Chapter 25

Critical Care Nutrition and Enteral-Assisted Feeding

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"Let food be your medicine and medicine be your food."
Hippocrates

CLINICAL IMPORTANCE

Patients of any age may become malnourished from inadequate nutrient intake. Malnutrition is any disorder with inadequate or unbalanced nutrition associated with either nutritional deficiencies or excesses. By most estimates, many hospitalized people and companion animals do not receive adequate nutrition. Hospitalized veterinary patients are more commonly malnourished due to decreased total food intake. The major consequences of malnutrition in all patients, but more prominently in sick or injured patients, are decreased immunocompetence, decreased tissue synthesis and repair and altered drug metabolism.

Immunocompetence

The reciprocal relationship between nutrition and immunity has been recognized for centuries. A malnourished patient is more susceptible to infections and a septic patient is more likely to be anorectic, which results in malnutrition. Nutrient imbalances suppress immune function, which increases the risk of disease; conversely, certain diseases alter some nutrient requirements (Semba et al, 1997; Burkholder and Swecker, 1990). Decreased protein-calorie intake is the most common cause of secondary immunodeficiency in people and can cause progressively poorer responses in several components of the immune system including significantly impaired cell-mediated responses, secretory IgA production, phagocytosis, complement function, antibody affinity and cytokine production (Shikora et al, 1994; Chandra, 1992; Redmond et al, 1991). Although fewer studies are available for review, similar alterations in the immune system are seen in pets with insufficient caloric intake (Freitag et al, 2000; Simon et al, 2000). Studies have shown that protein deficiencies that limit amino acid and nucleotide substrates for cell proliferation result in reduced numbers of circulating T-lymphocytes, helper cells and suppressor cells (Chandra and Kumari, 1994). Malnutrition also decreases immune function of existing cells through reduced complement secretions, less effective macrophage function and decreased killer cell activity (Saxena et al, 1984). Cytokine production and release are independently impaired in protein-calorie malnutrition and in several micronutrient (zinc, iron, pyridoxine, vitamin A, copper and selenium) deficiencies (Meydani, 1990; Chandra, 1992a).

Numbers of T_4 helper cells and T_8 cytotoxic suppressor cells in malnourished people return to normal quickly with refeeding (Chandra, 1983). Immunoglobulins and circulating antibodies are maintained at relatively low levels during malnutrition, but are highly responsive to appropriate refeeding stimuli. For example, investigators measured serum globulin concentrations in 12 healthy beagles before and 24 hours after small bowel resection. All dogs were fed via gastrostomy tube immediately after surgery. Six dogs received a monomeric food whereas the other six were fed an electrolyte solution. Twenty-four hours postoperatively, the dogs fed the monomeric food

Box 25-1. Regulation of Food Intake.

For many years it has been known that the hypothalamus is the center of appetite control. Although voluntary food intake can vary in amount and composition from day to day, over time, energy intake is matched to energy expenditure. Body weight is tightly conserved; therefore, food intake, meal frequency and size are highly regulated. The role of peripheral and central pathways involved in appetite control is being studied as obesity increases in human and pet populations.

Appetite is the desire for food and is often used synonymously with hunger. Satiety is the opposite of hunger and means that hunger has been satisfied. The body is normally in a state of hunger, which is intermittently relieved by eating. Hunger and satiety centers are found in the brain. The lateral hypothalamus contains the hunger center; stimulation of this area causes an animal to eat voraciously. The ventromedial hypothalamus contains the satiety center. Stimulation of this area causes complete satiety. Many neuroendocrine and metabolic factors affect these centers and therefore contribute to appetite control.

Short- and long-term input from the periphery including nutrients, gut vagal nerves, sensory spinal nerves, gut peptides and gut hormones act on the arcuate nucleus of the hypothalamus. The peptide, ghrelin, released from the stomach, is the "hormone of hunger." Ghrelin is released in response to gut nutrients rather than gastric distention. Peptide YY (PYY) is an appetite depressant. It affects gut motility centrally, acting as an "ileal brake." Neural control of PYY is demonstrated by its release shortly after food intake before nutrients reach the small intestine and colon, the site of highest PYY concentrations. Glucagon like peptide-1 (GLP-1) acts on the pancreas to cause release of insulin and inhibit food intake. Its rapid enzymatic breakdown has limited its potential use in Type II diabetics. Other gut peptides, pancreatic peptide (PP) and oxyntomodulin (oxm) play roles in decreasing food intake and homeostatic regulation of body weight. The peripheral appetite depressant, cholecystokinin (CCK) is secreted in the duodenum in response to fat and protein ingestion. CCK, gut vagal nerves and sensory spinal nerves travel to the nucleus tractus solitarius (NTS) satiety center in the brainstem.

Insulin, released peripherally in proportion to body fat mass and blood glucose levels, acts directly on hypothalamic appetite centers. Another gut hormone, leptin, is released from adipocytes in direct relationship to body fat. Leptin decreases appetite and increases thermogenesis. Obese patients appear to be leptin resistant, which may impede their ability to regulate body weight.

Gut peptides (ghrelin, PYY, GLP-1, PP, oxm) and hormones (insulin and leptin) act directly on the arcuate nucleus of the hypothalamus. The arcuate nucleus has two populations of neurons. The orexigenic neurons release two neuropeptides, neuropeptide Y (NPY) and agouti-related peptide (AgRP), which stimulate feeding and promote obesity. The anorexigenic neuron center acts as an appetite inhibitor with the neuropeptides alpha-MSH and cocaine and amphetamine-regulated transcript (CART).

These neuron centers of the arcuate nucleus project to the paraventricular nucleus of the hypothalamus. The paraventricular nucleus receives direct input from other peripheral signals. Peripheral CCK, nutrients, vagal and sensory spinal signals travel to the NTS, which then acts directly on the paraventricular nucleus.

Central signals to the paraventricular nucleus include the appetite stimulator, melanin-concentrating hormone (MCH) of the lateral hypothalamus. The cortex and limbic system integrate appetite signals with the paraventricular nucleus. The paraventricular nucleus then coordinates both central and peripheral signaling for feeding, energy metabolism, sympathetic nervous system activity and the endocrine axis.

In addition, the special senses of taste and smell are involved in the regulation of food intake. Taste is mediated through taste buds and free nerve endings. Taste bud cells are constantly renewed by dividing epithelial cells surrounding the taste buds. Taste buds are located on the tongue, soft palate, pharynx, larynx, epiglottis, cranial esophagus and even on the lips and cheeks of some species. Gustatory information received from taste buds is projected by cranial nerves to several areas of the brain including the lateral hypothalamus. Olfaction occurs via axons of bipolar neurons that course through the small holes of the cribriform plate of the ethmoid bone and form connections in the olfactory bulb. As with taste, there are olfactory projections to the hypothalamus.

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The Bibliography for **Box 25-1** can be found at www.markmorris.org.

synthesized more than twice the amount of globulin (12 g) than those dogs fed the electrolyte solution (5.3 g) (Moss, 1978). Other cell populations and specific cell functions are likewise quickly responsive to daily nutrient intake. Cats fed 25% of their resting energy requirement (RER) for seven days had significantly decreased total white blood cell (WBC) count, lymphocytes and monocytes; major histocompatibility complex (MHC) class II expression; phagocytic activity; lymphocyte proliferative capacity and delayed-type hypersensitivity response by Day 4. These alterations were reversed by Day 4 of refeeding to meet RER (Freitag et al, 2000; Simon et al, 2000). The immune system depends on and is responsive to adequate nutrition.

Tissue Synthesis and Repair

Tissue synthesis and wound healing are a function of local and whole body nutritional status (Crane, 1989). On the cellular level, amino acids and carbohydrates are needed for collagen and ground substance synthesis. Fibroblasts require energy to synthesize the RNA, DNA and ATP necessary for protein anabolism. Migration of fibroblasts and epithelial and endothelial cells also requires energy. On the organ level, the liver has energy and protein needs specifically for synthesis of fibronectin, complement and glucose. The bone marrow requires nutrients for production of platelets, red blood cells (RBC) and leukocytes. Transportation of these necessary

components and oxygen to wound sites requires the muscular activities of respiration and cardiac work. Tissue trauma and healing alter the normal cycle of protein turnover (synthesis and degradation) in the body. In rats, the rates of protein synthesis and degradation increased after trauma (Stein et al, 1976). Studies in perioperatively fed people indicate a 91% increase in protein synthesis with only a 10% increase in protein degradation (net synthesis) (Kien et al, 1978). Conversely, perioperatively fasted people had only a 50% increase in protein synthesis with a 79% increase in protein degradation (net loss) (Birkham et al, 1980). Therefore, proper nutrition on the cellular level depends on whole body nutrition for net tissue synthesis and wound healing.

Drug Metabolism

Cellular activities depend on and are regulated by the coordinated actions of peptides, lipids, vitamins and minerals as substrates, enzymes, coenzymes and cofactors of intermediary metabolism. Therefore, all nutrients are essential for the maintenance of normal cellular structure and function (Parke, 1991). Nutrient deprivation alters the normal metabolic synergy responsible for ion gradients, membrane potentials, production of high-energy phosphate compounds and antioxidant defenses. Enteral and parenteral nutritional support with products containing little or no lipid decreases hepatic cytochrome P-450 concentration and activity, which significantly decreases specific drug clearances (Knodell, 1990; Raftogianis et al, 1995). Protein-calorie deficiencies may result in decreased: 1) hepatic biotransformation of certain antibiotics, 2) concentrations of serum proteins that bind and transport drugs throughout the body and 3) renal blood flow, which decreases the rate of drug elimination and increases the possibility of drug overdose (Walter-Sack and Klotz, 1996). Therefore, protein-calorie malnutrition may alter the expected metabolism of certain drugs, which may increase or decrease their therapeutic effect even when given at recommended dosages (Pelissier et al, 1993; Krishnaswamy, 1989) (Chapter 69). Patients receiving sufficient calories and protein are expected to have better, or near normal, drug distribution, metabolism and elimination than patients with protein-calorie malnutrition.

Inadequate nutritional support can suppress the immune response, cause organ dysfunction, impair wound healing, result in muscle wasting and weakness, increase the incidence of acquired infections and increase mortality. As an example, a 50% decrease in jejunal mucosal mass and thickness normally occurs after burn injuries when no enteral feedings are given for 24 hours. Early feeding prevents this mucosal atrophy. Malnutrition in people, even imprecisely defined, is associated with prolonged ventilatory dependence and increased complication rates with longer hospital stays and higher costs (Remillard and Martin, 1990). Similarly in veterinary patients, malnutrition is thought to increase morbidity and mortality. Diseased and debilitated patients require nutrients daily to maintain optimal immune function, tissue synthesis and repair and proper drug metabolism.

ANOREXIA, CACHEXIA AND ACCOMMODATION

Normally, satiety occurs after a patient's caloric needs have been met. Conversely, anorexia is the loss of desire for food before caloric needs have been satisfied (**Box 25-1**). Anorexia may be partial or complete. The anorexia is complete if a patient consumes no food for a period beyond that considered normal. The anorexia is partial if the patient consumes some food but less than that considered a normal daily intake.

The flavor of food results from chemical stimulation of taste buds and free nerve endings in the nose, mouth and throat. "Taste" disorders often result from abnormalities in olfaction. Disorders of taste or smell can impair appetite and occur because of:

- Old age. The number of taste buds declines with age. Olfaction is usually the first sensory system to show an aging effect.
- Damage to neural connections due to surgery or traumatic head injury. Accidental blows to the head can shear the fine olfactory nerves that pass through the cribriform plate and are a common cause of anosmia (inability to smell) in people.
- Impaired renewal of taste buds and olfactory epithelium. Decreased chemosensory cell turnover is consistent with the decreased cell renewal reported to occur in the small intestinal epithelium as a result of food deprivation, radiation therapy, uremia, vitamin B_{12} deficiency and therapy with methotrexate. Many endocrine factors also depress cell proliferation. These factors and many conditions and drugs (**Table 25-1**) probably impair regeneration and function of taste buds and olfactory cells in the same manner that they impair regeneration of intestinal epithelium. The turnover time of taste buds and olfactory cells is about 10 days. Therefore, a return to normal taste function after mitosis is interrupted requires at least 10 days and usually longer.
- Modification of receptor cells as a result of a chronic change in local environment (e.g., an alteration in saliva or the fluids bathing the olfactory mucosa) due to drugs or metabolic agents such as urea.

Numerous medical problems including organic disease, inflammation, trauma and neoplasia can cause anorexia. In addition, pain, fear and other components of emotional stress inhibit the desire for food (Schiffman, 1983). If anorexia persists, depletion of body nutrient stores occurs. Nutritional depletion may also result from facial or oral injuries, or obstruction or dysfunction of the gastrointestinal (GI) tract, liver or pancreas so that the patient is incapable of ingesting, chewing, swallowing, digesting or absorbing food. In general, patients not eating for more than 48 hours or those consuming less than 50% of normal intake for more than three days should be of concern and noted as having a form of anorexia. Cats and dogs with a history of complete anorexia for three or more days or those with a history of partial anorexia for several weeks warrant further nutritional assessment.

