American Heartworm Society Feline Guidelines for the

Prevention, Diagnosis, and Management of Heartworm (*Dirofilaria immitis*) Infection in Cats

AMERICAN HEARTWORM SOCIETY
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Prepared by Dr. C. Thomas Nelson, Dr. John W. McCall, Dr. Andrew Moorhead, Dr. Lindsay Starkey, Dr. Dr. Marisa Ames, and Dr. W. Mark Cousins, and approved by the Executive Board of the American Heartworm Society (Officers: Dr. Jennifer Rizzo, President; Dr. Chris Duke, Past President; Dr. Chris Adolph, Vice President; Dr. Angele Bice, Secretary-Treasurer; Dr. Lindsay Starkey, Editor; Dr. C. Thomas Nelson, Research Chair; Dr. Andrew Moorhead, Symposium Program Chair; Board Members: Dr. Marisa Ames, Dr. Blue Brawner, Dr. Elizabeth Clyde, Dr. W. Mark Cousins, Dr. Uri Donnett, Dr. Aliya Magee; and Ex Officio Members: Dr. Jason Drake, Dr. Doug Carithers, Dr. Lisa Young, Dr. John W. McCall, and Paola Domínguez-López, CVT).

Preamble

These recommendations supersede previous editions and are based on the information presented at the 2022 Triennial Symposium of the American Heartworm Society and recently completed studies. The recommendations for the prevention, diagnosis, and treatment of heartworm infection in dogs are contained in a companion document (available on the AHS website).

EPIDEMIOLOGY

Heartworm infection in cats is a risk wherever heartworm infection occurs in dogs, making it a global threat. In the United States, its territories, and protectorates, heartworm is considered at least regionally endemic in each of the contiguous 48 states, Hawaii, Puerto Rico, US Virgin Islands, and Guam (Bowman et al., 2009; Kozek et al., 1995; Ludlam et al., 1970). There are regions in Alaska that have mosquito vectors and climate conditions to support the transmission of heartworms for brief periods, and cats have tested positive within the state (Darsie and Ward, 2005; Slocombe et al., 1995; Terrell, 1998; AHS incidence survey 2022; CAPC, 2024). Thus, the introduction of microfilaremic dogs or movement of wild canids to non-endemic locations could set up a nidus of

ABBREVIATIONS

ARDS, acute respiratory distress syndrome

AHS, American Heartworm Society

EPA, Environmental Protection Agency

FDA, US Food and Drug Administration

FHWD, feline heartworm disease

HARD, heartworm-associated respiratory disease

L1, first-stage larvae

L2, second-stage larvae

L3, third-stage larvae

L4, fourth-stage larvae

NAbD, no antibody detected

NAgD, no antigen detected

POCUS, point of care ultrasound

KEY POINTS: EPIDEMIOLOGY

- Heartworm infection has been diagnosed in all 50 states and around the globe.
- Environmental and climatic changes, both natural and those created by humans, relocation of microfilaremic dogs, and expansion of the territories of microfilaremic wild canids continue to be important factors contributing to further spread of the parasite.
- A pivotal prerequisite for heartworm transmission is a climate that provides adequate temperature and humidity to support a viable mosquito population and can also sustain sufficient heat to allow maturation of ingested microfilariae into infective, third-stage larvae (L3) within the intermediate host.
- The length of the heartworm transmission season in the temperate latitudes also depends on factors such as the influence of microclimates, unique biological habits and adaptations of the mosquito vector, variations in time of larval development, mosquito life expectancy, and temperature fluctuations.
- Heartworm transmission does decrease in colder months, but the presence of microenvironments in urban areas suggests that the risk of heartworm transmission never reaches zero.
- Cats have a very low and transient microfilaremia and thus become infected when bitten by an L3-infected mosquito that has previously bitten a microfilaremic dog.

Environmental and climatic changes, both natural and those created by humans, and animal movement have increased heartworm infection potential.

infection for local transmission of heartworm (see box on page 4 of the canine guidelines for more on the role of transport of infected dogs). Such relocation of microfilaremic dogs and expansion of the territories of microfilaremic wild canids in other areas continue to be important factors contributing to further dissemination of the parasite, as the ubiquitous presence of one or more species of vector-competent mosquitoes makes transmission possible wherever a reservoir of infection and favorable climatic conditions co-exist. Change in any of these factors can have a significant effect on the transmission potential in a specific geographic location.

Environmental and climatic changes, both natural and those created by humans, and animal movement have increased heartworm infection potential. Commercial and residential real estate development of non-endemic areas and areas of low incidence has led to the resultant spread and increased prevalence of heartworms by altering drainage of undeveloped land and by providing water breeding sites in new urban home sites. In the Western United States, irrigation and planting of trees has expanded the habitat for *Aedes sierrensis* (Western treehole mosquito), a primary vector for transmission of heartworms in those states (Scoles & Dickson, 1995; Scoles et al., 1993).

Aedes albopictus (Asian tiger mosquito), which was introduced into the Port of Houston in 1985, has now spread northward and eastward, including portions of Canada (Khan et al., 2020), and isolated populations have been identified in areas in the western states (Couper & Mordecai, 2022). This urban-dwelling mosquito is capable of reproducing in small containers, such as flowerpots (Benedict et al., 2007).

Urban sprawl has led to the formation of "heat islands," as buildings and parking lots retain heat during the day (**Figure 1**), creating microenvironments with potential to support the development of heartworm larvae in mosquito vectors during colder months, thereby lengthening the transmission season (Morchón et al., 2012; Nelson, 2016; Ledesma & Harrington, 2015).

As mosquito vectors expand their territory and new non-native vectors are introduced, such as *Aedes notoscriptus* (Peterson and Campbell, 2015; Metzger et al., 2022), the number of animals infected will continue to increase. A pivotal prerequisite for heartworm transmission is a climate that provides adequate temperature and humidity to support a viable mosquito population and can also sustain sufficient heat to allow maturation of ingested microfilariae into the infective, third-stage larvae (L3) within this intermediate host. It has been shown in three mosquito species that maturation of larvae ceases at temperatures below 57 °F (14 °C) (Christensen and Hollander, 1978; Fortin and Slocombe, 1981). Heartworm transmission does decrease in colder months, but the presence of microclimates in urban areas suggests that the risk of heartworm transmission never reaches zero (Nelson, 2016). Furthermore, some species of mosquitoes overwinter as adults (Hudson, 1978; Bolling et al., 2007; Farajollahi et al., 2005; Hawley et al., 1989, Hanson & Craig, 1995; Romi et al., 2006). While heartworm larval development in these mosquitoes may cease in cool temperatures, development quickly resumes with

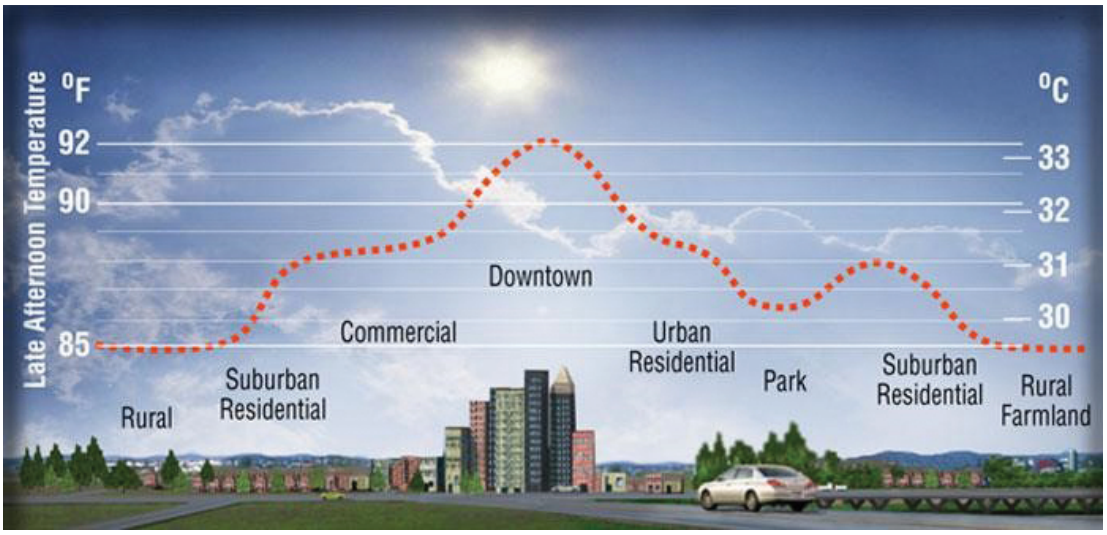


Figure 1. Urban heat island profile showing the elevation in urban air temperature compared with rural air temperature. (Image courtesy of Heat Island Group, Lawrence Berkeley National Laboratory).

subsequent warming (Christensen and Hollander, 1978; Ernst and Slocombe, 1983).

