Feline Symposium

In association with the
North American Veterinary Conference

Tuesday, January 22, 2013

Objectives

The 2013 ROYAL CANIN® Feline Symposium will provide a practical look at many of the most common preventive and therapeutic topics affecting feline medicine today. Experts from many fields will translate the latest diagnostic and therapeutic advancements into useful ideas that can be immediately utilized in your practice.

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MULTIPLE CATS, MULTIPLE NEEDS

The goal within a multi-cat household is to: 1) achieve a feeding strategy that puts no one at risk nutritionally having the base/common denominator diet available to everyone, as well as to 2) meet any additional individual nutritional needs of each member of the group as closely as possible twice a day behind closed doors. This requires analyzing the clowder’s nutritional needs, personalities and physical abilities.

In order to determine what doesn’t put anyone at risk, we need to think about what disease condition is most responsive to or, the reverse, most damaged by feeding the wrong diet. In other words, which cat is most fragile, from a nutritional point of view? Cats are obligate carnivores. This concept is central to understanding the nutritional needs of cats and planning dietary therapies for health disorders, especially when dealing with multiple cats with differing health considerations. We need to review basic nutritional needs of this species before we can decide what modifications can be made safely.

FOOD, FEEDING AND NUTRITION IN A FELINE CONTEXT

Cats diverged from dogs approximately 30 million years ago, evolving metabolically into obligate carnivores with unique strategies for the utilization of protein and amino acids, fats and vitamins. This concept must be at the centre of trying to understand the nutritional needs of cats and planning dietary therapies for health disorders. Domestic cats have not evolved from the wild cat model. They are anatomically and physiologically adapted to eating as many as 10-20 small meals, (a reflection of their hunting behaviour), throughout the day and night. This allows them to hunt and eat when their prey are active. Small rodents make up the majority of their diet, with rabbits, birds, insects, frogs and reptiles making up a smaller proportion. The average mouse provides ~8% of an average feral (i.e. active, unaltered) cat’s requirements. Repeated hunting throughout a 24-hour period is needed to meet this need, resulting in the normal grazing behaviour of domestic cats.

Being obligate carnivores has affected everything about cats: their hunting behaviour; that they eat many small meals a day alone; the small size of their stomach; their lack of salivary amylase; their social structure. Cats are hunters, yet the drive to hunt is independent from the need to eat. Hence, feeding more food doesn’t stop them from killing birds or mice, it merely makes them gain weight. Most cats needs 10-15 attempts to be successful at killing prey, thus the drive to “eye, stalk, pounce and kill” is permanently turned-on to avoid starvation. The average mouse provides 30-35 kcal of energy. Needing 50 kcal/kg ideal weight/day, the 5 kg cat needs 250 kcal or 8 mouse-sized portions/day. These meals are spread out throughout the day, not consumed all at once.

Feeding twice a day or having a bowl that is never empty are not “natural” ways for cats to eat. A 30 kcal meal is approximately 10 pieces of an average maintenance dry food; even eating 10 extra pieces/day results in a 10% (1 lb) weight gain/year. Our need for interaction with our cats also contributes to obesity. Cats generally interact with us frequently and at a low intensity/casually; people generally want fewer, more intense/focused periods of interaction with them. Eating is not a social activity for cats. We may feel like a bad provider or rejected if our cats don’t eat their food eagerly and seek second helpings. And, because their meals are so small, we misunderstand and want them to eat more. We try different diets until we have “evidence “ that they enjoy their food. We train them to ask for food and they train us to respond to their boredom or other unmet needs by feeding them.

Opportunities to express hunting behaviour are a basic need for a cat. If a cat doesn’t have the opportunity to hunt, toys meeting appropriate criteria are small (prey-sized), make high-pitched squeaks or cheeps and move in a rapid, unpredictable fashion. The Indoor Pet Initiative offers an informative piece on choosing the correct toy for an individual cat: http://indoorpet.osu.edu/cats/basicneeds/preypref/index.cfm. Allowing them to hunt for their food (bowl) or using a feeding toy are mentally stimulating activities. Examples of feeding toys include:

- Multivet Slim Cat (http://www.petsafe.net/Products/Feeders/SlimCat.aspx)
- Cat Activity Fun Board (http://www.traininglines.co.uk/cat-activity-fun-board-3397-0.html)
- Go!CatGo! Play-N-Treat balls
- FUNkitty Egg-Cersizer: (http://www.premier.com)
- Aikiou Stimulo (http://aikiou.com/stimulo-cat-bowls-and-feeder/)
Cats are very sensitive to the feel of a food (physical form), its odor and taste. They eat their prey head-first. This is a tactile response to the sensation from the direction of the hair/feathers. Most cats prefer foods that are solid and moist, like flesh, not powdery, sticky or greasy. They prefer their food at fresh-killed body temperature rather than room temperature or out of the refrigerator or hot.

While other species are able to rest their metabolic pathways from the efforts of glucose (energy) synthesis when they have been fed, cats must continue gluconeogenesis in both the fed and fasted states. When anorectic, they catabolize body proteins. Protein supplementation during fasting will slow hepatic lipid accumulation. Urea cycle enzymes in the liver of cats are always "turned on". Adult cats have a much higher requirement for protein than dogs or humans. Expressed as a percentage of diet, adult cats need 29% vs. the adult canine requirement of 12% or the human need for 8%. Over the long-term, cats can adapt to lower protein diets and use carbohydrates as an alternate energy source.

Under stressful situations, cats will refuse a novel food; under other circumstances, the same cat may be very adventurous and chose a new diet over their familiar food. A new diet is more likely to be accepted if it is offered at home rather than in the clinic setting.

Numerous studies have been performed all showing that spaying and neutering/castration decrease energy expenditure by 7-33%. It is, therefore, very important to counsel clients to change from a growth to an adult formulation and to restrict the caloric intake after surgical altering. In general, unaltered cats need 60-80 kcal/kg/day; after altering, they need about 40-50 kcal/kg ideal body weight/day.

**Quantity to feed**

50 kcal/kg/day provides a rough guide and refers to ideal body weight. If a cat is overweight, calculate their caloric requirement for maintenance at their ideal weight. This “rule-of-thumb” is adequate for calculations to determine how much a patient should be getting on a daily basis in clinic and as a starting point for the patient when they are discharged. The client should be advised of the actual amount of food to feed when sent home with canned or dry food. Make sure that you are communicating with common vocabulary as what one person thinks of as a “cup” may not be an 8 oz/250ml measuring cup.

Once feeding any therapeutic diet, it is very important to check and see how the individual patient is responding to the diet by reevaluating them, just as we would recheck a patient on any other medical therapy. Checking body weight and condition cannot be done over the phone. For cats outside of the 4-5kg (8.8-11lb) range in ideal condition, the 50 kcal/kg/day isn’t accurate enough. Caloric requirements for maintenance are closer to: 70 (BW in kg)0.75 (raised to the 0.75 power)

Example: for an 18 lb (8.1 kg) cat:

8.1 X 8.1 X 8.1 = BW cubed = 534.4
Hit square root button twice on calculator => 23 then
=> 4.8 X 70 = 336.
Using 50 kcal X 8.1 kg = 405 kcal

**Feeding for life-stage or using therapeutic diets as part of disease management**

Let’s apply this overview of very basic nutrition and feline feeding facts to a multi-cat home with multiple nutritional needs to a household consisting of the following eleven individuals:

1. An elderly cat with International Renal Interest Society (IRIS) Stage 3 renal insufficiency
2. A thin, arthritic cat
3. A 4 month old healthy kitten
4. A 2 year old healthy cat
5. A 7 year old obese cat (BCS 8/9)
6. An adult cat with “IBD” who vomits and gets diarrhea readily
8. A 7 year old chronically constipated cat
9. A 9 year old cat with CaOx history
10. A 2 year old cat with struvite cystalluria
11. A 14 year old hyperthyroid cat

What dietary strategy can accommodate what appears to be completely disparate nutritional needs?

**FEEDING CATS WITH RENAL DISEASE**

We would like to feed first cat, an elderly individual with Stage 3 renal disease, a protein-restricted diet suitable for renal insufficiency. Do all cats with renal disease have the same etiologic cause for their decline in renal function? Are they all at the same stage? Do they have identical nutritional requirements? Could this cat, perhaps, benefit from being fed a protein enhanced diet, a recuperative diet, a growth diet, a senior diet or a maintenance diet?

**Protein: calorie malnutrition** occurs when a cat is getting enough calories but not enough of them come from protein. As a result, there may or may not be weight loss, but there will be muscle wasting as well as a deterioration in the hair coat quality. Because protein is component in antibodies, immune function may be compromised; anemia may be exacerbated due to the lack of building blocks for hemoglobin; albumin levels may decrease and tissue healing will be affected. Protein is a preferred flavour, so if a cat is already inappetent, restricting protein may result in inadequate intake of all nutrients, and the protein intake may fall below that required for normal function.

As an obligate carnivore, if a cat doesn’t get enough dietary protein to meet metabolic requirements, he must draw on endogenous (stored) protein sources to meet those needs. Over months cats can down regulate their protein needs and switch to other pathways, but in the short and intermediate term, muscle will be catabolized. The resulting muscle wasting and decreased mass reduces the serum level of creatinine (Cr) measured. This makes it difficult to know how much of that decrease in Cr in a cat fed a restricted protein diet is from improvement in renal function and how much is because there is less functional muscle producing Cr.
Managing Differing Nutritional Needs in the Multi-Cat Household

Despite numerous experimental studies and clinical trials having been performed, questions about feeding protein to the cat with renal disease still remain. These include the following five:

1. What is optimal amount of protein for cat with renal disease?
2. When should protein restriction be implemented?
3. Does the type of protein make a difference?
4. How much restriction is necessary?
5. Will a cat in > stage II benefit if phosphorus is restricted by other means?

Protein levels in “restricted” and “high” protein diets fall within the nutritional guidelines, merely at the low or at the high end of the range. Protein-restricted therapeutic diets are not all the same; there are some marked differences in their composition, not just in protein sources and quantities, but also in the calorie source, in their phosphorus, potassium, and sodium content.

Dietary protein is not, in and unto itself, toxic to kidneys. Because of inherent progression of chronic renal insufficiency, IRIS staging focuses on factors which, when managed, are known to slow progression. These are: azotemia, metabolic acidosis, hyperphosphatemia, proteinuria and hypertension.

Azotemia, metabolic acidosis and, to some degree, hyperphosphatemia are affected by hydration, thus optimizing hydration through the use of canned diets, adding water to food, encouraging drinking by use of flavoured liquids or a fountain along with the use of daily subcutaneous fluids are beneficial to the well-being of the patient. The patient should enjoy the diet offered, regardless of what illness he/she has. It is always more important that they eat, rather than what they eat. And the amount consumed must be monitored. This requires calculating the caloric requirements for each individual. 50 kcal/kg/day is a reasonable goal. By being made aware of how much food this is equivalent to, they can notify the veterinarian should the cat be eating less than that amount. This helps prevent confusion regarding weight loss associated with progressing disease vs. that associated with inadequate nutrient intake.

Returning to the cat in question, we do not know from the description (Stage 3 chronic kidney disease) whether the cat is proteinuric or protein replete, nor what the phosphorus or potassium levels are. A protein-restricted diet (which one?) may be appropriate, but one of the other aforementioned diet types (protein enhanced, recuperative, growth, senior or maintenance) might be the correct diet for this individual cat. Just because someone has a specific illness does not automatically mean that the diet designed for that condition is the best diet for that individual.

Every time we send home a therapeutic diet, we are performing a feeding trial with one subject in it (n=1). We have to get the cat back into the clinic and see how he/she is doing on that food. How is his weight? Increased? Decreased? How is his coat? Does he eat with enjoyment or vigor? What are his stools like (moist logs or dry pellets, cow patties or colored water)? How energetic is he since he has been on this diet? Has there been a change in his PCV and proteins? In this case, have the BUN and Cr, the phosphorus and calcium or usg changed? Is he proteinuric and potentially protein deficient? What about his blood pressure? Have these parameters increased or decreased?

Feeding for arthritis

What are the nutritional requirements for cat #2 who is thin and arthritic? Options include a mobility/joint die or, for weight gain, a kitten diet, a recuperative diet, or possibly a senior diet. Assuming that the physical examination and diagnostics do not reveal a cause for her weight loss, it is reasonable to try a variety of diets including all of above in case she has become bored with her food.

Feeding growing cats and elderly cats

Young cats have growth requirements, which include an increased proportion of animal based protein and more calcium and phosphorus. The 4-month old kitten (#3) and the 2-year old healthy adult (#4) would ideally be fed a kitten diet and a maintenance diet respectively. Elderly cats over 12 years of age have been shown to have an increased need for protein, relative to adult cats. They also need more calories from fat than during their adult stage. In part this is because of a decreased ability to digest and absorb fat and protein.

