



2007 Guidelines for the Diagnosis, Prevention and Management of Heartworm (*Dirofilaria immitis*) Infection in Cats

Prepared and approved by the Executive Board of the American Heartworm Society (Officers: Dr. Charles Thomas Nelson, President; Dr. R. Lee Seward, Interim Past President; Dr. John W. McCall, Vice President; Dr. Sheldon B. Rubin, Secretary-Treasurer; Dr. Lynn F. Buzhardt, Dr. Wallace Graham, and Dr. Susan L. Longhofer, Board Members; Dr. Jorge Guerrero, Symposium Chair; Dr. Carol Robertson-Plouch, Symposium Co-Chair; Dr. Allan Paul, Editor; and Dr. Doug Carithers, Ad-hoc Member.)

Preamble

These recommendations are based on the latest information presented at the 2004 triennial symposium of the American Heartworm Society and recently completed studies. Revisions to the last recommendations, published in 2005, are based on new research and additional clinical experience.

Epidemiology

Heartworms are considered at least regionally endemic in each of the contiguous 48 states, Hawaii, Puerto Rico, U.S. Virgin Islands and Guam. Heartworm transmission has not been documented in Alaska and even with the importation of microfilaremic dogs, it is doubtful the climate this far north will permit maturation of infective larvae. Relocation of infected, microfilaremic dogs appears to be the most important factor contributing to further dissemination of the parasite. 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.

A climate that provides adequate temperature and humidity to support a viable mosquito population, and also sustain sufficient heat to allow maturation of ingested microfilariae to the infective, third-stage larvae (L3) within the intermediate host is a pivotal prerequisite for heartworm transmission to occur. Intermittent diurnal declines in temperature below the developmental threshold of 57°F (14°C) for only a few hours retard maturation, even when the average daily temperature supports continued development. At 80°F (27°C), 10 to 14 days are required for development of microfilariae to the infective stage.

The heartworm transmission season in the temperate latitudes is greatly influenced by the amount of accumulated heat in the environment during the incubation of the larvae in the mosquito. The peak months for heartworm transmission in the Northern Hemisphere are July and August. Algorithmic predictions based on analysis of historical temperature records have consistently overestimated actual transmission periods, confirmed independently by a variety of field studies, and appear to represent conservative guidelines. Under the most favorable conditions, these estimates range from less than four months in southern Canada to potentially all year

in the subtropical zones of southern Florida and the Gulf Coast. The model predicts that heartworm transmission in the continental U.S. is limited to six months or less above the 37th parallel, i.e., the Virginia-North Carolina state line.

Where the prevalence is low, a nidus of heartworm infection may be detected which usually represents both a focal spread of infection and heightened awareness through increased testing. Once a reservoir of microfilaremic domestic and wild canids is established beyond the reach of veterinary care, eradication becomes improbable.

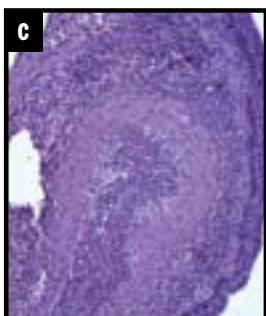
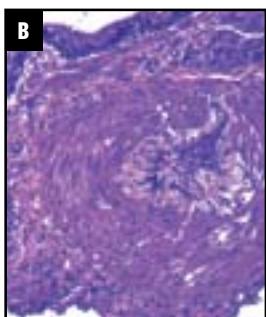
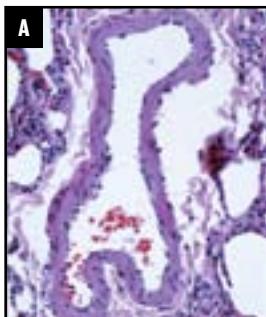
Biology of Feline Heartworm Infection

There are significant differences 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 to infection with adult *Dirofilaria immitis* than are dogs. When dogs not previously exposed to heartworms are injected with 100 L3 larvae, approximately 75 adult worms develop in almost 100% of the dogs whereas in cats, 3 to 10 adult worms develop in 75% of the cats. These L3 larvae molt to L4 and L5 stages with some loss along the way but there is a very high mortality rate of the L5 as they reach the lungs 3 to 4 months after infection. Most heartworm infections in cats are comparatively light and consist of less than six adult worms. 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. 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. Some clinical surveys and data from experimentally infected cats have documented a slight preponderance of infection in male cats, but it has not been determined



