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Short Bowel Syndrome in Adults – Part 3

Hydrating the Adult Patient with Short Bowel Syndrome



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Fluid and electrolyte abnormalities are a major cause of morbidity in short bowel syndrome. When untreated or inadequately treated, these patients often suffer dehydration, fatigue, weight loss, and if prolonged, renal insufficiency that may be irreversible. The degree of fluid and electrolyte abnormalities varies depending upon the remaining bowel anatomy; specifically, the length and presence of remaining disease in the residual small intestine and the presence of a colon-in-continuity. Part three of this five-part series on short bowel syndrome is dedicated to the challenges involved in keeping the patient with short bowel syndrome hydrated. Strategies to improve hydration to prevent morbidity and enhance quality of life are presented.

INTRODUCTION

Achieving adequate hydration status (euvoemia) can be very difficult for patients with ileostomies.^{1,2} It is even more challenging for those with short bowel syndrome (SBS). Identifying dehydration is often straightforward in patients who have high output ostomies, as the clinician can estimate the volume of stool output by the number of times the patient empties their ostomy appliance in a 24 hour period (Clinical pearl: it is important to ask patients, “In a 24 hour period,” or “during the day, and then, what about at night?”—As clinicians may only get half the

story otherwise). In contrast, recognizing dehydration in a patient without an ostomy can be difficult, as the clinician cannot rely on the number of bowel movements per day to accurately quantitate the volume of output.

About 4 liters of fluid (0.5L saliva, 2L gastric acid and 1.5L pancreaticobiliary secretions) are normally secreted into the intestinal lumen each day in response to food and drink. Because of regional gut differences in water and sodium handling, SBS patients without a colon may be ‘net secretors’ (they lose more water and sodium from their stoma than they take in by mouth). In patients who have < 100cm of residual jejunum, daily jejunostomy output can be more than four liters per day.

Electrolyte disturbances are a major cause of morbidity in patients with SBS. In particular, those with an end-jejunostomy lose large amounts of sodium in the

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stool, often resulting in chronic sodium depletion and dehydration. It is imperative that clinicians teach SBS patients not only what to expect in terms of stool/ostomy output (see Table 1), but also the basic symptoms and risks of dehydration (see Table 2).

Fluid and electrolyte disorders predominate the early clinical course following massive intestinal resection. These issues may continue long term, particularly in patients without a colon who can suffer from substantial enteric volume loss resulting in severe dehydration, nephrolithiasis, renal insufficiency/failure,^{3,4} persistent metabolic acidosis, hypokalemia, hypomagnesemia, and hypocalcemia.

Assessment of Hydration Status

The initial evaluation of all SBS patients should include a history of weight change, medication usage, signs/symptoms of electrolyte deficiencies, gastrointestinal or other symptoms that may affect oral intake or fluid loss (nausea, vomiting, bloating, distension, etc.). The physical examination should also assess for malnutrition and signs of dehydration and nutrient deficiencies. Serial weight measurements are useful to track trends and serve as a warning of nutritional and/or hydrational compromise. It is imperative that SBS patients are instructed to measure and record their daily fluid intake and urine/stool output to help to guide fluid needs. Adequate hydration is considered to be present when urine output is > 1 L/day and urinary sodium concentration is > 20 mEq/L.⁴⁻⁶

The usual laboratory parameters to assess hydration such as serum sodium, creatinine, and blood urea nitrogen are unreliable in SBS as they become abnormal only after severe dehydration occurs. This is due to the normal homeostatic mechanisms including elevation of plasma renin and secondary hyperaldosteronism that occur in response to a subtle decrease in serum sodium or blood volume. Sodium, and hence water, are avidly conserved by the kidney, so a rise in BUN to creatinine ratio is a late response and only occurs after the patient is significantly dehydrated.⁷

Management of Dehydration

Patients (and some health care providers) often believe that large quantities of water should be ingested to make up for stool losses in the setting of SBS. This misconception, however, generally leads to increased ostomy outputs and creates a vicious cycle that further

Table 1. Typical Ostomy/Stool Output/ 24 Hours with Each Type of Resection⁸

- Normal stool output = 200mL
- Colostomy = 200-600 mL
- Jejunostomy = up to 6000mL
- Ileostomy = > 1200 mL (post-op; decreasing to an average of 750mL)
 - Ileostomy output is considered “diarrhea” when losses are > 1000mL/day

exacerbates fluid and electrolyte disturbances. Patients are often surprised to find that stool/ostomy output is significantly reduced following a twenty-four hour trial where they ingest only appropriate solids and NO oral fluids (IV fluids may be needed during the trial to prevent dehydration).