The length of the heartworm transmission season in the temperate latitudes is critically dependent on the accumulation of sufficient heat to incubate larvae to the infective stage in the mosquito (Knight and Lok, 1998). While model-based predictions of transmission using climatic data are academically appealing, they typically fail to consider several potentially important factors, such as influence of microclimate, unique biological habits and adaptations of the mosquito vector, variations in time of larval development, mosquito life expectancy, and temperature fluctuations. Predictive risk maps assume that mosquito vectors live for only one month; however, several significant mosquito vectors live and breed for much longer periods, including:

- *Aedes albopictus* (3 months) (Löwenberg-Neto and Navarro-Silva, 2004),
- *Aedes sticticus* (3 months) (Gjullin et al., 1950),
- *Ochlerotatus* (formerly *Aedes*) *trivittatus* (2 months) (Christensen and Rowley, 1978),
- *Aedes vexans* (2 months) (Gjullin et al., 1950),
- *Ochlerotatus* (formerly *Aedes*) *canadensis* (several months) (Pratt and Moore, 1960) and *Anopheles quadrimaculatus* (4 to 5 months) (Hinman and Hurlbut, 1940).

Survey studies of trapped mosquitoes collected at various locations have demonstrated heartworm infection rates in mosquitoes ranging from 2.1% to 19.4% (McKay et al., 2013; Holderman et al., 2021). When mosquito sampling was restricted to kennel structures where known positive dogs were being housed, the infection rates of the mosquitoes in these restricted samplings resulted in rates of 74% inside the facilities (McKay et al., 2013). Based upon these data, it is important to protect pets from mosquito exposure (see Vector Control, page 10) in addition to administering year-round heartworm preventive.

Once a reservoir of microfilaremic domestic and wild canids is established beyond the reach of veterinary care, or without the knowledge of the local practitioners (i.e., not testing), the ubiquitous presence of one or more species of vector-competent mosquitoes makes transmission possible and eradication improbable.

BIOLOGY OF FELINE HEARTWORM INFECTION

KEY POINTS: BIOLOGY

- Significant differences exist between feline heartworm disease and its canine counterpart. Although cats are susceptible hosts, they are more resistant to infection with adult *Dirofilaria immitis* than are dogs.
- Most adult heartworm infections in cats are comparatively light and consist of less than six worms. Usually only one or two worms are present, and approximately one third of these consist of worms of the same sex.
- Nevertheless, because of their relatively small body size, cats with only a few worms are still considered to be heavily infected in terms of parasite biomass.
- The true prevalence of heartworm infection in cats is probably understated due to diagnostic limitations and the greater tendency of cats to exhibit only transient clinical signs or die without confirmation of infection.
- Heartworm antibody (HW Ab) levels nationwide appear to be in the 15–17% range but have been reported to range from a low of 3.5% to as high as 44%.
- Circulating microfilariae are seldom found in infected cats. It appears that feline infections become occult due to host immune-mediated clearance of the microfilariae.
- Aberrant migration occurs more frequently in cats than in dogs. Although uncommon, heartworms are found disproportionately often in the eyes, body cavities, systemic arteries, and central nervous system of cats.
- The life span of the parasite in cats is thought to be 2 to 4 years, which is considerably shorter than that in dogs.

Significant differences exist between feline heartworm disease and its classical canine counterpart and these are consistent with characteristics of partially adapted host–parasite relationships. Although cats are susceptible hosts, they are more resistant than dogs are to infection with adult *Dirofilaria immitis*. When dogs

not previously exposed to heartworms were inoculated with 100 L3, an average of 60 adult worms developed in almost 100% of the dogs (Blagburn et al., 2011); in 177 experimentally infected cats injected with 100 L3, however, 22.6% of the cats had no worms, 26.0% had one worm, and only 51.4% had two or more worms with an average of 5.6 per infected cat and a median of 5.9 heartworms per cat (McCall et al, 1992, Dillon et al 2007, McTier et al, 2019; McCall & McTier, 2020). These L3 molt to L4 and later to sexually immature adults with some loss along the way, but it is important to note there is a very high mortality rate of the sexually immature adult worms as they reach the pulmonary arteries, which takes 70 to 120 days after infection in dogs (Kotani and Powers, 1982). Most adult heartworm infections in cats are comparatively light and consist of less than six worms (Genchi et al., 1992b). Although much heavier infections occur occasionally, usually only one or two worms are present, and approximately one third of these consist of worms of the same sex (McTier et al., 1992; Ryan and Newcomb, 1995). Nevertheless, because of their relatively small body size, cats with only a few worms are still considered to be heavily infected in terms of parasite biomass.

Host preference by some of the most abundant vectors does favor the dog and may contribute to the lower prevalence of infection in cats (Di Sacco et al., 1992; Genchi et al., 1992a). In a study that collected mosquitoes from baited traps using a 28 kg male dog and an 8 kg male cat found four times as many mosquitoes from the trap containing the dog and they were three times more likely to have fed. This resulted in a 12-fold higher exposure rate in the dog (Genchi et al., 1992a). Furthermore, the *Culex* spp. of mosquitoes including *C. quinquefasciatus*, the Southern house mosquito, which are the most common mosquitoes in many urban areas, feed on both cats and dogs without preference (Wilke et al., 2021; Fitzpatrick et al., 2019).

The true prevalence of heartworm infection in cats is probably understated due to diagnostic limitations and the greater tendency of cats to exhibit only transient clinical signs or die without confirmation of infection. Necropsy surveys of shelter cats have placed the prevalence of adult heartworm infections at 5% to 15% of the rate in unprotected dogs in a given area (Ryan and Newcomb, 1995). Multiple serological surveys of both owned and shelter cats have been conducted (**Table 1**). Heartworm antibody (HW Ab) levels nationwide appear to be in the 15–17% range but have been reported to range from a low of 3.5% to as high as 44%.

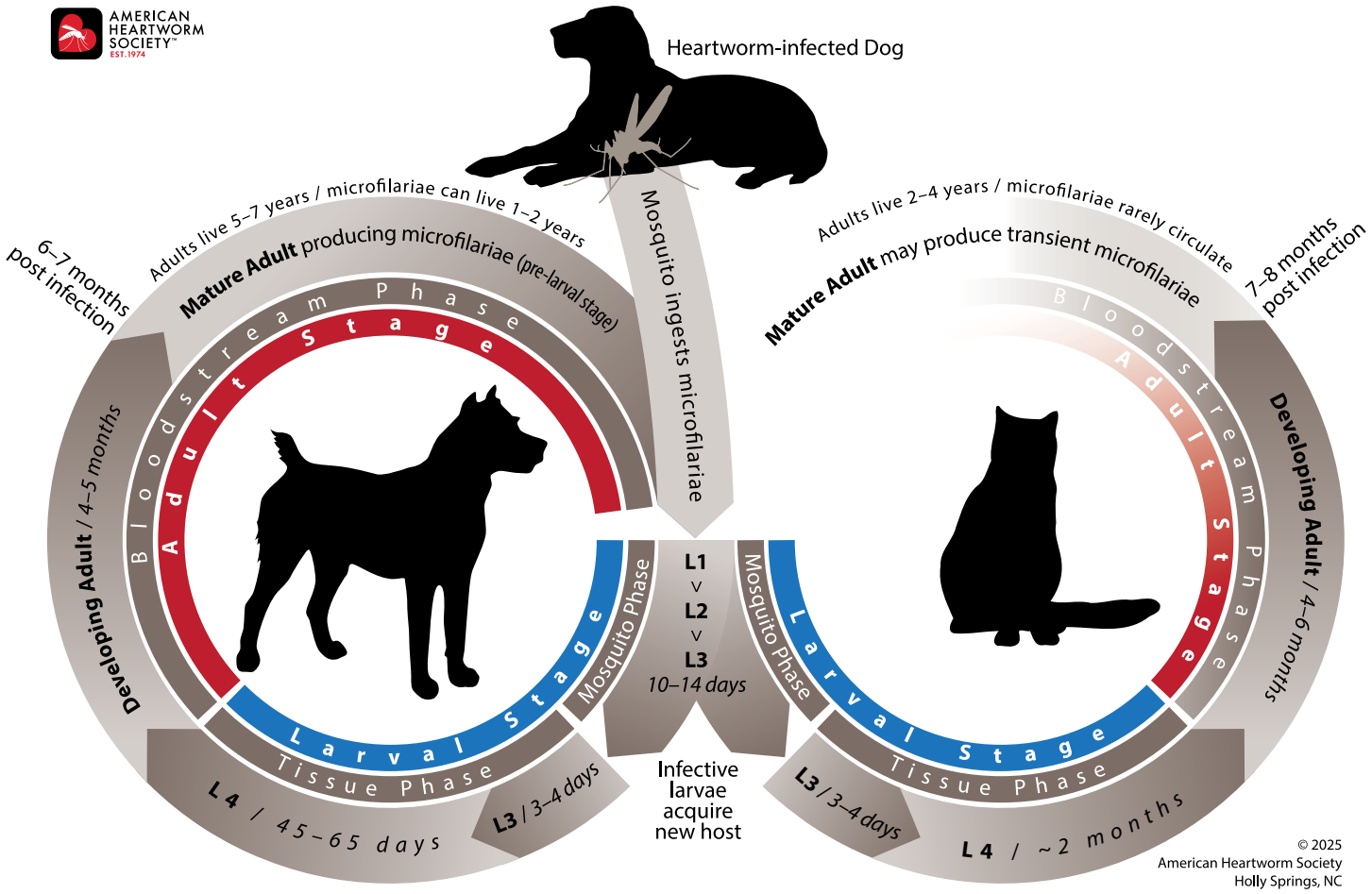
Circulating microfilariae are seldom found in infected cats (Browne et al., 2005). When microfilaremias do develop in cats, they appear only about one week later than in dogs (195 days post infection at the earliest) and seldom persist beyond 228 days post infection