Cachexia is a state of general illness, malnutrition and profound disability. For nearly 50 years, investigators and nutri-

Table 25-1. Disorders and drugs that affect taste and smell in people.*

Disorders		
Adrenocortical insufficiency	Cushing's syndrome	Nasal polyposis
Allergic rhinitis	Diabetes mellitus	Niacin deficiency
Bronchial asthma	Head trauma	Radiation therapy
Burns	Hepatic cirrhosis	Sinusitis
Cancer	Hypertension	Viral hepatitis (acute)
Chronic renal failure	Hypothyroidism	Zinc deficiency
Cobalamin deficiency	Influenza-like infections	
Drugs		
Drug classification	**Examples**	
Amebicides	Metronidazole	
Antiepileptic drugs	Phenytoin	
Anesthetics (local)	Benzocaine, procaine hydrochloride, tetracaine hydrochloride	
Antihistamines	Chlorpheniramine maleate	
Antimicrobial agents	Amphotericin B, ampicillin, cephalosporins, chloramphenicol, gentamicin, griseofulvin, kanamycin, lincomycin, neomycin, nitrofurantoin, sulfonamides, streptomycin, tetracyclines	
Antineoplastic agents	Doxorubicin, methotrexate, vincristine sulfate	
Antirheumatic, analgesic, antipyretic, antiinflammatory, immunosuppressive agents	Allopurinol, azathioprine, colchicine, levamisole, D-penicillamine, phenylbutazone	
Antithyroid agents	Propylthiouracil, thiouracil	
Diuretics and antihypertensive agents	Captopril, furosemide, thiazides	
Opiates	Codeine, morphine	
Sympathomimetic drugs	Amphetamines, ephedrine	
Others	Digitalis glycosides, estrogens, iron sorbitex, oral antidiabetic agents, vitamin D	

*Adapted from Schiffman SS. Taste and smell in disease (first of two parts). New England Journal of Medicine 1983; 308: 1275-1279. Schiffman SS. Taste and smell in disease (second of two parts). New England Journal of Medicine 1983; 308: 1337-1343.

tionists have recognized that cachexia and a resultant low body condition score (BCS) are associated with increased risk of complications in people (Windsor, 1993). Loss of skeletal and visceral proteins can have adverse anatomic and functional consequences in food-deprived patients. These adverse effects include anemia, reduced heart muscle mass and function, decreased pulmonary mechanical function and diminished respiratory drive, altered intestinal morphology and mildly impaired absorptive abilities (Biden and Taylor, 1983). Cachexia may affect dogs and cats with long-standing cancer, cardiac disease or renal disease (Chapters 30, 36 and 37, respectively). A state of metabolic "accommodation" that prolongs survival has been recognized in people with chronic diseases. A similar state of metabolic accommodation probably occurs in chronically ill dogs and cats.

Accommodation occurs when the energy equilibrium is reestablished at a constant but lower food intake and lean-tissue wasting is arrested before protein deficiency becomes fatal. Accommodation in people is successful when: 1) total lean-tissue depletion is less than that considered critical, 2) weight is low but stable and 3) albumin levels and total peripheral WBC counts are normal with an intact delayed cutaneous hypersensitivity response (Hoffer, 1994).

Accommodation with the exception of an intact delayed cutaneous hypersensitivity response accurately describes the condition of some chronically ill patients (i.e., those with chronic renal, hepatic or cardiac insufficiency). Some chronically ill, cachectic cats and dogs may be maintained at a less than optimal body weight and condition for some time, even though important organ function deficits are apparent. In these cases, metabolic rate has been down regulated and protein turnover has been altered to establish a fragile homeostasis. This homeostasis can be maintained until a new stress supervenes. Affected patients very often do not survive additional stresses such as trauma, surgery, infection or tumors, as might a previously healthy dog or cat.

Metabolic Changes Through Days of Food Deprivation

Simple Starvation

Simple starvation includes metabolic changes that occur in mammals, in the absence of disease, as days of food deprivation continue. The time course of metabolic changes and associated alterations in nutrient usage should guide how these hospitalized patients are fed. An understanding of the metabolic changes that take place (particularly in the liver) during starvation is essential to understanding metabolic alterations present during anorexia and disease states.

In the postprandial period of well-fed patients, exogenous dietary nutrients are used to meet immediate metabolic needs, thus sparing endogenous fuels stored as glycogen, adipose and muscle protein. After these needs are met, replenishment of glycogen reserves (in the liver, fat and muscle) as well as proteins catabolized since the last meal, takes place. Any excess energy in the form of carbohydrate, fat or protein is then converted to triglycerides for storage as fat in adipose, muscle and liver tissue. In the fed state when serum glucose is high, the liver becomes a net importer of glucose, trapping it in hepatocytes by phosphorylation via glucokinase. Glucokinase is an inducible hepatic enzyme whose maximal enzymatic activity (Km = 180 mg/dl) for glucose is reached with the help of insulin, at high blood glucose concentrations (Engelking and Anwer, 1992).

Patients undergoing food deprivation display almost complete reversal of the metabolic processes described for the well-fed patient. Due to the lack of exogenous dietary sources, endogenous sources become the primary fuel for meeting immediate metabolic needs. Glycogen stores, instead of being replenished, become exhausted as the initial energy source. Then in order to preserve vital functions as long as possible, patients use different proportions of stored body fat and protein to maintain blood glucose concentrations. Which fuel or mixture of fuels the patient uses depends on the length of time the patient was food deprived and the quantity of each of the fuel stores available to the patient (**Figure 25-1**). The adaptation from fed to starved state is one in which fuel use by the patient shifts from primarily a mixture of fuels to one in which the primary fuel is fatty acids.

Carbohydrate metabolism is profoundly altered during the first week of starvation. During the first few days, omnivores (i.e., dogs) maintain blood glucose levels through glycogenolysis and gluconeogenesis. In simple, uncomplicated starvation of mammals, a decrease in blood glucose below 120 mg/dl decreases activity of hepatic glucokinase. This triggers hepatic glycogenolysis during which the liver becomes a net exporter of glucose to preserve serum glucose levels (**Figure 25-2**). This is observed after four to five hours of fasting and will only maintain blood glucose levels for another 12 to 28 hours (Cahill and Owen, 1968). Thereafter, gluconeogenesis must maintain blood glucose concentrations because hepatic glycogen stores will have been depleted. In contrast, carnivores (i.e., cats) must rely solely on gluconeogenesis, beginning intraprandially for maintenance of blood glucose levels because of their decreased hepatic glycogen reserves. This decreased reserve is due in part to lower glycogen synthase and glucokinase concentrations (**Table 25-2**).

Gluconeogenesis is initiated by glucagon and later glucocorticoids as serum glucose levels decrease (**Figure 25-3**). This process is carried out predominately in the liver and kidneys using substrates (glycerol, lactate and glucogenic amino acids) resulting from the catabolism of adipose and muscle tissue. Adipose tissue supplies glycerol for glucose production and fatty acids for oxidation to supply energy. Muscle catabolism releases glucogenic amino acids, lactic acid and pyruvate for glucose production by the liver (Welborn and Moldawar, 1997). Once available in the circulation, extrahepatic tissues are able to trap glucose intracellulary due to the presence of hexokinase, the enzyme present in all mammalian cells. Hexokinase has a low Km (1 mg/dl) for glucose compared to glucokinase and it does not require insulin to be effective. Therefore, intracellular trapping of glucose via hexokinase activity can happen at very low blood glucose levels (Engelking and Anwer, 1992).

In an effort to conserve circulating glucose for glucose-dependent tissues, the liver releases ketone bodies as an alternate fuel for non-glucose dependent tissues. Ketones are oxidation products of long-chain fatty acids, which originate from triglycerides in adipose stores. Unlike fatty acids, which are water insoluble and must be carried in the blood by albumin, ketones are water soluble and thus have a very wide distribution

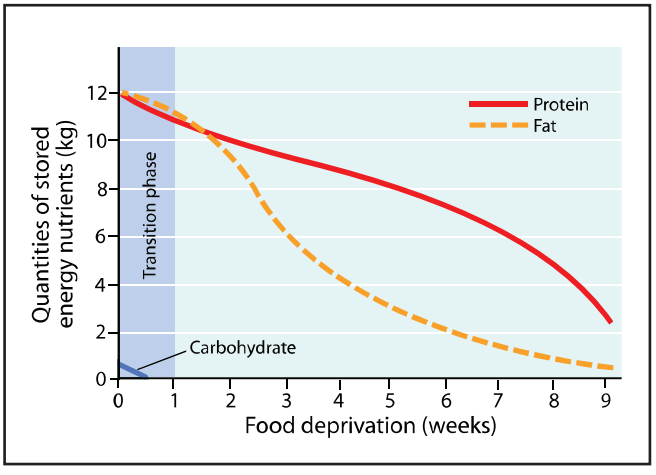

Figure 25-1. Disappearance of nutrient stores during starvation. (Adapted from Lewis LD, Morris ML Jr, Hand MS. Anorexia. In: Small Animal Clinical Nutrition III. Topeka, KS: Mark Morris Associates, 1987; 5-6.)

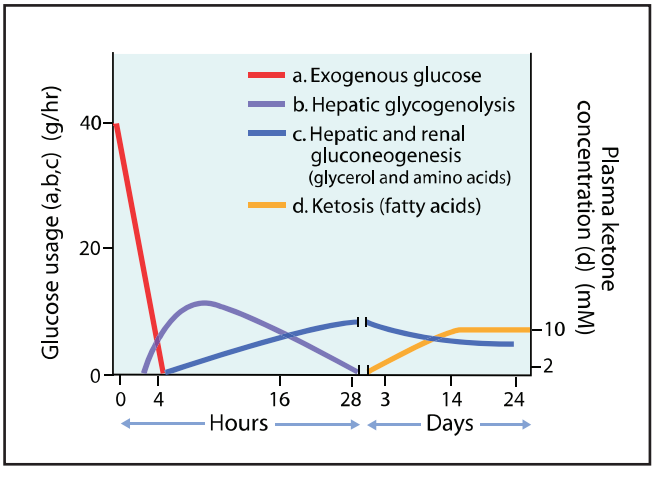

Figure 25-2. Graph of glucose use and source during starvation. (Adapted from Engelking LR, Anwer MS. Liver and biliary tract. In: Anderson NV, Sherding RG, Merritt AM, et al, eds. Veterinary Gastroenterology, 2nd ed. Philadelphia, PA: Lea & Febiger, 1992; 211-274.)

Table 25-2. Relative hepatic enzyme concentrations.

Enzymes	Dogs	Cats
Glycogen synthase	13*	1*
Glucokinase	55	5
Hexokinase	1.2	1

*Relative activity levels.

within the body and are able to diffuse across cell membranes. In this way they act to serve as a direct source of energy for vital organs such as the brain. Additionally the insoluble nature of fatty acids and their dependence on albumin limits their serum concentration. Thus the advantages of converting fatty acids to ketones are threefold: ketones are water soluble, not dependent on albumin for transport and can provide lipid fuel to cells at a much higher blood and interstitial fluid concentration. In

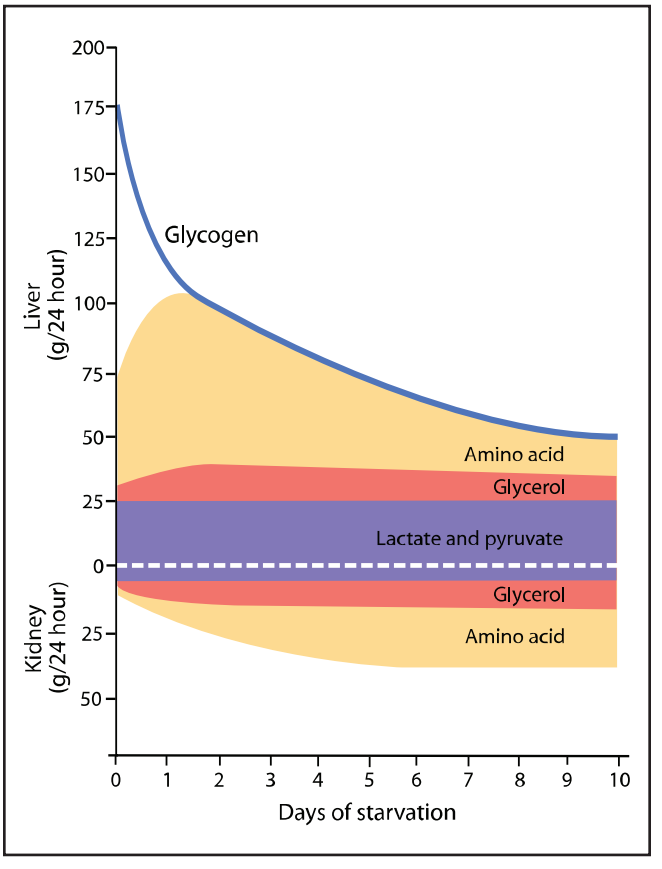

Figure 25-3. Graph of glucose production and source by the liver and kidneys. (Adapted from Owen OE, Felig P, Morgan AP, et al. Liver and kidney metabolism during starvation. Journal of Clinical Investigation 1969; 48: 574-583.)

mammals, serum ketones can reach 2 to 3 mM within a few days of starvation, with levels increasing to between 7 and 8 mM after one week. This is greater than the normal glucose concentration of 5 mM (Engelking and Anwer, 1992).