In SBS patients with excessive thirst due to dehydration, oral fluids should be restricted to < 1500mL/day and supplemental intravenous hydration provided to maintain euolemia.⁸ SBS patients may benefit from substituting regular beverages/fluids with glucose-electrolyte oral rehydration solution (ORS) to enhance intestinal absorption and reduce secretion (see ORS section below).

The ability to maintain euolemia while ingesting common oral liquids is often dependent upon the presence or absence of a colon. Most SBS patients with a colon can tolerate ingesting hypotonic fluids. They can usually maintain adequate hydration and sodium balance without excessive fluid loss.⁹ Patients without a colon often require additional sodium (~ 90mEq or ~ 2 g sodium (7/8 teaspoon of table salt) *per liter of stool lost*—in enterally fed patients, the sodium content of the formula should be brought up to ~ 90-100mEq/liter⁷ (1/4 teaspoon table salt = 600mg/26mEq of sodium) if no other sodium source is available.¹⁰

Special Considerations

Hypomagnesemia

Like chronic sodium depletion, hypomagnesemia can also be problematic in SBS. It occurs as a consequence of multiple factors including malabsorption of magnesium that is exacerbated by the binding of magnesium by unabsorbed fatty acids and increased renal excretion due to sodium/water depletion (and the

Table 2. Potential Signs, Symptoms and Risk Factors of Dehydration

- Urine output < 1000 - 1200mL) per day
- Rapid weight loss
- Reduced frequency of urination
- Darker urine color
- Ostomy or stool output > 2 liters per day
 - Stool output that is more than the total amount of fluid consumed
- Kidney stones (especially oxalate in those with remaining colon)
 - In patients who have formed kidney stones, it is important to maintain a urine output of 1500-2000 mL
- Worsening kidney function
- Feeling tired all the time
- Hypotension
- Thirst! Dry mouth, sticky or thick saliva
- Lightheadedness on standing

hyperaldosteronism that follows). The major clinical manifestations include tetany, tremor, weakness, apathy, convulsions, coma, and electrocardiographic abnormalities. Hypomagnesemia may contribute to hypocalcemia as a result of impaired parathyroid hormone (PTH) release.¹¹ Hypokalemia occurs in nearly half of those with chronic hypomagnesemia. The correction of sodium depletion is critical in treating hypomagnesemia. Measurement of urinary sodium may assist in the assessment of sodium balance in some patients; a random urinary sodium concentration of < 20 mEq/L is generally a good indicator of sodium depletion.

Oral magnesium salts can be administered in doses of 12-24 mEq/day and do not appear to increase stomal output, particularly when taken at night when intestinal transit is at its slowest. Higher doses are frequently needed, however, and may be difficult to use due to the laxative effects of oral magnesium causing a worsening of diarrhea. Magnesium heptogluconate is available as a liquid that may be added to an ORS (see section below) at a dose of 30 mEq/L. The oral administration of 1 α -hydroxycholecalciferol may also be useful as it can increase both intestinal absorption and renal

Table 3. Common Hypotonic and Hypertonic Fluids²⁶⁻²⁸

Beverage	(mOsm/kg)
Hypotonic fluids (< 300mOsm/kg)	
Water, purified, mineral tap	0-28
Sugar free tea, iced tea	13-44
Coffee, black	28-53
Sugar free:	
• Sodas, Lemonade, Punch, Kool-Aid®	13-44
Hypertonic Fluids (> 300mOsm/kg)	
Malted milk	940
Ice cream	1905
Eggnog	695
Fruit yogurt	871
Sherbet	1225
Popsicles	720
Ensure®/Boost®	590/640
Ensure® Plus/Boost® Plus	680/720
Resource® Breeze	750
Enteral formulas	250-700
Prune juice	1265
Grape juice	863
Apple/ orange juice	683/ 614
Tomato juice	595
Punch with sugar	448
Broth	445
Flavored gelatin	735
Energy drinks, Red Bull®, etc.	673-1030
Sodas, fruit drinks or other sugary drinks	537-1112

absorption of magnesium.¹² If moderate to severe hypomagnesemia (< 1 mg/dL) persists, parenteral magnesium sulfate may be necessary. Intravenous magnesium replacement should be given over 8-12 hours (rather than the usual IV piggy back bolus over 1-4 hours) to prevent significant renal excretion when the renal threshold is exceeded.¹³

