The feline lungworm, *Aelurostrongylus abstrusus*, is often a difficult diagnosis, and it is probably very underdiagnosed in cats around the country. Also, many forget that the *Cuterebra* larva that appears in the warble in the neck or cheek of a cat has migrated through the body of the cat for several weeks to more than a month before it appears at its subcutaneous location. California and the western US has many more species of *Cuterebra* in rodents and rabbits than the eastern US, but less cases in dogs and cats, and it is unclear if this is because these species do not do well in domestic animals or are less commonly diagnosed when in aberrant locations.

*Aelurostrongylus abstrusus*

*Aelurostrongylus abstrusus* is the common lungworm of the cat around the world. It is not the only lungworm of cats, but by far the most common. Other fairly uncommon feline lungworms include *Troglostrongylus brevior*, *Troglostrongylus subcrenatus*, *Troglostrongylus wilsoni*, and *Oslerus rostratus*. These other lungworms are larger and live in the trachea and bronchi, whereas *A. abstrusus* lives threaded through the terminal bronchioles and alveolar ducts. Due to the other worms being larger and more robust, you will often see images of the whole adult worms in publications. However, in the case of *A. abstrusus* the worms are smaller and threaded through the tissue, and it is difficult or impossible to often collect whole worms at necropsy, so one almost never sees an image of an intact worm. The adults of *A. abstrusus* (females 9 to 10 mm long, males 4 to 6 mm long) are dark reddish-brown to black in color, and can be seen at necropsy within the lesions made on the surface of the feline lung.

All of these lungworms of the cat require a snail or slug for the larvae passed in the feces of the cat to develop to the infective stage. The life cycle that has been best described is that of *A. abstrusus*. In the *A. abstrusus* life cycle, the females lay eggs that contain a single cell when laid and which embryonate within the alveolar ducts and the surrounding alveoli. First-stage larvae hatch from the eggs, are carried up the ciliary escalator, swallowed, and passed in the feces. Larvae are quite active and easy to recover in the feces using a Baermann funnel apparatus. The larva is approximately 360-390 μm long and has a characteristic dorsal spine on the tail. The larvae will not develop unless they are ingested by a snail or slug where they mature to third-stage larvae infective to paratenic hosts or directly to the cats; the temperature determines how fast the larvae mature in the snail with no development at 8°C and development to the infective stage in 18 or 19 days at 30°C. Once snails are infected, it has been shown that the larvae within can remain infective for at least two years. Paratenic hosts have been shown experimentally to include frogs, toads, snakes, lizards, ducklings, chickens, sparrows, and mice, and it is believed that most cats are infected by the ingestion of mice and birds. In the mice, the larvae were found throughout the intestinal tract embedded in the submucosa, subserosa, in the neighboring mesentery, in the pleural cavity, in the body cavity, and in the serous coverings of the pericardium, lungs, diaphragm, and especially in the fatty connective tissues of ligaments.

After a cat ingests infective third-stage larvae, the worms migrate directly to the lungs via the peritoneal and thoracic cavities, and are found within the lung tissue within 24 hours of infection. The worms then begin to develop, and the females begin to lay eggs as early as 25 days after infection. The first larvae will appear in the feces of an infected cat as early as 28 days after infection, i.e., the prepatent period is as short as 28 days. Cats seem to shed for a few months, with peak larval production being between 2 to 4 months after infection, but some cats will shed larvae for up to a year. An infection that clears does not preclude a cat being infected a second time, although during the second infection, there is often a longer lag between infection and the beginning of larval production (Ribeiro and Lima, 2001).

The prevalence of *A. abstrusus* in cats can be quite high. A necropsy survey of 126 cats in upstate NY published in 1954 found 15.1% of the cats infected (Baughn and Bliznick, 1954). A survey of 108 shelter cats in Florida by Baermann funnel and necropsy in 1988 found 20 infected with *A. abstrusus* (Willard et al, 1988). Using centrifugal flotation, of 1322 fecal samples from shelter cats in NY, 6.2% were found infected with *A. abstrusus* (Lucio-Forster and Bowman, 2011), but a study in Connecticut using stationary flotation found only 0.2% of 450 shelter and client-owned cats infected (Rembiesa and Richardson, 2003). Through the years it has been reported from throughout the United States, being found in 1.1% of 1400 cats necropsied in New Jersey, 4% of 107 cats in Hawaii, and has also been reported from AL, CA, CT, FL, GA, HI, MD, NJ, NY, SC, TN, TX, VA, WA, PR. It was found in 2% of 520 cats in San Francisco in 1935 (Hobmaier & Hobmaier, 1935). It has also been reported from Europe, Asia, Africa, South America, and Australia. This is probably one of the most unrecognized causes of respiratory disease in cats around the world.