Feeding obese cats

For the fifth cat, #5, the 7-year old obese kitty with body condition score (BCS) 8/9 (or 4.5/5) the therapeutic strategies may include a high fiber diet, a high protein, low carbohydrate balanced diet, or a low fat diet. Exceeding a cat’s protein needs beyond maintenance requirements helps induce satiety. In a study by Laflamme et al, when cats were fed a diet with 45% of calories from protein, cats lost more fat and less lean mass compared with cats fed a diet with 35% of calories from protein, despite similar total weight loss and rate of weight loss.

Ultimately, it is the calories ingested versus expended that is required for loss of weight. In fact, it doesn’t matter which approach we choose, (making this cat very flexible) as long as the caloric intake is reduced, the diet is balanced, the cat isn’t feeling deprived and pestering the client and the diet is balanced. Given the benefits of achieving lean body mass by feeding a high protein diet, a goal of at least 40% protein, dry basis, in a low-fat diet (6% to 10% fat) is a healthy approach to take.

The thermic effect of food (TEF) refers to the energy cost of digesting and absorbing food. TEF is higher when meals are small and frequent, so feeding multiple small meals is preferable to feeding one or two large meals. One way to incorporate this into the diet—and give the cat a little challenge (and exercise)—is to divide the day’s food into six or seven small portions, using feeding balls or placing it on saucers throughout the home as if the cat were on a “treasure hunt”. This feeding strategy makes the cat less likely to gorge and entices him or her to look for more, all of which has a higher TEF cost.
Managing Differing Nutritional Needs in the Multi-Cat Household

Calculating the quantity of dry and/or canned food to prescribe for weight loss:

1. Determine or approximate the cat’s ideal body weight in kg
2. Calculate the number of calories needed to maintain that ideal weight (wt in kg × 50 kcal/kg/day)
3. Multiply this number by 60-70% to get the amount of calories to feed for weight loss.

Include the calories in the treats and supplements, people food and pill pockets that the kitty is being given when you figure out the quantity of food to recommend. As with the protein-restricted diets, the composition of therapeutic diets designed for weight loss are very different from each other.

**Feeding cats with intestinal sensitivity**

Our dietary choices for cat #6, the adult with the sensitive gastrointestinal tract and a diagnosis of “IBD” are either a limited antigen or a “hypoallergenic” diet.

**Feeding cats with diabetes**

Cat #7 is the 10-year old diabetic. Feeding strategies include a high protein, low carbohydrate diet or a high fiber diet. However, a diabetic cat can be controlled with insulin as long as the diet fed is consistent from day to day.

Neither carbohydrates nor dry extruded diets are cause of diabetes or obesity. Exchanging dietary carbohydrate for protein appears to be useful for weight loss treatment and management of non-insulin dependent diabetes in cats.

In a prospective, randomized, double blinded 10-week study (Hall et al), 12 cats (7/12 obese) of whom six were newly diagnosed and six were poorly controlled diabetics evaluated standard maintenance diet vs. lower carbohydrate, higher protein (LCHP) diets. The cats ate dry or canned based on their preference. All were treated with glargine and assessed at weeks 1, 2, 4, 6, and 10 with fructosamine, BG curve and clinical signs. One cat from each diet group achieved remission by week 10. All cats improved clinically, increased weight and achieved good glycemic control. Those fed the LCHP had a significantly greater decrease in fructosamine. The conclusion, based on this small study was that using insulin, “frequent monitoring is key to achieving glycemic control in diabetic cats; potential benefits of dietary modification require further evaluation”. The author summarizes all of the preceding studies and approaches: high fiber & low fat, high insoluble fiber vs. low fiber, LCHP canned, low carbohydrate diet vs. low carbohydrate diet plus acarbose, low carbohydrate & low fiber diet vs. moderate carbohydrate & high fiber diet. None of these approaches appears to make a meaningful difference in the small numbers of cats in each study.

**Feeding the constipated cat**

Constipation is, first and foremost, treated by rehydrating cells. As long as cellular dehydration is present, the need will exist to resorb water from renal and gastrointestinal systems. Addition of fiber to the diet should be avoided until the patient is adequately hydrated. Use of enemas, promotility agents and laxatives prior to addressing this underlying problem is ineffective at best and has the potential for exacerbating the problem. Once that has been accomplished (or simultaneously to rehydration), once can focus on assisting the passage of the feces by mechanical or pharmacologic means.

Soluble fibers are helpful in diarrhea; insoluble fibers are beneficial for constipation. Dietary fiber is a combination of soluble and insoluble fibers. Recently a dry diet enhanced with psyllium has been marketed for the treatment of constipation. Along with rehydration, feeding this diet alleviated obstipation in cats with megacolon allowing them to cease medication, avoid surgery or euthanasia.

Another approach is to reduce fiber feeding a low residue diet.

**Feeding cats with lower urinary tract disease**

Ensuring that urine is in a neutral pH and stays dilute enough so that mineral components don’t come out of solution will help reduce the chance of either CaOx or struvite crystals from forming.

**Feeding for hyperthyroidism**

Use of the extremely low iodine-containing diet in a multi-cat household such as this is inappropriate.

For all cats in the household:

Make sure that water, the most important nutrient, is readily accessible. Have lots of water stations around the home. They should be in places other than the “kitchen” as well, so that cats don’t have to compete and because cats like to eat and drink in different places.

**Baseline diet**

The first of the two goals for feeding a multi-cat household is to achieve a feeding strategy that puts no one at risk nutritionally having the base/common ground diet available to everyone. Of these seven cats, the one at greatest risk if fed the wrong diet is the cat with “IBD”. If the cat with renal disease were in IRIS stage 4, he may well be the most delicate, but just getting adequate calories into a uremic cat becomes the main concern at that point and placing a feeding tube would allow us to deliver an appropriate diet. We would also have to think about a different strategy for restricting access to other diets if he were feeling well enough to be roaming the house. If he is hyperphosphatemic as well as being in stage 3, using intestinal phosphate binders is a viable and necessary alternative to using a restricted protein diet as the baseline, everyone eats, diet. (He can still get the restricted protein diet twice a day.)

**Supplementing requirements**

The second goal is to meet the individual nutritional needs of each member of the household as closely as possible twice a day “behind closed doors”. Certainly the “IBD”-safe diet can be left out during the day for all cats to eat. Twice daily all cats other than the cat with gastrointestinal disease, can be placed in separate rooms to be supplemented with their different or additional needs. This requires analysis of the clowder’s needs as well as their personalities and physical abilities. The elderly cat who is less able to jump can be prevented from eating the food of an agile youngster if the growth diet is placed high
Managing Differing Nutritional Needs in the Multi-Cat Household

up. An overweight cat can be prevented from getting to any food other than that designed for weight loss (the base diet) by putting a latch on a door, building a creep feeder or using a “keyed” cat flap (such as one that responds to the cat’s pre-existing microchip: www.sureflap.ca) so only the thinner cats can get through the narrower space. Treasure hunts using small quantities of food as well as feeding balls (which some cats won’t want to use) will also help. Figuring out creative strategies to use based on the strengths and weaknesses of the individuals is an intriguing challenge and needs to take the cats’ physical, personality and nutritional profiles into consideration.

Reducing stress in the multi-cat household must always be a focus. Cats are social but with strict social rules and restrictions to keep distance in order to avoid confrontation. Environmental enrichment is extremely important. Ellis writes eloquently about this. http://www.sciencedirect.com/science/article/pii/S1098612X09002538

KEY POINTS
1) Don’t assume that a diet designed for a particular clinical condition is necessarily the best diet for every cat with that condition.
2) The quantities to be fed listed in product guidelines are a starting point. Each cat is different.
3) Monitor the clinical response of the individual patient to the dietary prescription.

ADDITIONAL READING


Notes
Cardiopulmonary diseases and respiratory distress are common in feline practice, and identification of the specific underlying cause is important for both diagnostic and treatment success. A specific diagnosis is established based on the results of history, physical examination, thoracic radiographs, ECG, echocardiography, cardiac biomarkers and other ancillary tests.

### HISTORY AND PHYSICAL EXAM

Historical findings with respiratory distress in cats can be variable, but often include cough, labored breathing, lethargy, weakness, reduced interaction with people or housemates, infrequent vomiting and reduced appetite. On exam cats with CHF may have increased respiratory rate and effort, loud pulmonary crackles (pulmonary edema), lung sounds may be muffled ventrally (pleural effusion), and jugular vein distension or hepatomegaly may be noted (esp. in cats with pleural effusion). Hypothermia is common in cats with CHF and low cardiac output, while fever is very uncommon in cats with CHF.

A small volume ascites may be present in cats with biventricular heart failure; however marked cardiogenic ascites is only seen in cats with isolated right-sided CHF (e.g., heartworm disease with CHF). Cats often appear to be dehydrated based on skin turgor despite the fact that they clearly have excessive circulating intravascular volume based on the presence of pulmonary edema and/or pleural effusion. A summary of key historical, physical exam and diagnostic testing results that may help distinguish between CHF and pulmonary disease are listed in Table 1.

### THORACIC RADIOGRAPHS

**Cardiac size and shape** - On a dorsoventral (DV) or ventrodorsal (VD) radiographic view, the heart should occupy no more than 60% of the width of the thorax at its maximal dimension. On the lateral projection, the normal cardiac size is often less than 2.5 rib spaces in the cat. Measurement of the cardiac silhouette using the Vertebral Heart Size (VHS), as described by Buchannan et al, indexes the cardiac size to the vertebral length. Using this

<table>
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<tr>
<th>Clinical Finding</th>
<th>Cat CHF</th>
<th>Cat Lung Disease</th>
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<tr>
<td>Cardiac murmur</td>
<td>Often sternal location</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Cardiac gallop</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>Heart rate</td>
<td>Slower if hypothermic</td>
<td>Variable</td>
</tr>
<tr>
<td>Jugular vein</td>
<td>Distended if pleural effusion</td>
<td>Normal</td>
</tr>
<tr>
<td>Pulmonary crackles</td>
<td>Loud if pulmonary edema</td>
<td>Absent or soft</td>
</tr>
<tr>
<td>ECG findings</td>
<td>Tall p wave</td>
<td>Variable</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Sinus/slow sinus, APC or VPC</td>
<td>Sinus to sinus tachycardia</td>
</tr>
<tr>
<td>Cardiac size CXR</td>
<td>Biatral enlargement</td>
<td>N or RVE</td>
</tr>
<tr>
<td>Pulmonary vessels CXR</td>
<td>Enlarged PA and PV</td>
<td>N or enlarged PA</td>
</tr>
<tr>
<td>NT-proBNP</td>
<td>↑↑↑: &gt; 270 pmol/L</td>
<td>N or ↑: &lt; 270 pmol/L</td>
</tr>
</tbody>
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Modified from many other people, esp. R.L. Hamlin. APC = atrial premature depolarization; VPC = ventricular premature depolarization; CXR = Thoracic radiograph; PA = pulmonary artery; PV = pulmonary vein; N = normal; NT-proBNP = N-terminal B-type natriuretic peptide.
Feline Cardiopulmonary Disease: Who’s To Blame?

The shape of the heart is influenced by the chambers of the heart that are enlarged, although picking specific cardiac chamber enlargement in cats is often more difficult from radiographs compared to dogs. Left atrial enlargement is noted on the DV/VD view as a bulge at the 2:00 location of the clockwise, and the left atrium is best appreciated on the lateral view as enlargement at the caudal dorsal aspect of the heart. Right heart enlargement is noted as a bulge in the 6:00 position to the 11:00 location on the DV/VD view and enlargement of the pulmonary artery is seen at the 2:00 location. On the lateral view, right heart enlargement causes prominence in the craniodorsal and cranioventral quadrants of the cardiac silhouette. In cats with pericardial effusion, the shape of the heart is often very round, especially on the DV or VD view.

PULMONARY RADIOGRAPHIC PATTERNS

Patterns that may be detected on thoracic radiography include bronchial, interstitial, alveolar and vascular. In many diseases there is a combination of these radiographic patterns.

Interstitial patterns - Interstitial diseases primarily affect the connective tissues of the lung, rather than the alveoli or bronchi. Diseases affecting the interstitium include CHF and a variety of primary pulmonary diseases. The lung interstitium may fill with inflammatory products, edema, neoplasia or fibrosis. Interstitial diseases should not mask anatomy so you should be able to see regional vessels and borders of the heart or diaphragm.

Bronchial patterns - Bronchial infiltrates represent disease in the medium to larger airways and are often due to allergic and parasitic diseases. Cats with asthma or chronic bronchitis often have a bronchial pattern. Testing for heartworm disease and respiratory parasites is indicated in animals with bronchial patterns.

