Nutritional Plan: Matching Diet to Disease

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Institution of appropriate, timely nutritional support in the anorexic or critically ill patient has become accepted medical practice in people and animals. This article focuses on the benefits of appropriate nutrient intake in critically ill animals, recommended nutrient requirements for dogs and cats receiving enteral feeding, and mechanics of food preparation and delivery for a variety of feeding tubes. General nutrient requirements for all patients, specific recommendations for certain illnesses such as renal failure, pancreatitis, and hepatic disease, and nutritional alterations for critical illness are reviewed. Commercial liquid diets manufactured for people and pets, and pet-food diets practical for formulation of gruel are presented. Institution of and weaning from feeding are explained.

Food is love. Although this adage learned from our grandmothers may not be completely correct, there is indisputable evidence that malnutrition is detrimental to health and healing.[1] Provision of appropriate nutritional support for critically ill humans and animals improves organ function, decreases infections, hospital stay and cost, and improves overall outcome.[1,2] Goals of nutritional support should include satisfaction of nutritional requirements with concurrent anticipation and prevention of associated complications.[3]

An understanding of the detrimental effects of inadequate nutritional support on critically ill patients requires recognition of the difference between simple starvation and stressed starvation. Simple starvation occurs in normal, healthy animals when denied nutritional intake. During simple starvation the body undergoes multiple adaptations that ultimately result in a decreased metabolic rate, resulting in energy conservation to maintain body mass. These adaptations include increased production of inactive triiodothyronine (T3) compared with active thyroid hormones, and utilization of glucose and fat as the primary sources of energy. In contrast to simple starvation, stressed starvation occurs during illness or injury. Counterregulatory hormones such as glucagon, epinephrine, and inflammatory cytokines are released during serious illness. These hormones alter the physiologic response to starvation; energy expenditure does not decline appropriately and tissue catabolism results.[1,4]

Human studies have documented hypermetabolic states with specific disease processes such as neoplasia, thermal burns, and septicemia. An inverse relationship between body-mass index and mortality in critically ill patients suffering from acute respiratory failure, multiple organ failure, congestive heart failure, and coma has been established.[2,5] The deleterious effects of negative energy balance on critically ill patients occur in multiple body systems (Table 1).[3]

The detrimental effects of malnutrition during serious illness should prompt the clinician to diligently provide appropriate nutritional support for critically ill patients. Selecting the appropriate diet as part of a feeding protocol will be influenced by multiple factors including clinical status of the patient, nature of the disease process, availability of certain diets, and route of administration. In patients with a functional gastrointestinal tract, studies clearly demonstrate that enteral nutrition is the preferred method of nutrient delivery.[5-7] In other words, if the gut works, use it. If a part of the gut works, then use the part that works. This article will focus on the appropriate nutrient composition and consistency of food for various feeding devices and medical conditions, recommended feeding schedules, and return to voluntary food intake.

Meeting Nutritional Needs

Feeding protocols should be tailored to meet the needs of each individual patient, as determined by the clinician's assessment of historical and physical examination parameters, body weight and body condition score, interpretation of complete blood count and serum biochemical profile, acid/base status, and the particular disease process diagnosed. Nutrient requirements include water and electrolytes, energy-supplying nutrients (eg, carbohydrates, lipids, and proteins), minerals, and vitamins.[8]

Before initiation of a specific feeding protocol, the patient should be euvolemic, hydrated, and electrolyte abnormalities should be resolved. Hypovolemia should be addressed with appropriate therapy and monitoring tailored for each individual. Ongoing fluid requirements should be calculated to satisfy maintenance needs, correct dehydration, and address ongoing loss.[9] Feeding should be delayed until preexisting fluid and electrolyte abnormalities are corrected to avoid exacerbating gastrointestinal hypoxia secondary to increasing cellular metabolism, and to prevent hypophosphatemia and hypokalemia related to refeeding syndrome (see article by Dr. Michel, pp. 49-53, this issue).[10] The energy-supplying nutrients, carbohydrates (including starches, celluloses, and oligosaccharides), lipids (fats), and proteins should be provided in appropriate ratios for the given species (Table 2).[8,11,12]

Carbohydrates play important roles in health maintenance. Disruption of homeostasis may alter the ability to utilize or synthesize carbohydrates appropriately. Starches are used as an energy source. Insoluble fibers such as cellulose (also referred to as crude fiber or total fiber) promote normal mechanical function of the gastrointestinal tract, and soluble fiber main-

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TABLE 1. Deleterious Effects of Negative Energy Balance on Various Body Systems

Body System	Effect of Negative Energy Balance
Gastrointestinal	Increased transit times, villous atrophy, reduction in absorptive capabilities, increased risk of bacterial translocation
Renal	Increased urinary calcium and phosphorus excretion, decreased ability to excrete acid, increased gluconeogenesis, decreased glomerular filtration rate
Immune	Decreased humoral immunity, decreased barrier function (skin and mucosal surfaces), decreased inflammatory response, decreased leukocyte motility and bactericidal activity
Pulmonary	Decreased response to hypoxia, decreased lung elasticity, decreased secretion production, altered permeability, decreased tidal volume
Cardiovascular	Increased incidence of arrhythmias, decreased weight of the heart muscle
Musculoskeletal	Increased muscle weakness, increased muscle wasting

tains intestinal hygiene. Oligosaccharides act through fermentation and are a direct source of nutrients for the large intestines. Fermentation promotes the growth of normal gastrointestinal bacterial flora while inhibiting the growth of pathogenic bacteria, and improves digestion and nutrient absorption. Fermentation also helps to maintain an intact gastrointestinal mucosal barrier, which plays a vital role in preventing bacterial translocation from the gut to the systemic circulation.[8]

Lipids (including omega 3 and omega 6 fatty acids) are a primary source of energy for cats and dogs (1 g lipid = 9 kcal energy) and a have an important structural role in cellular membranes and lipoproteins. Omega 3 and 6 fatty acids are not synthesized by the body, and are therefore essential fatty acids. Omega 3 fatty acids have anti-inflammatory properties that inhibit the synthesis of chemical inflammatory mediators. Omega 6 fatty acids are essential for prostaglandin synthesis and some hormonally active molecules, and they have a positive effect on the immune barrier function of skin.[8]

Proteins are molecules composed of amino acids connected by chemical bonds. The arrangement of these bonds results in a 3-dimensional configuration that determines the nature and role of each protein. Proteins have numerous functions, including rebuilding and maintaining tissues and serving as carrier molecules, message transmitters, enzymes, antibodies, and sources of energy. A decrease in protein availability in a sick or injured animal that causes a negative nitrogen balance compromises the ability of the body to heal or fight infection.[8]

The following 3 amino acids are of special importance in critical veterinary patients: arginine, taurine, and glutamine. Arginine is a required intermediate of the urea cycle, and it is important in wound healing and immune function. Arginine is an essential amino acid in both cats and dogs; however, cats demonstrate clinical signs of deficiency more rapidly than dogs. Arginine is an intermediary in the urea cycle, and its absence leads to hyperammonemia from failure of urea production. Ultimately, hepatic encephalopathy occurs. Clinical signs associated with hepatic encephalopathy include vomiting, ataxia, vocalization, hypersalivation, and tremors. For cats, serious signs can develop within hours of consuming an arginine-deficient meal.[3,10,13]

