DIARRHEA
The most common causes of diarrhea in puppies and kittens are infectious, endoparasitic, and dietary. True congenital intestinal diseases, such as intestinal atresia, are rarely encountered, because affected individuals die shortly after birth.

Infectious Diarrhea
Infectious agents associated with diarrhea in young dogs and cats are typically bacterial or viral. Viral infections in dogs include canine parvovirus, canine distemper, coronavirus, and rotavirus, whereas parvovirus (feline panleukopenia), coronavirus, and retroviruses (feline leukemia virus [FeLV] and feline immunodeficiency virus [FIV]) are important in young cats. Bacterial organisms responsible for neonatal diarrhea can include Salmonella spp, Escherichia coli, Clostridium spp, Yersinia enterocolitica, and Campylobacter spp [1,2]. Diagnosis in most patients is usually uncomplicated and is based on typical clinical signs, serology or demonstration of virus in feces, or specialized fecal cultures for certain bacterial organisms. Treatment consists of supportive care, fluid therapy, and specific antibacterial drugs.

Canine parvovirus is the most significant cause of infectious diarrhea in young dogs; canine parvovirus type 1 (CPV-1, minute virus) is usually only considered pathogenic in puppies less than 6 weeks of age, whereas the more commonly encountered CPV-2 can cause disease in dogs of any age. Pathogenesis and transmission of the two organisms are similar. Histologic changes seen in CPV-1 are similar, although less severe, than those seen in CPV-2, and CPV-1 is capable of crossing the placenta and causing early fetal death or birth defects. CPV-1 is most commonly encountered in extremely young puppies, and presenting clinical signs include diarrhea, emesis, dyspnea, constant vocalizing, and sudden death. There are no routine diagnostic tests available, although CPV-1 infection can be confirmed by fecal electron microscopy, virus neutralization, or hemagglutination inhibition. Treatment is primarily
supportive, including fluid therapy and adequate nutrition, but is often unrea-
doning because of rapid progression of the disease. Prophylactic vaccines are
not available for CPV-1 at present [3].

CPV-2 infection is typically seen in young dogs without protective antibody
titers, because of a lack of or an incomplete series of vaccinations. A window of
susceptibility also occurs in puppies, in which maternal antibody falls below
protective levels but vaccine-induced immunity is lacking. A breed predilection
in Rottweilers, Doberman Pinschers, and Staffordshire Terriers has been re-
ported, and male dogs may be more commonly affected [4]. Additionally, a sea-
sonal prevalence in the summer months occurs in temperate climates. Clinical
signs of CPV-2 infection include lethargy, anorexia, vomiting, bloody diarrhea,
fever, and dehydration, and the clinical course is often protracted. Leukopenia
characterized by decreased neutrophil and lymphocyte counts is considered a
laboratory hallmark of the disease and results from viral lymphocytolysis,
bone marrow precursor destruction, and peripheral neutrophil consumption.
Anemia and hypoproteinemia are common as a consequence of gastrointestinal
(GI) blood loss and prolonged malnutrition. Significant secondary complica-
tions of CPV-2 infection include intestinal intussusception, sepsis, and dissemin-
ated intravascular coagulation (DIC). A diagnosis of CPV-2 infection is
confirmed by virus isolation, electron microscopy, fecal hemagglutination, or
fecal ELISA testing. The ELISA antigen test (CITE, Idexx, Sacramento, Cali-
ifornia) performed on fecal material is the most common and practical diagnos-
tic test in a clinical setting; negative test results can be seen early in the course of
disease, and modified-live virus vaccines can cause weak positive test results
from 5 to 15 days after vaccination [5]. Treatment of CPV-2 infections includes
aggressive intravenous fluid therapy, parenteral nutrition, antibiotics, and anti-
emetics. Fluid therapy is directed toward maintenance of hydration and control
of metabolic consequences, such as hypokalemia or hypoglycemia. Colloid
therapy (eg, hetastarch, plasma) is indicated in cases with severe hypoprotein-
emia, and blood product administration may be necessary if anemia becomes
sufficiently severe. Parenteral broad-spectrum antibiotic therapy is mandatory
because of disruption of the GI mucosal barrier and concomitant severe neutro-
penia; ampicillin or a first- or second-generation cephalosporin in combination
with an aminoglycoside (eg, cefazolin, amikacin) is quite effective. Recom-
mented antiemetics include metoclopramide as a constant-rate infusion or pro-
chlorperazine (Table 1). Both are centrally acting agents, and metoclopramide
also acts as a GI prokinetic agent. With aggressive treatment, the prognosis for
recovery from CPV-2 infection is considered good, and 80% to 90% of affected
dogs recover. Certain breeds (eg, Rottweilers) may experience higher mortality,
however. Prophylactic vaccines are available and are effective if the manufac-
turer’s directions are followed carefully.

