

Nutritional Management of Diseases

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The American College of Veterinary Nutrition recommends a three-step approach to patient assessment that includes assessment of patient factors, dietary factors, and feeding factors. After the assessment phase, a nutritional treatment plan is developed and instituted, and serial monitoring and adjustment ensue (an iterative process).²⁵⁹ This chapter focuses on the nutritional management of feline disorders. Additional information on each disorder can be found in other chapters in this book.

CARDIOVASCULAR DISEASES

Cardiovascular disease commonly occurs in cats with myocardial disease, occurring more commonly than valvular disease. The prevalence of dilated cardiomyopathy has decreased after the discovery of its association with taurine deficiency;²¹³ hypertrophic and restrictive cardiomyopathies occur most frequently. Systemic arterial hypertension may also result in left ventricular hypertrophy and myocardial failure. Cats with myocardial disease may be asymptomatic or may present with evidence of venous congestion, usually pleural effusion. For more information on cardiac disease, see Chapter 20.

Animal Factors

Cats with myocardial disease may be optimally conditioned or may be underconditioned or overconditioned depending on the severity and chronicity of the disease. Obesity results in blood volume expansion with elevated cardiac output, increased plasma and extracellular fluid volume, increased neurohumoral activation, reduced urinary sodium and water excretion, tachycardia, abnormal systolic and diastolic ventricular function, exercise intolerance, and systemic arterial hypertension.⁸⁶ This may result in progression of the disease. Likewise, cachexia may occur with myocardial failure. Cachexia associated with heart disease or failure results in negative nitrogen and energy balance.³⁹ The pathogenesis of cardiac cachexia is multifactorial, involving increased sympathetic tone, increased tumor necrosis factor and interleukin-1 levels, decreased physical activity with an increased resting energy requirement (RER), decreased tissue perfusion, venous congestion, and adverse effects of medications. Decreased nutrient intake and possibly increased nutrient losses (e.g., potassium loss with diuretic therapy) and loss of body weight and, more important, lean body mass occurs, resulting in an inability to respond to medical therapy and an increase in morbidity and mortality rates.

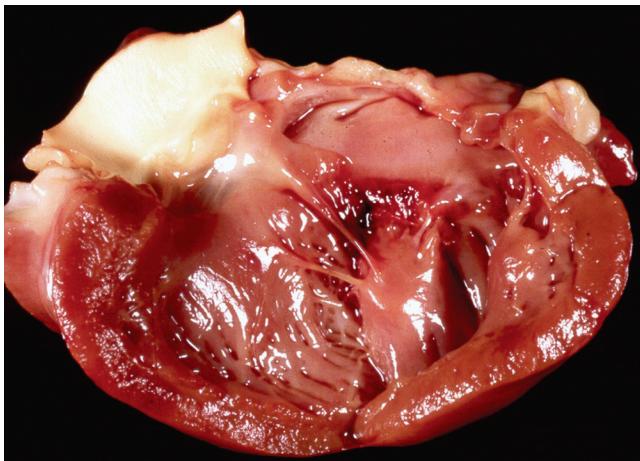


FIGURE 18-1 Dilated cardiomyopathy in a 4-year-old spayed female domestic shorthair cat with taurine deficiency.

Cats have a dietary requirement for taurine because they have limited ability to synthesize it from cysteine and methionine and because it is used exclusively for bile acid conjugation. Taurine deficiency results in dilated cardiomyopathy in predisposed cats (Figure 18-1). The mechanism of heart failure in taurine-deficient cats is poorly understood. Taurine may function in osmoregulation, calcium modulation, and inactivation of free radicals.²¹³ Other factors are likely involved because many cats fed taurine-deficient foods for prolonged periods fail to develop myocardial dysfunction. Additionally, there is an association between taurine and potassium balance.⁶⁷ Inadequate potassium intake may induce significant taurine depletion, resulting in myocardial dysfunction. Male cats may be more prone to developing taurine deficiency-associated myocardial failure than are female cats, or male cats may be more prone to developing clinical signs at higher plasma taurine concentrations.⁸³

L-Carnitine is a conditionally essential nutrient involved with transport of long-chain fatty acids from the cytosol into the mitochondria, where they undergo beta oxidation for energy production. L-Carnitine deficiency has been associated with dilated cardiomyopathy in some dogs¹³²; however, it has not been associated with this condition in cats.

Sodium intake is often restricted with heart disease; however, this may not be necessary until later in the course of the disease. Sodium restriction should occur concurrently with chloride restriction because chloride salt of sodium has more effect on blood pressure and plasma volume than non-chloride sodium salts.²⁶ Salt sensitivity has not been documented to occur in cats. Hypokalemia and hypomagnesemia are associated with arrhythmias, decreased myocardial contractility, and muscle weakness. Additionally, inadequate potassium intake may be associated with taurine deficiency.

BOX 18-1

Dietary Recommendations for Cats with Cardiac Disease

1. Restrict calories if obese; however, increase caloric intake if cachectic.
2. Protein content must be adequate or greater than normal (30% to 45% protein on a dry matter basis).
3. Omega-3 fatty acids may be beneficial in an omega-6:omega-3 ratio of 5:1.
4. Sodium restriction (0.07% to 0.3% on a dry matter basis) with chloride restriction (to 1.5 times the sodium content) is indicated with congestive heart failure.
5. Adequate potassium (>0.5% on a dry matter basis), phosphorus (0.3% to 0.7% on a dry matter basis), magnesium (>0.04% on a dry matter basis). With diuretic therapy, additional potassium supplementation may be required to prevent hypokalemia.
6. Taurine should be present in the diet at >0.3% on a dry matter basis but can be supplemented at 250 to 500 mg by mouth every 12 to 24 hours.

Dietary Factors

Dietary recommendations for cats with cardiac disease are designed to optimize body condition and are summarized in Box 18-1. Commercial diets formulated for cats with cardiovascular disease are available.

Feeding Factors

Some cats require feeding of frequent small meals because of decreased appetite. Pharmacologic appetite stimulation (Table 18-1) or assisted feeding through the use of feeding tubes may be required. Additional nutrients that may be beneficial include coenzyme Q10, which is required for energy reactions and is an antioxidant, and other antioxidants, which may decrease oxidative stress with dilated cardiomyopathy.

DENTAL AND ORAL DISEASES

Primary oral diseases are subdivided into conditions affecting the tooth, periodontium, and other oral tissues. In many cases dental disease is secondary to a systemic condition such as chronic renal disease in cats, although primary disorders such as lymphoplasmacytic gingivitis-stomatitis, tooth resorption, and neoplasia occur in cats. For more information, see Chapter 21.

Animal Factors

Oral disease occurs more commonly in older cats and usually is associated with systemic disease; therefore

TABLE 18-1 Appetite Stimulants for Use in Cats

Agent	Dose	Route	Frequency
Mirtazapine	1/8-1/4 of 15 mg tab	PO	q72h
Maropitant	2-4 mg/kg	PO	q24h × 5 days
Diazepam	1-2 mg/cat 0.05-0.1 mg/kg 0.5-2.0 mg	PO IV IV	As needed As needed As needed
Oxazepam	0.3-0.4 mg/kg 2-2.5 mg	PO PO	q12-24h q12-24h
Flurazepam	0.2-0.4 mg/kg	PO	q4-7d
Chlordiazepoxide	2 mg	PO	q12-24h
Cyproheptadine	2 mg	PO	q8-12h
Prednisone	0.25-0.5 mg/kg	PO	q48h
Boldenone undecylenate	5 mg	IM/SC	q 7 days
Nandrolone decanoate	10 mg	IM	q 7 days
Stanozolol	1-2 mg 25-50 mg	PO IM	q12h q 7 days
B vitamins	1 mL/L fluids	IV	—
Cobalamin	0.5 mg/kg	SC	q24h
Elemental zinc	1 mg/kg	PO	q24h
Potassium	0.5-1 mEq KCl/kg 3 mEq K gluconate	PO PO	q12h q6-8h
Interferon alfa 2b	3-30 IU	PO	q12h

PO, By mouth; IV, intravenously; IM, intramuscularly; SC, subcutaneously.

oral disease may be related to systemic effects of those disorders and to nutritional imbalances caused by those disorders or eating difficulties. Historical information should include diet, eating behavior, and access to toys and other foreign bodies. The veterinarian should perform a complete oral examination, which may require sedation or anesthesia, in addition to a physical examination to evaluate for systemic disease.

Dietary Factors

Several dietary factors have been implicated with oral disease. Food texture and composition can directly affect oral cavity health through the following:

- Maintenance of tissue integrity
- Alteration of bacterial plaque metabolism
- Stimulation of salivary flow
- Cleaning of tooth and oral surfaces by physical contact
- Chelation of calculogenic constituents¹⁶²

Claims of dry food being better for prevention of dental plaque than moist foods are unsubstantiated.³⁰

Likewise, there are no data to support the notion that natural diets and foods are better for oral health than commercial foods. Dietary texture can be modified by increasing fiber content with a size and texture that promotes chewing and mechanical cleansing of teeth.^{30,161,272} Dental treats do not offer an advantage over dry foods; however, some contain hexametaphosphate, a calcium chelator, which may decrease calculus formation, although data are contradictory.^{114,256} Hexametaphosphate has not been evaluated in cats. Many oral diseases are inflammatory, and modification of the inflammatory process may be beneficial. Antioxidants, vitamins E and C, and selenium may be beneficial, but data are lacking in cats. Nutritional deficiencies of such elements as calcium and vitamins A, B, C, D, and E are associated with oral cavity disease, but these are uncommon (see Chapter 17).

Nutrition has been implicated as a cause of feline tooth resorption. Acid coating of foods has been suggested to cause tooth resorption, although this has not been proved.^{5,226,278} Dry food may cause microfractures that predispose teeth to infection and inflammation; however, this has not been proved. Dietary vitamin D has been implicated in tooth resorption. Evidence to support this assertion includes correlation between cats with tooth resorption and increased blood levels of 25-hydroxyvitamin D and histologic comparisons of the effects of excessive intake of vitamin D to the effects of bone resorption.^{226,227} Although a direct effect of vitamin D has not been established, there is evidence of an active vitamin D signaling in the pathophysiology of tooth resorption.^{27,28}

Feeding Factors

Feeding a “dental” diet that carries the Veterinary Oral Health Council (<http://www.vohc.org/>) seal for plaque control may be beneficial for cats that are prone to periodontal disease. Additional recommendations include the following:

1. Vitamin E: >500 IU/kg
2. Vitamin C: 100 to 200 mg/kg
3. Selenium: 0.5 to 1.3 mg/kg
4. Phosphorus: 0.5% to 0.8% on a dry matter basis
5. Sodium: 0.2% to 0.5% on a dry matter basis
6. Magnesium: 0.04% to 0.1% on a dry matter basis

SKIN DISORDERS

The most common feline skin disorders are abscesses, parasitic dermatoses, allergy (flea bite hypersensitivity and atopic dermatitis), miliary dermatitis, eosinophilic granuloma complex, fungal dermatitis, adverse reactions to food, psychogenic dermatoses, seborrheic conditions, neoplasia, and immune-mediated dermatoses.^{116,243}

Animal Factors

Clinical signs associated with nutritional abnormalities include a sparse, dry, dull, and brittle hair coat that epilates easily; slow hair growth; abnormal scale accumulation; alopecia; erythema; crusting; decubital ulcers; and slow wound healing. Other clinical signs may be present with nutrient-deficient dermatoses (see Chapter 17). For more information on skin diseases, see Chapter 22.

Dietary Factors

Inadequate energy intake is associated with keratinization abnormalities, depigmentation, changes in epidermal and sebaceous glands, and increased susceptibility to trauma. Protein deficiency is associated with similar clinical signs. Essential omega-6 fatty acids include linoleic acid (>0.5% on a dry matter basis) and arachidonic acid (>0.02% on a dry matter basis).¹⁶⁹ Omega-3 fatty acids can supply part of the omega-6 fatty acid component. Clinical signs of essential fatty acid deficiency include scaling, matting of hair, loss of skin elasticity, dry and dull hair coat, erythema, epidermal peeling, otitis externa, and slow hair growth. Certain mineral deficiencies may affect the skin (see Chapter 17). Copper deficiency is associated with loss of normal hair coloration, decreased density or lack of hair, and rough or dull hair coat. Many dermatologic conditions may occur with zinc deficiency and respond to zinc supplementation. Dietary phytate binds zinc, resulting in clinical signs of deficiency. Clinical signs of zinc deficiency include erythema, alopecia, and hyperkeratosis. Certain vitamin deficiencies may affect the skin. Vitamin A deficiency is associated with skin lesions and focal sloughing of skin. Vitamin E deficiency occurs in cats in association with steatitis. Clinical signs include erythema and keratinization defects. Vitamin E-responsive dermatoses include discoid lupus erythematosus, systemic lupus erythematosus, pemphigus erythematosus, sterile panniculitis, acanthosis nigricans, dermatomyositis, and ear margin vasculitis. Dermatologic conditions also arise from food allergies (see Chapter 17).

Feeding Factors

In cats with dermatologic disease, the veterinarian should evaluate the quality and quantity of the diet fed, including treats, snacks, and table food. A complete and balanced diet may be made incomplete or unbalanced when fed with other food stuffs. Homemade diets must be evaluated carefully.²²⁸ If a nutritional deficiency is suspected, the veterinarian should discuss with the owner the possibility of changing the diet to one of a higher quality. With suspected adverse food reaction, a diet change should be considered (see Chapter 17). If a specific nutrient deficiency is identified, the diet might

be changed or supplemented accordingly. Zinc is supplemented for zinc-responsive dermatoses (zinc sulfate: 10 to 15 mg/kg per day by mouth; zinc methionine: 2 mg/kg per day by mouth). Vitamin A is supplemented for vitamin A-responsive dermatoses (tretinoin topically every 12 to 24 hours; isotretinoin: 1 to 3 mg/kg per day by mouth; etretinate: 0.75 to 1 mg/kg per day by mouth).

Fatty acid supplementation is often recommended for managing inflammatory skin disease. Cats have a limited capacity to convert 18-carbon long-chain fatty acids to 20-carbon long-chain fatty acids owing to low activity of delta-6-desaturase.²⁰¹ It is 20-carbon long-chain fatty acids that are incorporated into cellular membranes and subsequently metabolized to prostaglandins, leukotrienes, and thromboxanes. To alter levels of these cytokines in cats, it is necessary to supplement 20-carbon long-chain fatty acids. Insertion of omega-3 fatty acid (eicosapentaenoic acid [EPA]) into cell membranes results in production of cytokines of the odd-number series (e.g., prostaglandin E₃, leukotriene B₅) in place of the even-number series of cytokines produced from omega-6 long chain fatty acid, arachidonic acid (e.g., prostaglandin E₂, leukotriene B₄). These odd-numbered cytokines promote less inflammation and are more vaso-dilatory than the even-numbered cytokines. Cats being supplemented with omega-3 fatty acids must receive 20- and 22-carbon fatty acids owing to their limited ability to convert 18-carbon to 20-carbon fatty acids. The 20-carbon omega-3 fatty acid is EPA, and the 22-carbon omega-3 fatty acid is docosahexaenoic acid (DHA). Oils derived from marine life are high in EPA and DHA. Oils derived from plant life, such as flax seed and borage, contain primarily 18-carbon fatty acids, therefore limiting their conversion to the required 20-carbon fatty acid and their effectiveness in managing inflammation. There are no data on the effectiveness of omega-3 fatty acids in managing inflammatory skin diseases in cats.⁴¹

GASTROINTESTINAL DISEASES

Many disorders of the gastrointestinal system may respond to dietary management, whether or not the disorder is due to diet. For more information on gastrointestinal diseases, see Chapter 23. For information on adverse reactions to food, see Chapter 17.

Types of Foods Used in Managing Gastrointestinal Disease

Gastrointestinal Diets

Gastrointestinal diets are highly digestible with consistent ingredient and nutrient profiles. Highly digestible implies protein digestibility above 87% and fat and carbohydrate digestibility above 90%. These diets contain refined meat and carbohydrate sources with

carbohydrates in the largest amount, fortified with fat-soluble vitamins, and contain less than 5% fiber (dry matter basis). The fiber is usually a soluble fiber or mixed fiber source providing substrate for intestinal microbial fermentation.

Fiber-Enhanced Diets

Fiber-enhanced diets contain 15% to 25% fiber (dry matter basis), often insoluble fiber. Soluble fiber increases the viscosity of intestinal contents, delays gastric emptying, slows intestinal transit time, undergoes microbial fermentation, and binds toxins and bile acids. Insoluble fiber is slowly fermentable, has little to no effect on gastric emptying, normalizes intestinal transit time, and increases fecal bulk.

Restricted- and Moderate-Fat Foods

Dietary fat is more digestible and more energy dense than carbohydrate. Average fat digestibility is 74% to 91% in cats. Moderate-fat diets containing 15% to 22% (dry matter basis) are tolerable. Low-fat diets (<10% dry matter basis) necessitate increased food intake to meet caloric needs.

Elimination Foods

Many diets are available with novel protein sources, including protein hydrolysates, duck, venison, rabbit, kangaroo, lamb, and fish.

Gluten- and Gliadin-Free Foods

Several potential antigens are found in flour when cereal grains are processed. Gliadin, a polypeptide, is responsible for gluten-sensitive enteropathies and is found in wheat, barley, rye, buckwheat, and oat flours. It is not present in whole grains and flours produced from rice and corn.

Monomeric Foods

Monomeric foods are water-soluble liquid foods containing nutrients in simple forms. They are hypoallergenic because they require minimal digestion for absorption; however, they are expensive and not very palatable.

Animal Factors

Cats with pharyngeal or esophageal disease have difficulty swallowing food; these diseases are, however, uncommon in cats. Cats with gastric or small intestinal disease often vomit, and they may have diarrhea characterized by large-volume watery feces or inappetence. Poor body condition and weight loss may occur because of the inability to eat, vomiting, or loss of nutrients with diarrhea. Inflammatory bowel disease is the most common cause of small intestinal disease; however, other conditions, such as neoplasia and foreign body

obstruction, may occur. Large intestinal disease is usually associated with small-volume firm feces with mucous and/or blood or with constipation or obstipation.

Dietary Factors

Cats with pharyngeal and esophageal disease should be given a high-protein food (>40% on a dry matter basis) because protein increases lower esophageal tone. Feeding a high-fat (>25% on a dry matter basis) diet increases the caloric density of the diet; however, it delays gastric emptying. Therefore if gastric motility and emptying is a concern, a high-fat diet may not be indicated.

Several options exist for nutritional management of cats with inflammatory gastroenteritis.^{106,280} In addition to pharmacologic therapy, cats may respond to elimination diets, whether it is a diet containing a novel protein source, a protein hydrolysate diet, or a homemade simple-ingredient diet. Inflammatory reactions are thought to occur through interaction of a protein with an antibody directed against it. *Novel protein* refers to a single dietary protein source that the cat has not been fed before; therefore an inflammatory response would not be evoked.¹⁰⁶ A protein hydrolysate diet involves feeding a diet where the protein has been hydrolyzed to a size that is not recognized by antigen processing cells and antibodies, typically below 12,000 daltons.⁴³ A homemade diet often comprises single unprocessed ingredients. In processing of foods, glycated protein end-products may be produced through the Mallard reaction and these glycated end-products may induce an inflammatory response. Feeding unprocessed foods decreases exposure to these glycation end products and subsequent inflammatory response.¹⁵⁶

Cats with large intestinal disease may respond to an elimination diet or to a higher fiber (>5% on a dry matter basis) diet.^{63,202,247,280} Dietary fiber increases fecal bulk, which stimulates colonic contraction; however, it increases fecal volume, which could exacerbate constipation.²⁴⁷ Dietary recommendations for cats with inflammatory bowel disease are summarized in Box 18-2.⁶¹

Feeding Factors

A change of diet or formulation of diet, including a homemade diet, may be necessary to manage cats with intestinal disease. Feeding smaller meals or restricting the amount consumed at a meal may be beneficial.