Increased serum ketones contribute to the adaptive response of the patient towards conservation of endogenous protein and glucose sparing for the non-adaptive, glucose-dependent tissues (erythrocytes and renal medullary cells). Enzymatic changes in peripheral tissues and brain caused by increased serum ketone concentrations promote ketone use and decrease the demand for glucose. Ketosis in food deprivation is an appropriate physiologic response and may not lead to severe ketoacidosis except in diabetic dogs and cats. Thus, ketone bodies serve as a readily diffusible lipid-source fuel for muscles, kidney cortex, peripheral nerves and the brain during periods of starvation. Ketone body production is usually maintained until adipose tissue is depleted.

In addition to adaptation from mixed fuels to fatty acids as a primary fuel source, as food deprivation continues metabolism begins to decrease. By the third day of food deprivation in all mammals, the basal metabolic rate decreases to promote conservation of resources for long-term survival. Decreases in blood glucose levels result in a decrease in serum insulin release. The conversion of thyroxine (T_4) to triiodothyronine (T_3) is insulin responsive and thus declines in the face of decreased insulin (Gavin and Moeller, 1983). T_3 levels begin to decrease within 24 hours of fasting and may be 40 to 50% lower as compared to values in fed animals by Day 3 of food deprivation (Vagenakis et al, 1975). Thus the net effect is a hypometabolic state, which becomes a beneficial adaptation allowing conservation of body functions until appropriate caloric intake resumes.

By Day 5 of food deprivation in all mammals, endocrine changes have mandated a metabolic shift from exogenous fuel usage in the fed state to endogenous fuel usage in the forms of fatty acids and ketone bodies. Decreased insulin levels trigger lipolysis in an effort to conserve protein stores and maintain blood glucose levels for glucose-dependent tissues. By undergoing fatty acid oxidation, amino acids are partially spared, which helps maintain muscle protein stores throughout starvation until the end stages. Body proteins are only partially spared because muscles will catabolize branched chain amino acids. Nitrogen is exported with pyruvate as alanine and to a lesser extent glutamine, which may be deaminated and transaminated for hepatic protein synthesis. Additionally, their ketoanalogs are used for hepatic and renal glucose synthesis (Felig et al, 1969).

The respiratory quotient (RQ) can provide an indication of which substrate(s) are undergoing catabolism. An RQ of 0.7 indicates fat catabolism, 0.8 indicates protein catabolism and 1.0 indicates that carbohydrates are being used as the primary fuel. Studies involving well-fed dogs measured an RQ of 0.94 at rest, indicating high carbohydrate use. The RQ of the same dogs after five to 15 days of starvation was 0.8 indicating a shift from primarily carbohydrate use to that of lipid and/or protein catabolism (Himwich and Rose, 1927). This is a testament to the importance of fat as a primary fuel source after three to five days of food deprivation.

Refeeding management of the food-deprived patient is aimed at matching the number of "no food" days with the pattern of fuel use (i.e., adaptation from using a mixture of fuels to primarily using fat). This is often in the patient's dietary history. As described, the different proportions of stored carbohydrate, fat and protein that is/are used to maintain blood glucose and provide an energy source mainly depends on the duration of food deprivation. To minimize the metabolic complications of refeeding, the refeeding formula should contain a complete balance of nutrients and should have a carbohydrate, fat and protein source similar to that which the liver has become adapted or is estimated to be using from body stores. Oftentimes this requires a feeding formula that is lower in carbohydrate and higher in fat and protein sources as compared to a maintenance-type food. As described, patients deprived of food are in a state of catabolism, which can often be reversed by refeeding if body protein losses have not exceeded 25 to 30%.

Disease State

In contrast to patients undergoing simple starvation, patients in disease states are often inappetent or anorectic due to their disease process. Although similar in their decreased dependence on glucose for fuel and increased propensity for lipolysis, patients in disease states have increased energy requirements

and are more dependent on proteolysis than are patients that are simply food deprived. The hypermetabolic state that results from illness and anorexia is generated by general neuroendocrine responses and local mediators common to stressful stimuli. The specific hormonal and subsequent physiologic changes that occur in a stressed patient are unique to that disease condition, the time course of the disease process and other complicating diseases or conditions. The metabolic responses have best been characterized in conditions with a known acute onset such as trauma or infection (sepsis) (Cuthbertson, 1979). The response has been described as having an acute (ebb) phase followed by an adaptive (flow) phase (Popp and Brennan, 1983). These phases can vary in duration and intensity depending on the severity of the condition. Duration is usually shorter with trauma as compared to infection during which the acute phase remains until the infection has been eliminated.

The acute phase is characterized by catabolism and generally occurs within the first 24 to 72 hours. As mentioned, the hypermetabolic state that occurs results from a milieu of neuroendocrine and locally activated mediators stimulated by the sympathetic nervous system as part of the stress response. As in simple starvation, glucose use is somewhat reserved; however, part of this reservation is by consequence of increased cortisol levels perpetuating insulin resistance and hyperglycemia. Simultaneous sympathetic nervous system stimulation and release of catecholamines, cortisol, adrenocorticoids, glucagon, growth hormone and antidiuretic hormone induce metabolic and physiologic responses including:

- Insulin antagonism leading to insulin resistance.
- Hyperglycemia from glycogenolysis and gluconeogenesis, which provides an energy source for the "fight or flight" phenomenon.
- Increased lipolysis to provide fatty acids and glycerol for glucose and energy production.
- Increased proteolysis and net protein catabolism from albumin and skeletal muscle sources to supply gluconeogenic amino acids for glucose production and hepatic and immune cell proteins.
- Increased rate and depth of respiration and increased cardiac work to maintain perfusion to muscles and wound sites.

These responses are amplified in infection in which a toxic response results from invasive organisms and resorption of necrotic tissue. The amplification is caused by release of chemical mediators and lysosomal enzymes including histamine, kinins, prostaglandins, cytokines and serotonin. Interleukin-1 induces fever, which increases energy expenditure 10 to 13% per degree Celsius increase in body temperature. Endotoxins released from dying gram-negative bacteria trigger coagulation cascades that profoundly affect carbohydrate metabolism and cytokines stimulate production of hepatic acute-phase proteins. The net result is a hypermetabolic state with increased energy requirements that if not met, result in catabolism of endogenous fat and protein stores.

In the *acute phase*, glucose is the preferred fuel; however, muscles are insulin resistant and hyperglycemia is maintained for net immune and hepatic tissue anabolism. This is beneficial to the patient because it ensures an energy source in the face of hypoperfusion or poorly perfused organs (Daley and Bistrian, 1994). General feelings of malaise and anorexia signal the patient to reduce activity of skeletal muscle and signal the GI tract to conserve energy for essential tissue maintenance and repair. Although in the short-term, hyperglycemia appears to benefit the patient, maintaining this state is not conducive to recovery. Thus, the acute phase is characterized by decreased exogenous nutrients in the face of increased energy demands (hypermetabolic state) and a net negative energy balance. Breakdown or catabolism of protein and fat stores is necessary to address this imbalance between expenditure and intake.

This catabolic phase will continue until the neuroendocrine stimuli and cytokine mediators are removed. During severe head trauma, burns, multiple trauma and sepsis, lean tissue loss and overall body weight loss are rapid and unremitting in the absence of feeding. It is difficult to reverse the ongoing catabolism and achieve nitrogen balance in patients with these injuries and conditions. The goal in providing nutrition to these patients is to feed the catabolism with exogenous sources of protein and fat while sparing endogenous sources. The latter is critical because loss of 25 to 30% of body protein stores has been associated with reduced heart muscle mass and function, decreased pulmonary function, diminished respiratory drive, compromised immune function and therefore mortality (Matthews and Fong, 1993).

There is a definite turning point in which clinicians note a subjective improvement in their patients. This noted improvement is associated with the adaptive phase in which net anabolism occurs. The adaptive phase is characterized by increased metabolic rate, nitrogen gain and normal body temperature and may last for several days, weeks or years (Daley and Bistrian, 1994). The convalescent anabolic phase rebuilds damaged and catabolized lean tissue and therefore requires exogenous energy and protein sources. RQ values determined in resting postoperative and severely traumatized dogs were 0.76, indicating that fat was the preferred energy fuel, while dietary protein is used for anabolic processes (Walton et al, 1996).

In the catabolic and adaptive phases, fat and protein will more effectively address nutrient needs of the patient. Therefore, provision of dietary carbohydrates should be minimized, whereas fat and protein calories are maximized in refeeding formulas (provided there is no contraindication). Increased food carbohydrate fractions may lead to electrolyte disturbances and hyperglycemia, though the latter happens rarely in dogs. Cats, however, have very low glucokinase activity and cannot effectively transport glucose into hepatic cells given high intravenous and occasionally high enteral concentrations (**Table 25-2**). Subsequently, hyperglycemia is a commonly observed phenomenon during refeeding of cats. Additionally, specific disease states will affect the type and/or degree of metabolic and hormonal alterations in the patient, which subsequently influence substrate usage. In consideration of these factors, the recovery phase is patient dependent, which underscores the need for continual and consistent reassessment

Table 25-3. Examples of hospital feeding orders.

1. Offer 2 cans of product XX every 6 hr PO.
2. Give 100 ml of product YY gruel every 6 hr via PEG (percutaneous gastrostomy) tube.
3. Administer 300 ml of parenteral solution IV every 8 hr.

Sometimes the feeding orders should contain special conditions:

4. Begin feeding liquid product ZZ at 10 ml/hr via NG (nasogastric) tube. D/C (discontinue) all feeding if vomiting begins.
5. Administer 300 ml of parenteral solution IV every 8 hr. Check urine glucose and decrease rate to 150 ml every 8 hr if urine is positive. Recheck serum potassium daily and increase to 40 mEq/l if below normal.
6. Give 30 ml of product YY gruel every 6 hr by PEG. Increase meal volume fed by 10 ml every 24 hr. decrease volume by 50% if vomiting begins.

Table 25-4. Laboratory data of a dog after four months of starvation.

Tests	Results	Reference ranges
Complete blood cell count*		
RBC (x 10^6/mm^3)	2.73	4.62-8.3
HGB (g/dl)	6.5	11.6-20.6
HCT (%)	18.2	33.1-66.4
Reticulocyte (%)	0.0	0-3
WBC (x 10^3/mm^3)	3.4	4.8-16.2
Fibrinogen (mg/dl)	430	88-380
Serum biochemistry profile**		
Glucose (mg/dl)	172	65-110
AST (U/l)	79	9-43
ALT (U/l)	75	14-50
Alkaline phosphatase (U/l)	230	5-125
Total protein (g/dl)	4.0	4.6-7.0
Albumin (g/dl)	2.1	2.6-4.2
Calcium (mg/dl)	8.5	8.9-11.1
Phosphorus (mg/dl)	2.9	3.0-5.9
BUN (mg/dl)	28	7.0-25.0
Creatinine (mg/dl)	0.2	0.6-1.6
Urinalysis		
Specific gravity	1.052	1.015-1.045
pH	7.0	6.0-7.5
Ketones	Trace	-

Key: HGB = hemoglobin, HCT = hematocrit, AST = aspartate aminotransferase, ALT = alanine aminotransferase, BUN = blood urea nitrogen.
*MCV, MCH, MCHC, platelet numbers, WBC differential, blood lead and coagulation profile were normal.
**Serum K, Mg, Na, Cl and total bilirubin concentrations were normal.

of the patient to optimize the nutrition support plan.

PATIENT ASSESSMENT

Malnutrition can be recognized in patients through use of a nutritional assessment protocol. Nutritional assessment helps identify those patients that require assisted feeding to avoid or reduce nutrient deficiencies and the associated complications. Although inadequate nutrient intake may complicate many disorders, anorexia has been traditionally viewed as a secondary problem that will improve when the primary disease problem has resolved, i.e., "They'll eat when they feel better." However, it is better to be proactive and recognize the value of administering nutrients to veterinary patients and realize that, "They'll feel better sooner when they eat."

Diseased and debilitated patients (hospitalized or not) need to be assessed frequently, regardless of their age or lifestage. Assessment uses a number of parameters taken together to give an overall impression of whether a patient is experiencing malnutrition and requires specific nutritional intervention. Useful parameters to be assessed have been identified in large populations of people; however, no such parameters have been specifically formulated for dogs and cats. A veterinary nutritional assessment protocol should include history, physical examination (with special attention given to certain risk factors), body condition assessment (BCS) and laboratory tests (Buffington, 1994). Weight and dietary history, physical examination and body condition are relatively easy parameters to obtain. However, specific laboratory and immunologic tests that correlate well with nutritional status have not been identified. To date, very few clinical studies have been performed in veterinary patient populations to determine which parameters are applicable and their accuracy in determining nutritional status and predicting outcome (Michel, 1993).