Taurine is an amino acid found in body tissues and cells, including the brain, retina, myocardium, liver, platelets, leukocytes, and skeletal muscle. It is required by the liver to synthesize bile acids to aid in the absorption of dietary fats. Other functions include regulation of calcium flux, neurotransmission, antioxidant function, and stabilization of cell membranes. Cats lack the ability to synthesize taurine and sustain ongoing losses in the bile and feces, making taurine an essential amino acid in this species. Although the exact mechanism is unknown, taurine is thought to play an important role in bioelectrical potentials for the myocardium and retina. Clinical signs associated with taurine deficiency include reproductive failure, developmental abnormalities, retinal degeneration, and dilated cardiomyopathy.[10,12,14]

Glutamine, the most abundant amino acid in blood and body tissues, is considered nonessential in times of health. This amino acid becomes essential during disease and illness, however, because the glutamine synthesis pathway becomes attenuated.[10] When this occurs, the pool of available glutamine becomes significantly decreased. Glutamine plays a role in nitrogen transport, gluconeogenesis, RNA and DNA synthesis, and renal ammoniogenesis. It is a key fuel for rapidly dividing cells such as enterocytes, endothelial cells, renal tubular cells, and lymphocytes. There is an inter-organ glutamine cycle to mobilize glutamine stores in muscle and provide glutamine for mucosal repair, to generate substrate for renal ammoniogenesis, and to support lymphocyte function.[5,10,15,16] Simple starvation, metabolic acidosis, trauma, sepsis, endotoxemia, and malignant neoplasia alter the stability of this cycle by changing the ability of various organs to release or uptake glutamine (Table 3).[16] Because glutamine is an important fuel for rapidly dividing cells, enterocytes may not be able to heal and regenerate appropriately after injury. Clinical signs of glutamine depletion include protein catabolism leading to muscle wasting and cachexia, decreased gastrointestinal barrier function with increased bacterial translocation, and immune-system dysfunction with stimulation of inflammatory cytokines. The combination of these negative consequences can lead to multiorgan dysfunction.[17] Supplementation of glutamine in humans has been associated with shorter gastrointestinal healing times after radiotherapy or chemotherapy.[18,19]

TABLE 2. General Requirements for Protein, Fat, and Carbohydrate for Normal Healthy Cats and Dogs (DM = dry matter basis)

Nutrient	Cats	Dogs
Protein	30% to 45% of DM	15% to 30% of DM
Fat	10% to 30% of DM	10% TO 20% OF DM
Carbohydrates	<50% of DM	50% OF DM

Diet Selection and Preparation

The most physiologically sound, cost effective, and safest means of providing nutritional support is voluntary oral intake. When this is not possible, then nutritional support can be provided by enteral tube feeding. There are various enteral feeding methods available, including nasoesophageal, nasogastric, esophagostomy, gastrostomy, jejunostomy, nasojejunal, and gastrojejunal feeding tubes. Placement of these devices has

TABLE 3. Glutamine Release or Uptake in Various Body Tissues During Health and Disease

Cell Type	Health	Simple Starvation	Metabolic Acidosis	Trauma	Sepsis Endotoxemia	Malignancy
Lung	Release	Release	Release	Increased release	Increased release	Release
Skeletal muscle	Release	Release	Release	Increased release	Increased release	Increased Release
Intestine	Absorption	Increased absorption	Decreased absorption	Increased absorption	Decreased absorption	Decreased Absorption
Liver	Absorption	Release	Release	Increased absorption	Increased absorption	Increased Release
Kidney	Absorption	Increased absorption	Increased absorption	Absorption	Absorption	Absorption
Lymphocyte macrophage	Absorption	Absorption	Absorption	Absorption	Increased absorption	Absorption
Tumor	—	—	—	—	—	Increased absorption

been discussed elsewhere (see the article by Dr. Han, pp. 22-31, this issue and Dr. Heuter, pp. 32-42, this issue). Although there is no single ideal enteral formulation for all animals, there are some desirable characteristics for an enteral formula. These characteristics include isotonicity, caloric density of 1.0 kcal/mL or greater, protein content of at least 4.0 g/100 kcal or 16% of total calories, 30% of calories from fat, and a fiber component of 1.0-1.5 g/100 kcal.[5,7]

As veterinary medicine has evolved and the sophistication of care that clients demand for pets has increased, the number of enteral veterinary diets has grown tremendously. Diet selection is based on the disease process as discussed below, and also on the formulation, nutrient composition, and consistency of the diet. Prescription diets have been specifically formulated to address a multitude of health issues in dogs and cats. Based on formulation, diets are classified as either polymeric (containing intact protein, polysaccharides, and long-chain triglycerides), or monomeric (containing nutrients in small, hydrolyzed absorbable forms). Monomeric or elemental diets are liquid diets containing nutrients that are readily available for absorption, with little or no digestion required. These diets are primarily composed of amino acids, glucose, and oligosaccharides, and also include a source of fatty acids essential for humans. These diets are sometimes indicated for treatment of severe gastrointestinal diseases such as pancreatitis, short bowel syndrome, severe inflammatory bowel disease, or demonstrated intolerance of a polymeric diet. Disadvantages associated with the use of monomeric diets include expense, dietary intolerance, and diarrhea caused by high osmolarity. There are no monomeric diets formulated for dogs and cats. Commercially available human products include Peptamen® (Baxter Health Care, Deerfield, IL), Vivonex HN® (Norwich-Eaton Pharmaceuticals, Norwich, NY), and Vital HN® (Ross Products Division, Abbot Laboratories, Abbot Park, IL).[20]

All of the commercially available pet-food diets are polymeric diets. Routinely, polymeric diets are utilized for enteral feeding because they are readily available, more affordable, and well tolerated by most animals.[7] Examples of polymeric diets used for enteral nutrition are outlined in Table 4. Theoretically, monomeric diets should be preferable for jejunal feeding, wherein the absorptive functions of the upper gastrointestinal tract are bypassed. However, polymeric diets seem to be well tolerated by veterinary patients with jejunal feeding tubes.

Diet selection is also based on the size of the tube chosen for nutrient delivery. Large-bore tubes (esophagostomy and gastrostomy tubes) will accommodate either liquid or gruel consistency diets. Iams Eukanuba® Maximum Calorie (Iams Company, Dayton, OH) and Prescription Diet® Canine/Feline a/d® (Hill's Pet Nutrition, Topeka, KS), both classified as veterinary recovery diets, are commonly used for enteral feeding of critically ill dogs and cats.[10] Both of these diets have a smooth consistency that makes them ideal for large-bore tube feeding with minimal preparation. They are both rich in conditionally essential nutrients (taurine, arginine, and omega-3 fatty acids) for critically ill animals and have a caloric density of 1.75 kcal/mL and 1.0 kcal/mL, respectively. These diets have a consistency that makes feeding through 5- and 8-French tubes (eg, nasoesophageal, jejunal) impractical. Refrigeration also causes these foods to thicken. Warming to room temperature and blending with small amounts of water facilitates tube feeding; however, the calorie content per milliliter will decline when water is added (Table 4).

