

Nestlé Purina
PetCare
Handbook of
**Canine
and Feline
Clinical
Nutrition**

A Convenient
Reference Guide
for Everyday Use
in Veterinary Practice

 Nestlé PURINA



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Allergic Dermatitis – Canine

Stephen D. White, DVM, DACVD

Definition

Allergic dermatitis refers to any hypersensitivity disorder that causes an inflammatory condition of the skin. The most common of these disorders affecting dogs are **flea allergy**, **atopic dermatitis**, and **food allergy** (also known as adverse food reactions).

Key Diagnostic Tools and Measures

The history, including seasonality and diet or diet changes, is the first step in diagnosis of the cause of allergic dermatitis. During physical examination, the location of lesions can provide important diagnostic information: if the cause is flea allergy, lesions are likely to be found on the caudal half of body; with atopic dermatitis, lesions are found on the paws, axilla, face, and ears; and with food allergy, lesions may be similar to those seen in atopic dermatitis. Skin cytology is needed to check for secondary bacterial and *Malassezia* infections.

Pathophysiology

Atopic dermatitis is mediated by allergen-specific immunoglobulin E (IgE). Allergens, via the percutaneous route, bind to IgE antibodies bound to mast cells, which then release inflammatory substances. Recent work suggests that 1) some atopic dogs have a defect in filaggrin (a component of the stratum corneum), and 2) in normal dogs, the stratum corneum barrier function (against infection) may be helped by niacinamide supplementation.

The etiology of food allergy is not well understood, but both cell-mediated and antibody-mediated processes are probably involved. In flea allergy dermatitis, the allergens are proteins in the saliva of the flea.

Signalment

Golden retrievers, Labrador retrievers, terriers, Dalmatians, and Shar-Peis are predisposed breeds. In the UK, atopic retrievers were reported likely to have atopic offspring, especially atopic sires. Signs usually are observed in atopic dogs between 1 and 7 years of age (median 1.7). There are no known gender predilections.

Thirty percent of food-allergic dogs show signs by one year of age or earlier. Breed predilections are controversial; there are no known gender predilections. There are no age, breed, or gender predilections for flea allergy.

Key Nutrient Modifications

In dogs with atopic dermatitis, essential fatty acids (EFAs) may be used as antipruritics.¹ A difference in efficacy between EFA supplements of omega-3 fatty acids versus those containing a mixture of omega-3 and omega-6 is controversial. In dogs, EFAs may have as high as a 25% chance of reducing pruritus, particularly when combined with antihistamine treatment. When EFA supplements are included in the dog food, the success rate in one open trial was 42% (good to excellent control of pruritus); in another trial it was 44%. A recent article noted that improvement seen in atopic dogs with EFA supplementation did not always correlate with total fatty acid intake or with the ratio of omega 6:3 fatty acids. Another report documented the steroid-sparing effect of EFAs in some atopic dogs.²

Recommended Ranges of Key Nutrients

Nutrient	% DM	mg/100 kcal	% DM	mg/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Total n3 (from fish oil)	0.4–0.8	81–156	n/a	n/a
EPA	0.24–50	50–94	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

If supplements are used, an anecdotal recommendation is to use at least 36–44 mg/kg body weight/day of eicosapentaenoic acid (EPA) from fish oil, or approximately 1 g fish oil/5 kg body weight.

Therapeutic Feeding Principles

Food Allergy: Diagnosis and therapy is feeding an elimination (“hypoallergenic”) diet.³ The elimination diet is based on previous exposure to various food stuffs, and may be either home-made or commercial elimination (limited antigen) diets.

Other than fresh water, nothing else should be fed during the elimination diet trial: no vitamins, chewing toys, flavored medications, or toothpaste. Because a home-made elimination diet is not a balanced one, owners should be warned that the dog may lose weight, develop a “dull” hair coat or scaling, or be hungrier than usual.

The length of the elimination diet is usually from 8 to 12 weeks. Persistence of some pruritus at 12 weeks may indicate the presence of other concurrent hypersensitivities. In cases in which antibiotics are given to treat secondary infections, or oral corticosteroids for severe pruritus, the diet should continue 2 weeks past when these treatments are discontinued in order to properly judge its efficacy.

Upon resolution of clinical signs, the dog is challenged with its regular diet to confirm the diagnosis. Recurrence of clinical signs is usually noted within 2 weeks. The dog is then fed its elimination diet again, and the owner may challenge with suspected allergens, each being given 1 to 2 weeks at a time. The most common proven allergens in dogs are beef, chicken, milk, eggs, corn, wheat, and soy. Once the offending allergens are identified, commercial dog foods without them or a hydrolyzed protein diet may be fed. When the owners refuse to do provocative testing, a limited-antigen dog food may be used.

■ **Treats** – No treats except for those containing the same ingredients as the elimination diet can be used during the diet trial. Canned or dry commercial elimination diets may be baked (the latter mixed with water) in the form of dog biscuits. After a diagnosis of food allergy is made and an appropriate maintenance diet found, treats may be introduced on a weekly basis to evaluate any recurrence of clinical signs.

■ **Tips for Increasing Palatability** – Occasionally, heating the diet before feeding will improve palatability. If the dog refuses to eat an elimination diet after 2 to 3 days, it is best to try a diet with another protein.

■ **Diet Recommendations** – Elimination diets should avoid foodstuffs fed previously. For home-made diets, “novel” proteins such as pork, pinto beans, rabbit, duck, and tuna and carbohydrates such as potatoes, sweet potatoes, and rice are options. Commercial diets should specifically be marketed as limited-allergen. These may consist of “novel” proteins or

hydrolyzed proteins that are too small a molecular weight to trigger the dog's immune system.

Client Education Points

- **Atopic Dermatitis** – Clients should recognize that this disease needs to be controlled and managed throughout the dog's life; if EFAs are helpful in controlling pruritus, they will need to be given life-long.
- **Food Allergy** – Clients need to be very strict throughout the elimination diet trial. They should keep a daily record, noting pruritus, anything eaten that is not in the diet, and, ideally, any change in feces (e.g., consistency, odor).
- **Flea Allergy** – The role of the flea, and the various insecticides available to protect the dog from the flea's bite, should be explained. While the efficacy of EFAs as an aid in managing the pruritus of flea allergy has not been extensively investigated, the use of EFAs as an adjunct to parasitocidal treatment may be attempted.

Common Comorbidities

Atopic dermatitis and flea allergy often exist in the same dog. All three allergic dermatoses may have secondary bacterial (usually *Staphylococcus* spp) and/or *Malassezia* spp (yeast) infections. These will need to be treated, as well as the allergic dermatitis, as the infections in and of themselves may cause pruritus. Food allergy also may cause gastrointestinal signs. While frank diarrhea and/or vomiting probably only occur in 10% of dogs with food allergy-caused dermatitis, soft or poorly formed stools are noted more often; their occurrence should be specifically asked about in taking the patient's history.⁴ Rarely, idiopathic epilepsy has been noted to be caused by food allergy.⁵

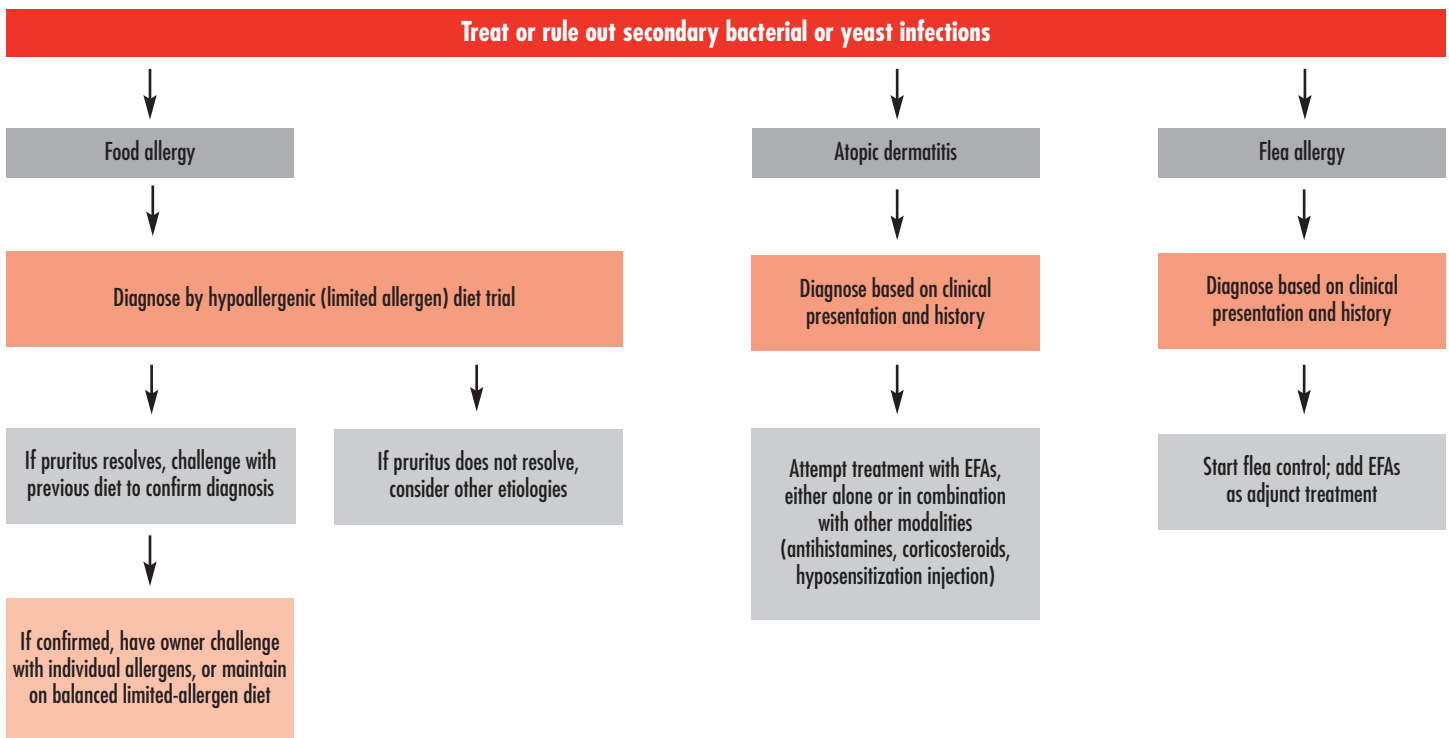
Interacting Medical Management Strategies

As noted above, antibiotics and anti-yeast medications (usually azoles) will need to be given to treat secondary infections. Such medications are usually administered for 4 to 8 weeks. Topical medications such as shampoos or dips (rinses) may also be used to control secondary infections, as well as an aid to treat pruritus. Corticosteroids may be needed to control pruritus, although their use should be limited to oral products with short acting effect, such as prednisone or prednisolone. The lowest possible every-other-day dosage should be the goal when corticosteroids are deemed necessary. Cyclosporine is often helpful in controlling clinical signs of atopic dermatitis; its efficacy in other allergic dermatoses has not been evaluated. Also relevant to atopic dermatitis is hyposensitization (immunotherapy, or "allergy shots") based on intradermal or serologic testing. Hyposensitization is not recommended for the treatment of food allergy; its role in the management of flea allergy needs further investigation.

Monitoring

Dogs with allergic dermatitis should be rechecked at least twice yearly following elimination of clinical signs; more frequent rechecks are indicated dependent upon the potential adverse effects of treatment (such as those seen with cyclosporine or corticosteroids). Dogs should also be evaluated if pruritus recurs, as this may mean either a relapse in the treatment of the allergic dermatitis (e.g., eating a "forbidden" allergen or a lapse in flea control), the recurrence of a secondary infection, or the emergence of another hypersensitivity (e.g., the food-allergic dog that develops atopic dermatitis).

Algorithm – Nutritional Management of Canine Allergic Dermatitis



Allergic Dermatitis – Feline

Stephen D. White, DVM, DACVD

Definition

Allergic dermatitis refers to any hypersensitivity disorder that causes an inflammatory condition of the skin. The most common of these disorders affecting cats are **flea allergy**, **food allergy** (also known as adverse food reactions), and **atopic dermatitis**.

Key Diagnostic Tools and Measures

The history, including seasonality and diet or diet changes, is the first step in diagnosis of the cause of allergic dermatitis. During physical examination, the location of lesions can provide important diagnostic information: if the cause is flea allergy, lesions are likely to be found on the caudal half of body and dorsal neck, and miliary dermatitis (encrusted papules) is often present. With atopic dermatitis and food allergy, lesions include facial, head, and neck pruritus; miliary dermatitis, eosinophilic granuloma complex; and self-induced alopecia. Skin cytology is needed to check for secondary bacterial and *Malassezia* infections.

Pathophysiology

Atopic dermatitis is mediated by allergen-specific immunoglobulin E (IgE). Allergens, via the percutaneous route, bind to IgE antibodies bound to mast cells, which then release inflammatory substances.¹

The etiology of food allergy is not well understood, but both cell-mediated and antibody-mediated processes are probably involved.

In flea allergy dermatitis, the allergens are proteins in the saliva of the flea.

Signalment

Age, breed or gender predilections have not been consistently proven for cats with atopic dermatitis, food allergy or flea allergy.

Key Nutrient Modifications

In cats with atopic dermatitis, essential fatty acids (EFAs) may be used as antipruritics.

Recommended Ranges of Key Nutrients

Nutrient	% DM	mg/100 kcal	% DM	mg/100 kcal
	Recommended dietary level	Minimum dietary requirement*		
Total n3 (from fish oil)	0.4–0.8	81–156	n/a	n/a
EPA	0.24–50	50–94	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

If supplements are used, an anecdotal recommendation for dogs is to use at least 36–44 mg/kg body weight/day of eicosapentaenoic acid (EPA) from fish oil, or approximately 1 g fish oil/5 kg body weight. There is no evidence to determine if this is or is not appropriate for cats.

Therapeutic Feeding Principles

Food Allergy: Diagnosis and therapy is feeding an elimination (“hypoallergenic”) diet. The elimination diet is based on previous exposure to various food stuffs, and may be either home-made or commercial elimination (limited antigen) diets.

Other than fresh water, nothing else should be fed during the elimination diet trial: no vitamins, chewing toys, flavored medications, or toothpaste. Because a home-made elimination diet is not a balanced one, owners should be warned that the cat may lose weight, develop a “dull” hair coat or scaling, or be hungrier than usual. The new diet should be introduced gradually over a few days by initially mixing it with the old diet. Owners should be warned that failure of the cat to eat the new diet for more than 2 days could result in disease (hepatic lipidosis) and should quickly prompt the search for a more palatable diet.

The length of the elimination diet is usually from 8 to 12 weeks. Persistence of some pruritus at 12 weeks may indicate the presence of other concurrent hypersensitivities. In cases in which antibiotics are given to treat secondary infections, or oral corticosteroids for severe pruritus, the diet should continue for 2 weeks past when these treatments are discontinued in order to properly judge its efficacy.

Upon resolution of clinical signs, the cat is challenged with its regular diet to confirm the diagnosis. Recurrence of clinical signs is usually noted within 2 weeks. The cat is then fed its elimination diet again, and the owner may challenge with suspected allergens, each being given 1 to 2 weeks at a time. The most common proven allergens in the cat are milk and dairy products, fish, and beef. Once the offending allergens are identified, commercial cat foods without them may be fed. When the owners refuse to do provocative testing, a limited-antigen cat food may be used.

■ **Treats** – No treats except for those containing the same ingredients as the elimination diet can be used during the diet trial. After a diagnosis of food allergy is made and an appropriate maintenance diet found, treats may be introduced on a weekly basis to evaluate any recurrence of clinical signs.

■ **Tips for Increasing Palatability** – Occasionally, heating the diet before feeding will improve palatability. If the cat refuses to eat an elimination diet after 2 days, it is necessary to try a diet with another protein.

■ **Diet Recommendations** – Elimination diets should avoid foodstuffs fed previously. For home-made diets, “novel” proteins such as pork, rabbit, and duck and carbohydrates such as sweet potatoes and tapioca are options. Commercial diets should specifically be marketed as limited-allergen. These may consist of “novel” proteins or hydrolyzed proteins that are too small a molecular weight to trigger the cat’s immune system.

Client Education Points

- **Atopic Dermatitis:** Clients should recognize that this disease needs to be controlled and managed throughout the cat’s life; if EFAs are helpful in controlling pruritus, they will need to be given life-long.
- **Food Allergy:** Clients need to be very strict throughout the elimination diet trial. They should keep a daily record, noting pruritus, anything eaten that is not in the diet, and, ideally, any change in feces (e.g., consistency, odor).
- **Flea Allergy:** The role of the flea, and the various insecticides available to protect the cat from the flea’s bite, should be explained. While the efficacy of EFAs as an aid in managing the pruritus of flea allergy has not been extensively investigated, the use of EFAs as an adjunct to parasitocidal treatment may be attempted.

Common Comorbidities

All three allergic dermatoses may have secondary bacterial (usually *Staphylococcus* spp) and/or *Malassezia* spp (yeast) infections.² These will need to be treated as well as the allergic dermatitis, as the infections in and of themselves may cause pruritus.³ Food allergy also may cause gastrointestinal signs, especially colitis. Rarely, angioedema or asthma-like signs have been noted to be caused by food allergy.⁴

Interacting Medical Management Strategies

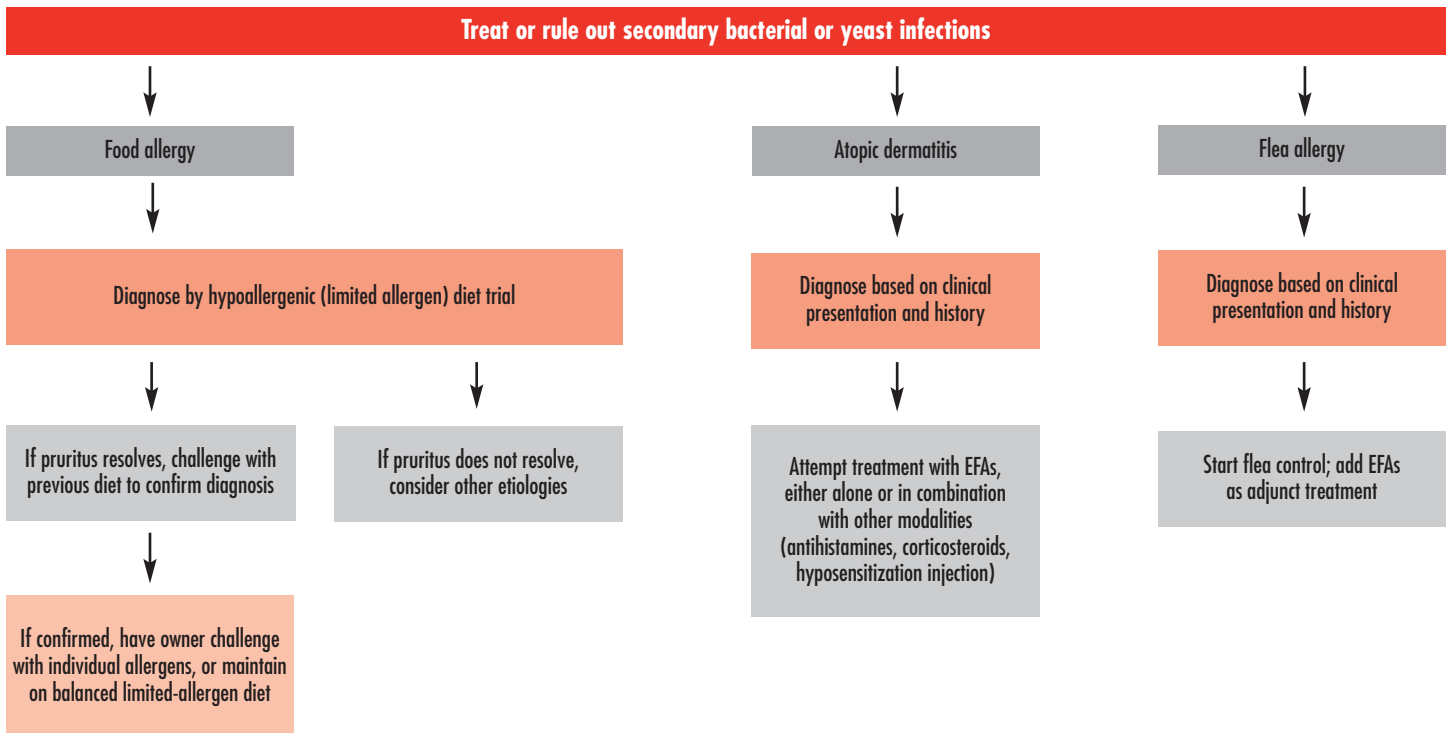
As noted above, antibiotics and anti-yeast medications (avoiding ketoconazole due to its reported hepatotoxic effects in cats) will need to be given to treat secondary infections. Such medications are usually administered for 4 to 8 weeks. Corticosteroids may be needed to control pruritus, although their use should be limited to oral products with short-acting effect, such as prednisolone (more effective in many cats than prednisone). The lowest possible every-other-day dosage should be the goal

when corticosteroids are deemed necessary. Cyclosporine is often helpful in controlling clinical signs of atopic dermatitis; its efficacy in other allergic dermatoses has not been evaluated. Also relevant to atopic dermatitis is hyposensitization (immunotherapy, or “allergy shots”) based on intradermal or serologic testing. Hyposensitization is not recommended for the treatment of food allergy; its role in the management of flea allergy needs further investigation.

Monitoring

Cats with allergic dermatitis should be rechecked at least twice yearly following elimination of clinical signs; more frequent rechecks are indicated dependent upon the potential adverse effects of treatment (such as those seen with cyclosporine or corticosteroids). Cats should also be evaluated if pruritus recurs, as this may mean either a relapse in the treatment of the allergic dermatitis (e.g., eating a “forbidden” allergen or a lapse in flea control), the recurrence of a secondary infection, or the emergence of another hypersensitivity (e.g., the food-allergic cat that develops atopic dermatitis).

Algorithm – Nutritional Management of Feline Allergic Dermatitis



Osteoarthritis – Canine

Denis J. Marcellin-Little, DEDV, DACVS, DECVS, DACVSMR

Definition

Osteoarthritis is a progressive joint disease with loss of cartilage integrity resulting in the production of new bone at the articular surface margins, in an increase in thickness of the joint capsule, and in pain.

Key Diagnostic Tools and Measures

- Changes in willingness or ability to exercise or play
- Pain response to joint manipulation, decrease in joint motion, or crepitus
- Lameness or weight shifting diagnosed using a force plate

Pathophysiology

Joint subluxation, excessive impact loading, articular fractures, or other causes trigger osteoarthritis. These triggering factors activate the division and multiplication of chondrocytes. Initially, the production of quantitatively and qualitatively abnormal proteoglycan and collagen is increased. Eventually, proteoglycan content is decreased, the collagen matrix structure is altered, and normal cartilage structure and function is lost. Osteophytes form around the joint.

Signalment

Osteoarthritis is common in dogs. It is primarily the consequence of hip dysplasia, elbow dysplasia, and the rupture of cranial cruciate ligaments. Larger dogs and dogs with lower ratio of muscle mass/subcutaneous tissue mass (Saint Bernards, for example) are predisposed. Overweight, older, and chondrodystrophic dogs are also predisposed.

Key Nutrient Modifications

Body condition is the factor that has most impact on the progression of osteoarthritis.¹ It is therefore critically important to maintain dogs with known or potential osteoarthritis at an optimal body condition score (BCS; see Appendix I) of 4 or 5 out of 9, by limiting caloric intake.⁶

An increase in the concentration of proteoglycan building blocks (glucosamine and chondroitin sulfate) led to a decrease in clinical signs in several large studies of people with moderate to severe osteoarthritis.^{2,3} The evidence supporting its use in dogs is more scant. An increase in the concentration of n3 polyunsaturated fatty acids, particularly eicosapentaenoic acid, led to a decrease in the lameness of dogs with osteoarthritis in one clinical trial.⁴ Several herbs have been shown to decrease the clinical signs of arthritic people but their safety and efficacy is not documented in dogs.⁵

Recommended Ranges of Key Nutrients

Nutrient	% DM	mg/100 kcal	% DM	mg/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Total n3 (from fish oil)	1.0–2.0	240–300	n/a	n/a
EPA	0.5–1.0	100–200	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Caloric intake should be controlled to keep arthritic dogs in a lean body condition. Dietary protein intake should not be restricted.

Therapeutic Feeding Principles

The first priority for the management of dogs with osteoarthritis is achieving and maintaining a lean body condition. Other priorities include the administration of compounds that may decrease joint pain through their anti-inflammatory properties. These include glucosamine sulfate or hydrochloride, chondroitin sulfate, and omega-3 fatty acids, particularly eicosapentaenoic acid.

■ **Treats** – Treats for dogs with osteoarthritis should be highly palatable and have a low caloric and low fat content to avoid weight gain.

■ **Tips for Increasing Palatability** – The fat content of food for dogs with osteoarthritis is not severely restricted, and thus palatability seems to be high overall.

■ **Diet Recommendations** – A diet designed for the dietary management of dogs with osteoarthritis contains omega-3 fatty acids and glucosamine, and has a moderate fat content (12.0% min of dry content), a high protein content (30% min of dry content), and a moderate fiber content (4.0% min of dry content). Obese dogs with osteoarthritis may be fed a diet that has a low fat content (4.0% to 8.5% of dry content), a moderate protein content (26.0% min of dry content), and a high fiber content (16.0% min of dry content).

Client Education Points

- Osteoarthritis negatively impacts the mobility of dogs and decreases their lifespan.⁷
- Osteoarthritis progresses more rapidly in overweight dogs than in fit dogs.¹
- The clinical signs of osteoarthritis decrease in overweight dogs who lose weight.⁸
- The signs of osteoarthritis may decrease after administration of nutritional supplements.⁴
- Low-impact activities may be beneficial to patients with osteoarthritis.^{4,9}
- Rapid changes in temperature may increase the pain perceived from arthritic joints.⁴

Common Comorbidities

Osteoarthritis can lead to chronic joint pain. The pain perceived from arthritic joints is variable among joints (the elbow joint is *less forgiving* than the hip joint), dog sizes (clinical signs are more severe in small and giant dogs compared with medium-sized dogs), and individuals. As a consequence of chronic joint pain, an affected joint may lose motion in specific directions, affected limbs become weaker, and affected patients lose muscle and cardiovascular fitness. The combination of painful, less mobile joints, weaker limbs, and unfit dogs lead to a loss of mobility and potential weight gain.

Interacting Medical Management Strategies

Osteoarthritis is not automatically associated with systemic diseases but, because the signs of osteoarthritis are amplified in older patients, osteoarthritis and other chronic diseases (for example, chronic renal insufficiency, heart failure, hyperadrenocorticism) are often managed simultaneously. The medical and nutritional management of chronic cardiac, renal, gastrointestinal, or other systemic diseases has priority over the medical and nutritional management of osteoarthritis. Other strategies, including therapeutic exercises, cold therapy, or stretching, may also be

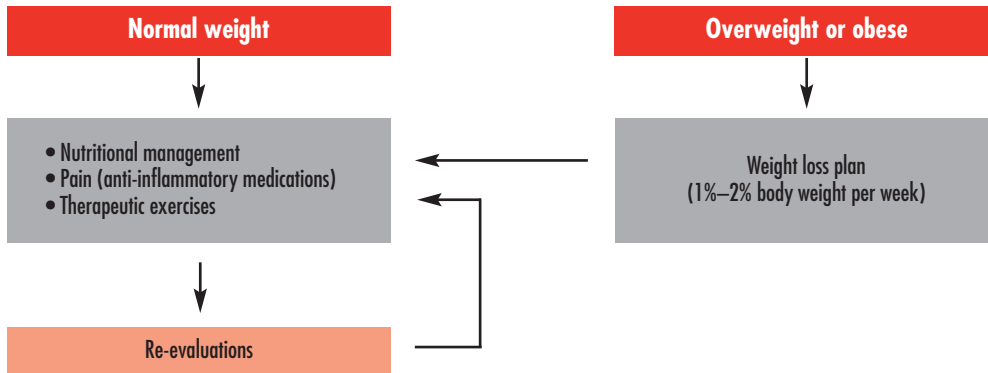
implemented based on specific patient needs. Therapeutic exercises may be used to strengthen, increase endurance, and stretch. The recommended weight loss rate in overweight, arthritic patients with systemic diseases is approximately 1% body weight per week. This is lower than the recommended weight loss rate of 1% to 2% body weight per week in overweight, arthritic patients free of systemic diseases.

Monitoring

Monitoring of the clinical signs and response to medical and nutritional

management of arthritic patients may be done at re-evaluations that include the assessment of locomotion (stance, gait at a walk and trot), and the pain response to joint palpation. The frequency of monitoring is linked to the severity of clinical signs. Younger, less severely affected patients may be re-evaluated intermittently (every 6 months, for example). Older, severely affected patients may be evaluated monthly. Weight loss should be assessed often (every 2 to 4 weeks). Patient monitoring should also include phone communication with the owner every week. Adjustments to medical, nutritional, and exercise programs can be made at that time.

Algorithm – Nutritional Management of Symptomatic Canine Osteoarthritis



Osteoarthritis / Degenerative Joint Disease – Feline

B. Duncan X. Lascelles, BSc, BVSC, PhD, MRCVS, CertVA, DSAS(ST), DECVS, DACVS

Definition

Arthritis is usually defined as inflammation of a joint usually characterized by swelling, pain, and restriction of motion. *Degenerative joint disease* (DJD) is a progressive destruction of the components of joints—cartilage, subchondral bone, ligaments, and joint capsule. DJD can affect both synovial joints and amphiarthrodial joints. We know little about joint disease in the cat, and so the term DJD will be used as arthritis invariably results in degrees of DJD.

Key Diagnostic Tools and Measures

History of Impaired Mobility & Activity. Very little work on the assessment of osteoarthritic pain has been performed in cats.^{1,2} From early work, however, it appears that an approach similar to that in dogs is likely to be most successful—that is, owners need to be centrally involved in the process. The difficult part of assessment of osteoarthritic pain in cats is that the activities that are altered by osteoarthritis are less fully understood than in dogs. A recent study of 28 cats with osteoarthritis showed that overt lameness was not the most common clinical feature.¹ Instead, features like jumping up, jumping down, height of the jump, general movement, “grumpiness” on handling, and seeking seclusion are likely to be activities and behaviors that should be followed. These findings were confirmed in a recently published randomized, blinded study using activity monitors as an objective measure of mobility.² In this study, the activities that owners picked out for the assessment were jumping up/down, playing (toys, cats), running (to food; from dog), lying down, moving up stairs, walking, sharpening claws, grooming, using litter tray, and hunting. More recent work by the same authors has shed more light on activities that may be appropriate to ask owners about when assessing DJD pain in cats.³

Performance Tests in Clinic. These tests can be difficult to do with cats, but some simple tests include 1) placing the cat down and watching it to move across the room, 2) encouraging the cat to jump off a table or chair, and 3) encouraging the cat to jump up to get to the carrier. Evaluating how the cat performs such tasks can, in some cases, provide valuable information, helping the clinician to locate the problem and assess the degree of the impairment.

Orthopedic Examination. Guidelines on how to perform a productive orthopedic examination in the dog are scarce, and virtually nonexistent for the cat. The following are a few pointers:

- Be prepared to put the required time in
- Have a calm approach
- Use a room that is quiet, away from barking dogs, and does not have “hiding places” where a cat can get lodged in
- Use a surface that is soft and will not slip around
- Minimize restraint
- Perform the examination in the position the cat is comfortable in (for example, standing, lying, or in the owners arms)
- Examination is often facilitated by having the owner present, but some cats may be more relaxed if the owner is not present.
- The examination should include every joint and the axial skeleton
- Be prepared to do the examination in parts and repeat it later if necessary.

Cats appear to resent extension of joints more than dogs, and will often react adversely to extension of the elbow and stifle. This reaction should not be over interpreted.

Goniometry. Goniometry appears to be a valid tool in the cat,⁴ but only one study has used goniometry in this species, probably because of the difficulty in defining pain reactions in cats.⁵ No studies have yet evaluated goniometry as a tool in assessing joint pain-free range of motion (ROM). The normal ranges of joint motion in the cat have been defined.⁴ Unpublished data from the author (BDXL) indicates that decreased ROM is significantly associated with radiographic evidence of DJD.

Radiographic Assessment. Orthogonal views of painful joints should be obtained. If radiographic signs consistent with degenerative joint disease are not seen, however, joint disease should not be ruled out. The author has found that there is only moderate overlap between the joints that appear painful clinically, and those that have radiographic signs of degenerative joint disease.

Pathophysiology

Although Allan outlines common causes of osteoarthritis in cats,⁶ there is very little documented evidence of the cause of feline DJD. Two primary forms of osteoarthritis are fairly well recognized in cats: Scottish Fold osteochondrodysplasia⁷⁻⁹ and mucopolysaccharidosis. Currently, the documented secondary causes of DJD in cats are nutritional (secondary to excess vitamin A), hip dysplasia, and the noninfectious polyarthropathies and infectious arthropathies.

Signalment

It appears that the incidence of DJD in the cat increases with increasing age,¹⁰⁻¹² and this was confirmed in a recent prospective cross-sectional study.¹³ No other signalment associations are known to exist, but there have been suggestions that hip dysplasia leading to DJD is more common in purebred cats.¹⁴

Key Nutrient Modifications

Diets containing high levels of the omega-3 fish oil, especially docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), are likely to be beneficial. One study in cats has suggested a beneficial effect of a diet high in EPA and DHA on some postulated serum markers of joint disease in arthritic cats.¹⁵ There is one blinded, prospective, placebo-controlled published study investigating the pain relieving effects of a ‘DJD diet’ in cats.¹⁶ The study evaluated a DJD diet containing high levels of DHA and EPA and also chondroitin sulfate, glucosamine hydrochloride, and green-lipped mussel extract. Activity significantly decreased in the group fed the control diet and significantly increased in the group fed the DJD diet. Much work is still to be done in this area, but dietary modulation may be an effective means of treating DJD-associated pain in cats.

Recommended Ranges of Key Nutrients

Nutrient	% DM		mg/100 kcal	
	Recommended dietary level	Minimum dietary requirement*		
Total n3 (from fish oil)	1.0–2.0	240–300	n/a	n/a
EPA	0.5–1.0	100–200	n/a	n/a

If omega-3 fatty acids are used, preformed EPA from fish oil should be provided. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

As indicated, the science of joint protection or preservation in cats is not advanced enough to be able to recommend any therapeutic feeding principles. Anecdotal suggestions include the feeding of supplements such as glucosamine-chondroitin sulfate and avocado/soybean unsaponifiables (ASU).

Client Education Points

- Weight has been associated with clinically apparent lameness in cats,¹⁷ with overweight cats being 4.9 more times likely to develop lameness requiring veterinary care. No cause and effect relationship has been established between obesity as a cause of joint disease in cats. However, as in other species, it makes sense to keep arthritic cats as light as possible.
- Traditionally, owners have been counseled to avoid table foods and snacks for their obese cats. However, one study suggested little or no advantage to restricting table foods and snacks as a part of weight-control or weight-loss programs.¹⁸ Indeed, the authors of that study suggested

that permitting owners to offer snacks and treats may improve compliance with weight management programs while maintaining the owner-pet bond.¹⁸

- It is important to strive to alleviate the pain associated with DJD in cats to improve their comfort and quality of life.

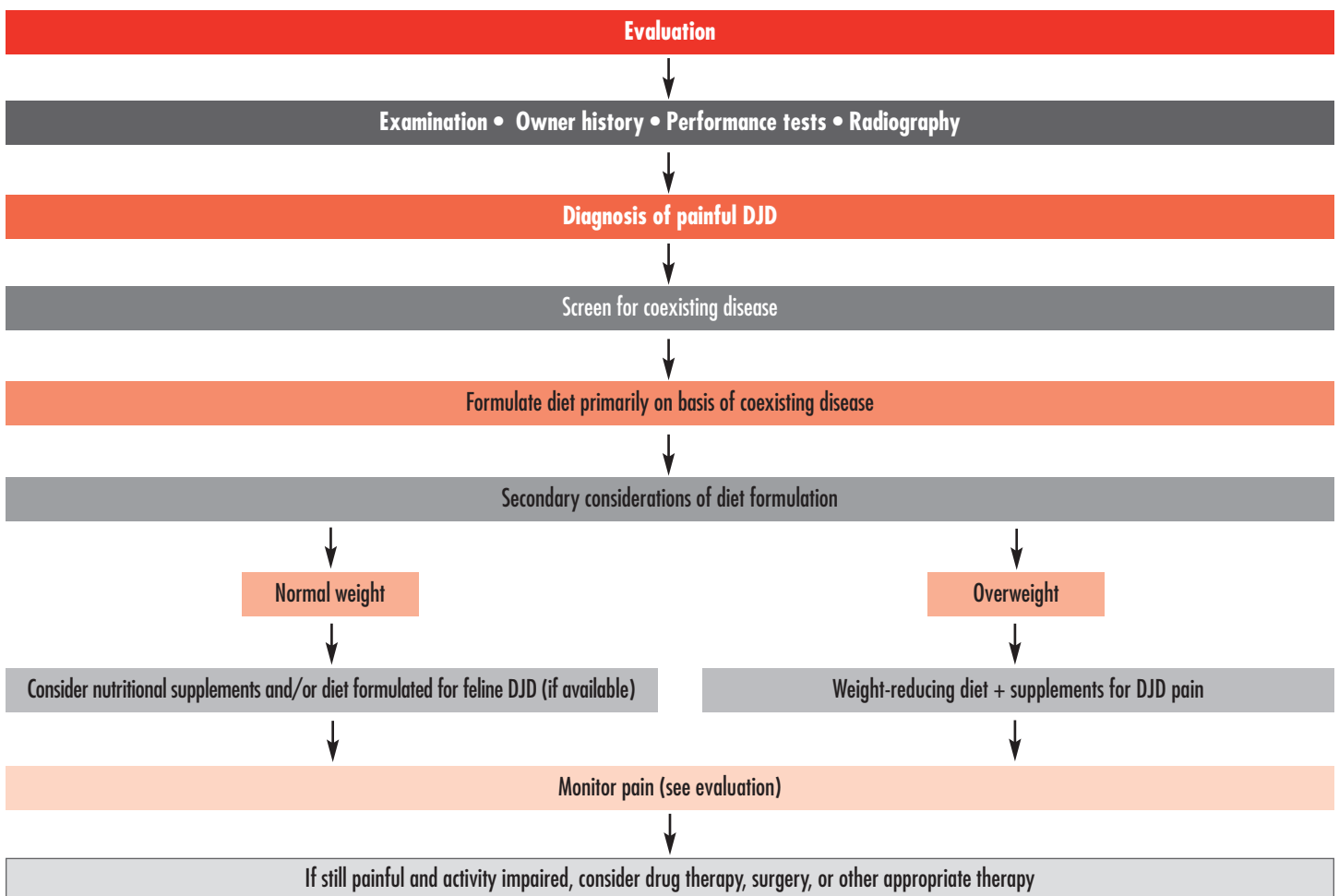
Common Comorbidities

No associations have been demonstrated for common comorbidities with feline DJD. However, given that it appears feline DJD increases in incidence with age, as discussed recently,¹³ all the diseases seen more frequently in older cats (for example, renal compromise, cardiac disease, diabetes, inflammatory bowel disease) should be considered as possible comorbidities, and diets should be formulated to take into account coexisting diseases in the individual cat.

Monitoring

Monitoring of feline DJD and the pain associated with it should be as outlined under *Key Diagnostic Tools and Measures*.

Algorithm – Nutritional Management of Suspected Feline Arthritis / Degenerative Joint Disease



Cardiac Disease – Canine

Lisa M. Freeman, DVM, PhD, DACVN

Definition

Cardiac diseases are common in dogs, affecting approximately 11% of all dogs, and can be congenital or acquired, and may affect the heart valves, the myocardium, or conduction pathways. *Heart failure* occurs when the cardiac disease becomes severe enough that the heart cannot pump blood to sufficiently supply all tissues. Clinical signs, ranging in severity from mild to severe, typically accompany heart failure. In *congestive heart failure* (CHF), the impaired cardiac function results in elevated venous pressure and resultant fluid accumulation (e.g., pulmonary edema, pleural effusion, ascites). Heart failure is the final common pathway of most cardiac diseases. The most common cardiac disease in dogs (>75% of all cardiac disease) is *chronic valvular disease* (CVD), while the second most common is *dilated cardiomyopathy* (DCM). *Congenital heart diseases* also occur, particularly in certain breeds. All of these cardiac diseases can result in heart failure. There are a number of systems to classify the severity of cardiac disease. One is the International Small Animal Cardiac Health Council (ISACHC) classification of heart failure (Table 1).

Key Diagnostic Tools and Measures

Body weight (BW), body condition score (BCS; see Appendix I), muscle wasting, appetite/food intake (diet history; see Appendix II), clinical signs (e.g., coughing, difficulty breathing, ascites, weakness, syncope, vomiting, diarrhea), and laboratory values including BUN, creatinine, electrolytes, hematocrit, and taurine (plasma and whole blood if taurine deficiency is suspected) should be considered in the diagnosis of cardiac disease in dogs. Other tests, if indicated, could include thoracic radiographs, blood pressure, electrocardiography, Holter monitor, and echocardiography.

Pathophysiology

Alterations in animals with heart failure that impact nutritional management include the following.

Calories. Many animals with cardiac disease, particularly when CHF arises, have reduced food intake. This can be the result of increased production of inflammatory mediators (e.g., cytokines, oxidative stress), side effects of cardiac medications, or poor control of heart failure signs.

Protein/Amino Acids. Muscle loss (cachexia) occurs in animals with heart failure as a result of reduced appetite, increased energy requirements, and pro-inflammatory cytokines. Taurine deficiency may be present in certain breeds of dogs with DCM (e.g., cocker spaniel, golden retriever, Newfoundland, St. Bernard, Portuguese water dog puppies), and has also

been associated with lamb meal and rice, high-fiber, or very-low-protein diets. Arginine may be a factor, as endothelial function may be impaired in animals with CHF, contributing to impaired exercise tolerance.

Fat. Dogs with CHF have been shown to have a relative deficiency of the n-3 fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), compared with healthy controls. n-3 fatty acids reduce inflammatory mediators and have anti-arrhythmic effects.

Minerals. Sodium and water retention occur in heart failure due to activation of the renin-angiotensin-aldosterone system. Hypokalemia may occur in dogs receiving loop (e.g., furosemide) or thiazide (e.g., hydrochlorothiazide) diuretics. Hypokalemia can increase the risk for cardiac arrhythmia. Hyperkalemia can occur in dogs receiving angiotensin-converting enzyme (ACE) inhibitors or potassium-sparing diuretics (e.g., spironolactone). Dogs receiving high doses of diuretics are at risk for hypomagnesemia, which can increase the risk for cardiac arrhythmia.

Vitamins. Increased urinary losses of B vitamin may occur in dogs receiving diuretics.

Other Nutrient Issues. Carnitine deficiency has been reported in a family of Boxers with DCM. Carnitine supplementation may improve energy metabolism in animals with CHF. In addition, dogs with CHF have increased oxidative stress (i.e., an imbalance between oxidant production and antioxidant protection)

Signalment

Chronic valvular disease (CVD) is the most common cardiac disease in dogs and typically affects small- and medium-sized breeds. Some breeds at increased risk for CVD include the Cavalier King Charles spaniel, Chihuahua, dachshund, miniature schnauzer, and toy and miniature poodles. Dilated cardiomyopathy (DCM) generally is found in large- and giant-breed dogs, such as the Doberman pinscher, Boxer, Irish wolfhound, and Great Dane. Some dogs with DCM may have taurine deficiency; predisposed breeds include the cocker spaniel, St. Bernard, golden retriever, Newfoundland, and Portuguese water dog puppies. Breeds with DCM that do not typically develop DCM (e.g., dachshund, corgi) also may have taurine deficiency.

Key Nutrient Modifications

Calories. Ensuring adequate calorie intake to maintain optimal BW is critical. Obesity can be present, particularly in dogs with early (asymptomatic) cardiac disease. As CHF develops, weight (and muscle) loss becomes common so ensuring adequate calorie intake often is critical at this stage.

Table 1. International Small Animal Cardiac Health Council (ISACHC) Classification of Heart Failure*

1. Asymptomatic

Heart disease is detectable but patient is not overtly affected and does not demonstrate clinical signs of heart failure. Diagnostic findings could include a cardiac murmur, arrhythmia, or cardiac chamber enlargement that is detectable by radiography or echocardiography

1a Signs of heart disease are present but no signs of compensation, such as volume or pressure overload ventricular hypertrophy, are evident

1b Signs of heart disease are present in conjunction with radiographic or echocardiographic evidence of compensation, such as volume or pressure overload ventricular hypertrophy

2. Mild to Moderate Heart Failure

Clinical signs of heart failure are evident at rest or with mild exercise and adversely affect quality of life. Typical signs of heart failure include exercise intolerance, cough, tachypnea, mild respiratory distress (dyspnea), and mild to moderate ascites. Hypoperfusion at rest is generally not present.

3. Advanced Heart Failure

Clinical signs of advanced congestive heart failure are immediately obvious. These clinical signs include respiratory distress (dyspnea), marked ascites, profound exercise intolerance, or hypoperfusion at rest. In the most severe cases, the patient is moribund and suffers from cardiogenic shock. Death or severe debilitation is likely without therapy.

3a Home care is possible

3b Hospitalization is mandatory because cardiogenic shock, life-threatening pulmonary edema, refractory ascites, or a large pleural effusion is present.

*From International Small Animal Cardiac Health Council. Recommendations for the Diagnosis of Heart Disease and the Treatment of Heart Failure in Small Animals, 1994.

Protein/Amino Acids. Normal to increased protein intake should be the goal to help counteract muscle loss. Protein restriction should be avoided unless severe concurrent renal disease is present. In dogs with DCM that are of breeds predisposed to taurine deficiency or breeds that do not typically develop DCM, taurine concentrations should be determined and taurine supplementation should be initiated while waiting for results. Taurine also may have some benefits due to its antioxidant and positive inotropic effects. Arginine supplementation has been shown in other species to improve the endothelial dysfunction associated with CHF. This has not yet been tested in dogs.

Fat. The anti-inflammatory and antiarrhythmic effects of n-3 fatty acids have many potential benefits in animals with cardiac disease. n-3 fatty acid supplementation reduces muscle loss in dogs with CHF and improves appetite in some animals. In addition, n-3 fatty acids have been shown to reduce ventricular arrhythmias in Boxers with arrhythmogenic right ventricular cardiomyopathy. n-3 fatty acids can be provided in diets that are highly enriched with this form of fat or as a dietary supplement.

Minerals. Severe sodium restriction is not recommended in early (asymptomatic) cardiac disease as sodium restriction activates the renin-angiotensin-aldosterone system. In early cardiac disease, the goal should be to avoid excessive sodium intake and to educate the owner about treats and table foods high in sodium. As the cardiac disease progresses and CHF develops, greater sodium restriction is indicated; this can help to reduce the diuretic doses required to control clinical signs. Controlling sodium intake from the dog food is important but it is also critical to ensure that other sources of sodium intake are addressed, such as treats, table foods, and foods used to administer medications.

Recommended dietary potassium modifications will depend upon medications being administered and serum potassium concentrations. Canine diets have a wide range of potassium content so using one appropriate for the individual patient is important (e.g., avoiding high-potassium diets in dogs with hyperkalemia). Consequently, serum potassium should be monitored, especially as more medications are administered to a patient.

Serum magnesium should be monitored, especially in dogs receiving high doses of diuretics. Magnesium should be supplemented in dogs with hypomagnesemia.

Vitamins. Most cardiac diets contain increased levels of B vitamins. If high doses of diuretics are being administered, B-vitamin supplementation may be indicated.

Other nutrients. L-carnitine supplements can be offered to owners who wish to be able to provide dietary supplements to their dogs with DCM *in addition* to the dog's cardiac medications (be careful to avoid a situation in which an owner gives supplements in place of cardiac medications). The prevalence of primary carnitine deficiency as a cause for DCM is likely low although Boxers may be a breed that is predisposed. Carnitine, however, also may improve myocardial energetics in dogs with CHF.

Coenzyme Q10 is sometimes recommended for dogs with DCM as an antioxidant and to aid in myocardial energy metabolism. No controlled studies have been performed in dogs with spontaneous cardiac disease and results from other species are conflicting on the potential benefits of supplementing coenzyme Q10.

Antioxidant supplementation was shown to reduce oxidative stress in one study of dogs with CVD; however, the effects on disease progression and clinical outcome are not known.

Recommended Ranges of Key Nutrients

Nutrient	mg/100 kcal	mg/100 kcal
	Recommended dietary level	Minimum dietary requirement*
Protein (g)	5.5–8	5.1
Sodium (mg)	35–100 (depends on stage of disease)	17

Modified intake of certain nutrients may help address alterations induced by cardiac disease or medications used to manage the disease. The recommended dietary composition is shown as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake and energy intake, except for those otherwise noted in the text. Correction of negative energy balance, if present, is critical.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

Avoid making big dietary changes when a dog is hospitalized for an acute episode of CHF. Continue feeding the dog's usual diet (unless very high in sodium), but have the owner discontinue any high-sodium treats or table foods. When the dog returns in 7 to 10 days for re-evaluation, a gradual change to a more appropriate diet can be instituted. This helps to avoid food aversions that can develop when a new diet is imposed on an acutely sick dog.

Calories should be adjusted to maintain optimal body condition (e.g., reducing calorie intake in obese animals; increasing calorie intake in animals that are below optimal body weight/condition).

Protein/Amino Acids. The diet should contain ≥ 5.1 g protein/100 kcal. Note that many canine cardiac diets are low in protein. Diets < 5.1 g protein/100 kcal should be avoided unless concurrent renal disease is present. In dogs with DCM that are of breeds predisposed to taurine deficiency or breeds that do not typically develop DCM, taurine concentrations should be determined and taurine supplementation should be initiated while waiting for results (250–1000 mg every 8–12 hours).

Fat. The optimal dose of n-3 fatty acids has not been determined; however, the author currently recommends a dosage of fish oil to provide 40 mg/kg EPA and 25 mg/kg DHA for animals with anorexia or cachexia. Unless the diet is one of a few specially designed therapeutic diets, supplementation will be necessary to achieve this n-3 fatty acid dose. Fish oil supplements vary in their concentration of EPA and DHA so the author recommends a 1-gram capsule that contains 180 mg EPA and 120 mg DHA. At this concentration, fish oil can be administered at a dose of 1 capsule per 10 pounds of body weight. Alternatively, a liquid form of n-3 fatty acids (e.g., Cardiguard, Boehringer Ingelheim, which contains 420 mg EPA and 280 mg DHA per gram) can be used. It should be noted that if the owner cannot administer the capsule, the dog will be exposed to the very strong flavor of the fish oil. While some dogs appear to enjoy the taste, others do not. In dogs that dislike the flavor, administration of n-3 fatty acids may not be possible due to adverse effects on food intake. Fish oil supplements should contain vitamin E as an antioxidant, but other nutrients should not be included to avoid toxicities. Cod liver oil and flax oil should not be used to provide n-3 fatty acids.

Minerals. With regard to sodium:

- ISACHC Stage 1: Counsel the owner to avoid diets high in sodium (> 100 mg/100 kcal) and to avoid high-sodium treats and table food.
- ISACHC Stage 2: The goal should be for < 80 mg/100 kcal in the dog food. Sodium intake from other foods (e.g., treats, table food, foods used for medication administration) also will be important.
- ISACHC Stage 3: The dog food should be < 50 mg/100 kcal although anorexia may require more leniency in sodium content in the diet (< 80 mg/100 kcal) in order to provide greater choice. Controlling sodium

intake from other foods (e.g., treats, table food, foods used for medication administration) is important as these can be major sources of sodium.

Recommended dietary potassium modifications will depend upon medications being administered and serum potassium concentrations. Diets high in magnesium or an oral magnesium supplement should be used in dogs with hypomagnesemia.

Vitamins. If high doses of diuretics are being administered, B vitamin supplementation may be indicated.

Other Nutrients. The minimum or optimal dose of L-carnitine necessary to replete a dog with low myocardial carnitine concentrations is not known, but the dose that has been recommended is 50–100 mg/kg orally (PO) every 8 hours. For coenzyme Q10, the current recommended (but empirical) dose in dogs is 30–90 mg PO twice daily, depending upon the size of the dog.

■ **Treats** – Most dogs with cardiac disease (>90% in one study) receive treats and table foods. Therefore, this issue is important to address with owners. Making specific recommendations to owners regarding treats that are appropriate (and those that should be avoided) is important as most commercial dog treats are high in sodium and most people are unaware of the sodium content of “people food.” Foods to be avoided include most commercial dog treats (unless specifically determined to be low in sodium), baby food, pickled foods, bread, pizza, lunch meats and cold cuts, condiments (e.g., ketchup, salsa), most cheeses, processed foods (e.g., rice mixes, macaroni and cheese, frozen meals), canned vegetables (unless the label states “sodium free” or “very low sodium”), rawhide chews, canned soups, and snack foods (e.g., potato chips, crackers). Acceptable treats include fresh fruits (e.g., apples, oranges, bananas; avoid grapes), fresh vegetables (e.g., carrots, green beans; avoid onions or garlic), dog treats that are determined to be low in sodium (<20 mg sodium/treat for medium-large sized dogs; <10 mg/treat for small dogs). Note that even low-sodium treats and foods can provide large doses of sodium if they are fed in large quantities, particularly for small dogs.

It also is important to provide the owner with appropriate methods for administering medications as many dog owners use foods to administer medications and many common foods used are high in sodium. The owner can be taught to administer the pill without using foods (either by hand or using a device designed for this purpose). Alternatively, foods such as fresh fruits (e.g., bananas, melon), low-sodium canned pet food, peanut butter (labeled as “no salt added”), or home-cooked meat (without salt; not lunch meats) can be used. Finally, a compounded liquid medication can be considered although the pharmacokinetics of compounded medications may be significantly altered.

■ **Tips for Increasing Palatability** – Dogs with CHF often have cyclical appetites (i.e., they will eat a food well for 7 to 14 days but then stop eating it). While reductions in appetite in a dog that was previously eating well can indicate the need for reassessment and medication adjustment, sometimes providing a different food will increase appetite again. Communicating with the owner about these issues can help to reduce anxiety.

Palatability enhancers such as home-made, low-sodium broth (e.g., chicken, beef) can enhance palatability. Most store-bought broths are high in sodium, even if labeled “low sodium.” Cooked chicken, beef, or fish can be added to the food. Dogs with CHF often prefer sweet flavors. Therefore, adding vanilla or fruit yogurt, maple syrup, or applesauce to the food often improves palatability and food intake. n-3 fatty acids will often improve appetite in dogs with CHF; however, it will take 2 to 4 weeks to see effects. Appetite stimulants can be considered (e.g., cyproheptadine, mirtazepine). Dogs with CHF often have preferences for food temperature (i.e., some will only eat foods at room temperature, some prefer cold foods, some prefer warmed foods). Encourage the owner to experiment to determine which temperature works best for their dog. Sometimes feeding the dog from a dinner plate (rather than the dog food bowl) and in a place different from their usual site can improve appetite.

Client Education Points

- Make specific diet and treat recommendations (types and amounts).
- Warn owner about common alterations in appetite in dogs with heart failure.
- Give the owner appropriate methods for medication administration.
- 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.
- In addition to safety and efficacy issues, there are significant concerns about the quality control of dietary supplements (e.g., quality control, bioavailability). Therefore, veterinarians should consider recommending specific brands of dietary supplements that bear the logo of the United States Pharmacopeia Dietary Supplement Verification Program (DSVP), which tests human dietary supplements for ingredients, concentrations, dissolvability, and contaminants. Another good resource is Consumerlab.com, which performs independent testing of dietary supplements (primarily human supplements but also some pet products).

Common Comorbidities

In one study, 61% of dogs with cardiac disease had at least one concurrent disease. Therefore, the nutritional goals may need to be modified for a dog with heart failure that has a concurrent, nutrient-sensitive disease (e.g., a dog with CHF and chronic renal failure or gastrointestinal disease).

Interacting Medical Management Strategies

Drug–nutrient interactions are common in CHF. Loop (e.g., furosemide) or thiazide (e.g., hydrochlorothiazide) diuretics can increase the risk for hypokalemia and hypomagnesemia, while ACE inhibitors and potassium-sparing diuretics (e.g., spironolactone) can increase the risk for hyperkalemia. Azotemia can result from overzealous use of diuretics. Anorexia can be a side effect of many cardiac medications (e.g., diuretics, digoxin, ACE inhibitors).

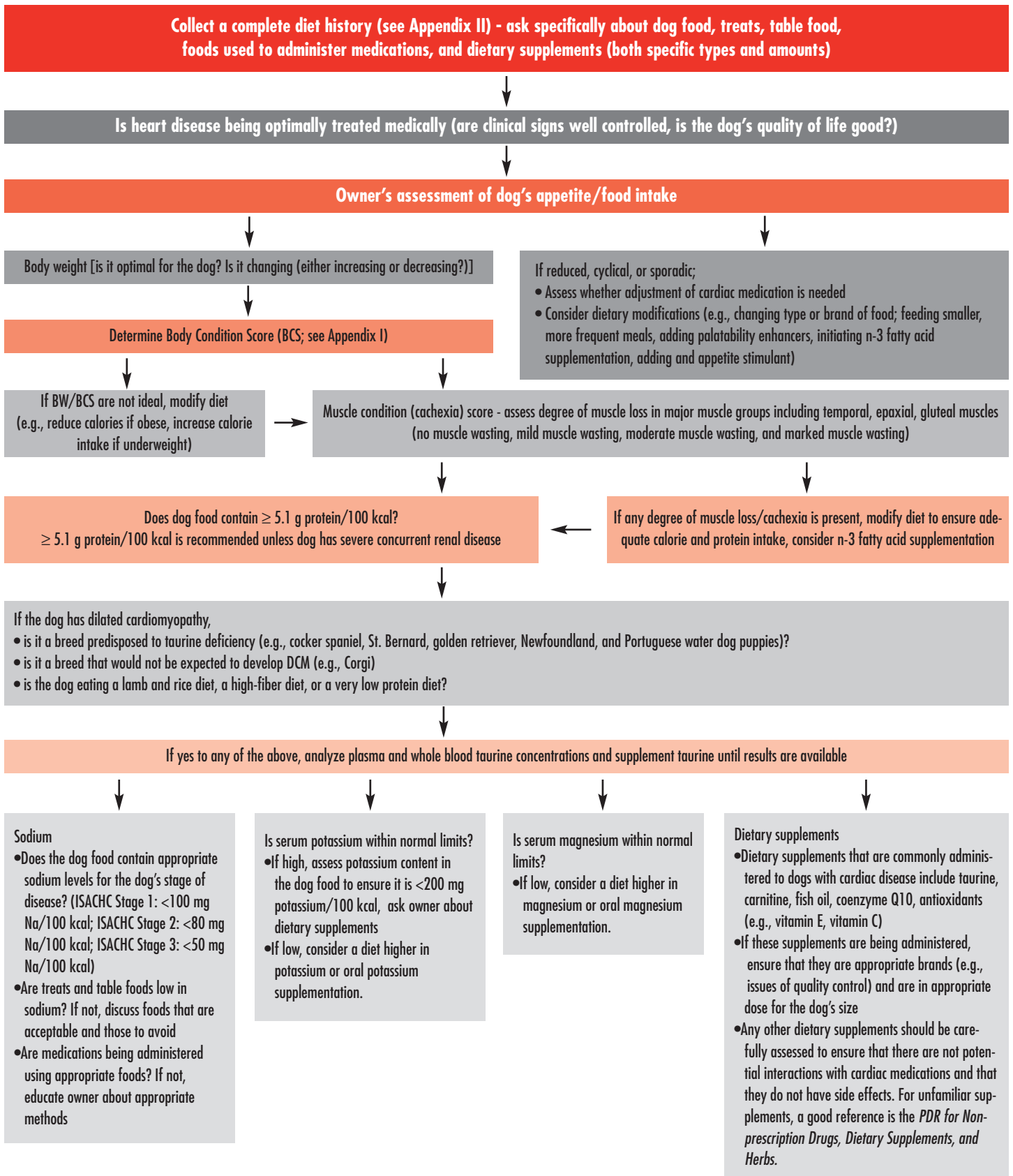
Monitoring

Reductions in appetite/food intake may indicate the need for dietary modifications but also may be an early sign of decompensation of the cardiac disease or the need for medication adjustment. Body weight should be monitored in obese dogs in order to achieve optimal weight. In animals with cachexia that are losing weight/muscle, dietary modifications are needed to minimize weight/muscle loss. Note that in animals with right-sided CHF, fluid accumulation (pleural or peritoneal effusion) can mask weight loss but weight and muscle loss is very common in these dogs so “dry weight” and muscle loss should be carefully monitored.

Body condition score (BCS) is helpful for monitoring animals with asymptomatic disease and those that are overweight or obese. Note that BCS systems assess fat stores but not muscle, so an animal can be overweight or obese but still have muscle wasting. Therefore, monitoring BW, BCS, and the degree of muscle wasting is important. Muscle loss is typically first noted in the temporal, epaxial, and gluteal muscles. A muscle condition score is being developed that subjectively categorizes muscle mass into four categories: No muscle wasting, mild muscle wasting, moderate muscle wasting, and marked muscle wasting. Intervening at an early stage (mild or moderate muscle wasting) provides improved opportunities for reversing or minimizing the degree of muscle wasting.

Clinical signs (e.g., coughing, difficulty breathing, weakness, syncope, vomiting, diarrhea), laboratory values (BUN, creatinine, electrolytes, hematocrit), and other measures, if indicated (e.g., thoracic radiographs, blood pressure, electrocardiography, Holter monitoring, echocardiography) should also be monitored.

Algorithm – Evaluation of Nutritional Issues in Canine Cardiac Disease



Cardiac Disease – Feline

Lisa M. Freeman, DVM, PhD, DACVN

Definition

Hypertrophic cardiomyopathy (HCM) is the most common cardiac disease in cats and occurs at a high frequency, especially in some regions of the country. Other cardiac diseases (e.g., *congenital cardiac diseases*) occur at much lower incidence. *Dilated cardiomyopathy* (DCM) is now an uncommon disease in cats unless the cat is eating a vegetarian or nutritionally unbalanced diet. HCM can result in *congestive heart failure* (CHF), *arterial thromboembolism* (ATE), *syncope*, or *sudden death*. There are a number of systems to classify the severity of cardiac disease. One is the International Small Animal Cardiac Health Council (ISACHC) classification of heart failure (see Table 1 on page 14).

Key Diagnostic Tools and Measures

Body weight (BW), body condition score (BCS; see Appendix I), muscle wasting, appetite/food intake (diet history; see Appendix II), clinical signs (e.g., difficulty breathing, weakness, syncope, vomiting, diarrhea), and laboratory values (e.g., BUN, creatinine, electrolytes, hematocrit) should be considered in the diagnosis of cardiac disease in cats. Other tests, if indicated, could include thoracic radiographs, blood pressure, electrocardiography, and echocardiography.

Pathophysiology

Alterations in cats with cardiac disease that impact nutritional management include the following.

Calories. When cats with cardiac disease are asymptomatic, appetite usually is unaltered. When CHF arises, however, most cats will have alterations in appetite and food intake. This may result in changes in food preferences (e.g., type of food, flavors) or in a reduction in the amount eaten. These alterations can be the result of increased production of inflammatory mediators (e.g., cytokines, oxidative stress), side effects of cardiac medications, or poor control of heart failure signs.

Protein/Amino Acids. Muscle loss (cachexia) can occur in cats with heart failure as a result of reduced appetite, increased energy requirements, and increased production of inflammatory cytokines. Taurine deficiency causes DCM in cats. While DCM is now an uncommon disease in cats, taurine deficiency should not be ruled out as a cause in cases of DCM, especially if the cat is eating a vegetarian or home-made diet.

Fat. The n-3 fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), reduce inflammatory mediators and have anti-arrhythmic and anti-thrombotic effects, all of which may be beneficial in cats with heart failure.

Minerals. Sodium and water retention occur in heart failure due to activation of the renin-angiotensin-aldosterone system. Hypokalemia may occur in cats receiving loop (e.g., furosemide) diuretics. Hyperkalemia can occur in cats receiving angiotensin converting enzyme inhibitors. High doses of diuretics increase the risk for hypomagnesemia.

Vitamins. Increased urinary losses of B vitamins may occur in cats receiving diuretics. Studies have shown, however, that vitamins B6, B12, and folate were lower in cats with HCM compared with controls whether or not CHF was present.

Signalment

While certain breeds are at increased risk for HCM and genetic mutations have been identified (i.e., Maine Coon cats, Ragdolls), most cats with this disease are domestic short- or long-haired cats.

Key Nutrient Modifications

Calories. Ensuring an appropriate calorie intake to maintain optimal body weight is critical. Obesity can be present, particularly in cats with early (asymptomatic) cardiac disease. As CHF develops, weight (and muscle) loss becomes common so ensuring adequate calorie intake is critical at this stage.

Protein/Amino Acids. Normal to increased protein intake should be the goal to help counteract muscle loss. Protein restriction should be avoided unless severe concurrent renal disease is present. Although DCM is now uncommonly seen in cats, taurine deficiency and DCM can occur in cats fed vegetarian or nutritionally unbalanced home-made diets. If taurine deficiency is present, taurine supplementation can reverse the disease in many cases.

Fat. The anti-inflammatory and antiarrhythmic effects of n-3 fatty acids have many potential benefits in cats with cardiac disease. n-3 fatty acid supplementation can reduce muscle loss and improve appetite via its anti-inflammatory effects. n-3 fatty acids can be provided in diets that are highly enriched with this form of fat or as a dietary supplement.

Minerals. Severe sodium restriction is not recommended in early (asymptomatic) cardiac disease as sodium restriction activates the renin-angiotensin-aldosterone system. In early cardiac disease, the goal should be to avoid excessive sodium intake and to educate the owner about treats and table foods high in sodium. As the cardiac disease progresses and CHF develops, greater sodium restriction is indicated and this can help to reduce the diuretic doses required to control clinical signs. Controlling sodium intake from the cat food is important but it is also critical to ensure that other sources of sodium intake are addressed, such as treats, table foods, and foods used to administer medications.

Recommended dietary potassium modifications will depend upon medications being administered and serum potassium concentrations. Feline diets have a wide range of potassium content so using one appropriate for the individual patient is important (e.g., avoiding high-potassium diets would be recommended for cats with hyperkalemia). Consequently, serum potassium should be monitored, especially as more medications are administered to a patient.

Serum magnesium should be monitored, especially in cats receiving high doses of diuretics. Magnesium should be supplemented in cats with hypomagnesemia.

Vitamins. If high doses of diuretics are being administered, B vitamin supplementation may be indicated.

Recommended Ranges of Key Nutrients

Nutrient	mg/100 kcal	mg/100 kcal
	Recommended dietary level	Minimum dietary requirement*
Protein (g)	7–9.5	6.5
Taurine (g)	0.025–0.050	0.025–0.050
Sodium (mg)	35–100 (depends on stage of disease)	50

Modified intake of certain nutrients may help address alterations induced by cardiac disease or medications used to manage the disease. The recommended dietary composition is shown as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake except for those otherwise noted in the text. Correction of negative energy balance, if present, is critical.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

Avoid making big dietary changes when a cat is hospitalized for an acute episode of CHF. Continue feeding the cat's usual diet (unless very high in sodium), but have the owner discontinue any high-sodium treats or table foods. When the cat returns in 7 to 10 days for re-evaluation, a gradual change to a more appropriate diet can be instituted. This helps to avoid food aversions that can develop when a new diet is imposed on a cat that is acutely sick.

Calories should be adjusted to maintain optimal body condition (e.g., reducing calorie intake in obese animals; increasing calorie intake in animals that are below optimal body condition).

Protein/Amino Acids. The diet should contain at least 6.5 g protein/100 kcal, unless severe concurrent renal disease is present. In cats with DCM and taurine deficiency (or while waiting for taurine results), taurine should be supplemented (125–250 mg q 12 hours).

Fat. The optimal dose of n-3 fatty acids has not been determined; however, the author currently recommends a dosage of fish oil to provide 40 mg/kg EPA and 25 mg/kg DHA for cats with anorexia or cachexia. Most cat foods do not achieve this dose so supplementation will be necessary. Fish oil supplements vary in their concentration of EPA and DHA so the author recommends a 1-gram capsule that contains 180 mg EPA and 120 mg DHA. At this concentration, fish oil can be administered at a dose of 1 capsule per 10 pounds of body weight. The capsule can be administered whole (although they are very large) or the oil can be expressed from the capsule and given as a treat or in the food. Alternatively, a liquid form of n-3 fatty acids (e.g., Cardiguard, Boehringer Ingelheim, which contains 420 mg EPA and 280 mg DHA per gram) can be used. It should be noted that if the owner cannot administer the capsule intact, the cat will be exposed to the very strong flavor of the fish oil. While some cats appear to enjoy the taste, others do not. In cats that dislike the flavor, administration of n-3 fatty acids will likely not be possible due to adverse effects on food intake. Fish oil supplements should contain vitamin E as an antioxidant, but other nutrients should not be included to avoid toxicities. Cod liver oil and flax oil should not be used to provide n-3 fatty acids (cod liver oil is high in vitamins A and D, which can cause toxicity, while cats are unable to convert the n-3 fatty acids in flax oil to EPA and DHA).

Minerals. With regard to sodium:

- ISACHC Stage 1: Counsel the owner to avoid diets high in sodium (>100 mg/100 kcal) and to avoid high sodium treats and table food.
- ISACHC Stage 2: The goal should be for <80 mg/100 kcal in the cat food. Sodium intake from other foods (e.g., treats, table food, foods used for medication administration) also will be important.
- ISACHC Stage 3: The cat food should be <50 mg/100 kcal although anorexia may require a slightly higher sodium content in the diet (<80 mg/100 kcal) in order to provide greater choice. Controlling sodium intake from other foods (e.g., treats, table food, foods used for medication administration) remains important.

Recommended dietary potassium modifications will depend upon medications being administered and serum potassium concentrations. Diets high in magnesium or an oral magnesium supplement should be used in cats with hypomagnesemia.

Vitamins. If high doses of diuretics are being administered, B vitamin supplementation may be indicated.

■ **Treats** – Fewer cats with cardiac disease receive treats on a regular basis compared with dogs (33% of cats vs. 92% of dogs); however, it is important to make specific recommendations to owners regarding treats that are

appropriate (and those that should not be fed) if owners wish to provide treats. Foods to be avoided include most commercial cat treats (unless specifically determined to be low in sodium), baby food, lunch meats and cold cuts, canned fish, and most cheeses. Acceptable treats include cat treats that are determined to be low in sodium (<5 mg/treat). Note that even low-sodium treats and foods can provide large doses of sodium if they are fed in large quantities to cats.

It also is important to provide the owner with appropriate methods for administering medications as cats are difficult to pill and many common foods used to administer medications are high in sodium. Fewer cats than dogs with cardiac disease receive medications in foods (34% of cats vs. 57% of dogs) so owners should be taught to administer the pill without using foods (either by hand or using a device designed for this purpose). Alternatively, foods such as low-sodium canned cat food or home-cooked meat (cooked without salt; not lunch meats) can be used. A compounded liquid medication can be considered although the pharmacokinetics of compounded medications may be significantly altered.

■ **Tips for Increasing Palatability** – Cats with CHF often have variable appetites (i.e., they may eat a food well for a week but then stop eating it). While reductions in appetite in a cat that was previously eating well can indicate the need for reassessment and medication adjustment, sometimes providing a different food will increase appetite again. Communicating with the owner about these issues can help to reduce anxiety and to provide the owner with specific strategies to address the problem (and when to bring the cat in for reassessment).

Home-made, low-sodium broth (e.g., chicken, beef, fish) can enhance palatability. Most store-bought broths are high in sodium, even if they say "low sodium." Cooked chicken, beef, or fish can be added to the food. n-3 fatty acids will often improve appetite in cats with CHF; however, it will take 2 to 4 weeks to see an effect. Appetite stimulants can be considered (e.g., cyproheptadine, mirtazepine). Cats with CHF often prefer warmed foods but encourage the owner to experiment to determine which food temperature works best for their cat. Sometimes feeding the cat from a dinner plate (rather than the pet bowl) and in a place different from their usual site can improve appetite.

Client Education Points

- Make specific diet and treat recommendations (both types and amounts).
- Warn the owner about common alterations in appetite in cats with heart failure.
- Give the owner appropriate methods for medication administration.
- 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) 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 (e.g., quality control, bioavailability). Therefore, veterinarians should consider recommending specific brands of dietary supplements that bear the logo of the United States Pharmacopeia Dietary Supplement Verification Program (DSVP), which tests human dietary supplements for ingredients, concentrations, dissolvability, and contaminants. Another good resource is Consumerlab.com, which performs independent testing of dietary supplements (primarily human supplements but also some pet products).

Common Comorbidities

In one study, 56% of cats with cardiac disease had at least one concurrent disease. Therefore, the nutritional goals may need to be modified for a cat with heart disease that has a concurrent, nutrient-sensitive disease (e.g., a cat with CHF and urolithiasis or chronic renal failure).

Interacting Medical Management Strategies

Drug–nutrient interactions are common in CHF. Loop (e.g., furosemide) diuretics can increase the risk for hypokalemia and hypomagnesemia, while ACE inhibitors can increase the risk for hyperkalemia. Azotemia can result from overzealous use of diuretics. Anorexia can be a side effect of many cardiac medications (e.g., diuretics, digoxin, ACE inhibitors).

Monitoring

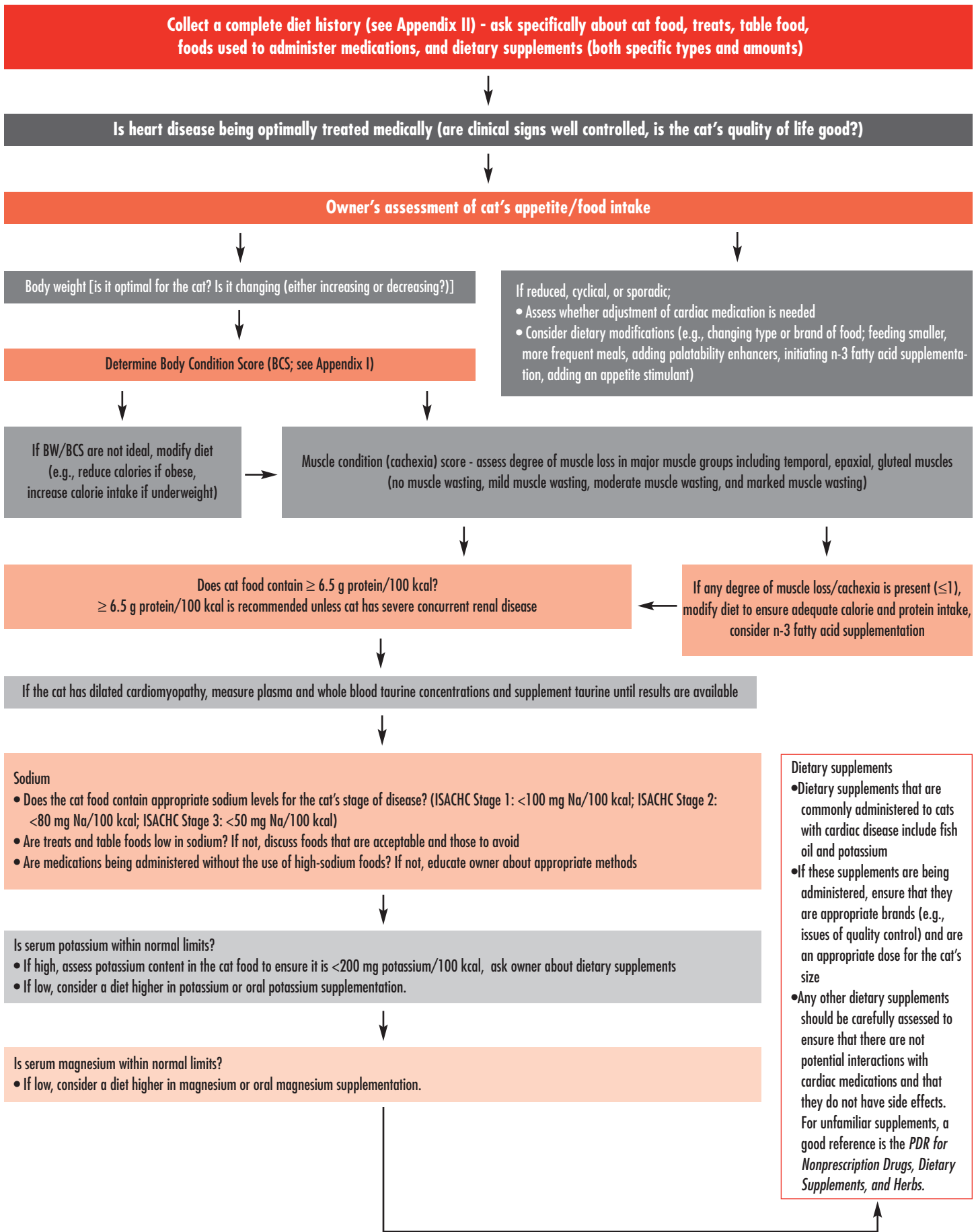
Reductions in food intake may indicate the need for dietary modifications but also may be an early sign of decompensation of the cardiac disease or the need for medication adjustment. Body weight should be monitored in

obese cats in order to achieve optimal weight. In cats with cachexia that are losing weight, dietary modifications are needed to minimize weight loss.