HEPATIC DISEASE

The liver is a metabolically active organ involved with digestion and nutrient metabolism, synthesis (e.g. albumin), storage (e.g., glycogen), removal of environmental and endogenous noxious substances, and

BOX 18-2**Dietary Recommendations for Cats with Inflammatory Bowel Disease**

1. Fat: 15% to 25% when feeding a highly digestible diet or 9% to 18% on a dry matter basis when feeding a fiber-enhanced diet.
2. Protein: >35% on a dry matter basis. When using a limited protein (elimination) diet, restrict protein to one or two sources and use a protein source that the cat has not consumed previously (novel protein).
3. Fiber: <5% on a dry matter basis for highly digestible diet or 7% to 15% for foods with increased fiber.
4. Digestibility: >87% for protein and >90% for fat and digestible carbohydrate for highly digestible diet, or >80% for protein and fat and >90% for carbohydrate for high fiber diet.

metabolism of drugs and toxins. The liver influences nutritional status through bile acid synthesis and excretion into the gastrointestinal tract and its central role in intermediary metabolism of proteins, carbohydrates, fat, and vitamins. The most common causes of feline liver disease include inflammatory conditions (cholangitis-cholangiohepatitis complex), lipidosis, neoplasia (particularly lymphoma), and portovascular anomalies.¹⁹⁴ For more information on liver diseases, see Chapter 23. Nutritional management of hepatobiliary disease is usually directed at clinical manifestations of the disease rather than the specific cause. Goals of nutritional management of cats with liver disease include the following:

- Maintaining normal metabolic processes and homeostasis
- Avoiding and managing hepatoencephalopathy
- Providing substrates to support hepatocellular repair and regeneration
- Decreasing further oxidative damage to damaged hepatic tissue
- Correcting electrolyte disturbances⁴⁵

Animal Factors

Animals with liver disease may demonstrate a variety of clinical signs, from none to hepatoencephalopathy (characterized by ptalism, vomiting, depression, and possibly seizures). Weight loss and poor body condition may or may not be present depending on the severity and chronicity of the underlying disease. Cats may or may not have hyperbilirubinemia.

Dietary Factors

Maintenance of body condition and weight is important; therefore adequate caloric intake is paramount. Hepatic

BOX 18-3**Dietary Recommendations for Cats with Hepatobiliary Disease**

1. An energy-dense diet containing >4.2 kcal/g
2. Protein: 30% to 45% on a dry matter basis unless hepatoencephalopathy is present: 25% to 30% on a dry matter basis
3. Arginine: 1.5% to 2% on a dry matter basis
4. Taurine: >0.3% on a dry matter basis
5. Potassium: 0.8% to 1.0% on a dry matter basis
6. L-carnitine: >0.02% on a dry matter basis
7. Vitamin E: >500 IU/kg
8. Vitamin C: 100 to 200 mg/kg

lipidosis is a result of negative energy balance with mobilization of peripheral adipose tissue and accumulation of intrahepatic lipid.⁴⁶ In managing cats with hepatic lipidosis, reversing the negative energy balance is most important in reversing the disease process. Protein restriction is not necessary unless hepatoencephalopathy and hyperammonemia are present. Hypokalemia may occur with liver disease and has been reported in approximately one third of cats with hepatic lipidosis⁴⁶; therefore diet should be potassium replete. Many hepatic diseases are associated with oxidative stress that may induce further hepatocellular damage. Feeding diets with additional antioxidants or supplementing with antioxidants may be beneficial. Hepatic dysfunction entails a dysregulation of lipid metabolism; this is particularly prominent with hepatic lipidosis. L-Carnitine is involved with lipid metabolism, and although L-carnitine deficiency does not occur with hepatic lipidosis,¹²⁶ L-carnitine supplementation at 250 to 500 mg daily may be beneficial in cats with hepatic lipidosis.⁴⁴

Dietary recommendations for cats with hepatobiliary disease are summarized in Box 18-3.¹⁹⁴

Feeding Factors

Feeding small meals or facilitating food intake using pharmacologic stimulation or feeding tubes may be required. Caution must be exercised with administration of medications that require hepatic metabolism, such as appetite stimulants, because side effects may occur.

ENDOCRINOLOGIC DISEASES: OBESITY

Obesity is the most important nutritional disease of cats. With prevalence rate estimates of up to 40%,^{8,166,242} obesity must be considered a significant hazard to cats. Increased emphasis on pet health and preventive health programs

makes obesity prevention an important aspect of health maintenance programs in dogs and cats. Treatment for obesity varies from frustrating to rewarding, and evaluating and prescribing for successful, long-term weight loss and maintenance usually require management of multiple, interrelated patient and client factors. Diagnosis of disease secondary to obesity and the major task of client education and motivation are the province of the veterinarian.

Obesity is a condition of positive energy balance and excess adipose tissue accumulation that adversely affects the quality and quantity of life. *Obesity* literally means increased body fatness, but measurement of fat fractions of body composition is difficult in practice. Therefore *obesity* can be defined as body weight in excess of 15% to 20% of ideal, owing to the accumulation of body fat.²⁸¹ Negative health manifestations often begin at this level of weight excess and are a virtual certainty at a 30% excess over ideal weight. Associated health risks include musculoskeletal and cardiovascular disease, diabetes mellitus, hyperlipidemia, hepatic lipidosis, higher incidence of cancer, possible anesthetic and surgical complications, decreased heat tolerance and stamina, and reproductive problems. Obesity is a proinflammatory condition, and adipose tissue is an active endocrine organ that produces cytokines called adipokines.^{168,221} This may explain in part the association of obesity with inflammatory conditions such as osteoarthritis.

The pathogenesis of obesity is multifactorial and is more than just "too much energy in and not enough energy out."¹⁴⁵ There are genetic, gender, and environmental influences. Apartment dwelling, inactivity, middle age, being male, neutered status, mixed parentage, and certain dietary factors are associated with being overweight.^{166,242} The pet owner's contribution to the problem may be significant and must be understood and addressed. In one survey of more than 18,000 dog and cat owners in Australia and the United States, almost a third of owners reported their pets as overweight or obese, but fewer than 1% felt that obesity was a health problem.⁸⁷ In another study of 120 German owners of indoor cats, questionnaire responses of owners of cats with normal body weight were compared with responses from owners of overweight cats.¹³³ Owners of overweight cats were more likely to watch their cats eat and relented more frequently when their cats begged for food. Owners of overweight cats were less likely to spend time playing with their cats and appeared to have a different relationship with them, being more likely to anthropomorphize them and consider them a substitute for human companionship.

Diagnosis of obesity is the first step in managing the disease. Determining whether a cat is overweight is not difficult; however, accurately determining the degree of overweight and the cat's ideal weight is challenging. Many owners underestimate their cat's body condition,

and veterinarians may overlook obesity. Documenting body weight in the medical record is important; in fact, veterinarians may be part of the problem. In one study medical records dramatically underreported overweight and obesity in cats when body condition scoring (BCS) results were compared to reported diagnoses.¹⁶⁶ For example, the prevalence of obesity defined by BCS in the population studied was 6.4% compared with 2.2% when defined by a recorded diagnostic code in the medical record. In addition to recording the body weight, it may be helpful to calculate the percentage change in weight since the last visit and compare it to a similar weight gain in a person. For example, an 8.8-lb (4-kg) cat that has gained 1 lb (0.5 kg) has increased its body weight by approximately 12%; this is equivalent to a 14-lb weight gain for a 120-lb person.

Muscle condition scoring and BCS provide additional information regarding the appropriateness of the cat's body weight to its overall condition.* Several BCS systems are available; the most widely used are the 5-point and 9-point scales (see Table 16-2).[†] In both scales the middle value (3/5 or 5/9) is considered optimal condition, and these cats have 15% to 25% body fat. Lower values on the scale are degrees of undercondition (cats having 2/5 or 3/9 have 5% to 15% body fat, and cats having 1/5 or 1/9 have <5% body fat), whereas higher values on the scale are degrees of overcondition (cats having 4/5 or 7/9 have 25% to 35% body fat, and cats having 5/5 or 9/9 have higher than 35% body fat).²⁶⁵ As additional data are generated, it is likely that revisions of the scales will occur.²⁶⁵ Muscle condition scoring assesses muscle mass and tone.¹⁹⁶ Evaluation of muscle mass includes visual examination and palpation over the temporal bones, scapulae, lumbar vertebrae, and pelvic bones. Decreased muscle mass may increase morbidity and mortality rates associated with disease.⁵²

Animal Factors

The most important step is to recognize that a cat is overweight or obese. The veterinarian should compare the BCS with body weight, especially with historical data from annual examinations. Often a cat's ideal body weight can be determined by finding its weight at about 1 year of age in the medical record. Cats that are obese may show clinical signs of related conditions such as diabetes mellitus, hepatic lipidosis, and osteoarthritis. The veterinarian should take a good dietary history, including the type(s) of food fed, amount(s), and frequency.¹⁹⁵ It is important to gather information about snacks, treats, and table foods that may be fed, as well as access to food in the outdoors if the cat is allowed

*References 10, 40, 56, 144, 145, 167, 239, 241, 242, 274.

†References 10, 40, 143-145, 239, 241.

BOX 18-4**Questions to Ask When Taking a Diet History**

1. What types and amounts of food are fed?
 - Include all treats and table food, other pets' food that may be eaten, prey, and food fed by neighbors for outdoor cats.
 - Include food or treats (e.g., Greenies Pill Pockets) used to administer medication.
2. How is the food measured?
 - Who feeds the cat its regular meals? Who gives the cat treats?
 - When and where is the cat fed?
 - Are there other pets in the home? If so, does each pet have a separate feeding station, or can they access each other's food?

access (Box 18-4). It may be helpful to have the owner keep a food diary for 1 or 2 weeks before the weight loss program is initiated. Collecting the information may help make owners aware of the role they play in the cat's obesity, as well as provide useful information.

Dietary Factors

To achieve weight loss to ideal body weight, a change in diet is necessary. Feeding less of the same food is usually unsuccessful because the cat is used to eating the diet, it does not induce a shift in metabolism, and it may lead to deficiencies. There are two dietary strategies for inducing weight loss in cats:

1. Increased-fiber/high-carbohydrate diets:
 - Carbohydrates: <40% on a dry matter basis. Avoid simple sugars and starch.
 - Fiber: 7% to 18% on a dry matter basis
 - Fat: <15% to 17% on a dry matter basis
 - Protein: 30% to 55% on a dry matter basis
 - Food form: Avoid semi-moist foods. Canned foods may facilitate weight loss better than dry foods.
2. Increased-protein/low-carbohydrate diets²⁶⁷:
 - Carbohydrates: <20% on a dry matter basis. Avoid simple sugars and starch.
 - Fiber: Usually <5% on a dry matter basis
 - Fat: 12% to 25% on a dry matter basis
 - Protein: 30% to 55% on a dry matter basis
 - Food form: Avoid semi-moist foods. Canned foods may facilitate weight loss better than dry foods.

Which dietary strategy will work in an individual cat is unknown. If one dietary strategy does not work, the veterinarian should switch to the other.

Recently, the first drug licensed for weight loss in veterinary medicine was approved for dogs. Dirlotapide

(Slentrol, Pfizer Animal Health) is a selective microsomal triglyceride transfer protein inhibitor. The drug reduces fat absorption and increases satiety signals. However, dirlotapide is contraindicated in cats because it increases the risk of hepatic lipidosis. In multi-pet households in which a dog may be receiving the drug, client education is important to avoid administration of the drug to overweight or obese resident cats.

Feeding Factors

Weight-reduction programs are a multistep approach involving owner commitment, a feeding plan, and repeated communications and monitoring.^{145,281} Owners must recognize that their cat is obese and understand associated health risks. Before instituting a weight loss program, the veterinarian should perform a thorough physical examination and obtain a minimum database (complete blood count, chemistry panel, urinalysis) to detect concurrent diseases. Then a feeding plan should be instituted. The veterinarian should first set the amount of calories to be fed on the basis of known or estimated energy requirements (see Chapter 15). Calculate the RER:

$$\text{RER (kcal/day)} = (\text{Body weight}_{\text{kg}})^{0.75} \times 70$$

or

$$\text{RER (kcal/day)} = (\text{Body weight}_{\text{kg}} \times 30) + 70$$

This number is multiplied by a factor of 0.8 to induce a weight loss of 1% to 2% body weight per week. The veterinarian should compare this estimated energy requirement with current caloric intake because some animals require further restriction to induce weight loss.²⁶⁸ The calculations for an 8-kg cat with an ideal body weight of 5 kg would be as follows:

$$(8 \text{ kg} \times 30) + 70 = 310 \times 0.8 = 248 \text{ kcal/day}$$

Achieving a safe weight loss of 3 kg will take 5 to 9 months.

The veterinarian should choose a commercial diet, as previously described, and recommend that it be fed to meet the energy requirements estimated to induce weight loss. The veterinarian should eliminate or account for additional food and treats in the caloric intake, which should be less than 5% of total daily caloric intake. Some animals tolerate an abrupt change in diet with little problem, although some appear to have fewer gastrointestinal issues if food is gradually changed over a 7- to 10-day period. A new diet may be readily accepted by some cats, but patience will be required with others (Box 18-5).

BOX 18-5**How to Change a Cat's Diet**

A transition to a new diet should be performed slowly, especially in cats that have become accustomed to one type or flavor of food. It is easiest to transition cats to a new food that is similar in texture and shape to the old food. Avoid changing a cat's diet when it is stressed by pain, illness, or separation from the owner (e.g., while hospitalized or boarding). Wait until the cat's condition is improved, it is eating normally, and it is at home before switching to a new diet. Patience is a virtue when changing diets; it may take 1 or 2 months for a successful transition in some cases. Educating owners about realistic expectations can help improve compliance. Monitor the cat's body weight during diet transitions, and intervene by returning to the old diet for a few weeks if weight loss greater than 10% occurs.

1. Meal feeding may make the transition to a new diet easier than *ad libitum* feeding because the cat is more likely to be hungry at mealtime. The transition to meal feeding can be made by leaving food out for 1 hour two to three times per day. It is often easiest to start this during the time of day when the owner is normally away from home and cannot be tempted to feed the cat off schedule.
2. Offer the new food along with the old food, rather than abruptly discontinuing the old food. Ideally, both foods should be in the same type of familiar bowl. It may be necessary to offer the new food for several days to a week or even longer before the cat will try it. Once the cat starts consuming the new food, decrease the amount of the old food offered by a small amount each day, with the aim of transitioning totally to the new diet over a 1- to 2-week period.
3. Another method of introducing a new diet involves mixing the old and new foods together. For the first few days, the cat is offered a mix of 75% current food and 25% new food. Then the ratio is changed to 50:50 for the next few days. By the end of the first week, it may be possible to offer 25% current diet and 75% new diet. The amount of the new food is increased thereafter until the cat is consuming 100% of the intended diet.
4. Cats must be exposed to both the smell and the taste of a new food to overcome neophobia. If the new diet is a canned food, it may be helpful to smear a small amount on a front paw to encourage the cat to lick and taste the food.
5. Enhancing the smell and flavor of the new food can be accomplished by warming it slightly or adding small quantities (approximately 1 tablespoon) of tuna or clam juice or low-salt chicken broth.



FIGURE 18-2 Food puzzle to encourage energy expenditure by a cat to acquire food. (Courtesy Steve Dale.)

The goal of weight reduction is to reduce the cat's excess adipose tissue; however, loss of lean muscle mass occurs as well.⁹⁸ Feeding a high-protein diet is associated with less lean muscle loss.²⁶⁷ Additionally, increased dietary fiber is associated with decreased protein digestibility⁷⁵; therefore high-fiber diets are formulated to account for this. It is important that owners ensure that the obese cat continues to eat because of the risk of hepatic lipidosis. L-Carnitine (250 to 500 mg/day, by mouth) has been shown to be beneficial in preventing hepatic lipidosis with weight loss in obese cats.^{24,48} Diets should contain more than 500 ppm.

Although meal feeding is associated with more consistent weight loss, this may not be possible depending on the cat. Providing a measured amount of food during a 24-hour period achieves the same goal. Using food puzzles or hiding food in various locations both provides a more stimulating environment and encourages energy expenditure (Figures 18-2 and 18-3). When feeding dry diets, some owners find using pre-weighed portions of food more acceptable than measuring the amount of food daily in a cup.²³ In multicat households the owner should strive to keep the obese cat from eating food provided for nonobese cats. This can be accomplished by separating the cats and limiting the time for meal consumption. Another strategy is to provide food for the nonobese cats in an area that the obese cat cannot enter (e.g., a box with a hole that only the nonobese cats can fit through). Many owners have become accustomed to using food and treats to enhance the bond with their cat and mistakenly believe that eating is a social event for cats, as it is for humans. In addition, owners often mistake any vocalization as a cry for food. Teaching owners to interact with their cats through play or training sessions may be an important part of the program.



FIGURE 18-3 Placing small amounts of food in different compartments of an egg carton encourages cats to expend energy to acquire food. (Courtesy Steve Dale.)

Environmental enrichment for indoor cats can be an integral part of a weight loss program.

Communication and monitoring are important. The cat should be weighed every other week and the body weight charted. Owners should be aware that it may be necessary to adjust the amount of food, especially given that cats often lose weight steadily at the outset of the diet and then plateau. Many obese cats will require up to 12 months for safe weight loss. Many owners find that the cat's learned hunger behaviors (e.g., vocalizing, attention seeking) increase in frequency and intensity once a weight loss plan is in place, and some are unable to cope with this. It is important for the owner to have realistic expectations and to understand and anticipate some of the problems that may arise.

The veterinarian should use positive reinforcement with the client and reward successes. Charting the cat's weight loss on a graph will make progress more apparent. A photograph of the cat can be taken before and after weight loss is achieved and then displayed in the waiting room. Some clients are motivated by certificates of achievement. Once the target body weight has been achieved, the food intake should be adjusted to maintain that weight. It is important that the owner does not revert to old habits, such as letting the cat eat *ad libitum*, feeding it treats, and not ensuring that it gets exercise. Owners must understand that long-term portion control will be necessary.

Prevention of obesity is easier than treating obesity in cats. The veterinarian should teach owners how to keep their cat's body condition from lean to optimally conditioned during growth.^{9,19,111,120,182} It will be necessary to adjust food intake after neutering because gonadectomy reduces energy requirements, although food intake increases within weeks of surgery. At every veterinary

visit, a cat's body weight and BCS should be recorded in the medical record.^{111,129,233}

ENDOCRINOLOGIC DISEASES: DIABETES MELLITUS

Diabetes mellitus is the most common endocrine disease in cats. It may be insulin-dependent diabetes mellitus (IDDM), in which absolute insulin deficiency occurs, or non-insulin-dependent diabetes mellitus (NIDDM), in which insulin antagonism occurs; between 50% and 70% of cats with newly diagnosed diabetes mellitus have NIDDM. The goals of managing a cat with diabetes mellitus include achieving and maintaining optimal body condition and maintaining euglycemia. Obese cats with NIDDM may become nondiabetic with weight loss and dietary management.^{20,85,135,231} For more information on diabetes mellitus, see Chapter 24.

Animal Factors

Insulin is a major anabolic hormone involved with energy, protein, carbohydrate, and lipid metabolism. With insulin deficiency or insulin antagonism, anabolic metabolic pathways are disrupted, resulting in polyuria/polydipsia, polyphagia, weight loss, muscle mass loss, decreased body condition, and clinical signs of ketoacidosis with progression of IDDM (e.g., vomiting, anorexia, seizures). Cats with NIDDM are typically obese and do not generally develop ketoacidosis. Risk factors identified for NIDDM in cats include indoor confinement and decreased physical activity, likely resulting in obesity and insulin resistance; type of food consumed is not necessarily a risk.^{223,248}

Dietary Factors

Dietary management of cats with diabetes mellitus depends in part on whether IDDM or NIDDM is present. For cats with IDDM, timing of meals with insulin administration is advantageous; however, some cats with diabetes mellitus eat small meals even when fed *ad libitum*.¹⁸⁰ Diets that are higher in fiber may increase insulin sensitivity and blunt postprandial hyperglycemia.^{135,203} In cats that are underconditioned as a result of unregulated IDDM, feeding a calorically dense diet to increase body weight and condition may be necessary while regulating the IDDM with insulin.

