Respiratory System Parasites of the Dog and Cat (Part I): Nasal Mucosa and Sinuses, and Respiratory Parenchyma  (20 Apr 2000)

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This Part I of a review that covers the majority of parasitic disease of the respiratory system of the dog an 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.

Part I: NASAL MUCOSA AND SINUSES

- *Eucoleus boehmi* [nematode]
- *Mammomonogamus* [nematode]
- *Cuterebra* spp. [arthropod]
- *Pneumonyssoides caninum* [arthropod]
- *Linguatula serrata* [arthropod]

Part II: TRACHEA AND BRONCHI

- *Crenosoma vulpis* [nematode]
- *Oslerus osleri* [nematode]
- *Oslerus rostratus* [nematode]
- *Oslerus pararostratus* [nematode]

Part I: RESPIRATORY PARENCHYMA

- *Toxoplasma gondii* [protozoan]
- *Strongyloides* spp. [nematode]
- *Aelurostrongylus abstrusus* [nematode]
- *Bronchostrongylus subcrenatus* [nematode]
- *Troglostrongylus subcrenatus* [nematode]
- *Filaroides hirthi* [nematode]
- *Paragonimus kellicotti* [trematode]

Part II: PULMONARY VESSELS

- *Cytauxzoon felis* [protozoan]
- *Angiostrongylus vasorum* [nematode]
- *Toxocara* spp. [nematode]
- *Eucoleus aerophilus* [nematode]

Nasal Mucosa and Sinuses

*Eucoleus boehmi* (Nematode)

This is a trichinelloid nematode parasite that is in the group of worms better known as the capillarids. The several mm long adult worms live threaded through the mucosa of the nasal sinuses. The adults are about a tenth of a millimeter in diameter, and this causes them to appear as very fine threads when they are removed from the mucosa at necropsy. The eggs were carefully illustrated by Campbell [1]. The eggs can be distinguished from other capillarid eggs found in the feces, because when passed, they have already undergone partial embryonation of the developing larvae, and this development causes the enclosed embryo to be shaped like a peanut still within its shell (Fig. 1). Also, the eggshell surface bears a small series of pits which distinguishes it from the other capillarid eggs passed in the feces of dogs which have shells with striations rather than pits (Fig. 2). Work by Schoning et al. [2] suggests that the eggs of this worm are difficult to recover by nasal swabs although they were recovered using nasal washings.
The life cycle of *Eucoleus boehmi* is not known. Some related species of *Eucoleus*, such as *Eucoleus aerophilus* which is in the lungs of dogs and cats, are found to require an earthworm intermediate host [4]. Other species of *Eucoleus* have direct life cycles. It is difficult to understand how dogs become infected through the ingestion of earthworms, unless that ingestion is accidental.

There have been several cases where this parasite has caused disease. King et al. [4] initially examined a dog that was found positive for this parasite because of a complaint of chronic nasal discharge. Evinger et al. [5] reported that an infected dog was forcefully tapping its nose against the ground, apparently trying to expel the induced nasal discharge from its nose.

Treatment for this infection appears to be ivermectin. Evinger et al. [5] treated a dog with nasal capillariasis with 0.2 mg/kg, a single oral dose. King et al. [4] treated a dog for nasal capillariasis with both ivermectin and febendazole, and although the infections were not cleared, these authors felt that the cause was reinfection of the animals.

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**Mammomonogamus (Nematode)**

This genus is distinguished from the genera such as *Syngamus* in birds by the fact that they are found in mammals and that there are longitudinal ribs on the inner walls of the buccal capsule of those found in mammals [6, 7]. Cases in cats occur in the Caribbean; cases have not been reported from dogs. Human infections with *Mammomonogamus* have been ascribed to *Mammomonogamus nasicola* [8] or *Mammomonogamus laryngeus* [9, 10]. Cases in cats have typically been ascribed to other species such as *Mammomonogamus ierei*. It is possible that *Mammomonogamus ierei* is a synonym of the ruminant parasites, *Mammomonogamus nasicola* and *Mammomonogamus laryngeus*.

