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*Prof. Celia Holland*

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Recent Findings of *Toxocara* Eggs in the Coats of Dogs

*Toxocara canis* (Figure 1), the common intestinal nematode of dogs, wolves, and foxes, is the parasite primarily responsible for human toxocariasis. Adult worms live in the dog’s small intestine, and highly fecund female worms are capable of producing millions of eggs in the feces. These eggs are not infective at the time they are shed, but rather require a period of time under appropriate conditions to become infective (Figure 2).

Considerable attention has been given to the epidemiology of toxocariasis in domesticated dogs. The prevalence of infection varies greatly, depending on the age and geographic location of the dog, whether or not it is stray, and how the infection was detected. Despite these variations, *Toxocara* infection is highly prevalent among pet dogs that are kept in close proximity to their human families. A national survey of American households estimated that approximately 60% owned dogs, cats, or both, and the highest rate of dog and cat ownership occurred in families with young children.

Human infection is a consequence of the ingestion of embryonated eggs followed by the hatching of third-stage larvae. In so-called paratenic hosts, such as humans and small rodents, larvae undergo a somatic migration through the various organs of the body but fail to reach maturity as adult worms. Although our understanding of the pathology in humans is incomplete, results of seroprevalence studies suggest that exposure levels are high among those who are disadvantaged and those who live in tropical environments. Larval involvement in the eye, termed *ocular toxocariasis*, remains the most devastating sequela of infection in humans, frequently leading to visual impairment and occasionally resulting in blindness.

Although contact with contaminated soil is considered to be the primary route of transmission to humans, recent research has suggested that direct contact with dogs may be an underestimated alternative route. In a study by Wolfe and Wright, hair samples were collected from 60 dogs from various regions of the United Kingdom and Ireland. The dogs, which ranged in age from 8 weeks to...
15 years, were a mixture of strays, working farm dogs, and in-home puppies. Of these 60 dogs, 11 were found to have *Toxocara* eggs in their coats (4 puppies and 11 adult dogs). The greatest number of eggs (300) were detected on a puppy; of these, 20 were embryonated, 180 were embryonating, and 100 were viable. The average eggs per gram of hair for puppies was elevated compared with that of adult dogs. While these are interesting findings, the study does have its flaws. The method of detection was not standardized, and the authors did not address whether the eggs were believed to be embryonated *in situ* or acquired from the environment.

Subsequently, Roddie and colleagues studied hair samples obtained from stray dogs from County Cork, Ireland. By using a standardized method of detection (albeit with a recovery rate of 52%), these authors sampled both puppies (*n*=25) and adult dogs (*n*=71), then performed postmortem examinations to count the number of worms harbored per infected host. Four key findings emerged:

1. All stray puppies harbored *Toxocara* eggs on their hair, and the intensity of eggs was markedly higher than in adult dogs.
2. There was no significant statistical relationship between the number of eggs detected on the hair and the intestinal worm burden of *Toxocara* for adult dogs, whereas there was a significant relationship for puppies. This finding suggests that adults may be acquiring some eggs from the environment, whereas puppies are more likely to be contaminating their own fur.
3. Virtually all the embryonated eggs were found on the hair of puppies (of the 120 eggs found, 117 of them were from puppies). The embryonation rate for adult dogs was 0.12% versus 0.31% for puppies.
4. There was no significant difference between the number of eggs detected on the hair of the dorsum and those on the hair around the anus of the dog.

Ayenizoz-Ozkayhan and colleagues focused their recent research on owned dogs in Turkey. The authors initially sought to determine whether breed and coat type influenced the presence of eggs on hair. Their sample included 26 puppies and 17 adult dogs, but a lack of variation in dog breed and coat precluded rigorous testing of the hypothesis. The prevalence of *Toxocara* eggs on the hair and the average number of eggs detected were similar to those found by Wolfe and Wright. The authors concluded that the age of the dog was a key risk factor for the presence of *Toxocara* eggs on fur because the vast majority of eggs were found on puppies.