The role of pathogenic bacteria in acute or chronic diarrheal disease remains
unclear, because organisms may be isolated with similar frequency from
healthy and ill animals. Most likely, these organisms are opportunists and
establish pathogenicity when some other insult has occurred to disrupt the
GI microenvironment. The incidence of infections seems to be highest in young kenneled animals or in immunocompromised patients. Clinical signs typically include watery, mucoid, or hemorrhagic diarrhea; in some cases (eg, *Salmonella*), systemic spread resulting in sepsis can occur. The diagnosis is established by specific fecal culture (eg, *Salmonella* spp, *Campylobacter* spp) or toxin assays (eg, *Clostridia* spp), whereas in some instances (eg, enteropathogenic *E coli*), there are no practical clinically available diagnostic tests. Treatment is appropriate antibiotic therapy; however, antibiotic resistance and carrier states represent significant concerns, and a decision to treat is based on the animal’s clinical status rather than merely laboratory detection of a potentially pathogenic organism [1].

**Endoparasitism**

Clinically significant endoparasitism of young dogs and cats includes roundworms (*Toxocara canis, Toxocara cati*, and *Toxascaris leonina*), hookworms (*Ancylostoma caninum, Ancylostoma tubaeforme*, and *Uncinia stenocephala*), tapeworms (*Dipylidium caninum, Echinococcus granulosus*, and *Taenia* spp), and *Strongyloides stercoralis* as well as protozoal organisms, such as coccidia (*Isospora* spp), *Cryptosporidium parvum*, and *Giardia* spp. Clinical signs are variable and range from asymptomatic to life threatening; most common clinical signs include diarrhea, weight loss, or failure to gain weight. *A caninum* can be associated with severe hemorrhagic enteritis and anemia in puppies. Diagnosis of endoparasitic infestations is most commonly made by fecal flotation, and centrifugation techniques may improve diagnostic accuracy. *S stercoralis* may be demonstrated by the Baermann flotation technique or by observation of larvae in fresh fecal smears. *Giardia* infections can be diagnosed by observation of motile trophozoites in fresh fecal smears or by detection of cysts using zinc sulfate fecal flotation; however, a more recently developed fecal ELISA test that detects *Giardia* antigen is the preferred diagnostic test.