The worms live in the nares and nasopharynx. The females are about 20 mm long; with the maximum length reported by Buckley [11] being 23.8 mm. The males are 5 to 6.9 mm long and rather stocky in appearance. The worms are found with the bursa of the male attached at the level of the vulva of the female. There is a large buccal capsule that has eight large teeth at its base. The eggs of *Mammomonogamus ierei* were illustrated by Buckley [11] as being ovoid with a shell that is clear and marked with fine irregular transverse striations. The eggs averaged 49.5 (48 - 52) µm by 92 (84 - 100) µm. The eggs when passed in the feces are typically in a four to six celled stage. Cuadrado et al. [12] reported that the eggs of *Mammomonogamus* were typically larger than those of *Ancylostoma*, had a thicker shell, and that the shell in a salt flotation preparation was typically found with attached debris.

There has not been a complete description of the life cycle of any of the species of *Mammomonogamus*. Species of the related avian genera, *Cyathostoma* and *Syngamus*, have been found to infect birds through the ingestion of their embryonated eggs, free larvae, or the ingestion of earthworm paratenic hosts [13]. Attempts to infect sheep with earthworms infected with *Mammomonogamus nasicola* or with free larvae of this parasite failed to induce infections with this parasite [14]. Human cases have been reported in the USA in individuals within a week after short trips to Jamaica, Martinique, and St. Lucia; this would indicate that the adults are capable of developing within a few days to two weeks [15, 16].

No signs have been reported for infected cats. Cuadrado et al. [12] report that histologically there is evidence of chronic inflammation of the nasopharynx. Humans with this infection have reported had non-productive and sometimes violent coughs [15, 17, 18]. Humans have been treated with mebendazole [10, 18].
**Cuterebra spp. (Arthropod)**

The large maggots that are often observed in the skin of dogs and cats represent the larva of the rodent and rabbit bots. These are dipteran parasites of the western hemisphere with some 34 different species being present in North America [19]. Adult flies lay there eggs on various materials around entrances to rodent burrows, and in response to the passing host, the small first-stage maggot hatches and jumps on board. The larva enters the host through the mouth, nose, eyes, or anus [20, 21]. Once within the rodent host, the larvae remains as first-stage larvae with the nasopharyngeal region [22, 23], and at this time these small transparent larvae can be found at the posterior end of the soft palate and in the nasal passages of experimentally infected mice and rats. Some species appear to make a migration to the area of the trachea, migration though the tracheal wall into the thoracic cavity, followed by migration through the diaphragm into the abdominal cavity prior to their ultimate subcutaneous location [24]. The time from the time of infection, until the larva leaves the warble is between 3 to 8 weeks. The seasonality of the infection is due to the activity of the adult flies that have only a single emergence in late spring in the more temperate parts of the United States. Cats and dogs are probably infected as they hunt, and young kittens and puppies may be infected by larvae carried on the pelage of the mother.

Clinical signs depend mainly on where the larva locates. The usual presentation is to have the larvae discovered in a warble on the cheek, neck, or back; of course, the warbles can appear at other sites (Fig. 3).

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**Pneumonyssoides caninum** *(Arthropod)*

This is the canine nasal mite. This mite was first described from a dog in the United States as *Pneumonyssus caninum*, but this was later reclassified as *Pneumonyssoides caninum* [32, 33]. This mite has been reported on numerous occasions from dogs in the United States [34-36]. This mite has also been reported from dogs from Canada [47], Australia [48], South Africa [49], Japan [50], and Europe [51-56].