Based on their composition, recovery diets may not be ideal for all hospitalized patients. For example, a patient with pancreatitis should not receive a high-fat diet. Other veterinary diets are available for use both in certain disease processes and general maintenance. Moist diets require blending with water to convert to a gruel that can be administered through a feeding tube. For example, to prepare a gruel using Hill's Prescription Diet® Feline p/d® (Hill's Pet Nutrition), mix half a can (224 g) of Feline p/d with three fourths of a cup (170 mL) of water, blend at high speed for 60 s, and then strain twice through a kitchen strainer (approximately 1-mm mesh) to remove any remaining large food particles (Fig 1). This yields approximately 390 mL of gruel with a caloric density of 0.9 kcal/mL and a protein content of about 11 g/100 kcal. Table 4 lists multiple diets available commercially, caloric density per can, caloric density when prepared as gruel, and some medical conditions for which each diet is indicated. Addition of water facilitates gruel passage through a feeding tube, but decreases the caloric density and therefore increases the volume of food an animal requires.

Small-bore tubes (nasoesophageal, nasogastric, and jejunal feeding tubes) will accommodate only liquid diets. Commercially available polymeric liquid diets formulated specifically for veterinary use include canine or feline CliniCare®, and feline CliniCare RF® (Abbot Laboratories). Examples of polymeric diets formulated for people include Ensure Plus®, Pulmocare®, and Jevity® (Ross Products Division, Abbott Laboratories; Table 4). These diets can be used in dogs; however, they should be used only on a short-term basis because they do not meet canine nutrient requirements. Diets formulated for people

TABLE 4. Canine and Feline Diets, Calorie Contents Per Can and Milliliter of Gruel, and Indications for Use in Enteral Feeding Protocols

Diet	Kcal/can	kcal/mL Blenderized	Osmolarity	Indications
Eukanuba Recovery (6.0-oz can)[A]	340 kcal/can	1.75kcal/mL‡		Tra, Sep, Sx
Hills n/d (canine 14.75-oz can)[B]	538 kcal/can			Tra, Sep, Sx, HP, Neo
p/d (canine 14.75-oz can)	675 kcal/can	0.9 kcal/mL		Tra, Sep, Sx, HP
p/d (feline 14.75-oz can)	663 kcal/can	0.9 kcal/mL*		Tra, Sep, Sx, HP
a/d (5.5-oz can)	197 kcal/can	1.0 kcal/mL***		Tra, Sep, Sx, HP
k/d (canine 14.25-oz can)	527 kcal/can	0.62 kcal/mL†		Kid, Hep, Cv
k/d (feline 14.25-oz can)	584 kcal/can	0.90 kcal/mL†		Kid, Hep, Cv
h/d (canine & feline 14.25-oz)	506 kcal/can			Cv
u/d (canine 14.75-oz can)	593 kcal/can	0.66 kcal/mL†		Uro, Kid, Hep
c/d (feline 5.5-oz can)	162 kcal/can	0.62 kcal/mL†		Uro, Gi
i/d (canine 14.5-oz can)	544 kcal/can	0.57 kcal/mL†		Gi, Pan
l/d (canine 14.75-oz can)	534 kcal/can			Hep
l/d (feline 5.5-oz can)	164 kcal/can			Hep
Purina CV (canine 12.5-oz can)[C]	638 kcal/can			Cv, HP, Tra, Sx
CV (feline 5.5-oz can)	223 kcal/can			Cv, HP, Tra, Sx
NF (canine 12.5-oz can)	500 kcal/can			Kid, Hep, Cv
NF (feline 5.5-oz can)	234 kcal/can			Kid, Hep, Cv
EN (canine 12.5-oz can)	424 kcal/can			Gi, Hep§, Pan
EN (feline 1.5-oz pouch)	117 kcal/can			Gi, Hep§, Pan
CliniCare (canine 8-oz can)[D]	237 kcal/can	1.0 kcal/mL	340 mOsm/kg	
CliniCare RF (feline 8-oz can)	237 kcal/can	1.0 kcal/mL	368 mOsm/kg	
Ensure (8-oz can)[E]	355 kcal/can	1.5 kcal/mL		
Jevity (8-oz can)[F]	250 kcal/can	1.06 kcal/mL	310 mOsm/kg	
Osmolite HN	355 kcal/can	1.06 kcal/mL	310 mOsm/kg	
Vital HN	355 kcal/can	1.0 kcal/mL	460 mOsm/kg	
Vivonex HN[G]	355 kcal/can	1.0 kcal/mL	810 mOsm/kg	

*blenderized 1/2 can + 170 mL water.
†blenderized 1/2 can + 284 mL water.
‡mix well 1 can + 32 mL water.
Abbreviations: Trauma = Tra; Sepsis = Sep; Surgery = Sx; Hypoproteinemia = HP; Neoplasia = Neo; Kidney = Kid; Hepatic = Hep; Hep§ = (not associated with signs of encephalopathy); Cardiovascular = Cv; Urogenital = Uro; Gastrointestinal = Gi; Pancreatitis = Pan
A. Iams Company, Dayton, OH
B. Hill's Pet Products, Topeka KS
C. Purina, St. Louis, MO
D. Abbott Laboratories, Chicago, IL
E. Abbott Laboratories, Columbus, OH
F. Ross Laboratories
G. Norwich-Eaton Pharmaceuticals.

should be used with extreme caution in cats because of their nutritional inadequacy.

Selection of Diet for Special Situations

Certain medical conditions may benefit from dietary therapy that is somewhat altered from general feeding recommendations. Animals with renal disease, gastrointestinal disorders, pancreatitis, hepatic disease, or neoplasia can benefit from diets that are specifically tailored for digestive and metabolic abnormalities caused by or associated with the underlying medical disorder.[21-24] When enteral feeding is required for medical management of individuals with these diseases, the following special considerations can provide a guideline for diet selection.

Renal failure

Normal renal functions include excretion of nitrogenous waste products, regulation of acid–base and electrolyte status, stimulation of erythropoiesis, and regulation of calcium homeostasis.[25] Varying degrees of renal failure result in impairment of one or more of these functions. Appropriate diet therapy can both ameliorate clinical signs of uremia and electrolyte imbalance, and delay the inevitable progression of renal failure.[21,26] Anorexia and nausea occur secondary to the progressive effects of uremia and loss of homeostasis. Nutritional support via the enteral or parenteral route provides essential nutrients to restore homeostasis in patients with acute or chronic renal failure.[27]

Specific dietary considerations for nutritional support in animals with renal failure include controlling dietary protein, phosphorous, calcium, and sodium intake, maintaining serum potassium, and preventing metabolic acidosis.[28] Dogs with naturally occurring chronic renal failure were evaluated for clinical response to a diet specifically formulated to meet the above requirements. Dogs were separated into two groups, with one group consuming a balanced adult maintenance-type diet, and the other group consuming a renal food (Prescription Diet® Canine k/d®, Hill's Pet Nutrition). The group consuming the renal food experienced fewer uremic crises and were less likely to die of renal failure than the control group over a 24-month period.[21] This study is in agreement with previous studies that documented the importance of protein restriction in the long-term medical management of renal failure. Advantages of diets specifically formulated for renal disease include amelioration of clinical signs of uremia, restriction of dietary phosphorous to minimize renal secondary hyperparathyroidism, reduction of acid production by the kidneys, and delay of end-stage renal failure.[28] There are a variety of commercially available canned and dry renal diets for dogs and cats that can be adapted for enteral feeding via esophagostomy or gastrostomy tube (Table 4). Renal diets contain significantly reduced protein compared with maintenance diets. A liquid protein-restricted diet for cats (Abbot CliniCare RF Feline®) is suitable for feeding via a naso-