Because cats with NIDDM are typically obese, weight loss is an important component of management. Many cats can go into diabetic remission with a combination of weight loss and insulin treatment¹⁷⁸ or weight loss alone. Traditional diabetic cat diets have been fortified with fiber to reduce postprandial glucose absorption and control weight.^{135,203} Many cats respond to being fed

BOX 18-6**Dietary Recommendations for Cats with Diabetes Mellitus****Increased-Fiber/High-Carbohydrate Diets:**

1. Carbohydrates: <40% on a dry matter basis. Avoid simple sugars and starch.
2. Fiber: 7% to 18% on a dry matter basis
3. Fat: <15% to 17% on a dry matter basis
4. Protein: 30% to 55% on a dry matter basis
5. Food form: Avoid semi-moist foods

Increased-Protein/Low-Carbohydrate Diets:

1. Carbohydrates: <20% on a dry matter basis. Avoid simple sugars and starch.
2. Fiber: Usually <5% on a dry matter basis
3. Fat: 12% to 25% on a dry matter basis
4. Protein: 30% to 55% on a dry matter basis
5. Food form: Avoid semi-moist foods

low-carbohydrate, high-protein diets. In studies so far, low-carbohydrate, high-protein diets are associated with improved remission rates compared with higher-fiber diets (68% versus 41%) and maintain more lean body mass during weight loss.* However, in cats that do not go into remission and require long-term therapy, there appears to be little difference between the diets. In addition, some cats respond better to high-fiber diets, and weight control may be easier with a less calorically dense food. Canned food is preferred in diabetic cats to maintain hydration, lower carbohydrate content, and improve satiety. Dietary recommendations for cats with diabetes mellitus are summarized in Box 18-6.²⁷⁹

The following supplements have been suggested for the management of cats with diabetes mellitus, although they are for the most part unproven:

- Carnitine (250 to 500 mg per day, orally) is important for the breakdown of long-chain fatty acids. By facilitating energy utilization of fats, carnitine protects against muscle catabolism during weight loss. Carnitine has also been shown to suppress ketogenesis and acidosis in starving dogs and protect liver function in fasting cats.^{24,48,124}
- Chromium is thought to increase insulin receptor numbers and activity. There are no studies in cats with diabetes, only in healthy cats.⁶
- Vanadium is thought to have insulin-like activity. One study showed vanadium lowered fructosamine, insulin requirements, and clinical signs in diabetic

cats; however, vomiting and anorexia were significant side effects.¹⁷⁹

- The role of taurine in diabetes is still controversial. Taurine is thought to exert antioxidant and antiinflammatory properties that decrease the incidence of diabetic complications such as neuropathy, retinopathy, and cardiovascular disease. Little research has evaluated taurine for use in diabetic cats.
- Supplementation with omega fatty acids in human studies has shown improved lipid metabolism and increased glycolysis in cells; however, they have not been evaluated in cats with diabetes mellitus.

Feeding Factors

The goal is to achieve and maintain optimal body condition and body weight. For cats with IDDM, dietary intake should be matched with insulin administration. For cats with NIDDM, body weight should be decreased from obese body condition to optimal body condition.

**ENDOCRINOLOGIC DISEASES:
HYPERTHYROIDISM****Animal Factors**

Hyperthyroidism is a clinical condition associated with excessive production and secretion of thyroxine (T_4). Most cats are older, with an average age at diagnosis of 13 years. Clinical signs associated with hyperthyroidism are usually polyphagia with weight loss, loss of muscle mass, polyuria/polydipsia, and hyperactivity; hyperthyroidism is also associated with cardiomyopathy. Because hyperthyroidism occurs in older cats, it may be associated with other diseases, most commonly chronic kidney insufficiency, which may become unmasked when the hyperthyroidism is treated. For more information on hyperthyroidism, see Chapter 24.

Dietary Factors

Most cats with hyperthyroidism are underweight and underconditioned; therefore feeding a calorically dense diet may be helpful in restoring body condition and weight. Increasing the fat content of the diet increases the caloric content. Underweight cats should receive a diet containing higher levels of protein; however, caution is necessary because of the association of renal disease with hyperthyroidism. Concentrations of blood urea nitrogen and creatinine should be monitored. If renal azotemia develops with treatment of hyperthyroidism, then dietary protein should be restricted (see the section on renal disease). Nutritional recommendations for feeding underweight cats with hyperthyroidism are summarized in Box 18-7.²⁷⁹

*References 20, 85, 135, 185, 200, 260.

BOX 18-7**Dietary Recommendations for Feeding Underweight Cats with Hyperthyroidism**

1. Calories: Feed to adult maintenance requirements at estimated or known optimal weight; typically, 1.2 × resting energy requirement
2. Fat: 15% to 25% on a dry matter basis
3. Protein: 30% to 55% on a dry matter basis unless renal disease is present or unmasked with treatment of hyperthyroidism
4. Fiber: <5% on a dry matter basis

Nutritional factors have been implicated in the pathogenesis of hyperthyroidism, although the etiopathogenesis is not known. Epidemiologic studies have identified consumption of commercial canned foods, especially fish or liver and giblets, as a risk for development of hyperthyroidism, which suggests that a goitrogenic compound may be present in the diet.^{130,181,207,270} However, no specific goitrogenic factor has been identified. Iodine is one potential dietary goitrogen; however, most commercially prepared cat foods contain adequate amounts of iodine. It is important to note that such studies show association but not necessarily a cause-and-effect relationship. Additional non-nutritional risk factors have been identified, including use of cat litter; being an indoor cat; sleeping on the floor; presence of dental disease; presence of a smoker in the house; use of flea products; and exposure to herbicides, pesticides, or plant pesticides.^{130,181,207,270}

Feeding Factors

Cats with untreated hyperthyroidism are often ravenous, although some may show hyporexia (so-called “apathetic hyperthyroidism”), and they often vomit. With treatment appetite often decreases; therefore clients must ensure adequate dietary intake during treatment. Additionally, medical therapy with methimazole or unmasking of renal disease with therapy may occur, resulting in hyporexia or anorexia.

**MUSCULOSKELETAL DISEASES:
OSTEOARTHRITIS**

Osteoarthritis (OA) has multiple etiologies and is characterized by pathologic changes of synovial or diarthrodial joints that are accompanied by pain and disability. Although the prevalence of OA in cats is unknown, radiographic evidence was found in 63 of 292 cats (22%) in one study.¹⁰¹ Clinical signs of OA in cats include decreased activity, reluctance to jump or climb stairs,

decreased grooming, lameness, inappropriate elimination, decreased appetite, and lethargy.¹⁸ For more information on osteoarthritis, see Chapter 26.

Animal Factors

OA occurs more commonly in cats older than 10 years of age. In one study of cats older than 12 years that were examined for reasons other than lameness, 90% of radiographs taken demonstrated OA.¹⁰⁹ Overweight cats are approximately 3 times more likely to present for lameness not associated with cat bite abscess.²⁴¹ Obesity may cause excessive forces on joints and articular cartilage, resulting in inactivity and further weight gain; however, obesity is a proinflammatory condition.^{38,69,105,282} Therefore obesity may result not only in abnormal mechanical forces on joints but also in production of adipokines, and upregulation of inflammatory pathways associated with obesity may promote joint inflammation and progression of OA.²²²

Dietary Factors

There is a paucity of information concerning nutritional management of OA in cats. Weight loss is an important component of nutritionally managing OA in cats (see the section on obesity). In one study consumption of a diet containing high levels of n3 fatty acids (eicosapentaenoic and docosahexaenoic acid) and supplemented with green-lipped mussel extract and glucosamine/chondroitin sulfate improved activity in cats with OA.¹⁴⁹ There are no studies evaluating supplementation with n3 fatty acids, glucosamine/chondroitin sulfate, antioxidants, or nutraceuticals in cats with OA. As mentioned, n3 fatty acids may alter inflammation through production of odd-numbered cytokines.^{15,38,246} Antioxidants scavenge free radicals that are increased with OA and may be beneficial.^{17,38} Chondromodulating agents such as chondroitin sulfate and glucosamine may slow progression or alter processes involved with OA, including stimulating cartilage matrix synthesis, inhibiting catabolic enzymes, and increasing fluidity of synovial fluid.^{17,38}

Feeding Factors

Obesity should be managed if present. This may require changing from *ad libitum* feeding to meal feeding or changing the diet.

ONCOLOGY

Cancer is among the most common causes of non-accidental death in cats. Nutritional management of cats with cancer has several goals. Nutritional support can

reduce or prevent toxicosis associated with cancer therapy (medical, surgical, radiation, or combination) and ameliorate presumed alterations in metabolism associated with cancer; possibly, specific nutrients can be used to treat cancer directly or indirectly.²⁴⁰ There are four stages of metabolic alterations that may occur with cancer:

1. Phase 1 is a preclinical phase with no obvious clinical signs; metabolic changes include hyperlactatemia, hyperinsulinemia, and altered amino acid profiles.
2. Phase 2 is associated with early clinical signs, such as anorexia, lethargy, and weight loss; metabolic changes are similar to those of phase 1.
3. Phase 3 is associated with advanced clinical signs, such as cachexia, anorexia, lethargy, and increased morbidity associated with cancer treatment; metabolic changes are more profound than in phases 1 and 2.
4. Phase 4 is recovery and remission; metabolic changes usually persist.²⁴⁰

Cancer may cause alterations in metabolism.^{206,276} Abnormalities in carbohydrate metabolism include glucose intolerance, insulin resistance, delayed glucose clearance, abnormal insulin secretion, increased glucose turnover, increased gluconeogenesis, hyperlactatemia, and increased Cori cycle activity (the pathway where lactic acid is recycled). Abnormalities of fat metabolism include excessive depletion of body fat relative to protein loss, decreased total body lipid content, increased lipolysis, decreased lipogenesis, hyperlipidemia, increased free fatty acid and glycerol turnover rates, failure of glucose to suppress free fatty acid oxidation, and decreased serum lipoprotein lipase activity. Abnormalities in protein metabolism include increased whole-body protein turnover, increased liver protein fractional synthetic rates, reduced muscle fractional synthetic rates, decreased incorporation of amino acids into muscle, increased hepatic protein synthesis, muscle breakdown, and decreased plasma branched-chain amino acids. Depending on the location and distribution of the cancer, malnutrition may result from interference with eating and digestion (Figure 18-4).

Treatment of cancer also induces problems. Surgery increases nutritional requirements, especially for energy and protein, and may impair food intake or result in malassimilation. Chemotherapy may induce anorexia, vomiting, mucositis, infections, and organ injury. Radiation therapy may cause mucositis or dermatitis. Complications may increase if multimodality therapy is used. For more information on feline oncology, see Chapter 28.

Animal Factors

Depending on the type of cancer, whether it is disseminated, and what treatments are undertaken, a cat with

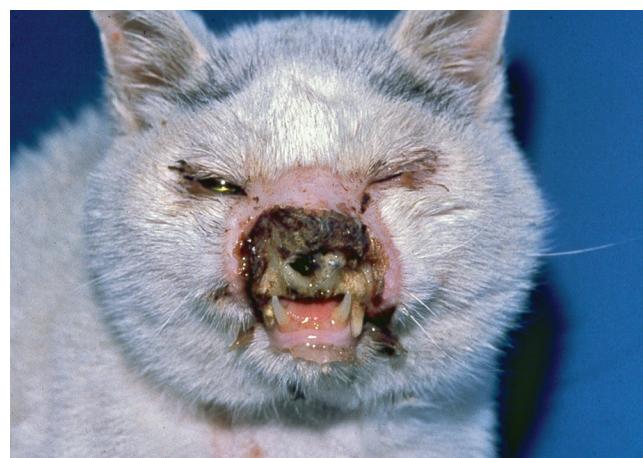


FIGURE 18-4 A 15-year-old castrated male domestic shorthair cat with inability to eat because of facial squamous cell carcinoma.

BOX 18-8

Dietary Recommendations for Cats with Cancer

1. Carbohydrates: <25% on a dry matter basis
2. Fat: 25% to 40% on a dry matter basis
3. n3 fatty acids: >5% on a dry matter basis with an n6:n3 fatty acid ratio of approximately 1:1
4. Protein: 40% to 50% on a dry matter basis
5. Arginine: >2% on a dry matter basis

cancer may be optimally conditioned or extremely underconditioned; however, obesity may be associated with certain cancers.²⁸² In one study of feline cancer patients, fat mass was reduced in 60% and muscle mass was reduced in 91%; cats with suboptimal body condition had a median survival time that was approximately 5 times less than cats that were at least optimally conditioned.¹⁰

Dietary Factors

There are no studies on nutritional requirements of cats with cancer. Information on nutritional factors for cats with cancer is derived from information generated primarily from dogs with lymphoma.²⁰⁵ Cats with cancer may not utilize carbohydrates efficiently but may utilize fat preferentially; therefore a low-carbohydrate, high-fat diet may be beneficial. Protein requirements may be higher to help maintain lean muscle mass. Other nutrients that may be of benefit include n3 fatty acids, antioxidants, and arginine. Nutritional recommendations for cats with cancer are summarized in Box 18-8.²⁴⁰

Preventing obesity decreases the risk of cancer in general and of certain types of cancer (e.g., mammary cancer).^{117,282}

Feeding Factors

Because many animals have decreased appetite or anorexia, a good dietary history is important. Getting an animal to eat may involve changing food type, texture, or feeding patterns, or it may involve stimulating appetite. Nutritional support is individualized. If possible, the cat should be fed enterally; however, parenteral nutrition may be required. Goals of nutrition include maintaining body condition and lean muscle mass and minimizing nutritional support of the actual cancer.¹⁰

OPHTHALMOLOGIC DISEASES: HERPESVIRUS INFECTION

Nutritional association with ophthalmologic diseases in cats is minimal, other than those associated with nutrient deficiencies (see Chapter 17); however, lysine and probiotics are associated with management of cats with herpesvirus infection.^{68,148,175} For more information on herpesvirus infections, see Chapter 29.

Animal Factors

Cats with active herpesvirus infection usually present for upper respiratory signs and punctate keratitis.^{97,172,261} Many cats infected with herpesvirus have latent infections with occasional flare-ups, particularly of ocular disease.²³⁰ During active infections, cats may be anorexic.

Dietary Factors

Lysine, an amino acid, has been shown to be beneficial in managing cats with herpesvirus, although data are contradictory.^{68,173-175,224,252} An oral dose of 400 to 500 mg of L-lysine every 12 to 24 hours has been recommended.^{174,252} Two studies have been published evaluating L-lysine addition to diets at 5% on a dry matter basis; neither showed benefit.^{68,175} There is one small study evaluating a probiotic (*Enterococcus faecium* SF68) in cats with herpesvirus infection; data were inconclusive, although some cats had lessened morbidity.¹⁴⁸

Feeding Factors

Dietary change is not usually necessary, although cats that are anorexic with active herpesvirus infection may require a calorically dense and palatable diet. Supplemental lysine may be beneficial in some cats.



FIGURE 18-5 Chylous pleural effusion in a 13-year-old castrated male domestic longhair cat with idiopathic chylothorax.

PULMONARY AND THORACIC MEDICINE: CHYLOTHORAX

Chylothorax refers to the accumulation of high-fat fluid in the thoracic cavity (Figure 18-5). It may occur as an idiopathic disease or in association with cardiac or intra-thoracic disease.* Management of the underlying condition, if identified, may be all that is required to manage chylothorax in a cat. In some cats, however, no identifiable cause is found, and the condition is termed *idiopathic chylothorax*.^{21,22,80} Nutritional management of cats with idiopathic chylothorax is directed at decreasing lymphatic flow from the intestinal tract and decreasing accumulation of chyle in the thoracic cavity. For more information on chylothorax, see Chapter 30.

Animal Factors

Cats with idiopathic chylothorax are typically middle-aged.^{21,81} Depending on length of duration of chylothorax, the cat may or may not be in optimal body condition.

Dietary Factors

Dietary fat restriction may decrease lymphatic drainage and amount of chyle accumulation in the thoracic cavity. Diets should contain less than 12% to 15% fat on a dry matter basis. Rutin is a citrus flavonoid glycoside found in buckwheat that has been shown to benefit some cats with idiopathic chylothorax and is dosed orally at every 8 hours until resolution of chylothorax; in some cases, indefinitely.^{102,139,264} Suggested mechanisms of action for rutin include reduced leakage of lymph from lymphatic vessels, increased protein removal by

*References 21, 78, 81, 82, 113, 118, 157, 189, 191, 271.

lymphatics, increased phagocytosis by stimulation of macrophages, increased recruitment of macrophages in tissues, and increased proteolysis and removal of protein from tissues.²⁶⁴

Feeding Factors

Cats with idiopathic chylothorax require a dietary change to a low-fat diet, unless the cat is severely under-conditioned. Feeding smaller amounts may decrease chylous effusion. Rutin is beneficial in some cats.

TOXICOLOGY

Foods, whether for human or animal consumption, are supposed to provide nutrients for maintenance of health; however, sometimes the food may be the source of the problem. There are basically two types of toxicities that can occur with pet foods, as with human foods: infectious agents and toxins. Additional adverse food reactions may occur as a result of immunologic and non-immunologic reactions, such as consumption of an incomplete or unbalanced food or immunologic reaction to a food-borne allergen. Aflatoxin involved in a recent pet food recall was primarily hepatotoxic and often fatal; however, not all dogs exposed to aflatoxin develop disease.²⁵¹ Potentially pathogenic and zoonotic bacteria have been identified in dog food, including *Escherichia coli* O157:H7 (enterotoxigenic *E. coli*),⁸⁸ *Salmonella*,²⁵³ *Yersinia enterocolitica*, *Campylobacter*, *Clostridium*, and *Listeria*.²⁸⁰ Additionally, parasites may be transmitted in food, especially uncooked food, such as *Echinococcus*, *Taenia*, *Toxocara*, *Toxoplasma*, *Trichinella*, and *Neospora*. The Centers for Disease Control and Prevention (CDC) (<http://www.cdc.gov>) provides information on infectious disease-related food problems (<http://www.cdc.gov/ncidod/diseases/food/index.htm>). Toxins may be inadvertently or intentionally added to a pet food, or a pet may consume them independent of its diet. Examples of ingested toxins include onions,²³² lilies,^{32,107,146,238,254} and melamine-cyanuric acid.^{55,155,220,275}

Recognizing food-associated illness can be difficult because cases often present sporadically with no apparent connection. Recognizing clusters of cases geographically (e.g., regionally) or during the same time period (e.g., animals in the same household) is important. The veterinarian should take a good diet history from the owners. Introduction of a new food or a new bag of food, poor palatability, or acceptance of the food by the pet(s), and pets eating the same food whether in the same household or different households may provide clues to problems with diet. The veterinarian should keep in mind that animals may present with similar clinical signs and histories but consume different diets and snacks or treats. The veterinarian should discuss cases

BOX 18-9

Reporting Food-Related Illnesses and Adverse Effects

1. Contact the manufacturer. A representative should be willing to listen and take information as well as answer questions regarding other complaints, if any.
2. In the United States, contact the Food and Drug Administration (FDA): <http://www.fda.gov/AnimalVeterinary/default.htm>. Pet food safety issues can be reported to the Safety Reporting Portal: <https://www.safetyreporting.hhs.gov>
3. Contact the American Veterinary Medical Association (AVMA) to report adverse events with drugs, vaccines, and pet food: http://www.avma.org/animal_health/reporting_adverse_events.asp
4. To report an adverse event associated with pet food (or other animal feed), contact your state FDA consumer complaint coordinator(s). Contact information can be found at <http://www.fda.gov/Safety/ReportaProblem/ConsumerComplaintCoordinators/default.htm>. When reporting, include as much information as possible, including the specific product name, lot numbers, veterinarian's report and diagnosis, and any other pertinent information.

with colleagues because they may be having similar experiences. Guidelines for reporting food-related illnesses and adverse effects are summarized in Box 18-9.