Body condition score (BCS) is helpful for monitoring cats with asymptomatic disease and those that are overweight or obese. Note that BCS systems assess fat stores but not muscle, so a cat can be overweight or obese but still have muscle wasting. Therefore, monitoring BW, BCS, *and* the degree of muscle wasting is important. Muscle loss is typically first noted in the temporal, epaxial, and gluteal muscles. A muscle condition score is being developed that subjectively categorizes muscle mass into four categories: No muscle wasting, mild muscle wasting, moderate muscle wasting, and marked muscle wasting. Intervening at an early stage (i.e., mild or moderate muscle wasting) provides improved opportunity for reversing or minimizing the degree of muscle wasting.

Clinical signs (e.g., difficulty breathing, weakness, syncope, vomiting, diarrhea), laboratory values (BUN, creatinine, electrolytes, hematocrit), and other measures, if indicated (e.g., thoracic radiographs, blood pressure, electrocardiography, echocardiography) should also be monitored.

Algorithm – Evaluation of Nutritional Issues in Feline Cardiac Disease



Chylothorax – Feline

Kathryn E. Michel, DVM, MS, DACVN

Definition

Chylothorax is the accumulation of a chylomicron-containing effusion in the pleural space.

Key Diagnostic Tools and Measures

The presence of pleural effusion will be noted on thoracic radiography. Patients with sufficient effusion to impede lung expansion will present with clinical signs of tachypnea, dyspnea, exercise intolerance, and muffled heart sounds on auscultation. Diagnosis is confirmed by performing a thoracocentesis and an analysis and cytology on the resulting fluid. Further imaging, blood work, and diagnostic testing may be indicated to determine an underlying cause.

A complete diet history should be taken that includes information about typical food intake and any commercial pet foods and treats that the patient receives including table foods or scraps (see Appendix II).

The patient's nutritional status should be assessed with special attention paid to the presence and duration of anorexia, evidence of weight loss (in particular, muscle wasting), feasibility of assisted feeding, and concurrent medical conditions.

Pathophysiology

Normally the chyle, which originates from intestinal lymphatic drainage, flows into the thoracic duct via the cisterna chyli and, in turn, empties into the venous system in the cranial thorax. Any condition causing an imbalance between chyle production and clearance that leads to increased pressure within the lymphatics can result in leakage of chyle into the pleural space. Underlying causes reported in cats include heart disease and the presence of a cranial mediastinal mass; however, idiopathic disease is not uncommon.

Patients with chylothorax are often lethargic and have a poor appetite. The impact of poor food intake on nutritional status is exacerbated by the loss of protein, fat, vitamins, electrolytes, and water if repeated palliative thoracocentesis becomes necessary to relieve respiratory distress.

Signalment

Older cats and oriental breeds (Siamese and Himalayan) are reported to be at increased risk of developing chylothorax.

Key Nutrient Modifications

Fat-restricted diets have been advocated as part of the medical management of chylothorax, the rationale being that a reduction in dietary fat absorption could translate into a reduction in chyle production. In addition to fat restriction, dietary supplementation with medium chain triglycerides (MCT) has been recommended. The rationale for MCT use is that these triglycerides can be absorbed directly into the portal blood and therefore can serve to increase the energy density of the diet without increasing chyle production.

While there have been case reports and retrospective studies of patients that were fed fat-restricted diets as part of the treatment they received, there are no prospective clinical trials investigating the efficacy of dietary fat restriction in companion animals. An investigation of normal dogs found that while varying the quantity and type of fat in the diet could affect the triglyceride makeup of the chyle, it did not alter the rate of lymph flow.¹ Furthermore, studies have found that when the diet of normal dogs

is supplemented with MCT, significant amounts of MCT appear in the chyle.^{2,3}

In conclusion, there is neither strong evidence nor a strong rationale for the use of dietary fat restriction for the management of feline chylothorax. It may be possible that by reducing the amount of chylomicrons in the lymph through dietary fat restriction, the resulting effusion is more readily resorbed from the pleural space. However, cat foods that are fat-restricted are also low in caloric density and this must be taken into consideration when managing a patient that is likely to have a poor appetite and may have already experienced significant deterioration in nutritional status.

Recommended Ranges of Key Nutrients

Nutrient	% DM	mg/100 kcal	% DM	mg/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Total dietary fat	9–12	<3.5	9.0	2.25

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

* Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Note that nearly all fat-restricted cat foods have been purposely calorie-restricted for weight control and are not indicated for patients that are experiencing anorexia and weight loss.

Therapeutic Feeding Principles

Therapeutic options for patients with idiopathic chylothorax include both medical and surgical management. In some cases, the effusion may spontaneously resolve after several weeks or months; however, the prognosis for cats with this condition should be considered guarded.

As previously stated, there is neither strong evidence nor a strong rationale for the use of dietary fat restriction for the management of feline chylothorax and the use of such a feeding strategy may further compromise nutritional status in a patient with poor food intake, particularly if it is undergoing repeated palliative thoracocentesis.

Diet selection should be predicated on finding a complete and balanced cat food that is acceptable to the patient. Unless contraindicated due to a concurrent condition, a high-protein diet may be beneficial, particularly for patients that are undergoing repeated thoracocentesis.

If a patient's weight is stable and the patient is in good body condition, a fat-restricted diet (<30% fat, energy basis) could be introduced on a trial basis. Care should be taken to give specific feeding directions and to monitor food intake and body weight to ensure that the patient is able to consume enough of the food to meet energy needs.

Patients who refuse food or have inadequate voluntary intake should be evaluated as candidates for assisted feeding. Ideally these patients should receive a complete and balanced diet by the enteral route. Parenteral nutritional support has been used to treat human patients with chylothorax. The etiology of this condition in humans, however, generally differs from that in cats so there is no clear rationale to feed by the parenteral route when enteral nutrition is feasible.

■ **Treats** – When selecting treats, it is relatively easy to avoid very high-fat

items (e.g., fat trimmings from meat, fried foods, cream) and it may be prudent to do so. Acceptable treats would include lean meats or fish (e.g., baked chicken breast, tuna packed in water), low-fat dairy products, and fresh fruits and vegetables (with the exception of grapes and onions).

■ **Tips for Increasing Palatability** – Unless there are clear indications for feeding a fat-restricted food (<30% fat, energy basis), diet selection should be predicated on finding a complete and balanced cat food that is acceptable to the patient. Adding some warm water to a dry food or slightly warming a canned food may enhance acceptance.

■ **Diet Recommendations** – Many cats diagnosed with chylothorax will present with a history of anorexia. It is essential to monitor the patient's food intake and body weight to ensure that the patient's voluntary consumption is adequate. The goal will be to find a diet that the patient eats readily and is appropriate for any concurrent condition that the patient may have (e.g., heart disease). Low-fat, calorie-restricted diets should only be used in patients that will readily consume them in amounts sufficient to meet their energy needs

For patients in good body condition, feeding portions should be based on previous caloric intake. For underweight patients, calories offered should be increased by 20% above previous intake to promote weight gain and adjusted as necessary based upon response.

Client Education Points

- All members of the household should understand that while dietary management may not play a direct role in the management of chylothorax, patients with this condition are at risk of becoming malnourished. Therefore, it is of particular importance to monitor the patient's food intake and body condition to enable early detection of weight loss.

Common Comorbidities

Heart Disease. Feline chylothorax has been reported in association with cardiomyopathy (particularly secondary to hyperthyroidism), heartworm disease, congenital cardiac abnormalities, and pericardial effusion. Therefore a complete cardiac workup is indicated in cats diagnosed with a chylous pleural effusion as treatment of the underlying heart condition could lead to resolution of the chylothorax.

Neoplasia. Conditions causing an anterior mediastinal mass including lymphosarcoma and other forms of neoplasia can lead to chylothorax. If a mass is identified it should be biopsied or aspirated to obtain a diagnosis in order to permit appropriate therapeutic measures to be taken.

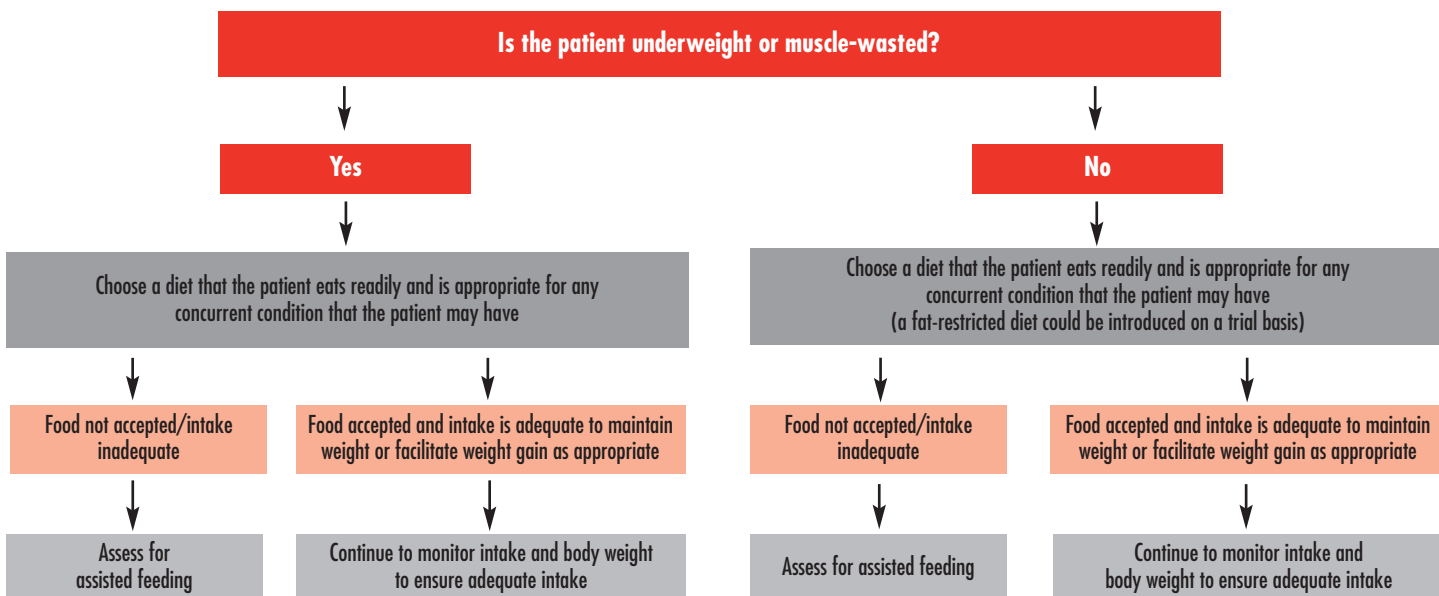
Interacting Medical Management Strategies

As previously mentioned, patients with chylothorax may require repeated palliative thoracocentesis leading to the loss of fluid, electrolytes, protein, fat, and vitamins. Patients with chylothorax secondary to an underlying condition such as heart disease may benefit from dietary therapy targeted at that condition.

Monitoring

Patients with idiopathic chylothorax will require monitoring on a regular basis to assess the rate of accumulation of fluid in the pleural cavity and whether there is a need for repeat thoracocentesis or other therapeutic intervention. Patients undergoing repeated thoracocentesis are at risk of developing dehydration, electrolyte imbalance (hyponatremia, hyperkalemia), hypoproteinemia, and malnutrition. Therefore, assessment of fluid and electrolyte balance as well as voluntary food intake, body weight, and body condition should be performed during patient rechecks.

Algorithm – Nutritional Management of Feline Chylothorax



Critical Care Nutrition – Canine

Daniel L. Chan, DVM, MRCVS, DACVECC, DACVN

Definition

Critical illnesses have a significant impact on nutritional status of dogs and often lead to overt malnutrition. Nutritional support of critically ill dogs is an important part of medical therapy and may play a role in improving outcome. Dogs with critical illnesses often have reduced nutritional intake, vomiting, diarrhea, and possibly altered nutritional requirements, all of which can impact their nutritional status.

Key Diagnostic Tools and Measures

Designing a nutritional plan entails performing a nutritional assessment to establish the specific needs and considerations for the patient. Body Condition Scoring (BCS) is an important part of nutritional assessment. Biochemistry profiles are also helpful in identifying important considerations for the nutritional plan. For the majority of critically ill dogs, nutritional support should be aimed at meeting resting energy requirements (RER) initially and adjusting the calories provided based on frequent re-assessment.

Pathophysiology

Critical illness imparts various changes in metabolism that impact the nutritional state of the patient. In a response to inflammation and injury, there are alterations in carbohydrate, protein, and lipid metabolism. The various changes in metabolism, combined with the effects of reduced dietary intake, result in a negative energy balance or a catabolic state. Dogs in a catabolic state may experience more complications and may have poorer rates of recovery. Reversal of the catabolic condition requires addressing the primary cause of disease and provision of adequate nutritional support.

Signalment

Patients that may be at greater risk for malnutrition include very young and geriatric patients. This may reflect the relative greater difficulty in providing nutritional support for patients in these age categories.

Key Nutrient Modifications

- Critically ill patients with a negative energy balance may have a greater need for protein to maintain lean body mass.
- Protein should be of good quality and highly digestible.
- Special considerations may include patients with comorbidities such as renal or hepatic failure, where increased protein may be contraindicated.
- More specific nutrient requirements will depend on the nature of the underlying disease.
- Antioxidants may be an important component of therapies intended for critically ill animals, however, specific dosages have not been determined.
- Other nutrients such as glutamine, arginine, omega-3 fatty acids may also be helpful in certain conditions, but specific or optimal dosages have not been established.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	20–45	6–9	18	5.1
Fat	25–35	5–7	5	1.4

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Correction of negative energy balance is critical.

Therapeutic Feeding Principles

An important part of the nutritional plan is establishing the most appropriate route of nutritional support. In the absence of a contraindication, it is preferable to feed the dog via the enteral route. Contraindications for enteral nutrition include protracted vomiting, regurgitation or diarrhea, inability to guard the airway, and intolerance for feedings (e.g., gastric atony). In patients that could tolerate enteral feedings but there is no adequate voluntary intake (approximately 75% of energy requirements), a feeding tube should be placed. For short-term assisted feeding (<3 days) a nasoesophageal feeding tube may be appropriate but this requires a liquid diet. Should nutritional support be required for longer than 3 days, use of an esophagostomy or gastrostomy feeding tube is usually indicated.

Once a feeding tube is in place, feedings should aim to provide 50% of calculated calories in the first day, and gradually increasing to 100% of calculated calories over next 2 days. In severely affected patients, initial nutritional support should start at 33% of calculated calories. Diets typically recommended for tube feeding are high in calories, protein, and fat. It is very important to ensure that the diet chosen is appropriate for the feeding tube being used (i.e., the consistency of the diet must not occlude the tube). In patients in which the enteral route cannot be used, parenteral nutrition is necessary. Formulation of parenteral nutrition is tailored to the patient and requires special formulation and handling.

■ **Treats** – For critically ill animals, the use of treats is usually ineffective at meeting energy and nutrient requirements and only delays more appropriate nutritional support. Animals receiving tube feedings may be offered treats to assess return of appetite. Provision of enteral and/or parenteral nutrition does not interfere with the assessment of appetite.

■ **Tips for Increasing Palatability** – The use of appetite stimulants in critically ill animals is not recommended as they are ineffective at restoring adequate nutritional intake. Techniques such as hand feeding or warming the food may be attempted but are also typically ineffective at achieving adequate nutritional support.

■ **Diet Recommendations** – Diets typically used in nutritional support of critically ill dogs are usually energy dense, high in protein and fat

content, and have high digestibility. Many prescription diets recommended for tube feeding are also very high in water content and are amenable for tube feeding with minimal modification. Most diets, however, will need to be modified in order to be used effectively with feeding tubes. With small-gauge tubes, typically used for nasoesophageal access, complete liquid diets are the only acceptable diets.

Client Education Points

- As many recovering patients can be discharged from the hospital with the feeding tube in place (e.g., esophagostomy, gastrostomy tubes), clients need to be instructed how to use and care for the feeding tubes.
- Clients need to be made aware of possible complications and how to detect them.
- Clients should be provided with detailed and specific instructions for how to use feeding tubes. This should include instructions of how to prepare the diet and how to administer the feedings.

Common Comorbidities

Patients with critical illness often have several affected organ systems which may impact the nutritional plan. More serious comorbidities include

concurrent congestive heart failure, renal failure, hepatic failure, respiratory failure, gastrointestinal dysfunction, neurologic dysfunction, and systemic infection.

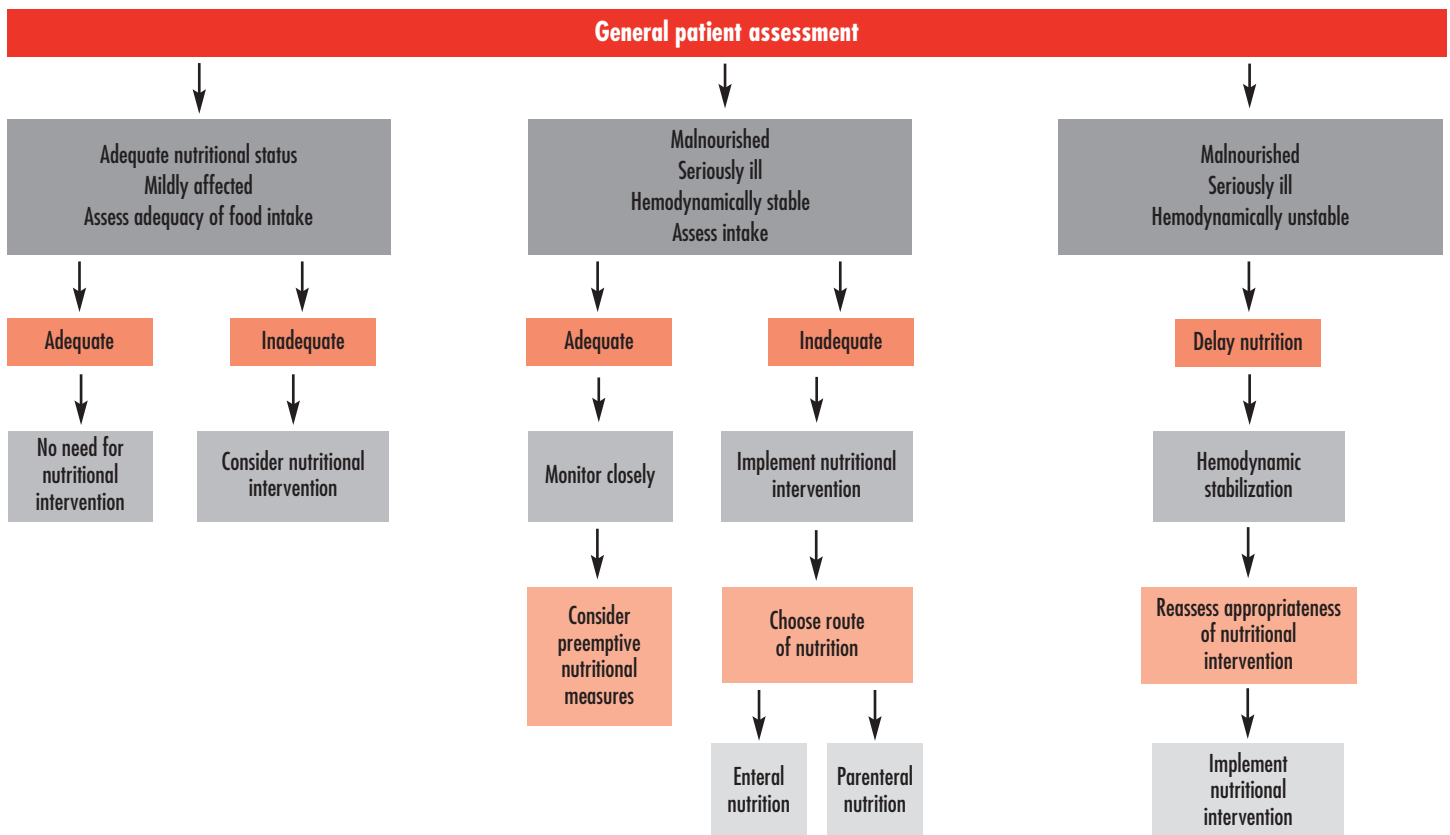
Interacting Medical Management Strategies

Various antibiotics may cause nausea, vomiting, or diarrhea. Chemotherapeutic agents may cause severe gastrointestinal complications. Diuretics and angiotensin-converting enzyme (ACE) inhibitors may also decrease appetite.

Monitoring

All critically ill patients receiving nutritional support should be closely monitored for possible complications related to nutritional support. Patients with feeding tubes should be inspected for infection/inflammation at the surgical exit site. Biochemical and hematologic tests may also be helpful in identifying metabolic complications. Although body weight and body condition scores are essential in patients receiving nutritional support, weight gain per se is not necessary.

Algorithm – Nutritional Support of Critically Ill Canine Patients



Critical Care Nutrition – Feline Hepatic Lipidosis

Daniel L. Chan, DVM, MRCVS, DACVECC, DACVN

Definition

Critical illnesses can have a significant impact on nutritional status in cats. In *hepatic lipidosis*, a syndrome resulting from excessive and pathologic accumulation of lipid within the liver, critical care nutritional support is an important part of medical therapy and reversal of this condition.

Key Diagnostic Tools and Measures

Physical examination findings (e.g., dehydration, icterus, mental depression, neurologic signs) may indicate a more severe degree of lipidosis. Nutritional assessment should be performed to guide the nutritional plan, and this usually requires obtaining a biochemical profile. Alkaline phosphatase (ALP) activity, alanine aminotransferase (ALT) activity, aspartate aminotransferase (AST) activity, and total bilirubin concentrations are significantly elevated in this condition and are supportive of the diagnosis. Biopsy or fine-needle aspirates of the liver can confirm the diagnosis.

Pathophysiology

The exact mechanism responsible for triggering hepatic lipidosis in cats is unclear; however, anorexia is a common feature of the disease and is believed to be a major contributing factor. Protein and calorie malnutrition is believed to overstimulate lipolysis resulting in mobilization of free fatty acids that overwhelms the liver's capacity to process the influx of triglycerides. Excessive lipid accumulation within hepatocytes disrupts cellular function and may result in overt liver failure.

Signalment

Middle-aged to older cats are most commonly affected. Male cats have been suggested to be over-represented but there is no significant increased risk associated with gender. Obesity is perhaps the most important major predisposing factor.

Key Nutrient Modifications

A negative energy balance is a major component of this disease and nutritional support is paramount for effective reversal of the condition. Protein should be of good quality and highly digestible. Protein levels should not be restricted unless there is a clear contraindication, such as signs of hepatic encephalopathy which includes neurologic impairment, excessive salivation, and seizure activity. Although this condition features a limited capacity by the liver to process fatty acids, some authors advocate fat restriction; however, as the provision of adequate calories is so critical for recovery, diets high in fat are commonly used without complications. Supplements such as S-adenosylmethionine (SAMe), taurine, and carnitine have been advocated for the management of hepatic lipidosis; however, the benefit of these supplements in this condition has not been evaluated.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	40–60	8–13	26	6.5
Fat	15–35	4–6	9	2.3

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

Ensuring adequate nutritional intake is the most important therapeutic feeding principle for this condition. The vast majority of cats with hepatic lipidosis require placement of a feeding tube (e.g., esophagostomy, gastrostomy feeding tube). With the exception of nasoesophageal feeding tubes, placement of feeding tubes require general anesthesia, which carries a high risk of complications in hemodynamically unstable patients. Cats that are deemed unstable for anesthesia may benefit from placement of a nasoesophageal tube or may be candidates for parenteral nutrition. Parenteral nutrition is usually reserved for patients that cannot tolerate enteral feeding (e.g., vomiting, severe diarrhea).

Initiation of feeding should commence once dehydration and electrolyte and acid–base imbalances have been addressed. Feedings should aim to meet resting energy requirements (RER):

$$\text{RER} = 70 \times (\text{Body weight in kg})^{0.75}$$

Initial feedings should begin at 33% to 50% RER for the first day, and gradually increase to meet RER over next 48 hours. If the patient tolerates feedings (i.e., is not vomiting), feeding targets could be increased by 10% to 20% RER to achieve a stable body weight during hospitalization. Tube feeding is usually required for several weeks and the decision to discontinue tube feedings is made once the cat is eating adequate amounts of food voluntarily.

■ **Treats** – For critically ill animals, the use of treats is usually ineffective at meeting energy and nutrient requirements and only delays more appropriate nutritional support. Animals receiving tube feedings may be offered treats to assess return of appetite.

■ **Tips for Increasing Palatability** – The use of appetite stimulants in critically ill animals is not recommended as they are ineffective at restoring adequate nutritional intake. Techniques such as hand feeding or warming the food may be attempted but are also typically ineffective at achieving adequate nutritional support. Appetite stimulants such as ciproheptadine or mirtazapine may have a role in cats that have recovered from their underlying disease process and are being transitioned to oral feedings.

■ **Diet Recommendations** – Diets typically used to treat cats with hepatic lipidosis are energy dense and high in protein and fat. Diets intended for critical care patients are used most commonly. Most diets, however, will need to be modified (adding water, blenderized) in order to be used effectively with feeding tubes. With small-gauge tubes, typically used for nasoesophageal access, complete liquid diets are the only acceptable diets.

Client Education Points

- As many recovering patients can be discharged from the hospital with the feeding tube in place (e.g., esophagostomy, gastrostomy tubes), clients need to be instructed how to use and care for feeding tubes.
- Clients need to be made aware of possible complications and how to detect them.
- Clients should be provided with detailed and specific instructions for how to use feeding tubes – this should include instructions on how to prepare the diet and how to administer the feedings.

Common Comorbidities

Comorbidities in cats requiring critical care nutrition for hepatic lipidosis include pancreatitis, cholangiohepatitis, and inflammatory bowel disease.

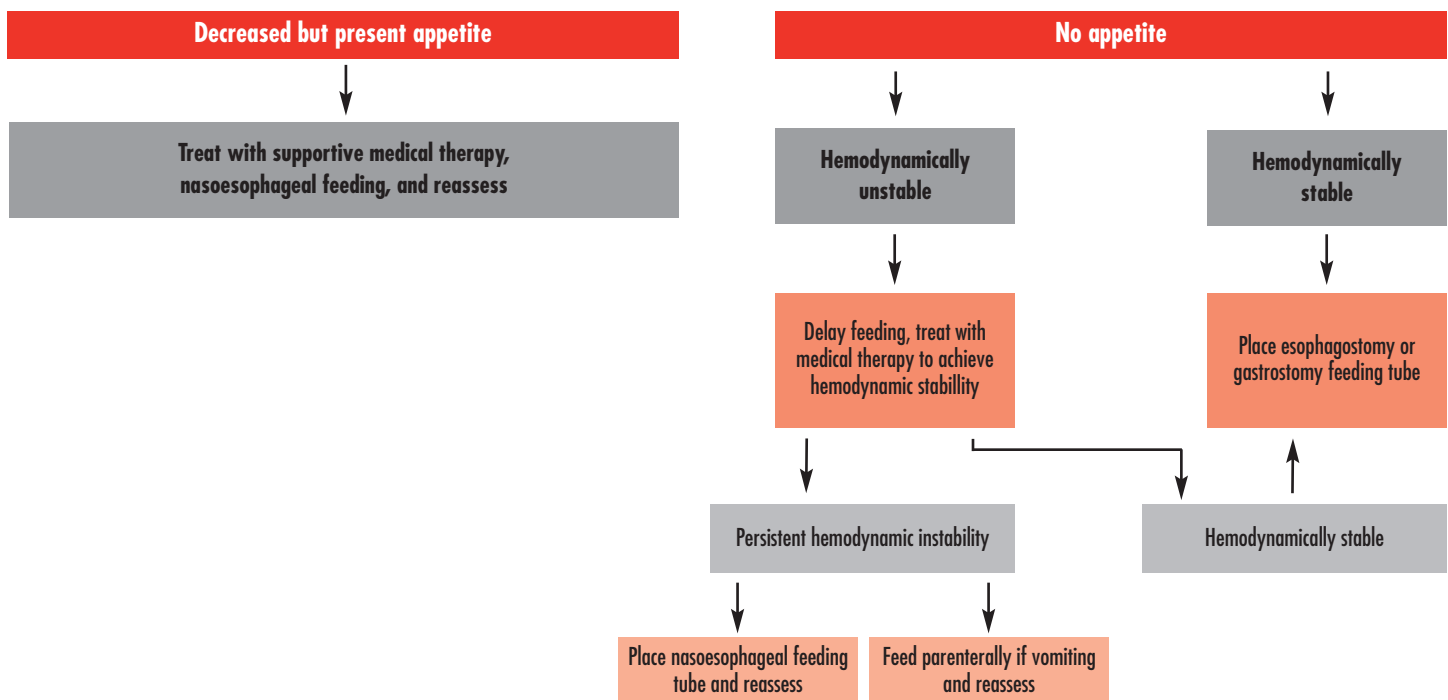
Interacting Medical Management Strategies

Caution must be exercised if nutritional supplements (e.g., taurine, carnitine, S-adenosylmethionine) are administered via the feeding tube; these may occlude the feeding tube and necessitate tube replacement.

Monitoring

All critically ill patients receiving nutritional support should be closely monitored for possible complications related to nutritional support. Patients with feeding tubes should be inspected for infection/inflammation at the surgical exit site. Biochemical and hematologic tests may also be helpful in identifying metabolic complications. Although body weight and body condition scores are essential in patients receiving nutritional support, weight gain per se during hospitalization is not necessary.

Algorithm – Nutritional Support of Critically Ill Feline Patients with Hepatic Lipidosis



Diabetes Mellitus – Canine

Linda Fleeman, BVSc, MACVSc, PhD
Jacquie Rand, BVSc, DVSc, DACVIM

Definition

Diabetes mellitus is caused by absolute or relative deficiency of insulin. This results in altered carbohydrate, fat, and protein metabolism, which manifests as hyperglycemia, hyperlipidemia, polyuria, lethargy, weight loss, polyphagia, poor hair coat, and reduced immunity.

Key Diagnostic Tools and Measures

Diagnosis of diabetes mellitus is based on hyperglycemia and glucosuria with compatible clinical signs. Measurement of canine pancreatic lipase immunoreactivity (cPLI) may aid in the identification of concurrent pancreatitis. Canine trypsin-like immunoreactivity (cTLI) may identify concurrent exocrine pancreatic destruction. Continued monitoring of body condition, glycemia, and serum triglyceride concentration is recommended.

Pathophysiology

Type 1 and other specific types of diabetes have been reported in dogs; the relative frequency varies with geographic location and particularly with neutering rates in female dogs. In North America, approximately 50% of dogs have type 1 diabetes mellitus caused by immune destruction of pancreatic beta cells. In approximately 30%, diabetes is due to extensive pancreatic damage from chronic pancreatitis. Canine diabetes also occurs secondary to corticosteroid therapy, hyperadrenocorticism, or progesterone-induced acromegaly. In intact bitches, a form analogous to human gestational diabetes can occur during diestrus or pregnancy and is common in countries with low neutering rates, such as Sweden.

Signalment

Most diabetic dogs are over 5 years of age with the highest prevalence occurring between 8 and 12 years of age. Intact females are at increased risk, especially if they are also overweight. Mixed-breed dogs have increased risk compared with most pure breeds. The following breeds are at increased risk: Australian terrier, standard schnauzer, Samoyed, miniature schnauzer, fox terrier, keeshond, bichon frise, Finnish spitz, cairn terrier, miniature poodle, Siberian husky, and toy poodle.

Key Nutrient Modifications

Total carbohydrate content of the diet is the major determinant of the glycemic response of typical commercial dog foods, and so a moderately carbohydrate-restricted diet (<30% metabolizable energy [ME]) is recommended; and meals should have consistent carbohydrate content. The guaranteed analysis statement on pet foods does not provide information on the carbohydrate content of the food, and so this must be inferred (see **Appendix III**). Low glycemic index carbohydrate sources are likely preferable; recommended sources include sorghum and barley. Rice is unlikely to be an optimal carbohydrate source. Diets with corn syrup should be excluded.

Although several studies indicate that high-fiber diets, compared with low-fiber diets, might be associated with improved glycemic control, there has been no clear demonstration of clinical benefit for diabetic dogs of feeding a high-fiber formulation compared with feeding a typical adult maintenance diet with moderate-fiber content (30–40 g/1000 kcal). Dietary fat restriction (<30% ME) is recommended for diabetic dogs with concurrent chronic pancreatitis or persistent hypertriglyceridemia. Nutrient requirements for concurrent diseases usually have priority over those for diabetes mellitus.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	25–50	6–10	18	5.1
Fat	8–12 [#]	3–5 [#]	5	1.4
Carbohydrate	0–40	0–8	n/a	n/a
Crude Fiber	5–15	2–4	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]Dietary fat restriction is recommended for diabetic dogs with concurrent chronic pancreatitis or persistent hypertriglyceridemia. Dietary fat restriction should not be routinely recommended for diabetic dogs with thin body condition.

Therapeutic Feeding Principles

Exogenous insulin therapy is the mainstay of clinical management of diabetes mellitus in dogs, and the primary goals are long-term resolution of all clinical signs and avoidance of insulin-induced hypoglycemia. A successfully managed diabetic dog will have no polyphagia, lethargy, or polydipsia, and will be able to maintain body weight. A typical management regimen includes twice-daily insulin dosages and consistent meals, which should be highly palatable so that food intake is predictable.

Commercial dog foods usually result in postprandial elevation of plasma glucose for less than 90 minutes following consumption by dogs and meals should be timed so that maximal exogenous insulin activity occurs during the postprandial period. Thus, dogs should be fed within 2 hours of administration of lente or NPH insulin or within 6 hours of protamine zinc insulin. When a twice-daily insulin dosing regimen is used, a feasible compromise is to feed the dog immediately following the insulin injection.

If mild signs of hypoglycemia develop, the owner should feed a meal of the dog's usual food or high-carbohydrate treats. Hand-feeding might be necessary to encourage the dog to eat. If the dog is unwilling or unable to eat, syrup containing a high glucose concentration can be administered orally. Suitable syrups are marketed for use by human diabetics and should be kept in reserve by all owners of diabetic dogs. When the dog recovers, a meal of the dog's usual food should be fed immediately, and then the owner should contact their veterinarian before the next insulin injection is due. Half the usual dose of insulin should be administered when a diabetic dog does not eat the accompanying meal.

■ **Treats** – Feeding consistent meals at fixed times each day is an important aspect of management; owner compliance should be encouraged. If treats are fed, they should be consumed during the expected period of maximal exogenous insulin activity. Treats containing high sugar or fat should be avoided.

■ **Tips for Increasing Palatability** – The majority of diabetic dogs will readily consume meals twice daily following the insulin injections if the meals are highly palatable and contain half the daily caloric requirement. For finicky eaters, the meal should be fed at the time of insulin administration and remain available until the expected end of the period of maximal exogenous insulin activity. Diabetic dogs are more likely to readily accept a diet that has a formulation similar to the diet they were consuming before diagnosis of diabetes. Foods with high sugar or fat content should not be used to improve the palatability of food prescribed for diabetic dogs. An example of a suitable alternative is warm low-fat chicken broth.

■ **Diet Recommendations** – Diets formulated for canine adult maintenance

with moderate dietary fiber and carbohydrate content will be suitable for most diabetic dogs. A fat-restricted diet should be considered for diabetic dogs with concurrent chronic pancreatitis or persistent hypertriglyceridemia. High-fiber, restricted-fat diets should not be routinely recommended for diabetic dogs with thin body condition. Most well-managed diabetic dogs require a similar amount of food per day as healthy non-diabetic dogs of similar age, gender, and lifestyle. Diabetic dogs with reduced exocrine pancreatic function have increased caloric requirement compared with healthy dogs.

Client Education Points

- Feeding consistent meals at fixed times each day is crucial to successful management of diabetes in dogs. Ideally, every meal should contain the same ingredients and calorie content.
- Timing of meals must be matched with timing of insulin injections.
- The importance of avoiding an insulin overdose cannot be over-emphasized. If some insulin is spilt during injection the owner should never give more at that time, even if it appears that the dog has received no insulin. If the owner is ever uncertain, the safest option is to withhold the injection, as the consequences of missing a single dose are negligible.
- Owners must be aware of the nutritional strategies for management of hypoglycemia as described earlier.
- Owners should seek prompt veterinary advice whenever a diabetic dog shows inappetence or anorexia because there is an increased risk of hypoglycemia if insulin is administered when the dog does not eat.

Common Comorbidities

Urinary tract infection, pancreatitis, hyperadrenocorticism, dermatitis, otitis externa, and exocrine pancreatic insufficiency are common conditions occurring in dogs with diabetes mellitus, as well as diestrus, pyometra, and

obesity in intact diabetic females.

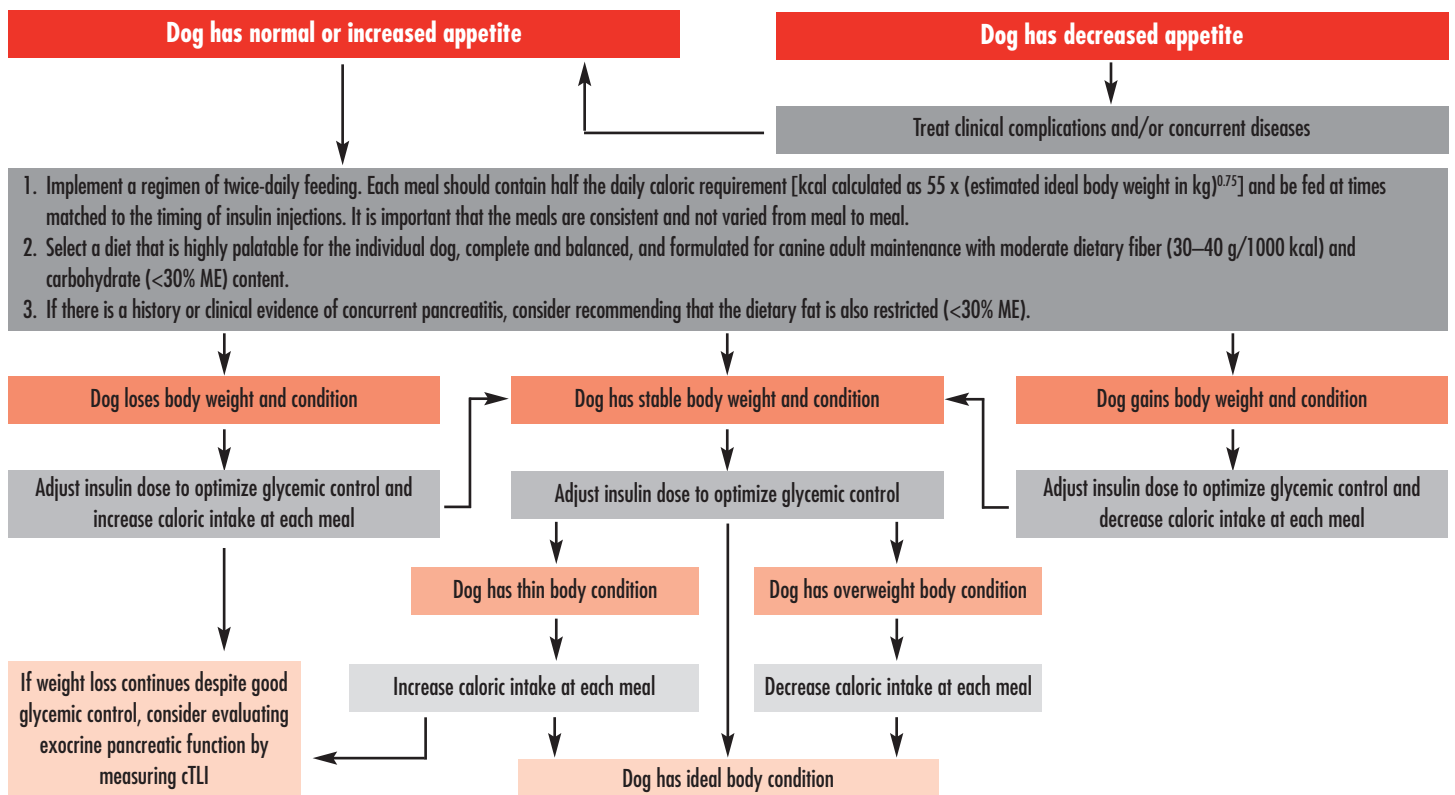
Interacting Medical Management Strategies

Exercise can be associated with increased risk of hypoglycemia in insulin-treated diabetic dogs. This can be managed with reduced insulin dose and/or increased feeding prior to exercise. Management strategies must be individualized for each dog.

Monitoring

One of the key clinical signs of untreated diabetes mellitus is loss of body weight and condition, despite polyphagia. With institution of appropriate medical and nutritional therapy, weight loss is usually arrested before optimal glycemic control is achieved. It is, therefore, important to monitor both body weight and body condition at each re-assessment. Glycemic monitoring is used to evaluate response to the insulin and dietary regimen. Fasting serum triglyceride concentration can be monitored to identify persistent hypertriglyceridemia, and to monitor the response to feeding a fat-restricted diet. Exogenous insulin therapy will result in resolution of hypertriglyceridemia in some diabetic dogs, while others require dietary fat restriction in addition to insulin therapy. Dietary fat restriction <30% ME should be recommended for all diabetic dogs with fasting serum triglyceride concentration >500 mg/dL because of the association with pancreatitis. For diabetic dogs with good glycemic control, dietary fat restriction <30% ME is recommended if fasting serum triglyceride concentration is >400 mg/dL. It is expected that fasting triglyceride levels will decrease in response to dietary fat restriction. Therefore if fasting serum triglyceride concentration is >400 mg/dL when the dog is being fed a diet with <30% ME fat, further restriction of dietary fat to <20% ME is recommended. If body weight loss continues despite adequate glycemic control, it is recommended that serum cTLI concentration be assayed to evaluate exocrine pancreatic function.

Algorithm – Nutritional Management of Canine Diabetes Mellitus



Diabetes Mellitus – Feline

Jacque Rand, BVSc, DVSc, DACVIM
Linda Fleeman, BVSc, MACVSc, PhD
Rebecca Remillard, PhD, DVM, DACVN

Definition

In feline *diabetes mellitus*, hyperglycemia occurs because of inadequate insulin secretion from pancreatic beta cells. Peripheral insulin resistance secondary to genotype, obesity, physical inactivity, disease, or drugs is often a contributing factor.

Key Diagnostic Tools and Measures

Persistent hyperglycemia is indicative of diabetes mellitus. Concurrent clinical signs of polyuria, polydipsia, and a history of weight loss are common and support a diagnosis of diabetes. Glucosuria and ketonemia help confirm the diagnosis; however not all cats are ketonuric or ketonemic. If blood glucose concentration is only moderately elevated (200–300 mg/dL; 12–17 mmol/L), persistent hyperglycemia should be documented on two to three successive blood samples taken at least 4 hours apart over 1 to 2 days to rule out stress-induced hyperglycemia. Fructosamine concentrations of ≥ 400 to 500 $\mu\text{mol/L}$ are supportive of diabetes, while those greater than 500 $\mu\text{mol/L}$ are highly associated with diabetes.

Pathophysiology

More than 80% of cats are thought to have type 2 diabetes mellitus which results from beta cell failure that occurs secondary to prolonged demand for increased insulin secretion as a result of peripheral insulin resistance. Eventually this damages beta cells and insulin secretion fails. The remaining cases are associated with other specific types of diabetes, such as pancreatic carcinoma, pancreatitis, acromegaly or hyperadrenocorticism, which either destroy beta cells directly or cause marked insulin resistance. Once blood glucose increases, it further suppresses insulin secretion and damages beta cells (termed glucotoxicity). Most cats are insulin dependent at the time of diagnosis; however depending on the underlying cause and duration of diabetes and management, 20% to 90% of diabetic cats can achieve non-insulin dependence (termed remission).

Signalment

Cats with diabetes are typically older, with a peak onset between 10 and 13 years. Cats that are overweight or obese, neutered, male, and domestic breed are at increased risk. In the US, Maine Coon, domestic long-haired, Russian Blue, and Siamese are over-represented, whereas Burmese cats are four times more likely to be diabetic in Australia, New Zealand, and the UK compared with domestic cats.

Key Nutrient Modifications

Diets low in soluble carbohydrate^a (CHO) (<20% on a dry matter [DM] basis; <15% metabolizable energy) are considered superior for the management of diabetes and have proven efficacious in managing feline diabetes. Grains suggested to have a lower glycemic index in the cat include corn, sorghum, oats, and barley. By limiting dietary carbohydrates, blood glucose is maintained primarily from hepatic gluconeogenesis and blood glucose fluctuations after a meal are minimized. Recently diagnosed diabetic cats usually do well on a low-CHO high protein diet. Cats require protein ($\geq 30\%$ DM and $>85\%$ digestible) of high biologic value (egg, meat, liver). Achieving diabetic remission is an advantage in these cats and is an important goal in any cat that has been diabetic for less than one year. The probability of remission is low in cats that have been diabetic for more than one year.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	40–60	10–17	26	6.5
Carbohydrate	0–20	0–5	n/a	n/a
Fat	10–35	3–7	9	2.3

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

The management goals in feline diabetes are to avoid insulin-induced hypoglycemia and to optimize the chance of achieving remission by minimizing hyperglycemia. It is generally recommended that diabetic cats be fed twice daily at the time of the insulin injections, although it is acceptable to provide smaller meals more frequently. The postprandial period of cats is very long and blood glucose remains elevated for more than 14 hours after a meal containing 50% to 100% of daily energy requirements.

Feeding low-CHO foods is associated with increased rates of remission in newly-diagnosed diabetic cats compared with feeding high-fiber foods. Because remission has such great advantages for the client and cat, diabetic management should be initially directed at maximizing the probability of remission. Therefore, low-CHO food should be fed in newly-diagnosed diabetics and to all diabetic cats in remission. Relapse of diabetes has occurred in cats when changed to a higher-CHO diet. However glucose control is not significantly different in cats that remain insulin-dependent when a high-fiber, high-CHO diet rather than a low-CHO diet is fed, although insulin dose is usually lower in those fed low-CHO foods. Dietary management of the co-morbidities must also be considered when selecting diets for the diabetic patient.

Overweight and obesity are associated with insulin resistance; therefore maintaining or achieving an ideal body weight is important in facilitating remission in diabetic cats. Overweight and obese cats should be fed restricted amounts of a diet with a low caloric density (fat) with the least amount of CHO (NFE) as possible (<20% DM).^{*} The energy intake needs to be restricted so that 1% to 2% loss of body weight occurs per week although more commonly 0.3% to 0.5% per week is achieved.

Cats with mild to moderate signs of hypoglycemia, such as weakness, trembling, and wobbliness, that are still able to eat should immediately be fed a palatable, highly digestible, high-CHO, low-fiber “intestinal” diet. If signs are severe, such as seizure or coma, glucose syrup designed for human diabetic patients can be applied to the gums and owners should seek veterinary attention immediately.

■ **Treats** – Maintaining a constant and low CHO intake is important, and high CHO treats should be avoided. Suitable examples include portions of the cat’s usual low-CHO diet or home-cooked meat or fish treats with a fat and protein content (high or low) suitable for the co-morbidities.

■ **Tips for Increasing Palatability** – Transition the diet change from the

^a Soluble CHO (mostly starch) is measured and reported as Nitrogen Free Extract (NFE) whereas CHO as fiber is reported as Crude Fiber.

regular diet to the suitable diabetic diet over 5 to 14 days; a longer period may be needed for cats that are more resistant to change. The palatability of food generally increases with increased temperature, water, and nutrients (fat, protein, and salt). Warm (microwave) food or lightly heat canned food. Add warm chicken or beef broth (+/- sodium) or add water or oil from canned fishes (sardine, tuna, mackerel) if appropriate to enhance taste.

■ **Diet Recommendations** – Nutrient ranges of low-CHO diets recommended for diabetic cats are <20% CHO, 40% to 60% protein, and 10% to 35% fat on a DM basis. Cats should be fed to maintain or achieve an ideal body weight. Canned foods are generally more palatable, contain more water and fat, and less CHO than kibble, and facilitate weight loss in some overweight and obese cats.

Client Education Points

- Feed meals at the time of insulin injection at 12-hour intervals. It is recommended that only food products designed for a diabetic cat are fed, and that the food is obtained from a reliable source for quality control and product consistency.
- Cats can become non-insulin dependent (remission), hence close monitoring is essential. It is essential to continue feeding a low-CHO diabetes diet to cats in remission to minimize demand on beta cells to secrete insulin.
- Cats with mild to moderate signs of hypoglycemia such as weakness, trembling, and wobbliness that are still able to eat should be immediately fed a palatable highly digestible diet, high-CHO, low-fiber “intestinal” diet. If signs are severe such as seizure or coma, glucose syrup designed for human diabetic patients can be applied to the gums, and owners should seek veterinary attention immediately.

Common Comorbidities

In lowering the CHO fraction, the dietary protein, fat, fiber, or some combination thereof must increase to account for the difference. Low CHO diets with varying levels of fat, protein, and fiber are useful in providing a multitude of dietary options depending upon the comorbidities for each case. Underweight diabetic cats should be fed CHO (<20%DM), protein (~55% DM), low fiber (1% DM), high fat (20–30% DM) and energy density (4–5 kcal/g DM metabolizable energy [ME]) diets. Overweight and obese cats can be managed by restricting the total amount of energy (as fat) fed. Insulin sensitivity should improve with the loss in body fat. Overweight and obese diabetic cats are best managed with CHO (<20% DM) diets with moderate fiber (10–15% DM) and low fat (~10 % DM) and caloric density (3–3.5 kcal/g DM ME). Feeding canned

diets may be more satiating than dry food for some cats due to the water content although canned foods generally contain more fat than the comparable dry version.

In cats with renal insufficiency (IRIS 2), try a low-CHO diet (<15% DM) because azotemia improves in many cats with improved glycemic control. If azotemia worsens, these cats may do better on a CHO (30–40% DM) diet with 35–40% DM protein and <1% DM phosphorus. Cats with advanced renal disease (IRIS 3 or 4) with systemic signs associated with azotemia are better managed with a restricted protein diet (CHO <30% DM, protein ~30% DM, fat ~30% DM, and <1% DM phosphorus) with phosphate binding agents to further control plasma phosphate concentrations in conjunction with acarbose to reduce glucose absorption from the gastrointestinal tract. Home-made diets that control the CHO, protein and restrict phosphorous are also an option for some clients.

Other common conditions in diabetic cats include pancreatitis or cancer (adenocarcinoma) (can still be fed low-CHO, high-protein diabetic foods); bacterial cystitis and urinary tract infections; hyperlipidemias (change to a lower-fat, low-CHO diabetic food); endocrinopathies (hyperadrenocorticism, acromegaly, hyperthyroidism); drug-induced conditions (glucocorticoids, progestins); and stress-induced hyperglycemia associated with illness (manage these cats as diabetics until resolved).

Interacting Medical Management Strategies

Corticosteroids predispose to diabetes, and repeated long-acting injections of corticosteroid are particularly important to avoid in diabetic cats, or cats in remission. Similarly progestins, such as megestrol acetate, decrease insulin sensitivity and predispose to diabetes.

Monitoring

Blood glucose concentrations need to be monitored to determine the level of glycemic control and appropriate insulin dosage. Blood glucose is best monitored at home using a portable glucose meter, preferably one calibrated for feline blood. Where this is not possible, home monitoring of urinary glucose and ketone concentrations is helpful. Exogenous insulin is administered with a low-CHO, high-protein diet (preferably) to control blood glucose concentrations, and is adjusted accordingly to maintain as close to a normal blood glucose concentration as possible while avoiding hypoglycemia. Monitor body weight and adjust energy intake to achieve an ideal weight.

See **Algorithm - Nutritional Management of Feline Diabetes Mellitus** on page 34.

Overweightness / Obesity – Canine

Sean J. Delaney, DVM, MS, DACVN

Definition

Obesity, defined as the excessive accumulation and storage of adipose tissue, is common in dogs, affecting over a third of dogs,¹ and is the number one nutritional disease encountered by veterinarians. On the nine-point Nestlé PURINA Body Condition Scoring system (see Appendix I), dogs with a body condition score (BCS) of 6 or more are considered overweight, dogs with a score of 8 or more are obese, and those with a score of 9 are morbidly obese.

Key Diagnostic Tools and Measures

Body condition scoring utilizes both visual and tactile cues to assign a numeric value to a patient's degree of adiposity. Since body condition scoring can be readily explained to clients, it is an effective tool to increase a client's awareness of the dog's degree of overweightness or obesity. Before starting a patient on a weight loss plan, it is recommended that a physical examination, complete blood count, and biochemical panel be performed to rule out any underlying diseases, especially hypothyroidism.

Pathophysiology

Typically, weight gain occurs gradually over many years as a result of the ingestion of excessive calories, as well as increasingly sedentary lifestyles, leading to the accumulation of adipose tissue. Hypothyroidism, hyperadrenocorticism, and administration of glucocorticoids can also lead to overeating.

Signalment

The likelihood of overweightness/obesity increases with age and with neutering. Certain breeds, including cocker spaniels, dachshunds, Dalmatians, Labrador retrievers, golden retrievers, Shetland sheepdogs, and Rottweilers, are at greater risk.²

Key Nutrient Modifications

Determining a dog's specific energy requirement is crucial to establishing a successful weight loss regimen. In a dog whose weight is fairly stable, current intake may be the best indicator of a patient's actual energy requirement due to large individual variation (\pm 50% of calculated requirement based on body weight). Collecting an accurate diet history (see Appendix II) can allow the veterinarian to calculate the patient-specific energy requirements. Once this has been established, weight loss can be achieved by feeding fewer calories than are required for weight stability. Ideally, a weight loss diet should have low energy density and an increase in the essential nutrients per kilocalorie. See Therapeutic Feeding Principles for detailed strategies.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	25–50	10–20	18	5.1
Fat	5–15	2–3.5	5	1.4

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should be increased relative to energy content of the diet in order to meet normal requirements adjusted for calorie restriction.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

The rationale for feeding a diet with decreased energy density achieved with additional fiber, water, and/or “air puffing,” is that it will lead to satiety by gut fill; the larger volume may be more acceptable to client (greater bowel and bowl fill). The rationale for increased essential nutrients per kilocalorie is that caloric restriction without concurrently maintaining the amount of essential nutrients being fed may lead to deficiencies; these deficiencies may be frequently recognized as poor coat quality during weight loss.

Feed fewer calories than are required: either 80% of current calories fed if this can be accurately determined with the diet history (see Appendix II) or calculate the dog's resting energy requirement (RER) based on current weight:

$$\text{RER} = 70 \times \text{weight in kg}^{0.75}$$

(see Appendix III for useful equations in clinical nutrition). Adjust the amount fed based on weigh-ins at 2-week intervals with the goal of 1% to 2% of body weight lost per week; faster weight loss decreases muscle mass, decreases compliance, and increases risk of rebound weight gain. If the dog is gaining weight on this diet, check compliance and decrease all amounts fed by 20%. If weight is stable, decrease all amounts by 10%. If the dog is losing weight too fast, check general health and increase all amounts fed by 10% to 20%.

Recommend a concurrent increase in activity with play or walks.

■ **Treats** – When giving commercial treats designed for weight loss, select treats that are less than 30 kcal/treat to allow for multiple treating opportunities daily. High-moisture vegetables and fruit, such as 3½ cups boiled, sliced zucchini (100.8 kcal), 20 raw baby carrots (105 kcal; avoid in dogs with calcium oxalate urolith concerns), 2 cups cubed cantaloupe (108.8 kcal), or 2 cups peeled and sliced apple (105.6 kcal), are healthy treats. Lean and low-sodium meats, such as ½ cup chopped or diced chicken breast (112 kcal) or 1½ ounces pork chop, meat only, no fat or bone (99.5 kcal), can also be given as treats.

■ **Tips for Increasing Palatability** – High-moisture foods that are canned or pouched may be preferred over dry foods. Dry food can also be soaked with water or sodium-free meat broth. Top dressing of a small amount of lean and low-sodium meat (see Treats) that does not exceed 10% of daily calories may be added or mixed in to prevent selective eating. A small amount of syrup can be mixed into food as long as it does not exceed 10% of daily calories (e.g., 1½ tbsp light corn syrup, 100.7 kcal)

■ **Diet Recommendations** – A food marketed and designed for active weight loss is strongly recommended. The diet should have less than 280 kcal/cup or less than 22 kcal/oz if canned or pouched if using the low energy density strategy, and less than 25% carbohydrate calories if using low carbohydrate strategy. Avoid OTC foods that use the words/phrases “less calories,” “reduced calorie,” “lean,” “low fat,” “less fat,” or “reduced fat” as terms relate only to food being compared to, have no set calorie definition, and do not indicate that the food has a low energy density. Avoid OTC foods that use the word “light,” “lite,” or “low calorie” as these are for prevention of weight gain and not for active weight loss; light foods are not as increased in essential nutrients per kilocalorie as foods designed for weight loss. Home-made recipes are not typically needed for patients in need of weight loss nor are they recommended. Their increased digestibility and palatability can be counterproductive in dogs that need to lose weight.

Client Education Points

- Dogs have a large variation in individual energy requirements ($\pm 50\%$). Obese patients often are very efficient users of calories. Historically when food was scarce, efficiency was a desirable trait (“your dog is genetically superior”), but now efficiency in an environment of abundant food leads more easily to weight gain.
- The initial phase of a weight loss plan is determining how efficient the dog is. Therefore, feeding the prescribed amount is very important and tailoring the plan based on weigh-ins is crucial; imperfect rates of weight loss should be initially expected.
- The goal is of the weight loss plan is improved quality and quantity of life.³

Common Comorbidities

Osteoarthritis is common in overweight and obese dogs; joint disease is managed with medication and weight loss. Consider nutritional management of joint disease after weight loss has been achieved. Fish oil and/or chondroprotective supplementation of food designed for weight loss can be considered if necessary. For **tracheal collapse**, **brachycephalic**

syndrome, or **laryngeal paralysis**, attempt to maximize rate of weight loss to 2% of body weight per week, especially if needed weight loss can be achieved before warmer seasonal temperatures. In dogs with **diabetes mellitus**, weight loss may enable improved glycemic control.

Interacting Medical Management Strategies

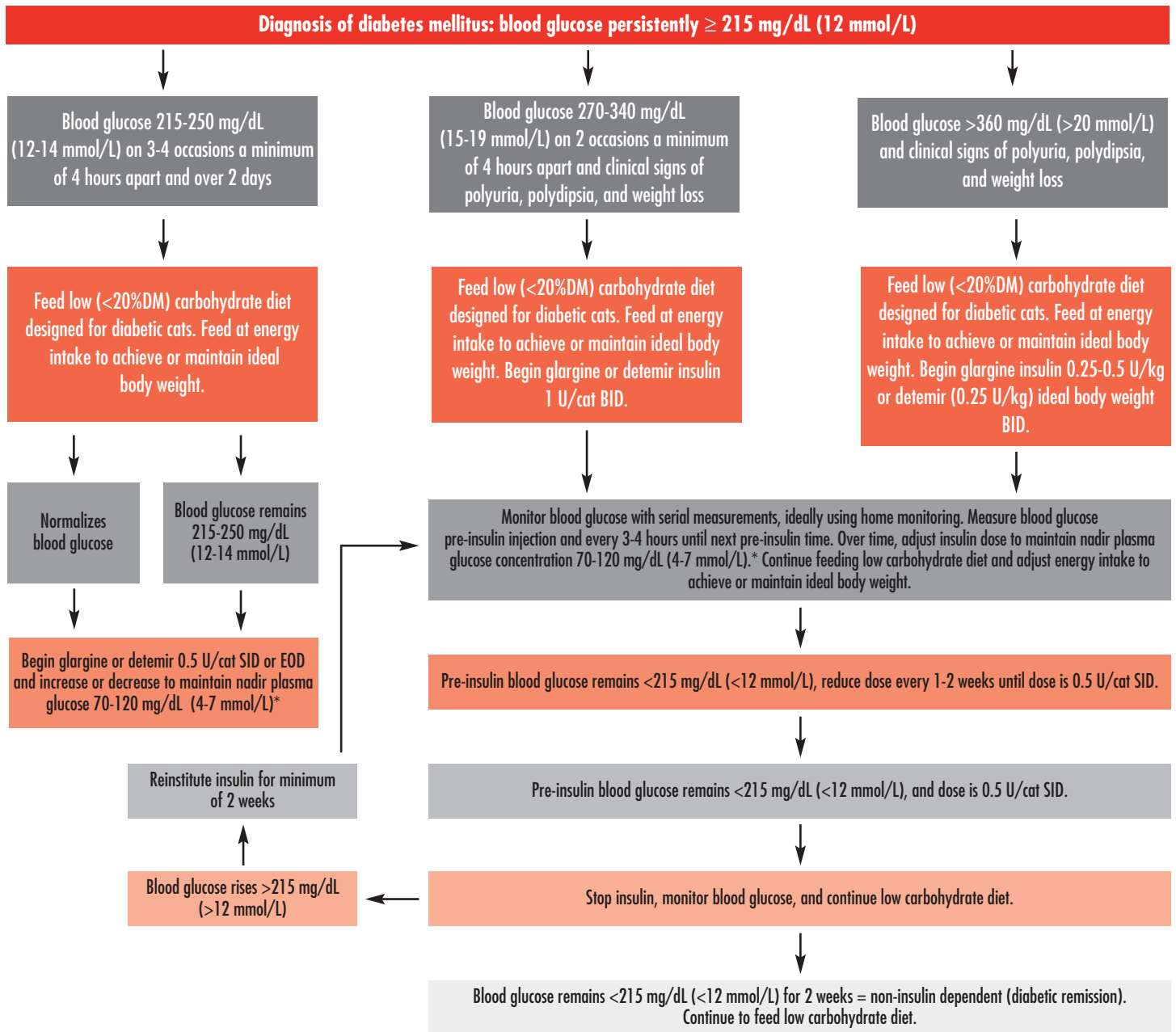
Comorbidities resulting in inappetence should be the primary focus of nutritional management to prevent unintentional weight loss due to illness being ignored and attributed to the weight loss regimen.

Monitoring

Reweigh the dog every 2 weeks and adjust the amount fed accordingly (*see Therapeutic Feeding Principles*). The goal weight loss rate is 1% to 2% of body weight per week. If a comorbidity is expected to improve with weight loss, check if it is & offer positive reinforcement. If an overweight, arthritic dog is more active after some initial weight loss, congratulate and remind client that it is attributable to the weight loss.

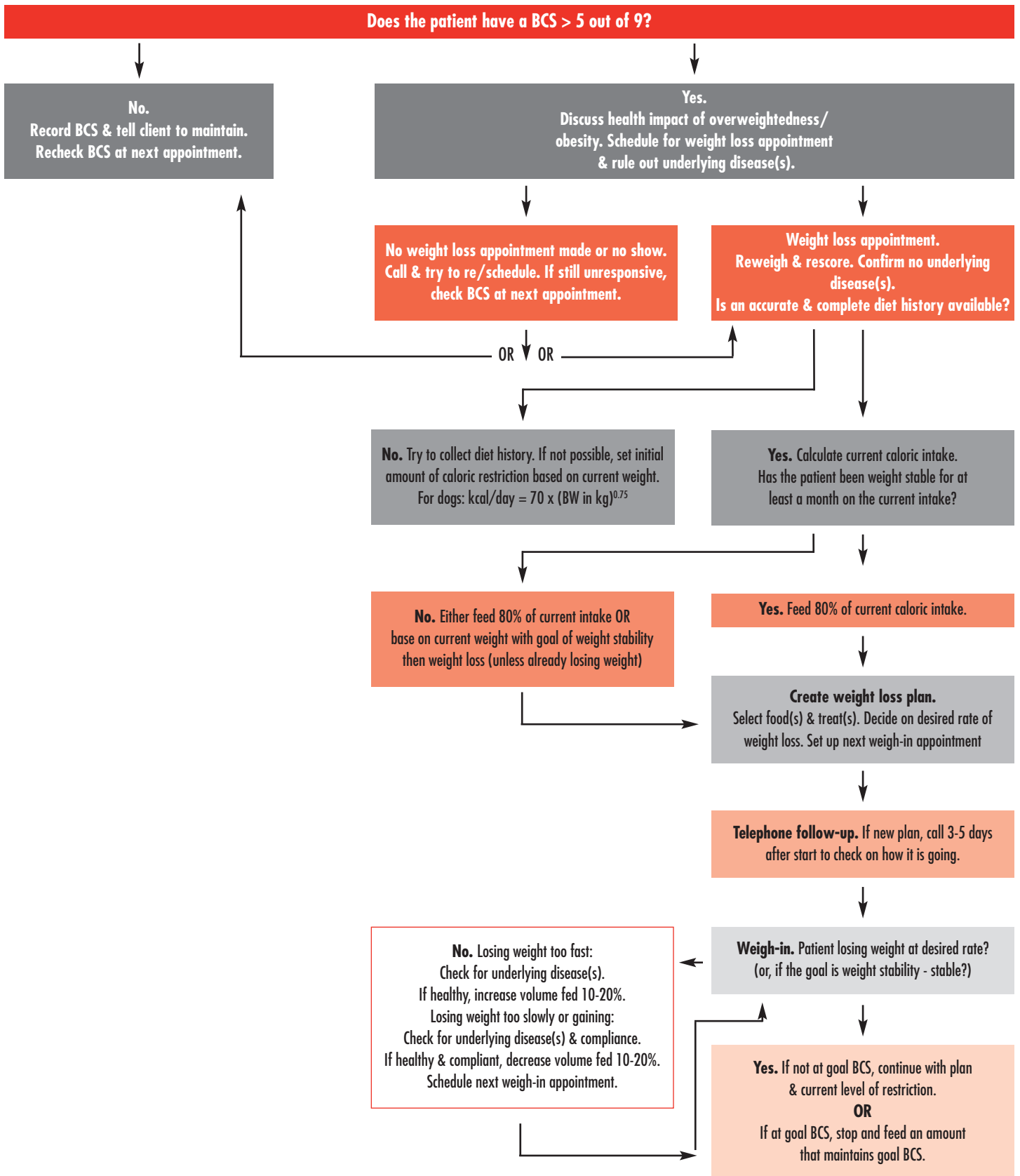
See **Algorithm – Nutritional Support for Canine Overweightness & Obesity** on page 35.

Algorithm – Nutritional Management of Feline Diabetes Mellitus



*Measured with a glucose meter calibrated for feline blood or a serum chemistry analyzer; glucose meters calibrated for human blood read 18–36 mg/dL (1–2 mmol/L) lower than actual blood glucose concentration.

Algorithm – Nutritional Support for Canine Overweightness & Obesity



Overweightness / Obesity – Feline

Rebecca Remillard, PhD, DVM, DACVN

Definition

Obesity is qualitatively defined as an excess of body fat sufficient to contribute to disease. *Overweight* cats (body condition score [BCS] of 6 or 7/9) carry 25% and 30% body fat, respectively. *Obese* cats (BCS 8/9) carry 35% fat, whereas morbidly obese cats (BCS 9/9) carry 40% or more fat (see Appendix I for more on body condition scoring).

Key Diagnostic Tools and Measures

Body weight, body weight history (Appendix II), and BCS estimate the degree of body fat excess. Body condition scoring is performed visually and through palpation. Routine blood and urine measures are performed to rule out causative and concurrent medical conditions.

Pathophysiology

In the absence of endocrine or metabolic disease, pet obesity is an iatrogenic disease due to daily maintenance energy (MER) intake exceeding daily energy expenditure. MER for most indoor neutered cats is approximated by $MER = 85 \times (BW \text{ kg})^{0.75}$ (see Appendix III). Daily food intake history for overweight or obese cats will show chronic caloric excess.

Signalment

The majority of overweight or obese cats are between 2 and 15 years old, with most between 5 and 10 years of age, neutered, and primarily housed indoors. Mixed breeds are more likely to be overweight or obese than purebred cats.

Key Nutrient Modifications

The necessary nutrient change is a caloric deficiency while not restricting other non-energy nutrients unless necessary to address comorbidities. A variety of commercial therapeutic foods have been used for the past 20 years in the treatment of feline obesity. Methods of weight management have traditionally included the use of low-fat, high-fiber foods to reduce caloric intake and body weight while maintaining satiety. There are large variations in fiber (soluble and crude) content among feline weight loss foods. Increased dietary protein appears to promote weight loss and reduce loss of lean body mass during weight loss in cats. Foods with added L-carnitine may aid fat loss depending on the level of protein in the diet. More recent concepts of weight loss in cats include using high-protein, low-carbohydrate foods.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	40–60	10–18	26	6.5
Fat	8–20	2.5–5.0	9	2.3

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should be increased relative to energy content of the diet in order to meet normal requirements adjusted for calorie restriction.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

Although it is ultimately calorie restriction that induces weight loss, it is important to avoid excessive restriction of essential nutrients. Therefore, a low-calorie product with increased nutrient/calorie ratios should be considered. It is also important to promote fat loss while minimizing loss of lean tissue, which is influenced by diet composition. Most of the available foods contain 40% to 50% protein calories and 25% to 40% fat calories.

Traditional methods of weight management have used low-fat, high-fiber foods to reduce caloric intake and body weight while attempting to maintain satiety. Several different nutrient profiles, however, have been successfully used for feline weight loss: 1) low-fat, low-fiber kibble food; 2) low-fat, high-fiber canned and kibble foods; 3) high-fat, low-carbohydrate, moderate-fiber canned and kibble foods; and 4) high-protein, low-fat, moderate-fiber kibble food. All diet strategies have shown significant decreases in weight and body fat with insignificant loss in lean body mass when fed to obese cats. There are 10 to 15 different feline weight loss products to choose from depending on diet palatability and product availability; however, restricting daily calories and food intake is essential to all weight loss programs.

Overweight cats should be transitioned onto a weight loss food over a 5- to 10-day period. A measured daily food allotment should be offered to the cat in multiple (three to six) meals per day if possible. Feeding meals separately from other house pets and people is key to a successful weight loss program. The cat is weighed monthly and compared with an estimated ideal body weight.

■ **Treats** – Most owners appreciate a treat allowance of 20 to 25 kcal per day. The treats should be limited to low-calorie options, such as raw vegetables and fruits, which initially the cat may refuse until weight loss begins. Kibble pieces of the same or different feline weight loss diet or feline dental food may be used as a treat between meals. Any and all low-calorie treats allowed must be counted as part of the daily calorie intake and the daily food amount must be reduced accordingly.

■ **Tips for Increasing Palatability** – There is rarely a problem with cats not eating the total allotment of weight loss food if calorie restriction is in place. If there is food refusal of a weight loss diet, consider changing the form of diet (kibble vs. canned) to the form the cat prefers. Cats do have preferences for the texture, consistency and ‘mouth feel’ of food. Also consider using a high-protein, low-calorie weight loss food as cats have specific taste receptors for animal protein.

■ **Diet Recommendations** – Weight loss begins when cats are fed 50% to 75% of ideal weight MER calories per day using one of the many therapeutic feline weight loss diets. The estimated rate of weight loss is approximately 1% per week or 0.5 to 1 lb per month. Continued weight loss may require feeding 200 or fewer kcal per day as the cat approaches ideal body weight. The food must be measured and a daily food diary is illustrative.

Client Education Points

- Cat owners have indicated that feeding the cat is an important positive factor in their relationship with the pet yet the majority did not perceive their cats as overweight. The owner must be able and willing to control caloric intake for a weight loss program to succeed. Use of an automatic feeder is one option. Numerous options are available; thus, another key to success is a flexible design with regular follow-up with the client.
- Obesity treatment programs that include dietary changes, measured food allotments (using a gram scale is ideal), and monthly body weight checks by a veterinary health care team are successful. Weight loss is a slow steady progress which may take 6 to 12 months to reach a goal weight.

- In cats, weight rebound occurs after rapid weight loss and when cats are allowed free access to energy-dense food after weight reduction. Most often the diet used during weight loss is recommended for long-term weight maintenance of the goal weight. The only difference is a greater daily amount of food is used for weight maintenance.

Common Comorbidities

Recent research has suggested a mechanism for the link between excess body weight and many diseases. Obesity is now seen as a chronic pro-inflammatory state producing oxidative stressors. It seems that adipose tissue, once considered to be physiologically inert, is an active producer of hormones, such as leptin and resistin, and numerous cytokines. Of major concern are the pro-inflammatory cytokines for adipose (adipokines) tumor necrosis factor- α (TNF- α) and interleukins 1 β and 6.

Common comorbidities in overweight and obese cats include diabetes (insulin resistance and glucose intolerance) directly associated with the degree of adiposity and circulating inflammatory mediators; osteoarthritis, FLUTD, cardiovascular disease and pancreatitis due to a chronic low-grade inflammation and oxidative state; orthopedic (cruciate tears) injuries due to excessive weight; hyperlipidemias and hepatic lipidosis due to derangements in lipid metabolism; and non-allergic dermatitis due to the inability to properly self-groom.

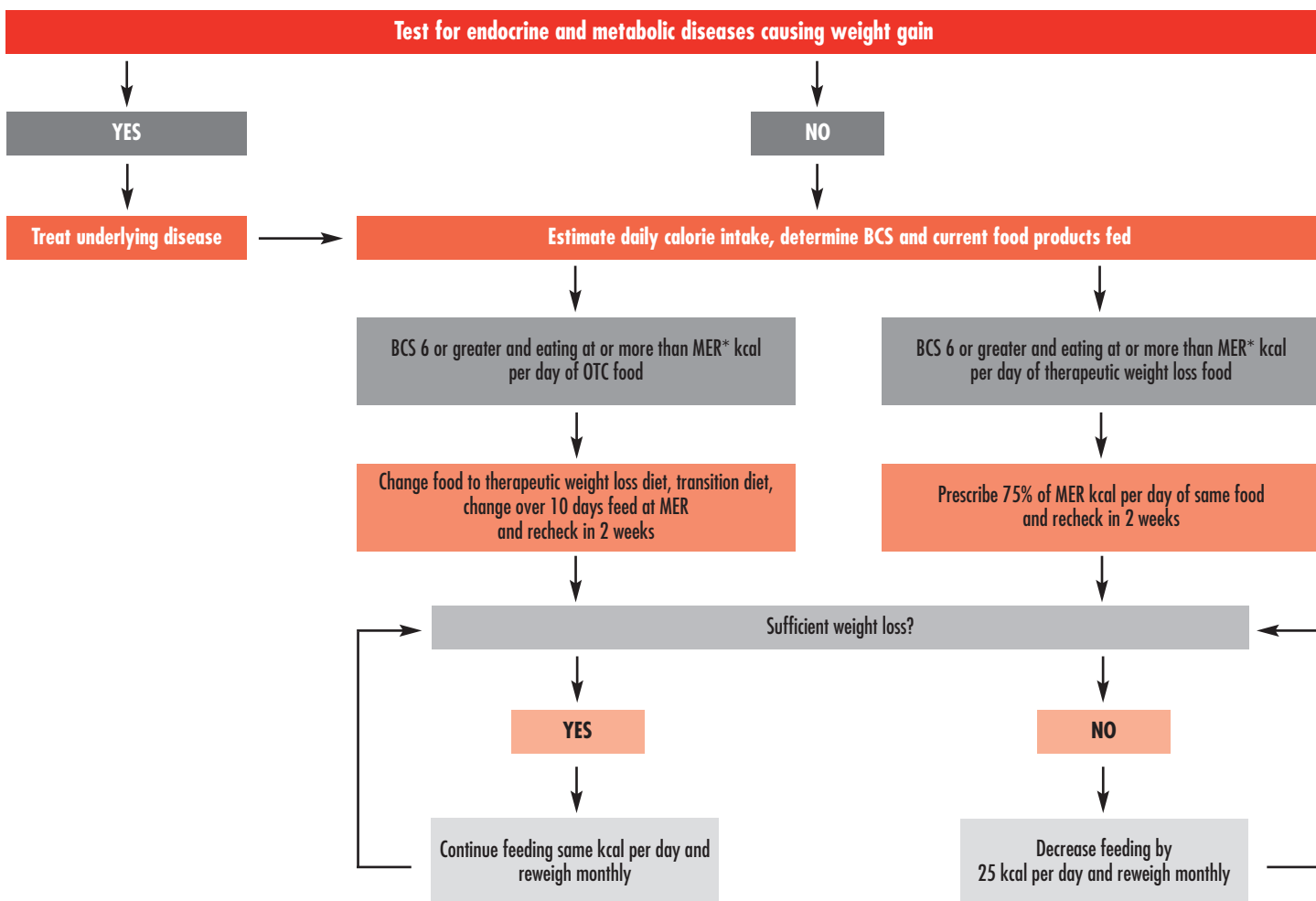
Interacting Medical Management Strategies

Feline hepatic lipidosis is rare in healthy cats fed fewer calories for weight loss as long as the cat consumes the entire daily allotment. If the cat refuses to eat the weight loss diet for several days, the possibility of hepatic lipidosis increases. Diabetic cats receiving insulin must be monitored carefully as insulin requirements decrease as insulin sensitivity returns. Cats receiving any medications based on body weight must be monitored carefully for dose adjustment as weight loss succeeds.

