Feline Hypertrophic Cardiomyopathy

“Treatment”

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Treatment of Feline Hypertrophic Cardiomyopathy

LOST DREAMS

Consensus Statement - Human
Because of the relatively low prevalence of HCM in general cardiologic practice, its diverse presentation, and mechanisms of death and disability and skewed patterns of patient referral, the level of evidence governing management decisions for drugs or devices has often been derived from non-randomized and retrospective investigations. Large-scale controlled and randomized study designs, such as those that have provided important answers regarding the management of coronary artery disease and congestive heart failure, have generally not been available in HCM as a result of these factors. Therefore, treatment strategies have necessarily evolved based on available data that have frequently been observational in design, sometimes obtained in relatively small patient groups, or derived from the accumulated clinical experience of individual investigators, and reasonable inferences drawn from other cardiac diseases. Consequently, the construction of strict clinical algorithms designed to assess prognosis and dictate treatment decisions for all patients has been challenging and has not yet achieved general agreement.

In some clinical situations, management decisions and strategies unavoidably must be individualized to the particular patient.

Treatment of Feline HCM Prior To The Onset Of Heart Failure
The Need to Treat

TV Star Spokesperson and Feed My Brain user
Lisa Whelchel

First Do No Harm

A DANGEROUS PLAN IS BETTER THAN NO PLAN
Treatment of Feline HCM Prior To The Onset Of Heart Failure

- There is a long list of drugs that veterinarians use in an attempt to try to prevent the progression of the feline hypertrophic cardiomyopathy
  - Beta blockers (atenolol)
  - Diltiazem
  - ACE inhibitors
  - Spironolactone
  - Supplements
  - Acupuncture
  - Homeopathy
  - Witchcraft

Treatment of Feline HCM Prior To The Onset Of Heart Failure

- Severe HCM with no heart failure
  - No evidence that any medical therapy alters the natural history of the disease
  - Beta blockers commonly administered to decrease SAM, if severe
    - Also, however are administered by some when SAM is not present
    - Decreases heart rate and so prolongs filling period
  - Diltiazem administered for theoretical reasons
    - Occasionally may result in ↓ in wall thickness (??)
    - Older studies suggested benefit
    - Currently not used by most veterinary cardiologists
  - ACE inhibitors
    - Two older studies suggested they can decrease wall thickness
    - Theoretically might be able to reduce interstitial myocardial fibrosis
    - Recent study shows neither of these happen
Treatment of Feline HCM Prior To The Onset Of Heart Failure

- Beta blockers
  - Reduce heart rate or prevent tachycardia during stress
    - Better than diltiazem
  - Reduce systolic anterior motion (SAM) of the mitral valve
    - Better than diltiazem
  - Do not directly improve diastolic function
  - Atenolol (25 mg tablets) – 6.25 to 12.5 mg q12 hours

- Beta Blockers
  - Most common medical treatment for HCM in humans
  - Traditionally given to patients with and without obstruction (SAM)
  - Assessment of benefit usually relies on the patient’s subjective perception
    - With day-to-day variability in symptoms it becomes very difficult to tell what to ascribe to drug and what to ascribe to chance in any individual patient
  - Propranolol is the traditional beta blocker used
    - Doses in adults range up to 480 mg (7 mg/kg) per day and some have used up to 1000 mg/day
    - Atenolol, metoprolol and nadolol are also used
Treatment of Feline HCM Prior To The Onset Of Heart Failure

- Systolic anterior motion of the mitral valve – how important is it?
  “In humans, although it has previously been subject to periodic controversy, there is now widespread recognition that the sub-aortic gradient (30 mm Hg or more) and associated elevations in intra-cavity LV pressure reflect true mechanical impedance to outflow and are of pathophysiological and prognostic importance to patients with HCM. Indeed, outflow obstruction is a strong, independent predictor of disease progression to HCM-related death (relative risk vs. non-obstructed patients, 2.0), to severe symptoms of New York Heart Association (NYHA) class III or IV, and to death due specifically to heart failure and stroke (relative risk vs. non-obstructed patients). Outflow gradients are responsible for a loud apical systolic ejection murmur associated with a constellation of unique clinical signs.” American College of Cardiology/European Society of Cardiology Clinical Expert Consensus Document on Hypertrophic Cardiomyopathy. Journal of the American College of Cardiology Vol. 42, No. 9, 2003.