It is important to gather as much information as possible and to save as much as food as possible. The veterinarian should document the product name, type of food, manufacturer/distributer information, and date code or best-by code. A copy of the packaging should be retained if possible. If the owner has a copy of the purchase receipt, this may be helpful. The veterinarian should retain samples of the food, keeping at least four cans or pouches of canned or semi-moist food and 1 kg of dry food. The veterinarian should not send all of the samples for analysis but should instead keep or have the owner keep some. The owner should be asked to document consumption of the food by pet(s) with as much detail as he or she can recall. Thorough records, including signalment, clinical signs, and test results, should be kept. If a pet dies, the veterinarian should perform a necropsy or have a necropsy performed, making sure to tell the diagnostic laboratory performing the necropsy that toxicity is suspected. Tissue and fluid samples should be taken, if possible. All communication with the manufacturer and with the Food and Drug Administration (FDA) and the American Veterinary Medical Association (AVMA) should be documented. If other pets may have been exposed, they should also be tested.

Pet food toxicities occur commonly, although less so than in human beings. The CDC estimates that 76 million Americans get sick, more than 300,000 are hospitalized, and 5000 die from food-borne illnesses annually. After eating contaminated food, people can develop anything from a short, mild illness, often mistakenly referred to as "food poisoning," to a life-threatening illness. The veterinarian should be suspicious of a food-related disease, especially if trends in households, food consumed, or clusters of cases geographically are evident. Food and information should be saved as described. Owners should be instructed to discontinue feeding the suspected food immediately. As a short-term solution, owners can give their pets home-prepared meals, if they wish. To minimize risk of food poisoning in pets, owners should do the following:

- Prevent pets from eating garbage or carrion.
- Cover and refrigerate unused portions of wet food.
- Refrain from feeding pets foods that have a suspicious appearance or odor.
- Use stainless steel bowls and utensils, and clean them well.
- Store dry foods in a cool, dry location free of pests.

The Veterinary Information Network (<http://www.vin.com>) has a list of frequently asked questions

and information about homemade pet foods. It was developed during the pet food crisis triggered by the melamine–cyanuric acid recall (Box 18-10).

CRITICAL CARE

Critical illnesses may be caused by factors such as trauma and acute or chronic disease and often result in inappetence. Nutritional support is indicated in animals that have not eaten for 3 to 5 days, have poor body condition, or have increased needs for nutrition.^{49,50,212} A few days of food deprivation is not detrimental to healthy cats; however, it may be detrimental to a sick cat. Short periods of not eating are not a problem because the body is able to utilize endogenous energy substrates such as glycogen. During prolonged food deprivation, the body shifts to a hypometabolic state to conserve structural and functional proteins as much as possible; thus glucose and fatty acids become the major energy sources. During periods of food deprivation associated with stress and illness, however, the body cannot utilize fatty acids or glucose efficiently. Therefore amino acids are mobilized and used for gluconeogenesis, for DNA and RNA synthesis, and for acute phase protein production. Malnutrition occurs rapidly.^{123,190,193,217} It is important to remember

BOX 18-10

Generic Adult Dog and Cat Homemade Food Recipe*

This recipe should be fed for not more than 2 months. Clinicians are advised to set up a consultation with the client at the end of this period to revisit feeding requirements and consider either re-instituting commercial food products or consulting with a clinical nutritionist.

This diet is adequate for healthy dogs and cats over 6 months of age:

- 1 pound fresh boneless skinless chicken breast
- 2½ cups cooked white rice
- 1 tablespoon safflower oil
- ¼ teaspoon Morton Lite Salt (Morton International, Inc.)
- ¼ teaspoon iodinated salt
- 3 grams of calcium carbonate without vitamin D: Use Tums Regular Strength (GlaxoSmithKline), 6 tablets (each contains 500 mg calcium carbonate, which provides 200 mg of elemental calcium). Other calcium preparations contain different amounts of calcium. For example, Tums 500 contains 1200 mg of calcium carbonate, providing 500 mg of elemental calcium.
- 1 Centrum (Wyeth Consumer Healthcare) adult multivitamin–mineral supplement (do not use Silver, Ultra Women's, Ultra Men's, or any other version)

- ¼ teaspoons taurine powder (or 500-mg tablet) (Taurine is optional for dogs but essential for cats.)

Sauté chopped chicken breast in oil until thoroughly cooked. Add rice and salt. Grind Tums (calcium carbonate), multivitamin–mineral tablet, and taurine supplement together. Add to cooled mixture. Store in refrigerator. Larger batches may be prepared in advance and stored in the freezer.

Nutritional Profile

- 40% protein (dry matter basis [DMB])
- 12% fat DMB
- 6% calcium DMB
- 4.3% phosphorus
- 1.4:1 calcium :phosphorus
- Calories: 1046 kcal per batch or 1.12 kcal/g
- Batch size: 932 g

To feed, calculate caloric needs, and divide into two or more daily feedings. One recipe batch should provide adequate intake for a 40- to 45-pound dog for 1 day. Adjust intake to maintain ideal body weight.

*Courtesy Veterinary Information Network, <http://www.vin.com>.

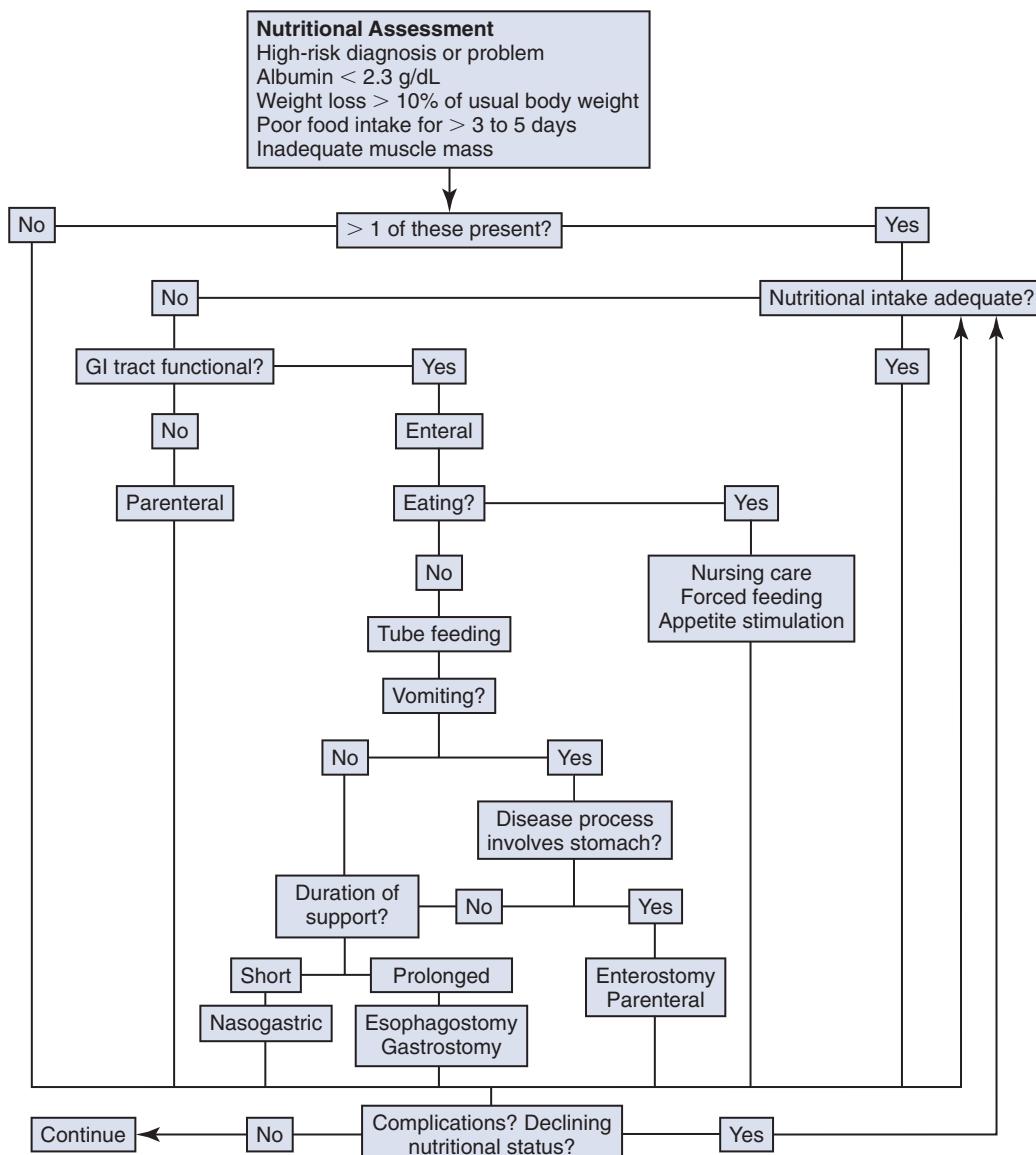


FIGURE 18-6 Algorithm for nutritional support.

that administration of parenteral fluids containing 5% dextrose does not provide nutritional support as 1 mL of 5% dextrose provides 0.17 kcal. For a 5-kg cat, it would require 1294 mL of 5% dextrose per day to meet RERs.

Goals of nutritional support for critical care patients are as follows:

1. To minimize metabolic derangements
 - Maintain hydration
 - Attenuate acid-base disorders
 - Attenuate electrolyte disturbances
 - Provide disease-specific nutrients
2. To provide nutrients to facilitate recovery
 - Suppress hypermetabolic response
 - Restore or reverse protein catabolism and negative nitrogen balance

- Maintain gastrointestinal tract integrity and function
- Optimize immune function

3. To maintain lean body mass and body weight
4. To avoid complications associated with refeeding

There are two main “Golden Rules” of nutrition: (1) If the gut works, use it; and (2) keep it simple (Figure 18-6). Nutrition should be provided enterally, when possible, because it is the easiest, safest, and most physiologic route. As much of the gastrointestinal tract should be used as possible. Not only does the enteral route provide nutrients for the whole animal; it also provides nutrition to enterocytes. Maintenance of enterocyte health is important to prevent bacterial translocation from the gastrointestinal tract into the systemic circulation or lymphatics and to facilitate recovery by the

animal.⁶² There are some potential disadvantages to providing nutritional enterally: Some patients cannot tolerate enteral feeding, some patients cannot assimilate nutrients when provided enterally, and there is a risk of aspiration pneumonia if regurgitation or vomiting occurs. Parenteral nutrition is limited to comatose or paralyzed patients or those with severe gastrointestinal dysfunction such as intractable vomiting, malassimilation syndromes, severe pancreatitis, and peritonitis. Parenteral nutrition should be considered in animals in which provision of nutrients is not feasible using the enteral route.

Animal Factors

The first step in providing nutritional support is to determine whether nutritional support is indicated (see *Figure 18-6*). This decision is based on historical and physical examination findings. A thorough history and complete physical examination with appropriate diagnostic testing is required before initiating nutritional support. It is important to ensure that the cat is able to eat. Severe periodontal disease and oral cancer are examples of reasons that cats may be unable to eat.

The RER is estimated by the following:

$$\text{RER (kcal/day)} = (\text{body weight}_{\text{kg}})^{0.75} \times 70$$

or

$$\text{RER (kcal/day)} = (\text{body weight}_{\text{kg}} \times 30) + 70$$

In critical care, hospital, or illness-associated conditions, RER is multiplied by 1. In other words, the goal is to provide RER to most patients. Exceptions to this include cases of severe trauma, head injury, and burns, in which energy requirements are greater than RER, often being more than twice the RER.^{57,76,186,225} Protein requirements vary depending on whether enteral nutritional support or parenteral nutritional support is provided. For enteral nutritional support, adult cats should be fed 6 g of protein/body weight_{kg} per day. Cats requiring protein restriction (e.g., renal failure or hepatoencephalopathy) are fed less (3 to 5 g of protein/body weight_{kg} per day), whereas cats requiring protein supplementation (e.g., those with protein-losing nephropathy or peritonitis) are fed more (>6 g of protein/body weight_{kg} per day).^{158,159,212,229,262} For parenteral nutritional support, protein requirements are based on caloric intake. Adult cats should be provided 6 g of protein/100 kcal per day, whereas cats requiring protein restriction are provided 3 to 5 g of protein/100 kcal per day and cats requiring protein supplementation are provided more than 6 g of protein/100 kcal per day. Taurine is an essential amino acid for cats and is not provided in parenteral solutions. Water is provided by nutritional support because most anorexic cats do not drink.

Maintenance fluid requirements for adult cats are approximately 60 mL/kg per day but may be more with diseases associated with fluid loss (e.g., renal failure, diarrhea, peritonitis).¹⁸³ Finally, the veterinarian should determine whether there are other nutrients of interest such as taurine and n3 fatty acids that should be added or restricted from nutritional support.

Dietary Factors

Choice of diet depends on several factors, including provision or exclusion of disease-specific nutrients and route of nutritional support.

Feeding Factors

Nutritional support may be accomplished using the enteral or parenteral routes. The route chosen depends on the individual patient and its ability to use the gastrointestinal tract (see *Figure 18-6*).

Enteral Route

Enteral nutrition may be accomplished by several means.

NURSING CARE AND COAXING

Sometimes good nursing care is all that is necessary to stimulate an animal to eat. This may include hand feeding the cat, using a highly palatable energy-dense food, warming the food to body temperature (especially important for cats), adding water to food, providing praise when the cat eats, and feeding the cat in a stress-free environment that is away from competition. Many cats will not eat while hospitalized but will eat voluntarily at home. Although anorectic cats often require hospitalization for diagnostic and therapeutic purposes, they should be hospitalized for the least amount of time possible. When food is offered, the cat should be stroked and given vocal reassurance to encourage voluntary food consumption. Although changing the diet or mixing diets may stimulate appetite, some cats may not accept a change in diet texture. If a cat has a nasal discharge, the nares should be cleaned. Sometimes these efforts are successful, particularly if it is an acute disease that is transient; however, if anorexia persists, other means should be considered.

APPETITE STIMULANTS

Some drugs may stimulate appetite (see *Table 18-1*). They do not work all the time, but when they do work, more aggressive means of nutritional support may not be necessary. Several pharmacologic agents have been used with variable success in veterinary medicine. Mirtazapine is, in part, a serotonin antagonist that has antiemetic and appetite-stimulatory properties. Diazepam¹⁷¹ may cause sedation and should be used cautiously in cats with liver disease because it may induce hepatic



FIGURE 18-7 Administration of a bolus of a homogeneous canned food using a syringe.



FIGURE 18-8 Nutritional support using an orogastric feeding tube in a 4-year-old castrated male cat.

failure.⁴⁷ Glucocorticoids are associated with side effects such as catabolism and insulin antagonism, which limits their usefulness as appetite stimulants.¹⁹⁷ Anabolic steroids are not as effective as benzodiazepines and may be associated with hepatotoxicity when used for prolonged periods.¹¹⁰ Also, there may be a lag phase between beginning their administration and onset of appetite stimulation. Megestrol acetate⁵⁸ may induce diabetes mellitus, adrenal suppression, and mammary neoplasia in cats.^{54,119,170,197,219} None of these drugs works consistently, and none has been evaluated under controlled conditions.

FORCED FEEDING

Boluses of food in the oropharynx will stimulate swallowing. This may be accomplished by forming the canned food into a meatball shape or using a syringe. Canned food gruels or convalescent canned veterinary products may be administered through a syringe (Figure 18-7). Forced feeding is easy to perform; however, it may add additional stress to a sick animal. Furthermore, it is difficult to do for more than a few days, and most cats do not tolerate it well.

TUBE FEEDING

Enteral feeding may be done by placing a tube within the gastrointestinal tract.^{7,49,212} Such tubes include orogastric, nasoesophageal, pharyngostomy, esophagostomy, gastrostomy, and enterostomy tubes.

OROGASTRIC FEEDING TUBE

Passing a feeding tube through the mouth into the distal esophagus or stomach is technically easy to do (Figure 18-8); however, it is usually stressful to adult cats. It is often used to provide nutrition to orphaned kittens.



FIGURE 18-9 Nasoesophageal feeding tube.

NASOESOPHAGEAL FEEDING TUBE

Nasoesophageal feeding tubes are technically easy to place and can be used safely in many animals (Figure 18-9).¹ They should not be used if the patient is comatose or lacks a gag reflex because of risk of aspiration. Nasoesophageal feeding tubes probably should not be used in animals with esophageal motility disorders. These tubes may be placed without general anesthesia. Complications of nasoesophageal feeding tubes include rhinitis, dacryocystitis, esophageal reflux, vomiting, aspiration, pneumonia, inadvertent tube removal, and obstruction of the tube.

PLACEMENT²⁹

- Place 0.5 to 1 mL 0.5% proparacaine hydrochloride into one of the nasal cavities of the cat. Tilt the head up to encourage the local anesthetic to coat the nasal



FIGURE 18-10 Insertion of a red rubber feeding tube into the ventral nasal cavity of a cat for nutritional support using a nasoesophageal feeding tube.

mucosa. Repeat the application to ensure adequate local anesthesia.

- If the cat is too stressed to place the tube, induce light sedation or a light plane of anesthesia.
- In general, for cats weighing less than 3 kg, choose a 5 Fr tube. For cats above 4 kg, an 8 Fr tube can often be inserted. Polyvinyl chloride feeding tubes or red rubber catheters are best because of their flexibility. Polyurethane tubes may be preferable for long-term feedings because of their resistance to degradation.²⁴⁵ Use a tube that is radiopaque to facilitate radiographic confirmation of placement.
- Measure the length of the tube on the side of the animal from the nasal planum to the last rib. If the tube is to terminate in the stomach, mark the tube at the proximal end with tape as a butterfly. If the tube is to terminate in the thoracic esophagus, pull the tube back 1 to 5 cm and place the butterfly tape at the proximal end. Nasogastric intubation is not usually associated with complications because of the small diameter of the tube.
- Lubricate the tube with 5% lidocaine jelly before insertion. Maintain the cat's head in a normal position to avoid tracheal intubation. A guidewire may be used for small and flexible tubes.
- Insert the tube caudoventrally and medially into the nasal cavity (Figure 18-10). Flex the cat's head to facilitate passage into the oropharynx and esophagus. The tube should pass into the oropharynx and stimulate a swallowing reflex. When the cat swallows, pass the tube into the esophagus to the predetermined distance.
- Inject 3 to 5 mL of sterile saline into the tube to confirm placement in the esophagus. If a cough is elicited, remove the tube and attempt placement again. For additional confirmation, inject 6 to 12 mL

of air in the tube and auscult for borborygmus at the xiphoid. If there is still uncertainty or if the cat is under general anesthesia, a lateral thoracic radiograph gives definitive confirmation.

- Secure the correctly positioned tube to the lateral aspect of the nose with the preplaced butterfly tape and again at the zygomatic arch. This can be done with sutures. Most cats will tolerate the tube without an Elizabethan collar.
- Place a column of water in the tube and cap it when not in use, and occlude the end with an infusion cap, three-way stopcock, or hypodermic needle cap (without the needle). This prevents intake of air, reflux of esophageal contents, and occlusion of the tube by diet.

ESOPHAGOSTOMY FEEDING TUBE

Esophagostomy tubes are easy to place, and a large-bore (>12 Fr) feeding tube may be placed in most animals.* Advantages of an esophagostomy feeding tube are that there is no interference with voluntary consumption of food, and gruels may be used because of diameter of the tube. Esophagostomy tubes must be placed under heavy sedation or general anesthesia. The distal tip of the tube should terminate in the distal esophagus and not the stomach to prevent gastroesophageal reflux and esophagitis.⁵⁹ They should not be used in cats with esophageal motility disorders. Additional complications include inflammation and infection at the tube exit site and vomiting.¹⁵⁴ Vomiting may occur as a result of the underlying disorder, overly rapid administration of food, or failure to warm food to almost body temperature.