Adult mites are about a millimeter long and about one-half millimeter wide (Fig. 5, Fig. 6). On the adults, the
first pair of legs each terminate in a pair of large hooks, while legs two, three, and four each terminate in a sucker armed with a pair of smaller hooks. The other stage which has been described is the six-legged larval stage. It appears that the female is ovoviviparous and that there is no nymphal stage in the life cycle of this parasite. It is believed that dog-to-dog transmission is by the direct transfer of larvae from one infested dog to another.

The clinical sign most typically reported in dogs infested with *Pneumonyssoides caninum* is sneezing. Other clinical signs that have been reported have included facial pruritus, snuffling, and snorting. Occasionally nasal discharge has been reported in dogs with this infestation [38]. Excessive lacrimation has been reported [14]. Also, the infestation has occasionally been associated with central nervous system disturbances [39, 40, 53]. It has been reported once that this mite has also been associated with lung and liver lesions [57], but this observation warrants further confirmation.

Treatment seems to be easily induced by the administration of ivermectin (200 mcg/kg) administered subcutaneously [36, 43, 47]. Another successful treatment that has been used is the administration of dichlorvos vapors [50]. Treatments that have failed to induce cures have included the infusion of rotenone into the nasal passages [46] and the administration of phenclorphos orally at 50 mg/kg body weight [56].

**Linguatula serrata (Arthropod)**

The pentastomid parasites are now considered to actually represent a group of specialized crustacean-like arthropods. The adults have a worm-like body and a mouth with a pair of hooks on each side. The adults are all associated with the respiratory system of the final host, being found in the air sacks of birds, the lungs of reptiles, and the nasal turbinates of dogs. These are large parasites, the adult female is about 8 to 10 cm long and about 1 cm in diameter. The male is about 2 cm long. The body appears superficially annulated, and the worms tend to be tan to brown in color. Infected dogs have been reported from North Africa, the Middle East, Turkey, Greece, east-central Europe, Brazil, Chile, and Argentina, Tanzania, and even Australia. Diagnosis can be performed by finding the eggs which are yellowish oval objects, about 80 $\mu$m, surrounded by a bladderlike envelope and containing a four-legged larva, in the nasal secretions or around the nares. The life cycle of *Linguatula serrata* involves a required intermediate host. The eggs that are passed by the female contain a four-legged larva. According to Hobmaier and Hobmaier [58], the eggs do not appear in the feces of the dog but instead are found in the nasal secretions. The possibility that the eggs are not shed in the feces should receive additional verification because in other hosts, the eggs are passed in the feces. If the egg is ingested by a mammalian intermediate host, e.g., rodent, ruminant, or primate, the larvae hatch, and migrate to the liver, lungs, lymph nodes, and peritoneal mesenteries where they develop into nymphs that can be several cm in length and which are encased in a host tissue reaction. It is suspected that most dogs obtain their infections by the ingestion of sheep offal. When dogs ingest infected tissues, the nymphs migrate up the back of the throat into the nasal turbinates. Once swallowed, the nymphs do not migrate back up the esophagus. The prepatent period is about six months, and it is believed that the adult worms live about two years. The early signs of infection with *Linguatula serrata* are sneezing and slight nasal discharge. The parasites become large, lie in the recesses of the nasal turbinates, and attach themselves firmly to the mucous membranes with their four hook. The adults apparently feed on respiratory mucosal cells and blood. When fully grown, the parasites are capable of causing impairment of respiration. In more chronic infections, the nasal discharge will increase in volume, and there is the possibility of obstruction of the nasal passages. Humans have on rare occasions been host to the same stage that is found in the dog. Humans present with signs that would be suspected from a several cm long larva migrating from the mouth into the nasal sinuses,
e.g., throat discomfort, coughing, sneezing, dysphagia, and vomiting.

Treatment of infections with *Linguatula serrata* is typically by the removal of the organisms from the sinuses by physical means. In reindeer, ivermectin has been shown to be effective in killing the deer-sinus pentastomid, *Linguatula arctica* [59].

