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REVIEW ARTICLE

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SHORT BOWEL SYNDROME AND INTESTINAL FAILURE IN ADULTS: ANATOMY-DRIVEN PHENOTYPES, ADAPTATION BIOLOGY, AND CONTEMPORARY INTESTINAL REHABILITATION

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SAŽETAK

Short bowel syndrome (SBS) is the leading cause of chronic intestinal failure in adults and most commonly follows extensive small-bowel resection. The resulting loss of absorptive surface and regulatory feedback leads to a spectrum of malabsorption syndromes, ranging from compensated intestinal insufficiency to intestinal failure requiring long-term parenteral support. Clinical outcomes are determined more by postoperative anatomy than by residual length alone, particularly the presence of colon continuity and the preservation of the distal ileum and the ileocecal region. The pathophysiology of SBS integrates reduced absorptive capacity, accelerated transit, gastric hypersecretion, disordered bile acid handling, microbiome perturbation, and impaired enteroendocrine signalling. Intestinal adaptation, driven by luminal nutrients and trophic hormones such as GLP-2, can partially restore function over months to years, enabling stepwise reduction of parenteral support in selected patients. Modern care is centred on structured intestinal rehabilitation delivered by multidisciplinary teams, combining individualised fluid–electrolyte strategy, optimised oral/enteral nutrition, targeted pharmacotherapy to control secretion and motility, prevention and management of catheter-related complications, and selective reconstructive surgery. Disease-modifying therapy with GLP-2 analogues can reduce parenteral support requirements in SBS-associated intestinal failure, while transplantation remains reserved for refractory, life-threatening complications. This review summarises current concepts and evidence-based principles for diagnosis, phenotype stratification, rehabilitation, and long-term surveillance in adult SBS.

Keywords: short bowel syndrome; chronic intestinal failure; home parenteral nutrition; colonic continuity; intestinal adaptation; teduglutide; high-output stoma

INTRODUCTION

Short bowel syndrome is best understood as a functional disorder arising from a mismatch between intestinal absorptive capacity and the patient's physiologic needs. Although definitions based on residual length are often used pragmatically, contemporary guidelines emphasise a functional framework in which intestinal failure is defined by the need for intravenous supplementation of fluids, electrolytes, and/or nutrients to maintain health, particularly in metabolically stable patients managed outside the acute hospital setting (chronic intestinal failure) (1). This distinction matters clinically because the same postoperative anatomy may evolve from early dependence on parenteral support to partial or complete enteral autonomy as adaptation proceeds and rehabilitation strategies are implemented. European guidance underscores that chronic intestinal failure management requires complex technologies and a multiprofessional, multidisciplinary model, frequently delivered through home parenteral nutrition programs (2).

SBS accounts for most cases of chronic intestinal failure in adults. Other etiologies, such as intestinal dysmotility, mucosal disease, fistulizing disease, and mechanical obstruction, remain relevant, but SBS predominates in most registries and guideline frameworks (2). This dominance has driven the evolution of "intestinal rehabilitation" as a structured, staged care paradigm designed not only to replace what the gut cannot absorb, but to actively maximise remaining gut function, prevent complications of parenteral support, and improve long-term quality of life (3).

2. Definitions and contemporary classification

The term "short bowel syndrome" is widely applied when residual small-bowel length is approximately 200

cm or less, but the clinical implications of a given length depend strongly on which segments remain, whether the colon is preserved, and whether intestinal continuity has been restored (2). In parallel, the ESPEN framework distinguishes intestinal insufficiency from intestinal failure, with chronic intestinal failure representing a persistent state in metabolically stable patients requiring long-term intravenous supplementation (2).

The classification of intestinal failure is not purely semantic. It informs prognosis, selection of monitoring strategies, and allocation of therapies such as home parenteral nutrition (HPN), glucagon-like peptide-2 (GLP-2) analogues, and consideration of reconstructive or transplant options (4). European guidance explicitly frames the primary objective of chronic intestinal failure therapy as maintaining health through intravenous supplementation, frequently delivered as home parenteral nutrition (2).