Table 1. Reported Heartworm Antibody (HW Ab) Levels in Cats

Study	# Cats/Status	Location	HW Ab Test	% Positive
Nelson & Self, 1998	98 SC	SE Texas	HC	22.50%
Piché et al., 1998	25,277 OC	46 states	HC	15.90%
Watkins et al., 1998	7,969 OC	30 states & Puerto Rico	AD	17%
Dillon et al., 1998	215 SYM	FL, SC, TN, TX	HC / AD	13.2% / 39.5%
Miller et al., 1998b	129 OC/ASYM	College Station, TX	AD	28%
Miller et al., 1998a	2,181 OC	Midwest, CA, TX & FL	AD	11.9% / 7.9% IN / 19.7% OUT
Hays et al., 2020	100 SC	Florida	HC / SYN	17% / 2%
Murrilo et al., 2023	2,165 OC	47 states	HC	3.50%
CAPC Map, 2023	215,714 OC	50 states	SYN	0.79%
Nelson & Johnson, 2024	50 SC	Alabama	SYN / HW Ab1	0% / 26%

Abbreviations: Heska (an Antech Company) (HC), Synbiotics Corp. (SYN), Animal Diagnostics (AD), Shelter Cats (SC), Owned Cats (OC), Symptomatic (SYM), Asymptomatic (ASYM), Indoor 100% (IN), Outdoor 100% (OUT), HW Ab Experimental (HW Ab1)

Figure 2. The heartworm life cycle in cats (right).



(McCall et al., 1992). Heartworms transplanted from cats are capable of resuming production of circulating microfilariae in dogs; thus it appears that feline infections become occult due to host immune-mediated clearance of the microfilariae and perhaps a reversible suppression of microfilariae production.

There are other indications that the cat is an imperfect host for heartworms. Aberrant migration occurs more frequently in cats than in dogs (McCall et al., 1992). Although uncommon, heartworms are found disproportionately often in the eyes, body cavities, systemic arteries, and central nervous system of cats. Additionally, the life span of the parasite in cats is thought to be 2 to 4 years, which is considerably shorter than that in dogs (**Figure 2**) (Genchi et al., 2008; McCall et al., 1992). Nevertheless, heartworms are capable of causing severe disease in cats.

PATHOPHYSIOLOGY OF FELINE HEARTWORM DISEASE

Feline heartworm infection is the presence of migrating heartworm larvae in tissues or adult worms within the pulmonary vasculature. Feline heartworm disease (FHWD), also known as *heartworm-associated respiratory disease* (HARD), is pathology caused by a current or past infection. The clinical importance of heartworms is amplified in cats because even a small number of heartworms are potentially life-threatening. Although live adult worms in the pulmonary arteries cause local arteritis, some cats never manifest clinical signs. When signs are evident, they usually develop during three stages of the disease. The first stage coincides with the arrival of sexually immature adult worms in the pulmonary arteries and arterioles approximately 75 to 90 days post infection. These early signs are due to an acute vascular and parenchymal inflammatory response to the newly arriving worms and the subsequent death of most of these same worms (Blagburn and Dillon, 2007; Nelson et al., 2007; Nelson, 2008; Dillon et al., 2014; Dillon et al., 2017a; Dillon et al., 2017b).

The intense inflammatory response is due to the presence of pulmonary intravascular macrophages (PIMs). The cat's reticuloendothelial system differs from the dog in that 70% of phagocytic activity is attributed to PIMs. In the dog, 80% of phagocytosis

KEY POINTS: PATHOPHYSIOLOGY

- Feline heartworm infection is the presence of migrating heartworm larvae in tissues or adult worms within the pulmonary vasculature.
- Feline heartworm disease (FHWD), also known as heartworm-associated respiratory disease (HARD), is pathology caused by a current or past infection.
- Although live adult worms in the pulmonary arteries cause local arteritis, some cats never manifest clinical signs. When signs are evident, they usually develop during one or more of the three stages of the disease.
- In the first stage, which occurs with the arrival of sexually immature adult worms in the pulmonary arteries and arterioles approximately 75 to 90 days post infection, there is an intense inflammatory response due to the presence of pulmonary intravascular macrophages (PIMs).
- In the second stage, the mature worms begin to die and the suppression of the immune system ends; the degenerating parasites result in pulmonary inflammation and thromboembolism, which often leads to fatal acute lung injury.
- The third stage of disease occurs if the cat survives the death of an adult worm. Hyperplasia of Type 2 alveolar cells replaces normal Type 1 alveolar cells, potentially leading to permanent lung injury.
- Caval syndrome occurs rarely in cats because infections are usually light; however, even one or two worms may cause tricuspid regurgitation and resultant heart murmur.

occurs by Kupffer cells in the liver. When heartworm larvae die in tissue, small particles are carried by lymph into the circulation and are phagocytized by PIMs resulting in the release of cytokines leading to an inflammatory response (Dillon et al., 1996a; Dillon et al., 2008; Warner, 1996). This may lead to coughing or dyspnea and is often misdiagnosed as asthma or allergic bronchitis but in actuality is part of a syndrome known as FHWD/HARD. Clinical signs associated with this acute phase subside as the worms mature; but, demonstrable histopathologic lesions are evident even in those cats that clear the infection or have never been definitively diagnosed (Browne et al., 2005, Nelson and Johnson, 2024). The most notable microscopic lesion is occlusive medial hypertrophy of the small pulmonary arterioles (**Figure 3**); but other changes are also noted in the bronchi, bronchioles, alveoli (**Figure 4**), and pulmonary arteries (Dillon et al., 2017a, Dillon et al., 2014).

Once an adult pulmonary infection is established, evidence suggests that live heartworms are able to suppress PIM activity and down-regulate immune function (Dillon et al., 1996a; Dillon et al., 2008; González-Miguel et al., 2010; Nelson, 2008; Simón et al., 2001). This allows many cats to tolerate their infection without apparent ill effects—until the mature worms begin to die and the suppression of the immune system ends, which initiates the second stage of disease expression. The degenerating parasites result in pulmonary inflammation and thromboembolism, which often leads to fatal acute lung injury (Atkins et al., 2000; McCall et al., 1992; McCall et al., 2008; Nelson 2008). Such reactions in cats did occur even in single-worm infections as the result of the death of that worm. It has also been shown that the death of pre-cardiac stages (L3 and L4) can lead to smooth muscle hypertrophy of pulmonary arterioles and increases in pulmonary interstitial myofibrocytes (Dillon et al., 2014, Browne et

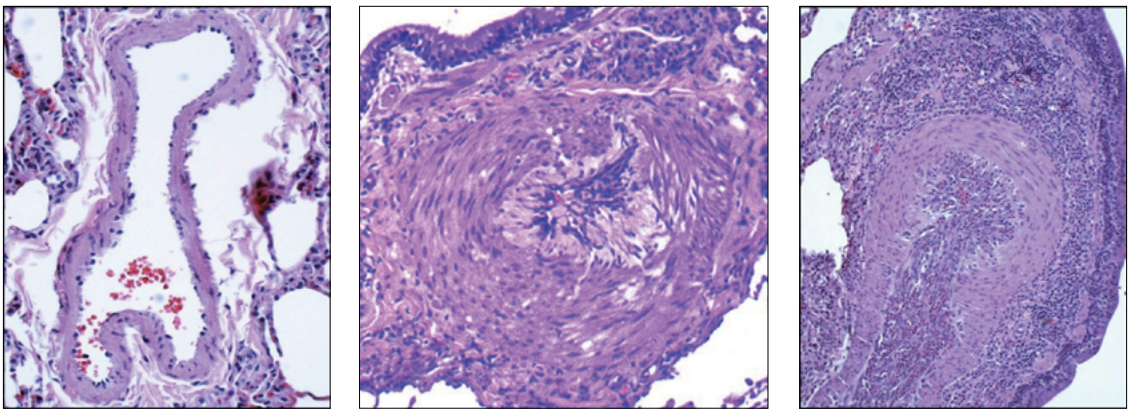


Figure 3. Small pulmonary arterioles. A, Adult heartworm and antibody (Ab) negative. B, Adult heartworm negative and antibody positive. C, Adult heartworm positive.