Effect of a Beta Blocker on SAM

![Effect of a Beta Blocker on SAM](image)
How important is SAM?

- In humans, the clinical signs associated with SAM primarily occur during exercise/exertion.

<table>
<thead>
<tr>
<th>Table 1. Effects of Nadolol and Verapamil on Exercise Capacity</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Exercise duration (min)</td>
<td>30 ± 4</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>26 ± 7</td>
</tr>
<tr>
<td>Mean arterial blood pressure (mmHg)</td>
<td>17 ± 6</td>
</tr>
</tbody>
</table>

- Cats are said to sleep 85% of their life
  - An indoor cat may never exert itself
  - An indoor/outdoor cat may exert itself once a month (?)
  - What’s better at calming down the sympathetic nervous system?
    - A beta blocker?
    - Sleep?
Treatment of Feline HCM Prior To The Onset Of Heart Failure

- Beta blockers are used in humans for symptomatic relief and most of the improvement comes in symptoms seen during some degree of exercise. Most pet cats don’t exercise at all and they are said to spend 85% of their life asleep. IMHO sleep is much better at keeping sympathetic tone low than is any drug.

Treatment of Feline HCM Prior To The Onset Of Heart Failure

Rarely a cat will get extremely stressed (e.g., cat fight, chased by a dog, taken to a veterinarian) and it’s possible that having a beta blocker on board might be beneficial a these times.
Treatment of Feline HCM Prior To The Onset Of Heart Failure

Diltiazem
- Myocardial (L-type) calcium channel blocker
- Experimental (theoretical) evidence
  - Improves ventricular relaxation in cats with HCM (Bright, et al)
  - No effect on compliance
  - Prevents diastolic dysfunction in one transgenic mouse model of HCM
  - Restores SR proteins and normal calcium cycling in another transgenic mouse model of HCM
- Clinical evidence of efficacy in cats
  - Lacking
Calcium Channel Blockers – Effect on myocardial relaxation

Verapamil and Propranolol Have No Effect on Diastolic Stiffness

Hess OM, Grimm J, Krayenbuehl HP Eur Heart J 1983; 4: F-47-56
Treatment of Feline HCM Prior To The Onset Of Heart Failure

Diltiazem

- My general clinical impression over years of use is that diltiazem is not an effective drug for feline HCM, either when a cat is in heart failure or prior to the onset of heart failure
- In humans, the calcium channel blocker verapamil is used almost exclusively.
  - It’s primary use is to relieve chest pain and exertional dyspnea
  - I’ve never see a cat that I thought had chest pain due to HCM and cats rarely exert themselves

SAM - Calcium Channel Blocker vs. Beta Blocker

COMPARISON OF THE EFFICACY OF INTRAVENOUS DILTAZEM AND ESMOLOL TO REDUCE LEFT VENTRICULAR OUTFLOW TRACT VELOCITY AND HEART RATE IN CATS WITH HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY: Aaron C. Wey and Mark D. Kittleson
University of California, Davis.

Diltiazem and beta-adrenergic blockers are frequently used to treat cats with hypertrophic cardiomyopathy (HCM) and systolic anterior motion (SAM) but evidence is lacking as to which drug or drug class is more efficacious at reducing heart rate (HR) and left ventricular outflow tract (LVOT) velocity in these cats. This study was performed to determine the relative efficacies of an intravenously administered beta-blocker (esmolol) and intravenously administered calcium channel blocker (diltiazem) for reducing HR and LVOT velocity in cats with HCM and SAM. Seven cats were selected for study based on echocardiographic evidence of HCM and LVOT obstruction secondary to SAM. Cats selected had to have a minimum LVOT velocity of 2.0 m/sec (range = 2.3 to 6.3 m/sec) with a late-peaking continuous wave Doppler tracing characteristic of a dynamic obstruction. Baseline measures of HR and LVOT velocity (left apical view) were recorded over time until the measures were stable. A slow IV bolus of esmolol (0.5 mg/kg) was then administered while monitoring the patient’s electrocardiogram and LVOT velocity. The minimum HR and LVOT velocity were recorded. Following a washout period of fifteen minutes or a return to baseline HR and LVOT velocity, the procedure was repeated using a slow IV bolus of diltiazem (1mg/kg). Statistically significant differences between treatment groups were determined using one-way repeated measures ANOVA and Bonferroni Dunn post-ANOVA testing. A p-value < 0.05 was considered significant. Pre-treatment HR and LVOT velocity were 190 +/- 25 (mean +/- SD) bpm and 4.0 +/- 1.4 m/sec, respectively. Treatment with esmolol resulted in significant reductions in HR (to 165 +/- 22 bpm; p=0.007) and LVOT velocity (to 1.5 +/- 0.5m/sec; p=0.001), respectively, whereas treatment with diltiazem only caused a significant reduction in LVOT velocity to 2.6 +/- 0.9 m/sec (p=0.003). In addition, esmolol significantly decreased LVOT velocity more than did diltiazem (p=0.008). This study is limited in that systemic arterial blood pressure was not measured during drug administration. Pressure gradient across the LVOT can be altered by changes in blood pressure, and both drugs can cause a decrease in blood pressure. Except for a mild and transient increase in heart rate following diltiazem administration, there was no clinical evidence of systemic hypotension during this study. Consequently, these data suggest that esmolol may be a significantly more efficacious drug for acutely reducing HR and LVOT velocity than diltiazem in cats with SAM due to HCM.
Treatment of Feline HCM Prior To The Onset Of Heart Failure

