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Respiratory System Parasites of the Dog and Cat (Part II): Trachea and Bronchi, and Pulmonary Vessels (20 Apr 2000)

D.D. Bowman
Department of Microbiology & Immunology, College of Veterinary Medicine, Cornell University, Ithaca, New York, USA.

This is Part II of a review that covers the majority of parasitic disease of the respiratory system of the dog and cat. Excluded from this review is the canine heartworm which is important and common enough to warrant separate coverage. Also excluded from this review are the lesions due to Spirocerca lupi, although sometimes included as parasite of the respiratory tract, the adults are parasites of the stomach and esophageal wall, and the lesions produced by the migrating larvae are in the aorta and will be covered under parasites of the vascular system.

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Part II: TRACHEA AND BRONCHI

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Part II: PULMONARY VESSELS

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Toxocara spp. [nematode]
Eucoleus aerophilus [nematode]

Trachea and Bronchi

Crenosoma vulpis (Nematode)
This is a metastrongyloid nematode parasite of bronchi of dogs and other canids. This parasite occurs in dogs throughout North America and Eurasia [1, 2]. These white worms are characterized by having a crenated cuticle that is thrown into folds on the anterior end making this portion of the worm appearing superficially segmented. The males are 4 to 8 mm long and possess a bursa. The females are 12 to 16 mm long and have the vulva located near mid body. The adults are 0.3 to 0.5 mm in width, and therefore, appear much stockier than parasites such as Aelurostrongylus which have a similar length. The females lay eggs that develop and hatch within the respiratory tract. The larvae are then coughed up and swallowed to be passed in the feces (Fig. 1). Diagnosis can be made by the recovery of the larvae from the feces by the Baermann procedure or by zinc-sulfate centrifugal flotation. Larvae and adult worms may be found in trachobronchial mucus or bronchoalveolar lavage samples. The larvae are characterized by possessing a very pointed tail; they measure 250 to 300 microns in length.
This parasite has been shown to utilize gastropod intermediate hosts [3-5]. Attempts to infect mice with the larvae from snails were unsuccessful, and it is believed that dogs are probably infected by ingesting the gastropod intermediate host [6]. After dogs ingest the infective-stage larvae from the snail, the larvae migrate to the lungs by way of the visceral lymphatics [4, 5] or via the hepatic portal system [7]. Females deposit larvae about 19 days after infection, and the prepatent period is 18 to 21 days. The adult worms probably live eight to nine months or longer.

Infection with this worm typically produces a dry, nonproductive cough that can be elicited by tracheal palpation [8]. In some cases, the cough may be chronic and productive [9]. The worms can be present in considerable numbers. Radiographic changes include enhanced definition in the hilus and shoulder regions of the bronchial walls; marked bronchial patterns with prominent interstitial markings, and in cases where the cough is productive cardiomegaly with interstitial densities in the diaphragmatic lobe [2, 8, 9]. Bronchoscopy may reveal no reaction of inflammation with moderate mucoid to mucopurulent discharge in the airways [9, 10]. Cytological examination of tracheal wash samples will reveal signs of eosinophilia indicative of parasitic bronchitis. Stockdale and Hullund [7] showed that the third-stage larvae migrating through the liver cause the formation of necrotic foci during the first days of infection, and that the discarded cuticles of these larvae after they reach the lungs cause granulomas to form as centers of interstitial pneumonia.

Treatment of infections with *Crenosoma vulpis* have been effected with fenbendazole [1, 9, 11]. Doses used have included 50 mg per kg body weight once-a-day of three days and 20 mg per kg body weight daily for 14 days. Other successful treatments have included the subcutaneous injection of 7.5 mg of levamisole per kg bodyweight followed by as second injection 2 days later [10]. Treatment with febantel as part of Drontal Plus (50 mg praziquantel, 144 mg pyrantel embonate, and 150 mg febantel) once daily for 7 days has also been shown to clear the infection [8].