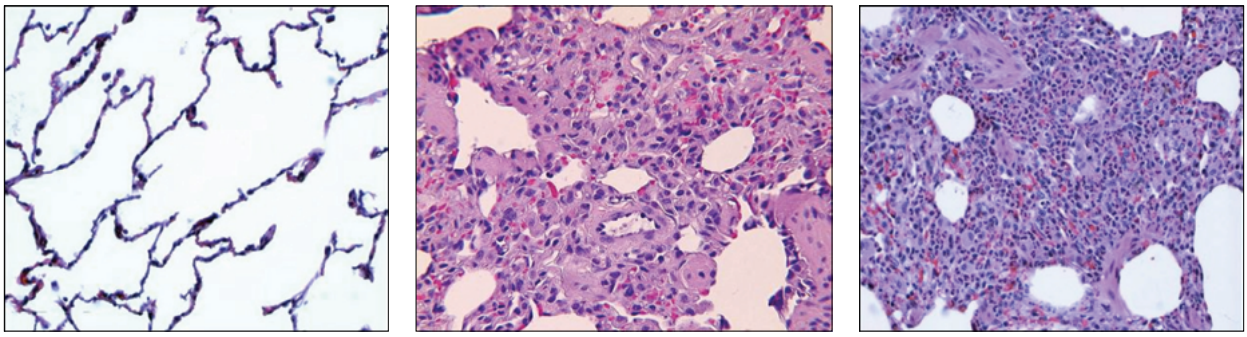


Figure 4. Alveolus, A, Adult heartworm and antibody (Ab) negative. B, Adult heartworm negative and antibody positive. C, Adult heartworm positive

al., 2005). Cats that were on heartworm preventive prior to infection, however, did not exhibit any significant pathologic changes.

The third stage of disease occurs if the cat survives the death of an adult worm as hyperplasia of Type 2 alveolar cells replaces normal Type 1 alveolar cells, potentially leading to permanent lung injury (Dillon et al., 1995).

Arterial intimal proliferation resembling the characteristic heartworm arteritis found in dogs also develops in the major lobar and peripheral pulmonary arteries of cats. Because heartworm infections in cats usually have a small number of worms and are of relatively short duration, these lesions are localized and ordinarily fail to cause sufficient obstruction to produce clinically significant pulmonary hypertension. Consequently, right ventricular hypertrophy and right heart failure are less common in heartworm-infected cats than in dogs. Even when narrowing of a lumen is compounded by worm-induced thrombosis, bronchopulmonary collateral circulation usually is adequate to prevent infarction of the lung (Dillon et al., 1995).

In dogs, caval syndrome (dirofilarial hemoglobinuria) results partly from large numbers of heartworms relocating to the cavae and right atrioventricular junction, interfering with tricuspid valve function. Caval syndrome occurs rarely in cats because infections are usually light; however, even one or two worms may cause tricuspid regurgitation and resultant heart murmur (Bowman and Atkins, 2009).

PREVENTION

KEY POINTS: PREVENTION

- The AHS continues to recommend that *all* cats be on year-round heartworm prevention.
- Any cat living where heartworm-positive dogs and wild animals are in the vicinity is at risk. Given that mosquitoes can enter homes, this includes indoor cats.
- Heartworm chemoprophylaxis can be achieved in cats with monthly doses of milbemycin oxime orally, or topical eprinomectin, moxidectin or selamectin; an every other month topical formulation of moxidectin is also available.
- Heartworm preventives should be administered year-round for the following reasons: 1) added activity against some common intestinal parasites +/- external parasites that may also have zoonotic potential, 2) increased compliance, and 3) some retroactive efficacy as a safeguard for inadvertently missed doses.
- Whereas vector control to supplement prevention of infection typically includes mosquito-repellent products *applied* to dogs, in cats a multimodal vector-control program is usually geared toward reducing the *risk* of mosquitoes in the environment rather than mosquito repellents on the cat itself.

(continued on next page)

There is a risk for any cat, regardless of lifestyle, to become infected with heartworms if heartworm-positive wild or domestic dogs are in the vicinity. The risk increases further for cats that spend time outdoors and in regions where heartworm is endemic. Even cats

- Vector control measures include eliminating sources of standing water or treating them with chemical and/or biological tools such as insect growth regulators, *Bacillus* species, and mosquito fish.
- Utilize local environmental application of insecticidal sprays/fogs and adult mosquito traps.
- Other measures include reducing exposure of cats by limiting outdoor activities and using FDA- or EPA-approved ectoparasiticide products designed for use in cats as many products approved for dogs contain levels of permethrin or related compounds that can be toxic if applied to or accidentally ingested by cats
  - o DEET, which is approved for human use, is **NOT** recommended for use on cats. (Dorman, 1990; Gwaltney-Brant, 2004)
- While vector mitigation strategies and lifestyle management alone or together are helpful, they are not completely effective as monotherapy for heartworm prevention with a macrocyclic lactone.

that spend their entire lives indoors remain at risk, as mosquitoes do enter into homes. In one retrospective study, approximately 25% of cats diagnosed with adult heartworms were considered indoor cats as defined by the owner (Atkins et al., 2000). To help mitigate the risk for feline heartworm infection, several safe and effective options are available for cats that provide protection not only against heartworm, but other parasites as well. Caregivers should be advised objectively of the potential risk of heartworm infection in their community and for their cat's living conditions. When heartworm chemoprophylaxis is elected, it should be administered year-round for the following reasons: 1) added activity against some common intestinal parasites +/- external parasites that may also have zoonotic potential (Arther et al., 2005; Bishop et al., 2000; Bowman et al., 1991; Boy et al., 2000; Humbert-Droz et al., 2004; Nolan et al., 1992) ; 2) increased compliance, and 3) some retroactive efficacy as a safeguard for inadvertently missed doses. (For a more detailed explanation, consult the Canine Guidelines under the heading Macrocyclic Lactones, and https://animaldrugsatfda.fda.gov/adafda/views/#/foiDrugSummaries.) Resistant heartworm isolates have been documented. For more information see the canine guidelines.

Drugs

Heartworm chemoprophylaxis can be achieved in cats with monthly doses of milbemycin oxime orally, or topical eprinomectin, moxidectin or selamectin (see Preventives for Cats chart on the AHS website). An every other month topical formulation of moxidectin is also available. Preventives should be started in kittens as early as 6 weeks of age and be administered to all cats. The individual minimum monthly prophylactic dose of ivermectin is 24 µg/kg (Longhofer et al., 1995), milbemycin oxime 2 mg/kg (Genchi et al., 2004), eprinomectin 0.48 mg/kg (Baker et al, 2021), moxidectin 1 mg/kg (Arther et al., 2003) or 2 mg/kg (Freedom of Information Summary: Bravecto® Plus, 2019) depending on duration of the formulation, and selamectin 6 mg/kg of body weight (McTier et al., 2000). Administration of these drugs in cats may not be precluded by antibody or antigen seropositivity, nor is heartworm testing prior to administration required for cats as it is in dogs, but testing is still **HIGHLY RECOMMENDED**. These drugs have been evaluated for safety in heartworm-positive cats. One should always read the label when administering these products in heartworm-positive cats. Selamectin has been approved for use in heartworm-positive cats.

Vector Control

Heartworm disease is likely grossly underreported in cats. There are a number of broad-spectrum products available to prevent heartworm disease in cats; however, the number of cats being prescribed heartworm prevention is low, and for those that are prescribed preventives, lack of compliance further erodes at the number of protected cats. Because the mosquito is an obligate intermediate host and vector for heartworms, the opportunity to interrupt the chain of transmission at the level of the vector should not be ignored by the pet owner, the veterinarian, or the local municipalities responsible for environmental health and mosquito abatement.

A macrocyclic lactone preventive in addition to vector control measures will provide more complete protection from susceptible heartworms.