**Renin-angiotensin-aldosterone system**

- **ACE Inhibition**
  - Two small studies suggested the an ACE inhibitor can decrease LV wall thickness in cats with HCM
    - 19 cats studied *retrospectively*
    - Included any cat with HCM that was examined in a 6 year period that was placed on enalapril for at least 3 months that had a follow-up exam within 6 months of diagnosis
    - Septal and free wall thicknesses were thought to decrease 1 mm on average
    - Left atrial size decreased 2 mm
Treatment of Feline HCM Prior To The Onset Of Heart Failure

• ACE Inhibition
    • 28 cats in an open-label trial
    • 5 different veterinary hospitals enrolled patients
    • M-mode used to measure LV wall thicknesses
    • Supposed to be randomly assigned to benazepril plus standard therapy (diltiazem +/- furosemide) or standard therapy plus benazepril (0.5 mg/kg/day)
    – 19 cats in benazepril group and 9 in the other group
    • Variable times in study from 3 to 12 months
    • Resultant plots of LV wall thicknesses over time look like what one would expect if one were to look at a group of cats with HCM over time – a lot of random variability with a few cats that had what appeared to be marked changes in wall thickness
    • Concluded that cats on benazepril experienced a 0.1 mm/month decrease in wall thickness

• ACE Inhibition
  – Methods
    • 26 Maine Coon and Maine coon cross cats with HCM
    • LV Mass measured using cardiac MRI
    • Cats divided into two equal groups and matched for LV mass index
    • One group on ramipril (0.5 mg/kg/day) and one on placebo for one year
    • Measured LV mass index and delayed enhancement (myocardial fibrosis) at baseline, 6 months and one year
    • Measured DTI, plasma aldosterone %
    • BNP concentrations and systolic blood pressure every 3 months
    • Plasma ACE activity measured in 16 cats
Treatment of Feline HCM Prior To The Onset Of Heart Failure

- **ACE Inhibition**
  

  - Results
    - No change in LV mass index over time
    - No change in diastolic enhancement over time
    - No change in early diastolic velocity measured via DTI over time
    - No change in systolic blood pressure
    - Appropriate therapeutic decrease in plasma ACE activity documented the dose used was correct
    - Despite the decrease in plasma ACE activity there was no decrease in plasma aldosterone concentration

  - Conclusion
    - ACE inhibition has no apparent effect on LV mass or diastolic function in cats with HCM

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**Ramipril in HCM**

![Graphs showing treatment effects](image-url)
**Spironolactone**

**Spironolactone in HCM**

- **Spironolactone is an aldosterone antagonist**
  - Usually classified as a diuretic (poor)
  - Aldosterone has effects other than sodium and water retention
    - Promotes the formation of interstitial fibrosis in the myocardium
    - Stimulates myocardial hypertrophy
  - In a transgenic rat model of HCM (troponin T) spironolactone reversed myocardial fibrosis and reduced myofiber disarray (Circulation 2004)
  - TOPCAT (Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist) is a current ongoing trial to look at spironolactone in human patients with severe diastolic dysfunction
  - Study was designed to look at the effect of spironolactone in a group of research cats with HCM
Spironolactone in HCM