PLACEMENT²⁶⁶

- Anesthetize and intubate the cat, and position it in right lateral recumbency; shave and prep the cervical area from the base of the ear to the point of the shoulder (Figure 18-11, A).
- Insert a speculum to hold the mouth open.
- Pass the curved end of a curved Carmalt forceps through the oral cavity and into the mid to proximal esophagus.
- Palpate the curved end of the forceps, and make a 1-cm skin incision over the end (see Figures 18-11, B and C).
- Continue the incision through the subcutaneous tissue and esophageal wall, and exteriorize the curved tips of the forceps.
- Grab the distal end of the feeding tube with the curved forceps, and pull the tube through the incision into the oral cavity and out of the mouth (see Figures 18-11, D and E).

*References 60, 64, 108, 125, 128, 154, 269.

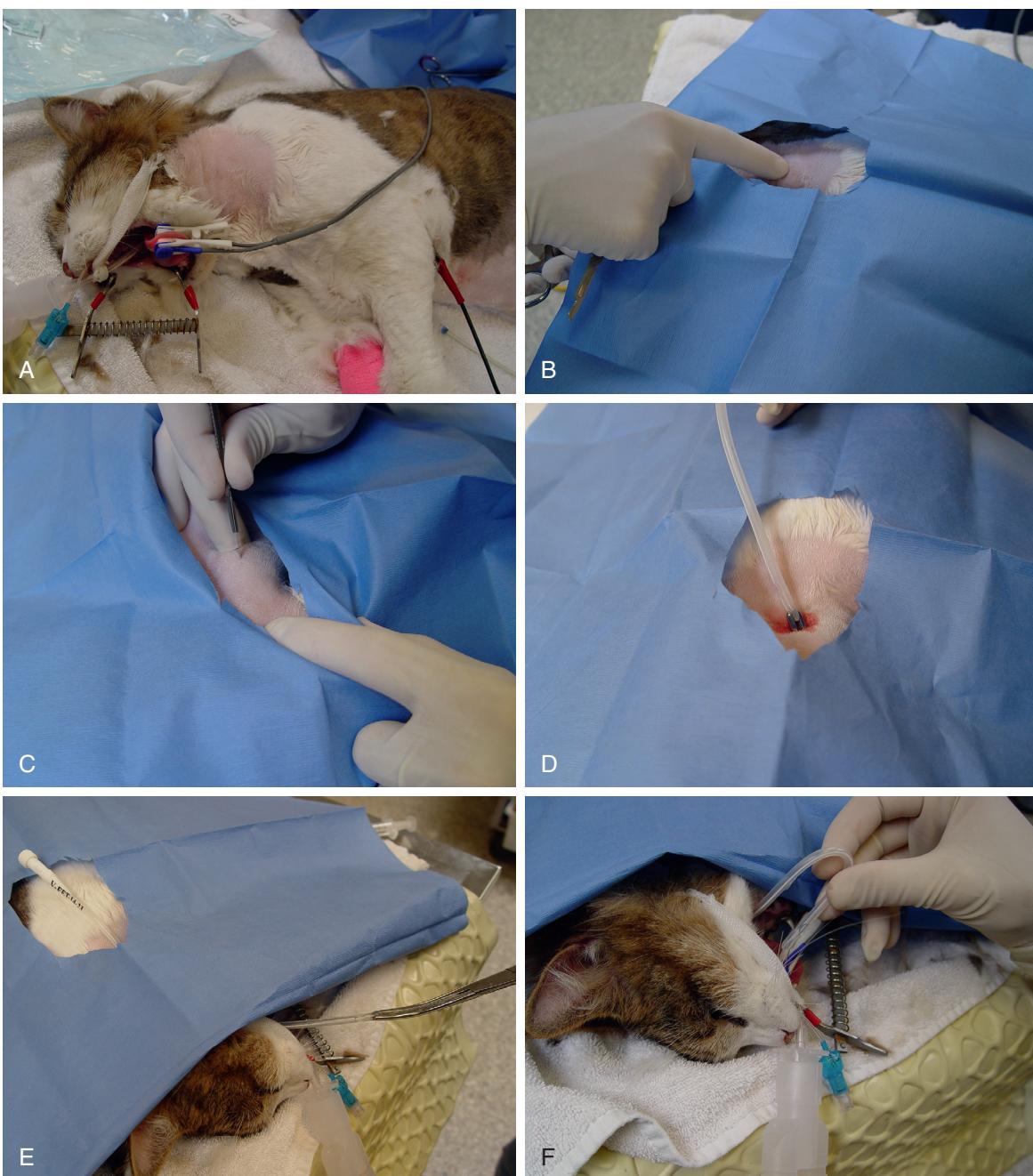


FIGURE 18-11 A, Placement of an esophagostomy feeding tube. Anesthetize and intubate the cat, and position in right lateral recumbency; shave and prep the cervical area from the base of the ear to the point of the shoulder; insert a speculum to hold the mouth open. B and C, After passing the end of a curved Carmalt forceps through the oral cavity and into the mid-esophagus, palpate the end of the forceps through the skin and make a 1-cm incision. D and E, Grab the distal end of the feeding tube with the forceps, and pull the tube through the incision into the oral cavity and out of the mouth. F, Redirect the distal end of the feeding tube into and down the esophagus by hand or using the forceps to the premeasured mark.

Continued



FIGURE 18-11, cont'd **G** and **H**, Secure the tube to the patient's neck at the exit point with a finger trap suture and bandage lightly. (From Chan DL: The inappetent hospitalised cat: clinical approach to maximising nutritional support, J Feline Med Surg 11:925, 2009.)

- Redirect the distal end of the feeding tube into and down the esophagus to the premeasured mark. The distal end is grasped as it exits the oral cavity and is inserted down the esophagus by hand or using the forceps (see [Figure 18-11, F](#)).
- Secure the tube to the patient's neck at its exit point with a finger trap friction suture (see [Figures 18-11 G and H](#)). The tube may be incorporated into a light neck bandage,
- A column of water is placed in the tube after food administration and the end closed with a three-way stopcock, infusion plug, or hypodermic needle cap (without the needle),
- When the tube is ready to be removed, no sedation is required. The sutures are removed, and the tube is gently pulled out. The stoma site is left to close by second intention.

GASTROSTOMY FEEDING TUBE

Gastrostomy feeding tubes may be placed surgically through a small laparotomy incision or at the time of abdominal surgery, or non-surgically using an endoscope (percutaneous endoscopic gastrostomy tube) or nonendoscopically.* Advantages of a gastrostomy tube are that they can be used in animals with esophageal or higher disease, a large bore feeding tube (16 to 24 French) can be used so pet food gruels may be administered, they can be used for extended periods of time (months to years), and there is no mechanical inhibition of voluntary food consumption ([Figure 18-12](#)). Cats tend to tolerate gastrostomy feeding tubes well. In addition, a low profile gastrostomy feeding tube device may be used for extended periods of time ([Figure 18-13](#)). Complications with use of gastrostomy feeding tubes include vomiting with risk of aspiration pneumonia



FIGURE 18-12 Providing nutritional support to a 6-year-old spayed female domestic shorthair cat with idiopathic hepatic lipidosis using a percutaneously endoscopically placed gastrostomy feeding tube.

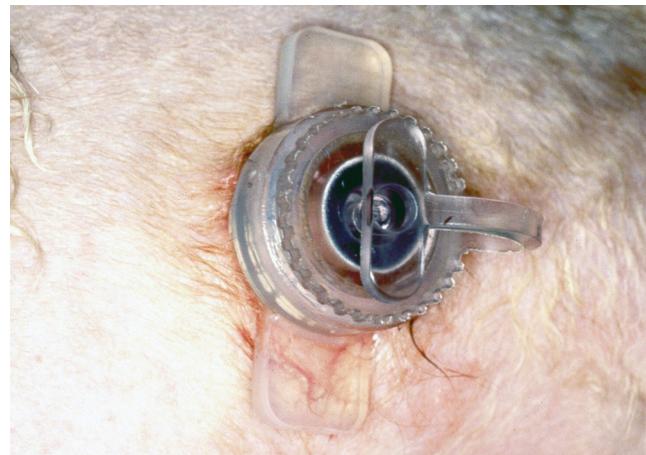


FIGURE 18-13 Low-profile gastrostomy feeding tube.

(often associated with administering cold food or with administering food too quickly), dislodgement of the tube which may result in peritonitis or cellulitis, peristomal infections, and difficulties in maintaining bandages on cats.

*References 7, 33, 90, 100, 127, 164, 192, 250, 263.

ENTEROSTOMY FEEDING TUBE

Enterostomy feeding tubes are usually 5 Fr tubes that are placed directly into the duodenum or jejunum either surgically or percutaneously using endoscopy.^{7,84,115,127,176} Placement of an enterostomy feeding tube can be done at the time of surgery; therefore careful planning is necessary to avoid a second surgery. Advantages of an enterostomy feeding tube are that they bypass the stomach and therefore can be used in animals undergoing gastric surgery or in cats with pancreatitis. Usually, liquid enteral diets are administered through a 5 Fr feeding tube using constant-rate infusion.

Selection of a diet administered through a feeding tube depends on the diameter of the feeding tube and location of its termination within the gastrointestinal tract. Liquid diets or homogenous gruels of canned foods can be administered through 5 Fr and larger feeding tubes. Gruels of canned foods can be administered through 10 Fr and larger feeding tubes. When using gruels, the veterinarian should ensure that no large pieces of food or dietary constituents are present that may clog the tube.

Begin tube feeding by administering warm water through the tube. If this is well tolerated, then divide the first day's administration of diet into 6 meals, and administer every 4 hours. If this is tolerated, increase the volume of food administered and decrease the frequency of feeding. Many cats can tolerate 2 to 3 bolus feedings daily by feeding tube. Constant-rate infusions are used for enterostomy tubes and are begun at one half the calculated administration rate for the first 6 to 12 hours and then increased to full rate if well tolerated. Ensure that the feeding tube is properly situated before feeding by infusing approximately 6 to 12 mL of warm water through the tube and observing for clinical signs of discomfort. After administration of the meal, flush the tube with 6 to 12 mL of warm water and cap the tube, leaving a column of water inside the tube; this will help prevent occlusion of the tube with food.

PARENTERAL NUTRITION

Parenteral nutrition may be used for short-term nutritional support, if enteral nutrition cannot be used or to supplement enteral food intake.* Parenteral nutrition is often considered to be "total" if all components are used and "partial" if selected components are used. In reality, parenteral nutrition in veterinary medicine is not complete and is therefore not "total." It is better to consider parenteral nutrition as either centrally administered, in which the solution is hypertonic, or peripherally administered, in which the solution is isotonic to plasma. Components of parenteral nutrition include 3.5% to 10% amino acids with or without electrolytes (protein source);

5% to 50% dextrose (carbohydrate source); 20% lipid emulsion (fat source); and vitamins, minerals, and electrolytes (Figure 18-14). Frequently, vitamins, other than B-complex vitamins (1 mL per liter of parenteral fluids), and minerals are excluded from parenteral nutrition formulations in cats.¹⁹⁸ Isotonic fluids used in parenteral nutrition are 3.5% amino acids and 5% dextrose. Lipid emulsions do not exert tonicity, although they can decrease the tonicity of the final solution. As a general rule, solutions that are above 600 mOsm/kg (approximately two times plasma osmolality) should be administered only through a centrally placed venous catheter.

Parenteral nutritional solutions must be prepared aseptically and are administered for no longer than 3 days owing to potential contamination and breakdown of the lipid emulsion. Prepared parenteral nutritional solutions may be refrigerated for up to 7 days. Venous catheters used for administration of parenteral nutritional solutions should be used only for nutritional support; they should not be used for blood sample collection or administration of medications.

CENTRALLY ADMINISTERED PARENTERAL NUTRITION

Centrally administered parenteral nutrition solutions are formulated and compounded to provide amino acids, dextrose, lipids, electrolytes, and B-complex vitamins; they may contain trace minerals and other vitamins (total admixture). They are hypertonic and must be administered through a centrally located venous catheter. Energy requirements are calculated as previously explained, and dextrose and lipid are used to meet these energy requirements; some recommend including calories provided by amino acids. The caloric content of 50% dextrose is 1.7 kcal/mL and that of 20% lipid is 2 kcal/mL. In most cases the ratio of dextrose to lipid ranges from 40:60 to 60:40, although other ratios may be used depending on the patient.^{158,159,229} Protein requirements



FIGURE 18-14 Components of parenteral nutrition, including multivitamin for infusion, lipid emulsion, dextrose, and amino acid solution.

*References 49, 51, 53, 158-160, 212, 262.

are calculated as previously described, and volume of amino acid infused is determined by the solution used; 8.5% amino acid solution provides 0.085 g/mL, and 10% amino acid solution provides 0.1 g/mL. Electrolytes are provided as a component of the amino acid solution, added as a supplement, or added as a balanced crystalloid to the parenteral nutrition solution. Vitamins are added as 1 mL of B-complex vitamins/L solution, although multivitamin and trace mineral solutions are available for addition to parenteral nutrition solutions.¹⁹⁸ Begin the infusion at $\frac{1}{2}$ of estimated rate for the first 8 to 12 hours. At that time, check concentrations of blood glucose and urea nitrogen and inspect the plasma for lipemia; if concentrations are within normal reference range and lipemia is not apparent, increase the rate to full administration and re-evaluate a biochemical panel after an additional 8 to 12 hours. Do not abruptly cease administration of centrally administered parenteral nutrition; decrease the rate by $\frac{1}{2}$ for 8 to 12 hours, and then discontinue. Complications of centrally administered parenteral nutrition are related to the catheter or solution (e.g., sepsis, thrombophlebitis, inadvertent catheter removal) or to metabolic complications (e.g., hyperglycemia or hypoglycemia, azotemia, metabolic acidosis, electrolyte imbalances).^{51,160,184,199}

PERIPHERALLY ADMINISTERED PARENTERAL NUTRITION

Peripherally administered parenteral nutrition refers either to administration of one or two components, such as lipids alone, or administration of total admixture at less than what is required for the maintenance of the patient.^{53,212,229} Peripherally administered parenteral nutrition solutions may be administered alone or used in combination with enteral nutrition. Commonly, 20% lipid is administered as a calorie source, or 5% dextrose with 3.5% amino acid solution is administered as a caloric and protein source. Peripherally administered parenteral nutrition solutions must be less than 2 times plasma osmolality (<600 mOsm/kg), or thrombophlebitis may occur.²²⁹

Refeeding syndrome may occur in cats that have experienced prolonged anorexia, such as cats with hepatic lipidosis. Prolonged anorexia induces a hypometabolic state, as described previously. Refeeding stimulates the release of insulin, causing dramatic shifts in electrolytes from the extracellular to the intracellular space. This affects primarily phosphorus, potassium and magnesium. Hypophosphatemia may also induce hemolytic anemia in cats. To prevent refeeding syndrome, identify patients at risk, especially those that have experienced anorexia for more than 5 to 7 days. Initial feeding rates should not exceed the patient's basic RER. Serum phosphorus, potassium, and magnesium should be monitored at least daily for the first few days of refeeding, and supplemental therapy should be provided as

needed. Patients should also be monitored carefully for fluid overload.

URINARY TRACT DISORDERS: CHRONIC RENAL DISEASE

Animal Factors

Cats with chronic renal disease may show clinical signs consistent with a chronic disease such as weight loss, loss of muscle mass, poor hair coat, and pale mucous membranes; however, they may appear clinically normal.^{70,104,150,215} Chronic renal disease may occur in association with other conditions, such as hyperthyroidism.^{4,25,138,147} A staging system and recommended treatment plan were developed by the International Renal Insufficiency Society (<http://iris-kidney.com/>). Staging of chronic kidney disease (CKD) is undertaken after the diagnosis of CKD to facilitate appropriate treatment and monitoring of the patient. Staging is based initially on fasting plasma creatinine, assessed on at least two occasions in the stable patient. The patient is then substaged on the basis of proteinuria and systemic blood pressure. This staging system has been shown to correlate with survival.³¹ Additional variables associated with decreased survival in cats with chronic renal failure include degree of azotemia, proteinuria, leukocytosis, anemia, systemic arterial hypertension, and ureteral calculi.* Nephrolithiasis is not associated with progression of renal failure.²³⁶ For more information on CKD, see Chapter 32.

Dietary Factors

Dietary modification has been shown to improve survival and quality of life of cats with CKD.[†] Because cats with CKD have polyuria and polydipsia, feeding them a canned diet may be beneficial. Fresh water should be available at all times. Some cats with CKD do not drink adequately and require supplemental subcutaneously administered crystalloid solutions (75 to 150 mL per day). If subcutaneous fluid administration is not possible, placement of a feeding tube (esophagostomy or gastrostomy) provides a means to administer supplemental fluids, nutrition, and medications. Some cats with CKD have decreased appetite; therefore they should be fed a calorically dense diet (i.e., a high-fat diet). Restrict dietary protein to 3.8 to 4.4 g/kg per day or 28% to 32% on a dry matter basis. Hypokalemia may occur in cats with CKD (Figure 18-15); therefore a diet replete in potassium (0.8% to 1.2% on a dry matter basis) should

*References 72, 134, 140, 142, 257, 258.

†References 2, 3, 11, 73, 74, 112, 214, 216, 218, 235.



FIGURE 18-15 Hypokalemic polymyopathy in a 16-year-old castrated male domestic shorthair cat with chronic kidney disease.

be fed, or supplementation provided using potassium gluconate or potassium citrate. Renal disease diets are typically sodium restricted, which may be beneficial with associated fluid retention and systemic arterial hypertension; however, there is evidence that it may contribute to hyperkalemia.³⁹ Metabolic acidosis occurs with CKD, although it may not occur until late in the course of the disease. Feed an alkalinizing diet to offset metabolic acidosis or supplement with an alkalinizing agent (e.g., potassium citrate). Phosphorus restriction has been shown to modify progression of CKD.²³⁴ Modification of dietary fatty acids, including increased n3 fatty acid intake, has been shown to be beneficial in dogs,^{16,35-37} but the efficacy has not been proved in cats. CKD is a pro-oxidative condition, and antioxidants may be beneficial.^{34,131,277}

The recommended dietary formulation for feeding cats with chronic renal failure is summarized in **Box 18-11**.⁷⁷

Feeding Factors

Decreased appetite may result from uremic gastroenteritis. Management includes feeding a highly palatable, calorically dense diet, administering histamine₂ receptor antagonists or antacids, feeding small meals frequently, and using feeding tubes.⁷¹

BOX 18-11

Dietary Recommendations for Cats with Chronic Renal Disease

1. Protein: 28% to 35% on a dry matter basis
2. Phosphorus: 0.3% to 0.6% on a dry matter basis
3. Sodium: <0.4% on a dry matter basis, if hypertensive
4. Chloride: 1.5 times sodium level on a dry matter basis
5. Potassium: 0.7% to 1.2% on a dry matter basis
6. n3 fatty acids: 0.4% to 2.5% on a dry matter basis. Ratio of n6:n3 fatty acids in the diet should be 1.1-7:1
7. Vitamin E: >500 IU/kg of diet
8. Vitamin C: 100 to 200 mg/kg of diet

URINARY TRACT DISORDERS: UROLITHIASIS

Animal Factors

Uroliths may be composed of several different types of minerals; struvite and calcium oxalate occur most commonly in adult cats.^{42,121,210} In kittens, infection-induced struvite and urate uroliths occur most commonly. In young adults struvite uroliths are typically not associated with a bacterial urinary tract infection. In older cats infection-induced struvite and calcium oxalate occur more commonly.¹² Uroliths occurring in the upper urinary tract are typically composed of calcium oxalate.^{141,151,237}

Urolith formation is associated with varying underlying causes. Sterile struvite uroliths are associated with the feeding of meals versus *ad libitum* feeding, dry foods, increased carbohydrate, certain protein sources, and alkaluria.* Conditions necessary for the formation of struvite crystals and uroliths include sufficient concentration of the composite minerals (i.e., magnesium, ammonium, and phosphate) and retention of the components in the urinary tract for sufficient time to allow crystallization. Thus production of small volumes of concentrated urine would seem to be a contributing factor. In addition, a pH favorable for struvite precipitation must exist (7 and above). However, many cats with struvite urolithiasis have a neutral or acidic urine pH at presentation.