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**Respiratory Parenchyma**

*Toxoplasma gondii* (Protozoan)

This protozoan is typically considered a parasite that causes little or no disease in the cat (Fig. 7), and when cats serve only as the host for the enteric stages of this parasite, they will usually show no signs of infection while they pass the oocyst stages into the environment. Cats can, however, develop systemic toxoplasmosis, and this very often presents as respiratory disease. On rare occasions, dogs also will develop pneumonia forms of toxoplasmosis. In both dogs and cats, systemic toxoplasmosis often develops in the presence of concomitant viral infections or some other form of immunosuppression. However, agents other than *Toxoplasma gondii* have not always been identified in affected animals, and experimental studies would indicate that in some situations, cats may develop systemic infections with this parasite without being infected with another pathogen.

![Figure 7. Toxoplasma gondii. Bradyzoites in a cyst in the lung of a cat. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

In dogs, pneumonic toxoplasmosis is a rare presentation. A typical presentation is a dog that is one year of age with pneumonia and concomitant distemper infection [60]. In these cases, histological lesions of congestion are found in the lungs of these animals [60, 61]. In other cases, there does not seem to be an underlying infection when dogs develop disease [62]. It appears that many cases that were once considered to be toxoplasmosis in dogs are actually neosporosis. So at this time, it is difficult to ascertain which is the offending agent in many cases without biopsies, however, it appears that neosporosis seldom presents as pneumonic disease.

Disseminated toxoplasmosis has been known to occur in cats for a long time. Olafson and Monlux [63] was the first to identify the organisms in the lungs of a cat at necropsy. Meier et al. [64] describe acute and subacute forms of toxoplasmosis in cats that presented initially as signs of inappetence, lethargy, weight loss and fever, followed by the development of severe dyspnea. Hirth and Nielson [65] reported that the most common finding at necropsy in affected cats was an interstitial pneumonia with foci of necrosis and a fibrinous exudate. Feeney et al. [66] describe pneumonic toxoplasmosis in a one-year-oldcat inn Minnesota that presented with progressivdyspnea, depression, and anorexia that die in spite of symptomatic treatment. In this cat, the radiographic signs were diffuse, symmetrical, homogenous opacities of the interstitium with alveolar coalescence; the authors note that the signs observed in this cat were in contrast to the usual patchy patterns of alveolar densities observed in cats with pneumonic toxoplasmosis. Dubey and Carpenter [67] examined tissues from 100 cats that had been verified by histology to have systemic toxoplasmosis, these authors felt that 36 of these 100 cases represented cases of actual generalized toxoplasmosis. Presenting signs in these cats includedyspnea, polypnea, and abdominal discomfort. The primary lesions were pulmonary lesions which occurred in 72% of the cats with generalized disease. Of the 100 cats initially examined 77% of the 86 lung tissue samples that were available had organisms present. Dubey and Carpenter [68] described neonatal toxoplasmosis in 5 litters of kittens. In one litter the Siamese queen died of generalizetoxoplasmosissi, and her kittens died or were killed 20 to 28 days later. In the second litter, two of three Abyssinian kittens died of toxoplasmosis about 4 months after birth. In the third litter, an Aysinnian kitten delivered by caesarean section, died 19 days after the delivery from toxoplasmic pneumonia and hepatitis. In the fourth litter, three kittens were found to be shedding oocysts in their feces and organisms were identified in tissues of one of these kittern. In the fifth litter, one 3-week-old kitten of the four littermates became ill, oocysts were found in its feces and organisms were found in its tissues at necropsy.