Mosquito control in cats is centered less toward products applied to cats and more so on implementing a multimodal strategy to reduce the risk of transmission. This involves evaluating the threat of

infection and disease, both in dogs (reservoirs) and cats, followed by coordinated and reasonable application of countermeasures to mitigate each of those threats. Since cats are more at risk when in the vicinity of positive canines, it is useful to assess the local threat of heartworm infection in dogs using the AHS Incidence Maps (heartwormsociety.org) and information provided in the Canine Guidelines, as well as other data sources for information related to canine heartworm.

Several tactical approaches can be employed to support the overall strategy of vector control. Vector biology has been addressed elsewhere in these guidelines. The first community-based approach should be elimination of mosquito larval habitats, such as standing water sources, wherever possible or treatment of these habitats with chemical and/or biological tools such as, but not limited to, insect growth regulators, *Bacillus* species, and mosquito fish. Local application of insecticidal sprays and fogs and deployment of adult mosquito traps are other approaches. Low winds greatly disturb internally directed flight patterns of mosquitoes, and fan-generated wind has been shown to dilute attractants like carbon dioxide and is a practical approach to protecting people and pets in back yard settings (Hoffman and Miller, 2003). Public municipal organizations as well as private professional businesses can provide expert guidance and tools for these efforts.

Veterinarians should be encouraged to make recommendations for heartworm infection and disease countermeasures that are commensurate with the known or anticipated level of threat. For example, a cat residing in an area of low incidence or exclusively indoors may be administered a macrocyclic lactone product as a reasonable year-round countermeasure. As the threat increases, the application of reasonable mosquito abatement measures is a reasonable addition to the year-round macrocyclic lactone.

Using a multimodal risk-management approach to address the threat of heartworm infection and disease enhances the potential to break the cycle of heartworm transmission and protect the cat while also addressing the challenges of resistant phenotypes in the heartworm population.

Direct protective measures that can be recommended to the cat owner include risk-behavior modification such as eliminating outdoor access or limiting outdoor activities during peak mosquito feeding times and avoidance of known mosquito habitats. A highly effective direct protective measure is the use of topically applied ectoparasiticide products with demonstrated mosquito repellency and insecticidal claims specifically **APPROVED FOR USE IN CATS**. Some canine ectoparasiticide products are toxic to cats, thus caution should be exercised when choosing an appropriate ectoparasite product.

While vector mitigation strategies and lifestyle management alone or together are helpful, they are not completely effective as monotherapy for heartworm prevention. Thus, a macrocyclic lactone preventive in addition to vector control measures will provide more complete protection from susceptible heartworms.

PHYSICAL DIAGNOSIS

KEY POINTS: PHYSICAL DIAGNOSIS

- Clinical signs associated with FHWD may be only a vague malaise or can consist of respiratory, gastrointestinal (e.g., emesis), or occasionally neurologic manifestations, either chronically or acutely.
- Signs of chronic respiratory disease such as persistent tachypnea, intermittent coughing, and increased respiratory effort are most common.
- A right sternal border systolic heart murmur may be present in cats when worms reside in the right atrioventricular junction interfering with tricuspid valve function.
- Anorexia and weight loss occur in some cats. Intermittent vomiting unrelated to eating is reported frequently and in endemic areas when no other cause is evident should raise suspicion of heartworm infection.
- A peracute syndrome consisting of some combination of signs including respiratory distress, ataxia, collapse, seizures, hemoptysis, or sometimes sudden death may arise without warning.

Clinical Signs and Physical Findings

Heartworm infection in cats is a serious disease that can result in fatality, although many cats tolerate their infection without any noticeable clinical signs, or with signs manifested only transiently. Clinical signs associated with FHWD may be only a vague malaise or can consist of respiratory, gastrointestinal (e.g.,

emesis), or occasionally neurologic manifestations, either chronically or acutely. Signs of chronic respiratory disease such as persistent tachypnea, intermittent coughing, and increased respiratory effort are most common.

A right sternal border systolic heart murmur may be present in cats when worms reside in the right atrioventricular junction interfering with tricuspid valvular function. Anorexia and weight loss occur in some cats. Intermittent vomiting unrelated to eating is reported frequently and in endemic areas when no other cause is evident should raise suspicion of heartworm infection. Other abnormalities, such as ascites, pleural effusion (possibly chylous), pneumothorax, ataxia, seizures, and syncope, have been reported but are uncommon. A peracute syndrome consisting of some combination of signs including respiratory distress, ataxia, collapse, seizures, hemoptysis, or sometimes sudden death may arise without warning (Atkins et al., 2000; Dillon, 1984; Dillon et al., 1996b, 1997a,b; McCall et al., 2008).

DIAGNOSTIC TESTING

KEY POINTS: DIAGNOSTIC TESTING

- Heartworm infection in cats is a more elusive diagnosis than in dogs and can be easily overlooked; therefore, we **STRONGLY RECOMMEND** annual testing of cats for heartworm.
- In the cat, no single test will detect all heartworm cases. While the antigen tests are highly specific for detecting adult heartworm antigen, they will not detect infections with only live male worms. The clinician must use a combination of test results to determine the likelihood of heartworm disease as the etiology of the cat's symptoms.
- Since microfilaremia in cats is uncommon, transient, and below concentration levels that might trigger an adverse reaction to microfilaricidal chemoprophylactic drugs, pretesting for microfilariae prior to prescribing a heartworm preventive is unnecessary.
- The preferred method for screening includes the use of both a heat-treated antigen and a Heska (an Antech company) antibody test.
- Antibody tests have the advantage of being able to detect infection by both male and female worms, as larvae of either sex can stimulate a detectable immune response as early as 2 months post infection
- The antigen test is the "gold standard" in diagnosing heartworms in dogs but because unisex infections consisting of only male worms or symptomatic immature infections are more common in cats, none of the presently available antigen tests can be relied upon to rule out heartworm disease in cats.
- The routine heating of blood samples for antigen recovery **IS RECOMMENDED** for cats and is available at most reference laboratories. This recommendation is different than the AHS Canine Guidelines and may interfere with the results of combination tests that include an antibody test for detection of other infectious agents.
- Heartworm test results should only be recorded as positive, no antigen detected (NAgD), or no antibody detected (NAbD) and should not be recorded as "negative."
- Radiographs and echocardiography are the most useful tests to evaluate cardiopulmonary structures in the cat suspected of having heartworm disease. However, point of care ultrasound (POCUS) is useful in the cage-side evaluation of cats with respiratory distress.
- Making an antemortem diagnosis of heartworm infection may be difficult and thus necropsy confirmation should be attempted in cats suspected of dying of the disease or in which the cause of death is unexplained.

Heartworm infection in cats is a more elusive diagnosis than in dogs and can be easily overlooked. A conscious awareness of its existence is critical; therefore, we **STRONGLY RECOMMEND** annual testing of cats for heartworm. The preferred method for screening includes the use of both a heat-treated antigen and a Heska (an Antech company) antibody test. A willingness to pursue this high index of suspicion frequently entails application of multiple diagnostic tests, some of which may need to be repeated on several occasions. Of these, heartworm serology, thoracic radiography, and echocardiography are the most useful methods of clinical confirmation. However, even the use of multiple

diagnostic modalities may fail to confirm heartworm infection in some cats.

The primary reasons for heartworm testing in cats are:

1. To establish an etiologic diagnosis in those individuals that, based on other clinical evidence, are suspected of being infected;
2. To monitor the clinical course of those cats that have already been diagnosed with feline heartworm disease;
3. To establish a baseline reference prior to initiating chemoprophylaxis;
4. To inform veterinarians and pet owners of the risk for heartworm infection by tracking the number of local animals testing positive.

Serology

Interpretation of antibody and antigen test results is complicated and a thorough understanding of the limitations of both tests is necessary in order to use these assays in a clinical setting with any confidence.

Antibody Testing

Antibody tests have the advantage of being able to detect infection by both male and female worms, as larvae of either sex can stimulate a detectable immune response as early as 2 months post infection (McCall et al., 1995). Antibody tests do not offer an indication of the continued existence of an infection, however, just that an infection occurred. Initial research reported the sensitivity and specificity of the feline antibody tests to be as high as 98% in experimentally infected cats with adult worms (Bestul et al., 1998; McCall et al., 1992). Necropsy surveys of naturally infected cats from shelters, however, have indicated a lower sensitivity ranging from 32% to 89% (Berdoulay et al., 2004; Snyder et al., 2000). The different antibody tests vary in their sensitivity to each stage of larval development, thus discordant results between test methods are common. In a necropsy survey in which six different antibody tests were evaluated, 21 of 31 heartworm-infected cats were negative on at least one antibody test (Snyder et al., 2000). These tests were performed on postmortem samples, which may have some effect on the sensitivity; but in another necropsy survey involving 10 heartworm-positive cats, 50% were antibody negative on antemortem samples (Nelson and Self, 1998). A third report of 50 clinical cases from a university referral center had a 14% antibody false-negative rate (Atkins et al., 2000).