Calcium oxalate uroliths are associated with hypercalcemia (idiopathic hypercalcemia is the most common cause in cats) or hypercalciuria. The factors predisposing cats without hypercalcemia to hypercalciuria are not well understood and may involve consumption of high

*References 14, 65, 91-96, 211, 249.

BOX 18-12**Dietary Recommendations for Dissolution of Sterile Struvite Uroliths in Cats**

1. Water: Canned foods induce polyuria and excretion of minerals
2. Magnesium: 0.04% to 0.09% on a dry matter basis
3. Phosphorus: 0.45% to 1.1% on a dry matter basis
4. Protein: 30% to 50% on a dry matter basis
5. Sodium: 0.3% to 0.6% on a dry matter basis
6. Urinary pH: 5.5 to 6.5

levels of sodium (discussed later) and the vitamin D and ascorbic acid levels in the diet. Some cats with calcium oxalate uroliths are mildly acidemic, which may result in mobilization of calcium that is excreted in the urine. Most cats with calcium oxalate uroliths have normal serum calcium, but some have moderate hypercalcemia, which may promote urinary calcium excretion. Urate uroliths are associated with liver disease or an underlying defect in uric acid metabolism.^{13,188} Cystine uroliths are associated with an inborn error of cystine reabsorption in the renal proximal tubule.⁶⁶ For more information on urolithiasis, see Chapter 32.

Dietary Factors

Struvite uroliths can be dissolved medically with a calculolytic diet. For dissolution of struvite uroliths, feed a low-magnesium, low-phosphorus, low-protein, acidifying (pH 5.5 to 6.5), and diuretic diet, and administer antibiotics if the uroliths are associated with infection.^{13,208,209} The average time for dissolution of sterile struvite uroliths is 2 to 4 weeks.^{13,209}

The recommended dietary formulation for inducing dissolution of sterile struvite uroliths in cats is summarized in Box 18-12.⁷⁹

At the time of this writing, no therapeutic diets are available for dissolution of calcium oxalate uroliths in cats. Other methods of urolith removal from the lower urinary tract must be employed, such as cystoscopic basket retrieval, voiding urohydropropulsion, and cystotomy.

Prevention of uroliths may involve dietary changes insofar as many types of feline uroliths are recurrent.

For infection-induced struvite uroliths, no dietary change is required. Infection-induced struvite uroliths result from a microbial infection with an organism that produces urease. Therefore if an infection does not recur, infection-induced struvite uroliths will not recur.^{13,244} Sterile struvite uroliths are recurrent if diet is not modified. The recommended dietary formulation for prevention of sterile struvite uroliths in cats is summarized in Box 18-13.⁷⁹

BOX 18-13**Dietary Recommendations for Prevention of Sterile Struvite Uroliths in Cats**

1. Water: Canned diet may increase urine volume and dilute potential calculogenic compounds
2. Magnesium: 0.04% to 0.14% on a dry matter basis
3. Phosphorus: 0.5% to 0.9% on a dry matter basis
4. Protein: 30% to 50% on a dry matter basis
5. Urinary pH: 6 to 6.8

BOX 18-14**Dietary Recommendations for Prevention of Calcium Oxalate Uroliths in Cats**

1. Water: Canned diets increase urine volume and dilute potential calculogenic compounds
2. Magnesium: 0.07% to 0.14% on a dry matter basis
3. Phosphorus: 0.5% to 1% on a dry matter basis
4. Calcium: 0.6% to 1% on a dry matter basis
5. Protein: 30% to 40% on a dry matter basis
6. Urinary pH: >7
7. Crude fiber: >7% on a dry matter basis if idiopathic hypercalcemia is present

Dietary management to prevent recurrence of calcium oxalate uroliths depends on whether the cat is normocalcemic or hypercalcemic. In cats that are normocalcemic, feed an oxalate-preventive diet that is low in calcium and replete in magnesium and that induces a neutral to alkaline urine pH (>7).^{13,14,99,165} Cats with idiopathic hypercalcemia should be fed a higher-fiber diet supplemented with potassium citrate.¹⁸⁷ The recommended dietary formulation for prevention of calcium oxalate uroliths in cats is summarized in Box 18-14.⁷⁹

A current controversy in the management of calcium oxalate uroliths is dietary content of sodium. Consumption of high levels of sodium may augment renal calcium excretion in human beings. Epidemiologic evidence suggests that low dietary sodium levels in cat and dog foods increase the risk for calcium oxalate urolithiasis, and diets containing high dietary sodium levels decrease the risk.^{152,153} Recent studies in healthy cats did not find increased urine calcium excretion in response to high dietary salt intake (minimum 1.2% sodium dry matter basis).¹³⁷ In humans with hypocitraturia, sodium supplementation increased urine volume and decreased urinary saturation for calcium oxalate²⁵⁵; however, in a study of humans with hypercalciuria, sodium restriction decreased urinary calcium excretion and risk of stone formation.²⁰⁴ In one study cats with naturally occurring

BOX 18-15**Dietary Recommendations for Prevention of Urate Uroliths in Cats**

1. Protein: 28% to 35% on a dry matter basis
2. Phosphorus: 0.3% to 0.6% on a dry matter basis
3. Sodium: <0.4% on a dry matter basis, if hypertensive
4. Chloride: 1.5 times sodium level on a dry matter basis
5. Potassium: 0.7% to 1.2% on a dry matter basis
6. Urinary pH: >7

calcium oxalate uroliths excreted less urine calcium when fed a food lower in sodium.¹⁶⁵ In healthy cats or those with marginal renal function and hypercalciuria, increased dietary sodium exacerbated calcium excretion with¹³⁶ and without^{39,122,163,273} increasing azotemia or blood pressure. Furthermore, in another study restriction of dietary sodium was associated with kaliuresis, an increased risk of hypokalemia, and a decrease in glomerular filtration rate in cats with induced chronic renal failure.³⁹ Until further data are available, orally administered sodium chloride or loop diuretics, which promote renal sodium excretion, for diuresis should be used cautiously and with careful monitoring because they may increase the risk of calcium oxalate urolith formation or worsening azotemia in some patients. Recommended levels of sodium in foods for cats predisposed to calcium oxalate formation is unknown; diets containing as low as 0.4 g/1000 kcal sodium and as high as 3.5 g/1000 kcal sodium are available commercially.

For urate uroliths not associated with underlying liver disease, cats should be fed a low-protein, alkalinizing, and diuretic diet. Allopurinol has not been evaluated in cats. The recommended dietary formulation for prevention of urate uroliths in cats is summarized in **Box 18-15**.

For management of cystine uroliths, cats should be fed a low-protein, alkalinizing, and diuretic diet similar to that used to manage cats with urate uroliths.

Feeding Factors

Free-choice feeding is associated with persistent aciduria because ingesting multiple small meals per day reduces the magnitude of the postprandial alkaline tide. As the size of the meal increases, so does the postprandial urine pH. Therefore *ad libitum* feeding may be most appropriate for management of struvite urolithiasis. Feeding a canned diet may decrease the risk of recurrence of sterile struvite, calcium oxalate, urate, and cystine uroliths by decreasing concentrations of calculogenic compounds in the urine. For cats that will not eat a canned diet, other

BOX 18-16**How to Increase Water Consumption**

1. Fresh water should be provided every day.
 - Some cats will drink more water if offered filtered water rather than tap water.
 - Water can be flavored with ice cubes made from tuna or clam juice.
2. Provide water in more than one location.
 - Ensure that water bowls are placed away from food dishes, if possible, and definitely away from litter boxes.
3. Try a different type of water bowl.
 - Wide or shallow bowls may be preferred by some cats; try using a dog water bowl.
 - Keep the water filled to near the top; some cats dislike putting their face into a bowl.
 - Clean food and water bowls at least every other day.
 - Use stainless steel or ceramic bowls; plastic is more difficult to clean and retains odors.
4. Try providing a source of running water, such as a dripping tap or a pet water fountain.
 - Pet water fountains require meticulous cleaning.
5. Increase the frequency of meals.
 - Feed a larger number of small meals, ensuring that the total quantity fed is not increased.
6. Add water to canned food, but do not make it too thin; some cats will even accept water added to dry diets.

methods may be employed to increase water intake (**Box 18-16**). Monitor response to dietary therapy using urine specific gravity, urine pH, and absence of crystalluria.

URINARY TRACT DISORDERS: IDIOPATHIC CYSTITIS**Animal Factors**

Idiopathic cystitis is a sterile inflammatory process that usually occurs in young adult cats and is the most common diagnosis for young cats with lower urinary tract disease signs. Clinical signs include pollakiuria, dysuria, hematuria, and perirenia. Clinical signs tend to be self-limiting, resolving in 3 to 7 days. However, recurrence is common. Males may obstruct from matrix-crystalline urethral plugs that are often composed of struvite. The pathophysiology of idiopathic cystitis is not well understood but appears to involve derangements of the interactions between the central nervous system and the endocrine system, with the bladder as target organ. For more information on idiopathic cystitis, see Chapter 32.

Dietary Factors

Dry diets may result in concentrated urine and increased concentration of crystallogenic compounds. Although cats consuming dry diets may drink more water than cats consuming canned diets, they do not increase water intake in sufficient amounts to compensate for the lower moisture content of dry diets. This is an important concept for owners to understand. Canned diets increase water intake and urine volume and decrease urine specific gravity, which may be beneficial.^{13,103,177} Increased water intake may also dilute noxious components in urine that could irritate the bladder mucosa. The treatment goal should be to decrease the cat's urine specific gravity to 1.030 or less. Acidifying diets per se are of no value in treating idiopathic cystitis unless significant struvite crystalluria is present.

Feeding Factors

Cats fed *ad libitum* may have increased frequency of urination and greater total urine volume than meal-fed cats. Feeding a canned diet also increases volume and frequency of urination and may be beneficial in cats with idiopathic cystitis. Changing diets is a stressor and must be approached carefully for cats with idiopathic cystitis (see Box 18-5). For cats that will not eat a canned diet, other methods may be employed to increase water intake (see Box 18-16).

References

- Abood SK, Buffington CA: Enteral feeding of dogs and cats: 51 cases (1989-1991), *J Am Vet Med Assoc* 201:619, 1992.
- Adams LG, Polzin DJ, Osborne CA et al: Effects of dietary protein and calorie restriction in clinically normal cats and in cats with surgically induced chronic renal failure, *Am J Vet Res* 54:1653, 1993.
- Adams LG, Polzin DJ, Osborne CA et al: Influence of dietary protein/calorie intake on renal morphology and function in cats with 5/6 nephrectomy, *Lab Invest* 70:347, 1994.
- Adams WH, Daniel GB, Legendre AM et al: Changes in renal function in cats following treatment of hyperthyroidism using ^{131}I , *Vet Radiol Ultrasound* 38:231, 1997.
- Anderson JG, Harvey CE, Flax B: Clinical and radiographic evaluation of external odontoclastic resorptive lesions in cats (abstract), *J Vet Intern Med* 7:134, 1993.
- Appleton DJ, Rand JS, Sunvold GD et al: Dietary chromium tripicolinate supplementation reduces glucose concentrations and improves glucose tolerance in normal-weight cats, *J Feline Med Surg* 4:13, 2002.
- Armstrong PJ, Hand MS, Frederick GS: Enteral nutrition by tube, *Vet Clin North Am Small Anim Pract* 20:237, 1990.
- Armstrong PJ, Lund EM: Changes in body composition and energy balance with aging, *Vet Clinical Nutr* 3:83, 1996.
- Backus RC, Cave NJ, Keisler DH: Gonadectomy and high dietary fat but not high dietary carbohydrate induce gains in body weight and fat of domestic cats, *Br J Nutr* 98:641, 2007.
- Baez JL, Michel KE, Sorenmo K et al: A prospective investigation of the prevalence and prognostic significance of weight loss and changes in body condition in feline cancer patients, *J Feline Med Surg* 9:411, 2007.
- Barber PJ, Rawlings JM, Markwell PJ et al: Effect of dietary phosphate restriction on renal secondary hyperparathyroidism in the cat, *J Small Anim Pract* 40:62, 1999.
- Bartges JW: Lower urinary tract disease in older cats: What's common, what's not? In *Health and nutrition of geriatric cats and dogs*, Proceedings, North American Veterinary Conference, Orlando, Fla, 1996.
- Bartges JW, Kirk CA: Nutrition and lower urinary tract disease in cats, *Vet Clin North Am Small Anim Pract* 36:1361, 2006.
- Bartges JW, Kirk CA, Moyers T: Influence of alkalinization and acidification on urine saturation with calcium oxalate and struvite and bone mineral density in healthy cats, *Urol Res* 32:172, 2004.
- Bauer JE: Responses of dogs to dietary omega-3 fatty acids, *J Am Vet Med Assoc* 231:1657, 2007.
- Bauer JE, Markwell PJ, Rawlings JM et al: Effects of dietary fat and polyunsaturated fatty acids in dogs with naturally developing chronic renal failure, *J Am Vet Med Assoc* 215:1588, 1999.
- Beale BS: Use of nutraceuticals and chondroprotectants in osteoarthritic dogs and cats, *Vet Clin North Am Small Anim Pract* 34:271, 2004.
- Beale BS: Orthopedic problems in geriatric dogs and cats, *Vet Clin North Am Small Anim Pract* 35:655, 2005.
- Belsito KR, Vester BM, Keel T et al: Impact of ovariohysterectomy and food intake on body composition, physical activity, and adipose gene expression in cats, *J Anim Sci* 87:594, 2009.
- Bennett N, Greco DS, Peterson ME et al: Comparison of a low carbohydrate-low fiber diet and a moderate carbohydrate-high fiber diet in the management of feline diabetes mellitus, *J Feline Med Surg* 8:73, 2006.
- Birchard SJ, McLoughlin MA, Smeak DD: Chylothorax in the dog and cat: a review, *Lymphology* 28:64, 1995.
- Birchard SJ, Smeak DD, McLoughlin MA: Treatment of idiopathic chylothorax in dogs and cats, *J Am Vet Med Assoc* 212:652, 1998.
- Bissot T, Servet E, Vidal S et al: Novel dietary strategies can improve the outcome of weight loss programmes in obese client-owned cats, *J Feline Med Surg* 12:104, 2010.
- Blanchard G, Paragon BM, Milliat F et al: Dietary L-carnitine supplementation in obese cats alters carnitine metabolism and decreases ketosis during fasting and induced hepatic lipidosis, *J Nutr* 132:204, 2002.
- Boag AK, Neiger R, Slater L et al: Changes in the glomerular filtration rate of 27 cats with hyperthyroidism after treatment with radioactive iodine, *Vet Rec* 161:711, 2007.
- Boegehold MA, Kotchen TA: Relative contributions of dietary Na^+ and Cl^- to salt-sensitive hypertension, *Hypertension* 14:579, 1989.
- Booij-Vrieling HE, Ferbus D, Tryfonidou MA et al: Increased vitamin D-driven signalling and expression of the vitamin D receptor, MSX2, and RANKL in tooth resorption in cats, *Eur J Oral Sci* 118:39, 2010.
- Booij-Vrieling HE, Tryfonidou MA, Riemers FM et al: Inflammatory cytokines and the nuclear vitamin D receptor are implicated in the pathophysiology of dental resorptive lesions in cats, *Vet Immunol Immunopathol* 132:160, 2009.
- Bosworth C, Bartges JW, Snow P: Nasoesophageal and nasogastric feeding tubes, *Vet Med* 99:590, 2004.
- Boyce EN, Logan EI: Oral health assessment in dogs: study design and results, *J Vet Dent* 11:64, 1994.
- Boyd LM, Langston C, Thompson K et al: Survival in cats with naturally occurring chronic kidney disease (2000-2002), *J Vet Intern Med* 22:1111, 2008.

