The Canine Gastrointestinal Tract: Small Intestine

From The WALTHAM Course on Dog and Cat Nutrition.

KEY POINTS

- **Dietary rest for 24 hours is indicated in acute gastroenteritis to give relief from diarrhea and vomiting.**
- **This should be followed for several days with feeding a highly digestible, "bland" diet to minimize the risk of acquired dietary hypersensitivity before normal food is gradually reintroduced.**
- **Feeding a "bland," "hypoallergenic" diet is particularly important after a bout of hemorrhagic gastroenteritis as this is associated with a high incidence of acquired hypersensitivities.**
- **In chronic inflammatory bowel disease (IBD), such as lymphocytic-plasmacytic or eosinophilic enteritis, total dietary rest is not indicated; rather, feeding novel dietary ingredients, as indicated for dietary sensitivities is recommended.**
- **In both acute and chronic enteritis, acquired dietary hypersensitivities may develop to dietary components and dietary changes may be necessary throughout the treatment.**
- **Some small intestinal diseases are associated with specific dietary intolerances or hypersensitivities (e.g., lactose intolerance, gluten-sensitive enteropathy, true allergies), and dietary changes have to be made accordingly.**
- **Increased levels of high-quality, highly digestible dietary protein are particularly indicated in the protein-losing enteropathies, such as those that occur with lymphangiectasia, lymphosarcoma, and severe IBD.**
- **Generally, moderate dietary fat levels are indicated in chronic small intestinal disease with fat maldigestion/malabsorption, particularly with villi atrophy, small intestinal bacterial overgrowth (SIBO), and lymphangiectasia.**
- **Patients with SIBO also require a highly digestible diet to reduce undigested nutrients left in the lumen for bacterial utilization. "Hypoallergenic" diets have also been recommended in SIBO.**
- **In lymphangiectasia, feeding a low-fat diet is the cornerstone of treatment, reducing lymphatic flow.**

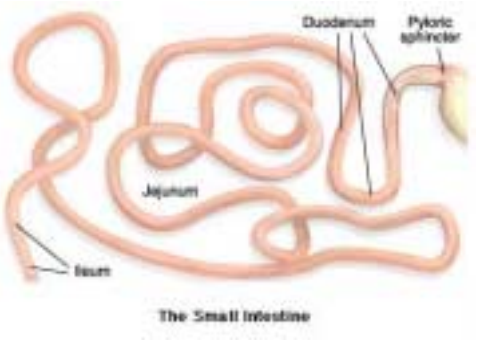
- **A low-fiber diet is generally indicated in chronic small intestinal disease, although some forms of soluble fiber may be beneficial.**
- **Parenteral application of vitamin B_{12} and folate may be indicated in those patients with reduced levels.**
- **Feeding little and often helps overcome the reduced digestive and absorptive capabilities of the diseased small intestine, thus reducing diarrhea and increasing nutrient intake.**

The small intestine (SI) is where the majority of food digestion and absorption occurs. Enzymes and secretions discharged into the SI from the brush border cells, pancreas, and liver are crucial for the digestion of nutrients; diseases of these organs are therefore often linked.

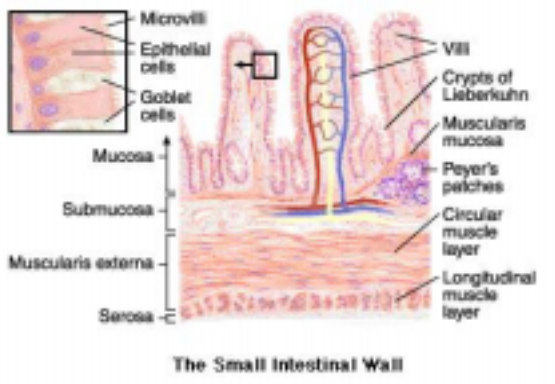
Food intake is vital to small intestinal health and function, and the SI is vital for "food" intake for the rest of the body. It is not surprising, therefore, that diet is very important in the pathogenesis and treatment of a great number of small intestinal diseases.

ANATOMY

The SI received its name from being comparatively small in diameter, not length. The SI follows on from the stomach and is split into three distinct sections: the duodenum, jejunum, and ileum. The duodenum comprises approximately 10% of the SI, with the jejunum and ileum making up the remaining 90%, although there is no clear anatomic demarcation among them.



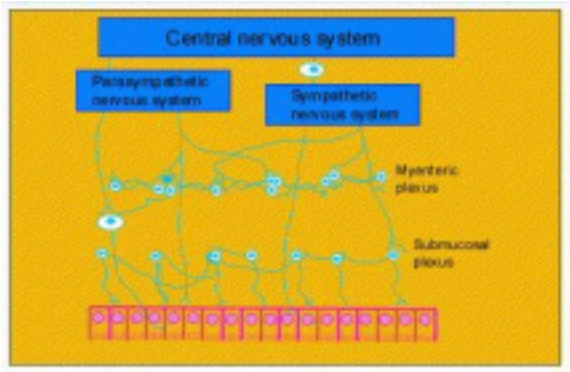
The Small Intestine



The Small Intestinal Wall

The intestinal wall consists of several layers, namely the:

- **Mucosa**
- **Submucosa**
- **Muscularis** with an outer longitudinal and an inner circular layer
- **Serosa**



Integration and Control of GI Responses to Food.

Neurologic control of the intestinal muscles is by both local and vagally mediated reflexes via the submucosal nerve plexus and myenteric plexus between the circular and longitudinal muscles. The parasympathetic nervous system regulates normal intestinal function, whereas the sympathetic nervous system plays a minor role. Local control and coordination of motility and secretions by the intestine and associated glands is very complex, with contributions from nerves and paracrine and endocrine chemicals - at least 15 different types of SI paracrine/endocrine cells have been identified so far.

FUNCTION

General

The SI is the primary area of food digestion and absorption. Digestion is achieved using a combination of enzymes from the small intestinal luminal brush border cells and the pancreas. Additionally, bile released from the gallbladder is important for fat digestion.

The majority of digestion and absorption in the SI occurs in the first quarter, so there is considerable functional reserve. The SI epithelium is folded into villi and has a luminal brush border to increase the surface area for absorption and production of brush border enzymes. Goblet cells dispersed among the epithelium secrete mucus and have fewer microvilli.

The cells at the base of the villi are dividing, undifferentiated cells and the main area of fluid secretion. The cells differentiate as they push up to the tips of the villi, where they become absorptive cells. It takes about 2-3 days for the cells to travel from the base to the tip of the villus - the entire surface of the SI is shed every 3 days. Shedding becomes more rapid if an insult stops epithelial growth and differentiation and can result in villi atrophy, e.g., in small intestinal bacterial overgrowth (SIBO), inflammatory disease, toxins, and some drugs, such as chemotherapy agents. Vitamin B_{12} and folate deficiency will make shedding more rapid. It has also been shown that the normal gut microflora slows down the replacement rate - small intestinal epithelial cells have double the lifespan in gnotobiotic animals. Occasionally, hypertrophy of the mucosa occurs as a result of trophic substances (e.g., gastrin), increasing epithelial growth.

The dog's bacterial population of the SI is low but stable at $<10^5$ colony forming units (CFU)/ml, with mainly aerobes and gram-positive bacteria present. Bacterial numbers are increased in SIBO, and the types of bacteria present can also change.