A number of effective anthelmintics are available for the treatment of helminth infestations (see Table 1), and routine treatment of young animals is recommended, even without diagnostic confirmation. It can safely be assumed that almost all puppies, for example, have *T canis* infection because of transplacental or transmammary larval transmission [1], and some regimens suggest treatment every 2 to 4 weeks until 16 weeks of age and then every 6 months thereafter. Control of many helminth infections can be easily accomplished by monthly administration of combined anthelmintic and heartworm preventative (eg, ivermectin plus pyrantel pamoate). Treatment of *Dipylidium* infections must include adequate flea control as well as an appropriate anthelmintic, such as praziquantel. Coccidial infections are treated with sulfa-containing drugs, such as sulfadimethoxine or trimethoprim sulfa; although frequently diagnosed and treated in the clinical setting, most coccidial infections are likely self-limiting. Fenbendazole is the recommended treatment for *Giardia* infection; although metronidazole is commonly used, it is proven less effective than fenbendazole and has greater potential for side effects [6]. A *Giardia* vaccine is also
<table>
<thead>
<tr>
<th>Generic name</th>
<th>Trade name</th>
<th>Indications</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthelmintics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fenbendazole</td>
<td>Panacur (Hoechst-Roussel)</td>
<td>Ascarids, hookworms, whipworms, Giardia, Taenia</td>
<td>50 mg/kg/d PO for 3 consecutive days</td>
</tr>
<tr>
<td>Ivermectin</td>
<td>Ivomec, Heartgard (Merck AgVet)</td>
<td>Ascarids, hookworms, whipworms, heartworm preventative</td>
<td>200 μg/kg PO as anthelmintic but not approved, 6 μg/kg PO monthly for heartworm preventative</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>Flagyl (Searle, Geneva Pharmacueticals)</td>
<td>Giardia, Trichomonas</td>
<td>15 mg/kg PO twice daily</td>
</tr>
<tr>
<td>Milbemycin oxime</td>
<td>Interceptor (Ciba-Geigy Animal Health)</td>
<td>Heartworm preventative, ascarids, hookworms, whipworms</td>
<td>0.5 mg/kg PO monthly</td>
</tr>
<tr>
<td>Praziquantel</td>
<td>Droncit (Miles)</td>
<td>Tapeworms</td>
<td>Dogs: 5 mg/kg PO, SQ, IM</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cats: 11 mg 1–3 lb, 22 mg 3–11 lb, 33 mg &gt;11 lb</td>
</tr>
<tr>
<td>Praziquantel + pyrantel pamoate</td>
<td>Drontal (Bayer)</td>
<td>Ascarids, hookworms, tapeworms</td>
<td>See package insert (cats)</td>
</tr>
<tr>
<td>Praziquantel + pyrantel pamoate + febantel</td>
<td>Drontal Plus (Bayer)</td>
<td>Ascarids, hookworms, whipworms, tapeworms</td>
<td>See package insert (dogs)</td>
</tr>
<tr>
<td>Pyrantel pamoate</td>
<td>Nemex, Pfizer</td>
<td>Ascarids, hookworms</td>
<td>15 mg/kg PO</td>
</tr>
<tr>
<td>Sulfadimethoxine</td>
<td>Albon (Hoffman-La Roche), Bactrim (Pitman-Moore)</td>
<td>Coccidia</td>
<td>55 mg/kg PO first day, then 12.5 mg/kg PO twice daily for 14–21 days</td>
</tr>
<tr>
<td>Thiabendazole</td>
<td>Mintezol (Merck)</td>
<td>Strongyloides</td>
<td>50–100 mg/kg PO once daily for 3–5 days</td>
</tr>
<tr>
<td>Antiemetics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chlorpromazine</td>
<td>Thorazine (SmithKline Beecham Pharmaceuticals)</td>
<td>Effective phenothiazine</td>
<td>0.5 mg/kg IM q 8 hours</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>Benadryl (Parke-Davis)</td>
<td>Effective antihistamine, give before travel</td>
<td>0.05 mg/kg IV (dogs)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2–4 mg/kg PO q 8 hours</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2 mg/kg IM q 8 hours</td>
</tr>
<tr>
<td><strong>Metoclopramide</strong></td>
<td>Reglan (A.H. Robins Company)</td>
<td>Gastric motility disorders, esophageal reflux, give 30 minutes before meals and at bedtime</td>
<td>0.2–0.4 mg/kg PO, SQ q 8 hours 1–2 mg/kg q 24 hours CRI Do not exceed single doses of 1 mg/kg</td>
</tr>
<tr>
<td><strong>Ondansetron Prochlorperazine</strong></td>
<td>Compazine (SmithKline Beecham Pharmaceuticals)</td>
<td>Effective phenothiazine</td>
<td>0.1 mg/kg IM q 6 hours</td>
</tr>
<tr>
<td><strong>Antimicrobials</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Amikacin</strong></td>
<td>Many</td>
<td>Systemic infections</td>
<td>10–20 mg/kg IV, IM, SQ q 24 hours 22 mg/kg PO q 12 hours</td>
</tr>
<tr>
<td><strong>Amoxicillin</strong></td>
<td>Many</td>
<td>Combine with aminoglycoside for systemic infections</td>
<td>10–20 mg/kg PO q 6–8 hours 5–10 mg/kg IV, IM, SQ q 6–8 hours 22 mg/kg PO q 12 hours</td>
</tr>
<tr>
<td><strong>Ampicillin</strong></td>
<td>Many</td>
<td>Combine with aminoglycoside for systemic infections</td>
<td>22 mg/kg PO q 12 hours</td>
</tr>
<tr>
<td><strong>Cefadroxil</strong></td>
<td>Cefa-Tabs (Fort Dodge Laboratories)</td>
<td>Combine with aminoglycoside for systemic infections</td>
<td>22 mg/kg PO q 12 hours</td>
</tr>
<tr>
<td><strong>Cefoxitin sodium</strong></td>
<td>Mefoxin (Merck)</td>
<td>Combine with aminoglycoside for systemic infections</td>
<td>22 mg/kg IV, IM</td>
</tr>
<tr>
<td><strong>Cephalexin</strong></td>
<td>Keflex (Dista Products)</td>
<td>Combine with aminoglycoside for systemic infections</td>
<td>20 mg/kg PO, SQ, IV q 8 hours</td>
</tr>
<tr>
<td><strong>Cephradine</strong></td>
<td>Velosef (Bristol-Myers Squibb Company)</td>
<td>Combine with aminoglycoside for systemic infections</td>
<td>10–20 mg/kg PO q 8 hours</td>
</tr>
<tr>
<td><strong>Chloramphenicol</strong></td>
<td>Many</td>
<td>Salmonella, Campylobacter, Yersinia</td>
<td>50 mg/kg IV, IM, SQ, PO q 8 hours in dogs, q 12 hours in cats</td>
</tr>
<tr>
<td><strong>Clindamycin</strong></td>
<td>Antirobe (Pharmacia &amp; Upjohn)</td>
<td>Campylobacter, Toxoplasma, Cryptosporidium</td>
<td>3–5 mg/kg PO, IV, IM q 12 hours</td>
</tr>
<tr>
<td><strong>Erythromycin</strong></td>
<td>Many</td>
<td>Can cause vomiting, anorexia</td>
<td>10 mg/kg PO q 8 hours 2 mg/kg IM, SQ q 8–12 hours</td>
</tr>
<tr>
<td><strong>Gentamicin</strong></td>
<td>Many</td>
<td>Systemic infections, Shigella, Yersinia, Salmonella</td>
<td></td>
</tr>
<tr>
<td><strong>Metronidazole</strong></td>
<td>Flagyl (Searle, Geneva Pharmaceuticals)</td>
<td>Anaerobic infections</td>
<td>7.5 mg/kg PO, IV q 8–12 hours</td>
</tr>
</tbody>
</table>