Monitoring

By recording body weight and BCS, ideal body weight can be more easily determined. Physical examinations and weight checks are suggested monthly with a discussion about daily feeding regime and food measurements. Behavioral changes in feeding the cat are essential. Discuss logistical feeding problems within the household (multi-cat household, boarding, family members, visitors, etc.). Changing food and adjusting calorie intake as needed to address problems and ensure continued weight loss. Clients should be encouraged to develop non-food related bonding activities to reduce the intake of calories and increase calorie expenditure.

Algorithm – Nutritional Management of Feline Overweightness/Obesity (BCS>5/9)



*MER = $85 \times (\text{BW kg})^{0.75}$

Constipation – Canine

Sally Perea, DVM, MS, DACVN

Definition

Constipation is characterized by absent, infrequent, or difficult defecation associated with retention of feces within the colon and rectum. Severe constipation can progress to *obstipation* when the feces become excessively hard and impacted within the colon. *Megacolon* refers to dilation and hypomotility of the colon, but is rarely seen in dogs.¹

Key Diagnostic Tools and Measures

A diagnosis of constipation is generally made during history collection and physical examination. Clinical signs may include tenesmus, anorexia, vomiting, weight loss, lethargy, and poor coat condition. Rectal and abdominal palpation will likely reveal firm stool within the rectum and colon. Abdominal radiographs can be used to further define the extent of the constipation, and to rule out foreign bodies, enlarged prostate, and pelvic or spinal lesions that may be contributing to the constipation. Serum chemistry, thyroxine (T4), complete blood count, and urinalysis are also indicated to rule out underlying metabolic abnormalities.

Pathophysiology

Constipation can occur with any condition that impairs the movement of feces through the colon. When feces are retained within the colon for an extended period of time, water continues to be absorbed, resulting in a progressively harder and drier fecal mass. Constipation can occur secondary to rectocolonic obstruction (such as prostatic hypertrophy, pelvic fracture, neoplasia, diverticulum), painful defecation (anal wounds, orthopedic disorders), environmental factors (confinement/boarding, inactivity), medications (opioids, diuretics, others), neuromuscular dysfunction, fluid and electrolyte abnormalities, ingestion of foreign material, or inadequate water intake.

Signalment

Constipation can occur in dogs at any age, and is seen in both males and females of all breeds. The signalment may help to narrow the differential diagnoses. For example, neoplasia is more commonly seen in older animals, and prostatic hypertrophy is only seen in males.

Key Nutrient Modifications

Increasing fiber and moisture content of the diet are the key nutrient modifications that can be made to address constipation in dogs. Obstruction or partial obstruction of the colon should be ruled out prior to initiating a high-fiber diet or fiber supplementation. Fiber is classified as soluble or insoluble. Soluble fiber has the ability to hold water, which helps to increase the moisture content of dry feces and normalize gastrointestinal transit time. Some soluble fibers are fermentable and support the growth of normal gastrointestinal flora and production of short chain fatty acids that provide energy to colonocytes and stimulate longitudinal colonic smooth muscle contractions.² Insoluble fiber has a low ability to hold water and is not readily degraded by gastrointestinal bacteria. Insoluble fiber adds bulk to the feces and can help to stimulate colonic motility.³

Dogs with constipation may be dehydrated; therefore, increasing moisture in the diet can help to maintain appropriate hydration and soften dry stools.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Crude fiber [#]	7–15	2–8	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]The crude fiber analysis includes most insoluble fibers, but does not include soluble fibers. Therefore, crude fiber has limited usefulness when evaluating the total fiber content of foods. The ingredient list should be evaluated for sources of soluble fiber.

Diets high in moisture may be helpful in this condition: canned foods contain about ≥75% water, versus dry foods which provide ~10% water.

Therapeutic Feeding Principles

Hydration status should be corrected prior to initiating dietary treatment. If the patient is prone to dehydration, feeding a canned diet or adding water to a dry kibble (two to three parts water to one part dry kibble) is recommended. A diet providing a combination of soluble and insoluble fiber sources is ideal for the management of constipation in dogs. Soluble fiber increases stool moisture, while the insoluble fiber provides fecal bulk and stimulates motility. Because most pet foods do not report soluble and insoluble fiber levels, the ingredient list can be evaluated to gain a better understanding of the fiber types in the diet. Examples of soluble fiber include citrus pulp and other fruits (provide pectins), gums (such as guar gum), and oligosaccharides (such as carrageenan); insoluble fibers include cellulose, brans (such as rice and wheat), oat fiber, and peanut hulls; and mixed fibers include beet pulp, soy fiber, pea fiber, and psyllium. If the patient cannot be transitioned to a high-fiber diet, supplemental fiber can be added to the current diet.

- Psyllium is a readily available mixed fiber supplement that can be added to the diet in amounts of 1–3 tablespoons per day.
- If a source of soluble fiber is needed, Benefiber (guar gum) can be added to the diet in amounts of 2–4 teaspoons per day.
- If a source of insoluble fiber is needed, coarse wheat bran can be added to the diet in amounts of 1–3 tablespoons per day.

The amount of supplementation needed to correct constipation can vary in individual patients; therefore, it is generally recommended to start at the low end of the dosage range and titrate to effect. Adjustments to the fiber dosage should be made every 5 to 7 days as needed until the desired affect is achieved.

■ **Treats** – Fruits and vegetables are good sources of both soluble and insoluble fibers, and are generally high in moisture.

- 1 medium baby carrot = 3 kcal, 180 mg total dietary fiber (60 g/1000 kcal), 90% moisture
- ¼ cup sliced/chopped apple with skin = 16 kcal, 750 mg total dietary fiber (47 g/1000 kcal), 96% moisture

Whole grains and bran cereal are also good sources of insoluble fiber.

- ¼ cup Mini-Wheats = 48 kcal, 1.66 g total dietary fiber (33.3 g/1000 kcal)

Care should be taken to ensure that unbalanced treats are limited to ≤10% of the total daily calories.

- If feeding a high-fiber diet, additional high-fiber treats may be contraindicated. Some fiber supplements designed for human use may be sweetened. Supplements sweetened with xylitol should be avoided.

■ Tips for Increasing Palatability

- Slightly heat food to enhance food odor and texture.
- Add a low-sodium chicken or beef broth to the food to increase both moisture and palatability (limit to $\leq 10\%$ of the total daily calories, and avoid broths made with onion or garlic).
- If adding water to food to increase moisture, start by adding a small amount and then slowly increase over 1 to 2 weeks to allow the patient to become accustomed to the change in dietary moisture and texture.

■ **Diet Recommendations** – Foods providing moderate to high dietary fiber with mixed soluble and insoluble fiber sources are recommended. Veterinary therapeutic foods designed for diabetes mellitus, colitis, and weight loss generally provide increased dietary fiber levels. In general, weight loss foods will provide a greater proportion of the fiber from an insoluble source. Canned foods and/or the addition of water to the food is recommended for patients prone to dehydration.

Client Education Points

- Proper hydration is key to the management of constipation. Free access to fresh water should be provided at all times. Water intake can also be enhanced by feeding canned foods or adding two to three parts water to one part dry kibble.
- Confinement or lack of activity can contribute to constipation.
- Implement a regular exercise routine or walking schedule.
- Feeding a higher-fiber diet or adding a fiber supplement will result in increased fecal volume and frequency of defecation.
- Transitioning to a higher-fiber diet or adding a fiber supplement should be done slowly over 4 to 5 days.
- The level of fiber supplementation needed can vary from pet to pet, so adjustments may be required to achieve the desired response.

Common Comorbidities

Common comorbidities are generally predisposing factors for constipation. Conditions that cause pain on defecation (such as anorectal and orthopedic disorders), rectocolonic obstruction (such as pelvic fractures or neoplasia), or neuromuscular dysfunction (such as lumbosacral spinal cord disease or dysautonomia), are commonly seen with constipation. For disease conditions associated with colonic obstruction or partial obstruction, feeding a high-fiber, fecal bulking diet may be contraindicated. In these cases, feeding a highly digestible diet to decrease fecal mass is more appropriate. When constipation is seen with fluid or electrolyte abnormalities (such as hypokalemia or hypercalcemia) correction of the hydration status and electrolyte imbalances should take priority in the treatment plan.

Interacting Medical Management Strategies

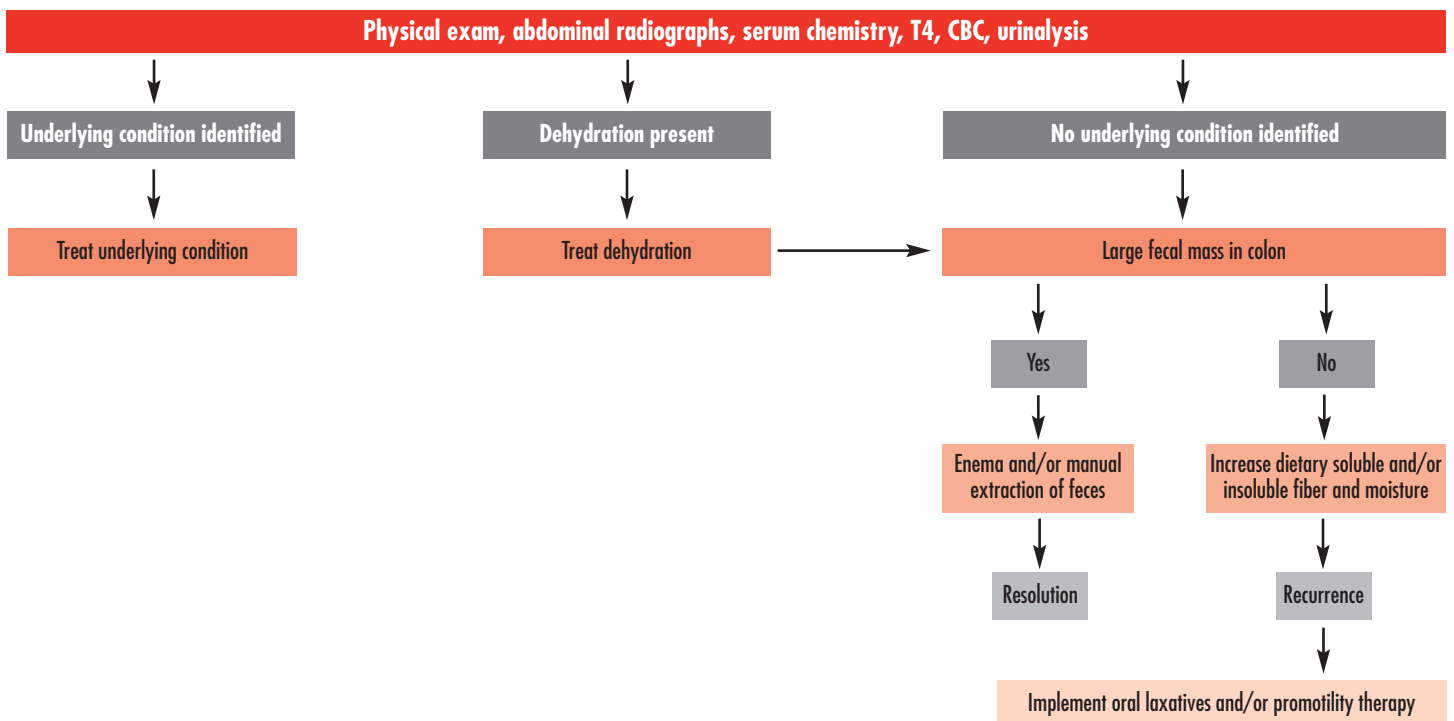
Medical management should be aimed at eliminating or controlling any identified underlying conditions. If a large fecal mass is present, enema treatment and/or manual extraction may be required. If dehydration or electrolyte abnormalities are identified, proper fluid therapy is indicated.

If dietary therapy alone is not successful in preventing recurrence of constipation, oral laxative medications can be implemented. Generally, a mild emollient laxative such as docusate sodium, or osmotic laxative such as lactulose is recommended to help soften the stool. Proton pump inhibitors, such as cisapride, may also be beneficial in dogs with decreased colonic motility.

Monitoring

Response to treatment can be monitored by having the owner record daily bowel movements and fecal characteristics. Success of treatment is characterized by return of daily bowel movements, absence of straining or pain on defecation, and normal fecal consistency.

Algorithm – Nutritional Management of Canine Constipation



Constipation – Feline

Sally Perea, DVM, MS, DACVN

Definition

Constipation is characterized by absent, infrequent, or difficult defecation associated with retention of feces within the colon and rectum. Constipation can progress to *obstipation* when the feces become excessively hard and impacted within the colon. *Megacolon* refers to dilation and hypomotility of the colon, and is usually seen with severe constipation and may include colonic smooth muscle or neurologic abnormalities.¹

Key Diagnostic Tools and Measures

A diagnosis of constipation is generally made during history collection and physical examination. Clinical signs may include tenesmus, anorexia, vomiting, weight loss, lethargy, and poor coat condition. Rectal and abdominal palpation will likely reveal firm stool within the rectum and colon. Abdominal radiographs can be used to further define the extent of the constipation, and to rule out megacolon, foreign bodies, and pelvic or spinal lesions that may be contributing to the constipation. Serum chemistry, thyroxine (T4), complete blood count, and urinalysis are also indicated to rule out underlying metabolic abnormalities.

Pathophysiology

Constipation can occur with any condition that impairs the movement of feces through the colon. When feces are retained within the colon for an extended period of time, water continues to be absorbed, resulting in a progressively harder and drier fecal mass. Constipation can occur secondary to rectocolonic obstruction (such as pelvic fracture, neoplasia, diverticulum), painful defecation (anal wounds, orthopedic disorders), environmental factors (dirty litter box, inactivity), medications (opioids, diuretics, others), neuromuscular dysfunction, fluid and electrolyte abnormalities, ingestion of hair or foreign material, or inadequate water intake.

Signalment

Constipation can occur at any age, and has not been reported to have a breed or sex predilection. Increased frequency of megacolon, however, has been reported in middle-aged male cats, with domestic short-haired, domestic long-haired, and Siamese being the most commonly affected breeds.¹

Key Nutrient Modifications

Increased dietary fiber and moisture are the key nutrient modifications that can be made to address mild to moderate cases of constipation in cats. For cats with megacolon, feeding a highly digestible diet to decrease fecal mass is recommended. Megacolon and obstruction or partial obstruction of the colon should be ruled out prior to initiating a high-fiber diet or fiber supplementation.

Fiber is classified as soluble or insoluble. Soluble fiber has the ability to hold water, which helps to increase the moisture content of dry feces and normalize gastrointestinal transit time. Some soluble fibers are fermentable and support the growth of normal gastrointestinal flora and production of short chain fatty acids that provide energy to colonocytes and stimulate longitudinal colonic smooth muscle contractions.² Insoluble fiber has a low ability to hold water and is not readily degraded by gastrointestinal bacteria. Insoluble fiber adds bulk to the feces which can help to stimulate colonic motility.^{3,4} Many cats with constipation are dehydrated; therefore, increasing moisture in the diet can help to maintain appropriate hydration and help to soften the stool.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Crude Fiber [#]	5–8	1.3–5.5	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]The crude fiber analysis includes most insoluble fibers, but does not include soluble fibers. Therefore, crude fiber has limited usefulness when evaluating the total fiber content of foods. The ingredient list should be evaluated for sources of soluble fiber.

Diets high in moisture may be helpful in this condition: canned foods contain about ≥75% water, versus dry foods which provide ~10% water.

Therapeutic Feeding Principles

Hydration status should be corrected prior to initiating dietary treatment. If the patient is prone to dehydration, feeding a canned diet or adding water to a dry kibble (two to three parts water to one part dry kibble) is recommended. For cats without megacolon, a diet providing a combination of soluble and insoluble fiber sources is ideal for the management of constipation. Soluble fiber increases stool moisture, while the insoluble fiber provides fecal bulk and stimulates motility. Cats with megacolon should be fed a low-fiber, highly digestible diet to help reduce fecal mass. Because most pet foods do not report soluble and insoluble fiber levels, the ingredient list can be evaluated to gain a better understanding of the fiber types in the diet. Examples of soluble fiber include citrus pulp and other fruits (provide pectins), gums (such as guar gum), and oligosaccharides (such as carrageenan). Insoluble fibers include cellulose, brans (such as rice and wheat), oat fiber, and peanut hulls. Mixed fibers include beet pulp, soy fiber, pea fiber, and psyllium. If the patient cannot be transitioned to a high-fiber diet, supplemental fiber can be added to the current diet.

- Psyllium is a readily available mixed fiber supplement that can be added to the diet in amounts of 1–4 teaspoons per day.
- If a source of soluble fiber is needed, Benefiber (guar gum) can be added to the diet in amounts of ½–2 teaspoons per day.
- If a source of insoluble fiber is needed, coarse wheat bran can be added to the diet in amounts of 1–4 teaspoons per day.

The amount of supplementation needed to correct constipation can vary in individual patients; therefore, it is generally recommended to start at the low end of the dosage range, and titrate to effect. Adjustments to the fiber dosage should be made every 5 to 7 days as needed until the desired affect is achieved. Some fiber supplements are sweetened with fructose or sucrose (which contains fructose). Because fructose is poorly metabolized by cats, formulations containing fructose or sucrose should be avoided. Supplements that contain xylitol should also be avoided.

■ **Treats** – Pumpkin is commonly recommended for fiber supplementation in cats. The amount of fiber provided by canned pumpkin (0.4 g/tablespoon) is small compared with the levels that are provided by fiber supplements such as psyllium (9 g/tablespoon). Therefore, pumpkin alone may not provide the amount of fiber needed to see a clinical response but may serve as a good supplemental treat.

- Ensure that clients select canned pumpkin as opposed to pumpkin pie filling.
- Care should be taken to ensure that unbalanced treats are limited to

≤10% of the total daily calories.

- One tablespoon of canned pumpkin provides 5 kcal.

■ **Diet Recommendations** –

- Slightly heat food to enhance food odor and texture.
- Add a low-sodium chicken or beef broth to the food to increase both moisture and palatability (limit to ≤10% of the total daily calories, and avoid broths made with onion or garlic).
- If adding water to food to increase moisture, start by adding a small amount and then slowly increase over 1 to 2 weeks to allow the patient to become accustomed to the change in dietary moisture and texture.

■ **Diet Recommendations** – Canned foods and/or the addition of water to dry food are recommended. Foods should provide moderate dietary fiber with mixed soluble and insoluble fiber sources. Veterinary therapeutic foods designed for diabetes mellitus and weight loss may provide increased dietary fiber levels. In general, weight loss foods will provide a greater proportion of the fiber from an insoluble source. If megacolon is present, a low-fiber/highly digestible diet, such as those designed for gastrointestinal disease, is recommended.

Client Education Points

- Proper hydration is key to the management of constipation. Free access to fresh water should be provided at all times. Water intake can also be enhanced by feeding canned foods or adding two to three parts water to one part dry kibble.
- Many cats will avoid using a dirty litter box, which can contribute to constipation; therefore, daily cleaning of the litter box (with thorough cleaning and replacement of litter weekly) is important in the management of constipation.
- Transitioning to a higher-fiber diet or adding a fiber supplement should be done slowly over 4 to 5 days.
- The level of fiber supplementation needed can vary from pet to pet, so adjustments may be required to achieve the desired response.

Common Comorbidities

Common comorbidities are generally predisposing factors of constipation. Conditions that cause pain on defecation (such as anorectal and orthopedic disorders), rectocolonic obstruction (such as pelvic fractures or neoplasia), or neuromuscular dysfunction (such as lumbosacral spinal cord disease, or dysautonomia), are commonly seen with constipation.

For disease conditions associated with colonic obstruction or partial obstruction, feeding a high-fiber, fecal bulking diet may be contraindicated. In these cases, feeding a highly digestible diet to decrease fecal mass is more appropriate. When constipation is seen with fluid or electrolyte abnormalities (such as hypokalemia or hypercalcemia) correction of the hydration status and electrolyte imbalances should take priority in the treatment plan.

Interacting Medical Management Strategies

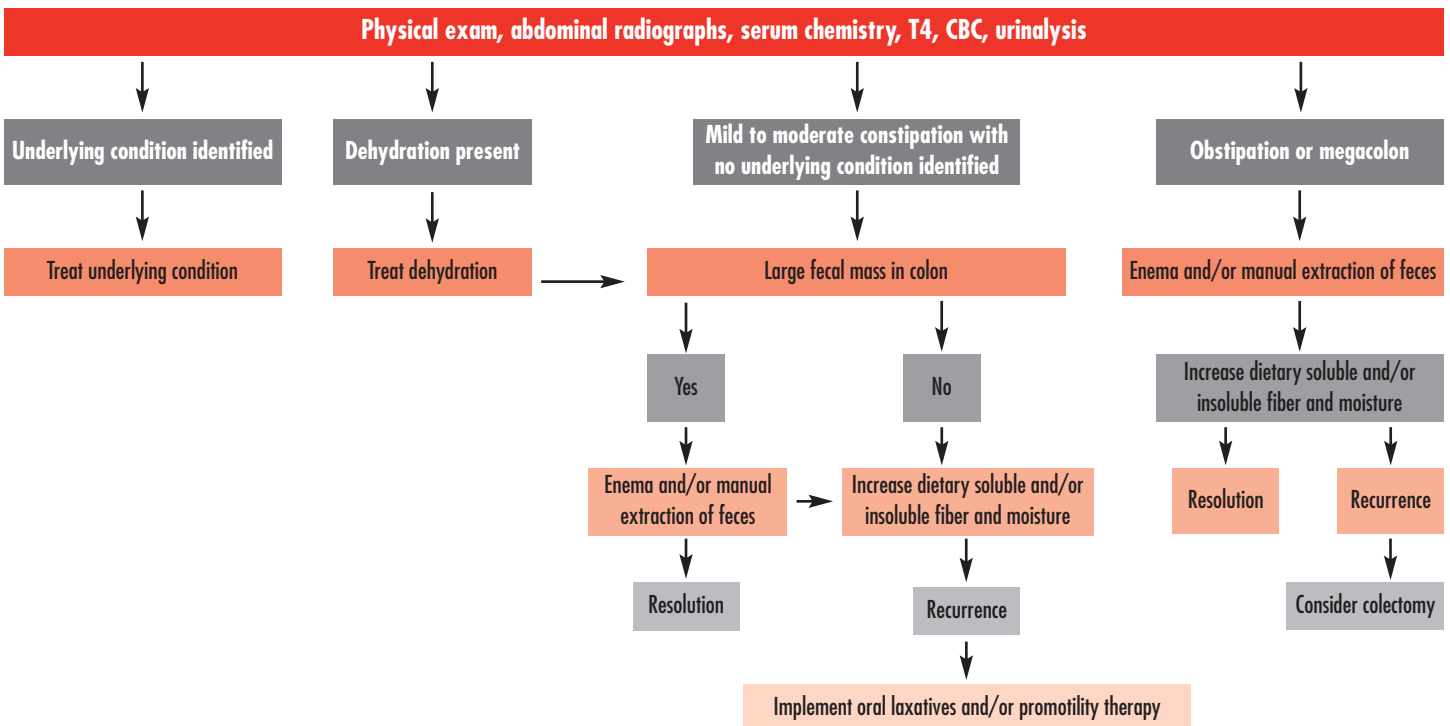
Medical management should be aimed at eliminating or controlling any identified underlying conditions. If a large fecal mass is present, enema treatment and/or manual extraction may be required. If dehydration or electrolyte abnormalities are identified, proper fluid therapy is indicated.

If dietary therapy alone is not successful in preventing recurrence of constipation, oral laxative medications can be implemented. Generally, a mild emollient laxative such as docusate sodium, or osmotic laxative such as lactulose is recommended to help soften the stool. Proton pump inhibitors, such as cisapride, may also be beneficial in cats with megacolon⁵ or decreased colonic motility. Intractable cases of megacolon may require colectomy.

Monitoring

Response to treatment can be monitored by having the owner record daily bowel movements and fecal characteristics. Success of treatment is characterized by return of daily bowel movements, absence of straining or pain on defecation, and normal fecal consistency.

Algorithm – Nutritional Management of Feline Constipation



Small Bowel Diarrhea – Canine

Debra L. Zoran, DVM, PhD, DACVIM

Definition

Diarrhea is defined as an increase in the water content, frequency, or volume of feces. *Small bowel diarrhea* is characterized by normal to increased volume of liquid or unformed feces that may be associated with weight loss (chronic) or vomiting, but is not necessarily associated with straining or increased frequency of defecation. If blood is present, it will be digested (melena). Small bowel diarrhea is also characterized by its cause (e.g., infectious, inflammatory, parasitic, mechanical, dietary, neoplastic) or duration (acute or chronic).

Key Diagnostic Tools and Measures

Diagnosis of small bowel diarrhea in dogs begins with a complete history, including dietary history and drug therapy, and physical examination, including rectal examination.

Fecal stream analysis (e.g., fecal flotation, cytology, enzyme-linked immunosorbent assay [ELISA]/polymerase chain reaction [PCR] analysis) is an especially important aspect of the analysis of acute diarrhea in young dogs, but is essential in all dogs due to their scavenging nature and exposure to parasites. In acute diarrhea, symptomatic or supportive therapy may be all that is needed (e.g., highly digestible diet, probiotics, deworming, gastrointestinal [GI] protectants). In chronic (>2 weeks) diarrhea, diagnostic evaluation is necessary to determine the cause, which may include imaging (radiographs or ultrasound), GI function testing (trypsin-like immunoreactivity [TLI], cobalamin, folate), or possibly biopsy (endoscopic or abdominal exploratory).

Pathophysiology

By definition, small bowel diarrhea results from diseases affecting the small intestine; however, small bowel diarrhea can occur in dogs with a large variety of inciting causes, including bacterial or viral infections, parasitic or protozoal infections (*Giardia* or other parasites), mechanical dysfunction (foreign bodies or intussusception), endocrinopathies (Addison's disease), infiltrative diseases (inflammatory bowel disease [IBD], fungal infections, or cancer such as lymphoma), diseases of the lymphatic system (lymphangiectasia), maldigestion of nutrients (exocrine pancreatic insufficiency [EPI]) or dietary sensitivities (food allergy, food intolerance).

Signalment

Acute diarrhea is more common in young dogs due to the increased risk of dietary indiscretion, parasitic infection, or viral diseases such as parvoviral enteritis. Chronic diarrhea is most common in middle-aged or older dogs and may occur due to a variety of dietary, endocrine, inflammatory, or neoplastic causes. German shepherd dogs have an increased incidence of diarrhea caused by EPI or antibiotic-responsive enteritis (also called tylosin-responsive enteritis).

Key Nutrient Modifications

The most important nutrients of concern in dogs with diarrhea are carbohydrates and fat. The goal is to increase digestion and absorption of both nutrients to prevent worsening diarrhea due to disruption of the bacterial flora or the osmotic effects of maldigestion. Undigested fat is also a cause of additional diarrhea via steatorrhea. As a result, ideal diets for diarrhea should contain moderate amounts of highly digestible carbohydrate sources and moderate to very low amounts of fat. The lowest amounts of fat are needed in dogs with lymphangiectasia or other severe

diseases causing a protein-losing enteropathy (PLE).

Cooked white or blended rice is often an ideal carbohydrate source for dogs with intestinal disease because it is highly digestible and does not contain gluten, which may be antigenic in some dogs. Other gluten-free carbohydrate sources are potato, tapioca, and corn, but they are slightly less digestible than rice, and corn may cause hypersensitivities in some dogs.

Protein becomes a major concern in dogs when diarrhea is due to a food allergy or as a result of a disease causing a PLE (e.g., severe IBD, infiltrative diseases such as lymphosarcoma, or lymphangiectasia). In these conditions, dogs may not digest or absorb protein normally and thus become hypoproteinemic (especially low albumin). To prevent protein malnutrition and edema formation, feeding a highly digestible or hydrolyzed protein is often essential to successful management of the disease. In dogs with dietary sensitivity, allergy to protein, rather than the quantity of protein, is the cause of the intestinal inflammation. The key to successful management of dogs with diarrhea caused by food allergy is identifying a novel protein source (or one that is less antigenic, such as a hydrolyzed protein diet) using appropriately planned and executed dietary trials.

Reduced insoluble fiber in the diet is indicated in dogs with small bowel diarrhea, as this type of fiber reduces the digestibility of foods and may increase the risk of maldigestion or malabsorption of nutrients. This is particularly true in dogs with lymphangiectasia or PLE where digestibility of the diet is especially important for the uptake of nutrients. Soluble fiber sources may be beneficial in some dogs as they are digested by the normal flora and may function as prebiotics to help maintain a healthy intestinal flora. Studies in healthy dogs suggest increased numbers of beneficial bacteria using prebiotics or soluble fiber sources, but studies in dogs with small intestinal disease are lacking.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Fat	5–15	1.4–5	5	1.4

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

- Nutrients should be highly digestible (>90% digestibility) to minimize osmotic diarrhea, bacterial fermentation of foods, and reduce intestinal gas.
- Source of medium chain triglycerides (MCTs) as an easily digested and absorbed source of fat.
- Use a high-quality, single-source hydrolyzed protein if IBD or food sensitivity is likely.
- The carbohydrate source should be high quality, gluten-free, and lactose free; sources contributing low protein are beneficial if food sensitivity is likely.
- The diet should contain low fat (less than 5 g/100 kcal at minimum, in dogs with lymphangiectasia, less than 3.5 g/100 kcal if PLE is often needed to achieve successful management of signs).
- Increased omega 3 fatty acids to improve eicosanoid profiles and reduce inflammation in the intestinal mucosa.

- Low insoluble fiber, moderate soluble, or mixed fiber (3–7% total) increases short chain fatty acids and improves bacterial flora.
- Probiotic supplement to restore microflora balance.

■ **Treats** – In general, treats should be avoided in dogs with intestinal disease until a definitive diagnosis is made. For example, if diarrhea is due to food sensitivity, an elimination diet trial will be necessary and this includes treats. If treats are important for the dog’s daily routine, treats made using the therapeutic diet or based on the principles above can be given.

■ **Tips for Increasing Palatability** – If the dog will not eat the suggested diet, a small amount of low-sodium chicken broth can be added to the food. Alternatively, a small amount of the canned version of the dry food can be mixed with the food to increase interest.

■ **Diet Recommendations** – Several veterinary prescription products are available that can be used for this purpose, but the primary theme is that the diets are highly digestible, have reduced or no additives or flavorings that can be associated with food intolerance, are low in insoluble sources of fiber, and have reduced amounts of fat. In severely affected dogs, such as dogs with lymphangiectasia or other severe intestinal diseases affecting absorption, diets containing hydrolyzed protein sources, ultra-low levels of fat, or novel protein sources may be indicated.

Client Education Points

- Feed only the recommended foods.
- Feed small amounts of the food more frequently—three to four times per day— because large amounts of food increase the workload of the

GI tract and may contribute to diarrhea or vomiting.

- Make sure plenty of water is available at all times. If vomiting occurs or the dog stops eating or drinking, a recheck with the veterinarian is recommended to prevent dehydration from the ongoing diarrhea

Common Comorbidities

Conditions that commonly occur concurrently in dogs with small bowel diarrhea include IBD and PLE, IBD and food allergy, and EPI and antibiotic-responsive diarrhea.

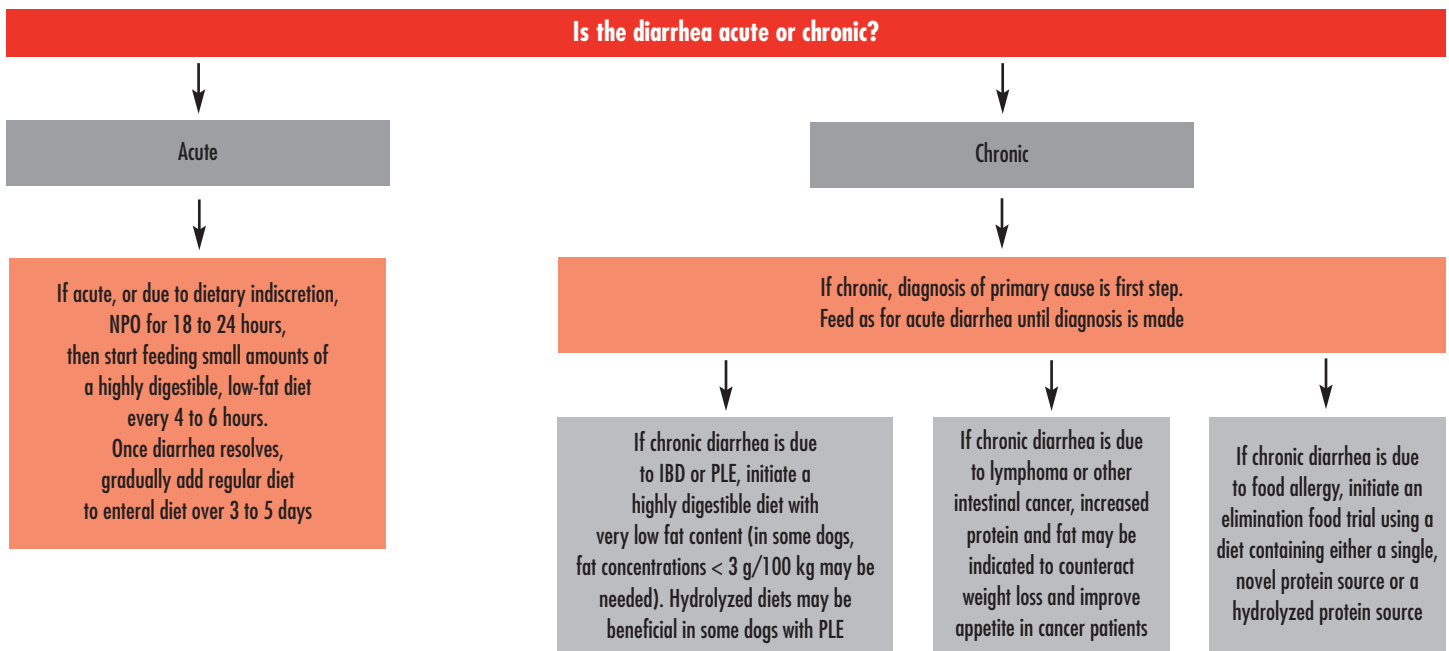
Interacting Medical Management Strategies

Steroid therapy in IBD will increase thirst and appetite and may result in unintended weight gain or hepatopathy. Immunosuppressive therapy for IBD or lymphoma may result in GI toxicity (common clinical signs can be vomiting or diarrhea). Antibiotic therapy may disrupt the bacterial flora and cause diarrhea due to bacterial disruption.

Monitoring

Fecal composition should be assessed to determine if normal stool character is returning or if new problems (e.g., melena, hematochezia) are developing. Assessment of clinical condition is important to be sure the dog is not dehydrated and is continuing to eat, with no new signs of illness (e.g., lethargy, weight loss, reduced or no appetite, or vomiting). If the dog is losing weight or becoming dehydrated, the feeding method and treatment should be re-evaluated and adjusted to the needs of the particular patient.

Algorithm – Nutritional Management of Canine Small Bowel Diarrhea



Small Bowel Diarrhea – Feline

Debra L. Zoran, DVM, PhD, DACVIM

Definition

Diarrhea is defined as an increase in the water content, frequency, or volume of feces. *Small bowel diarrhea* is characterized by normal to increased volume of liquid or unformed feces that may be associated with weight loss (chronic) or vomiting, but is not necessarily associated with straining or increased frequency of defecation. If blood is present, it will be digested (melena). Small bowel diarrhea is also characterized by its cause (infectious, inflammatory, parasitic, mechanical, dietary, neoplastic) or duration (acute or chronic).

Key Diagnostic Tools and Measures

Diagnosis of small bowel diarrhea in cats begins with a complete history, including dietary and drug history and other risk factors (such as environment, age, previous problems), and physical examination, including assessment of hydration, body condition, and careful palpation of abdomen. If indicated, rectal examination should be performed under sedation. Fecal stream analysis (fecal flotation, cytology, enzyme-linked immunosorbent assay [ELISA]/polymerase chain reaction [PCR] analysis) is especially important in young or indoor/outdoor cats. In acute diarrhea, symptomatic or supportive therapy may be all that is needed (e.g., highly digestible diet, probiotics, deworming). In chronic (>2 weeks) diarrhea, imaging (radiographs or ultrasound), GI function testing (trypsin-like immunoreactivity [TLI], cobalamin, folate), endocrine testing (thyroid), viral screening (FeLV/FIV), testing for specific infections (e.g., fungal) or endoscopy/surgery with biopsy may be indicated.

Pathophysiology

By definition, small bowel diarrhea results from diseases affecting the small intestine; however, small bowel diarrhea can occur in cats with a large variety of inciting causes, including antibiotic-responsive diarrhea, viral infections, parasitic or protozoal infections (*Giardia*, coccidian, *Cryptosporidia*, *Tritrichomonas*, or other parasites), endocrinopathies (hyperthyroidism), mechanical dysfunction (foreign bodies or intussusception), infiltrative diseases (inflammatory bowel disease, fungal infections, or cancer such as lymphoma), maldigestion of nutrients (exocrine pancreatic insufficiency [EPI]) or dietary sensitivities (food allergy, food intolerance).

Signalment

Acute diarrhea is more common in young cats due to the increased risk of dietary indiscretion (eating string, foreign bodies), parasitic infection, or if from a non-closed colony, viral diseases such as feline immunodeficiency virus (FIV), feline infectious peritonitis (FIP), or panleukopenia. Chronic diarrhea is most common in middle-aged or older cats and may occur due to a variety of dietary, endocrine, inflammatory, or neoplastic causes, including hyperthyroidism, lymphoma of the intestinal tract, inflammatory bowel disease (IBD), food allergy or other food sensitivities, and bacterial, fungal, or protozoal infections. There are no specific breed predispositions for IBD or lymphoma, but purebred cats are more likely to have FIP, *Tritrichomonas*, or other diseases of multiple-cat households or colonies.

Key Nutrient Modifications

Protein is a particular concern in feline diets because of the increased need for protein, and of all of the nutrients present in feline diets, the protein digestibility is the most affected by quality and preparation. Even in healthy cats, protein of poor quality or sub-standard processing is less digestible

and will result in a larger amount of this nutrient reaching the large bowel, where bacterial digestion of these undigested proteins will result in significant decline in fecal quality (increased water, odor) and increased production of by-products (e.g., phenols) that may be harmful to colonocytes. The end result is development of poor fecal quality simply by the presence of reduced protein quality. Thus, a major first step in the management of feline diarrhea is assuring a highly digestible, good quality protein source in the diet. Protein digestibility is even more important in senior cats (>10 years of age) that have reduced ability to digest and absorb nutrients simply because of their increased age and the changes that occur in their digestive functions. Finally, in cats with significant GI disease that may have abnormal enzyme function or absorptive capacity due to the disease process, the protein digestibility of the diet has an even more profound effect.

The protein source becomes a particular concern when feline diarrhea is suspected to be due to a dietary allergy, intolerance, or to IBD. In one study,³ nearly 60% of cats with diarrhea responded to a diet change, and of those cats 20% to 25% had food allergy. This study was the first to illustrate the importance of diet in the management of feline GI disease, and emphasized that all dietary responses were not food allergy. It is key to remember that in cats with a food allergy, the protein itself is the source of the intestinal inflammation, while in a food intolerance results in development of GI disturbance due to any part of the food (e.g., nutrient, additive, preservative) that causes disrupted function (e.g., maldigestion of nutrients, release of histamine or other reactions). Successful diagnosis and management of cats with diarrhea due to food allergy requires identification of an appropriate novel or hydrolyzed protein source, a process which requires a carefully performed food trial of appropriate duration (8–12 weeks in some cats). Cats with a food intolerance will often respond to a change to a diet containing highly digestible nutrients without additional additives, preservatives, or foods that are known to be potential sources of intolerance (e.g., excessive complex carbohydrates, wheat or other grains containing gluten, lactose).

In normal cats, fat is a highly digestible food component that is not likely to be associated with malabsorption. This fact was supported by a recent study by Laflamme and others which showed that the concentration of fat in the diet was not important in cats with chronic, nonspecific diarrhea.⁶ As long as the diet contained increased amounts of highly digestible protein, 55% to 60% of the cats improved. Based on the results of that study, diets for cats with diarrhea should contain moderate to increased amounts of highly digestible protein and moderate to very low amounts of highly digestible carbohydrate. Because cats have specific requirements for increased amounts of certain fats in the diet, and apparently are not as sensitive to the presence of higher fat amounts in their diets, these studies suggested that diets containing reduced amounts of fat were not necessary (as is the case in many dogs with intestinal disease), and may result in less acceptance of the food, considering that fat is the primary palatability enhancer in cat foods.

The role of carbohydrates (CHO) in feline diets is receiving increased scrutiny as more information on the affect of this readily available energy source on feline GI function and overall metabolism is gained. Cooked white or blended rice or potato are frequently used CHO sources for cats with intestinal disease because they are highly digestible sources of energy and do not contain gluten, which may be antigenic in some cats. In healthy cats, these CHO may be acceptable when fed in small quantities; however, if CHO intolerance or significant infiltrative disease is suspected that would affect CHO digestibility and lead to CHO malabsorption and its attendant adverse effects, reduction of CHO in the diet to less than 2 to 3 g/100 kcal (less than 15% CHO) is a reasonable approach.

Reduced insoluble fiber in the diet is indicated in cats with small bowel diarrhea as this type of fiber reduces the digestibility of foods and may increase the risk of maldigestion of nutrients as well as reduced intake (lower palatability). Soluble fiber sources may be beneficial in some cases as these types of fiber are digested by the normal flora and may function as prebiotics that help maintain a healthy intestinal flora. Most studies using prebiotics, however, have been used in normal cats with no signs of GI disease; thus their effectiveness in cats with small bowel diarrhea is unknown.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Crude fiber [#]	7–16	2.0–5.0	n/a	n/a
Fat	10–15	3–5	5	1.4

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]Sources should include both soluble and insoluble fibers. The crude fiber analysis includes most insoluble fibers, but does not include soluble fibers. Therefore, crude fiber has limited usefulness when evaluating the total fiber content of foods. The ingredient list should be evaluated for sources of soluble fiber.

Therapeutic Feeding Principles

- Nutrients should be highly digestible (>90% digestibility) to minimize osmotic diarrhea, bacterial fermentation of protein or CHO, and reduce production of intestinal gas.
- High-quality, highly digestible protein (can be single source and novel or hydrolyzed if IBD or food sensitivity is likely).
- Carbohydrate source is high quality, gluten-free, and lactose free, and in most cats, low quantity (<15% of overall diet).
- Low-fat diets appear not to be necessary in cats with diarrhea, unless the intestinal disease is severe and steatorrhea is observed.
- Increased omega-3 fatty acids to improve eicosanoid profiles in the intestinal mucosa.
- Low insoluble fiber, low to moderate soluble or mixed fiber (3–5% total) to increase short-chain fatty acids and improve bacterial flora.
- Probiotic supplement to restore microflora balance.

■ **Treats** – In general, treats should be avoided in cats with intestinal disease until a definitive diagnosis is made. For example, if diarrhea is due to food sensitivity, an elimination diet trial will be necessary and this includes treats. However, treats can be prepared from the therapeutic diet or based on the principles above if deemed necessary.

■ **Tips for Increasing Palatability** – In general, food preference and mouth feel are the first aspects of feeding cats that must be considered. Cats will not typically consume foods that are different from their normal (e.g., dry food eaters will not eat canned) and they have very specific preferences for flavors, odors, and temperature. Most cats prefer room temperature canned

food or slightly warm (think dead mouse). Fat is a palatability enhancer for cats, so switching to a food with more fat may be necessary to get the cat to consume the therapeutic diet recommended, or adding a small amount of animal fat (not plant oil) to the food may increase acceptance. Force feeding cats is to be avoided as it can cause food aversion

■ **Diet Recommendations** – Diets that may be selected for cats with diarrhea can be a highly digestible moderate protein profile, or a highly digestible high protein/low carbohydrate profile. A number of novel antigen diets are available for cats with food allergy or IBD; protein sources include venison, lamb, duck, rabbit, or fish. Only two hydrolyzed diets currently are available in the US for cats. A probiotic nutritional supplement has been shown to be effective in restoring normal intestinal health and balance.

OTC products that are suitable for nonspecific diarrhea (high protein, low carbohydrate) included canned foods in several categories.

Client Education Points

- Feed only the recommended foods.
- Feed small amounts of the food more frequently—three to four per day—large amounts of food increase the workload of the GI tract and may contribute to diarrhea or vomiting.
- Make sure plenty of water is available at all times. If vomiting occurs or the cat refuses the diet, stops drinking, or is more lethargic, a recheck with your veterinarian is recommended.

Common Comorbidities

Conditions that commonly occur concurrently in cats with small bowel diarrhea include IBD and lymphoma, IBD and food allergy, hyperthyroidism and diarrhea, and FIV or other viral infections and diarrhea in young or cattery cats.

Interacting Medical Management Strategies

Steroid therapy in IBD will increase appetite and may result in development of diabetes (especially obese cats) or secondary infections. Immunosuppressive therapy for IBD or lymphoma may result in GI toxicity (common clinical signs can be vomiting or diarrhea). Antibiotic therapy may disrupt the bacterial flora and cause diarrhea due to bacterial flora disruption. Treatment with methimazole can cause GI problems (vomiting or diarrhea).

Monitoring

Fecal composition should be assessed to determine if normal stool character is returning or if new problems (e.g., melena, hematochezia) are developing. Assessment of clinical condition is important to be sure the cat is not dehydrated and is continuing to eat, with no new signs of illness (e.g., lethargy, weight loss, reduced or no appetite, or vomiting). If the cat is losing weight or becoming dehydrated, the feeding method and treatment should be re-evaluated and adjusted to the needs of the particular patient.

See Algorithm – Nutritional Management of Feline Small Bowel Diarrhea on page 50.

Large Bowel Diarrhea – Canine

Debra L. Zoran, DVM, PhD, DACVIM

Definition

Diarrhea is defined as an increase in the water content, frequency, or volume of feces. *Large bowel diarrhea*, however, is classically noted to be associated with increased tenesmus or urgency with defecation, increased frequency of defecation of usually smaller volumes, and hematochezia or increased mucus may be present. These characteristics serve to differentiate large bowel diarrhea from small bowel diarrhea. Large bowel diarrhea can also be characterized by cause: infectious (clostridial colitis), parasitic (whipworm colitis), dietary (fiber-responsive colitis), inflammatory (lymphoplasmacytic colitis or inflammatory bowel disease [IBD]), and neoplasia. Conditions causing large bowel diarrhea often are associated with inflammation and thus are termed *colitis*.

Key Diagnostic Tools and Measures

Diagnosis of large bowel diarrhea begins with a complete history, including dietary history and drug therapy, and physical examination, including rectal examination. Fecal stream analysis (e.g., fecal flotation, cytology, enzyme-linked immunosorbent assay [ELISA]/polymerase chain reaction [PCR] analysis) or therapeutic deworming is very important; whipworms are especially difficult to find and are important causes of signs of colitis. In acute large bowel diarrhea, symptomatic or supportive therapy is often all that is needed (e.g., added dietary fiber, probiotics, deworming, or possibly motility modifiers). In dogs with chronic (>2 weeks) large bowel diarrhea where symptomatic or supportive therapy is not effective in controlling the clinical signs, imaging (radiographs or ultrasound), more specific tests for gastrointestinal (GI) parasites or bacteria, or endoscopy (with biopsy) are indicated.

Pathophysiology

The clinical signs of large bowel diarrhea are a reflection of proximity of the disease to the end of the GI tract. As a result the clinical signs of colon diseases are all quite similar, while their inciting causes may be quite different. For example, hematochezia occurs instead of melena because the blood is not in the tract long enough to be digested by bacteria or enzymes, increased mucus is often present in the feces due to the increased number of mucus-secreting glands in the colon which increase their production when the epithelium is disrupted, and increased straining or frequency of defecation occurs due to disrupted colonic motility reducing the storage time for feces to form normal-sized, reduced-water feces. Thus, while large bowel diarrhea can occur in dogs due to a large variety of inciting causes, the response to the disruption of the mucosa in the colon, regardless of cause, is essentially the same.

Signalment

Acute large bowel diarrhea is more common in young or middle-aged dogs due to the increased risk of dietary indiscretion, parasitic infection, or infectious causes such as clostridial colitis from dietary changes or boarding. Small breeds of dogs may be at a greater risk of developing stress colitis or irritable bowel syndrome, although this condition can occur in any breed. Chronic large bowel diarrhea is most common in middle-aged or older dogs and may occur in any breed due to a variety of dietary, inflammatory, or neoplastic causes including colonic IBD or various forms of colon cancer. Boxer dogs have an increased incidence of histiocytic ulcerative colitis (HUC), a specific type of antibiotic-responsive IBD due to bacterial overgrowth in this breed.

Key Nutrient Modifications

The most important dietary modifications in dogs with large bowel diarrhea are to provide highly digestible nutrients so that excess carbohydrates, fat, and protein do not reach the colon undigested, and second, to modify (increase) the amount and type of dietary fibers and prebiotics to maximize colonic epithelial and bacterial health.

The goal for providing a diet with highly digestible nutrients (>85–90% digestible) is to maximize digestion and absorption of carbohydrates and fats in the small bowel to prevent an exacerbation of large bowel diarrhea due to bacterial disruption or the osmotic effects of maldigestion of carbohydrates (CHO). As a result, ideal diets for large bowel diarrhea should contain moderate amounts of highly digestible CHO sources and moderate to low amounts of fat. Cooked white or blended rice or potatoes are often ideal CHO sources for dogs with intestinal disease because they are highly digestible and do contain gluten or other potential sources of antigenic stimulation. Other gluten-free CHO sources are tapioca and corn, but they are slightly less digestible than rice, and corn may cause hypersensitivities in some dogs.

Protein becomes a concern when diarrhea is suspected due to a food allergy. Most dogs with food allergy have both large and small bowel diarrhea, vomiting, or cutaneous manifestations of allergic disease. The key to successful management of dogs with diarrhea due to food allergy is identifying a novel protein source (or one that is less antigenic, such as a hydrolyzed protein diet).

In some dogs with large bowel diarrhea, the single most important dietary modification may be the addition of dietary fiber to the diet. Dietary fibers are complex carbohydrates primarily from plant sources that are not easily digested by mammalian digestive enzymes. Digestion of these foods is accomplished by the help of bacteria in the GI tract, and most efficiently occurs in the colon of dogs. Because different types of dietary fiber are digested (also called *solubility* or *fermentability*) more or less efficiently by bacteria, they have often been classified by this characteristic. It is important to realize, however, that many dietary fibers have characteristics of both groups and thus are termed *mixed fiber*.

Soluble (or highly fermentable) fiber sources are beneficial in dogs with colonic disease because they are broken down into short-chain fatty acids (an essential nutrient source for colonocytes) and also serve as a nutrient source for beneficial bacteria (also known as a *prebiotic*). Addition of soluble fiber sources has been shown to increase the numbers of beneficial bacteria and reduce pathogenic bacteria, a key issue when disruption of the normal environment occurs, no matter what the inciting cause. Soluble fiber sources cannot be added in large amounts, however, as they are highly fermentable and will cause increased flatus and fecal water content.

Insoluble (or poorly fermentable) fiber sources are also beneficial in dogs with colonic disease, but for very different reasons. Insoluble fibers increase fecal bulk and, as a result of the stretching and distention, improve motility (both normal segmentation as well as propulsion) in the colon. The result of adding insoluble fiber to diets is to decrease frequency and straining associated with aberrant motility in colitis; the disadvantage is that they do not provide a nutrient source for fecal bacteria or colonocytes, which is important in dogs with severe diseases of the colon.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Crude fiber [#]	7–16	2.0–5.0	n/a	n/a
Fat	10–15	3–5	5	1.4

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle and energy intake. *Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]Sources should include both soluble and insoluble fibers. The crude fiber analysis includes most insoluble fibers, but does not include soluble fibers. Therefore, crude fiber has limited usefulness when evaluating the total fiber content of foods. The ingredient list should be evaluated for sources of soluble fiber.

Therapeutic Feeding Principles

- Key nutrients should be highly digestible (>90% digestibility) to minimize osmotic diarrhea, bacterial fermentation of undigested foods, and reduce intestinal gas.
 - Use a high-quality, single-source hydrolyzed protein if IBD or food sensitivity is likely, but in most cases of colitis this is not necessary.
 - Moderate to increased amounts of insoluble fiber are indicated to improve colonic motility, unless constipation or colonic obstruction occurs due to cancer or stricture.
 - Addition of a mixed or soluble fiber source is indicated to improve colonic epithelial cell health and normalize bacterial populations disrupted by disease or therapy. The optimum ratio of soluble and insoluble fibers in diets for colon disease is debated; however, addition of both fiber sources is generally accepted to be ideal.
 - Increased omega-3 fatty acids to help reduce eicosanoids associated with intestinal inflammation.
 - Moderate levels of dietary fat (should be high digestible).
 - Probiotic supplement to restore microflora balance.
- **Treats** – In general, treats should be avoided in dogs with intestinal disease until a definitive diagnosis is made. If treats are important for the dog's daily routine, treats made using the therapeutic diet or based on the principles above can be given.
- **Tips for Increasing Palatability** – If the dog will not eat the suggested diet, a small amount of low-sodium chicken broth can be added to the food. Alternatively, a small amount of the canned version of the dry food can be mixed with the food to increase interest. If the dog refuses to consume the therapeutic diet, a mixed-fiber source such as psyllium mucocolloid (Metamucil®, Proctor & Gamble) can be added to the dog's usual diet.
- **Diet Recommendations** – Diets used for large bowel diarrheas can be one of two dietary types: 1) diets containing highly digestible ingredients as is typical of small bowel diarrhea, or 2) diets containing increased amounts of dietary fiber. Diets containing increased dietary fiber can be either primarily insoluble (bulking) fiber diets or mixed (both soluble and

insoluble) fiber diets. Addition of a probiotic nutritional supplement has been shown to be effective in restoring normal intestinal health and balance.

Several OTC diets are potentially suitable for dogs with large bowel diarrhea, as they contain increased amounts of insoluble fiber – these diets are typically classified as weight management diets. The individual diets may contain insoluble or mixed fiber sources, so the label must be evaluated to determine the fiber source.

Client Education Points

- Feed only the recommended foods for the time recommended.
- It may be helpful to feed small amounts of the food more frequently—three to four times per day—because large amounts of food increase the workload of the GI tract and may contribute to clinical signs; however, this is not universally true for all dogs.
- Make sure plenty of water is available at all times; adding fiber to the diet may cause feces to become too dry and hard to pass in some dogs.
- Counsel owners on the effects of adding dietary fiber to the diet: insoluble fiber will increase fecal volume, while soluble fibers generally contribute to a softer, smaller stool, but may be associated with more flatus.

Common Comorbidities

Conditions that commonly occur concurrently in dogs with large bowel diarrhea include colonic IBD and bacterial overgrowth, and clostridial colitis and recent boarding or diet change.

Interacting Medical Management Strategies

Steroid therapy in IBD will increase thirst and appetite and may result in unintended weight gain or hepatopathy. Immunosuppressive therapy for IBD or lymphoma may result in GI toxicity (common clinical signs can be vomiting or diarrhea). Nonsteroidal anti-inflammatory therapy for colonic IBD (e.g., sulfasalazine) may result in sulfonamide toxicity (i.e., dry eye, liver or bone marrow disease, immune-mediated diseases such as immune-mediated thrombocytopenia, immune-mediated hemolytic anemia). Antibiotic therapy may disrupt the bacterial flora and cause worsening diarrhea due to bacterial overgrowth.

Monitoring

Fecal composition should be assessed to determine whether normal stool character is returning or if new problems are developing. Assessment of clinical condition is important to be sure the dog is not dehydrated and is continuing to eat, with no new signs of illness (e.g., lethargy, weight loss, reduced or no appetite, or vomiting). If the dog is losing weight or becoming dehydrated, the treatment should be re-evaluated and adjusted to the needs of the particular patient.

See [Algorithm – Nutritional Management of Canine Large Bowel Diarrhea on page 51](#).

Large Bowel Diarrhea – Feline

Debra L. Zoran, DVM, PhD, DACVIM

Definition

Large bowel diarrhea is classically noted to be associated with increased tenesmus or urgency with defecation, increased frequency of defecation of usually smaller volumes, and hematochezia or increased mucus may be present – these characteristics differentiate large bowel diarrhea from that of small bowel character. Large bowel diarrhea can also be characterized by cause: infectious (clostridial colitis), parasitic (whipworm colitis), dietary (fiber-responsive colitis), inflammatory (lymphoplasmacytic colitis or inflammatory bowel disease [IBD]), and neoplasia. Conditions causing large bowel diarrhea often are associated with inflammation and thus termed *colitis*.

Key Diagnostic Tools and Measures

Diagnosis of large bowel diarrhea begins with a complete history, including dietary history and drug therapy, and physical examination, including rectal examination. Fecal stream analysis (e.g., fecal flotation, cytology, enzyme-linked immunosorbent assay [ELISA]/polymerase chain reaction [PCR] analysis) or therapeutic deworming is important. In acute large bowel diarrhea: symptomatic or supportive therapy is often all that is needed (e.g., added dietary fiber, probiotics, or deworming). In cats with chronic (>2 weeks) large bowel diarrhea where symptomatic or supportive therapy is not effective, imaging (radiographs or ultrasound), more specific tests for gastrointestinal (GI) parasites or bacteria, or endoscopy (with biopsy) are indicated.

Pathophysiology

The clinical signs of large bowel diarrhea are a reflection of proximity of the disease to the end of the GI tract. As a result the clinical signs of colon diseases are all quite similar, while their inciting causes may be quite different. For example, hematochezia occurs instead of melena because the blood is not in the tract long enough to be digested by bacteria or enzymes, increased mucus is often present in the feces due to the increased number of mucous secreting glands in the colon which increase their production when the epithelium is disrupted, and the observation of increased straining or frequency of defecation occurs due to irritation of colonic epithelium or disrupted colonic motility reducing the storage time for feces to form normal sized, reduced water feces. Thus, while large bowel diarrhea can occur in cats due to a large variety of inciting causes, the response to the disruption of the mucosa in the colon is essentially the same.

Signalment

Acute large bowel diarrhea is more common in young or middle-aged cats due to the increased risk of dietary indiscretion (feathers, bones or other foreign objects), parasitic or protozoal (*Tritrichomonas*) infection, or infectious causes such as clostridial colitis. Long-haired breeds of cats have an increased incidence of hair-induced colitis – a condition that occurs presumably due to irritation of the colon from passage of large amounts of hair. Chronic large bowel diarrhea is most common in middle aged or older cats and may occur in any breed due to a variety of dietary, inflammatory or neoplastic causes including colonic IBD or various forms of colon cancer. Siamese cats appear to be at an increased risk of development of colonic adenocarcinoma.

Key Nutrient Modifications

The most important dietary modifications in cats with large bowel diarrhea are to provide highly digestible nutrients so that excess carbohydrates, fat and protein do not reach the colon undigested, and secondly, to increase the concentration of dietary fibers to maximize colonic epithelial and bacterial health.

The goal for providing a diet with highly digestible nutrients (> 85–90% digestible) is to maximize digestion and absorption of carbohydrates and fats in small bowel to prevent an exacerbation of large bowel diarrhea due to bacterial overgrowth or the osmotic effects of maldigestion of carbohydrates.

Protein becomes a concern when diarrhea is suspected to be due to a food allergy. The key to successful management of cats with diarrhea due to food allergy is identifying a novel protein source (or one that is less antigenic, such as a hydrolyzed protein diet). It is a rare occurrence for cats with food allergy to have only large bowel diarrhea, and no signs of small bowel disease (vomiting or diarrhea) or dermatologic signs of allergy.

In cats with large bowel diarrhea, the single most important dietary modification may be the addition of dietary fiber to the diet. Dietary fibers are complex carbohydrates primarily from plant sources that are not easily digested by mammalian digestive enzymes. Digestion of these foods is accomplished by the help of bacteria in the GI tract, and most efficiently occurs in the colon of cats. Because different types of dietary fiber are digested (also called solubility or fermentability) more or less efficiently by bacteria, they have often been classified by this characteristic. It is important, however, to realize that many dietary fibers have characteristics of both groups and thus are termed *mixed fiber*.

In general, soluble (or highly fermentable) fiber sources, which are those fibers that are readily broken down by bacteria to form short chain fatty acids, water and gases, are beneficial in cats with colonic disease for the same reasons that they are in dogs. The soluble fibers, however, must be added in small amounts to feline diets because the increased fecal bacteria results in increased flatus and fecal water content resulting in undesirable stool characteristics and odors.

Insoluble (or poorly fermentable) fiber sources are also beneficial in cats with colonic disease, but for very different reasons. Insoluble fibers increase fecal bulk and, as a result of the stretching and distention, improve motility (both normal segmentation as well as propulsion) in the colon. The result of adding insoluble fiber to diets is to decrease frequency and straining associated with aberrant motility in colitis and they are helpful in moving hair through the colon more effectively – reducing hair-induced colitis or obstruction. The disadvantage of insoluble fiber sources is that they do not provide a nutrient source for fecal bacteria or colonocytes – and in cats that do not consume enough water, may produce feces that is too dry and more difficult to pass – creating the possibility of development of constipation, which is a frequent complication of high insoluble fiber diets in cats.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Crude fiber [#]	2–8	0.5–2.5	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]Sources should include both soluble and insoluble fibers. The crude fiber analysis includes most insoluble fibers, but does not include soluble fibers. Therefore, crude fiber has limited usefulness when evaluating the total fiber content of foods. The ingredient list should be evaluated for sources of soluble fiber.

Therapeutic Feeding Principles

- Key nutrients should be highly digestible (>90% digestibility) to minimize osmotic diarrhea, bacterial fermentation of undigested foods, and reduce intestinal gas.
- High quality, single source hydrolyzed protein if IBD or food sensitivity is likely, but in most cases of colitis this is not necessary.
- Moderate to increased amounts of insoluble fiber are indicated to improve colonic motility, unless constipation or colonic obstruction occurs due to cancer or stricture.
- The optimum ratio of soluble and insoluble fibers in diets for colon disease is debated, however, addition of small amounts both fiber sources is generally accepted to be ideal.
- Increased omega 3 fatty acids to help reduce eicosanoids associated with intestinal inflammation.
- Probiotic supplement to restore microflora balance.

■ **Treats** – In general, treats should be avoided in cats with intestinal disease until a definitive diagnosis is made. If treats are important for the cat's daily routine, treats made using the therapeutic diet or based on the principles above can be given.

■ **Tips for Increasing Palatability** – If the cat will not eat the suggested diet, a small amount of low sodium chicken broth can be added to the food. Alternatively, a small amount of the canned version of the dry food can be mixed with the food to increase interest. If the cat refuses to consume the therapeutic diet, a mixed fiber source such as psyllium mucocolloid (Metamucil[®]) can be added to the usual diet to increase the fiber content.

■ **Diet Recommendations** – Therapeutic diets suitable for cats with large bowel diarrhea may include diets that have highly digestible ingredients that reduce the amount of ingesta reaching the colon, or can include diets with increased insoluble dietary fibers present. Whether or not fiber will be an effective therapy depends on the individual situation, as some cats consuming high fiber diets develop hard, dry feces and constipation. In

these situations, the diet should be changed to a low fiber diet, as development of this problem indicates fiber intolerance. A probiotic nutritional supplement has been shown to be effective in restoring normal intestinal health and balance.

Several OTC diets are potentially suitable for cats with large bowel diarrhea. These diets contain added insoluble fiber, and most are marketed as either formulas for management of weight or hairballs. Most cat formulas that contain added fiber have insoluble fiber as the primary fiber source; however, some foods contain mixed fiber sources, and the practitioner must examine the label to determine the fiber source.

Client Education Points

- Feed only the recommended foods for the time recommended.
- It may be helpful to feed small amounts of the food more frequently, 3 to 4 times a day, as with small bowel diarrhea, large amounts of food increase the workload of the GI tract and may contribute to clinical signs; however, this is not universally true for all cats.
- Make sure plenty of water is available at all times; adding fiber to the diet may cause feces to become too dry and hard to pass in some cats. The best way to increase water consumption in cats is to feed canned foods.
- Counsel owners on the effects of adding dietary fiber to the diet: insoluble fiber will increase fecal volume, while soluble fibers generally contribute to a softer, smaller stool, but may be associated with more flatus.

Common Comorbidities

Colonic IBD and bacterial overgrowth, and clostridial colitis and recent boarding or diet change, are common comorbidities in cats with large bowel diarrhea.

Interacting Medical Management Strategies

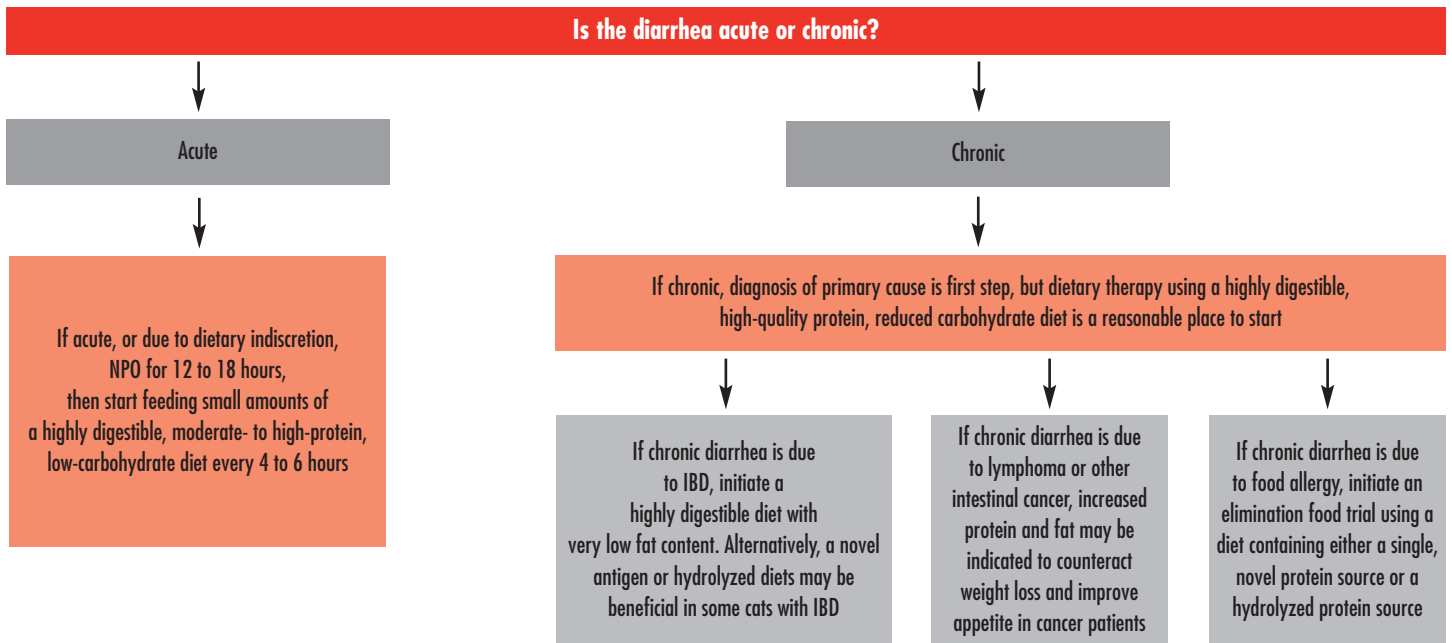
Steroid therapy in IBD will increase thirst and appetite and may result in unintended weight gain or hepatopathy. Immunosuppressive therapy for IBD or lymphoma may result in GI toxicity (common clinical signs can be vomiting or diarrhea). Antibiotic therapy may disrupt the bacterial flora and cause worsening diarrhea due to bacterial overgrowth

Monitoring

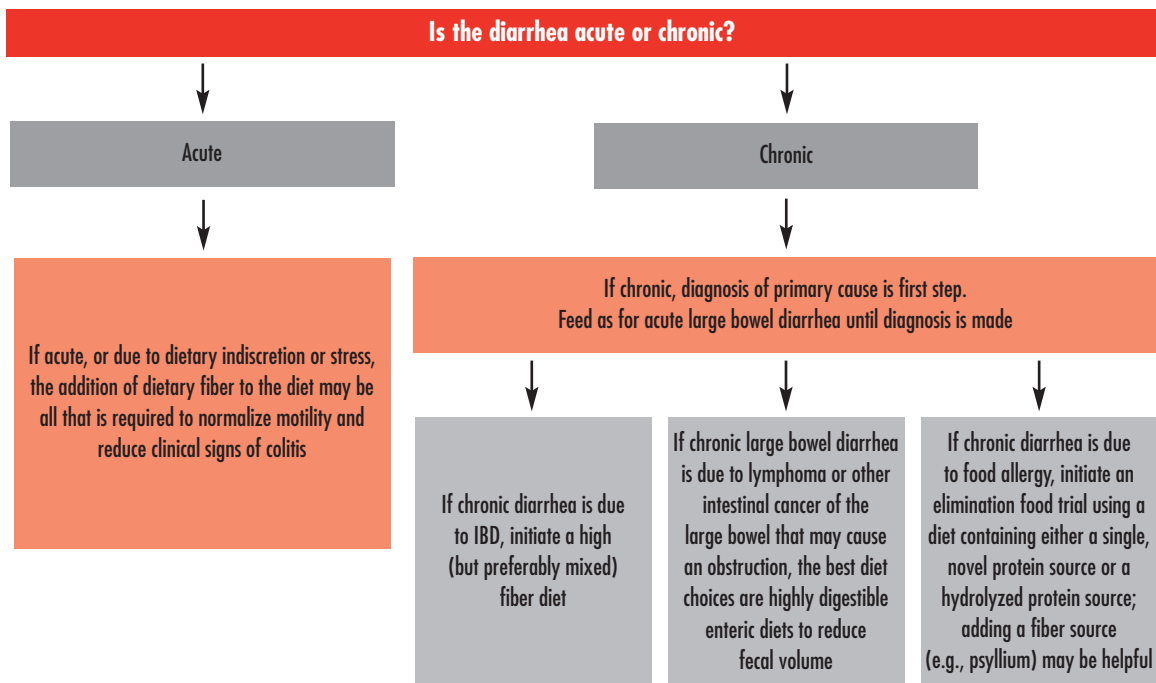
Fecal composition should be assessed to determine if normal stool character is returning or if new problems are developing. Assessment of clinical condition is essential to be sure the cat is not dehydrated and is continuing to eat, with no new signs of illness (e.g., lethargy, weight loss, reduced or no appetite, or vomiting). If the cat is losing weight or becoming dehydrated, the treatment should be reevaluated and adjusted to the needs of the particular patient.

See [Algorithm – Nutritional Management of Feline Large Bowel Diarrhea on page 51](#).

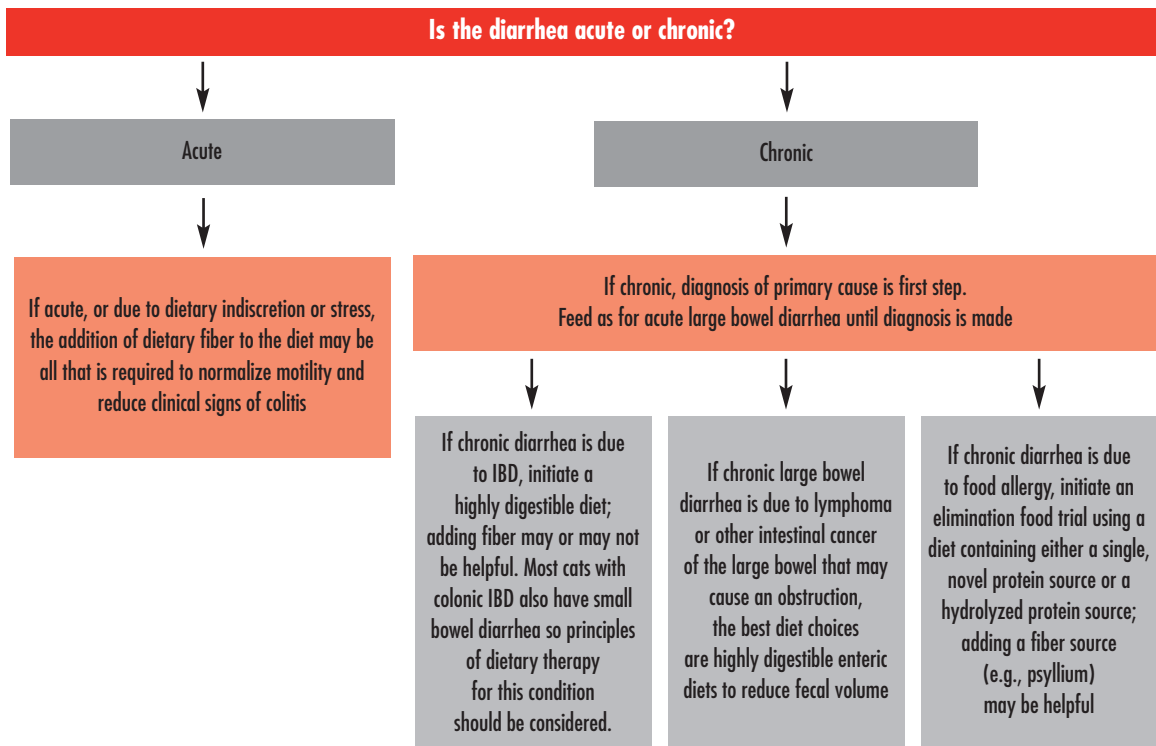
Algorithm – Nutritional Management of Feline Small Bowel Diarrhea



Algorithm – Nutritional Management of Canine Large Bowel Diarrhea



Algorithm – Nutritional Management of Feline Large Bowel Diarrhea



Colitis – Canine

Scott Campbell, BVSc, MACVSc, DACVN

Definition

Colitis is an inflammation of the colon that impairs absorption of water and electrolytes and results in tenesmus, dyschezia, hematochezia, mucoid feces, diarrhea, and/or constipation. Colitis may be caused by parasitic, fungal, or clostridial infection, neoplasia, or may be idiopathic.

Key Diagnostic Tools and Measures

History, physical examination, fecal analysis, hematology, and serum biochemistry are routinely indicated. Colonoscopy and biopsy, and occasionally full-thickness biopsies via laparotomy, are often necessary for diagnosis. A high-fiber, highly digestible, or limited-antigen diet can be essential to manage clinical signs.

Pathophysiology

The pathophysiology of colitis is multifactorial and dependent on etiology. Electrolyte and water absorption by the inflamed mucosa is impaired and active secretion of electrolytes may also occur. Colonic motility is typically compromised and mucous secretion amplified by an inciting pathogen or secondary to inflammation itself. Advanced inflammation may result in colonic erosion and ulceration and more severe clinical signs.