Acute toxoplasmosis has been examined in experimentally infected cat, kittens, and neonatally infected kittens. In cats, pneumonitis was sever and sometimes fatal early in the induced infections [69]. In the lungs of
these cats examined in the first few days after infection, the lesions were represented by randomly distributed infiltrated of neutrophils and mononuclear cells into the alveolar walls. In cats that were examined later in the course of the disease, there was diffuse alveolar necrosis, pneumocytic hyperplasia, and fibrillar exudates in the alveoli. Organisms were found in fibroblasts, macrophages, Type I and Type II pneumocytes, bronchiolar epithelial cells, bronchiolar smooth muscle cells, endothelial cells, neutrophils, eosinophils, and circulating monocytes (Fig. 8). When newborn kittens were infected with cysts from muscle [70], they tended to develop enteritis, hepatitis, myocarditis, myositis, pneumonitis, and encephalitis and were usually moribund by the 9th day after infection. Older kittens, tended not to develop pneumatic signs and usually survived, while adult cats tended to remain asymptomatic. The inoculation of cysts subcutaneously into newborn kittens caused them to die of acute toxoplasmosis with severe pneumonia, myocarditis, encephalitis and hepatitis. In nine cats infected orally with Toxoplasma gondii, 6 with tissue cysts and 3 with oocysts, none showed any clinical signs of infection, but organisms were later recovered from the lungs of all nine animals [71]. Three cats inoculated intraperitoneally with tissue cysts develop fatal infections with signs of fever, anorexia, lethargy, and dyspnea with large amounts of fluid accumulation in the pleural and peritoneal cavities. Dubey et al. [72] reported on the experimental inoculation of five pregnant queens with tissue cysts. From these queens, 22 live and 3 dead kittens were born 16 to 31 days after the queens were infected. Of the 22 live kittens, 21 died or were killed by 29 days after birth. In all kittens, there were lesions of proliferative interstitial pneumonia (Fig. 9).

Pneumonic toxoplasmosis often develops in cats that have concomitant infections with other pathogens. Work by Davidson et al. [73] showed that cats with FIV that were given intraperitoneal inoculations of Toxoplasma gondii tachyzoites developed generalized severe toxoplasmosis with the major lesion at necropsy being interstitial pneumonia and hepatic necrosis. Only transient clinical signs developed in cats given tachyzoites which had not previously been infected with FIV.

***Strongyloides spp. (Nematode)***

Strongyloides stercoralis can on occasion cause significant pulmonary changes in dogs and cats (Fig. 10). Typically, these changes appear following a course of some form of long-term immunosuppression. In these dogs, it appears that internal autoinfection occurs and the infective stage larvae migrate to various ectopic sites within the dog [74, 75]. In dogs receiving prednisolone treatments superimposed on chronic Strongyloides stercoralis infections, diffuse deposits of calcium phosphate have been observed [76]. It has also been noted that a dog without any marked underlying immunosuppressive problem may have radiographs showing evidence of diffuse interstitial infiltrates that have been verified upon necropsy [77]. In cats infected with Strongyloides felis, interstitial pneumonia and focal pulmonary granulomas have been found to occur in association with this infection [78].
Aelurostrongylus abstrusus (Nematode)
This is a metastrongyloid nematode parasite of the cat around the world [79-85]. The adult females are 9 to 10 mm long with a vulval opening which, unlike most nematodes, occurs near the anus. The males are smaller, being 4 to 6 mm long, and have a small bursa. The worms are less than a millimeter wide and have a dark brown to black appearance when observed in fresh tissue. Due to the small worms being deeply within the terminal respiratory bronchioles and alveolar ducts, it is difficult to remove entire worms by dissection. The females lay eggs that contain a single cell when laid and which embryonate within the alveolar ducts and the surrounding alveoli (Fig. 11). The larvae hatch from the eggs, are carried up the ciliary escalator, swallowed, and pass in the feces (Fig. 12). Eggs can sometimes be found in tracheal wash or sputum samples (Fig. 13). The larvae of Aelurostrongylus abstrusus are quite active larvae which are easy to recover in the feces using a Baermann apparatus. The larva is approximately 360-390 µm long and has a characteristic dorsal spine on the tail.

