Proceeding of the NAVC
North American Veterinary Conference
Jan. 8-12, 2005, Orlando, Florida

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HEART DISEASE: PROGNOSTIC INFORMATION FOR OWNERS

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INTRODUCTION
One of the first questions a client will ask their veterinarian after their pet receives a diagnosis of heart disease is “How long will she live?” As human beings, we tend to remember and prognosticate based on the most extreme cases or the last disastrous case, we are swayed by our feelings for the pet and the clients and are concerned about what decisions a client might make if we misstate the situation. Hard scientific data on survival of cats and dogs with naturally-occurring heart disease is available, but often represents a small population of purebred animals with specific diseases peculiar to the breed, animals treated in state-of-the-art facilities or with unlimited funds or animals treated with a specific medication (with no information on survival of the same disease treated differently). Despite this daunting list of problems related to prognostication, veterinarians perform this service every day, with profound consequences.

A closer examination of common findings affecting prognosis in cardiovascular disease allows identification of key moments in the pet’s course of disease and allows the clinician to individualize a prognosis to the patient.

THE CLINICIAN’S ROLE IN COMMUNICATING PROGNOSIS
As veterinarians, we are trained to consider medical factors in patients with disease. As the professional on a given case, we are responsible for relaying as much information as we can about the patient’s medical situation. Sociologic factors affecting prognosis begin with the information-relay step; the client’s understanding of the information has a large impact on decision-making. Several perceptions about the client may affect the manner in which information is relayed:

- client’s apparent understanding of medical terminology (any medical background?)
- client’s apparent understanding of the situation during the conversation (are questions appropriate?)
- client’s level of distraction (due to children, emotional responses, noise)
- client’s apparent emotional ability to “handle” the information
- client’s apparent financial ability to cope with further diagnostics or therapy

To avoid miscommunication, serious conversations involving important diagnostic and prognostic questions occur most optimally in quiet surroundings and without the pet present in most situations. Information should be given in bite size “chunks” with frequent verbal checks of understanding. It is helpful to stay sensitive to the client’s emotional state during the conversation, delivering the most necessary information first. Premature judgments regarding the client’s level of financial commitment or what would be “best” for the client’s perceived situation should be avoided.

It is the clinician’s responsibility to remain up-to-date on published data regarding survival times in common diseases. When evaluating survival studies, median survival time is usually reported. Median survival time is the interval at which 50% of the patients are dead, but it is important to remember that this number does not convey how long the remaining 50% lived and is not the “average” survival time. In diseases where survival of an initial episode is critical (i.e. acute aortic thromboembolism in a cat), many animals die or are euthanized immediately but those who do survive live significantly longer. In these animals, median survival time is an inaccurate reflection of a given animal’s prognosis.

THE CLIENT’S ROLE IN AFFECTING PROGNOSIS
Many pets with cardiovascular disease require therapy for their clinical signs (e.g. cough, difficulty breathing). Medications for these problems can vastly improve the quality of the patient’s life, but come at a cost to both client and patient. Most symptomatic cardiac diseases are ultimately fatal, but survival is artificially shortened when the client perceives that the recommended medications are making the patient more ill, are not improving the situation, cost too much, are too difficult to administer or are causing intolerable side effects in the patient or the household. Some methods of preventing these sorts of problems from affecting the patient’s survival include:

- informing the client in advance (and preferably in writing via a prepared handout) about a drug’s desired effect, possible side effects and ways to cope with the side effects
- assuring the client that changes in drugs and doses are common in the first two weeks of therapy and the need for changes is not a “bad sign”
- informing the owners which drugs need to be given on a regular schedule (usually drugs in which a stable blood concentration is necessary for optimal effects, e.g. anti-arrhythmic drugs) and which drugs can be safely administered an hour early or late (drugs with a very long half-life e.g. digoxin, or drugs not intended to attain steady state e.g. furosemide)
- prioritizing medications if cost is an issue

PROGNOSIS IN CASES OF CONGENITAL HEART DISEASE
Little information has published regarding the natural history of most types of congenital heart disease, especially if the disease is untreated. Survival information regarding patients with treated patent ductus arteriosus (PDA) or dogs with subaortic stenosis has been published. Most other congenital heart diseases such as mitral dysplasia (MVD), tricuspid dysplasia (TVD), ventricular septal defect (VSD) atrial septal defect (ASD) or pulmonic stenosis (PS) follow some general rules that can be adapted for the individual animal. Because of the paucity of specific survival information in many congenital heart diseases, the clinician is required to concentrate more on analyzing “good signs” and “bad signs” regarding a patient’s condition and projected survival rather than concentrating on remembering specific timeframes (Table 1).