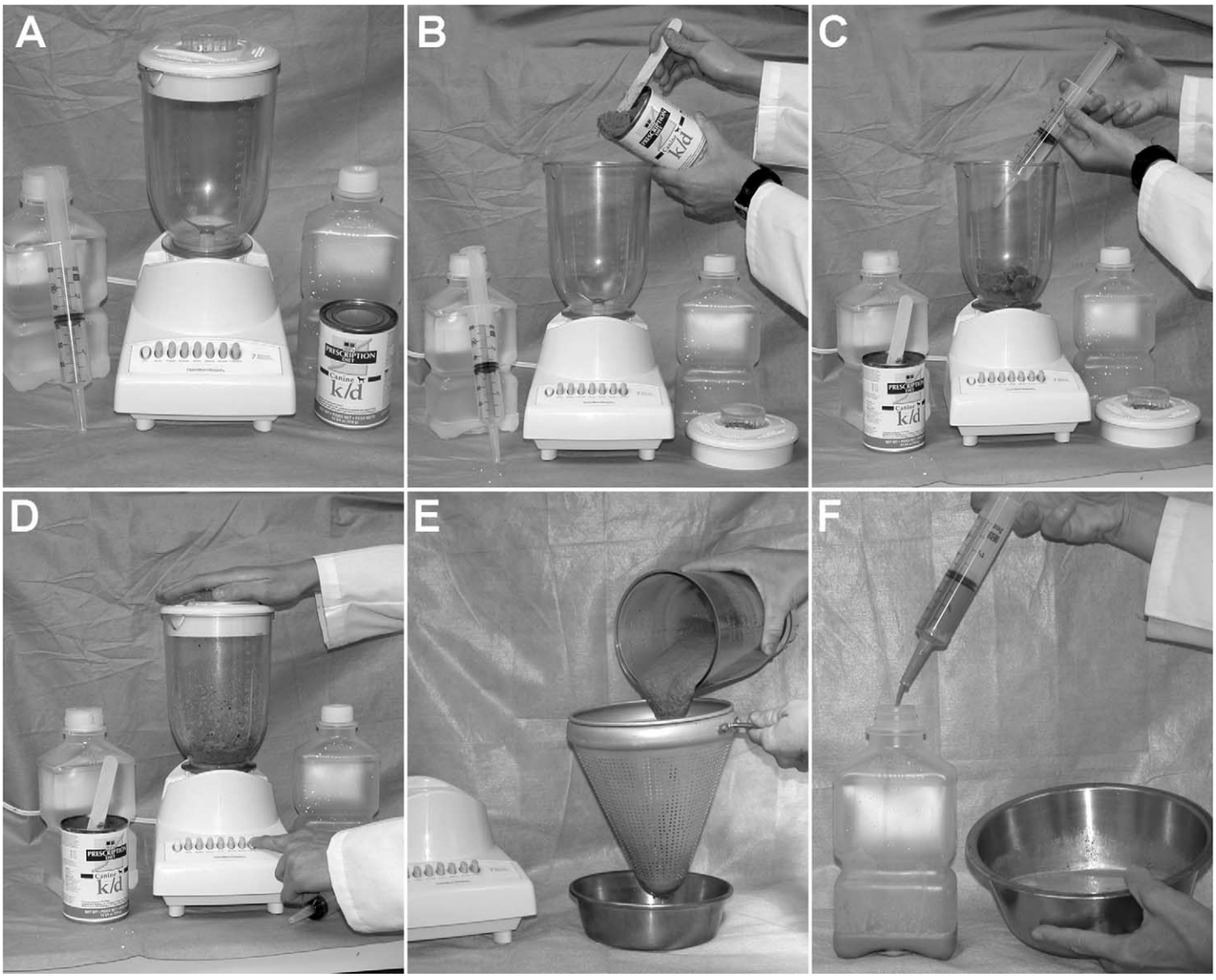

Fig 1. Steps to prepare gruel from canned pet food: a) Assemble ingredients: selected diet, blender, water, measuring device for water, clean containers for gruel; b) Measure food into blender jar; c) Add measured amount of water; d) Blend until smooth; e) Strain gruel twice to remove particles; f) Place in storage container.

esophageal or jejunal feeding tube. This diet contains about two thirds the amount of protein as the liquid diet product manufactured for adult cats without renal failure.[10]

Gastrointestinal Disease

Dietary modification for gastrointestinal diseases provides a powerful tool for clinical management.[22,29] Certain diets contribute to gastrointestinal disease in particular animals by containing allergenic or antigenic proteins for that individual, and by altering absorption, motility, enzyme secretion, luminal ammonia production, or colonic volatile fatty-acid content.[29] Animals with gastrointestinal disease can present acutely or chronically, with inappetance, weight loss, vomiting, or diarrhea. For purposes of this discussion, nutritional recommendations for chronic small-bowel diarrhea will be included, because acute gastroenteritis is likely to resolve before the need for enteral feeding, and large-bowel diarrhea rarely requires enteral nutritional support.

Diets designed for the management of chronic small-bowel diarrhea should be composed of highly digestible food sources, with high biologic value protein. Limiting proteins and carbohydrates to one or two novel sources facilitates identification of dietary hypersensitivity to certain foods. Dietary fat digestion is frequently compromised in animals with small-bowel disease; therefore, moderate fat restriction is recommended. Small quantities of soluble or mixed fiber are recommended for distal small-bowel and colonic health. General functions of dietary fiber include modifying gastric emptying, normalizing intestinal motility and transit rate, buffering gastric acid and intraluminal toxins, binding excess water, promoting normal gastrointestinal flora, and altering viscosity of gastrointestinal luminal contents.[22,29] Weight loss is a common feature of small-intestinal disease, and should be addressed by insuring appropriate calorie and nutrient intake. Small, frequent feedings are often better tolerated by animals with small-intestinal disease.[29] A variety of commercial dry and moist diets that meet the above recommendations are available for dogs and cats. Moist diets commonly recommended for small canine small-intestinal disease include Hill's Prescription Diet® Canine i/d® (Hill's Pet Nutrition), Iams Eukanuba® Low Residue® Canine (Iams

Company), and Purina CNM® and Canine E/N-Formula® (Nestle-Purina, St. Louis, MO). Feline moist products include Hill's Prescription diet® Feline i/d®, Iams Eukanuba Low-Residue Feline®, and Purina CNM® Feline EN-formula.[22]

Pancreatitis

Pancreatitis is a disease of widely varying severity. Definitive diagnosis is difficult and often based on consistent historical and physical examination findings and supportive diagnostic testing. Classic clinical findings of pancreatitis in dogs include being middle-aged and overweight, with acute or chronic vomiting, anorexia, and abdominal pain. Complete blood count and serum biochemical findings consistent with pancreatitis in dogs include inflammatory leukogram, elevated transaminases, total bilirubin, lipemia, hypocalcemia, and elevated amylase and lipase. Findings on diagnostic imaging include radiographic evidence of loss of detail in the right cranial quadrant and duodenal ileus, and ultrasonographic mottled echotexture in the pancreatic region. If ascites is present, then a sterile suppurative exudate would be expected. Idiopathic hypertriglyceridemia and high dietary fat intake have been associated with pancreatitis in dogs.[30] Cats with pancreatitis are more challenging to diagnose because clinical findings and supportive diagnostics are widely variable.[31] Weight loss and anorexia might be the only signs associated with chronic pancreatitis, whereas acute pancreatitis may present with similar findings to the dog.[22,30] Feline pancreatitis may occur concurrently with diabetes mellitus, hepatic lipidosis, cholangiohepatitis, or inflammatory bowel disease.[22,31,32]

