The Scoop on Poop: Diagnostic and Therapeutic Approach to the Diarrheic Dog and Cat

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Diarrhea is generally regarded as the most consistent clinical sign of intestinal disease in the dog and cat. The history and physical examination are paramount for determining whether the diarrhea is due to primary disease of the gastrointestinal tract or secondary to extraintestinal diseases such as chronic renal failure or hyperthyroidism.

HISTORY

The history can indicate the location, severity, and probable cause of the disease process. An important goal of the history is to categorize the diarrhea into “small bowel” or “large bowel” in origin, because this will have a direct impact on the diagnostic and therapeutic approach to the patient. A careful history should also indicate the presence of extraintestinal disease as the underlying cause of diarrhea or may identify important predisposing factors, such as diet, environmental factors, exposure to parasites, infectious agents, drugs or toxins. The history should focus on the duration of the diarrhea, the patient’s diet (and diet changes), the appearance of the faeces (color, volume, mucus, presence of fresh blood), worming and vaccination history, defecation frequency, aggravating or alleviating factors, and defecation urgency. Adverse reactions to food are a relatively common cause of chronic diarrhea in dogs and cats, and the history is a simple way to determine incriminating diets or protein sources.

PHYSICAL EXAMINATION

Physical examination should emphasize the detection of fever, cachexia, dehydration, weakness or depression, pallor (blood loss anemia) and effusions or edema (hypalbuminemia). Intestinal loops should be carefully palpated for masses, thickening (inflammatory or neoplastic infiltration), distension, pain, or associated lymphadenopathy. Palpation of the rectum may reveal rectal masses or thickening of the rectal wall.

DIAGNOSTIC APPROACH

For undiagnosed chronic diarrhea, the minimum database should include a complete blood count (CBC), a serum biochemistry profile, a urinalysis, a fecal floatation for parasitic ova, and a direct smear of saline admixed fresh feces for protozoa. Fecal flotations should always be done on fresh fecal specimens (< 1 hour ideally), or on fresh fecal specimens that have been refrigerated for < 48 hrs. Flotations are excellent for recovering common nematode ova, oocysts of coccidia, and Giardia cysts. The main limitation of flotations is their inability to float organisms whose diagnostic stage has a specific gravity higher than that of the flotation medium. Most commonly these are the heavy ova of trematodes and acanthocephalans (thorny-headed worms). Fragile cysts and larvae, though recovered, may be too distorted to identify. It is always preferable to use a centrifugation flotation technique. This is probably the single most important change one can institute to improve routine recovery of parasite stages by flotation. Flotation methods that do not utilize a centrifugation technique are often not sensitive enough to recover small numbers
of organisms in the feces. Always use a coverslip rather than a loop or glass rod to transfer the meniscus to a slide. If your centrifuge has free-swinging buckets, use a coverslip on the final centrifugation to recover parasites. If your centrifuge has a fixed-angle, carefully add more flotation medium after the final spin to create a meniscus and set the coverslip on top for several minutes before lifting it off for examination. If at this point you are still determined to do standing flotations, use a coverslip to transfer the meniscus. Coverglass improves the optics of any microscope. Check the specific gravity of flotation medium. If possible, switch to zinc sulphate (sg 1.18 or 1.2) in order to improve detection and morphology of *Giardia* spp. Remember that regardless of the flotation solution all eggs do not float. Choice of flotation solution is less important than the actual method of flotation. Examine preparations as soon as possible after preparing them. Delay will result in distortion of some parasite stages and incorrect or missed diagnoses, especially with delicate cysts and oocysts.

Encysted forms of *Giardia* organisms in a fecal specimen may be detected most reliably with zinc sulfate floatation, as opposed to examination of fresh saline smears. Preliminary studies in *Giardia*-infected cats at the University of California, Davis, have revealed that the ProSpecT® Microplate ELISA Assay compares favorably in sensitivity and specificity to the zinc sulfate floatation technique; however, the sensitivity of the ProSpecT® *Giardia* Rapid ELISA Assay is significantly less than that of the Microplate Assay and zinc sulfate floatation methods. The importance of evaluating 2 to 3 consecutively obtained fecal specimens cannot be overemphasized as our studies in dogs and cats have confirmed intermittent shedding of oocysts, and significant increases in sensitivity in all diagnostic tests when more than one stool specimen is examined.