Mild infections often present with only minimal signs, however, heavy infections can cause severe bronchopneumonia with cats having rapid, open-mouthed abdominal breathing (Vig and Murray, 1986). A retrospective study of 312 cases of cats with eosinophilia revealed that 2% of the cases were infected with *A. abstrusus*, while the majority of cases, 20.5%, had eosinophilia as a
result of flea-bite allergy dermatitis (Center et al., 1990). Hamilton (1963) reported on one cat that was presumed to have died as a result of its lungworm infection; the 6-month-old cat developed signs of respiratory disease when three-months-old and the disease persisted. During this three month period, the cat was observed to be coughing and sneezing with a mucopurulent discharge. As the disease progressed, the cat became dyspneic, anorexic, emaciated, and with hydrothorax.

Heavy infections can cause severe pulmonary disease with heavy infections (Stockdale, 1970). Cats infected with 100 larvae showed early radiographic changes 2 weeks after infection (Mahaffey, 1979). The most severe disease was noted 5 to 15 weeks after infection and presented as alveolar disease. In 1980, 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 (Rawlings et al., 1980); however, recent work (see below) suggests that this is not the case. Pulmonary arteries in experimentally infected kittens show 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 (Naylor et al., 1984).

The clinical effects of A. abstrusus infection are manifest. Cats that are infected have blood gas changes suggesting hypoventilation and respiratory acidosis that may not resolve until 60 days after treatment (Yildiz et al, 2011). Radiographs typically reveal a misted pattern of alveolar, bronchial, interstitial, and vascular disease, with bronchial disease being most common, and alveolar disease was more common in cats with clinical respiratory signs (Losonsky et al, 1978). Following cats with induced infections with 100 larvae revealed the earliest radiographic changes at 2 weeks after infection, and the most severe changes with alveolar disease was 5 to 15 weeks after infection. As the alveolar patten resolved, the interstitial and bronchial patterns became more obvious. Computed tomography has now been used in cats and has shown that affected cats have significant lung disease, but that it cannot be used to make a specific diagnosis at least with the two cats that were examined (Payo-Puente et al, 2005).

Medial hypertrophy of the pulmonary arteries is a common lesion seen in cats upon the examination of tissues at necropsy (Stunzi et al., 1966); often the changes will progress to nodular disease (Dubey et al, 1968). It has also been shown that the right ventricles of chronically infected cats are increased in weight that is believed to be a result of increased work load due to chronic interstitial pneumonia (Swerczek, 1969). Examination of the development of these lesions in experimentally infection kittens from 10 days to 24 weeks after experimental infection has shown that there was progressive disruption of the vascular endothelium resulting in vacuolation and later proliferation of the endothelial cells (Naylor et al., 1984). Eosinophils were observed migrating through the artery walls as early as 10 days after infection, resulting in disruption of the internal elastic laminae and contributing to the disruption of the endothelium. Hypertrophy and hyperplasia of the smooth muscle cells of the media were evident and this, together with intimal proliferation, resulted in complete occlusion of the lumen of many of the vessels by 24 weeks after infection. Seven of 10 cats with experimental A. abstrusus infections were shown to have pulmonary artery pressures or pulmonary vascular resistance indices above those of normal animals (Jonas et al., 1972). Recently, such vascular changes were noted to be important relative to feline anesthesia-associated deaths in a spay-neuter program (Gerdin et al., 2011). In 33% of 54 such cases, significant gross and/or microscopic pre-existing disease, including pulmonary, cardiac, and systemic disease, was detected. Pulmonary disease was most frequently diagnosed (24% of cases), including 5 cases (9% of cases) of A. abstrusus infection). Heart disease, including two cases of hypertrophic cardiomyopathy, was less frequent (11% of cases).

A recent report on two 10-week old kittens presented with dyspnea that worsened over the next two weeks with the development of heart murmurs (Dirven et al., 2012). Examination of one kitten by echocardiography showed severe eccentric hypertrophy of the right ventricle and a moderately dilated right atrium, and color Doppler echocardiography showed a large systolic regurgitant jet originating from the tricuspid valve. The kitten was placed in an oxygen cage, but died, and the necropsy revealed severe A. abstrusus infection. The second cat was examined and revealed a respiratory rate of 50 BPM with an abdominal breathing pattern. Thoracic auscultation revealed end-inspiratory crackles and a grade III/IV systolic heart murmur with it point of maximum intensity on the right hemithorax. Radiographs showed a complex pulmonary pattern throughout the lung fields, predominantly characterized by marked bronchial and interstitial changes but also some alveolar infiltrates. The heart showed right-sided cardiomegaly; echocardiography revealed moderate to severe right ventricular dilation and a moderately enlarged right atrium compared to the left atrium. Right ventricular diameter in diastole was 7.7 mm (average reference value: 2.6 mm, range: 2.0–3.2 mm); left atrial dimensions were considered normal. There was pulmonary artery dilatation. Color Doppler echocardiography showed a large systolic tricuspid regurgitation. Mild pulmonic valve insufficiency was also noted. These findings were interpreted as mild systolic pulmonary hypertension. The cat was treated with milbemycin oxime and praziquantel three times at two-week intervals, and after 6 weeks, larvae were no longer present in the feces. The resolution of the heart murmur and echocardiographic abnormalities with treatment of A. abstrusus, suggest that pulmonary hypertension was associated with A. abstrusus infection. Even when the kitten was 36 weeks old, right-sided cardiomegaly was still apparent although reduced.