The mainstay of treatment for pancreatitis in dogs consists of supportive care and minimization of pancreatic secretion. This goal can be met by either postgastric enteral feeding or parenteral nutrition. Jejunal feeding causes less pancreatic stimulation than gastric feeding of similar diets. Diets for animals with pancreatitis should be highly digestible with moderate protein and fat restriction, because these nutrients have been shown to increase pancreatic secretion more than carbohydrates. Most commercial diets suitable for gastrointestinal disease are appropriate for animals with pancreatitis.[22] Jejunal diets must be liquid because jejunal feeding tubes are typically of small diameter (5-8 French). Commercial veterinary liquid diets can be used for jejunal feeding; however, appropriate selection for the species being treated is critical because the feline product is relatively higher in fat than the canine product. Alternatively, monomeric liquid diets can be used for jejunal feeding.[22,30]

Relatively little is known about appropriate feeding for cats with documented pancreatitis. In the authors' experience, cats with suspected pancreatitis tolerate nasogastric feedings well with minimal exacerbation of clinical signs. The commercially available feline liquid diet for renal failure should be avoided in cats with pancreatitis because of its high-fat content. Jejunal feeding or parenteral nutrition can be instituted in individuals with severe clinical signs that do not tolerate nasogastric feeding.[31]

Liver Failure

The liver performs many unique metabolic functions to maintain homeostasis. These include digestion and metabolism of nutrients and vitamins, maintenance of immunologic defense, hormone metabolism, hematologic functions, detoxification, and mineral and energy storage.[33] Liver disease can affect any of these normal functions, and can be acute or chronic in onset, with a wide range of severity. Acute etiologies include various infectious organisms, drug or toxin insult, circulatory abnormalities, idiopathic hepatic lipidosis, inflammatory and neoplastic diseases, and hypoxic injury.[34] Chronic diseases include breed-related storage abnormalities, chronic inflammation, neoplasia, chronic biliary obstruction, and end-stage fibrosis.[35] Depending on the underlying etiology, acute liver failure may be reversed with time and appropriate treatment. The liver has the ability to regenerate after acute injury, and it has a large functional reserve capacity.[34] Chronic liver disease is frequently incurable; however, long-term dietary therapy can improve some animals' quality of life by controlling secondary effects including hypoproteinemia, hypoglycemia, and hepatic encephalopathy. Liver disease causes anorexia, and certain diseases such as cirrhosis induce a catabolic state that causes muscle wasting and weight loss. Hepatic carbohydrate metabolism functions include glucose uptake, glycogen synthesis and storage, glycogenolysis, and gluconeogenesis, and are instrumental to maintaining euglycemia. Hepatic failure can result in hypoglycemia. Hepatic protein synthesis provides 20% of total body protein turnover, and 25% of all protein synthesized by the liver is albumin. Hepatic protein synthesis is influenced primarily by amino-acid availability. Liver failure commonly results in hypoalbuminemia.[33] Hepatic encephalopathy is a disorder of the central nervous system that develops in association with hepatic insufficiency. In general, hepatic dysfunction results in increased production of hepatotoxins (ammonia, glutamine, aromatic amino acids, short-chain fatty acids, false neurotransmitters, and gaba-aminobutyric acid) with decreased toxin degradation and increased toxin accumulation. Toxins are derived primarily from the failure of dietary protein metabolism and enter the systemic circulation to cause impaired cerebral function.[33] Clinical signs can be acute or chronic, and include anorexia, lethargy, depression, mental dullness, seizures, coma, or death.[35]

General goals for nutritional support include preventing malnutrition, reducing hepatic workload, and avoiding the production of neurotoxins that exacerbate hepatic encephalopathy.[23,29,35] These goals can be achieved by selecting a diet that provides adequate energy with increased simple and complex carbohydrates. Protein should be reduced if protein intolerance (hepatic encephalopathy) is evident. Contrary to recommendations of protein with high biologic quality (meat and fish protein) with renal failure, protein sources with hepatic disease should consist of dairy and vegetable proteins. These proteins have lower concentrations of encephalopathic components and increased dietary fiber to limit gastrointestinal ammoniogenesis.[35] There is no universal recommendation to restrict fat intake with hepatic disease. Multiple small-volume feedings might provide a more regular source of nutrients to maintain euglycemia. Enteral nutritional therapy has become the mainstay of therapy for cats with hepatic lipidosis, because anorexia both predisposes to and complicates therapy for this disease.[23,29,35,36] Commercial moist diets available for the management of liver disease in dogs includes Hill's Prescription Diet® Canine l/d® and Purina CNM™ Canine NF-formula™. Feline commercial diets appropriate for liver disease include Hill's Prescription Diet® Feline l/d® and Purina CNM™ Feline NF-Formula™.[23]

Step 1: Calculate resting energy requirement (RER)

For patients of any size: 70 x $(BWKg)^{3/4}$ = RER

70 x (_____)$^{3/4}$ = _____ Kcal/day

For patients between 2 and 30 kg: (30 x BWKg) + 70 = RER

(30 x _____) + 70 = _____ Kcal/day

Step 2: Multiply RER by illness factor to identify metabolizable energy requirement (MER)

Illness factors: Dogs: 1.0 (Mild illness/ injury) to 1.5 (Severe illness/ injury)

Cats: 1.0 (Mild illness/ injury) to 1.25 (Severe illness/ injury)

RER x _____ = _____ Kcal/day

Step 3: Calculate milliliters feeding formula required/ day

MER (Kcal/ day) / Feeding formula (Kcal/ ml) = ml formula / day

_____ / _____ = _____ ml/ day

Step 4: Calculate milliliters per feeding

(Milliliters/day) (Desired number of feedings / day) = Milliliters / feeding

_____ x _____ = _____ml/feeding

Fig 2. Worksheet to calculate energy requirements.