32. Brady MA, Janovitz EB: Nephrotoxicosis in a cat following ingestion of Asiatic hybrid lily (*Lilium* sp), *J Vet Diagn Invest* 12:566, 2000.
33. Bright RM: Percutaneous endoscopic gastrostomy, *Vet Clin North Am Small Anim Pract* 23:531, 1993.
34. Brown SA: Oxidative stress and chronic kidney disease, *Vet Clin North Am Small Anim Pract* 38:157, 2008.
35. Brown SA, Brown CA, Crowell WA et al: Does modifying dietary lipids influence the progression of renal failure? *Vet Clin North Am Small Anim Pract* 26:1277, 1996.
36. Brown SA, Brown CA, Crowell WA et al: Effects of dietary polyunsaturated fatty acid supplementation in early renal insufficiency in dogs, *J Lab Clin Med* 135:275, 2000.
37. Brown SA, Finco DR, Brown CA: Is there a role for dietary polyunsaturated fatty acid supplementation in canine renal disease? *J Nutr* 128:276S, 1998.
38. Budberg SC, Bartges JW: Nutrition and osteoarthritis in dogs: does it help? *Vet Clin North Am Small Anim Pract* 36:1307, 2006.
39. Buranakarl C, Mathur S, Brown SA: Effects of dietary sodium chloride intake on renal function and blood pressure in cats with normal and reduced renal function, *Am J Vet Res* 65:620, 2004.
40. Burkholder WJ: Use of body condition scores in clinical assessment of the provision of optimal nutrition, *J Am Vet Med Assoc* 217:650, 2000.
41. Campbell KL: Fatty acid supplementation and skin disease, *Vet Clin North Am Small Anim Pract* 20:1475, 1990.
42. Cannon AB, Westropp JL, Ruby AL et al: Evaluation of trends in urolith composition in cats: 5,230 cases (1985-2004), *J Am Vet Med Assoc* 231:570, 2007.
43. Cave NJ: Hydrolyzed protein diets for dogs and cats, *Vet Clin North Am Small Anim Pract* 36:1251, 2006.
44. Center SA: Hepatic lipidosis, glucocorticoid hepatopathy, vacuolar hepatopathy, storage disorders, amyloidosis and iron toxicity. In Guilford WG, Center SA, Strombeck DR et al, editors: *Strombeck's small animal gastroenterology*, ed 3, Philadelphia, 1996, Saunders, p 766.
45. Center SA: Nutritional support for dogs and cats with hepatobiliary disease, *J Nutr* 128:2733S, 1998.
46. Center SA, Crawford MA, Guida L et al: A retrospective study of 77 cats with severe hepatic lipidosis: 1975-1990, *J Vet Intern Med* 7:349, 1993.
47. Center SA, Elston TH, Rowland PH et al: Fulminant hepatic failure associated with oral administration of diazepam in 11 cats, *J Am Vet Med Assoc* 209:618, 1996.
48. Center SA, Harte J, Watrous D et al: The clinical and metabolic effects of rapid weight loss in obese pet cats and the influence of supplemental oral L-carnitine, *J Vet Intern Med* 14:598, 2000.
49. Chan DL: The inappetent hospitalised cat: clinical approach to maximising nutritional support, *J Feline Med Surg* 11:925, 2009.
50. Chan DL, Freeman LM: Nutrition in critical illness, *Vet Clin North Am Small Anim Pract* 36:1225, 2006.
51. Chan DL, Freeman LM, Labato MA et al: Retrospective evaluation of partial parenteral nutrition in dogs and cats, *J Vet Intern Med* 16:440, 2002.
52. Chandler ML: Nutritional support for the hospitalised small animal patient, *Practice* 30:442, 2008.
53. Chandler ML, Guilford WG, Payne-James J: Use of peripheral parenteral nutritional support in dogs and cats, *J Am Vet Med Assoc* 216:669, 2000.
54. Chastain CB, Graham CL, Nichols CE: Adrenocortical suppression in cats given megestrol acetate, *Am J Vet Res* 42:2029, 1981.
55. Cianciolo RE, Bischoff K, Ebel JG et al: Clinicopathologic, histologic, and toxicologic findings in 70 cats inadvertently exposed to pet food contaminated with melamine and cyanuric acid, *J Am Vet Med Assoc* 233:729, 2008.
56. Colliard L, Paragon BM, Lemuet B et al: Prevalence and risk factors of obesity in an urban population of healthy cats, *J Feline Med Surg* 11:135, 2009.
57. Cook AM, Peppard A, Magnuson B: Nutrition considerations in traumatic brain injury, *Nutr Clin Pract* 23:608, 2008.
58. Cooper JH: Megestrol acetate and appetite gain, *Vet Rec* 102:45, 1978.
59. Crowe DT, Downs MO: Pharyngostomy complications in dogs and cats and recommended technical modifications: Experimental and clinical investigations, *J Am Anim Hosp Assoc* 22:493, 1986.
60. Crowe DT, Jr, Devey JJ: Esophagostomy tubes for feeding and decompression: clinical experience in 29 small animal patients, *J Am Anim Hosp Assoc* 33:393, 1997.
61. Davenport DJ, Jergens AE, Remillard RL: Inflammatory bowel disease. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute, p 1065.
62. De-Souza DA, Greene LJ: Intestinal permeability and systemic infections in critically ill patients: effect of glutamine, *Crit Care Med* 33:1125, 2005.
63. Dennis JS, Kruger JM, Mullaney TP: Lymphocytic/plasmacytic colitis in cats: 14 cases (1985-1990), *J Am Vet Med Assoc* 202:313, 1993.
64. Devitt CM, Seim HB III: Clinical evaluation of tube esophagostomy in small animals, *J Am Anim Hosp Assoc* 33:55, 1997.
65. Devois C, Biourge V, Morice G et al: Struvite and oxalate activity product ratios and crystalluria in cats fed acidifying diets. In *Urolithiasis 2000*, Capetown, South Africa, 2000.
66. DiBartola SP, Chew DJ, Horton ML: Cystinuria in a cat, *J Am Vet Med Assoc* 198:102, 1991.
67. Dow SW, Fettman MJ, Smith KR et al: Taurine depletion and cardiovascular disease in adult cats fed a potassium-depleted acidified diet, *Am J Vet Res* 53:402, 1992.
68. Drazenovich TL, Fascetti AJ, Westermeyer HD et al: Effects of dietary lysine supplementation on upper respiratory and ocular disease and detection of infectious organisms in cats within an animal shelter, *Am J Vet Res* 70:1391, 2009.
69. Eckersley RM: Losing the battle of the bulge: causes and consequences of increasing obesity, *Med J Aust* 174:590, 2001.
70. Elliott DA: Nutritional management of chronic renal disease in dogs and cats, *Vet Clin North Am Small Anim Pract* 36:1377, 2006.
71. Elliott DA, Riel DL, Rogers QR: Complications and outcomes associated with use of gastrostomy tubes for nutritional management of dogs with renal failure: 56 cases (1994-1999), *J Am Vet Med Assoc* 217:1337, 2000.
72. Elliott J, Barber PJ: Feline chronic renal failure: clinical findings in 80 cases diagnosed between 1992 and 1995, *J Small Anim Pract* 39:78, 1998.
73. Elliott J, Barber PJ, Rawlings JM et al: Effect of phosphate and protein restriction on progression of chronic renal failure in cats, *J Vet Intern Med* 12:221(abstract), 1998.
74. Elliott J, Rawlings JM, Markwell PJ et al: Survival of cats with naturally occurring chronic renal failure: effect of dietary management, *J Small Anim Pract* 41:235, 2000.
75. Fekete S, Hullar I, Andrasofszky E et al: Reduction of the energy density of cat foods by increasing their fibre content with a view to nutrients' digestibility, *J Anim Physiol Anim Nutr (Berl)* 85:200, 2001.
76. Flynn MB: Nutritional support for the burn-injured patient, *Crit Care Nurs Clin North Am* 16:139, 2004.
77. Forrester SD, Adams LG, Allen TA: Chronic kidney disease. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute, p 865.

78. Forrester SD, Fossum TW, Rogers KS: Diagnosis and treatment of chylothorax associated with lymphoblastic lymphosarcoma in four cats, *J Am Vet Med Assoc* 198:291, 1991.
79. Forrester SD, Kruger JM, Allen TA: Feline lower urinary tract diseases. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute, p 925.
80. Fossum TW: Chylothorax in cats: is there a role for surgery? *J Feline Med Surg* 3:73, 2001.
81. Fossum TW, Forrester SD, Swenson CL et al: Chylothorax in cats: 37 cases (1969-1989), *J Am Vet Med Assoc* 198:672, 1991.
82. Fossum TW, Miller MW, Rogers KS et al: Chylothorax associated with right-sided heart failure in five cats, *J Am Vet Med Assoc* 204:84, 1994.
83. Fox PR, Trautwein EA, Hayes KC et al: Comparison of taurine, alpha-tocopherol, retinol, selenium, and total triglycerides and cholesterol concentrations in cats with cardiac disease and in healthy cats, *Am J Vet Res* 54:563, 1993.
84. Francis H, Bartges JW, Tobias K et al: Enterostomy feeding tubes, *Vet Med* 99:627, 2004.
85. Frank G, Anderson W, Pazak H et al: Use of a high-protein diet in the management of feline diabetes mellitus, *Vet Ther* 2:238, 2001.
86. Freeman LM: Interventional nutrition for cardiac disease, *Clin Tech Small Anim Pract* 13:232, 1998.
87. Freeman LM, Abood SK, Fascetti AJ et al: Disease prevalence among dogs and cats in the United States and Australia and proportions of dogs and cats that receive therapeutic diets or dietary supplements, *J Am Vet Med Assoc* 229:531, 2006.
88. Freeman LM, Michel KE: Evaluation of raw food diets for dogs, *J Am Vet Med Assoc* 218:705, 2001.
89. Freeman LM, Rush JE: Nutrition and cardiomyopathy: lessons from spontaneous animal models, *Curr Heart Fail Rep* 4:84, 2007.
90. Fulton RB, Jr, Dennis JS: Blind percutaneous placement of a gastrostomy tube for nutritional support in dogs and cats, *J Am Vet Med Assoc* 201:697, 1992.
91. Funaba M, Matsumoto C, Matsuki K et al: Comparison of corn gluten meal and meat meal as a protein source in dry foods formulated for cats, *Am J Vet Res* 63:1247, 2002.
92. Funaba M, Oka Y, Kobayashi S et al: Evaluation of meat meal, chicken meal, and corn gluten meal as dietary sources of protein in dry cat food, *Can J Vet Res* 69:299, 2005.
93. Funaba M, Tanak T, Kaneko M et al: Fish meal vs. corn gluten meal as a protein source for dry cat food, *J Vet Med Sci* 63:1355, 2001.
94. Funaba M, Uchiyama A, Takahashi K et al: Evaluation of effects of dietary carbohydrate on formation of struvite crystals in urine and macromineral balance in clinically normal cats, *Am J Vet Res* 65:138, 2004.
95. Funaba M, Yamate T, Hashida Y et al: Effects of a high-protein diet versus dietary supplementation with ammonium chloride on struvite crystal formation in urine of clinically normal cats, *Am J Vet Res* 64:1059, 2003.
96. Funaba M, Yamate T, Narukawa Y et al: Effect of supplementation of dry cat food with D,L-methionine and ammonium chloride on struvite activity product and sediment in urine, *J Vet Med Sci* 63:337, 2001.
97. Gaskell R, Dawson S, Radford A et al: Feline herpesvirus, *Vet Rec* 38:337, 2007.
98. German AJ, Holden S, Bissot T et al: Changes in body composition during weight loss in obese client-owned cats: loss of lean tissue mass correlates with overall percentage of weight lost, *J Feline Med Surg* 10:452, 2008.
99. Gisselman K, Langston C, Palma D et al: Calcium oxalate urolithiasis, *Compend Contin Educ Vet* 31:496, 2009.
100. Glaus TM, Cornelius LM, Bartges JW et al: Complications with non-endoscopic percutaneous gastrostomy in 31 cats and 10 dogs: a retrospective study, *J Small Anim Pract* 39:218, 1998.
101. Godfrey DR: Osteoarthritis in cats: a retrospective radiological study, *J Small Anim Pract* 46:425, 2005.
102. Gould L: The medical management of idiopathic chylothorax in a domestic long-haired cat, *Can Vet J* 45:51, 2004.
103. Grant DC: Effect of water source on intake and urine concentration in healthy cats, *J Feline Med Surg* 12:431, 2009.
104. Grauer GF: Early detection of renal damage and disease in dogs and cats, *Vet Clin North Am Small Anim Pract* 35:581, 2005.
105. Greenberg AS, Obin MS: Obesity and the role of adipose tissue in inflammation and metabolism, *Am J Clin Nutr* 83:461S, 2006.
106. Guilford WG, Matz ME: The nutritional management of gastrointestinal tract disorders in companion animals, *N Z Vet J* 51:284, 2003.
107. Hadley RM, Richardson JA, Gwaltney-Brant SM: A retrospective study of daylily toxicosis in cats, *Vet Hum Toxicol* 45:38, 2003.
108. Han E: Esophageal and gastric feeding tubes in ICU patients, *Clin Tech Small Anim Pract* 19:22, 2004.
109. Hardie EM, Roe SC, Martin FR: Radiographic evidence of degenerative joint disease in geriatric cats: 100 cases (1994-1997), *J Am Vet Med Assoc* 220:628, 2002.
110. Harkin KR, Cowan LA, Andrews GA et al: Hepatotoxicity of stanozolol in cats, *J Am Vet Med Assoc* 217:681, 2000.
111. Harper EJ, Stack DM, Watson TD et al: Effects of feeding regimens on bodyweight, composition and condition score in cats following ovariohysterectomy, *J Small Anim Pract* 42:433, 2001.
112. Harte JG, Markwell PJ, Moraillon RM et al: Dietary management of naturally occurring chronic renal failure in cats, *J Nutr* 124:2660S, 1994.
113. Hayes G: Chylothorax and fibrosing pleuritis secondary to thyrotoxic cardiomyopathy, *J Small Anim Pract* 46:203, 2005.
114. Hennet P, Servet E, Soulard Y et al: Effect of pellet food size and polyphosphates in preventing calculus accumulation in dogs, *J Vet Dent* 24:236, 2007.
115. Heuter K: Placement of jejunal feeding tubes for post-gastric feeding, *Clin Tech Small Anim Pract* 19:32, 2004.
116. Hill PB, Lo A, Eden CA et al: Survey of the prevalence, diagnosis and treatment of dermatological conditions in small animals in general practice, *Vet Rec* 158:533, 2006.
117. Hill RC: Conference on "Multidisciplinary approaches to nutritional problems". Symposium on "Nutrition and health." Nutritional therapies to improve health: lessons from companion animals, *Proc Nutr Soc* 68:98, 2009.
118. Hinrichs U, Puhl S, Rutteman GR et al: Lymphangiosarcomas in cats: a retrospective study of 12 cases, *Vet Pathol* 36:164, 1999.
119. Hinton M, Gaskell CJ: Non-neoplastic mammary hypertrophy in the cat associated either with pregnancy or with oral progestagen therapy, *Vet Rec* 100:277, 1977.
120. Hoenig M, Ferguson DC: Effects of neutering on hormonal concentrations and energy requirements in male and female cats, *Am J Vet Res* 63:634, 2002.
121. Houston DM, Moore AE: Canine and feline urolithiasis: examination of over 50 000 urolith submissions to the Canadian veterinary urolith centre from 1998 to 2008, *Can Vet J* 50:1263, 2009.
122. Hughes KL, Slater MR, Geller S et al: Diet and lifestyle variables as risk factors for chronic renal failure in pet cats, *Prev Vet Med* 55:1, 2002.
123. Iapichino G, Radrizzani D, Destrebecq A et al: Metabolic support of the critically ill: 2008 update, *Minerva Anestesiol* 74:709, 2008.
124. Ibrahim WH, Bailey N, Sunvold GD et al: Effects of carnitine and taurine on fatty acid metabolism and lipid accumulation in the liver of cats during weight gain and weight loss, *Am J Vet Res* 64:1265, 2003.
125. Ireland LM, Hohenhaus AE, Broussard JD et al: A comparison of owner management and complications in 67 cats with

- esophagostomy and percutaneous endoscopic gastrostomy feeding tubes, *J Am Anim Hosp Assoc* 39:241, 2003.
126. Jacobs G, Cornelius L, Keene B et al: Comparison of plasma, liver, and skeletal muscle carnitine concentrations in cats with idiopathic hepatic lipidosis and in healthy cats, *Am J Vet Res* 51:1349, 1990.
 127. Jergens AE, Morrison JA, Miles KG et al: Percutaneous endoscopic gastrojejunostomy tube placement in healthy dogs and cats, *J Vet Intern Med* 21:18, 2007.
 128. Kahn SA: Placement of canine and feline esophagostomy feeding tubes, *Lab Anim (NY)* 36:25, 2007.
 129. Kanchuk ML, Backus RC, Calvert CC et al: Neutering induces changes in food intake, body weight, plasma insulin and leptin concentrations in normal and lipoprotein lipase-deficient male cats, *J Nutr* 132:1730S, 2002.
 130. Kass PH, Peterson ME, Levy J et al: Evaluation of environmental, nutritional, and host factors in cats with hyperthyroidism, *J Vet Intern Med* 13:323, 1999.
 131. Keegan RF, Webb CB: Oxidative stress and neutrophil function in cats with chronic renal failure, *J Vet Intern Med*, 2010.
 132. Keene BW, Panciera DL, Atkins CE et al: Myocardial L-carnitine deficiency in a family of dogs with dilated cardiomyopathy, *J Am Vet Med Assoc* 198:647, 1991.
 133. Kienzle E, Bergler R: Human-animal relationship of owners of normal and overweight cats, *J Nutr* 136:1947S, 2006.
 134. King JN, Tasker S, Gunn-Moore DA et al: Prognostic factors in cats with chronic kidney disease, *J Vet Intern Med* 21:906, 2007.
 135. Kirk CA: Feline diabetes mellitus: low carbohydrates versus high fiber? *Vet Clin North Am Small Anim Pract* 36:1297, 2006.
 136. Kirk CA, Jewell DE, Lowry SR: Effects of sodium chloride on selected parameters in cats, *Vet Ther* 7:333, 2006.
 137. Kirk CA, Ling GV, Osborne CA et al: Clinical guidelines for managing calcium oxalate uroliths in cats: medical therapy, hydration, and dietary therapy. In *Managing urolithiasis in cats: recent updates and practice guidelines*, Topeka, Kan, 2003, Hill's Pet Nutrition Inc, p 10.
 138. Kobayashi DL, Peterson ME, Graves TK et al: Hypertension in cats with chronic renal failure or hyperthyroidism, *J Vet Int Med* 4:58, 1990.
 139. Kopko SH: The use of rutin in a cat with idiopathic chylothorax, *Can Vet J* 46:729, 2005.
 140. Kuwahara Y, Ohba Y, Kitoh K et al: Association of laboratory data and death within one month in cats with chronic renal failure, *J Small Anim Pract* 47:446, 2006.
 141. Kyles AE, Hardie EM, Wooden BG et al: Clinical, clinicopathologic, radiographic, and ultrasonographic abnormalities in cats with ureteral calculi: 163 cases (1984-2002), *J Am Vet Med Assoc* 226:932, 2005.
 142. Kyles AE, Hardie EM, Wooden BG et al: Management and outcome of cats with ureteral calculi: 153 cases (1984-2002), *J Am Vet Med Assoc* 226:937, 2005.
 143. Laflamme D: Development and validation of a body condition score system for cats: a clinical tool, *Feline Pract* 25:13, 1997.
 144. Laflamme DP: Nutrition for aging cats and dogs and the importance of body condition, *Vet Clin North Am Small Anim Pract* 35:713, 2005.
 145. Laflamme DP: Understanding and managing obesity in dogs and cats, *Vet Clin North Am Small Anim Pract* 36:1283, 2006.
 146. Langston CE: Acute renal failure caused by lily ingestion in six cats, *J Am Vet Med Assoc* 220:49, 2002.
 147. Langston CE, Reine NJ: Hyperthyroidism and the kidney, *Clin Tech Small Anim Pract* 21:17, 2006.
 148. Lappin MR, Veir JK, Satyraj E et al: Pilot study to evaluate the effect of oral supplementation of *Enterococcus faecium* SF68 on cats with latent feline herpesvirus 1, *J Feline Med Surg* 11:650, 2009.
 149. Lascelles BD, Depuy V, Thomson A et al: Evaluation of a therapeutic diet for feline degenerative joint disease, *J Vet Intern Med* 24(3):487, 2010.
 150. Lees GE: Early diagnosis of renal disease and renal failure, *Vet Clin North Am Small Anim Pract* 34:867, 2004.
 151. Lekcharoenk C, Osborne CA, Lulich JP et al: Increased frequency of calcium oxalate uroliths in the upper urinary tract of cats: 1981 to 1999. In *Managing urolithiasis in cats: recent updates and practice guidelines*, Davis, Calif, 2003.
 152. Lekcharoenk C, Osborne CA, Lulich JP: Epidemiologic study of risk factors for lower urinary tract diseases in cats, *J Am Vet Med Assoc* 218:1429, 2001.
 153. Lekcharoenk C, Osborne CA, Lulich JP et al: Association between dietary factors and calcium oxalate and magnesium ammonium phosphate urolithiasis in cats, *J Am Vet Med Assoc* 219:1228, 2001.
 154. Levine PB, Smallwood LJ, Buback JL: Esophagostomy tubes as a method of nutritional management in cats: a retrospective study, *J Am Anim Hosp Assoc* 33:405, 1997.
 155. Lewin-Smith MR, Kalasinsky VF, Mullick FG et al: Melamine-containing crystals in the urinary tracts of domestic animals: sentinel event? *Arch Pathol Lab Med* 133:341, 2009.
 156. Lin L: RAGE on the Toll Road? *Cell Mol Immunol* 3:351, 2006.
 157. Lindsay FE: Chylothorax in the domestic cat—a review, *J Small Anim Pract* 15:241, 1974.
 158. Lippert AC: Enteral and parenteral nutritional support in dogs and cats with gastrointestinal disease, *Semin Vet Med Surg (Small Anim)* 4:232, 1989.
 159. Lippert AC, Faulkner JE, Evans AT et al: Total parenteral nutrition in clinically normal cats, *J Am Vet Med Assoc* 194:669, 1989.
 160. Lippert AC, Fulton RB, Jr, Parr AM: A retrospective study of the use of total parenteral nutrition in dogs and cats, *J Vet Intern Med* 7:52, 1993.
 161. Logan EI: Dietary influences on periodontal health in dogs and cats, *Vet Clin North Am Small Anim Pract* 36:1385, 2006.
 162. Logan EI, Wiggs RB, Scherl DS et al: Periodontal disease. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute, p 979.
 163. Luckschander N, Iben C, Hosgood G et al: Dietary NaCl does not affect blood pressure in healthy cats, *J Vet Intern Med* 18:463, 2004.
 164. Luhn A, Bartges JW, Snow P: Gastrostomy feeding tubes: percutaneous endoscopic placement, *Vet Med* 99:612, 2004.
 165. Lulich JP, Osborne CA, Lekcharoenk C et al: Effects of diet on urine composition of cats with calcium oxalate urolithiasis, *J Am Anim Hosp Assoc* 40:185, 2004.
 166. Lund EM, Armstrong PJ, Kirk CA et al: Prevalence and risk factors for obesity in adult cats from private veterinary practices, *Int J Appl Res Vet Med* 3:88, 2005.
 167. Lund EM, Armstrong PJ, Kirk CA et al: Health status and population characteristics of dogs and cats examined at private veterinary practices in the United States, *J Am Vet Med Assoc* 214:1336, 1999.
 168. Lusby AL, Kirk CA, Bartges JW: The role of key adipokines in obesity and insulin resistance in cats, *J Am Vet Med Assoc* 235:518, 2009.
 169. MacDonald ML, Rogers QR, Morris JG: Role of linoleate as an essential fatty acid for the cat independent of arachidonate synthesis, *J Nutr* 113:1422, 1983.
 170. MacDougall LD: Mammary fibroadenomatous hyperplasia in a young cat attributed to treatment with megestrol acetate, *Can Vet J* 44:227, 2003.
 171. Macy DW, Gasper PW: Diazepam-induced eating in anorexic cats, *J Am Anim Hosp Assoc* 21:17, 1985.