Alveolar pattern - Alveolar disease is readily recognized by the appearance of air bronchograms, which develop due to the inherent contrast between aerated bronchi and fluid-filled lung parenchyma. Alveolar disease most commonly represents pneumonia, pulmonary edema, pulmonary hemorrhage or pulmonary contusion. Alveolar pulmonary edema may be cardiogenic or non-cardiogenic, but alveolar edema is a later stage of CHF. With pulmonary edema, the pattern often progresses from an interstitial pattern to an alveolar pattern in later states of CHF. Cardiogenic pulmonary edema should be accompanied by cardiomegaly and left atrial enlargement, pulmonary vessel enlargement, and classically the edema is usually perihilar. In cats, cardiomegaly is not always appreciated and edema distribution may be patchy, and may even be found to have a predominantly ventral distribution.

Vascular pattern - The pulmonary arteries and veins are often abnormal in animals with cardiac disease. When the pulmonary vein is enlarged then left sided CHF is likely present or imminent. Pulmonary vein enlargement and tortuosity is often seen in cats on the lateral thoracic radiograph and this finding is often associated with marked left atrial enlargement. Cats with left sided CHF often have enlargement of both pulmonary arteries and pulmonary veins. When the pulmonary arteries alone are enlarged then pulmonary hypertension or heartworm disease may be present. Enlargement of both the pulmonary artery and vein may indicate fluid overload, or the presence of a left-to-right shunting congenital cardiac defect. The caudal vena cava and often the liver become enlarged in cases with pericardial effusion or right-sided CHF.

CARDIAC BIOMARKERS

Natriuretic Peptides - The natriuretic peptides are a family of structurally similar proteins that regulate salt and water homeostasis. Atrial natriuretic peptide (ANP) and B-type natriuretic peptide (BNP, or brain natriuretic peptide) are manufactured, stored, and released from the myocardium in response to cardiac stretch or hypertrophy. As natriuretic peptides are released they are cleaved into an active molecule (e.g., C-terminal BNP) and an inactive fragment (e.g., NT-proBNP). NT-proBNP is characterized by higher serum concentrations and a longer half-life than C-BNP. These proteins counteract the renin-angiotensin-aldosterone and sympathetic nervous systems and exert actions of natriuresis, diuresis, and balanced vasodilatation. Normal cats have very low levels of circulating ANP and BNP, but chronic pressure or volume overload leads to increased cardiac synthesis and release of BNP. Thus, cardiac disease and CHF result in increased plasma concentrations of natriuretic peptides, thus they can be used for differentiation of cardiac from respiratory disease in the emergency setting. NT-proBNP is greater than 90% correct in differentiating cardiac from non-cardiac causes of dyspnea or other respiratory signs in cats. In addition, veterinarians in practice were noted to have significantly more accurate diagnostic assessments when NT-proBNP was added to history, physical examination, thoracic radiographs and ECG.

Cardiac Troponins - Cardiac troponins, specifically cardiac troponin I (cTnI), may offer some utility in identification of cardiac disease. These troponins are released in response to cardiac injury, and mild increases in cardiac troponins can be seen in cats with CHF. Severe elevations occur in setting of acute or ongoing myocardial injury such as shock, cardiac trauma, myocardial infarction, or cardiac hemangiosarcoma. Cardiac troponin I may have some utility in cats to discriminate between cardiac and respiratory causes of shortness of breath or cough, but is not as accurate as NT-proBNP. The combination use of cardiac troponins and natriuretic peptides might prove to be very helpful in the future.

ECHOCARDIOGRAPHY IN THE EMERGENCY ROOM SETTING

Ultrasoundography is increasing available in the emergency setting, and there is also growing evidence that focused
echocardiography can improve diagnostic accuracy and patient care. A focused echocardiographic exam consists of a short exam, performed in an emergency setting, is most useful if it aims to answer two key questions: 1) Is pleural effusion present, and 2) does the cat have left atrial enlargement or not.

Pleural effusion is found as a relatively echo-free density around the heart, however there is no pericardial sac surrounding the heart. There are often thin, wispy “fibrin tags” floating in pleural effusions, however these wispy floating fibrin tags are rarely found in animals with pericardial effusion. To distinguish between pleural and pericardial effusion, alter the depth such that the heart gets very “small” and search for a pericardial sac. Next, aim the ultrasound probe cranial and caudal to the heart in an attempt to locate and better characterize pleural effusion – the volume of pleural fluid can often be better estimated by imaging in front of or behind the heart.

Left atrial enlargement is almost always present on echocardiography for cats with radiographic pulmonary infiltrates due to L-sided CHF. If the diagnosis of CHF remains in doubt in the emergency setting, a quick echocardiographic exam to assess left atrial size can help guide clinical decisions. In the absence of left atrial enlargement, dyspnea and/or pulmonary infiltrates are much less likely to be due to cardiac disease and other differentials should be pursued.

MANAGEMENT OF CHF IN THE EMERGENCY ROOM SETTING

Keys to success with respiratory distress in the emergency setting include rest, supplemental oxygen, limitation of stress, diuresis if indicated, and thoracocentesis if indicated. A combination of low doses of diazepam or midazolam and narcotic can be used for thoracocentesis if sedation is required (the author recommends against the use of propofol in severe CHF). Vasodilators, positive inotropes, thrombolytics or anticoagulants, bronchodilators, antibiotics or steroids may also be indicated, depending upon the etiology. Fluids rarely have a role in the care of cats in active CHF and in many cases have a limited role in other causes of feline respiratory distress.

Common causes of CHF in cats include congenital heart disease, endocarditis, hyperthyroidism, and various forms of cardiomyopathy (hypertrophic, restrictive, and dilated cardiomyopathy). Excess fluid administration and injectable repositol glucocorticoids can also lead to CHF in cats. Hypertrophic cardiomyopathy, the most common form of feline heart disease, can be associated with left ventricular outflow tract obstruction, and this feature can complicate management of CHF in the emergency setting. Aggressive diuresis can result in dehydration and anorexia; due to the high incidence of ATE in cats with myocardial disease, especially since volume contraction can result in worsening vascular stasis, the author sometimes employs heparin at 250 units/kg subcutaneously 3 times a day, or another antithrombotic, while cats are in the hospital for management of CHF.

Furosemide - Furosemide, likely the most useful drug for the management of acute CHF, is the diuretic of choice for the management of severe pulmonary edema due to CHF. In cats with acute severe pulmonary edema, high doses of furosemide (2-4 mg/kg IV every 1-2 hours for 2-3 doses) may be required to induce an adequate diuresis. The dose must be titrated to the cat’s clinical response, and the dose required to clear significant edema accumulations is often close to a dose that might result in electrolyte disturbance, dehydration, or pre-renal azotemia. Once improvement is noted in breathing rate and effort then the dose of furosemide should be reduced to avoid azotemia, hypotension, electrolyte depletion, and metabolic alkalosis. In cats, we often will give high doses in the first 12-24 hours, and then additional furosemide might not be needed for 2-3 days. In the emergency setting, an alternative to pulsatile furosemide dosing is to administer furosemide using a continuous rate infusion in a small volume of fluid. The dose is usually adjusted to between 0.1 and 1 mg/kg/hour.

Pimobendan - Pimobendan is a calcium sensitizing drug that is useful as a positive inotrope in addition to having properties as a phosphodiesterase inhibitor with vasodilating effects. Pimobendan can result in clinical improvement in cats with CHF, but the optimal dose and ideal situations for use are unclear in cats. While there is growing consensus that the drug could be an adjunct to management of emergent CHF, the use in cats with severe CHF is a bit less clear. There are concerns about the use of pimobendan in cats with serious CHF, especially in the absence of an echocardiographic exam to determine whether left ventricular outflow tract obstruction (LVOTO) exists. Cats with LVOTO are reported to be at risk for adverse effects from worsening obstruction due to either positive inotropic or vasodilating drugs, and pimobendan has both of these actions. Thus, in the absence of an echocardiogram to determine whether LVOTO is present, and until further studies are available, pimobendan should likely not be used a routine drug for life-threatening pulmonary edema in cats.

Sodium Nitroprusside - Nitroprusside can be initiated in cats with life-threatening pulmonary edema and minimal clinical improvement after 1 to 3 hourly doses of furosemide at 4 mg/kg IV. This drug can be very effective in this setting, although blood pressure monitoring is ideal. Close observation of the animal and frequent re-evaluation of the animal’s condition is needed to find an effective dose. Doses ranging between 1 and 5 mcg/kg/min are often successful. The mean arterial pressure should ideally be maintained above 65 mmHg, although if cats with severe pulmonary edema can still stand then the author thinks that blood pressure is probably reasonable for short term (8 to 12 hour) infusions. Sodium nitroprusside is often initiated at 1 mcg/kg/minute in a fluid volume equal to approximately 1/16th to 1/8th of the cat’s calculated maintenance fluid needs.

Dobutamine - Dobutamine can be given as a continuous rate infusion to cats with CHF, hypothermia and azotemia. These cats typically have CHF and concurrent low cardiac output, and this clinical presentation is manifest by cold extremities, hypothermia, pallor and slow capillary refill time, azotemia, weakness and hypotension. Dobutamine has a short half-life and must be given by continuous IV infusion, preferably administered through an infusion pump. Dobutamine in our hospital is often given to cats in low doses (1-3 mcg/kg/min) and the infusion is often
Feline Cardiopulmonary Disease: Who’s To Blame?

kept to less than 24 hours as side effect frequency seems to rise after the first 24 hours. Side effects in cats with CHF can include vomiting, arrhythmias, and possibly seizures. The development of tachycardia above 220 beats/minute or arrhythmia formation dictates dose reduction. Positive inotropes such as dobutamine or dopamine are usually not employed in cats with known hypertrophic cardiomyopathy and left ventricular outflow tract obstruction.

**Angiotensin-Converting Enzyme Inhibitors** - Angiotensin-converting enzyme (ACE) inhibitors are commonly used in the management of chronic CHF and the main benefits of ACE inhibitors are not seen in an emergency setting. However, enalaprilat can be given IV and we have used the injectable formulation of enalaprilat in a number of cats with severe pulmonary edema, typically only in cases where nothing else seems to be working.

**Nitroglycerin** - Nitroglycerin acts as a nitric oxide donor to create vasodilatation. The venous vasodilatation is thought to move blood from the heart and lungs to the capacitance veins, and the drug is most often used in the setting of acute severe pulmonary edema, for 1-3 days, until other medications can be initiated. Typical doses of 1/8” to 2” are applied topically (transdermally), using gloves to avoid absorption to the person administering medication, and the drug is given q 6 to 8 hours. There is controversy as to the effectiveness of this transdermal drug administration in the setting of CHF. The most common side effects are hypotension, weakness, and lethargy. The author suspects that nitroglycerin causes headache in cats, and that its use may be associated with a slightly higher incidence of azotemia.

Most animals with CHF are markedly improved in 24 to 48 hours; if improvement is not detected, both the current therapeutic plan and the diagnosis should be reconsidered.

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**Notes**
Top 10 Treatment Tips for Feline Heart Disease: Feeding and Pharmacology

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INTRODUCTION
Despite the increasingly common diagnosis of cardiac disease in cats, managing cardiac disease in this species can be particularly challenging. Successful management requires careful selection of treatment strategies, regular monitoring, and clear client communication. The authors have identified a number of tips that can be helpful in the care of feline cardiac patients.

TOP TEN TIPS

1. **Body condition AND muscle condition scores should be determined for every patient with cardiac disease at every visit.** Body weight and body condition score, which primarily assesses body fat, are important because they have been associated with overall survival. Cats that are underweight and cats that are significantly overweight have the shortest survival while cats in moderate body condition (4-6/9) had the longest survival (Finn et al, 2010). The muscle condition score is a subjective score which assesses muscle condition in the areas of the epaxial, gluteal, scapula, and temporal areas and graded as normal muscle condition or mild, moderate, or severe muscle loss. Because of increased production of inflammatory cytokines in congestive heart failure (CHF), muscle is lost preferentially to fat. Therefore, even in overweight cats, significant muscle wasting can be present. This muscle loss has important clinical implications as if negatively impacts strength, immune function, wound healing, and survival. By assessing muscle condition at every visit, muscle loss can be identified at its early stages when intervention is more likely to be successful. Optimizing diet (e.g., ensuring nutritionally balanced diet with adequate calorie and protein intake) and omega-3 fatty acid supplementation can be beneficial for cats with CHF by reducing muscle loss.

2. **Don’t make major dietary changes while the cat is hospitalized.** Most cats with acute CHF will not eat but should begin eating when the CHF is better controlled. While hospitalized, avoid diets that are high in sodium but don’t make major dietary changes during hospitalization. Once the cat is home, feeling better, and stabilized on medications, a gradual change to a new and more optimal diet can be made. Forced dietary changes when the cat is sick can induce food aversions. Knowing what the cat eats at home can help to know what food preferences will be (e.g., canned vs dry, flavor, kibble shape) since some cats have very distinct preferences.