History and Physical Examination

All patients should receive a physical examination including an accurate determination of body weight and an estimate of body condition. Weight changes must be viewed as a proportion or percentage of "normal, usual or optimal" weight within a certain time period as opposed to absolute changes in units (e.g., g or kg lost). Weight loss of more than 10% within a week is clinically significant and warrants further assessment. As a point of reference, a weight change of 10 to 15% within several days is most likely a hydration problem and should be corrected first with medical or fluid management. Pets on a designated weight-loss program can safely lose 1 to 4%, more typically 1 to 2%, of their body weight per week (Laflamme, 1993) (Chapter 27). A 10% (5 kg) weight loss within a week for a 50-kg dog is easily recognized as significant, but a similar percent weight loss over seven days for a 5-kg cat (i.e., 0.5 kg) is not easily recognized. This weight loss should be considered as serious as the same percentage weight loss in the dog. It is more difficult to accurately determine a 0.5-kg weight change than a 5-kg change; therefore, cats should be weighed on a scale that is accurate between 0 and 15 kg.

Body weight is an objective measurement, whereas body condition is a more subjective evaluation of the patient's tissue composition relative to its weight (i.e., fat, muscle and bone) (Chapter 1). Body condition scoring adds valuable information to body weight data. Decreasing fat stores indicate low energy intake and vice versa. Muscle wasting implies protein intake has been insufficient because skeletal muscle mass supports hepatic protein synthesis when dietary intake is inadequate. In one human study, using three independent clinicians' nutritional assessment of the same 64 patients, there was a 77% agreement among clinicians, and their clinical judgment of nutritional risk correlated well with

objective data such as albumin, transferrin and cholesterol concentrations and weight loss history (Lupo et al, 1993).

Survival rates of people have been directly correlated with available muscle mass. Loss of more than 25 to 30% of body protein compromises the immune system and muscle strength, and death results from infection, pulmonary failure or both (Matthews and Fong, 1993). Decreased muscle mass may occur before serum protein levels drop below normal in chronic states because overall muscle wasting is less life threatening than decreased serum protein concentrations. Muscle atrophy due to protein malnutrition occurs bilaterally and should involve several muscle groups. Bedridden patients can develop muscle atrophy due to decreased use just as astronauts develop muscle atrophy of anti-gravity muscles because muscle size depends on exercise and gravity (Lane et al, 1993). Selected muscle groups may be atrophied in animals that have limited use of a limb. Therefore, lack of activity should be considered when evaluating the muscles of a patient, particularly one that is partially paralyzed or has a long-term illness.

Recording the food intake of hospitalized patients helps determine whether or not assisted feeding is necessary. In addition to having complete feeding orders, the medical record should also contain the time of day and amount of food actually consumed by the patient. Consumption can be simply recorded as some percentage of the food offered (e.g., 0%, 50%, 100%). If feeding orders are properly written and food consumption is recorded, it will be apparent after 24 hours of hospitalization whether or not the patient is consuming sufficient food to meet its RER, and whether assisted feeding is necessary. In a study of 276 hospitalized dogs, a positive-energy balance (>95% RER) was achieved in only 27% of 821 dog days recorded, whereas a negative-energy balance (<95% RER) was observed on the majority (73%) of the dog days. The primary reasons for the 601 negative-energy balance dog days were: 1) dogs refused to eat any or all of the food offered (43%) and 2) the attending veterinarian ordered nil per os (NPO) (34%) (Remillard et al, 1998). Currently, many hospitalized dogs do not consume their RER primarily because they refuse to eat the food offered to them. Also, feeding orders for hospitalized patients should be clear and complete. Properly written hospital feeding orders identify a specific food product with the amount, frequency and the route of intake specified, if not per os (**Table 25-3**). In the same study, fewer than 20% of approximately 1,000 written feeding orders were complete and accurate.

Assisted feeding should be considered for any patient with a suspected or documented food intake below the calculated daily RER for more than three days. Nutritional support should initially deliver sufficient amounts of a nutritionally balanced food to meet the RER of the patient at its current weight when the BCS is 3/5 or less. RER is primarily determined by total weight of metabolically active tissues such as skeletal and smooth muscle and visceral organs. BCS is primarily a measure of body fat stores. RER and BCS taken together are used to initially estimate the patient's daily caloric requirement. Animals with a BCS of 4/5 or 5/5 generally have the same muscle and organ mass as those with a BCS of 3/5; however, these animals have increased fat stores, which do not increase RER. It may be prudent, therefore, to calculate RER on an estimate of optimal weight in overweight patients to prevent overfeeding (Chapter 27). After several days, the food intake may be increased if warranted.

Figure 25-4. This dog experienced four months of starvation because its owner was unable to care for it due to a chronic terminal illness. **Table 25-4** presents laboratory data from this animal.

Laboratory Data and Other Clinical Information

The changes in most laboratory data due to malnutrition are indistinguishable from those occurring in some disease processes; however, malnutrition should be considered when examining the patient and reviewing the data (**Figure 25-4** and **Table 25-4**). RBC number, hemoglobin content, urea nitrogen, potassium, albumin and total protein concentrations, total WBC and lymphocyte count are useful in nutritional assessment of adequately hydrated patients. RBCs, hemoglobin, albumin and total protein have moderately long half-lives of one to eight weeks and are an indication of the energy and protein status of the patient over the preceding weeks to months. In one study, dogs fed a protein-deficient food (4% dry matter

Box 25-2. Future Laboratory Tests for Nutritional Assessment.

DELAYED-TYPE HYPERSENSITIVITY TESTING

The delayed-type hypersensitivity (DTH) skin test has been promoted as an inexpensive and simple bedside preoperative test for people with sepsis-related mortality risk, again relating the close tie between immunocompetence and patient outcome. Patients who did not have an appropriate skin reaction to a multi-antigen intradermal injection had a sepsis rate of 34% and a mortality rate of 38% vs. a 7% sepsis rate and 3% mortality rate for patients who reacted to the test injection. Several diseases and drugs, however, may alter the specificity of this test as an indicator of malnutrition.

Delayed-type intradermal hypersensitivity testing is not currently used in dogs and cats; however, some preliminary work in cats has shown promise. Cats infected with feline leukemia and feline immunodeficiency viruses had a DTH response less than that of normal cats. In another study, healthy cats receiving no food had a significantly reduced response to an intradermal injection of feline rhinotracheitis-calicivirus-panleukopenia antigens on Day 4 vs. when they received food daily.[a]

LYMPHOCYTE FUNCTION TESTING

Other promising indicators of nutritional status in the development stage for dogs and cats are specific immune function tests. A battery of immune function tests has been developed for use in cats, including: 1) immunophenotyping to identify relationships between immunosuppressor and helper cells, 2) measuring membrane calcium flux to evaluate membrane function, 3) immunophenotyping to identify cells expressing major histocompatibility class II surface antigen, 4) measuring phagocytic capabilities of monocytes and 5) assessing neutrophil activation. Preliminary data indicate differences in these lymphocyte function tests among normal-fed, normal-fasted and ill anorectic cats.[b]

ACUTE-PHASE PROTEIN TESTING

Laboratory tests available in other species, but not yet fully investigated as parameters of nutritional assessment in dogs and cats include serum prealbumin, transferrin, retinol-binding protein, fibronectin and cholesterol concentrations and total iron-binding capacity. A group of down-regulated proteins (prealbumin, transferrin, fibronectin and retinol-binding protein) and up-regulated proteins (ceruloplasmin, α-1-antitrypsin, α-1-acid glycoprotein and C-reactive protein) may prove useful in nutritional assessment. These proteins have relatively short half-lives (two hours to 10 days) in people, and have been suggested as indicators of the patient's energy and protein status. The half-lives of these proteins are unknown in cats and dogs but are assumed to be relatively short and related to the nutritional status of the patient.

GENE EXPRESSION TESTING

Gene expression of metabolic enzymes and hormones in the fed vs. fasted state can be differentiated. The means by which food affects genetic activity probably differs among responding organs but also depends on the duration of the fast and the composition of the refeeding food. Many nutritional studies using animals have demonstrated the expression of enzymatic genes using a three-day fast followed by refeeding specific dietary formulations. For example, this starvation-refeeding paradigm has demonstrated that fasting causes adaptive increases in the concentrations of many hepatic and renal enzymes that convert amino acids to precursors of glucose and fatty acids. Conversely, feeding a carbohydrate diet decreases the activity of those enzymes involved in gluconeogenesis and amino acid catabolism. Fasting and refeeding alter the structure of chromatin in regions near the structural genes involved in metabolic regulation. The alterations in chromatin also depend on the amount of carbohydrate, protein and fat in the refeeding food. The method of refeeding affects transcriptional regulation of certain genes. In the future, it should be possible to more accurately assess the metabolic state (i.e., nutritional status) of animal patients by measuring the activities of specific enzymes, cell receptors and gene signaling pathways and then to administer an appropriate refeeding formulation.

ENDNOTES

a. Saker KE, Virginia-Maryland Regional College of Veterinary Medicine, Blacksburg, VA, USA. Unpublished data. October 1997.
b. Saker KE, Remillard RL, Virginia-Maryland Regional College of Veterinary Medicine, Blacksburg, VA, USA, and Angell Memorial Animal Hospital, Boston, MA, USA. Unpublished data. January 1997.

The Bibliography for **Box 25-2** can be found at www.markmorris.org.

[DM] protein) with adequate caloric intake (19% DM fat) had below normal serum albumin and total protein levels but normal globulin concentrations after four weeks (Davenport et al, 1994). As an indicator of morbidity and mortality, serum albumin concentration is a reliable tool (Mendez et al, 2005). Numerous studies in people have demonstrated that a low serum albumin value correlates with complications during recovery. It is not recommended, however, to use serum albumin levels alone to determine nutritional status in critical care patients, because multiple factors can lead to hypoalbuminemia without malnutrition (Makhija and Baker, 2008). Establishment of an accurate and sensitive nutritional assessment protocol for veterinary patients, such as the Subjective Global Assessment used in people, would provide a valuable tool for veterinary practitioners.

Decreased serum protein levels may occur in more acute states of inadequate protein intake relative to a large protein loss (e.g., protein-losing enteropathies, open abdomen). In starving animals, loss of muscle mass decreases the body's protein reserves and, together with a slower rate of protein turnover in the remaining muscle, decreases the body's ability to synthesize proteins in response to metabolic needs (Tomkins et al, 1983). Such patients are poor surgical candidates because the body's protein reserves (muscle mass) have

been catabolized to maintain the higher priority protein pools. If surgery can be safely postponed, several days of preoperative nutritional support in such patients is advisable. Only one to three days of adequate energy and protein intake may be required to up-regulate hepatic and muscle anabolic enzymes (Zeiderman et al, 1989).

Serum potassium and urea nitrogen concentrations may also be lower in anorectic patients because these variables are largely affected by food intake on a day-to-day basis. Urea nitrogen, however, tends to increase in endstage starvation because muscle is catabolized for energy when fat stores are depleted. Serum creatine kinase levels have also been evaluated as a possible marker in feline malnutrition and refeeding (Fascetti et al, 1997). Creatine kinase concentrations, however, will also increase and decrease in many disease states (Kitagawa et al, 1991). Several different types of tests that may lead to better nutritional assessment are currently under investigation (**Box 25-2**).

Risk Factors

Physiologic State

The physiologic status of the patient should be noted. This is relatively simple but rarely noted in the medical record. Knowing the gender, reproductive status, age and activity level of a patient aids in the nutritional evaluation. For example, a neutered bitch at less than optimal weight and body condition (BCS 2/5) is clearly very different from one currently lactating for eight puppies. Dietary recommendations should reflect the obvious difference in energy requirement. Neuter status can alter metabolic rate and energy needs (Root et al, 1996; Flynn et al, 1996). The metabolic processes of growth, gestation and lactation do not necessarily cease when a dog or cat becomes acutely ill. Several days of inadequate energy intake may be necessary before the hormonal milieu for growth, gestation or lactation is down regulated. Environmental temperature is usually a minor risk factor because most hospitalized dogs and cats are kept indoors.

History of Malnutrition

Patients fed homemade foods, table foods, vegetarian or single item foods are at greater risk for developing subclinical nutritional imbalances and warrant further nutritional assessment. Foods designed, formulated or prepared by owners may not be nutritionally complete, balanced or consistent (Chapter 10). These patients may not only have protein-calorie malnutrition, but are more likely to have several vitamin and mineral imbalances concurrently (e.g., calcium and certain micromineral deficiencies and/or subclinical vitamin A and D toxicoses).

Patients with a history of nausea, vomiting and diarrhea are at increased risk of malnutrition because nutritional intake probably has been less than optimal before admission. Nutrient intake may be voluntarily decreased with nausea, whereas vomiting and diarrhea can compromise nutrient digestion and absorption. Such clinical signs are also associated with additional losses of body protein.

Key Nutritional Factors

The primary focus of the key nutritional factor discussion is on enteral foods. However, some information regarding parenteral nutrition (Chapter 26) is included here and in the "Other Nutritional Factors" section that follows.