From this recent research, we can conclude that the status of the dog and its age are clearly important factors influencing the prevalence of *Toxocara* eggs on its coat.
still relatively few studies of the prevalence and intensity of *Toxocara* eggs on the hair of owned puppies, and there is little data comparing contaminated dog hair and contaminated soil as modes of transmission to humans. Additional research is needed to determine the relative success of embryonation in soil versus hair under experimental conditions.

References

Intestinal helminths, pentastomids, and intestinal and blood protozoa are common parasites of captive and wild reptiles. (Reptiles may serve as definitive, intermediate, accidental, or paratenic hosts). Generally, these parasites are commensal in wild animals and do not cause illness. However, they may become pathogenic when present in high concentrations or if their host is weakened by inadequate environmental conditions in captivity (Table 1).

**Anthelmintic Compounds for Reptiles**

When treating reptiles with nematodiasis, cestodiasis, trematodiasis, or acanthocephalidiasis, veterinarians generally rely on deworming protocols that originated in domestic animal medicine. Over time, some of these compounds were tested and became empirically recommended because of their efficacy and safety, and there is now much documented evidence to support their use in reptiles.

Since the 1960s, 3 classes of broad-spectrum anthelmintic drugs have been predominantly used to control helminths in mammals: Benzimidazoles (eg, fenbendazole), tetrahydropyrimidines, imidazothiazoles (eg, pyrantel, morantel-levamisole), and macrocyclic lactones (eg, ivermectin). While effective, many are distributed primarily as oral formulations that are viewed unfavorably by pet owners. As with many veterinary pharmaceuticals, the safety profiles for some agents are still emerging. In a study of 6 male Hermann’s tortoises (*Testudo hermanni*) treated with oral fenbendazole, blood analysis showed transient hypoglycemia, hyperuricemia, and hyperphosphatemia. Consequently, the authors recommended that baseline serum studies be performed before administering fenbendazole, and hematologic parameters be monitored in treated animals.

Emodepside and praziquantel (Table 2) are now available in combination as a topical formulation (Profender®, Bayer HealthCare Group, bayer-ah.com) that was first found to be highly effective in cats and is now being used more widely in reptiles. Case reports involving snakes, aquatic turtles, agamids, varanids, and geckonids have demonstrated...
that the agent is effective against nematodes belonging to the families Oxyridae, Ascaridae, Strongylidae, Trychostrongylidae, and Capillaridae.12

Efficacy
To objectively determine the clinical efficacy of emodepside-praziquantel in reptiles, we selected 16 animals of various species confirmed to be harboring nematodes of different families (oxyurids, ascarids, rhabdiasids, and strongylids) and belonging to herpetoculturists or sellers of exotic animals. All subjects had positive parasitic fecal examinations through direct examination and flotation before receiving treatment with topical emodepside-praziquantel, 0.56 mL/kg as a single administration. The tested animals were kept on newspaper in dry conditions at approximately 86°F during the 15-day study, and the bedding was replaced after every defection to avoid reinfection. Repeat fecal examinations were performed on days 7 and 15. As shown in Table 3, emodepside-praziquantel was only partially effective at this dosage, particularly in cases of severe infestations by pinworms or ascarids and in reptiles with thicker integument (such as terrestrial tortoises); no adverse effects were observed. These results correlate with those from a German study performed on a substantially larger sample.13

Pharmacokinetics. In a separate study, 11 healthy adult reptiles (3 green iguanas, 1 Argentine black-and-white tegu, 1 ball python, 1 corn snake, 2 Savannah monitor lizards, 1 Hermann’s tortoise, 1 spur-thigh tortoise, 1 red-eared slider turtle) were tested. The animals were kept at 86°F to 90°F for the duration of the study. Blood was collected before treatment and at 3 intervals (5, 24, and 48 hours) after treatment with topical emodepside-
praziquantel, 2 drops/100 g body weight. Blood samples were heparinized, centrifuged, and frozen (-14°C to -18°C). Pooled serum from 20 untreated turtles and tortoises (control animals) was collected for comparison.