3. Aetiology and clinical context of SBS in adults

Adult SBS most often follows resections for mesenteric ischemia, Crohn's disease, and complex surgical complications, with radiation enteritis, neoplasia-related resections, and trauma contributing in selected populations (5). Contemporary expert reviews emphasise that the phenotype after resection is shaped by the extent and location of disease, the number of prior operations, and the feasibility of restoring continuity, particularly the ability to include the colon in continuity (3).

4. Anatomy-driven phenotypes and prognostic thresholds

The postresection configuration is a major determinant of fluid balance, energy salvage, and the likelihood of achieving enteral autonomy. Classic clinical descriptions highlight two common adult phenotypes: patients with jejunum in continuity with a functioning colon and those with a jejunostomy, with the latter typically experiencing greater nutritional and fluid challenges because they lose the colon's absorptive and fermentative capacity (6). Modern intestinal failure practice often operationalises three anatomy types: end-jejunostomy, jejuno-colonic anastomosis (colon in continuity), and jejuno-ileo-colonic anastomosis (colon in continuity with some ileum preserved) (7).

Clinicians frequently use residual-length "thresholds" as probabilistic guides rather than absolute cutoffs. For end-jejunostomy, the residual length required to avoid chronic dependence on parenteral support is generally higher than for colon-in-continuity configurations, reflecting the loss of colonic water and sodium absorption and the absence of energy salvage from fermentation (3). This anatomy-based stratification is central to counselling and to designing rehabilitation plans, because it predicts whether the dominant challenge will be high-output fluid loss (typical for jejunostomy) or energy malabsorption with complex colonic fermentation issues (more common when the colon is preserved) (3).

5. Pathophysiology: integrated mechanisms of malabsorption and instability

5.1 Reduced absorptive surface and accelerated transit

The fundamental driver of SBS is the abrupt reduction in absorptive surface area for macronutrients, micronutrients, water, and electrolytes. This loss is compounded by reduced contact time between luminal contents and mucosa due to accelerated transit, particularly when the distal ileum and colon are removed and the "ileal brake" is lost. Contemporary expert guidance emphasises that symptom severity, especially diarrhoea and dehydration, reflects both malabsorption and dysregulated motility and secretion (2).

5.2 Gastric hypersecretion and high-output states

Gastric hypersecretion is a recognised early feature after massive small-bowel resection and contributes to increased fluid delivery to the small intestine, worsening diarrhoea and stoma output. Expert reviews in SBS management consistently prioritise acid suppression as a practical strategy in early phases and in patients with high-output stomas, while acknowledging the physiologic trade-off that gastric acid normally helps limit proximal bacterial overgrowth (8).

5.3 Segment-specific nutrient and bile acid handling

Loss of the terminal ileum impairs enterohepatic bile acid recycling and predisposes to fat malabsorption and steatorrhea; it also increases the risk of vitamin B12 deficiency due to loss of the site of intrinsic factor–B12 uptake. These segment-specific consequences are emphasised in clinical management frameworks because they determine monitoring priorities and supplementation strategies (3).

5.4 Microbiome perturbation, bacterial overgrowth, and D-lactic acidosis

Altered anatomy, impaired motility, blind loops, and medication exposures can promote small intestinal bacterial overgrowth, aggravating malabsorption and contributing to bloating, gas, abdominal discomfort, and diarrhoea. Reviews and expert guidance recognise that diagnostic accuracy is imperfect and that empiric or cyclic antibiotic strategies are used in practice when suspicion is

high, with recurrent disease being common (9). D-lactic acidosis is an uncommon but clinically important complication, typically associated with colon in continuity and carbohydrate malabsorption, where colonic fermentation generates D-lactate that can be systemically absorbed and cause episodic neurologic symptoms with metabolic acidosis. Recent clinical reports reinforce the mechanistic link between carbohydrate delivery to the colon and D-lactate generation in SBS (10).