Direct wet mounts (direct smears) should be performed on all diarrheic specimens. The study must be done on fresh feces (< 1 hour) to prevent desiccation of motile trophozoites, and to preserve motility of the organism for detection. Morphological differentiation of *Giardia* trophozoites from *Tritrichomonas foetus* trophozoites can be difficult in unstained smears. *Giardia* trophozoites typically have a “falling leaf” motion, whereas *T. foetus* trophozoites have an irregular hap-hazard motion. Diagnosis of *T. foetus* can also be facilitated by implementation of a commercially available culture system, which is specific for growth of *T. foetus*. *Tritrichomonas foetus* has been associated with a “large-bowel” type diarrhea in dogs and cats, which has been shown to be refractory to antibiotic therapy.

Macroscopic examination of a fresh fecal specimen is essential for assessment of bulk, color, consistency, and detection of blood and mucus. Small bowel diarrhea is generally free of grossly visible mucus or red blood, but prominent steatorrhoea may cause the faeces to appear lighter in color. Rapid intestinal transit time can be associated with yellow or green stools due to incomplete metabolization of bilirubin.

The complete blood count may reveal an eosinophilia secondary to endoparasitism, eosinophilic enteritis, or abdominal mast cell neoplasia. Anemia may result from enteric blood loss or from depressed erythropoiesis caused by systemic disease or chronic inflammation. Underlying systemic diseases such as chronic renal failure or liver disease may be detected on the serum biochemical profile. In addition, panhypoproteinemia (low serum albumin and globulin) may be seen with severe infiltrative bowel diseases such as inflammatory bowel disease, intestinal
lymphoma, and Histoplasmosis. The finding of steatorrhoea and weight loss in the face of a normal to increased appetite is consistent with a malassimilation disorder such as exocrine pancreatic insufficiency. This disorder is best confirmed by performing a serum trypsin-like immunoreactivity assay. Animals exhibiting signs of large bowel diarrhea should have a rectal scraping and stained fecal smear performed to evaluate for inflammatory cells or fungal hyphae.

Survey abdominal radiographs are a relatively low yield procedure in most patients with chronic diarrhea, but are indicated in dogs suspected of having partial obstructions due to foreign bodies, intussusceptions, or masses. Abdominal ultrasound is complimentary to survey abdominal radiographs and is more sensitive for the detection of abdominal masses, intestinal mural thickening, intussusceptions, and mesenteric lymphadenopathy. In addition, ultrasound-guided percutaneous biopsy or aspiration of masses is an effective diagnostic procedure. Contrast radiography and fluoroscopy are occasionally indicated for identification of partial obstructions and intestinal motility disorders, respectively.

The clinical documentation of intestinal bacteria causing diarrhea in dogs is clouded by the occasional presence of these organisms existing as normal constituents of the indigenous intestinal flora. Fecal isolation of Clostridium perfringens and C. difficile is therefore a low-yield procedure in animals with diarrhea, and commercially available toxin assays are recommended. Fecal cultures are best performed to isolate Campylobacter spp. and Salmonella spp. Fecal enteric panels should be reserved for dogs and cats developing diarrhea after kenneling or show attendance, in animals with an acute onset of bloody diarrhea in association with evidence of sepsis, or in diarrhea outbreaks occurring in more than one pet in a household. Caution should be exercised when interpreting the presence of C. perfringens endospores in fecal smears obtained from animals with diarrhea, because healthy dogs can harbor large numbers of C. perfringens endospores in their stools. In addition, the currently accepted opinion that ≥2 to 3 endospores/oil immersion field is associated with enterotoxin appears unsubstantiated in light of recent studies documenting a poor correlation between fecal endospore numbers and the presence of enterotoxin.

Dogs and cats with no evidence of life-threatening disease, whose diarrhea remains undiagnosed after initial laboratory and imaging procedures, are further evaluated with elimination diets for 2 to 3 weeks. The diet selected should be free of additives and preservatives, and contains a novel protein source that is highly digestible. The protein should be highly digestible because intact proteins are far more antigenic than polypeptides and amino acids. If signs resolve following several weeks of dietary therapy, the diet should be balanced or alternatively a complete and balanced commercial diet with the same novel protein source as the home-made diet should be selected. Hydrolyzed protein diets are commercially available and appear beneficial for some patient's refractory to controlled diets containing intact protein sources. If signs do not resolve, a more extensive diagnostic approach is warranted.