Cancer

Cancer can cause weight loss and malnutrition in animals by a variety of mechanisms including physical factors (eg, reduced food intake because of the physical presence of oral mass, nausea from antineoplastic therapy or gastrointestinal neoplasia), and endocrine or metabolic factors (eg, neoplasia causing gland or organ dysfunction or failure). Cancer cachexia is a term that describes progressive weight loss despite appropriate calorie intake observed with neoplasia in animals and humans.[37,38] Metabolic derangements associated with cancer cachexia include increased resting metabolic rate and altered nutrient metabolism that cause nutrients to be consumed by the tumor at the expense of the host.[24,38] These metabolic derangements result from increased activity of cytokines (interleukin-1, interleukin-6, tumor necrosis factor-α, and interferon-γ) and glucocounter-regulatory hormones by certain types of cancer.[38]

Carbohydrate metabolic alterations are characterized by peripheral insulin resistance resulting from high concentrations of glucose counterregulatory hormones and decreased insulin release. Elevated blood lactate concentrations can be observed, as well as intolerance to administration of exogenous lactate.[39,40] Hyperlactatemia can be caused by the following two mechanisms: poor tissue perfusion, resulting in tissue hypoxia and lactic acid production, and increased lactate production from an unusual source (eg, neoplasia). Tumor cells are not efficient at oxidative metabolism and typically produce lactic acid from anaerobic glycolysis after a carbohydrate challenge. In situations of poor tissue perfusion such as hypovolemic shock, elevated blood lactate concentrations imply tissue hypoxia and are a negative prognostic indicator. When lactate production is unrelated to tissue hypoxia or poor perfusion, the detrimental effects of hyperlactatemia and the relationship of hyperlactatemia to prognosis is unclear.[41] Diets high in simple carbohydrates have been shown to increase mean insulin and lactate levels in dogs with lymphosarcoma compared with dogs fed a diet with more energy from fat.[42]

Lipid metabolism alterations include increased lipolysis and decreased lipogenesis, with decreased lipoprotein lipase activity. These changes result in hyperlipidemia. Protein metabolic derangements include excessive degradation resulting in negative nitrogen balance. Amino acids are utilized for hepatic gluconeogenesis to maintain serum glucose, causing depletion of

Day 1: *Initiate feeding at 1/3 of desired energy requirement*

Metabolizable energy requirement (MER) (Kcal/day) x 1/3 = Kcal/ day, day 1

_____MER x 1/3 = _____ Kcal/day, day 1

Calculate milliliters food/day for day 1

Kcal/day for day 1 / Kcal/ml of food = ml food/day, day 1

_____Kcal/day / _____ Kcal/ml = _____ml food/day 1

Calculate milliliters per feeding for day 1

Milliliters food/day 1 / desired # feedings day 1 = milliliters food/feeding, day 1

_____ml food/day 1 / _____ feedings/day = _____ml food/feeding day 1

Day 2: *Increase feeding to 2/3 of desired energy requirement if patient tolerating feeding*

Metabolizable energy requirement (MER) (Kcal/day) x 2/3 = Kcal/ day, day 2

_____MER x 2/3 = _____ Kcal/day, day 2

Calculate milliliters food/ day for day 2

Kcal/day for day 2 / Kcal/ml of food = ml food/day, day 2

_____Kcal/day 2 / _____ Kcal/ml = _____ml food/day 2

Calculate milliliters per feeding for day 2

Milliliters food/day 2 / desired # feedings day 2 = milliliters food/feeding, day 2

_____ml food/day 2 / _____ feedings/day = _____ml food/feeding day 2

Day 3: *Increase to full energy requirement if patient tolerating feeding*

Calculate milliliters food/ day for day 3 and beyond

Kcal/day MER / Kcal/ml of food = ml food/day, day 3

_____Kcal/day MER / _____ Kcal/ml = _____ml food/day 3 and beyond

Calculate milliliters per feeding for day 3

Milliliters food/day MER / desired # feedings day 3 = ml food/feeding, day 3 and beyond

_____ml food/day 3 / _____ feedings/day = _____ml food/feeding

Fig 3. Worksheet to initiate feeding.

body protein stores.[37] Systemic effects of protein loss include impairments of humoral and cell-mediated immunity, gastrointestinal function, and wound healing.

Metabolic derangements causing cancer cachexia have been associated with testicular, hepatic, colonic, gastric, esophageal, pancreatic, salivary, prostatic, ovarian, renal, nonsmall-lung-cell carcinomas, and T-cell lymphomas in people.[24,43] Similar mechanisms for cancer cachexia are thought to exist for dogs and cats; however, there is no definitive evidence of particular tumors more likely to cause this syndrome in these species. A study evaluating the influences of dietary alterations was completed in dogs with lymphoblastic lymphoma. Longer survival time and disease-free interval was seen in dogs with stage III lymphoma fed the experimental diet compared with control dogs; however, there was no difference in these parameters between the experimental and control group in dogs with stage IV lymphoma.[44]

If metabolic derangements related to cancer cachexia are suspected, then dietary recommendations include moderate amounts of highly bioavailable protein, modest amounts of

Your pet has been discharged from the hospital with a feeding device in place. With this device, you can feed your pet even though he or she is not eating voluntarily. Please follow these recommendations for feeding.

1) Food

Please feed _____ diet. This diet _____ (does/ does not) require any additional preparation prior to feeding. (If additional preparation needed, see step A).

Step A: Please add _____ (amount of food) and _____ (amount of tap water) to a blender jar, and mix until a smooth consistency results. After mixing, strain the mixture through a food strainer two times to remove any large particles. This will make enough food mixture to feed your pet for _____ day(s).

2) Getting Ready to Feed

The diet should be fed at room temperature. Unused portions should be refrigerated in a clean container until you are ready to feed. Please allow refrigerated food to come to room temperature before feeding. To warm food, please place the food container in a bowl of warm water for 10-15 minutes, shaking gently every 5 minutes. Do not heat this food in the microwave. Apply a small amount of food to your skin to insure that overheating has not occurred; food should be comfortable to the touch. When food is warm, fill the feeding syringe(s) with _____ mL of food (see below). Fill an additional syringe with _____ mL of water to flush the tube after feeding.

3) Feeding

Place your pet in a quiet, comfortable area for feeding. Remove the cap from the end of the syringe. Attach the syringe to the tube, and slowly administer the food into the syringe. Food should pass with very little resistance, and no food should drip from the attachment site or from the skin entry site. If food is seen at these areas, discontinue the feeding session and contact your veterinarian. During feeding, your pet should be comfortable. If any discomfort, drooling, or vomiting occurs, please discontinue feeding, and administer food at a slower rate for the next feeding session. If these signs occur at the next feeding session, please discontinue feeding until you speak with your veterinarian. If any rapid or uncomfortable breathing occur, or if there is a sudden change with breathing at any time during feeding, discontinue feeding immediately, and do not administer more food until you speak with your veterinarian. If breathing difficulties persist after discontinuing feeding, seek emergency veterinary care.

3) Feeding Schedule

Day 1: Your pet should receive _____ mL of food every _____ hours
Day 2: Your pet should receive _____ mL of food every _____ hours
Day 3: Your pet should receive _____ mL of food every _____ hours
Day 4: Your pet should receive _____ mL of food every _____ hours
Day 5 and beyond: Feed _____ mL of food every _____ hours
In addition to tube feeding, please offer your pet _____ (type of food) by mouth _____ times a day, and monitor for voluntary food intake

4) Daily Duties

Inspect tube entry site and bandage or T-shirt once a day. Gently wipe the tube entry site with warm water and gauze if the entry site is moist or crusted. Please contact your veterinarian if the entry site has a foul odor or excessive discharge. Change the T-shirt as needed. See your veterinarian if the bandage is soiled.

Your pet _____ (should/should not) wear an Elizabethan collar at all times. Please clean food and debris from the collar as needed.