Signalment

Inflammatory bowel disease (IBD) is most common in middle-aged dogs. German shepherd dogs are over-represented but IBD is reported in most dog breeds as well as mixed breeds. An uncommon form of colitis, *histiocytic ulcerative colitis*, occurs mostly in young boxer dogs less than two years of age.

Key Nutrient Modifications

Most dogs affected with colitis maintain appetite and body weight. As such, modification of key dietary nutrients aims to reduce or abolish clinical signs. Depending on the etiology of colitis in an individual dog, a high-fiber, highly digestible, or limited-antigen diet may be beneficial.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Crude Fiber#	7–16	2.0–5.0	n/a	n/a

Modified fiber intake may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Individual dogs may have improvement in clinical signs when fed a diet high in crude fiber, a diet that is highly digestible, or a diet that has limited ingredients novel to the individual or hydrolyzed protein sources.

Therapeutic Feeding Principles

As most dogs with colitis maintain appetite and body weight, the principal aim of dietary modification is to reduce the clinical signs of tenesmus, dyschezia, bloody mucoid feces, diarrhea, and/or constipation. This is achieved by increasing the digestibility of the diet, increasing the fiber

content of the diet, or minimizing dietary antigens.

A highly digestible diet attenuates the clinical signs of colitis by limiting the volume of ingesta delivered to the compromised colon.

Dietary fiber is metabolized by colonic flora to short-chain fatty acids (acetate, butyrate, and propionate) and this provides a direct energy source to damaged colonocytes. By binding luminal water, increased dietary fiber content favors normalization of colonic motility. Dietary fiber also protects the mucosa from contact with physical irritants in bile acids and ingested material to limit colonocyte injury. These effects of dietary fiber also restrict the virulence mechanisms of *Clostridium perfringens*, which has been implicated in some cases of colitis.

Colon inflammation may be caused or exacerbated by dietary antigens; therefore, dietary restriction to a single novel protein or hydrolyzed protein effects clinical improvement in some dogs. Identification of these individuals requires a trial elimination diet whereby this new diet is fed solely for at least 4 weeks before clinical improvement may be expected.

Studies in human patients suggest that antioxidants, omega-3 polyunsaturated fatty acids, fructo-oligosaccharides, prebiotics, and probiotics may be beneficial in the dietary management of colitis.

■ **Treats** – While treats can be important in maintaining the human-animal bond, treats that differ at all in composition from the primary diet should be avoided during initial assessment phase so that efficacy of the base diet alone can be accurately evaluated. Dogs found to respond to high-fiber diets may then be offered high-fiber treats such as vegetables. Dogs that are managed with a highly digestible diet may be fed many treats also containing highly digestible ingredients. Animals fed uncommon/limited-ingredient diets should only be fed treats containing the same ingredients as the base diet. Likewise, dogs receiving hydrolyzed-protein diets should only be fed treats containing similar hydrolyzed ingredients. Canned forms of any base diet can be sliced and baked to form cookies for treats. Alternatively, some of the primary diet may be offered outside regular feeding times and utilizing alternative feeding methods as a treat. Affection and attention can be provided as a substitute for food treats. If treats are given, they should be incorporated slowly, with consistency maintained in type of treat given each day, and the dog should be monitored closely for recurrence of colitis signs. As always, it is suggested that all treats and supplements supply less than 10% of the total daily calories.

■ **Tips for Increasing Palatability** – The majority of dogs with colitis maintain an excellent appetite and diets suitable for managing colitis do not typically lack palatability. If a particular commercial preparation is not accepted, the dog may find other comparable diets suitably tempting. Alternatively, warming the food to body temperature or adding a sweetener such as corn syrup may increase palatability. Appetite stimulants or assisted-feeding devices are occasionally necessary in patients in which persistent anorexia precludes necessary caloric intake.

■ **Diet Recommendations** – A number of highly digestible, high-fiber, uncommon/limited-ingredient, and hydrolyzed-protein diets are available from the major therapeutic diet manufacturers in the United States. If uncommon/limited-ingredient diets are to be used it is preferred that they contain ingredients novel to the individual as determined from the diet history. The initial amount to be fed should be estimated by calculating the previous daily caloric intake when weight stable or by using calculated maintenance energy requirement where the previous determination is not possible.

Client Education Points

- Colitis is a common disease in dogs characterized by increased frequency of defecation, straining to defecate, and feces which may contain blood and/or mucous. It is not usually associated with weight loss or loss of appetite.

- Colitis is an inflammation of the large bowel (colon) that may be caused by infection with parasites, fungi, or bacteria; dietary allergy or intolerance; and rarely cancer. One of the most common types of colitis has no known cause and is termed *inflammatory bowel disease*.
- Several tests are often necessary to identify the underlying cause of colitis including blood tests, fecal analysis, endoscopy, and biopsy of the large bowel.
- Medical treatment is aimed at eliminating the underlying cause wherever possible. Dietary modification can be an essential and effective method of alleviating clinical signs irrespective of the inciting cause.

Common Comorbidities

Colitis can occur in combination with small intestinal or gastric inflammation.

Interacting Medical Management Strategies

Medical therapy for colitis may include parasiticides, antiprotozoals, antibiotics, gastrointestinal protectants, and anti-inflammatory and immunosuppressive therapy.

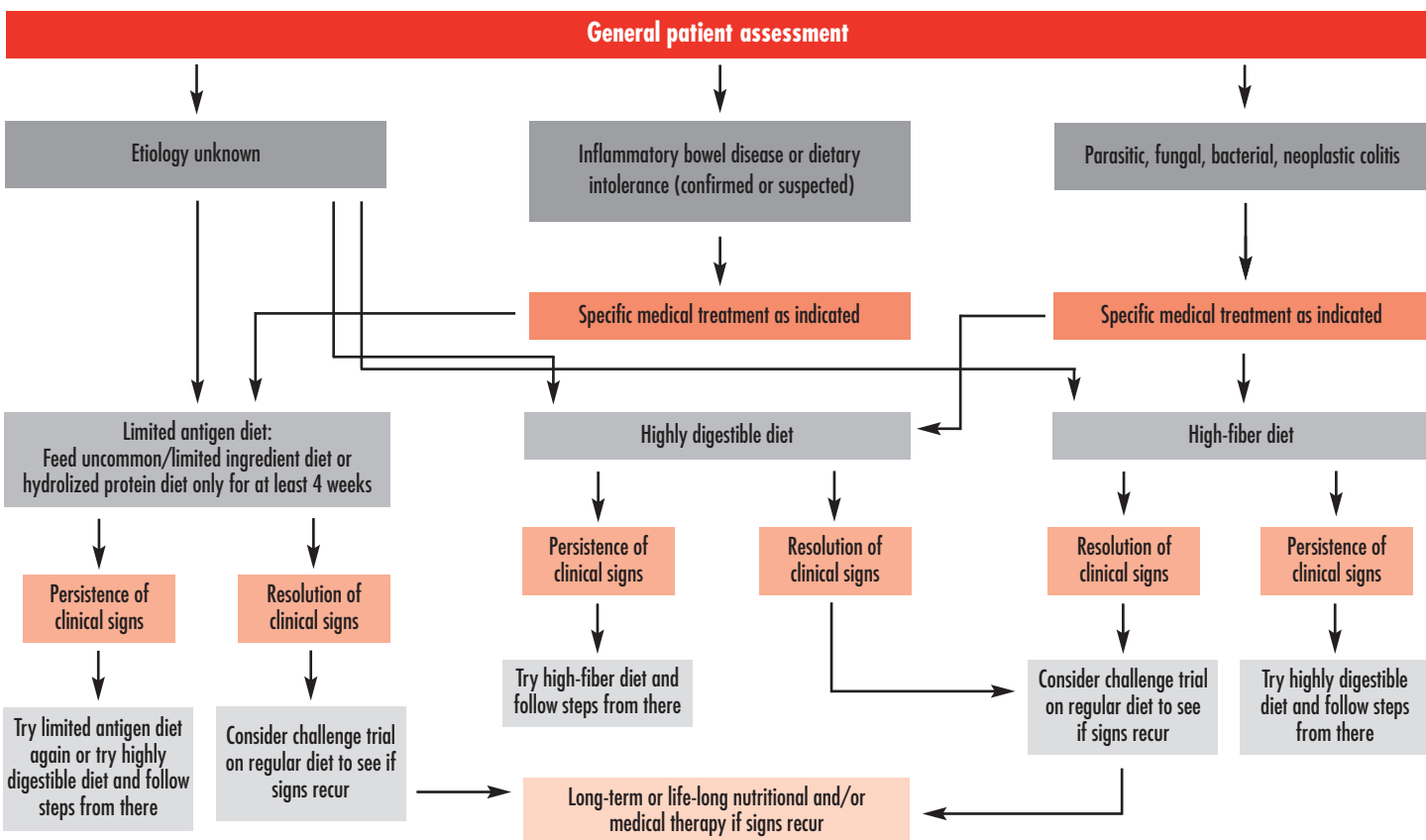
Polyphagia is a common side effect of corticosteroid administration and owners of dogs receiving elimination diets should be warned about indiscriminant eating or scavenging, which would counteract the benefits of dietary manipulation.

When prescribed, sulfasalazine, a drug with anti-inflammatory actions in the colon, should be administered with food to reduce the drug's emetic side effect. Conversely, some antibacterial agents are incompletely absorbed in the presence of food and efficacy is dependant on administration at least 1 hour before or 2 hours after meals.

Monitoring

The initial treatment of choice for colitis varies among clinicians, with some preferring dietary manipulation alone and others using medication(s) in addition. The efficacy of treatment is based on resolution or reduction in tenesmus, dyschezia, hematochezia, mucoid feces, diarrhea, and/or constipation. In patients where dietary modification alone is not effective, medical therapy should be instigated and continued for at least 2 to 4 weeks following control of clinical signs before gradual dosage reduction may be attempted. Dietary management, and for some dogs medical therapy, may be required long-term or life-long to control signs.

Algorithm – Nutritional Management of Canine Colitis



Colitis – Feline

Scott Campbell, BVSc, MACVSc, DACVN

Definition

Colitis is an inflammation of the colon that impairs absorption of water and electrolytes to produce clinical signs that may include tenesmus, dyschezia, hematochezia, mucoid feces, diarrhea, and/or constipation.

Key Diagnostic Tools and Measures

History, physical examination, fecal analysis, hematology, and serum biochemistry are routinely indicated. Colonoscopy and biopsy, and occasionally full-thickness biopsies via laparotomy, are necessary for diagnosis. A highly digestible or limited-antigen diet may be essential to manage clinical signs.

Pathophysiology

The pathophysiology of colitis is multifactorial and dependent on etiology. Electrolyte and water absorption by the inflamed mucosa is impaired and active secretion of electrolytes may also occur. Colonic motility is typically compromised and mucous secretion amplified by an inciting pathogen or secondary to inflammation itself. Advanced inflammation may result in colonic erosion and ulceration and more severe clinical signs.

Signalment

Colitis is reported in all breeds and ages of cats. Inflammatory bowel disease is reported to occur with greater frequency in purebred cats.

Key Nutrient Modifications

Most cats affected with colitis maintain their appetite and body weight. As such, modification of key dietary nutrients aims to reduce or abolish clinical signs. Depending on the etiology of colitis in an individual cat, a highly digestible or limited-antigen diet may be beneficial.

Recommended Ranges of Key Nutrients

All essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake. Consideration should be given to the use of novel or hydrolyzed protein, or highly digestible, diets.

Therapeutic Feeding Principles

As most cats with colitis maintain their appetite and body weight, the principal aim of dietary modification is to reduce the clinical signs of tenesmus, dyschezia, bloody mucoid feces, diarrhea, and/or constipation. Unlike dogs, cats with colitis do not benefit from increased dietary fiber. Instead, increasing the digestibility of the diet to limit the volume of ingesta delivered to the compromised colon and minimizing dietary antigens by restriction to a single novel protein or hydrolyzed protein to limit colonic inflammation forms the basis of dietary management. Studies in human patients suggest that antioxidants, omega-3 polyunsaturated fatty acids, fructo-oligosaccharides, prebiotics, and probiotics may be beneficial in the dietary management of colitis.

■ **Treats** – While treats can be important in maintaining the human–animal bond, treats that differ at all in composition from the primary diet should be avoided during the initial assessment phase so that efficacy of the base diet alone can be accurately evaluated. Cats that are managed with a highly digestible diet may be fed many treats also containing highly digestible ingredients. Cats fed uncommon/limited ingredient diets should only be fed treats containing the same ingredients as the base diet. Likewise,

cats receiving hydrolyzed-protein diets should only be fed treats containing similar hydrolyzed ingredients. Canned forms of any base diet can be sliced and baked to form cookies for treats. Alternatively, some of the primary diet may be offered outside regular feeding times and utilizing alternative feeding methods as a treat. Affection and attention can be provided as a substitute for food treats. If treats are given, they should be incorporated slowly, consistency maintained in type of treat given each day, and the cat should be monitored closely for recurrence of colitis signs. As always, it is suggested that all treats and supplements supply less than 10% of the total daily calories.

■ **Tips for Increasing Palatability** – Most cats with colitis maintain a good appetite and diets suitable for managing colitis do not typically lack palatability. If a particular commercial preparation is not accepted, the cat may find other comparable diets suitably tempting. Alternatively, warming the food to body temperature or adding moisture may increase palatability. Appetite stimulants or assisted-feeding devices are occasionally necessary in patients in which persistent anorexia precludes necessary caloric intake.

■ **Diet Recommendations** – A number of highly digestible, uncommon/limited-ingredient, and hydrolyzed-protein diets are available from the major therapeutic diet manufacturers in the United States. If uncommon/limited-ingredient diets are to be used it is preferred if they contain ingredients novel to the individual as determined by examining the diet history. The initial amount to be fed should be estimated by calculating the previous daily caloric intake when weight stable or by using calculated maintenance energy requirement where the previous determination is not possible.

Client Education Points

- Colitis in cats is characterized by increased frequency of defecation, straining to defecate, and feces that may contain blood and/or mucus. It is not usually associated with weight loss or loss of appetite.
- Colitis is an inflammation of the large bowel (colon) that may be caused by infection with parasites, fungi, or bacteria, dietary allergy or intolerance, or cancer. One of the most common types of colitis has no known cause and is termed *inflammatory bowel disease*.
- Several tests are often necessary to identify the underlying cause of colitis including blood tests, fecal analysis, endoscopy, and biopsy of the large bowel.
- Medical treatment is aimed at eliminating the underlying cause if it can be determined. Dietary modification can be an essential and effective method of alleviating clinical signs irrespective of the inciting cause. In some forms of colitis, such as that associated with inflammatory bowel disease, dietary management alone may prevent recurrence of clinical signs.

Common Comorbidities

Colitis can occur in combination with small intestinal or gastric inflammation.

Interacting Medical Management Strategies

Medical therapy for colitis may include parasiticides, antiprotozoals, antibiotics, gastrointestinal protectants, and anti-inflammatory and immunosuppressive therapy.

Polyphagia is a common side effect of corticosteroid administration and owners of cats receiving elimination diets should be warned about indiscriminant eating or scavenging, which would counteract the benefits of dietary manipulation.

When prescribed, sulfasalazine, a drug with anti-inflammatory actions

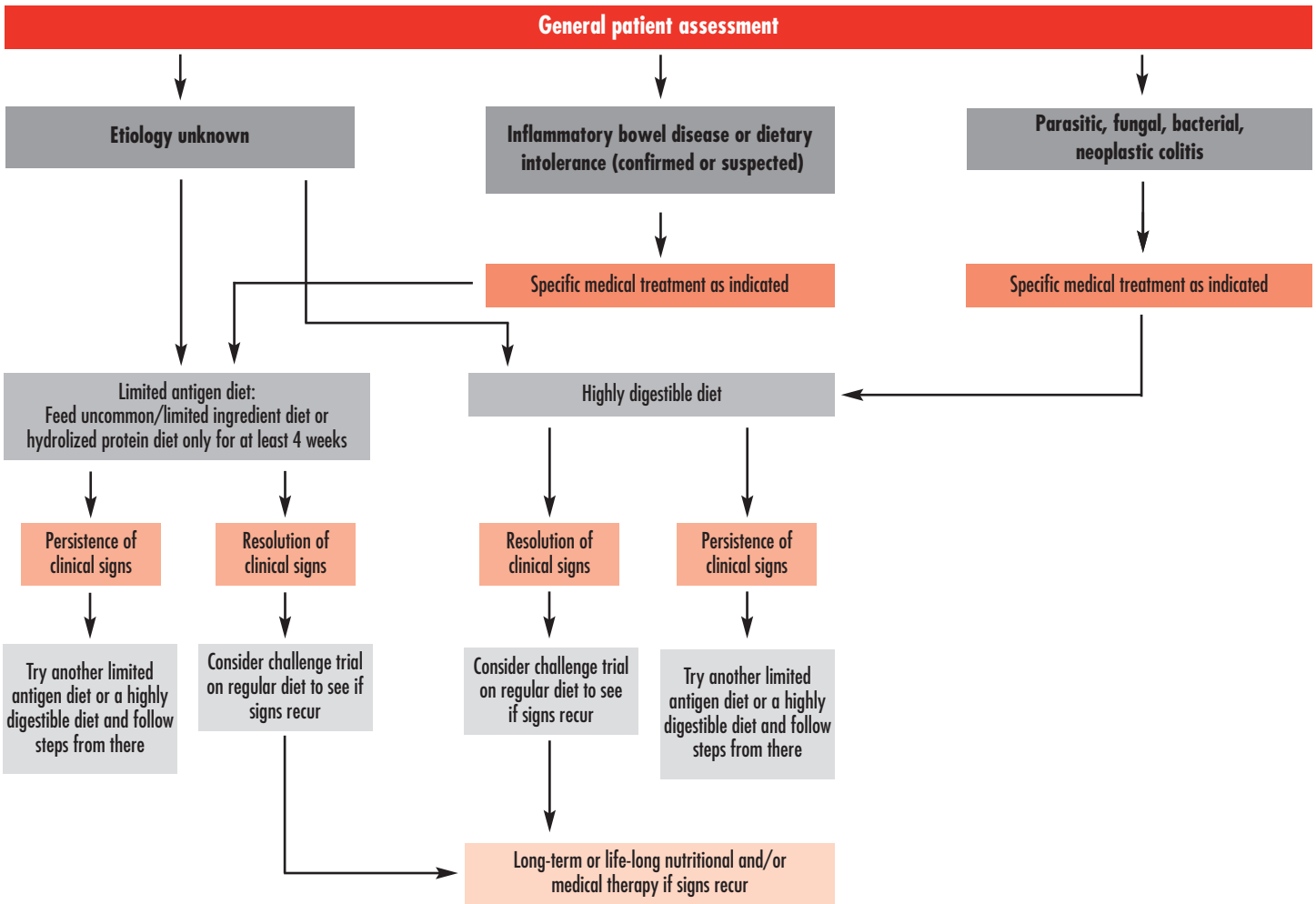
in the colon, should be administered with food to reduce the drug's emetic side effect. Conversely, some antibacterial agents are incompletely absorbed in the presence of food and efficacy is dependent on administration at least 1 hour before or 2 hours after meals.

Monitoring

The initial treatment of choice for colitis varies among clinicians, with some preferring dietary manipulation alone and others using medication(s) in

addition. The efficacy of treatment is based on resolution or reduction in tenesmus, dyschezia, hematochezia, mucoid feces, diarrhea, and/or constipation. When dietary modification alone is not effective, medical therapy should be instigated and continued for at least 2 to 4 weeks following control of clinical signs before gradual dosage reduction may be attempted. Dietary management, and for some cats, medical therapy, may be required long-term or life-long to control signs.

Algorithm – Nutritional Management of Feline Colitis



Exocrine Pancreatic Insufficiency – Canine

Scott Campbell, BVSc, MACVSc, DACVN

Definition

Exocrine pancreatic insufficiency (EPI) is a reduction in pancreatic acinar synthesis and secretion of digestive enzymes, including lipases, amylase, trypsin, and proteases, that results in retardation of normal intestinal digestion of fats, carbohydrates, protein and vitamins.

Key Diagnostic Tools and Measures

Trypsin-like immunoreactivity (TLI) measures the fasted level of serum trypsinogen (and active trypsin) and reflects exocrine pancreatic function. Fecal elastase and pancreatic histopathology obtained via laparoscopic or surgical biopsy can be useful adjunct tests for EPI. Common comorbidities with EPI are mentioned elsewhere.

Pathophysiology

Genetically based immune-mediated pancreatic acinar destruction precedes development of EPI in certain breeds.³ EPI can also occur secondary to chronic pancreatitis.² With either etiology the reduction in pancreatic exocrine function results in fewer enzymes being available to digest intestinal contents. Maldigestion of nutrients results in malnutrition, diarrhea, intestinal mucosal irritation, bacterial overgrowth, and toxin absorption. Subclinical reductions in digestive function precede the onset of characteristic clinical signs.

Signalment

German shepherd dogs and rough-coated collies comprise almost 90% of all canine EPI cases in the United States.¹ These breeds often develop the condition as young dogs secondary to immune-mediated pancreatic acinar destruction. Other breeds can develop EPI subsequent to chronic pancreatitis at any age.

Key Nutrient Modifications

Various nutrient modifications can be useful in individual dogs with EPI. Some dogs, particularly those that develop EPI secondary to chronic pancreatitis, may benefit from feeding a diet that is low in fat. The level of fat restriction should be considered relative to the previous intake ascertained from the diet history. Other dogs may benefit from feeding a higher-fat (more calorie dense) diet, highly digestible (low-fiber) diet, or a high-fiber diet. Many dogs may be well managed while remaining on their regular diet when supplemented with pancreatic enzymes. Regardless of which diet approach is used, it is important to feed a complete and balanced diet. Because of the variety of diets that may be acceptable to dogs with EPI, concurrent conditions may take priority in diet selection initially although modification may be required if significant undesirable signs are noted.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Fat	7–15	3–5	5	1.4

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy (ME). All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Diets for dogs with pancreatitis often contain less fat. Not all dogs with pancreatitis need this level of fat restriction and factors other than fat content (such as digestibility, protein content) may be important for dogs with pancreatitis.

Therapeutic Feeding Principles

Some resources recommend feeding a highly digestible fat-restricted diet to dogs with EPI to reduce flatulence and fecal volume, but many animals can be maintained on a variety of diets with appropriate digestive enzyme supplementation. In fact, recent studies have shown no significant benefit with feeding a highly digestible fat-restricted diet to dogs with EPI. Unnecessarily restricting the fat content of the diet will reduce the caloric density of the diet requiring the animal to ingest a larger volume of food in order to consume adequate calories to arrest further weight loss. A recent small study found that 40% of dogs with EPI were best controlled when maintained on their regular diet, 25% were best controlled on a high-fat diet, 20% were best controlled on a high-fiber diet and 10% were best controlled on a highly digestible diet.⁴ Recognizing that individual dogs with EPI respond differently to various feeding regimes, but all dogs likely benefit from consistency in the diet fed, will allow the clinician to individually formulate appropriate feeding strategies. Supplementation of the diet with parenteral vitamin B12 should be considered in deficient animals.

■ **Treats** – As dogs with EPI require supplementation of digestive enzymes with any food, and individual animals may respond undesirably to certain nutrients such as fat and fiber, treats are not generally recommended. If treats are given, they should be incorporated slowly, consistency maintained in type of treat, and the dog monitored closely for adverse effects. As always, it is suggested that all treats and supplements supply less than 10% of the total daily calories.

■ **Tips for Increasing Palatability** – The majority of dogs with EPI maintain an excellent appetite. If a particular commercial preparation is not accepted, the dog may find other suitable diets tempting. Alternatively, warming the food to body temperature or adding a sweetener such as corn syrup may increase palatability. Appetite stimulants or assisted-feeding devices are occasionally necessary in patients where persistent anorexia precludes necessary caloric intake.

■ **Diet Recommendations** – Many of the intestinal-disease type diets from the veterinary therapeutic diet manufacturers are considered to be highly digestible, but they have markedly variable fat contents. Clinicians should check the fat content of diets (ideally on a ME basis) with the manufacturers to ensure that they are appropriate for fat-intolerant animals. Some dogs with EPI may have improved clinical signs when fed a higher-fat diet, a high-fiber diet, or their regular diet.

Client Education Points

- EPI is a common disease in dogs whereby the pancreas fails to produce the products necessary for normal digestion of food.
- Diagnosis is often suggested on the basis of breed, age, and signs, but confirmatory laboratory testing is required.
- Clinical signs commonly seen in dogs with EPI including weight loss despite appropriate daily caloric intake, diarrhea, and a poor haircoat.
- These signs can often be alleviated with appropriate therapy.
- Animals diagnosed with EPI require life-long management, but can achieve extended survival times (median survival time >5 years in a recent study) particularly if they achieve a rapid initial response.
- Feeding of a consistent diet and supplementation of the diet with digestive enzymes are generally recommended.

- Various diet types can be effective for individual dogs so a few separate diet trials may be suggested.
- A number of secondary conditions can complicate management and are frequently checked for to ensure optimal control of clinical signs associated with EPI.
- Often rapid response to treatment can be seen, but some animals respond poorly to even the most complete treatment plans.

Common Comorbidities

Measurement of serum cobalamin and folate may be useful to assess for concurrent vitamin B12 malnutrition or intestinal bacterial overgrowth. Both of these conditions occur commonly in dogs with EPI and may have clinically relevant effects. Parenteral administration of vitamin B12 every 1 to 4 weeks may be used in dogs found to be deficient, as oral supplementation is unlikely to be beneficial in animals with deranged intestinal handling of this vitamin. Dogs with intestinal flora alterations secondary to maldigestion of nutrients may benefit from treatment with an antibiotic such as tylosin or a probiotic supplement. It may also be useful to routinely assess breeds other than those known to have a genetic basis for developing EPI for evidence of underlying pancreatitis and concurrent diabetes mellitus.

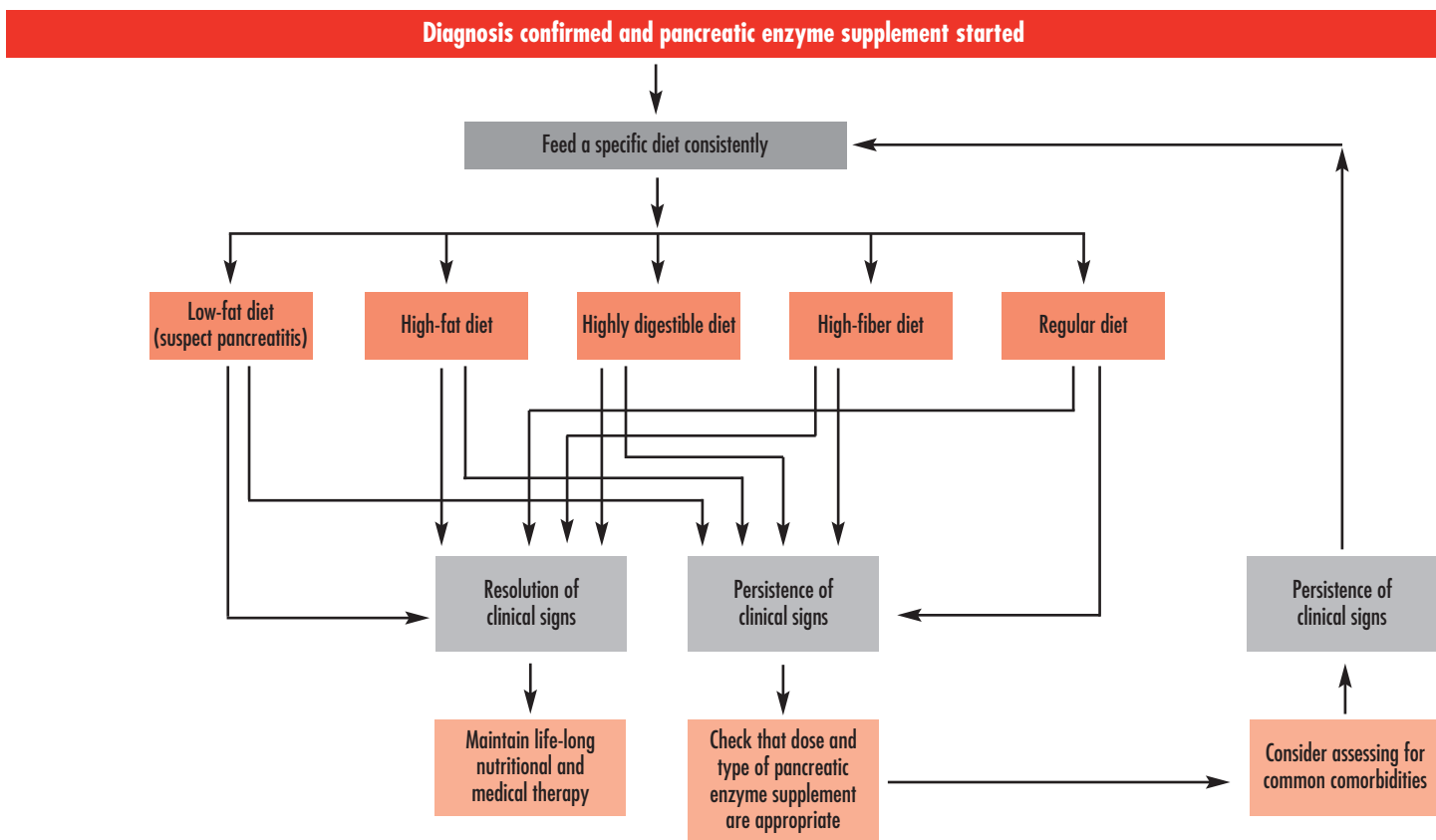
Interacting Medical Management Strategies

Supplementation of the diet with digestive enzymes, as powder, raw pancreas, or tablets/capsules, prior to consumption is essential in ensuring effective management of EPI. Inadequate digestive enzyme supplementation is a common cause of treatment failure. Powdered supplements are considered by some clinicians to be more effective in some dogs. Much of the supplemented enzyme is digested in the stomach so sufficient amounts must be supplied to overcome this anticipated loss. Some animals may benefit from concurrent administration of H-2 antagonists such as famotidine to reduce gastric acidity.

Monitoring

Primary clinical markers indicating attainment of a desired therapeutic effect are resolution of diarrhea, maintenance of or increase in body weight, and improvement in haircoat. It may also be beneficial to monitor serum cobalamin, folate, and pancreatic lipase immunoreactivity (PLI) concentrations depending on the results of initial testing. Animals that respond poorly to appropriate diets, supplementation, and medication should be evaluated for intestinal neoplasia, intestinal inflammation, or adverse reactions to food.

Algorithm – Nutritional Management of Canine Exocrine Pancreatic Insufficiency



Gastroenteritis / Vomiting – Canine

Korinn E. Saker, DVM, PhD, DACVN

Definition

Gastroenteritis is an inflammatory disease of the stomach and intestine. It is commonly characterized by either cellular infiltrates (eosinophils, lymphocytes, plasma cells) into the lamina propria, submucosa and/or muscularis, gastric ulcers/erosions, or severe, explosive bloody diarrhea (hemorrhagic). The predominant type of cells indicates the type of disease (i.e., eosinophilic or lymphocytic-plasmacytic [LPL] gastroenteritis)

Key Diagnostic Tools and Measures

Key features include a history of intermittent chronic vomiting and/or diarrhea, loss of body weight/condition, panhypoproteinemia, and neutrophilic leukocytosis with LPL. Diagnostic tools include minimum database plus fecal examination, bacterial culture and antigenic testing, fasting serum trypsin-like immunoreactivity (TLI), folate and cobalamin assays, and a hypoallergenic diet trial. Barium contrast studies, abdominal ultrasound, and endoscopy can help evaluate disease distribution and stomach/intestinal wall thickness and facilitate biopsy diagnosis.

Pathophysiology

Irritation/injury to mucosa and/or abnormal immune response leads to disruption of the mucosal barrier and recruitment and infiltration of inflammatory cells. Ongoing mucosal damage results in loss of gastrointestinal (GI) barrier function, decreased blood flow, and altered gut motility. Dietary indiscretion/intolerance, foreign body ingestion, toxins, drugs, and infectious agents are common primary GI causes. Renal and liver disease, endocrine disorders, shock, and sepsis are common systemic causes.

Signalment

Dietary, foreign body, and infectious etiologies are common in younger dogs, while metabolic, neoplastic, and drug-induced etiologies are common in older dogs. LPL gastroenteritis affects dogs over 5 years of age. Breed predilections include German shepherd dogs and the Shar-Pei with LPL; Irish setters with the gluten-sensitive form; and Lundehounds and Basenjis with non-LPL IBD. Eosinophilic gastroenteritis has a lower incidence and is seen predominantly in dogs less than 5 years of age, particularly German shepherd dogs, Rottweilers, and soft-coated wheaten terriers.

Key Nutrient Modifications

The inflammation characteristic of gastroenteritis adversely alters nutrient digestion and absorption. Dietary modifications should focus on overall diet digestibility, choosing a very highly digestible diet ($\geq 90\%$). Alter the dietary protein source to something the pet has not previously been exposed to and limit the content to one or possibly two high-quality protein sources fed at minimally adequate levels. Alter the dietary fat level based on the location of GI inflammation. Highly digestible soluble carbohydrate sources fed at reduced quantities are preferable. Diets with increased moisture content (canned vs. dry) help counter fluid losses. Increased levels of potassium, chloride, and sodium in the diet help to correct electrolyte alterations. Omega-3 fatty acids can provide an anti-inflammatory function.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	16–30	4.5–7.5	18	5.1
Fat	10–15	2.6–4.7	5	1.4
Fiber [#]	1–2.5	0.2–0.4	n/a	n/a
		mg/100 kcal		mg/100 kcal
Potassium	0.6–1.3	140–230	17	170
Sodium	0.3–0.5	85–120	0.06	17
Omega-3 fatty acid	1–1.5	200–300	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]Soluble fibers are preferred. The crude fiber analysis includes most insoluble fibers, but does not include soluble fibers. Therefore, crude fiber has limited usefulness when evaluating the total fiber content of foods. The ingredient list should be evaluated for sources of soluble fiber.

Therapeutic Feeding Principles

Goals of nutritional management are to minimize gastric irritation/vomiting, reduce gastric/intestinal secretions, promote gastric emptying, normalize gut motility, minimize residue, and meet determined nutritional requirements.

Initial treatment for acute, non-life-threatening vomiting includes nothing by mouth (NPO) for 12 to 24 hours with intravenous correction of fluid and electrolyte deficits if severely dehydrated. As vomiting resolves, offer small volumes of water or ice cubes orally (PO). If tolerated, gradually reintroduce an enteral diet. Initial re-feeding targets are 25% to 33% of resting energy requirement (RER) calories, with gradual increases over several days to provide RER, then daily energy requirement (DER) at current body weight (BW). Small meals, multiple times per day (three to six) minimize any adverse GI response and increase diet assimilation. Specific diet characteristics and target nutrient levels include:

- Total diet digestibility $\geq 90\%$.
 - Novel or hydrolyzed protein source. Ideally, a single, high-quality protein source with high digestibility ($>87\%$). Target protein intake between 4.5 and 7.5 g/100 kcal consumed; diet 16–30% on a DM basis.
 - Target fat intake between 2.6 and 4.7 g/100 kcal; diet $\leq 15\%$ fat, DM.
 - Low insoluble fiber content to increase digestibility. Target fiber intake between 0.2 and 0.35 g/100 kcal ($\sim 1.0\%$, DM) with fermentable fiber sources such as pectin, guar gum, gum arabic, beet pulp.
 - Adjust potassium (K⁺), sodium (Na), and chloride content. Target electrolyte intake between 140 and 230 mg K⁺/100 kcal; 0.6% to 1.3%, DM basis and 85 to 120 g Na/100 kcal; 0.35% to 0.5%, DM.
 - Mild fluid losses replaced orally with clean, fresh water and/or moist diet. Moderate to severe losses restored parenterally with appropriate crystalloid solutions.
 - Omega-3 fatty acid content ~ 250 mg/100 kcal; 75 to 100 mg/kg BW.
 - Target dietary omega-6:omega-3 at $\leq 2:1$.
 - Probiotic supplementation can be considered, but generally it is more commonly indicated in clinical diarrhea.
- Treats – Treats are not routinely recommended when managing gastrointestinal disorders. If treats are a necessary component of the daily feeding regime, choose highly digestible treats, providing a novel or

hydrolyzed protein source and moderate fat content. A small portion of the chosen dry diet or a complementary hydrolyzed dry kibble product can be presented as a treat. Bite-sized, baked treats can be made from the chosen canned diet. Caloric contribution from treats should not exceed 10% of total daily calories.

■ **Tips for Increasing Palatability** – Vomiting can be associated with food aversions. To circumvent this issue, review the ingredient list/nutrient content of the patient’s diet pre-GI disturbance, then identify disease management diets with different nutrient sources (i.e., protein, fat) for the re-feeding process.

Warm the canned diet to slightly above room temperature to enhance aroma. Add warm water or warmed low sodium broth or cooking juice to the dry food. If feeding a novel or hydrolyzed protein source, the broth or juice should be derived specifically from that protein source.

■ **Diet Recommendations** – Highly digestible therapeutic diets formulated to manage gastroenteritis can be referred to as *gastroenteric, gastrointestinal, intestinal, or low residue diets*. Novel or hydrolyzed protein/carbohydrate diets are commonly referred to as *anti-inflammatory, food allergic, hypoallergenic, limited antigen, low allergen, or skin and coat formula diets*. Senior life-stage diets may be acceptable.

Start re-feeding at or below RER (current BW) with the long-term goal of delivering DER calories at optimal BW.

Client Education Points

- Dogs with infectious or parasitic causes of gastroenteritis may be contagious to other animals and/or zoonotic. Exposure to other animals and handling of these patients should be done with caution.
- Animals diagnosed with IBD may have a genetic component and/or an immune system disorder predisposing these animals to other diseases.
- Spaying and neutering of affected animals should be discussed due to hereditary potential.
- IBD is not cured but is managed medically and through dietary focus.

- Treatment(s) and follow-up may be life-long. Dietary changes should be done gradually. Hospitalization and parenteral nutrition may be necessary for debilitated patients.

Common Comorbidities

Protein-losing enteropathy, kidney failure, liver failure, hypoadrenocorticism, pancreatitis, and exocrine pancreatic insufficiency are common comorbid conditions in dogs with gastroenteritis or vomiting.

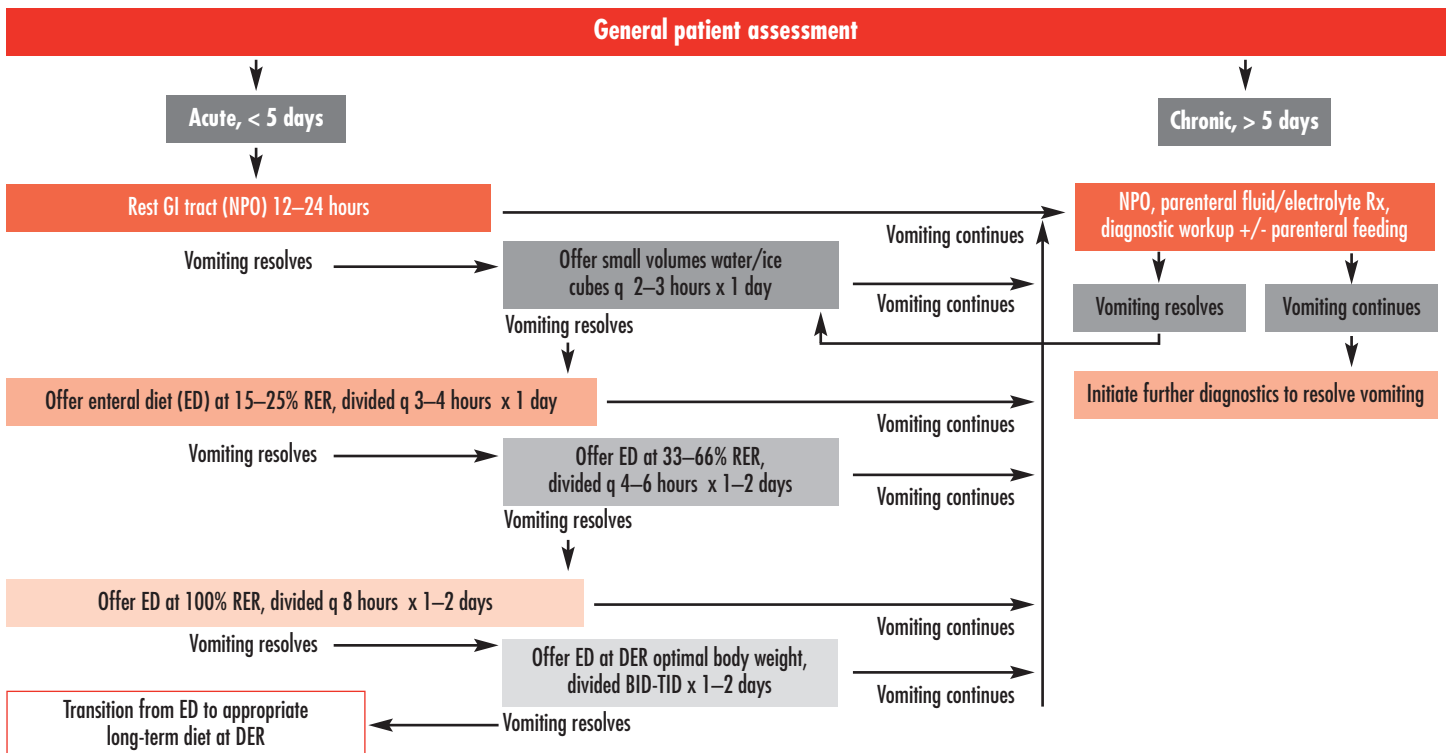
Interacting Medical Management Strategies

H₂ receptor antagonism and proton pump inhibition can decrease absorption of B vitamins and iron due to decreased acid release. Cytoprotectants form a mucosal barrier, inactivate pepsin, absorb bile salts and may bind to nutrients causing decreased nutrient absorption. Prokinetics alter the rate of food delivery and absorption in the small intestine by influencing gut motility. Chronic administration of steroids can decrease calcium absorption. Metronidazole can cause reversible neurotoxicity at high doses. Cyclosporine can irritate the GI tract and gingiva. Antibiotic administration changes microbial flora of the digestive tract and can chelate minerals (Ca, Mg, Fe, Zn) affecting nutrient metabolism and absorption.

Monitoring

Monitor hydration through sequential packed cell volume (PCV), urine specific gravity, and/or skin tent response. Utilize serum chemistry values to evaluate electrolyte status and renal function. Follow-up imaging can ascertain changes in stomach/intestinal wall thickness and disease distribution. Body weight and body condition score (BCS) changes reflect nutrient utilization and/or ongoing losses. If there is minimal or no improvement in key measures, reassess feeding plan components (patient, diet, feeding method) to reformulate a nutritional support plan based on new developments or previously overlooked parameters.

Algorithm – Nutritional Management of Canine Gastroenteritis / Vomiting



Gastroenteritis / Vomiting – Feline

Korinn E. Saker, DVM, PhD, DACVN

Definition

Gastroenteritis is an inflammatory disease of the stomach and intestine. Cellular infiltrates (eosinophils, lymphocytes, plasma cells) into the lamina propria, submucosa, and/or muscularis are characteristic and indicate the type (i.e., eosinophilic or lymphocytic-plasmacytic [LPL]) of gastroenteritis. Gastric ulcers/erosions may develop.

Key Diagnostic Tools and Measures

Key features of gastroenteritis in cats include a history of intermittent chronic vomiting and/or diarrhea, loss of body weight/condition, peripheral eosinophilia, nonregenerative anemia, and cobalamin deficiency. Diagnostic tools include minimum database plus thyroxine (T₄), FeLV/FIV serology, fasting serum trypsin-like immunoreactivity (TLI), and cobalamin assay. Barium contrast studies, abdominal ultrasound, and endoscopy can help evaluate disease distribution, stomach/intestinal wall thickness, and facilitate biopsy diagnosis.

Pathophysiology

Irritation/injury to the mucosa and/or abnormal immune response leads to disruption of the mucosal barrier and recruitment and infiltration of inflammatory cells. Ongoing mucosal damage results in loss of gastrointestinal (GI) barrier function, decreased blood flow, and altered gut motility. Foreign body ingestion, dietary intolerance, motility disorder, irritant or toxin ingestion, drugs, and infectious agents are common primary GI causes. Renal disease, liver disease, pancreatitis, and hyperthyroidism are common systemic causes.

Signalment

Lymphocytic-plasmacytic enteritis, an associated cause of inflammatory bowel disease (IBD), is more common in animals 2 years of age or older but is seen occasionally in kittens. No breed predilections are described. Hypereosinophilic syndrome occurs more frequently in middle-aged, female domestic short-haired (DSH) cats. Foreign body ingestion, parasitic, and infectious etiologies are more common in younger animals, while metabolic, neoplastic, or drug-induced causes are more common in older animals. Intestinal motility disorders are seen in middle-aged male DSH, domestic long-haired (DLH), and Siamese cats.

Key Nutrient Modifications

Inflammation associated with gastroenteritis adversely alters nutrient digestion and absorption. Dietary modifications should focus on overall diet digestibility, ideally choosing a very highly digestible diet ($\geq 90\%$). The protein source should be altered to something the pet has not previously been exposed to, and the content limited to one, possibly two, high-quality protein sources fed at reduced levels. The dietary fat level should be altered based on the location of GI inflammation. Highly digestible soluble carbohydrate sources fed at reduced quantities are preferable. Diets with increased moisture content (canned vs. dry) help counter fluid losses. Increased dietary potassium, chloride, and sodium can help correct electrolyte alterations. Omega-3 fatty acids can provide an anti-inflammatory function.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	32–42	7.5–9.5	26	6.5
Fat	15–24	3.5–6.0	9	2.3
Fiber [#]	1–2.5	0.2–0.5	n/a	n/a
		mg/100 kcal		mg/100 kcal
Potassium	0.75–1.1	180–250	0.6	150
Sodium	0.3–0.5	70–85	0.2	50
Omega-3 fatty acid	1–1.5	200–300	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]Soluble fibers are preferred. The crude fiber analysis includes most insoluble fibers, but does not include soluble fibers. Therefore, crude fiber has limited usefulness when evaluating the total fiber content of foods. The ingredient list should be evaluated for sources of soluble fiber.

Therapeutic Feeding Principles

Nutritional goals are to minimize gastric irritation/vomiting, reduce gastric/intestinal secretions, promote gastric emptying, normalize gut motility, minimize residue, and meet determined nutritional requirements.

Initial treatment for acute, non-life-threatening vomiting includes nothing by mouth (NPO) for 12 to 24 hours with intravenous (IV) correction of fluid and electrolyte deficits if severely dehydrated. As vomiting resolves, offer small volumes of water or ice cubes orally (PO). If tolerated, gradually reintroduce an enteral diet. Initial re-feeding targets are to gradually re-introduce an enteral diet. Initial re-feeding targets are 25% to 33% of resting energy requirement (RER) calories, with gradual increases over several days to provide RER, then daily energy requirement (DER) at current body weight (BW). Small meals, multiple times per day (three to six) minimize any adverse GI response and increase diet assimilation. Specific diet characteristics and target nutrient levels include:

- Total diet digestibility $\geq 90\%$.
- Novel or hydrolyzed protein source. Ideally, a single, high quality protein source of high digestibility ($>87\%$). Target protein intake between 7.5 and 9.5 g/100 kcal consumed; 32% to 42%, DM basis.
- Target fat intake between 3.5 and 6.0 g/100 kcal; diet 15% to 24% fat, DM.
- Low insoluble fiber content to increase digestibility. Target fiber intake between 0.2 and 0.5 g/100 kcal (1% to 2.5%, DM) with fermentable fiber sources such as pectin, guar gum, gum arabic, and beet pulp.
- Adjusted potassium (K⁺), sodium (Na), and chloride content. Target electrolyte intake between 180 and 250 mg K⁺/100 kcal; 0.75% to 1.1%, DM and 70 to 85 g Na/100 kcal; 0.3% to 0.45%, DM.
- Mild fluid losses should be replaced orally through clean, fresh water and/or moist diet. Moderate–severe losses should be restored parenterally with appropriate crystalloid solutions.
- Omega-3 fatty acid content ~ 250 mg/100 kcal; 75 to 100 mg/kg BW. Target dietary omega-6:omega-3 at $\leq 2:1$.
- Probiotic supplementation can be considered, but it generally is more commonly indicated with clinical diarrhea.

■ Treats – Treats are not routinely recommended when managing

gastrointestinal disorders. If treats are a necessary component of the daily feeding regime, choose highly digestible treats providing a novel or hydrolyzed protein source and moderate fat content. A small portion of the chosen dry diet or a complementary hydrolyzed dry kibble product can be presented as a treat. Bite-sized, baked treats can be made from the chosen canned diet. Caloric contribution from treats should not exceed 10% of total daily calories.

■ **Tips for Increasing Palatability** – Vomiting can be associated with food aversions. To circumvent this issue, review the ingredient list/nutrient content of the patient’s diet pre-GI disturbance, and identify disease management diets with different nutrient sources (i.e., protein, fat) for the re-feeding process.

Warm the canned diet to slightly above room temperature to enhance aroma. Add warm water or warmed low sodium broth or cooking juice to the dry food. If feeding a novel or hydrolyzed protein source, the broth or juice should be derived specifically from that protein source.

■ **Diet Recommendations** – Highly digestible therapeutic diets formulated for manage of gastroenteritis can be referred to as *gastroenteric, gastrointestinal, intestinal, or low residue diets*. Novel or hydrolyzed protein/carbohydrate diets are commonly referred to as *anti-inflammatory, food allergic, hypoallergenic, limited antigen, low allergen, or skin and coat formula diets*. Senior life-stage diets may be acceptable.

Start re-feeding at or below RER (current BW) with the long-term goal of delivering DER calories at optimal BW.

Client Education Points

- Cats with infectious or parasitic causes of gastroenteritis may be contagious to other animals or even zoonotic. Exposure to other animals and handling of these patients should be done with caution.
- Animals diagnosed with IBD may have a genetic component and/or immune system disorder and may be predisposed to other diseases. Spaying and neutering of affected animals should be discussed due to hereditary potential.

- Inflammatory GI conditions are not cured but rather managed medically and nutritionally. Treatment(s) and follow-up may be life-long. Dietary changes should be done gradually. Debilitated patients may require hospitalization and parenteral nutrition.

Common Comorbidities

Dehydration, malnutrition, hypoproteinemia, anemia, kidney failure, liver failure, hyperthyroidism, pancreatitis, pancreatic exocrine insufficiency, neoplasia, and FeLV/FIV are common comorbid conditions in cats with gastroenteritis or vomiting.

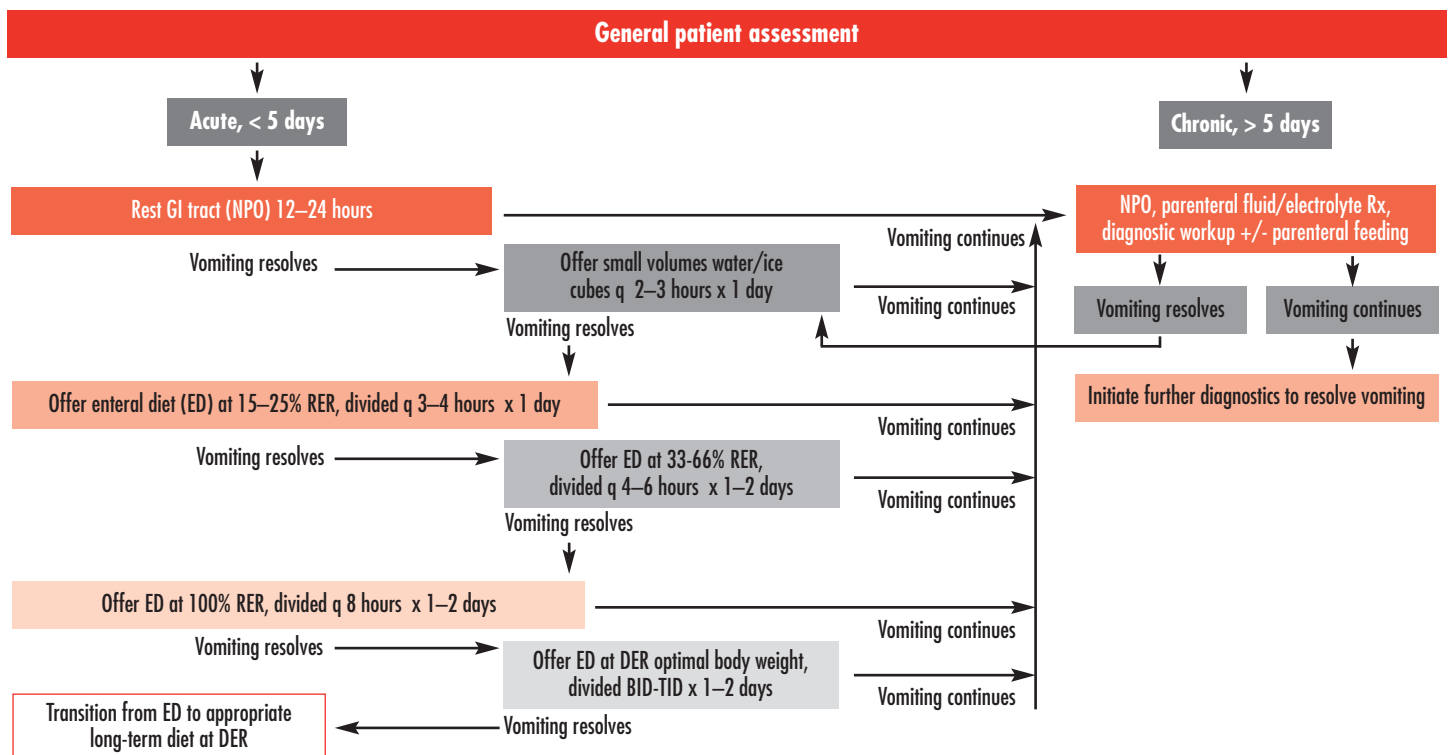
Interacting Medical Management Strategies

H₂ receptor antagonism and proton pump inhibition can decrease absorption of B vitamins and iron due to decreased acid release. Cytoprotectants form a mucosal barrier, inactivate pepsin, absorb bile salts, and may bind to nutrients causing decreased nutrient absorption. Prokinetics alter the rate of food delivery and absorption in the small intestine by influencing gut motility. Chronic administration of steroids can decrease calcium absorption. Metronidazole can cause reversible neurotoxicity at high doses. Antibiotic administration changes microbial flora of the digestive tract and can chelate minerals (Ca, Mg, Fe, Zn) affecting nutrient metabolism and absorption. Azathioprine can cause bone marrow suppression in cats.

Monitoring

Monitor hydration through sequential packed cell volume (PCV), urine specific gravity, and/or skin tent response. Utilize serum chemistry values to evaluate electrolyte status and renal function. Follow-up imaging can ascertain changes in stomach/intestinal wall thickness and disease distribution. Body weight and body condition score (BCS) changes reflect nutrient utilization and/or ongoing losses. If there is minimal or no improvement in key measures, reassess feeding plan components (patient, diet, feeding method) to reformulate a nutritional support plan based on new developments or previously overlooked parameters.

Algorithm – Nutritional Management of Feline Gastroenteritis / Vomiting



Chronic Enteropathies – Canine

Frédéric P. Gaschen, Dr.med.vet., Dr.habil., DACVIM, DECVIM-CA
Dottie Laflamme, DVM, PhD, DACVN

Definition

Chronic canine enteropathies include *adverse reactions to food*, *antibiotic-responsive diarrhea* (ARD), and *inflammatory bowel diseases* (IBD), and are defined by the occurrence of chronic, sometimes intermittent diarrhea with or without vomiting associated with an inflammatory infiltrate of variable severity in the gastrointestinal (GI) mucosa, in the absence of an identifiable cause.

Key Diagnostic Tools and Measures

The typical workup differs depending on the severity of disease. It is essential to initially rule out parasite infestation with serial fecal exams or administration of a broad-spectrum anthelmintic. Dogs with mild to moderate disease can undergo sequential treatment trials with an elimination diet, followed by antibiotics, before considering a more comprehensive workup. This option includes complete blood count (CBC) and chemistry profile, abdominal ultrasound, and sampling of mucosal biopsies. Severely affected dogs or those with signs of protein loss usually benefit from an aggressive initial diagnostic approach.

Pathophysiology

Adverse reactions to food include food intolerance (a non-immunologically mediated reaction) and food allergy (IgE or non-IgE-mediated).

Dogs with ARD benefit from a change in the commensal intestinal flora or disappearance of offending bacteria secondary to antibiotic treatment. Alternatively, selected antimicrobials may themselves directly influence the mucosal immunity.

The pathogenesis of canine IBD remains largely unknown. Abnormal intestinal flora and aberrant interactions between flora and the host immune system are most likely key players as exemplified by mucosa-adherent and invasive *E. coli* which have been directly implicated in the pathogenesis of histiocytic ulcerative colitis (HUC), a specific form of IBD.

Signalment

While dogs of many breeds have been diagnosed with chronic enteropathies, some breed predilections have been identified. The incidence of protein-losing enteropathies is reported to be high in soft-coated wheaten terriers, Shar-Peis, and Norwegian Lundehunds. Lymphangiectasia occurs frequently in Yorkshire terriers. Immunoproliferative enteritis is a disease of Basenjis. German shepherd dogs are at high risk for ARD. In a recent study, dogs with diet-responsive chronic enteropathies were generally young, large-breed dogs while those diagnosed with IBD were lighter and usually older.

Key Nutrient Modifications

Chronic enteropathies are a poorly defined syndrome with multiple, often unknown, etiologies. Hence, a single diet may not be right for all cases. A large proportion of dogs with chronic idiopathic enteropathies will respond to a diet change (diet-responsive disease). Among those, many dogs will benefit from being switched to an elimination diet (novel protein source or hydrolyzed proteins). Severe small intestinal IBD can result in malnutrition and malabsorption. A highly digestible, moderate- to low-fat diet should be fed in these patients. Omega-3 fatty acids from fish oil can reduce inflammation, and some patients with IBD may benefit from a diet with increased fish oil. If lymphangiectasia is part of the syndrome, feed a low-fat diet. Medium-chain triglycerides may be used to provide additional highly digestible calories. Probiotics may be of benefit in dogs with IBD.

Recommended Ranges of Key Nutrients

Nutrient	% DM	mg/100 kcal	% DM	mg/100 kcal
	Recommended dietary level		Minimum dietary requirement‡	
Total dietary fat	12–14%	2.5–3.5	5.0	1.43

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

#When necessary, e.g., in lymphangiectasia, fat may need to be restricted to below 12% dry matter, or less than 2.5 g/100 kcal

Therapeutic Feeding Principles

The goal of dietary therapy is to provide balanced nutrition for patients while helping to address clinical signs. GI inflammation can occur in response to dietary antigens, bacterial antigens, or other irritants. Any dietary change can result in alterations in these potential stimulants.

Dogs with chronic idiopathic enteropathies should undergo a dietary trial with a hydrolyzed or novel protein diet. Dogs may improve when fed these diets even if they do not have a food allergy (a final diagnosis of food allergy implies a clinical relapse upon challenge with ingredients from the prior diet). Improvement should be observed within 2 to 3 weeks.

A high percentage of dogs with suspected or confirmed IBD showed clinical improvement when fed either a hydrolyzed protein diet, or a diet containing 1% omega-3 fatty acids from fish oil.

For patients with protein-losing enteropathy or lymphangiectasia, a very low-fat, highly digestible diet should be used. A hydrolyzed protein diet, if low in fat, may also be of value in these patients.

Probiotics may be of value in IBD patients. By altering the GI microflora, they may change the bacterial antigens presented to the gut and may thereby reduce the inflammatory stimulus.

■ **Treats** – Treats, as well as flavored medications, should be completely omitted in dogs undergoing a dietary trial (for diet-responsive inflammation). When allowed, treats should not exceed 10% of daily calorie intake. If a dry diet is fed, kibble can be set aside to give as treats. If a canned diet is being fed, meatballs of this food can be used as is, or after broiling to create a crisper texture. For dogs with lymphangiectasia, low-fat treats may be fed. Commercial treats should contain <12% fat (dry basis). Other acceptable treats include bits of cooked chicken breast, raw or steamed vegetables, and fat-free yogurt.

■ **Tips for Increasing Palatability** – Addition of warm water can enhance the palatability of dry foods. Warming canned foods to body temperature releases aromatic compounds and can enhance palatability. Food may be sprinkled with a flavor-enhancing probiotic product.

■ **Diet Recommendations** – Novel protein diets are chosen on the basis of the dog's dietary history. Ideally, the animal should have had no prior exposure to the selected protein source. Secondary protein sources such as grains must be considered as well, as they may also contain allergenic proteins. While home-made diets may initially have some advantages, they do not provide balanced nutrition in the long term. Many nutritionally balanced products with proteins from various sources such as fish, venison, duck, rabbit, or kangaroo are currently available on the market. Novel protein diets are not inherently lower in allergenicity, so lack of prior exposure is critical. Some dogs may subsequently develop intolerance to the novel protein diets.

Hydrolyzed protein diets consist of smaller peptides that are less likely to elicit an immunologic reaction. As for novel protein diets, consider potential

protein contained in grains and other carbohydrates included in the diet. If no intact proteins are included in the diet, the diet can be used without knowing the patient's detailed diet history. While these diets are less allergenic than novel protein diets, a very small percentage of dogs may yet have an allergic response to their components.

Response to a diet change usually occurs within 2 to 3 weeks in dogs with diet-responsive chronic enteropathies. Some may be progressively reintroduced to a high-quality commercial diet after a successful elimination trial. Others will need to be fed an elimination diet on a long-term basis.

Highly digestible low-fat diets are indicated in dogs with severe protein-losing enteropathies (e.g., lymphangiectasia) and associated clinical signs. The addition of fermentable fiber to the diet provides additional benefits in dogs with chronic colitis. Fermentable fibers are metabolized to short chain fatty acids by the large intestinal flora and provide a useful source of energy. Overall, they enhance structure and function of the intestinal epithelium. Psyllium is an example of fermentable fiber that can be given as a supplement to the diet. Recommended dosages are: 0.5 tablespoon (T) for toy breeds, 1 T for small dogs, 2 T for medium dogs, and 3T for large dogs. Fiber should be added gradually (increasing to full amount over 4 to 7 days) to allow the GI microflora to adapt.

Client Education Points

- Strict dietary management of dogs with chronic intestinal disease is a central component of treatment. While this may prove challenging at times, it is essential to feed dogs exclusively with the recommended diet.
- Any treats that re-expose the dog to offending proteins may cause a relapse. This includes drugs such as chewable forms of heartworm preventative. They should be avoided during the dietary trial and until they have been shown not to induce an adverse reaction.
- Although home-prepared diets can be helpful, they generally do not provide balanced nutrition in the long term; this is why commercially available, nutritionally balanced diets are preferred.

Common Comorbidities

Moderate to severe chronic enteropathies affecting the small intestine are frequently associated with maldigestion and malabsorption due to failing intestinal function. This may result in malnutrition and weight loss. Recently, cobalamin (vitamin B12) deficiency has been documented in dogs with

chronic enteropathies. In such instances, parenteral cobalamin supplementation may be necessary for the treatment to be successful. The recommended dose in dogs with documented cobalamin deficiency is between 250 and 1,500 µg SC depending on the dog's size. Injections are administered weekly for 6 weeks, and every other week for 6 additional weeks. Regular reassessment of the dog's clinical status and cobalamin concentration is recommended to guide further treatment.

Interacting Medical Management Strategies

Antibiotics are recommended for the treatment of ARD. They greatly influence the composition of the intestinal flora which may have an impact on gastrointestinal function. The following antimicrobials are usually well tolerated: metronidazole, tylosin, or tetracycline. Enrofloxacin is the treatment of choice for HUC, a specific form of IBD.

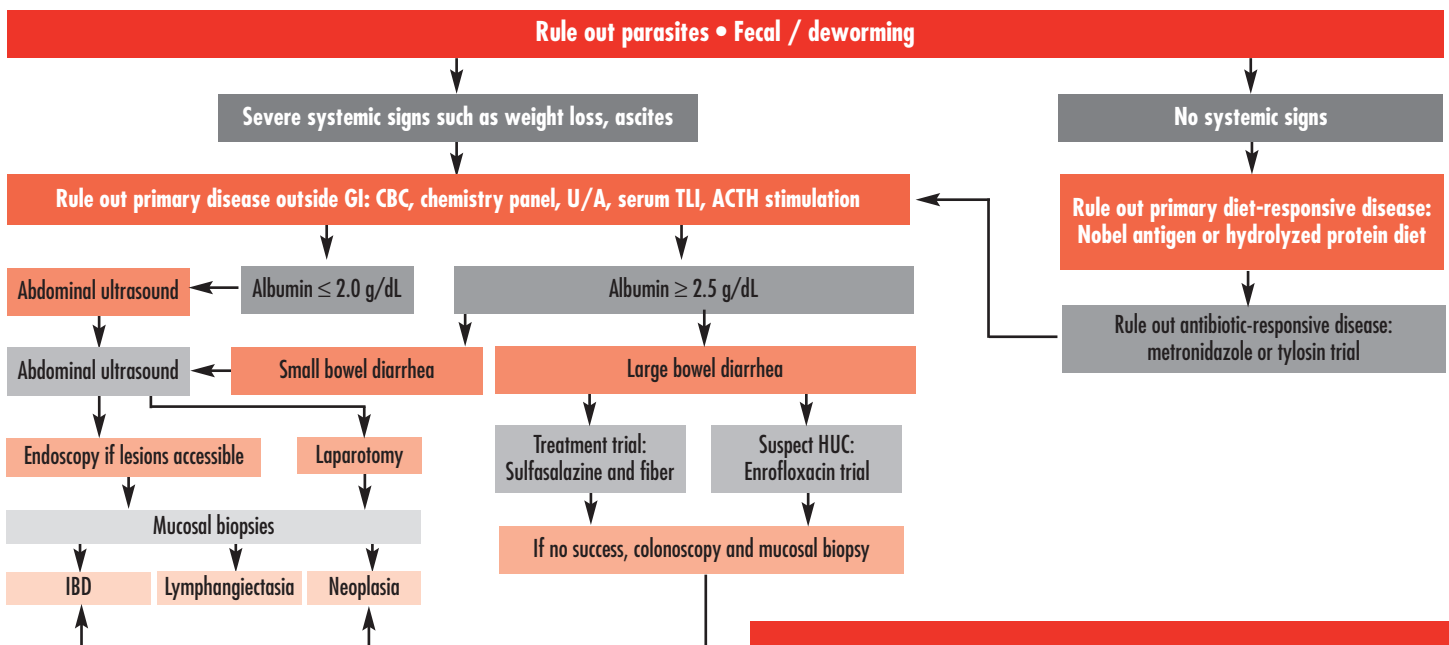
Corticosteroids are the mainstay for the treatment of idiopathic IBD, and are often administered at high doses (immunosuppressive dose of prednisone: minimum 2 mg/kg in two daily doses). Corticosteroids are catabolic hormones and are therefore not desirable in dogs suffering from GI dysfunction and its impact on the metabolism. In cases of IBD, however, their beneficial effects targeting the immune system by far outweigh the deleterious effects they may have on the metabolism.

Monitoring

Appropriate rechecks should be scheduled to reassess the dog's condition. Monitoring body weight (BW) and body condition scoring (BCS) will help ensure the dog receives adequate amounts of food. Clinical scoring indices grading various clinical and laboratory parameters are available in the literature, and may help the veterinarian measure changes from one visit to the next (canine IBD activity index [CIBDAI] or canine chronic enteropathy clinical activity index [CCECAI]).

In case of treatment failure despite adherence to the algorithm provided here, consider the following possibilities: (1) poor compliance with treatment; (2) presence of intercurrent disease such as exocrine pancreatic insufficiency, infection with refractory enteropathogens, or hypoadrenocorticism with glucocorticoid deficiency; (3) refractory IBD which may respond to a combination therapy with additional immunosuppressive drugs such as cyclosporine, azathioprine, chlorambucil; and (4) presence of diffuse GI neoplasia.

Algorithm – Nutritional Management of Canine Chronic Diarrhea



Chronic Enteropathies – Feline

Frédéric P. Gaschen, Dr.med.vet., Dr.habil., DACVIM, DECVIM-CA
Dottie Laflamme, DVM, PhD, DACVN

Definition

Chronic feline enteropathies include *adverse reactions to food* and *inflammatory bowel diseases* (IBD), and are defined by the occurrence of chronic, sometimes intermittent vomiting and/or diarrhea associated with an inflammatory infiltrate of variable severity in the gastrointestinal (GI) mucosa in the absence of an identifiable cause. Many cats present with vomiting and/or anorexia only.

Key Diagnostic Tools and Measures

It is essential to initially rule out parasite infestation. In cats with mild diarrhea and/or vomiting, an initial treatment trial with an elimination diet is appropriate before considering more invasive options. In cats with moderate to severe disease, a more aggressive approach is preferred. A variety of diseases originating outside the GI tract may cause vomiting in cats. They may be ruled out with a complete blood count (CBC) and chemistry profile including serum thyroxine (T4) concentration. An abdominal ultrasound exam may be helpful as well. If the disease is limited to the digestive tract, endoscopic or surgical sampling of mucosal biopsies may be required.

Pathophysiology

The pathophysiology of adverse reactions to food includes food intolerance (a non-immunologically mediated reaction) and food allergy (IgE mediated or non-IgE-mediated). The pathogenesis of feline IBD remains largely unknown. Abnormal intestinal flora and aberrant interactions with host immune system are most likely key players as exemplified by the recent finding of mucosa-adherent and invasive *E. coli* in association with feline IBD.

Signalment

Cats affected with chronic enteropathies are usually middle-aged, but the age range is wide and includes young and old animals as well. There is no documented breed predilection, although purebred cats such as Siamese, Persians, and Himalayans may be at increased risk. No gender predilection has been identified.

Key Nutrient Modifications

Food ingredients to which the cat may have an adverse reaction must be identified and avoided. Most allergic reactions occur to proteins in foods. The food ingredients most commonly recognized to be associated with adverse reactions in cats include beef, dairy, and fish products. Affected cats may respond to a novel protein or hydrolyzed protein diet. Highly digestible diets may be of benefit in animals with dietary sensitivities or IBD because these cats may have decreased gastrointestinal function. Omega-3 fatty acids from fish oil can reduce inflammation, and some patients with IBD may benefit from a diet with increased fish oil. Probiotics may be of benefit in cats with IBD.

Recommended Ranges of Key Nutrients

There is insufficient information available to recommend specific nutrient levels for cats with chronic enteropathies. Total digestibility should be $\geq 85\%$ (this information is not on labels, but should be available from the manufacturer). All essential nutrients should meet normal requirements adjusted for life stage, lifestyle and energy intake.

Therapeutic Feeding Principles

For *diet-responsive chronic enteropathies*, the goal of dietary therapy is to provide balanced nutrition for patients while helping to address clinical signs. Cats suspected to have adverse reactions to food should undergo a trial with an elimination diet. Two options exist: a diet composed of ingredients to which the cat has not previously been exposed (novel protein); or a hydrolyzed protein diet in which the antigens have been reduced in size below the point of recognition by allergen-specific antibodies (hypoallergenic). Selection of a novel protein diet must be based on a comprehensive diet history to avoid prior intake. Selection must also consider carbohydrate sources, such as grains, that also contain potentially allergenic proteins. Hydrolyzed hypoallergenic diets provide the advantages that a diet history is not critical to diet selection, and they reduce the risk for development of allergy to the novel diet. Most cats with diet-responsive diarrhea will show noticeable improvement within 1 to 3 weeks after being fed an appropriate elimination diet. Ongoing management depends on identifying and avoiding the specific food ingredients to which the patient reacts. Some cats, however, with food-responsive diarrhea may respond to a dietary change and not recrudescence when challenged. After a period of stabilization, some of these cats may return to their normal diet without problems.

For *IBD*, the goal of dietary therapy is to provide balanced nutrition for patients while helping to address clinical signs. GI inflammation can occur in response to food antigens, bacterial antigens, or other irritants. Any dietary change can result in alterations in these potential stimulants. A high percentage of cats with suspected or confirmed IBD showed clinical improvement when fed either a novel protein diet (see above), or a highly digestible diet containing either high or low dietary fat. Some cats with diarrhea respond positively to a low-carbohydrate diet. Probiotics may be of value in IBD patients. By altering the GI microflora, they may change the bacterial antigens presented to the gut and thereby reduce the inflammatory stimulus.

■ **Treats** – Treats, as well as flavored medications, should be completely omitted in cats undergoing a dietary trial (for food-responsive inflammation). Alternatively, treats can be composed of bits of the elimination diet. If a dry diet is fed, kibble can be set aside to give as treats. If a canned diet is being fed, meatballs of this food can be used as is, or after broiling to create a crisper texture. Other acceptable treats include bits of cooked chicken breast and fat-free yogurt.

■ **Tips for Increasing Palatability** – Warming canned foods to body temperature releases aromatic compounds and can enhance palatability. Food may be sprinkled with a flavor-enhancing probiotic product.

■ **Diet Recommendations** – For cats with adverse reaction to food, selection of novel protein diet is dependent on lack of prior exposure, so a thorough dietary history is required. Consider both primary and secondary protein sources, such as grains (see Appendix II). Commercial hydrolyzed protein diets are appropriate. In addition, limited-ingredient home-made diets can be good choices for short-term use during the elimination period.

For cats with IBD, select a highly digestible diet. Some cats respond to a low-carbohydrate (<15% dry basis) diet. For a dietary trial, use either a hydrolyzed protein or novel protein diet. Consider addition of probiotics for long-term management.

Client Education Points

- Strict dietary management of cats with chronic intestinal disease is a central component of the treatment. While this may prove challenging at times, it remains essential to feed cats exclusively with the recommended diet. If you own several cats, you may need to switch all

to the new diet as long as the other cats do not require a different specific dietary treatment. Any treats that re-expose the cat to offending proteins may cause a relapse and should be avoided during the dietary trial.

- Although home-prepared diets can be helpful, they generally do not provide a balanced nutrition in the long term. This is why commercially available, nutritionally balanced diets are preferred.

Common Comorbidities

It has been reported that cholangiohepatitis and pancreatitis may occasionally occur more frequently in cats with IBD. The term *triaditis* is used when intestine (IBD), liver (cholangiohepatitis), and pancreas (pancreatitis) are simultaneously affected. To date there is no confirmed theory describing a common pathomechanism for the three diseases.

Moderate to severe chronic enteropathies affecting the small intestine are frequently associated with maldigestion and malabsorption due to failing intestinal function. This may result in malnutrition and weight loss. Cobalamin (vitamin B12) deficiency has been documented in cats with chronic enteropathies. In such instances, parenteral cobalamin supplementation is necessary for the treatment to be successful. The recommended dose in cats with documented cobalamin deficiency is 250 µg given subcutaneously (SC). Injections are administered weekly for 6 weeks, and every other week for 6 additional weeks. Regular reassessment of the cat's clinical status and cobalamin concentration is recommended to guide further treatment.

Vitamin K deficiency was detected and attributed to intestinal malabsorption in cats with severe IBD but rarely resulted in bleeding

tendencies. Vitamin K1 supplementation was beneficial (1–5 mg/kg SC daily).

Interacting Medical Management Strategies

Corticosteroids are the mainstay for the treatment of idiopathic IBD, and are often administered at high doses (immunosuppressive dose of prednisolone: minimum 2 mg/kg daily). Corticosteroids are catabolic hormones and are, therefore, not desirable in cats suffering from gastrointestinal dysfunction. In cases of IBD, however, their beneficial effects targeting the immune system by far outweigh the deleterious effects they may have on the metabolism.