Metabolic Acidosis Bicarbonate

Metabolic acidosis may arise from excessive gastrointestinal bicarbonate loss. The acidosis may be

Table 4. Commercial Oral Rehydration Solutions (ORS)²⁹

Solution	Glucose (CHO) (g/L)	Sodium mEq/mg per liter	Potassium mEq/mg per liter	Citrate mEq/mg per liter	Osmolarity mOsm/L	Calories/liter	Company
WHO packet	20	90/ 2070	20/ 780	30	330	152	Jianas Brothers (816)421-2880 jianasp@aol.com
Reduced osmolarity	13.5	75/ 1725	20/ 780	30	245	126	
Rehydralyte®	25	75/ 1725	20/ 780	30	310	172	Abbott Nutrition (800)227-5767 abbottnutrition.com
Pedialyte®	25	45/ 1035	20/ 780	30	250	100	
EquaLyte®	25	78/ 1794	22/ 858	30	305	172	
Parent's Choice Pediatric Electrolyte	20	45/ 1035	20/ 780	n/a	262	100	Wal-Mart Brand
CeraLyte® 70	40	70/ 1610	20/ 780	30	< 260	160	CeraLyte® ceralyte.com (888)237-2598
CeraLyte® 90	40	90/ 2070	20/ 780	30	< 275	160	
DripDrop®	33	58/ 1330	20/ 780	160/251	235	130	DripDrop dripdrophydration.com (800)761-0321

Note: Citrate = about 2.4 calories per gram

further compounded by impaired renal homeostasis caused by profound salt and water depletion. Relevant to the SBS patient, chronic acidosis can lead to bone resorption and osteopenia, aggravation of secondary hyperparathyroidism, increased protein catabolism, reduced respiratory reserve and malaise.^{14,15} Metabolic acidosis can be detected on laboratory testing by the finding of low serum bicarbonate (or CO₂). SBS patients can have either a normal anion gap or hyperchloremic metabolic acidosis. In patients with metabolic acidosis, alkali therapy (usually with oral sodium bicarbonate) is used to maintain the serum bicarbonate concentration in the normal range. A bicarbonate solution such as bicitra may prove beneficial over sodium bicarbonate tablets due to the sheer number of tablets needed for the equivalent amount of bicarbonate in bicitra.¹⁵ Occasionally, parenteral alkali therapy may be needed.

D-lactic acidosis

D-lactic acidosis is a rare neurological syndrome associated with SBS that is characterized by altered mental status ranging from confusion to coma, slurred speech, seizures and ataxia. D-lactic acidosis results

from bacterial fermentation of unabsorbed carbohydrates seen in SBS patients, particularly children, with a remaining colon.¹⁶ Development of this syndrome requires carbohydrate malabsorption with increased delivery of nutrients to the colon, ingestion of a large amount of carbohydrate (usually concentrated sweets), microbes that produce D-lactate and impaired D-lactate metabolism. Excessive production of D-lactate occurs when abnormal gut microbes overwhelm the normal metabolism of D-lactate and results in the accumulation of this substance in the blood.¹⁷ Because measurement of D-lactate requires a special laboratory request (unlike L-lactate), a high level of suspicion is needed. This condition should be considered when an anion-gap metabolic acidosis with normal lactate (L-lactate) level is present in the SBS patient with a colon-in-continuity and typical clinical manifestation. Although the optimal treatment of this condition is unclear, options include a carbohydrate (sugar)-restricted diet and the use of antibiotics to reduce the production of D-lactate producing gut microbes.