5) Medications

Your pet should receive the following medications (list medication, frequency, and route of administration)

- _____
- _____
- _____

5) Emergencies

Please discontinue feeding and contact your veterinarian immediately if any of the following are observed:

- Breathing difficulties
- Vomiting that does not resolve with slower feeding rate, or any vomiting with a nasal tube in place in place
- Inadvertent tube removal or displacement (Save any parts of the tube you find to show your veterinarian)
- Sudden change of attitude or behavior

Emergency veterinarian contact information:

_____

Fig 4. Client Care Worksheet for Enteral Feeding at Home.

simple carbohydrates, and increased amounts of fat. Although derangements in lipid metabolism are seen with cancer cachexia, the use of a high-fat rather than high-carbohydrate diet is supported by evidence that cancer patients have insulin resistance, carbohydrate intolerance, and hyperlactatemia when challenged with a dietary carbohydrate load. High-fat diets limit the amount of lactic acid production, lessen insulin resistance, and provide an energy substrate preferentially used by the host.[24]

Recent research has supported the role of increased omega-3:omega-6 fatty acid ratios for nutritional support of cancer patients. When omega-3 fatty acids were administered in a research setting, fat uptake attenuated in neoplastic cells and fat release attenuated from inguinal adipocytes. The mechanism of

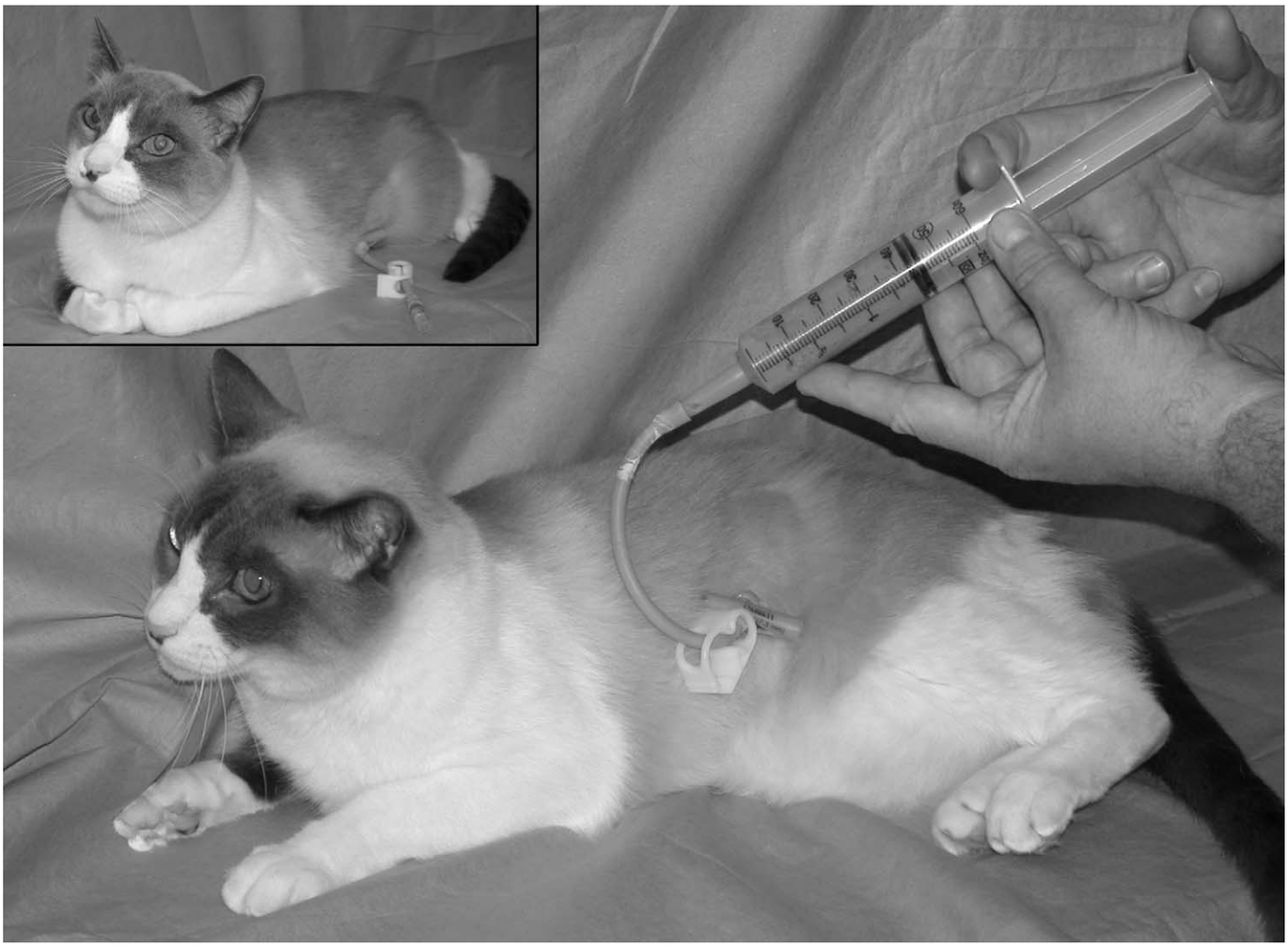

Fig 5. Animals should be comfortable and happy with a feeding tube in place, and have no onset of discomfort with administration of food.

this effect is thought to be related to altered fatty-acid receptor activity on neoplastic cells, causing an inability to utilize omega-3 fatty acids for energy. The anticipated clinical effect of these changes is to reduce the rate of cancer growth and minimize loss of stored body fat.[44,45] Hill's Prescription Diet® Canine n/d® is a commercially available food marketed specifically for use in animals with cancer.[46]

Initiation of Feeding

After placement of a feeding device, determination of nutritional needs, and selection and preparation of diet, feeding may begin. Calorie requirements should be based on the resting energy requirement, and adjusted for the nature and type of illness (Fig 2). Feeding should be instituted at less than the calculated calorie requirements initially, and then gradually increased to ensure patient tolerance of diet and feeding.[7,10] A reasonable plan for initiating feeding through all enteral feeding devices is feeding a third of the resting energy requirement for the first 24 hours, and then gradually increasing the amount fed over the next 3 to 5 days to achieve the desired calorie intake (Fig 3). In the author's experience, animals with prolonged anorexia or historical vomiting require a more gradual introduction to feeding than animals without these conditions. Even though complete calorie needs are not met for the first few days of feeding, initiation of enteral feeding supplies nutrients directly to enterocytes to maintain normal gut structure and function.[47]

Physical aspects of feeding include preparation of food, accurate measuring for mixing and feeding, and monitoring to ensure patient comfort during feeding. Because most feeding devices (eg, nasoesophageal, esophageal, gastrostomy) can be used for bolus feeding, patients are frequently discharged from the hospital for home care.[48] Client education for appropriate home care is crucial for a successful outcome. Fig 4 outlines steps for client care of animals receiving enteral nutritional support at home. Gruel must be prepared in a clean area to limit microbial contamination. Food should be administered at room temperature. Refrigerated food can be warmed in a water bath. Warming gruel in a microwave oven might cause hotspots, leading to thermal burns of the gastrointestinal tract. Food must be measured accurately into the feeding syringe. Minimal resistance to flow should be met while administering food through the tube, and the animal should remain comfortable (Fig 5). After any feeding, the tube should be flushed with enough water to remove food debris to prevent clogging. Most tubes require 5 to 10 mL for water flush. Water administered through a feeding tube supplies some patient water needs, and should be considered when intravenous fluid needs are calculated.