Ultimately, animals with severe clinical signs of non-surgical disease at a young age are more likely to have a very limited life span (weeks to months from the time of diagnosis). Animals with severe anatomic changes and cardiac enlargement but no clinical signs might be expected to live several years (exception: some dogs with severe tricuspid valve dysplasia may live to be middle aged despite horrifying echocardiographic findings). Animals with less severe anatomic changes and minimal cardiac enlargement...
may live well into middle age, and animals with small defects and relatively normal cardiac size radiographically and echocardiographically might live a normal life span. In all cases, the clients should be warned that complications such as arrhythmias or vegetative endocarditis might shorten the animal’s life span unexpectedly.

SPECIFIC CONGENITAL HEART DISEASE CONDITIONS

Patent Ductus Arteriosus

PDA remains the “best” congenital heart disease to have – surgical ligation or coil embolization of the ductus usually results in a relatively normal life span for the pet. Exceptions include animals with other concurrent congenital abnormalities or severe myocardial failure (systolic dysfunction) and congestive heart failure (CHF) accompanying the PDA. These animals may recover fully after ductal ligation, or may require lifelong therapy and still have a shortened life span. Most untreated PDAs will result in CHF within 1-3 years; some untreated animals may develop pulmonary hypertension at an early age that reverses flow in the PDA and renders it untreatable surgically (i.e. reversed PDA). Rarely, animals will live a normal life span with a very small untreated ductus.

Subaortic Stenosis

Subaortic stenosis is a common congenital heart defect and varies widely in severity. In 1994, a comprehensive survival study grouped dogs with SAS according to severity of obstruction. Dogs were divided into groups with “mild” (transvalvular gradient < 35 mmHg), “moderate” (gradient 36-80 mmHg) or “severe” (> 80 mmHg) stenosis. Dogs with mild obstructions usually remained asymptomatic and had normal life spans. Dogs with moderate obstructions were more likely to develop clinical signs of SAS, but still usually lived into middle age. The complications of left heart failure or infective endocarditis occurred more frequently in these groups than in the more severely affected dogs, perhaps because mild or moderately affected dogs lived longer. Severely affected dogs had a median survival significantly shorter (18.9 mos) than mildly (51.1 mos) or moderately (30.5 mos) affected dogs. Severely affected dogs were 16 times more likely to die a sudden death than less severely affected dogs.

PROGNOSIS IN CASE OF ACQUIRED HEART DISEASE

As is the case in congenital heart disease, several broad generalizations can be made that affect survival in pets with acquired heart disease in which the underlying cause is unknown or unfixable. These generalizations involve the nature of the individual disease as well as medical and owner-related factors (Table 2).

Table 1: Clinical findings in congenital heart disease patients and their effect on prognosis*

<table>
<thead>
<tr>
<th>Findings associated with a better prognosis</th>
<th>Findings associated with a worse prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• well-grown, energetic and inquisitive patient, the same size as littermates</td>
<td>• stunted, lethargic or excessively quiet patient, smaller than littermates</td>
</tr>
<tr>
<td>• no clinical signs of disease (exercise intolerance, cyanosis, difficulty breathing, ascites)</td>
<td>• clinical signs of heart failure, cyanosis or arrhythmias are present</td>
</tr>
<tr>
<td>• no concurrent disease (e.g. hydrocephalus, porto-systemic shunt)</td>
<td>• concurrent disease is known or suspected</td>
</tr>
<tr>
<td>• loud murmur is good if a VSD is present</td>
<td>• loud murmurs are bad in stenotic lesions (PS, SAS), regurgitant lesions (MVD, TVD) or if an ASD is present</td>
</tr>
<tr>
<td>• no arrhythmias</td>
<td>• radiographic and echocardiographic findings show major anatomic abnormalities accompanied by significant chamber enlargement</td>
</tr>
<tr>
<td>• radiographic and echocardiographic findings show minimal cardiac enlargement and contractile function is normal</td>
<td></td>
</tr>
</tbody>
</table>

* applicable to congenital heart diseases that are not correctable with surgery or other invention

Table 2: Clinical findings in acquired heart disease patients and their effect on prognosis