3. **Collect complete diet information at every visit to determine whether the diet is optimized.** A complete diet history includes the main cat food being provided, but also treats, table food, dietary supplements, and foods used for...
Top 10 Treatment Tips for Feline Heart Disease: Feeding and Pharmacology

4. Warn owners of cyclical/variable appetite in cats with CHF. Particularly as CHF becomes more severe, appetite often becomes more variable/cyclical (i.e., the cat will eat one diet well for a few days but then stop eating it. At this time, however, they’ll often eat a different food for a few days or more). It is helpful to warn the owner about appetite and to provide them with multiple appropriate diet options for the owner to feed (i.e., they can feed one diet for several days until the cat no longer has interest for that food and then switch to a different food. They’ll often be willing to eat the original food again so owners can use a “rotation” of foods to keep the cat eating). In addition to having different food options, other tips to increase appetite include:
   - Warm the food to body temperature.
   - Feed several small frequent meals during the day, rather than just one or two meals.
   - Feed the cat from a different type of dish (e.g., a new catfood dish or a human dinner plate).
   - Try feeding in a different location in the house.
   - Add homemade chicken, beef, or fish broth to the food (even low sodium store-bought broths are too high in sodium).
   - Add a small amount (1-2 teaspoons) of cooked meat (hamburger, chicken, or fish) to the food. Be sure to instruct the owner not to use any prepared foods, such as rotisserie chicken, deli meats, or canned meats or fish due to their high sodium content.
   - Fish oil, which is high in omega-3 fatty acids, reduces inflammatory cytokines and may have modest benefits for appetite. Fish oil (rather than flax or cod liver oil) should be used and a good quality brand should be used since there is little regulation of dietary supplements. The dose used by the authors is enough fish oil to provide 40 mg/kg eicosapentaenoic acid (EPA) and 25 mg/kg docosahexaenoic acid (DHA). For cats, a one gram capsule containing 180 mg EPA and 120 mg DHA can be easily used to provide this dose when given as one capsule per cat per day. Specific brands can be found on the Tufts HeartSmart website (www.tufts.edu/vet/heartsmart)

5. Be sure to specifically discuss treats with the owner since many owners give treats (either cat treats or “people food”). Most owners are unaware of treats that would be contraindicated (e.g., high sodium). The author typically provides a list of foods that are appropriate and foods to avoid as treats to assist the owner in wise selection. Specifically discuss (and demonstrate, if necessary) appropriate methods for medication administration and if foods are used, be sure to provide appropriate (low sodium) choices (e.g., fish, chicken). A final part of the diet history is dietary supplements. Ask at each visit if the owner is administering dietary supplements. If so, ensure that the supplements are safe, are not interacting with the diet or medications, and are being administered at an appropriate dose. Fewer cats than dogs with cardiac disease receive dietary supplements (13% of cats vs. 31% of dogs; Torin et al, 2007; Freeman et al, 2003) but addressing this issue with the owner is important as “Internet surfing” for alternative treatments of cardiac disease is common. In addition to safety and efficacy issues, there are significant concerns about the quality control of dietary supplements since they do not require proof of safety, efficacy, or quality control to be marketed. Therefore careful selection of type, dose, and brand is important to avoid toxicities or complete lack of efficacy. Much additional information is needed to define the role of dietary supplements in heart disease, when they should be used, when they should not be used, and optimal dosages.

6. The bigger the left atrium, the more an ACE inhibitor is indicated. Angiotensin-converting enzyme (ACE) inhibitors are commonly used in the management of CHF in cats. Blocking the action of the renin angiotensin system leads to diminished plasma levels of angiotensin II and reduced stimulation of aldosterone. As a result, fluid retention and vasoconstriction are blunted. The beneficial effects of ACE inhibition likely result from both the vasodilation and the drug’s effects to reduce cardiac remodeling. In well-designed human and canine heart failure trials, ACE inhibitors resulted in improved clinical signs and a prolonged survival or the time until additional therapy is needed. There are few well conducted clinical trials in cats with which to form solid guidelines regarding clinical use of ACE inhibitors, although one study indicated that in Maine coon cats with asymptomatic HCM, no benefit was seen on cardiac enlargement or remodeling. Most veterinary cardiologists would agree that ACE inhibitors are probably effective in cats with congestive heart failure. There is also general agreement that ACE inhibitors are useful when systemic hypertension complicates feline heart disease, especially when hypertension persists despite amiodipine. ACE inhibitors are also indicated in cats with asymptomatic heart disease and concurrent renal hypertension.

One note of caution: If the cat also is acting lethargic, is tachypneic, won’t eat any foods or if the cat has been eating very well and then suddenly stops eating, this usually indicates the need for reassessment and adjustment of medications.
disease that is associated with even modest proteinuria. Many veterinary cardiologists recommend an ACE inhibitor in cats with cardiomyopathy and moderate to marked left atrial enlargement, even if signs of CHF have not yet developed. Certainly there is no consensus on this topic, but the smaller the left atrium the less likely it is that a cat will benefit from ACE inhibition.

7. Pick the right dose of furosemide – Furosemide is indicated once clear cut congestive sign have developed – pulmonary edema, pleural effusion or ascites that is cardiogenic in origin. It has been documented that furosemide will activate undesirable neuroendocrine responses if administered to animals prior to the onset of CHF, and most cardiologists would avoid use of furosemide as the sole drug for chronic treatment of CHF. After the onset of CHF, almost all cats will need ongoing furosemide to prevent recurrence of signs of CHF. It can be difficult to define the exact dose of diuretic required by any individual cat with CHF, although the desired dose will clear significant edema accumulations and avoid undesirable side effects such as electrolyte disturbance, dehydration and the development of pre-renal azotemia. The combined use of ACE inhibitors and diuretics compromises one of the kidneys’ normal compensatory mechanisms (vasoconstriction of the efferent arteriole) and can lead to elevation of BUN and creatinine when 1) excessive diuretic dose is initiated or 2) significant pre-existing renal disease is present. Still, most cardiologists now concurrently use ACE inhibitors and diuretics for management of CHF; thus caution is advised to avoid excessive doses of furosemide. We usually try to use the lowest possible dose of furosemide which with control signs of CHF, and since this dose will change over time we give the owner upper and lower limits for acceptable furosemide dose, and carefully explaining to them that they should “give more for difficulty breathing or rapid respirations (> 35 breaths per minutes when not purring), and give less if the animal seems weak, lethargic, anorexic, or depressed”. In cats with relatively mild CHF a dose of 6.25 mg/cat q 24 to 48 hrs may be adequate. Cats with ascites or significant volume of pleural effusion often require more furosemide, especially those with enough pleural effusion to require thoracentesis, and a starting dose of at least 6.25 mg/cat q 12 hours is usually needed. When this dose does not control signs of CHF then diuretic resistance has likely developed and drugs such as spironolactone, pimobendan, torsemide, or eplerenone may be added into the furosemide. Alternatively, going to a higher dose of ACE inhibitor, reviewing dietary sodium intake, or taking away the beta-blocker can be alternative to going to higher doses of furosemide.

8. Pimobendan can be used in most cats for management of chronic CHF – Pimobendan, a calcium sensitizing drug that is useful as a positive inotrope, also has vasodilating properties due to concurrent phosphodiesterase III inhibition. The combined positive inotropism and vasodilation has been referred to as inodilation. Pimobendan use in cats with CHF has been reported, but the optimal dose and the optimal clinical situation has not yet been established. Pimobendan also seems to be associated with a low side effect profile in cats. The somewhat empiric dose for pimobendan in cats is 0.25 mg/kg q 12 hours. Several questions remain regarding pimobendan in cats, given the lack of approved drugs for treating heart disease and the retrospective nature of the studies on pimobendan to date. Most cardiologists would give pimobendan in combination with a diuretic and an ACE inhibitor to cats with CHF. Most would not endorse use in cats prior to the onset of CHF, especially in cats with asymptomatic hypertrophic cardiomyopathy. For cats with HCM and concurrent hypertension at the top of the interventricular septum causing obstruction the flow of blood through the left ventricular outflow tract (e.g., systolic anterior motion of the mitral valve, increased aortic outflow tract velocity, and often secondary mitral regurgitation), there are concerns that pimobendan might worsen the outflow tract obstruction. In general, cats with HCM and dynamic left ventricular outflow tract obstruction are thought to be at risk of hypotension or worsening obstruction when given drugs that are positive inotropes or vasodilators, thus drugs like pimobendan might not be advisable. Because the drug has a positive inotropic effect, there has been a tendency for veterinarians to use pimobendan in cats after myocardial failure, evidenced by a reduced fractional shortening, is apparent. Further prospective studies are needed to better outline whether this drug has benefits in cats with CHF and which clinical situations are most appropriate for the use of the drug. The author currently is most likely to use pimobendan in cats under the following circumstances: 1) the cat has advanced heart disease and congestive heart failure 2) there is no left ventricular outflow tract obstruction and 3) the cat has either echocardiographic evidence of myocardial failure or signs of CHF that have not fully responded to twice a day furosemide and a solid dose of an ACE inhibitor or 4) the cat has combined heart and renal failure and there has been a deterioration in renal function (and creatinine > 2.5-3 mg/dl) following initiation of an ACE inhibitor.

9. The bigger the left atrium, the more likely you should think about an antithrombotic - Arterial thromboembolism (ATE) is a common complication of feline heart disease, occurring in 30-40% of cats with cardiomyopathy. The thrombus may develop in either the left ventricle or the left atrium, however, a left atrial origin is most common. Three factors are thought to contribute to thrombus formation: altered coagulability, altered blood flow, and endothelial damage. Cats with cardiomyopathy often have dilatation of the left atrium with blood stasis, they may have endothelial disruption or endothelial dysfunction, and altered coagulation can also been seen in cats with cardiomyopathy. Once ATE develops, treatment is very difficult and often unsuccessful, therefore prevention of this devastating event becomes very important. Unfractionated heparin is a very familiar drug to most veterinarians, and this drug is commonly used to prevent further enlargement of a thrombus in cats with active ATE. While several doses of heparin have been proposed in this setting, and the author’s opinion is that heparin should be used at high doses (275-350 IU /kg q 6-8 hours) for active ATE. However, the
long term use of unfractionated heparin is not usually advisable due to the need for frequent administration and the number of side effects that can develop with chronic use. However, low molecular weight heparins (Dalteparin and Enoxaparin) have been recently used to prevent thrombus formation in cats at risk of ATE. We have used dalteparin (Fragmin; 160 to 175 U/kg) subcutaneously twice a day. Research has suggested that the high doses are needed to inactivate clotting factors for the entire day, and administration of the drug q 8 hrs may be advisable in cats with active ATE. Another low molecular weight heparin option is enoxaparin, which has been studied at 1 mg/kg subcutaneously q 12 hr. Both dalteparin and enoxaparin can be expensive to use on a long-term basis and these drugs must be given by subcutaneous injections. Yet, many owners prefer injections to oral medications in cats, and the drug is currently the author’s first choice for prevention of ATE in cats. Clopidogrel is an antiplatelet drug (Plavix) and is the other drug often used to prevent ATE in cats. This drug reduces platelet activation, reduces platelet degranulation, and inhibits modification of glycoprotein IIb/IIIa receptor which leads to reduced aggregation. Clopidogrel seems to be well tolerated in many cats, although foaming at the mouth or vomiting has been seen in some cats, and increased liver enzymes and bilirubin can also be seen with the drug. The author prefers clopidogrel to aspirin as the latter has been associated with altered renal function, and this may be especially concerning in cats taking furosemide and ACE inhibitors. There is no routine monitoring of clotting times or any other blood tests for these drugs. Clopidogrel is supplied as 75 mg tablets and the proposed dose (pending further clinical experience) is 2-4 mg/kg/day (¼ of a tablet orally once a day), although in cats less than 3-4 kg the drug should likely be compounded to avoid overdose.

10. Maintain quality of life - Cats with heart disease can have many factors that reduce their quality of life, including respiratory distress, weakness after arterial embolism, drug administration issues and drug side effects, trips to the veterinarian, and altered interactions with owners or other animals in the household. Identification of these factors, and determining which are most important for an individual cat and cat owner, can help limit the impact of heart disease on the quality of life. Reducing frequency of medication administration, and consider other alterations in drug administration method, can reduced disruption of the bond between cat and owner.

ADDITIONAL READING


Tufts HeartSmart website [information on heart disease for pet owners, including nutrition (eg, low sodium diets for dogs and cats, treats, omega-3 fatty acid supplements)]. Available at: www.tufts.edu/vet/heartsmart. Accessed July 23, 2012.


Notes
PREVALENCE OF WEIGHT LOSS IN SENIOR CATS

The AAFP/AAHA feline life stage guidelines define a senior cat as 11 to 14 years old and a geriatric cat as 15 years and older. The term ‘senior’ is often used to refer to all cats over about the age of 10 years and will be used as such in these notes. The easiest problems to detect in senior cats are weight loss and decline in body condition, but they are also problems that may challenge the clinician’s diagnostic and therapeutic skills. In addition, not all pet owners appreciate the significance of these signs in older animals. In a recently published study of responses to an internet survey, only 73% of respondents felt that weight loss warranted veterinary attention in a senior animal.