Small Pulmonary Arterioles

- A. Heartworm challenged cat on monthly chemoprophylaxis
- B. Abbreviated heartworm larval infection
- C. Adult heartworm positive



conclusively that male cats are at greater risk. No sex predilection for anti-*D. immitis* host antibody seropositivity has been proven within populations of naturally exposed cats, nor has a preference by vector mosquitoes for either sex, although some data suggest trends for each toward female cats. Host preference by some of the most abundant vectors does favor the dog and may contribute to the lower prevalence of infection in cats. The *Culex spp.* mosquito, which is the most common species in many urban areas, feeds on both cats and dogs without preference. 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. Circulating microfilariae are seldom found in infected cats. When microfilaremias do develop in cats, they appear only about one week later than in dogs (195 days postinfection at the earliest), but seldom persist beyond 228 days postinfection. Since heartworms transplanted from cats are capable of resuming production of circulating microfilariae in dogs, it appears 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. Although uncommon, heartworms are found disproportionately often in the body cavities, systemic arteries and central nervous system of cats. Additionally, in cats, the life span of the parasite is thought to be two to three years, which is considerably shorter than that in dogs. Despite this, heartworms are capable of causing severe disease in the cat.

Pathophysiology of Feline Heartworm Disease

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 a local arteritis, some cats never manifest clinical signs. When signs are evident, they usually develop during two stages of

the disease: 1) arrival of heartworms in the pulmonary vasculature and 2) death of adult heartworms. The first stage coincides with the arrival of immature adult worms in the pulmonary arteries and arterioles; approximately three to four months postinfection. 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. This initial phase is often misdiagnosed as asthma or allergic bronchitis but in actuality is part of a syndrome now known as Heartworm Associated Respiratory Disease (HARD). Clinical signs associated with this acute phase subside as the worms mature but demonstrable histopathological lesions are evident even in those cats which clear the infection. The most notable microscopic lesion is occlusive medial hypertrophy of the small pulmonary arterioles, but other changes are also noted in the bronchi, bronchioles, alveoli, and pulmonary arteries.

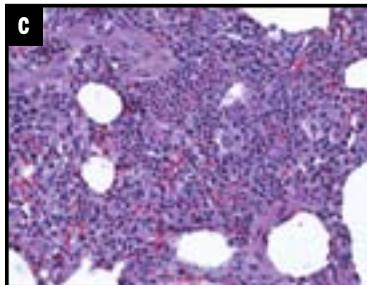
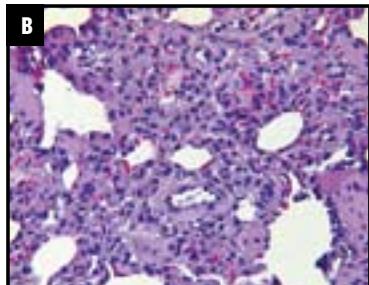
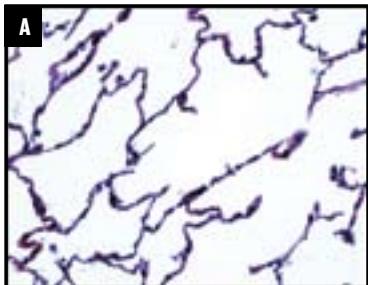
Once the pulmonary infection is established, evidence suggests that live heartworms are able to suppress immune function. This allows many cats to tolerate their infection without apparent ill effects. That is until the mature worms begin to die, which initiates the second stage of disease expression. The degenerating parasites result in pulmonary inflammation and thromboembolism which often leads to fatal acute lung injury. Such reactions in cats can occur even in single worm infections as the result of the death of that worm.

In dogs, the 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 as infections are usually light. However, even one or two worms may cause tricuspid regurgitation and resultant heart murmur.