The life cycle of Aelurostrongylus abstrusus has been shown to involve a required snail intermediate host [86]. It has also been shown that mice which ingest the infected snail can serve as a paratenic host [87]. Hobmaier and Hobmaier [88] also showed that if the larvae from snails were fed to frogs, toads, snakes, lizards, ducklings, chickens, or sparrows that the larvae could later be recovered from their tissues. Thus, it seems highly possible that cats become infected typically by the ingestion of infected mice or birds. Stockdale [89] found that the females began to lay eggs as early as 25 days postinfection; larvae have been found in the feces after 39 days of being infected with third-stage larvae [90]. Aelurostrongylus abstrusus is known to cause severe pulmonary disease with heavy infections [89]. Infections of cats with 100 larvae showed that the earliest radiographic changes were observed 2 weeks after infection [91]. The most severe disease was noted 5 to 15 weeks after infection and presented as alveolar disease (Fig. 14). Examination of experimentally infected cats followed for up to a year after infection revealed that there was neither pulmonary hypertension or associated right ventricular disease [92]. Examination of the pulmonary arteries in experimentally infected kittens has revealed that there is disruption of the vascular endothelium and proliferation of the endothelial cells, and as early as 10 days after infection, there is disruption of the internal elastic lamina. Hypertrophy and hyperplasia of the medial and intimal walls of the pulmonary vessels caused complete occlusion of many of the vessels by 24 weeks after infection [84].

Mild infections often present with only minimal signs, however, heavy infections can cause severe bronchopneumonia with cats having rapid, open-mouthed abdominal breathing [93]. A retrospective study of 312 cases of cats with eosinophilia revealed that 2% of the cases were infected with Aelurostrongylus abstrusus, while the majority of cases, 20.5%, had eosinophilia as a result of flea-bite allergy dermatitis [82]. In cats infected with Aelurostrongylus abstrusus, treatment with fenbendazole at 55 mg per kg daily for 21 days [93] and at 20 mg per kg daily for 5 days followed by a second 5 day treatment after a five day hiatus has
been reported to be successful [94]. Treatment of 15 cats experimentally infected with *Aelurostrongylus abstrusus* with fenbendazole at 50 mg per kg per day for three days stopped the shedding of larvae in the feces by 14 days after treatment, but a few days later, the larvae reappeared in small numbers in the feces of the infected cats [95]. Published reports on the efficacy of ivermectin in these infections has not produced conclusive results of the efficacy in treating cats with this infection [96]; however, there is a single report of a treatment with 200 (g per kilogram followed by a second treatment with 400 (g per kilogram clearing a cat of its infection with *Aelurostrongylus abstrusus* [97].

**Bronchostrongylus subcrenatus** (Nematode)
This is a metastrongyloid nematode parasite of the lungs of felids that is related to *Aelurostrongylus abstrusus* and which was originally reported from a leopard in the Congo. This has been reported on a single occasion from a cat in Africa, Blantyre, Nyasaland [98]. The males of this species have long slender spicules and the vulva from the female is posterior to the middle of the body rather than near the anus as in *Aelurostrongylus*. The adults are 10 to 23 mm long; about twice the length of the adults of *Aelurostrongylus abstrusus*. The life cycle of a related parasite *Bronchostrongylus brevoir* was described by Gerichter [90]. The adults are in *Felis ocreata* and *Catolynx chaus*. First-stage larvae in the feces enter a suitable mollusk, and infective larvae are present in about eight days. The patent period for this other species was found to be 28 days in a kitten fed snails containing infective larvae.

**Troglostrongylus subcrenatus** (Nematode)
This is a metastrongyloid nematode parasite of the lungs of felids that is similar to *Aelurostrongylus*. As with *Bronchostrongylus sucrenatus*, the male has very long spicules and the vulva of the female is posterior to midbody rather than near the anus. The adults are about a cm long. The life cycle of the related species, *Troglostrongylus brevoir*, which lives in the bronchi of wild felines around the Dead Sea has been shown to utilize gastropod intermediate hosts [90]. The feeding of infective larvae to domestic kittens produces patent infections in about a month.