6. The colon in continuity: fluid–electrolyte conservation, energy salvage, and endocrine adaptation

Preservation of colonic continuity is repeatedly identified as a favourable prognostic factor in SBS-associated intestinal failure because it materially increases net absorption of water and sodium chloride, thereby reducing the risk of dehydration, and enables energy salvage through fermentation of unabsorbed carbohydrates into short-chain fatty acids (SCFAs). Classic clinical syntheses describe the colon's contribution to energy balance via bacterial fermentation, with undernutrition risk being greater when the colon is absent (6, 7). A recent focused review highlights that colon continuity supports intestinal adaptation and influences microbiota composition and metabolism, with implications for both baseline physiology and responses to disease-modifying therapies such as GLP-2 analogues (7).

Endocrine signalling provides an additional explanation for the colon's beneficial role. The distal ileum and colon are rich in L-cells, which secrete glucagon-like peptide-1 (GLP-1), GLP-2, and peptide YY (PYY). These hormones collectively slow gastrointestinal transit, modulate intestinal secretion, and promote mucosal growth and adaptation. Preservation of this enteroendocrine signalling axis partly explains why patients with colon-in-continuity have a greater likelihood of reducing or even discontinuing parenteral support compared with those with an end-jejunostomy configuration (7).

7. Intestinal adaptation: biology, time course, and therapeutic leverage

Intestinal adaptation refers to structural and functional changes that enhance absorption in the remaining intestine after resection. While the magnitude of adaptation varies, the concept is foundational to rehabilitation: enteral nutrients provide luminal trophic stimulation, and enteroendocrine hormones and growth signals support mucosal remodelling and functional upregulation over months to years. Contemporary reviews emphasise that advances in understanding perturbed physiology have translated into improved care and outcomes, underscoring adaptation as a clinically actionable process rather than a passive phenomenon (11).

GLP-2 is central in this biology. Pharmacologic GLP-2 analogues exploit this pathway by enhancing absorptive capacity and reducing the need for parenteral support in appropriately selected patients with SBS-associated intestinal failure. The concept is now embedded in major guidelines and expert reviews as a disease-modifying option within multidisciplinary programs (3).

8. Clinical evaluation and longitudinal monitoring

Because SBS is dynamic, assessment must be longitudinal and phenotype-specific. Early management prioritises quantifying stool or stoma output and objectively assessing hydration status and renal perfusion, while long-term care integrates surveillance for micronutrient deficiencies, metabolic bone disease, renal complications, catheter-related complications, and intestinal failure–associated liver disease (IFALD). European Society for Clinical Nutrition and Metabolism (ESPEN) practical guidance emphasises structured clinical nutrition strategies and monitoring in chronic intestinal failure, reflecting the complexity and chronicity of care needs in this population (1).

Laboratory monitoring typically includes electrolytes, magnesium and phosphate, renal function, and markers relevant to nutritional status. Segment-specific risks require targeted testing, such as vitamin B12 after ileal resection and fat-soluble vitamins when fat malabsorption is suspected. Catheter-dependent patients require regular assessment for infection and thrombosis risk, while liver monitoring is central in chronic parenteral support (1).

9. Management principles: contemporary intestinal rehabilitation

9.1 Multidisciplinary model of care

Guideline-level consensus supports that chronic intestinal failure management requires multidisciplinary expertise, both to treat the underlying disease and to deliver home parenteral nutrition safely. This model typically integrates gastroenterology, surgery, nutrition, pharmacy, specialist nursing, and, where available, dedicated intestinal failure units that coordinate education, catheter care, and systematic reassessment for parenteral support reduction (2).

9.2 Fluid and electrolyte strategy

Fluid management is the most immediate determinant of stability in jejunostomy phenotypes. High-output states are characterised by large sodium and water losses that

cannot be corrected effectively by simply increasing free-water intake; expert guidance instead emphasises structured oral rehydration approaches and individualised intravenous supplementation when net absorption is insufficient (3). In colon-in-continuity phenotypes, the colon's capacity to absorb water and sodium can mitigate the risk of dehydration, but careful management remains necessary during intercurrent illness, dietary changes, or medication modifications (7).