(continued on next page)
### Table 1 (continued)

<table>
<thead>
<tr>
<th>Generic name</th>
<th>Trade name</th>
<th>Indications</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trimethoprim-sulfadiazine</td>
<td>Septra (GlaxoWellcome)</td>
<td>Salmonella, Yersinia</td>
<td>15 mg/kg PO, IM, SQ q 12 hours</td>
</tr>
<tr>
<td></td>
<td>Tylan (Elanco)</td>
<td>Intestinal bacterial overgrowth</td>
<td>20–40 mg/kg PO q 12 hours in dogs</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>5–10 mg/kg PO q 12 hours in cats</td>
</tr>
<tr>
<td>Tylosin</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastrointestinal protectants</td>
<td>Pepto Bismol (Procter and Gamble)</td>
<td>Nonspecific diarrhea</td>
<td>10–20 mg/kg q 8–12 hours</td>
</tr>
<tr>
<td></td>
<td>Carafate (Marion Merrell Dow)</td>
<td>Gastrointestinal ulceration, give 60 minutes before acid blockers</td>
<td>100–1000 mg PO q 6–8 hours in dogs</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>100–200 mg PO q 6–8 hours in cats</td>
</tr>
<tr>
<td>Bismuth subsalicylate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sucralfate</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cimetidine</td>
<td>Tagamet (SmithKline Beecham Pharmaceuticals)</td>
<td>Gastrointestinal ulceration, inhibits hepatic microsomal enzymes, avoid in animals less than 3 months of age</td>
<td>5 mg/kg PO, IV, IM q 8–12 hours</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>Zantac (Glaxo Pharmaceuticals)</td>
<td>Gastrointestinal ulceration, does not inhibit microsomal enzymes, avoid in animals less than 3 months of age</td>
<td>2–4 mg/kg PO, IV, SQ q 12 hours can be used as CRI</td>
</tr>
<tr>
<td>Famotidine</td>
<td>Pepcid (Merck)</td>
<td>Gastrointestinal ulceration, does not inhibit microsomal enzymes, avoid in animals less than 3 months of age</td>
<td>0.5–1.0 mg/kg PO q 12–24 hours</td>
</tr>
<tr>
<td>Omeprazole</td>
<td>Prilosec (Procter &amp; Gamble)</td>
<td>Gastrointestinal ulceration</td>
<td>0.5–1.0 mg/kg PO q 24 hours</td>
</tr>
<tr>
<td>Miscellaneou</td>
<td>Drug</td>
<td>Manufacturer</td>
<td>Use</td>
</tr>
<tr>
<td>-------------</td>
<td>------</td>
<td>--------------</td>
<td>-----</td>
</tr>
<tr>
<td>Diphenoxylate</td>
<td>Lomotil</td>
<td>Searle &amp; Company</td>
<td>Antidiarrheal</td>
</tr>
<tr>
<td>Loperamide</td>
<td>Imodium</td>
<td>McNeil Consumer</td>
<td>Antidiarrheal, narcotic</td>
</tr>
<tr>
<td>Lactulose</td>
<td>Cephulac, Chronulac</td>
<td>Marion Merrell Dow</td>
<td>Laxative, hepatoencephalopathy</td>
</tr>
<tr>
<td>Dioctyl sodium sulfosuccinate</td>
<td>Colace</td>
<td>Bristol-Myers Squibb</td>
<td>Laxative, 50- and 100-mg capsules</td>
</tr>
<tr>
<td>Bran</td>
<td></td>
<td></td>
<td>Fiber source, laxative, colitis</td>
</tr>
<tr>
<td>Psyllium</td>
<td>Metamucil</td>
<td>Procter &amp; Gamble</td>
<td>Fiber source, laxative, colitis</td>
</tr>
</tbody>
</table>

Abbreviations: CRI, constant-rate infusion; IM, intramuscular; IV, intravenously; PO, orally; q, every; SQ, subcutaneously.
available, and it has been suggested that it may be effective in clearing refractory infections [7]; however, vaccination against *Giardia* failed to decrease the incidence of diarrhea associated with giardiasis in one field study (A.D. Davidson, DVM, MS, personal communication, 2001). Some endoparasites may pose significant public health concerns, including *T. canis* (ie, visceral and ocular larval migrans) and *Giardia* spp, and appropriate client counseling is advised.