172. Maggs DJ: Update on pathogenesis, diagnosis, and treatment of feline herpesvirus type 1, *Clin Tech Small Anim Pract* 20:94, 2005.
173. Maggs DJ, Collins BK, Thorne JG et al: Effects of L-lysine and L-arginine on in vitro replication of feline herpesvirus type-1, *Am J Vet Res* 61:1474, 2000.
174. Maggs DJ, Nasisse MP, Kass PH: Efficacy of oral supplementation with L-lysine in cats latently infected with feline herpesvirus, *Am J Vet Res* 64:37, 2003.
175. Maggs DJ, Sykes JE, Clarke HE et al: Effects of dietary lysine supplementation in cats with enzootic upper respiratory disease, *J Feline Med Surg* 9:97, 2007.
176. Marks SL: The principles and practical application of enteral nutrition, *Vet Clin North Am Small Anim Pract* 28:677, 1998.
177. Markwell PJ, Buffington CA, Chew DJ et al: Clinical evaluation of commercially available urinary acidification diets in the management of idiopathic cystitis in cats, *J Am Vet Med Assoc* 214:361, 1999.
178. Marshall RD, Rand JS, Morton JM: Treatment of newly diagnosed diabetic cats with glargin insulin improves glycaemic control and results in higher probability of remission than protamine zinc and lente insulins, *J Feline Med Surg* 11:683, 2009.
179. Martin G, Rand J: Current understanding of feline diabetes: part 2, treatment, *J Feline Med Surg* 2:3, 2000.
180. Martin GJ, Rand JS: Food intake and blood glucose in normal and diabetic cats fed ad libitum, *J Feline Med Surg* 1:241, 1999.
181. Martin KM, Rossing MA, Ryland LM et al: Evaluation of dietary and environmental risk factors for hyperthyroidism in cats, *J Am Vet Med Assoc* 217:853, 2000.
182. Martin L, Siliart B, Dumon H et al: Leptin, body fat content and energy expenditure in intact and gonadectomized adult cats: a preliminary study, *J Anim Physiol Anim Nutr (Berl)* 85:195, 2001.
183. Mathews KA: The various types of parenteral fluids and their indications, *Vet Clin North Am Small Anim Pract* 28:483, 1998.
184. Mazzaferro EM: Complications of fluid therapy, *Vet Clin North Am Small Anim Pract* 38:607, 2008.
185. Mazzaferro EM, Greco DS, Turner AS et al: Treatment of feline diabetes mellitus using an alpha-glucosidase inhibitor and a low-carbohydrate diet, *J Feline Med Surg* 5:183, 2003.
186. McCarthy MS, Fabling J, Martindale R et al: Nutrition support of the traumatically injured warfighter, *Crit Care Nurs Clin North Am* 20:59, 2008.
187. McClain HM, Barsanti JA, Bartges JW: Hypercalcemia and calcium oxalate urolithiasis in cats: a report of five cases, *J Am Anim Hosp Assoc* 35:297, 1999.
188. McCue J, Langston C, Palma D et al: Urate urolithiasis, *Compend Contin Educ Vet* 31:468, 2009.
189. Meadows RL, MacWilliams PS, Dzata G et al: Chylothorax associated with cryptococcal mediastinal granuloma in a cat, *Vet Clin Pathol* 22:109, 1993.
190. Mehta NM, Duggan CP: Nutritional deficiencies during critical illness, *Pediatr Clin North Am* 56:1143, 2009.
191. Meincke JE, Hobbie WV, Jr, Barto LR: Traumatic chylothorax with associated diaphragmatic hernias in the cat, *J Am Vet Med Assoc* 155:15, 1969.
192. Mesich ML, Bartges JW, Tobias K et al: Gastrostomy feeding tubes: surgical placement, *Vet Med* 99:604, 2004.
193. Mesotten D, Van den Berghe G: Clinical benefits of tight glycaemic control: focus on the intensive care unit, *Best Pract Res Clin Anaesthesiol* 23:421, 2009.
194. Meyer HP, Twedt DC, Roudebush P et al: Hepatobiliary disease. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute, p 1155.
195. Michel KE: Focus on nutrition, *Compend Contin Educ Vet* 31:22, 2009.
196. Michel KE, Anderson W, Cupp C et al: Validation of a subjective muscle mass scoring system for cats, *J Anim Physiol Anim Nutr (Berl)* 93:806, 2009.
197. Middleton DJ, Watson AD: Glucose intolerance in cats given short-term therapies of prednisolone and megestrol acetate, *Am J Vet Res* 46:2623, 1985.
198. Miller CC, Bartges JW: Parenteral feeding products. In Kirk RW, Bonagura JD, editors: *Current veterinary therapy XIII*, Philadelphia, 1999, Saunders, p 80.
199. Miller CC, Bartges JW: Refeeding syndrome. In Kirk RW, Bonagura JD, editors: *Current veterinary therapy XIII*, Philadelphia, 1999, Saunders, p 89.
200. Mori A, Sako T, Lee P et al: Comparison of three commercially available prescription diet regimens on short-term post-prandial serum glucose and insulin concentrations in healthy cats, *Vet Res Commun* 33:669, 2009.
201. Morris JG: Idiosyncratic nutrient requirements of cats appear to be diet-induced evolutionary adaptations, *Nutr Rev* 15:153, 2002.
202. Nelson RW, DiPietro ME, Long GG: Lymphocytic-plasmacytic colitis in the cat, *J Am Vet Med Assoc* 184:1133, 1984.
203. Nelson RW, Scott-Moncrieff JC, Feldman EC et al: Effect of dietary insoluble fiber on control of glycemia in cats with naturally acquired diabetes mellitus, *J Am Vet Med Assoc* 216:1082, 2000.
204. Nouvenne A, Meschi T, Prati B et al: Effects of a low-salt diet on idiopathic hypercalciuria in calcium-oxalate stone formers: a 3-mo randomized controlled trial, *Am J Clin Nutr* 91:565, 2010.
205. Ogilvie GK, Fettman MJ, Mallinckrodt CH et al: Effect of fish oil, arginine, and doxorubicin chemotherapy on remission and survival time for dogs with lymphoma: a double-blind, randomized placebo-controlled study, *Cancer* 88:1916, 2000.
206. Ogilvie GK, Vail DM: Nutrition and cancer. Recent developments, *Vet Clin North Am Small Anim Pract* 20:969, 1990.
207. Olczak J, Jones BR, Pfeiffer DU et al: Multivariate analysis of risk factors for feline hyperthyroidism in New Zealand, *N Z Vet J* 53:53, 2005.
208. Osborne CA, Lulich JP, Bartges JW et al: Medical dissolution and prevention of canine and feline uroliths: diagnostic and therapeutic caveats, *Vet Rec* 127:369, 1990.
209. Osborne CA, Lulich JP, Kruger JM et al: Medical dissolution of feline struvite urocystoliths, *J Am Vet Med Assoc* 196:1053, 1990.
210. Osborne CA, Lulich JP, Kruger JM et al: Analysis of 451,891 canine uroliths, feline uroliths, and feline urethral plugs from 1981 to 2007: perspectives from the Minnesota Urolith Center, *Vet Clin North Am Small Anim Pract* 39:183, 2009.
211. Palma D, Langston C, Gisselman K et al: Feline struvite urolithiasis, *Compend Contin Educ Vet* 31:542, 2009.
212. Perea SC: Critical care nutrition for feline patients, *Top Companion Anim Med* 23:207, 2008.
213. Pion PD, Kittleson MD, Rogers QR et al: Myocardial failure in cats associated with low plasma taurine: A reversible cardiomyopathy, *Science* 237:764, 1987.
214. Plantinga EA, Beynen AC: A case-control study on the intake of polyunsaturated fatty acids and chronic renal failure in cats, *J Appl Res Vet Med* 1:127, 2003.
215. Plotnick A: Feline chronic renal failure: long-term medical management, *Compend Contin Educ Vet* 29:342, 2007.
216. Polzin DJ, Osborne CA, Ross S et al: Dietary management of feline chronic renal failure: where are we now? In what direction are we headed? *J Feline Med Surg* 2:75, 2000.
217. Powell-Tuck J: Nutritional interventions in critical illness, *Proc Nutr Soc* 66:16, 2007.
218. Pratt A: Effect of commercial diets on cats with chronic renal insufficiency, *Vet Rec* 157:455, 2005.
219. Pukay BP: A hyperglycemia-glucosuria syndrome in cats following megestrol acetate therapy, *Can Vet J* 20:117, 1979.

220. Puschner B, Poppenga RH, Lowenstein LJ et al: Assessment of melamine and cyanuric acid toxicity in cats, *J Vet Diagn Invest* 19:616, 2007.
221. Radin MJ, Sharkey LC, Holycross BJ: Adipokines: a review of biological and analytical principles and an update in dogs, cats, and horses, *Vet Clin Pathol* 38:136, 2009.
222. Rajala MW, Scherer PE: Minireview: The adipocyte—at the crossroads of energy homeostasis, inflammation, and atherosclerosis, *Endocrinology* 144:3765, 2003.
223. Rand JS, Fleeman LM, Farrow HA et al: Canine and feline diabetes mellitus: nature or nurture? *J Nutr* 134:2072S, 2004.
224. Rees TM, Lubinski JL: Oral supplementation with L-lysine did not prevent upper respiratory infection in a shelter population of cats, *J Feline Med Surg* 10:510, 2008.
225. Reid CL: Nutritional requirements of surgical and critically-ill patients: do we really know what they need? *Proc Nutr Soc* 63:467, 2004.
226. Reiter AM, Lewis JR, Okuda A: Update on the etiology of tooth resorption in domestic cats, *Vet Clin North Am Small Anim Pract* 35:913, 2005.
227. Reiter AM, Lyon KF, Nachreiner RF et al: Evaluation of calcitonin hormones in cats with odontoclastic resorptive lesions, *Am J Vet Res* 66:1446, 2005.
228. Remillard RL: Homemade diets: attributes, pitfalls, and a call for action, *Top Companion Anim Med* 23:137, 2008.
229. Remillard RL, Saker KE: Parenteral-assisted feeding. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute, p 477.
230. Richter M, Schudel L, Tobler K et al: Clinical, virological, and immunological parameters associated with superinfection of latently FeHV-1 infected cats, *Vet Microbiol* 138:205, 2009.
231. Rios L, Ward C: Feline diabetes mellitus: diagnosis, treatment, and monitoring, *Compend Contin Educ Vet* 30:626, 2008.
232. Robertson JE, Christopher MM, Rogers QR: Heinz body formation in cats fed baby food containing onion powder, *J Am Vet Med Assoc* 212:1260, 1998.
233. Root MV, Johnston SD, Olson PN: Effect of prepuberal and post-puberal gonadectomy on heat production measured by indirect calorimetry in male and female domestic cats, *Am J Vet Res* 57:371, 1996.
234. Ross LA, Finco DR, Crowell WA: Effect of dietary phosphorus restriction on the kidneys of cats with reduced renal mass, *Am J Vet Res* 43:1023, 1982.
235. Ross SJ, Osborne CA, Kirk CA et al: Clinical evaluation of dietary modification for treatment of spontaneous chronic kidney disease in cats, *J Am Vet Med Assoc* 229:949, 2006.
236. Ross SJ, Osborne CA, Lekcharoensuk C et al: A case-control study of the effects of nephrolithiasis in cats with chronic kidney disease, *J Am Vet Med Assoc* 230:1854, 2007.
237. Ross SJ, Osborne CA, Lulich JP et al: Canine and feline nephrolithiasis. Epidemiology, detection, and management, *Vet Clin North Am Small Anim Pract* 29:231, 1999.
238. Rumbeisha WK, Francis JA, Fitzgerald SD et al: A comprehensive study of Easter lily poisoning in cats, *J Vet Diagn Invest* 16:527, 2004.
239. Russell K, Sabin R, Holt S et al: Influence of feeding regimen on body condition in the cat, *J Small Anim Pract* 41:12, 2000.
240. Saker KE, Selting KA: Cancer. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute, p 588.
241. Scarlett JM, Donoghue S: Associations between body condition and disease in cats, *J Am Vet Med Assoc* 212:1725, 1998.
242. Scarlett JM, Donoghue S, Saidla J et al: Overweight cats: prevalence and risk factors, *Int J Obes Relat Metab Disord* 18(Suppl 1):S22, 1994.
243. Scott DW, Paradis M: A survey of canine and feline skin disorders seen in a university practice: Small Animal Clinic, University of Montreal, Saint-Hyacinthe, Quebec (1987-1988), *Can Vet J* 31:830, 1990.
244. Seaman R, Bartges JW: Canine struvite urolithiasis, *Compend Contin Educ Pract Vet* 23:407, 2001.
245. Seim HB, Bartges JW: Enteral and parenteral nutrition. In Tams TR, editor: *Handbook of small animal gastroenterology*, ed 2, Philadelphia, 2003, Saunders, p 416.
246. Simopoulos AP: Omega-3 fatty acids in inflammation and autoimmune diseases, *J Am Coll Nutr* 21:495, 2002.
247. Simpson JW: Diet and large intestinal disease in dogs and cats, *J Nutr* 128:2717s, 1998.
248. Slingerland LI, Fazilova VV, Plantinga EA et al: Indoor confinement and physical inactivity rather than the proportion of dry food are risk factors in the development of feline type 2 diabetes mellitus, *Vet J* 179:247, 2009.
249. Smith BH, Stevenson AE, Markwell PJ: Urinary relative supersaturations of calcium oxalate and struvite in cats are influenced by diet, *J Nutr* 128:2763S, 1998.
250. Smith SA, Ludlow CL, Hoskinson JJ et al: Effect of percutaneous endoscopic gastrostomy on gastric emptying in clinically normal cats, *Am J Vet Res* 59:1414, 1998.
251. Stenske KA, Smith JR, Newman SJ et al: Aflatoxicosis in dogs and dealing with suspected contaminated commercial foods, *J Am Vet Med Assoc* 228:1686, 2006.
252. Stiles J, Townsend WM, Rogers QR et al: Effect of oral administration of L-lysine on conjunctivitis caused by feline herpesvirus in cats, *Am J Vet Res* 63:99, 2002.
253. Stiver SL, Frazier KS, Mauel MJ et al: Septicemic salmonellosis in two cats fed a raw-meat diet, *J Am Anim Hosp Assoc* 39:538, 2003.
254. Stokes JE, Forrester SD: New and unusual causes of acute renal failure in dogs and cats, *Vet Clin North Am Small Anim Pract* 34:909, 2004.
255. Stoller ML, Chi T, Eisner BH et al: Changes in urinary stone risk factors in hypocitraturic calcium oxalate stone formers treated with dietary sodium supplementation, *J Urol* 181:1140, 2009.
256. Stokey GK, Warrick JM, Miller LL: Effect of sodium hexametaphosphate on dental calculus formation in dogs, *Am J Vet Res* 56:913, 1995.
257. Syme HM, Barber PJ, Markwell PJ et al: Prevalence of systolic hypertension in cats with chronic renal failure at initial evaluation, *J Am Vet Med Assoc* 220:1799, 2002.
258. Syme HM, Markwell PJ, Pfeiffer D et al: Survival of cats with naturally occurring chronic renal failure is related to severity of proteinuria, *J Vet Intern Med* 20:528, 2006.
259. Thatcher CD, Hand MS, Remillard RL: Small animal clinical nutrition: an iterative process. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute.
260. Thiess S, Becskei C, Tomsa K et al: Effects of high carbohydrate and high fat diet on plasma metabolite levels and on i.v. glucose tolerance test in intact and neutered male cats, *J Feline Med Surg* 6:207, 2004.
261. Thiry E, Addie D, Belak S et al: Feline herpesvirus infection. ABCD guidelines on prevention and management, *J Feline Med Surg* 11:547, 2009.
262. Thomovsky E, Reniker A, Backus R et al: Parenteral nutrition: uses, indications, and compounding, *Compend Contin Educ Vet* 29:76, 2007.
263. Thompson K, Bartges JW, Snow P: Gastrostomy feeding tubes: percutaneous, nonsurgical, nonendoscopic placement, *Vet Med* 99:619, 2004.
264. Thompson MS, Cohn LA, Jordan RC: Use of rutin for medical management of idiopathic chylothorax in four cats, *J Am Vet Med Assoc* 215:345, 1999.

265. Toll PW, Yamka RM, Schoenherr WD et al: Obesity. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute, p 501.
266. Vannatta M, Bartges JW, Snow P: Esophagostomy feeding tubes, *Vet Med* 99:596, 2004.
267. Vasconcellos RS, Borges NC, Goncalves KN et al: Protein intake during weight loss influences the energy required for weight loss and maintenance in cats, *J Nutr* 139:855, 2009.
268. Villaverde C, Ramsey JJ, Green AS et al: Energy restriction results in a mass-adjusted decrease in energy expenditure in cats that is maintained after weight regain, *J Nutr* 138:856, 2008.
269. von Werthern CJ, Wess G: A new technique for insertion of esophagostomy tubes in cats, *J Am Anim Hosp Assoc* 37:140, 2001.
270. Wakeling J, Everard A, Brodbelt D et al: Risk factors for feline hyperthyroidism in the UK, *J Small Anim Pract* 50:406, 2009.
271. Walberg J: Idiopathic chylothorax in a cat, *J Am Vet Med Assoc* 182:525, 1983.
272. Watson AD: Diet and periodontal disease in dogs and cats, *Aust Vet J* 71:313, 1994.
273. Xu H, Laflamme DP, Long GL: Effects of dietary sodium chloride on health parameters in mature cats, *J Feline Med Surg* 11:435, 2009.
274. Yang VK, Freeman LM, Rush JE: Comparisons of morphometric measurements and serum insulin-like growth factor concentration in healthy cats and cats with hypertrophic cardiomyopathy, *Am J Vet Res* 69:1061, 2008.
275. Yhee JY, Brown CA, Yu CH et al: Retrospective study of melamine/cyanuric acid-induced renal failure in dogs in Korea between 2003 and 2004, *Vet Pathol* 46:348, 2009.
276. Young CD, Anderson SM: Sugar and fat—that's where it's at: metabolic changes in tumors, *Breast Cancer Res* 10:202, 2008.
277. Yu S, Paetau-Robinson I: Dietary supplements of vitamins E and C and beta-carotene reduce oxidative stress in cats with renal insufficiency, *Vet Res Commun* 30:403, 2006.
278. Zetner K, Steurer I: The influence of dry food on the development of feline neck lesions, *J Vet Dent* 9:4, 1992.
279. Zicker SC, Nelson RW, Kirk CA et al: Endocrine disorders. In Hand MS, Thatcher CD, Remillard RL et al, editors: *Small animal clinical nutrition*, ed 5, Topeka, Kan, 2010, Mark Morris Institute, p 559.
280. Zoran D: Is it IBD? Managing inflammatory disease in the feline gastrointestinal tract, *Vet Med* 95:128, 2000.
281. Zoran DL: Feline obesity: recognition and management, *Compend Contin Educ Vet* 31:284, 2009.
282. Zoran DL: Obesity in dogs and cats: a metabolic and endocrine disorder, *Vet Clin North Am Small Anim Pract* 40:221, 2010.