9.3 Nutritional therapy: maximising enteral intake while using parenteral support judiciously

Nutrition strategies are individualised by anatomy. In colon-in-continuity SBS, complex carbohydrates can be particularly valuable because they supply substrate for SCFA production, supporting both energy salvage and potentially mucosal trophism, whereas excessive simple sugars may worsen osmotic diarrhoea and gas. In jejunostomy SBS, emphasis often shifts toward energy-dense intake, sodium supplementation, and strategies that reduce output and improve net absorption(6). Parenteral support remains lifesaving for intestinal failure, but modern rehabilitation aims to stabilise patients while creating conditions for gradual reduction when adaptation permits. ESPEN guidance positions home parenteral nutrition as a central therapy in chronic intestinal failure, provided within experienced programs that can minimise complications and reassess candidacy for reduction over time (2).

9.4 Pharmacologic control of secretion, motility, and overgrowth

Acid suppression is widely used to mitigate gastric hypersecretion, particularly early after surgery and in high-output states, and is explicitly discussed in expert guidance (3). Antimotility agents are used to prolong transit and improve absorptive contact time, while adjunct agents may be considered in refractory cases within specialist care pathways.
Bacterial overgrowth is managed pragmatically in many SBS programs with intermittent or cyclic antibiotics when clinical suspicion is high, acknowledging limitations of diagnostic testing and the frequent recurrence of symptoms (9).

9.5 GLP-2 analogue therapy as disease modification (teduglutide)

Teduglutide has the strongest evidence base among disease-modifying therapies for SBS-associated intestinal failure. A pivotal randomised controlled trial demonstrated that teduglutide reduces parenteral support requirements in patients with short bowel syndrome–associated intestinal failure (SBS-IF) (12). Earlier placebo-controlled trial data also supported reductions in parenteral support over 24 weeks (13). Long-term extension data and subsequent studies suggest sustained efficacy and a safety profile consistent with earlier findings, supporting ongoing reduction of parenteral support in responders under structured monitoring (14).
Expert reviews emphasise that GLP-2 analogue therapy should be embedded within an intestinal rehabilitation program, with deliberate, protocolized attempts to down-titrate parenteral support based on objective hydration and nutritional markers and with surveillance for adverse events (3).

10. Complications: prevention and management in long-term care

10.1 Catheter-related bloodstream infection and vascular access preservation

Catheter-related bloodstream infection is consistently described as the most common and clinically consequential complication of home parenteral nutrition in chronic intestinal failure (15). Contemporary reviews focus on standardised catheter-care protocols, education, and centre-level practices to reduce infection rates; recent analyses also explore nutrition delivery strategies as potential modifiers of infection risk (16).

10.2 Intestinal failure–associated liver disease (IFALD)

IFALD represents a spectrum of hepatic injury in patients receiving long-term parenteral nutrition. Reviews highlight that pathogenesis extends beyond parenteral nutrition itself and may involve inflammation, bile acid metabolism, dysbiosis, and bacterial overgrowth, suggesting that prevention and management require both nutrition optimisation and attention to gut-liver axis biology (17). Recent adult cohort work in home parenteral nutrition settings emphasises the prevalence of fibrosis and the need for systematic hepatic surveillance (18).

10.3 Renal complications and nephrolithiasis

Patients with SBS are at increased risk for renal complications, including dehydration-related kidney injury and nephrolithiasis. Hyperoxaluria-related stones are particularly associated with colon in continuity in the context of fat malabsorption, and recent work has examined oxalate burden and its clinical correlates in SBS (19). A 2022 study further supports the clinical relevance of urolithiasis risk in SBS cohorts (20).

10.4 Metabolic bone disease and micronutrient deficiency

Chronic malabsorption, vitamin D deficiency, disturbances in calcium and magnesium, and systemic

inflammation contribute to metabolic bone disease in chronic intestinal failure. ESPEN practical guidance incorporates monitoring and supplementation strategies within comprehensive chronic intestinal failure management (1).