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**Oslerus osleri** (Nematode)

This is a metastrogyloind nematode parasite causing nodules in the trachea and bronchi of dogs and other canids. These are very small worms that are found in nodules that tend to be located close to the bifurcation of the trachea (Fig. 2, Fig. 3). The males are only 4 to 7 mm long and the females are only slightly longer (Fig. 4). The vulva of the female is located just anterior to the anus. The stage passed in the feces is a first-stage larva (Fig. 5, (Fig. 6) which is virtually indistinguishable from the first-stage larva of *Filaroides hirthi*, and larvae are best found in fecal samples by using direct smears or zinc-sulfate centrifugal flotations [12]. The larva passed in the feces is characterized by having a tail that is characterized by having a constriction just anterior to its tip, which gives the very tip of the tail a kinked appearance. This parasite has been diagnosed in dogs from around the world [13-18]. The diagnosis of infection is be confirmed by the viewing of nodules in the trachea with a bronchoscope, and the presence of the fibrous nodular projections into the lumen of the trachea and bronchi is diagnostic.
Oslerus osleri

The life cycle of Oslerus osleri has been shown to be direct. Dogs are probably commonly infected as puppies by the transmission of larvae in sputum by the licking and cleaning of the mother or through regurgitated food [17, 19]. Dunsmore and Spratt [19] found fourth-stage larvae in an experimentally infected Dingo 14 days after infection and immature adults one month postinfection. An adult female was found in the tracheal mucosa 60 days after the experimental infection of a pup [18]. Dunsmore and Spratt [19] found adults in nodules 70 days after infection and nodules in experimentally infected dingos 18 months after infection. The signs of infection with Oslerus osleri is a dry cough that is precipitated by exercise. Laryngeal or tracheal massage does not tend to elicit a cough as in typical cases of bronchitis. There are usually no signs of serious disease until the nodules are enlarged to the point where they cause obstruction of air flow. Some dogs present with a persistent cough over a year-long period [14]; while in another case, a 5- to 6-month old Blue Heeler in Australia was diagnosed as having severe respiratory distress due to Oslerus osleri infection. Cases observed in Yorkshire terriers presented initially as a dry cough that had not responded to antibiotic treatment. After diagnosis, one dog ultimately had to be euthanatized due to the development of severe dyspnea [20]. A 7-month-old Pomeranian in Japan presented with a 2-week history of severe dyspnea, and in spite of supportive treatment which included the administration of supplemental oxygen, the animal died 5 days after presentation [13]. Another presentation has been recurrent pneumothorax, that was cured by the removal of the obstructing nodules from the trachea [21]. Nodules are often observed on radiographs , and then there presence confirmed by endotracheal observation.

Treatment of infections with Oslerus osleri appears to be successful by the subcutaneous administration of ivermectin at 0.2 mg per kg bodyweight [14]. There are three criteria for successful chemotherapy of infections with this parasite: disappearance of the signs associated with the infection, resolution of the nodules, and the disappearance of the larvae from the feces of the infected animal. Fenbendazole (50 mg per kg daily for seven days) has been reported to stop coughing in an infected dog [22]. Albendazole thiabendazole, and intravenous thiacetarsemide do not appear to clear all dogs of their infections. Thus, it would appear that at this time, ivermectin or fenbendazole would be the treatment of choice, but both require long-term follow-up to assure that the dog is actually cured of its infection.

Oslerus rostratus (Nematode)

This is a metastrongylid nematode parasite of felines that occasionally finds its way into the domestic cat. Oslerus rostratus is a large worm that is closely related to Oslerus osleri. It has been reported from cats in the United States, Pacific Islands, Southern Europe, and the Middle East [23-26]. The adult males are about 28-37 mm long, and the adult females are 48-64 mm long. The worms are typically found in the bronchial submucosa. The vulva in the female is located just anterior to the anus. The larvae found in the feces are 335 to 412 micrometer long and have a tail that is similar to the of Oslerus osleri. The life cycle has been described by Gerichter [23] and Klewer [27] who found that the larvae were capable of development in slugs. Seneviratna [25] has shown that the third-stage larvae from slugs are capable of infecting mice that serve as paratenic hosts. Similarly, he found that week-old chickens could be infected as paratenic hosts and that the larvae recovered from these chicks were capable of infecting a cat. Larvae were first observed in the cat 78
days after being given infective-stage larvae. There have been no studies on the signs of infection with this parasite or on effectiveness of different treatments.

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**Oslerus pararostratus (Nematode)**

This is a metastrogyloid nematode parasite that has been described from nodules found in the trachea of a dog in Mexico [28].

