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Esophageal, Gastric, and Intestinal Disorders of Young Dogs and Cats

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Esophagogastroenteric disorders, manifest as regurgitation, vomiting, or diarrhea, are common clinical complaints in young dogs and cats. The majority of these disorders in young animals are acute problems due to infectious or parasitic agents. Dietary changes or intolerances and congenital defects may also be responsible for gastrointestinal disease in young pets.

Regardless of the etiology, these diseases require prompt and appropriate symptomatic therapy. Because of their smaller total fluid volume compared with adults, young animals are particularly susceptible to dehydration and acid-base and electrolyte disturbances. For most infectious enteric diseases, symptomatic therapy is sufficient; for the viral enteritides, it is the only therapy available. Parasitic and congenital disorders, on the other hand, require specific diagnosis and treatment. For these reasons, we have divided this discussion into three sections: (1) infectious diseases, (2) parasitic diseases, and (3) congenital and other diseases of the gastrointestinal tract of young dogs and cats. For each disease, we have indicated the presentations for which it should be considered, as well as the means of definitive diagnosis, treatment, and prevention. There also is a brief discussion of general supportive therapy for gastroenteritis at the end of this article.

INFECTIONIOUS DISEASES OF THE GASTROINTESTINAL TRACT

Canine Parvovirus

Canine parvoviral enteritis is the most important, most frequent, and most problematic of the gastroenteric disorders of young dogs. It continues to occur sporadically among puppies despite rigorous vaccination programs using effective vaccines. It is endemic in most breeding kennels, successfully resisting efforts to eliminate it. The disease was unknown prior to 1977.

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Retrospective serologic surveys suggest that the virus first evolved at about the same time a mutant of the feline panleukopenia virus or some other closely related parvovirus.

**Clinical Indications.** Canine parvoviral enteritis must be considered in any dog with an acute onset of fever, vomiting, and diarrhea. It is especially likely in young dogs 6 to 20 weeks of age. Because of the blocking effect of maternal antibody (see below), a history of vaccination against parvovirus is not sufficient to rule out the diagnosis in pups up to about 1 year of age.

**Clinical Signs.** Clinical signs vary from mild or subclinical to acute and fulminating. The full spectrum of disease may be seen within a single litter. Stress, concurrent disease, and intestinal parasites have been purported to increase the severity of parvoviral enteritis, although the precise factors that control the outcome of infection remain unknown. All breeds are susceptible, although the Doberman Pinscher and Rottweiler appear to be at higher risk of developing clinical disease. In the Doberman Pinscher, parvoviral enteritis often occurs shortly after ear cropping.

Classically, there is a prodromal period of depression, inappetence, and lethargy lasting 12 to 36 hours. This is followed by vomiting and diarrhea, frequently severe and hemorrhagic. Affected puppies are usually febrile, often markedly so. Leukopenia is a distinguishing characteristic when present. The expression of disease is extremely variable, however, and it cannot be ruled out simply because some of the classic signs are missing. For example, leukopenia is observed in only about 33 per cent of dogs at the time of admission, although serial hemograms reveal leukopenia in about 85 per cent of clinically affected dogs. The more severe the leukopenia, the poorer the prognosis.

A second syndrome—sudden death from parvoviral myocarditis—appeared at the same time that parvoviral enteritis was first recognized. Affected puppies were either found dead or died shortly after the appearance of signs suggestive of acute heart failure. Canine parvovirus was isolated from the myocardium of such animals and the syndrome could be reproduced experimentally only if pups were infected within 1 to 2 weeks of birth. Parvoviral myocarditis has virtually disappeared as a clinical problem because most bitches are now immune; thus, puppies cannot be infected during the period critical for the development of myocarditis.

**Diagnosis.** Specific diagnosis is not essential for appropriate therapy. In cases in which it is important to establish diagnosis for other reasons, parvoviral enteritis can be confirmed by serology, histopathology, viral isolation, or other methods of detecting virus in feces (electron microscopy, ELISA, stool hemagglutination tests). For serologic diagnosis, it is essential to demonstrate the presence of specific antiparvovirus IgM in order to differentiate active infection from preexisting titers. Tests that rely on the detection of virus are only applicable during the period of clinical illness; the period of viral shed is brief (5 to 10 days).

**Treatment.** Treatment is supportive only and follows the guidelines given at the end of this article. In addition, prophylactic parenteral antibiotic therapy is indicated in severe cases, because secondary septicemia and other bacterial infections have been observed as fatal sequelae. A suggested regimen is ampicillin (11 to 22 mg per kg three times a day) and gentamicin...
(2.2 mg per kg three times a day) to provide broad-spectrum protection against both aerobic and anaerobic bacteria. If gentamicin or other aminoglycoside antibiotics are used, however, care must be taken to ensure adequate hydration to avoid serious nephrotoxicity.

**Prevention.** Prevention depends on vaccination combined with appropriate kennel management. A variety of effective vaccines are available from various manufacturers, either alone or in combination with distemper, hepatitis, leptospirosis, parainfluenza, and bordetella vaccines. Although concern has been expressed about potential immunosuppression by attenuated canine parvovirus in combined vaccines, these concerns have never been supported by appropriately controlled laboratory studies. Indeed, there is evidence that vaccinal parvovirus is not immunosuppressive.

The principal barrier to complete control through vaccination appears to be maternal antibody interference with immunization. It has been known since the late 1950s that the antibody that pups acquire from their dams, principally through the colostrum, can suppress response to vaccination. The unexpected finding with respect to canine parvovirus was the duration of this effect (in some cases, for more than 18 weeks after birth). Moreover, there is a period during which maternal antibody titers are too low to prevent infection but are still sufficient to preclude response to vaccination. These latter findings account for the continued occurrence of parvoviral enteritis among vaccinated pups and the inability to eradicate the disease in breeding kennels despite heroic vaccination strategies. Modified live canine parvovirus (CPV) vaccines appear to be less susceptible to maternal antibody interference, and low passage strains may be superior to more highly attenuated strains in this regard.

The best current recommendations seem to be to (1) keep pups relatively isolated through the critical period of susceptibility (6 to 20 weeks of age); (2) maintain high levels of sanitation and care; and (3) vaccinate pups routinely. Although it is not possible to eliminate the virus from a premise, even by vigorous cleaning, reduced exposure and better overall health of pups appear to increase the likelihood of mild or inapparent infection. Although there are no absolute, experimentally tested vaccination protocols, it appears that pups should be vaccinated first at 6 to 8 weeks of age and then periodically revaccinated until they are 18 to 20 weeks old. The number and timing of the intervening vaccinations depend on a number of factors, including economic considerations and the prevalence of disease in the area. Hence, specific vaccination protocols should be determined on an individual basis. Even so, no vaccination program can completely prevent parvoviral enteritis, and owners need to be informed of this unfortunate reality.