11. Surgical rehabilitation and intestinal transplantation

Restoration of intestinal continuity, particularly reconnection of the small bowel to the colon when feasible, is a long-standing surgical principle in SBS care because it can improve fluid absorption and energy salvage, and it is emphasised in authoritative technical reviews (21). In carefully selected patients, reconstructive procedures that slow transit or improve functional surface area may be considered within specialised centres as part of intestinal rehabilitation. Intestinal transplantation is reserved for patients with irreversible intestinal failure who develop life-threatening complications of parenteral nutrition, such as recurrent severe catheter-related sepsis, loss of venous access, or progressive liver disease, consistent with guideline frameworks for chronic intestinal failure (2).

In keeping with contemporary intestinal rehabilitation concepts, the major postoperative phenotypes and management priorities of short bowel syndrome are summarised in Table 1.

Domain	Key determinant	What to assess in practice	Clinical implication	Take-home message
Postresection anatomy	Colon in continuity vs end-jejunostomy	Stool/stoma output volume, urine output, urine sodium, weight trend	Colon-in-continuity generally improves fluid and energy salvage and increases the probability of reducing parenteral support; end-jejunostomy is dominated by high-output losses and sodium depletion	"Length matters, but anatomy matters more"—colon in continuity is a major prognostic advantage
Residual ileum and ileocecal region	Presence of distal ileum/ileocecal valve	Vitamin B12 status, bile acid–related symptoms, transit pattern, SIBO risk features	Preserved ileum supports bile acid recycling and vitamin B12 absorption; ileocecal valve slows transit and may reduce proximal bacterial migration	Preserve/restore distal segments when feasible; anticipate B12 and bile acid issues when ileum is lost
Early postoperative physiology	Gastric hypersecretion and rapid transit	Timing after surgery, output pattern, response to acid suppression	Hypersecretion can amplify diarrhea/high-output states and worsen dehydration early; acid suppression often improves net balance during the high-risk phase	Treat the "secretory storm" early to stabilize fluids and enable rehabilitation
Fluid strategy	Hypotonic vs sodium-rich oral fluids	Fluid types and timing, serum/urine electrolytes, renal function	Large volumes of hypotonic fluids can increase output and worsen sodium loss in high-output states; sodium–glucose solutions improve net absorption	Target net absorption, not volume—use structured rehydration rather than free water
Nutrition and energy salvage	Carbohydrate delivery to colon and fat handling	Diet composition, stool characteristics, weight trajectory, micronutrients, stone history	Complex carbohydrates may support SCFA-mediated energy salvage with colon in continuity, whereas excess simple sugars may worsen osmotic diarrhea and gas; fat malabsorption can increase hyperoxaluria risk when colon is present	Match diet to anatomy: "feed the colon wisely" and manage fats to reduce symptoms and protect kidneys
Microbiome complications	SIBO and colonic fermentation	Bloating/gas, fluctuating output, steatorrhea, metabolic acidosis episodes	SIBO can worsen malabsorption and symptoms; D-lactic acidosis is a colon-in-continuity risk with carbohydrate malabsorption and episodic neurocognitive symptoms	Consider SIBO when symptoms are disproportionate; check D-lactate in episodic acidosis with neurologic features
Disease-modifying therapy	GLP-2 analogue responsiveness	Baseline PS volume, objective down-titration trials, adverse-event surveillance	GLP-2 therapy can reduce PS requirements in selected SBS-IF patients but is most effective within a structured rehabilitation program	GLP-2 is an amplifier of rehabilitation—measure benefit by safe PS reduction
Long-term safety of parenteral support	Catheter and liver complications	CRBSI history, venous access status, liver biochemistry and fibrosis assessment when indicated	Catheter infection/thrombosis and IFALD drive long-term morbidity and may determine escalation to surgical rehabilitation or transplantation	Protect the line and the liver—prevention is as important as nutrient delivery

Table 1. Practical, anatomy-driven determinants of short bowel syndrome–associated intestinal failure, with key clinical implications and take-home messages.