Monitoring

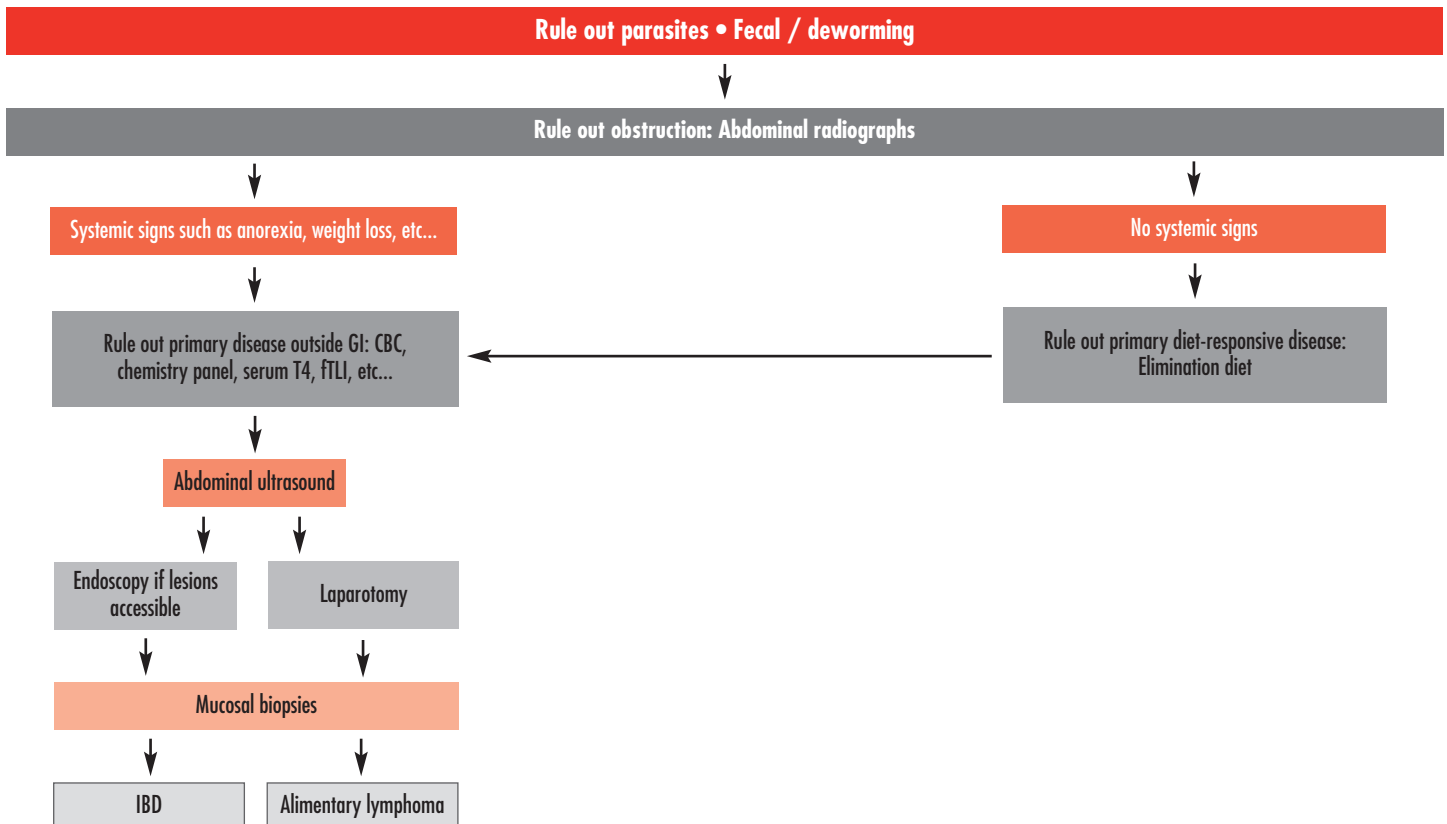
Response to a diet change usually occurs within 7 to 21 days in cats with diet-responsive chronic enteropathies. If dietary treatment fails, follow the algorithm provided.

In cats with documented IBD, prednisolone therapy usually initially includes high doses (2–4 mg/kg/day) which are progressively decreased in 2-week steps. Ideally, rechecks should be scheduled before each change in steroid therapy to reassess the cat's condition.

Monitoring body weight (BW) and body condition score (BCS) will help ensure the cat receives adequate amounts of food.

Differentiating IBD and alimentary lymphoma may be challenging in cats. Low-grade lymphoma may temporarily respond to steroid treatment but eventually relapse. Therefore, treatment-refractory IBD cats may in fact have lymphoma.

Algorithm – Nutritional Management of Feline Chronic Vomiting with or without Diarrhea



Lymphangiectasia – Canine

Debra L. Zoran, DVM, PhD, DACVIM

Definition

Lymphangiectasia is dilation of the lymphatic system, and in particular, the mesenteric lymphatics draining the small intestine, including the lacteals of the intestinal villi. The condition can be *primary* (caused by a congenital, presumed to be genetic defect), *secondary* (occurs secondary to another disease process that disrupts lymphatic flow), or *idiopathic*. In most dogs, the disease is idiopathic, and is often associated with a protein-losing enteropathy (PLE) that occurs as a result of the primary disease process (lymphangiectasia). Dogs with hereditary or congenital forms of the disease are often severely affected and may have a significantly shortened life span. Alternatively, the disease in dogs with idiopathic or secondary forms of lymphangiectasia is quite variable, ranging from milder, clinically manageable disease to severe, life-threatening disease.

Key Diagnostic Tools and Measures

Diagnosis of lymphangiectasia in dogs begins with a complete history, including dietary history and drug therapy, and physical examination, including rectal examination. Weight loss is a very important and early clinical sign of dogs with lymphangiectasia and PLE, and may precede development of diarrhea by months. Fecal stream analysis (e.g., fecal flotation, cytology, enzyme-linked immunosorbent assay [ELISA]/polymerase chain reaction [PCR] analysis) is important to rule out concurrent parasitic infections that may complicate the management of the disease.

A standard evaluation of the dog with weight loss—with or without diarrhea and hypoproteinemia—includes assessment for other systemic diseases, evaluation of liver function, ruling out proteinuria as the cause of hypoproteinemia, and evaluation of the gastrointestinal (GI) tract both functionally (trypsin-like immunoreactivity [TLI], cobalamin/folate, sugar absorption if available, possibly alpha-1 protease inhibitor) and structurally (imaging, histopathology obtained either via endoscopy or surgically). Once a diagnosis is made, supportive therapy (control of edema and protein loss) and nutritional management (ultra-low-fat diet to reduce exacerbation of the clinical disease in the intestine) is paramount unless a specific cause can be identified and corrected.

Pathophysiology

The clinical signs of lymphangiectasia (weight loss and diarrhea) occur as a result of maldigestion and malabsorption of nutrients (protein, fat, and carbohydrate) that occurs as a result of the lymphatic dilation (lymphangiectasia) or the combination of lymphatic dilation and inflammation (that may be present as a result of inflammatory bowel disease (IBD) or a reaction to lipogranulomas) that occur. In dogs with severe PLE, protein loss resulting in weight loss may precede development of diarrhea. In dogs with lymphangiectasia, the severe maldigestion and loss of nutrients may also lead to clinically significant hypocalcemia, hypocholesterolemia, and hypomagnesemia resulting in ascites or edema formation and seizures or muscle weakness in the most severely affected dogs.

Signalment

Common breeds of dogs affected by primary lymphangiectasia include Lundeheunds, Yorkshire terriers, and soft-coated wheaten terriers; these breeds may show clinical signs at a very early age. Any breed of dog may develop secondary lymphangiectasia and PLE, so there is not a typical signalment for this particular form.

Key Nutrient Modifications

Protein replenishment is the most pressing concern in dogs with edema or ascites due to severe hypoalbuminemia resulting from lymphangiectasia or PLE. As long as dogs with lymphangiectasia are continuing to eat, the best approach to replacement of protein is via **enteral nutrition**. Selecting a diet that is effective is the most challenging aspect of therapy of this disease. In dogs that are not eating, are too sick to eat, or cannot keep food down due to vomiting, **intravenous nutrition** may be necessary to provide protein and energy for metabolic function. The ideal diet will contain a moderate amount of highly digestible (or hydrolyzed) protein and carbohydrate, but is highly restricted in fat content. In the most severely affected dogs, an essentially ultra-low (<2 g/100 kcal fat) or no-fat diet may be essential to successful management of the disease, with gradual reintroduction of essential fatty acids and fat-soluble vitamins to prevent deficiency. In dogs with a suspected dietary allergy or concurrent IBD, a novel protein source (or one that is less antigenic, such as a hydrolyzed protein diet) may also be a necessary aspect of the dietary therapy. In addition to highly digestible protein sources, some dogs benefit from the addition of protein modules or elemental enteral diets such as Vivonex® T.E.N. (Nestlé Nutrition) to their hydrolyzed or ultra-low-fat diet.

The ideal carbohydrate (CHO) source for a dog with intestinal disease is generally believed to be cooked white rice or potato (without the skins) because they are highly digestible and do not contain gluten, which may be antigenic in some dogs. Other gluten-free CHO sources are tapioca and corn, but they are slightly less digestible than rice, and corn may cause hypersensitivities in some dogs. Dogs with severe lymphangiectasia often cannot handle corn unless it is completely ground into a mash.

In dogs with severe lymphangiectasia, the fat present in the diet should be in amounts that will supply the essential fatty acids (plant oils will provide some of these) and fat-soluble vitamins, but long-chain triglycerides should be avoided as much as possible. If additional fat is needed to supply energy, a source of medium-chain triglycerides can be used; however, these fat sources are often not palatable to dogs and may reduce the diet acceptability.

Reduced insoluble fiber in the diet is indicated in dogs with small bowel diarrhea, as this type of fiber reduces the digestibility of foods and may increase the risk of maldigestion or malabsorption of nutrients. This is particularly true in dogs with lymphangiectasia, as reducing the digestibility of protein and CHO sources may make the clinical signs worse (diarrhea), slow increase in protein levels and body weight, and increase the risk of bacterial disruption and thus may cause a new problem. Soluble fiber sources may be beneficial in some dogs as they are digested by the normal flora and may function as prebiotics to help maintain a healthy intestinal flora.

Recommended Ranges of Key Nutrients

Nutrient	% DM		g/100 kcal	
	Recommended dietary level	Minimum dietary requirement*		
Fat	5–15	5.0	1.5–4	1.4
Crude fiber	3–7	n/a	0.75–2.5	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

- Nutrients should be highly digestible (>90% digestibility) to minimize osmotic diarrhea, bacterial fermentation of undigested foods, and reduce intestinal gas.
- A high-quality, single-source protein (can be novel if IBD or food sensitivity is likely) or a hydrolyzed protein is indicated to maximize digestion and absorption.
- The carbohydrate source should be high quality, gluten free, and lactose free.
- Diets should contain low fat (less than 4 g/100 kcal at minimum; will likely need less than 3.5 g/100 kcal if severe PLE or lymphangiectasia is present).
- Increased omega-3 fatty acids (ratio of omega-6:omega-3 should be 5–10:1) can be added to improve eicosanoid profiles in the intestinal mucosa.
- Low insoluble fiber or moderate soluble or mixed fiber (3–7% total) is indicated to increase short-chain fatty acids and improve bacterial flora.
- Supplementation of fat-soluble vitamins (A, D, E, and K) is usually only necessary in severe cases of steatorrhea and long-term fat malabsorption.
- Adding a probiotic to the diet may be helpful to increase short chain fatty acid production and maintain microflora stability.

■ **Treats** – In general, treats should be avoided in dogs with intestinal disease until a definitive diagnosis is made. For example, if diarrhea is due to food sensitivity, an elimination diet trial will be necessary and this includes treats. If treats are important for the dog's daily routine, treats made using the therapeutic diet or based on the principles above can be given.

■ **Tips for Increasing Palatability** – If the dog will not eat the suggested diet, a small amount of low-sodium chicken broth can be added to the food. Alternatively, a small amount of the canned version of the dry food can be mixed with the food to increase interest.

■ **Diet Recommendations** – Prescription diets suitable for dogs with diarrhea are available through the veterinary clinic and should be formulated according to the therapeutic feeding principles listed above. Most low-fat OTC diets also have increased dietary fiber, and thus are not acceptable for this purpose.

Client Education Points

- Feed only the recommended foods.
- Feed small amounts of the food more frequently—three to four times per day. Large amounts of food increase the workload of the GI tract and may contribute to diarrhea or vomiting.
- Make sure plenty of water is available at all times. If vomiting occurs or the dog stops eating or drinking, a recheck with your veterinarian is recommended to prevent dehydration from the ongoing diarrhea.

Common Comorbidities

Conditions that commonly occur concurrently in dogs with lymphangiectasia include IBD and PLE, IBD and food allergy, and exocrine pancreatic insufficiency (EPI) and antibiotic-responsive enteropathy.

Interacting Medical Management Strategies

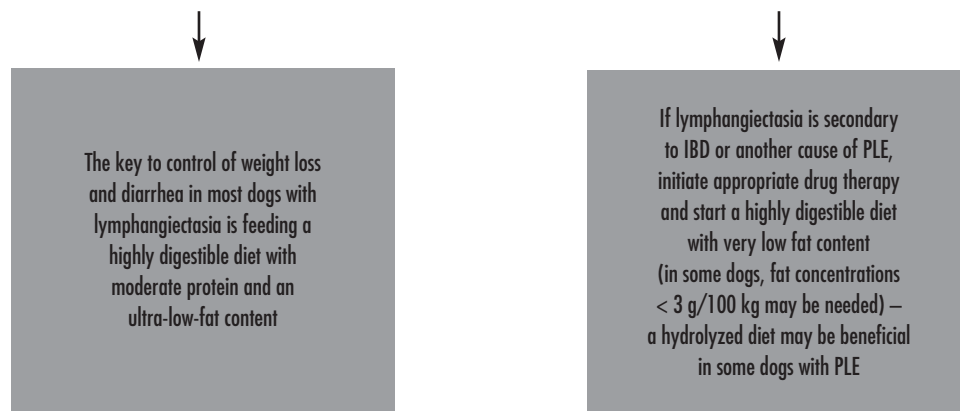
Steroid therapy in IBD will increase thirst and appetite and may result in unintended weight gain, loss of muscle mass, or hepatopathy. In dogs with lymphangiectasia, steroid therapy may worsen edema in some cases. Immunosuppressive therapy for IBD or lymphoma may result in GI toxicity, common clinical signs of which can be vomiting or diarrhea. Antibiotic therapy may cause diarrhea due to disruption of the normal flora and increased numbers of pathogenic species.

Monitoring

Fecal composition should be assessed to determine whether normal stool character is returning or if new problems (e.g., melena, hematochezia) are developing. Clinical condition must be monitored to be sure the dog is not dehydrated and is continuing to eat, with no new signs of illness (e.g., lethargy, weight loss, reduced or no appetite, or vomiting). If the dog is losing weight or becoming dehydrated, the feeding method and treatment should be re-evaluated and adjusted to the needs of the particular patient.

Algorithm – Nutritional Management of Canine Lymphangiectasia

Diagnosis of primary cause (if there is one) is first step, as it will alter the long-term treatment plan and prognosis



Pancreatitis – Canine

Kathryn E. Michel, DVM, MS, DACVN

Definition

Pancreatitis is an inflammatory condition that can be acute or chronic. Clinical signs can range from mild with minimal systemic effects to extremely severe disease characterized by pancreatic necrosis leading to the systemic inflammatory response syndrome (SIRS) and circulatory collapse.

Key Diagnostic Tools and Measures

Pancreatitis can be a challenging condition to diagnose as, aside from pancreatic biopsy, there are no specific diagnostic tests for this disease. To arrive at a diagnosis, the clinician will need to rely on his/her acumen utilizing the patient's clinical presentation (vomiting, abdominal pain, cardiovascular status), the results of clinical chemistry (serum chemistry, complete blood count [CBC], and urinalysis coupled with pancreatic biomarkers such as canine pancreatic lipase immunoreactivity [cPLI] and canine trypsin-like immunoreactivity [cTLI]), and imaging. A complete diet history should be taken that includes information about the commercial pet foods and treats the patient is fed including any table foods or scraps and whether there is any history of dietary indiscretion (see Appendix II).

The patient's nutritional status should be assessed with special attention paid to the duration of anorexia, evidence of weight loss (in particular, muscle wasting), severity of gastrointestinal (GI) signs, feasibility of assisted feeding, and concurrent medical conditions.

Pathophysiology

Often the inciting cause of pancreatic inflammation remains unknown although numerous drugs, dietary indiscretion, trauma, surgical manipulation, and ischemia have been implicated. The condition results from the failure of the protective mechanisms that normally ensure that the zymogens stored within the cells of the pancreas remain in an inactive form until they enter the duodenum. The activation of these zymogens within the pancreatic tissue unleashes their proteolytic effects resulting in tissue damage and inflammation.

The inflammatory response that accompanies severe pancreatitis produces a catabolic state that can cause a rapid deterioration in nutritional status. This decline in nutritional status is complicated by the fact that it is necessary to withhold food from patients that are experiencing nausea, vomiting, abdominal pain, ileus, or hemodynamic instability. Many patients with acute pancreatitis will present with hyperlipidemia.

Signalment

In general, older dogs (>5 years), obese dogs, and certain breeds (terriers, miniature schnauzers, Shetland sheepdogs) are reported to be at increased risk for developing pancreatitis.

Key Nutrient Modifications

Avoidance of oral intake of high-fat foods has been advocated for patients recovering from pancreatitis. Although this recommendation has not been evaluated by a prospective randomized clinical trial, it is based upon several different rationales. First, as fat (as well as protein) is a potent stimulant of pancreatic secretion, the concern in a convalescent patient is that pancreatic over-stimulation might lead to a relapse. Second, with regard to prevention of recurrent disease, investigations in dogs have shown that a low-protein, high-fat diet can induce pancreatitis and that pancreatitis is more severe when induced in dogs that had been fed a high-fat diet.^{1,2} Furthermore, there is evidence that hyperlipidemia may be an inciting factor for pancreatitis.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Fat	10–14	2–3.5	5	1.4

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

Classically food has been withheld from patients with moderate to severe acute pancreatitis with the aim of reducing pancreatic stimulation and thereby presumably reducing pancreatic secretions. Recently this dogma has been challenged in management of human patients. There is evidence that management schemes designed solely to promote pancreatic rest and minimize secretions have succeeded only in achieving pain relief and have not been shown to have an impact on patient outcome.³

Patients with moderate to severe pancreatitis are in a catabolic state and will experience a rapid decline in nutritional status. Furthermore, human prospective randomized clinical trials have shown improved outcomes when patients receive early enteral feeding via jejunostomy tube (j-tube) as opposed to parenteral nutrition (PN).⁴ Results have varied from trial to trial but most have found that enteral feeding via j-tubes attenuates the acute phase response and leads to a reduction in overall complications, including septic complications, when compared with PN.

The goal, therefore, for the management of canine pancreatitis is to encourage voluntary oral food intake in patients with mild or resolving clinical signs. Initially, the patient should be offered water, and based on response this is followed by a diet that is high in carbohydrates but low in fat and contains adequate but not high protein. Food should be offered in small meals 4 to 6 times per day.

In cases in which oral intake is contraindicated due to persistent nausea and vomiting, dogs should be evaluated as candidates for assisted feeding. If possible, they should receive a complete and balanced diet by the enteral route. Even patients with severe pancreatitis are able to tolerate j-tube feeding. When enteral feeding access is unobtainable in a patient for whom assisted feeding is indicated, parenteral nutritional support should be initiated.

■ **Treats** – Table foods and scraps that are high in fat should not be used as treats for patients that have recovered from a bout of pancreatitis. This includes most meats, dairy products (unless they are non-fat varieties), and fried foods or snacks. Appropriate treats include most dog biscuits, fresh fruits and vegetables (with the exception of grapes and onions), very lean meats (e.g., baked chicken breast), and pasta.

■ **Tips for Increasing Palatability** – There are a number of therapeutic and commercial dog foods to choose from that will meet the guidelines for moderate to low fat content, and it should be possible to find an appropriate diet that is acceptable to most patients. Adding some warm water to a dry food, slightly warming a canned food, or adding a few tablespoons of low-sodium, plain canned tomato sauce to a food may enhance acceptance.

■ **Diet Recommendations** – Transition the patient onto a complete and balanced diet that has adequate protein and moderate fat content (<30% fat, energy basis). Most dry and canned gastrointestinal therapeutic diets will meet this criterion and it is possible that the patient's normal diet will

as well. Patients with idiopathic hyperlipidemia may require greater dietary fat restriction (<20% fat, energy basis). Patients should be monitored for food acceptance, recurrence of clinical signs, and weight maintenance.

For patients in good body condition, feeding portions should be based on previous caloric intake. For underweight patients, calories offered should be increased by 20% above previous intake to promote weight gain during convalescence and adjusted as necessary based upon response. For overweight patients, a weight reduction program should be prescribed once the patient has fully recovered from pancreatitis.

Client Education Points

- All members of the household should understand that there is a potential risk of recurrent disease.
- The clients should be aware of any dietary and feeding recommendations and the reasons for them. Of particular importance is restricting the pet’s access to high-fat foods, treats, table scraps, and garbage.
- When a patient is under- or overweight upon discharge from the hospital, there should be discussion of what additional nutritional management will be necessary in the coming weeks to ensure a return to an optimal body weight. The owner should understand why taking such steps will be beneficial for the patient.

Common Comorbidities

Often a patient with acute pancreatitis will present with **hyperlipidemia**. While in many cases the elevation in serum lipid concentration is a consequence of the pancreatitis, there is evidence that pre-existing hyperlipidemia can be causative agent of this condition. Hyperlipidemia can be associated with a high-fat meal, a concurrent endocrinopathy (hyperadrenocorticism, diabetes mellitus, or hypothyroidism), or a breed

disposition (miniature schnauzers, Shetland sheepdogs). When hyperlipidemia is secondary to underlying condition, naturally the best therapeutic approach is to address that condition. Patients with primary hyperlipidemia may benefit from a very low fat diet (<20% fat, energy basis) as will some patients who have this condition secondary to other diseases (see pages 80–81). Pancreatitis may also occur concurrently in dogs with **diabetes mellitus** (see pages 28–29).

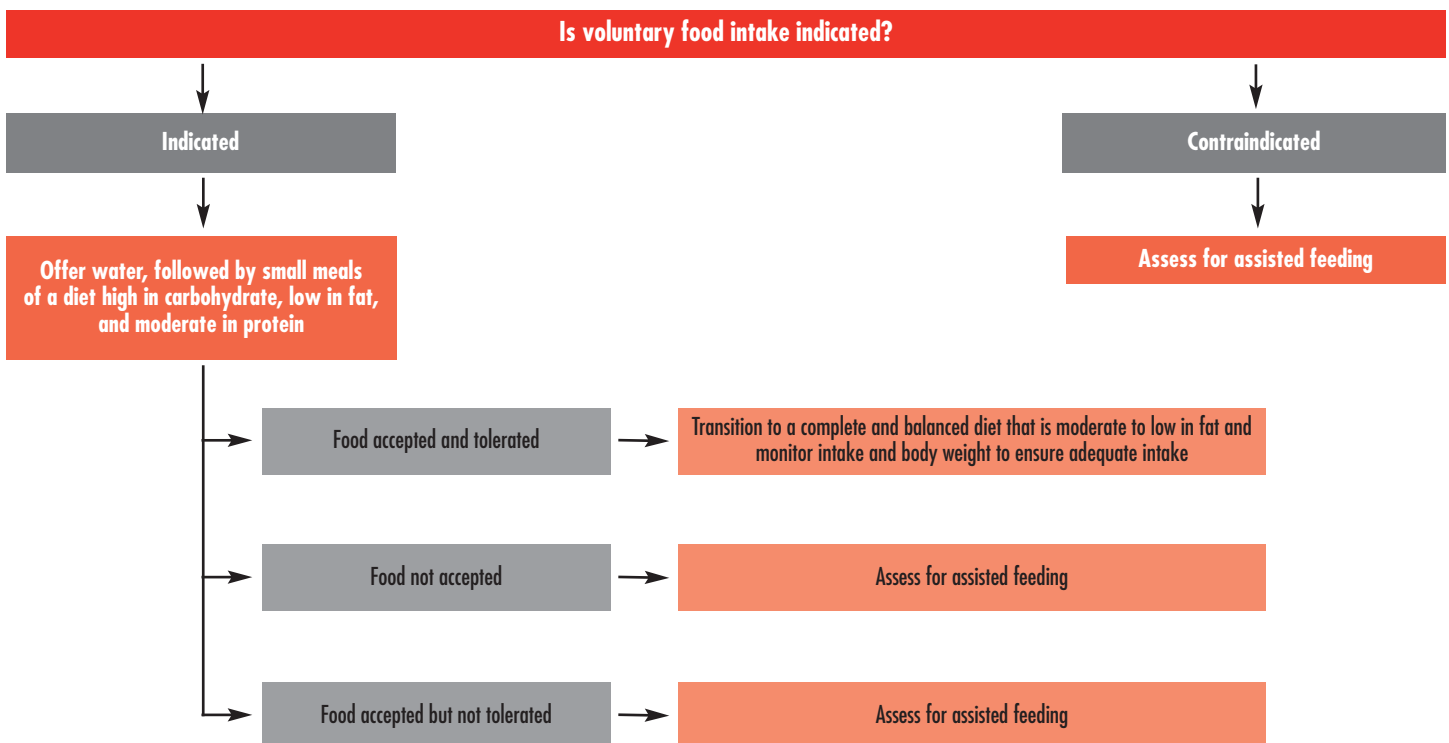
Interacting Medical Management Strategies

Patients with moderate to severe acute pancreatitis will require aggressive fluid resuscitation and supportive care that can include colloid support, antiemetics, gastroprotective agents, and analgesics. While pain control is an important aspect of the medical management of pancreatitis, some analgesic agents can cause gastrointestinal ileus. Ileus can depress a patient’s appetite and therefore delay the return of voluntary intake or complicate the delivery of enteral nutrition in a patient receiving tube feeding. Ileus can be addressed by weaning the patient off of analgesic medication as soon as feasibly possible or switching to a medication that has fewer gastrointestinal side effects.

Monitoring

Patients that either needed to regain or lose weight after discharge from the hospital should have their weight monitored to ensure that appropriate progress is being made. Patients that are being treated for primary hyperlipidemia will require rechecks to monitor serum lipid concentrations to determine if the level of dietary fat restriction is sufficient. Any future visit to the clinic, regardless of purpose, is an opportunity to inquire about the patient’s dietary management and chance to reinforce previous feeding recommendations.

Algorithm – Nutritional Management of Canine Pancreatitis



Pancreatitis – Feline

Kathryn E. Michel, DVM, MS, DACVN

Definition

Pancreatitis is an inflammatory condition that can be acute or chronic. Clinical signs can range from mild with minimal systemic effects to extremely severe disease characterized by pancreatic necrosis leading to the systemic inflammatory response syndrome (SIRS) and circulatory collapse.

Key Diagnostic Tools and Measures

Pancreatitis can be a challenging condition to diagnose as, aside from pancreatic biopsy, there are no specific diagnostic tests for this disease. Diagnosis is further complicated by the fact that the clinical presentation of this condition in cats differs significantly from dogs. In feline pancreatitis, the most common clinical signs are nonspecific (anorexia, lethargy, and dehydration), whereas the classic signs associated with canine pancreatitis (vomiting and abdominal pain) are relatively uncommon in cats.¹ The clinician will need to rely on his/her acumen utilizing the patient's clinical presentation, the results of clinical chemistry (serum chemistry, complete blood count [CBC], and urinalysis coupled with pancreatic biomarkers such as feline pancreatic lipase immunoreactivity [fPLI] and feline trypsin-like immunoreactivity [fTLI]), and imaging to arrive at a diagnosis.

A complete diet history should be taken that includes information about typical food intake and any commercial pet foods and treats that the patient receives, including table foods or scraps (see Appendix II).

The patient's nutritional status should be assessed with special attention paid to the duration of anorexia, evidence of weight loss (in particular, muscle wasting), severity of gastrointestinal signs, feasibility of assisted feeding, and concurrent medical conditions.

Pathophysiology

In the majority of cats, an inciting cause of pancreatic inflammation cannot be determined although infectious agents (*Toxoplasma gondii*, flukes, feline infectious peritonitis [FIP]), organophosphate pesticides, drugs, trauma, surgical manipulation, and ischemia have been implicated in the pathogenesis of this condition. There also has been speculation about the common association of feline pancreatitis with inflammatory bowel disease and cholangiohepatitis and the possibility of a related etiopathogenesis.²

Pancreatitis results from the failure of the protective mechanisms that normally ensure that the zymogens stored within the cells of the pancreas remain in an inactive form until they enter the duodenum. The activation of these zymogens within the pancreatic tissue unleashes their proteolytic effects resulting in tissue damage and inflammation.

In the acute form of the disease, the inflammatory response that accompanies severe pancreatitis produces a catabolic state that can cause a rapid deterioration in nutritional status. This decline in nutritional status is complicated by the fact that it is necessary to withhold oral food intake from patients that are experiencing nausea, vomiting, abdominal pain, ileus, or hemodynamic instability.

In the chronic form of feline pancreatitis, since anorexia is one of the most common clinical findings, patients often present with evidence of malnutrition, in particular, weight loss characterized by muscle wasting. It is imperative that muscle mass be assessed by physical palpation as patients can present with excess body fat and an obese appearance despite having experienced significant muscle wasting.

Signalment

There have not yet been any reports of definite associations between age, breed, or neuter status and the risk of feline pancreatitis.

Key Nutrient Modifications

Avoidance of oral intake of high-fat foods has been advocated for patients recovering from pancreatitis because fat (as well as protein) is a potent stimulus of pancreatic secretion and the concern is that, in a convalescent patient, pancreatic overstimulation might lead to a relapse. Cats have metabolic adaptations, however, that reflect this species' evolution on a diet rich in protein and fat but lacking any significant amounts of carbohydrates. As a consequence, most cat foods are relatively high in fat and protein. The lower-fat cat foods that are available will still contain moderate amounts of fat, high protein, and often have a low caloric density. While the impact of dietary fat intake on clinical outcome in cats diagnosed with feline pancreatitis has not been evaluated in a clinical trial, anecdotally, cats recovering from pancreatitis appear to tolerate typical cat foods including those containing high amounts of fat.

Recommended Ranges of Key Nutrients

There appears to be insufficient data upon which to identify any necessary nutrient modifications.

Therapeutic Feeding Principles

Feline pancreatitis is an emerging disease that was rarely diagnosed before 1990. Most practitioners are more familiar with treating the acute form of pancreatitis in canine patients. Classically, food is withheld from canine patients with moderate to severe acute pancreatitis followed by gradual reintroduction of oral intake of foods high in carbohydrates but low in fat and moderate in protein.

There are problematic aspects of using this approach in cats suffering from this disease. First, as anorexia is one of the most common clinical findings in cats with pancreatitis, patients often present with evidence of malnutrition. Further fasting will only serve to worsen the extent of malnutrition in these patients. In addition, idiopathic hepatic lipidosis (IHL) is a common concurrent disease or sequelae of feline pancreatitis and withholding food from a patient with IHL would be contraindicated.

Classically food has been withheld from patients with moderate to severe acute pancreatitis with the aim of reducing pancreatic stimulation and thereby presumably reducing pancreatic secretions. Recently this dogma has been challenged in management of human patients. There is evidence that management schemes designed solely to promote pancreatic rest and minimize secretions have succeeded only in achieving pain relief and have not been shown to have an impact on patient outcome.³

Therefore, voluntary food intake should be encouraged in patients in which oral intake is not contraindicated due to persistent nausea and vomiting. Patients who refuse food or have inadequate voluntary intake should be evaluated as candidates for assisted feeding. Ideally they should receive a complete and balanced diet by the enteral route. Even patients with severe pancreatitis and persistent vomiting are able to tolerate jejunostomy tube feeding. However, when enteral feeding access is unobtainable in a patient in which assisted feeding is indicated, parenteral nutritional support should be initiated.

■ **Treats** – The recommendation for dogs recovering from pancreatitis is to avoid commercial treats or table foods and scraps that are high in fat. It is unclear whether a similar recommendation should apply to cats recovering from this condition; however, it may be prudent to avoid very high fat items such as fat trimmings from meat, fried foods, or cream. Acceptable treats would include lean meats or fish (e.g., baked chicken breast, tuna packed in water), low-fat dairy products, and fresh fruits and vegetables (with the exception of grapes and onions).

■ **Tips for Increasing Palatability** – Unless there are clear indications for feeding a fat-restricted food (<30% fat, energy basis), diet selection should

be predicated on finding a complete and balanced cat food that is acceptable to the patient. Adding some warm water to a dry food or slightly warming a canned food may enhance acceptance.

■ **Diet Recommendations** – Most cats diagnosed with pancreatitis will present with a history of anorexia. It is essential to monitor the patient’s food intake, especially when transitioning from assisted feeding, to ensure that the patient’s voluntary consumption is adequate. The goal will be to find a diet that the patient eats readily, is tolerated by the gastrointestinal tract, and is appropriate for any concurrent condition that the patient may have (e.g., inflammatory bowel disease, liver disease, diabetes mellitus). Patients should be monitored for food acceptance, weight maintenance, and recurrence of clinical signs.

For patients in good body condition, feeding portions should be based on previous caloric intake. For underweight patients, calories offered should be increased by 20% above previous intake to promote weight gain during convalescence and adjusted as necessary based upon response. For overweight patients, a weight reduction program should be prescribed once the patient has fully recovered from pancreatitis.

Client Education Points

- All members of the household should understand that there is a potential risk of recurrent disease. They should be made aware of any dietary and feeding recommendations and the reasons for them. Of particular importance is monitoring the patient’s food intake and body condition to enable early detection of anorexia and weight loss.
- When a patient is under- or overweight upon discharge from the hospital, there should be a discussion of what additional nutritional management will be necessary in the coming weeks to ensure a return to an optimal body weight, and why taking such steps will be beneficial for the patient.

Common Comorbidities

It is not uncommon for feline patients to be diagnosed with concurrent pancreatitis and inflammatory bowel disease. These patients may benefit from a novel protein/limited antigen or hydrolyzed protein diet. Most patients diagnosed with concurrent pancreatitis and IHL will require assisted feeding. Adequate food intake is necessary for resolution of IHL and cats with this condition generally exhibit insufficient voluntary intake. Cats with hepatic failure, regardless the underlying etiology, may require dietary modification including protein restriction. Pancreatitis may also occur concurrently in cats with diabetes mellitus (see pages 30–31).

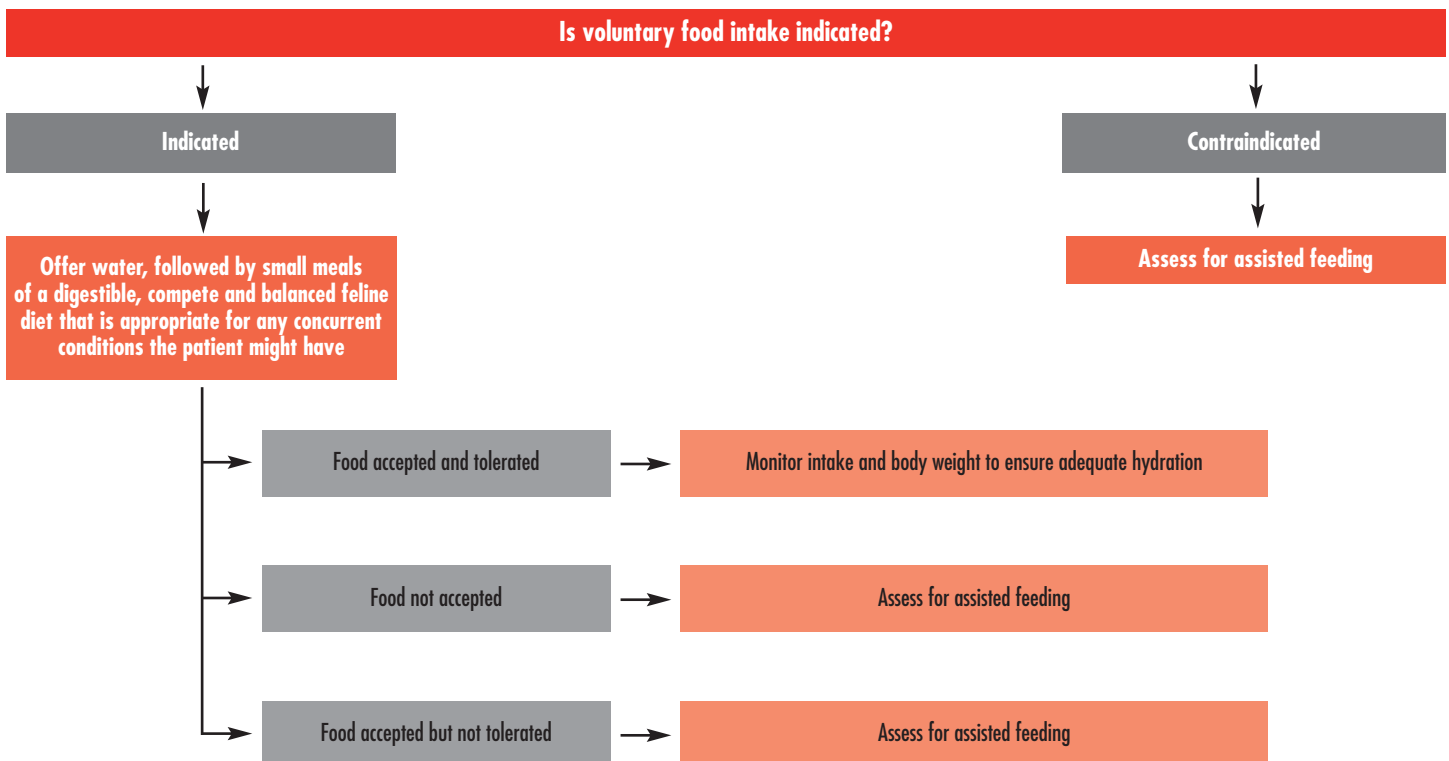
Interacting Medical Management Strategies

Patients with moderate to severe acute pancreatitis will require aggressive fluid resuscitation and supportive care that can include colloid support, antiemetics, gastroprotective agents, and analgesics. While pain control is an important aspect of the medical management of pancreatitis, some analgesic agents can cause gastrointestinal ileus. Ileus can depress a patient’s appetite and therefore delay the return of voluntary intake or complicate the delivery of enteral nutrition in a patient receiving tube feeding. Ileus can be addressed by weaning the patient off of analgesic medication as soon as feasibly possible or switching to a medication that has fewer gastrointestinal side effects.

Monitoring

Patients that either needed to regain or lose weight after discharge from the hospital should have their weight monitored to ensure that appropriate progress is being made. Any future visit to the clinic, regardless of purpose, is an opportunity to inquire about the patient’s dietary management and a chance to reinforce previous feeding recommendations.

Algorithm – Nutritional Management of Feline Pancreatitis



Hepatic Disease – Canine

David C. Twedt, DVM, DACVIM

Definition

Primary liver disease in dogs is generally either acute or chronic. *Acute hepatopathies* are often the result of various drugs, toxins, or secondary to metabolic diseases. If severe, acute hepatopathies can result in liver failure; if less severe, they may be reversible. The most common chronic liver disease in dogs is *chronic hepatitis* (CH), which in some cases can progress to cirrhosis. Abnormal copper accumulation has been implicated in many cases of CH. Congenital *portal systemic vascular shunt* (PSS) anomalies are common in dogs and result in a number of metabolic derangements and *hepatic encephalopathy* (HE).

Key Diagnostic Tools and Measures

The dietary history should be taken and energy requirements for the patient calculated based on ideal body weight (BW). The body condition score (BCS) and BW should be noted. Changes in laboratory tests that reflect liver function (e.g., glucose, albumin, BUN, cholesterol, and serum bile acids) may indicate significant hepatic dysfunction. Diagnosis of HE is based on clinical signs, disease condition, and elevated blood ammonia concentrations.

Pathophysiology

The liver performs a multitude of metabolic processes including the removal of toxic products, storage of nutrients, and the metabolism and regulation of carbohydrates, fats, and proteins. When there is abnormal hepatic metabolism of intestinal nitrogenous byproducts, HE will result. Coagulopathies occur due to a failure of the liver to produce clotting factors and hypoglycemia from altered carbohydrate metabolism. Hypoalbuminemia from decreased liver production will contribute to ascites formation and gastrointestinal ulceration in some patients. Alterations in copper and vitamin metabolism may also occur with various types of liver dysfunction.

Signalment

Liver disease can occur in any breed of dog. Congenital PSS are identified in young, most often small-breed dogs. Acute liver disease from drugs or toxins can occur at any age in any breed. CH is most often seen in middle-aged female dogs (generally from 3 to 10 years of age). Certain breeds of dogs are reported to have metabolic defects in copper metabolism causing a copper-associated CH, including the Bedlington terrier, Doberman pinscher, West Highland white terrier, Skye terrier, Dalmatian, and Labrador retriever.

Key Nutrient Modifications

It is important to ensure there is adequate caloric intake in the dog with liver disease. Diets should be selected for palatability and lipid restriction is not necessary. Carbohydrates should make up no more than 45% of total calories. When hypoglycemia is a concern (i.e., liver failure or PSS) multiple small frequent meals a day may help maintain glucose concentrations and lessen the metabolic impact on the liver. Restricting protein could be detrimental if the dog is in a negative nitrogen balance (i.e., weight loss and hypoalbuminemia). Provide a high quality and highly digestible protein source contributing 15% to 20% on a dry matter basis (DM). Protein restriction should only be instituted in the patient that has clinical evidence of protein intolerance (most often PSS or advanced liver failure). Proteins of milk or plant sources rather than meat-based proteins are suggested. When HE is present dietary protein restriction should be implemented.

In dogs with copper-associated CH, diets containing a high copper content should be avoided (ideally copper should be <5 mg/kg on a dry matter basis). Adequate vitamin supplementation is important because of the important metabolic roles of vitamins in the liver. Animals unwilling to eat often require enteral feeding tubes or parenteral feeding.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	15–30 [#]	3.5–8.0	18	5.1
Carbohydrate	35–50	5–13	n/a	n/a
Fat	10–25	3–6	5	1.4
	mg/kg diet		mg/kg diet	
Copper**	<5.0		7.3	

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary DM and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]When protein restriction is needed due to hepatic encephalopathy, meat proteins should be avoided and total protein limited to 15 to 20% of the diet dry matter.

**For dogs with copper-storage associated liver disease, copper should be restricted. Ideally, in these dogs, copper should be <5 mg/kg on a dry matter basis.

Therapeutic Feeding Principles

The goal of nutritional management of liver disease is basically supportive and requires a fine balance between promoting hepatocellular regeneration and providing nutrients to maintain homeostasis without exceeding the metabolic capacity that will lead to accumulation of toxic metabolites. It is vital that the animal with liver disease has adequate caloric intake in order to minimize catabolism and promote recovery of hepatic function, regeneration, and adequate protein synthesis. If necessary enteral tube feeding or parenteral feeding may be required to meet the patient's caloric requirements.

Next the protein content should be considered and protein restriction initiated only with clinical evidence of protein intolerance (i.e., hepatic encephalopathy). The digestibility of the protein and type of amino acids fed also appear to be important. Diets that are high in aromatic amino acids (AAA) promote the formation of false neurotransmitters and subsequent HE. In general, meat-based proteins are higher in AAA content and should be avoided while dairy- and vegetable-based proteins are higher sources of branched-chain amino acids (BCAA) and lessen the risk of HE.

Soluble fiber will undergo colonic bacterial fermentation producing organic acids lowering of the intraluminal colonic pH and, as a consequence, available acids convert NH_3 to the less readily absorbed NH_4^+ , in essence trapping ammonia in the colon. Fiber also functions as an osmotic laxative reducing absorption of ammonia and related nitrogenous derived encephalopathic factors from the gastrointestinal tract.

Many types of liver disease may benefit from support in the form of nutritional antioxidants. Nutritional supplements given for antioxidant function including vitamin E, zinc, and glutathione precursors such as S-adenosylmethionine (SAME) may be beneficial.

■ **Treats** – Vitamin supplementation is appropriate for dogs with liver disease because there may be either an increased demand for vitamins, altered conversion to the active form of the vitamin, or decreased hepatic storage in these patients. Treats or vitamin-mineral supplements containing copper, however, should be avoided especially for dogs having copper-associated CH. Treats containing poor-quality protein, such as rawhide chews, should be avoided in dogs having protein intolerance.

■ **Tips for Increasing Palatability** – Anorexia is often a concern in animals with liver disease. The cause could be associated with HE, gastrointestinal ulceration, or electrolyte abnormalities. Measures should be first taken to correct these conditions. Next, the palatability of the diet should be considered. There is often a misconception regarding fat content in diets for

liver disease; dogs generally have a good tolerance for fat and fat not only improves the palpability but also is an important source of energy density. Sometimes the use of specialty diets intended for liver disease may be rejected simply due to poor palatability and if so dietary flavorings should be tried.

■ **Diet Recommendations** – Without evidence of protein intolerance, premium commercial diets are recommended. The caloric requirements should be calculated to determine needs and the amount divided into four to six small meals a day. For dogs exhibiting protein intolerance or dogs with advanced liver disease, restricted-protein diets are necessary. Feeding commercial therapeutic diets for liver disease or renal disease is recommended for this purpose. Recipes for home-made diets for liver disease have also been published.

Client Education Points

- Dogs with clinical signs related to PSS that do not undergo surgery to correct the shunt should be fed a modified-protein diet, oral lactulose, and if necessary intestinal antibiotics to control the signs. The long-term prognosis is quite variable.
- CH secondary to copper accumulation should be treated with low-copper diets and copper chelation therapy. Following chelation therapy, oral zinc given at high doses can block further absorption of copper. If diagnosed early and with appropriate therapy, the prognosis is good.
- CH from other causes requires specific therapy and if the etiology is not identified an anti-inflammatory is often used. Dietary manipulation, vitamin supplementation, and antioxidant therapy are recommended for most cases. The prognosis is variable for CH.
- Acute liver toxicity is treated with general support and the prognosis is based on the reversibility of the condition. The use of antioxidants and measures to increase hepatic glutathione concentrations are indicated.

Common Comorbidities

Dogs with PSS often have concurrent urate renal or cystic calculi as a result of elevated blood ammonia concentrations. Removal of current cystic calculi, correction of the PSS, and use of measures to control elevated ammonia concentrations prevent further calculi formation. Both acute and chronic

liver disease can result in HE, GI ulceration, or coagulopathy. Ascites formation can occur with chronic liver disease due to the combination of portal hypertension and hypoalbuminemia. Ascites is treated with diuretics and sodium-restricted diets, such those used for the management of cardiac failure. With advanced acute or chronic liver disease, multi-organ failure can result including renal shutdown and cardiopulmonary failure; when the latter occurs the prognosis is grave.

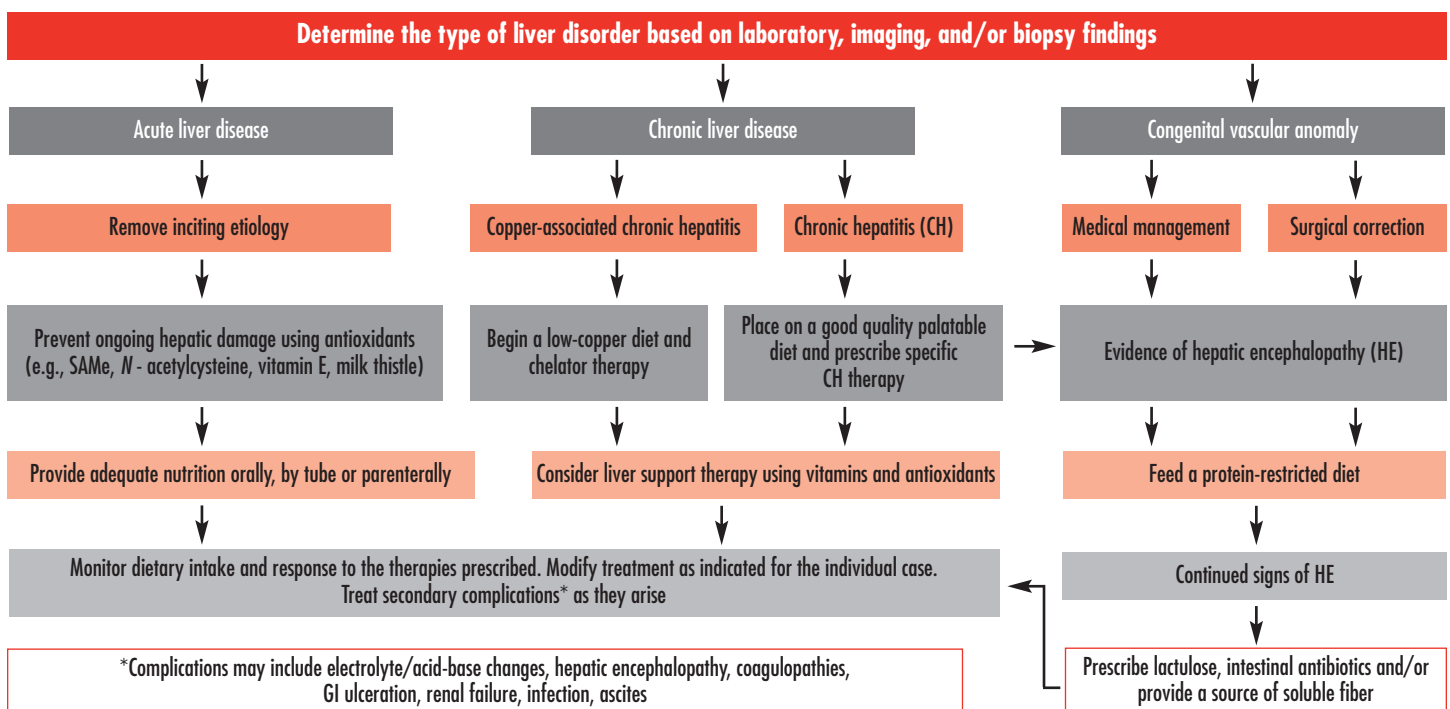
Interacting Medical Management Strategies

Dogs having congenital macroscopic PSS are generally treated surgically. Without surgery cases must be managed nutritionally using protein-restricted diets, soluble fiber source such as Metamucil® (Proctor & Gamble), lactulose, and intestinal antibiotics. Copper-associated CH should be treated with copper chelators such as penicillamine or trientine and diets having lower copper concentrations. With successful chelation therapy zinc supplementation at high concentrations will block intestinal copper absorption by inducing binding of copper to a specific intestinal binding protein in the enterocyte. Specific therapy for animals having CH generally involves anti-inflammatory therapy, such as corticosteroids and or immunosuppressive agents. Ursodeoxycholic acid and antioxidants such as vitamin E, SAMe, and milk thistle derivatives are often used as adjunct therapy. Acute liver toxicity involves removal of the offending agent, treating specific complications of the liver disease and providing support to promote liver regeneration. Supplementation using either SAMe or N-acetylcysteine is a means of providing glutathione, a major intracellular detoxifier. Other antioxidants, such as vitamin E and milk thistle or its derivatives, are often used as supplemental adjunct therapy.

Monitoring

The patient's daily caloric intake, in addition to BW and BCS, should be recorded. The liver enzymes and electrolytes should be evaluated periodically to observe improvement or need for modification of the treatment protocol. Repeat liver biopsies are recommended for CH and copper-associated CH to determine the effectiveness of the therapy and to direct modifications in the treatment protocol

Algorithm – Nutritional Management of Canine Hepatic Disease



Hepatic Disease – Feline

David C. Twedt, DVM, DACVIM

Definition

The two most common feline hepatic disorders include *lipidosis* and inflammatory (*cholangitis*) liver disease. Hepatic lipidosis occurs either as a primary idiopathic syndrome or secondary to other conditions causing inadequate nutritional intake or lipid metabolism.

Key Diagnostic Tools and Measures

The dietary history should be taken and energy requirements for the patient calculated based on ideal body weight (BW). The body condition score (BCS, see Appendix I) and BW should be noted. Cats with lipidosis frequently were obese prior to sudden weight loss and the current BW or BCS may not reflect severity of the disease so percent BW loss occurring from illness should also be calculated.

Pathophysiology

The high protein requirements in cats must be met to prevent breakdown and mobilization of muscle protein for energy. Specific nutritional deficiencies may include cobalamin, arginine, taurine, and possibly carnitine. Hypophosphatemia, hypokalemia, and hypomagnesemia can also occur in the anorexic cat.

Signalment

Cholangitis occurs in middle-aged to older cats and some may have concurrent chronic pancreatitis or inflammatory bowel disease. Secondary lipidosis is found in older cats as a result of another disease. Idiopathic lipidosis occurs in middle-aged to older cats with obesity a major risk factor for this condition.

Key Nutrient Modifications

An esophageal feeding tube should be placed to assure adequate caloric intake in an anorexic cat with hepatic lipidosis. The caloric needs are calculated based on ideal BW and the patient is fed a blenderized nutritionally balanced diet that meets the cat's caloric and protein requirements. In cats, approximately 50 to 55 kcal/kg BW is a good starting point for caloric needs. Dietary protein and fat restriction is generally not necessary in cats with hepatic lipidosis. When protein restriction is necessary (i.e., hepatic encephalopathy is suspected) the dietary protein content fed should be reduced to 25% to 30% on a dry matter basis. The feeding volume begins low and is increased over 3 to 4 days to reach calculated nutritional requirements. Electrolytes potassium, phosphorus, and magnesium are supplemented in the fluids. These electrolytes should be monitored closely in the first few days because a refeeding syndrome may occur with these electrolytes becoming dangerously low.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein [#]	30–45	7–12	28	6.5

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials.

[#]Protein should only be restricted when necessary to manage hepatic encephalopathy. In these patients, protein may be restricted to 25 to 30% of the diet dry matter (6 to 8 g/100 Kcal)

Therapeutic Feeding Principles

The nutritional recommendations for idiopathic hepatic lipidosis in cats are completely empirical and not well documented. Force-feeding a diet often results in inadequate caloric intake, undue stress, and food aversion and should be avoided. Numerous reports suggest feeding various diets (with a variety of protein and fat content recommendations) and numerous dietary supplements. Commercial feline diets or nutritional support diets formulated suitable for tube feeding are generally used. Some recommend L-carnitine supplementation for cats with hepatic lipidosis at 250 mg/cat/day, but additional studies are necessary to determine the benefit. Arginine levels in commercial diets should meet the minimum dietary allowance for adult maintenance (>1.0% of the diet DM per day) and consequently supplementation is not necessary. Some suggest arginine (1,000 mg/day), thiamine (100 mg/day), and taurine (500 mg/day) for 3 to 4 weeks. Cats with hepatic disease and especially those with lipidosis may develop a cobalamin deficiency; cobalamin should be given (250 µg) subcutaneously weekly until concentrations reach normal and the cat is eating on its own. B-complex vitamin supplementation is recommended as well. Other supplements to consider include S-adenosylmethionine (SAME) and silymarin (milk thistle products) for their antioxidant effects.

■ **Treats** – Supplemental treats generally are not appropriate for cats with liver disease or lipidosis.

■ **Tips for Increasing Palatability** – During the recovery period of tube feeding, a highly palatable diet should be offered prior to feeding through the tube. Only when adequate nutrition is taken voluntarily can the feeding tube be removed. Improving palatability by warming the food, offering the original diet, or using taste enhancers such as tuna juice should be tried.

■ **Diet Recommendations** – Diets include commercial therapeutic diets for feline gastrointestinal or liver disease or feline maintenance diets. When protein restriction is required, feeding a specialty renal diet with a protein content of 25% to 30% DM is suggested. When nasoesophageal tube feeding is necessary, a liquefied diet must be used. The amount fed is based on calculated caloric requirements.

Client Education Points

- Inflammatory liver disease (cholangitis) in cats often tends to wax and wane with flare-ups associated with anorexia and vomiting.
- Hepatic lipidosis can occur secondary to many other diseases and the prognosis for recovery is in part dependent on the nature of the primary disease.
- Idiopathic hepatic lipidosis requires aggressive nutritional management and owner compliance for home feeding. The prognosis is good for most cases.

Common Comorbidities

Cholangitis frequently is associated with chronic pancreatitis and/or inflammatory bowel disease. Although the therapy for these concurrent diseases is often similar to that of cholangitis, one should refer to the chapters on management of those specific disorders. Secondary hepatic lipidosis requires specific management of the primary disease with careful attention to nutritional management as described for the idiopathic form of hepatic lipidosis.

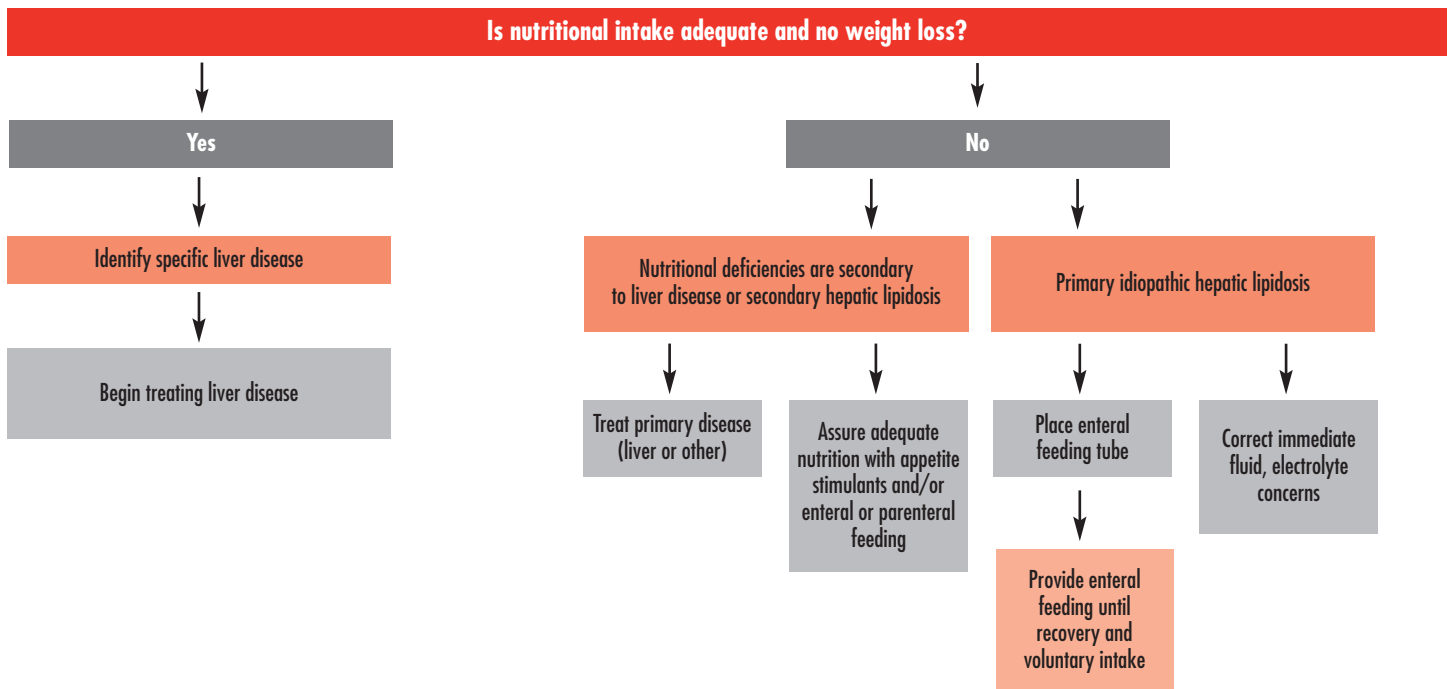
Interacting Medical Management Strategies

Cats with liver disease due to cholangitis are often treated with antibiotics, corticosteroids, and ursodeoxycholic acid. Vomiting is a frequent complication and the use of antiemetics for nausea and vomiting may improve nutritional intake of the patient. Nutritional aspects are paramount in the management of idiopathic hepatic lipidosis. Concurrent use of appetite stimulants is not recommended because of potential side effects and the fact that they are rarely effective in assuring adequate nutritional intake.

Monitoring

The patient's daily caloric intake in addition to BW and BCS should be recorded. Liver enzymes and electrolytes should be evaluated periodically to observe improvement or need for supplementation. Cobalamin concentrations should be determined monthly or until patient is eating on its own.

Algorithm – Nutritional Management of Feline Hepatic Disease



Hepatic Encephalopathy – Canine

David C. Twedt, DVM, DACVIM

Definition

Hepatic encephalopathy (HE) may be defined as a disturbance in central nervous system function resulting from nitrogenous substances derived from the gastrointestinal tract that gain access to the brain from decreased hepatic function or portal-systemic shunting of blood. HE can occur in dogs secondary to congenital portal systemic vascular shunts (PSS) or acute or chronic liver failure.

Key Diagnostic Tools and Measures

Diagnosis of HE is based on history, clinical signs, and elevated blood ammonia concentrations. Laboratory and imaging testing is required to determine if HE is the result of liver failure or PSS. Precipitating factors that can promote HE (see below) should also be investigated. The body condition score (BCS) and body weight (BW) should be noted and the energy requirements for the patient should be calculated based on ideal BW.

Pathophysiology

HE results from nitrogenous substances absorbed from the intestine that without adequate liver metabolism enter the brain and produce alterations of neurotransmission affecting consciousness and behavior. Ammonia is a key factor but other gut-derived toxins include benzodiazepine-like substances, short- and medium-chain fatty acids, phenols, and mercaptans. Alterations in the ratios of aromatic amino acids (AAA) to branched-chain amino acids (BCAA) in the brain may also contribute to HE as increased concentrations of AAA are thought to promote the formation of false neurotransmitters.

Signalment

Congenital PSS are usually identified in young, small-breed dogs. Acute liver disease can occur at any age or in any breed. Chronic liver disease is most often identified in older dogs. Certain breeds are at risk including the Bedlington terrier, Doberman pinscher, West Highland white terrier, Skye terrier, Dalmatian, Labrador retriever, standard poodle, and cocker spaniel.

Key Nutrient Modifications

Diet is key to the management of HE and should be selected to reduce the nitrogenous load in the gut by protein restriction. Severe protein restriction should be avoided but rather a moderate dietary protein content diet should be fed. Protracted nitrogen restriction contributes to malnutrition and is detrimental. A positive nitrogen balance can often be achieved in conjunction with adjunct HE therapy. A high quality highly digestible protein source contributing 15 to 20% of dry matter basis (DM). Vegetable and dairy sources are preferable to animal protein as they provide a higher calorie to nitrogen ratio and tend to be higher in BCAA than AAA. Further protein restriction should only be instituted in the patient that has clinical evidence of protein intolerance despite adequate HE therapy. Diets containing or supplemented with soluble fiber, a substrate for colonic bacteria, and subsequent colonic acidification may also be of benefit.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
Recommended dietary level		Minimum dietary requirement*		
Protein	15–20	3.5–6.0	18	5.1
Carbohydrate	50–65	10–15	n/a	n/a
Fat	15–30	3–7	5	1.4
Fiber [#]	2–4	0.3–2.0	n/a	n/a
	mg/kg diet		mg/kg diet	
Copper**	<5.0		7.3	

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary DM and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]Soluble fibers are preferred. The crude fiber analysis includes most insoluble fibers, but does not include soluble fibers. Therefore, crude fiber has limited usefulness when evaluating the total fiber content of foods. The ingredient list should be evaluated for sources of soluble fiber.

**For dogs with copper-storage associated liver disease, copper should be restricted. Ideally, in these dogs, copper should be <5 mg/kg on a dry matter basis.

Therapeutic Feeding Principles

The nutritional recommendation for the patient suffering from HE involves modification of the diet to limit production of nitrogenous byproducts that when absorbed fail to be metabolized by the liver and contribute to HE. First, it is important to assure that the dog is consuming adequate calories as this will minimize catabolism and promote recovery of hepatic function, regeneration and adequate protein synthesis. Next the protein content should be considered. Protein restriction is generally always initiated with clinical evidence of HE. The protein fed should be highly digestible and 15% to 20% of the DM fed. Diets high in aromatic amino acids (AAA) that promote the formation of false neurotransmitters should be avoided, while dairy- and vegetable-based proteins are higher sources of branched-chain amino acids (BCAA) and can lessen HE. Soluble fiber undergoes colonic bacterial fermentation producing organic acids. Fermentation promotes the conversion of luminal ammonia (NH₃) to ammonium (NH₄⁺), which, by virtue of its net charge, is less readily absorbed into the bloodstream. Fiber also functions as an osmotic laxative reducing absorption of ammonia and related nitrogenous derived encephalopathic factors from the GI tract. Fermentable fiber such as psyllium (1–3 teaspoons per meal) can be supplemented if fermentable fiber is not present in the diet fed.

Adequate vitamin supplementation, given as a B-complex product, is suggested due to their important metabolic roles vitamins play in the liver. Many types of liver disease may benefit from support in the form of nutritional antioxidants. Nutritional supplements given for antioxidant function include vitamin E, zinc, and glutathione precursors such as S-adenosylmethionine (SAMe) and may be beneficial. Zinc, a cofactor of urea cycle enzymes, is often deficient in humans with HE and supplementation could prove beneficial although this has not been documented in dogs. Feeding multiple small frequent meals a day may help maintain glucose concentrations and lessen the metabolic impact on the liver at one time. If the animal is anorexic enteral tube feeding or parenteral feeding may be required to meet the patient's nutritional requirements.

■ **Treats** – Treats generally are not recommended. Giving low-protein

treats or predominantly carbohydrate-based treats such as raw vegetables would be acceptable. Treats or vitamin-mineral supplements containing copper should be avoided. Rawhide or other chews should be avoided in most cases as they provide minimal nutritional benefit.

■ **Tips for Increasing Palatability** – Palatability of the diet is extremely important. First, one should investigate for liver-associated conditions that could contribute to anorexia such as gastrointestinal ulceration or electrolyte abnormalities and if identified they should be corrected. Dietary fat improves palatability and there is no need to restrict fat content in the patient having liver disease and HE. Fat not only improves the palatability but also is an important source of energy density. Warming the food or adding flavorings also may also be helpful.

■ **Diet Recommendations** – Recommended diets to feed the patient suffering from HE include the specialty liver or renal diets. Both contain highly digestible moderate-protein diets. Some geriatric diets or the hydrolyzed diets may also meet nutritional goals in HE therapy. The caloric requirements should be calculated to determine the patient’s needs and the amount divided into four to six small meals a day. Animals exhibiting protein intolerance and worsening clinical signs will require lower-protein diets and additional therapy for HE. Resources are also available on home-cooked diets to use for dogs with liver disease and HE.

Client Education Points

- For dogs with congenital PSS that have clinical signs of HE, the general recommendation is surgery to correct the anomaly. Dogs that do not have surgery must be treated medically, which includes feeding a modified-protein diet, oral lactulose, and if necessary intestinal antibiotics to control the signs. The long-term prognosis is quite variable.
- Acute liver disease that causes HE is generally severe and has a guarded prognosis. Acute liver disease has the potential to be reversible, however, if vital metabolic functions can be maintained until the liver has time to regenerate. Therapy involves providing basic liver support and treating complications as they occur.
- The prognosis for chronic liver disease is grave. When HE occurs in chronic liver disease, cirrhosis generally is present. In this situation therapy is only supportive, treating complications of chronic liver disease. With appropriate HE therapy the patient may improve in the short term.

Common Comorbidities

Dogs having congenital PSS often have concurrent urate renal or cystic calculi as a result of elevated blood ammonia concentrations. Removal of current cystic calculi, surgical correction of the PSS, and using measures to control elevated ammonia concentrations prevents further calculi formation. Occasionally dogs having one congenital anomaly will have additional anomalies.

Both acute and chronic liver disease can result in HE, GI ulceration, or a coagulopathy. Ascites formation can occur with chronic liver disease due to the combination of portal hypertension and hypoalbuminemia. Ascites is treated with diuretics and sodium-restricted diets such as those used for the management of cardiac failure. With advanced acute or chronic liver disease multi-organ failure can result including renal shutdown and cardiopulmonary failure; when the latter occurs the prognosis is grave.

Interacting Medical Management Strategies

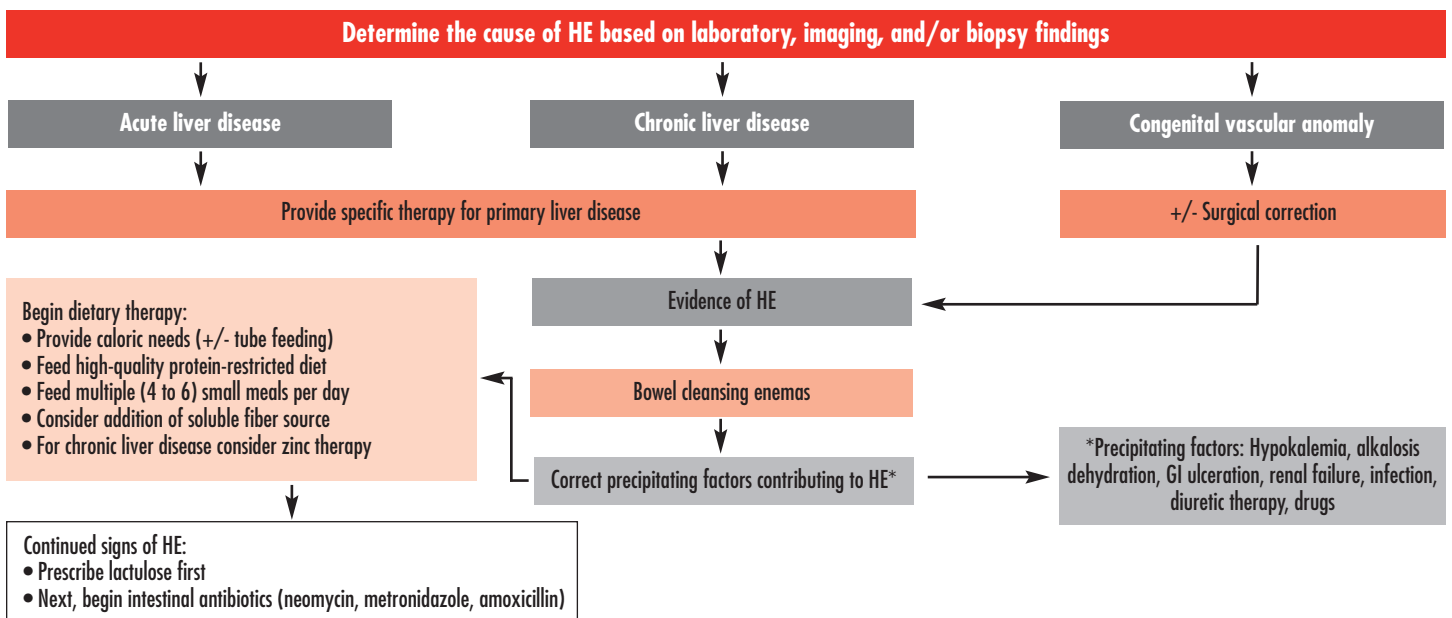
The medical management of HE usually always involves additional therapy beyond dietary manipulation using protein-restricted diets and a soluble fiber source. With acute HE, bowel cleansing is a mainstay of therapy because colonic evacuation enemas remove intestinal nitrogenous substrates. Chronic management of HE usually requires the use of oral lactulose and intestinal antibiotics. Precipitating factors in HE, including hypoglycemia, hypokalemia, alkalosis and gastrointestinal ulceration, should be avoided or prevented.

There is evidence in human cirrhotic patients with HE that zinc, a cofactor of urea cycle enzymes, may be deficient especially if associated with concurrent malnutrition. Zinc supplementation may be beneficial in chronic liver disease in dogs but this has not been documented.

Monitoring

The patient should be consuming adequate calories and specific therapies for the primary disease condition should be instituted. The clinical and neurologic status should be improved with appropriate diet and lactulose therapy. If these measures fail to improve the patient’s status then intestinal antibiotics should be initiated. Precipitating factors in HE should be excluded with a biochemical profile including electrolytes and with a fecal occult blood analysis. If abnormalities are identified they should be treated.

Algorithm – Nutritional Management of Canine Hepatic Encephalopathy (HE)



Hepatic Encephalopathy – Feline

David C. Twedt, DVN, DACVIM

Definition

Hepatic encephalopathy (HE) is a disturbance in function of the central nervous system resulting from nitrogenous substances derived from the gastrointestinal tract that gain access to the brain from decreased hepatic function or portal-systemic shunting of blood. HE is uncommon in cats but can occur as a result of congenital portal systemic shunts (PSS), idiopathic hepatic lipidosis (IHL), acute liver failure, and rarely from chronic inflammatory liver disease.

Key Diagnostic Tools and Measures

Diagnosis of HE is based on history, clinical signs, and elevated blood ammonia concentrations. Hypersalivation and seizures are common signs of HE in cats. Laboratory testing and imaging is required to determine if HE is the result of liver failure or PSS. Precipitating factors that can promote HE (see below) should also be investigated. The body condition score (BCS) and body weight (BW) should be noted and the energy requirements for the patient should be calculated based on ideal BW.

Pathophysiology

HE results from nitrogenous substances absorbed from the intestine that without adequate liver metabolism enter the brain and produce alterations of neurotransmission affecting consciousness and behavior. Ammonia is a key factor but other gut-derived toxins include benzodiazepine-like substances, short- and medium-chain fatty acids, phenols, and mercaptans. Alterations in the ratios of aromatic amino acids (AAA) to branched-chain amino acids (BCAA) in the brain may also contribute to HE as increased concentrations of AAA are thought to promote the formation of false neurotransmitters.

Signalment

Congenital PSS are usually identified in young cats. Some are reported to have unusual copper-colored irises. Acute liver disease can occur at any age or in any breed. Idiopathic hepatic lipidosis occurs in middle-aged obese cats. Chronic liver disease occurs in older cats with no breed predilection.

Key Nutrient Modifications

Diet for the management of HE should be selected to reduce the nitrogenous load in the gut. In most cases protein restriction should be avoided when feeding cats with HE because of their high protein requirements. A high-quality, highly digestible moderate dietary protein source contributing 25 to 30% on a dry matter basis (DM) is recommended instead. Vegetable and dairy sources of protein are preferable to animal protein as they provide a higher calorie to nitrogen ratio and tend to be higher in BCAA than AAA.

Cats with IHL often require B vitamin (including cobalamin) supplementation. Other supplements suggested include carnitine, arginine, thiamine, and taurine although their importance in various types of liver disease is not well documented. Carnitine functions to transport fatty acids into hepatic mitochondria for energy production. Arginine is an essential amino acid that must be derived from the diet and plays an important role in the urea cycle of cats. Taurine is also an essential nutrient for cats and is involved in CNS function and taurine deficiency could be confused with signs associated with HE.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein [#]	25–30	5–8	28	6.5
Fat	20–40	4–7	9	2.3
Carbohydrate	30–50	4–12	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary DM and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]Meat-based diets should be avoided. Highly digestible milk and plant based proteins are preferred when managing hepatic encephalopathy.

Therapeutic Feeding Principles

Nutrition is the key to the management of cats suffering from IHL as well as other causes of HE. A catabolic state develops quickly in the anorexic cat and prompt measures should be taken to correct this condition. Generally placement of a gastrointestinal (GI) feeding tube is indicated for management of the anorexic cat to meet nutritional requirements. Nasoesophageal, esophageal, or gastrostomy tubes are used for this purpose. Next the caloric needs should be calculated for the patient based on optimal BW; generally feeding 50 to 55 kcal/kg BW is adequate for most cats. Feeding the calculated caloric requirements will promote recovery of hepatic function, regeneration, and adequate protein synthesis.

The amount and protein content of the diet should be considered when HE is present. Unlike dogs, however, protein restriction is rarely initiated in cats with clinical evidence of HE because of the cat's innate protein requirements. The protein fed should be highly digestible and make up 25% to 30% DM. Meat-based proteins are higher in AAA content and should be avoided while dairy- and vegetable-based proteins are higher sources of branched-chain amino acids (BCAA) and can lessen HE. Diets high in fiber generally should be avoided for cats because they decrease the nutrient density of the diet.

Adequate vitamin supplementation, given as a B-complex product, is suggested due to the important metabolic roles vitamins play in the liver. Many types of liver disease may benefit from support in the form of nutritional antioxidants. Nutritional supplements given for antioxidant function including vitamin E and glutathione precursors such as S-adenosylmethionine (SAMe) may be beneficial. Feeding multiple small frequent meals a day may help maintain glucose concentrations and lessen the metabolic impact on the liver at one time.

■ **Treats** – Treats are generally not recommended or given to cats with liver disease.

■ **Tips for Increasing Palatability** – Palatability of the diet is extremely important. First, investigate for liver-associated conditions that could be contributing to anorexia such as GI ulceration or electrolyte abnormalities and correct as needed. Dietary fat improves palatability and there is no need to restrict fat content in the cat with IHL or other causes of HE. Fat also is an important source of energy density. Warming the food or adding flavorings may be helpful. Force-feeding anorexic cats with IHL should be avoided as it may cause food aversions.

