S
hort-bowel syndrome (SBS) refers to the various clinical signs that may result from removal of a large part of the small intestine with or without removal of part of the large intestine. SBS is common in humans and, especially, infants but is uncommon in dogs and cats because most animals that need large portions of intestine removed are euthanized. The decreased absorptive surface area of the small intestine due to extensive resection often results in inadequate digestion and absorption of nutrients and water as well as electrolyte imbalance and micro- and macroelement deficiencies. 

CAUSES

Some of the most frequent intestinal conditions that may necessitate removal of extensive segments of the small intestine in dogs and cats are presented in the box on page 183.

In humans, the main underlying disorders leading to intestinal resection include mesenteric infarction, severe Crohn's disease, radiation enteritis, tumors and congenital abnormalities in infants (e.g., gastroschisis, intestinal atresia, malrotation), and other conditions (e.g., midgut volvulus, necrotizing enteritis).

PATHOPHYSIOLOGY

The small intestine extends from the pylorus to the ileocolic valve. The function of the small intestine is to digest and absorb mainly nutrients and to move its contents without interruption in an aboral direction. The small intestine is divided anatomically into three arbitrary parts: the duodenum, jejunum, and ileum. Within the small intestine, these different parts have different functions. Extensive small intestine resections may lead to impaired digestion and absorption as well as hormone and peptide depletion. Gastric acid hypersecretion, vitamin

ABSTRACT: Short-bowel syndrome (SBS) is a state of malabsorption and malnutrition that usually results from extensive small intestinal resection and infrequently occurs in dogs and cats. The most common clinical signs include persistent watery diarrhea, polyphagia, weight loss, and steatorrhea. In time, the intestinal surface undergoes histologic and functional changes, known as intestinal adaptation, to keep the patient in nutritional balance. Total parenteral nutrition is often necessary in the early postoperative period until oral feeding can counterbalance the nutritional needs of the patient. Aggressive medical management should also be instituted in the early stages. The prognosis for patients with SBS is variable.

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D deficiency, cholesterol crystals, gallstones, kidney stones, and D-lactic acidosis are among the main complications of SBS in humans.

**Anatomy and Physiology of the Small Intestine**

The average length of the small intestine is three and a half times the length of the body. The small intestine measures approximately 80 cm to 1.3 m in cats and 1.8 to 4.8 m in dogs. In dogs, the duodenum is approximately 25 cm and the ileum is approximately the last 15 cm. In cats, the duodenum is approximately 10 cm and the jejunum and ileum are 70 cm to 1.2 m. The luminal surface area of all three parts is covered by the intestinal mucosa, which is characterized by villi, crypts, and absorptive cells with microvilli, local endocrine cells, and other histologic features. Nevertheless, there is a distinct difference in the morphology and function of the enterocytes of the proximal to the distal part of the small intestine. The villi are higher, the crypts are deeper, and the junctions between the epithelial cells are considerably looser in the jejunum than in the ileum, all of which increase the absorptive ability of the jejunum.

Protein, fat, carbohydrate, trace elements, vitamins, and water are mainly absorbed in the jejunum, especially within the first half of its length. The remaining surface of the jejunum functions as a reserve. The ileum is the site of absorption of bile acids and vitamin B₁₂ and secretion of specific hormones that regulate the motility and adaptation of the small intestine (Figure 1).

The normal physiologic functions of the small intestine include digestion of food; absorption of necessary nutrients, along with water and electrolytes; propulsion and mixing of food through the intestinal lumen; and production of gastrointestinal (GI) enzymes and site-specific hormones that regulate the proper work of the GI tract. The whole process of digestion begins in the stomach, where gastric secretions act on food and the forming chyme gradually enters the duodenum. The presence of fat and acid in this section of the small intestine causes the release of secretin and cholecystokinin. These two intestinal hormones and the presence of food stimulate the exocrine pancreas and gallbladder to secrete digestive enzymes into the descending duodenum. Amylase, lipase, proteases, and other pancreatic enzymes are responsible for the breakdown of protein and carbohydrates into smaller, water-soluble, and readily absorbed molecules such as amino acids, oligopeptides, glucose, maltose, and other monosaccharides. In addition, intestinal brush border enzymes contribute to digestion of nutrient molecules and promote their active transport across the intestinal membranes. Digestion and absorption of those nutrients predominantly occur in the jejunum. Fat digestion requires the presence of bile acids and pancreatic lipase that act on fatty acids to form micelles, which are water-soluble spheres that surround fatty acids and make their absorption from the ileum possible. Bile acids are synthesized in the liver and stored in the gallbladder. After bile acids have contributed to fat-absorption mechanisms, they are reabsorbed in the ileum and are transported back to the liver via the portal circulation.