**Feline Panleukopenia**

Feline panleukopenia is a contagious systemic disease of young cats, most often characterized by severe enteritis. It is caused by feline panleukopenia virus (FPV), a parvovirus closely related to, and perhaps even the progenitor of, canine parovirus.

**Clinical Indications.** Panleukopenia must be considered in the differential diagnosis for any severe, acute enteritis of young cats, especially those accompanied by severe systemic signs. The disease is most frequently en-
countered in the late summer and fall when the large spring crop of kittens are reaching the end of their maternal antibody protection. As with canine parvovirus, maternally derived antibodies can suppress response to vaccination; thus, in kittens, a history of vaccination for panleukopenia is not sufficient reason to rule out the disease.

**Clinical Signs.** Initial signs are anorexia and lethargy followed by persistent vomiting. Rectal temperatures commonly exceed 40°C. Diarrhea, often bloody, commences concurrent with or follows shortly after the onset of emesis.

The hallmark of panleukopenia in cats, as the name suggests, is a profound leukopenia, observed in nearly 100 per cent of field cases. A marked and progressive leukopenia begins on the 2nd or 3rd day after infection. By the 4th to 6th day, there may be fewer than 100 white blood cells per cubic mm of blood. As with canine parvovirus, there appears to be an inverse correlation between the severity of the leukopenia and the likelihood of survival; the prognosis for cats with extreme leukopenia is very poor. In animals that survive the infection, the white count rapidly returns to normal or greater than normal numbers in 5 to 7 days.

Feline panleukopenia infection of kittens either in utero or shortly after birth can result in a second distinct syndrome of chronic cerebellar ataxia. Signs are not apparent until the kittens first learn to walk at several weeks of age, but the signs are nonprogressive. Affected kittens have a base-wide stance and hypermetria. All actions are exaggerated and tend to “overshoot the mark,” oscillating on either side of their intended goal. The disease results from viral destruction of the flask (Purkinje) cells of the cerebellum at a critical stage of development. Feline panleukopenia-induced ataxia is now rarely encountered because most kittens are refractory to infection by virtue of maternal or maternally derived immunity during the critical period of susceptibility.

**Diagnosis.** Because the leukopenia that occurs in FPV infections is both consistent and profound, it is probably sufficient evidence to confirm the diagnosis in a young cat with enteric signs. The histopathologic lesions are also specific. As with CPV, serologic diagnosis or the identification of virus in the feces is possible by several means, although this is seldom necessary.

**Prevention.** Feline panleukopenia is well controlled by routine immunization with commercial vaccines. Both attenuated live virus and inactivated virus vaccines are available. Some published reports indicate that attenuated live virus vaccines provide both more rapid and more durable protection, especially in kittens with residual maternal antibody. There is insufficient evidence to conclude that this is true for all vaccines and, in any case, both types of vaccine appear to be effective in practice. The recommended immunization schedule begins with vaccination at 6 to 8 weeks of age and continues as biweekly vaccinations until the kittens are at least 12 weeks old.

**Canine Coronavirus**

The true incidence of coronaviral enteritis is not known. Serologic studies suggest that infection may be relatively common, but that serious clinical
disease is not. There is some evidence that coronaviral enteritis may be an endemic problem in certain kennels, but more study of the entire question is needed.

**Clinical Indications.** Coronaviral enteritis should be considered in cases of acute, mild to moderate enteritis, especially among young dogs, and particularly if more than one animal is affected simultaneously.

**Clinical Signs.** The clinical signs of coronaviral enteritis are nonspecific and include an acute onset of vomiting, diarrhea, depression, and anorexia. A “typical” color and odor of the stool have been described, but these hardly seem reliable diagnostic criteria. The absence of fever and leukopenia may be of some use in differentiating coronaviral from parvoviral enteritis, but their absence does little to distinguish coronavirus from any number of other causes of diarrhea in puppies. Reliable information about incidence, morbidity, and mortality is lacking.

**Diagnosis.** As with other viral enteritides, diagnosis is usually an academic exercise since it neither rules out other causes absolutely nor affects the treatment regimen. Coronaviral particles can be identified in feces by electron microscopy in appropriately equipped diagnostic laboratories. There is some question about the reliability of this method; coronaviral particles deteriorate rapidly, leading to false-negative results, and artifacts in the sample may mimic virions, leading to false-positive results. Coronaviral serology is also available from a few laboratories.

**Prevention.** An inactivated canine coronavirus vaccine has been recently introduced in the United States. In laboratory studies, dogs were challenged 2 weeks after two vaccinations and killed on the 5th day: coronavirus was either not recovered or was recovered in significantly reduced amounts from the intestine of vaccinated dogs compared with unvaccinated controls. At the time of this writing, however, appropriately controlled field trials of vaccine efficacy have not been published. Given the uncertainty about the true significance of coronaviral enteritis in dogs and the unanswered questions about the long-term field efficacy of the vaccine, it seems appropriate to proceed cautiously in recommending coronaviral vaccination. Practitioners may have to experiment, trying the vaccine in problem kennels and carefully monitoring the results.

### Feline Coronaviruses

A number of feline enteric coronaviruses have been recognized in recent years. These all share antigenic cross-reactivity with feline infectious peritonitis virus (FIPV). It is probable that virulent FIPV and the enteric coronaviruses represent opposite ends of a spectrum of virulence among a family of closely related viruses. The enteric coronaviruses appear to be widespread in the cat population, and at least some strains are capable of causing enteritis and diarrhea in kittens.

**Clinical Indications.** Feline enteric coronavirus infection should be considered in cases of relatively mild, self-limiting enteritis in kittens. Severe, fatal infections occur rarely and would have to be differentiated from panleukopenia.

**Clinical Signs.** Feline enteric coronaviruses produce an acute episode of diarrhea and occasional vomiting in young cats. Except for depression and
inappetence, systemic signs are usually absent. Coronaviruses are highly contagious and the entire litter is usually affected simultaneously.

**Diagnosis.** Feline enteric coronaviruses can be identified in fecal samples by electron microscopy. As with canine coronavirus, the viral particles deteriorate rapidly so that prompt processing of fresh specimens is essential. Antibodies to the enteric coronaviruses cross-react with FIPV extensively on serologic tests. Hence, it would be possible to use seroconversion to FIPV as evidence of feline coronavirus infection.