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*(continued from page 14)***Fluid Options for Those with SBS**

SBS patients can lose large volumes of fluids and electrolytes due to diarrhea/high ostomy output and, occasionally, from the presence of decompressive gastric/enterostomy tubes. Fluids should be given to cover all losses and maintain a urine output of at least 1 L/d. The sodium and glucose content of the fluid are important considerations, as inappropriate fluids will exacerbate fluid losses in SBS. Hyperosmolar fluids

(e.g., regular soda and fruit juices) are concentrated and induce secretion from enterocytes in an attempt to dilute the concentration of the luminal contents, which then contributes to increased diarrhea. In contrast, hypo-osmolar fluids (e.g., water) do not contain the sodium or glucose necessary to optimally facilitate absorption in an end-jejunosomy patient and may lead to dehydration if consumed in large amounts as they pull sodium (and hence water) into the lumen. In the normal subject, when water or other solutions with

Table 5. Sample Homemade Oral Rehydration Recipes

Sugar and salt water	1 quart water ¾ teaspoon salt 6 teaspoons sugar Optional: Crystal Light® to taste (especially lemonade or orange-pineapple flavors)		
World Health Organization Reduced Osmolality Oral Rehydration Therapy (Home Solution)	Need 1 liter container ½ teaspoon table salt ¼ teaspoon Morton® Salt Substitute (note caution in those with hyperkalemia) ½ teaspoon baking soda 2 tablespoons table sugar Add tap water to make one liter Directions: Use household measuring spoons, not silverware, to measure the ingredients. To the one liter container, add about 1/2 of the needed water. Add the dry ingredients, stir or shake well; add the remaining water to make a final volume of one liter. Add Nutrasweet® or Splenda® based flavoring of choice if desired. Best if chilled. Sip as directed. Discard after 24 hours.		
Gatorade® G2	4 cups Gatorade® G2 (or one, 32 ounce bottle) ¾ teaspoon salt		
Chicken Broth	4 cups water 1 dry chicken broth cube ¼ teaspoon salt 2 tablespoon sugar	OR	2 cups liquid broth 2 cups water 2 tablespoon sugar
Tomato Juice	2 ½ cups tomato juice 1 ½ cups water		
Homemade Cereal Based	½ cup dry, precooked baby rice cereal 2 cups water ¼ teaspoon salt Directions: Combine ingredients and mix until well dissolved and smooth. Refrigerate. Solution should be thick, but pourable and drinkable.		

➤ For more home-made “oral rehydration-like” solutions using alternative beverage base access: Parrish CR. A Patient’s Guide to Managing a Short Bowel. 3rd ed., 2015 Available free @ www.shortbowelsupport.com

a sodium concentration < 60-90mEq are consumed, sodium (and hence water— as water follows sodium) is secreted into the intestinal lumen during passage through the duodenum and jejunum in an effort to equilibrate the concentration gradient differences. The sodium is normally reabsorbed in the distal small bowel; however, in those with an end jejunostomy, both sodium and fluid are lost in the stool.¹⁸ See Table 3 for examples of both hypertonic and hypotonic fluids.

Oral Rehydration Solution (ORS)

The rationale to include sodium in oral rehydration solutions is to replace sodium losses and to promote water absorption. Water movement in response to a water gradient is about nine-times greater in the upper small bowel than in the distal small bowel.¹⁹

As the jejunum is more permeable to small molecules, osmolality makes a difference in fluid flux in this area and is the basis for use of ORS. Intestinal luminal sodium and glucose play important roles in promoting fluid absorption.^{20,21} Glucose in the gut lumen stimulates sodium absorption across the small intestine, which is followed by anions and water. For each cycle of this transport, two sodium ions and one molecule of glucose/galactose are transported together across the cell membrane and hundreds of water molecules move into the epithelial cell. Absorption of sodium occurs by 3 different mechanisms across the GI tract epithelium:

1. Passive absorption; probably through the intercellular junctions of the mucosal cells,
2. Active absorption of sodium, mediated by

the sodium-potassium pump, and

3. Glucose-coupled transport of sodium (most active in the jejunum).

Oral rehydration solutions are efficacious because they utilize the glucose-coupled transport system. Use of an ORS has been shown to enhance water and sodium absorption in patients with SBS,^{10,19,22-24} and it has allowed some patients to discontinue supplemental parenteral fluid support. The optimal sodium concentration of ORS to promote jejunal absorption has been demonstrated to be 90-120 mEq Na⁺/L²⁵ (with optimum carbohydrate: sodium ratio of 1:1).⁹