**Loss of Intestinal Absorptive Surface Area**

Removal of a significant length of the duodenum and jejunum results in decreased pancreatobiliary and mucosal enzyme concentrations, which are necessary for digestion and active transport of carbohydrates, fat, and protein. In addition to impaired digestion of food nutrients, there is also insufficient absorption of amino acids, glucose, monosaccharides, vitamins, and microelements. The faster-than-normal intestinal transit time after small intestinal resections also contributes to malabsorption and manifestation of diarrhea because it eliminates the contact time of nutrients with the intestinal mucosa. Humans with less than 100 cm of preserved jejunum are unable to control their nutritional deficiencies and water losses only by oral feeding and therefore depend on total parenteral nutrition (TPN). The hypertonic environment caused by undigested or
partially digested carbohydrates and fats is responsible for the flow of water from vascular space into the lumenal space of the intestine. In humans with small intestinal resection, the transit time of the luminal content through the ileum becomes faster than normal and the colon becomes unable to respond to increased loads of water, which contribute to manifestation of diarrhea, dehydration, hyponatremia, hypokalemia, and hypovolemia. In cases of ileal resection, bile salt reabsorption and its transport back to the liver via the portal vein are impaired, gradually leading to consumption of bile acid liver reserves with inevitable fat malabsorption and clinical demonstration of steatorrhea. Hydroxylation of the unabsorbed fatty acids and degradation of carbohydrates by the colonic flora stimulate colonic water secretion and diarrhea secondary to elevation of intraluminal osmotic pressures. Furthermore, bacterial deconjugation of bile salts delivered to the colon results in irritation of the intestinal mucosa and colonic secretion. In humans, ileal resections of more than 60 cm may contribute to vitamin B12 deficiency. However, in cats undergoing subtotal colectomy and jejunocolonic anastomosis, postoperative intestinal function has reportedly been normal. This evidence suggests that the physiologic length of the ileum in small animals may extend beyond its anatomic boundaries.

Humans undergoing small intestinal and colonic resections may have serious or life-threatening clinical manifestations of SBS because the colon represents the last borderline of water, sodium, magnesium, calcium, and fatty acid absorption. The colon is also a significant coordinator of energy production, via degradation of malabsorbed carbohydrates and proteins to short-chain fatty acids, by the local microflora. The presence of an intact colon is also beneficial to patients with SBS because of the colon’s ability to slow intestinal transit time and to promote intestinal adaptation. Subtotal colectomy can be performed in cats with minimal effects on intestinal function, but the outcome of colectomy in dogs is not well elucidated.

Additional Complications of SBS

Gastric acid hypersecretion because of hypergastrinemia is often reported in animals and humans with SBS, especially in the early postoperative period, but tends to improve with time. Many authors suggest that there is a strong connection between secretion of specific intestinal hormones of the small intestinal epithelium and inhibition of gastrin production. Others support the view that impaired gastrin metabolism because of intestinal resection is responsible for elevated gastrin levels in blood serum. High concentrations of gastric acid and a subsequent decrease of pH in gastric chyme may result in gastroesophageal reflux, GI...
ulceration, irritation of the ascending small intestinal mucosa, disturbed fat digestion (by inactivation of pancreatic lipase), and increased osmolality of the small intestine and GI motility.\textsuperscript{2,7,14,15,17}

The loss of significant length of the jejunum and ileum interferes with absorption of important trace elements (e.g., iron, zinc, copper) and vitamins (fat- and water-soluble).\textsuperscript{2,16} Normochromic nonregenerative anemia may also be present as a result of chronic disease.\textsuperscript{10}

Persistent malnutrition and negative nitrogen balance can finally lead to hypoalbuminemia and hypoproteinemia manifested as edema, ascites, decreased healing ability, and a depressed immune system.\textsuperscript{7,10}