**Treatment.** Most cases resolve spontaneously with simple supportive treatment.

**Prevention.** No vaccine is available. Vaccine development will not be possible until the relationships between FIPV and the enteric coronaviruses are clarified. Antibody to some enteric strains appears capable of sensitizing kittens to FIPV, predisposing them to more rapid and severe peritonitis when they are subsequently exposed to FIPV. Considering that feline coronaviral enteritis is sporadic and usually self-limiting, vaccination may not be warranted.

It is possible to maintain a coronavirus-free closed cattery. This can be done by using serologic tests for FIPV to exclude carriers. This is usually done for the purpose of eliminating FIPV and is necessary only in special circumstances (for example, research colonies).

**Canine and Feline Rotaviruses**

Rotaviruses have been identified as important causes of neonatal diarrhea in a large number of mammals. In man, they are believed to be the leading cause of enteritis in young children. The importance of rotaviruses in canine and feline pediatrics, however, is very much in doubt. It may be that we have failed to recognize their importance, but it seems more likely that they are only infrequent causes of neonatal diarrhea in young pets.

**Clinical Indications.** Rotaviral enteritis should be included in the differential diagnosis of acute enteritis in puppies and kittens. It appears to be an important problem only in very young animals (most reported cases have been younger than 6 weeks old).

**Clinical Signs.** The number of confirmed case descriptions is limited, but as with coronaviral infections, the signs appear to be those of a nonspecific small bowel enteritis. Systemic signs include diarrhea, vomiting, dehydration, depression, and inappetence.

**Diagnosis.** The simplest and surest means of diagnosis is identification of the virus in the feces. A commercial enzyme-linked immunoassay* is available. Rotaviruses can also be identified by electron microscopy, although care must be taken to differentiate them from apparently nonpathogenic reoviruses that are sometimes observed in dog feces.

**Treatment.** Treatment is supportive only.

**Prevention.** There is no commercial vaccine available at this time for the prevention of rotaviral enteritis in dogs or cats. Some success has been achieved in developing vaccines to protect human infants and calves, so that

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*Rotazyme. Abbott Laboratories, North Chicago, Illinois.*
an efficacious vaccine—at least one that could provide protection during the neonatal period—might be possible. If our present estimates of the incidence of rotaviral enteritis in puppies and kittens are correct, however, they do not appear to justify widespread vaccination.

Colibacillosis

Although colibacillosis is cited as a cause of enteritis in puppies and kittens by virtually every standard text, there is little reliable information about its true importance in clinical practice. It was probably overdiagnosed in the past, unsolved cases of pediatric diarrhea being lumped together under a diagnosis of "colibacillosis" without laboratory confirmation.

**Clinical Indications.** Colibacillosis should be suspected in cases of profuse watery diarrhea not accompanied by systemic signs of illness.

**Clinical Signs.** Colibacillosis produces classic scours (that is, profuse "rice water" diarrhea). There may be evidence of abdominal cramps or pain, but other systemic signs are usually absent; vomiting is not usually part of the clinical syndrome.

**Diagnosis.** Simple isolation of *Escherichia coli* from diarrheic feces is of no significance; the organism is a normal large bowel inhabitant. Definitive diagnosis of colibacillosis requires demonstrating that the recovered organisms are either invasive or enterotoxigenic by in vitro or in vivo laboratory tests. Serotyping can provide indirect evidence of pathogenicity since certain serotypes are associated with enterotoxemia. A new test, the colony blot hybridization method, has been developed for detecting enterotoxigenic strains of *E. coli*.\(^3\) The test uses a radiolabeled DNA probe to detect genes for stable toxin in *E. coli* colonies isolated from suspected cases of colibacillosis. Initially developed for use in identifying colibacillosis in swine, this relatively rapid, inexpensive, and reproducible test may also shed new light on the true importance of colibacillosis in young dogs and cats.

**Campylobacteriosis**

In the past decade, *Campylobacter jejuni* has emerged as one of the most important causes of acute enteritis in humans, rivaling *Salmonella* and *Shigella* in importance.\(^22\) All domestic animals, including dogs and cats, can act as reservoirs of infection, and animal to man transmission has been documented.\(^7\) Although pets are estimated to account for only about 5 per cent of human cases of campylobacter enteritis, puppies recently acquired from shelters seem to represent a human health hazard. A typical history is the acquisition of a new pup, the development of diarrhea by the puppy, and the subsequent development of diarrhea by the owner.\(^22\)

Several studies have revealed high rates of asymptomatic carriers among the dogs and cats. Carrier rates are higher among young animals and animals housed in crowded or unsanitary conditions. In most studies, fecal cultures from animals with diarrhea yielded *C. jejuni* significantly more often than did stool cultures from nondiarrheic animals. Nevertheless, campylobacteriosis does not appear to be a common primary cause of illness or mortality among young dogs and cats. Many reported clinical cases have had concurrent enteric protozoal, viral, or bacterial infections, leading to speculation that *Campylobacter* may be most often a secondary opportunistic pathogen.
**Clinical Indications.** Campylobacteriosis should be considered in young animals with acute or recurrent enteritis, especially in dogs recently obtained from a dealer or shelter. Concomitant enteric disease in the owner should increase the suspicion of campylobacteriosis.

**Clinical Signs.** Campylobacteriosis is characterized by loose, watery, mucus-laden diarrhea, which may be blood tinged and/or contain increased numbers of leukocytes. The diarrhea usually persists for 5 to 15 days, but may be recurrent or chronic. It is often accompanied by low grade fever and anorexia.

**Diagnosis.** Definitive diagnosis of campylobacteriosis is complicated by the high rate of subclinical carriers and the fastidious growth requirements of the organism. Various studies have reported isolation of *C. jejuni* from as many as 63 per cent of normal dogs in kennel situations. Thus, isolation itself is insufficient to implicate *C. jejuni* as the sole cause of the enteritis. The organism is microaerophilic and requires special growth medium and culture conditions. Anaerobic transport medium should be used when submitting samples. Broth enrichment procedures increase the rate of recovery. Growth and definitive identification take 2 to 5 days.

**Treatment.** Erythromycin is the drug of choice in man. A dosage of 80 mg per kg per day for 5 days has been used successfully in the dog and cat, although resistant strains are known to exist. Good supportive care (see below) is essential.

**Prevention.** No vaccine is available, and the incidence of disease does not appear to suggest the need for mass immunization. Too little is known about the natural history of campylobacteriosis to suggest specific prevention and control strategies. Because the disease is zoonotic and frequently severe in man, special care should be taken in handling suspected cases to avoid contamination of hospital personnel.