Protection and Immunity

The gastrointestinal (GI) tract has direct contact with the environment and therefore forms a very important protective barrier within the animal's organism. The gut has to stop foreign material, such as bacteria or antigens, entering the body and also prevent excessive loss of "body material" into the gut.

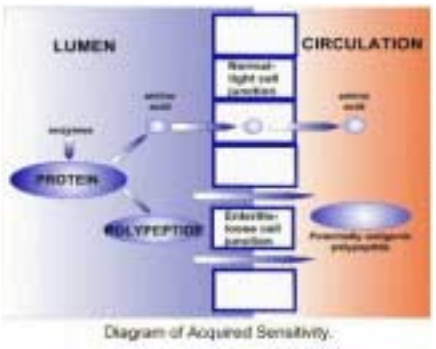
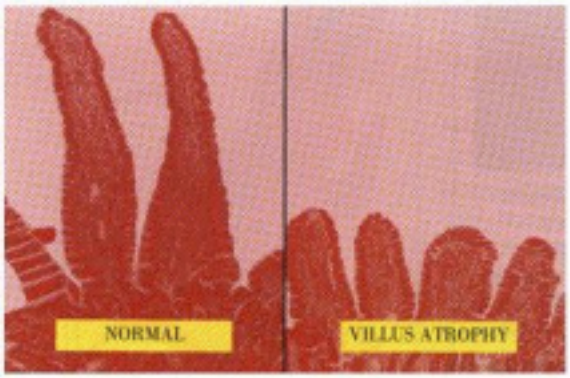


Diagram of Acquired Sensitivity.

The epithelial cells are joined by "tight junctions," which prevent large molecules, particles, or bacteria from penetrating the gut wall and entering the host. They also provide an effective barrier to uncontrolled loss of proteins, fluid, and electrolytes into the lumen. Even so, adults can absorb up to 2% of dietary protein intact (this is even higher in neonates) and 40% of normal daily plasma protein loss occurs in the GI tract. The amount of intact protein absorption increases in inflammatory bowel disease (IBD), thus increasing the risk of developing hypersensitivities.

Additional protection against absorption of bacteria and antigens include:

- Gastric acid
- Antibacterial effects of pancreatic and bile secretions
- Normal gut motility
- Normal gut bacterial flora
- Mucus and glycoproteins on the gut surface
- Local secretion of IgA
- Lymphocytes in the submucosa
- The hepatic reticuloendothelial system, which clears antigens and bacterial from portal blood ("second line of defense")



Small Intestinal Villi

A breakdown in any one of these mechanisms as a result of disease can result in bacterial translocation from the gut and subsequent septicemia; additionally, the impaired gut barrier may lead to an uptake of antigens and the development of dietary hypersensitivity. The presence of food in the GI lumen is essential for maintenance of this natural defense barrier, and the gut should therefore never be starved for any length of time unless truly indicated. Enterocytes obtain at least 50% of their energy requirements from the products of intraluminal digestion; glutamine is the main fuel in the SI and butyrate in the large intestine.

Motility

Normal gut motility involves two types of contraction; they occur in both the small and large intestines, but are slower more distally in the GI tract.

- ***Rhythmic segmentation*** is a random contraction of small areas of the smooth muscle, which slows down the forward motion of food and mixes it, allowing more effective digestion and absorption. This type of contraction usually occurs only when the gut is full, as it is stimulated by local stretch reflexes and the vagus.

- ***Peristalsis*** is an organized, forward moving wave of contraction, propelling food down the GI tract. Known as the "migrating motility complex" (MMC), it also occurs in the unfed state, when there may be infrequent but large contractions; these are then known as "interdigestive MMCs" or "housekeeping contractions."

Most motility diarrheas are a result of a reduction in rhythmic segmentation, *not* an increase in peristalsis. Reduced rhythmic segmentation results in a flaccid gut such that even normal peristaltic waves will propel food a long way easily.

CLINICAL DISORDERS

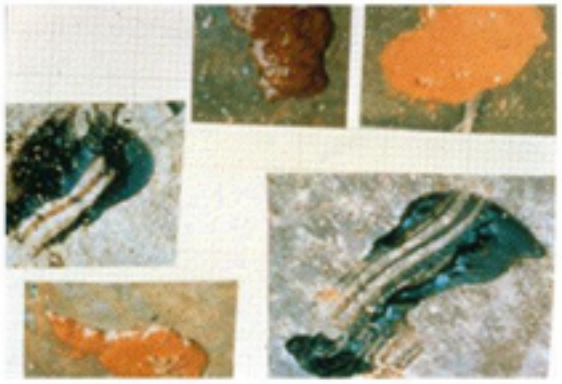
Classically, disorders of the SI result in small intestinal diarrhea. In many cases there is an increased mucosal permeability with the potential for the development of acquired dietary hypersensitivity. In some cases there may also be significant protein-losing enteropathy (PLE*)* or melena. However, the large functional reserve of the SI means that damage to a small area may produce no clinical signs at all.

The most common small intestinal disease encountered in practice is acute gastroenteritis, often due to dietary change or indiscretion, and this often responds to dietary management alone. Other causes of transient small intestinal diarrhea include infections, hemorrhagic gastroenteritis (HE), and functional diarrhea.

More chronic small intestinal diarrhea requires careful differentiation from large intestinal diarrhea and a thorough diagnostic workup to rule out diseases of other organs and to reach a diagnosis. Causes of chronic disease include dietary intolerance or hypersensitivity, chronic infections, partial obstruction, neoplasia, SIBO, and diseases of other organs. Diet is important in both the pathogenesis and treatment of most of these disorders.

Acute Gastroenteritis and Diarrhea

Acute diarrhea is very common in dogs and has been suggested as the main reason dogs are presented for treatment. The main cause is scavenging or dietary indiscretion, although lactose intolerance or transient infections may also be involved.



Watery Feces – Diarrhea

Sudden dietary change, scavenging, or overfeeding can lead to diarrhea because brush border and pancreatic enzymes need time to adapt to new foods. Sudden changes may “overwhelm” the existing enzymes, resulting in maldigestion and osmotic diarrhea. Additionally, toxins in contaminated foods may stimulate secretory diarrhea.

If the animal is otherwise well, acute gastroenteritis and acute diarrhea are usually managed symptomatically with a short period (24 hours) of dietary rest, oral fluids and electrolytes, and appropriate dietary management. As most cases respond to symptomatic therapy, a definite diagnosis is often not established.

Many patients with acute gastroenteritis may also vomit.

In severe and/or prolonged cases, a further diagnostic evaluation is indicated. Young puppies dehydrate very easily and may suffer significant electrolyte imbalance and hypoglycemia, even with transient diarrhea. These patients need to be monitored carefully and may require intensive treatment, even with mild to moderate clinical signs.

Hemorrhagic Gastroenteritis

This is a relatively common condition of unknown etiology and can be confused with canine parvovirus-2 infection. In spite of its name, the disease does not affect the stomach. HGE is mainly seen in young adult, small breed dogs of 2 to 4 years of age. There is usually no obvious trigger and often a sudden onset of vomiting and severe hemorrhagic diarrhea, which can be of the small and/or large intestinal type. This may be preceded by non-hemorrhagic diarrhea.

The diarrhea results from a sudden, marked increase in intestinal permeability with a resultant loss of plasma proteins and blood into the bowel lumen but very little inflammatory infiltrate in the mucosa. This lack of serious damage means that the prognosis for complete recovery from HGE is very good, provided adequate supportive therapy is given.