Results of this study show that both of the metabolites penetrated the skin and were found in the serum at levels similar to those seen in cats. However, reptiles with thicker integument (eg, terrestrial tortoises, green iguanas, ball pythons, Savannah monitors) showed relatively low concentrations for both praziquantel and emodepside 48 hours after administration; in some cases, emodepside concentrations were extremely low (< 2 mcg/L). In these species, a higher dosage may be needed to achieve therapeutic levels.

Discussion
On the basis of these clinical and pharmacokinetic studies, an increased dose of 4 drops/100 g (1 mL/kg) body weight is now recommended. Emodepside-praziquantel is distributed as a feline dewormer in single-dose pipettes available in 3 sizes for small (0.35 mL), medium (0.7 mL), and large (1.12 mL) cats. All 3 contain the same concentration of active ingredients. The topical route of administration is relatively simple and less traumatizing than orogastric intubation, reducing the stress to shy lizards and turtles (and their owners).

Despite the encouraging results from early case reports, veterinarians should exercise caution when dispensing this medication. Further research should be undertaken in a wide range of reptile species to verify the safety and efficacy of this promising drug.

Table 2. Emodepside & Praziquantel Profile

<table>
<thead>
<tr>
<th>Medication</th>
<th>Mode of Action</th>
<th>Effective Against</th>
</tr>
</thead>
<tbody>
<tr>
<td>Emodepside</td>
<td>Stimulates presynaptic receptors at neuromuscular junction</td>
<td>Gastrointestinal nematodes, trichostongylids, ascarids, hookworms, trichocephalids, large &amp; small strongyles, &amp; respiratory strongyles; nematodes resistant to ivermectin, benzimidazoles, and levamisole</td>
</tr>
<tr>
<td>Praziquantel</td>
<td>Induces severe muscular contractions, dislodging the worm. Within 5 minutes, major changes in the syncytial integument begin in the scolex and strobila, likely the result of interaction with phospholipids and proteins, leading to the reduction of glucose intake and accelerated depletion of energy reserves</td>
<td>Tapeworms, blood fluke, liver fluke (except Fasciola hepatica), lung fluke</td>
</tr>
</tbody>
</table>

Table 3. Efficacy of Emodepside-Praziquantel in individual Reptiles*

<table>
<thead>
<tr>
<th>Species</th>
<th>Parasites Observed*</th>
<th>Weight (g)</th>
<th>mL/kg</th>
<th>Drops</th>
<th>7 Days</th>
<th>15 Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eurotestudo hermanni</td>
<td>Ascaris species (+)</td>
<td>1025</td>
<td>0.57</td>
<td>23</td>
<td>+ (Pinworms)</td>
<td>+ (Pinworms)</td>
</tr>
<tr>
<td>Pogona henrilawsonni</td>
<td>Pinworms (+++)</td>
<td>45</td>
<td>0.02</td>
<td>1</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Boa constrictor</td>
<td>Rhabditidae species (++)</td>
<td>3225</td>
<td>1.806</td>
<td>71</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Testudo iberia</td>
<td>Pinworms (+)</td>
<td>750</td>
<td>0.42</td>
<td>15</td>
<td>+ (Pinworms)</td>
<td>–</td>
</tr>
<tr>
<td>Testudo marokkensis</td>
<td>Pinworms (+)</td>
<td>995</td>
<td>0.56</td>
<td>22</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Testudo graeca</td>
<td>Pinworms (+++)</td>
<td>360</td>
<td>0.2</td>
<td>7</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Iguana iguana</td>
<td>Pinworms (+++)</td>
<td>2100</td>
<td>1.17</td>
<td>45</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>Uromastyx acanthinurus</td>
<td>Pinworms (+)</td>
<td>320</td>
<td>0.18</td>
<td>7</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Eurotestudo hermanni</td>
<td>Ascaris species (+++)</td>
<td>1200</td>
<td>0.67</td>
<td>26</td>
<td>+ (Pinworms)</td>
<td>+ (Pinworms)</td>
</tr>
<tr>
<td>Geochelone sulcata</td>
<td>Ascaris species (+++)</td>
<td>4500</td>
<td>2.52</td>
<td>100</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>Geochelone pardalis</td>
<td>Ascaris species (+++)</td>
<td>2450</td>
<td>1.37</td>
<td>53</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>Eublepharis macularius</td>
<td>Pinworms (+++)</td>
<td>40</td>
<td>0.02</td>
<td>1</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Python molurus</td>
<td>Strongylidae species (+)</td>
<td>1750</td>
<td>0.98</td>
<td>39</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>Crotophytus collaris</td>
<td>Pinworms (+++)</td>
<td>55</td>
<td>0.03</td>
<td>1</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Pogona vitticeps</td>
<td>Pinworms (+++)</td>
<td>245</td>
<td>0.13</td>
<td>5</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>Pogona vitticeps</td>
<td>Pinworms (+)</td>
<td>230</td>
<td>0.13</td>
<td>5</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