The diagnosis of infection with A. abstrusus is best performed with a Baermann funnel on fresh feces. Centrifugal flotation using zinc sulfate is probably next best, and as seen above with the data from Connecticut, stationary flotation is probably not very sensitive.
Recently, a nested PCR was developed that gave excellent sensitivity and specificity using feces or pharyngeal swabs (Traversa et al., 2008); the test unfortunately is not readily available to the public.

There are no approved treatments for A. abstrusus in cats in the United States. Products that are approved for treating cats for other helminths that have been shown to have efficacy include: a single topical treatment with Profender (emodepside and praziquantel) producing an efficacy based on larval reductions 82% after treatment (Traversa et al., 2009a) and Advantage Multi for Cats (moxidectin and imidacloprid) producing larval reductions of 100% 28 days after treatment (Traversa et al., 2009b). The treatment of cats with fenbendazole (Panacur) for 3 days resulted in an efficacy of 99.29% (Traversa et al., 2009a,b).

**Cuterebra spp.**

Cuterebra spp. are included here because most veterinarians seem to forget that this parasite causes respiratory disease. In human beings, there have been three reports of tracheopulmonary myiasis due to Cuterebra (Cornet et al., 2003). One was in a lady who was from Florida and coughed up bloody sputum a mature third-stage maggot after having flown to Paris on holiday; a larva that was coughed up in Ontario that was a second or early third-stage maggot, and a case from Oregon where a person coughed up an early second-instar larva. They have been found in similar locations in cats (Bordelon et al., 2009).

For the common bot fly larvae in veterinary medicine, the large third-stage larva, the bot, comes out of the host and digs its way into the soil to pupate. The biology differs however as to what goes on with the different developing eggs and larvae that come out of the female fly. The disease that occurs in cats that is caused by Cuterebra is due to the biology of the early larvae in its host. The biology of Cuterebra is different from that of the larvae of Oestrus of sheep, Hypoderma of cattle, Gasterophilus of horses, and Dermatobia of South American mammals. Again these differences are of importance biologically, but also because the biology is related to the disease induced.

The larvae of Cuterebra cannot penetrate the skin. The larvae of Cuterebra migrate around inside their hosts before they hypoderms to form their warble. The female Cuterebra lays her eggs on sticks and blades of grass along rodent and rabbit runs or around the openings to rodent and rabbit burrows. The eggs hatch spontaneously when warm potential hosts pass by. The maggots are sticky and get onto the host’s hair, then they crawl around on the host until they find an orifice, and they enter the orifice – it does not matter which orifice. Researchers have placed the larvae of the bots into all the different orifices of mice, but it does not change where they ultimately migrate after they enter the host. Whether the young maggot enters via the mouth or the anus (or any other orifice), it makes its way ultimately to an area around the tracheal, then after a few days in this location, it will begin to migrate to its site of warble development. If it is headed for the inguinal region, it will migrate from the area around the trachea through the diaphragm to the inguinal region of the chipmunk to form its warble. The species that commonly occur in rodents seem to spend 2 weeks migrating around before they get to the warble site, and the ones in rabbits tend to spend 3 weeks or so migrating around before they get to the warble site. When the maggot has completed its growth in the warble, it will drop to the ground to burrow its way into the soil to pupate.

So, in the cat, it seems that sometimes the larvae get lost (Glass et al, 1998; Williams et al., 1998; Wyman et al., 2004). Perhaps when the larvae enter the nose, they make the mistake of directly entering the head through the cribriform plate. This seems to fit tracks we have seen on CT scans of cats with neurologic cuterebriasis. However, when they are found at T5 or T6 in the spinal cord, they probably went there, like Hypoderma, from the tracheal area around the lungs. So, the thing to remember is that when a bot in its warble is found on the chest, under the tail, between the legs, or on the cheek of a cat, the maggot was a lot of different places before it arrived at the site of development.

Cuterebriasis may be preventable. It is possible that the regular use of heartworm preventives might kill the migrating larvae in a host. It is possible that flea and tick products applied to the surface of cats might kill the small maggot while it is crawling about the surface of a cat looking for an orifice to enter. It is possible that lufenuron injectable might prevent the maggots within the cat from being able to develop. These are all possibilities, but completely speculative. There do seem to be many fewer cases of cuterebriasis in dogs these days. Have dogs changed their behavior, or are we doing something without knowing it that is protecting dogs from infection.

**Summary**

These are two parasites that can cause severe respiratory disease in cats and often may go unnoticed. Also, it is likely that routine preventives administered to cats may prevent or treat infections such that disease does not occur. They seem two very good reasons to keep cats indoors or to insure they are on preventive therapy if they do go outside.

**References**


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