In extensive jejunectomies, calcium and magnesium ions and vitamin D are poorly absorbed by the remaining intestinal mucosa. Vitamin D deficiency also contributes to impaired homeostasis of calcium, phosphorus, and magnesium. In addition, in patients with steatorrhea, these ions tend to form solid complexes with unabsorbed long-chain fatty acids in the colonic lumen, leading to additional mineral losses in the feces.\textsuperscript{16} Osteopenia may result from persistent hypocalcemia and hypomagnesemia.\textsuperscript{6,15}

Another consequence of fat and calcium malabsorption is the development of oxalate kidney stones. In SBS, in which fat malabsorption occurs, calcium ions are consumed mostly by fatty acids. Free oxalate is then absorbed by the colon epithelium and excreted by the kidneys, where hyperoxaluria and calcium oxalate renal stones may form.\textsuperscript{6,15,23}

In humans with ileal resections, reduction of bile acid concentrations resulting from impaired enterohepatic circulation and increased bile acid losses changes the composition of hepatic bile, makes the content of the gallbladder more soluble, and makes evacuation of the bladder more difficult. This often results in formation of cholesterol crystals and gallstones, which justifies resection of the gallbladder.\textsuperscript{2,6,15,23}

D-Lactic acidosis is a rare complication of SBS in humans. In patients with SBS, D-lactate is absorbed by the colonic mucosa and brought into the blood circulation, leading to metabolic acidosis and neurologic signs (e.g., nystagmus, ataxia, confusion).\textsuperscript{6}

### Resection of the Ileocolic Valve

The ileocolic valve is a physical barrier between the small and large intestines that increases the intestinal transit time. It also prevents reflux of colonic content and bacterial load into the jejunum. The combination of the loss of the ileocolic valve, intestinal dilation, and slower-than-normal transit results in small intestinal bacterial overgrowth (SIBO), which may become serious if left untreated.\textsuperscript{3} In a retrospective study\textsuperscript{24} of cats comparing preservation and removal of the ileocolic junction, cats that had the junction removed produced significantly looser stool in the long term. Small animals with SIBO may exhibit fever, deterioration of their nutritional status, weakness, a high leukocyte count, and, rarely, signs of generalized sepsis.\textsuperscript{2,27} Irritation of the intestinal epithelium by bacterial endotoxins may cause further water secretion and worsening of diarrhea.\textsuperscript{15} However, rapid emptying of the small intestinal contents into the colon in cases of ileocolic-valve resection is believed to be beneficial because the bacterial population and its toxins are excreted in the feces.\textsuperscript{6}

### Loss of Intestinal Hormones

Intestinal endocrine cells are specifically arranged along the small bowel epithelium, producing site-specific polypeptides and hormones. Gastrin, cholecys-

In small animals with short-bowel syndrome, early but limited oral intake may stimulate and enhance intestinal adaptation.

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Glucagon-like peptides 1 and 2, neurotensin, and peptide YY are produced in the ileum and colon after the stimulus of fat and carbohydrate digestion. These peptides normally interfere with gastric emptying and intestinal transit time by slowing down small intestinal evacuation into the colon and are characterized as the ileal brake.\textsuperscript{6,28} It is also believed that gastrin, cholecystokinin, secretin, and motilin contribute to intestinal adaptation and inhibition of gastric secretion in experimental animals.\textsuperscript{6,29}

### INTESTINAL ADAPTATION

The aim of intestinal adaptation is to increase absorption of nutrients in the remaining intestinal tract and
The ability of the small intestine to adapt after extensive resections is highly important to the survival of patients with SBS. Throughout the processes of adaptation, the intestinal surface undergoes histologic and functional changes to keep the organism in nutritional balance, as shown in experimental animals. During adaptation, villi elongation, deepening of the crypts, and increased numbers of cells per villus column are observed (Figure 2). Enlargement of crypts pertains to their total size and structural units, including an increase in proliferative cells, but not to the number of crypts. The rate of cell apoptosis is also altered, preserving a balance in cell proliferation or loss.

The ileum possesses a greater adaptive ability than do the duodenum and jejunum. In massive resections (>80%), the ileum can demonstrate an increased length and diameter and has a tendency to resemble the jejunal mucosa. Within the first 48 hours after intestinal resection, mucosa of the intestinal remnant shows the first signs of adaptation. Clinical studies in humans suggest that the whole process of adaptation is completed within the following 2 years. Clinical improvement of diarrhea and the nutritional status of patients with resected intestine represent increased absorptive intestinal ability and mucosal enzyme activity resulting from the adaptive changes.