Arterial intimal proliferation resembling the characteristic heartworm arteritis found in dogs also develops in the major lobar and peripheral pulmonary arteries of cats. Since heartworm infections in cats usually are light, and 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 cats than in dogs. Even when narrowing of a

Alveolus

- A. Heartworm challenged cat on monthly chemoprophylaxis
- B. Abbreviated heartworm larval infection
- C. Adult heartworm positive



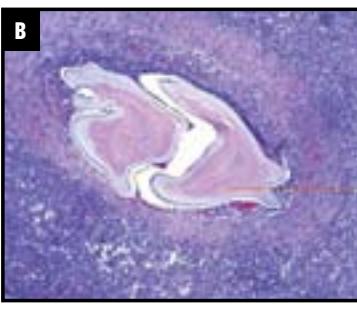
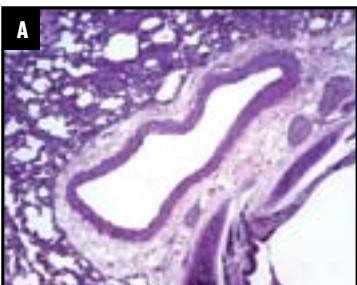


lumen is compounded by worm-induced thrombosis, bronchopulmonary collateral circulation usually is adequate to prevent infarction of the lung.

Physical Diagnosis

Clinical signs and physical findings

Many cats tolerate their infection without any noticeable clinical signs, or with signs manifested only transiently. Clinical signs associated with feline heartworm disease may be only a vague malaise or can comprise predominantly respiratory, gastrointestinal (e.g., emesis) or occasionally neurologic manifestations, chronically or acutely. Signs of chronic respiratory disease such as persistent tachypnea, intermittent coughing and increased respiratory effort are most common. A 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, hydrothorax, chylothorax, 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.



Pulmonary Artery

- A. Heartworm challenged cat on monthly chemoprophylaxis
- B. Adult heartworm positive

Diagnostic Testing

Heartworm infection in cats is a more elusive diagnosis than in dogs and can be overlooked easily. A conscious awareness of its existence is critical. 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.

Microfilariae

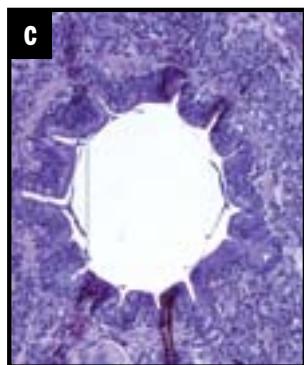
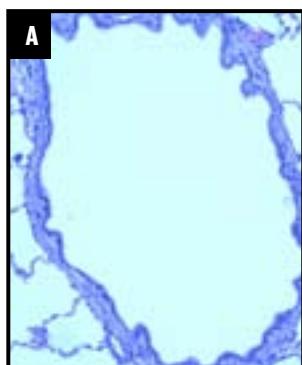
Cats are seldom microfilaremic when examined. In the Americas, only *D. immitis* microfilariae have been

identified in cats but in northern Italy, microfilariae of *Dirofilaria repens* also have been identified. Since few microfilariae are ever present, the chances of finding them are improved by using concentrations techniques (modified Knott or millipore filter).

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. 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 sensitivity of commercially available tests has increased and they are highly effective in detecting single adult female worm infections. In the cat, detectable antigenemia develops at about 5.5 to 8 months postinfection. Necropsy surveys of shelter cats have shown that 50 to 70% of infected cats have at least one female worm.

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 two months postinfection. However, antibody tests do not offer an indication of the continued existence of an infection, 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. However, necropsy surveys of naturally infected cats from shelters have indicated a lower sensitivity ranging from 32 to 89%. The different antibody tests vary in their sensitivity to each stage of larval development, thus discordant results between tests is common. In a necropsy survey where eight different antibody tests were evaluated, 21 of 31 heartworm infected cats were negative on at least one antibody test. These tests were performed on postmortem samples which may have some affect on the sensitivity but in another necropsy survey involving 10 heartworm positive cats, 50% were antibody negative on antemortem samples. A third report of 50 clinical cases from a university referral center had a 14% false antibody negative rate. These four studies reported a wide range in sensitivity.



Bronchiole

- A. Heartworm challenged cat on monthly chemoprophylaxis
- B. Abbreviated heartworm larval infection
- C. Adult heartworm positive



Interpretation of heartworm diagnostic procedures tests in the cat

Test	Brief Description	Result	Interpretations	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 have pulmonary arterial disease; confirms cat is at risk	
Antigen Test	Detects antigen produced by the adult female heartworm 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 young L5 and, later, hypertrophy), pulmonary parenchymal inflammation, and edema.	Normal	Lower index of suspicion	Radiographic signs subjective and affected by clinical interpretation.
		Signs consistent with FHD	Enlarged arteries greatly increases index of suspicion	
Echocardiography	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 symptomatology.