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**PARASITIC DISEASES OF THE GASTROINTESTINAL TRACT**

**Roundworms**

Nearly every puppy and kitten is infected with roundworms, either by transplacental migration, lactogenic spread, or both. Although most infections produce mild clinical signs (if any), heavily parasitized animals can become severely ill. Rarely, the infection is fatal. Importantly, roundworms in the intestine can predispose the gut to injury or insult by other causes that alone would not produce clinical disease.

**Clinical Indications.** The classic, unthrifty, "pot-bellied" puppy or kitten is recognized commonly in practice. The chief complaint is often the appearance of roundworms in vomitus. Abdominal palpation of cord-like thickening of the loops of small bowel in a young dog or cat is also indicative of ascariasis. Less commonly, a young pet with severe roundworm infection presents with signs suggestive of small bowel obstruction—anorexia, persistent vomiting, dehydration, lethargy, and no defecation.

**Clinical Signs.** In neonatal puppies, the clinical signs of roundworm infestation depend on the age of the pup, the route of infection, and the species of ascarid. Larvae of *Toxocara canis* encysted in the tissues of the
pregnant bitch are reactivated during the last trimester of pregnancy and migrate across the placenta to the pups in utero. These larvae enter the liver and lungs of the fetus where they wait until birth before moving to the gut. After whelping, the bitch also passes infective larvae to the pups via her milk.

The pulmonary phase of heavy prenatal and transmammary infections may prove fatal for pups within a few days of birth. More commonly, however, pups become unthrifty and develop respiratory distress, a dull coat, and a distended abdomen. They adopt a straddle-legged posture and experience various degrees of digestive disturbances. Heavy worm burdens interfere with digestion and nutrient adsorption and result in mild enteritis and subnormal growth. Heavy prenatal infections with *Toxocara canis* can also cause severe abdominal discomfort in young puppies, manifest as whimpering and crying. Large numbers of worms may appear in the vomitus or feces. Death can result from rupture or obstruction of the intestines and rarely, adult worms migrate into and block the pancreatic or biliary ducts.

The clinical signs of roundworm infection depend on the life cycle of the specific ascarid involved. Ingestion of infective eggs of *Toxocara canis* results in the release of larvae into the upper intestine. The larvae then migrate through the liver and lungs, disrupting the integrity and function of these organs. The larvae continue to migrate until they reach the respiratory tree, are coughed up, swallowed, and returned to the intestinal tract. In puppies younger than 5 weeks old, nearly all *T. canis* larvae take this route, eventually maturing in the gut. In older pups, larvae are distributed from the lungs in oxygenated blood to the somatic tissues where they encyst. In dogs 3 months of age and older, nearly all larvae will encyst rather than reenter the gut.

Larvae of *Toxascaris leonina* are released in the lumen of the stomach and invade the mucosa of the small intestine. Here they develop and molt before returning to the lumen of the bowel. Hence, infections with *T. leonina* produce only gastrointestinal disease, without the respiratory involvement characteristic of *T. canis*.

*Toxocara cati* infests kittens. Transmammary infection is the primary route of infection; transplacental migration does not occur. Like *T. leonina*, *T. cati* undergoes only mucosal migration and, thus, produces signs limited to the gastrointestinal tract.

**Diagnosis.** Definitive diagnosis of ascaridiasis depends on identification of ova in the feces of the affected puppy or kitten. In heavy infestations, ova are sufficiently numerous to be found on a direct fecal smear. In lighter infections, a concentration technique, such as a commercially available fecal flotation kit, may be necessary in order to find ova. The eggs are large, spherical, and easily recognized.

**Treatment.** A wide variety of effective anthelmintics are available for treating ascarid infections. Nursing pups seem to tolerate pyrantel pamoate best. It is available in a liquid suspension that facilitates the treatment of young dogs and cats. Another advantage is that pyrantel pamoate also has excellent activity against hookworms.

Effective treatment of heavy ascarid infestations in young pups and kittens requires repeated doses at 2-week intervals, beginning as early as 2
weeks of age. Three or four treatments may be necessary. Repeated treat-
ment is needed because none of the presently available anthelmintics has
any significant effect on the larval stages of ascarids and because transmam-
mary infection continues as long as the young continue to nurse. 24

Supportive treatment of puppies and kittens suffering from ascaridiasis
consists of attentive nursing care, warm, dry housing, and a palatable, bal-
anced diet.

Prevention. Attention must be focused on the bitch or queen since
they are the major source of infection. An anthelmintic effective against the
larval stages is needed. Recent studies have demonstrated that prenatal
transmission of *Toxocara canis* (and lactogenic transmission of *Ancylostoma
caninum*) from the bitch to her pups was reduced by more than 90 per cent
when fenbendazole (50 mg per kg) was administered once daily from the
40th day of pregnancy through the 2nd week after whelping. The cost of
this 37-day course of therapy is substantial so that it is probably only feasible
in valuable bitches that have previously had heavily infected litters or in
research colonies where the total elimination of ascarids is desired. 24

Hookworms

Hookworms are extremely common among puppies, especially in the
southern United States. For example, a survey of the prevalence of hook-
worms in pups younger than 6 months of age revealed an 85 per cent
prevalence in Georgia. 24 The severity of clinical disease varies greatly, how-
ever, from inapparent infection to mild persistent anemia to sudden death
from exsanguination.

The two major determinants of the severity of signs are the magnitude
of the challenge, as determined by the number and virulence of the hook-
worms, and the resistance of the host. The number of hookworms in a kitten
or puppy is largely determined by the degree of exposure to infective larvae.
The pathogenicity of any given number of worms is species-dependent. For
example, *Ancylostoma caninum* sucks much more blood than the other hook-
worms that infest dogs, and as a consequence is much more pathogenic. *A.
tubaeforme* is the most pathogenic hookworm of cats.

Host resistance reflects the animal’s ability to limit the number of hook-
worms maturing in the intestine. This, in turn, depends on the animal’s age,
immunity gained from previous exposure, and premunity engendered by
preexisting populations of hookworms. 9 Only the first factor has direct bear-
ing on the problem in young dogs and cats. In general, young animals are
much more likely to have clinical hookworm disease than are older animals.

The adult dog is the principal reservoir of infection, although infective
larvae can persist in warm moist environments for several weeks. Infection
occurs by ingestion or by direct cutaneous penetration of hookworm larvae.
In infected animals, larvae are continually leaving encystment in the muscles
and migrating to the intestines via the lungs. In the pregnant or lactating
bitch, this “larval leak” provides the two principal routes by which pups
become infected, namely, transplacental and transmammary infection. The
transmammary route is considered the most important source of infection
for neonates.