■ **Diet Recommendations** – Recommended diets for the patient suffering from HE include the specialty liver or renal diets, both of which contain highly digestible moderate proteins. If tube feeding is required, use

specialty high-density diets formulated for such use. Nasoesophageal feeding tubes require a liquefied feline formula. The caloric requirements should be calculated to determine the patient's needs and the amount divided into four to six small meals a day. Animals exhibiting protein intolerance and worsening clinical signs will require lower-protein diets and additional therapy for HE. Home-cooked diets for cats generally are not recommended because of the cat's unique nutrient requirements.

Client Education Points

- For cats with congenital PSS that have clinical signs of HE, the general recommendation is surgery to correct the anomaly. Cats that do not have surgery must be treated medically, which includes diet, oral lactulose, and if necessary intestinal antibiotics to control the signs. The long-term prognosis is quite variable.
- Acute liver failure causing HE is generally severe and has a guarded prognosis. Acute liver disease has the potential to be reversible, however, if vital metabolic functions can be maintained until the liver has time to regenerate.
- Cats having IHL require aggressive nutritional management and the owner must be involved in tube feeding at home until the cat begins eating on its own. The prognosis generally is good for these cases. If hepatic lipidosis occurs secondary to other conditions causing anorexia, the prognosis generally is not as good and is based on the underlying condition.
- The prognosis for chronic liver disease causing HE is grave. When HE occurs in chronic liver disease, cirrhosis generally is present. In this situation therapy is only supportive and involves treating complications of chronic liver disease. With appropriate HE therapy the patient may be improved in the short term.

Common Comorbidities

Cats with congenital PSS may have seizures associated with elevated blood ammonia concentrations. Occasionally seizures occur following surgical correction of the shunt and may require anticonvulsant therapy.

Cats with IHL occasionally may experience a refeeding syndrome, a

condition that results in metabolic electrolyte disturbances. With the introduction of food insulin secretion increases and causes intracellular uptake of phosphorus, potassium, and magnesium. Hypophosphatemia can result in muscle weakness and hemolytic anemia. Slow introduction of food and correction of electrolytes prevents the refeeding syndrome. Hyperglycemia and glucose intolerance are common in cats having IHL or other liver disorders and can be lessened by decreasing the carbohydrate content of the diet.

Chronic inflammatory liver disease (cholangitis) is often associated with concurrent chronic pancreatitis and or inflammatory bowel disease. On rare occasion chronic pancreatitis can result in exocrine pancreatic insufficiency requiring pancreatic enzyme supplementation.

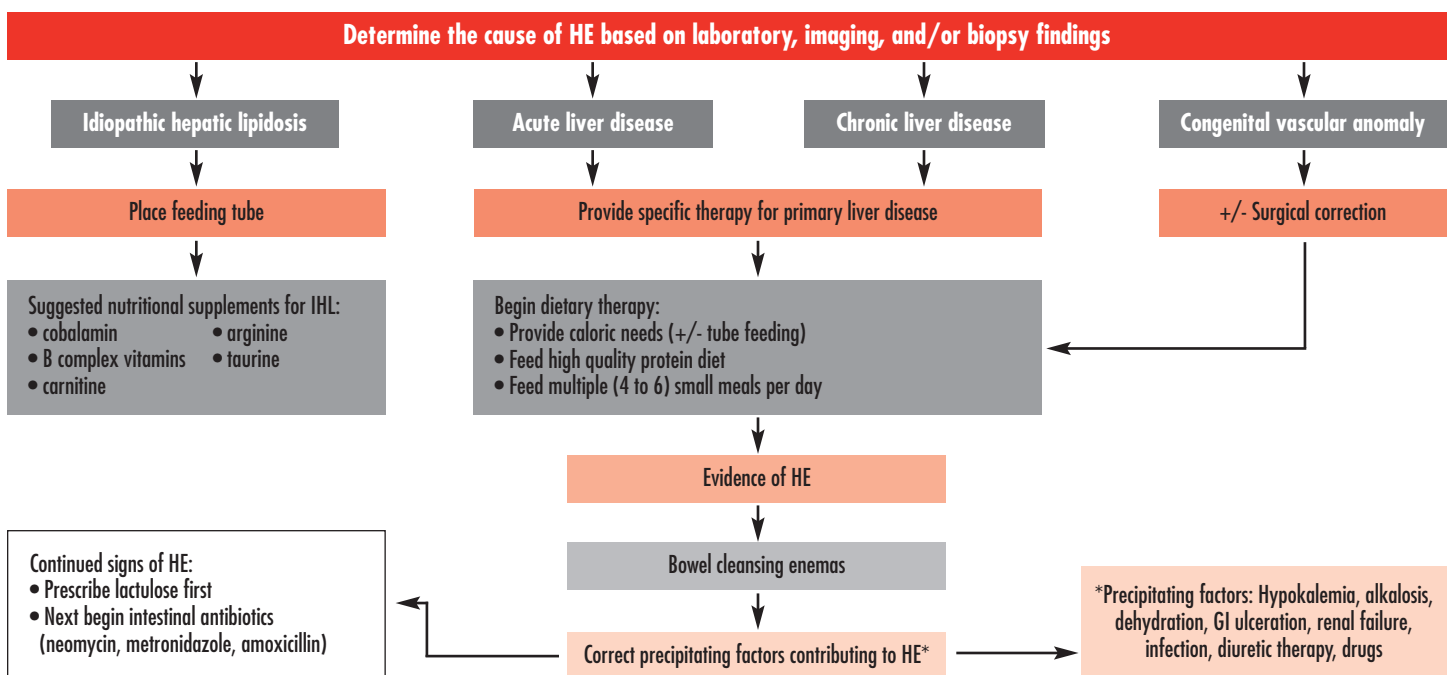
Interacting Medical Management Strategies

The medical management of HE usually involves additional therapy beyond dietary manipulation. If the initial diet results in continued signs of HE then further protein restriction should be instituted. With acute HE bowel cleansing is a mainstay of therapy because colonic evacuation enemas remove intestinal nitrogenous substrates. Chronic management of HE usually requires the use of oral lactulose and intestinal antibiotics. Lactulose can also be used in the final evacuation enema. Precipitating factors in HE including hypoglycemia, hypokalemia, alkalosis, and GI ulceration should be avoided or prevented. Chronic inflammatory liver disease is usually treated with corticosteroids and may result in glucose intolerance.

Monitoring

The patient should be consuming adequate calories and specific therapies for the primary disease condition should be instituted. The clinical and neurologic status should be improved with appropriate diet and lactulose therapy. If these measures fail to improve the patient status then intestinal antibiotics should be initiated. Precipitating factors in HE should be excluded with a biochemical profile including electrolytes and with a fecal occult blood analysis. If abnormalities are identified they should be corrected.

Algorithm – Nutritional Management of Feline Hepatic Encephalopathy (HE)



Hyperlipidemia – Canine

John E. Bauer, DVM, PhD, DACVN

Definition

Hyperlipidemia is defined as an increase in serum concentrations of triglyceride (TG), cholesterol, or both.

Key Diagnostic Tools and Measures

The presence of lipemic serum suggests hypertriglyceridemia but not hypercholesterolemia. In some cases, the triglyceride concentrations may be sufficiently elevated (typically >1000 mg/dL) and lipoprotein particles large enough to impart an opaque or milky appearance (*lactescence*). Pure hypercholesterolemia does not impart lactescence. Blood samples to confirm should be collected after a 12- to 18-hour fast. Refrigeration of the sample for 12 hours is helpful to determine whether the triglyceride is associated with postprandial (chylomicron) particles or endogenous (very low density lipoprotein [VLDL]) particles. Chylomicrons are less dense and will float under this condition while VLDL will remain suspended in the sera.

Pathophysiology

The most common type is *postprandial hyperlipidemia*; this is a normal phenomenon resulting from the appearance of chylomicrons in the circulation 2 to 6 hours after fat ingestion and usually resolving after approximately 10 hours. *Primary hyperlipidemias* may result from an inherited defect of lipoprotein metabolism (e.g., primary/familial hyperlipidemia including idiopathic forms). *Secondary hyperlipidemias* may exist in which triglyceride elevations develop due to increased chylomicron or VLDL production, decreased catabolism, or a combination of both. Cholesterol elevations are more typically associated with low-density lipoprotein (LDL) or high-density lipoprotein (HDL) elevations but rarely result in lactescent serum

Signalment

Miniature schnauzers are particularly susceptible to idiopathic forms of hypertriglyceridemia. Reports also exist in beagles and various terrier breeds. Idiopathic hypercholesterolemias have been described in Doberman pinschers and Rottweilers

Key Nutrient Modifications

Key nutrients for management of primary hyperlipidemias include low-fat diets (< 20% of metabolic energy) and increased long-chain omega-3 fatty acid intake. In the case of secondary forms (i.e., comorbidities), attempts should be made to identify the underlying cause (see below) and institute dietary management and other techniques to control the metabolic disturbances identified.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Fat	5–10	1.2–2.3	5.0	1.4
DHA+EPA [#]	0.3–1.25	75–330	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]Docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), long-chain omega-3 fatty acids from fish oil. Standard fish oil contains 30% EPA + DHA.

Therapeutic Feeding Principles

Diet histories should be obtained focusing on dietary fat content. Initial client discussion will ideally focus on efforts to restrict dietary fat content in the existing diet (elimination of treats or table scraps) and other recommendations made to restrict fat, such as a total diet change. Numerous therapeutic diets are available including those formulated primarily for obesity management, gastrointestinal disorders, and even hypoallergenic diets as long as they are reduced in dietary total fat.

Diets rich in marine omega-3 fatty acids may be especially helpful but only in the context of an overall low-fat dietary approach. Supplementation of existing low-fat diets with fish oils may be more appropriate rather than feeding a high-fat diet containing the omega-3 oils, which may preclude any benefit. Marine (or long-chain omega-3 fatty acids) are necessary rather than vegetable omega-3s because the triglyceride-lowering effect is primarily due to docosahexaenoic acid (DHA), which is poorly synthesized from vegetable based precursors.

The inclusion of soluble dietary fiber interferes with the enteric reabsorption of cholesterol and bile acids and may help reduce hypercholesterolemic states. Dry diets are usually lower in cholesterol than canned diets and may be lower in total fat overall. Table scraps, which are often high in fat, should be eliminated.

■ **Treats** – Treats are not recommended unless they are known to be low in fat. Human foods such as vegetables or low-fat crackers may be substituted.

■ **Tips for Increasing Palatability** – Obviously, the use of dietary oils to improve palatability is contraindicated. Moistening dry-expanded type foods is suggested, or a diet change within the low-fat category can be made. Using a low-fat canned food top-dressed on dry food may improve acceptance in some cases as long as total fat and calories remain controlled.

■ **Diet Recommendations** – Several forms of low-fat diets are available primarily as therapeutic diets. Both canned and dry-extruded varieties exist but fat content of these, even with the same product name, may be different so caution is urged in selecting the most appropriate commercial product. Canned products often have a higher fat content even though they have the same product name as the dry version, yet these may be a useful alternative in some instances. Amounts to be fed include those necessary to maintain a healthy non-obese body weight. In the case of obese animals, efforts to reduce caloric intake should be made. Home-prepared diets may be a necessary alternative in cases in which response to commercial low-fat diets is unsatisfactory.

Client Education Points

- Adherence to a low-fat diet, including all foods regularly consumed, such as human and pet food–based treats, is required.
- Total caloric intake, even using low-fat foods, should be discussed and alternatives suggested.
- Dogs will typically consume fish oil capsules as a treat, and these capsules may be a suitable treat substitute.

Common Comorbidities

Diseases associated with secondary hyperlipidemias include diabetes mellitus, hypothyroidism, hyperadrenocorticism, cholestasis, and nephrotic syndrome, or may be drug related (megestrol acetate, corticosteroids, anti-epilepsy drugs). Hypertriglyceridemia related to diabetes mellitus can be attributed to decreased lipoprotein lipase activity and decreased clearance from sera or increased production. Lack of insulin may also stimulate lipolysis and increase cholesterol genesis. Hyperadrenocorticism may induce hypertriglyceridemia due to increased lipid mobilization from storage sites mediated by stimulation of tissue hormone-sensitive lipase activities. Glucocorticoids may also inhibit lipoprotein lipase, reducing triglyceride clearance. Hypothyroidism is the most common cause of hypercholesterolemia. Dogs being treated to manage seizure activities often are presented with hypertriglyceridemias. Hypertriglyceridemia is a risk factor for pancreatitis.

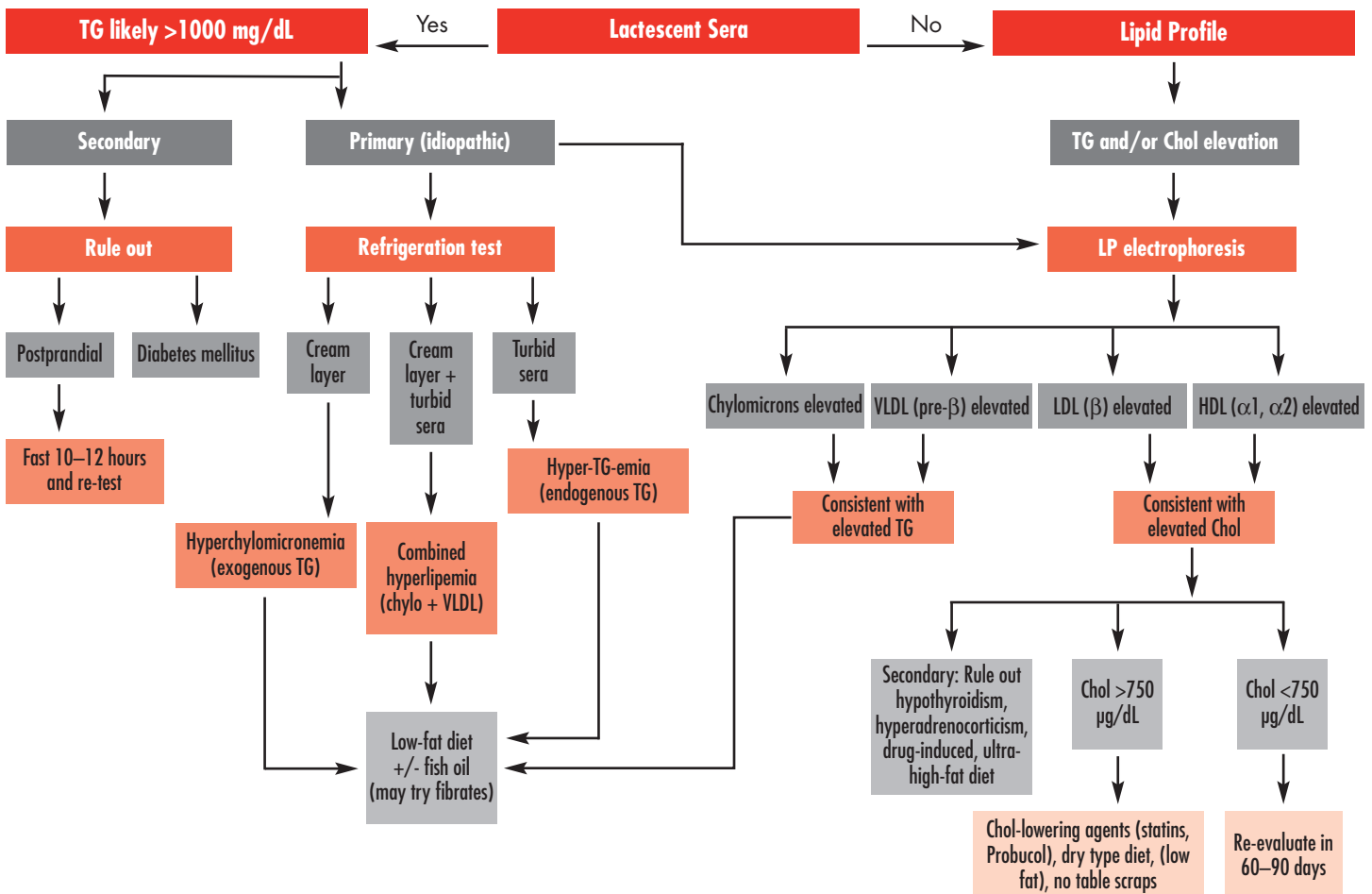
Interacting Medical Management Strategies

Several drug therapies may be useful in treating hyperlipemias but all may have undesirable side effects or have not been fully explored in dogs. Generally, dogs with triglyceride concentrations <500 mg/dL should not be treated pharmacologically. **Niacin** (100 mg/day) may reduce fatty acid release from adipocytes with resultant decrease in VLDL synthesis but may result in facial pruritus, erythema, and vomiting. The **fibrate derivatives** stimulate lipoprotein lipase activity and may be used at 10 mg/kg body weight twice daily; however, vomiting and diarrhea may also occur. The **statins** suppress cholesterol synthesis but adverse effects such as lethargy, muscle pain, and elevated liver enzymes may occur. **Probucol** is a cholesterol-lowering drug but it has been associated with arrhythmias. **Cholestyramine**, a bile acid sequestrant, may be effective in lowering serum cholesterol (1–2 g per os twice a day). Constipation may occur as well as increased VLDL and triglyceride synthesis. Hypercholesterolemia, when less than approximately 750 mg/dL generally poses no health risk so treatment efforts remain best focused on dietary fat reduction and fish oil supplementation.

Monitoring

Monitoring involves serum chemistry determinations including liver enzymes and lipid measurements.

Algorithm – Nutritional Management of Canine Hyperlipidemia



Hyperlipidemia – Feline

John E. Bauer, DVM, PhD, DACVN

Definition

Hyperlipidemia is defined as an increase in serum concentrations of triglyceride (TG) or cholesterol or both.

Key Diagnostic Tools and Measures

The presence of lipemic serum suggests hypertriglyceridemia but not hypercholesterolemia. In some cases, the triglyceride concentrations may be sufficiently elevated (typically >1000 mg/dL) and lipoprotein particles large enough to impart an opaque or milky appearance (*lactescence*). Pure hypercholesterolemia does not impart lactescence. Blood samples to confirm should be collected after a 12- to 18-hour fast. Refrigeration of the sample for 12 hours is helpful to determine whether the triglyceride is associated with postprandial (chylomicron) particles or endogenous (very low density lipoprotein [VLDL]) particles. Chylomicrons are less dense and will float under this condition while VLDL will remain suspended in the sera. Waxing and waning vomiting, diarrhea, or abdominal discomfort may be present.

Pathophysiology

The most common cause is *postprandial hyperlipidemia*. This is a normal phenomenon resulting from the appearance of chylomicrons in the circulation 2 to 6 hours after fat ingestion and usually resolving after approximately 10 hours. One recognized *primary hyperlipidemia* of cats results from an inherited defect of lipoprotein lipase metabolism. In this case, reduced clearance (catabolism) of triglyceride-rich postprandial (i.e., chylomicron) and/or endogenous (i.e., VLDL) particles occurs. *Primary hypercholesterolemia* also exists; cholesterol elevations are typically associated with low-density lipoprotein (LDL) or high-density lipoprotein (HDL) elevations but rarely result in lactescent serum.

Cutaneous xanthomas due to lipid-laden macrophages and foam cells are the most common manifestation of hyperlipidemias in cats. Severe hypercholesterolemia may be associated with lipemia retinalis, arcus lipoides, and, more rarely, atherosclerosis. *Secondary hyperlipidemias* may exist in which triglyceride elevations develop due to increased chylomicron or VLDL production, decreased catabolism, or a combination of both due to some other primary disorder

Signalment

No breed predispositions have been identified to date.

Key Nutrient Modifications

Key nutrients for management of primary hyperlipidemias include low-fat diets (< 20% of metabolic energy) and increased long-chain omega-3 fatty acid intake. In the case of secondary forms (i.e., comorbidities), attempts should be made to identify the underlying cause (see below) and institute dietary management and other techniques to control the metabolic disturbances identified.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Fat	5–10	1.2–2.3	9.0	2.2
		mg/100 kcal		
DHA+EPA#	0.3–1.25	75–330	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

#Docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), long-chain omega-3 fatty acids from fish oil. Standard fish oil contains 30% EPA + DHA

Therapeutic Feeding Principles

Diet histories should be obtained focusing on dietary fat content. Initial client discussion will ideally focus on efforts to restrict dietary fat content of an existing diet (elimination of treats or table scraps where fed), and other recommendations made to restrict fat, such as a total diet change. Numerous therapeutic diets are available including those primarily recommended for obesity management, gastrointestinal disorders, and even hypoallergenic diets as long as they are reduced in dietary total fat.

Diets rich in marine omega-3 fatty acids may be especially helpful in the context of an overall low-fat dietary approach but are not well studied to date. Supplementation of existing low-fat diets with fish oils may be more appropriate rather than feeding a high-fat diet containing the omega-3 oils, which may preclude any benefit. Marine (or long-chain omega-3 fatty acids) are necessary rather than vegetable omega-3s because the triglyceride-lowering effect is primarily due to docosahexaenoic acid (DHA), which is poorly synthesized from vegetable-based precursors.

The inclusion of soluble dietary fiber interferes with the enteric reabsorption of cholesterol and bile acids and may help reduce hypercholesterolemic states. Dry diets are usually lower in cholesterol than canned diets and may be lower in total fat overall.

■ **Treats** – Treats are not recommended unless they are known to be low in fat.

■ **Tips for Increasing Palatability** – Obviously, the use of dietary oils to improve palatability is contraindicated. Moistening dry-expanded type foods is suggested, or a diet change within the low-fat category can be made. Using a low-fat canned food top-dressed on dry food may improve acceptance in some cases as long as total fat and calories remain controlled.

■ **Diet Recommendations** – Several forms of low fat diets are available primarily as therapeutic diets. Both canned and dry-extruded varieties exist but fat content of these, even with the same product name, may be different so caution is urged in selecting the most appropriate commercial product. Canned products often have a higher fat content even though they have the same product name as the dry version; yet these may be a useful alternative in some instances. Amounts to be fed include those necessary to maintain healthy non-obese body weight. In the case of obese animals, efforts to reduce caloric intake should be made. Home-prepared diets may be a necessary alternative in cases in which response to commercial low-fat diets is unsatisfactory.

Client Education Points

- Adherence to a low-fat diet, including all foods regularly consumed such as human foods and treats, is required.
- Total caloric intake, even using low-fat foods, should be discussed and alternatives suggested.
- Some cats will refuse fish oil capsules as a treat but will often accept the oil when top-dressed on an existing diet.

Common Comorbidities

Diseases associated with secondary hyperlipidemias include diabetes mellitus, hyperadrenocorticism, cholestasis, nephrotic syndrome, or may be drug related (megestrol acetate, corticosteroids). Hypertriglyceridemia related to diabetes mellitus can be attributed to decreased lipoprotein lipase activity and decreased clearance from sera or increased production. Hypertriglyceridemia is a risk factor for pancreatitis. Lack of insulin may also stimulate lipolysis and increase cholesterol genesis. Glucocorticoids may also inhibit lipoprotein lipase, reducing triglyceride clearance. Lipemia retinalis may be seen, and cutaneous xanthomas (lipid-laden macrophages under the skin) or thoracic granulomas may be seen with hypercholesterolemia. Peripheral neuropathies (tibial nerve or radial nerve paralysis and Horner's syndrome) may be associated with hypercholesterolemia.

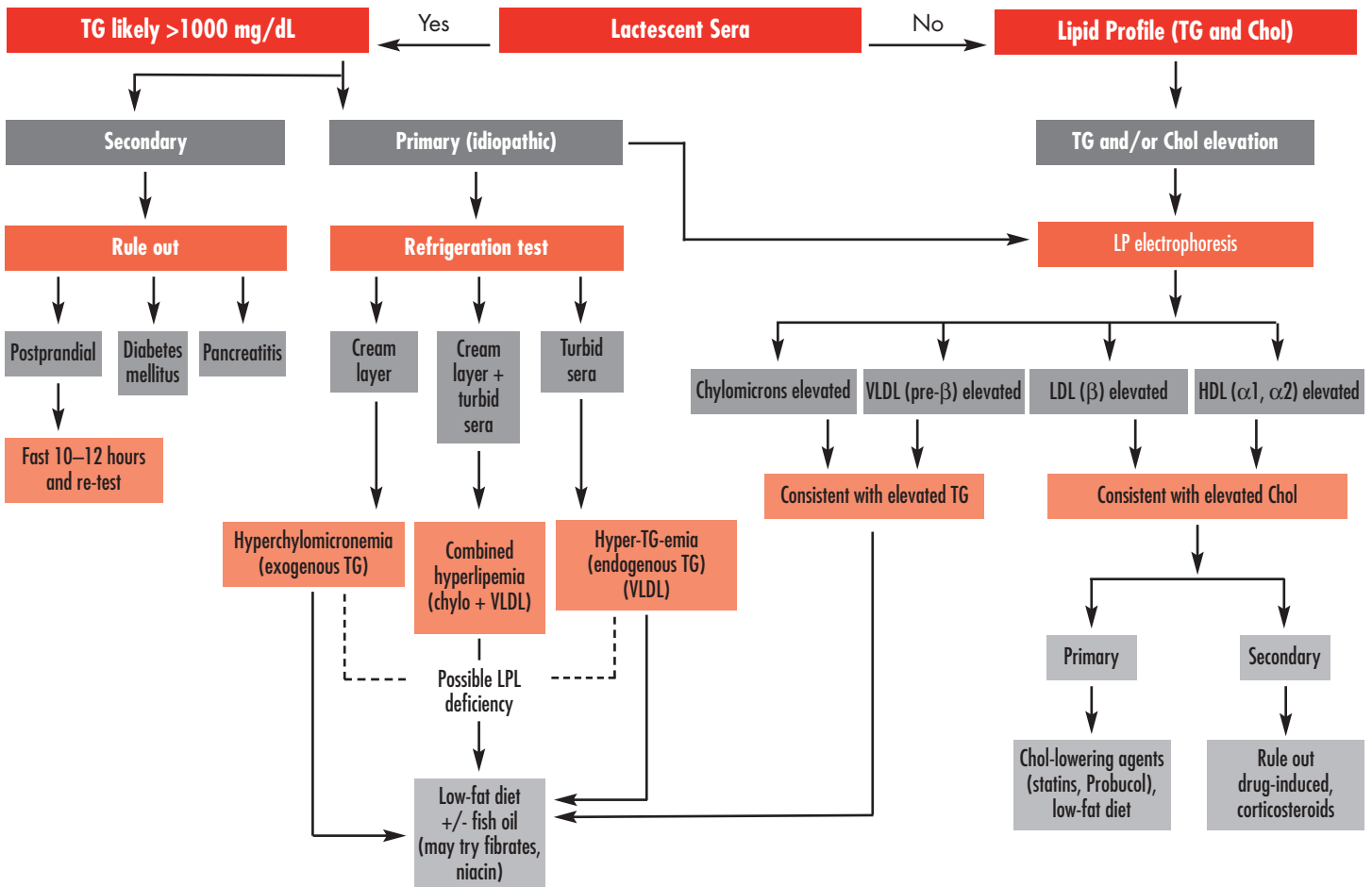
Interacting Medical Management Strategies

Several drug therapies may be useful in treating hyperlipemias but all may have undesirable side effects or have not been fully explored in cats. Generally, triglyceride concentrations <500 mg/dL need not be treated pharmacologically. **Niacin** (100 mg/day) may reduce fatty acid release from adipocytes with resultant decrease in VLDL synthesis but may result in facial pruritus, erythema, and vomiting. The **fibrate derivatives** stimulate lipoprotein lipase activity and may be used at 10 mg/kg body weight twice daily; however, vomiting and diarrhea may also occur. The **statins** suppress cholesterol synthesis but adverse effects such as lethargy, muscle pain, and elevated liver enzymes may be seen. **Probucol** is a cholesterol-lowering drug but it has been associated with arrhythmias. **Cholestyramine**, a bile acid sequestrant, may be effective in lowering serum cholesterol (1–2 g per os twice a day). Constipation may occur as well as increased VLDL and triglyceride synthesis.

Monitoring

Monitoring involves serum chemistry determinations including liver enzymes and lipid measurements.

Algorithm – Nutritional Management of Feline Hyperlipidemia



Chronic Kidney Disease – Canine

David J. Polzin, DVM, PhD, DACVIM

Definition

Kidney disease is the presence of functional or structural abnormalities in one or both kidneys. *Chronic kidney disease* (CKD) is kidney disease that has been present for 3 months or longer. Dogs with CKD and stable serum creatinine values (SC) < 1.4 mg/dL are considered CKD stage 1; SC values between 1.4 and 2.0 mg/dL are CKD stage 2; SC values between 2.0 and 5.0 mg/dL are CKD stage 3; and SC values > 5.0 mg/dL are CKD Stage 4.

Key Diagnostic Tools and Measures

Two to three determinations of serum creatinine, urea nitrogen, phosphorus, bicarbonate, calcium, potassium, and albumin concentrations; urinalysis and urine protein to creatinine ratio (UPC); arterial blood pressure; and assessment of hydration; diet history including food intake; body weight; body condition scoring are used in the diagnosis of CKD.

Pathophysiology

Loss of nephrons leads to waste product retention (especially nitrogenous wastes), impaired body fluid and electrolyte balance, and limited production of renal hormones (e.g., erythropoietin, calcitriol). Markedly impaired kidney function leads to uremia with anorexia, weight loss, and gastrointestinal signs. Excess intakes of protein, phosphorus, sodium, and acidifying nutrients may promote uremic signs and/or progressive kidney damage.

Signalment

Chronic kidney disease typically is found in dogs greater than 7 years of age, although it can occur in all age groups. Breeds predisposed to familial CKD include beagle, English foxhound, bull terrier, Doberman pinscher, Samoyed, Bernese mountain dogs, Brittany spaniel, Rottweiler, Shih Tzu, Lhasa Apso, Keeshond, Norwegian elkhound, cairn terrier, West Highland white terrier, soft-coated wheaten terrier, Basenji, English cocker spaniel, and Shar-Pei.

Key Nutrient Modifications

Limited amounts of high-quality **protein** are indicated in CKD stages 3 and 4. Excess protein intake promotes clinical signs of uremia. Protein intake should also be limited with protein-losing nephropathies. High protein intake does not cause CKD. Limiting dietary **phosphorus** intake slows progression of CKD; this is indicated in stages 2 through 4 CKD to meet serum phosphorus concentration targets (i.e., keep serum phosphorus below 4.5, 5.0, and 6.0 mg/dL in CKD stages 2, 3, and 4, respectively). Phosphorus intake can be limited further with intestinal phosphate binders. Diets enhanced in **omega-3 polyunsaturated fatty acids** (PUFA) slow progression of kidney disease. Excess omega-3 PUFA supplementation may impair coagulation and the immune system and increase the need for antioxidant support. Limit **sodium** intake. Excess salt intake may promote progressive kidney injury and limit hypertension (limited evidence). Excessive salt restriction may promote hypokalemia and dehydration. With regard to **energy**, higher caloric density facilitates ingestion of sufficient food to maintain appropriate body weight.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Phosphorus	0.2–0.4	0.04–0.08	0.5	0.14
Protein	14–18	3.0–4.5	18	5.1
Sodium	0.2–0.3	0.04–0.06	0.06	0.02

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

The goals of nutritional management of CKD are to 1) ameliorate signs of uremia, 2) maintain electrolyte and acid–base normalcy, 3) optimize nutrition, and 4) slow progressive loss of kidney function. Nutritional therapy using manufactured diets formulated for dogs with CKD has been shown to substantially extend lifespan and limit clinical signs of uremia in randomized controlled clinical trials in dogs with spontaneous CKD stages 3 and 4. Based on results of these clinical trials, nutritional therapy is indicated for dogs with CKD stages 3 and 4. Nutritional therapy may also be indicated for dogs with CKD stage 2 when serum phosphorus remains above 4.5 mg/dL. Because limiting protein intake and providing omega-3 PUFA may limit proteinuria, renal diets are recommended when UPC ratios exceed 2.0 in CKD stage 1 and 0.5 in CKD stage 2. The exact contribution of individual diet components to the beneficial effects of these diets remains to be established. Renal diets should contain ~14% to 17% protein, ≤0.3% phosphorus, and ~0.2% to 0.3% sodium. In addition, manufactured renal diets may also include increased omega-3 PUFA, fiber, vitamin D, and antioxidant contents, increased caloric density, and a neutral effect on systemic pH.

■ **Treats** – Most foods low in protein, phosphorus, and sodium may be used for treats in limited quantities. Kibbles of dry manufactured renal diets are a good choice. Avoid meats and dairy products. In general, treats should constitute no more than 5% of the patient's caloric intake. Commercial dog treats may be used, but only in very limited quantities as they may be high in sodium and phosphorus. Excessive intake of protein-rich treats may induce uremic signs in dogs with stage 4 CKD.

■ **Tips for Increasing Palatability** – Change from the previous diet to the renal diet should be gradual over 7 to 10 days by progressively adding the renal diet to the previous diet. Do not expose patients to long-term diets during periods of hospitalization for uremia. Enhance palatability by mixing small amounts of flavoring (gravy, low-sodium broth) or highly odorous foods into the renal diet. Warming food and stimulating eating by positive reinforcement with petting and stroking may facilitate food acceptance. Avoid associating unpleasant activities (e.g., undesirable medications, fluids) with feeding. When these methods fail to stimulate adequate food intake to maintain body weight, esophagostomy or gastrostomy tubes are indicated. Appetite stimulants rarely produce adequate food intake.

■ **Diet Recommendations** – Manufactured therapeutic diets designed specifically for patients with CKD are recommended. Senior diets are not recommended for these patients because they fail to include all dietary modifications. Dogs should initially be fed 132 x (body weight in kilograms)^{0.75} calories per day. Thereafter, serially monitor body weight and body condition score (BCS) and adjust caloric intake to maintain BCS between 4/9 and 5/9 (see Appendix I).

Client Education Points

- CKD is irreversible and will be present for the remainder of the pet's life.
- Most treatment recommendations for dogs with CKD will need to continue for the remainder of the pet's life.
- Regular follow-up visits are essential to detect changes in treatment needs.
- CKD is usually a progressive disease; however, proper treatment and monitoring can slow progression. Some dogs live many months to years with a good quality of life.
- Free access to fresh water at all times is essential. Never limit water intake.
- Limit excess protein and phosphorus intake (e.g., meats and dairy products).
- Limit intake of salt.
- Progressive weight loss must be addressed to avoid slow starvation.
- Minor gastrointestinal upsets may cause kidney function to abruptly worsen; treatment to prevent dehydration may be needed.
- High amounts of protein in urine and high blood pressure are harmful to the kidneys and require life-long therapy.

Common Comorbidities

Excess sodium intake promotes extracellular fluid volume expansion; excesses in calcium and phosphorus intake may promote vessel mineralization. Both have potential to promote **hypertension**. **Dental and other oral diseases** may be exacerbated by excess protein intake. Inadequate management of dental and oral diseases may impair food intake. In dogs with concurrent **urolithiasis**, renal diets are indicated because of strong evidence supporting renal protective effects while they may also be a reasonable choice for calcium oxalate uroliths. Struvite uroliths are usually of infectious origin rather than dietary. In dogs with concurrent **degenerative joint disease**, weight management and nutraceuticals are preferred over use of nonsteroidal anti-inflammatory drugs due to their potential to harm the kidneys. Finally, the high fat content of renal diets may promote **pancreatitis** in predisposed patients.

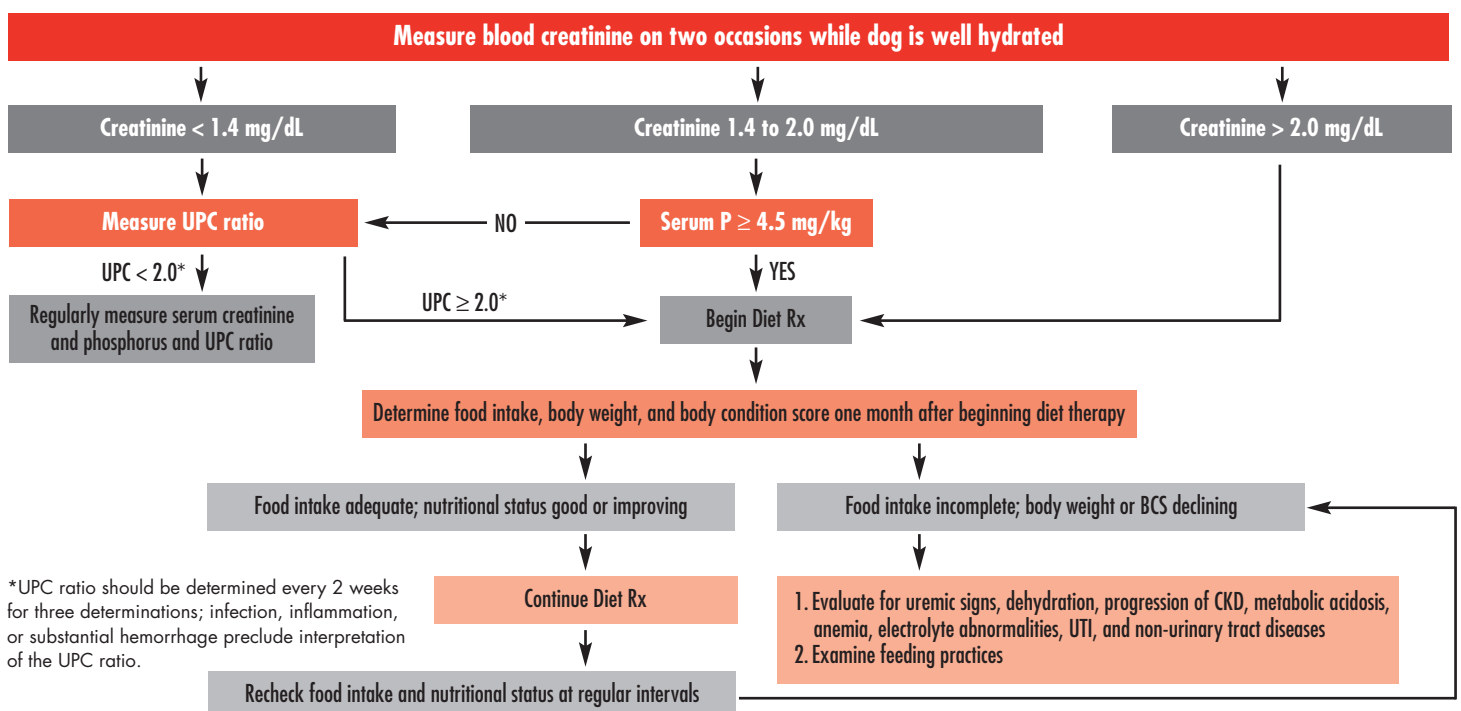
Interacting Medical Management Strategies

Intestinal **phosphate binders** (e.g., aluminum, calcium, or lanthanum salts) have an additive effect with dietary phosphorus restriction in reducing phosphorus intake. **Calcitriol** (dihydrocholecalciferol), which is produced by the kidneys and often deficient in patients with CKD, is often used to further suppress renal hyperparathyroidism and slow progression of CKD. Excess dietary vitamin D content may promote hypercalcemia in patients receiving calcitriol. **Angiotensin-converting enzyme (ACE) inhibitors** appear to have a greater salutary effect than protein restriction on mitigating glomerular hyperperfusion, proteinuria and, presumably, progression of CKD. It is unclear whether their effects on renal perfusion and proteinuria are additive. Metabolic acidosis reportedly impairs nutrition in humans receiving protein-restricted diets. **Oral sodium bicarbonate** is used to mitigate metabolic acidosis. As a non-chloride-containing sodium salt, it is not contraindicated with sodium-restricted diets or in hypertensive patients. **Corticosteroids** may impair the nutritional response to protein-restricted diets, enhance proteinuria, and promote gastrointestinal bleeding in dogs with CKD; use sparingly and with great caution. **Erythropoietin** therapy increases patient strength and appetite by increasing hematocrit. Iron supplementation and adequate protein and calorie intake is essential to maximize therapeutic response.

Monitoring

Food intake (food diary), body weight, BCS, serum chemistries (at least serum creatinine, urea nitrogen, phosphorus, calcium, albumin, potassium, and bicarbonate concentrations), hematocrit, and urinalysis (and urine protein:creatinine ratio if proteinuric) should be monitored within the first month after dietary intervention and every 3 to 4 months thereafter. Nutritional status and renal function should remain stable or improve. Ratio of BUN:creatinine should be less than 20; phosphorus should be less than 4.5, 5.0 or 6.0 mg/dL for CKD stages 2, 3 and 4, respectively. Steps should be taken to increase food intake in patients that fail to maintain stable, adequate nutritional status (including use of feeding tubes as indicated).

Algorithm – Nutritional Management of Canine Chronic Kidney Disease



*UPC ratio should be determined every 2 weeks for three determinations; infection, inflammation, or substantial hemorrhage preclude interpretation of the UPC ratio.

Chronic Kidney Disease – Feline

David J. Polzin, DVM, PhD, DACVIM

Definition

Kidney disease is the presence of functional or structural abnormalities in one or both kidneys. *Chronic kidney disease* is kidney disease that has been present for 3 months or longer. Cats with CKD and stable serum creatinine values (SC) < 1.6 mg/dL are considered CKD stage 1; SC values between 1.6 and 2.8 mg/dL are CKD stage 2; SC values between 2.8 and 5.0 mg/dL are CKD stage 3; and SC values > 5.0 mg/dL are CKD Stage 4.

Key Diagnostic Tools and Measures

Two to three determinations of serum creatinine, urea nitrogen, phosphorus, bicarbonate, calcium, potassium, and albumin concentrations; urinalysis and urine protein to creatinine ratio (UPC); arterial blood pressure; assessment of hydration; diet history including food intake; body weight; and body condition scoring are used in the diagnosis of CKD.

Pathophysiology

Loss of nephrons leads to waste product retention (especially nitrogenous wastes), impaired body fluid and electrolyte balance, and limited production of renal hormones (e.g., erythropoietin, calcitriol). Markedly impaired kidney function leads to uremia with anorexia, weight loss, and gastrointestinal signs. Excess intakes of protein, phosphorus, sodium, and acidifying nutrients may promote uremic signs and/or progressive kidney damage.

Signalment

Cats have an increasing rate of CKD beginning about 7 years of age with about two thirds of cases occurring in cats over 10 years of age. Reportedly, CKD occurs with increased frequency in Maine Coon, Abyssinian, Siamese, Russian Blue, and Burmese cats. Polycystic kidney disease is particularly prevalent in long-haired cats.

Key Nutrient Modifications

Limited amount of high quality **protein** are indicated in CKD stages 3 and 4. Excess protein intake promotes clinical signs of uremia. Protein intake should also be limited with protein-losing nephropathies. High protein intake does not cause CKD. Limiting dietary **phosphorus** intake likely slows progression of CKD and is indicated in stages 2 through 4 CKD to meet serum phosphorus concentration targets (keep serum phosphorus below 4.5, 5.0, and 6.0 mg/dL in CKD stages 2, 3, and 4, respectively). Phosphorus intake can be limited further with intestinal phosphate binders. Diets enhanced in **omega-3 polyunsaturated fatty acids (PUFA)** may slow progression of CKD. Excess omega-3 PUFA supplementation may impair coagulation and the immune system and increase need for antioxidant support. **Sodium** intake is typically limited. Excess salt intake may promote progressive kidney injury and limit hypertension (limited evidence). Excessive salt restriction may promote hypokalemia and dehydration. Adequate **potassium** intake is necessary to prevent development of hypokalemia. Finally, a neutral **acid–base** effect is desired. Acidifying diets are inappropriate for cats with CKD; diets producing a neutral effect on body pH should be fed.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Phosphorus	0.3–0.5	0.08–0.12	0.5	0.13
Protein	28–34	6–8	26	6.5
Sodium	0.2–0.3	0.04–0.07	0.2	0.05
Potassium	0.8–1.2	0.15–0.30	0.6	0.15

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

The goals of nutritional management of CKD are to 1) ameliorate signs of uremia, 2) maintain electrolyte and acid–base normalcy, 3) optimize nutrition, and 4) slow progressive loss of kidney function. Nutritional therapy using manufactured diets formulated for cats with CKD has been shown to substantially extend lifespan and limit clinical signs of uremia in randomized controlled clinical trials in cats with spontaneous CKD and serum creatinine values \geq 2.0 mg/dL. Based on results of these clinical trials, nutritional therapy is indicated for cats with CKD stages 2 through 4. Because limiting protein intake and providing omega-3 PUFA may limit proteinuria, renal diets should be considered when UPC ratios exceed 2.0 in CKD stage 1. The exact contribution of individual diet components to the beneficial effects of these diets remains to be established. Renal diets should contain ~28% to 34% protein, \leq 0.5% phosphorus, >0.8% potassium, and ~0.2% to 0.3% sodium. Manufactured renal diets may also include increased omega-3 PUFA, fiber, vitamin D, and antioxidant contents, increased caloric density, and a neutral effect on systemic pH.

■ **Treats** – Most foods low in protein, phosphorous, and sodium may be used for treats in limited quantities. Kibbles of dry manufactured renal diets are a good choice. Avoid meats and dairy products. In general, treats should constitute no more than 5% of the patient's caloric intake. Commercial cat treats may be used but only in very limited quantities as they may be high in sodium and phosphorus. Excessive intake of protein-rich treats may induce uremic signs in cats with stage 4 CKD.

■ **Tips for Increasing Palatability** – Changing from the previous diet to the renal diet should be gradual over 7 to 10 days by progressively adding the renal diet to the previous diet. Do not expose patients to long-term diets during periods of hospitalization for uremia. Enhance palatability by mixing small amounts of flavoring (gravy, tuna or clam juice, low-sodium broth) or highly odorous foods into the renal diet. Warming food and stimulating eating by positive reinforcement with petting and stroking may facilitate food acceptance. Avoid associating unpleasant activities (e.g., undesirable medications, fluids) with feeding. When these methods fail to stimulate adequate food intake to maintain body weight, esophagostomy or gastrostomy tubes are indicated. A trial with appetite stimulants may be considered, but rarely produces adequate food intake.

■ **Diet Recommendations** – Manufactured therapeutic diets designed specifically for patients with CKD are recommended. Senior diets are not recommended for these patients because they fail to include all dietary modifications. Cats should initially be fed 1.1 to 1.4 x [70 x (body weight in kilograms)^{0.75}] calories per day. Thereafter, serially monitor body weight

and body condition score (BCS) and adjust calorie intake to maintain BCS between 4/9 and 5/9.

Client Education Points

- CKD is irreversible and will be present for the remainder of the pet’s life.
- Most treatment recommendations for cats with CKD will need to continue for the remainder of the pet’s life.
- Regular follow-up visits are essential to detect changes in treatment needs.
- CKD is usually a progressive disease; however, proper treatment and monitoring can slow progression. Many cats live for many months to years with a good quality of life.
- Free access to fresh water at all times is essential. Never limit water intake.
- Limit excess protein and phosphorus intake (e.g., meats, dairy products).
- Limit intake of salt.
- Progressive weight loss must be addressed to avoid slow starvation.
- Minor gastrointestinal upsets may cause kidney function to abruptly worsen; treatment to prevent dehydration may be needed.
- High amounts of protein in urine and high blood pressure are harmful to the kidneys and require life-long therapy.

Common Comorbidities

Excess sodium intake promotes extracellular fluid volume expansion; excesses in calcium and phosphorus intake may promote vessel mineralization. Both have potential to promote **hypertension**. **Dental and other oral diseases** may be exacerbated by excess protein intake. Inadequate management of dental and oral diseases may impair food intake. **Hyperthyroidism** should generally be controlled as soon as it is recognized. Appetite commonly declines after treatment of hyperthyroidism, but, left untreated, it will ultimately prove fatal. In cats with concurrent **nephrolithiasis (almost always composed of calcium oxalate)**, renal diets are indicated because of strong evidence supporting renal protective effects while they may also be a reasonable choice for calcium oxalate uroliths. In cats with concurrent **degenerative joint disease**, weight management and nutraceuticals are preferred over use of nonsteroidal anti-inflammatory drugs due to their potential to harm the kidneys.

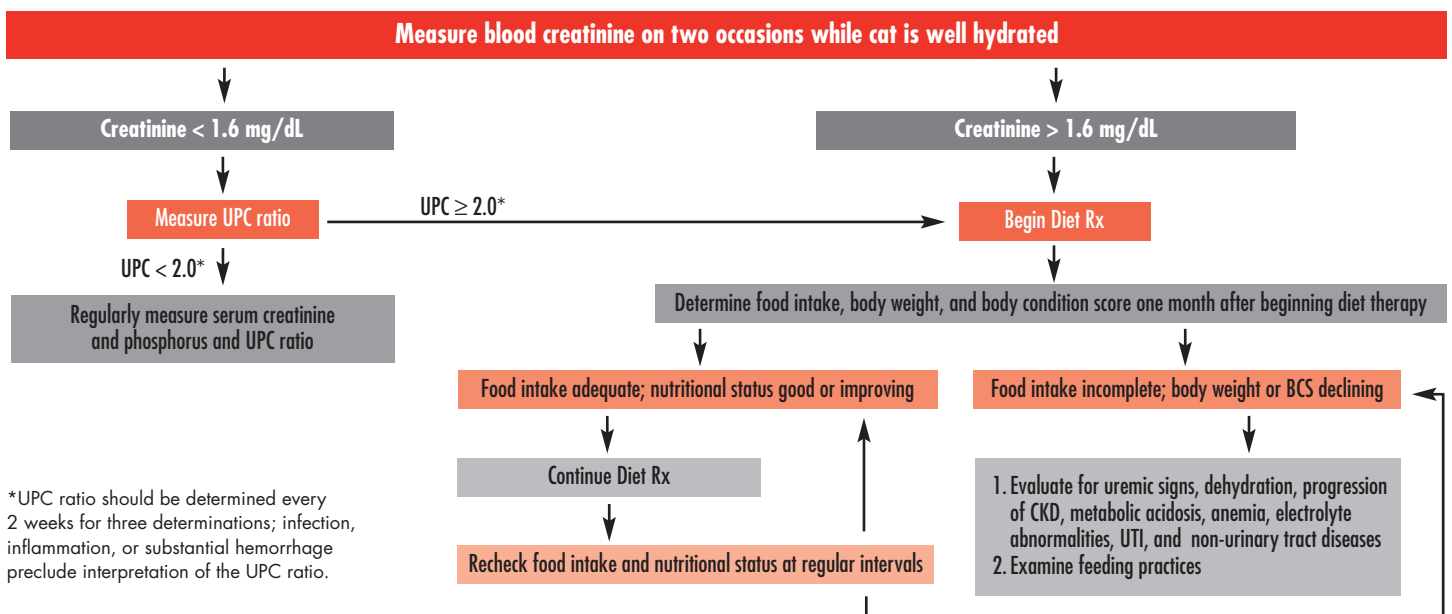
Interacting Medical Management Strategies

Intestinal **phosphate binders** (e.g., aluminum, calcium, or lanthanum salts) have an additive effect with dietary phosphorus restriction in reducing phosphorus intake. **Calcitriol** (dihydrocholecalciferol), which is produced by the kidneys and often deficient in patients with CKD, is often used to further suppress renal hyperparathyroidism and slow progression of CKD. Excess dietary vitamin D content may promote hypercalcemia in patients receiving calcitriol. **Angiotensin-converting enzyme (ACE) inhibitors** appear to have a greater salutary effect than protein restriction on mitigating glomerular hyperperfusion, proteinuria and, presumably, progression of CKD. It is unclear whether their effects on renal perfusion and proteinuria are additive. Metabolic acidosis reportedly impairs nutrition in humans receiving protein-restricted diets. **Oral sodium bicarbonate** is used to mitigate metabolic acidosis. As a non-chloride-containing sodium salt, it is not contraindicated with sodium-restricted diets or in hypertensive patients. **Corticosteroids** may impair the nutritional response to protein-restricted diets, enhance proteinuria and promote gastrointestinal bleeding in cats with CKD; use sparingly and with great caution. **Erythropoietin** therapy increases patient strength and appetite by increasing hematocrit. Iron supplementation and adequate protein and calorie intake is essential to maximize therapeutic response.

Monitoring

Food intake (food diary), body weight, BCS, serum chemistries (at least serum creatinine, urea nitrogen, phosphorus, calcium, albumin, potassium, and bicarbonate concentrations), hematocrit, and urinalysis (and urine protein:creatinine ratio if proteinuric) should be monitored within the first month after dietary intervention and every 3 to 4 months thereafter. Nutritional status and renal function should remain stable or improve. Ratio of BUN:creatinine should be less than 20; phosphorus should be less than 4.5, 5.0, or 6.0 mg/dL for CKD stages 2, 3, and 4, respectively. Steps should be taken to increase food intake in patients that fail to maintain stable, adequate nutritional status (including use of feeding tubes as indicated).

Algorithm – Nutritional Management of Feline Chronic Kidney Disease



Calcium Oxalate Urolithiasis – Canine

Joseph W. Bartges, DVM, PhD, DACVIM, DACVN

Definition

Urolithiasis is a condition in which crystals in the urine form stones or calculi called *uroliths*. In calcium oxalate urolithiasis uroliths are composed of calcium oxalate monohydrate, dihydrate, or both.

Key Diagnostic Tools and Measures

Obtain serum or plasma calcium concentration to evaluate for hypercalcemia; if present, determine blood ionized calcium, parathyroid hormone, and parathyroid hormone–related protein concentrations. Adrenal gland testing is recommended due to the association of hyperadrenocorticism and increased risk of calcium oxalate urolithiasis. Urinalysis should be done to evaluate urine pH and presence of calcium oxalate crystalluria. Abdominal radiography should reveal radiodense uroliths that often have an irregular surface contour. Calcium oxalate uroliths cannot be dissolved medically; therefore, they must be removed physically by surgery or voiding urohydropropulsion.

Pathophysiology

Calcium oxalate uroliths form when urine is oversaturated with calcium, oxalate, or both. They typically form in aciduria because the solubility of calcium oxalate is decreased when urine pH is <6.8. Hypercalcemia increases urinary calcium excretion and risk for calcium oxalate urolith formation. Approximately 4% of dogs with calcium oxalate urolithiasis have hypercalcemia. Malignant neoplasia is the most common cause of hypercalcemia in dogs; however, this is usually not associated with calcium oxalate urolith formation. Primary hyperparathyroidism is more commonly associated with canine calcium oxalate urolithiasis. In dogs with calcium oxalate uroliths and normocalcemia, the mechanism(s) for calcium oxalate urolith formation is not known. In one study of miniature schnauzers, increased absorption of calcium from the gastrointestinal tract relative to non-urolith-forming beagles was found. Hyperadrenocorticism is associated with calcium oxalate urolith formation because it promotes hypercalciuria, which results in urinary oversaturation for calcium oxalate and possibly urolith formation.

Signalment

Small-breed dogs such as miniature schnauzers, toy poodles, Lhasa Apsos, Shih Tzus, and Yorkshire terriers have breed predispositions to calcium oxalate urolithiasis. Uroliths are more common in middle-aged or older dogs, and there is a male-to-female ratio of 3:1.

Key Nutrient Modifications

Decreased dietary **calcium** may result in decreased degree of calciuresis. Decreased **protein** intake may decrease degree of calciuresis by decreasing calcium release from bone in response to acid load provided by dietary protein. High **fiber** may decrease absorption of calcium from the intestinal tract. Increased **sodium** intake may decrease urinary saturation for calcium oxalate by increasing urine volume. Dietary **potassium** citrate may decrease risk of calcium oxalate urolith formation by inducing alkaluria and by inhibiting calcium oxalate crystal and urolith formation.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	12–22	3–6	18	5.1
Fiber	8–15	2–5	n/a	n/a
Calcium	0.4–1.2	0.15–0.35	0.6	0.17
Sodium	1.0–1.5	0.25–0.35	0.06	0.02

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Increased water intake is recommended and dogs may benefit from either feeding a canned diet or adding water to dry food prior to feeding.

Therapeutic Feeding Principles

Inducing production of a greater volume of dilute urine may be beneficial. Mild to moderate dietary protein restriction is recommended, and some dogs respond to higher fiber intake. Feed a diet that induces alkiluria. If secondary to endocrine disease, management of that disease is the main treatment; dietary management of calcium oxalate disease is also indicated.

- **Treats** – Avoid high-protein treats and treats associated with increased oxalate levels (e.g., carrots and green leafy vegetables)
- **Tips for Increasing Palatability** – Water can be added to food to increase palatability.
- **Diet Recommendations** – A high-fiber, low-fat diet is recommended for dogs with calcium oxalate urolithiasis. A lower protein, alkalinizing diet is also appropriate.

Client Education Points

- Calcium oxalate uroliths occur when urine contains high levels of calcium and/or oxalate.
- Calcium oxalate uroliths are recurrent, with approximately 30% recurrence rate at 12 months and approximately 60% recurrence within 5 years.
- Dietary modification may decrease risk of recurrence of calcium oxalate uroliths

Common Comorbidities

Calcium oxalate urolithiasis occurs commonly in dogs with hyperadrenocorticism or hyperparathyroidism if hypercalcemic.

Interacting Medical Management Strategies

Potassium citrate:

- Urinary alkalinizing agent
- Increases calcium oxalate solubility with alkaluria
- Citrate may inhibit calcium oxalate crystal formation and aggregation
- 50–100 mg/kg orally (PO) every 12 hours, adjust to urine pH of approximately 7.5

Thiazide diuretics:

- Increases distal renal tubular reabsorption of calcium resulting in lowered urinary calcium excretion
- Can result in hypercalcemia
- No long-term studies in dogs on safety or efficacy
- Hydrochlorothiazide: 2 mg/kg PO every 12 hours

Vitamin B6:

- Involved with oxalate metabolism
- Deficiency does not likely occur and no data that supplementation helps
- 20 mg/kg PO every 24 hours

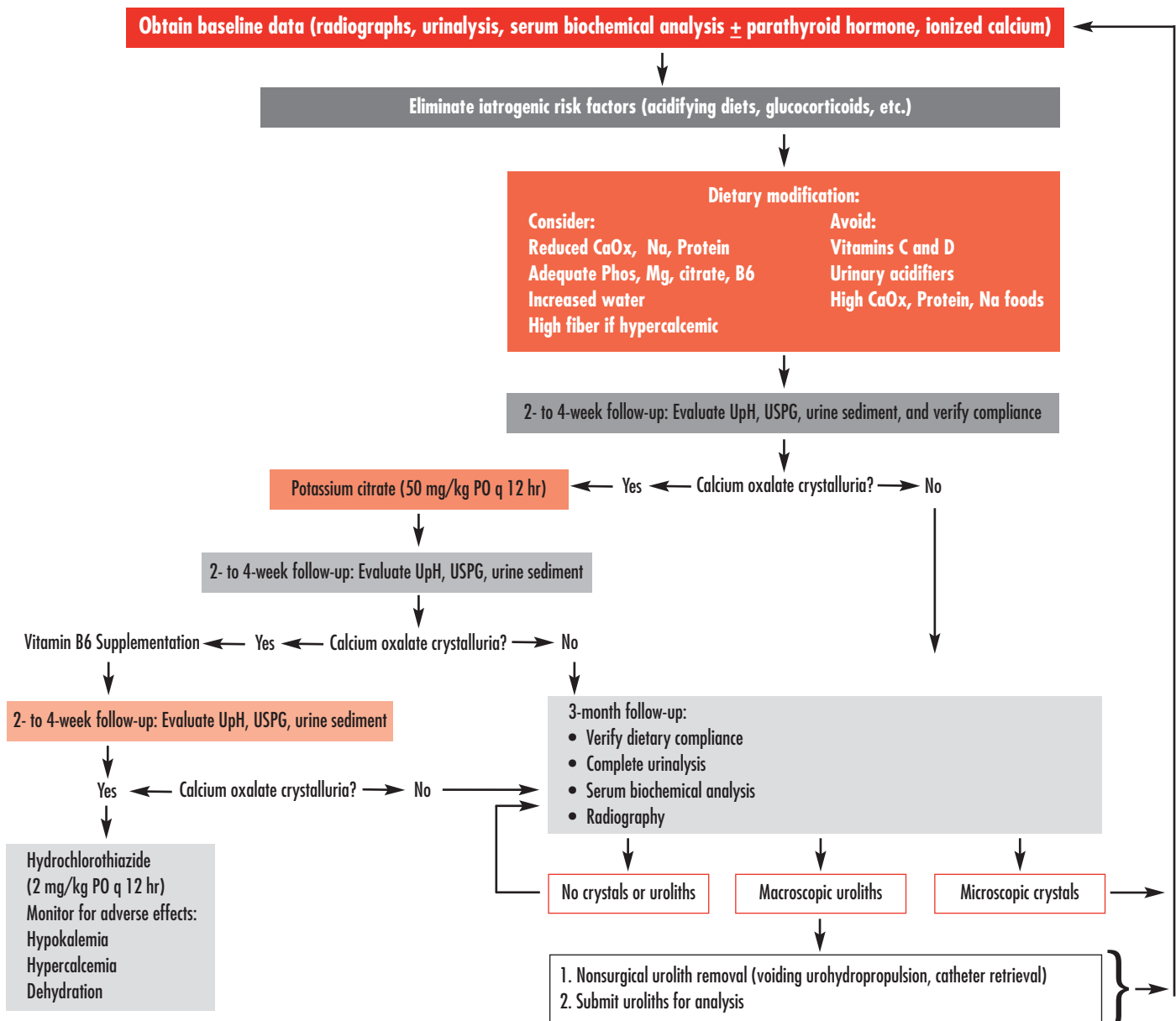
Monitoring

Urinalysis should be done monthly for 3 to 6 months to monitor response to treatment (Table 1). pH should be neutral to alkaline; specific gravity: should be dilute; and crystalluria should be absent. Survey abdominal radiography or ultrasonography should be performed at 6 and 12 months and then every 6 to 12 months depending on response. Serum calcium should be monitored 1 month after starting hydrochlorothiazide and then every 3 to 6 months. If urolithiasis is secondary to endocrine disease, the management of that condition should be monitored appropriately.

Table 1. Expected changes with therapy of calcium oxalate uroliths.

Factor	Pre Therapy	Prevention
Polyuria	±	variable
Pollakiuria	0 to 4+	0
Hematuria	0 to 4+	0
USPG	variable	1.004–1.025
Urine pH	< 7.0	> 7.0
Urine inflammation	0 to 4+	0
Calcium oxalate crystals	0 to 4+	0
Bacteriuria	0 to 4+	0
Culture	positive/negative	negative
BUN (mg/dL)	> 15	10–30
Urolith size and number	small to large	0

Algorithm – Nutritional Management of Canine Calcium Oxalate Uroliths



Feline Lower Urinary Tract Disease – Idiopathic Cystitis & Struvite/Calcium Oxalate Urolithiasis

Joseph W. Bartges, DVM, PhD, DACVIM, DACVN

Definition

Feline lower urinary tract disease (FLUTD) is often associated with *urolithiasis* (formation of struvite or calcium oxalate uroliths in the urine) or *idiopathic cystitis*. In cats less than 10 years of age, idiopathic cystitis is the most common type of nonobstructive lower urinary tract disease, with urolithiasis (usually sterile struvite) the next most common type. In cats older than 10 years of age, bacterial *urinary tract infection* (UTI) is the most common cause of nonobstructive lower urinary tract disease, with urolithiasis (usually calcium oxalate) the next most common type. If the UTI is caused by a urease-producing bacterial organism, infection-induced struvite uroliths may form. **Obstructive uropathy** may be due to urolithiasis or to matrix-crystalline *urethral plugs* (found only in male cats); struvite is the most common mineral observed to occur in plugs.

Key Diagnostic Tools and Measures

Diagnosis begins with urinalysis to determine urine pH and presence and type of crystalluria. Urine culture can rule out or confirm infection; less than 1% of cats under 10 years of age with lower urinary tract signs have a bacterial UTI, whereas approximately 45% of cats over 10 years of age with lower urinary tract signs have a bacterial UTI. In these cases, the infection is often associated with a predisposing factor (e.g., renal failure, diabetes mellitus, feline leukemia virus [FeLV] infection). Abdominal radiography is helpful in determining if uroliths are present because struvite and calcium oxalate, which account for over 90% of uroliths in cats, are typically radiodense. Serum calcium concentration determination is important in cats with calcium oxalate uroliths because 20% to 35% of cats with calcium oxalate uroliths are hypercalcemic. Ionized calcium (iCa) and parathyroid hormone (PTH) concentrations should be determined if hypercalcemia is present. Idiopathic hypercalcemia, characterized by increased total calcium, increased iCa, and low PTH concentrations, is the most common cause of hypercalcemia in cats. If a urolith or plug is retrieved, it should be analyzed. Advanced imaging such as cystoscopy, ultrasonography, or contrast urethrocytography may help to identify uroliths or rule out causes of FLUTD. Thickened urinary bladder wall and glomerulations may be observed by cystoscopy with idiopathic cystitis.

Pathophysiology

Many diseases result in signs of lower urinary tract disease; idiopathic cystitis and urolithiasis occur most commonly, especially in cats less than 10 years of age. Uroliths form when urine is oversaturated with minerals that precipitate to form the uroliths. **Struvite** is magnesium ammonium phosphate hexahydrate, and thus struvite solubility is dependent on concentrations of magnesium, ammonium, and phosphate, and urine pH. Struvite solubility decreases as urine pH exceeds approximately 6.8. Two forms of struvite occur: 1) **Infection-induced struvite uroliths** form secondary to a UTI with a urease-producing bacterial organism, typically *Staphylococcus* spp. Bacterial urease activity results in alkaluria, increased urinary ammonium concentrations, and change in ionization state of phosphate promoting struvite formation. 2) **Sterile struvite uroliths** form without a bacterial infection and occur with oversaturation of urine with struvite calcuogenic minerals and alkaluria. Alkaluria occurs secondary to postprandial alkaline tide or to persistent renal excretion of base. Sterile struvite uroliths occur more commonly in cats than infection-induced struvite; however, cats do form infection-induced struvite uroliths if they develop a UTI with a urease-producing bacterial organism.

Calcium oxalate uroliths may occur as the monohydrate or dihydrate forms or a combination of both; dihydrate form occurs most commonly. Calcium oxalate uroliths form when urine is oversaturated with calcium or oxalate or both, and form with aciduria. Approximately 20% to 35% of cats with calcium oxalate uroliths have hypercalcemia, most commonly idiopathic, which promotes hypercalciuria and urolith formation. The mechanism(s) for calcium oxalate urolith formation in cats that are normocalcemic is unknown; however, urinary oversaturation with calcium oxalate occurs.

Idiopathic cystitis may occur as a nonobstructive form that may or may not be associated with crystalluria or as an obstructive form due to a matrix-crystalline urethral plug in male cats. Idiopathic cystitis refers to clinical signs of FLUTD including hematuria, but without an identifiable cause. Viral infection and neurogenic inflammation are theorized causes. Matrix-crystalline urethral plug formation in male cats may represent an intermediate phase between urinary inflammation (e.g., idiopathic cystitis, bacterial cystitis) and urolithiasis.

Signalment

FLUTD occurs more commonly in cats between 4 and 10 years of age. Idiopathic cystitis and struvite urolithiasis occurs typically in cats less than 10 years of age; there is no gender or breed predisposition. Matrix-crystalline urethral plugs typically occur in male cats less than 10 years of age. Calcium oxalate urolithiasis typically occurs in cats older than 8 years of age; long-haired cats have a breed-associated predisposition, but there is no gender predisposition.

Key Nutrient Modifications

For cats with **struvite urolithiasis** and **matrix-crystalline urethral plugs**, increasing water intake will help to decrease concentration of calcuogenic minerals in urine. For dissolution, dietary protein restriction decreases urinary ammonia concentration; for prevention, dietary protein should be moderated. Phosphorous and magnesium should be restricted. With regard to dietary fat, increasing energy density results in decreased amount of food intake and, therefore, overall mineral intake; however, obesity increases the risk of FLUTD and should be avoided. With regard to urinary acidification, struvite is more soluble (that is, likelihood of precipitation is increased) when urine pH is <6.5–6.8; therefore, avoid alkalinizing foods and induce an aciduria.

For **calcium oxalate urolithiasis**, increasing water intake will help to decrease the concentration of calcuogenic minerals in urine. Excessive dietary protein intake should be avoided. Increasing energy density results in decreased amount of food intake and, therefore, overall mineral intake; however, obesity increases the risk of FLUTD and should be avoided. Calcium intake should be moderately restricted (see, for example, the diet fed in reference 6). Avoid dietary restriction of phosphorous and magnesium. Avoid excessive intake of vitamins C and D. With regard to urinary alkalinization, calcium oxalate is more soluble when urine pH is >6.8;¹¹ therefore, avoid acidifying diets and induce a neutral to alkaline urine pH. Increasing fiber intake may benefit cats with idiopathic hypercalcemia and calcium oxalate uroliths.

For **idiopathic cystitis**, increasing water intake may help to decrease recurrence of clinical signs.

See Algorithm – Nutritional Management of Feline Lower Urinary Tract Disease on page 94.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
For cats with struvite urolithiasis:				
Protein	30–48	8–13	26	6.5
Fat	14–28	3.5–6.5	9	2.3
Phosphorus	0.6–1.1	0.1–0.3	0.5	0.13
Magnesium	0.05–0.08	0.01–0.025	0.04	0.01
For cats with calcium oxalate urolithiasis:				
Protein	30–48	8–13	26	6.5
Fat	14–28	3.5–6.5	9	2.3
Calcium	0.6–1.1	0.1–0.3	0.6	0.15
Phosphorus	0.6–1.1	0.1–0.3	0.5	0.13
Magnesium	0.05–0.08	0.01–0.025	0.04	0.01
Fiber [#]	5–16	2–4	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake. Increased water intake should be encouraged. Use of canned, high moisture diets or sodium-supplemented diets may help increase water intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

[#]For hypercalcemic cats, select a high-fiber diet along with the listed modifications

Therapeutic Feeding Principles

For dissolution or prevention of struvite urolithiasis and matrix-crystalline urethral plugs, induce aciduria and restrict dietary magnesium, protein, and phosphorous to induce urinary undersaturation and dissolution of uroliths or crystals. For calcium oxalate urolithiasis, induce neutral to alkaline urinary pH, restrict dietary calcium, and increase urine volume. For idiopathic cystitis, increase urine volume.

■ **Treats** – For cats with struvite urolithiasis and matrix-crystalline urethral plugs, avoid alkalinizing treats and medications. For cats with calcium oxalate urolithiasis, avoid acidifying treats, such as those containing acidifiers or high protein content, and avoid excessive calcium and vitamins D and C intake. For cats with idiopathic cystitis, avoid alkalinizing treats as they may induce struvite crystalluria.

■ **Tips for Increasing Palatability** – Warming food to near, but not over, body temperature increases palatability. Flavoring agents, such as gravy or broth, can also be used.

■ **Diet Recommendations** – Commercial diets formulated to decrease the risk of struvite and calcium oxalate formation as well as a struvite dissolution diet are available for cats with struvite urolithiasis, matrix-crystalline urethral plugs, and calcium oxalate urolithiasis. Canned diets are recommended for cats with idiopathic cystitis.

Client Education Points

- Struvite uroliths usually occur without a bacterial UTI in cats, but can form secondary to an infection. Dietary modification is important to decrease crystalluria and potential for recurrence of uroliths and plugs.
- Approximately one in three to five cats with calcium oxalate stones has high blood calcium concentrations. Dietary modifications to decrease calcium in urine and increase urine pH help to decrease recurrence.

- The cause(s) of idiopathic cystitis is/are not known. From a diet perspective, increasing urine volume helps to decrease recurrence in many cats.

Common Comorbidities

Struvite uroliths can form secondary to UTIs with urease-producing bacteria (infection-induced struvite); however, in cats, they usually form without an infection. Idiopathic hypercalcemia occurs in 20% to 35% of cats with calcium oxalate uroliths. Stress and inappropriate behavior are part of the pathogenesis of idiopathic cystitis. Obesity is associated with increased incidence of struvite urolithiasis and matrix-crystalline urethral plugs, calcium oxalate urolithiasis, and idiopathic cystitis.

Interacting Medical Management Strategies

If dietary acidification is ineffective in cats with struvite urolithiasis and matrix-crystalline urethral plugs, urinary acidifiers can be used. For calcium oxalate urolithiasis, potassium citrate induces neutral to alkaline urine pH and citrate is an inhibitor of calcium oxalate formation. It should be administered to cats with idiopathic hypercalcemia that are fed a moderate- to high-fiber diet. Thiazide diuretics decrease renal excretion of calcium; however, data does not exist in cats concerning safety and efficacy

No treatment has been shown to be completely effective for idiopathic cystitis. Analgesics aid in keeping the cat comfortable until clinical signs spontaneously resolve. Amitriptylline may help some cats with recurrent or persistent idiopathic cystitis. Pentosan polysulfate sodium is used in women with interstitial cystitis and may help some cats with recurrent or persistent idiopathic cystitis. Pharmacological modification of stress may benefit some cats.

Monitoring

For cats monitored for **dissolution of struvite uroliths**, sterile struvite uroliths typically dissolve in 2 to 4 weeks when feeding a struvite dissolution diet. Infection-induced struvite uroliths typically dissolve in 8 to 10 weeks when feeding a struvite dissolution diet and administering an appropriate antimicrobial agent. Urinalysis and lateral abdominal radiographs should be monitored monthly until urolith dissolution. Urinalysis should indicate aciduria, dilute urine, no crystalluria, resolution of hematuria, and no inflammation. For **prevention of struvite urolithiasis**, urinalysis and lateral abdominal radiography should be performed 1 to 2 months after medical dissolution or surgical removal. Urinalysis should indicate aciduria, dilute urine, no crystalluria, resolution of hematuria, and no inflammation. Consider urinalysis every 4 to 6 months and abdominal radiography if clinical signs of FLUTD occur.

For cats with **struvite matrix-crystalline urethral plugs**, urinalysis should be performed 1 to 2 months after beginning dietary modification. Check for aciduria, dilute urine, no crystalluria, resolution of hematuria, and no inflammation. Consider urinalysis every 4 to 6 months, and abdominal radiography if clinical signs of FLUTD occur.

For cats with **calcium oxalate urolithiasis**, urinalysis and lateral abdominal radiography should be performed 1 to 2 months after surgical removal. Look for neutral to alkaline urinary pH, dilute urine, no crystalluria, resolution of hematuria, and no inflammation. Consider urinalysis every 4 to 6 months. In cats with idiopathic hypercalcemia, monitor total serum calcium 1 to 2 months after dietary modification is begun and then every 4 to 6 months; abdominal radiographs are needed if clinical signs of FLUTD occur.

For cats with **idiopathic cystitis**, urinalysis should be performed 1 to 2 months after beginning dietary modification; look for aciduria, dilute urine, no crystalluria, resolution of hematuria, and no inflammation. Consider urinalysis every 4 to 6 months, and abdominal radiography if clinical signs of FLUTD occur.

Urate Urolithiasis – Canine

Joseph W. Bartges, DVM, PhD, DACVIM, DACVN

Definition

In *urate urolithiasis*, uroliths are composed of uric acid or salts of uric acid; ammonium urate occurs most commonly. The condition may occur as a result of liver disease, most commonly portovascular shunt or microvascular dysplasia, or as a metabolic defect without liver disease, most commonly in Dalmatians and English bulldogs.

Key Diagnostic Tools and Measures

In cases associated with portovascular shunt or microvascular dysplasia, clinical and laboratory findings include stunted growth, microcytosis, low-normal or sub-normal BUN, hyperammonemia, abnormal provocative serum bile acid testing, possible signs of hepatoencephalopathy (ptyalism, depression, vomiting, seizures) particularly associated with eating, and urate crystalluria. Urate uroliths are marginally radiodense or radiolucent; identification requires ultrasonography or double-contrast urocytography. Microhepatica may be seen on survey abdominal radiography or abdominal ultrasonography. Contrast portography may reveal shunting of blood if a shunt is present. Portal scintigraphy is abnormal. Identification of extravascular or intravascular hepatic shunt if present is made at surgery.

In cases *not* associated with portovascular shunt or microvascular dysplasia, findings may include hyper-uric acidemia (healthy dogs: 0.1–0.3 mg/dL; with urate urolithiasis, >0.3 mg/dL, usually 0.8–1.5 mg/dL), and urate crystalluria. Urate uroliths are marginally radiodense or radiolucent; identification requires ultrasonography or double-contrast urocytography. Urate urolithiasis may be associated with miliary dermatitis in Dalmatians, and with dilated cardiomyopathy in English bulldogs.