* + = < 10 eggs per field; ++ = 10–20 eggs per field; +++ = > 20 eggs per field
Acknowledgements
The author gratefully acknowledges Dr. Roland Schaper (Bayer HealthCare AG, Leverkusen-Germany), Dr. Julien Rocher (Bayer Santé Division Animale, Puteaux-France), and Drs. Olivier Bétrémieux; Juliette Tiempé, and Soline Vénard (CVVA-France) for their contributions to this article.

References

To sufficiently penetrate the thick horny outer layer of the reptilian epidermis, emodepside-praziquantel must be administered in higher doses than those used for cats. Doses 15, 30, and even 50 times higher than feline dosages have been administered without adverse effects, but reptile guidelines recommend administering 2 drops/100 g (0.56 mL/kg) body weight. Because this may be insufficient to achieve therapeutic levels in reptiles—particularly those with thicker integument, I recommend increasing the dose to 4 drops/100 g (1 mL/kg) body weight. Apply the medication where the epidermis is relatively thin, such as the gular region in snakes, under armpits or skinfolds of the groin area in lizards, or in the prefrontal area or gural fossa in turtles. Aquatic species must be kept in a dry place for 48 hours following topical application.

Administration of Profender’ spot-on solution on the integument of a green iguana (Iguana iguana)
**Results of Recent *Giardia* Studies**

*Giardia duodenalis* is one of the most common parasites affecting dogs throughout the world.¹ One could debate the zoonotic potential of this parasite relative to humans and other animal hosts,² but few would disagree that dogs are highly likely to get infected from cysts shed by other dogs, or that the parasite can cause disease in an otherwise healthy canine host.³

In the developed world, humans are likely to develop symptomatic illness (namely, severe diarrhea) when exposed to the parasite because giardial cysts have been essentially removed from our regular diet thanks to cleaner drinking water. Consequently, we have not acquired immunity through repeated lifelong exposure.⁴ A similar scenario seems to be emerging among our canine companions that generally drink very clean water and do not get a regular dose of cysts from their environment.³ As a result, when they happily drink from a puddle, they are at risk for a disease that was not a concern just a few years ago.

There may be a seemingly obvious need to treat giardiasis in dogs that have signs of infection (particularly because some dogs appear to be in significant discomfort), but many consider the treatment of dogs without signs to be unwarranted. However, even without signs, dogs are potential carriers of infection for other dogs. Treatment of carriers reduces the risk that they will infect naïve dogs that share the same space.