Clinical Indications. The rapid deterioration and death of pups in the
2nd and 3rd weeks of life or the persistence of anemia in older pups receiving adequate nutrition are clues to the existence of a hookworm problem. In severely contaminated kennels, dermatitis of the lower limbs may develop in response to migrating larvae.

**Clinical Signs.** The principal clinical signs of hookworm infection result from the anemia caused by these blood-sucking parasites. Pups nursing heavily infected bitches develop pale mucous membranes and pass soft to liquid feces during the 2nd or 3rd week after birth. Pups can die of exsanguination during the prepatent period before any hookworm ova are passed in the feces. Older pups may develop severe anemia after exposure to larvae from the bitch or kennel environment. Again, clinical signs may be apparent before hookworm ova are present in the feces. Older pups that are better able to compensate for the blood loss may develop a persistent, initially responsive anemia.⁹

**Treatment.** In young dogs and cats that are anemic and debilitated as a result of hookworm infestation, the parasite must be eliminated as quickly as possible while at the same time providing supportive care. Anthelmintics that require only a single dose (pyrantel pamoate or disophenol) are the most rapid and effective. Pyrantel pamoate is preferred over disophenol which, given subcutaneously, requires up to 24 hours for effective elimination of the hookworms.²⁴

Blood transfusion may be necessary to keep the young animal alive long enough for the anthelmintic to take effect. Supportive fluid therapy, a palatable nutritious diet, and attentive nursing care are also beneficial.

**Prevention.** Routine sanitation of cages, pens, and runs and periodic administration of anthelmintic medication to adult animals are recommended to reduce the level of environmental contamination with hookworm larvae. In situations in which neonatal losses from hookworms have occurred, it is essential to closely observe the young on a daily basis from the first few days of life through weaning. The first signs of anemia should prompt administration of an effective anthelmintic.⁹

Alternatively, anthelmintic medication (pyrantel pamoate) can be given at 2-week intervals, beginning at 2 weeks of age and continuing until after the pups are weaned. This protocol will be effective against roundworms as well (see the preceding discussion on roundworms). The treatment of pregnant bitches with a prolonged course of fenbendazole (as described for roundworms) could be used to reduce the incidence of transmammary infection.²⁴

**Coccidiosis and Cryptosporidiosis**

Coccidial oocysts are frequent incidental findings during examination of fecal smears or flotations. Coccidiosis in pet puppies and kittens is usually self-limiting and seldom requires treatment, although it can be a serious problem in large kennels. Crowding, malnutrition, unsanitary conditions, and other parasitoses are believed to contribute to the development of clinical coccidiosis.

Recently, there has been increasing interest in *Cryptosporidium*, an intestinal protozoan parasite that causes diarrhea in man and a variety of other animals.¹⁴ The prevalence of cryptosporidiosis in puppies and kittens
remains to be elucidated, but preliminary evidence suggests that it may be an important cause of diarrhea in young animals.

**Clinical Indications.** Coccidiosis should be considered in unthrifty young dogs and cats with diarrhea, tenesmus, and small amounts of fresh blood in the feces.

**Clinical Signs.** Young pets with coccidiosis are usually unthrifty, small for their age and breed, and have poor hair coats. It is not clear whether these signs are the direct result of coccidiosis or merely a reflection of management conditions that predispose the animals to clinical coccidiosis. Diarrhea or loose stools flecked with bright red blood associated with straining and increased frequency of defecation are classic signs of the large bowel disease associated with coccidiosis. Systemic signs are usually absent, although dehydration, depression, and anemia may be seen in severe cases.

Cryptosporidiosis is characterized by watery diarrhea, anorexia, and weight loss. Young animals, especially neonates, are more prone to infection and are more likely to develop clinical illness than are older animals. Although adults appear to develop immunity to *Cryptosporidium*, this is not passively transferred to their young.14

**Diagnosis.** Diagnosis depends on detecting coccidial oocysts in the feces, either by direct smear or fecal flotation. Small numbers of oocysts in feces from adult animals or otherwise healthy puppies are insignificant. Free merozoites in feces or in colonic washes indicate epithelial destruction as the result of protozoal multiplication and differentiate clinical from incidental coccidiosis.

The diagnosis of cryptosporidiosis is difficult. The oocysts are small (3 to 5 μm) and easily overlooked or confused with yeast. The organisms in feces can be identified with a simple iodine-stained wet mount, but various acid-fast stains help to verify the diagnosis; cryptosporidial oocysts stain red, whereas yeast stains blue-green.14 Sheather’s sugar flotation concentration technique can be used if the results of a wet mount or stained smears are negative.

**Treatment.** Incidental coccidiosis requires no treatment. Even clinical coccidiosis is usually self-limiting and will spontaneously resolve when other predisposing conditions, such as poor hygiene, inadequate nutrition, and concurrent parasitoses, are corrected. Supportive symptomatic therapy and improved management are usually adequate. Sulfadimethoxine (55 mg per kg t.i.d. for 7 days) or amprolium (23 ml per gal in the water source for up to 10 days) have been recommended in severe cases or endemic kennel problems.12 There is no specific therapy known to be effective for cryptosporidiosis.

**Prevention.** Appropriate management, involving good hygiene, adequate nutrition, and control of other infectious and parasitic diseases, appears to be the key to controlling coccidiosis. Prophylactic use of coccidiostats may be necessary in large kennels, especially in the southern United States.

The resistance of cryptosporidial oocysts to disinfectants makes control of this organism difficult. Care should be exercised in handling infected animals and their feces. Cryptosporidia are not host-specific, and there is potential for zoonotic infection.14
Giardiasis

Giardiasis may well be one of the most important and underdiagnosed enteric diseases of young dogs and cats. It frequently is endemic in kennels and catteries and can lead to intermittent bouts of refractory diarrhea. *Giardia* were originally thought to be host-specific (that is, that one species of this parasite could infect only one vertebrate host). There is now clear evidence that *Giardia* cysts collected from one species of animal can infect other species, including man. Thus, the diagnosis of giardiasis in pets carries important public health significance.

The life cycle of *Giardia* is direct (that is, no intermediate host is required). The protozoan exists in two forms—the fragile, motile trophozoite and the durable cyst. The usual habitat of the flagellated, binucleate trophozoite is the mucosal surface of the upper small intestine, where the organism attaches by an adhesive disk. Here the parasite lives and reproduces by binary fission. The presence of large numbers of trophozoites in the small intestine is associated with clinical signs, although not all infected animals show signs consistently.