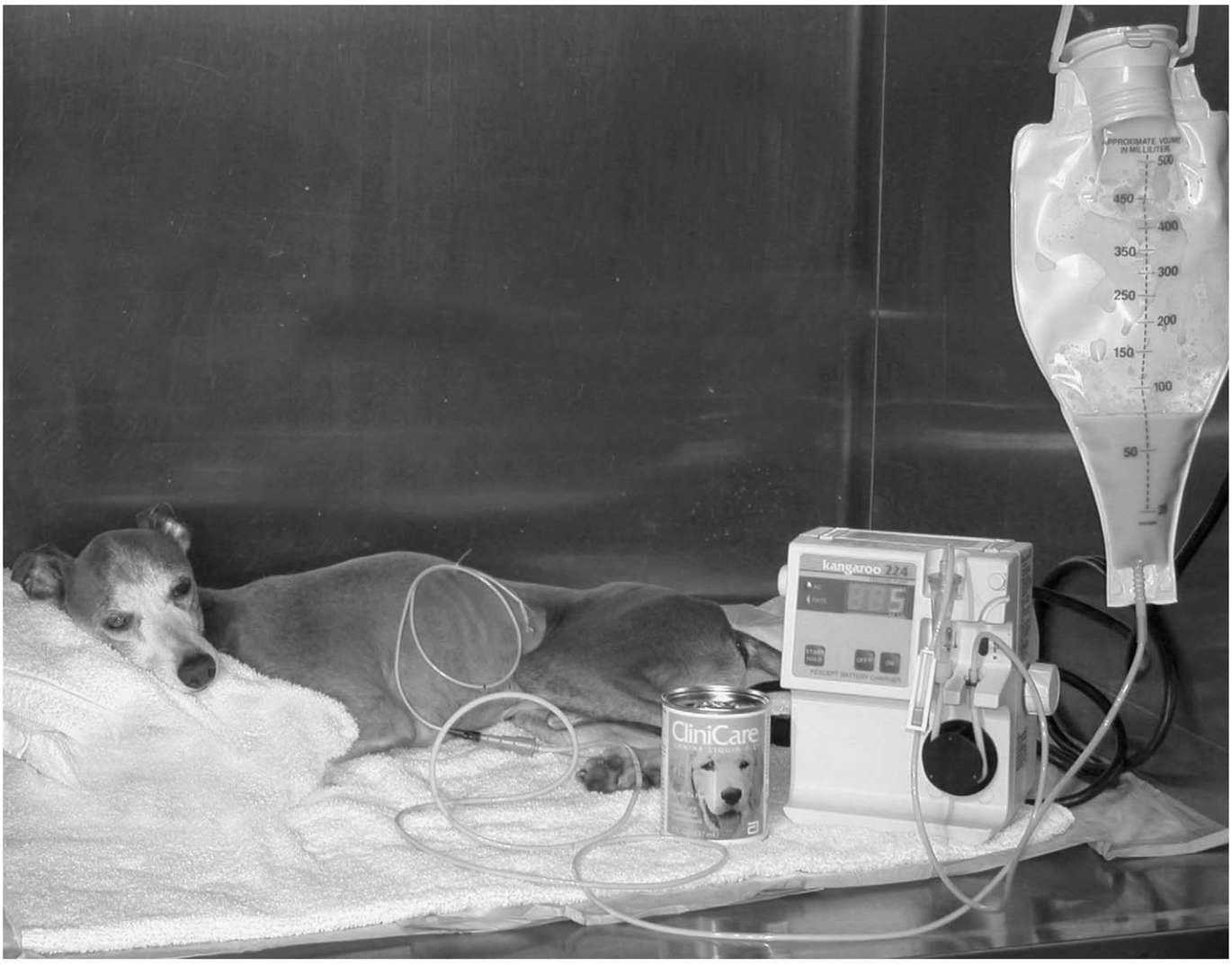

Fig 6. Continuous rate infusion of liquid food through a jejunostomy tube.

Any signs of nausea, discomfort, or ptyalism should be an indicator to temporarily suspend feeding until the discomfort resolves. If the discomfort does not resolve, then the client should contact the veterinarian. If respiratory distress is observed, then feeding should be suspended and emergency care sought. Small-diameter tubes (5-8 French) commonly used for nasal placement can be displaced with vomiting. If vomiting is observed, then feeding should be discontinued until appropriate tube placement can be ascertained. The owner should inspect the tube-insertion site daily for signs of localized abscess or food leakage.

Jejunal tubes should only be used in hospitalized patients. Because the jejunum is not distensible, continuous rate infusion of an appropriate liquid diet is indicated. Continuous rate infusion pumps are made for this application (Kangaroo Feeding Pump, Sherwood Medical, Norfolk, NE; Fig 6). Flow rates through this pump are only variable by 5-mL increments; therefore, a liquid diet might require dilution with water to achieve a desired calorie infusion in a small patient. Jejunal feeding should be initiated at a third to a half of the calculated calorie requirements. Dilution with an equal volume of water for initial feedings can improve intestinal tolerance for hyperosmolar diets. Jejunal feeding can be tapered and discontinued when other feeding methods can be instituted.[49]

When and How to Wean

The goal of enteral tube feeding is to supply nutrient requirements until the resumption of normal voluntary food intake. All types of enteral feeding devices allow the animal voluntary food consumption. Voluntary feeding should be encouraged in patients being fed via enteral feeding tubes, except when medically contraindicated (eg, severe oral or esophageal disease, severe vomiting, pancreatitis). No single documented plan applies for weaning because animals will resume voluntary food intake at different times and rates depending on the nature of the disease and the individual animal. Careful records of dietary habits, either in the hospital or at home, can be helpful to identify when voluntary food intake approaches the daily nutritional needs of the animal.

Most animals begin eating on their own during the recovery phase of disease. Nutritionally balanced and complete pet-food diets selected based on the underlying medical condition should be offered to initiate oral feeding. If a commercial diet is being used for tube feeding, then the same diet formulation can be offered for oral consumption. The morning or evening feeding can be delayed to offer the diet and evaluate voluntary consumption. If the patient was receiving a nutritionally incomplete diet before becoming ill (eg, "table scraps"), or a diet

not appropriate for the newly diagnosed medical condition, then the original diet can be offered initially until appetite becomes normal, and then gradual change to an appropriate commercial diet should be encouraged. Some animals prefer dry or canned food; therefore, different compositions of foods should be offered to entice the animal to eat. If the food consumed voluntarily has dramatic differences in nutrient composition, then diarrhea from dietary intolerance can occur. Food aversion may occur if a painful or unpleasant experience is associated with feeding. Attempting to feed animals that are vomiting or nauseous should be avoided to prevent food aversion. If a particular therapeutic veterinary diet is appropriate for medical management, then this diet should not be introduced for oral consumption until nausea has resolved. Appetite stimulants are occasionally helpful to restore voluntary food intake, but they will not dramatically improve appetite in most animals.[10]

Feeding tubes should be maintained until evidence of recovery from the underlying illness is observed, and voluntary food intake is 75% to 100% of desired food intake. Generally, several days are required to ascertain whether voluntary calorie intake is sustained. One method of weaning involves discontinuation of enteral feeding when voluntary food intake is 75% of normal, as well as monitoring body-weight and body-condition score for a week before tube removal to ensure continued weight maintenance or gain. If the patient cannot maintain body weight with voluntary food intake, then enteral feeding should be continued, and further diagnostic testing may be indicated to identify changes in medical status.

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