Restoration of intestinal continuity following stoma formation can increase absorptive surface area, promote fermentation of undigested carbohydrates into short-chain fatty acids by the colon, and slow intestinal transit, thereby improving nutrient absorption.[53,106,107] However, this procedure may also increase the risk of bile acid-induced diarrhea, nephrolithiasis, and perianal complications, necessitating careful patient selection. For patients with dilated bowel segments, intestinal lengthening procedures such as the Bianchi procedure or serial transverse enteroplasty (STEP) increase functional bowel length without sacrificing additional intestine, thereby enhancing absorptive capacity.[108,109] In selected patients without

bowel dilatation, segmental reversal of a short bowel segment creates an antiperistaltic region that prolongs transit time and increases nutrient absorption.[109,110]

Intestinal transplantation remains the final therapeutic option for patients who develop life-threatening complications related to prolonged PN or who cannot be successfully managed using conventional rehabilitation strategies.[25] Indications include irreversible PN-associated liver failure, recurrent catheter-related bloodstream infections, repeated central venous thromboses, and severe recurrent dehydration despite optimal medical therapy. Although advances in transplantation techniques have improved outcomes, significant risks including graft dysfunction, rejection, infection, and mortality remain.[111] Long-term survival following transplantation continues to be lower than that observed among well-managed patients receiving home PN, although early referral before irreversible liver disease develops may improve outcomes.[69,112] Recent therapeutic advances have focused on pharmacological enhancement of intestinal adaptation. Early studies demonstrated that combinations of human growth hormone, glutamine, and specialized dietary therapy could temporarily improve nutrient and fluid absorption in patients with SBS.[113] Somatropin, a recombinant human growth hormone, subsequently became the first pharmacological agent approved for short-term treatment of SBS. However, its beneficial effects generally diminish after treatment discontinuation, limiting its long-term clinical utility.[114]

Greater therapeutic success has been achieved through targeting glucagon-like peptide-2 (GLP-2), an endogenous intestinotrophic hormone that promotes intestinal epithelial growth and functional adaptation.[39,40,115] Teduglutide, a recombinant GLP-2 analogue with a prolonged biological half-life, enhances villus height, increases crypt depth, improves mucosal integrity, and significantly augments intestinal absorptive capacity.[117–121] Phase III clinical trials have demonstrated that daily subcutaneous administration of teduglutide at 0.05 mg/kg substantially reduces parenteral nutrition volume requirements and the number of weekly infusion days, with some patients achieving complete independence from PN.[119,122,123] Gastrointestinal adverse effects, including abdominal pain, nausea, and vomiting, represent the most frequently reported side effects. Because GLP-2 stimulates epithelial proliferation, colonoscopic evaluation before treatment initiation and periodic surveillance thereafter are recommended to detect colorectal polyps or malignancy.[37,125,126] Teduglutide is therefore indicated for clinically stable adults with non-obstructive, nonmalignant SBS who remain dependent on parenteral nutrition despite optimized dietary, hydration, and medical management, representing a major advancement in the long-term rehabilitation of patients with intestinal failure.

CONCLUSION

Short bowel syndrome represents one of the most complex forms of chronic intestinal failure, requiring comprehensive, individualized, and evidence-based management to address the profound nutritional, metabolic, physiological, and psychosocial consequences of extensive intestinal loss. Disease severity is influenced by multiple interacting factors, including the length and anatomical location of the remaining intestine, preservation of the colon and ileocecal valve, underlying disease, and the patient's capacity for intestinal adaptation. These factors determine long-term nutritional requirements, dependence on parenteral nutrition, and overall prognosis. Although home parenteral nutrition remains the cornerstone of treatment for patients with permanent intestinal failure, its long-term use requires meticulous monitoring because of the risks of catheter-related infections, hepatic complications, metabolic abnormalities, and diminished quality of life. Modern intestinal rehabilitation strategies, including optimized enteral nutrition, individualized dietary modification, pharmacological agents such as teduglutide, and carefully selected surgical interventions, have significantly improved intestinal function while reducing dependence on parenteral nutrition in many patients. Nurses remain fundamental members of the multidisciplinary intestinal rehabilitation team through comprehensive assessment, early detection of complications, nutritional monitoring, patient and caregiver education, catheter management, psychosocial support, and coordination of long-term care. Patient-centered management that integrates medical, nutritional, surgical, psychological, and nursing interventions offers the greatest opportunity to improve functional independence, enhance quality of life, reduce healthcare utilization, and achieve sustainable long-term clinical outcomes for individuals living with short bowel syndrome.

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