and 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 injected into heartworm naïve cats and the cats were followed for 6 to 9 months. 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 two-to-three year life span of an adult worm. The two necropsy studies represent cat populations more typical of those encountered in clinical practice. In the last study from a university referral center, 72% of the cats had clinical signs of disease. Limited evidence from these studies suggests that the antibody level in cats decreases with time as

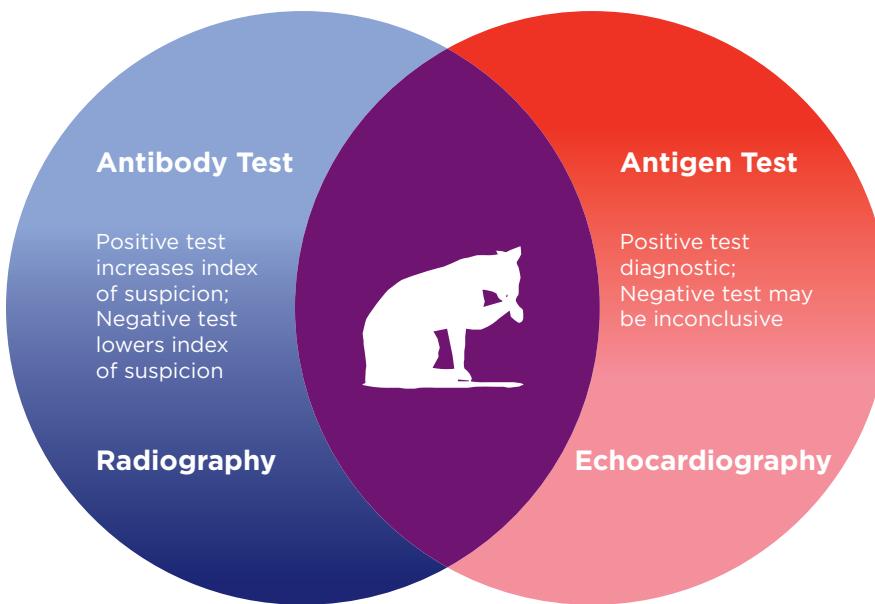
the parasite matures and that heartworm infected cats with clinical signs are more likely to be antibody positive than infected asymptomatic cats.

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. These findings have been confirmed in experimental models and are significant as they indicate pulmonary disease occurs even in those cats that do not develop adult worm infections.

Correct interpretation of antibody test results requires additional information and thoughtful analysis. However, when infection with adult female worms actually exists, antigen tests are more reliable than generally credited. Since both L5 larvae and adult worms are capable of causing clinical disease in the cat, both antibody and antigen tests are useful tools and when used together increase the probability of making appropriate diagnostic decisions.

Thoracic radiography

Independent of serologic test results, radiography may provide strong evidence of feline heartworm disease, 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 a sometimes 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. These vascular features are visualized best in the ventrodorsal view and may be visible only in the right caudal lobar artery, where heartworms are found most often. 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. Enlargement of the main pulmonary artery segment may occur in heavily infected cats but is not a reliable marker, since most cats do not develop pulmonary hypertension and because the main pulmonary artery is obscured by the cardiac silhouette. The cardiac silhouette itself is seldom enlarged. A bronchointerstitial lung pattern that may clear spontaneously within a few months is a common secondary feature, suggestive of, but not unique to feline heartworm disease. Other less commonly associated pulmonary findings include hyperinflation of the lungs with flattening of the diaphragm, focal parenchymal radiodensities, consolidated lung lobes, pleural effusion and pneumothorax. In some cases of feline heartworm disease, thoracic radiographs provide no evidence of infection. Radiographic features suggestive of feline heartworm disease 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 feline heartworm disease are antibody positive. 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.

Echocardiography

The chambers of the right side of the feline heart can be thoroughly interrogated by 2D ultrasonography and limited access also can be gained to the main pulmonary artery, and a long segment of the right and a short portion of the left pulmonary arteries. Although heartworms are found most often in the main and right lobar branch of the pulmonary artery, it is necessary to probe methodically all of these locations, since 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. Sometimes dead heartworms can be recognized by 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. 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. In suspected cases, the high specificity of this examination generally allows for confirmation of heartworm infection of at least five months duration. Quantification of worm burden is,

nevertheless, difficult because the potential serpentine positioning allows echo beams to transect the worm in multiple sites, giving multiple echo images and potentially overestimating worm burden.