Giardiasis in dogs has been treated using several different drugs, but none is actually approved for this purpose in animals. The most commonly prescribed drug for giardiasis is metronidazole,⁵ which is approved for use only in humans but is prescribed by veterinarians under the Animal Medicinal Drug Use Clarification Act. Some of the benzimidazoles (eg, fenbendazole and oxfendazole) have been shown to be efficacious against giardiasis.⁶,⁷ The combination of pyrantel, febantel, and praziquantel given for 3 consecutive days has also been found to be somewhat efficacious in clearing giardial cysts in dogs.⁸⁻¹⁰ However, another trial of the same dosing regimen was not efficacious.¹¹

Additional studies in dogs have investigated 3- and 5-day regimens of pyrantel, febantel, and praziquantel, both at the dosage approved in Europe for the treatment of canine helminth infections and at 3 times that dosage.¹²,¹³
In one U.S. study, treatment resulted in complete clearance of cysts in 8 of 12 treated dogs, whereas all 6 untreated dogs continued to shed cysts. Of 6 dogs receiving the low-dose regimen for 5 days, only 1 shed cysts 9 and 10 days after completing treatment; of 6 dogs receiving the high-dose regimen, 3 shed cysts after the last dose. A recent study performed in Spain examined use of a standard dose to dogs housed in a shelter. Eight dogs received treatment for 3 days; 1 dog continued to shed cysts during and after treatment, and 1 shed cysts only twice (3 and 5 days after the last dose). Of 8 dogs treated for 5 days, 3 shed cysts after the last treatment: 1 on the first day after the last treatment, 1 on the 5th day, and one on the 5th and 7th days. When considering duration of treatment, clinicians should be advised that some researchers have suggested that giving a dog a good bath and changing its environment after treatment may be more important to curing the infection than the number of doses of medication administered.

We report here on a study of dogs that received 3 days of treatment with the recommended dosage for confirmed* Giardia duodenalis infection. In this study, dogs were initially verified as being infected by use of a zinc sulfate concentration technique. The cysts shed were quantified in dilute feces through direct immunofluorescent labeling of the cysts rather than through the use of flotation or antigen detection.

### Methods

The study utilized 14 random-source dogs that were housed individually. The dogs, which were determined by physical examination to be otherwise healthy, had confirmed presence of giardial cysts in their feces. One group of 7 dogs was assigned to receive oral therapy with pyrantel/febantel/praziquantel at the recommended dosage for 3 days and a second group of 7 dogs remained untreated as controls. Cysts were enumerated from fecal samples collected on Days -7, -5, and -3, then daily from day 1 until the end of the trial on day 11 (7 days after the last treatment). Comparing the number of cysts in fecal samples collected from the 2 groups before treatment was begun, there was no significant difference in the mean number of cysts (P=.51).

### Results

The treated dogs had markedly fewer cysts in their feces within 1 day of beginning treatment (Table 1). Two days after treatment, cysts were found in the feces of only 1 dog. The mean number of cysts was significantly reduced in fecal samples collected from the treated dogs on the second and third days of treatment (P=.004), and the number of fecal oocysts were also significantly decreased within the samples collected 1 week after treatment.

<table>
<thead>
<tr>
<th>Day on Trial</th>
<th>Treated Dogs</th>
<th>Untreated Dogs</th>
<th>Calculated Efficacy (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>-7</td>
<td>21,679</td>
<td>10,291</td>
<td>N/A</td>
</tr>
<tr>
<td>-5</td>
<td>61,228</td>
<td>340,653</td>
<td>N/A</td>
</tr>
<tr>
<td>-3</td>
<td>104,111</td>
<td>368,594</td>
<td>N/A</td>
</tr>
<tr>
<td>1</td>
<td>10,507</td>
<td>371,805</td>
<td>97.2</td>
</tr>
<tr>
<td>2</td>
<td>105</td>
<td>537,504</td>
<td>100.0</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>296,362</td>
<td>100.0</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
<td>42,189</td>
<td>100.0</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>54,328</td>
<td>100.0</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>10,125</td>
<td>100.0</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>6161</td>
<td>100.0</td>
</tr>
<tr>
<td>8</td>
<td>5</td>
<td>63,272</td>
<td>100.0</td>
</tr>
<tr>
<td>9</td>
<td>4</td>
<td>73,483</td>
<td>100.0</td>
</tr>
<tr>
<td>10</td>
<td>43</td>
<td>36,813</td>
<td>99.9</td>
</tr>
<tr>
<td>11</td>
<td>321</td>
<td>284</td>
<td>N/A</td>
</tr>
</tbody>
</table>