While ORS therapy has been extremely successful in the treatment of diarrheal illnesses worldwide, it is not a panacea, and in some SBS patients, it too can increase stool output.²² Furthermore, some patients may find it unpalatable. To hydrate some patients, 2-3 liters per day may be required, however, would start with the goal of 500-1000 mL per day. If the patient will sip ORS over the course of a day and is willing to maintain this regimen day after day, the volume can be titrated as needed. To improve palatability, ORS can be made into ice cubes or popsicles. Both homemade recipes and commercial preparations are available (see Tables 4 & 5). If better hydration is achieved with use of an ORS, then it can be continued indefinitely; however, if output is increased without net gain of increased urine output, then it should be stopped. ORS has also been administered via a gastrostomy tube as a nocturnal infusion with success.²⁴ ORS should not be substituted with commercial sports drinks as sports drinks contain considerably higher carbohydrate and lower sodium

Table 6. Fluid Options for Those with Short Bowel Syndrome

Best Choices	Worth Trying Small Amounts of*	Will Aggravate Stool Output
<ul style="list-style-type: none"> • Oral rehydration solutions • See Tables 4 & 5 • Ceralyte® “jello” 	<ul style="list-style-type: none"> • G2 Gatorade or sport drink equivalents • Milk – 1% or 2% may be better in some • Broth or broth-based soups • Sugar free sodas • Diluted fruit juices, specifically grape, orange, pineapple/drinks • ½ strength jello (add twice as much water as called for in recipe) • Some water 	<ul style="list-style-type: none"> • Too much water • Fruit juice • Fruit-like beverages • Koolaid • Coffee, tea • Sweet tea • Ensure, Boost or equivalent product • Sodas • Alcohol

*NOT the same as oral rehydration solutions, but better than those in column 3

content than ORS. Potassium and magnesium may be added to the ORS as gluconate (12mmol/L of ORS) and heptogluconate (30 mmol/L of ORS) salts, respectively, where available. Realistically, despite our best efforts, there are some patients who just will not drink ORS. In those cases, it is best to at least give suggestions for better options (or the least “bad” options), rather than those that will definitely aggravate stool/ostomy output (see Table 6).

Parenteral Fluid

In some SBS patients following the acute stage, parenteral fluids without macronutrients may be needed for those who require the fluid, but not the calories. If a patient cannot maintain a urine output of > 1 liter daily, then supplemental parenteral fluids may be needed. Intravenous fluid is commonly provided as a liter of normal saline infused over 2 to 4 hours once daily as needed. Although the content of the fluid may include only sodium chloride, occasionally dextrose, other electrolytes, vitamins and bicarbonate may be added. Parenteral fluids will be necessary if the stool output consistently exceeds fluid intake (‘net secretors’), a situation most commonly seen in the SBS patient with an end-jejunostomy who has < 100cm of jejunum and an output of > 2 liters/day. As mentioned earlier, ORS may be administered via a gastrostomy tube as a nocturnal infusion.¹⁸ During the hot summer months, patients receiving parenteral nutrition overnight may require additional parenteral hydration during the day to prevent dehydration and reduce potential injury to the kidneys. Parenteral fluids may also be needed in the SBS patients who have successfully weaned from PN but still require occasional parenteral fluid support.

CONCLUSION

Maintaining hydration status is a central component in the care of the patient with SBS. Failure to do so can result in dehydration, rapid weight loss and fatigue. If chronic and untreated, it can also lead to nephrolithiasis and jeopardize renal function. Educating patients to identify signs of dehydration as well as to properly instruct them on measures to protect against it should be a high priority to clinicians taking care of these patients. ■