**Filaroides hirthi** (Nematode)
These are very small metastrongyloid nematodes that are deeply buried in the parenchyma of the lungs. The males and females are very small, and it is almost impossible to remove the worms from the tissue at necropsy. The stage passed in the feces is a first-stage larva (Fig. 15). Zinc-sulfate was found to be 100 times more efficient than the Baermann apparatus in the recovery of the larvae from feces [90]. This parasite was first recognized as a parasite of colony Beagle dogs [100], and it continues to remain a significant pathogen in such situations as evidenced by the report of 98% of research Beagles having lungworm-associated lesions [101].

![Figure 15. Filaroides hirthi. First-stage larva passed in the feces of dogs. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

The life cycle of *Filaroides hirthi* has been shown to be direct [90, 102-105]. Dogs are probably commonly infected as puppies sometime during the nursing period. After the larvae are ingested, the rapidly make there way to the lungs through the hepatic-portal or mesenteric lymph system. After reaching the lungs, there are four rapid molts with adults being present after nine days. After five weeks, larvae will appear in the feces of the infected dog. Larvae are capable of persisting within the mesenteric lymph nodes for extended period which was considered to be evidence of potential autoinfection [105]. This has since been confirmed by the occurrence of hyperinfection in exogenously immunosuppressed animals or in animals with endogenous
immunosuppression caused by hyperadrenocorticism [106, 107]. A case of massive infection was diagnosed at necropsy in a dog that had been receiving corticosteroid therapy for arthritis [108]. The signs of a light infection with *Filaroides hirthi* is a nonproductive cough. Signs in a young Chihuahua that presented to an animal clinic were rapid breathing and an unproductive cough [109]. Other signs that have been reported include dyspnea and exercise intolerance in a King Charles Spaniel, "kennel cough" in a Yorkshire Terrier, and nonproductive coughing in a male and a female Yorkshire Terrier that was worse in the female at night [110-112]. The changes in the lungs vary from small foci of granulomatous reaction to tumor-like lesions [101]. Thoracic radiographs show diffuse interstitial lung opacities and mixed alveolar patterns with consolidation [110, 112]. Radiographs of experimentally infected dogs revealed that at five weeks after infection there were linear and miliary infiltrates throughout the lung lobes (Fig. 16). After albendazole treatment of some of these experimentally infected dogs, the treated dogs developed lesions that were more severe in the untreated dogs [113].

![Figure 16. Filaroides hirthi. Radiograph of lung of dog showing milirtary infiltrates. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

Treatment has been most typically been by the administration of albendazole at 25 mg per kg twice daily for 5 days with a second treatment two to for weeks later [114]. Drugs that were initially tried without effect included piperazine, dichlorvos, thiabendazole, dithiazine iodide, and levamisole [104]. A Chihuahua was treated with fenbendazole at 50 mg per kg and prednisolone at 1.25 mg per kg for 14 days, and apparently cured of its infection [109]. In another case fenbendazole at 50 mg per kg was administered to a Yorkshire Terrier without apparent success, but treatment with ivermectin at 50 µg per kg greatly reduced the larval counts in the feces [111].

**Paragonimus kellicotti** (Trematode)

This is a trematode of the family *Troglotreatidae*. This trematode occurs in the lungs of cats and dogs in the Eastern United States, mainly in the areas of the drainage system of the Mississippi River and its tributaries. The natural hosts of the adults of this parasite are various mustellids that live within this area. The adult flukes are about one-quarter inch long and live within cysts within the lung parenchyma (Fig. 17, Fig. 18). There are usually two or more trematodes per cyst, but on occasion, cysts will be found that only contain a single worm (Fig. 19). The eggs of the trematodes, are deposited by the hermaphroditic trematodes into the lung tissue where they are coughed up and swallowed to be passed in the feces. When the eggs are passes, they are in a single celled stage.