* Treatment began on Day 0.
It was unclear at the end of the trial whether treated dogs had actually been cleared of infection. Six days after the last treatment (day 8) an increased number of dogs began to shed cysts. As shown in Figure 1, the mean number of cysts shed also increased. Simultaneously, 2 of the control dogs that had had long-standing infections were found to have significantly decreased numbers of cysts on the last day of the trial (day 11). Consequently, on the final day of the trial, 3 treated dogs and 3 control dogs were shedding cysts.

**Three consecutive days of treatment with the recommended dosage of pyrantel/febantel/praziquantel tablets seemed to halt shedding of giardial cysts in most dogs.**

**Discussion**

In our trial, we found that 3 consecutive days of treatment with the recommended dosage of pyrantel/febantel/praziquantel tablets seemed to halt shedding of giardial cysts in most dogs. However, beginning 6 days after treatment, there were more dogs shedding cysts and an increased mean number of cysts were shed; other studies have yielded similar results.\(^\text{13}\) We cannot say for sure whether the reappearance of cysts is due to insufficient clearance of trophozoites from the animals or to reinfection. Without stringent biocontainment of the individual animals, the possibility of reinfection cannot be dismissed because the prepatent period in dogs can be as short as 5 days.\(^\text{2}\) Also, the assay used here may not have had sufficient sensitivity to assess the total disappearance of organisms from the intestinal tract, although this flaw is not unique to the assay we used. Almost all studies (regardless of the assay used) have shown a significant suppression in cyst shedding, but none has been able to verify total clearance or reinfection because some animals always seem to become positive again shortly after treatment.

**References**


Endemic *Angiostrongylus vasorum* infections in dogs (canine angiostrongylosis) were first recognized in southern France in the early 1800s, giving rise to the term “French heartworm.” During the past decade, an increasing number of cases of this disease have been reported in North and South America, Africa, and across Europe. Most frequently, the infection manifests in dogs as verminous pneumonia and coagulopathy that can be fatal if left untreated. Recent studies have provided evidence-based knowledge of efficacy and safety of the various anthelmintic treatment options available, giving clinicians more confidence to tailor therapy to the patient.

A metastrongyloid nematode, *A. vasorum* uses snails and slugs as intermediate hosts (Figure 1). A wide range of definitive hosts have been described, most of which are closely related: dog, fox, wolf, coyote, and jackal.1 After a dog ingests a colonized snail or slug, the larvae are released into the stomach and penetrate the stomach wall to enter the mesenteric lymph nodes. The larvae travel through the lymphatic system to the right side of the heart and pulmonary arteries, where they mature and lay eggs. The eggs are carried into the pulmonary capillaries, where they eventually hatch and penetrate the capillary walls, entering alveoli. They larvae are then coughed up, swallowed, and excreted with feces.1

**Individual assessment of each case, including analysis of clinical and paraclinical findings, is necessary to give a precise prognosis for survival.**

**Clinical Presentation**

In the 2002 Copenhagen *Angiostrongylus* survey (CAS), the disease was diagnosed in dogs ranging in age from 3 months to 14 years, although more than 50% of affected dogs (*n*=163) were 18 months of age or younger. No gender predisposition was found.2 Although some infected dogs may show no clinical signs, others may have acute onset of severe, life-threatening disease. The various clinical features, which may be present alone or in combination, can broadly be characterized into 3 groups: respiratory signs, bleeding problems, and miscellaneous disease (Table 1). Respiratory signs are most predominant, due to verminous pneumonia and the inflammation caused by the larvae migration through the pulmonary parenchyma of the host. The most common respiratory signs (70% of reported cases) include cough, dyspnea, tachypnea, and exercise intolerance. Auscultatory findings may be unremarkable, but crackles may be heard in more severe cases.3