References

- Nagle D, Pare T, Keenan E, et al. Ileostomy pathway virtually eliminates readmissions for dehydration in new ostomates. *Dis Colon Rectum*. 2012;55(12):1266-72.
- Paquette IM, Solan P, Rafferty JF, et al. Readmission for dehydration or renal failure after ileostomy creation. *Dis Colon Rectum*. 2013;56:974-979.
- Banerjee A, Warwicker P. Acute renal failure and metabolic disturbances in the short bowel syndrome. *QJM* 2001;95(1):37-40.
- Lauverjat M, Hadj Aissa A, Vanhems P, et al. Chronic dehydration may impair renal function in patients with chronic intestinal failure on long-term parenteral nutrition. *Clin Nutr*. 2006;25(1):75-81.
- DiBaise JK, Matarese LE, Messing B, et al. Strategies for weaning parenteral nutrition in adult patients with short bowel syndrome. *J Clin Gastroenterol* 2006;40(Suppl):S94-98.
- O’Neil M, Teitelbaum DH, Harris MB. Total body sodium depletion and poor weight gain in children and young adults with an ileostomy: a case series. *Nutr Clin Pract*. 2014;29(3):397-401.
- Nightingale JMD. Management of a high-output jejunostomy. In: Nightingale JM (Ed). *Intestinal failure*. Greenwich Medical Media Limited. London, England; 2001:375-392.
- Hughes S, Myers A, Carlson G. Care of the Intestinal Stoma and Enterocutaneous Fistula. In: Nightingale JM (Ed). *Intestinal failure*. Greenwich Medical Media Limited. London, England; 2001:53-63.
- Kelly DG, Nadeau J. Oral rehydration solution: a “low-tech” oft neglected therapy. *Nutr Issues Gastroenterol* 2004;28:51-62.
- Nightingale JMD, Lennard-Jones JE, Walker ER, et al. Oral salt supplements to compensate for jejunostomy losses: comparison of sodium chloride capsules, glucose electrolyte solution, and glucose polymer electrolyte solution. *Gut* 1992;33:759-761.
- Anast CS, Winnacker JL, Forte LR, et al. Impaired release of parathyroid hormone in magnesium deficiency. *Clin Endocrinol Metab* 1976;42:707-717.
- Fukumoto S, Matsumoto T, Tanaka Y, et al. Renal magnesium wasting in a patient with short bowel syndrome with magnesium deficiency: effect of 1 alpha-hydroxyvitamin D3 treatment. *J Clin Endocrinol Metab* 1987;65:1201-1204.
- Karosanidze T. Magnesium – So Underappreciated. *Practical Gastroenterology* 2014;XXXVIII(1):28.
- Mitch WE. Influence of metabolic acidosis on nutrition. *Am J Kidney Dis* 1997;29(5):xlvi-xlviii.
- Rosner M. Metabolic Acidosis in Patients with Gastrointestinal Disorders: Metabolic and Clinical Consequences. *Practical Gastroenterology* 2009;XXXIII(4):42.
- Petersen C. D-lactic acidosis. *Nutr Clin Pract* 2005;20:634-645.
- Hove H, Mortensen PB. Colonic lactate metabolism and D-lactic acidosis. *Dig Dis Sci* 1995;40:320-330.
- Lennard-Jones JE. Review article: practical management of the short bowel. *Aliment Pharmacol Ther*. 1994;8(6):563-77.
- Lennard-Jones JE. Oral rehydration solutions in short bowel syndrome. *Clin Ther* 1990;12(Suppl A):129-37.
- Lin R, Murtazina R, Cha B, et al. D-glucose acts via sodium/glucose cotransporter 1 to increase NHE3 in mouse jejunal brush border by a Na⁺/H⁺ exchange regulatory factor 2-dependent process. *Gastroenterology* 2011;140:560-571.
- Elliott EJ, Cunha-Ferreira R, Walker-Smith JA, et al. Sodium content of oral rehydration solutions: a reappraisal. *Gut*. 1989;30(11):1610-21.
- Newton CR, Gonvers JJ, McIntyre PB. Effect of different drinks on fluid and electrolyte losses from a jejunostomy. *J R Soc Med*. 1985;78(1):27-34.
- Beaugerie L, Cosnes J, Veraerde, et al. Isotonic high-sodium oral rehydration solution for increasing sodium absorption in patients with short bowel syndrome. *Am J Clin Nutr* 1991;53:769-772.
- Nauth J, Chang CW, Mobarhan S, et al. A therapeutic approach to wean total parenteral nutrition in the management of short bowel syndrome: three cases using nocturnal enteral rehydration. *Nutr Rev*. 2004;62(5):221-31.
- Rodrigues Ca, Lennard-Jones JE, Thompson DG, et al. What is the ideal sodium concentration of oral rehydration solutions for short bowel patients. *Clin Sci* 1988;74:69.
- Parrish CR. Enteral Feeding: The Art and the Science. *Nutr Clin Pract* 2003;18(1):76-85.
- Bell S, Anderson FL, Bistran BR, et al. Osmolality of Beverages Commonly Provided On Clear and Full Liquid Menu. *Nutr Clin Pract* 1987;2:241-244.
- Dini E, De Abreu J, López E. Osmolality of frequently consumed beverages. *Invest Clin*. 2004;45(4):323-35.
- Parrish, CR. *A Patient’s Guide to Managing a Short Bowel*, 2nd Edition. Growth, Inc., Newark, DE; February 2013:1-65.