Demonstrations of increased absorption of glucose per unit of intestinal length in rats after small intestinal resection support the theories of the adaptive process. Enteral nutrition is considered the most important factor enhancing intestinal hyperplasia. The presence of food in the intestinal lumen stimulates trophic GI secretions, provides direct topical nutrition to the regenerating intestinal cells, and increases pancreatobiliary secretion, splanchnic blood flow, neuronal activity, and peristalsis of the intestinal remnant.

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Experimental studies on animals and clinical studies on humans have confirmed that oral feeding augments the mechanisms of intestinal adaptation. In SBS animal models, when no food was administered, no hyperplasia was observed; instead, atrophy of the luminal mucosa had occurred. In humans maintained exclusively with TPN, little adaptive ability was found compared with patients who started oral intake early after resection. In rat models, administration of food could reverse the intestinal atrophy and promote adaptation.

The endogenous effect of the GI hormones and the local growth factors also plays an important role in intestinal adaptation. Gastrin is likely to have trophic results in
the proximal intestine, enteroglucagon enhances mucosal hyperplasia, prolactin is suspected of cooperating in mucosal renewal during lactation, epidermal growth factor and insulin-like growth factor are known to increase DNA synthesis, and prostaglandins and polyamines may promote intestinal adaptation. Research has focused on the use of synthetic hormones to treat SBS by enhancing intestinal adaptation.

Pancreatobiliary secretions are considered a significant candidate in the process of intestinal adaptation. Studies on rat intestines showed that there seems to be a relationship between pancreatic secretions and the length and cellularity of the villi in the intestinal remnant. Many authors support the idea that extensive ileal hyperplasia in small intestinal resections ensues because the ileum is displaced closer to the pancreatic duct and is influenced by trophic pancreatobiliary secretions.

The use of elemental diets in treating humans is still questionable. Glutamine, fatty acids, proteins, fibers, and other nutrients are being investigated for their beneficial effects on intestinal adaptation and the nourishment of patients with SBS.

**CLINICAL SIGNS AND DIAGNOSIS**

The main clinical signs after extensive resection include diarrhea, electrolyte imbalances, malabsorption, and weight loss. Diagnosis of SBS is based on clinical signs and the amount of the intestine that was resected.

The clinical manifestations of SBS in small animals depend not only on the site and the extent of the intestine removed but also on the presence of the ileocolic valve, the length and adaptive ability of the remaining absorptive intestinal surface area, and preservation of the colon. In dogs and cats, watery diarrhea is the dominant clinical sign soon after extensive intestinal resection. Within the first 24 to 48 hours, diarrhea may lead to abrupt weight loss because of significant water loss. Dehydration and electrolyte imbalances necessitate immediate fluid therapy. Hyponatremia, hypokalemia, increased creatinine and blood urea nitrogen levels, and metabolic acidosis may be detected in serum biochemistry analysis. During the following weeks, persistent diarrhea, evidence of malnutrition, constant weight loss, hypergastrinemia, and steatorrhea may be observed.

Malabsorption can easily be confirmed by microscopic examination of fecal samples. Contrast radiography of the GI tract may show dilated intestinal segments and a fast intestinal transit time. As soon as postoperative shock subsides and especially if there are no surgical complications, animals with SBS may become alert and have an increased appetite (i.e., polyphagia). Over time, intestinal hyperplasia occurs, resulting in nutritional and clinical improvement. It has been reported that intestinal hyperplasia takes about 2 months to occur in dogs. The frequency of defecation may decrease, and the texture of feces may become pulπer, leading to less water loss. Patients eventually gain weight but never reach optimal body weight. Laboratory findings may reveal hypoalbuminemia and anemia. Sporadic worsening of diarrhea may result from SIBO, especially in humans undergoing ileocolic-valve resection. The diagnosis of SIBO is very difficult because many of the intestinal bacteria are not culturable.

**MEDICAL TREATMENT**

The purpose of medical treatment of SBS is provision of adequate nutritional support, acceleration of intestinal adaptation, and control of diarrhea. Hormones, growth factors, and other substances (e.g., octreotide, ursodeoxycholic acid [UDCA]) may play a role in enhancing the adaptation response.