Effect of Spironolactone on Diastolic Function and Left Ventricular Mass in Maine Coon Cats with Familial Hypertrophic Cardiomyopathy

- K.A. MacDonald, M.D. Kittleson, and P.H. Kass

• Methods
  - 26 cats randomized (paired and based on diastolic function) to either placebo or spironolactone (2 mg/kg PO q12 hours for 4 months)
  - All cats had moderate to severe HCM
  - Echocardiogram used to estimate LV mass at baseline and at 2 and 4 months
  - Tissue Doppler imaging performed to assess diastolic function
    - All cats had an E/Em < 10 cm/s with the majority in the 5-8 cm/s range
  - Plasma aldosterone measured in the morning after being sedated and lying quietly for 20 minutes
  - Blood pressure normal in all cats
Spironolactone in HCM

Results

- There was no effect on diastolic function as measured by tissue Doppler imaging.
- There was no effect on left ventricular mass index.

Spironolactone in HCM

Results

- Plasma aldosterone concentration increased dramatically in the cats on spironolactone at 2 and 4 months.
- This documented that the cats were receiving the medication and the dose was adequate.
Spironolactone in HCM

Adverse Effects
- Four of the 13 cats on spironolactone developed skin lesions in 2-3 weeks
  - Severe ulcerative facial dermatitis
  - Severe, diffuse, subacute necrotizing and ulcerative dermatitis with superficial to deep eosinophilic and neutrophilic perivascular dermatitis
  - Herpes virus on PCR present in 1 cat
  - Lesions severe enough to necessitate stopping the drug
  - Resolved in 4-6 weeks on antibiotic and corticosteroid therapy

Treatment of Feline HCM Prior To The Onset Of Heart Failure


The course of myocardial hypertrophy in hypertrophic cardiomyopathy. Results of a 10-year follow-up.

Gregor P, Widimský P
2nd Department of Internal Medicine, 3rd School of Medicine, Charles University, Prague, Czech Republic.

Sixty-nine patients with hypertrophic cardiomyopathy were followed up for a minimum period of 10 years, with detailed quantitation of myocardial hypertrophy using two-dimensional echocardiography at a two-year interval. The parameters determined included maximum myocardial wall thickness, mean myocardial wall thickness (defined as the arithmetic mean of myocardial wall thickness values measured in ten areas of approximately the same size the LV and septal myocardium had been divided into), and the extent of hypertrophy (percentage of the myocardium affected by hypertrophy). Other echocardiographic parameters as well as clinical, ECG and Holter data were also assessed. Seven patients (10%) showed a gradual increase in mean myocardial wall thickness (associated with an increase in maximum myocardial wall thickness and in the extent of hypertrophy in two). By contrast, a progressive decrease in hypertrophy was observed in six patients (9%). In either case, the observed changes tended to involve areas in segments outside the zone of maximum wall thickness. Generally, the changes in hypertrophy were not related to the development of clinical or Holter manifestations, and were unaffected by verapamil or beta-blocker therapy. There was no death among these patients unlike the five deaths in a group with stationary hypertrophy. Based on their results, the authors conclude that changes in myocardial hypertrophy (regardless of whether there is progression or regression) probably are part of natural variations in the course of the disease, and have no clinical or prognostic implications for the majority of patients.
Treatment of Feline HCM Prior To The Onset Of Heart Failure

My experience in my colony of Maine Coon and Maine Coon cross cats over the past 15 years

- No cat in the colony has been given a beta blocker, diltiazem or an ACE inhibitor prior to the onset of heart failure (and then usually only furosemide)
- Even cats with severe HCM have only been observed
- The natural progression of the disease is highly variable
  - There are a few cats that progress rapidly to heart failure and death
  - There are others that develop severe HCM and then stabilize for years
- I'm firmly convinced that it I had decided to treat these cats with a standard drug or drug regimen that I would firmly believe by now that whatever drug(s) I chose worked quite well.

Treatment Decision Making in Cats with HCM Prior to HF
Treatment of Feline HCM

So what do I do?