Diet-associated Diarrhea

Dietary causes of enterocolitis and diarrhea in young animals include dietary intolerance, dietary hypersensitivity, ingestion of spoiled foods (“garbage gut”), ingestion of foreign material, and abrupt dietary changes. Additionally, many drugs (eg, nonsteroidal anti-inflammatory drugs [NSAIDs], corticosteroids, antibiotics, anthelmintics), chemicals like cleaning agents or fertilizers, and plants or plant toxins can cause enterocolitis and associated diarrhea. Most nonspecific enterocolitis of young animals (ie, dietary indiscretions) are acute and self-limiting, although clinical signs can be fulminant and even life threatening in certain cases. The diagnosis in such cases is usually straightforward and is based on the history, typical clinical signs, and examination findings. Symptomatic treatment is warranted in most cases before extensive diagnostic procedures, and most patients with nonspecific enterocolitis show marked improvement in 24 to 48 hours with little or no treatment. General principles of treatment include removal or avoidance of the inciting cause, correction of metabolic or electrolyte disturbances, promotion of GI mucosal repair, and control of secondary complications (ie, vomiting or abdominal pain). Dietary restriction represents the primary mode of treatment, and withholding of food for 24 to 48 hours is typically recommended. In theory, withholding of food leads to more rapid restoration of mucosal integrity and return to normal function as well as decreasing diarrhea by eliminating irritant or osmotic effects of undigested nutrients. There is an opposing school of thought that espouses “feeding through the diarrhea” on the premise that continued provision of micronutrients hastens restoration of mucosal integrity; benefits of this approach may be more relevant in secretory diarrhea, which is likely uncommon in small animal patients [1]. If clinical signs abate with an appropriate period of GI rest, gradual introduction of a highly digestible, reduced-fat, low-fiber diet is done using cooked rice or cereal supplemented with low-fat cottage cheese, chicken or lean ground beef, or baby foods. Alternatively, there are many commercial or prescription diets formulated for GI disease that may be used. A normal diet may be reintroduced over several days if clinical signs have resolved.

Bismuth subsalicylate can be used empirically as a GI protectant if necessary (see Table 1), and there is evidence that salicylates may decrease intestinal hypersecretion by inhibition of prostaglandin synthesis. Salicylate-containing compounds should be used cautiously in cats.

Parenteral fluid therapy may be indicated in some patients with enterocolitis if dehydration or electrolyte or acid-base imbalances have resulted. Attention is
paid to the routine guidelines of fluid therapy: correct dehydration, supply
daily maintenance, and replace ongoing losses. Hypokalemia is a common find-
ing in patients with continued vomiting and diarrhea, and supplementation
should be adjusted using frequent measurement of serum potassium levels.

Adverse Food Reactions

Adverse reactions to food may represent an immunologic reaction to a dietary
antigen (ie, food allergy) or a nonimmunologic reaction (ie, dietary intoler-
ance). Clinical signs are similar for both and include vomiting, diarrhea, or
abdominal discomfort, which may be immediate or delayed in onset. Dermatol-
ologic signs, such as papules, erythema, pruritus, pyoderma, military dermatitis
(cats), and eosinophilic granuloma (cats), may coexist with GI signs in patients
with a dietary allergy, although this concurrence has been infrequently docu-
mented [1,8].

Diagnosis of food allergy or intolerance relies on the clinical response to an
exclusion diet and recurrence of clinical signs with reintroduction of the offend-
ing dietary component. Often, the diagnosis must be considered presumptive,
because many other GI diseases improve with dietary manipulations, and most
clients decline rechallenge of their pets with suspect diets. Additionally, histo-
logic changes seen in GI biopsy samples do not distinguish food allergy from
other chronic mucosal infiltrative diseases, such as inflammatory bowel disease
(IBD). Indirect tests for food allergy have been studied and are advocated by
some individuals; however, none of these are considered reliable in veterinary
patients. Such indirect tests for food allergy include measurement of antigen-
specific serum antibodies (eg, RAST [radioallergosorbent], ELISA), skin test-
ing, and gastroscopic food sensitivity testing [1,9,10].

Although dietary trials are used to diagnose food allergies and intolerances,
they do not distinguish between the two, nor do they necessarily eliminate
from consideration other dietary-responsive GI diseases, such as lymphangiec-
tasia or IBD. The primary principle of a dietary exclusion trial is to feed dietary
components to which the patient has not been previously exposed, and this diet
should represent the sole nutrition source for the period of the trial. The rec-
ommended exclusion diet should consist of a single protein and carbohydrate
source. Typically, a “novel protein” combined with rice is used; diets may be
homemade, although there are also a plethora of appropriate commercial exclu-
sion diets that consist of chicken, soy, fish, venison, or duck combined with car-
bohydrates, such as rice, corn, tapioca, or potato. Hydrolyzed protein diets (eg,
Hill’s Ultra Z/D, Hill’s, Topeka, Kansas; Purina HA or LA, Purina, St. Louis,
Missouri) are also available, in which proteins have been hydrolyzed into com-
ponents of a molecular weight purportedly too small to elicit an immune re-
sponse, although there is not proof that these diets are superior to the more
standard novel protein diet. The trial diet is typically fed for a period of
3 weeks [1], although it has been shown that cases of food-allergic skin disease
may require up to 10 weeks for remission. Treatment consists of continuation
of a diet formulated based on the response to the exclusion trial; many suitable
commercial diets are available and may be preferable because they are nutritionally balanced and more convenient.

During episodes of severe enteritis, the mucosal barrier is breached, and dietary proteins gain access to the mucosal-associated immune system; in this way, patients may become sensitized and subsequently intolerant of certain dietary proteins. Accordingly, some individuals recommend the feeding of a simplified or hydrolyzed protein diet for several weeks after such illness. Alternatively, switching to a different protein-based diet several weeks after an episode of enteritis has also been advocated.