<table>
<thead>
<tr>
<th>Findings associated with prolonged survival</th>
<th>Findings associated with a worse prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• minimal cardiac enlargement</td>
<td>• presence of CHF</td>
</tr>
<tr>
<td>• no CHF or arrhythmias</td>
<td>• indicates that disease has been present chronically</td>
</tr>
<tr>
<td>• no concurrent diseases that affect drug therapy (e.g. renal failure)</td>
<td>• indicates that the heart’s ability to compensate has been exceeded</td>
</tr>
<tr>
<td>• brisk response to initial therapy with minimal side effects</td>
<td>• introduces the effects of medication to survival</td>
</tr>
<tr>
<td>• hearty appetite</td>
<td>• significant cardiac enlargement</td>
</tr>
<tr>
<td>• dedicated, observant and creative caregiver</td>
<td>• decreased systolic function</td>
</tr>
<tr>
<td></td>
<td>• severe anatomic derangements</td>
</tr>
<tr>
<td></td>
<td>• disease is associated with catastrophic complications (e.g. aortic thromboembolism)</td>
</tr>
<tr>
<td></td>
<td>• concurrent diseases limit drug therapy or lead to side effects</td>
</tr>
<tr>
<td></td>
<td>• drugs are poorly tolerated or administered inconsistently</td>
</tr>
</tbody>
</table>
SPECIFIC ACQUIRED HEART DISEASES

Feline Hypertrophic Cardiomyopathy (HCM)

Asymptomatic cats with idiopathic HCM can span a wide range of phenotypes and therefore, the asymptomatic form of the disease has an unpredictable course. Cats with echocardiographically evident HCM but no clinical signs have a median survival time of 5 years or more, but cats with heart failure have a shorter median survival, ranging from 92-563 days.5,6

HCM with Thromboembolic Complications

Development of aortic thromboembolism (ATE) severely decreases survival times for cats with HCM for several reasons. Cats that develop ATE as a complication of HCM frequently have severe cardiac pathology and CHF, contributing to decreased survival. In addition, the clinical signs of ATE are often dramatic and painful, with a history of limited success with therapy and a well-publicized poor prognosis for recovery. This constellation of findings and facts often leads to rapid euthanasia in affected animals. Published survival times for cats with HCM and ATE range from 61 to 184 days.5,6 When all feline cardiac diseases are included, the mean survival for cats with ATE who survived the initial thrombotic event (37% of cats) was 11.5 months, with 28% of cats dying during the initial event and 35% euthanized7.

Feline Atrial Fibrillation (AF)

Atrial enlargement provides a substrate for the occurrence of AF in all species and is frequently associated with significant significant heart disease and heart failure. In a recent retrospective study of 50 cats with AF, the most common underlying disease was restrictive cardiomyopathy and 56% of cats with AF were presented with signs of heart failure. Interestingly, in 23% of cats studied, AF was an incidental finding. Survival was longer than expected, with an overall median survival of 165 days. Presence of clinical signs at the time of diagnosis did not have a significant effect on survival8.

Canine Mitral Valve Insufficiency (MVI)

The prognosis for survival in dogs with MVI due to endocardiosis is generally better than survival in dogs with dilated cardiomyopathy. Typically, the onset of the heart murmur of MVI is first noted in middle age (approximately 6-9 years old). Dogs may remain without clinical signs for years or indefinitely, but once clinical signs are apparent, some generalizations can be made.9,10 The median survival for dogs presented with severe CHF due to MVI is approximately 7 months, with 75% of dogs dead by one year. If the CHF is less severe, median survival is approximately one year, with 75% dead by 21 months. Cavalier King Charles Spaniels have a more accelerated version of MVI secondary to endocardiosis. Typically, dogs of this breed tend to develop murmurs at a young age (2-4 years old) and seem to progress more rapidly to heart failure.

CANINE DILATED CARDIOMYOPATHY (DCM)

The presence of CHF at the time of diagnosis has a significant negative effect on survival in dogs with idiopathic DCM. Overall, a diagnosis of DCM in a dog has an overall survival rate of approximately 20% at one year,11 but once a dog with DCM has signs of CHF, the median survival is between 2 and 3 months and about 75% of the dogs are dead by 6 months.9,12 Approximately 20% of dogs with mild CHF will survive to one year. While these statistics are daunting, it is important to remember that many dogs are euthanized soon after diagnosis due to poor prognosis rather than because of severe clinical signs. This group is likely to skew the survival statistics downward. Doberman pinscher DCM is particularly malignant with median survival times post-diagnosis of approximately 6 weeks13, about 20% of affected dogs die suddenly during this period. Newer medications (e.g. pimobendan) show promise for increasing survival times in DCM dogs14.

REFERENCES