Necropsy confirmation

Since making an antemortem diagnosis of heartworm infection may be difficult, 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 vena cavae, right side of the heart and pulmonary arteries must be performed since one or two worms easily can be overlooked, particularly if immature, dead and fragmented. 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. 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.

Treatment

Medical options

If a cat displays no overt clinical signs despite radiographic evidence of pulmonary vascular/interstitial lung disease consistent with feline heartworm disease, it may be prudent to allow time for a spontaneous cure to occur. The course of infection in these subclinical cases can be monitored periodically at 6 to 12 month intervals by repeat antibody and antigen testing, and thoracic radiography. In those cats destined to recover, regression of radiographic signs and especially seroconversion of a positive antigen test to negative status provide evidence that the period of risk probably has passed. Prednisone in diminishing doses often is effective medical support for infected cats with radiographic evidence of lung disease, whether or not they appear ill. Also, this should be initiated whenever antibody and/or antigen positive cats display clinical signs. An empirical oral regimen is 2 mg/kg body weight/day, declining gradually to 0.5 mg/kg every other day by two weeks and then discontinued after an additional two weeks. At that time the effects of treatment should be reassessed based on the clinical response and/or thoracic radiography. This treatment may be repeated in cats with recurrent clinical signs.

Cats that become acutely ill need to be stabilized promptly with supportive therapy appropriate for treating shock. Depending on the circumstances, this may include intravenous corticosteroids, balanced electrolyte solutions, bronchodilators and oxygen via intranasal catheter or closed cage. Diuretics are inappropriate, even for infected cats with severe interstitial or patchy alveolar lung patterns. Aspirin and other non-steroidal anti-inflammatory drugs have failed to produce demonstrable benefit and actually may exacerbate the parenchymal pulmonary disease.



Adulicide administration is considered the treatment of last resort for cats in stable condition, but which continue to manifest clinical signs that are not controlled by empirical corticosteroid therapy. 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.

Ivermectin at 24 ug/kg monthly given for 2 years has been reported to reduce worm burdens by 65% as compared to untreated cats. Since 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.

To date, there are no studies that indicate any form of medical adulcicidal therapy increases the survival rate of cats harboring adult heartworms.

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, basket catheters, or loop snares via right jugular venotomy, or after left thoracotomy, alligator forceps can be inserted through a right ventricular purse string incision. Before attempting either approach, heartworms should be identified ultrasonographically in locations that can be reached with these inflexible instruments. When probing from the right jugular vein, worms must be present within the cavae or right atrium since achieving access to the right ventricle is difficult with these instruments. Through a ventriculotomy incision, both atria and ventricle as well as the main pulmonary artery can be reached 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 adulicide treatment of cats that are heavily infected and/or are in critical condition. Surgery is specifically indicated in those few cases that develop the caval syndrome. Care must be taken to remove the worms intact since partial or complete traumatic transection of a worm may result in acute circulatory collapse and death.

Additional Considerations for Adulicide Therapy

Wolbachia

Most filarial nematodes, including *D. immitis*, harbor obligate, intracellular, gram-negative bacteria belonging to the genus *Wolbachia* (*Rickettsiales*). In infections with other filarial parasites, treatment with tetracyclines during the first month of infection was lethal to some *Wolbachia*-harboring filariae, but not to filariae that

did not harbor *Wolbachia*, and treatment of *Wolbachia*-harboring filariae suppressed microfilaremia. Similar prophylaxis studies with *D. immitis* have not been reported, but in one study, tetracycline treatment of heartworm-infected dogs resulted in infertility in the female worms. These bacteria also have been implicated in the pathogenesis of filarial diseases, possibly through their endotoxins. Recent studies have shown that a major surface protein of *Wolbachia* (WSP) induces a specific IgG response in hosts infected by *D. immitis*. It is hypothesized that *Wolbachia* contribute to pulmonary and renal inflammation through its surface protein WSP, independently from its endotoxin component. Studies to determine the effects of suppressing *Wolbachia* populations with doxycycline prior to adulicide therapy are in progress to determine the clinical utility of this therapeutic approach.