**IDIOPATHIC CHRONIC DIARRHEA OF YOUNG CATS**

A refractory idiopathic chronic diarrhea is seen in young cats and is characterized by watery and voluminous small bowel diarrhea. Affected cats are usually normal otherwise and maintain a good appetite and body condition. Routine diagnostics are typically unrewarding, and the disorder may result from intestinal functional immaturity, poor feeding practices, or undiagnosed infectious or endoparasitic diseases. Treatment strategies include administration of appropriate anthelmintics and dietary trials with novel protein source foods. Fiber supplementation to the diet may be helpful in some cases, and in the author’s experience, some patients may improve with antibiotic therapy with metronidazole or amoxicillin. These cases can be frustrating for the client and clinician; however, in most affected cats, resolution occurs by 1 year of age [3].

**MEGAESOPHAGUS**

Congenital megaesophagus is characterized by generalized esophageal hypomotility, dilatation, and unsuccessful passage of ingesta from the oropharynx to the stomach. A predilection is reported in the Miniature Schnauzer, Fox Terrier, German Shepherd, Chinese Shar-Pei, Great Dane, Labrador Retriever, Newfoundland, and Irish Setter breeds [11]. Congenital megaesophagus is rare in cats but has been seen in association with dysautonomia or pyloric dysfunction; Siamese and Siamese-related breeds have a higher incidence [12]. The pathogenesis of congenital megaesophagus is enigmatic, with some studies suggesting vagal afferent innervation defects, whereas others have suggested intrinsic esophageal biomechanical dysfunction [13].

Regurgitation is the most common clinical sign seen in affected individuals and may vary in frequency from only occasional to multiple daily episodes. Regurgitation is often described as passive or effortless and may occur within minutes to hours postprandially, and regurgitated material often appears undigested and mixed with mucus or saliva. Because of the frequent regurgitation, affected individuals are often malnourished and cachectic on examination. Aspiration pneumonia is a common complication of megaesophagus and may be reflected in fever, cough, or pulmonary adventitious sounds heard during thoracic auscultation.

Routine laboratory diagnostics are typically unremarkable in animals with congenital megaesophagus, although an inflammatory leukogram is seen in some cases with aspiration pneumonia. The diagnosis is usually easily made
with survey thoracic radiographic findings in concert with the typical presenting signs, but a barium contrast study is recommended for diagnostic confirmation and to exclude possible other causes of megaesophagus, such as a foreign body or congenital anomaly like a persistent right aortic arch. Endoscopic examination of the esophagus is not typically considered contributory, although it may certainly confirm the diagnosis; in a small percentage of cases, secondary esophagitis may be documented by endoscopy.

Therapy for congenital megaesophagus consists of nutritional management and treatment of aspiration pneumonia episodes. A high-calorie diet in small frequent feedings from an elevated or upright position is recommended. A period of trial and error may be needed to determine the most appropriate dietary consistency, because some individuals tolerate a completely liquid diet best, whereas others perform better when given solid foods. Being held in an upright position for 15 to 30 minutes after eating may be helpful in some animals by facilitating gravity drainage of the esophagus. Individuals that cannot maintain an adequate nutritional level orally may require gastrostomy tube placement, surgically or endoscopically, for feeding.

Aspiration pneumonia is treated with appropriate antibiotic therapy, which is ideally selected on the basis of culture and sensitivity of samples collected by a tracheal wash or endoscopic bronchoalveolar lavage. Supportive treatment measures for aspiration pneumonia include supplemental oxygen administration, thoracic coupage, and perhaps nebulization therapy. It is recommended that antibiotic therapy be continued for at least 7 to 10 days beyond radiographic resolution of the pneumonia.

GI prokinetic agents, such as metoclopramide and cisapride, have been used empirically in the treatment of megaesophagus; however, they typically result in significant worsening of signs of regurgitation. This clinical worsening occurs from a marked increase in lower esophageal sphincter resting pressure in response to these drugs, thereby increasing resistance to esophageal outflow, whereas improvement in esophageal motility, if any, is insubstantial.

The prognosis for congenital megaesophagus is considered fair. Many affected animals eventually show improvement in esophageal motility and function if adequate nutritional support is provided. Repeated episodes of aspiration pneumonia are, unfortunately, common, and a decision to euthanatize because of these is a leading cause of mortality.