The Association of American Feed Control Officials (AAFCO) allowances (2008), and the "recommended allowances" listed in the National Research Council (NRC) Nutrient Requirements of Dogs and Cats (2006) are based on healthy animals, but are often referenced when estimating average nutrient requirements of critically ill dogs and cats to be fed enterally. This approach has been considered appropriate because most foods used in assisted feeding have nutrient digestibilities greater than those of typical pet foods (AAFCO, 2008) and, therefore, the actual available nutrient level provided by these foods would be greater than the referenced estimate. Assessment of the critical care patient may reveal nutritional factors that are not accounted for by AAFCO or the NRC; therefore, the practice of using these references for critically ill patients should be approached with caution. When estimating nutrient intakes for patients receiving parenteral nutrition, the NRC "minimal requirement" recommendations are probably better estimates than AAFCO allowances, because NRC minimum requirement recommendations were typically determined using synthetic foods, which better approximate 100% availability (2006). In addition to assuring that enteral foods intended for critically ill patients meet AAFCO (or some other credible regulatory agency) allowances, special emphasis is placed on the key nutritional factors and their recommended levels discussed below and summarized in **Table 25-5**.

Unlike the key nutritional factor recommendations for normal and clinical conditions described in the rest of this book, in critical care nutrition, nutrient requirements are conventionally expressed on an energy rather than on a DM basis (Chapter 1). This designation is primarily an extension of the units used in actual clinical metabolic trials. In addition, nutrient profiles of oral liquid products and parenteral solutions used in nutritional support/recovery are more commonly expressed on an energy rather than on a DM basis.

Fluid and Electrolyte Therapy

Initial support often involves management of fluid, electrolyte and acid-base disorders. The water requirements in ml for normal healthy animals approximate their daily energy requirement (DER) in kcal (i.e., 1 kcal [4.184 kJ] DER = 1 ml of water). Fresh, clean water should be available to patients at all times, unless the patient requires a period of nothing per os. Most patients in an intensive care unit (ICU) have venous catheters in place and receive crystalloid fluid therapy. These patients may have fluid restrictions or, conversely, may require diuresis. In these cases, the water or fluid administered will not be equal to the patient's DER. Daily maintenance fluid requirements are approximately 60 ml/kg body weight/day.

The patient's fluid and electrolyte (sodium, potassium, calcium, magnesium and phosphorus) balance should be near normal limits before assisted feeding is begun. Nutritional support

Table 25-5. Key nutritional factors for commercial liquid or blended foods for canine and feline patients requiring enteral nutrition (EN) support.

Factors	Recommended food levels
Water	Correct dehydration with parenteral fluid therapy before starting assisted feeding. Supply at 1 ml/kcal DER unless patient requires fluid restriction or diuresis. Typical daily maintenance fluid requirement is 60 ml/kg body weight.
Electrolytes	Major electrolyte disorders, acid-base abnormalities and blood glucose levels should be corrected before instituting EN support.
Osmolarity	250 (optimal) to 400 mOsm/liter.
Energy density	Supply 1 kcal/ml (as standard minimum). If the patient is not eating at least RER per os, provide nutritional support by assisted-feeding techniques to meet this requirement. By the fifth day of food deprivation or longer, patients should receive the majority (60 to 90%) of their calculated RER as lipid. If using a liquid or blended food, select a product that provides 1.0 to 2.0 kcal/ml (1.0 to 2.0 kcal/g), as fed.
Digestible carbohydrate	Dogs and cats: 2 to 4 g/100 kcal is a safe starting point for refeeding. Increase to 6 to 10 g/100 kcal 3 to 4 days into the refeeding process.
Protein	Dogs: Use a food that provides 5.0 to 12.0 g protein/100 kcal. Cats: Use a food that provides 7.5 to 12.0 g protein/100 kcal.
Arginine	≥146 mg arginine/100 kcal for dogs. ≥250 mg arginine/100 kcal for cats.
Glutamine	≥500 mg/100 kcal.
Fat	Provide a calorically dense food (5 to 7.5 g fat/100 kcal), except in cases in which high fat content is not tolerated. Provide a low-fat content food (2.0 to 3.5 g fat/100 kcal) if fat restriction required*

Key: DER = daily energy requirement, RER = resting energy requirement, to convert kcal to kJ, multiply kcal by 4.184.
*For example, patients with pancreatitis.

should not be initiated until the patient is hemodynamically stable because administering enteral or parenteral nutrition may further compromise the patient. Nutritional support should not be initiated as a "last ditch" effort in unstable patients. Major electrolyte disorders, acid-base abnormalities and blood glucose levels should be corrected before instituting enteral or parenteral nutritional support. It is also desirable to correct severe tachycardia, hypotension, colloid and volume deficits before starting assisted feeding (Minard and Kudsk, 1994). A practical goal is to begin nutritional assessment and support within 24 hours of hospitalization for the injury or illness (Burkholder, 1995).

Osmolarity

Osmolarity refers to or represents the number of solute particles per liter of solution. Serum concentrations greater than 310 mOsm/l in dogs and greater than 330 mOsm/l in cats are usually considered hyperosmolar (Tilley and Smith, 2004). During enteral nutritional support, the osmolarity of a food appears to have the most significant clinical impact on GI function and stool character (i.e., presence of diarrhea) (Pasulka and Crockett, 1994). In general, osmolarity of commercial pet foods is not reported. Instead digestibility of typical dry or moist foods is evaluated and, among other things, reflects the potential of a food to be tolerated by the GI tract. As digestibility increases, the osmolarity decreases, allowing for greater absorption of ingesta/digesta and minimizing the draw of excess water into the GI tract. Conversely, the osmolarity of liquid foods is reported for veterinary and human products. To optimize GI function, transit time and stool character, liquid foods of 250 to 400 mOsm/l are recommended.

Along with GI tolerance, another clinical concern affects critical care patients fed hyperosmolar foods. As described previously, these patients often exhibit insulin resistance associated with the stress response to illness and/or trauma. Liquid foods providing increased digestible (soluble) carbohydrate-derived calories are hyperosmolar (>400 mOsm/l). This promotes and perpetuates a hyperglycemic state, thus increasing the risk of the hyperglycemic hyperosmolar syndrome (Schaer, 2005). Specific concerns for delivery of hyperosmolar nutrient solutions intravenously are discussed in Chapter 26.

Energy and Energy Density

Knowing a patient's approximate caloric requirement is important because feeding more of any food than is necessary may cause metabolic complications. Overfeeding patients is possible through a feeding tube or with parenteral nutritional support. In people and several animal models, excessive carbohydrate intake was associated with hyperglycemia, hypercarbia, fatty liver, increased ventilatory drive and failure to wean from a ventilator (Deitel et al, 1983). Excessive fat administration has been associated with hyperlipidemia, hypoxia, increased rate of infection and higher postoperative mortality (Lowry and Brennan, 1979).

The proportion of fat and carbohydrate supplying calories to hospitalized patients should be similar to that which the liver is estimated to be using from body stores (**Figure 25-2**). Caloric density is important in both enteral and parenteral feedings when volume is limited. Enterally fed patients can be volume restricted by gastric or intestinal sensitivities. Parenterally fed patients can be fluid restricted due to cardiorespiratory diseases and functional disabilities. In general, most dogs and cats tolerate the volume of food or solutions that meet the patients' RER within easily tolerated volumes when the caloric density is approximately 1 kcal/ml.

In malnutrition, without disease or injury, decreased T_3 concentrations decrease the metabolic rate in an effort to conserve functional protein and energy stores. However, with an ongoing disease process or traumatic injury, the neuroendocrine responses to stress increase the metabolic rate above that found in simple starvation. Respiration calorimetry measurements of more than 3,000 people with a wide variety of diseases, specifically excluding hyperthyroidism, showed that 90% of the patients had energy requirements from 15% above to 15% below RER (Boothby and Sandiford, 1924). The energy expenditure in people with trauma peaks in three to four days

and then subsides by Days 7 to 10 unless complicated by sepsis (Moore and Moore, 1994). Energy expenditure of people with other disease conditions probably follows a similar pattern with varying requirements above RER that may occur over time.

Hospitalized veterinary patients are assumed to be similar to ill people and their DER is very near their RER. Results of a few preliminary respiration calorimetry measurements in dogs with specific disease conditions suggested that most had requirements near RER (Walton et al, 1996; Ogilvie et al, 1996). Estimating the RER of hospitalized patients should be calculated by the equation RER = $70(BW_{kg})^{0.75}$ (Chapter 5). Most hospitalized veterinary patients should be fed at their calculated RER, realizing their actual energy requirement is likely to change over the course of the disease process and recovery. In human surgical patients, there was relatively little additional benefit to increasing intake after half of the caloric requirement of patients had been achieved (Elwyn et al, 1981). Therefore, initially feeding patients at RER, or at least 60% of RER, if 100% RER is not possible, is a rational and safe recommendation that decreases the probability of metabolic complications. Regular nutritional assessment of the patient is strongly recommended to adjust initial feeding rates.

There are exceptions when the caloric requirement will be greater than RER. Particular cases have energy requirements 1.3 to 2.1 x RER as determined by bedside respiration calorimetry in people (Moore and Moore, 1994). For example, according to indirect respiration calorimetry, people with severe closed-head and brain injury have energy requirements 40 to 60% above their calculated RER (Ott et al, 1990). Brain injury apparently increases oxygen consumption and acute-phase protein synthesis, which subsequently increase patients' caloric and protein requirements significantly above RER. Energy requirements of twice RER appear to be the upper limit in the most severe head injuries. Energy expenditure may be 30 to 50% above RER in patients with multisystem trauma. Severely burned patients also have energy and protein requirements 80 to 100% above RER, relative to the extent of skin damage and surface area exposed (Moore and Moore, 1994). The body loses heat, moisture and protein through wounds that have little or no epithelial covering. The patient's actual metabolic rate and resultant energy requirement are related to the degree of trauma, disease and/or complications and can only be approximated in a clinical setting.

The energy density of foods intended for patients requiring assisted feeding is often reported relative to the water or fluid content of the food. This is because an animal's energy requirement in kcal is approximately equal to its water requirement in ml and most critical care feeding is done in a liquid form. Thus, when patients are fed a sufficient amount of food to meet their energy requirements, they also meet their requirements for water. The recommended energy density of a food intended for assisted feeding (enteral or parenteral nutrition) is 1 to 2 kcal/ml.

Adjusting for Protein Calories

Calculating and adjusting for protein calories is of minor consequence when feeding at RER. If one assumes that part of the caloric intake is to be supplied by protein, then that fraction of protein intake that is used for energy vs. that which is used for protein synthesis must be estimated because the same amino acid cannot do both. Theoretically, if protein were supplied at 4 g/100 kcal to the patient but all of it was oxidized for energy with none going to synthesis, the protein could only account for 14% (4 g x 3.5 kcal/g) of the total caloric intake at best.

The most conservative and simplest method is to first provide the entire caloric need with fat and carbohydrate, and then meet the protein requirement entirely with amino acids, and not estimate the fraction of the protein that may be catabolized vs. anabolized. This method will not shortchange either the caloric or protein requirement because the fraction of amino acids actually used for energy will provide only a small amount (<15%) of additional calories. Essential amino acids provided by food are most efficiently used in protein synthesis and should not be used for energy, if at all possible. In summary, protein calories may be taken into account, however, the contribution is small and not significant.

Carbohydrate

Carbohydrate usage during the healthy fed state results in energy storage (glycogen) or energy production (ATP) in a very efficient manner. Conversely, use of this nutrient in refeeding scenarios during the unfed state is less efficient and can result in adverse metabolic and physiologic states, particularly in diseased or injured patients. Insulin resistance, presence of bacteria with infection, diminished production of digestive enzymes, altered GI absorptive capacity and alteration of gut microbiota complicate the recovery process and influence dietary carbohydrate tolerance. The two major clinical manifestations currently associated with carbohydrate intake include altered glucose control and diarrhea. Consequently, most foods formulated for recovery are low in carbohydrate content.

Tight glucose control in ICU patients has been regarded as beneficial because both hyperglycemia and hypoglycemia have detrimental effects on tissue function and clinical outcome, but recently this goal has been challenged (Elia and De Silva, 2008). Adverse effects of hyperglycemia that can predispose the patient to infection and delay recovery from illness include osmotic shifts, glucosuria, altered immune and endothelial cell function and promotion of free radicals. Conversely, hyperglycemia is a normal response to injury or stress. Glucose is needed for wound healing and inflammatory/immune cells that are involved in the metabolic response to injury, as well as other physiologic functions. A review of several human ICU-based studies indicated that narrowly controlled glucose did not significantly reduce hospital mortality (Elia and DeSilva, 2008). Circulating glucose is the major energy source for the brain. Although in most ICU patients, during the unfed state and during diabetic ketoacidosis, the brain can use ketones as an energy source. Once re-fed, the circulating insulin acts to suppress ketone body concentrations, which once again leaves glucose as the major energy source for the brain. Reducing the blood glucose concentration in some patients (lower than seen with stress/injury response) could have detrimental effects such

as neuronal dysfunction, neuronal death or cerebral infarction (Gandhi et al, 2007). In contrast, partial or complete cessation of nutrition has been identified to be one of the major risk factors for developing hypoglycemia (Elia and De Silva, 2008). A low-carbohydrate food can amplify the onset of the hypoglycemic state, particularly in small-breed dogs.