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**Eucoleus aerophilus (Nematode)**

This is a trichinelloid nematode that like *Eucoleus boehi* is better known as a capillarid. The worms which are several mm long are found threaded within the mucosa of the trachea, bronchi, and bronchioles (Fig. 7). The "capillarid" eggs are 59 to 83 µm long by 26 to 40 µm wide with a surface bearing a net-like ornamentation (Fig. 8, Fig. 9). The adults are typically seen most commonly in histologic sections. When whole worms are removed the genus can be recognized by the long, spined spicular sheath of the male.

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Figure 7. *Eucoleus aerophilus*. Section through an adult worm in the tracheal mucosa of a cat. - To view this image in full size go to the IVIS website at www.ivis.org . -

Figure 8. *Eucoleus aerophilus*. Egg passed in the feces. - To view this image in full size go to the IVIS website at www.ivis.org . -

Figure 9. *Eucoleus aerophilus*. Surface of egg showing the fine reticulate markings. - To view this image in full size go to the IVIS website at www.ivis.org . -

It is believed that direct ingestion of the egg with an infective larva is the most common route of infection [60]. The prepatent period is between three to five weeks. The eggs deposited by the female within the tracts they have made in the mucosa slowly work their way to the surface. The freed eggs are then coughed up, swallowed, and passed in the feces. Eggs require a period of development in the soil to become infectious. The main clinical signs in the cat and dog are coughing and wheezing due to bronchiale disease. A bronchial pattern may be present on chest radiographs. Levamisole has been successfully used to treat infected cats [61, 62]. Also, levamisole has successfully been used to treat dog [63]. It is expected that long-term fenbendazole therapy would also be effective.

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**Pulmonary Vessels**

**Cytauxzoon felis (Protozoan)**

This is an apicomplexan protozoan parasite of wild felids that occasionally finds its way into domestic cats. This parasite was first reported as a parasite of the domestic cat by Wagner in 1976 [29]. The natural host is the bobcat [30]. Most of the cases have been reported from the southeastern United States. There is a stage which circulates in the blood, the merozoites. This is a small blue-staining parasite within the red-blood cell which has a dark staining nucleus. It is very difficult to distinguish this parasite form *Babesia* spp. which have been reported from cats. Due to the typically fulminant presentation of this infection in the domestic cat, the disease is typically diagnosed at post mortem by the finding of the large schizontongous stages that occur in the
walls of the venous system [31]. These schizonts are commonly found in the venules of the lungs (Fig. 10, Fig. 11).

![Figure 10. *Cytauxzoon felis*. Schizonts developing in vascular system of feline lung in a fatal case. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

![Figure 11. *Cytauxzoon felis*. Schizonts developing in histocytes which are highly increased in size causing vascular plugging. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

The life cycle has been only poorly described, and to a great extent has been modeled after what is known about the related parasites of the genus *Theileria*. Merozoites circulate within red blood cells, and are taken up by a feeding tick [32]. It seems that the gametes undergo development and sexual fusion in the stomach of the tick host, and ultimately produce sporozoites that are found in the salivary glands. Once inoculated into a cat, it is not known where the sporozoites first take up residence, but within a few days, schizonts are found in histiocytes of the veins and venules of the lungs and other tissues. Merozoites appear in the peripheral blood 6 to 8 days after inoculation [33].

The typical presentation is a severely ill cat with signs that included anemia and depression. These signs may be accompanied by fever, dehydration, icterus, splenomegaly, and hepatomegaly. Packed cell volumes decrease markedly, but the reticulocyte counts and mean cell volumes remain normal [33]. Platelets are decreased in number. Lymphocyte and eosinophil counts become decrease near the time of death, about 8 days after infection. Bone marrow aspirates are a good place to recover the large, diagnostic schizonts. Almost all cats die between 9 and 15 days after being infected. Typically, between 1 to 4% of blood cells have organisms, but up to 25% of red blood cells may be parasitized. Attempts have been made to treat experimentally induced cytauxzoonosis with parvaquone (Clexon) and buparvaquone (Butalex); both drugs have been shown to be successful in treating theileriosis in cattle [34]. In the regimen employed, the disease still proved a fatal one. Over a 24-h period, a cat that presented with a 2-day history of lethargy and anorexia became seriously icteric and had dark brown urine [35]. The cat was treated with a 10-day course of enrofloxacin followed by a 5-day course of tetracycline. Organisms were present in the cat after the 10-day course of enrofloxacin, but were not present in blood samples collected 6 and 15 weeks after discharge. It is not known why this cat survived; enrofloxacin is not known to be effective against protozoa.