PORTOSYSTEMIC SHUNTS
A portosystemic shunt (PSS) is a congenital malformation of the hepatic portal venous drainage system and can have a familial (ie, genetic) or random occurrence. A congenital PSS can be intrahepatic or extrahepatic; breed predilections for extrahepatic shunts include the Yorkshire Terrier, Maltese, Poodle, Miniature Schnauzer, Dachshund, Lhasa Apso, Pekingese, Pug, and Shih Tzu. Intrahepatic shunts are more commonly identified in large-breed dogs, such as Golden Retrievers, German Shepherds, Irish Wolfhounds, Irish Setters, and Samoyeds [14]. PSSs are uncommon in cats.
Clinical signs attributable to a PSS are most often seen before 2 years of age, although they may not be evident in some individuals until much later in life. Clinical signs of a PSS result from loss of hepatic metabolic functions and often include drug (anesthetics or sedatives) intolerance, hepatic encephalopathy, anorexia, vomiting, and diarrhea. Hepatic encephalopathy may lead to seizures, stupor, coma, amaurosis, or mentation changes. Ammonium biurate calculi may develop in some individuals and can cause stranguria, pollakiuria, and hematuria, and the finding of urate calculi in a non-Dalmation breed should raise suspicion of a PSS. Individuals with a PSS are often stunted in growth. Ptyalism is often a prominent clinical sign in cats with PSSs. Other clinical findings in cats include cardiac murmurs, renomegaly, and a characteristic “golden” or copper discoloration of the iris [15].

Biochemical abnormalities commonly associated with a PSS include mild to moderate liver enzyme elevations, hypoglycemia, low blood urea nitrogen (BUN), hypoalbuminemia, and hypocholesterolemia. Red blood cell microcytosis or mild nonregenerative anemia may be seen. Ammonium biurate crystalluria, hematuria, and pyuria can occur in animals with urate uroliths. Although uncommon, routine blood work and urinalysis can be completely normal in some individuals with a PSS. Other tests of liver function include measurement of serum bile acids and blood ammonia levels. Bile acid tolerance testing is recommended, in which fasting and 2-hour postprandial levels are measured. Although not pathognomonic for a PSS, marked elevations in bile acid levels are characteristic and should greatly elevate the index of suspicion in patients of appropriate age and clinical presentation. Blood ammonia levels are measured postprandially after ingestion of a high-protein meal or after oral ammonia challenge, and hyperammonemia is a frequent finding.

Abdominal ultrasonography is a useful diagnostic technique and is routinely done when a PSS is suspected. It is noninvasive and requires no anesthesia; however, diagnostic accuracy is highly operator dependent, and the PSS is confirmed in only approximately 60% to 80% of cases [16]. Transcolonic portal scintigraphy is another useful diagnostic tool and has a high degree of accuracy. Disadvantages include limited availability, extensive patient preparation, and the potential need for sedation or anesthesia during the procedure. Transsplenic portography after intrasplenic contrast material injection can also be used to identify a PSS successfully and is a relatively simple procedure, although it also does require anesthesia. Finally, mesenteric portography, although it is the most invasive technique and requires general anesthesia, is a highly reliable method of confirming and localizing a PSS.

Treatment of a PSS may be palliative by medical management or definitive by surgical ligation. It must be emphasized that medical management is only a symptomatic therapy and that a delay in surgical ligation may lead to further deterioration in liver function or ability to recover after shunt ligation.

Medical therapy of patients with a PSS is directed primarily at controlling signs of hepatoencephalopathy (HE) by limiting absorption of toxins from the GI tract and manipulation of dietary proteins. Lactulose is a nonabsorbed
synthetic disaccharide that is degraded to organic acids by colonic bacteria. This leads to a decrease in colonic luminal pH, resulting in ionic trapping of ammonia as ammonium ion, which is incapable of diffusing into the portal circulation. Additionally, the organic acid byproducts of lactulose digestion act as an osmotic cathartic, thereby decreasing colon retention time and absorption of toxic metabolites. Lactulose as well as neomycin or povidone iodine may be used in retention enemas in the treatment of patients in encephalopathic crisis. Antibiotics like metronidazole, neomycin, ampicillin, or amoxicillin are routinely used to suppress intestinal anaerobic flora responsible for enteric ammonia production (see Table 1).

Blood within the GI tract is a potent source of ammonia production, and treatment of GI hemorrhage is a key component in the successful management of HE. Treatment considerations include H₂-receptor blockers or proton-pump inhibitors to decrease acid secretion, coating agents like sucralfate (see Table 1), and treatment of any primary causes like endoparasitism.

Restriction of dietary protein intake is a critical element of the medical management of a PSS. Diets high in branched-chain amino acids and limited in aromatic amino acids are recommended, with a recommended daily protein intake of 2.0 to 2.5 mg/kg [15]. Milk and vegetable proteins, such as cottage cheese and tofu, are excellent protein sources, with the bulk of dietary caloric intake consisting of carbohydrates, such as boiled white rice. Small frequent feedings are recommended to maximize digestion and absorption. Commercial low-protein diets, such as L/D, K/D or I/D (Hill’s, Topeka, Kansas), are suitable for use in dogs and cats with a PSS, and there are also homemade diets that can be beneficial.