HGE may be complicated by secondary clostridial overgrowth and endotoxemia due to an increased gut permeability. The cause of the increased permeability is unknown - anaphylactic shock to a food allergen has been suggested. It is therefore recommended to feed a “hypoallergenic” diet after recovery, particularly as increased permeability increases the risk of acquired dietary hypersensitivity in these dogs.

Vaccination history helps differentiation from proviruses, and HGE cases are normally not leukopenic whereas canine proviruses usually are. HGE typically (and rather surprisingly) causes a markedly increased packed cell volume (PCV) due to splenic contraction with normal levels of plasma proteins.

Infectious Causes of Diarrhea

Infectious organisms may cause acute or chronic diarrhea, which can range from self-limiting to life-threatening and may involve only the SI or both the small and large intestines.

Viral causes of enteritis include canine parvovirus (type 2) and canine distemper virus. Also, canine coronavirus causes diarrhea, which is usually transient and mild. Rotavirus may be involved in some mild, self-limiting small intestinal diarrheas in dogs.

Bacteria causing diarrhea include *Campylobacter jejuni*, *Salmonella*, and others. *Campylobacter* is particularly found in kenneled dogs (30% positive fecal cultures in healthy kenneled dogs compared with 1% in household pets) and also in man. The infection may be asymptomatic, or cause self-limiting or chronic diarrhea. *Campylobacter*

colonizes the jejunum, ileum, cecum, and colon; however, the clinical signs are largely large intestinal.

Salmonella infection in the dog is usually caused by *Salmonella enteritidis* serotype *typhimurium*. Asymptomatic carriage is possible but occurs mainly in young, stressed, or diseased animals; there is a very low carriage rate in healthy dogs and cats. *Salmonella* invades the mucosa of the ileum and occasionally the colon and may cause septicemia. It can cause mild to severe gastroenteritis, fresh blood in the feces, and even septicemia.

Other bacterial causes of diarrhea include *Clostridium perfringens* and *Escherichia coli;* both are normal residents of the GI tract but may become pathogenic in some circumstances. *Clostridium* overgrowth and production of enterotoxin is encouraged by intestinal stasis and can result in hemorrhagic diarrhea and rapid death. *E. coli* is also normally found in the gut, but pathogenic serotypes may be important in some cases of diarrhea, especially in young animals with poor immunity. *E. coli* septicemia is the major cause of death in puppies with proviruses.

Protozoa that cause small intestinal diarrhea include *Giardia* and Coccidia.

Giardia are anaerobic, lumen-dwelling protozoa that may have some mucosal attachment. They live in the upper SI in dogs and thus generally cause acute or chronic small intestinal diarrhea. Diarrhea is mainly due to damage of the epithelium and the brush border enzymes and may lead to secondary SIBO.

Coccidial infections are caused by *Cystoisospora* (*Isospora*) and *Cryptosporidium*. In both cases, there is a high level of asymptomatic infections; clinical signs are seen especially in young or immunosuppressed animals and involve small intestinal diarrhea with or without mucus and fresh blood in the feces.

Of the nematode infections, ascarids very rarely cause a clinical problems in adults. In puppies, however, they may cause weight loss, vomiting, and soft feces. Similarly, hookworms are mainly a problem in puppies, where they cause anemia but rarely GI signs.

Diagnosis of Chronic Diarrhea

- **Dietary**
  - Chronic or intermittent scavenging
  - Chronic dietary intolerance (e.g., lactose intolerance, gluten-sensitive enteropathy, other dietary sensitivities)
- **Chronic infections**
  - *Salmonella, Campylobacter, Trichuris, Giardia,* and others

- **Small intestinal disease**
  - IBD (lymphocytic-plasmacytic, eosinophilic, granulomatous)
  - SIBO
  - SI neoplasia (lymphoma, mast cell tumors, adenocarcinomas)
  - SI partial obstruction (strictures, foreign bodies, intussusceptions [most commonly ileo-cecal], jejuno-jejunal, hernias, adhesions)
  - Lymphangiectasia
  - Increased permeability due to portal congestion, as with right-sided chronic heart failure, liver disease
  - Rare brush border enzyme deficits
  - "Short bowel syndrome" after extensive SI resection (SIBO, maldigestion, and malabsorption)
- **Pancreatic disease**
  - Exocrine pancreatic insufficiency (EPI)
  - Chronic pancreatitis
  - Pancreatic adenocarcinoma
- **Liver disease**
  - Portal congestion with or without a lack of bile salts
- **Renal disease**
  - Uremia with or without hypoalbuminemia/edema of nephrotic syndrome
- **Endocrine disease**
  - Hypoadrenocorticism
- **Miscellaneous toxemias**
  - Pyometra
  - Toxicities
  - Certain drugs, including some antibiotics

Dietary Sensitivity

The term dietary sensitivity encompasses both food intolerance, a nonimmunologic abnormal response to a food, as well as dietary hypersensitivity, an immunologically mediated phenomenon. Although animals are exposed daily to a great variety of potential dietary allergens, only a small number will develop an abnormal immune response to a particular dietary ingredient. Once sensitized, further exposure to the allergen results in the development of clinical signs.

The GI mucosa presents a barrier that limits absorption of the vast array of potential allergens contained within the digesta. This mechanism is imperfect, however, and a significant proportion of dietary antigens may be absorbed across both the healthy and the abnormal gut and are then tackled by the gut-associated lymphoid tissue (GALT).

Absorption is limited by the local immune response, predominantly involving the production of IgA antibody. Backup mechanisms help to clear the system of any antigenic material that is absorbed, including the formation of immune complexes with IgA antibodies, which are transported to the liver, secreted in bile, and returned to the intestine. A systemic response involving IgG antibodies may also occur but, in normal individuals, is not associated with adverse clinical effects. The local immune response may be enhanced by immunization, which limits absorption of macromolecules. Conversely, absorption may be increased as a result of local vasodilatation, such as that which may occur with allergic or other inflammatory GI disease.

Small intestinal diarrhea may improve nonspecifically with the use of a "hypoallergenic" diet. This does not necessarily imply that there is an allergic basis to the disease but may simply be due to the fact that these diets tend to be "bland" and highly digestible.

True food allergies do exist but are very hard to define and diagnose. Confirmed food allergy in dogs usually produces pruritic skin diseases rather than GI signs. However, there are concurrent GI signs in 10-15% of dogs with food hypersensitive skin disease, although the GI signs may be mild and easily overlooked.

In addition, it is worth considering that any infiltrative IBD involving lymphocytes, plasma cells, or eosinophils may have an allergic basis and feeding a "hypoallergenic" diet in conjunction with other treatments may therefore be beneficial.

Inflammatory Bowel Disease

Lymphocytic-plasmacytic enteritis is the most common form of IBD in dogs. The cause is unclear, although dietary sensitivity is often implicated. IBD may also involve the stomach and/or colon or may occur secondary to other conditions, such as SIBO or Giardia infection.

There is also potential for confusion between severe lymphocytic-plasmacytic enteritis and lymphoma on biopsy. Severe cases of lymphocytic-plasmacytic enteritis show PLE. Some Basenji dogs develop a particularly severe form of lymphocytic-plasmacytic enteritis.