The prevalence of obesity decreases with age in cats; in fact, senior cats have a tendency to be underweight. In a report of 191 cats at the Waltham Centre for Pet Nutrition ranging in age from 1-13 years, the heaviest cats were neutered males aged 5-8 years while cats over 11 years had a tendency to exhibit lower body weights than younger cats. In a survey of over 2,000 cats presented to veterinary hospitals in the Northeastern United States, the proportion of overweight cats increased until 7 years of age, after which it declined, especially in cats over 10 years of age. Similar patterns were found in another study, where the proportion of overweight cats peaked at 7 years and the proportion of underweight cats increased sharply at 11 years. Longitudinal data collected on 53 healthy cats over 11 years of age at the Waltham Centre indicates that for most cats, weight loss or weight maintenance rather than weight gain is a feature of old age. After 8 years of age, 50% of cats in that report maintained weight and 30% lost weight. It appears that a significant proportion of obese middle-aged cats die before reaching old age (e.g., from diseases such as diabetes mellitus or hepatic lipidosis) and a similar proportion lose weight into their senior years. Those senior cats that are obese have probably been obese for most of their lives.

CAUSES OF WEIGHT LOSS IN SENIOR CATS

The reasons for a tendency to weight loss with aging in cats are probably complex and interrelated. Maintenance energy requirements (MERs) in cats decrease by about 3% per year up until about 11 years old. After 11 years of age, MERs actually increase in many cats and may contribute to the tendency of senior cats to be underweight if their energy needs are not met. An investigation of changes in body composition with aging found that lean body mass drops dramatically after 12 years of age, and that by age 15, cats may have a mean lean tissue mass under 2 kg (4.4 lb), one-third less than cats aged 1-7 years (mean 3 kg [6.6 lb]). This loss of lean body mass in aging cats in the absence of disease is termed sarcopenia. Mean percentage body fat also decreases progressively after 12 years of age so that the lean body mass to fat ratio does not show significant changes with aging. The combination of reduced lean mass and body fat contributes to the frail look of many elderly cats.

A healthy young to middle-aged adult cat requires at least 5 grams of protein/kg body weight/day whereas older cats probably require more. Cats can certainly utilize carbohydrates as an energy source, but they have a limited ability to spare protein utilization by replacing it with carbohydrate. Changes in digestive efficiency occur with age and may contribute to weight loss and increased protein requirements. Older cats are less efficient at digesting fats and proteins. In one study, 22% of cats over 14 years old had protein digestibility of less than 77% and 33% of cats over 12 years old had fat digestibility of less than 80%. To compensate, senior cats may need to increase their daily food intake by as much as 25%. In a study of 85 senior cats on a long-term feeding study (over 7 years), there was a significant increase in total kcal/kg body weight ingested and total daily food consumption in cats from 10 to 15 years of age.

Despite the increase in caloric intake, body weight decreased with age, particularly after age 13. The nutritional profile of commercially available senior diets varies widely, so not all senior diets are equal. A feeding plan must be tailored to body condition and the presence of diseases as well as life stage. Considering the available data on metabolism and body weight in aging cats, it seems likely that many elderly cats, particularly those over 12 years of age that are not overweight, would benefit from frequent small meals of energy-dense, highly digestible diets with protein of high biological value to maintain body weight and lean tissue mass and avoid protein/calorie malnutrition. The AAFCO minimum for cats is 6.5 g/100 kcal of diet, but higher levels are likely more optimal. Despite the numerous advantages of feeding canned diets to
Evaluating Weight Loss in Senior Cats

senior cats (e.g., increased water content, higher proportion of animal source protein), most canned diets have a lower caloric density based on volume fed than dry diets. Therefore, attention must be paid to ensure the caloric intake of cats on canned diets is appropriate.

Other reasons for the susceptibility of senior cats to lose weight may include the presence of diseases (including those causing pain) and decreased appetite due to dulling senses of taste and smell. Cachexia is the loss of lean body mass associated with chronic disease. In senior cats, cachexia and sarcopenia may occur together. Early detection of weight loss and loss of lean body mass is important as it may lead to early detection of disease. A study of 258 cats in a Nestle Purina colony that died of cancer, renal failure and hyperthyroidism determined weight loss started about 2.5 years before death.a Cats dying from other causes started losing body weight even earlier, about 3.75 years before death. Body weight loss two years prior to death was over 6% in cats with cancer, renal failure and hyperthyroidism. During the last year of life, the average weight loss was over 10% for cats dying of all causes. Gradual weight loss is often overlooked by owners. Therefore, the body weight and body condition score should be determined and recorded at every opportunity, since weight loss may be the earliest sign of disease. Percentage weight change is an easily performed calculation ([previous weight – current weight]/previous weight) that detects subtle trends. Muscle condition scoring is also useful in senior cats for early detection of loss of body condition. It is important to note that diseases causing weight loss in senior cats are not always associated with inappetence. Since weight loss can occur with either an increased or decreased appetite, it is important to encourage owners to report any change in appetite.

Finally, cognitive dysfunction syndrome (CDS) is suspected to occur in cats as it does in aging dogs, although formal diagnostic criteria for cats are lacking.15, 16 The signs of CDS are mainly behavioral and include disorientation, altered interactions and sociability, disrupted sleep-wake cycles, altered activity levels and patterns, excessive vocalization, anxiety and irritability, and decreased grooming. In addition, alternations in appetite could lead to weight loss.

DIAGNOSIS OF WEIGHT LOSS IN SENIOR CATS

Diagnosis of weight loss in senior cats is dependent on thorough data gathering since there are many potential causes. A complete history, including a nutritional history, is the first step in diagnosis. The diet history should include brands, flavors, format (dry, canned, semi-moist), as well as quantity and frequency of meals, treats and table food, nutritional supplements, and whether food is used to administer medication. Attention should be paid to trends in food and water consumption and questions should be asked about signs of pain, behavior changes, changes in elimination patterns, and changes in mobility as well as presence of vomiting or diarrhea. Senior cats often have more than one health problem and may be receiving multiple medications, many of which cause gastrointestinal distress including anorexia, such as NSAIDs, antibiotics, and cardiac medications. A detailed behavioral history should be obtained for clues to medical problems including CDS. A thorough physical examination should include assessment of weight and body condition, an orthopedic examination, and blood pressure measurement.

Causes of weight loss in senior cats may be categorized by quality of appetite. With a normal or increased appetite, diseases causing malabsorption or maldigestion (e.g., inflammatory bowel disease, gastrointestinal lymphoma) or excessive protein loss (e.g., protein-losing nephropathy or enteropathy, diabetes mellitus, hyperthyroidism) must be considered. With a diminished appetite, investigations must focus on oral cavity disease, systemic diseases (e.g., neoplasia, chronic kidney disease, liver disease, gastrointestinal disease, retroviral infection) and diseases causing pain (e.g., degenerative joint disease).

The minimum laboratory database for investigation of weight loss in senior cats includes a complete blood cell count, serum biochemistries and electrolytes, total T4, complete urinalysis, and retroviral testing. Most common causes of weight loss in this age group will be quickly diagnosed or eliminated with this minimum database. Cats with protein/calorie malnutrition may have lymphopenia, anemia, lower than expected BUN and creatinine, and increased liver enzymes and bilirubin. In severely protein deficient animals, serum total protein and albumin may be reduced. Serum creatinine kinase activity may be a useful marker for assessment and monitoring of nutritional status in cats. In one study, serum CK was significantly increased in hospitalized anorectic cats compared to non-anorectic cats, and was significantly lower after 48 hours of nutritional support via nasoesophageal tube.17

Depending on the physical examination findings and results of initial testing, further laboratory investigations may include bile acid testing and extended thyroid hormone testing. Pancreatic (e.g., chronic pancreatitis) and gastrointestinal disease (e.g., inflammatory bowel disease, lymphoma) are common causes for weight loss that may not be readily apparent from the history, physical examination and initial laboratory testing. Blood tests for pancreatic and gastrointestinal disease include cobalamin, folate, and feline pancreatic lipase immunoreactivity. Finally, more advanced diagnostics will be indicated for a subset of patients, such as abdominal imaging or endoscopy. Exploratory laparotomy or laparoscopy should be considered when the cause of weight loss remains undiagnosed. During surgery, samples for histopathology should be collected even if the tissue appears grossly normal. Sites to sample include liver, pancreas, lymph nodes, stomach and multiple areas of the small intestine.

NON-SPECIFIC MANAGEMENT OF WEIGHT LOSS

The best chance to reverse weight loss is to diagnose and treat underlying diseases. However, non-specific measures for nutritional support are often part of the treatment plan. Encouraging increased food intake can be accomplished by feeding an energy-dense, nutrient-dense diet that is palatable to the cat – some cats prefer diets with high moisture content while others prefer dry diets. Examples of appropriate diets include diets designed for growth and recovery or critical care. Even if a therapeutic diet is recommended, initially it is best to feed familiar foods as learned aversions may be induced by feeding
novel foods to sick or hospitalized cats. It is better for a sick cat to eat any food rather than no food at all. Slow introduction of the recommended diet can be made once the cat’s condition and appetite have improved and it has been discharged from hospital. Short-term use of appetite stimulants may be helpful in some hyporexic cats. If adequate food intake cannot be achieved, nutritional support via tube feeding should be considered. Nutritional support should be considered earlier rather than later for moderately to severely malnourished cats. Some anorectic patients with diseases such as neoplasia, advanced renal disease, hepatopathy, protein-losing gastrointestinal disease or protein-losing glomerular disease will benefit from early nutritional support before significant weight loss occurs.

Cats are solitary feeders by nature and elderly cats often do not cope well with competition and stressors. Therefore, many older cats in multi-cat homes would benefit from being fed separately or being offered supplemental meals. Owners should be educated to monitor the daily food intake of senior cats carefully. One way to do this when cats are fed individually is to weigh food bowls before and after feeding. Other ways to encourage senior cats to eat include:

1. Offer fresh favorite and familiar foods to avoid learned aversions
2. Use wide, shallow food and water bowls
3. Warm the food to body temperature
4. Moist the food
5. Feed in a quiet, stress-free environment
6. Use encouragement (e.g., petting and praise) during feeding

REFERENCES
Nutritional Management of Endocrine Disease in Cats

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Dr. Mark Peterson graduated from the University of Minnesota in 1976, moved to NYC to do an internship and medical residency at The Animal Medical Center (AMC), and then completed a post-doctoral fellowship in endocrinology and nuclear medicine at The New York Hospital-Cornell Medical Center and the AMC. Mark obtained board certification from the American College of Veterinary Internal Medicine in 1981.

Following his training, Mark stayed on as head endocrinologist at the AMC for over 30 years. In 2009, Mark opened the Animal Endocrine Clinic in New York City, a specialty referral hospital devoted exclusively to the diagnosis and treatment of cats and dogs with endocrine disease, where he now cares for his endocrine patients.

Over the last 35 years, most of Dr. Peterson’s clinical and research efforts has been directed toward advancing our understanding of endocrine disorders of the cat and dog, including hyperthyroidism, diabetes mellitus, and adrenal disease. He has published more than 475 journal articles, book chapters, and research abstracts. With more than 300 lecture presentations to his credit, Dr. Peterson is a frequent speaker at veterinary colleges and scientific seminars both in both the United States and abroad.

When treating cats with endocrine disease, most of us concentrate on the latest or best drug that can be used to cure or manage the disease. However, it is also important to realize that proper nutritional support also plays an important and integral role in management of these diseases.

Whenever considering feline nutrition, it is paramount to remember that cats are strict carnivores that rely on nutrients in animal tissues to meet their specific and unique nutritional requirements. In their natural habitat, cats consume small prey high in protein (50-60%) with moderate amounts of fat (30-50%) but very minimal amounts of carbohydrates (1-2%). Evolution has shaped the cat’s core metabolism to adapt to this carnivorous diet of animal tissues over a period of millions of years, so it is not very “natural” for cats to eat many of the commercial diets that contain moderate to high amounts of carbohydrates (35-50%) and much lower amount of protein, especially animal protein.

In this review, I will cover the 3 most common endocrine problems of cats seen in clinical practice (i.e., hyperthyroidism, diabetes, and idiopathic hypercalcemia) and discuss the way I integrate nutrition into the management of these common feline diseases. My goal is to use the cat’s diet as a mean to support their overall body and metabolism and, therefore, best manage their underlying endocrine disease.

HYPERTHYROIDISM IN CATS

Hyperthyroidism is the most common endocrine disorder of cats, and is one of the most common medical problems seen in small animal practice. Surprisingly, despite the fact that nutritional factors and cat food has been proposed to have a role in the etiopathogenesis of this disease, there are only limited published recommendations about what to feed these cats. The question, “What’s the best diet to feed my hyperthyroid cat?” is an extremely common one that we all get from concerned cat owners.

The Many Metabolic Problems Facing the Hyperthyroid Cat

When secreted in excess, thyroid hormones have profound metabolic effects on the whole body, and dysfunction of multiple organ systems (CNS, cardiac, gastrointestinal, hepatic, and renal) is common in hyperthyroid cats.