- I used to flip a coin in cats with moderate to severe HCM that were not in heart failure
  - Heads got diltiazem and tails got atenolol
- I’ve stopped doing that
- Currently I only treat a cat that is not in heart failure if there is a severe (>80 mmHg) pressure gradient across the dynamic left ventricular outflow tract obstruction and that cat gets atenolol
- No other cat with HCM without heart failure gets treated

Heart Failure Due to HCM
Treatment of Heart Failure Due to HCM

- No definitive therapy
- Palliative therapy for heart failure
  - Furosemide
  - Angiotensin converting enzyme (ACE) inhibitor
  - Pleurocentesis
  - No reason to stop beta blocker when the cat goes into heart failure
- Try to prevent clot formation in left atrium
  - Classically aspirin is administered
    - Unfortunately it does not work
  - Plavix – current study under way (FATCAT)
  - Low molecular weight heparins (expensive)
- Prevent sudden death (?)

Treatment of Heart Failure Due to HCM

- Patients in heart failure
  - Furosemide
    - Primary therapy for pulmonary edema and pleural effusion
    - Most efficacious drug for treating heart failure
    - 1 to 2 mg/kg q 8-12 hours most commonly
  - ACE inhibitors
    - Anecdotal evidence suggests that these drugs help improve clinical signs and prolong life
    - Enalapril (0.5 mg/kg q12-24 hours)
      - Most commonly give 2.5 mg/day/cat
  - Pleurocentesis
Rules of Furosemide Therapy

- Keep the patient dry
- Keep the sleeping respiratory rate less than 40 breaths/min
- Do not worry about mild to even moderate pre-renal azotemia
  - Furosemide is not nephrotoxic
- Do not give parenteral fluid therapy and furosemide simultaneously
  - This makes no sense
  - You need to decide at any point in time, which is needed
    - For example, if a cat is severely dehydrated and moderately azotemic and the sleeping RR is normal – stop furosemide and give fluids

Feline HCM
Feline HCM
Feline HCM

ACE Inhibition in Cats with Heart Failure Due to HCM

- Which ACE Inhibitor?
  - Whichever one is least expensive and easiest to administer
  - There is no evidence that one ACE inhibitor is better than another
  - Benazepril does not have fewer renal side effects (i.e., functional azotemia) than other ACE inhibitors in cats or dogs
Benazepril

About 50% of benazepril is metabolized by the liver and 50% is excreted via the kidneys vs. 100% excretion by the kidneys for other ACE inhibitors

- ACE inhibitors are all or nothing drugs
- You have to suppress over 80% of ACE to have a pharmacologic effect and 100% is no better than 80%.
- It takes 30 times the normal dose of enalapril to produce true renal toxicity.
- So what happens if not as much enalapril is excreted renally in a patient that is azotemic? Even if the plasma concentration were to go up 10-fold there would be no untoward effects.

What About the Use of Diltiazem or a Beta Blocker in Cats with HCM That Are in Heart Failure?
Diltiazem (Bright et al; 1991)

Evaluation of the Calcium Channel-Blocking Agents Diltiazem and Verapamil for Treatment of Feline Hypertrophic Cardiomyopathy

To determine the efficacy of and clinical response to several pharmacologic agents for treatment of idiopathic hypertrophic cardiomyopathy in cats, 17 symptomatic cats were randomized to treatment with either propranolol, diltiazem, or verapamil. Clinical, laboratory, radiographic, electrocardiographic, and echocardiographic data were obtained before treatment and after 3 and 6 months of chronic oral therapy. Too few of the cats receiving propranolol or verapamil survived long enough to obtain long-term data needed to make statistical comparisons between groups. However, all 12 cats ultimately treated with diltiazem became asymptomatic, and no adverse effects from this drug were noted in any of these cats. Treatment with diltiazem was associated with a significant reduction of pulmonary congestion assessed radiographically (P < 0.01), and improved ventricular filling based on echocardiographic measurements of left atrial size (P < 0.05), left ventricular internal diastolic dimension (P < 0.05), and relaxation time index (P < 0.001). There was also a drug-related improvement in jugular venous oxygen tension (P < 0.001) and blood lactate concentration (P < 0.01) suggesting improved peripheral perfusion in the cats receiving diltiazem. The results indicate that diltiazem provides an effective and apparently safe treatment for the management of feline hypertrophic cardiomyopathy.

Diltiazem (Bright et al; 1991)
Diltiazem (Bright et al; 1991)

Diltiazem (Bright et al; 1991)
Fox – ACVIM 2003

Prospective, Double-Blinded, Multicenter Evaluation of Chronic Therapies for Feline Diastolic Heart Failure: Interim Analysis.