In humans, three clinical stages have been recognized during the recovery period, requiring a different therapeutic approach than that for animals (Table 1). During the recovery period, animals with SBS need adequate medical and nutritional support. In the early postoperative phase, treatment of most small animal patients involves intravenous fluid administration and electrolyte restoration. Sodium and potassium blood levels should be evaluated daily. TPN can provide optimal fluid, electrolyte, vitamin, and nutritional sup-

**Table 1. Stages of the Recovery Period in Humans with Short-Bowel Syndrome**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinical Signs</th>
<th>Management</th>
</tr>
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<tbody>
<tr>
<td>I (initial period)</td>
<td>Massive diarrhea, Weight loss, Electrolyte depletion</td>
<td>TPN exclusively</td>
</tr>
<tr>
<td>II (early adaptation)</td>
<td>Decreased diarrhea</td>
<td>Oral feeding and TPN</td>
</tr>
<tr>
<td>III (maximal adaptation)</td>
<td>—</td>
<td>Oral feeding is well tolerated; TPN is discontinued</td>
</tr>
</tbody>
</table>
port during this period, although it is not frequently preferred in veterinary medicine because of its high cost.\textsuperscript{1,14,43} TPN is routinely used in humans with intestinal failure, especially in infants, prolonging the patient’s life until intestinal adaptation occurs. Nevertheless, some patients cannot be successfully weaned from TPN and others have severe chronic complications (e.g., hepatobiliary disease, catheter-mediated sepsis, venous thrombosis, catheter damage).\textsuperscript{2,4,44}

Medical treatment including anti(diarrheal agents (e.g., loperamide, diphenoxylate, opioids) should be initiated to increase intestinal transit time and decrease the volume of diarrhea.\textsuperscript{1,7,14} Gastric acid hypersecretion should be controlled with proton pump inhibitors (e.g., omeprazole) or H\textsubscript{2}-blockers (e.g., cimetidine, ranitidine), which block histamine receptors and inhibit gastric acid secretion.\textsuperscript{1,14} In animals with pancreatic duct disruption after extensive resections of the duodenum, pancreatic enzyme supplementation may be needed.\textsuperscript{14} Bile-salt binding agents (cholestyramine, fiber) could be effective in animals with bile acid–related diarrhea.\textsuperscript{6,26} In cases of bacterial overgrowth, a combination of antibiotics (e.g., metronidazole, tylosin, amoxicillin, and/or tetracycline) should be effective against aerobic and anaerobic bacteria.\textsuperscript{7,43} During the management of SIBO, antibiotics should be frequently changed to prevent antibiotic resistance.\textsuperscript{4}

As soon as the patient is stable, it is advisable to start limited oral intake because food administration is thought to be one of the most important factors in stimulating and enhancing intestinal hyperplasia.\textsuperscript{14,15} Animals with SBS may have an increased appetite soon after surgery, but food administration should be reasonable because large amounts could result in vomiting and worsening of diarrhea.\textsuperscript{7} An elemental diet seems to be beneficial for some humans because it is highly digestible and easily absorbed,\textsuperscript{1} but it may be too expensive for pet owners. Polymeric diets could also be used in animals during the initial period and administered orally or through a rhinosophageal or gastrostomy tube. In the long term, small meals of a low-fat, highly digestible diet could be given three to four times per day.\textsuperscript{1,7,14} Micronutrient supplementation (e.g., vitamins, calcium, zinc, magnesium) of the food may be needed. The food tolerance and nutritional status of the animal should be re-evaluated frequently to avoid starvation. Partial administration of TPN is advisable in animals with a delayed response to oral feeding.\textsuperscript{1,7,14,15} Some animals start to gain weight soon after surgery, but unfortunately others never stabilize and have a poor prognosis.\textsuperscript{1}