Weight loss and muscle wasting: Weight loss, despite a normal to increased appetite, is the classic and most common sign seen in cats with hyperthyroidism. These cats lose weight because their hyperthyroidism accelerates their metabolic rate and body’s energy expenditure; they are burning up their food calories faster than they can consume their daily meals. It’s important to realize that hyperthyroidism is a catabolic state. The progressive weight loss and muscle wasting that is so characteristic of feline disease is caused by increased protein catabolism leading to a negative nitrogen balance.

When hyperthyroid cats first lose weight, the case can usually be first noticed as a loss of muscle mass in the cat’s lumbar paravertebral area. Despite this loss of muscle mass, most mildly hyperthyroid cats retain their “belly” during the initial stages of their thyroid disease and may even have a higher than ideal body condition score. With time, severe muscle wasting, emaciation, cachexia, and death from starvation can occur if the cat’s hyperthyroidism is left untreated.

In hyperthyroidism, the cat’s body consumes its own muscle tissue to get the protein it needs to sustain its carnivorous life. Even with treatment of hyperthyroidism, recovery of muscle mass and function may be prolonged, lasting several weeks to months. This is especially true if these cats are not provided with enough dietary protein to rebuild and maintain their lost muscle mass.

Hyperglycemia, glucose intolerance, insulin resistance, and overt diabetes: Hyperthyroid cats commonly develop profound changes in glucose and insulin metabolism. Mild to moderate hyperglycemia is common in hyperthyroid cats, which is generally attributed to a “stress” reaction. However, the underlying metabolic changes are actually much more complicated: hyperthyroidism frequently causes moderate to severe “endogenous” insulin resistance, as demonstrated by high resting serum insulin concentrations and an exaggerated insulin response during an IV glucose tolerance test. This insulin resistance is associated with a decreased glucose clearance (impaired glucose tolerance), which is indicative of a prediabetic state.

Occasionally, an untreated hyperthyroid cat will develop overt diabetes mellitus. Many of these diabetic cats will develop moderate resistance to the injected insulin, with poor diabetic...
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control.10,11 Surprisingly, the insulin resistance and prediabetic state so common in hyperthyroid cats does not always improve and may even worsen despite successful treatment of hyperthyroidism.8,9 This indicates that hyperthyroid cats may have long-lasting alterations of glucose tolerance and insulin secretion that cannot always be reversed by treatment. In accord with that, some of these hyperthyroid cats (not diabetic at time of diagnosis) will go on to develop overt diabetes mellitus in the months to years after treatment of hyperthyroidism.

Sarcopenia of aging: In addition to loss of muscle mass from the catabolic effects of thyroid hormone excess, cats also tend to lose muscle mass as they age, independent of their thyroid status. This phenomenon, referred to as sarcopenia of aging, is also common in elderly human beings.12,13 The term age-related sarcopenia is derived from Greek (meaning “poverty of flesh”) and is characterized by a degenerative loss of skeletal muscle mass and strength, as well as increased muscle fatigability.

In adult cats, maintenance energy requirements decrease by about 3% per year up until the age of 11 years, and then actually start to increase again.14 This contributes to a tendency of senior cats to lose muscle mass if their energy needs are not met. Lean body mass of aging cats drops dramatically after 12 years of age, and by age 15, cats may have a mean lean tissue mass that is a third less than cats aged 7 years or less.14,15 Body fat also tends to progressively decrease in cats after the age of 12 years; this combination of reduced lean mass and body fat contributes to weight loss experienced by many elderly cats.

The ability to digest protein is also compromised in many geriatric cats. After the age of 14 years, one-fifth of geriatric cats have reduced ability to digest protein.14-16 Reduced protein digestibility in geriatric cats seems to occur in parallel with reduction of lean tissue and it might predispose them to negative nitrogen balance.17

Although moderation of calorie intake might be suitable for some mature cats, it does not appear to match the needs of most geriatric cats. In contrast, it seems more logical to use highly digestible, energy-dense food for geriatric cats in order to prevent or slow their decline in body weight and lean body tissue.14,17,18 Reducing protein intake in geriatric cats, at a time when lean tissue has been lost, is contraindicated. Geriatric cats seem to have nutritional requirements closer to kittens than to mature adult cats.

Diet Recommendations for Hyperthyroid Cats

High dietary protein: Obligate carnivores, such as the cat, are unique in their need for large amounts of dietary protein (specifically, dispensable nitrogen) that separates them from omnivores and herbivore species.1,2,19,20 This absolute requirement for dietary protein intake in cats is critically important when formulating a diet for hyperthyroid cats, in which protein catabolism and muscle wasting is universally present.

Protein is the primary macronutrient responsible for maintenance of muscle mass. Restoring and preserving any remaining muscle tissue in cats treated for hyperthyroidism depends upon the cat consuming a diet with sufficient amounts of high-quality protein (40-60% calories as protein).

This recommendation for higher amounts of dietary protein does not change once euthyroidism has been restored. The dogma that all older cats should be fed reduced energy “senior” diets must be questioned based on what is now known about the increasing energy requirements and nutritional needs of older cats.15,18 In most geriatric cats, logic dictates the use of highly digestible, energy-dense food to mitigate the decline in body weight and lean body tissue and to avoid protein:calorie malnutrition.13,17,18 Reducing protein intake in geriatric cats, at a time when lean tissue has been lost, is contraindicated. Geriatric cats seem to have nutritional requirements closer to kittens than to mature adult cats. Remember that when deprived of protein, carnivores will continue to break down muscle tissue to create the energy they need.1,19,20 By feeding only high-quality protein diets, we will help restore the cat’s muscle mass and improve strength and agility.

Low Dietary Carbohydrates: Since most of these cats also have subclinical diabetes —as evidenced by their mild hyperglycemia, glucose intolerance, and insulin resistance— feeding a low carbohydrate diet (<10% of total calories) also is strongly recommended.21 Feeding a low carbohydrate diet will lessen postprandial hyperglycemia, improve insulin sensitivity, reduce the need for exogenous insulin, and help stabilize glucose metabolism in these cats.21-27 This may prevent the development of overt diabetes and control long-term obesity in these cats after successful control of the hyperthyroidism.

This website (www.catinfo.org) provides a breakdown of the nutritional composition of the various prescription and over-the-counter diets (Click on link entitled “Protein/Fat/Carbohydrate Chart” on right panel of home page). It turns out that many commercial canned diets have a composition of protein and carbohydrates that is more than adequate for the hyperthyroid cat. However, the composition of almost all dry food cat diets is much too high in carbohydrates, and most are too low in protein content. That is why I believe it’s best to limit the amount of dry food that is fed to hyperthyroid cats, or even better, not feed dry food at all.

DIABETES MELLITUS

Evolutionary events shaped the cat’s core metabolism such that their systems are uniquely set up to metabolize a diet which is high in moisture, high in protein, and very low in carbohydrates. Because this is the diet they have relied upon for tens of thousands of years, they do not have the ability to process carbohydrates very efficiently.1,2,19,20 This becomes extremely important when selecting a diet for cats with diabetes (see below).

Normal Glucose Metabolism and Postprandial Glycemia in Cats

So what are these specific feline adaptive mechanisms that have developed to meet the requirements of a carnivorous diet? First, cats’ gluconeogenic pathway provides an almost continuous source of carbon skeletons for glucose or energy production.1,2,19,20 Secondly, glucokinase concentrations are markedly reduced or absent, whereas hexokinase activity is increased. This is in marked contrast to the liver of omnivores
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(dogs, man), which contains both glycolytic enzymes that act to catalyze the phosphorylation of glucose during glycolysis. In addition, cats have reduced amylase and disaccharidase activity in the small intestine, reduced and delayed insulin secretion, and delayed gastric emptying.1,19,20

As a result of these differences, plasma glucose clearance rates are longer in cats compared to dogs or humans after feeding a moderate to high carbohydrate meal — in other words, even normal cats have much more prolonged postprandial period of hyperglycemia than might be expected. In healthy humans and dogs, postprandial hyperglycemia normally persists for 2 to 6 hours.26,27 In contrast, a recent study of healthy cats found that both serum glucose and insulin concentrations remained significantly increased for a median time of 12 hours following ingestion of a moderate carbohydrate meal (25% ME), and that both glucose and insulin concentrations remained above baseline values for 24 hours in approximately 20% of the cats.30 Most feline diets contain even higher amounts of carbohydrate and, therefore, would be expected to result in more severe postprandial hyperglycemia and a longer time to return to baseline.

Dietary Management of Cats with Diabetes Mellitus: Key Part of Treatment!

In cats with diabetes, a primary goal of therapy is to minimize the degree of hyperglycemia that develops after feeding in order to lessen the subsequent demand on beta-cells to secrete insulin.21,31 By doing this, we decrease the effect of “glucose toxicity” and allow the pancreatic islet cell to hopefully recover.

But how do we do this? It has long been known and is a well accepted “dogma” by most practicing veterinarians who specialize in feline medicine that feeding a low-carbohydrate diet is the mainstay in the treatment of diabetes mellitus, especially if remission of the diabetic state is the goal. Feeding a low carbohydrate diet will improve insulin sensitivity, reduce or eliminate the need for exogenous insulin, and help stabilize glucose metabolism in these cats.21-27,30 Again, one major way a low-carbohydrate diet improves the diabetic state is by helping to prevent severe and prolonged postprandial hyperglycemia.

The sooner one starts the diabetic cat on a low-carbohydrate diet, the better. By “low-carbohydrate,” I mean a diet that provides <10% of the calories as carbohydrate. Some cats will do fine on a slightly higher-carbohydrate diet (10-15%), whereas others do best on a diet containing <5% carbohydrate.21 To achieve these low carbohydrate levels, we must formulate a homemade diet or feed a canned food (go to www.catinfo.org and review the “Protein/Fat/Carbohydrate Chart” to select an appropriate low-carbohydrate diet). None of the available dry cat foods are very low in carbohydrates, and most are too low in protein. It’s best to limit the amount of dry food that is fed to diabetic cats, or even better, not feed dry food at all.

When we reduce the content of carbohydrates in a cat food, we must raise the content of either protein or fat, or both. I recommend feeding diabetic cats a diet that mimics the composition of their prey in the wild: about 40-60% of protein with the remaining amount in fat and almost no carbohydrate. This higher than average protein level also helps restore and maintain lost muscle mass, since many diabetic cats will develop “sarcopenia” as they age.33,34,35

Another plus for feeding low-carbohydrate, high-protein diets in cats with relatively early diabetes is that this diet composition (together with insulin treatment) greatly improves the diabetic remission rate.23-27 If the diabetic cat goes into remission (no more insulin needed to maintain euglycemia), we recommend maintaining a restricted carbohydrate diet for life to help prevent relapse of the diabetic state.

If a change in feeding to a low-carbohydrate diet is made in a diabetic cat already stabilized on insulin, it is extremely important to realize that this will result in a lowered daily insulin dosage — often significantly.21 If not closely monitored — ideally with home glucose monitoring — severe hypoglycemia can develop in these cats because they become more sensitivity to insulin after the diet composition is changed.

IDIOPATHIC HYPERCALCEMIA IN CATS

Over the last two decades, a syndrome of idiopathic hypercalcemia in cats has emerged and appears to be increasing in frequency.32-37 The term “idiopathic hypercalcemia” refers to a high serum ionized calcium concentration of unknown cause, even after extensive medical evaluation has been undertaken to rule out other known causes of hypercalcemia, such as primary hyperparathyroidism and neoplasia.38,39

Multiple factors have been considered in relation to the underlying cause of idiopathic hypercalcemia. It is still unclear if increased intestinal calcium absorption, increased bone resorption, or decreased renal calcium excretion (or some combination thereof) is the key factor leading to the development of the ionized hypercalcemia in this syndrome.

Despite the fact that the underlying cause of idiopathic hypercalcemia remains elusive, this has clearly become the most common type of hypercalcemia in cats.

Postulated Causes for Idiopathic Hypercalcemia

It has been suggested that the diet fed may predispose cats to development of idiopathic hypercalcemia, as well as formation of calcium oxalate calculi found in 10-15% of cats with this syndrome.38,39

Many believe that feeding of acidifying, magnesium-restricted diets predisposes cats to idiopathic hypercalcemia.35,39 In support of this hypothesis is the fact that both calcium oxalate stones and hypercalcemia first became prevalent in the 1990’s, shortly after the introduction of feline acidifying diets designed for prevention of struvite crystals.32-34 In addition, 3 of 5 cats in one series32 and all 14 cats for which diet history was available in another report33 had been fed acidifying diets designed to minimize struvite crystalluria and urolithiasis.