Eosinophilic enteritis/gastroenteritis is usually a more severe condition than lymphocytic-plasmacytic enteritis, but it is rare. It may also be caused by dietary sensitivity. Eosinophilic enteritis often causes PLE.

Small Intestinal Bacterial Overgrowth

Normal small intestinal bacterial populations are small and stable, and SIBO in dogs is defined as $>10^5$ CFU/ml bacteria in duodenal culture or $>10^4$ CFU/ml anaerobes. The

most commonly seen form is a “primary” condition in young, large breed dogs, especially German Shepherds. The exact cause is unknown but may be related to a defective local immunity or IgA deficiency in this breed.

Possible reasons for pathologic bacterial overgrowth include:

- Deranged gastric and small intestinal motility, (e.g., “gastric dumping” or small intestinal ileus)
- Deranged small intestinal defense mechanisms, as with the use of steroids
- Reduction and disturbance of the normal flora, as with the use of antibiotics
- Increased presence of unabsorbed nutrients, (e.g., in EPI or IBD

The clinical signs include chronic diarrhea, typically steatorrhea with some large intestinal character due to fat maldigestion, weight loss, and possibly vomiting. Some patients show a reactive increase in hepatocellular enzymes. The histologic examination shows only mild changes and may even appear normal in 60% of all cases; 30% of affected dogs show partial villi atrophy and mild lymphocytic-plasmacytic infiltrate.

The clinical signs seen in SIBO are based on:

- **Bacterial deconjugation** of bile salts reducing fat emulsification and hence digestion and absorption; deconjugated bile salts also cause colonic irritation and secretory diarrhea
- **Bacterial breakdown** of fat to hydroxy fatty acids, which also cause secretory diarrhea
- **Bacterial interference** with brush border enzymes and enterocyte function

Gluten-Sensitive Enteropathy

This condition is reported most commonly in Irish Setters but may also occur in other breeds. Susceptible Irish Setters show signs at 4-7 months of age, which include diarrhea and weight loss. It is a malabsorption disorder, and affected dogs fed on gluten-containing diets develop an impairment of the mucosal barrier with partial villous atrophy. These morphologic changes responds to removal of wheat (and hence gluten) from the diet and can be reversed by feeding a gluten-free diet. It is not yet clear whether this has an immunologic basis or it is attributable mainly to other mechanisms of food intolerance. Gluten-sensitive enteropathy shows similarities to celiac disease in humans, where the pathogenesis involves interactions between genetic and environmental factors. These include genetic predisposition, enteric infections, type and quantity of gluten, and age of first gluten intake.

Lactose Intolerance

Lactose intolerance is relatively common in dogs and occurs because of a reduction in the small intestinal brush border lactase activity after weaning. This means that lactose (milk sugar) cannot be digested, and the undigested lactose in the small intestinal lumen causes osmotic diarrhea.

Protein-Losing Enteropathy

Any disturbance resulting in a marked increase in mucosal permeability can cause protein-losing enteropathy (PLE), including some infections, portal hypertension, congestive heart failure, ulceration and chronic intussusceptions, obstructions, or foreign bodies. The most common causes of PLE include:

- Eosinophilic enteritis and severe lymphocytic-plasmacytic enteritis
- Intestinal lymphoma (and other neoplasms)
- Lymphangiectasia

Dietary management is very important in patients with PLE, and it is particularly important not to restrict dietary protein intake in these dogs.

Lymphangiectasia

This is quite an uncommon condition in dogs; both congenital and acquired forms are recognized.

Acquired cases show a blockage, dilation, leakage, and rupture of the lymphatics with surrounding granulomatous inflammation. The condition is seen in some cases of IBD or portal hypertension; however, the underlying cause is usually unknown.

The condition results in a loss of lymph into the GI lumen. Since lymph contains high levels of fat (triglycerides as chylomicrons), protein, and lymphocytes, affected dogs have low plasma total proteins (albumin and globulin), lymphopenia, and hypocholesterolemia. Patients may also suffer from a deficiency of fat-soluble vitamins. Major clinical signs are weight loss, with or without ascites, pleural effusion, and/or edema. Diarrhea, particularly steatorrhea is variable and not always present. Appropriate dietary management is the only effective treatment in most cases, but the prognosis is still generally poor.

Neoplasia

The most commonly diagnosed small intestinal tumors in dogs are adenocarcinoma, lymphoma, leiomyoma, and leiomyosarcoma.

The latter two and adenocarcinoma are often local, annular-type lesions that may present as total or partial small intestinal obstructions.

Lymphoma may be localized, but it is more often seen as a diffuse infiltrate in the small intestinal wall; this usually presents as PLE and often melena. Lymphoma may involve the SI alone or form part of a multicentric problem.

The prognosis with adenocarcinoma depends on time of diagnosis and removal. If local resection is possible in early stages, the prognosis may be reasonable; however, it will be poor if the tumor has metastasized at surgery.

Leiomyomas and leiomyosarcomas often have an excellent prognosis after surgical resection.

Lymphoma carries a very poor prognosis; the response of GI lymphoma to chemotherapy is reported to be poor. Even when remission is achieved, the outlook is poor because removing the neoplastic cells renders the gut wall very “leaky.”

Conditions Requiring Surgical Management

A number of causes of acute or chronic small intestinal disease require surgical management. These include small intestinal foreign bodies and other causes of obstruction, such as external compression or small intestinal tumors, ileo-ceco-colic and jejuno-jejuno intussusceptions, strictures, torsions, hernias, strangulations, small intestinal perforations, and adhesions.

Postsurgical dietary management is important in all cases to ensure optimal GI healing, and it is particularly important to feed dogs as soon as possible after GI surgery.

A particular syndrome is recognized after extensive surgical resection of the SI; it is called “short bowel syndrome.” This condition results in chronic maldigestion and/or malabsorption and diarrhea due to a combination of SIBO, failure of digestion and absorption, hypersecretion of gastric acid, and enterocyte malnutrition.

DIAGNOSIS

There are a number of procedures in the workup of chronic diarrhea and small intestinal disease, starting with a thorough history and baseline investigation, excluding EPI, investigating the small intestinal damage, and examining the SI directly.

Small Intestinal Diarrhoea	Large Intestinal Diarrhoea
Vomiting common	Sometimes vomit (30%)
Weight loss common	Usually no weight loss
Polydipsia common	No polydipsia
Often appetite increased or reduced	Appetite often normal
Watery/bulky faeces	Faecal type varies
Faecal volume increased	Volume normal or increased
Defaecate 1-3 times a day	Defaecate >6 times a day
Faecal fat ± starch may be present	No faecal fat (unless secondary)
Tenesmus not present	Tenesmus often present
Urgency not present	Urgency often present
Mucus not present	Mucus often present
If blood present, melaena	If blood present, fresh
Minimal flatulence	Flatulence common

Diagnosis to Establish Small Intestine Diarrhea

Small intestinal diarrhea needs to be carefully differentiated from large intestinal diarrhea to allow appropriate further workup. Differentiation may be difficult: Small intestinal diarrhea may be combined with secondary large intestinal diarrhea due to undigested fat reaching the colon and being metabolized by colonic bacteria to hydroxy fatty acids, which are potent secretagogues.