These studies reported a wide range in sensitivity; to understand the differences, the population tested and the timing of the test must be examined. In the first study on experimentally infected cats, 50 to 100 L3 were inoculated into heartworm-naïve cats and the cats were followed for 6 months (Bestul et al, 1998). This is a much larger challenge than occurs in nature and no data are available on whether the antibody level will decrease over the expected 2- to 4-year life span of an adult worm in a naturally infected cat. The two necropsy studies represent cat populations more typical of those encountered in clinical practice (Snyder et al, 2000; Nelson and Self, 1998). In the last study from a university referral center, 72% of the cats had clinical signs of disease (Atkins et al, 2000). Limited evidence from these studies suggest that the antibody level in cats decreases with time as the parasite matures and that heartworm-infected cats with clinical signs are more likely to be antibody positive than asymptomatic infected cats. Necropsy studies of shelter cats indicate a distinct correlation to antibodies and occlusive medial hypertrophy of substantial numbers of small pulmonary arterioles. These pathologic changes are evident in 79% of necropsy-confirmed adult worm infections and 50% of adult heartworm-negative but antibody-positive cats (Browne et al., 2005). These findings have been confirmed in a study utilizing an experimental model and are significant as they indicate pulmonary disease occurs even in those cats that do not develop adult worm infections (Blagburn and Dillon, 2007; Dillon et al., 2017a). In the model, cats were infected with 100 L3 and were then treated with ivermectin at 150 µg/kg every 2 weeks starting at day 84 post infection to abbreviate the infection. The study revealed 50% of the cats were antibody negative 8 months later when necropsied. These cats developed radiographic and histopathologic changes indicative of FHWD/HARD. A second group of cats using the same protocol but necropsied 18 months later were all antibody negative even though radiographic and histopathologic changes could still be detected (Dillon et al., 2017b).

Heartworm infection in cats is a more elusive diagnosis than in dogs and can easily be overlooked; therefore, we STRONGLY RECOMMEND annual testing of cats for heartworm.

Historically, there were a half dozen heartworm antibody tests and their target antigen varied among the tests. Currently there are only two antibody tests remaining, one provided by Heska (an Antech company) and the second manufactured by Synbiotics. Several studies comparing the two tests have shown the Heska (an Antech company) test to be more sensitive. Snyder et al. (2000) reported the Heska (an Antech company) HW Ab test used with 330 Florida shelter cats detected HW Ab in 87 of 330 cats (26.4%) while the Synbiotics HW Ab test detected HW Ab in 35 of 330 cats (10.6%) (Snyder et al., 2000). A second study performed on blood samples from 100 cats obtained from Florida animal shelters reported the Heska (an Antech company) HW Ab test was positive in 17 of 100 cats while the Synbiotics HW Ab test was only positive in 2 of 100 cats (Hays et al., 2020). A third shelter cat study on 50 cats reported the Synbiotics test did not detect any HW antibodies despite 20% being heat-treated heartworm antigen (HW Ag) positive; it is obvious why obtaining a diagnosis for FHWD/HARD is so elusive (Nelson and Johnson, 2024).

Antigen Testing

The antigen test is the "gold standard" in diagnosing heartworms in dogs but because unisex infections consisting of only male worms or symptomatic immature infections are more common in cats, none of the presently available antigen tests can be relied upon to rule out heartworm disease in cats. In the cat, if detectable antigenemia develops, this occurs at about 5.5 to 8 months post infection (McCall et al., 1998; Stewart et al., 1992). Necropsy surveys of shelter cats have shown that 50% to 70% of infected cats have at least one female worm (Berdoulay et al., 2004; Snyder et al., 2000). False-negative test results occur most commonly when infections are light, female worms are still immature, only male worms are present, and/or the test kit instructions have not been followed (Genchi et al., 1998; McCall et al., 1998). There are also documented cases of antigen-antibody complexes interfering with antigen testing, resulting in false-negative tests. Heating the sample test tube in a warm water bath to 104 °C for 10 minutes will break these complexes down, releasing any antigen, resulting in more accurate test results (Little et al., 2014). The routine heating of blood samples for antigen recovery **IS RECOMMENDED** for cats and is available at most reference laboratories (Little et al., 2014; Nelson and Johnson, 2024; Gruntmeir et al., 2017). This recommendation is different than the AHS Canine Guidelines and may interfere with the results of combination tests that include an antibody test for detection of other infectious agents. It should be noted that there have been cases reported that have had heat-treated antigen positive test results where no worms were recovered on necropsy (Nelson and Johnson, 2024). In these cases, 86% had vascular lesions consistent with FHWD/HARD. Ectopic infections are more common in cats and worm fragments from worms that have recently died can be forced into the very distal regions of the pulmonary arteriole tree and can be missed on necropsy. Gruntmeir et al. also reported that in the cats that became HW Ag positive after heat-treatment, 77% were Hw Ab positive on the Heska (an Antech company) Solo Step test (Gruntmeir et al., 2017). Due to this and other mentioned considerations, heartworm test results should only be recorded as positive or no antigen detected (NAgD), no antibody detected (NAbD) and should not be recorded as "negative."

The routine heating of blood samples IS RECOMMENDED for cats and is available at most reference laboratories.

Thoracic Radiography

Independent of serologic test results, radiography may provide strong evidence of FHWD and is valuable for assessing the severity of disease and monitoring its progression or regression. The most characteristic radiographic features of heartworm disease in cats, as in dogs, are subtle enlargement of the main lobar and peripheral pulmonary arteries, characterized by loss of taper, and sometimes tortuosity and truncation in the caudal lobar branches (Brawner et al., 1998; Donahoe et al., 1976a,b; Schafer and Berry, 1995). These vascular features are visualized best in the ventrodorsal and dorsoventral views and may be visible only in the right caudal lobar artery, where heartworms are found most often. Quantitatively, a ratio of greater than 1.6 when comparing either caudal lobar pulmonary artery to the size of the 9th rib at the 9th intercostal space is indicative of pulmonary arterial enlargement, which could be caused by FHWD (Schafer & Berry, 1995). The characteristic morphology of the pulmonary arteries in infected cats, unlike dogs, tends to normalize and may disappear completely, leaving no residual evidence of infection (Selcer et al., 1996). Enlargement of the main pulmonary artery segment may occur in heavily infected cats but is not a reliable marker because most

cats do not develop pulmonary hypertension (Winter et al., 2017) and because the main pulmonary artery is obscured by the cardiac silhouette. The cardiac silhouette itself is seldom enlarged. A bronchial and/or unstructured interstitial lung pattern that may clear spontaneously within a few months is a common secondary feature suggestive of, but not unique to, FHWD. Other less commonly associated pulmonary findings include alveolar pattern, hyperinflation of the lungs with flattening of the diaphragm, focal parenchymal radiodensities, focal hyperlucency, consolidated lung lobes, pleural effusion, and pneumothorax (Schafer and Berry, 1995; Selcer et al., 1996). In some cases of FHWD, thoracic radiographs provide no evidence of infection (Selcer et al., 1996).

Radiographic features suggestive of FHWD can be found in about half of the cats suspected of being infected based on historical and physical signs. Also, about half of those cats with pulmonary arterial enlargement indicative of FHWD are antibody positive (Brawner et al., 1998). Temporal differences in the development of the parasite, host immune responses and organic disease, as well as spontaneous regression of lesions, may account for discrepancies between radiographic, clinical and serologic findings. Based on an experimental model of pulmonary larval dirofilariasis (HARD), the arrival and death of early stages of sexually immature adult heartworms in the pulmonary vasculature induce pulmonary airway, interstitial, and arterial lung lesions (Dillon et al., 2017a). These lesions lead to radiographic changes that are similar to those in cats with mature adult infections, yet radiographic lesions improved over time (Dillon et al., 2017b). Infection with *Toxocara cati* and feline lungworm species can cause similar radiographic patterns and must be considered in a differential diagnosis (Browne et al., 2005; Mackenzie, 1960; Swerczek et al., 1970).