Periodically, the trophozoites detach from the mucosa of the upper small bowel and enter the intestinal stream. They can reattach elsewhere in the small bowel or, as they flow along the lumen of the intestine, they can become spherical and elaborate a cyst wall. The resulting cysts are the form usually excreted in the feces. If the flow of ingesta through the intestine is very rapid, motile trophozoites may also be passed in the feces. Excreted trophozoites deteriorate rapidly and are unable to transform into cysts outside the host.

Freshly passed cysts are immediately infective and remain so for several weeks under favorable conditions (warmth and humidity). A new host acquires the infection by accidentally ingesting infective cysts. Excystation occurs in the small intestine, each cyst yielding two trophozoites. Once freed from the cysts, the trophozoites attach to the mucosa, completing the life cycle.

**Clinical Indications.** A diagnosis of giardiasis should be considered in animals of any age with prolonged histories of intermittent bouts of diarrhea or soft, mushy stools. Affected animals are often thin or unthrifty despite good appetites.

**Clinical Signs.** All of the signs of giardiasis are referable to the gastrointestinal tract. The most prominent sign is diarrhea. The stool is soft, light-colored, greasy, and mixed with mucus. The diarrhea may be intermittent, chronic, or both, persisting for weeks or months. Weight loss usually accompanies the diarrhea, even though most animals maintain a good appetite and adequate food intake. In young animals, growth retardation may result from malabsorption of nutrients. Occasionally, signs of colitis (straining, fresh blood) are also observed in animals suffering from giardiasis.

**Diagnosis.** The standard methods of fecal examination used in clinical practice desiccate and destroy giardial cysts and trophozoites, rendering them unrecognizable. Motile trophozoites can be visualized only by microscopic examination of a saline suspension of fresh feces, but recent studies indicated that only about 20 per cent of infections in dogs can be detected.
in this way. The same studies found that a modified zinc sulfate concentration technique detected nearly 70 per cent of infected dogs. It was recommended that the direct fecal smear be used as a screening test and that the zinc sulfate concentration technique be employed for further evaluation of stool samples from dogs suspected of having giardiasis. Response to therapy may provide support for a diagnosis of giardiasis when one cannot otherwise be definitively established.

**Treatment.** Although several medications have been recommended for the treatment of giardiasis in dogs, their efficacy has not been rigorously established. Recently completed studies indicate that quinacrine (3 mg per kg twice a day for 5 days) produced a 100 per cent cure rate in dogs in the trial, although side effects (anorexia, lethargy, and fever) were seen in almost half the animals treated. Also, quinacrine had no effect on the prevalence of trichomoniasis, another intestinal protozoan infection that was present in nearly 30 per cent of the dogs with giardiasis. Metronidazole is the medication most commonly recommended to treat giardiasis in dogs. At a dose of 22 mg per kg twice a day for 5 days, metronidazole had a cure rate of 67 per cent against giardiasis and 100 per cent against trichomoniasis without discernible side effects. In light of these data, the current recommendation is to use metronidazole as the first choice for treating dogs with giardiasis. Those animals (approximately 33 per cent) that fail to respond to metronidazole should then receive a course of quinacrine therapy.

In a kennel situation, therapy will not be effective unless the environment is treated simultaneously in order to break the cycle of continual contamination and reinfection. Preliminary data from our laboratory indicate that quaternary ammonium compounds, present in many commercial disinfectants, are effective in destroying giardia cysts and should be used to reduce the likelihood of reinfection.

**Prevention.** It is believed that wildlife in many parts of the United States (world?) are infected and represent a reservoir of infection. Since giardiasis results from ingestion of food or water contaminated with cysts, prevention entails not allowing pets access to surface water (streams, creeks, ponds) and otherwise preventing contact with feces from potentially infected animals.

In kennels and shelters, isolating infected or potentially infected animals until they can be effectively screened and/or treated will minimize the risk of the spread of infection.

**CONGENITAL AND MISCELLANEOUS ENTERIC DISORDERS**

**Megaesophagus, Vascular Ring Anomalies, and Esophageal Foreign Bodies**

Young dogs are often presented with the chief complaint of “vomiting,” which careful evaluation of the client’s description or direct observation proves to be regurgitation. Vomiting is an active process accompanied by retching and abdominal contraction. Regurgitation is passive, wherein undigested food appears to just “fall out” of the animal’s mouth. In the veterinary literature, regurgitation has become synonymous with esophageal
disease and has been defined as the relatively passive emptying of the dilated esophagus, regardless of cause.\textsuperscript{25}

Regurgitation can result from either a mechanical or functional obstruction of the esophagus. The most frequent cause in young dogs is idiopathic megaesophagus, a neuromuscular abnormality that precludes the normal propagation of waves of peristalsis along the body of the esophagus. This results in the ineffective passage of food, which pools in the esophagus, leading to megaesophagus and regurgitation. Vascular ring anomalies and esophageal foreign bodies can also cause regurgitation in young dogs.

**Clinical Indications.** Regurgitation, whether intermittent, persistent, or progressive, suggests esophageal disease.

**Clinical Signs.** The hallmark of esophageal disease is regurgitation, differentiated from vomiting by its relatively passive nature. Owners of affected animals usually comment about how easily the ingesta "rolls" out of the mouth when a combination of gravity and head position allows the cranial esophagus to empty. Besides its passive nature, regurgitation differs from vomiting in that the regurgitated food is undigested and lacks a sour odor. Pups with esophageal problems are usually thin, but are otherwise healthy, bright, and alert. They are often hungry and may begin eating the regurgitated material immediately. The interval between eating and regurgitation may be quite variable, from a few minutes to several hours.

Untreated megaesophagus and vascular ring anomalies can lead to severe stunting and emaciation. Aspiration pneumonia, manifested as pyrexia, coughing, rales, or silent lung fields, is a frequent complication. The dilatation of the cervical esophagus may be detectable on physical examination.

The clinical signs of esophageal foreign bodies are quite variable, depending on the size, shape, and location of the object. Complete obstructions usually present as acute problems, whereas partial obstructions may have clinical histories spanning several days or even weeks. The pain produced by attempts to swallow causes anorexia. Esophageal colic—spastic contraction of the esophagus at the level of the foreign body—can produce restlessness and episodic cries of pain. Complete obstruction blocks the normal swallowing of saliva, resulting in drooling and hypersalivation.

Pressure necrosis can also result. If the inflammatory reaction extends through the esophageal wall, mediastinitis or pleuritis results, producing lethargy, dyspnea, abdominal breathing, and reluctance to move about. Pain will be evident on compression of the rib cage.