Endoscopy is a valuable procedure for the diagnosis of intestinal mucosal diseases that are associated with morphologic changes. Endoscopy, however, does not differentiate intestinal motility disorders, secretory diarrheas, or brush border enzyme defects, and is likely to miss lesions in the intestinal submucosa and muscularis. In addition, endoscopy is limited by the working length of the scope, limiting endoscopic examination of the jejunum. Rigid proctoscopy
is preferred over flexible colonoscopy for the initial evaluation of large bowel disease. Rigid proctoscopy entails less risks, time, and cost than colonoscopy, and is able to diagnose the majority of large bowel disorders because of the diffuse nature of the disease. Flexible colonoscopy is indicated for evaluation of upper colonic disease, including cecal inversion, ileocolic neoplasia, and occult *Trichuris* infection.

Tests for malabsorption do not give a specific causal diagnosis for the diarrhea, and many of these tests are limited by relatively poor sensitivity and specificity. In contrast to the endoscopic procedure that typically diagnoses abnormal mucosal morphology, malabsorption tests evaluate intestinal function. They identify abnormal carbohydrate or fat assimilation secondary to motility disorders, bacterial overgrowth, or brush border enzyme deficiencies. Screening tests of malassimilation include the indirect quantitative analysis of fecal fat; the breath hydrogen analysis, and the direct/indirect Sudan stain for fecal fat. The use of the fat absorption test and Sudan stain for fecal fat are not recommended due to the insensitivity of the tests and frequency of misleading results.

**PRINCIPLES OF DIARRHEA THERAPY**

Symptomatic therapy includes restoration and maintenance of fluid and electrolyte balance, dietary modification, and empiric deworming. Antibiotics and immunosuppressive drugs are introduced based on evidence of inflammatory bowel disease or bacterial enteritis. Motility modifiers are only indicated if the diarrhea is intractable and should not be used if the diarrhea is due to invasive microorganisms. The opiate and opioid narcotic analgesics such as loperamide (Imodium; 0.1-0.2 mg/kg TID, PO) are the most effective motility modifiers for managing diarrhea. Anticholinergic agents are contraindicated because they may cause generalized suppression of all motility and may potentiate ileus. Most dogs with moderate to severe **inflammatory bowel disease (IBD)** will require adjuvant pharmacologic therapy in combination with dietary management. It is important to understand that the therapy of IBD must be tailored according to each patient’s response.

**Antibiotics**

Use of antibiotics as empirical therapy in the management of uncomplicated or noninfectious diarrhea is not recommended because of the adverse effects of the antibiotics on the normal intestinal microflora and their tendency to promote resistant strains of bacteria. Antibiotics are indicated when specific bacterial or protozoan enteropathogens, such as *Campylobacter*, *Clostridium*, or *Giardia* are isolated from the feces (Table 1). In addition, antibiotics should be considered in conditions associated with severe mucosal damage and a high risk of secondary sepsis or endotoxemia. Metronidazole (Flagyl) has several beneficial properties, including broad spectrum activity against anaerobic bacteria, antiprotozoal activity, and potent inhibition of cell-mediated immunity. Metronidazole may be used as a single agent in mild cases of IBD; however, it should be combined with prednisone to manage moderate to severe cases or to allow a reduction in prednisone dosage. The dose of metronidazole is 10 to 15 mg/kg q 8 to 12 hours. Metronidazole is supplied as 250 and 500 mg tablets or as a 5 mg/ml oral suspension. Metronidazole tablets have a sharp, unpleasant, metallic taste when scored that can cause severe salivation. Scored tablets can be placed in empty gelatin capsules or compounded into a
palatable suspension to minimize this problem from occurring. Side-effects are rare, although metronidazole has been associated with a peripheral neuropathy in both humans and dogs, and less commonly in cats. Less common side effects include inappetence, nausea, vomiting, ataxia, seizures, and reversible neutropenia. **Tylosin** (Tylan) is a macrolide antibiotic that has been reported to be effective in managing canine IBD. Although the drug’s mechanism of action is unknown, it appears to be effective in some dogs refractory to other forms of therapy. Tylosin is supplied in a highly concentrated powdered form (approx. 3.5 g per teaspoon) marketed for poultry. The dose range is 15 to 20 mg/kg q 12 hours. Tylosin appears safe and no side effects have been reported.