Echocardiography

The chambers of the right side of the feline heart, the main pulmonary artery, and a long segment of the right and a short portion of the left pulmonary arteries can be thoroughly interrogated by echocardiography (Venco et al., 1998b). Although heartworms are found most often in the main and right lobar branch of the pulmonary artery, it is necessary to methodically examine all of these locations because worms in a typical light infection may occupy only one or two sites and may escape detection. The body wall of an adult heartworm is strongly echogenic and produces short, segmented, parallel linear artifacts where the imaging plane transects the parasite's body, producing the signature signs of live worms (Selcer et al., 1996). Sometimes dead heartworms can be recognized by the collapse of the parallel sides of the body wall. An adult heartworm is relatively long compared with the length of the pulmonary arteries in cats. Therefore, there is a better chance in cats than in dogs of finding heartworms extending from peripheral branches into proximal segments where they can be visualized (Atkins et al., 1998). An experienced sonographer has a very good chance of making a definitive diagnosis in cats that are actually infected with adult heartworms, particularly when there are several worms (DeFrancesco et al., 2001; Genchi et al., 1998). In suspected cases, the high specificity of this examination generally allows for confirmation of heartworm infection of at least 5 months' duration (Selcer et al., 1996). Quantification of worm burden is, nevertheless, virtually impossible because the potential serpentine positioning allows echo beams to transect the worm in multiple sites, giving multiple echo images and potentially overestimating worm burden. Furthermore, even though highly specific, the sensitivity of visually detecting a worm via echocardiography is highly variable for a number of reasons; thus heartworm infection cannot be ruled out due to lack of visualization with this diagnostic method.

Radiographs and echocardiography are the most useful tests to evaluate cardiopulmonary structures in the cat suspected of having heartworm disease.

Point of Care Ultrasound

Radiographs and echocardiography are the most useful tests to evaluate cardiopulmonary structures in the cat suspected of having heartworm disease. However, point of care ultrasound (POCUS) is useful in the cage-side evaluation of cats with respiratory distress. Left-sided heart disease is a common cause of respiratory distress and an important differential diagnosis. The finding of an increased left atrium to aortic ratio (e.g., a ratio ≥ 2) increases the likelihood of a left-sided heart disease as the cause of respiratory distress and makes an airway or primary pulmonary cause of respiratory distress less likely. Right-sided heart enlargement would be more consistent with cardiac dysfunction secondary to chronic pulmonary disease (including heartworm disease) and subsequent pulmonary hypertension. The body wall of adult heartworms

is highly echogenic and produces distinctive, short parallel-sided images with the appearance of "equal signs" where the imaging plane cuts across loops of the parasite. Given that the heartworm is relatively long compared to the cat's pulmonary vasculature, it is possible a worm may be seen in the right ventricle and atrium in a cat with adult heartworm infection. POCUS is also used to evaluate for pleural effusion when decreased or absent lung sounds are noted ventrally. However, cats with left-sided heart disease (e.g., hypertrophic cardiomyopathy) can develop pulmonary edema and/or pleural effusion. Pleural effusion does not always indicate right-sided heart failure in the cat and, furthermore, severe pulmonary hypertension and right-sided heart failure is uncommon in the cat.

Necropsy Confirmation

Making an antemortem diagnosis of heartworm infection may be difficult and thus necropsy confirmation should be attempted in cats suspected of dying of the disease or in which the cause of death is unexplained. A thorough search of the distal pulmonary arteries, venae cavae, and right side of the heart must be performed because one or two worms easily can be overlooked, particularly if immature, dead, or fragmented (Atkins et al., 1998; Genchi et al., 1998; Levy, 2007; McCall et al., 1992; McCall et al., 1995). Special attention should be paid to examining the distal extremities of the pulmonary arteries as any dead worms would be forced and compressed, by blood flow, into the most distal and smallest possible space (Miller et al., 1998a). Because heartworms occasionally are restricted to ectopic sites, the systemic arteries, body cavities, and, if neurologic signs were present, the brain and spinal canal should also be examined thoroughly (Snyder et al., 2000).

Microfilariae

Cats are seldom microfilaremic when examined. It is important to recognize that other microfilaria could be present in the blood of cats, including *D. repens* and *D. striata* (Genchi et al., 1992b; Genchi et al., 1993, Hays et al., 2020, Wyatt et al., 2020). Since few microfilariae are ever present, the chances of finding them are improved by using concentration techniques (modified Knott or membrane filter). For clinics that still have access to a filter apparatus, solutions mentioned above can be used in combination with filter membranes (polycarbonate, 25 mm diameter, 5 µm pore size), most readily available through commercial scientific supply sources. PCR is also available as a send-out option for detection of microfilariae through commercial or research laboratories and provides the benefit of sensitivity as well as specificity, using genetic sequences to differentiate species rather than morphologic characteristics.

Feline heartworm diagnostics are illustrated in **Figure 5** and heartworm diagnostic procedures and tests are summarized in **Table 2**.

Testing Prior to Chemoprophylaxis

Pretesting (screening) dogs is limited to documenting either heartworm antigenemia or circulating microfilariae, both of which are specific indicators of adult worm infection in the dog; however, neither of these are guaranteed to be present in a cat. Since microfilaremia in cats is uncommon, transient, and below concentration levels that might trigger an adverse reaction to microfilaricidal chemoprophylactic drugs, pretesting for microfilariae prior to prescribing a heartworm preventive is unnecessary. Heat-treated antigen and a Heska (an Antech company) antibody test is **HIGHLY RECOMMENDED** prior to administration of preventives for cats. As stated above, these drugs have been evaluated for safety in heartworm-positive cats. Based on the results of studies with other heartworm preventive drugs, there have been no contraindications to their use in heartworm-positive cats.

Interpretation of antibody test results may be difficult if the animal is currently on chemoprophylaxis due to the potential of the presence of L3/L4 that were killed by preventives (Donoghue et al., 1998). Many heartworm preventive products do not maintain residual activity

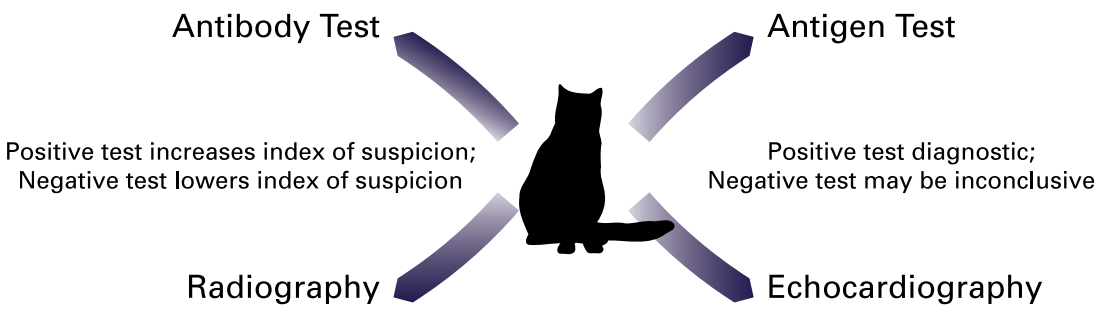


Figure 5. Summary of feline heartworm diagnostics

Table 2. Interpretation of Heartworm Diagnostic Procedures and Tests in Cats

Test	Brief Description	Result	Interpretation	Limitations
Antibody Test	Detects antibodies produced by the cat in response to presence of heartworm larvae. May detect infections as early as 8 weeks post transmission by mosquito	Negative	Lower index of suspicion	Antibodies confirm infection with heartworm larvae, but do not confirm disease causality.
		Positive	Increasing index of suspicion; 50% or more cats will hace pulmonary arterial disease; confirms cat is at risk	
Antigen Test	Detects antigen produced by the adult femaleheartworm or from the dying male (>5) or female heartworms	Negative	Lower index of suspicion	Immature or male-only worm infections are rarely detected.
		Positive	Confirms presence of heartworms	
Thoracic Radiography	Detects vascular enlargement (inflammation caused by juvenile worms and, later, hypertrophy), pulmonary parenchymal inflammation, and edema [the latter only in acute respiratory distress syndrome (ARDS)-like syndrome]	Normal	Lower index of suspicion	Radiographic signs are subjective and affected by clinical interpretation.
		Signs consistent with feline heartworm disease	Enlarged arteries greatly increase index of suspicion	
Echocardiograaphy	Detects echogenic walls of the immature or mature heartworm residing in the lumen of the pulmonary arterial tree, if within the visual window of the ultrasound	No worms seen	No change to index of suspicion	Ultrasonographer experience with heartworm detection appears to influence accuracy rate.
		Worms seen	Confirms presence of heartworms in the structure	

Note: In the cat, no single test will detect all heartworm cases. While the antigen tests are highly specific for detecting adult heartworm antigen, they will not detect infections with only live male worms. The clinician must use a combination of test results to determine the likelihood of heartworm disease as the etiology of the cat's symptoms.

against larval heartworm stages following dosing, and since mosquito bites/transmission of larval stages can occur between dosages, the need for consistently timed and year-round dosing schedules is imperative to reduce the risk for larval stages developing to the point of inciting HARD or other heartworm-related issues. It is those transmitted larval stages that are capable of inciting an immune response that will result in a positive antibody test, even in a cat that is compliantly administered a heartworm preventive product.