When fed to normal cats, such acidifying diets may result in a state of systemic acidosis,30,34 which, in turn, promotes increased calcium resorption from bone, leading to reduced bone mineral density and a state of negative calcium balance. The calcium salts present in bone represent the largest store of alkaline base
in the body and, therefore, act as a buffer in states of metabolic acidosis.\textsuperscript{42} Therefore, when human subjects are fed a diet that produces a net acid load, excessive calcium salt may be released from bone, resulting in increased urinary calcium excretion.\textsuperscript{43-44} In agreement with these human studies, the induction of metabolic acidosis in cats fed an acidifying diet may result in both mild ionized hypercalcemia and hypercalciuria.\textsuperscript{45}

Another plausible hypothesis is that excessive dietary vitamin D content in some cat foods may contribute to this syndrome.\textsuperscript{36} Cats have a low requirement for vitamin D (1.4 \textmu g [56 IU] cholecalciferol per 100 kcal diet),\textsuperscript{45} at least when fed a diet with adequate concentrations (and a correct ratio) of calcium and phosphorus.

The vitamin D levels in commercial cat diets are not listed on the label and are not always included on product guides or company websites. However, if the label states that a cat diet is “complete and balanced,” the vitamin D levels must be between 3.1-62.5 \textmu g [125-2,500 IU] per 1000 kcal to comply with AAFCO guidelines.\textsuperscript{46} Therefore, the amounts of vitamin D added to commercial cat foods could range from 2- to 50-fold higher than the minimal requirement recommended by the NRC.\textsuperscript{47} Most commercial cat foods likely contain relatively high amounts of vitamin D, which could result in hypervitaminosis D in some cats and contribute to ionized hypercalcemia in at least some of them.\textsuperscript{45,47}

The finding of “normal” serum concentrations of 25-hydroxyvitamin D and calcitriol in most cats with idiopathic hypercalcemia goes against this hypothesis that excessive dietary vitamin D levels in the diet contributes to this syndrome.\textsuperscript{33,48} However, it is important to realize that reference range limits for 25-hydroxyvitamin D have all been established in clinically normal cats fed commercial diets which again may be heavily supplemented with vitamin D.

**Treatment of Idiopathic Hypercalcemia**

In almost all cats with idiopathic hypercalcemia, clinical signs are usually relatively mild, at least at diagnosis. In general, the severity of hypercalcemia in these cats tends to be slowly progressive. Therefore, most cats can be treated as outpatients with either dietary therapy, alone or in combination with drug therapy—i.e., glucocorticoids or bisphosphonates.\textsuperscript{35-39,49}

I generally start with diet modification as a first-line treatment. If an acidifying diet is being fed, it should be discontinued. However, it may not always be clear that the cat food being fed is an acidifying diet—one should always closely examine the ingredient list to look for the presence of an added urinary acidifier, such as dl-methionine, phosphoric acid, and ammonium chloride. Although feline urine is normally mildly acidic, feeding cats commercial diets containing high amounts of carbohydrate (e.g., starch and fiber) will result in an alkaline urine pH.\textsuperscript{30,33} Therefore, many commercial cat food diets contain added acidifiers in order to “counteract” the alkalizing effects of the high carbohydrate diet, even when it is not promoted as a urinary tract diet.

No matter what type of diet is chosen, it is best to feed a wet-only diet to promote urinary dilution and lessen the chance for calcium oxalate stones.\textsuperscript{52} To this end, we have 5 different types of cat food diets that have been proposed to help lower calcium in cats with idiopathic hypercalcemia.\textsuperscript{26-28}

High fiber diets (e.g., Purina OM Overweight Management, Iams Intestinal Plus Low-Residue, Hill’s w/d) will restore normocalcemia in some cats with idiopathic hypercalcemia and calcium oxalate urolithiasis.\textsuperscript{32,36} The effects of fiber on intestinal absorption are complex and depend on the type and amount of fiber, as well as the interactions with other nutrients in the diet. However, these “high fiber diets” are usually supplemented with extra calcium; therefore, calcium content does not explain why these diets are occasionally helpful in treating idiopathic hypercalcemia. Again, changing from an acidifying diet to any diet that is less acidifying (such as high fiber) would be expected to be beneficial.

Another option, of course, would be to feed a lower calcium diet and add fiber to the diet (e.g., psyllium for a mixed-fiber source or guar gum for an all-soluble source).\textsuperscript{30} However, because high-fiber diets tend to be lower in protein, cats with idiopathic hypercalcemia chronically fed these diets can lose lean muscle mass to become muscle wasted.\textsuperscript{3,14} This is especially true if the cat’s appetite is poor, a sign present in some cats with this syndrome.\textsuperscript{33-39} Overall, I do not find high-fiber diets to be helpful in the vast majority of cats with idiopathic hypercalcemia and no longer recommend these diets.

Prescription kidney diets (e.g., Purina NF Kidney Function, Royal Canin Renal LP Modified, Iams Renal Plus, Hill’s k/d) also may result in normocalcemia in some cats with idiopathic hypercalcemia.\textsuperscript{36} Although these renal diets appear less acidifying than most maintenance or high-fiber diets, many renal diets still contain added dl-methionine. Most renal diets are low in calcium, so its decreased consumption should lead to a decrease in the amount of calcium absorbed.\textsuperscript{36}

Remember, however, that renal diets are also restricted in phosphorus, which may lead to increased calcitriol (active vitamin D) synthesis by the kidney; the action of this increased serum calcitriol could offset the advantage of the decreased calcium absorption in cats with idiopathic hypercalcemia.\textsuperscript{35,53} Overall, because these diets are lower in protein, renal diets are not my first choice—with time, cats with idiopathic hypercalcemia can become muscle wasted on these diets.

Canned diets developed to prevent calcium oxalate urolithiasis (e.g., Royal Canin Urinary SO, Purina UR Urinary St/Ox, Iams Urinary-O Plus Moderate pH/O, Hill’s c/d) may be beneficial in the treatment of cats with idiopathic hypercalcemia.\textsuperscript{36,37} These diets are restricted in calcium and tend to be less acidifying, resulting in a neutral urine pH in most cats. However, some still contain dl-methionine, which should definitely be avoided. Some of these diets are also restricted in oxalic acid, which may help prevent the calcium oxalate stones that develop in 10-15% of cats with idiopathic hypercalcemia. However, I do not find any of these “calcium oxalate” diets to be very helpful in normalizing the high ionized calcium concentrations found in cats with idiopathic hypercalcemia. Therefore, I cannot strongly recommend these diets, especially if no calcium oxalate stones are present.
Commercial canned diets with a composition similar to what cats would eat in the wild (i.e., 40-60% protein, 30-50% fat, and <15% carbohydrates) may also be beneficial in lowering serum calcium concentrations in some cats, particularly those with mild forms of idiopathic hypercalcemia. Again, one can use the online “Protein/Fat/Carbohydrate Chart” found at www.catinfo.org to select a canned cat food that provide a nutritional composition similar to what cats would ingest in small prey (e.g., small rodents, birds, and insects). Although this diet composition will result in an acidic urine pH (normal for cats), a high-protein diet is preferable to added acidifiers for prevention of struvite crystal formation in cats and is not associated with the same degree of metabolic acidosis.

In addition to the macronutrient composition, one should ensure that the canned food selected does not have any added acidifiers (e.g., dl-methionine, phosphoric acid, or ammonium chloride) and is not a magnesium-restricted diet. A diet with a relatively low vitamin D content (<5 μg [<200 IU]/1000 kcal) is recommended. Although feeding a low-calcium diet is ideal, none of the available commercial cat foods are calcium-restricted.

For more control over the exact macronutrient, mineral, and vitamin D content of the cats’ diet, feeding a specially formulated, home-prepared diet is recommended. Some cats, especially those with mild ionized hypercalcemia, will show a good response to a diet restricted in both calcium and Vitamin D. Such diets must be specially formulated, since none of the commercial cat food diets could be low in either calcium or vitamin D content and still meet AAFCO guidelines to be a “complete and balanced” diet.

Again, I recommend formulating this diet to have a macronutritional composition similar to what cats would eat in the wild (i.e., 40-60% protein, 30-50% fat, and <10% carbohydrates). Products containing high concentrations of vitamin D, such as organ meats and fish oil, should be avoided. Calcium content should be kept restricted to 600 mg per 1000 kcal of diet (in contrast, the minimal adult maintenance requirement set by AAFCO is 1500 mg per 1000 kcal). Magnesium should not be restricted, and acidifiers should never be added.

Ideally, this home-prepared diet is formulated under the guidance of a veterinary nutritionist to ensure that it is nutritionally adequate for the cat. If no response is detected after a month or two on this restricted calcium diet, alternative therapies (e.g., glucocorticoids, alendronate) should be considered.

REFERENCES
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Notes
Special-Needs Cats

PART 1: SUPPORTING CATS WITH CANCER

Cancer is a common cause of death in senior cats. However, advances in veterinary oncology have increased effective treatment choices, so that greater numbers of feline cancer patients are under long term care. Since cancer can be a painful and debilitating disease, supportive care to enhance quality of life (QOL) is important. In one study, 87% of cats undergoing chemotherapy for lymphoma experienced adverse effects.1

However, QOL as perceived by the owners was higher during treatment than before starting chemotherapy and the majority of owners (83%) were happy they treated their cat.2

Dr. Susan Little received her BSc degree from Dalhousie University (Nova Scotia, Canada) in 1983 and her DVM in 1988 from the Ontario Veterinary College, University of Guelph. She has been in feline practice since 1990 and achieved American Board of Veterinary Practitioners certification in Feline Practice in 1997, re-certifying in 2006. She is part owner of two feline specialty practices in Ottawa, Canada. She serves on the board of the Winn Feline Foundation and the American Assoc. of Feline Practitioners, and is a feline medicine consultant for the Veterinary Information Network. Her other activities include committee work for the National Board of Veterinary Medical Examiners, the Pet Nutrition Alliance, and the Canadian Veterinary Medical Association. She is a peer reviewer for the Canadian Veterinary Journal and the Journal of Feline Medicine and Surgery. In 2010, Dr. Little was the recipient of the Canadian Veterinary Medical Assoc. Small Animal Practitioner Award. She is the editor and co-author of a new textbook, The Cat – Clinical Medicine and Management, published by Elsevier in 2011.

Anorexia and weight loss are among the most common reasons owners give for making the decision to euthanize feline cancer patients. Weight loss is characterized by loss of both fat and muscle mass. Weight loss may be due to various factors such as functional impairment due to the tumor location (e.g., oral squamous cell carcinoma), impairment of digestion (e.g., gastrointestinal lymphoma), complications of cancer therapy (e.g., nausea, vomiting, mucositis, early satiety), inappetence due to pain, and cancer cachexia.

Early detection of weight loss and loss of lean body mass in senior cats is important as it may lead to early detection of disease. A study of 258 cats in a nutrition colony that died of cancer, renal failure, and hyperthyroidism determined that weight loss started about 2.5 years before death.2 Body weight loss two years prior to death was over 6% in cats with cancer, renal failure, and hyperthyroidism. Body condition score (BCS) is also prognostic – normal weight or obese cats with cancer live longer than underweight cats. It is important to institute nutritional support early as it may improve both quality of life and response to treatment.

Cancer cachexia is a poorly understood, paraneoplastic process characterized by progressive weight loss and decreasing body condition despite adequate nutritional intake. This type of cachexia is a form of protein-calorie malnutrition associated with delayed wound healing, immunosuppression, and compromise of gastrointestinal, cardiovascular, and pulmonary function. In humans, it is associated with less favorable responses to treatment, reduced survival times, and reduced quality of life.

The goals of nutritional support for cats with cancer are to reduce or prevent toxicosis associated with therapy, ameliorate metabolic alterations, and improve QOL. Cancer patients should undergo regular nutritional assessment that includes a detailed diet history (brand names, format (canned, dry, semi-moist), flavors, amount fed, frequency of feeding, supplements, etc.). The extent of voluntary food intake should also be assessed, as well as gastrointestinal signs associated with the

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Dr. Mark Peterson graduated from the University of Minnesota in 1976, moved to NYC to do an internship and medical residency at The Animal Medical Center (AMC), and then completed a post-doctoral fellowship in endocrinology and nuclear medicine at The New York Hospital-Cornell Medical Center and the AMC. Mark obtained board certification from the American College of Veterinary Internal Medicine in 1981.

Following his training, Mark stayed on as head endocrinologist at the AMC for over 30 years. In 2009, Mark opened the Animal Endocrine Clinic in New York City, a specialty referral hospital devoted exclusively to the diagnosis and treatment of cats and dogs with endocrine disease, where he now cares for his endocrine patients.

Over the last 35 years, most of Dr. Peterson’s clinical and research efforts has been directed toward advancing our understanding of endocrine disorders of the cat and dog, including hyperthyroidism, diabetes mellitus, and adrenal disease. He has published more than 475 journal articles, book chapters, and research abstracts. With more than 300 lecture presentations to his credit, Dr. Peterson is a frequent speaker at veterinary colleges and scientific seminars both in both the United States and abroad.
disease or with therapy, assessment for pain, and assessment for food aversion. Periodic evaluation of a minimum database is recommended to monitor for anemia, increased serum creatine kinase (CK) activity, hypokalemia, or decreasing serum urea nitrogen and creatinine.