**Immunosuppressive Drugs**

**Corticosteroids**

The main indication for corticosteroid administration appears to be in dogs and cats with lymphoplasmacytic or eosinophilic gastroenteritis, and in dogs with colitis that is only partially responsive to sulfasalazine or metronidazole.12,13 Prednisone is the corticosteroid most frequently used for the therapy of canine and feline IBD. The dosage and duration of therapy is based on the severity and duration of clinical signs, the severity and type of inflammation, the clinical response, and tolerance to the drug. The initial dosage of prednisone for therapy of IBD in dogs is 1 to 2 mg/kg q 12 hours. The drug is gradually tapered over a 4- to 10-week period once clinical remission is attained. Some dogs will require higher dosages to maintain clinical remission; however, the deleterious side effects of prednisone in dogs (polyuria, polydipsia, polyphagia, weakness, and other clinical signs of iatrogenic hyperadrenocorticism) often preclude its prolonged administration. Combination therapy with a controlled diet, azathioprine, or metronidazole is undertaken with the goal of reducing the dose of prednisone.

Poorly absorbed oral steroids have been developed for human patients to reduce the systemic side effects of oral glucocorticoids. Budesonide, an orally administered corticosteroid structurally related to 16-hydroxy prednisolone, has high topical anti-inflammatory activity and low systemic activity because of its high affinity to the steroid receptor and rapid hepatic conversion to metabolites with minimal or no steroid activity. Controlled prospective clinical trials comparing the efficacy of prednisone versus budesonide in dogs with IBD are warranted before the routine use of this drug can be recommended. Parenteral corticosteroid therapy is reserved for vomiting patients, or animals with severe nonresponsive disease. Depot preparations of glucocorticoid should be avoided because of their rapid and severe depression of the hypothalamic-pituitary axis and their inconsistent control of clinical signs in animals with moderate to severe IBD. In glucocorticoid resistant cases, the addition of metronidazole or chlorambucil (Leukeran) may result in remission of disease.

**Azathioprine**

Azathioprine is an antimetabolite that is converted to 6-mercaptopurine in the liver and then to thioinosinic acid. The latter compound impairs purine biosynthesis and this biochemical reaction inhibits cellular proliferation and reduces natural killer cell cytotoxicity. The onset of these immunological effects is slow, paralleling the human clinical data which shows benefits for these drugs when given for 3 to 6 months or longer. Despite the lack of clinical trials showing efficacy of azathioprine in the treatment of canine and feline IBD, clinical and anecdotal experience
suggests that the drug is most useful in dogs as adjunctive therapy in severe or refractory IBD. Azathioprine can also be used for its steroid-sparing effects when the adverse effects of prednisone are unacceptably high. The dose for dogs is 50 mg/m² or 1-2 mg/kg once daily for 2 weeks, followed by alternate-day administration. The most significant side effect of azathioprine is bone marrow suppression, particularly in cats. Frequent hematologic monitoring is warranted with temporary discontinuation of the drug if thrombocytopenia or neutropenia develop. Other side effects include anorexia, pancreatitis, and hepatic dysfunction.