Pathophysiology

Uric acid is a metabolite of purine metabolism from endogenous and exogenous sources. Purines include the nucleic acid bases adenine and guanine. Purines enter into the purine metabolic pathway and are metabolized to hypoxanthine to xanthine and xanthine to uric acid by xanthine oxidase. Uric acid is metabolized to allantoin by hepatic uricase. Allantoin is the typical end-product of purine metabolism in dogs.

Urate uroliths form with urinary oversaturation with uric acid. With liver disease, conversion of uric acid by uricase and ammonia by the urea cycle is decreased. In dogs without liver disease, conversion of uric acid to allantoin is decreased and decreased reabsorption or increased secretion of uric acid by proximal renal tubules occurs.

Urinary and serum uric acid concentrations can be decreased by restricting dietary purine (protein) intake. Dietary protein restriction also decreases ammonia production. Urate uroliths form typically in acidic urine, and decreasing dietary protein intake results in alkaluria.

Signalment

Young dogs, especially those of small breeds (e.g., Yorkshire terriers, miniature schnauzers), are typical signalment for dogs that form urate uroliths secondary to congenital liver disease. Dalmatians and English bulldogs have increased incidence of urate urolith formation without congenital liver disease. Urate uroliths occur more commonly in males, with the highest incidence in 1- to 5-year-old dogs.

Key Nutrient Modifications

Uric acid comes from endogenous and exogenous purines. Exogenous purine sources are highly cellular nutrients, such as animal-based protein

sources. Organ meat is particularly high in purine content. Ammonia originates from colonic bacterial metabolism of protein and from ammonia excretion by kidneys. Ammonia from intestinal bacteria is normally metabolized to urea by the hepatic urea cycle. Kidneys excrete ammonia as a buffer for acid by filtration and deamination of glutamine. Dietary protein restriction results in decreased purine intake, decreased ammonia production, alkaluria (uric acid is more soluble in alkaline pH) and diuresis, which results in dilution of calculogenic compounds in urine.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	14–17	3.0–4.0	18	5.1

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

For dogs with urate uroliths, but without liver disease, nutritional management includes feeding a low or ultra-low protein diet that induces alkaluria and diuresis. Crude protein (on an as fed basis) should be 11% to 16% (dry food) and 3% to 4% (canned food). These diets also usually are formulated to contain an alkalinizing agent, often potassium citrate. In theory, a vegetarian diet may be beneficial but this is unproven. A hydrolyzed vegetable protein diet, which does not contain organ meats that are high in protein, can be fed. Nutritional management principles are similar in dogs with urate uroliths and liver disease.

■ **Treats** – Low-protein treats, such as carbohydrates or vegetables, can be fed. Avoid high-protein treats, such as meats or cheeses, or treats that result in aciduria.

■ **Tips for Increasing Palatability** – Adding water to food may increase palatability and result in polyuria that may dilute calculogenic components. Adding light salt (potassium chloride) may stimulate thirst resulting in polyuria that may dilute calculogenic components.

■ **Diet Recommendations** – Low-protein or ultra-low protein diets that are alkalinizing and induce a diuresis, such as renal failure diets and advanced renal failure diets, are recommended. Feed to maintain body weight and body condition.

Client Education Points

For dogs without liver disease:

- Urate uroliths form because of an inborn error of metabolism of uric acid.
- Urate uroliths are highly recurrent without dietary modification and possibly administration of allopurinol, a xanthine oxidase inhibitor that decreases the amount of uric acid in urine.
- Diets formulated for management of urate uroliths are effective, but not 100%.
- Treats even in small amounts can result in recurrence of uroliths and should be given sparingly.

For dogs with liver disease:

- Urate uroliths form due to an underlying liver condition.
- Management of the liver condition, especially if surgical correction of a

- blood vessel bypassing the liver, is often curative.
- In other dogs, medical management of the liver disease often helps to prevent recurrence of or formation of urate uroliths.

Common Comorbidities

Hepatoencephalopathy and liver failure are seen in dogs with congenital liver disease and urate urolithiasis. Dilated cardiomyopathy has been associated with urate urolithiasis in English bulldogs and Dalmatians. Dalmatians with urate uroliths often have miliary dermatitis.

Interacting Medical Management Strategies

The xanthine oxidase inhibitor allopurinol competitively inhibits conversion of hypoxanthine to xanthine and xanthine to uric acid, thereby decreasing urinary uric acid concentration. The dosage for dissolution of urate uroliths is 15 mg/kg orally (PO) every 12 hours; for prevention, if used, 7–10 mg/kg PO every 12 to 24 hours can be given. The most common complication is xanthine crystalluria and urolith formation; occasionally dermatitis may occur. Xanthine oxidase is not effective in dogs with liver disease.

The urinary alkalinizing agent potassium citrate increases solubility of uric acid in urine and decreases urinary ammonia excretion.

In dogs with urate uroliths secondary to congenital liver disease with hyperammonemia and hyper-uric academia, antibiotics (e.g., neomycin, amoxicillin) to decrease intestinal bacterial counts and/or lactulose may be necessary to decrease absorption of ammonia from the intestinal tract. Other treatments to consider include S-adenosylmethionine (SAMe), vitamin E, and milk thistle or silymarin.

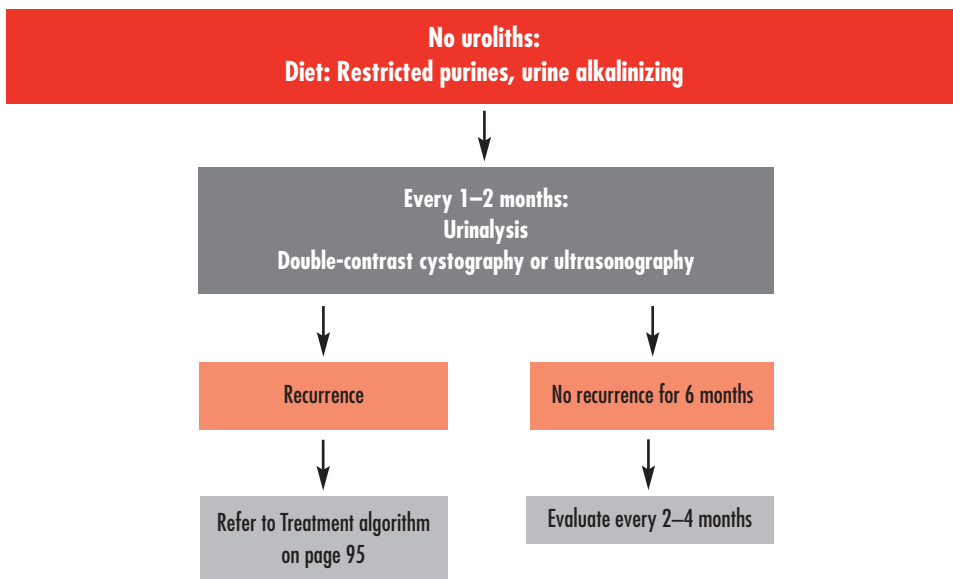
Monitoring

Dogs with urate uroliths but without liver disease should be monitored monthly during dissolution with abdominal ultrasonography or double-contrast cystography and urinalysis (pH should be alkaline, specific gravity should be dilute, crystalluria should be absent). For prevention, for first 6 months following dissolution or surgical removal of uroliths, perform urinalysis every 1 to 2 months (pH should be alkaline, specific gravity should be dilute, crystalluria should be absent). Consider monitoring BUN because low protein intake should result in decreased BUN concentration. Abdominal ultrasonography or double-contrast cystography is recommended at 6 months and 12 months. If no recurrence, consider monitoring urinalysis every 4 to 6 months thereafter unless there is recurrence of clinical signs.

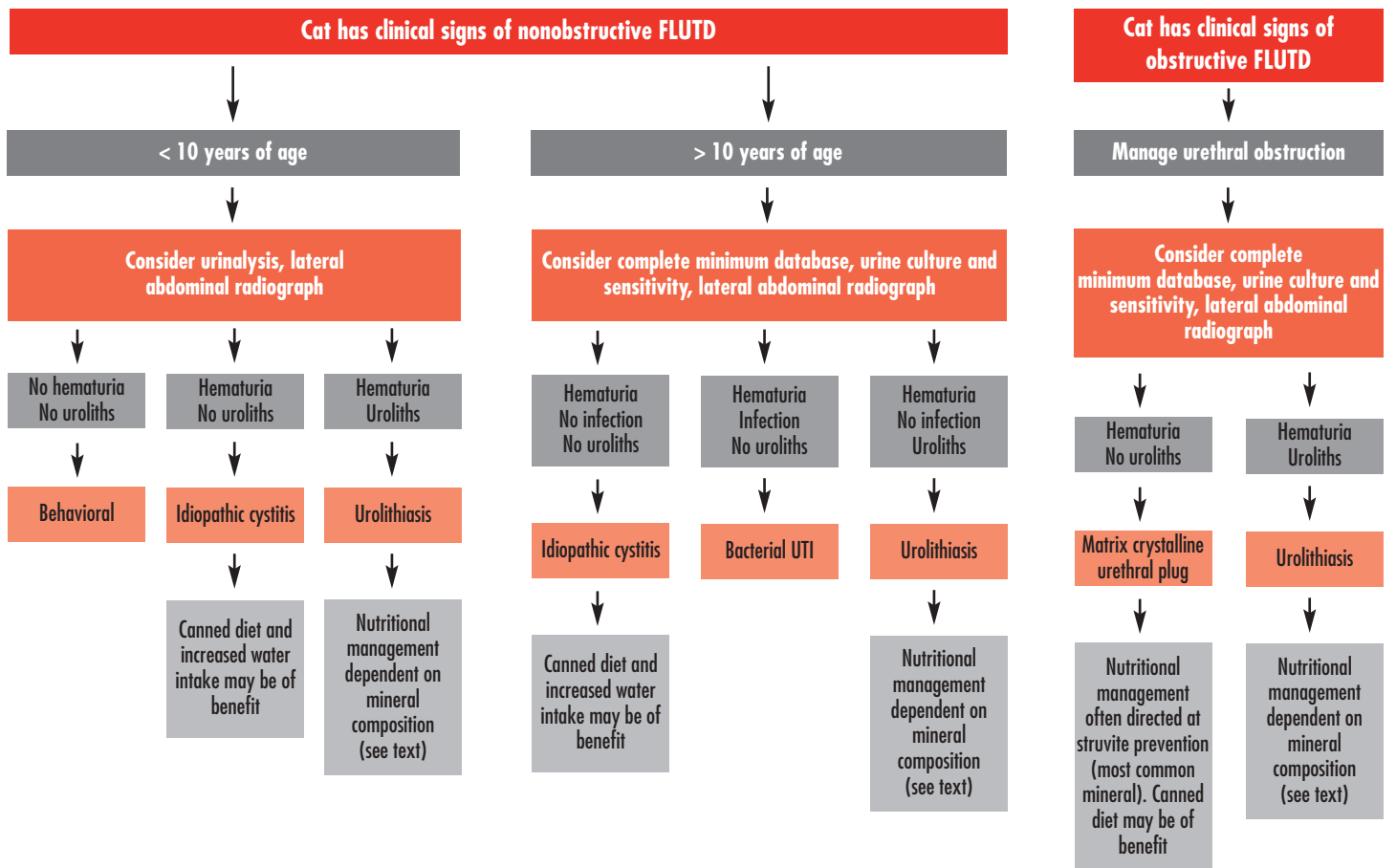
In dogs with urate uroliths and liver disease, if surgical correction of portovascular shunt is possible, no further monitoring may be required as correction of the shunt should eliminate urate urolith recurrence. If surgical correction is not possible, monitoring will depend on severity of clinical signs and response to treatment. Monitor urinalysis and blood work every 3 to 6 months.

See Algorithm – Nutritional Management for Treatment of Canine Urate Urolithiasis on page 95.

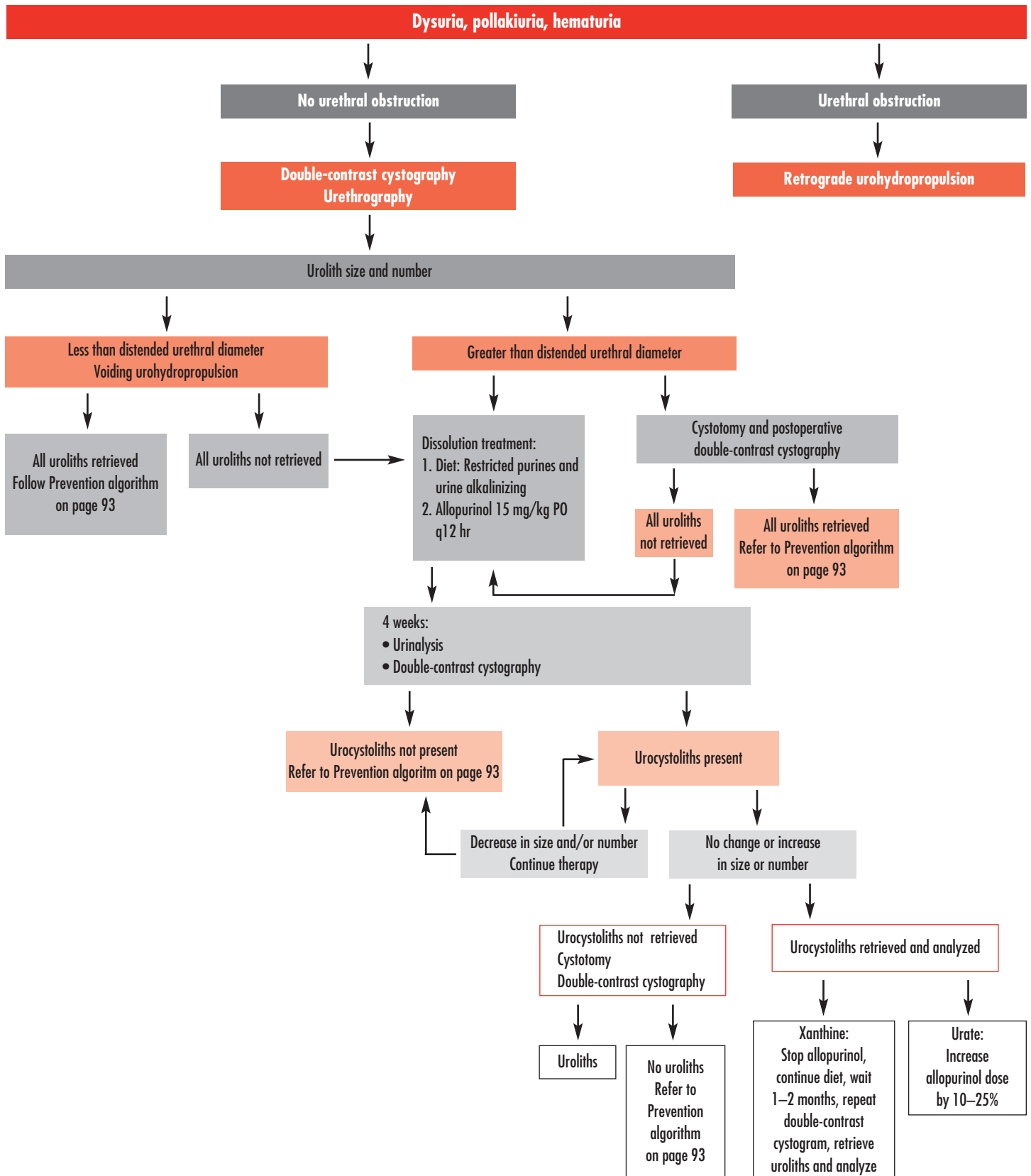
Algorithm – Prevention of Canine Urate Urocystolithiasis



Algorithm – Nutritional Management of Feline Lower Urinary Tract Disease



Algorithm – Nutritional Management for Treatment of Canine Urate Urolithiasis



Diabetes Mellitus & Kidney Disease – Feline

Rebecca Remillard, PhD, DVM, DACVN

Definition

In feline *diabetes mellitus* blood glucose concentrations cannot be maintained in the normal range as a result of low insulin secretion from pancreatic beta cells. *Renal disease* is abnormal renal function with a decreased glomerular filtration rate (GFR) and possibly urine-concentrating ability without azotemia. *Renal insufficiency* is abnormal renal function with a decreased GFR, urine concentration, and mild azotemia. *Renal failure* is severe azotemia associated with some or all of the systemic manifestations of uremia. For more on diabetes in cats, see pages 30–31; for more on feline kidney disease, see pages 86–87.

Key Diagnostic Tools and Measures

Persistent hyperglycemia over 2 days is indicative of diabetes mellitus. Renal disease is assessed by urine specific gravity of 1.014 or less and increased serum creatinine. Both conditions have concurrent clinical signs of polyuria, polydipsia, and history of weight loss.

Pathophysiology

More than 80% of cats are thought to have Type II diabetes mellitus, which is a relative insulin deficiency because the amount of insulin actually secreted may be increased, decreased, or normal but is always inadequate relative to serum glucose levels. Diabetes mellitus is characterized by peripheral insulin resistance combined with dysfunctional beta cells. The incidence of renal disease increases with age and generally is associated with renal cell injury and death. Dysfunction occurs in one or more categories of renal function: glomerular filtration, membrane selectivity, urine concentration, tubular resorption, or endocrine function. Diabetic nephropathy as seen in people is not well recognized in cats.

Signalment

Diabetes mellitus affects cats of any age and gender but is diagnosed more commonly in neutered male cats older than 6 years of age (usually between 10 and 13 years) with no particular breed predilection. Renal disease occurs in all age groups but is seen more commonly in cats over 10 years of age (7.7% of cases), and more so over 15 years of age (15.3% of cases). In one report, renal disease was recognized more than twice as often in Maine coon, Abyssinian, Siamese, Russian Blue, and Burmese breeds.

Key Nutrient Modifications

Diets low in soluble carbohydrate^a (CHO) (<20% on a dry matter [DM] basis) are considered superior for the management of diabetes mellitus. Grains suggested to have a lower glycemic index in the cat include corn, sorghum, oats, and barley. By limiting dietary CHO, blood glucose is maintained primarily from dietary protein using hepatic gluconeogenesis which releases glucose into the circulation at a slow and steady rate. Diabetic cats require high-quality protein ($\geq 30\%$ DM and $>85\%$ digestible) of high biologic value.

The mainstay of dietary management of renal disease is to reduce the nitrogenous waste products, hence avoiding an excessive (30–35% DM) quantity of protein is recommended. Dietary protein intake should be adjusted to minimize azotemia. Increasing protein quality decreases deamination of nonessential amino acids, thereby decreasing production of nitrogenous waste. Dietary phosphorous restriction (0.3–0.5% DM) has been shown to slow the progression of renal disease. The CHO, fat, and

^a Soluble CHO (mostly starch) is measured and reported as Nitrogen Free Extract (NFE) whereas CHO as fiber is reported as Crude Fiber.

fiber fraction can be adjusted to allow for a lower protein as needed to control azotemia. Hence, low CHO and phosphate diets with moderate protein should be adequate in most cases.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	30–35	6–8	26	6.5
Fat	15–30	4–9	9	2.3
Carbohydrate	15–35	3–6	n/a	n/a
Phosphorus	0.3–0.5	0.07–0.12	0.5	0.13

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary DM and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

The management goals of feline diabetes are to avoid insulin-induced hypoglycemia and hyperglycemic episodes and to optimize the chance of achieving diabetic remission. It is generally recommended that diabetic cats be fed twice daily at the time of the insulin injections, although it is acceptable to provide smaller meals more frequently.

It is not yet clear which food profile (high-fiber vs. low-carbohydrate) provides optimal glycemic control. Feeding low-carbohydrate foods is associated with a reversion rate of clinical diabetes to a non-insulin-dependent state by threefold compared with feeding high-fiber foods. Reversion has occurred, however, when feeding the high-fiber option and glucose control is not significantly different in cats that remain insulin-dependent. Dietary management of the comorbidities must also be considered when selecting diets for the diabetic patient.

The goals of management for renal disease are to minimize the azotemia and electrolyte aberrations. Cats should be fed a canned diet to increase water intake fortified with B vitamins and antioxidants. Sodium and potassium intakes need be adjusted on an individual basis but a diet containing alkalinizing agents will help to manage the acidosis. Cats generally do better fed multiple small meals (four to six per day) to minimize the postprandial load to the kidney.

■ **Treats** – Maintaining a low carbohydrate, protein and phosphorous intake is important. Suitable examples include portions of the cat's recommended diet or kibble forms of comparable diets.

■ **Tips for Increasing Palatability** – Transition the diet change from the regular diet to the suitable diabetic diet over 5 to 14 days; longer times for cats that are more resistant to change. The palatability of food generally increases with increased temperature, water, and the nutrients fat, protein, and salt. Warm (microwave) food or lightly warm canned food. Add warm chicken or beef broth (low salt), water or oil from canned fishes (sardine, tuna, mackerel), if appropriate, to enhance taste.

■ **Diet Recommendations** – Nutrient ranges of diets recommended for diabetic cats with renal disease are <35% CHO, 30% protein, and 30% fat with <0.5% phosphorous DM basis. Cats should be fed to maintain or achieve an ideal body weight. Canned foods are generally more palatable; contain more water, fat, and less CHO than kibble. Consider a specifically designed home-made diet to meet multiple dietary needs if commercial products are unsuccessful.

Client Education Points

- Feed meals at the time of insulin injection at 12-hour intervals. It is recommended that only food products designed for a diabetic cat with low phosphorous be fed, and that the food is obtained from a reliable source for quality control and product consistency.
- Cats can become non-insulin-dependent, hence close monitoring is essential.
- Cats may be fed smaller meals between insulin if tolerated.
- Cats with mild to moderate signs of hypoglycemia such as weakness, trembling, and wobbliness that are still able to eat should be immediately fed a renal type diet. If signs are severe, such as seizure or coma, glucose syrup designed for human diabetic patients can be rubbed into the gums, and owners should seek veterinary attention immediately.

Common Comorbidities

Comorbidities are very common in diabetic cats with renal disease. For cats that are also overweight or obese, feed a high-fiber, low-CHO food. Insulin sensitivity may return as adiposity decreases. Cats that also have pancreatitis or cancer (adenocarcinoma) can still be fed low-CHO, high-protein

diabetic foods. Other common comorbidities include bacterial cystitis and urinary tract infections, hyperlipidemias (change to a lower-fat, low-CHO diabetic food), endocrinopathies (hyperadrenocorticism, acromegaly), and drug-induced conditions (glucocorticoids, progestins). For stress hyperglycemia associated with illness, manage as a diabetic until resolved.

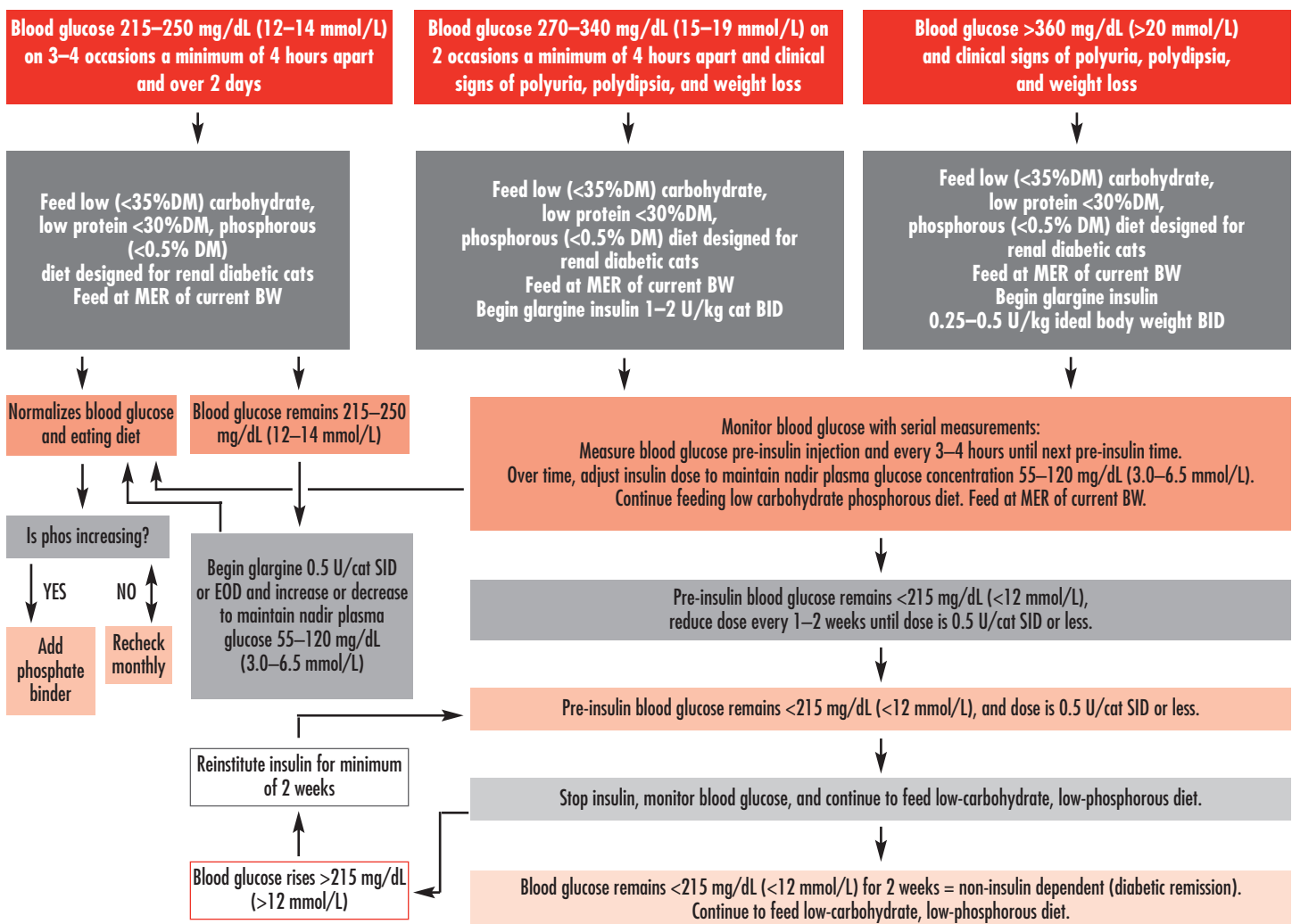
Interacting Medical Management Strategies

Normalizing serum phosphorous should first be attempted through feeding a lower phosphorous diet for 2 to 4 weeks before adding intestinal phosphate binders. Acarbose is a useful adjunct for managing diabetic cats with advanced renal disease that are meal-fed a restricted-protein diet. It is less effective for cats eating multiple small meals daily.

Monitoring

Blood and urinary glucose and ketone concentrations need to be monitored to determine the level of glycemic control, along with regular blood and urine specific gravity tests to monitor progression of renal disease. Monitor body weight and adjust energy intake to achieve an ideal weight.

Algorithm – Nutritional Management of Feline Diabetes Mellitus with Creatinine >2.0



Diabetes Mellitus & Obesity – Feline

Rebecca Remillard, PhD, DVM, DACVN

Definition

In feline *diabetes mellitus* blood glucose concentrations cannot be maintained in the normal range as a result of low insulin secretion from pancreatic beta cells. *Obesity* is qualitatively defined as an excess of body fat sufficient to contribute to disease. For more on diabetes in cats, see pages 30–31; for more on overweight and obesity in cats, see pages 36–37.

Key Diagnostic Tools and Measures

Persistent hyperglycemia over 2 days is indicative of diabetes mellitus. Concurrent clinical signs of polyuria, polydipsia, and history of weight loss are common although the cat may still be overweight. Obesity has been linked to peripheral insulin resistance. Overweight cats (body condition score [BCS] of 6/9 or 7/9) carry 25% and 30% body fat, respectively. Obese cats (BCS 8/9) carry 35% fat, whereas morbidly obese cats (BCS 9/9) carry 40% or more fat (see Appendix I).

Pathophysiology

The diet history for overweight or obese cats will show chronic caloric excess. Obesity has been shown to result in abnormal glucose tolerance and peripheral insulin resistance. These factors are known to precede the development of Type II diabetes mellitus in cats. The risk of developing diabetes increases by nearly fourfold in obese cats. More than 80% of cats with diabetes are thought to have Type II diabetes, with the remaining cases secondary to other conditions.

Signalment

Diabetes mellitus affects cats of any age and gender but is diagnosed more commonly in neutered male cats older than 6 years of age (usually between 10 and 13 years) with no particular breed predilection, which is the same population most at risk for obesity.

Key Nutrient Modifications

Diets low in soluble carbohydrate^a (CHO) (<20% on a dry matter [DM] basis) are considered superior for the management of DM. In lowering the CHO fraction, the content of protein, fat, fiber, or some combination thereof must increase to account for the difference. It is logical to replace the carbohydrates with protein as opposed to fat in overweight cats because dietary fat is known to increase insulin resistance and decrease glucose tolerance. Low CHO diets with low levels of fat and calories but higher in protein and fiber can be used to achieve weight loss in diabetic cats.

Diabetic cats losing adipose require adequate protein ($\geq 30\%$ DM and $>85\%$ digestible) of high biologic value to maintain lean tissue. Low calorie, high-fiber diets provide similar protein levels to low-carbohydrate, high-protein foods as a percent of metabolizable energy (ME). Fiber aids in glycemic control by promoting slow and sustained gastrointestinal absorption of glucose after meals. A diet including moderate amounts of mixed fiber (5%–12% DM) also aids in weight management. Hence, low CHO and fat diets with $\geq 30\%$ protein should be adequate in most overweight diabetic cases.

^a Soluble CHO (mostly starch) is measured and reported as Nitrogen Free Extract (NFE) whereas CHO as fiber is reported as Crude Fiber.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	35–60	8–17	26	6.5
Fat	6–20	2.5–5	9	2.3
Carbohydrate	5–25	1.5–6	n/a	n/a
Fiber	5–12	2–4	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary DM and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

The management goals in feline diabetes are to avoid insulin-induced hypoglycemia and hyperglycemic episodes and optimize the chance of achieving diabetic remission. For weight management, low-fat, high-fiber foods are used to reduce caloric intake and body weight while attempting to maintain satiety. These two dietary strategies are not exclusive.

Fiber-fortified foods originally designed for weight control (3.5 kcal/g DM) are appropriate for weight reduction and glycemic control in overweight cats. Cats can regulate their food intake and these foods have low caloric densities. Owner and patient compliance may be easier to achieve when feeding a larger food volume (e.g., fiber-fortified foods).

Grains with a lower glycemic index in the cat that also add fiber to the diet include sorghum, oats, and barley. Reversion to a non-insulin-dependency has occurred when feeding the high-fiber option, and for cats that remain insulin dependent, glucose control is not significantly different from those fed low-fiber diets. Hence, low-CHO, high-protein, low-fat with moderate fiber foods are suitable for weight loss and blood glucose control in the overweight diabetic cat.

■ **Treats** – Maintaining a constant and low carbohydrate intake is important, and high-CHO treats should be avoided. Suitable examples include portions of the cat's usual low-CHO, moderate-fiber diet or home-cooked lean meat jerky treats with little or no fat. Calories provided by treats should be part of the calculated daily energy intake (white meat chicken, game meat, jerky treats with little or no fat). Calories provided by treats should be part of the calculated daily energy intake.

■ **Tips for Increasing Palatability** – Transition the diet change from the regular diet to the suitable diabetic diet over 5 to 14 days; longer times for cats that are more resistant to change. The palatability of food generally increases with increased temperature, water and nutrients fat, protein and salt. Warm (microwave) canned food and/or add warm chicken or beef broth (+/- sodium) to enhance taste to dry food.

■ **Diet Recommendations** – Nutrient ranges of diets recommended for overweight diabetic cats are ~15% CHO, ~55% protein, ~10% fat with 5% to 10% fiber on a dry matter (DM) basis. Cats should be fed at the maintenance energy requirement (MER) of current weight while changing the diet and then decrease daily food intake by 25 kcal to lose 0.5% to 1% body weight per week until a BCS of 5/9 or 6/9 has been achieved (see Appendix III). Consider a specifically designed home-made diet to meet multiple dietary needs if commercial products are unsuccessful.

Client Education Points

- Feed meals at the time of insulin injection at 12-hour intervals. It is recommended that only food products designed for an overweight diabetic cat be fed, and that the food is obtained from a reliable source for quality control and product consistency.
- Cats can become non-insulin-dependent as they lose weight, hence close monitoring is essential.
- The owner must recognize the cat's obesity and then be able and willing to control calorie intake for a weight loss program to succeed. Another key to success is a flexible design with regular follow-up with the client.
- Weight loss that includes dietary changes, measured food allotments (using a gram scale is ideal), and regular body weight checks by a veterinary health care team are successful. Weight loss is a slow steady progress which may take 12 to 18 months to reach a goal weight.

Common Comorbidities

Comorbidities are very common in obese diabetic cats. Cats that also have pancreatitis or cancer (adenocarcinoma) can still be fed low-CHO, moderate-fiber diabetic foods. Other comorbidities include renal insufficiency (change to a lower phosphorous diabetic food), bacterial cystitis and urinary tract infections, hyperlipidemias (change to a lower fat diabetic food if possible), endocrinopathies (hyperadrenocorticism, acromegaly), and drug-induced conditions (glucocorticoids, progestins). For stress hyperglycemia associated with illness, manage as a diabetic until resolved.

Recent research has suggested a mechanism for the link between excess body weight and many diseases. Obesity is now seen as a chronic pro-inflammatory state producing oxidative stressors. It seems that adipose tissue, once considered to be physiologically inert, is an active producer of hormones, such as leptin and resistin, and numerous cytokines. Of major concern are the pro-inflammatory cytokines for adipose (adipokines) TNF- α and interleukins

1 β and 6. Conditions linked to obesity include osteoarthritis, idiopathic cystitis, cardiovascular disease and pancreatitis due to a chronic low grade inflammation and oxidative state; orthopedic (cruciate tears) injuries due to excessive weight; idiopathic, bacterial, and crystal-related cystitis; hyperlipidemias and hepatic lipodosis; and non-allergic dermatitis due to the inability to properly self-groom.

Interacting Medical Management Strategies

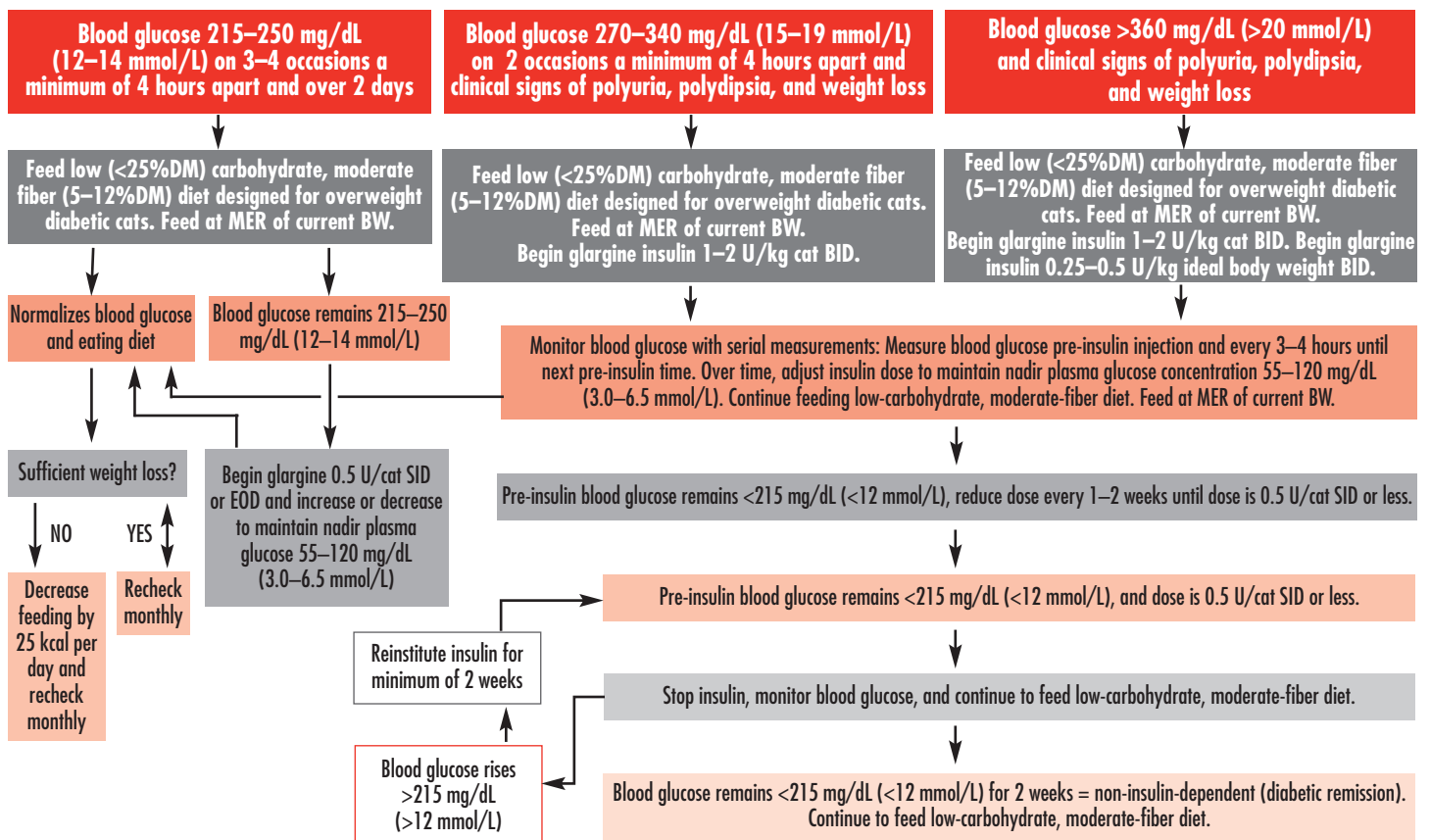
Feline hepatic lipodosis is rare in cats fed fewer calories for weight loss as long as the cat consumes the entire daily allotment. If the cat refuses to eat the weight loss diet within 24 hours, the possibility of hypoglycemia increases if insulin treatment continues. Diabetic cats receiving insulin must be monitored carefully as insulin requirement decreases as weight loss occurs and insulin sensitivity returns. Cats receiving any medications based on body weight must be monitored carefully for dose adjustment.

Monitoring

Blood and urinary glucose and ketone concentrations need to be monitored to determine the level of glycemic control. Exogenous insulin is administered with a low-CHO diet to control blood glucose concentrations, and is adjusted accordingly to maintain as close to a normal blood glucose concentration as possible.

By recording body weight and BCS, ideal body weight can be more easily determined. Physical examinations and weight checks are suggested monthly with a discussion about daily feeding regime and food measurements. Behavioral changes in feeding the cat are essential. Discuss logistical feeding problems within the household (e.g., multiple-cat household, boarding, family members, visitors). Change food and adjust calorie intake as needed. Clients should be encouraged to develop non-food-related bonding activities.

Algorithm – Nutritional Management of Feline Diabetes Mellitus with BCS >6/9



Diabetes Mellitus and Crystal-Related Cystitis – Feline

Rebecca Remillard, PhD, DVM, DACVN

Definition

In feline *diabetes mellitus* blood glucose concentrations cannot be maintained in the normal range as a result of low insulin secretion from pancreatic beta cells with and without peripheral insulin resistance. Cats with *lower urinary tract disease* exhibit hematuria, stranguria, pollakiuria without an identifiable cause. For more on diabetes in cats, see pages 30–31; for more on lower urinary tract disease in cats, see pages 90–91.

Key Diagnostic Tools and Measures

Hyperglycemia on three to four successive blood glucose measurements taken at least 4 hours apart over 2 days is indicative of diabetes mellitus. Concurrent clinical signs of polyuria, polydipsia, and history of weight loss are common. Cats are rarely ketonuric but may have glucosuria and ketonemia. Fructosamine concentrations between 400 and 500 $\mu\text{mol/L}$ are supportive of diabetes while concentrations greater 500 $\mu\text{mol/L}$ are highly associated with diabetes. Clinical signs of stranguria and pollakiuria with a positive urinalysis (pH, specific gravity, presence of blood, crystals, bacteria) are indicative of feline lower urinary tract disease (FLUTD).

Pathophysiology

More than 80% of cats with diabetes are thought to have Type II diabetes mellitus, which is a relative insulin deficiency because the amount of insulin actually secreted may be increased, decreased, or normal, but is always inadequate relative to serum glucose levels. FLUTD is a syndrome of clinical signs with a multitude of etiologies. The causes of the cystitis (idiopathic, bacteria, crystals) appear to be different in cats of different ages. Cats with calcium oxalate-related cystitis should be checked for hypercalcemia.

Signalment

Diabetes affects cats of any age and gender but is diagnosed more commonly in neutered male cats older than 6 years of age (usually between 10 and 13 years) with no particular breed predilection. FLUTD in cats less than 10 years of age is most often idiopathic; in cats under 1 and over 10 years of age it is likely to be bacterial cystitis. In cats with crystal-related cystitis, 90% of cases are due to struvite or calcium oxalate.

Key Nutrient Modifications

Diets low in soluble carbohydrate^a (CHO) (<20% on a dry matter [DM] basis) are considered superior for the management of DM. Grains suggested to have a lower glycemic index in the cat include corn, sorghum, oats, and barley. By limiting dietary carbohydrates, blood glucose is maintained primarily from hepatic gluconeogenesis, which releases glucose into the circulation at a slow and steady rate. Blood glucose fluctuations after a low CHO meal are minimized. In lowering the CHO fraction, the content of protein, fat, fiber, or some combination thereof must increase to account for the difference. Diabetic cats with struvite crystal-related cystitis require lower-magnesium, urine-acidifying canned diets. Diabetics with calcium oxalate-related cystitis require potassium citrate in addition to a canned diet. There are no dietary changes for idiopathic and bacterial-related FLUTD.

^a Soluble CHO (mostly starch) is measured and reported as Nitrogen Free Extract (NFE) whereas CHO as fiber is reported as Crude Fiber.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	40–60	10–17	26	6.5
Fat	15–30	3.5–6	9	2.3
Carbohydrate	10–35	3–7	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary DM and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

The goals of management in feline diabetes are to avoid insulin-induced hypoglycemia and hyperglycemic episodes and to optimize the chance of achieving diabetic remission. It is generally recommended that diabetic cats be fed twice daily at the time of the insulin injections, although it is acceptable to provide smaller meals more frequently.

It is not yet clear which food profile (high fiber vs. low carbohydrate) provides optimal glycemic control. Feeding low-carbohydrate foods is associated with a reversion rate of clinical diabetes to a non-insulin-dependent state by threefold compared with feeding high-fiber foods. Reversion has occurred, however, when feeding the high-fiber option and glucose control is not significantly different in cats that remain insulin dependent. Dietary management of the comorbidities must also be considered when selecting diets for the diabetic patient.

Diabetic cats with crystal-related cystitis should be fed canned diabetic diets. Cats with calcium oxalate-related cystitis may do better on a high-fiber (less acidifying), low-oxalate (less acidifying), low-carbohydrate diabetic food with 40 to 75 mg potassium citrate per kg BW added to each BID meal. Cats with struvite-related FLUTD should be fed a lower-magnesium, low-carbohydrate urine-acidifying canned diet.

■ **Treats** – Maintaining a constant and low-CHO intake is important, and high-CHO treats should be avoided. Suitable examples include portions of the cat's usual low-CHO diet or home-cooked meat or fish treats for struvite-related cystitis. Meat treats may not be suitable for calcium oxalate-related cystitis as they may lower urine pH.

■ **Tips for Increasing Palatability** – Transition from the regular diet to the suitable diabetic diet should be done over 5 to 14 days or longer for cats that are more resistant to change. The palatability of food generally increases with increased temperature, water, and nutrients (fat, protein, and salt). Warm (microwave) food or lightly warm canned food. Add warm chicken or beef broth (low salt) or add water or oil from canned fishes (sardine, tuna, mackerel) if appropriate to enhance taste.

■ **Diet Recommendations** – Nutrient ranges of low-CHO diets recommended for diabetic cats with struvite-related cystitis are <20% CHO, 30% to 60% protein, and 10% to 25% fat with <0.08% magnesium (Mg) (DM basis) or <20 mg Mg per 100 kcal basis. Cats should be fed to maintain or achieve an ideal body weight. Canned foods are generally more palatable and contain more water, fat, and less CHO than kibble. Consider a specifically designed home-made diet with potassium citrate to meet multiple dietary needs if commercial products are unsuccessful.

Client Education Points

- Feed meals at the time of insulin injection at 12-hour intervals. It is recommended that only food products designed for a diabetic cat be fed, and that the food is obtained from a reliable source for quality control and product consistency.
- Cats can become non-insulin-dependent, hence close monitoring is essential.
- Cats with mild to moderate signs of hypoglycemia such as weakness, trembling, and wobbliness that are still able to eat should be immediately fed a palatable highly digestible, high-CHO, low-fiber “intestinal” diet. If signs are severe, such as seizure or coma, glucose syrup designed for human diabetic patients can be rubbed into the gums, and owners should seek veterinary attention immediately.
- Monitor for clinical signs of FLUTD.

Common Comorbidities

Comorbidities are very common in diabetic cats with FLUTD. Cats that are also overweight or obese should be fed a high-fiber, low-CHO food. Insulin sensitivity may return as adiposity decreases. Cats that also have pancreatitis or cancer (adenocarcinoma) can still be fed a low-CHO, high-protein diabetic foods. For renal insufficiency, change the diet to a

lower-protein, low-CHO diabetic food. For hyperlipidemias, change to a lower-fat, low-CHO diabetic food. Other common comorbidities include endocrinopathies (hyperadrenocorticism, acromegaly) and drug-induced conditions (glucocorticoids, progestins). For stress hyperglycemia associated with illness, manage as a diabetic until resolved.

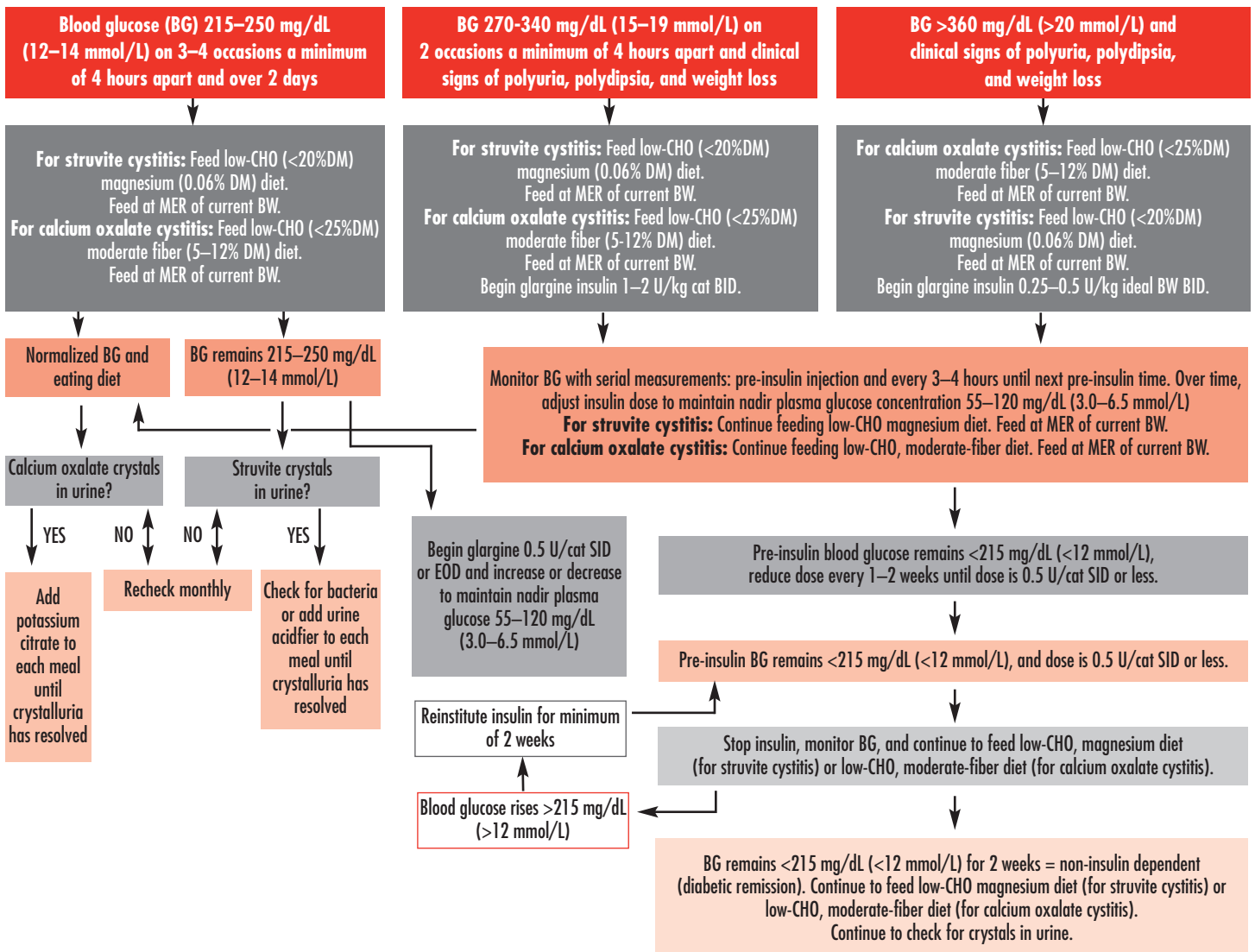
Interacting Medical Management Strategies

Monitor urine sediment and pH. If calcium oxalate crystals are present and pH is too low, add urine alkalinizer (potassium citrate) for calcium oxalate-related cystitis. If struvite crystals are present and pH is too high, add a urine acidifier for struvite-related cystitis.

Monitoring

Blood and urinary glucose and ketone concentrations need to be monitored to determine the level of glycemic control. Exogenous insulin is administered with a low-CHO, high-protein diet (preferably) to control blood glucose concentrations, and is adjusted accordingly to maintain as close to a normal blood glucose concentration as possible. Monitor body weight and adjust energy intake to achieve an ideal weight. On urinalysis, sediment should be crystal free, with a pH <6.5 for struvite prevention or pH 6.6-7.5 for calcium oxalate prevention.

Algorithm – Nutritional Management of Feline Diabetes Mellitus and Crystal-Related Cystitis



Pancreatitis & Chronic Kidney Disease – Canine

Jennifer Larsen, DVM, PhD, DACVN

Definition

Dogs with both *pancreatitis* and *chronic kidney disease* (CKD) suffer from a mild to severe inflammatory process affecting exocrine or endocrine functions, or both, of the pancreas. Glomerular and/or renal tubular disease resulting in azotemia, reduced urine-concentrating ability, and polyuria/polydipsia (PU/PD), with or without associated clinicopathologic abnormalities (anemia, proteinuria, hypoproteinemia, hypercalcemia, hyperphosphatemia, hypo- or hyperkalemia) may exist concurrently. For more on pancreatitis in dogs, see pages 68–69; for more on chronic kidney disease, see pages 84–85.

Key Diagnostic Tools and Measures

Consider history, physical examination, clinical signs, and minimum database: serum biochemical analysis, complete blood count (CBC), urinalysis, and diet history. Abdominal ultrasound, serum canine pancreatic lipase immunoreactivity (cPLI) and, ideally, biopsy can provide valuable additional information, and can confirm a preliminary diagnosis.

Pathophysiology

The inciting cause of canine pancreatitis often is unknown, but it is frequently associated with higher-fat diets, dietary indiscretion, hyperlipemia, and obesity. Gastrointestinal (GI) signs are common (inappetence, vomiting, diarrhea, pain). If chronic, it can lead to exocrine pancreatic insufficiency.

Chronic kidney disease is associated with accumulation of metabolic products of protein catabolism and other compounds usually excreted in urine. While creatinine is considered a crude index of glomerular filtration, BUN is a marker of dozens of other nitrogenous waste compounds that impact appetite, smell, and taste. Hypergastrinemia from reduced renal clearance leads to GI mucosal irritation and ulceration, as well as acidosis. Other uremic toxins and reduced renal function result in many other abnormalities, including alterations in mineral metabolism (impaired urinary phosphorus clearance, secondary hyperparathyroidism, altered vitamin D metabolism, hypercalcemia).

Signalment

Middle-aged or older dogs, particularly Yorkshire terriers and miniature schnauzers, appear to be predisposed to pancreatitis. Obesity, concurrent metabolic diseases (such as diabetes mellitus, hyperadrenocorticism, hypothyroidism), drugs (such as thiazide diuretics, furosemide, azathioprine, tetracycline), ischemia, hyperlipemia, and bile duct obstruction or trauma can also predispose dogs to pancreatitis and CKD.

Key Nutrient Modifications

Fat restriction is required to decrease pancreatic stimulation; the level of restriction is determined by diet history and if possible, identifying the fat level of the diet or treat(s) eaten when pancreatitis occurred. For treatment and prevention, provide a dietary fat level significantly less than that in the prior diet. Protein restriction is needed to manage azotemia, and phosphorus restriction is indicated to reduce the risk of soft tissue mineralization and slow progression of CKD. Supplementation of B vitamins replenishes losses secondary to polyuria. Sodium restriction helps manage hypertension and/or fluid retention. Supplementation of omega-3 fatty acids has an anti-inflammatory effect and is renoprotective. If necessary, a canned diet can help attenuate dehydration.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Fat	8–12	2.0–3.5	5	1.4
Protein	15–24	3.5–6.0	18	5.1
Phosphorus	0.25–0.4	0.05–0.1	0.5	0.14
Sodium	0.2–0.3	0.04–0.07	0.06	0.02

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

As for all patients, the primary goal of nutritional management of the dog with both pancreatitis and renal failure is to meet energy requirements with a diet inclusive of nutrient modifications specific for the disease. The pancreas is stimulated by gastrin release, sensory stimuli (anticipation of feeding), dietary fat, and protein. Reducing these stimuli, especially dietary fat, can help control clinical signs and avoid further exacerbation of inflammation. Vomiting is a common clinical sign in dogs and enteral feeding is contraindicated during episodes of severe vomiting (as during acute pancreatitis) to reduce the risk of aspiration and decrease further stimulation of emesis. Once rehydrated and stabilized, treatment should include pain management, gastric protectants, and anti-emetic therapy, if necessary. Severe cases involving refractory vomiting and/or prolonged anorexia (longer than 5 days) should be fed parenterally or post-gastrically with an enteral device (jejunostomy tube). Once water is tolerated orally, small amounts of highly digestible, high carbohydrate foods can be reintroduced. For longer-term management and for chronic cases, dietary fat should be restricted to below the level in the diet associated with the onset of disease. Therefore, the level of fat restriction should be determined for each individual patient based on history.

Feeding strategies for CKD focus on slowing the progression of disease (phosphorus restriction and supplementation with long-chain omega-3 fatty acids) as well as managing clinical signs (avoiding acidification, protein and sodium restriction, supplementation with B vitamins, and modifying levels of calcium, vitamin D, and potassium, if necessary). Achieving these goals in the face of acute or chronic pancreatitis is not usually counter to providing a low-fat diet; however, the energy density of the diet will be lower if the patient requires severe fat restriction. In addition, if the patient requires fat restriction in excess of that provided by commercially available diets formulated for the management of CKD, then a home-cooked diet formulation is indicated. However, consider that the canned and dry versions of commercially available prescription diets formulated for the management of CKD will typically vary in fat content.

■ **Treats** – Treats should be low in fat, protein, phosphorus, and sodium. Total daily treats should be provided at no more than 10% of the daily caloric intake. Examples include fruits (apple, berries, peaches), vegetables (green beans, carrots, sugar snap peas), and Kellogg's Frosted Mini-Wheats. Avoid foods known to be toxic to dogs: grapes and raisins, onions and garlic, macadamia nuts, bread dough, and chocolate.

■ **Tips for Increasing Palatability** – Altering the moisture level of the diet by soaking kibble or baking canned diets can increase acceptance in some pets. Heating the diet can also be useful. The owner should provide a calm

and safe environment for eating and can also try positive reinforcement. If necessary, the daily treat allowance (up to 10% of the daily calories) can be used to add appropriate food items to the meals.

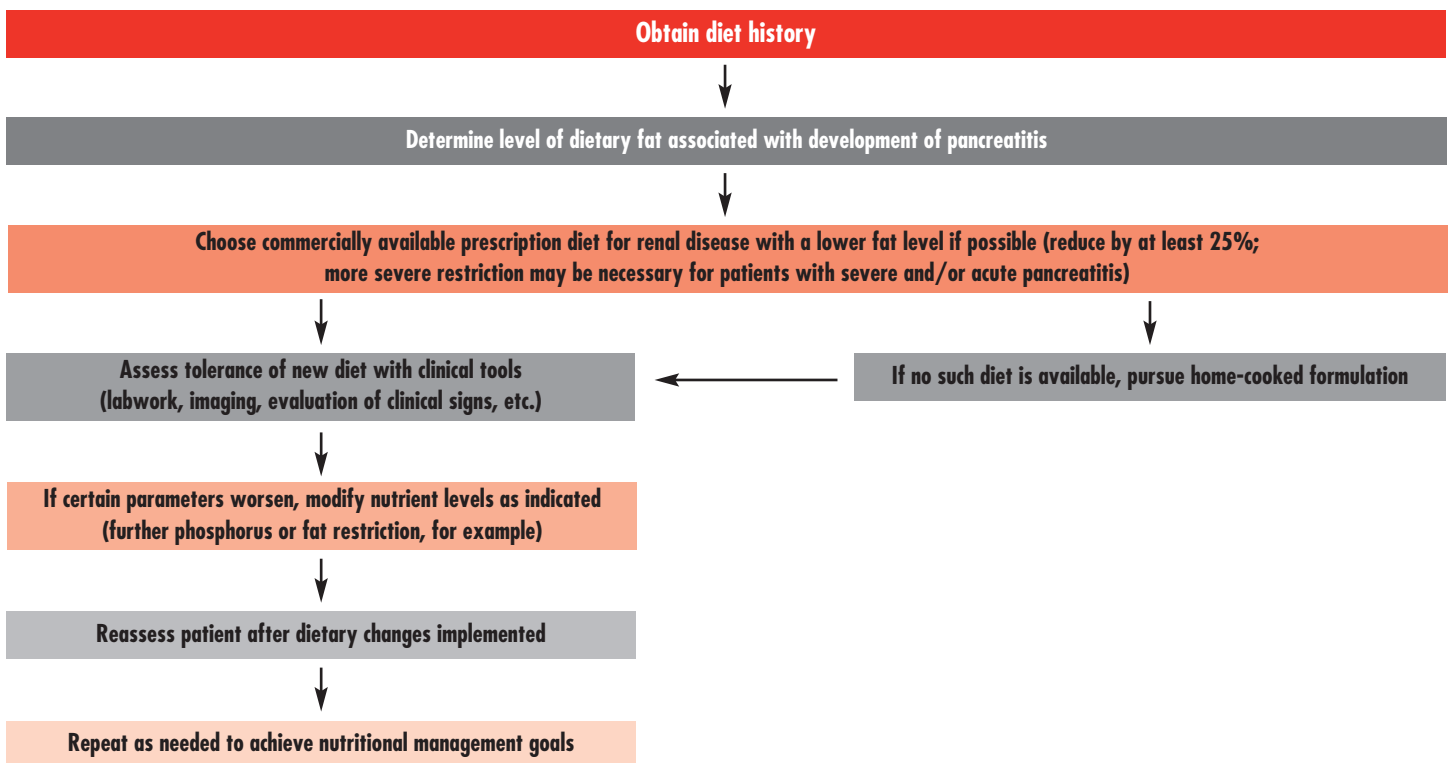
■ **Diet Recommendations** – For critically ill animals, initially feed resting energy requirements (RER; $70 \times BW$ in $kg^{0.75}$), monitor body weight, and adjust as necessary. For more chronic and stable cases, provide true maintenance energy requirements, if possible (MER; the amount of calories that has maintained stable body weight). If the diet history is not complete enough to determine this, then estimate MER by calculation. MER can be determined by calculating RER for the current weight and multiplying by the appropriate factor: 1.4 for dogs prone to obesity, 1.6 for neutered dogs, and 1.8 for intact dogs (see Appendix III).

Choose a diet that has been formulated for the management of CKD and that supplies fat levels below that in the diet suspected of contributing to the pancreatitis. If there is a history of excessive or inappropriate treating (especially with high-fat food items) or of dietary indiscretion, the fat level in the current diet may still be tolerated by the patient.

Client Education Points

- Monitoring and reassessment will be necessary on a regular basis.
- Diet compliance is important for patients with both diseases.
- Avoid treats high in fat, protein, sodium, or phosphorus.
- A feeding tube may be needed if the patient will not eat adequate amounts of an appropriate diet.
- Pancreatitis can recur despite a restricted fat diet and control of concurrent diseases.
- CKD in dogs progresses at variable rates in different patients, and comorbidities may negatively impact prognosis.

Algorithm – Nutritional Management of Concurrent Canine Pancreatitis and Chronic Kidney Disease



Common Comorbidities

Hypertriglyceridemia, GI upset from uremia or hypergastrinemia, calcium oxalate urolithiasis, obesity, and hypertension may occur in dogs with pancreatitis and CKD.

Interacting Medical Management Strategies

Use of angiotensin-converting enzyme (ACE) inhibitors in dogs with CKD may contribute to hyperkalemia. Phosphate binders may decrease palatability and cause constipation, anorexia, nausea, or vomiting. The sodium content of any parenteral fluids should be considered in hypertensive or otherwise sodium-sensitive patients.

Monitoring

cPLI and abdominal ultrasound (in addition if clinical signs) are useful for monitoring recovery from acute pancreatitis and for assessing chronic cases. Monitoring serum BUN/creatinine, phosphorus, calcium, and potassium levels, urine specific gravity, and blood pressure is useful for managing CKD. Regular assessment of the patient for urinary tract infections is indicated; also add serum albumin concentration and urine protein:creatinine ratio (UPC) for patients with glomerular disease. If diet is not effective in lowering serum phosphorus, add a binder. If mineral/electrolyte values are persistently deranged, a diet change may be indicated. If pancreatitis does not resolve with the current diet and control of comorbid conditions, further fat restriction is indicated. If azotemia or UPC is worsening, and diet compliance is confirmed, further protein restriction is indicated. Consider concurrent disease if UPC does not improve (e.g., Lyme nephritis).

Allergic Gastroenteritis/Inflammatory Bowel Disease & Chronic Kidney Disease – Canine

Jennifer Larsen, DVM, PhD, DACVN

Definition

Dogs with both *allergic gastroenteritis/inflammatory bowel disease* (IBD) and *chronic kidney disease* (CKD) suffer from a diet-responsive inflammatory process affecting immune, digestive, and absorptive functions of the gastrointestinal (GI) tract as well as glomerular and/or renal tubular disease resulting in azotemia, reduced urine-concentrating ability, and polyuria/polydipsia (PU/PD), with or without associated clinicopathologic abnormalities (anemia, proteinuria, hypoproteinemia, hypercalcemia, hyperphosphatemia, hypo- or hyperkalemia). For more on allergic gastroenteritis/IBD in dogs, see pages 62–63; for more on CKD, see pages 84–85.

Key Diagnostic Tools and Measures

Consider history, physical examination, clinical signs, and minimum database: serum biochemical analysis, complete blood count (CBC), urinalysis, and diet history. Serum tests for the diagnosis of food allergy are not reliable and should not be used. Response to a dietary change is likewise not diagnostic, as improvement or resolution of clinical signs of many GI diseases can occur due to a response to changes in fat or fiber levels, fiber types, digestibility, or secondary effects on intestinal microflora. Abdominal ultrasound, serum folate and cobalamin concentration, and ideally dietary elimination-rechallenge trials and biopsies can provide valuable additional information, and can confirm a preliminary diagnosis.

Pathophysiology

The inciting cause of allergic gastroenteritis/IBD often is unknown, but it may be a reaction to food components, bacterial antigens, and/or self-antigens. Upper or lower GI signs can be seen (inappetence, vomiting, diarrhea, borborygmus, flatulence). Chronic kidney disease is associated with accumulation of metabolic products of protein catabolism and other compounds usually excreted in urine. BUN is a marker of dozens of other nitrogenous waste compounds that impact appetite, smell, and taste. Hypergastrinemia from reduced renal clearance leads to GI mucosal irritation and ulceration as well as acidosis and abnormalities in mineral metabolism (impaired urinary phosphorus clearance, secondary hyperparathyroidism, altered vitamin D metabolism, hypercalcemia).

Signalment

Breeds predisposed to these conditions include Irish setters (for gluten-sensitive enteropathy) and German shepherd dogs and Shar-Pei (for lymphoplasmacytic enteritis).

Key Nutrient Modifications

Feeding a highly digestible, novel antigen, and/or a low-fat diet appear to be valuable strategies (novel ingredients determined by thorough diet history of individual patient). Altering the dietary fiber type and level can also have a beneficial effect. Pre- and probiotic therapy is useful for many cases. Protein restriction is needed to manage azotemia, and phosphorus restriction is indicated to reduce the risk of soft tissue mineralization and slow progression of CKD. Supplementation of B vitamins replenishes losses secondary to polyuria. Sodium restriction helps manage hypertension and/or fluid retention. Supplementation of omega-3 fatty acids has an anti-inflammatory effect and is renoprotective. If necessary, a canned diet can help attenuate dehydration.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	Recommended dietary level	
			% DM	g/100 kcal
Fat	8–12	2.0–3.5	5	1.4
Protein	15–24	3.5–6.0	18	5.1
Phosphorus	0.25–0.4	0.05–0.1	0.5	0.14
Sodium	0.2–0.3	0.04–0.07	0.06	0.02

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

As for all patients, the primary goal of nutritional management of the dog with both allergic gastroenteritis/IBD and CKD is to meet energy requirements with a diet inclusive of nutrient modifications specific for the disease. Although not all patients will demonstrate an immune response to diet ingredients per se, positive response to a diet change may still be noted since diets likely differ in fat and fiber levels, fiber types, and digestibility of ingredients. It is not always possible to discern which aspect of the successful diet is responsible for the positive effect. Choose hydrolyzed or novel ingredient diets if possible; some patients will do well on known tolerated ingredients even if prior exposure is documented. A thorough diet history is crucial to determining a list of potential novel ingredients for an individual patient (see Appendix II). Sometimes less exotic ingredients will be options. Limit the number of antigens the patient is exposed to (consider flavored medications, treats, access to food for other pets or table scraps). Patients can lose tolerance to ingredients over time, so it is useful to maintain a list of novel options specific to the animal. For very severe disease, malabsorption of fat-soluble vitamins as well as folate and cobalamin may occur.

Feeding strategies for CKD focus on slowing the progression of disease (phosphorus restriction and supplementation with long-chain omega-3 fatty acids) as well as managing clinical signs (avoiding acidification, protein and sodium restriction, supplementation with B vitamins, and modifying levels of calcium, vitamin D, and potassium if necessary). Consider that in many cases, the ingredients of the canned and dry versions of a prescription diet formulated for the management of CKD will be different. Thus, it may be helpful to compare the list of novel or tolerated foods for the patient to both the dry and canned versions. If the appropriate commercially available diet is not tolerated by the patient, then a home-cooked diet formulation is indicated.

■ **Treats** – Treats should be low in protein, phosphorus, and sodium. The treats should not introduce an ingredient that is not present in the base diet. For many patients, treats should be avoided, especially in the initial stages of diet evaluation. If provided, total daily treats should be provided at no more than 10% of the daily caloric intake. Avoid foods known to be toxic to dogs: grapes and raisins, onions and garlic, macadamia nuts, bread dough, and chocolate.

■ **Tips for Increasing Palatability** – Altering the moisture level of the diet by soaking kibble or baking canned diets can increase acceptance in some pets. Heating the diet can also be useful. The owner should provide a calm

and safe environment for eating and can also try positive reinforcement. If necessary, the daily treat allowance (up to 10% of the daily calories) can be used to add appropriate food items to the meals.

■ **Diet Recommendations** – For critically ill animals, initially feed resting energy requirements (RER; $70 \times BW$ in $kg^{0.75}$), monitor body weight, and adjust as necessary. For more chronic and stable cases, provide true maintenance energy requirements if possible (MER; the amount of calories that has maintained stable body weight). If the diet history is not complete enough to determine this, then estimate MER by calculation. MER can be determined by calculating RER for the current weight and multiplying by the appropriate factor: 1.4 for dogs prone to obesity, 1.6 for neutered dogs, and 1.8 for intact dogs (see Appendix III). Choose a diet that has been formulated for the management of CKD and that supplies ingredients known to be tolerated or that are novel to the individual patient.

Client Education Points

- Monitoring and reassessment will be necessary on a regular basis.
- Diet compliance is important for patients with both diseases.
- Avoid treats high in protein, sodium, or phosphorus. Do not feed treats that contain ingredients not present in the main diet. Consider feeding part of the daily diet as a treat, or use the alternate form of the diet if appropriate (canned or dry).
- A feeding tube may be needed if the patient will not eat adequate amounts of an appropriate diet.
- Chronic kidney disease in dogs progresses at variable rates in different patients, and comorbidities may negatively impact prognosis.

Common Comorbidities

Pancreatitis, GI upset from uremia or hypergastrinemia, allergic dermatitis, hypertension, and protein-losing enteropathy (secondary lymph-

angiectasia) may occur in dogs with allergic gastroenteritis/IBD and CKD.

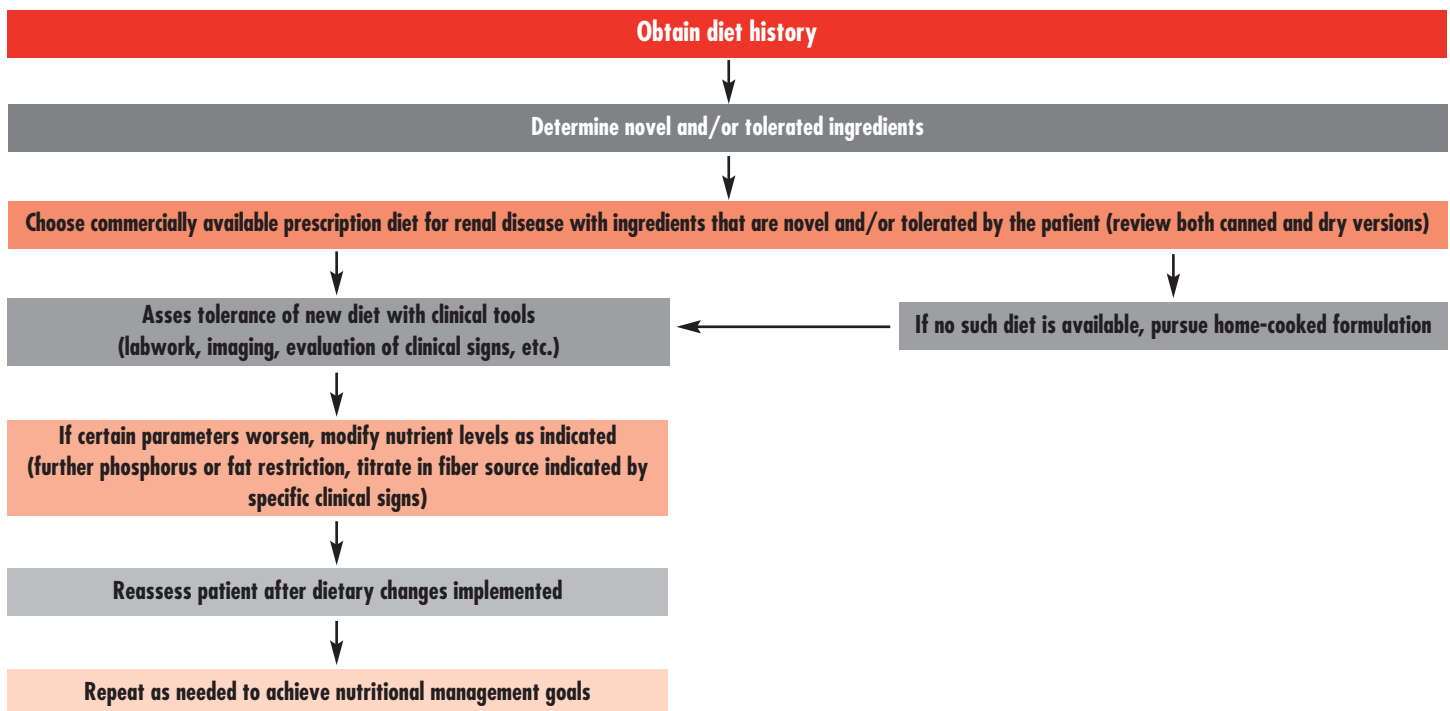
Interacting Medical Management Strategies

Use of angiotensin-converting enzyme (ACE) inhibitors in CKD may contribute to hyperkalemia. Phosphate binders may decrease palatability and cause constipation, anorexia, nausea, or vomiting. Sodium content of any parenteral fluids should be considered in hypertensive or otherwise sodium-sensitive patients. Flavored and/or compounded medications can be a source of undesirable antigens (also consider the source of gelatin capsules).

Monitoring

Clinical signs of GI disease appear to be correlated with severity of inflammation within the GI tract. Monitoring serum BUN/creatinine, phosphorus, calcium, and potassium levels, urine specific gravity, and blood pressure is useful for managing CKD. Regular assessment of the patient for urinary tract infection is indicated. Add serum albumin concentration for patients with concurrent lymphangiectasia and/or glomerular disease and urine protein:creatinine ratio (UPC) for patients with glomerular disease. If diet is not effective in lowering serum phosphorus, add a binder. If mineral/electrolyte values are persistently deranged, a diet change may be indicated. If GI signs do not resolve with the current diet, and diet compliance is confirmed, consider altering ingredients, fiber levels and types, or concurrent/secondary disease. Consider the use of probiotics (however, consider any flavorings or other associated antigens in product). If azotemia or UPC is worsening, and diet compliance is confirmed, further protein restriction is indicated. Consider concurrent disease if UPC does not improve (e.g., Lyme nephritis).

Algorithm – Nutritional Management of Concurrent Canine Allergic Gastroenteritis/IBD and Chronic Kidney Disease



Allergic Gastroenteritis/Inflammatory Bowel Disease & Chronic Kidney Disease – Feline

Jennifer Larsen, DVM, PhD, DACVN

Definition

Cats with both *allergic gastroenteritis/inflammatory bowel disease* (IBD) and *chronic kidney disease* (CKD) suffer from a diet-responsive inflammatory process affecting immune, digestive, and absorptive functions of the gastrointestinal (GI) tract as well as glomerular and/or renal tubular disease resulting in azotemia, reduced urine-concentrating ability, and polyuria/polydipsia (PU/PD), with or without associated clinicopathologic abnormalities (anemia, proteinuria, hypoproteinemia, hypercalcemia, hyperphosphatemia, hypo- or hyperkalemia). For more on allergic gastroenteritis/IBD in cats, see pages 64–65; for more on CKD, see pages 86–87.

Key Diagnostic Tools and Measures

Consider history, physical examination, clinical signs, and minimum database: serum biochemical analysis, complete blood count (CBC), urinalysis, and diet history. Serum tests for the diagnosis of food allergy are not reliable and should not be used. Response to a dietary change is likewise not diagnostic, as improvement or resolution of clinical signs of many GI diseases can occur due to a response to changes in fat or fiber levels, fiber types, digestibility, or secondary effects on intestinal microflora. Abdominal ultrasound, serum folate and cobalamin concentration, and ideally dietary elimination-rechallenge trials and biopsies can provide valuable additional information, and can confirm a preliminary diagnosis.

Pathophysiology

The inciting cause of allergic gastroenteritis/IBD often is unknown, but it may be a reaction to food components, bacterial antigens, and/or self-antigens. Upper or lower GI signs can be seen (inappetence, vomiting, diarrhea, borborygmus, flatulence). Chronic kidney disease is associated with accumulation of metabolic products of protein catabolism and other compounds usually excreted in urine. BUN is a marker of dozens of other nitrogenous waste compounds that impact appetite, smell, and taste. Hypergastrinemia from reduced renal clearance leads to GI mucosal irritation and ulceration as well as acidosis and abnormalities in mineral metabolism (impaired urinary phosphorus clearance, secondary hyperparathyroidism, altered vitamin D metabolism, hypercalcemia).

Signalment

Breeds predisposed to these conditions include Siamese cats (for lymphoplasmacytic enteritis).