Laboratory Tests

The laboratory tests used in the workup of small intestinal diarrhea include (listing in the usual order of investigation):

- Routine blood screens
- Fecal samples
- Vitamin B_{12}, folate, and trypsin-like immunoreactivity (TLI) measurement
- Tests for malabsorption/maldigestion
- Additional tests for SIBO
- Tests for PLE
- Tests for increased small intestinal permeability

The workup for small intestinal disease may also involve plain and contrast radiography, ultrasonography and biopsy.

Routine Blood Screens

Blood samples for hematology and biochemistry screens are indicated in chronic small intestinal disease to rule out endocrine and metabolic diseases and other organ disorders, including those involving the pancreas and liver. They also allow assessment of electrolyte, plasma protein, acid-base, and hydration status and anemia in the presence of

significant melena. Hypokalemia is particularly common with small intestinal disease, particularly if there is concurrent vomiting. Hypokalemia leads to GI hypomotility, which may further inhibit recovery.

Fecal Samples

The gross evaluation of fecal samples helps to differentiate between small and large intestinal diarrhea. If there is no grossly obvious melena but a suspicion of blood loss, feces can be tested for occult blood. For this purpose, a vegetarian diet is fed for 3 days, so that there is no cross-reaction with dietary sources of blood.

Fecal culture and sensitivity are essential with all cases of chronic diarrhea, especially when requesting a culture for *Salmonella* and *Campylobacter*.

Feces may also be assessed for clostridial toxin or pathogenic serotypes of *E. coli*. A zinc sulfate flotation will give indication of *Giardia;* however, the diagnosis of *Giardia* is difficult and requires three feces samples taken every other day because excretion can be intermittent. Fecal samples should also be assessed for evidence of nematodes and coccidia.

Vitamin B_{12}, Folate, and Trypsin-like Immunoreactivity

These tests are usually offered as one package by the laboratory.

TLI assesses pancreatic disease, while B_{12} and folate assess the SI.

Low TLI is an indication of EPI, while very high levels indicate acute or chronic pancreatitis

Folate is absorbed by the jejunum, so levels will be reduced in all jejunal disease causing malabsorption, such as IBD. On the other hand, folate levels are increased in cases of SIBO, as it is synthesized by some bacteria.

Vitamin B_{12} is absorbed in the ileum and is therefore reduced in ileal disease causing malabsorption. These are, however, unusual, and it is more commonly reduced in SIBO as bacteria bind B_{12}. Reduced levels are also seen in EPI, partly due to secondary SIBO but also due to the lack of sufficient pancreatic intrinsic factor necessary to allow absorption. Vitamin B_{12} is required for normal small intestinal health, so supplementation is advisable if levels are low.

However, while high folate and/or low vitamin B_{12} levels are indicative of SIBO, only 65% of SIBO cases show changes in these vitamins; further tests may therefore be used for a definite diagnosis of SIBO.

Tests for Malabsorption

These tests are rather nonspecific and include the following:

- **Assessment of fecal fat and starch** (rather insensitive)
- **Fat absorption test:** The patient is fed 3 ml/kg corn oil after fasting for 24 hours. Blood samples are collected before and 2 hours after feeding. In normal animals the second sample should be lipemic; the absence of lipemia is indicative of fat malabsorption/maldigestion (or delayed gastric emptying). However, this test is rather crude and nonspecific.
- O**ral glucose tolerance test:** 2 g/kg of 20% glucose solution are applied by a stomach tube. Blood samples are taken before and then every 30 minutes for 3 hours. Normal animals show peak levels of 6-9 mmol in 30-60 minutes, whereas small intestinal malabsorption results in a flat curve. However, false low curves can be caused by delayed gastric emptying and SIBO. Some cases of EPI are due to chronic pancreatitis, and a prediabetic curve with prolonged high levels will be observed. Again, this is a crude, nonspecific test and is complicated by changes in peripheral glucose use and excretion.
- **Oral xylose absorption test:** A 5% solution of xylose is given at 0.5 g/kg. Blood samples are taken in the same way as with the glucose tolerance test. The response in normal animals shows a peak above 3.0 at 60-90 minutes. The main advantage of this test over the glucose tolerance test is that xylose is not metabolized peripherally or lost in the urine, although levels are still prone to change with SIBO and delayed gastric emptying. The test shows improved sensitivity if both xylose and glucose are given; to rule out malabsorption, the ratio measured should be >0.6.

Tests for Small Intestinal Bacterial Overgrowth

- **Vitamin B12:** Vitamin B_{12} is most commonly reduced in SIBO as bacteria bind B_{12}. However, while high folate and/or low vitamin B_{12} levels are indicative of SIBO, only 65% of SIBO cases show changes in these vitamins; further tests may therefore be necessary for a definite diagnosis of SIBO. Reduced levels are also seen in EPI, partly due to secondary SIBO but also due to the lack of sufficient pancreatic intrinsic factor necessary to allow absorption. Vitamin B_{12} is required for normal small intestinal health, so supplementation is advisable if levels are low.

- **Duodenal culture:** This can be done by endoscopic collection of duodenal juice and is regarded as the "gold standard" test. Dogs with SIBO show a minimum of 10^5 CFU/ml (>10^4 CFU/ml anaerobes).
- **Breath hydrogen tests:** This test is carried out in referral centers only. Carbohydrate substrate (e.g., xylose) is administered after fasting, and breath hydrogen is measured. Normally, hydrogen is produced only in the gut and absorbed after carbohydrate/fiber metabolism by bacteria in the large intestines. In normal dogs, this leads to a breath hydrogen peak 3-5 hours after ingestion of a meal (time varies with substrate used). An earlier peak (i.e., after 1-2 hours) shows SIBO with bacteria in the SI fermenting the food to hydrogen. Significant carbohydrate malabsorption also results in raised breath hydrogen, but this is due to increased large intestinal fermentation; thus a somewhat higher peak is observed at the expected time for the large intestine. However, these values are abnormal in only 65% of cases of SIBO, as some cases of SIBO ferment to methane and other gases rather than to hydrogen.

Tests for Protein-Losing Enteropathy

- **Low plasma total proteins:** This is the assessment of both the albumin and globulin fractions. It is important to differentiate from other causes of low plasma protein levels, such as malnutrition, protein-losing nephropathy or chronic liver disease.
- The "gold standard" for diagnosis of PLE is the **loss of intravenously administered 51chromium albumin into gut**; however, this is performed in referral centers only.
- **Assessment of fecal -1-protease inhibitor** in a single fecal samples is a test used in humans. The plasma protein is approximately the same size as albumin, which is not normally lost into the gut. It also inhibits digestive enzymes, so it is not digested, even if it does enter the GI tract. Its presence in feces therefore indicates PLE.

Permeability Tests

These are carried out in selected referral centers only. There are two possible tests to show increased small intestinal permeability, as seen in IBD or gluten-sensitive enteropathy. The permeability may be measurably increased before there are any clinical signs, so these tests may be useful measure of early disease or recurrence.

- **Lactulose/rhamnose differential sugar absorption:** Lactulose (L) is a large disaccharide that is not normally digested or absorbed. Instead, it is fermented by bacteria in the large intestine. However, lactulose will be absorbed if there is an increase in the small intestinal permeability due to reduced integrity of tight junctions between enterocytes. Rhamnose (R) is a smaller molecule than lactulose and is therefore absorbed in the SI; absorption depends on the surface area available. Normal

ratio values for L:R lie below 0.12; large values show increased permeability with or without reduced absorption

- **Radiolabels:** This is based on (1) the oral administration of ^{51}chromium EDTA and measuring 24-hour urinary excretion or (2) loss of intravenously administered ^{51}chromium albumin into the gut in cases of PLE.