![Figure 17. Paragonimus kellicotti. Live adult recovered from the lung of a cat. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

![Figure 18. Paragonimus kellicotti. Live adult recovered from the lung of a cat. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

![Figure 19. Paragonimus kellicotti. Section through a nodule showing that there are two adult flukes present within the nodule. - To view this image in full size go to the IVIS website at www.ivis.org.](image)

The eggs have a thick brown shell, a distinct operculum, and occasionally a knob on the abopercular end (Fig. 20). If the eggs enter fresh water, they begin to develop and produce a ciliated miracidium. The miracidium hatches and seeks out a young snail host, *Pomatiopsis lapidaria*. 
After undergoing asexual multiplication in the snail, the cercarial stage is produced. The cercariae leave the snail, penetrate the shell of a crayfish, and encyst in an area near the heart of this crustacean. Within the crayfish, the cercaria forms a cyst wall and becomes a metacercaria. When the crayfish is ingested by an appropriate final host, the excised metacercariae rapidly penetrate the intestinal tract and enter the peritoneal cavity [115]. The young flukes migrate about in the peritoneal cavity for a week to 10 days and then enter the pleural cavity through the diaphragm. The worms begin to enter the lungs around two weeks after infection. By three weeks after infection, there will typically be pairs of flukes found together in the lung tissue. Dogs and cats typically become infected by eating metacercariae in crayfish, however, it has been shown that rats can serve as paratenic hosts of *Paragonimus kellicotti* [116]; and it is possible that cats are infected by the ingestion of infected rodents. It has also been postulated due to the appearance of paragonimiasis in kittens of infected queens when only a few months of age, that transmammary or transplacental transmission of the juvenile flukes might be possible [117]; however, examination of 4 kittens born 67 days after the infection of the queen revealed no stages of *Paragonimus kellicotti* in the tissues [118]. Dogs and cats will begin to shed eggs 5 to 7 weeks after infection. Clinical signs are typically quite mild, e.g., occasional coughing, although bouts of paroxysmal coughing and dyspnea due to pneumothorax from migrating flukes has been described [119]. High numbers of eosinophils appear in the blood, especially 3 weeks after infection which is when the worms would first enter the lungs. Radiographs of early lesions will reveal indistinct nodular densities containing small air cavities and having irregular, sharply defined margins, older cysts are typical air-filled pneumatoeysts (Fig. 21). However, the presentation my often be simply ill-defined interstitial nodular densities [120, 121]. Occasionally, sudden death may occur from the rupture of the pleural space [122].

The treatment of choice is likely to be praziquantel (25 mg/kg orally every 8 hours for a total dose of 150 mg/kg [123]. Experimentally infected cats have been treated with praziquantel at 23 mg/kg three times each day for 3 days [118]. Albendazole has also been used to treat experimentally infected cats (50 to 100 mg/kg for 14 to 21 days) and naturally infected cats (25 mg/kg twice a day for 11 to 24 days) [118, 124].

*Paragonimus kellicotti* has traveled with dogs outside of the United States, and a case has been reported in a dog that died suddenly in Israel [122]. The concern is that this fluke could establish itself in either the Middle East or Europe. Various mustellids and wild canids are capable of cycling the disease in the wild, and if adequate snail hosts are present, this is a real possibility if infected dogs are introduced into an area. There are many other species of *Paragonimus* found around the world. In Asia, are the species *P. westermani*, *P. pulmonalis*, *P. miyazakii*, *P. heterotremus*, *P. siamensis*, *P. skrjabini*, and *P. ohirai*. Other species in the Americas include *P. mexicanus*, *P. inca*, *P. caliensis*, and *P. amazonicus*. The species in Africa are *P. africanus* and *P. uterobilateralis*. These species utilize fresh-water crabs and crayfish as the intermediate host. Most of these species are capable of infecting dogs and cat hosts, and dogs and cats have been found infected naturally with a number of these species.

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