Note: Abbreviations: **CRBSI**, catheter-related bloodstream infection; **GLP-2**, glucagon-like peptide-2; **IFALD**, intestinal failure–associated liver disease; **PS**, parenteral support; **SBS**, short bowel syndrome; **SBS-IF**, short bowel syndrome–associated intestinal failure; **SCFA**, short-chain fatty acids; **SIBO**, small intestinal bacterial overgrowth.

12. Conclusions

SBS in adults is a prototypical cause of chronic intestinal failure and exemplifies the clinical importance of anatomy-driven physiology. Residual length alone cannot predict outcomes without considering the presence of colon in continuity, preservation of distal ileum and ileocecal function, and the balance between secretion and transit. Intestinal adaptation provides a biological basis for functional recovery, which can be therapeutically leveraged through optimised oral/enteral intake, targeted pharmacotherapy, and disease-modifying GLP-2 analogue therapy in selected patients. Long-term success hinges on multidisciplinary intestinal rehabilitation programs that minimise complications of parenteral support, preserve vascular access, monitor for IFALD and renal complications, and repeatedly reassess candidacy for reduction of parenteral support or surgical reconstruction. Transplantation remains an option of last resort for refractory, life-threatening complications.

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PREGLEDNI RAD

SINDROM KRATKOG CREVA I INTESTINALNA INSUFICIJENCIJA KOD ODRASLIH: ANATOMIJOM USLOVLJENI FENOTIPOVI, BIOLOGIJA ADAPTACIJE I SAVREMENA INTESTINALNA REHABILITACIJA

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SAŽETAK

Sindrom kratkog creva (SKC) predstavlja vodeći uzrok hronične intestinalne insuficijencije kod odraslih i najčešće nastaje nakon opsežne resekcije tankog creva. Nastali gubitak apsorptivne površine i regulatornih mehanizama dovodi do spektra sindroma malapsorpcije, u rasponu od kompenzovane intestinalne insuficijencije do intestinalne insuficijencije koja zahteva dugotrajnu parenteralnu nutritivnu podršku. Klinički ishod u većoj meri zavisi od postoperativne anatomije nego od same preostale dužine creva, a pogotovo od postojanja kontinuiteta debelog creva i očuvanja distalnog ileuma i ileocekalne regije. Patofiziologija SKC obuhvata smanjeni apsorptivni kapacitet, ubrzan intestinalni tranzit, gastričnu hipersekreciju, poremećaj metabolizma žučnih kiselina, promene crevne mikroflore i poremećenu enteroendokrinu signalizaciju. Intestinalna adaptacija, podstaknuta luminalnim nutrijentima i trofičkim hormonima poput GLP-2, može tokom meseci i godina delimično obnoviti funkciju, omogućavajući postepeno smanjenje potrebe za parenteralnom podrškom kod odabranih pacijenata. Savremeni pristup lečenju zasniva se na struktuiranoj intestinalnoj rehabilitaciji koju sprovode multidisciplinarni timovi uz primenu individualizovane strategije nadoknade tečnosti i elektrolita, optimizovane oralne i enteralne ishrane, ciljane farmakoterapije uz kontrolu sekrecije i motiliteta, prevencije i lečenja komplikacija vezanih za katetere, kao i selektivnih rekonstruktivnih hirurških zahvata. Terapija koja modifikuje tok bolesti primenom GLP-2 analoga može smanjiti potrebu za parenteralnom nutritivnom podrškom kod intestinalne insuficijencije povezane sa SKC, dok je transplantacija rezervisana za refraktorne, životno ugrožavajuće komplikacije. Ovaj pregledni rad sumira savremene koncepte i principe zasnovane na dokazima u dijagnostici, fenotipskoj stratifikaciji, rehabilitaciji i dugoročnom praćenju odraslih bolesnika sa SKC.

Ključne reči: sindrom kratkog creva, hronična intestinalna insuficijencija, kućna parenteralna ishrana, kontinuitet kolona, intestinalna adaptacija, teduglutid, stoma sa visokim izlazom