Radiography in Small Intestinal Disease

Initially, plain abdominal radiographs should be taken to look for evidence of partial obstructions, foreign bodies, intussusceptions, or abnormalities of other organs. Additionally, plain thoracic radiographs may be indicated to check for tumor metastases (e.g., of adenocarcinoma).

Contrast barium radiographs may then follow; however, these are of limited usefulness in chronic diarrhea and only indicated when there is no access to ultrasonography and there is a suspicion of local, annular gut wall thickening, or intussusception. Contrast radiography is very insensitive to generalized gut wall infiltration. It is contraindicated in suspected bowel rupture, as barium will leak out and not be absorbed, causing chronic peritoneal irritation. Additional contraindications exist with gas-filled bowel loops on plain film suggesting obstruction. These gas-filled loops show bowel hypomotility, which means that the administered barium will not move down the GI tract and will present an added hazard at laparotomy (barium leakage into the peritoneum must be avoided at surgery). In the presence of gas-filled bowel loops (and after having ruled out parvovirus enteritis), surgery is indicated, without the administration of barium.

Ultrasonography in Small Intestinal Disease

Ultrasonography is very useful in the investigation of chronic small intestinal disease and is often more helpful than radiographic contrast studies. It allows the assessment of gut wall thickening and gut wall layers. In lymphosarcoma, for example, there tends to be a loss of the normal five wall layers whereas they are retained in IBD; the distinction, however, is not always absolute. Any local areas of thickening may be amenable to fine-needle aspiration under ultrasound guidance to allow cytologic assessment, although the differentiation of severe lymphocytic-plasmacytic enteritis from lymphoma may be difficult even on histology and cytology. Furthermore, intussusceptions and foreign bodies may be visualized. Ultrasonography also allows assessment of the pancreas, liver, stomach, and other abdominal organs.

Biopsy in Small Intestinal Disease

Endoscopic biopsy is only possible from the duodenum, and the biopsy is then only mucosal. Unfortunately, this does not allow inspection of the rest of the GI tract or other abdominal organs.

Laparotomy may be preferable in many cases as it allows visual inspection of other organs, including the stomach, pancreas, liver, and mesenteric lymph nodes. Also, multiple full thickness biopsies can be collected from the length of the gut. However, laparotomy is highly invasive and should performed only after extensive diagnostic workup. Patients with PLE and low plasma proteins show a high risk of wound breakdown and peritonitis.

PATHOPHYSIOLOGY OF DIARRHEA

Diarrhea is defined as an increase in the frequency, fluidity, and volume of feces. A 10-20% increase in fecal fluid cause obvious diarrhea. However, large breed dogs tend to have softer feces and this should not be confused with diarrhea.

The daily volume of fluid absorbed from the jejunum has been measured as 1350 ml. This represents approximately 50% of the volume that enters the jejunum daily, and 75 % of the remaining fluid will be absorbed in the ileum. Liquid is then further absorbed very efficiently in the colon. The colon also has a large reserve capacity, as it can increase its fluid absorption threefold if necessary. Therefore, quite a small change in fluid production by the colon can result in diarrhea.

Underlying causes of small and large intestinal diarrhea may be classified as:

- **Osmotic diarrhea**
- **Secretory diarrhea**
- **Permeability diarrhea**
- **Motility diarrhea**

However, there may be some overlap with more than one type of diarrhea present at any one time.

Osmotic Diarrhea

This is the most common form of diarrhea in small animals. It can be caused by dietary indiscretion, some laxatives, “gastric dumping,” and maldigestion/malabsorption. There

is an increase in unabsorbed solutes causing an increase in fecal water. The diarrhea does not contain blood or excess protein and typically ends with dietary rest.

Secretory Diarrhea

Secretory diarrhea is caused by an increased secretion of fluid and ions (in the crypts) or reduced absorption (at the villi tips). It can be caused by bacterial enterotoxin damage, viral damage to the villi, the presence of undigested hydroxy fatty acids, bile salts, and some laxatives.

It classically continues when food is withheld, although it may stop if the secretagogues, (e.g., hydroxy fatty acids) are the result of maldigestion. The feces do not contain blood or excess protein.

Permeability Diarrhea

Permeability diarrhea is caused by an increased permeability of the epithelial cells and tight cell junctions. Possible causes include an increase in the mucosal blood pressure (e.g., with portal hypertension or right-sided heart failure), mucosal damage associated with IBD and neoplasia, gluten enteropathy, infections, and some toxins.

Depending on the severity of the condition, the following stages can be distinguished:

- Mild permeability diarrhea→ electrolytes leak into the gut.
- More severe permeability diarrhea → small plasma proteins leak into the gut.
- Most severe permeability diarrhea → proteins and white and red blood cells leak into the gut.

Severe cases can therefore develop PLE, with or without melena, as well as HGE. Increased gut permeability encourages the absorption of potential antigens into the body with a possible hypersensitive reaction and a breakdown of immune tolerance. This type of diarrhea usually improves but does not stop with dietary rest.

Motility Diarrhea

This type of diarrhea is caused by deranged GI motility decreasing (or increasing) the intestinal transit time and is usually characterized by reduced rhythmic segmentation rather than increased frequency of peristalsis. Causes include many inflammatory and

parasitic diseases, canine dysautonomia, canine megacolon, and colitis. The condition does occur frequently but is usually very poorly characterized in animals; thus there is no "typical" clinical picture, although it tends not to be associated with PLE or melena and tends to improve on fasting.

MANAGEMENT

Dietary management is vitally important in all small intestinal diseases in order to limit the development of acquired hypersensitivities seen with increased mucosal permeability, to maximize nutrient absorption, to allow mucosal healing, and to help resolution of clinical signs. Dietary management should be used in combination with appropriate fluid therapy and medical or surgical management, depending on the disease involved.

Fluid Therapy

Appropriate fluid therapy is important in all small intestinal diseases, since increased mucosal permeability, diarrhea, and vomiting can result in the loss of large amounts of water and electrolytes. Fluids and electrolytes may be provided orally in mild disease or intravenously in more severe cases, such as surgical patients with obstructions, torsions, HE, or parvovirus enteritis. Careful attention to fluid therapy is particularly important in puppies, as they dehydrate rapidly due to their small size and the inability of their kidneys to concentrate urine as effectively as adult dogs. After restoring fluid and electrolyte balance, consideration should be given to dietary, medical, and/or surgical management, as appropriate.

Medical Management

Fluid therapy and dietary management are central to the treatment of small intestinal disease. In addition, the following drug therapies may be used where appropriate:

- **Antiinflammatory drugs:** Antiinflammatory doses are recommended in lymphocytic-plasmacytic enteritis, immunosuppressive doses in eosinophilic enteritis. A hypoallergenic diet should be tried in all cases of idiopathic IBD as it may render antiinflammatory drugs unnecessary or at least lower the required dose. In cases of concurrent SIBO, the use of steroids should be delayed until SIBO is treated.
- **Antibiotics:** The use of antibiotics should be avoided in small intestinal disease, except in those cases where there is a specific indication, such as *Campylobacter* or *Giardia*, SIBO, parvovirus enteritis, or HGE with a significant risk of gram-negative

septicemia. Antibiotics should not be used in cases of uncomplicated acute diarrhea as their discriminate use can cause a number of possible problems.