**Angiostrongylus vasorum** (*Nematode*)

This is a meatastrongyloid nematode parasite of the pulmonary arteries of the dog and other canids. The parasite is most commonly seen in dogs in southern Europe, but it has also been reported from the United Kingdom and northern Europe, South America (Brazil and Colombia), and Uganda in Africa [36-39]. This worm has also been reported in naturally infected animals in the Canadian provinces on the Atlantic coast. The adult females are about 18 to 25 mm long, and the males are 14 to 18 mm long. The spicules of the males are 300 to 400 micrometer long, and the vulva of the female is located just anteriad to the anus. When seen in necropsy (Fig. 12), the worms appear red to dark brown in color with the white ovaries and uteri coiled around the intestine giving the worm a 'barber pole' appearance. The females lay eggs that contain a single cell, and the eggs are carried to the pulmonary capillaries where they lodge and develop. The larvae that hatch from the eggs, penetrate the alveoli, and are carried up the respiratory escalator, are swallowed, and passed in the feces. The larvae of *Angiostrongylus vasorum* are apparently indistinguishable from those of *Aelurostrongylus abstrusus* [12] and have a recognizable kink to the tip of the tail. The larvae passed in the feces are very active larvae which are easy to recover in the feces using a Baermann apparatus. The larva is approximately 310-400 micrometer long and has a characteristic dorsal spine on the tail.

![Figure 12. *Angiostrongylus vasorum*. Lungs of a dog at necropsy showing the dark red worms in the open artery. - To view this image in full size go to the IVIS website at www.ivis.org.](image)
The life cycle of *Angiostrongylus vasorum* has been shown to involve a required snail intermediate host [40-42]. When infective larvae are ingested by a dog, they migrate to the mesenteric lymph nodes where they undergo two molts and become immature adults by five days after infection [43]. These larvae then migrate to the right ventricle and pulmonary artery by days nine and ten postinfection (Fig. 13). The adults are mature by about a month after infection. Larvae appear in the feces about 49 to 57 days after the dog is infected [41, 44]. Following experimental infection, rodents will occasionally maintain larvae in their mesenteric lymph nodes [41], and these rodents could perhaps serve as paratenic hosts. Frogs have recently been shown to possibly act as intermediate hosts for this parasite [45]. Dogs probably maintain the infection for life [37]; an experimentally infected dog shed larvae for five years after infection [42].

![Figure 13. Angiostrongylus vasorum. Histologic section showing transverse sections through several adult worms. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

Older dogs with chronic angiostrongylosis will typically present with clinical signs of gradually progressing pulmonary disease and cardiac failure [37]. These dogs will show signs including depression, stunted growth, weight loss, decreased activity tolerance, coughing, dyspnea, and perhaps edema. Occasionally, chronic infections can be accompanied by asthmatic respiratory distress [46]. On other occasions, chronic infections have been associated with coagulopathies [47]. This condition can be associated with anemia, hemoptysis, melena, and subcutaneous hematomas. A dog with a history of bleeding episodes and a severe regenerative anemia was found to have a coagulopathy that consisted of a consumptive intramuscular process that resembled disseminated intravascular coagulation and later a diagnosis of angiostrongylosis was made [48]. Clinical signs are very rarely associated with acute infections with *Angiostrongylus vasorum*, although pulmonary changes, which can be quite severe, can occur quite soon after the infection is initiated [37, 44]. Signs from acute infections seem to occur mainly in younger dogs that present with dyspnea, coughing fits, bronchopneumonia, and dilation of the right heart, and on rare occasions the acute course can lead to death two weeks after presentation [49]. It is believed that this acute type of presentation is due to the ingestion of a large number of infective larvae on a single occasion. The experimental infection of 4-month-old puppies with 150 larvae induced significant pneumatic changes but little in the way of clinical signs [44]. The first changes were observed in the lungs prior to egg production, and it has been suggested that the worms may elaborate some substance that provoked the observed eosinophilic response associated with nodules of poorly circumscribed foci of interstitial pneumonia and thickened alveolar wall. The most sever changes in the lung were noted to occur at the time of patency. At 60 days postinfection, there were numerous granulomas containing viable and necrotic parasite eggs and irregular areas of fibroplasia that obliterated alveoli and were interpreted as resolving granulomas. At this time in the infection, there are vascular changes that include thrombi and intense proliferation of the vascular walls. Radiographs taken at this time reveal an alveolar pattern associate with the hemorrhage into the alveolar spaces at the time of patency [50]. As the infections progressed, the alveolar patter regressed and left an interstitial pattern. Treatment of dogs infected with *Angiostrongylus vasorum* has been performed using ivermectin at 200 mcg per kg [51, 52]. Fenbendazole at 20 mg per kg once or twice daily for 2 to 3 weeks has also proven successful [51, 53]. The drug that has historically been utilized to treat this infection has been levamisole. The levamisole is given at 7.5 mg per kg for 2 consecutive days, followed by 2 days at 10 mg per kg, and if the infection is not cleared, the regimen is repeated [37]. Problems encountered with levamisole therapy have been problems with dosing due to its apparent unpleasant taste, failure of some infections to respond, temporary neurologic disturbances in some dogs, and anaphylactic reactions that are believed due to large numbers of rapidly dying worms.