Surveillance of infected cats

Serologic retesting at 6- to 12-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 adulcicidal therapy. Once adult heartworm infection has been diagnosed, monitoring will be most informative if both antibody and antigen testing are performed. The retesting interval should be consistent with the clinical circumstances. For asymptomatic cats, an annual retest may be adequate. Spontaneous or adulcicidal induced elimination of infection in antigen positive cats ordinarily will be followed within four to five months by disappearance of detectable antigenemia. Once cats become antigen negative and are clinically normal, further antibody retesting becomes optional since antibody may persist for an indefinite period after the parasites are gone and because continued exposure, even with preventive therapy, will result in a positive test. In those cats with pulmonary vascular and/or parenchymal lung disease, or in which heartworms have been identified echocardiographically, radiography and ultrasonography also may be very useful for monitoring the course of infection and disease.

Chemoprophylaxis

Monthly chemoprophylaxis is a safe and effective option for cats living in areas where heartworm infection is considered endemic in dogs and exposure to infective mosquitoes is possible. Many cats live more sheltered lives than do most dogs and are often confined indoors. Unless the home environment provides an effective barrier to the entrance of mosquitoes, these so called "indoor" cats may also be at risk. In one retrospective study, approximately twenty-five percent of cats diagnosed with adult heartworms were considered indoor cats. Care givers should be advised objectively of the potential risk of heartworm infection in their community and for their cat's living conditions. When monthly heartworm chemoprophylaxis is elected, it should at least be administered within 30 days following the estimated seasonal onset of transmission and be continued within 30 days after



that period has ended. Administering a preventive year-round also has merit due to the following reasons: (1) activity against some common intestinal parasites and in the case of selamectin, external parasites, (2) increased compliance, and (3) retroactive efficacy as a safeguard for inadvertent missed doses. (For a more detailed explanation, consult the Canine Guidelines under the heading "Macrocyclic Lactones")

Drugs

Heartworm chemoprophylaxis can be achieved in cats with monthly doses of either oral (ivermectin or milbemycin oxime), or topical (moxidectin or selamectin) preventive medications. Preventives should be started in kittens at eight weeks of age and be administered to all cats in heartworm endemic areas during the heartworm transmission season. The individual monthly prophylactic dose of ivermectin is 24 µg/kg, milbemycin oxime 2 mg/kg, moxidectin 1 mg/kg and selamectin 6-12 mg/kg of body weight. Administration of these drugs in cats is not precluded by antibody or antigen seropositivity.

Serologic testing

Since seroepidemiologic data for most communities is presently meager, it behooves veterinarians to become familiar with the local risk potential, by testing cats before initiating heartworm chemoprophylaxis. While guidelines are still being developed and evaluated, it is considered prudent to establish this serologic benchmark for future reference, in the event it becomes necessary to retest a cat receiving chemoprophylaxis. Although testing cats before starting chemoprophylaxis is recommended, there is less utility in doing so than is the case for dogs. This apparent contradiction reflects the differences in testing methods and test performance in the two hosts. Pretesting (screening) dogs is limited to documenting either heartworm antigenemia or circulating microfilariae, both of which are specific indicators of adult worm infection in a host that is

significantly more likely to become infected. Many, if not most, cats that are antibody positive have only been transiently infected to the 4th larval stage. Evidence of exposure of a cat to at least 4th stage larvae confirms the potential risk of developing Heartworm Associated Respiratory Disease (HARD) and reinforces justification for recommending chemoprophylaxis. The use of an antigen test to screen healthy cats is also an option, if one is fully aware of its limitations. (Refer to chart in Diagnostic Testing section for limitations of both antibody and antigen tests). However, the preferred method for screening includes the use of both an antigen and an antibody test.

Since microfilaremia in cats is uncommon, transient and below concentration levels that might trigger an adverse reaction to microfilaricidal chemoprophylactic drugs, pretesting for microfilariae is unnecessary. Furthermore, antibody retesting of cats already committed to chemoprophylaxis provides no assurance of efficacy since sensitization from repetitive aborted precardiac larval infections is possible in cats that are repetitively exposed. Therefore, the primary reasons for heartworm testing 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 that have already been diagnosed with feline heartworm disease,
3. and to establish a baseline reference prior to initiating chemoprophylaxis.

These guidelines are based on the latest information on heartworm disease. In keeping with the objective of the Society to encourage adoption of standardized procedures for the diagnosis, treatment and prevention of heartworm disease, they will continue to be updated as new knowledge becomes available.