Diarrhea appears to be a common problem in critically ill patients during refeeding. Malabsorption of dietary carbohydrate or fat, hyperosmolar formulas (generally high carbohydrate content), and feeding high volumes of enteral fluid have all been reported as causal factors (Mutlu et al, 2001). Complications of diarrhea include effects on hydration, acid/base status, mineral balance, contamination of wounds, decreased colonic fermentation and reduced butyric acid production (Thaklar et al, 2005; Kien et al, 1999). Traditional attempts to minimize the osmotic diarrhea, believed due to carbohydrate malabsorption, have focused on limiting the dietary carbohydrate intake (Kein et al, 2004). Small intestinal carbohydrate malabsorption (breath H_2) and colonic fermentation, stool volume and total enteral fluid volume were measured in burn patients receiving a high carbohydrate (Vivonex TEN) enteral food over a four-week period. Although all patients had diarrhea over several weeks, the lack of correlation of either carbohydrate intake or breath H_2 with stool volume suggested diarrhea was due to factors other than carbohydrate malabsorption (Thaklar et al, 2005). Notwithstanding, prevention and possibly treatment of osmotic diarrhea has been addressed by delivery of lower osmolarity nutrient solutions.

The value of dietary carbohydrate in maintaining an adequate and healthy population of gut microbiota cannot be overlooked. The intestinal microflora has been proposed as an environmental factor responsible for control of body weight and energy metabolism. Fermentation of non-digestible dietary fiber (insoluble carbohydrate) and resistant starches (oligosaccharides) along with numerous other mechanisms are linked to the health of gut microflora and energy metabolism. The major part of the microbiota is present in the colon where food products have escaped digestion, so the biologic functions controlled by this microflora seem to relate to effectiveness of bacteria to harvest energy that has been ingested, but not digested, by the patient. Human and rodent studies similarly conclude that microbiota can extract energy from non-digestible carbohydrate based on species (Turnbaugh et al, 2006; Ley et al, 2006), suggesting a benefit of providing adequate insoluble carbohydrate in the diet of critically ill patients. Additionally, fructooligosaccharides taken in the diet (5 to 20 g/day) improved mucosal barrier function, improved glucose tolerance and insulin homeostasis in human patients and rodents (Cani and Delzenne, 2007). Another study further highlights the value of resistant starches and dietary fiber as sources of short-chain fatty acids in critically ill/injured patients. Increased short-chain fatty acids, in particular butyrate, significantly enhanced colonic anastomosis healing and increased intestinal bursting pressure postoperatively in rats (Campos et al, 2008).

Perioperative carbohydrates minimize postoperative complications. Intracellular tight junctions maintain the intestinal epithelial barrier. Formation of dysfunctional tight junctions after stressful events such as surgery, contribute to postsurgical complications and delayed recovery (Bouritius et al, 2008). Adequate intestinal blood flow along with the mononuclear phagocytic system, located predominately in the liver, work in tandem to protect against bacterial translocation. Studies have indicated that glucose supplementation increases intestinal blood flow and that hepatic glycogen content contributes to increased survival rate by maintaining the liver system. Clinically these findings were substantiated when rats receiving a carbohydrate drink consisting of glucose, maltose and polysaccharides (12 g carbohydrate/100 ml) for six days before major abdominal surgery retained intestinal barrier function and were protected from translocation of bacteria to distant organs compared to cohorts not fed carbohydrates (Bouritius et al, 2008). Based on these studies, dietary carbohydrates maintain the mucosal barrier, hasten tissue healing, minimize complications and shorten hospital stays.

The dietary carbohydrate level in the initial refeeding of critically ill patients should be based on patient assessment and timing. Dampening the body's natural response of hyperglycemia to illness/stress may not be as beneficial as previously thought; carbohydrates in various forms have metabolic and physiologic value to patients. Conversely, promotion of a severe hyperglycemic state in these patients is contraindicated to recovery. On average, "recovery" type foods provide 2 to 4 g carbohydrate/100 kcal, with increased fat and/or protein content; this appears to be a safe starting point for refeeding. Then, consider transitioning to a higher carbohydrate food (6 to 10 g/100 kcal) and evaluating the insoluble carbohydrate (fiber) source three to four days into the refeeding process, based on patient reassessment.

Protein

Protein in the body is always in flux between synthesis and breakdown. Protein synthesis requires that amino acids be present within cells at the correct time and ratio so that a protein may be constructed successfully. Protein degradation involves the release of amino acids, and if the amino acid is deaminated, the ketoacid analog is converted to glucose or fat and the amino group enters the hepatic urea cycle and is ultimately excreted in the urine. Under most circumstances, about 15% of the RER comes from the oxidation of amino acids (Kinney, 1988). Providing a dietary protein source to patients in catabolic states spares endogenous skeletal muscle protein and supplies essential amino acids and amino groups for acute-phase proteins and the immune response. Excessive dietary protein should be avoided in patients with kidney or liver disease (Chapters 37 and 68). However, high dietary protein intakes are handled well by most canine and feline critical care patients to replace dietary carbohydrate when carbohydrates are not well tolerated.

Protein administration should complement nonprotein calories because amino acids will be oxidized for energy when a patient's total energy need has not been met first. Sufficient calories must be available from fat and/or glucose before ingest-

ed amino acids will be used for tissue synthesis and repair (Mallet, 1984). Excessive protein feeding requires energy expenditure to rid the body of excess nitrogen, which, in certain patients, may or may not be handled well by the liver (urea cycle) and kidneys and can result in hyperammonemia with accompanying clinical signs of encephalopathy. Conversely, insufficient protein has been linked to low albumin concentrations, poor immune response, impaired healing and increased risk of wound dehiscence and muscle wasting. The most efficient use of protein in people occurs when 2 to 6 g protein/100 kcal are administered (Stein, 1986).

Commercial products intended for enteral support of canine and feline critical care patients provide between 5.5 and 14.3 g protein/100 kcal. Due to a lack of evidence to the contrary and because these products appear to work well in critical care patients, a range of 5.0 to 12.0 g protein/100 kcal is recommended for canine patients and 7.5 to 12.0 g protein/100 kcal is recommended for feline patients. Because of the overlap of these recommendations, several commercial products intended for enteral support are designated for use in both canine and feline critical care patients.

When formulating parenteral nutritional support, it is prudent to first provide for total caloric needs with carbohydrate and fat, and then meet the protein requirement. If sufficient calories are supplied to patients as either fat or carbohydrate, then most of the essential amino acids will be used for protein synthesis and not burned for energy. A starting point of 2 to 3 g protein/100 kcal parenterally (Remillard and Thatcher, 1989) can be used for most dogs that can excrete protein waste products and do not have an extraordinary protein loss. A lower range (1 to 2 g/100 kcal parenterally) is a more reasonable estimate for patients with kidney or liver diseases. A higher range (3 to 4 g/100 kcal parenterally) is a more reasonable estimate for cats because of their constant state of gluconeogenesis from amino acids. Protein intake can then be adjusted based on the patient's needs and ability to handle the initial protein recommendation (e.g., decreasing serum albumin concentration or encephalopathic signs).

In addition, specific nutrients affect immunocompetence. Some nutrients act directly on the lymphoid system and immune cell function, thereby altering host immune response to pathogens. As an example, arginine, glutamine and dietary nucleotide-enriched foods are associated with significant reduction in wound infection and length of hospital stay in human burn patients.

Arginine

Arginine is essential to traumatized patients. It has a marked immunopreserving effect in the face of immunosuppression induced by protein malnutrition and cancer. In postsurgical patients, arginine supplementation enhances T-lymphocyte response and augments T-helper cell numbers, with a rapid return to normal T-cell function postoperatively, compared with findings in control patients (Bower et al, 1995). These data taken together suggest that arginine supplementation may increase or preserve function in high-risk surgical patients and theoretically enhance the host's capacity to resist infection. Arginine enrichment stimulates the immune system, improves wound healing and decreases morbidity and mortality in burn patients. A feeding regimen with arginine as 9% of the protein source has been suggested and tested in burn patients. Those receiving the arginine-enriched food had a significant reduction in the incidence of wound infection and shorter hospital stays. As a nutrient substrate, arginine appears nontoxic and may benefit surgical patients at increased risk of infection (Goffschlich et al, 1990). The optimal arginine intake for people is unknown, so selection of enteral foods based solely on arginine content is not recommended for human patients.

Numerous studies in a variety of animal models demonstrated the efficacy of arginine-supplemented foods in reducing the catabolic response to major trauma, sepsis and injury and in improving the immune response after a variety of adverse stimuli. For example, a food containing arginine as 2% of the total nonprotein calories significantly increased survival after 30% surface burns (Irenton-Jones and Baxter, 1990). Furthermore, in animal studies, exogenous arginine supplementation consistently improved nitrogen retention, protein turnover and wound healing. Arginine augments cellular immunity, as evidenced by enhanced skin allograft rejection in normal mice, and improves delayed hypersensitivity responses.

Arginine is an essential amino acid in dogs, cats and people. Therefore, most pet foods meeting AAFCO nutrient concentrations should contain at least 146 mg arginine/100 kcal for adult dogs and 250 mg arginine/100 kcal for adult cats (providing approximately 80 to 200 mg/kg body weight). Arginine content of human enteral products is variable but usually stated on the label. Human enteral products and parenteral nutrition solutions must contain at least adequate amounts of arginine if used for more than a few days in dogs or cats.

Glutamine

Glutamine is an amino acid that plays an important role in many cellular processes. Human studies suggest that glutamine concentrations in whole blood and skeletal muscle decrease markedly following injury and other catabolic states, thus making it "conditionally" essential during serious injury or illness (Lacey and Wilmore, 1990). Numerous clinical trials suggest that intervention with glutamine reduces rates of infectious complications in postsurgical patients and complications and mortality rates in critically ill patients (Novak et al, 2002). Replicating cells such as fibroblasts, lymphocytes and intestinal epithelial cells have high glutaminase activity and consume glutamine, but the intracellular level of glutamine remains low. The mechanism linking the beneficial effect of glutamine on attenuating cellular metabolic dysfunction and enhancing cell survival depends on glutamine-induced enhancement of specific heat shock proteins (Peng et al, 2006). These findings may be important for patients with large wounds or inflammation associated with infection.

The controversy persists as to which route of glutamine administration (enteral or parenteral) is most effective at

improving clinical outcomes in critically ill patients. At least 80% of the published data in laboratory animals demonstrate a positive effect with glutamine-enriched feedings. Positive effects include enhanced protein metabolism, intestinal and pancreatic repair and regeneration, nutrient absorption, gut-barrier function, systemic and intestinal immune function and animal survival. The mechanism(s) for these effects are not well clarified, but studies suggest several possibilities. First is the inter-organ conversion of glutamine-derived citrulline to renal arginine synthesis (Ligthart-Melis et al, 2007). A second mechanism is through glutamine attenuation of the gut-derived inflammatory response (Wischmeyer, 2006).

Numerous animal studies have demonstrated the value of enteral glutamine during stress. For example, rats undergoing abdominal radiation and fed glutamine orally for eight days following the stress had significantly increased jejunal villous number and height and an increased number of mitoses per crypt, whereas non-irradiated control rats fed the same food without glutamine supplementation had no significant increase in mucosal cell activity (Klimberg et al, 1990). Similarly, dogs had an increased intestinal requirement for glutamine during the immediate postoperative phase (less than seven days), but uptake rates returned to normal later during the recovery phase (after 10 days) (Souba et al, 1987). Oral glutamine supplementation influences GI function, along with cell morphology. For example, rats undergoing ischemia-reperfusion injury maintained small intestinal barrier function when provided with enteral glutamine (Kozar et al, 2004).

Provision of exogenous glutamine to stressed patients might better support the metabolic requirements of the small intestine and possibly decrease the rate of systemic protein catabolism (Wischmeyer, 2006), therefore, supplementation is warranted. The optimal concentration of glutamine for different disease states is still under study. It is presently unclear whether glutamine must be in the free form to be beneficial or if the protein-bound form is also beneficial in maintaining gut integrity. Most enteral foods contain some protein-bound glutamine but the glutamine concentration of these products must be estimated. Some enteral products have added free glutamine. The glutamine content of these products is often stated on the label. Glutamine levels in commercial enteral foods intended for critical care canine and feline patients should be at least 500 mg/100 kcal.