TREATMENT

Medical Options

To date, there are no studies that indicate any form of medical adulticidal therapy increases the survival rate of cats harboring adult heartworms. That being said, utilizing heat-treated antigen tests and the Heska (an Antech company) HW Ab test will inevitably lead to more cats being diagnosed with FHWD/HARD and the demand to provide some semblance of therapy to mitigate clinical signs and the real possibility of sudden death from an acute lung injury. Below is a discussion of potential treatment options that may be employed that are based on a combination of theory, anecdotal

KEY POINTS: TREATMENT

- While there is no approved adulticide treatment for cats as there is for dogs, there are medical options to manage cats infected with heartworms.
- These measures can relieve the clinical signs of disease and, in cases of adult infection, prevent sudden death of the cat.
- Medications to consider include the following:
  - o Prednisolone to relieve coughing and other respiratory signs
  - o Doxycycline to eliminate *Wolbachia* organisms from heartworms, which contribute to the pathogenesis of the disease
  - o Supportive therapy with corticosteroids, bronchodilators, oxygen, fluids and thermal support to relieve respiratory distress
  - o Antileukotrienes to prevent respiratory crisis
- Melarsomine is not recommended for use in cats; preliminary data suggests that melarsomine is toxic to cats at doses as low as 3.5 mg/kg.
- Adult heartworms in cats can be surgically removed if the precise location of the worms can be identified using ultrasonography.
- Serologic retesting at 6-month intervals for the purpose of monitoring infection status is recommended for all infected cats whether or not they have clinical signs that are treated empirically or are given medical/surgical adulticide therapy.

reports, and/or extrapolated from canine heartworm treatment protocols.

Prednisolone in tapering doses often is effective medical support for infected cats with radiographic evidence of lung disease whether or not they appear ill. Prednisolone also should be initiated whenever antibody- and/or antigen-positive cats display clinical signs such as cough and tachypnea. An empirical oral regimen is 1–2 mg/kg body weight/day, declining gradually to 0.5 mg/kg every other day by 2 weeks or the lowest possible dose to control clinical signs, with potential discontinuation pending clinical response. This treatment may be repeated in cats with recurrent clinical signs as needed. If clinical signs recur or persist, then alternate-day glucocorticoid therapy is given at the lowest dose effective in controlling signs (Nelson, 2023). A transition to fluticasone 110–220 µg by metered-dose inhaler with spacer and face mask, 1 puff q 12h, can be considered in cats who are successfully tapered to alternate-day glucocorticoid therapy.

Cats that become acutely ill and present in respiratory distress need to be stabilized promptly with supportive therapy appropriate for treating shock. Additional supportive care may also include corticosteroids (dexamethasone 0.1 to 1.0 mg/kg per cat IV or IM), bronchodilators (aminophylline 6.6 mg/kg q12h IV or IM; or terbutaline 0.01 mg/kg IV, IM, or SC), and oxygen. The upper end of the dose range for dexamethasone may seem excessive, yet the acute respiratory distress syndrome seen after death of an adult worm can progress rapidly without time to re-administer additional dosages of corticosteroids. Maintenance of supportive therapy (IV fluids and thermal support) is needed until stabilization of the patient. Please note that the acute respiratory distress syndrome (ARDS) reaction that occurs when the adult heartworm dies will not be eliminated by the use of low-dose alternate doses of prednisolone (Nelson, 2008; Nelson, 2023). Diuretics are inappropriate, even for infected cats with severe interstitial or patchy alveolar lung patterns. These lung patterns are usually the result of inflammation and thromboembolic processes rather than a cardiogenic pulmonary edema (left-sided heart failure) (Davidson et al., 2006; Schermerhorm et al., 2004; Venco et al., 2008). Heart failure is a rare sequela in cats with heartworm disease, and when present, is usually a right-sided heart failure where increased systemic venous pressure leads to the development of cavitary effusions. In these rare cases, centesis and diuretics are indicated. Aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) have failed to produce demonstrable benefit and actually may exacerbate the parenchymal pulmonary disease (Rawlings, 1990).

Ivermectin at a dose of 24 µg/kg monthly given for 2 years has been reported to reduce worm burdens by 65% as compared with untreated cats (Guerrero et al., 2002). Because most cats have small worm burdens, it is not worm mass alone that is problematic but the "anaphylactic" type reaction that results when the worms die. This will likely also occur when the ivermectin-treated worms die but the extent of the reaction is unknown.

Administration of antileukotrienes has been hypothesized as a treatment, due to the fact that cytokines are an important component of the FHWD process. Host cells produce interleukin-8 (IL-8) in response to *Wolbachia*. Interleukin-4 (IL-4) also has been

found in the secretions recovered by bronchoalveolar lavage from heartworm-infected cats. Anecdotally, antileukotrienes (e.g., montelukast) at an oral dosage of 2 mg every day could aid in preventing respiratory crisis in heartworm-infected cats (Feldman et al., 2011; Nelson, 2008; Nelson, 2023).

There is insufficient experience with melarsomine dihydrochloride at this time; thus melarsomine is not recommended for use in cats. Preliminary data suggests that melarsomine is toxic to cats at doses as low as 3.5 mg/kg (Goodman, 1996; McLeroy, 1998).

Most filarial nematodes, including *D. immitis*, harbor obligate, intracellular, gram-negative bacteria belonging to the genus *Wolbachia* (Rickettsiales) (Taylor et al., 2005). These bacteria also have been implicated in the pathogenesis of filarial diseases, possibly through their endotoxins (Bouchery et al., 2013). Doxycycline is a component of the AHS treatment recommendation for heartworm in dogs and has markedly reduced pulmonary pathology and post-treatment complications. The use of doxycycline in cats with heartworm disease is still being explored. However, in cases where the owner would consider unproven adjunct therapies, doxycycline could be considered. Doxycycline would be administered at 10 mg/kg, given q24h or 5 mg/kg q12h for 30 days, in conjunction with a macrocyclic lactone heartworm preventive. A doxycycline suspension is preferred over a pill to prevent esophagitis and potential esophageal strictures that can occur if a pill becomes lodged in the esophagus. Six milliliters of water should be given if a pill is used (Nelson, 2023).

It would be prudent to supply an owner of a known heartworm-positive cat with a syringe containing dexamethasone sodium phosphate (1 mg/kg) to be given subcutaneously in case of severe respiratory distress (Nelson, 2008, Nelson, 2023). The cat should then be taken immediately to a veterinary emergency facility.

Surgical Options

In principle, it is preferable to remove heartworms rather than destroy them in situ. This can be accomplished successfully by introducing brush strings (Venco et al., 1998a), basket catheters (Borgarelli et al., 1997), or loop snares (Small et al., 2008) via right jugular venotomy or, after left thoracotomy, alligator forceps can be inserted through a right ventricular purse-string incision (Glaus et al., 1995; Rawlings et al., 1994). Before attempting either approach, heartworms should be identified ultrasonographically in locations that can be reached with these instruments (Borgarelli et al., 1997). Worms within the vena cavae, right atrium, and right ventricle are usually accessible from jugular vein access, whereas worms within the pulmonary artery or branches are harder to retrieve with currently available instruments. Both atria and ventricles as well as the main pulmonary artery can be reached through a ventriculotomy incision with straight alligator forceps.

Although it may not be possible to retrieve every worm, the surgical option may be a reasonable alternative to symptomatic support or adulticide treatment of cats that are heavily infected and/or in critical condition (Rawlings et al., 1994; Small et al., 2008). Surgery is specifically indicated in those few cases that develop caval syndrome. Care must be taken to remove the worms intact because partial or complete traumatic transection of a worm may result in acute circulatory collapse and death (Venco et al., 1998a).

Adult heartworms in cats can be surgically removed if the precise location of the worms can be identified using ultrasonography.

Surveillance of Infected Cats

Serologic retesting at 6-month intervals for the purpose of monitoring infection status is recommended for all infected cats whether or not they have clinical signs that are treated empirically or are given medical/surgical adulticide therapy. Once adult heartworm infection has been diagnosed, monitoring will be most informative if both Heska (an Antech company) antibody and heat-treated antigen testing are performed. Spontaneous or adulticide-induced elimination of infection in antigen-positive cats ordinarily will be followed within 4 to 5 months by disappearance of detectable antigenemia (Levy et al., 2003). Once cats become antigen negative and are clinically normal, further antibody retesting becomes problematic because antibodies may persist for an indefinite period after the parasites are gone and because continued exposure, even with preventive therapy, may result in a positive test. Radiography and ultrasonography also may be very useful for monitoring the course of infection and disease in those cats with pulmonary vascular and/or parenchymal lung disease, or in which heartworms have been identified with echocardiography.

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