- **Wormers:** Effective endoparasite control should be given as necessary.
- **Antidiarrheals and absorbents:** These may be used but have limited usefulness. Motility modifiers include parasympatholytics, which paralyze the GI tract, and opiates, which reduce motility by increasing segmental contractions and are also antisecretory. These drugs are typically used to make the condition more acceptable to the owner and are best avoided in most acute diarrheas as they are contraindicated in infectious diarrhea and also predispose to SIBO. Opiates may be preferred to parasympatholytics as the latter predispose to ileus.
- **Probiotics:** Diarrhea is associated with a change in small intestinal microflora with a decrease in *Lactobacillus* spp. and an increase in bacteroides and enterobacteria. Probiotics or live yogurts have been given to try to regain the normal flora, but there is little evidence for their effectiveness. The bacteria in probiotics are unlikely to survive gastric acid and even if they do, there is no evidence they displace normal or abnormal bacteria in the SI. Some forms of soluble fiber, however, have shown to affect the SI microflora.

Problems with Indiscriminate Use of Antibiotics

- Suppression of normal bowel flora delays recovery, predisposes to SIBO, and may allow pathogens such as *Salmonella* to colonize the SI.
- Antibiotics used in *Salmonella* infection may prolong the carrier state and can be recommended only in cases of septicemia.
- The antibiotic itself may be associated with significant GI side effects that might be hard to distinguish from the primary disease, such as vomiting or diarrhea (through direct effects and effects on the microflora). Antibiotics may also interfere with action of pancreatic lipase and reduce bile acid resorption.

Surgical Management

Surgery is indicated in a number of small intestinal diseases such as partial or total obstruction by foreign bodies or tumors, intussusceptions, torsions, hernias, adhesions, strictures, and small intestinal perforations. It is also indicated for full thickness small intestinal biopsy. Before performing surgery, it is vital to ensure the patient is stabilized and treat shock and fluid and electrolyte imbalances. It is also vital to restore blood albumin levels in any dog with very low preoperative plasma proteins, as seen in PLE. This is either done by dietary means or by perioperative plasma transfusions, as low blood albumin levels are associated with a significant increase in postoperative wound dehiscence.

Postoperatively, it is important to feed the patient as soon as possible, ideally within 24 hours, unless a specific contraindication to early feeding exists. Early feeding after enteropathy significantly reduces the incidence of complications such as wound dehiscence or GI bacterial translocation causing septicemia. Bacterial translocation may occur, as the small intestinal epithelium obtains 50% of its nutrient requirements from the intraluminal products of digestion, particularly glutamine, and adequate protein and calories are needed to maintain the integrity of the mucosal tight junctions, the function of brush border enzymes, and efficient local immunity. Contrary to popular belief, feeding does not increase “stress” on sutures in the small intestinal wall. In fact, the opposite is true, as contractions that predominate in the fasting state are larger and more forceful than those that occur when fed.

Dietary Management

Dietary management is important as primary or supportive therapy in all small intestinal diseases, including those managed surgically. Short-term dietary rest followed by feeding of an appropriate highly digestible, single-protein diet is indicated in acute uncomplicated gastroenteritis.

Dietary rest is inappropriate in chronic small intestinal diseases, except for short-term control of vomiting or osmotic diarrhea.

Dietary manipulations are very important, since changes in the components of the food have profound effects on small intestinal digestion and absorption . Diet is important in both the pathogenesis of small intestinal disease and for nutrition and healing of the small intestinal mucosa. Dietary components of particular importance in small intestinal disease are fat and protein. Also, recently interest has focused on the use of soluble fiber to modify the small intestinal microflora.

Acute Gastroenteritis/Feeding a “Bland” Diet

Acute gastroenteritis is often due to dietary indiscretion, change of diet, or overfeeding. In many cases of acute diarrhea, a definitive diagnosis is not determined because symptomatic treatment is effective in reversing the clinical signs.

Classically, advice is to allow dietary rest for 24-48 hours, as this will help the resolution of any vomiting and/or osmotic diarrhea. Food is then reintroduced in form of a “bland” diet, fed little and often. A “bland” diet can be defined as a high-quality, highly digestible, and nonspicy diet containing components that pose a low risk of adverse reactions.

There has been some controversy about whether total dietary rest (i.e., starvation) is actually necessary in nonvomiting patients. Studies in human infants suggest that "feeding through" diarrhea speeds the healing process by maintaining better mucosal barrier integrity, by providing energy for active electrolyte uptake, and by minimizing malnutrition. However, although the duration of diarrhea is not prolonged by "feeding through," it certainly increases diarrhea production, especially in cases of osmotic diarrhea. To many dog owners, an initial deterioration of diarrhea is not acceptable (as dogs do not wear diapers), so short-term starvation is usually indicated to stop diarrhea.

The use of oral rehydration formulas is one way of avoiding total GI rest and is important to ensure effective rehydration.

Once feeding is resumed, a "bland," "hypoallergenic" diet minimizes the risk of acquiring dietary hypersensitivities. The GI tract is exposed to a wide variety of potentially allergenic food components but has a system of defense mechanisms to prevent allergens from penetrating the intestinal mucosa and initiating an allergic response. Antigens (e.g., dietary proteins) are broken down by intestinal enzymes and entry is prohibited by tight cell junctions of the intestinal lining. It has been estimated that only 0.002% of dietary protein is absorbed intact, which is then tackled by antibodies from the GALT. A hypersensitive reaction occurs with increased mucosal uptake of antigens bypassing the normal antigen presentation system. When the intestinal tract is damaged by a period of inflammation, as in enteritis, protective mechanisms break down. There is a reduction in enzyme action combined with an increase in intestinal permeability due to weakening of the tight cell junctions, facilitating antigen uptake. During this period, the patient could acquire a dietary hypersensitivity.

In order to minimize the risk of this process, it may be important to minimize the number of protein sources fed, so that any potentially harmful protein will be easily identified. It has even been recommended to choose a protein source that is not normally part of the animal's diet (i.e., one that is not commonly found in pet foods) and "sacrifice" this protein as a potential dietary allergen; protein sources used in this way are also called "sacrificial proteins." The risk of dietary hypersensitivity can be further reduced by feeding a highly digestible protein, thus reducing the amount of luminal antigen available for absorption.

Formulating and Feeding a Home-Prepared Hypoallergenic Diet

- A detailed list of the animal's diet, including everything that the dog has eaten during its lifetime, should be compiled; by studying the list, it may be possible to identify foods that have not previously been fed and are therefore "novel" and can form the basis of a diet "hypoallergenic" for that individual.
- Novel sources of protein and ideally also carbohydrate are then chosen; however, rice is often used as it has a very low allergenicity even if it is usually not novel to the dog.
- There is no single diet that will be "hypoallergenic" for all patients; however, certain

ingredients such as chicken and rice are highly digestible and therefore have a low allergic potential.