Surgical shunt ligation is the definitive treatment for a PSS, and the current recommended method of ligation is by placement of an amaroid constrictor. The amaroid constrictor consists of a dehydrated casein core surrounded by a metal ring. After placement of the amaroid constrictor, the casein core absorbs water and enlarges, leading to gradual occlusion of the shunting vessel over a period of several weeks to months. This avoids the major complication of portal hypertension seen in many cases after abrupt shunt occlusion. Postoperative complications are uncommon but can include portal hypertension, postoperative seizures, or rupture of the shunting vessel, all of which have a high mortality rate; however, overall mortality with surgical shunt ligation is less than 10%.

**CHRONIC ENTEROPATHIES**

A genetic predilection to small intestinal disease has been reported in several breeds of dogs. An immunoproliferative enteropathy is seen in the Basenji breed, which is characterized by lymphangiectasia, intermittent diarrhea, weight loss, hypoalbuminemia and hyperglobulinemia, and lymphoplasmacytic mucosal infiltrates throughout the GI tract [17]. Other reported clinical signs include paresis, seizures, facial muscle contracture, buccal lymphadenomegaly, and adverse vaccine reactions. The diagnosis is confirmed by intestinal histopathologic examination obtained via a laparotomy or endoscopically obtained
biopsy samples. Treatment options include dietary manipulation, corticosteroids, and antibiotic therapy. Diets are typically formulated as fat restricted and high-quality protein and may include commercial GI diets, weight control diets, or appropriate homemade formulas. Frequent small feedings and fat-soluble vitamin supplementation are recommended. Medium-chain triglycerides are used as a dietary caloric source that bypasses lymphatic absorption and are usually added to the diet in an oil form; however, vomiting and diarrhea can be problematic side effects. Corticosteroids are usually administered initially at a higher dosage to "induce remission" and are then tapered slowly to a lower and more tolerable maintenance level. Antibiotic therapy with agents like metronidazole, amoxicillin, or tylosin (see Table 1) may be beneficial in individuals with suspected or confirmed bacterial overgrowth.

Wheat-sensitive or gluten enteropathy has been reported in Irish Setter dogs and is clinically typified by weight loss or poor weight gain as well as by intermittent diarrhea episodes [18,19]. This disorder is attributable to selective deficiencies in certain brush border enzymes, resulting in hypersensitivity to wheat in the diet. Histopathologic changes are often patchy or segmental and are typified by villous atrophy without a significant inflammatory infiltrate. Treatment is elimination of wheat from the diet.

Chinese Sharpei dogs have been identified with an enteropathy that is characterized by poor weight gain, weight loss, or intermittent diarrhea episodes, with onset of signs typically between 2 and 6 months of age [2]. Histopathologic examination of intestinal biopsy samples shows lymphoplasmacytic-eosinophilic mucosal infiltrates. Treatment is dietary management and immunosuppressive therapy with corticosteroids; however, the response to therapy is variable, and more potent immunosuppressives, such as azathioprine, may be necessary in certain individuals. Bacterial overgrowth is not infrequent, as reflected in elevated serum folate and decreased serum cobalamin levels, and some affected dogs may benefit from long-term antibiotic therapy.

Selective cobalamin malabsorption has been identified in Giant Schnauzer dogs with clinical signs of anorexia, lethargy, and poor weight gain or weight loss, usually beginning between 3 and 6 months of age; nonregenerative megaloblastic anemia and neutropenia are also characteristic [20]. This disorder results from a defect in transport of the intrinsic factor cobalamin receptor complex to the brush border membrane in the ileum. Treatment is parenteral cobalamin administration weekly for 4 to 6 weeks and then every 3 months thereafter. Recovery is usually complete.

**JUVENILE PANCREATIC ATROPHY**

Pancreatic atrophy or hypoplasia of unknown but perhaps genetic cause is reported in young dogs; German Shepherd dogs are predisposed, accounting for approximately one half of the reported cases [21]. Pancreatic exocrine insufficiency (PEI) results, and clinical signs of diarrhea, weight loss, and steatorrhea usually develop between 6 and 12 of age. Diagnosis is typically straightforward
and is established by finding markedly decreased serum trypsin–like immuno-reactivity levels.

Primary treatment consists of dietary pancreatic enzyme replacement using agents like pancreatin or pancrelipase. Powdered formulations or crushed non–enteric-coated tablets are recommended, because absorption of enteric-coated tablets is unpredictable. Mixing of pancreatic enzyme replacements with food 30 to 60 minutes before feeding is recommended so as to circumvent the loss of enzyme activity that occurs in the stomach. Reduced-fat high-carbohydrate diets are typically recommended, because lipids are the most severely mal-absorbed dietary nutrient; however, some afflicted individuals respond better to non–fat-restricted diets. Frequent smaller feedings (eg, three times a day) are recommended. High-fiber diets are avoided, because dietary fiber has been shown to decrease intestinal pancreatic enzyme activity and perhaps pancreatic secretion.

Many patients also benefit from acid secretion inhibition with drugs like cimetidine, ranitidine, famotidine, or omeprazole (see Table 1). Intestinal bacterial overgrowth often accompanies PEI, and antibiotic therapy may be useful in refractory patients. The prognosis for PEI is considered good.

References