Although based on human studies indicating the gut preferably takes up enterally administered glutamine compared with intravenously provided glutamine (Ligthart-Melis et al, 2007), evidence suggests intravenous glutamine can provide benefits as well. In protein-depleted rats, intravenous glutamine supplementation resulted in increased villous height, increased small-bowel mucosal weight, enhanced DNA activity (O'Dwyer et al, 1989) and improved DNA content and sucrase and lactase activities. Parenteral admixture supplemented with 2% glutamine and administered for 48 hours before and 72 hours postintestinal abdominal surgery in undernourished dogs improved ileal morphology, increased CD4:CD8 cells, select immunoglobulins and mononuclear cell function and resulted in fewer postsurgical diarrhea days compared to dogs administered non-glutamine supplemented parenteral admixture (Saker et al, 2001). In short-term studies in rats and pigs, adding glutamine to nutritional intravenous solutions reduced some aspects of disuse intestinal atrophy and enhanced intestinal immune function (Remillard et al, 1998). Intravenous glutamine supplementation immediately following hemorrhagic shock partially restored the depletion of hepatic ATP, reduced cellular apoptosis and oxidative stress-associated cell damage in rat and feline models (Yang et al, 2007; Krizova et al, 2004), suggesting additional benefit from intravenous-glutamine supplementation during critical illness. Intravenous glutamine should probably be limited to short-term use (one week or less) just before oral refeeding. Inclusion of 2% L-glutamine via the intravenous route has been safely used in human and veterinary patients. However, it should be noted that inclusion of glutamine in parenteral nutrition solutions can be difficult to achieve due to solubility constraints.

Fat

Supplying the majority of calories as fat to critically ill patients has several benefits. Fat contains 8.5 kcal metabolizable energy/g and is therefore calorically dense compared to carbohydrate and protein. Therefore, more calories may be provided in a smaller volume to patients. After three to five days of not eating, the liver has shifted from glucose to fat metabolism, therefore providing more fat and less dextrose at this time reduces metabolic complications of nutritional support. Additionally, providing calories as fat rather than dextrose reduces CO_2 production, which noticeably reduces respiratory work in patients requiring oxygen therapy. On average, "recovery" type foods provide 5 to 7.5 g fat/100 kcal; this appears to be a safe starting point for refeeding. The exceptions include patients with pancreatitis or other conditions in which enteral intake of a high-fat food is not tolerated.

Other Nutritional Factors

Other nutritional factors can be important considerations for enteral foods or parenteral fluids for critical care patients. Factors such as vitamins and minerals are typically included in adequate amounts in commercial veterinary therapeutic enteral foods formulated for dogs and cats. However, these nutrients are important to consider in parenteral nutrition support. Other factors (nucleotides, essential fatty acids and antioxidants) may benefit veterinary patients based on the human critical care literature and case reports. The optimal daily dose and duration of provision have yet to be standardized for dogs and cats. Typical ingredients used in many enteral foods contain nucleotides, essential fatty acids and antioxidants. Some critical care foods have been specifically enriched, whereas others have not been and would need exogenous supplementation if they were deemed valuable. Provision enterally is more efficient and practical than through parenteral fluids.

B Vitamins

Folic acid, thiamin, riboflavin, niacin, pantothenic acid, pyri-

doxine and B_{12} are essential for hepatic metabolism of glucose, fat and protein. These are coenzymes for the tricarboxylic acid (TCA) cycle, ATP production and RBC metabolism. B vitamins are required in small amounts relative to other nutrients, but they are required daily and are necessary for efficient energy metabolism. Most commercial pet foods contain adequate amounts of these nutrients, so deficiency should not be of concern if the patient is eating or being fed enough food to meet its RER. B vitamins should be added to the fluids (1 to 2 ml of vitamin-B complex/1,000 ml of crystalloid fluid) of all patients that are not eating but receiving fluid therapy or parenteral nutrition support.

Microminerals

Zinc, copper, manganese, chromium and selenium are vital cofactors for optimal hepatic and peripheral metabolism of energy substrates. Microminerals (i.e., trace minerals or trace elements) are important cofactors (metalloenzymes) and participate in tissue repair and albumin synthesis; therefore, zinc, copper and manganese should be included in all food forms used for assisted feeding. Most pet foods contain adequate amounts of these nutrients, thus deficiency should not be of concern if the patient is eating enough food to meet its RER. Specialized solutions containing essential trace (zinc, copper and manganese) minerals can easily be added to parenteral nutrition solutions at approximately 1 ml per 100 kcal of solution.

Fat-Soluble Vitamins and Macrominerals

Hospitalized patients rarely need fat-soluble vitamins and macrominerals. Most patients have fat and hepatic stores of the fat-soluble vitamins sufficient to meet metabolic needs for months to years. However, administering fat-soluble vitamins should be considered in cases of prolonged malnutrition in which the patient is severely underweight with little to no fat stores (i.e., BCS 1/5). Most pet foods contain adequate amounts of these nutrients, thus deficiency should not be of concern if the patient is eating enough food to meet its RER. Fat-soluble vitamins are not added to parenteral nutrition solutions due to insolubility problems. It is easiest to administer a single dose of vitamins A, D and E by deep intramuscular injection to patients needing these vitamins. Such an injection supplies approximately one to two months of daily requirements.

Macrominerals (i.e., calcium, phosphorus, magnesium, sodium and potassium) are rarely needed by patients above that required to maintain serum electrolyte levels. Whole body stores of these minerals can be depleted but are usually easily corrected by intravenous administration. The distribution between the intracellular and extracellular fluid space can be a problem and imbalances should be corrected before assisted feeding is begun (**Box 25-3**). Sodium, potassium and magnesium levels may

Box 25-3. Refeeding Syndrome.

Refeeding syndrome in people is characterized by generalized muscle weakness, tetany, myocardial dysfunction, dysrhythmias, seizures, excessive sodium and water retention, hemolytic anemia and death due to cardiac or respiratory failure. A similar syndrome occurs less commonly in veterinary patients. When it does occur, it is most often seen in patients receiving parenteral nutrition or during inappropriate assisted enteral feeding and most commonly presents as hypokalemia or hypophosphatemia. Significant electrolyte shifts occur from extracellular to intracellular compartments as energy and amino acids are reintroduced. This electrolyte shift will occur regardless of the route of administration (i.e., enteral or parenteral). Often serum ion levels are deceptively normal in anorectic patients before refeeding begins (**Table 25-4**). However, when calories are reintroduced, particularly from carbohydrate, potassium and phosphate shift intracellularly with glucose resulting in hypokalemia and hypophosphatemia.

Potassium moves into cells with refeeding because glucose stimulates insulin release, which in turn stimulates the Na-K ATPase pump and glycogen synthesis, which requires 0.33 mEq potassium/g of glycogen. Phosphate moves into cells with refeeding to support the increased production of phosphorylated intermediary compounds of energy metabolism. Severe hypophosphatemia, hemolytic anemia and death have occurred in cats within 12 to 72 hours of refeeding with either an apparently normal or phosphorus-deficient diet. The refeeding formula should contain at least the Association of American Feed Control Officials recommended minimum allowance of 0.5% dry matter phosphorus.

In people, hypomagnesemia is another common electrolyte complication that must be corrected. Hypomagnesemia increases urinary excretion of potassium, exacerbates hypokalemia and causes hypocalcemia, which is refractory to supplementation until the hypomagnesemia is corrected. Little information is available about magnesium status in hospitalized dogs and cats; however, serum magnesium levels should probably be monitored in veterinary patients with abnormal serum electrolytes.

RECOMMENDATIONS FOR AVOIDING COMPLICATIONS OF THE REFEEDING SYNDROME

1. Anticipate the potential for the problem and re-feed with formulations known to contain adequate potassium, phosphate and magnesium levels and lowered digestible (soluble) carbohydrate content.
2. Use initial nutritional refeeding rates that do not exceed the patient's resting energy requirement (RER) and 2 to 6 g protein/100 kcal (parenterally) or 5.5 to 7.5 g protein/100 kcal (enterally). These rates can be increased as needed over subsequent days. Consider refeeding a high-fat, low-carbohydrate formula to patients that have not eaten for four to five days or more.
3. Monitor serum potassium, phosphate and magnesium levels as needed. Once a day is sufficient for most cases.
4. Supply water-soluble vitamins free choice, particularly thiamin, to facilitate energy metabolism.
5. Monitor patients daily for signs of fluid overload and congestive heart failure.

The Bibliography for **Box 25-3** can be found at www.markmorris.org.

become a concern in patients experiencing excessive urinary loss of those minerals due to intensive diuretic therapy. Most pet foods contain adequate amounts of these nutrients, thus deficiency should not be of concern if the repleted patient continues eating enough food to meet its RER. Calcium and magnesium are not added to parenteral nutrition solutions due to insolubility problems; however, phosphorus, sodium and potassium can be added to parenteral nutrition solutions at maintenance concentrations or for repletion if needed.

Nucleotides

Nucleotides are precursors of DNA and RNA, but they also participate in a number of metabolic reactions fundamental to cellular activity. Dietary nucleotides appear to be important for maintenance of normal cellular immunity and are vital to maintain host defenses against bacterial and fungal pathogens. Dietary nucleotides appear essential to the normal maturation of lymphocytes (Hall et al, 1998). In vitro mixed lymphocyte culture response and mitogen stimulation are suppressed in patients supported on a casein-based laboratory food. Such foods are nucleotide free. Mice maintained on nucleotide-free foods are much more susceptible to lethal infections caused by *Candida albicans* and *Staphylococcus aureus* and exhibit depressed macrophage bactericidal activity compared to nucleotide-fed counterparts. Similarly, animals fed a nucleotide-free food for six weeks had significant immunosuppression as demonstrated by enhanced cardiac allografts and diminished ability to survive a fungal challenge. These findings are significant because all commercially available parenteral and nearly all enteral human products are devoid of nucleotides.

The clinical value of nucleotides was evaluated in two separate studies that investigated the effects of a human enteral product enriched with arginine, nucleotides and omega-3 fatty acids (Impact[a]). In one study, researchers investigated the effects of this enriched enteral product on immune parameters of patients undergoing major abdominal surgery. In general, patients receiving the enriched product had enhanced immunocompetence and fewer infectious complications than patients in other groups. In the other study, a subset of patients with sepsis who were fed the enriched enteral product (Impact) had shorter hospital stays and a major reduction in the frequency of acquired infections vs. other groups (Bower et al, 1995). Though clinical gain was evident from the nucleotide-enriched food, it is not clear if the benefit was from nucleotides alone or from the combination of special nutrients provided in the food. There are no reported studies evaluating the clinical value of nucleotide-enriched foods for critical care veterinary patients, likely because pet foods that use meats and cereal grains as ingredients should provide adequate levels of dietary nucleotides. Despite the limiting data substantiating their clinical value in veterinary patients, dietary nucleotides are a vital component of regimens to maintain or restore immune function and host defense and, therefore, should be considered when choosing a critical-care food.

Omega-3 Fatty Acids

The effect of dietary fatty acids on the immune system depends on which fatty acid is fed and what specific aspects of the immune system are evaluated. Dietary fatty acids are thought to affect the immune system by three mechanisms: 1) altered eicosanoid synthesis, 2) changes in cell membranes that affect membrane-associated protein and receptor function and 3) changes in intracellular nonesterified fatty acid pools that affect cytokine production. Generally, omega-3 (n-3) fatty acids produce fewer inflammatory cytokines, whereas omega-6 (n-6) fatty acids produce more proinflammatory cytokines (Lands, 1992).

The capacity of tissues and WBC to produce pro- or anti-inflammatory prostaglandins and lipoxygenase products is largely determined by the amount and type of fatty acids present, which is mostly determined by concentration of dietary fatty acids. Omega-3 fatty acids, once incorporated into the plasma membrane, affect immune cell function by altering membrane fluidity and second messenger function, and by increasing production of dienoic prostaglandins, the 3-series prostaglandins and 5-series leukotrienes. These changes may be responsible for alterations in such cell functions as phagocytosis, production of interleukins and production of superoxides. A significant reduction in dietary omega-6 polyunsaturated fatty acids will lower production of proinflammatory eicosanoids and appears to be a prudent approach in nutritional support of immunocompromised, traumatized, postoperative or infected patients. Conversely, the inclusion of omega-3 fatty acids in such foods would seem to be beneficial in increasing antiinflammatory eicosanoid production. Findings suggest that marked improvement can be made in foods by adjusting the omega-6 and omega-3 components to ensure optimal immune function.

Clinical evidence suggests that dietary omega-3 fatty acids may benefit the management of severe inflammatory and autoimmune disorders in rodents and people. These less inflammatory metabolites alter immune function and may improve survival in patients in which the inflammatory process threatens to cause irreversible damage, as in septic shock or endotoxemia. Omega-3 fatty acids shift the response away from intense inflammation. In other studies, fish oil protected guinea pigs from endotoxic shock and lactic acidosis, providing them with a survival advantage (Fritsche and McGuire, 1996).