Diastolic heart failure (HF) presenting as pulmonary edema is the most prevalent cause of symptomatic heart disease in felines. Optimal treatment strategies have been confounded by lack of comparative outcome data, and no clinical trials are available to support the use of one agent over another.

To initiate an evidence-based approach to chronic heart failure management, we began a prospective, double-blinded, placebo-controlled, multicenter clinical trial in May, 1998.

Cats that survived first onset of HF were assigned to one of 4 treatment groups in a complete randomized block design. Accordingly, all cats received furosemide (1.1-2.5mg/kg q24h). In addition, each received either placebo (referred to as placebo group), atenolol (1.1-2.5mg/kg), Dilacor (30mg/cat), or enalapril (0.4-0.6mg/kg) once daily. We assessed time to HF recurrence, time on study, and mortality. Data is analyzed on the intention-to-treat principle. This study is ongoing and blinding is maintained for active treatment groups (referred to as group 2, 3, and 4).

We enrolled 118 cats with HF (57 HCM, 37 HOCM, and 24 RCM or UCM). Proportion of disease categories (HCM, HOCM, Other) were not different across treatment groups (P=0.18). Patient characteristics with respect to groups placebo, 2, 3, and 4 were: body weight (mean: 5.5, 5.1, 5.7, 4.9kg; P=0.088); age (median: 8.7, 6, 8, 5 years; P=0.154); breed (DSH: 70%, 73%, 88%, 66%; P=0.86); and gender (male, 77%, 54%, 78%, 66%; P=0.8), respectively. Comparing time to 25% HF recurrence in a cohort of 39 cats indicated that the placebo group (furosemide alone) remained compensated the longest (254 days). Group 4 did much worse than placebo (92 days, hazard ratio, 202%, P=0.03). Group 2 (99 days, hazard ratio, 144%) and group 3 (153 days, hazard ratio, 124%) appeared to have a poorer outcome vs. placebo, though probabilities were P=0.22 and P=0.47, respectively. Time on study (comparing placebo to group 2, 3, and 4) was shortest for group 4 (hazard ratio 139%, P=0.11); group 3 was slightly worse than placebo (hazard ratio 113%, P=0.6), while group 2 performed slightly more favorably (hazard ratio 74%, P=0.19). All cause mortality was 58.5%. Death due to heart failure and/or thromboembolism occurred in 63 of these 69 cats. Overall mortality was not affected by treatment when considering the entire study population (P=0.8) or subgroups (DSH cats only, P=0.6, or just HOCM and HCM cohorts, P=0.8).

Thus, at interim analysis, there was little evidence to suggest that chronic administration of furosemide plus either atenolol, Dilacor, or enalapril at doses specified, provided any major advantage in clinical outcome vs. administration of furosemide alone.

Fox – ACVIM 2003

Enrolled cats with cardiogenic pulmonary edema due to either HCM (n=94) or unclassified cardiomyopathy (n=24) over several years (total=118)

Randomly assigned to receive:

- Placebo
- Atenolol (1.1-2.5 mg/kg once a day)
- Dilacor (sustained release diltiazem; 30 mg once a day)
- Enalapril (0.4-0.6 mg/kg once a day)

Of the 118 cats, 69 (58%) died during the study

- 63 of these died of heart failure and/or thromboembolism

Neither diltiazem or enalapril had any appreciable effect

Atenolol appeared to negatively affect outcome (survival)
Fox – ACVIM 2003

Problems with the study
- Both cats with HCM and UCM were enrolled
- Atenolol was only given once a day (effect lasts 12 hours or so)
- Dilacor was only given once a day (effect lasts 12-14 hours)
- Furosemide dose was limited to a maximum of 2.5 mg/kg per day

Feline Hypertrophic Cardiomyopathy

• Prognosis
  - Mild to moderate HCM
    • May progress to severe HCM, especially in a young cat
    • If no progression occurs the prognosis is often good
  - Severe HCM
    • Short-term prognosis is guarded
    • Long-term prognosis is guarded to poor
      - Heart failure may occur
      - Sudden death may happen
      - Systemic thromboembolism may rear its ugly head
  - Severe HCM with heart failure
    • Long-term prognosis is poor (usually months)
The Art of Wishful Thinking

• I can't think of a more powerful but false diagnostic and therapeutic tool than wishful thinking." (Kittleson 2008)

• Nothing is easier than self-deceit. For what each man wishes, that he also believes to be true. (Demosthenes; circa 385-322 BC)