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**Toxocara spp. (Nematode)**

*Toxocara canis* and *Toxocara cati* are mainly considered to be of importance because of their potential to cause intestinal disease in their respective canine and feline hosts. Also, both of these worms have importance in that both have the potential to cause visceral larval toxocariasis in humans. Less well recognized is the fact that both of these parasites are capable of causing pulmonary disease in dogs and cats.
Toxocara canis is capable of causing pulmonary disease in both the dog and the cat. In the dog, the larvae of Toxocara canis regularly make a liver-lung migration before the larvae take up residence in either the intestinal tract as adults or within somatic tissues as arrested larvae. As the worms migrate through the lungs, they cause petechial hemorrhages, and larvae are found in the pleural cavity and diaphragm [54]. Also, Toxocara canis larvae have been reported on rare occasions to cause myocarditis with associated pulmonary arterial thrombi in the canine host [55]. In the cat, Toxocara canis has been shown to cause significant changes in the pulmonary arteries [56, 57]. The work of Pankavitch [56] carefully describes how the medial hypertrophy of the pulmonary arteries that occurs with larval migration through the lungs is markedly greater with Toxocara canis than it is with either Toxocara cati and Aelurostrongylus abstrusus. As of this time, there have been no cases where the pulmonary effects of these infections have been diagnosed and treated antemortum.

Figure 14. Toxocara canis. Medial hypertrophy of the pulmonary arteries in a cat infected with Toxocara canis (transverse section stained with Verhoff’s Van Giesen stain). - To view this image in full size go to the IVIS website at www.ivis.org.

Toxocara cati is known to cause pulmonary changes in cats, and it appears that there has been no examination of its effects on the lungs of dogs. The work of Pankavitch [56] showed that the histopathologic effects of the migrating larvae of Toxocara cati were minimal changes in the lungs of cats, especially when compared to the changes induced by Toxocara canis and Aelurostrongylus abstrusus. Jonas et al. [58] found similar results although using other methods. These authors examined the hemodynamic changes in cats experimentally infected with Toxocara cati and Aelurostrongylus abstrusus. Seven of 10 cats with Aelurostrongylus abstrusus had increased pulmonary artery pressures or pulmonary vascular resistance indices. Only 6 of the 16 cats infected with Toxocara cati became moderately hypertensive. Weatherley and Hamilton [59] reported that in kittens inoculated with eggs of Toxocara cati, that there was a cellular inflammatory activity that progressively increased up to 4 weeks after infection. By 6 weeks after infection, there was a gross thickening of the arterial walls with pronounced intimal proliferation. By 8 weeks after infection, there was complete occlusion of some vessels. Thus, it would seem that in the case of Toxocara cati, it is not yet resolved as to the severity of the lesions induced in the lungs of cats by migrating larvae. Also, as with Toxocara canis, there has been no examination of the effects of different treatments on reducing the pathology caused by these migrations.

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