Clinical reports on small animals and experimental studies\textsuperscript{1,26} on dogs and rats have shown that resections of less than 70% of the small intestine are well tolerated. On the contrary, resections of more than 75% to 80% of the total small intestine length may lead to SBS.\textsuperscript{7,10,14,15} It has been reported\textsuperscript{10} that dogs with 30 to 40 cm of remaining small intestine and cats with approximately 20 cm have a great chance of surviving without intensive treatment. In humans, resections of up to 50% to 70% of the total length of the small intestine, especially when the colon remains intact, may lead to clinical signs that can be easily treated.\textsuperscript{10} In general, it can be estimated that humans with more than 100 cm of the small intestine and an intact colon can maintain nutritional balance via oral feeding.\textsuperscript{3,17} Infants seem to tolerate more extensive resections because they have greater adaptive ability as a result of their growth.\textsuperscript{11} It has been reported\textsuperscript{1,5} that infants with more

\textbf{Patients with short-bowel syndrome should be treated aggressively on an individual basis.}

nidazole, tylosin, amoxicillin, and/or tetracycline) should be effective against aerobic and anaerobic bacteria.\textsuperscript{7,43} The use of octreotide, a long-acting analogue of somatostatin, may be beneficial in animals with SBS. It has been shown that octreotide inhibits GI and pancreatic secretions and increases intestinal transit time, resulting in reduced fluid loss via diarrhea.\textsuperscript{41,45} Although the results from experimental trials are promising, octreotide may inhibit intestinal adaptation\textsuperscript{45} and further studies must be conducted before its wide therapeutic use.

UDCA has also been studied for its effectiveness in maintaining acceptable nutritional status in patients with SBS. UDCA reportedly changes the composition of bile\textsuperscript{17} and plays a liver-protective role.\textsuperscript{14} It has been postulated that UDCA assists in gastrin suppression in patients with hypergastrinemia after intestinal resection.\textsuperscript{19} These effects of UDCA may explain the mechanism of enhancing food absorption in experimental animals.\textsuperscript{19} In our clinical experience, UDCA administration (10 to 15 mg/kg PO) in a limited number of cats with SBS increased fecal consistency and reduced the frequency of defecation.
Growth hormone, glutamine, and a modified diet seemed to have a positive effect on gut adaptation, providing encouraging data for patients being weaned from TPN. Peptide YY analogues and glucagon-like peptide 2 administration may also be beneficial for patients with SBS, but further investigation is needed. Menhaden oil, long-chain fatty acids, and low-fat diets may be preferred, according to others. Current recommendations concerning drug therapy for SBS in dogs and cats are presented in Table 2.

### SURGICAL TREATMENT

Surgical therapy may be considered when medical and dietary management fail to control clinical signs. Selection of the appropriate surgical procedure for patients with SBS depends on the remaining intestinal length, intestinal function, and diameter of the remaining intestinal lumen. The goal of surgical treatment is to increase intestinal absorptive capacity either by improving absorption of the remaining intestine or by increasing the absorptive intestinal area. These procedures should be performed after the initial adaptive period and not during initial resection. The goal of surgical procedures that have been used in experimental animals has been to increase either the absorptive surface of the intestinal mucosa or the intestinal length and to slow the intestinal transit speed. A reversed small intestinal segment has been used in beagle puppies with extensive small intestinal resections to increase transit time; improved water, lipid, and nitrogen absorption has been reported. Colonic interposition in dogs has been found to increase transit time and improve growth. Creation of intestinal valves by telescoping a proximal intestinal segment into a distal one and apposing seromuscular layers with sutures has been reported in dogs with experimental SBS; weight loss and steatorrhea were improved over a period of 2 to 3 months after surgery. Reversed electrical intestinal pacing has been implemented in dogs and resulted in improved absorption of water and electrolytes. Long-term efficacy of these procedures in humans is lacking, and they are clinically associated with significant morbidity and mortality. Clinical data concerning the use of these techniques in small animal patients are not available.

### PROGNOSIS

Aggressive management in the early stages of SBS may determine a patient’s prognosis. The prognosis may depend on the amount of intestinal resection, which varies among individuals; preservation of the