**Diagnosis.** Definitive diagnosis can seldom be established solely on the basis of history and physical examination because the clinical signs of all three diseases overlap to a considerable degree. Plain radiographs of the thorax, however, often provide a sound basis for a definitive diagnosis.\textsuperscript{25} The vast majority of esophageal foreign bodies are bones and, thus, appear as radiopaque objects in the thorax. Idiopathic megaesophagus is characterized by a large, flaccid, dilated esophagus filled with ingesta, fluid, and air. In megaesophagus resulting from a neuromuscular disorder, the dilatation is evident throughout the length of the thoracic esophagus; in vascular ring anomalies, the dilatation is restricted to the esophagus cranial to the heart. Plain radiographs can also reveal the presence and degree of complications such as aspiration pneumonia, mediastinitis, and pleuritis.
Further evaluation by contrast radiography is warranted in some patients to better define the degree of distension and length of the esophagus affected or to detect concurrent vascular ring anomalies in patients with generalized megaesophagus. In patients with esophageal foreign bodies, contrast studies will help delineate the size, shape, and location of the object. Special care must be taken when performing oral contrast studies on patients with esophageal dysfunction, as there is an increased risk of aspiration of the contrast medium. Iodine-based contrast medium should be substituted for barium if there is any possibility of esophageal laceration or necrosis secondary to a foreign body. Leakage of barium-containing contrast medium through a rent in the esophageal wall could induce severe, granulomatous mediastinitis and pleuritis.

**Treatment.** Dietary and medical management have generally been more satisfactory than surgery for young dogs with idiopathic megaesophagus. The puppies should be trained to eat from an elevated platform and remain upright for 10 to 20 minutes after eating. In this position, gravity can assist peristalsis in emptying the esophagus and delivering food to the stomach.

Soft foods or slurries are usually easier to swallow, but affected dogs differ greatly in their ability to manage foods of different consistencies. Owners should be encouraged to experiment with different food types (dry bulky foods, canned food, gruels, or slurries) to find which produces the least problems for their particular pet.

Medical management of pups with idiopathic megaesophagus is directed principally toward preventing and controlling aspiration pneumonia. Surgical correction (a modified Heller’s myotomy) should be considered only if more conservative therapy fails.

In contrast to idiopathic megaesophagus, vascular ring anomalies can only be successfully treated surgically. Prognosis depends on the degree of esophageal dilatation oral to the vascular ring, and careful evaluation is necessary before predicting the likelihood of success. If the cranial thoracic esophagus is severely dilated, it may never regain normal function, even after the mechanical obstruction is surgically corrected. Regurgitation may continue, and affected dogs may require management similar to animals with idiopathic megaesophagus.

Removal of an esophageal foreign body, either surgically or by means of an endoscope, is only the first phase of the treatment for this problem. Equally important is the medical management of the accompanying esophagitis. Failure to treat the foreign body-induced esophagitis effectively can lead to slowly progressive esophageal scarring and stricture.

The medical management of esophagitis consists of dietary alteration, antibiotic therapy, and an H2-receptor blocker (cimetidine or ranitidine). Food should be withheld for 1 to 5 days to minimize the possibility of further physical trauma to the esophagus, with the animal maintained on intravenous or subcutaneous fluids during this period. Thereafter, the diet should consist of slurried, protein-enriched meals given frequently in small amounts for 1 to 3 weeks. Broad-spectrum antibiotic therapy (ampicillin) should be administered to eliminate secondary bacterial contamination of the ulcerated esophageal mucosa by the constant stream of oral flora in swallowed saliva.
H₂-antagonists are used to reduce gastric acidity and protect the damaged mucosa against further insult that could result from esophageal reflux.

**Prevention.** Animals with vascular rings or idiopathic megaesophagus should not be used for breeding. Evidence of a genetic factor in the development of megaesophagus has been reported in at least two breeds of dogs, and certain lines appear to have a higher incidence than others. It has been suggested that vascular ring anomalies also have a genetic basis, although definitive evidence is lacking.

Owners should be warned about the various hazards associated with giving their dogs bones, especially relatively small bones with sharp projections. Pork chop bones are especially likely to cause esophageal foreign bodies.

**Pyloric Outflow Dysfunction**

Persistent vomiting by a young dog or cat can have many causes. The two most frequently encountered are gastric foreign bodies and pyloric stenosis or dysfunction. The inherent curiosity of young pets and their propensity to prehend and swallow objects make them frequent victims of pyloric outflow obstruction due to foreign objects in the stomach. Gross muscular hypertrophy of the pylorus, resulting in pyloric stenosis, occurs in young pups, probably as a primary congenital abnormality.¹⁵

Likewise, a syndrome of pyloric and esophageal dysfunction has been described in cats, particularly Siamese. The clinical signs and radiographic appearance are similar to pyloric stenosis in the dog, but no muscular hypertrophy could be detected at the time of surgery. This syndrome apparently represents a functional rather than a mechanical abnormality, probably a result of autonomic nervous system dysfunction.¹⁶

**Clinical Indications.** Any young puppy or kitten with a history of persistent vomiting should be evaluated for the presence of gastric foreign bodies and pyloric obstruction or dysfunction.

**Clinical Signs.** The signs of congenital pyloric stenosis or dysfunction develop soon after weaning. The predominant sign is vomiting, which may vary in frequency from several times a day to only once or twice a week. In individual animals, the interval between eating and vomition is often fairly constant, but varies from 30 minutes to as long as 8 hours. The vomitus may be easily recognized as part of a previous meal or even food eaten as long as 2 or 3 days before. In contrast to the regurgitation associated with esophageal dysfunctions, the vomiting is active, even projectile, and the vomitus is seldom reingested, although affected animals may have ravenous appetites immediately after vomiting. Abdominal distension due to gastric dilatation, loud borborygmi, and abdominal discomfort or pain are other common clinical signs. These are often relieved by vomiting. Young animals become unthrifty, thin, and stunted as a result of inadequate protein and caloric intake.

Gastric foreign bodies may cause similar signs. A history of chewing on or swallowing foreign objects is sometimes obtained, which may be helpful in differentiating foreign bodies from congenital dysfunction. If the foreign body passes through the pylorus and obstructs the small bowel, the severity of signs may suddenly worsen and the animal's condition deteriorates rapidly.
Diagnosis. Radiographic evaluation is usually necessary to establish a definitive diagnosis. If a radiopaque foreign body is present, plain films may be sufficient, but contrast studies are often necessary to detect rubber balls, socks, plastic wrap, and other objects that puppies and kittens may ingest. However, care must be taken in the interpretation of contrast studies. The presence of a foreign body may be obscured on the early films of a barium series. Repeated radiographs should be taken until the barium empties sufficiently to evaluate the entire lumen.