Bleeding disorders such as chronic low-grade disseminated intravascular coagulation,4 coagulation in consumption,5,6 and immune-mediated thrombocytopenia may be common but their mechanisms in *A. vasorum* infections are not yet fully understood.5–11
In the absence of clinical signs related to the respiratory tract or bleeding problems, canine angiostrongylosis can be a challenge to diagnose. The miscellaneous disease group includes several nonspecific signs, such as depression, anorexia and weight loss, lethargy, and exercise intolerance. Involvement of other organ systems, such as the gastrointestinal tract (vomiting and diarrhea), the central nervous system (seizures, circling, ataxia, tetraparesis, and others) and the musculoskeletal system (lameness) demonstrate that *A. vasorum* infection can be a great imitator.

**Diagnosis**

The definitive diagnosis of canine angiostrongylosis is made by demonstration of either adult worms or first-stage larvae in the definitive host. With this in mind, a detailed knowledge of the morphology of the different stages of *A. vasorum* and the L1 larvae in particular, is essential (Figure 2, page 14). The Baermann sedimentation test is the gold standard for detecting *A. vasorum* because of its simplicity and rapid results, but L1 larvae can also be found by tracheal wash or bronchoalveolar lavage. Such diagnostic methods as polymerase chain reaction or enzyme-linked immunosorbent assay–based techniques are not yet commercially available.

**Thoracic Radiographs**

Presence of adult worms and larval migration through lung parenchyma cause an inflammatory process and verminous pneumonia. Pulmonary radiography is an important and valuable diagnostic tool. Depending on the magnitude and duration of infection, the radiographic appearance will vary—including interstitial, bronchial, and alveolar lung patterns. Lesions are commonly located in the periphery of the lungs, particularly in the dorsal and caudal lung fields.

**Hematologic and Biochemical Parameters**

Results of hematologic and biochemical studies are generally nonspecific. Eosinophilia, neutrophilia, mild thrombocytopenia, hyperglobulinemia, and reduced serum fructosamine levels are the most frequent findings. Veterinarians should keep in mind, however, that a normal hematologic

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**Table 1. Clinical Signs in Dogs at Time of Diagnosis with Angiostrongylus**

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Koch et al, 2005² Frequency, % (n=160)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coughing</td>
<td>68.1</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>23.1</td>
</tr>
<tr>
<td>Depression</td>
<td>21.9</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>15.6</td>
</tr>
<tr>
<td>Exercise intolerance</td>
<td>15.6</td>
</tr>
<tr>
<td>Anorexia</td>
<td>14.4</td>
</tr>
<tr>
<td>Weight loss</td>
<td>11.3</td>
</tr>
<tr>
<td>Vomiting</td>
<td>10.0</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>5.6</td>
</tr>
<tr>
<td>Neurologic signs</td>
<td>4.4</td>
</tr>
<tr>
<td>Subcutaneous swelling</td>
<td>2.5</td>
</tr>
<tr>
<td>Clearing throat</td>
<td>1.9</td>
</tr>
<tr>
<td>Others</td>
<td>5.6</td>
</tr>
<tr>
<td>No clinical signs</td>
<td>6.9</td>
</tr>
</tbody>
</table>

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**Indirect Life Cycle of *Angiostrongylus vasorum*¹⁶**

*Figure 1.* Indirect life cycle of *Angiostrongylus vasorum*. L1 larvae are transferred to the intermediate host after a prepatent period of 35 to 60 days. The larvae then molt twice in order to become infective L3 larvae. The definitive host becomes infected after ingestion of infected intermediate hosts.

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**Bleeding Commonly Associated with *Angiostrongylus vasorum***

- Petechiae or ecchymosis
- Traumatic or postoperative hematomas
- Intracranial hemorrhage
- Epistaxis
- Hemoptysis
- Hematuria
- Gastrointestinal bleeding
- Scleral or conjunctival hemorrhage
and biochemical blood profile cannot exclude *A. vasorum* as the cause of disease.