**Sulfasalazine**

The drug consists of sulfapyridine linked to mesalamine (previously called 5-aminosalicylic acid) by an azo bond and is poorly absorbed in the upper gastrointestinal tract. In humans, approximately 75% of sulfasalazine passes unabsorbed to the colon, where bacterial azoreductases cleave the azo bond and release the active moiety of the drug, mesalamine. Sulfapyridine is almost completely absorbed in the colon, metabolized in the liver, and excreted in the urine. The mesalamine moiety is locally absorbed and acetylated in the cytosol of the colonic epithelial cell and inhibits the formation and degradation of inflammatory mediators, including leukotrienes, prostaglandins, thromboxane, platelet activating factor, histamine, and a number of cytokines, including interleukin 1-alpha and interferon-gamma. Sulfasalazine is of no value in managing small bowel inflammation because colonic bacterial metabolism is needed to release the active moiety. Sulfasalazine is supplied as 500-mg tablets or as a 50-mg/ml liquid formulation. The usual initial dose in dogs is 20 to 40 mg/kg q 8 hours for 3 weeks, followed by 20 to 40 mg/kg q 12 hours for 3 weeks, and 10 to 20 mg/kg q 12 hours for 3 weeks. The most common side-effects of sulfasalazine include anorexia, vomiting, cholestatic jaundice, allergic dermatitis, and keratoconjunctivitis sicca (KCS). These side effects have been attributed to the sulfapyridine moiety; however, studies in beagles have documented the association of KCS with mesalamine administration.

**Dietary therapy**

**Elimination Diets**

Several studies in the veterinary literature suggest that some patients may benefit from diets providing novel, highly digestible protein sources. Selecting a protein source not commonly found in the animal’s diet is recommended because it reduces the likelihood of feeding a protein to which the animal is allergic. The ideal elimination diet for dogs with chronic small bowel diarrhea is based on a highly digestible single protein and carbohydrate source that is gluten and lactose free. There are a number of commercially available elimination diets that can be utilized. The use of computer-generated home-made diets formulated by a qualified veterinary nutritionist is typically reserved for cats failing to respond to the commercial diets. It is important that the ingredients list of a potentially hypoallergenic diet be thoroughly evaluated, because diets with several protein sources (lamb, beef, rice, and wheat) are commonly marketed with a claim to hypoallergenicity.

Dietary recommendations for the management of large bowel diarrhea are controversial. The response to dietary therapy can vary dramatically from one patient to another, with some animals showing improvement on low residue, “hypoallergenic” diets, and others improving on less digestible diets containing soluble or insoluble fiber sources.¹⁵,¹⁶ There is evidence to suggest
that some forms of colitis may be associated with a dietary sensitivity similar to that observed with small bowel disease. The theoretical benefit for utilizing highly digestible “hypoallergenic” diets for patients with colitis includes reducing the digestive challenge to the large intestine and minimizing the likelihood of dietary antigens actually reaching the colon, thus lessening the likelihood of an immunological reaction. The author utilizes the same elimination diets for large intestinal disease as those recommended for management of small-bowel type disease. The supplementation of fermentable fiber sources such as psyllium or oat bran may be necessary in cats with large-bowel type disease showing partial resolution of their clinical signs. Failure to respond to these recommendations may necessitate selecting a “hypoallergenic” diet with a different novel protein source or adding insoluble fiber to the diet.

**Dietary Fiber**

There is increasing evidence that fermentable fiber sources can also be beneficial because of their pre-biotic effect in reducing or preventing inflammation in experimental models of IBD. Therefore, a fermentable fiber source should probably be included as part of dietary therapy, although information regarding which (e.g. resistant starch, fructosoligosaccharides, inulin) and how much is lacking. Fructooligosaccharides (FOS) are carbohydrates that resist digestion by the enzymes in the gastrointestinal tract and can be metabolized by the microbial species that colonize the distal small intestine and colon. The addition of FOS to feline diets at 0.75% (DM) did not affect duodenal flora, but it did increase the numbers of lactobacilli and reduce the numbers of *E. coli* in the fecal flora of healthy cats. 

Recently, treatment of chronic idiopathic large bowel diarrhea with a highly digestible diet and soluble fiber was reviewed in a retrospective study of 37 dogs. Treatment with a soluble fiber source (Metamucil), added to a highly digestible diet, resulted in a very good to excellent response in 23 of the 27 dogs that received supplementation. Dogs classified as having a very good or excellent response to soluble fiber supplementation received no other additional therapy except for occasional loperamide or diphenoxylate. Fiber supplementation was later reduced or eliminated in 11 dogs; diarrhea returned in 6 of them.