The nutritional support plan should include estimation of daily water needs (typically 50-100 mL/kg/day, depending on activity, diet, concurrent disease, etc.). For adult cats, the starting point for daily resting energy requirement (RER) is calculated by the equation \[30 \times \text{body weight in kg} + 70\]. The amount fed should then be adjusted according to monitoring of body weight and BCS.

Dietary recommendations for cats with cancer include (on a dry matter basis): carbohydrate <25\%, fat 25-40\%, n-3 fatty acids >5\% (n6:n3 ratio of 1:1), protein 40-50\%, arginine >2\%, crude fiber >2.5\%. Typically, diets designed for enteral feeding, convalescence, or kitten growth will be most appropriate for cancer patients providing there is no contraindication due to concurrent disease. All that having been said, the most important factor is palatability – will the patient eat the diet? Feline cancer patients should also be monitored for hypocobalaminemia, especially patients with gastrointestinal lymphoma.

Basic supportive care may be required for hyporexic/anorexic cats and could include improving hydration, vitamin B12 supplementation, and treatment of fever, pain, or nausea if present. Nutritional intervention should be implemented if the cat is eating less than 85\% of RER, the cat is anorexic for 3 or more days, or the cat has lost 10\% BW or more in a short period of time. Food aversion occurs readily in cats if they learn to associate eating or the sight or smell of food with feeling sick, nauseous, or painful. It is important to recognize signs of nausea in cats (e.g., gulping, drooling, dropping food from the mouth, turning away from food) and institute treatment early. Feeding strategies can be grouped into three levels based on degree of intervention:

**Level 1 - Simple interventions:** Enhancing smell and palatability can be accomplished by feeding fresh canned foods that are warmed no higher than body temperature, and adding water or chicken or tuna broth. Human baby foods are a popular choice for tempting cats to eat and are acceptable as long as they do not contain onion and are used for less than 2 weeks. Hand-feeding, petting, and praise may also be helpful. Appetite stimulating drugs are not very reliable and many have undesirable side effects so their usefulness is limited.

**Level 2 - Syringe feeding:** Syringe feeding is best for cats that are not totally anorexic and will tolerate the procedure. It may be difficult to meet daily caloric needs with this method, and many cats will struggle and resist. Orogastric feeding with a mouth speculum and tube is not recommended by this author as it is too stressful.

**Level 3 - Assisted feeding:** Early institution of tube feeding has a better outcome than waiting until the patient has end stage disease or is debilitated. Tube feeding is indicated when nutritional support will be needed for more than a few days. Food quantity (mL) required per 24 hours = total energy requirement (kcal) divided by caloric density of diet (kcal/mL). Nasoesophageal (NE) or nasogastric (NG) tubes are easy to place, typically without anesthesia or sedation, and are safe. They are used for short-term nutritional support (no more than 2-3 days). Only liquid diets can be fed using NE/NG tubes. Esophagostomy tubes are well-tolerated in the long term, economical, and easy to place. Diets that are nutritionally complete and balanced may be administered through large bore tubes. No long-term complications (such as esophagheal stricture or diverticulum, esophagitis) have been reported. Other types of feeding tubes, such as gastrostomy tubes, may be appropriate for certain patients.

Pain assessment and control is an essential part of supportive care for cancer patients. An important barrier is that recognizing the signs of pain and its impact on quality of life is challenging in cats. The effects of pain are detrimental to the animal’s wellbeing in the short term, and pain interferes with healing and causes disease in the long term. In cancer patients, pain may be caused by the disease itself or by treatment (e.g., radiation damage or chemotherapy-induced neuropathy).

**REFERENCES**

PART 2: FEEDING THE DIABETIC CAT WITH CHRONIC KIDNEY DISEASE

Normal Glucose Metabolism and Postprandial Glycemia in Cats

Cats are strict carnivores that rely on nutrients in animal tissues to meet their specific and unique nutritional requirements. In their natural habitat, cats consume small prey high in protein (50-60%) with moderate amounts of fat (30-50%) but very minimal amounts of carbohydrates (1-2%). This is in marked contrast to many commercial diets that contain moderate to high amounts of carbohydrates (35-50%).

Evolutionary events shaped the cat’s core metabolism to adapt to this carnivorous diet of animal tissues over a period of millions of years. Their systems are uniquely set up to metabolize this diet which is high in moisture, high in protein, and very low in carbohydrates. Because this is the diet they have relied upon for tens of thousands of years, they do not have the ability to process carbohydrates very efficiently. This becomes extremely important when selecting a diet for cats with diabetes (see below).

So what are these specific feline adaptive mechanisms that have developed to meet the requirements of a carnivorous diet? First, cat’s gluconeogenic pathway provides an almost continuous source of carbon skeletons for glucose or energy production. Secondly, glucokinase concentrations are markedly reduced or absent, whereas hexokinase activity is increased. This is in marked contrast to the liver of omnivores (dogs, man), which contains both glycolytic enzymes that act to catalyze the phosphorylation of glucose during glycolysis. In addition, cats have reduced disaccharidase activity in the small intestine, reduced and delayed insulin secretion, and delayed gastric emptying.

As a result of these differences, plasma glucose clearance rates are longer in cats compared to dogs or humans after feeding a moderate to high carbohydrate meal — in other words, even normal cats have much more prolonged postprandial period of hyperglycemia than might be expected. In healthy humans and dogs, postprandial hyperglycemia normally persists for 2 to 5 hours. In contrast, a recent study of healthy cats found that both serum glucose and insulin concentrations remained significantly increased for a median time of 12 hours following ingestion of a moderate carbohydrate meal (25% ME), and that both glucose and insulin concentrations remained above baseline values for 24 hours in approximately 20% of the cats. Most feline diets contain ever higher amounts of carbohydrate and therefore would be expected to result in more severe postprandial hyperglycemia and a longer time to return to baseline.

Dietary Management of Cats with Diabetes Mellitus: Key Part of Treatment!

In cats with diabetes, a primary goal of therapy is to minimize the degree of hyperglycemia that develops after feeding in order to lessen the subsequent demand on beta-cells to secrete insulin. By doing this, we decrease the effect of “glucose toxicity” and allow the pancreatic islet cell to hopefully recover. But how do we do this? It has long been known and is well accepted “dogma” by most practicing veterinarians who specialize in feline medicine that feeding a low-carbohydrate diet is the mainstay in the treatment of diabetes mellitus, especially if remission of the diabetic state is the goal. Feeding a low carbohydrate diet will improve insulin sensitivity, reduce or eliminate the need for exogenous insulin, and help stabilize glucose metabolism in these cats. Again, one major way a low-carbohydrate diet improves the diabetic state is by helping to prevent severe and prolonged postprandial hyperglycemia.

The sooner one starts the diabetic cat on a low-carbohydrate, high-protein diet, the better. By “low-carbohydrate,” I mean a diet that provides <10% of the calories as carbohydrate. Some cats will do fine on a slightly higher-carbohydrate diet (12-14%) whereas others do best on a diet containing <7% carbohydrate. To achieve these low carbohydrate levels, we must formulate a homemade diet or feed a canned food — none of the available dry cat foods are very low in carbohydrates and most are too low in protein. That is why I believe it’s best to limit the amount of dry food that is fed to diabetic cats or even better, not feed dry food at all.

When we reduce the content of carbohydrate in a cat food, we must raise the content of either protein or fat, or both. I like to do both, feeding my diabetic cats a diet that mimics the composition their prey in the wild: about 40-60% of protein with the remaining amount in fat. This higher than average protein level also helps restore and maintain lost muscle mass, since many diabetic cats will develop “sarcopenia” as they age.

Another plus for feeding a low-carbohydrate, high-protein diets in cats with relatively early diabetes is that this diet composition (together with insulin treatment) greatly improves the diabetic remission rate. If the diabetic cat goes into remission (no more insulin needed to maintain euglycemia), we recommend maintaining a restricted carbohydrate diet for life to help prevent relapse of the diabetic state.

Dietary Management of Cats with Diabetes Mellitus that Develop Concurrent Renal Disease

Chronic renal failure (CKD) is common in geriatric cat, so it should not be surprising that many diabetic cats with develop concurrent CKD as they age. In addition to medical therapy, nutritional modification is one of the mainstays of long-term management of CKD. These commercial renal diets made for cats are restricted in protein and phosphorus and are generally supplemented with potassium, omega 3 fatty acids, and water-soluble vitamins.

As most renal diets are restricted in protein, adequate caloric density is achieved by increasing the amount of carbohydrate and fat in the diet. None of the commercial renal diets are “low-carb.” As noted above, this becomes an issue when selecting the best diet for a cat with diabetes, in which low-carbohydrate diets are preferred. Switching the diabetic cat from a low-carbohydrate diet to a typical renal diet certainly sets up the
cat for deterioration of diabetic management or relapse of the diabetes due to the high-carbohydrate content of these low-protein renal diets.6,7

Although it has long been one of the cornerstones of diet therapy for CRD, protein restriction is controversial at best.8,9 Reducing consumption of non-essential amino acids theoretically results in decreased production of nitrogenous waste, reduced solute workload on the kidney, and improvement of clinical signs of uremia. However, we have no evidence that feeding low-protein diets slows progression of disease or is even needed in the early stages of CKD in cats.

Stated again (because most veterinarians find it impossible to believe), dietary protein does not cause CKD or alter the course of kidney disease. The terms “renal protective” or “renal friendly” diet are often used which reinforces the fact that many people think that feeding low protein diets actually has a protective effect on kidneys. Low dietary protein only decreases the symptoms associated with severe, end-stage kidney failure; it does not slow it or cure it.9

Another major concern that I have with some of the prescription kidney diets is that they restrict protein to the point that some cats—especially those with a catabolic concurrent disease such as diabetes—will begin to catabolize their own muscle mass to fulfill their protein needs. Remember that cats also tend to lose muscle mass as they age, a phenomenon is called sarcopenia.8 Therefore, geriatric cats require more protein than younger animals as they age, not less! Studies have shown that diets containing higher levels of protein increases the percentage of muscle tissue and decreases sarcopenia in geriatric subjects. Feeding a low-protein diet would do the opposite and increase muscle loss. Therefore, I believe that protein restriction only becomes necessary in the late stages of renal failure (late IRIS stage 3 to Stage 4 CKD).

So what diets do I recommend for feeding cats with diabetes that develop concurrent renal disease? I continue feeding a diet low in carbohydrates (<10-15%) and relatively high in protein (35-50%) to best manage the diabetic state (either active or in remission) and to prevent the development of loss of lean muscle mass. In some cats, it is best to feed a homemade diet that is formulated for them. This, by far, is my favorite way to feed my CKD cats, but it is more practical to feed commercial canned cat foods.

Phosphate restriction is key for cats with CKD so I formulate a low-phosphate diet or select an OTC cat food relatively low in phosphorus.11 For this purpose, a phosphate content that is < 100 mg per 100 kcal is ideal. If an OTC diet is selected, look for one that contains < 250 mg of phosphate per 100 kcal (see http://binkyspage.tripod.com/CanFoodNew.html to determine phosphate content of many cat foods). You may have to have to call a company for specific information. Fish-based cat foods are often high in phosphorus, so I stay away from these diets for cats with CKD.

If a low-phosphate diet cannot be fed or serum phosphate concentrations remain above 4.5 mg/dl, use of phosphate binders is started.11 Aluminum hydroxide (100 mg/kg/day) is commonly used and can be mixed with canned or dry food. I prefer use of lanthanum (Renalzin) due to its efficacy and low toxicity, but this drug is not available in the US at this time.

Other supplementation for OTC diets to consider include the following: 9 1) Fish oil 1,000 mg/day (1 capsule of most products) for the omega-3 fatty acids. Poke a pin in a capsule, drip it onto the food, and mix well. 2) Potassium gluconate (or citrate if acidic), as needed for hypokalemia. 3) Water-soluble B-complex vitamins are also given to most cats.

Finally, I also monitor and treat both hypertension and proteinuria in these cats. In some cats, we also start the cats on calcitriol (the serum phosphatase must be < 6 mg/dl before we start).

REFERENCES

Nutritional Support For Each Condition

Feline

Joint support
MOBILITY SUPPORT

Kidney failure
RENAL LP MODIFIED

Diabetes mellitus
DIABETIC

Liver failure
HEPATIC

Stress management
CALM

Lower urinary tract disease
URINARY SO
URINARY SO MODERATE CALORIE

Weight management
CALORIE CONTROL
SATIETY SUPPORT
CALORIE CONTROL HC
HIGH FIBER

Gastrointestinal disorders
FIBER RESPONSE
INTESTINAL MODERATE CALORIE
INTESTINAL HIGH FIBER
INTESTINAL HIGH PROTEIN

Disorders causing skin disease
HYPOALLERGENIC
SELECTED PROTEINS
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