**Therapy**
The treatment of canine angiostrongylosis is generally 2-pronged: Direct anthelmintic therapy and supportive therapy. Several different treatment protocols have been used and reported to be efficacious (Table 2). During the past decade, fenbendazole has become widely used in a large number of dose regimens, ranging from 20 to 50 mg/kg for 5 to 21 days, and seems to have replaced levamisole and ivermectin as the preferred treatment. The recent introduction of novel formulations of macrocyclic lactones, such as milbemycin oxime (Milbemax®; Novartis Animal Health, ah-novartis.com) and moxidectin has increased treatment options available for certain breeds of dogs that are at increased risk for ivermectin toxicosis. Prospective studies of fenbendazole (Panacur®; intervet.com) and imidacloprid/moxidectin (Advocate®; Bayer Animal Health, advocate-spot-on.com [marketed as Advantage Multi in the U.S.; bayer-ah.com] have shown that these treatments offer similar efficacies.

Whereas comparisons of anthelmintic therapeutic options for canine angiostrongylosis are becoming increasingly evidence-based, reports of supportive treatment are still anecdotal. The use of antibiotics, bronchodilators, diuretics, heparin, and corticosteroids are often reported. Certainly the use of immunosuppressive dosages of corticosteroids in cases of immune-mediated thrombocytopenia and anaphylactic reactions seems to be indicated. Some authors have suggested that corticosteroids may also be beneficial for reducing pulmonary inflammation and secondary fibrosis of the lungs. For initial stabilization, dogs with life-threatening bleeding problems, thrombocytopenia, or disseminated intravascular coagulation may be supported with transfusions of fresh frozen plasma or whole blood while anthelmintic treatment is initiated. For unknown reasons, bleeding disorders generally seem to resolve within 24 to 48 hours after the onset of anthelmintic treatment.

**Prognosis and Future Management**
Nearly all dogs with mild to moderate cases of angiostrongylosis will survive. In contrast, 25% of dogs with severe disease will die naturally or be euthanized. In most cases of fatal disease, the reported cause of death is coagulopathies, severe dyspnea, or acute heart failure. Individual assessment of each case, including analysis of clinical and paraclinical findings, is necessary to give a more precise prognosis for survival.

The usefulness of prophylactic anthelmintic treatment is still under investigation. The availability of a simple, efficacious regimen will determine which protocol to use. Local conditions (such as licensing requirements) may dictate why one treatment is prescribed over another. As a preventive measure, veterinarians should also educate clients about measures to take to protect their dogs (and others) from infection. To prevent environmental contamination by L1 larvae, proper disposal of the affected dog’s feces will effectively break the life cycle of *A. vasorum*.

**Table 2. Anthelmintic Treatment Protocols for Canine Angiostrongylosis**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fenbendazole</td>
<td>50 mg/kg, Q 24 H for 5 to 21 days³</td>
</tr>
<tr>
<td>Fenbendazole</td>
<td>25 mg/kg, Q 24 H for 20 days¹⁵</td>
</tr>
<tr>
<td>Milbemycin oxime</td>
<td>0.5 mg/kg, once weekly for 4 weeks¹⁴</td>
</tr>
<tr>
<td>Imidacloprid/moxidectin</td>
<td>0.1 mL/kg, single topical dose¹⁵</td>
</tr>
</tbody>
</table>

³ After initial anthelmintic treatment, repeat anthelmintic treatment in 30 days is recommended. The addition of corticosteroids for clinical improvement is recommended for high-risk cases. Final anthelmintic treatment is repeated in 30 days. 

¹⁴ In mild cases of angiostrongylosis, a single treatment is recommended. 

¹⁵ In severe cases of angiostrongylosis, 30 mg/kg for 3 days is recommended. Repeat anthelmintic treatment in 14 days is recommended.
By educating dog owners, we can promote more testing on asymptomatic dogs, thereby preventing the development of serious clinical signs. Considering the increased transportation of dogs between locations, owners may consider prophylactic treatment or Baermann testing if they are moving to an area where *A. vasorum* is not yet established.

**References**