**Polyunsaturated fatty acids**

Fish oil has been reported to be beneficial in ulcerative colitis and Crohn’s disease patients, but the results are controversial. Only two of these studies found significant decreases in rectal leukotriene B4 (LTB₄) concentrations, the others simply reported clinical improvement. The differences between the reports regarding study design, supplement composition, dose and assessment of clinical improvement may in part explain the conflicting results. A recent study compared the efficacy of n-3 fatty acids in fish oil to sulfasalazine in the treatment of mild to moderate active ulcerative colitis in humans. Treatment with n-3 fatty acids resulted in greater disease activity as detected by a significant increase in platelet count, erythrocyte sedimentation rate, C-reactive protein, and total fecal nitrogen excretion. An often overlooked concern is the increase in lipid peroxidation after fish oil supplementation is instituted. Antioxidant supplementation may be able to counteract the potentially adverse effects of n-3 fatty acids. Most of the literature regarding n-3 fatty acid administration fails to address the amount of attendant antioxidant supplementation. There are no reports in the veterinary literature to date demonstrating the efficacy of n-3 fatty acid supplementation in managing canine or feline patients with IBD. Studies in healthy dogs fed diets with n-6 to n-3 ratios of 5:1 and 10:1 demonstrated a decreased production of LTB₄ in plasma, neutrophils and skin. Increases in
certain long chain n-3 fatty acids and decreases in arachidonic acid were identified in the small intestine and colonic mucosa of healthy Beagles fed the same ratios. Further research is necessary to determine the clinical benefits in dogs and cats with large bowel diseases.

**Vitamin B12**

Low serum B12 or cobalamin has often been regarded solely in the context of its diagnostic utility in identifying dogs with small intestinal bacterial overgrowth. However, low serum B12 has been described in dogs and cats in association with a wide variety of gastrointestinal diseases including IBD and intestinal lymphoma.\(^{20}\) It is likely that mucosal repair is impeded in the initial management of IBD when B12 is deficient and its absorption impaired, however this has not been investigated. Consideration should be given to B12 assays in the initial evaluation of cats with chronic intestinal disease, and parenteral administration during the initial management of IBD if low serum cobalamin is identified. Dogs are typically supplemented with B12 at a dose of 500–1000µg per dose, subcutaneously, for 4 to 5 weeks on a weekly basis. Serum concentrations of B12 should be rechecked q 3-4 months if clinical signs of diarrhea are still persistent, or alternatively, B12 can be empirically administered at the same dose.

Water-soluble vitamins are often depleted by the fluid losses associated with diarrhea and fat-soluble vitamin loss can be significant in animals with steatorrhoea. Deficiencies of vitamins A, C, D, E and have been reported in the human literature. Common mineral deficiencies in patients with ulcerative colitis and Crohn’s disease include selenium, zinc and magnesium. Published recommendations for vitamin B\(_{12}\), K and E supplementation in gastrointestinal disease are available, but further investigation into vitamin and mineral status and requirements for dogs with IBD is warranted.

### TABLE 1

**ANTIBIOTICS FOR TREATMENT OF BACTERIAL-ASSOCIATED DIARRHEAS**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enrofloxacin</td>
<td>5 mg/kg BID, PO</td>
<td>Sepsis (Gram negative bacteria)</td>
</tr>
<tr>
<td>Enrofloxacin</td>
<td>5 mg/kg BID, PO</td>
<td>Campylobacter spp.</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>10-15 mg/kg TID, PO</td>
<td>Campylobacter spp.</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>10-20 mg/kg TID, PO</td>
<td>Clostridium perfringens</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>10-20 mg/kg TID, IV, SC</td>
<td>Sepsis (use with aminoglycosides)</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>2.2 mg/kg TID, IV, SC</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Cephalothin</td>
<td>22-44 mg/kg TID, IV, IM</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>10-15 mg/kg BID, PO</td>
<td>C. perfringens, C. difficile, IBD</td>
</tr>
<tr>
<td>Tylosin</td>
<td>15 mg/kg SID-BID, PO</td>
<td>C. perfringens, IBD</td>
</tr>
<tr>
<td>Trimethoprin-sulfa</td>
<td>15 mg/kg BID, PO, IV, SC</td>
<td>Salmonella spp.</td>
</tr>
<tr>
<td>Enrofloxacin</td>
<td>5 mg/kg BID, PO</td>
<td>Salmonella spp.</td>
</tr>
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REFERENCES