Plain radiographs of the young animal with inherent pyloric obstruction often reveal a stomach still dilated with food and fluid long after the ingesta should have moved into the small bowel. Concretions of mineralized debris and food may be present if the delayed emptying has persisted long enough. Studies using liquid contrast medium sometimes reveal delayed passage of the medium out of the stomach, but in many patients with pyloric dysfunction, the passage of liquid may be normal or even enhanced. A more physiologic assessment of gastric function can be made using barium sulfate powder admixed with a palatable canned food. Such a meal should normally clear the stomach in 8 hours or less. In young cats with pyloric dysfunction, esophageal motility should also be evaluated. The esophagus will frequently retain contrast medium in such cases, comparable with the radiographic appearance of megaesophagus in the dog.

Treatment. Gastric foreign bodies should be removed either surgically or, if relatively small, via the esophagus by means of an endoscope. Patients suffering from pyloric dysfunction or stenosis generally respond well to pyloroplasty. In the authors’ experience, the beneficial effects of pyloroplasty were of longer duration than those of pyloromyotomy. Animals treated with the latter procedure often suffer a recurrence of delayed gastric emptying within a year.

Prevention. Ideally, young animals should have limited access to potential gastric foreign bodies, but this is difficult, if not impossible. Some animals will repeatedly ingest specific objects (for example, stones). Limiting access to these objects, at least temporarily, may help break the habit. Animals diagnosed as having pyloric outflow abnormalities should not be used for breeding. The increased prevalence of these conditions in some breeds and lines suggests a genetic predisposition.

SYMPTOMATIC THERAPY FOR ACUTE ENTERITIS

Goals of Therapy

The objectives of symptomatic therapy for acute enteritis are to (1) rest the gut, (2) restore and maintain fluid and electrolyte balance, and (3) minimize continuing losses.

Triage

The first and perhaps most important treatment decision is whether the case can be handled on an outpatient basis or whether hospitalization and more aggressive therapy are needed. In general, patients with acute enteritis can be treated as outpatients unless they have clinical findings, such as severe
dehydration or anemia, seizures, high fever, marked depression, and so on, that suggest a life-threatening condition. Patients with such findings and patients that have failed to respond to conservative treatment should be hospitalized, and an intensive therapeutic and diagnostic program should be begun. Young puppies and kittens are more likely to require hospitalization than adults because of the rapidity with which they can become dehydrated and moribund.

**Outpatient Therapy**

Withholding food for 12 to 24 hours is one of the most important aspects of symptomatic therapy. Water should also be withheld if vomiting is severe; otherwise, animals may begin a vicious cycle of drinking and emesis that only exacerbates dehydration. Subcutaneous or intravenous fluids (see below) should be used to prevent dehydration. Severely ill kittens and puppies should definitely not be given milk, especially if they have become chilled; it will not be digested and can cause fatal bloat.

Food should be gradually reintroduced after 12 to 24 hours as a warm bland gruel. Commercially available bland diets (for example, Prescription Diet i/d*) are convenient, but cottage cheese and boiled rice are a satisfactory homemade substitute. If feeding causes a recurrence of vomiting or diarrhea, the fast should be continued for an additional 12 to 24 hours. After food can be tolerated, frequent small meals of the bland diet should be continued for 2 to 3 days, gradually introducing the regular diet.

Fluid therapy is an important aspect of treatment, especially in young animals. It should be initiated promptly and continued for as long as the vomiting or diarrhea persists. Oral electrolyte solutions are the simplest form of therapy, providing that fluids can be tolerated by mouth. Commercial preparations, such as those for calves (for example, Resorb†) or people (Gatorade‡), are excellent, but a homemade formula of 3.5 gm table salt, 3.5 gm sodium bicarbonate, 1.5 gm potassium chloride, and 20 gm glucose per liter of water can also be used. Maintenance requirements are about 70 ml per kg per day, but more will be necessary if there is severe diarrhea.

Subcutaneous fluids can be used on an outpatient basis for animals that are mildly dehydrated but cannot tolerate oral electrolyte therapy. Severely dehydrated animals should be hospitalized and given intravenous fluid therapy (see below).

Recent research in both human and veterinary medicine has called into question the value of many of the medications traditionally used for vomiting and diarrhea. For example, no benefit could be demonstrated for kaolin-pectin preparations in controlled clinical trials. Similarly, the routine use of motility modifiers, such as anticholinergics, and opiate agonists, is no longer recommended. Anticholinergics, in particular, may be detrimental because they decrease segmentation to a greater extent than peristalsis, thereby reducing rather than increasing resistance to the flow of ingesta.

Antiemetics may be useful in patients with prolonged or severe vomiting. Metoclopramide, a new antidopaminergic antiemetic, has proven es-

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*Hill's Pet Products, Topeka, Kansas.
†Beecham Laboratories, Bristol, Tennessee.
‡Stokley-Van Camp, Chicago, Illinois.
pecially useful in patients with viral enteritis. A dose of 0.2 to 0.4 mg per kg can be given subcutaneously every 8 hours, or in very severe cases, the drug can be given as a continuous intravenous drip at the rate of 1 to 2 mg per kg every 24 hours.

**In-Hospital Therapy**

Fasting is also important for hospitalized patients and should follow the general guidelines given above. Oral medications should be avoided; vomiting, delayed gastric emptying, and altered mucosal integrity make absorption unpredictable and unreliable.

Intravenous fluid therapy should be initiated promptly for severely ill or dehydrated patients. Ideally, acid-base and electrolyte imbalances should be monitored and corrected, but even if this is not possible, simple volume expansion with a balanced electrolyte solution is often life-saving.

Routine use of antibiotics for simple enteritis is unwarranted. Oral antibiotics may alter the intestinal flora in favor of enteric pathogens. They may also prolong shedding of enteropathogens like *Salmonella* and favor the development of resistant strains. Parenteral antibiotics, on the other hand, probably have a place in the management of severe parvoviral enteritis and feline panleukopenia. Animals with these diseases appear to be prone to secondary septicemia and generalized bacterial infection. A combination of ampicillin and gentamycin has been recommended because of the broad spectrum of activity against both aerobes and anaerobes. Care must be taken to ensure adequate hydration if any of the aminoglycosides are used because they can cause substantial nephrotoxicity. The urine sediment should be monitored frequently to detect tubular casts that herald the onset of tubular nephrosis.

**SUMMARY**

Digestive tract disorders are common diagnostic and therapeutic problems among young dogs and cats. Prompt and effective symptomatic therapy is necessary in all cases, and is sufficient in many. Parasitic and protozoal problems require attention to kennel management as well as to individual treatment. Chronic and congenital disorders are often extremely challenging diagnostic dilemmas.

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