<table>
<thead>
<tr>
<th>Drug</th>
<th>Canine Dosage</th>
<th>Feline Dosage</th>
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<tbody>
<tr>
<td>Loperamide</td>
<td>0.1 mg/kg PO q8–12h</td>
<td>0.08–0.16 mg/kg PO q12h</td>
</tr>
<tr>
<td>Diphenoxylate</td>
<td>0.1–0.2 mg/kg PO q8–12h</td>
<td>0.05–0.1 mg/kg PO q12h</td>
</tr>
<tr>
<td>Cisapride</td>
<td>0.1–0.5 mg/kg PO q8–12h</td>
<td>2.5–5 mg/cat PO q8–12h</td>
</tr>
<tr>
<td>Omeprazole</td>
<td>20 mg/dog PO sid or 0.7 mg/kg PO q24h</td>
<td>Not recommended</td>
</tr>
<tr>
<td>Cimetidine</td>
<td>10 mg/kg IV, IM, or PO q6–8h</td>
<td>10 mg/kg IV, IM, or PO q6–8h</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>2 mg/kg IV or PO q8h</td>
<td>2.5 mg/kg IV q12h or 3.5 mg/kg PO q12h</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>15 mg/kg PO q12h or 12 mg/kg q8h</td>
<td>10–25 mg/kg PO q24h</td>
</tr>
<tr>
<td>Tylosin</td>
<td>7–15 mg/kg PO q12–24h</td>
<td>7–15 mg/kg PO q12–24h</td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>6–20 mg/kg PO q8h</td>
<td>6–20 mg/kg PO q8h</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>15–20 mg/kg PO q8h</td>
<td>15–20 mg/kg PO q8h</td>
</tr>
<tr>
<td>Pancrelipase</td>
<td>2 tsp powder with food per 20 kg body weight or 1–3 tsp/0.45 kg of food 20 min before feeding</td>
<td>—</td>
</tr>
<tr>
<td>Octreotide</td>
<td>10–20 µg/dog SC q8–12h</td>
<td>10–20 µg/cat SC q8–12h</td>
</tr>
<tr>
<td>Ursodeoxycholate</td>
<td>10–15 mg/kg PO q24h</td>
<td>10–15 mg/kg PO q24h</td>
</tr>
<tr>
<td>Psyllium</td>
<td>1 tsp/5–10 kg (added to each meal)</td>
<td>1 tsp/5–10 kg PO</td>
</tr>
<tr>
<td>Vitamin B₁₂</td>
<td>100–200 µg/day IM</td>
<td>50–10 µg/day IM</td>
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duodenum and ileocolic valve; amount of adaptation; condition of the remaining GI tract; preoperative state of the animal; and client determination to pursue long-term management with significant cost. Each patient should be treated on an individual basis, and a hasty prognosis should not be made.3

REFERENCES


**ARTICLE #1 CE TEST**

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1. Which intestinal condition requires massive intestinal resection, possibly leading to development of SBS in dogs and cats?
   a. intestinal perforation associated with a linear foreign body
   b. diffuse neoplasia
   c. small intestinal infarction
   d. all of the above

2. _______ absorbed in the ileum.
   a. Bile acids are
   b. Fat is
   c. Vitamins A, D, E, and K are
   d. Carbohydrates are

3. Patients with SIBO may have
   a. a fever.
   b. an elevated leukocyte count.
   c. deterioration of their nutritional status.
   d. all of the above

4. Which is not a histologic change of the intestine via the adaptation process?
   a. villi elongation
   b. decreased villi size
   c. crypt enlargement
   d. balanced apoptosis

5. The presence of enteral nutrients in the intestinal lumen may
   a. increase pancreaticobiliary secretions.
   b. stimulate trophic GI secretions.
   c. provide topical nutrition.
   d. all of the above

6. Which statement regarding the amount of resected intestine leading to SBS is incorrect?
   a. Resection of less than 70% of the small intestine is well tolerated.
   b. Resection of more than 75% to 80% of the small intestine may lead to SBS.
   c. Subtotal resection of the colon alone may lead to SBS.
   d. none of the above

7. Which is not considered a complication of SBS?
   a. oxalate renal stones
   b. gallstones
   c. hypergastrinemia
   d. ulcerative colitis

8. Clinical signs of SBS do not include
   a. watery diarrhea.
   b. weight loss.
   c. steatorrhea.
   d. hemorrhagic gastroenteritis.

9. The goal of surgical management of SBS is to
   a. increase intestinal length.
   b. decrease intestinal length.
   c. increase intestinal transit speed.
   d. decrease absorptive capacity of the intestine.

10. The prognosis of SBS depends on
    a. aggressive medical management.
    b. the amount of adaptation.
    c. the preoperative state of the patient.
    d. all of the above

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