- "Common" allergens will vary with time and part of the world as they are largely dependent on the frequency with which these protein sources are fed to pets.
- Dietary hypersensitivities are most commonly caused by proteins, mainly by cow's milk, beef, or cereal, alone or in combination.
- Hypoallergenic diets that have been successfully used in dogs include chicken, rabbit, lamb, horsemeat, and fish as sources of protein; these are typically fed with rice, potatoes, or another source of carbohydrate. The carbohydrate source should be free of cereal gluten.
- It is important to stress to pet owners that the same source of protein and carbohydrate must be used for every meal when preparing a home-cooked diet, including the same type of fish. So, if the chosen diet is based on cod and rice, for example, it is crucial that cod is used consistently and not replaced by another type of white fish. It may therefore be necessary to stock up on more exotic ingredients.
- It is important to ensure that the diet is balanced, particularly when used in young, immature animals. Nutritional deficiencies can be irreversible and may only manifest at a much later stage, and it is therefore crucial to ensure a balanced intake of all nutrients.
- Home-prepared diets usually require appropriate supplementation with vitamins and minerals.
- Patients may develop a hypersensitivity to the protein source used in the diet if the intestinal tract remains inflamed and leaky; in these cases the protein source ought to be changed after 6 weeks or as soon as the condition recurs.

Energy

Chronic small intestinal diseases associated with chronic vomiting and/or PLE will predispose to the development of protein-calorie malnutrition. It is therefore important to feed an energy-dense diet to enable sufficient caloric intake to maintain lean body mass and to allow small intestinal mucosal healing and efficient immune function. Energy intake can be optimized if the diet is fed little and often; this also helps to avoid overwhelming the compromised small intestinal digestive and absorptive capabilities.

Protein

Protein intake should be restricted only if there is a specific indication, such as concurrent renal disease or hepatic encephalopathy. Adequate protein intake promotes good epithelial cell turnover, production of small intestinal brush border enzymes, and GI

immunity. Protein restriction can have a negative impact on all these processes and thus increase the risk bacterial translocation from the gut.

The amino acid glutamine is a particularly important energy source for the small intestinal epithelial cells. Enterocytes receive about 50% of their energy requirements from glutamine, so adequate dietary levels are very important in all small intestinal disease, after small intestinal surgery, and in all enteral critical care diets.

It is particularly important not to restrict dietary protein intake in cases of PLE. In these dogs, it is advisable to feed high levels of high-quality, highly digestible protein to help restore plasma protein levels. Low plasma protein levels not only predispose to the development of cavity effusions and edema but also have serious deleterious effects on the gut, which may prolong diarrhea and predispose to wound breakdown after biopsy or surgery. The protein fed should always be very digestible in small intestinal diseases, both acute and chronic, to reduce the risk of acquired hypersensitivities developing to undigested antigens.

Fat

Dietary fat restriction does not appear to be necessary in acute gastroenteritis, although fat restriction is often important in chronic small intestinal disease.

Dogs with chronic small intestinal diarrhea may develop fat maldigestion/malabsorption for one or more of the following reasons:

- In EPI, fat digestion is reduced due to the reduction of pancreatic lipase, and dietary fat restriction is therefore crucial.
- In SIBO, bacteria deconjugate bile salts, reducing fat emulsification and therefore digestion. Bacteria also break down the undigested fat to hydroxy fatty acids. These and deconjugated bile salts irritate the colonic mucosa and can cause secretory large intestinal diarrhea.
- Fat malabsorption will occur in any condition causing villi atrophy and hence reduced small intestinal surface area.
- Fat malabsorption occurs in lymphangiectasia, where the lymphatics are dilated and not functioning. Feeding a low-fat diet is the cornerstone in treating this condition as reducing dietary fat results in reduced lymphatic flow.
- Disease of the distal ileum may result in defective bile salt reabsorption with reduced bile salts and lipid maldigestion.

Dietary fats are essential for the supply of essential fatty acids and fat-soluble vitamins, and fat usually contributes a significant proportion of the daily energy intake, as it is very energy dense. It is therefore important to ensure adequate caloric intake with low-fat

diets, particularly in active dogs, and to avoid weight loss. It may be possible to substitute fat with medium-chain triglycerides (MCTs), such as coconut oil, fed with or between meals. MCTs contain chains of 8-12 carbons, compared with 16-18 in fatty acids. They do not require bile acids for absorption, as they are taken up directly into the capillaries rather than the lymphatics. It is, however, important to carefully control the level of intake as unabsorbed MCTs can cause osmotic diarrhea, and ¼ to 4 teaspoons a day in divided doses has been recommended for dogs. MCTs do not carry fat-soluble vitamins, which may have to be supplemented additionally. In some individuals, MCTs may cause vomiting.

Carbohydrate and Fiber

Carbohydrate provides the main source of calories in many chronic small intestinal diseases where a low-fat diet is indicated and should therefore be readily digestible. Easy digestion of the dietary components also allows the distal SI to “rest.”

Some carbohydrates, such as gluten, can cause specific dietary sensitivities and thus need to be avoided in the patient’s diet.

Using a single source of a highly digestible carbohydrate can help to reduce the risk of acquired hypersensitivities.

Generally, a low-fiber diet is indicated in small intestinal disease, as fiber interferes with small intestinal absorption and the activity of brush border enzymes. Fiber also impairs the activity of pancreatic enzymes. However, some specific soluble fiber sources, such as sugar beet pulp, have been shown to have a potentially beneficial action in SIBO, as they are broken down by bacteria in the SI and promote the growth of “beneficial” bacteria.

Vitamins and Minerals

Specific vitamin requirements in small intestinal disease are as follows:

- **B Vitamins**
  - Folate levels may be reduced in jejunal disease as it is absorbed in the jejunum; however, levels are increased in SIBO.
  - Vitamin B_{12} may be reduced in SIBO, ileal disease, and EPI due to a deficiency of pancreatic intrinsic factor needed for its absorption.
  - Both folate and vitamin B_{12} are necessary for small intestinal health, so if they are found to be low in the course of diagnostic tests, it is important to ensure supplementation. Parenteral supplementation is more effective than enteral, as small intestinal absorption is impaired

- **Fat soluble vitamins** should be supplemented in diseases associated with fat maldigestion/malabsorption. Sufficient intake needs to be ensured when feeding a low-fat diet. Most commercial low-fat diets, however, are balanced and further supplementation is not recommended and can even be toxic.
- Adequate amounts and a good balance of all **essential vitamin**s should be included in long-term diets for chronic small intestinal disease. This is particularly important when using home-prepared "hypoallergenic" diets. Home-prepared diets are often used short term for diagnostic purposes; however, they are best replaced with suitable balanced commercial diets in order to avoid nutrient deficiencies.

The most important **mineral** to consider in small intestinal disease is potassium, as chronic diarrhea and vomiting often result in hypokalemia. This is particularly important in dogs with concurrent anorexia and/or receiving intravenous fluids low in potassium. Dietary potassium supplementation may therefore be necessary after assessing the patient's blood levels.

SUMMARY

With acute small intestinal diarrhea, a definitive diagnosis is rarely established, as patients usually respond well to short-term dietary rest followed by feeding a highly digestible, "bland" diet. Feeding a "bland" diet with a minimum number of ingredients minimizes the risk of the patient developing an acquired dietary hypersensitivity while the gut is inflamed and permeable.

With chronic IBD, total dietary rest is not usually indicated; for most chronic small intestinal diseases, feeding a "bland," highly digestible diet little and often is beneficial. Moderate fat and fiber levels are usually best. These general recommendations are appropriate for most small intestinal problems; there are, however, diseases that require specific dietary management.

Diseases requiring a specific nutrient modification include SIBO and lymphangiectasia. These disorders respond best to a highly digestible, low-fat diet.