Key Nutrient Modifications

Feeding a highly digestible, novel antigen, and/or a low fat diet appear to be valuable strategies (novel ingredients determined by thorough diet history of individual patient). Altering dietary fiber type and level can also have a beneficial effect. Pre- and probiotic therapy is useful for many cases. Protein restriction is needed to manage azotemia. Phosphorus restriction is indicated to reduce the risk of soft tissue mineralization and slow progression of CKD. Supplementation of B vitamins replenishes losses secondary to polyuria. Sodium restriction helps manage hypertension and/or fluid retention. Supplementation of omega-3 fatty acids has an anti-inflammatory effect and may be renoprotective in cats as in dogs. If necessary, a canned diet can help attenuate dehydration.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Protein	28–32	6–8	26	6.5
Phosphorus	0.35–0.6	0.07–0.13	0.5	0.13
Fat	12–30	3–6	9	2.3
Sodium	0.15–0.3	0.03–0.07	0.2	0.05

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

As for all patients, the primary goal of nutritional management of the cat with both allergic gastroenteritis/IBD and CKD is to meet energy requirements with a diet inclusive of nutrient modifications specific for the disease. Although not all patients will demonstrate an immune response to diet ingredients per se, positive response to a diet change may still be noted since diets likely differ in fat and fiber levels, fiber types, and digestibility of ingredients. It is not always possible to discern which aspect of the successful diet is responsible for the positive effect. Choose hydrolyzed or novel ingredient diets if possible; some patients will do well on known tolerated ingredients even if prior exposure is documented. A thorough diet history is crucial to determining a list of potential novel ingredients for an individual patient. Sometimes less exotic ingredients will be options. Limit the number of antigens the patient is exposed to (consider flavored medications, treats, access to food for other pets or table scraps). Patients can lose tolerance to ingredients over time, so it is useful to maintain a list of novel options specific to the animal. For very severe disease, malabsorption of fat-soluble vitamins as well as folate and cobalamin may occur.

Feeding strategies for CKD focus on slowing the progression of disease (phosphorus restriction and supplementation with long-chain omega-3 fatty acids) as well as managing clinical signs (avoiding acidification, protein and sodium restriction, supplementation with B vitamins, and modifying levels of calcium, vitamin D, and potassium if necessary). Consider that in many cases, the ingredients of the canned and dry versions of a prescription diet formulated for the management of CKD will be different. Thus, it may be helpful to compare the list of novel or tolerated foods for the patient to both the dry and canned versions. If the appropriate commercially available diet is not tolerated by the patient, then a home-cooked diet formulation is indicated.

■ **Treats** – Treats should be low in protein, phosphorus, and sodium. The treats should not introduce an ingredient that is not present in the base diet. For many patients, treats should be avoided, especially in the initial stages of diet evaluation. If provided, total daily treats should be provided at no more than 10% of the daily caloric intake. Avoid foods known to be toxic to cats, especially those containing onions and garlic.

■ **Tips for Increasing Palatability** – Altering the moisture level of the diet by soaking kibble or baking canned diets can increase acceptance in some pets. Heating the diet can also be useful. The owner should provide a calm and safe environment for eating and can also try positive reinforcement. Cats generally prefer to eat multiple small meals per day. If necessary, the

daily treat allowance (up to 10% of the daily calories) can be used to add appropriate food items to the meals.

■ **Diet Recommendations** – For critically ill animals, initially feed resting energy requirements (RER; $70 \times BW$ in $kg^{0.75}$), monitor body weight, and adjust as necessary. For more chronic and stable cases, provide true maintenance energy requirements if possible (MER; the amount of calories that has maintained stable body weight). If the diet history is not complete enough to determine this, then estimate MER by calculation. MER can be determined by calculating RER for the current weight and multiplying by the appropriate factor: 1.0 for cats prone to obesity, 1.2 for neutered cats, and 1.4 for intact cats (see Appendix III). Choose a diet that has been formulated for the management of CKD and that supplies ingredients known to be tolerated or that are novel to the individual patient.

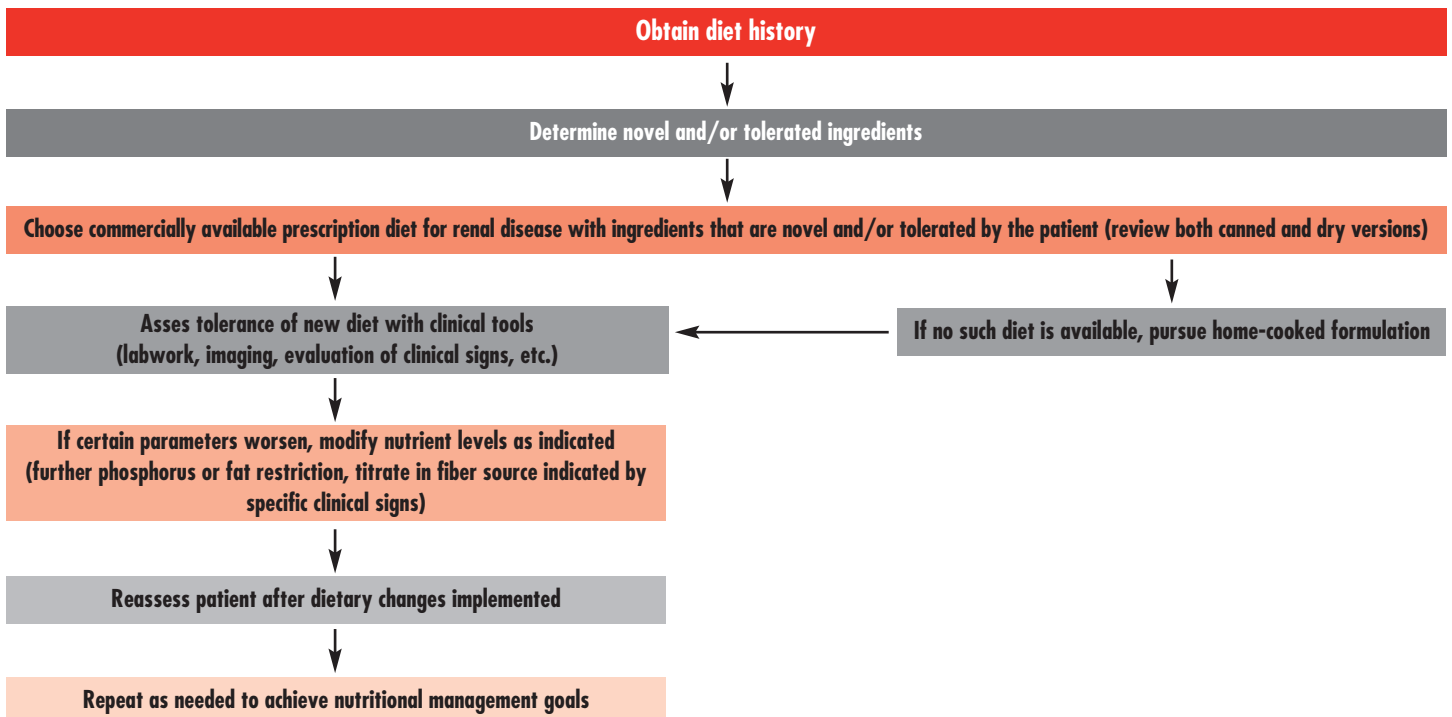
Client Education Points

- Monitoring and reassessment will be necessary on a regular basis.
- Diet compliance is important for patients with both diseases.
- Avoid treats high in protein, sodium, or phosphorus. Do not feed treats that contain ingredients not present in the main diet. Consider feeding part of the daily diet as a treat, or use the alternate form of the diet if appropriate (canned or dry).
- Do not allow prolonged periods of anorexia or suboptimal food intake due to the risk of hepatic lipidosis.
- A feeding tube may be needed if the patient will not eat adequate amounts of an appropriate diet.

Common Comorbidities

Pancreatitis, hepatic disease, GI upset from uremia or hypergastrinemia, allergic dermatitis, hypertension, and urolithiasis may occur in cats with allergic gastroenteritis/IBD and renal disease.

Algorithm – Nutritional Management of Concurrent Feline Allergic Gastroenteritis/IBD and Chronic Kidney Disease



Interacting Medical Management Strategies

Use of angiotensin-converting enzyme (ACE) inhibitors in renal disease may contribute to hyperkalemia. Phosphate binders may decrease palatability and cause constipation, anorexia, nausea, or vomiting. Sodium content of any parenteral fluids should be considered in hypertensive or otherwise sodium-sensitive patients. Flavored and/or compounded medications can be a source of undesirable antigens (also consider the source of gelatin capsules).

Monitoring

Clinical signs of GI disease appear to be correlated with severity of inflammation within the GI tract. Monitoring serum BUN/creatinine, phosphorus, calcium, and potassium levels, urine specific gravity, and blood pressure is useful for managing renal disease. Regular assessment of the patient for urinary tract infections is indicated. Add serum albumin concentration (for patients with concurrent lymphangiectasia and/or glomerular disease) and urine protein:creatinine ratio (UPC) for patients with glomerular disease. If diet is not effective in lowering serum phosphorus, add a binder. If mineral/electrolyte values are persistently deranged, a diet change may be indicated. If GI signs do not resolve with the current diet, and diet compliance is confirmed, consider altering ingredients, fiber levels and types, or concurrent/secondary disease. Consider the use of probiotics (consider any flavorings or other associated antigens in product). If azotemia or UPC is worsening, and diet compliance is confirmed, further protein restriction is indicated. Consider concurrent disease if UPC does not improve (e.g., infectious disease).

Calcium Oxalate Urolithiasis and Hyperlipidemia – Canine

Joseph W. Bartges, DVM, PhD, DACVIM, DACVN

Definition

Hyperlipidemia is defined as an increase in serum concentrations of triglyceride (TG), cholesterol, or both. In *calcium oxalate urolithiasis*, uroliths composed of calcium oxalate monohydrate, dihydrate, or both form in the dog's urine. Calcium oxalate uroliths are often found in dogs with concurrent hyperlipidemia, typically *hypertriglyceridemia*. For more on hyperlipidemia in dogs, see pages 80–81; for more on calcium oxalate urolithiasis, see pages 88–89.

Key Diagnostic Tools and Measures

Serum or plasma calcium concentration is checked to evaluate for hypercalcemia. Serum or plasma triglyceride and cholesterol concentrations may require a minimal 12-hour fast prior to collection. In the refrigeration test, collect a serum sample after a 12-hour fast and refrigerate the sample for 12 hours. If a creamy layer collects on top of clear serum, then hyperchylomicronemia is present. If the sample is turbid, then other lipoproteins are present. Endocrine testing for hyperlipidemia includes thyroid testing for hypothyroidism, adrenal gland testing for hyperadrenocorticism, and blood and urine glucose and ketones for diabetes mellitus. Chronic pancreatitis may result in hyperlipidemia; therefore, testing includes determination of serum or plasma amylase and lipase activities, serum canine pancreatic lipase immunoreactivity, and abdominal ultrasonography. A serum lipoprotein profile can be performed to further differentiate the concentration of lipoproteins present.

Pathophysiology

Hyperlipidemia does not cause calcium oxalate urolith formation; however, certain associations occur. Miniature schnauzers have a breed predisposition to hyperlipidemia and calcium oxalate urolithiasis. The etiology is unknown; however, lipoprotein lipase deficiency or decreased activity is suspected.

Hyperadrenocorticism can cause both conditions to occur. Hyperlipidemia occurs because of insulin resistance induced by hypercortisolemia resulting in alteration in lipid metabolism. Hypercortisolemia promotes hypercalciuria, which results in urinary oversaturation for calcium oxalate and possibly urolith formation.

Signalment

Miniature schnauzers have a breed predisposition to primary hyperlipidemia and calcium oxalate urolithiasis. Breeds at risk for hyperadrenocorticism include, but are not limited to miniature schnauzers, miniature and toy poodles, dachshunds, and Boston terriers.

Key Nutrient Modifications

A low-fat diet may help decrease the degree of hyperlipidemia. A high-fiber diet may decrease absorption of fat. Fiber may also reduce the absorption of calcium from intestinal tract, but currently there is no evidence that this beneficially influences urinary calcium. Low-calcium diets are a risk factor for calcium oxalate urolithiasis so should be avoided. Increased sodium intake may decrease urinary saturation for calcium oxalate. Increased urine volume may reduce the risk for urolithiasis so water intake should be encouraged. Increased water intake may be promoted by feeding wet food, or adding water to a canned or dry food.

Recommended Ranges of Key Nutrients

Nutrient	% DM		g/100 kcal	
	Recommended dietary level	Minimum dietary requirement*		
Fat	7–12	1.5–3.5	5	1.4
Fiber	7–14	2–8	n/a	n/a
Calcium	0.6–1.5	0.2–0.35	0.6	0.17
Sodium	0.2–1.5	0.06–0.35	0.06	0.02

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake. Increased water intake should be encouraged. Use of canned, high moisture diets or sodium-supplemented diets may help increase water intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

With primary hyperlipidemia, dietary fat restriction is recommended with or without higher fiber. Increased fiber is sometimes recommended for the management of calcium oxalate uroliths although no studies have been published to support this. If hyperlipidemia is secondary to endocrine disease, management of disease is the main treatment. Dietary management to minimize risk factors that promote recurrent calcium oxalate disease also is indicated.

■ **Treats** – Avoid high-fat treats.

■ **Tips for Increasing Palatability** – Water can be added to food to increase palatability.

■ **Diet Recommendations** – A high-fiber, low-fat diet or a very low-fat diet is recommended for dogs with hyperlipidemia. Dogs with calcium oxalate urolithiasis should be fed a non-acidifying diet that reduces calcium oxalate urinary supersaturation. A high-moisture, low-fat diet would be recommended for the patient with both of these problems.

Client Education Points

- Hyperlipidemia (high levels of circulating fat in blood) can occur as a primary disease, as in miniature schnauzers, or secondary to other diseases, such as hyperadrenocorticism.
- Calcium oxalate uroliths occur when urine contains high levels of calcium and/or oxalate. This can occur in association with diseases that cause hyperlipidemia.
- Clients should avoid giving supplements containing calcium or vitamin C, as well as human-food treats high in oxalate.

Common Comorbidities

Hyperadrenocorticism, hypothyroidism, chronic pancreatitis, seizures with hypertriglyceridemia, and hyperparathyroidism, if hypercalcemic, are common comorbidities in dogs with hyperlipidemia and calcium oxalate urolithiasis.

Interacting Medical Management Strategies

For the hyperlipidemia, the following may be required if dietary management does not appropriately decrease triglyceride concentrations: omega-3 fatty acids (may decrease synthesis of certain lipoproteins; 10–30 mg/kg orally (PO) every 24 hours); gemfibrozil (decreases certain

lipoprotein and triglyceride synthesis; 100–300 mg PO every 12 hours); chitin or chitosan (may bind certain dietary lipids in the intestinal tract thereby decreasing their absorption; no good studies in dogs; 150–300 mg PO 30 minutes prior to feeding); and niacin (reduces triglyceride synthesis; vasodilatory resulting in erythema and pruritus; 25–150 mg PO every 12 hours).

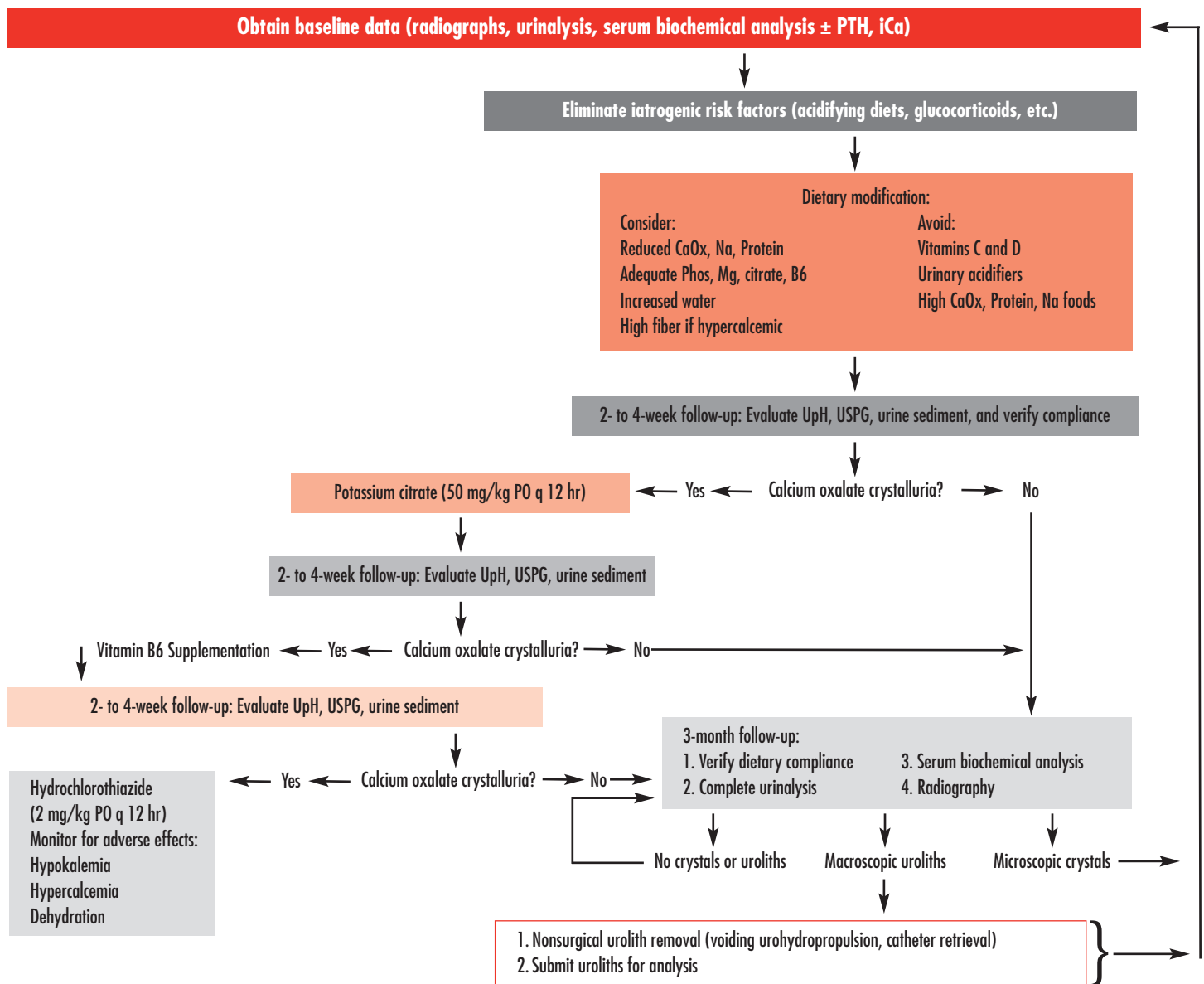
For calcium oxalate urolithiasis, potassium citrate, a urinary alkalinizing agent, can be given to increase calcium oxalate solubility with alkaluria. Citrate may inhibit calcium oxalate crystal formation and aggregation at a dose of 50–100 mg/kg PO every 12 hours; adjust to urine pH of approximately 7.5. Thiazide diuretics increase distal renal tubular reabsorption of calcium resulting in lowered urinary calcium excretion, but can result in hypercalcemia. There are no long-term studies in dogs on safety or efficacy. Hydrochlorothiazide is given at 2 mg/kg PO every 12 hours. Vitamin B6 is involved with oxalate metabolism, although there is no evidence that vitamin B6 supplementation provides any benefit in dogs.

Monitoring

For hyperlipidemia, check for resolution of clinical signs (if present). Fasting triglyceride and cholesterol concentrations should be monitored; if secondary to endocrine disease, treatment for that disease should be monitored.

For calcium oxalate urolithiasis, urinalysis should be done monthly for 3 to 6 months to monitor response to treatment; pH should be neutral to alkaline, specific gravity should be dilute, and crystalluria should be absent. Survey abdominal radiography or ultrasonography should be performed at 6 and 12 months and then every 6 to 12 months depending on response. Serum calcium should be monitored 1 month after starting hydrochlorothiazide and then every 3 to 6 months. If secondary to endocrine disease, appropriate monitoring of management is indicated.

Algorithm – Nutritional Management of Concurrent Canine Calcium Oxalate Uroliths and Hyperlipidemia



Chronic Kidney Disease and Obesity – Canine

Donna M. Raditic, DVM, CVA

Joseph W. Bartges, DVM, PhD, DACVIM, DACVN

Definition

Obesity is defined quantitatively as 15% to 20% above ideal body weight. Functionally, obesity impairs health and is sufficient to cause diseases. Specific distribution of fat in the body is known to be important as seen in “metabolic disease” in humans. **Chronic kidney disease** (CKD) is a more common disease in older dogs and a congenital form is seen occasionally in younger dogs. For more on obesity in dogs, see pages 32–33; for more on CKD in dogs, see pages 84–85.

Key Diagnostic Tools and Measures

Current body weight, body condition scoring (BCS) (see Appendix I), and a complete diet history should be obtained in evaluation of an obese cat with suspected CKD (see Appendix II). Serum or plasma concentrations of creatinine, urea nitrogen, phosphorus, calcium, electrolytes (sodium, potassium, chloride); urine protein-to-urine-creatinine ratio (UP:UC); urinalysis (sample collected by cystocentesis), and urine sediment analysis (urine collected by cystocentesis and analyzed immediately; false results can be obtained with cooled urine) are used in the diagnosis of CKD.

Additional measures include testing for leptospirosis (ideally 2-week then 4-week titer intervals or polymerase chain reaction [PCR]); tick-borne diseases (titers or PCR), including babesiosis (*B. canis* or *B. gibsoni*; PCR testing is most sensitive) and *Borrelia burgdorferi*. *B. burgdorferi* infection has been associated with azotemia, but the pathophysiology has not been established. It is known that seropositive dogs can demonstrate no illness from the exposure; therefore, testing must be interpreted judiciously. The 4Dx test (IDEXX) tests for the C6 antibody and is both sensitive and specific for *Borrelia* exposure.

Ethylene glycol test must be done within 48 hours of possible ingestion. For plasma zinc testing, use royal blue top tubes. Other tests include indirect arterial blood pressure for hypertension, abdominal radiography for potential uroliths or neoplasia, ACTH stimulation testing for Cushing’s disease, serum total T4 and TSH or full thyroid panel for hypothyroidism, and blood and urine glucose for primary or concurrent diabetes mellitus. Advanced testing could include ultrasonography, dual energy x-ray absorptiometry (DEXA) analysis for lean body mass to fat mass ratio, and blood gas analysis.

Pathophysiology

Obesity occurs when caloric intake exceeds the dog’s energy requirements, such as basal metabolic rate, exercise and other energy expenditures. Obesity is a disease with increases in inflammatory mediators, insulin resistance and abnormal blood lipids. Diseases such as diabetes mellitus, cardiovascular changes, pancreatitis, lipidosis, osteoarthritis, cancer, constipation, and lower urinary tract disease have been associated with obesity. Obesity does not cause renal failure, but these conditions are associated. The multiple effects of obesity suggest a link between obesity and renal failure although the exact pathophysiology is not known. Epidemiologically both obesity and renal failure are increasing at similar rates in dogs. Early glomerular changes have been documented in obese human beings. Low birth weight babies have a higher risk of reduced nephron numbers, obesity, and hypertension.

Signalment

Obesity occurs most often in dogs between 5 and 10 years of age. These obese dogs are at a greater risk of early morbidity and mortality. Renal failure in young dogs can result from dietary indiscretion (zinc, lilies, raisins, grapes, antifreeze, and other medications). Glomerular disease is the leading type of renal failure in dogs; it is most frequently seen in male dogs with a mean age of 8 years of age or older. Dogs with diabetes mellitus are often obese and have an increased risk of developing renal failure. The Shar-Pei breed is predisposed to renal failure involving amyloid deposition in the glomerular and medulla of the kidneys. Fanconi syndrome in Basenjis often progresses to renal failure. Congenital renal failure has been described in many breeds and usually manifests in dogs less than 3 years of age.

Key Nutrient Modifications

Low-phosphorus diets have been shown to slow progression of renal failure. Moderate levels of high-quality protein are beneficial in slowing progression of renal failure in cats. High dietary intake of omega-3 fatty acids has anti-inflammatory effects in primary renal failure. Diets with moderate restriction of sodium may decrease systemic arterial hypertension and degree of azotemia.

Antioxidants play a role in obesity-related inflammatory mediators and increased oxidative stress on normal cell function; therefore, diets fortified with balanced antioxidants will be beneficial. Low-calorie diets must provide all essential nutrients balanced to the calorie intake. Low-fat diets decrease dietary calorie content because fat provides two times more calories per gram than protein or carbohydrates. High-fiber diets are used to decrease caloric intake and increase satiation for weight loss and improve gastrointestinal health in renal patients. Canned diets with more water may increase satiety and improve fluid balance in renal patients.

Recommended Ranges of Key Nutrients

Nutrient	% DM		g/100 kcal	
	Recommended dietary level	Minimum dietary requirement*		
Phosphorus	0.2–0.4	0.04–0.1	0.5	0.14
Sodium	0.1–0.35	0.02–0.12	0.06	0.02
Protein	13–20	3.5–5.5	18	5.1
Fat	6–11	1.5–3.0	5	1.4
Fiber	9–22	2–7	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

- Renal failure is the primary management concern. It requires strict phosphorus restriction and moderate high quality protein intake that has been shown to slow progression of this disease.
- Renal diets tend to be higher in fat and lower in fiber content to ensure adequate caloric intake; dogs with renal failure often have poor appetites and concurrent gastrointestinal disease.

- The high fat levels in renal diets can improve palatability, but can cause gastrointestinal issues such as pancreatitis.
 - Obese dogs require low calorie, low fat, and moderate to high fiber diets, but the palatability of these diets can be poor in dogs with renal failure.
 - The dietary goal of managing an obese dog with renal failure is to ensure adequate caloric intake for weight maintenance and safe weight loss; this weight loss diet must be low in phosphorus with moderate high quality protein levels to slow renal failure progression.
- **Treats** – Avoid table scraps and human foods. Avoid treats that are high in protein, salt or phosphorus (% ash), calories, and fat.
- **Tips for Increasing Palatability** – Some dogs prefer canned foods; mix appropriate canned and dry foods. Warm canned foods or offer fresh. Add low-salt, low-fat broth or low-salt tuna water to diet. Offer small meals frequently.
- **Diet Recommendations** – Diets that meet the following criteria are recommended: Low phosphorus, moderate high-quality protein, moderate sodium, low fat, moderate to high fiber, and high omega-3 content.

Client Education Points

- Management of CKD may supersede a weight loss program for obese dogs in renal failure.
- Close monitoring of weight is paramount in obese dogs with renal failure. Monitoring includes body condition scoring and body weight.
- Dogs in renal failure can easily lose weight in the form of lean body mass rather than fat, which is a poor prognosis for survival.
- “Eating some” is not enough; the dog must consume enough for maintaining weight or achieving safe weight loss if this is a goal.
- Monitor renal values closely if weight loss occurs.
- Weight loss can signify dehydration and/or the progression of renal failure.
- Malnutrition is a major cause of morbidity and mortality in dogs with renal failure.

Common Comorbidities

Bacterial urinary tract infection (cystitis or pyelonephritis), nephrolithiasis (usually calcium oxalate), diabetes mellitus, hypothyroidism, hyperparathyroidism, pancreatitis, inflammatory bowel disease, colitis, and osteoarthritis are seen in obese dogs with renal failure.

Interacting Medical Management Strategies

Angiotensin-converting enzyme (ACE) inhibitors are used to decrease proteinuria, but may induce hyperkalemia. **Amlodipine** is used to decrease systemic arterial hypertension, but may induce hypotension. **Phosphate binders** are used to decrease hyperphosphatemia, but may cause constipation, hypercalcemia, or hypophosphatemia depending on the type used. **Calcitriol** is used to treat renal secondary hyperparathyroidism, but may induce hypercalcemia. **Antibiotics** used to treat bacterial infections may be nephrotoxic (e.g., aminoglycosides). **Nonsteroidal anti-inflammatory drugs (NSAIDs)** used to decrease inflammation may be nephrotoxic. **H2 blockers** (e.g., metoclopramide or ranitidine) are used to decrease nausea, vomiting, and gastrointestinal ulceration and bleeding but may cause sedation or hyperactivity in rare cases. **Recombinant human erythropoietin (EPO)** is used to correct anemia, but can cause anti-EPO antibodies leading to a nonregenerative anemia. **Potassium salts** (e.g.,

gluconate or citrate) is used to treat hypokalemia or metabolic acidosis, but may induce hyperkalemia especially when used with an ACE inhibitor. **Omega-3 fatty acids** used to decrease inflammatory response can increase caloric intake. **Sodium bicarbonate** is used to treat metabolic acidosis, but is unpalatable for cats and may increase blood pressure due to increased sodium intake.

Monitoring

Adequate **hydration** is needed to maintain renal perfusion. Many dogs with CRD require supplemental fluid administration, including feeding canned diets that contain more than 75% moisture; oral administration of water or flavored fluids; subcutaneous administration of lactated Ringer’s solution or other balanced crystalloid solution; or enteral administration of water by feeding tube.

Maintain a stable **creatinine** concentration. Clinical signs do not often correlate with the degree of azotemia in dogs with CRD because they have adapted to it. Creatinine concentrations that increase by more than 0.2 mg/dL between measurements in dogs with CRD that are adequately hydrated indicates progression.

A **urine protein:creatinine ratio (UP:UC)** maintained below 0.5 is ideal. UP:UC ratios should be followed serially; interpretation is dependent on absence of hematuria, pyuria, or infection. ACE inhibitors should be administered to dogs with chronic renal failure with UP:UC ratios greater than 0.5.

Phosphorus levels should be maintained below 5.5 mg/dL. Phosphate binding agents can be used to initiate treatment if levels continue to increase.

Blood pressure (systolic) should be less than 160 mmHg, and should be measured serially over weeks or months. Initiate treatment if blood pressure continues to increase; slowly lower sodium intake further if levels are moderate. In dogs on ACE inhibitors and/or amlodipine therapy, increases in creatinine > 0.5 mg/dL suggest an adverse drug reaction. ACE inhibitors or amlodipine should never be given to a dehydrated patient.

Hematocrit should be maintained between 38% and 48%. The use of recombinant human EPO or darbepoetin may be appropriate. Other factors, such as gastrointestinal ulcerations, iron deficiency, poor nutrition, hyperparathyroidism, and infections, should be considered in anemic dogs.

Serum potassium levels should be between 3.5 and 5.5 mEq/L. **Hypokalemia** can be treated with oral potassium gluconate or citrate. For hyperkalemia, decrease dietary intake of potassium and consider reduction in dosage of ACE inhibitors or potassium supplement. If **metabolic acidosis** is not controlled by diet, initiate treatment with oral sodium bicarbonate or potassium citrate.

Malnutrition is a concern; an obese dog with CKD that experiences rapid weight loss (more than 1% to 2% body weight per week) is losing lean muscle mass (which can increase azotemia) in addition to fat or is severely dehydrated. Enteral feeding tubes should be considered to supply enough calories to prevent rapid weight loss. The enteral diet should be formulated with adequate calories, but low in phosphorus, sodium, and moderate high-quality protein or as necessitated by the patient and renal staging. Aggressive rehydration via intravenous fluid administration is needed.

See **Algorithm: Nutritional Management of Concurrent Canine Obesity and Chronic Kidney Disease** on page 114.

Chronic Kidney Disease and Obesity – Feline

Donna M. Raditic, DVM, CVA

Joseph W. Bartges, DVM, PhD, DACVIM, DACVN

Definition

Obesity is defined quantitatively as 15% to 20% above ideal body weight. Functionally, obesity impairs health and is sufficient to cause diseases. Specific distribution of fat in the body is known to be important as seen in “metabolic disease” in humans. **Chronic kidney disease** (CKD) is a more common disease in older cats and seen occasionally in younger cats. It is often irreversible and slowly progressive. For more on obesity in cats, see pages 36–37; for more on kidney failure in cats, see pages 86–87.

Key Diagnostic Tools and Measures

Current body weight, body condition scoring (BCS) (see Appendix I), and a complete diet history should be obtained in evaluation of an obese cat with suspected CKD (see Appendix II). Serum or plasma concentrations of creatinine, urea nitrogen, phosphorus, calcium, electrolytes (sodium, potassium, chloride); urine protein-to-urine-creatinine ratio (UP:UC); urinalysis (sample collected by cystocentesis), and urine sediment analysis (urine collected by cystocentesis and analyzed immediately; false results can be obtained with cooled urine) are used in the diagnosis of CKD. Additional measures include indirect arterial blood pressure for hypertension, abdominal radiography for potential uroliths or neoplasia; blood and urine glucose for primary or concurrent diabetes mellitus; and serum total T4 or free T4 for hyperthyroidism. Advanced testing could include ultrasonography, dual energy x-ray absorptiometry (DEXA) analysis for lean body mass to fat mass ratio, and blood gas analysis.

Pathophysiology

Obesity occurs when caloric intake exceeds the cat’s energy requirements, such as basal metabolic rate, exercise and other energy expenditures. Obesity is a disease with increases in inflammatory mediators, insulin resistance and abnormal blood lipids. Diseases such as diabetes mellitus, cardiovascular changes, pancreatitis, lipidosis, osteoarthritis, cancer, constipation, and lower urinary tract disease have been associated with obesity. Obesity does not cause kidney failure, but these conditions are associated. The multiple effects of obesity suggest a link between obesity and kidney failure although the exact pathophysiology is not known. Epidemiologically both obesity and kidney failure are increasing at similar rates in cats. Early glomerular changes have been documented in obese human beings. Low birth weight babies have a higher risk of reduced nephron numbers, obesity, and hypertension.

Signalment

Obesity occurs most often in cats between 5 and 10 years of age. Kidney failure typically occurs in cats over 10 years of age. Obese cats are at a greater risk of early morbidity and mortality. Cats with diabetes mellitus are often obese and have an increased risk of developing kidney failure. Neutered male cats greater than 6 years of age have a higher incidence of diabetes mellitus and the same population of cats has a higher risk for obesity. Persian cats have a higher than normal incidence of polycystic CKD.

Key Nutrient Modifications

Low-phosphorus diets have been shown to slow progression of kidney failure. Moderate levels of high-quality protein are beneficial in slowing progression of kidney failure in cats. High dietary intake of omega-3 fatty acids has anti-inflammatory effects in primary kidney failure. Diets with moderate restriction of sodium may decrease systemic arterial hypertension and degree of azotemia.

Antioxidants play a role in obesity-related inflammatory mediators and increased oxidative stress on normal cell function; therefore, diets fortified with balanced antioxidants will be beneficial. Low-calorie diets must provide all essential nutrients balanced to the calorie intake. Low-fat diets decrease dietary calorie content because fat provides two times more calories per gram than protein or carbohydrates. High-fiber diets are used to decrease caloric intake and increase satiation for weight loss and improve gastrointestinal health in kidney patients. Canned diets with more water may increase satiety and improve fluid balance in kidney patients.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Phosphorus	0.4–0.6	0.08–0.15	0.5	0.13
Sodium	0.1–0.36	0.03–0.08	0.2	0.05
Protein	25–36	6–10	26	6.5
Fat	7–16	2–5	9	2.3
Fiber	4–13	1.2–4	n/a	n/a

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

Chronic kidney disease is the primary management concern. It requires strict phosphorus restriction and moderate levels of high-quality protein, which has been shown to slow progression of CKD. Kidney diets tend to be higher in fat and lower in fiber content to ensure adequate caloric intake; cats with CKD often have poor appetites and concurrent gastrointestinal disease. Protein catabolism is seen in obese kidney cats; therefore, weight loss from lean body tissue is a concern. Obese cats need low-calorie, low-fat, and moderate- to high-fiber diets. Some obese cats seem to respond to high-protein diets for weight loss, but these diets would be inappropriate for a cat with CKD. The dietary goal of managing obese cats with kidney failure is to ensure decreased caloric intake (using a moderate- to high-fiber, low-fat approach) while maintaining low phosphorus and moderate high-quality protein levels.

■ **Treats** – Avoid treats that are high in protein, salt or phosphorus (% ash), calories, and fat.

■ **Tips for Increasing Palatability** – Some cats prefer dry food, while others prefer canned; feed accordingly. Add low-salt, low-fat broth to the diet. Warm canned foods or offer fresh. Add low-salt tuna water for cats that prefer fish-flavored diets. Offer small meals frequently.

■ **Diet Recommendations** – Diets that meet the following criteria are recommended: Low phosphorus, moderate high-quality protein, moderate sodium, low fat, and moderate fiber.

Client Education Points

- Cats cannot be “starved” into eating a recommended diet.
- Slow diet transition is necessary.
- Management of CKD may supersede a weight loss program for obese cats with CKD.
- Close monitoring of weight is paramount in obese cats with CKD.
- “Eating some” is not enough; the cat must consume enough for maintaining weight or achieving safe weight loss if this can be a goal.
- Monitoring includes body condition scoring and body weight.
- Weight loss can signify dehydration and/or the progression of CKD.
- Cats with CKD can easily lose weight in the form of lean body mass rather than fat, which carries a poor prognosis for survival.
- Malnutrition is a major cause of morbidity and mortality in cats with CKD.

Common Comorbidities

Bacterial urinary tract infection (cystitis or pyelonephritis), nephrolithiasis (usually calcium oxalate), diabetes mellitus, hyperthyroidism, pancreatitis, hepatic lipidosis, idiopathic hypercalcemia, inflammatory bowel disease, chronic constipation, and osteoarthritis are seen in obese cats with CKD.

Interacting Medical Management Strategies

Angiotensin-converting enzyme (ACE) inhibitors are used to decrease proteinuria, but may induce hyperkalemia. **Amlodipine** is used to decrease systemic arterial hypertension, but may induce hypotension. **Phosphate binders** are used to decrease hyperphosphatemia, but may cause constipation, hypercalcemia, or hypophosphatemia depending on the type used. **Calcitriol** is used to treat kidney secondary hyperparathyroidism, but may induce hypercalcemia. **Antibiotics** used to treat bacterial infections may be nephrotoxic (e.g., aminoglycosides). **Nonsteroidal anti-inflammatory drugs (NSAIDs)** used to decrease inflammation may be nephrotoxic. **H2 blockers** (e.g., metoclopramide or ranitidine) are used to decrease nausea, vomiting, and gastrointestinal ulceration and bleeding but may cause hyperactivity or disorientation in rare cases. **Recombinant human erythropoietin (EPO)** is used to correct anemia, but can cause anti-EPO antibodies leading to a nonregenerative anemia. **Potassium salts** (e.g., gluconate or citrate) are used to treat hypokalemia or metabolic acidosis, but may induce hyperkalemia especially when used with an ACE inhibitor. **Omega-3 fatty acids** used to decrease inflammatory response can increase caloric intake. **Sodium bicarbonate** is used to treat metabolic acidosis, but is unpalatable for cats and may increase blood pressure due to increased sodium intake.

Monitoring

Adequate hydration is needed to maintain kidney perfusion. Many cats with CKD require supplemental fluid administration, including feeding

canned diets that contain more than 75% moisture; oral administration of water or flavored fluids; subcutaneous administration of lactated Ringer’s solution or other balanced crystalloid solution; or enteral administration of water by feeding tube.

Maintain a stable **creatinine** concentration. Clinical signs do not often correlate with the degree of azotemia in cats with CKD because they have adapted to it. Creatinine concentrations that increase by more than 0.2 mg/dL between measurements in cats with CKD that are adequately hydrated indicates progression.

A **urine protein:creatinine ratio (UP:UC)** maintained below 0.4 is ideal. UP:UC ratios should be followed serially; interpretation is dependent on absence of hematuria, pyuria, or infection. ACE inhibitors should be administered to cats with chronic kidney failure with UP:UC ratios greater than 0.4.

Phosphorus levels should be maintained <5.5 mg/dL. Phosphate binding agents can be used to initiate treatment if levels continue to increase.

Blood pressure (systolic) should be less than 160 mmHg, and should be measured serially over weeks or months. Initiate treatment if blood pressure continues to increase; slowly lower sodium intake further if levels are moderate. In cats on ACE inhibitors and/or amlodipine therapy, increases in creatinine > 0.5 mg/dL suggest an adverse drug reaction. ACE inhibitors or amlodipine should never be given to a dehydrated patient.

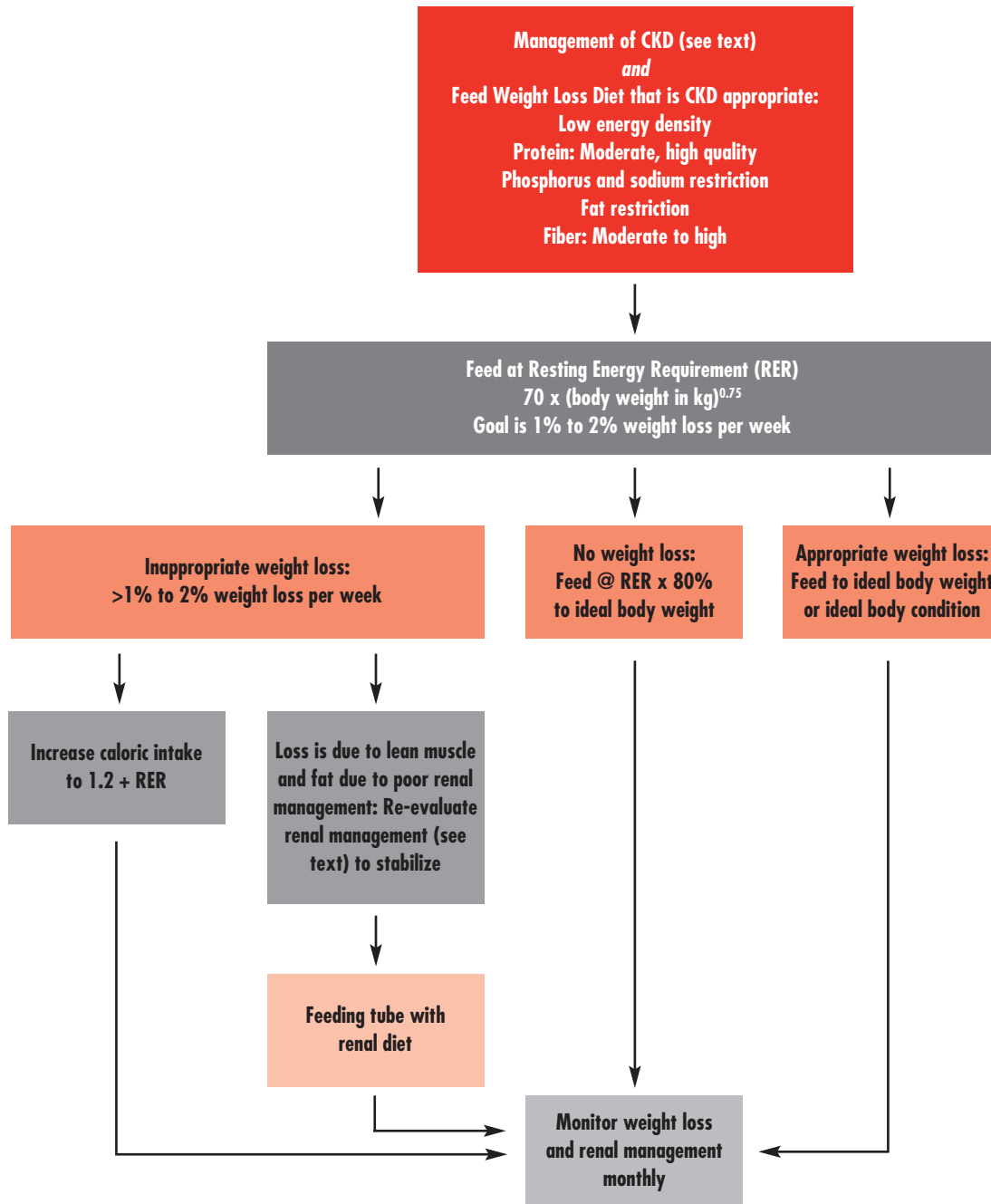
Hematocrit should be maintained between 30% and 40%. The use of recombinant human EPO or darbepoetin may be appropriate. Other factors, such as gastrointestinal ulcerations, iron deficiency, poor nutrition, hyperparathyroidism, and infections, should be considered in anemic cats.

Serum potassium levels should be between 3.5 and 5.5 mEq/L. **Hypokalemia** is more common in cats, and can be treated with oral potassium gluconate or citrate. For **hyperkalemia**, decrease dietary intake of potassium and consider reduction in dosage of ACE inhibitors or potassium supplement. If **metabolic acidosis** is not controlled by diet, initiate treatment with oral sodium bicarbonate or potassium citrate.

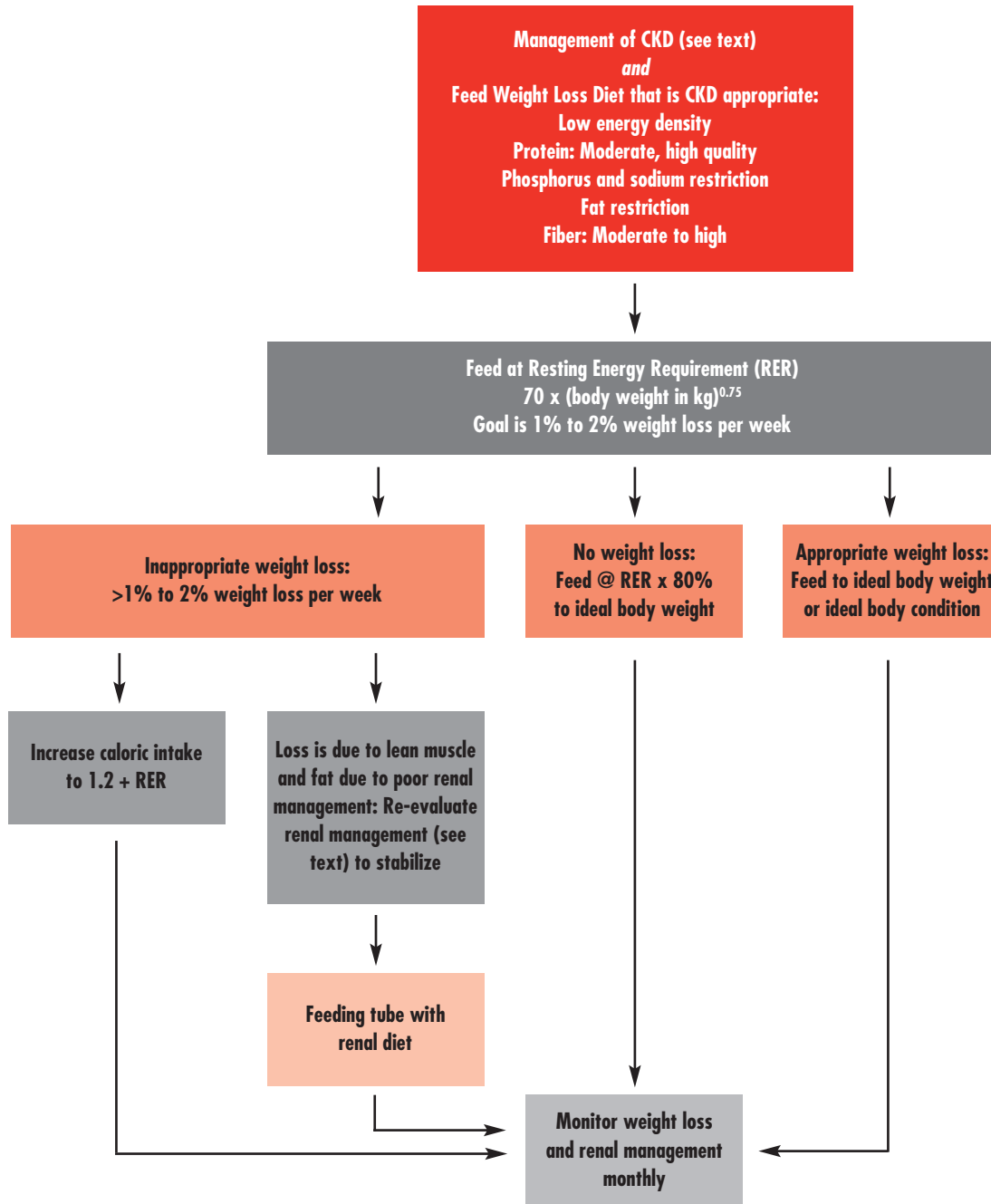
Malnutrition is a concern; an obese cat with CKD that experiences rapid weight loss (more than 1% to 2% body weight per week) is losing lean muscle mass (which can increase azotemia) in addition to fat or is severely dehydrated. Enteral feeding tubes should be considered to supply enough calories to prevent rapid weight loss. The enteral diet should be formulated with adequate calories, but low in phosphorus, sodium, and moderate high-quality protein or as necessitated by the patient and kidney staging. Aggressive rehydration via intravenous fluid administration is needed.

See **Algorithm: Nutritional Management of Concurrent Feline Obesity and Chronic Kidney Disease** on page 115.

Algorithm – Nutritional Management of Concurrent Canine Obesity and Chronic Kidney Disease (CKD)



Algorithm – Nutritional Management of Concurrent Feline Obesity and Chronic Kidney Disease (CKD)



Struvite Urolithiasis and Obesity – Feline

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Definition

Obesity is defined quantitatively as 15% to 20% above ideal body weight. Functionally, obesity impairs health and is sufficient to cause diseases. Specific distribution of fat in the body is known to be important as seen in “metabolic disease” in humans. **Struvite urolithiasis** is a lower urinary tract disease often resulting in one or more of the signs of hematuria, dysuria, pollakiuria, urethral obstruction, and inappropriate urination. For more on obesity in cats, see pages 36–37; for more on struvite urolithiasis in cats, see pages 90–91.

Key Diagnostic Tools and Measures

Current body weight, body condition scoring (BCS) (see Appendix I), and a complete diet history should be obtained in evaluation of an obese cat with suspected struvite urolithiasis (see Appendix II). Urinalysis (sample collected by cystocentesis), analysis of urolith or plug if retrieved, and urine sediment analysis (this test should be done immediately after collection to evaluate crystalluria; false results are often obtained with cooled urine) should be evaluated. On urine culture, less than 1% of cats less than 10 years of age have bacterial cystitis whereas 45% of cats over 10 years of age with lower urinary tract signs have bacterial cystitis. Bacterial infections are often secondary to renal failure, diabetes, mellitus, feline leukemia virus, etc. Abdominal radiography for potential uroliths (struvite uroliths are radiodense) and blood testing to rule out other diseases related to obesity or secondary to bacterial cystitis are recommended. Advanced testing could include cystoscopy (limited to urethral diameter), ultrasonography, and dual energy x-ray absorptiometry (DEXA) analysis for lean body mass to fat mass ratio.

Pathophysiology

Obesity occurs when caloric intake exceeds the cat’s energy requirements such as basal metabolic rate, exercise and other energy expenditures. Obesity is a disease with increases in inflammatory mediators, insulin resistance and abnormal blood lipids. Diseases such as diabetes mellitus, cardiovascular changes, pancreatitis, lipidosis, osteoarthritis, lower urinary tract disease, cancer, constipation and lower urinary tract disease have been associated with obesity.

The multiple effects of obesity suggest a link between obesity and lower urinary tract diseases such as struvite urolithiasis, although the exact pathophysiology is unknown. The most common struvites in cats are sterile; however, cats do form struvites uroliths if they develop a urinary tract infection with a urease-producing bacterium, typically a *Staphylococcus* spp. “Struvite” mineral composition is magnesium, ammonium phosphate hexahydrate. Struvite solubility is dependent on concentrations of magnesium, ammonium, phosphate, and urine pH. Struvite solubility decreases as urine pH exceeds 6.8, and sterile struvite uroliths form with concurrent urine oversaturation of minerals and alkaline urine. Struvite is the most common mineral seen in urethral obstruction due to a urolith or urethral plugs. Sterile struvite uroliths form because of dietary composition as well as other unknown risks factors for urolith formation.

Signalment

Obesity occurs most often in cats between 5 and 10 years of age. These cats are at a greater risk of early morbidity and almost 3 times the average rate of mortality. Overall lower urinary tract disease in cats is seen between 4 and 10 years of age. Struvite urolithiasis typically occurs in cats less than 10

years of age with no gender or breed predisposition. Bacterial-induced struvite urolithiasis is seen most often in cats greater than 10 years of age and is often secondary to another disease. Neutered male cats less than 10 years of age have a higher incidence of urethral plugs, which most commonly contain struvite.

Key Nutrient Modifications

In nutritional management of obesity, low-calorie diets must provide all the essential nutrients balanced to the calorie intake. Low-fat diets decrease dietary calorie content because fat provides two times more calories per gram than protein or carbohydrates. High-fiber diets are used to decrease caloric intake and increase satiation for weight loss. Weight loss programs have been achieved with low-carbohydrate, high-protein diets in some cats. Canned diets with more water may increase satiety and increase fluid intake decreasing concentration of minerals in the urine.

Key modifications in management of struvite urolithiasis include dietary phosphorus restriction, dietary magnesium restriction, avoidance of excess protein intake as this decreases urinary ammonia concentration, avoidance of alkalinizing foods (e.g., renal failure diets or plant-based protein diets), and inducing acid urine. Cats produce an alkaline urine after a meal that is more prolonged with meal feeding rather than ad libitum. Thus, cats that consume small quantities of food rather than one or two large meals per day reduce less struvite crystalluria. Ad libitum feeding still requires calorie management in struvite-forming, obese cats. Sodium intake decreases urine saturation of struvites; sodium’s exact role in urine saturation and resulting struvite and calcium oxalate urolithiasis is an area of investigation.

Recommended Ranges of Key Nutrients

Nutrient	% DM	g/100 kcal	% DM	g/100 kcal
	Recommended dietary level		Minimum dietary requirement*	
Fat	7–10	2.3–4	9	2.3
Fiber	4–13	1.2–4	n/a	n/a
Protein	30–44	9–15	26	6.5
Phosphorus	0.6–1.1	0.1–0.3	0.5	0.13
Magnesium	0.06–0.08	0.01–0.02	0.04	0.01
Sodium	0.1–1.1	0.05–0.35	0.2	0.05

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Increased water intake should be encouraged. Use of canned, high moisture diets or sodium-supplemented diets may help increase water intake.

Therapeutic Feeding Principles

Obese cats need low-calorie, low-fat, and moderate- to high-fiber diets. Some obese cats seem to respond to high-protein diets for weight loss, but these diets would be inappropriate for a cat with struvite urolithiasis. The dietary goal of managing obese cats with struvite uroliths is to ensure decreased caloric intake (using a moderate- to high-fiber, low-fat approach) while maintaining low phosphorus and moderate high-quality protein levels. Ad libitum feeding must be calorie controlled. Restrict dietary levels

of magnesium, protein, and phosphorus to produce less urine saturation of these minerals and dissolution of struvite uroliths or crystals. Induce and maintain acid urine for both under saturation and dissolution. Encourage water intake for both urine saturation and the cat's satiation.

■ **Treats** – Avoid treats that are high in calorie, fat, protein and phosphorus and magnesium (% ash). Encourage water intake with canned diets or flavored water. Avoid alkalinizing treats and medications (such as potassium citrate or sodium bicarbonate).

■ **Tips for Increasing Palatability** – Attempt to transition the cat to an appropriate canned diet. Add flavoring agents such as appropriate broth or gravy. Warm canned foods. Offer fresh canned food frequently. Put small bowls of dry food around the house

■ **Diet Recommendations** – Diets that meet the following criteria are recommended: Low phosphorus and magnesium; acidifying; moderate amounts of high-quality protein; and low-fat, high-fiber diets for weight loss. Struvite dissolution and preventative diets are commercially available.

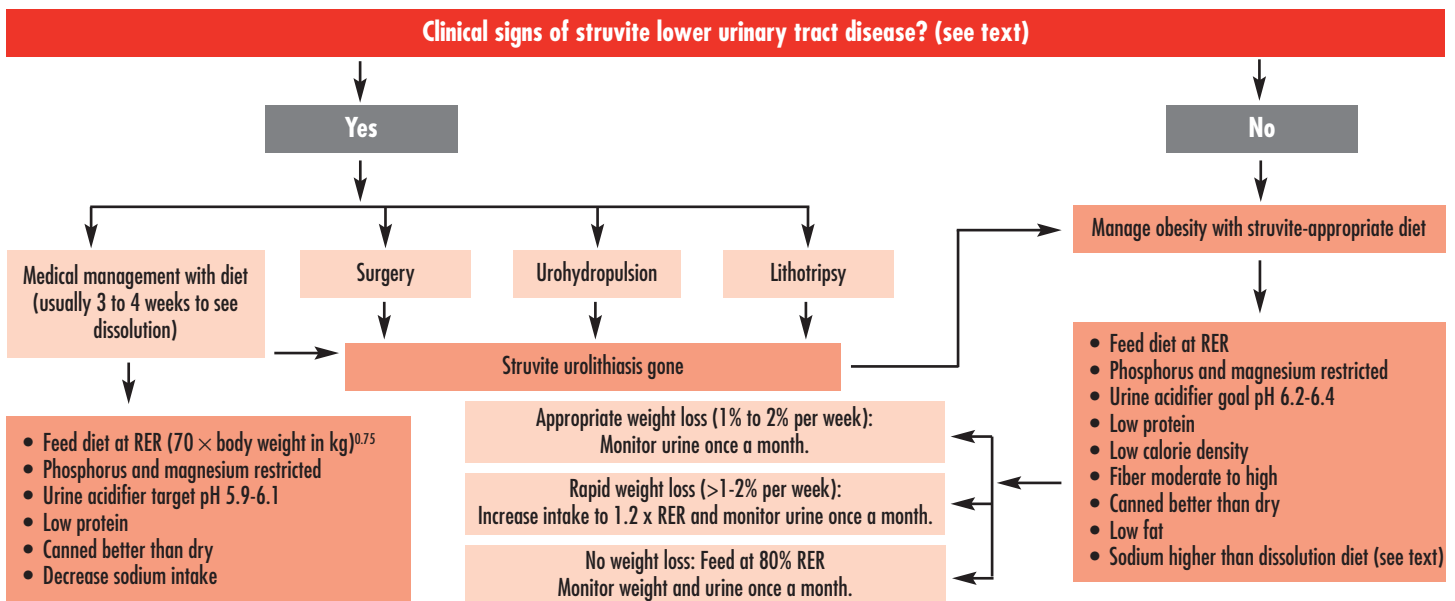
Client Education Points

- Obesity in cats is related to lower urinary tract disease such as struvite urolithiasis.
- A weight loss program should be designed for a 1% to 2% weight loss per week using an appropriate struvite prevention or dissolution diet.
- Cats cannot be “starved” into eating a recommended diet; slow diet transition is necessary.
- If diet modification is not maintained, reformation of struvite uroliths and crystals can occur.
- Older cats can have struvite secondary to bacterial infections.

Common Comorbidities

Bacterial urinary tract infection can be seen in older cats with struvite urolithiasis. Urethral strictures may occur concurrently with struvite urolithiasis. Conditions commonly seen in obese cats include diabetes mellitus, pancreatitis, inflammatory bowel disease, chronic constipation, osteoarthritis, and other lower urinary tract diseases.

Algorithm – Nutritional Management of Concurrent Feline Obesity and Struvite Urolithiasis



Interacting Medical Management Strategies

Steroids for administered for post-obstruction inflammation increase susceptibility to bacterial cystitis. **Appetite stimulants** for diet transition are contraindicated in a weight loss program. **Nonsteroidal anti-inflammatory drugs (NSAIDs)** may cause acute anorexia, gastrointestinal ulcers, and perforation. **Urinary acidifiers** may be used if dietary acidification is not adequate.

Monitoring

Utilizing an appropriate diet for struvite urolithiasis, a weight loss program should be designed to obtain safe weight reduction of 1% to 2% per week. Monthly weights should be charted and dietary intake adjusted accordingly. Once optimal weight and body condition has been achieved the cat's weight can be checked every 4 to 6 months with urinalysis and to assure weight is maintained.

When monitoring for **dissolution** of struvite uroliths, keep in mind that sterile struvite uroliths typically dissolve in 2 to 4 weeks when feeding a struvite dissolution diet. Infection-induced struvite uroliths typically dissolve in 8 to 10 weeks when feeding a struvite dissolution diet and administering appropriate antibiotics. Urinalysis and radiographs should be monitored monthly until uroliths are dissolved. Look for acid urine (pH <6.8), urine specific gravity < 1.030, no crystalluria on sediment evaluation (urine collected by cystocentesis and analyzed immediately; false results are often obtained with cooled urine), resolution of hematuria, and no inflammation.

When monitoring for **prevention** of struvite uroliths, urinalysis and radiographs should be checked monthly to evaluate effectiveness of the diet. Look for acid urine (pH <6.8), urine specific gravity < 1.030, no crystalluria on sediment evaluation (urine obtained by cystocentesis and analyzed within 15 minutes of collection), resolution of hematuria, and no inflammation or signs of lower urinary tract disease. If the diet seems to be effective, urinalysis can be done every 4 to 6 months. With any signs of lower urinary tract disease, radiographs, urinalysis, and blood work as discussed above should be performed.

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Nestlé PURINA

BODY CONDITION SYSTEM

TOO THIN

1 Ribs, lumbar vertebrae, pelvic bones and all bony prominences evident from a distance. No discernible body fat. Obvious loss of muscle mass.

2 Ribs, lumbar vertebrae and pelvic bones easily visible. No palpable fat. Some evidence of other bony prominence. Minimal loss of muscle mass.

3 Ribs easily palpated and may be visible with no palpable fat. Tops of lumbar vertebrae visible. Pelvic bones becoming prominent. Obvious waist and abdominal tuck.

IDEAL

4 Ribs easily palpable, with minimal fat covering. Waist easily noted, viewed from above. Abdominal tuck evident.

5 Ribs palpable without excess fat covering. Waist observed behind ribs when viewed from above. Abdomen tucked up when viewed from side.

TOO HEAVY

6 Ribs palpable with slight excess fat covering. Waist is discernible viewed from above but is not prominent. Abdominal tuck apparent.

7 Ribs palpable with difficulty; heavy fat cover. Noticeable fat deposits over lumbar area and base of tail. Waist absent or barely visible. Abdominal tuck may be present.

8 Ribs not palpable under very heavy fat cover, or palpable only with significant pressure. Heavy fat deposits over lumbar area and base of tail. Waist absent. No abdominal tuck. Obvious abdominal distention may be present.

9 Massive fat deposits over thorax, spine and base of tail. Waist and abdominal tuck absent. Fat deposits on neck and limbs. Obvious abdominal distention.

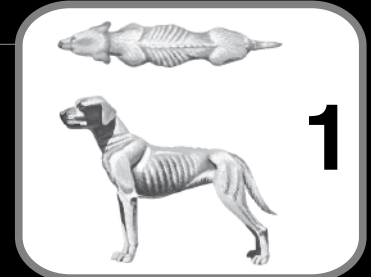
The **BODY CONDITION SYSTEM** was developed at the Nestlé Purina Pet Care Center and has been validated as documented in the following publications:

Mowby D, Bartges JW, Moyers T, et. al. *Comparison of body fat estimates by dual-energy x-ray absorptiometry and deuterium oxide dilution in client owned dogs.* *Compendium* 2001; 23 (9A): 70

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Nestlé PURINA



Nestlé PURINA

BODY CONDITION SYSTEM

TOO THIN

1 Ribs visible on shorthaired cats; no palpable fat; severe abdominal tuck; lumbar vertebrae and wings of ilia easily palpated.

2 Ribs easily visible on shorthaired cats; lumbar vertebrae obvious with minimal muscle mass; pronounced abdominal tuck; no palpable fat.

3 Ribs easily palpable with minimal fat covering; lumbar vertebrae obvious; obvious waist behind ribs; minimal abdominal fat.

4 Ribs palpable with minimal fat covering; noticeable waist behind ribs; slight abdominal tuck; abdominal fat pad absent.

IDEAL

5 Well-proportioned; observe waist behind ribs; ribs palpable with slight fat covering; abdominal fat pad minimal.

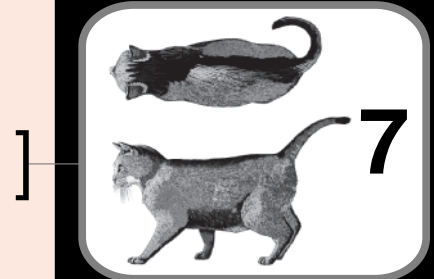
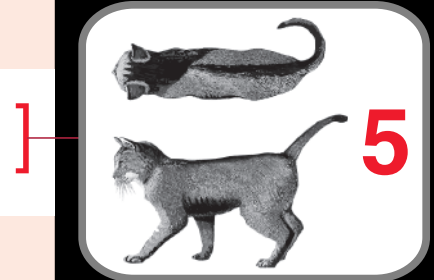
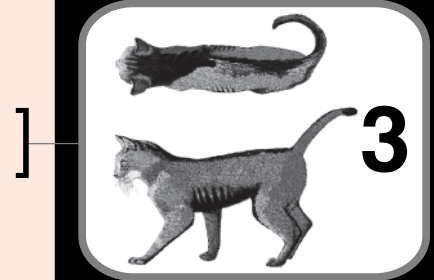
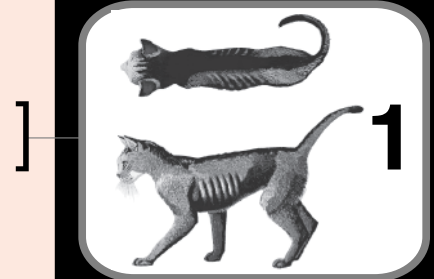
TOO HEAVY

6 Ribs palpable with slight excess fat covering; waist and abdominal fat pad distinguishable but not obvious; abdominal tuck absent.

7 Ribs not easily palpated with moderate fat covering; waist poorly discernible; obvious rounding of abdomen; moderate abdominal fat pad.

8 Ribs not palpable with excess fat covering; waist absent; obvious rounding of abdomen with prominent abdominal fat pad; fat deposits present over lumbar area.

9 Ribs not palpable under heavy fat cover; heavy fat deposits over lumbar area, face and limbs; distention of abdomen with no waist; extensive abdominal fat deposits.



Call 1-800-222-VETS (8387), weekdays, 8:00 a.m. to 4:30 p.m. CT



Nestlé PURINA

Appendix II – Diet History Form

Date: _____ Pet Name: _____ Client Name: _____ Patient ID Number: _____	Reason for Today's Visit: Weight: _____ & _____ kg or lb <div style="text-align: center;"><i>Current</i> <i>Ideal</i></div> Body Condition Score (1-9): _____
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▼ BELOW TO BE COMPLETED BY CLIENT ▼

1. Is your pet housed: Indoors Outdoors Both Outside mainly for walks or exercise
2. Please describe pet's activity level (i.e., type, duration & frequency): _____

3. Do you have other pets? Yes No If yes, please list: _____

4. Is your pet fed in the presence of other animals? Yes No
If yes, please describe: _____
5. Is food left out for your pet during the day or taken away after the meal? _____

6. Does your pet have access to other unmonitored food sources (i.e., food from a neighbor, cat food etc.)? Yes No If yes, please describe: _____

7. Who typically feeds your pet? _____
8. How do you store your pet's food? _____
9. Please list your pet's current and past medical problems, if any, and whether they have resolved:

10. Please list all the medications your pet is currently receiving and any administered over the past three months (indicate medications that are current): _____

11. Please indicate whether your pet has experienced any of the following before today's visit:
Recent involuntary or unintended weight gain OR weight loss
 How much? _____ kg or lb Over what time period? _____
 Vomiting _____ times/day _____ times/week
 Diarrhea _____ times/day _____ times/week
12. Have you observed changes in any of the following:
 Urination **OR** Drinking What was the specific change? _____
 Since when? _____
 Defecation What was the specific change? _____
 Since when? _____
 Appetite What was the specific change? _____
 Since when? _____
13. Does your pet have? allergies **OR** difficulty chewing swallowing
 If so, please describe: _____

Current Diets

Please list below the brand or product names (if applicable) and amounts of ALL foods, snacks, and treats your pet **currently** eats. Please separate out each ingredient in a home-cooked diet, listing each ingredient on its own line. *This description should provide enough detail that we could go to the store and purchase the food. It should include human foods given as treats or at the table. Examples are given in italics.*

Brand/Product/Food	Form	Amount Fed Per Meal	# of Meals	Fed Since
EXAMPLES:				
<i>Brand Name Dog Chow</i>	<i>dry</i>	<i>1 1/2 cups</i>	<i>twice a day</i>	<i>May 2000</i>
<i>Boneless Chicken (white meat)</i>	<i>boiled</i>	<i>2 ounces</i>	<i>3x a week</i>	<i>June 1998</i>

Previous Diets and Supplements

Please list other diets and treats your pet has received **in the past**, indicating the approximate time period when they were fed. *An example is given in italics.*

Brand/Product/Food	Form	From	To	Reason Stopped
EXAMPLES:				
<i>Brand Name Kitten Diet</i>	<i>can</i>	<i>June 1999</i>	<i>March 2000</i>	<i>became an adult</i>

Please list the name of each additional supplement your pet receives, indicate how much and how often your pet receives it (i.e., herbal product, fatty acid, vitamin or mineral supplement):

Pet Dietary Preferences/Restrictions: (What ingredients will/can your pet eat?)

Please fill out this section **ONLY** if a home-cooked diet formulation is being requested or may be needed. If diet formulation is needed due to an adverse reaction to food(s), please provide us with some options of protein and carbohydrate sources that are both **palatable** AND **tolerated** by this animal. This will need to be determined prior to submitting this consult.

Protein Sources

- beef
- chicken
- cottage cheese
- crab
- egg
- lamb
- other: _____
- pork
- salmon*
- tofu
- tuna*
- turkey
- whitefish

Carbohydrate Sources

- barley
- millet
- oatmeal
- pasta, spaghetti
- peas, green
- potato, sweet
- potato, white
- quinoa
- rice, brown
- rice, white
- tapioca
- corn

* These ingredients may contain high levels of mercury – not recommended for long-term feeding

Appendix III – Useful Calculations in Clinical Nutrition

Calculating Energy Requirements

Simple equations can be used to calculate a pet's energy requirements. However, the energy needs of dogs and cats are not a linear function of body weight; energy requirements of animals per kilogram of body weight decrease as the size of the animal increases. In addition, adjustments are needed to account for variations in the daily energy needs of dogs and cats due to factors that include age and life stage, breed, activity level, reproductive status, environment, and health status.

- **Metabolizable energy (ME)** is the portion of the total energy available to the body from food, essentially the usable calories and their concentration, or density. ME is typically measured in calories or joules. A caloric statement is included on most pet food labels, although it is not required. Calories are stated in terms of metabolizable kilocalories per kilogram (ME kcal/kg) of food and may also be expressed as calories per unit of household measure such as per cup or per can. A higher ME indicates a higher concentration of calories, and a more energy-packed food.
- **Energy density** refers to the energy content of the food per unit weight and is usually expressed as kcal/100 g.
- **Resting Energy Requirement (RER)**
The resting energy requirement is the basic amount of energy that used in a day by a pet remaining at rest. For pets weighing between 2 and 45 kg (5–99 pounds), RER, expressed in kilocalories of metabolizable energy (ME) per kilogram body weight per day, can be calculated by:

$$\text{RER (kcal/day)} = 30 \times (\text{current body weight in kg}) + 70, \text{ or}$$

$$\text{RER (kcal/day)} = 70 \times (\text{current body weight in kg})^{0.75}$$

(Convert body weight from pounds to kilograms by dividing by 2.2.) Any activity other than rest will require an increase in energy and an increase in calories to meet the energy needs.

Maintenance Energy Requirement (MER)

The maintenance energy requirement (MER) is based on the RER plus the energy required to move and to digest and absorb food from the intestinal tract. Numerous equations have been developed to calculate MER in healthy dogs and cats but there is no consensus among nutritionists as to which is the most accurate. For an individual animal, the MER provides an initial guideline; ongoing assessment of body condition score (BCS) should then be used to adjust food intake up or down as needed.

To calculate MER, expressed in kilocalories of metabolizable energy (ME) per kilogram body weight per day, begin with the equations for RER shown above. The mathematical equation will overestimate MER in very small dogs and in large-breed dogs, so the exponential equation should be used in these animals. Because there is less variation in body weight among adult cats, MER can be estimated as:

$$\text{MER} = 80 \times (\text{BW in kg}) \text{ for active cats}$$

$$\text{MER} = 70 \times (\text{BW in kg}) \text{ for inactive cats}$$

To calculate MER, individual variables must be accounted for—such as age and life stage, reproductive status (intact vs. neutered), body condition

(obese or underweight), gestation and lactation, or illness—by multiplying by a specific factor. For example:

Puppies	
Birth to half mature weight	RER × 2.2
Half mature weight to adult	RER × 1.5
Neutered adult dog	RER × 1.8
Obese-prone adult dog	RER × 1.2 to 1.4
Pregnant bitches	RER × 1.3
Lactating bitches	
Up to 1 week post-whelping	RER × 1.2
Peak (3–4 weeks post-whelping)	RER × 3.5
Up to 6 weeks	RER × 1.5
Neutered adult cat	RER × 1.2 to 1.4
Intact adult cat	RER × 1.4 to 1.6
Obese-prone adult cat	RER × 1.0

Once an animal's maintenance energy requirement—the number of kilocalories needed per day—has been calculated, the amount of the appropriate diet to feed can be determined by checking for the number of kcal/cup or kcal/can on the pet food label and calculating the serving size to meet those needs..

Converting from As Fed to Dry Matter Basis

The “guaranteed analysis” (GA) on pet food labels lists nutrient levels on an “as fed” basis, that is, the amounts of nutrients, as a percentage, present in the bag or can. When comparing the labels of dry and canned products, the levels of crude protein and most other nutrients are much lower for canned products. For example, a canned food guarantees 8% crude protein and 75% moisture (or 25% dry matter), while a dry food contains 27% crude protein and 10% moisture (or 90% dry matter). It may not be obvious which diet has more protein, the dry or canned? The difference can be explained by looking at the relative moisture contents: Canned foods typically contain 75% to 78% moisture, whereas dry foods contain only 10% to 12% moisture.

The most accurate means of comparing nutrient levels of dry and canned diets is to convert the guarantees for both products to a moisture-free or dry matter (DM) basis. To calculate dry matter percentages, start by subtracting the amount of moisture from the total, leaving the amount of “dry matter.” Then divide the amount of the nutrient, for example, protein or fat, by the amount of dry matter. Thus, if the canned diet has 75% moisture:

$$(100\% - 75\%) = 25\% \text{ dry matter}$$

The protein content can be calculated by dividing it by 25. For example, if the label says it contains 8% protein:

$$8 / 25 \times 100 = 32\% \text{ protein on a dry matter basis}$$

For the dry diet,

$$100\% - 10\% = 90\% \text{ dry matter}$$

If the label for the dry diet lists 27% crude protein,

$$27 / 90 \times 100 = 30\% \text{ protein on a dry matter basis}$$

Continues

Thus, although it looks like the dry diet in this example has a lot more protein, the canned diet actually has a little more. A quick comparison can be made by estimating that the amount of dry matter in a dry food is about four times the amount in a canned product.

Using the Guaranteed Analysis Statement on Pet Food Labels to Determine the Carbohydrate Content of the Food*

The guaranteed analysis statement on pet foods does not provide information on the carbohydrate content of the food, and this must be inferred. For example, a guaranteed analysis statement for a dog food might be as follows:

Crude Protein Min.	8.0%
Crude Fat Min.	5.0%
Crude Fiber Max.	1.5%
Moisture Max.	75%
Ash Max.	3.0%

The dry matter (DM) portion of the food contains all of the non-water nutrients and can be roughly calculated using the moisture content, which in this case is 75%. This is typical for moist foods. The typical moisture content for dry foods is approximately 10%.

Crude Protein Min.	$8.0\% / (1.00 - 0.75) = 32\% \text{ DM}$
Crude Fat Min.	$5.0\% / (1.00 - 0.75) = 20\% \text{ DM}$
Crude Fiber Max.	$1.5\% / (1.00 - 0.75) = 6\% \text{ DM}$
Ash Max.	$3.0\% / (1.00 - 0.75) = 12\% \text{ DM}$

The total %DM accounted for by protein, fat, fiber, and ASH is thus 70% (32% + 20% + 6% + 12%). The remainder can be assumed to primarily contribute the carbohydrate content of the food.

$$\text{Carbohydrate } 100\% - (\text{total \%DM of other nutrients}) = 30\% \text{ DM}$$

* This section provided by Linda Fleeman, BVSc, MACVSc, PhD, and Jacquie Rand, BVSc, DVSc, DACVIM.