Timing of dietary omega-6 and omega-3 fatty acid manipulation is critical to influencing patient inflammatory response. The literature varies when reporting dosing route, concentration and species. Nonesterified fatty acids in tissues have been effectively altered within hours of oral dosing with omega-3 fatty acids. In cats, concentrations of specific fatty acids were altered in immune cell membranes within 28 days of enteral feeding (Saker, 2002); whereas in pigs, plasma phospholipid profiles differed significantly within eight days (Murray et al, 1991). Intestinal mucosa and plasma had an altered fatty acid profile within four weeks, whereas an alteration in the fatty acid profile of skin

generally occurred after six to 12 weeks of supplementation. In dogs, however, investigators found plasma fatty acid profile changes within two weeks after the onset of omega-3 dietary supplementation (Campbell and Dorn, 1992). Thus, there may not be enough time for dietary omega-3 fatty acid therapy to affect an acute inflammatory process, depending on the affected tissue unless the fatty acids were incorporated into the patient's dietary regimen before the onset of disease. The dietary dose that favors a less inflammatory cascade during a disease process is still not standardized across veterinary patients, but is suggested as an omega-6:omega-3 fatty acid ratio ranging between 5:1 to 1:1, depending on patient assessment.

On the other hand, chronic suppression of the inflammatory and/or immune response by feeding high levels of omega-3 fatty acids should be done cautiously and is not warranted in disease states in which a fully competent immune system is essential for survival and recovery. Studies in which mice were pre-fed (two to four weeks) extremely high levels of omega-3 fatty acids (40% of calories as fish oil) compromised their resistance in an infectious disease state (Chang et al, 1992). Platelet function was significantly diminished in healthy cats fed an enriched omega-3 fatty acid food (omega-6:omega-3 ratio of 1.3:1) for eight weeks (Saker et al, 1998). As with many other nutrients, excessive levels of omega-3 fatty acids can be detrimental.

FEEDING PLAN

The feeding plan discussion assumes that the health care team has determined that the patient is a candidate for nutritional support (see History and Physical Examination section, above).

Nutrients can be supplied enterally or parenterally (**Figure 25-5**). Enteral feeding provides adequate nutrition simply and cost effectively whether done orally or by feeding tube. Enteral feeding is usually preferred to parenteral feeding because it is less expensive, stimulates the systemic and GI immune systems, helps to maintain GI mucosal integrity and avoids most metabolic complications. However, nutrients must be administered parenterally when the GI tract is inaccessible or not functioning adequately enough to meet the patient's nutrient requirements enterally. Chapter 26 covers parenteral-assisted feeding.

Enteral-assisted feeding is providing nutrients to the patient using some portion of the GI tract. Patients that cannot or will not eat but who can digest and absorb nutrients from the small intestine should receive enteral-assisted feeding. Feeding via the GI tract is often the simplest, fastest, easiest, safest, least expensive and most physiologic method of feeding patients. Prior knowledge that a patient requires other medical and surgical procedures should also be considered when formulating an enteral assisted-feeding plan. For example, feeding tubes can easily be placed at the end of a procedure requiring anesthesia or tranquilization. Feeding tube placement must consider the treatment plan and owners' expectations. Some feeding tubes can only be used when the patient is in the hospital whereas other tubes may also be used for at-home feeding.

The two methods, enteral and parenteral, are not mutually exclusive; supplementing what the patient consumes voluntarily with parenteral calories and protein infusion is possible in many veterinary practices. Therefore, overall patient assessment, including evaluating a patient's ability to eat and assimilate food, is the first step in developing a feeding plan because it dictates the route, enteral, parenteral or both, for providing assisted feeding.

The food choice should be made based on a food's key nutritional factor profile and form of the food (i.e., liquid, moist) that best accommodates the specific nutritional support feeding method. For example, if a small nasogastric feeding tube were to be used, a liquid food of appropriate viscosity and key nutrient make-up would be selected. This is an added consideration compared to developing feeding plans for patients that do not require assistance.

Select the Feeding Method

Enteral Feeding Routes

ORAL FEEDING

Several routes exist for enteral feeding, but the first attempt usually should be oral feeding. Placing a bolus of food in the proximal portion of the mouth may stimulate the swallowing reflex and, if the patient offers no resistance, is a good method as long as the patient receives enough food to meet its RER. Simple syringe feeding of a liquid product is also a good method, if tolerated. For dogs, the syringe tip is placed between the molar teeth and cheek with the head held in a normal or lowered position; for cats, the syringe tip is placed between the four canine teeth (**Box 25-4**). The patient may choose to swallow the liquid or allow it to flow from the mouth down the esophagus by gravity. Some patients refuse to swallow boluses of food; therefore, forced feeding may increase the risk of food aspiration. Oral feeding should be discontinued if the patient does not swallow food voluntarily. Appetite stimulants may be used to induce food consumption in some patients; however, voluntary food intake rarely continues and their RER is often not met (**Table 25-6**).

Orogastric tubes require placement at each feeding but may provide a useful option for one or two days of feeding. They can be used as long as there is no nasal, pharyngeal or esophageal trauma or disease. Anesthesia is not required; therefore, this route can be used in patients that are an anesthetic risk. Neonates appear to tolerate multiple daily oral tube feedings better than adults. A red rubber or polyvinyl chloride tube (8 to 24 Fr.)[b] may be used with the tip placed in either the caudal esophagus or stomach. An indwelling feeding tube is the method of choice if enteral-assisted feeding is necessary for more than two days.

Feeding through an indwelling tube is easier and less stressful on the patient than forced feeding or repeated placement of an orogastric tube. Nasoesophageal, pharyngostomy, esophagostomy, gastrostomy and enterostomy are potential placement sites. Tubes should be placed in the most proximal functioning portion of the GI tract possible by the least invasive method. The stomach should be used whenever possible.

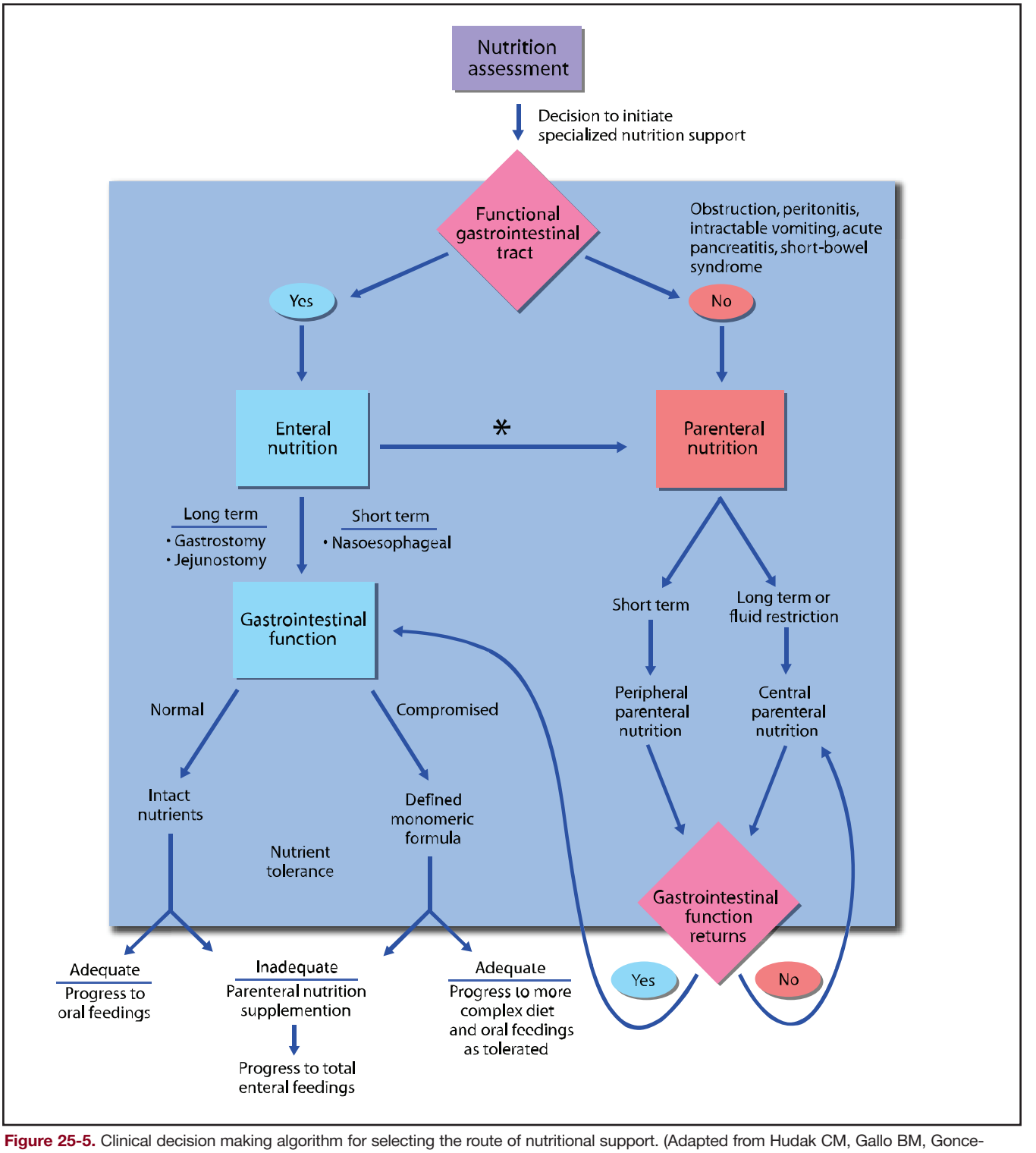

Figure 25-5. Clinical decision making algorithm for selecting the route of nutritional support. (Adapted from Hudak CM, Gallo BM, Gonce-Morton P, eds. Patient management: Gastrointestinal system. Critical Care Nursing, 7th ed. Philadelphia, PA: Lippincott, 1998; 771.)
*Nasoesophageal tube not tolerated or anesthesia not possible.

NASOESOPHAGEAL TUBES

Nasoesophageal tubes are generally used for three to seven days but are occasionally used longer (weeks, if moved to the opposite side every seven days). Polyurethane tubes (6 to 8 Fr., 90 to 100 cm) with or without a tungsten-weighted tip[c] and silicone[d] feeding tubes (3.5 to 10 Fr., 20 to 105 cm) may be placed in the caudal esophagus or stomach. The preferred placement of all tubes originating cranial to the stomach is in the caudal esophagus to minimize gastric reflux and subsequent esophagitis. An 8-Fr. tube will pass through the nasal cavity of most dogs. A 5-Fr. nasoesophageal tube is more comfortable for cats. Nasoesophageal feedings may be used in anorectic

Box 25-4. Syringe Feeding.

Patients are often fed a liquid or moist homogenized product by syringe on a short-term basis. For dogs, the syringe tip is placed between the molar teeth and cheek with the head held in a normal or lowered position (**Figure 1**). For cats, the syringe tip is placed between the four canine teeth (**Figure 2**). The patient may choose to swallow the liquid or allow it to flow down the mouth into the esophagus by gravity. Some patients refuse to swallow the liquid or food; therefore, force feeding may increase the risk of aspiration. Syringe feeding should be discontinued if the patient does not swallow food voluntarily.

Figure 1. Syringe-feeding technique for administering liquid or moist homogenized foods to dogs.

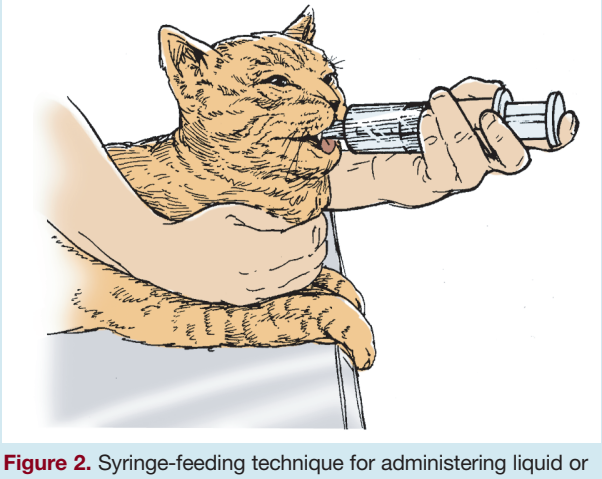

Figure 2. Syringe-feeding technique for administering liquid or moist homogenized foods to cats.

patients that do not have nasal, oral or pharyngeal disease or trauma. Anesthesia or tranquilization is not necessary to place a nasoesophageal tube, so this route provides enteral access to patients considered anesthetic risks. These tubes are most often used in the hospital, although conscientious owners can use nasoesophageal tubes at home (**Box 25-5**).

Table 25-6. Pharmacologic appetite stimulants.*

Cyproheptadine	Antihistamine and anti-serotonin effects. Dose cats at: a) 2 to 4 mg per cat per os once or twice daily, b) 2 mg per cat every 12 hours. May take up to 24 hours for a response. Give at this dosage for one week and then taper.
Diazepam	Short-lived appetite stimulant with sedative properties. Dose in cats varies: a) 0.05 to 0.15 mg/kg IV once daily to every other day or 1 mg per os once daily, b) 0.05 to 0.4 mg/kg IV, IM or per os. Eating may begin within a few seconds after IV administration. Food should be readily available.
Mirtazapine	Increases central nervous system serotonin while antagonizing serotonin activity in the GI tract. Dose cats at: 3.5 mg per cat per os every three days. May take up to 36 hours for a response.
Oxazepam	Short-lived appetite stimulant with sedative properties. Dose cats at 2 mg per cat (total dose) every 12 hours.
Prednisolone	Dose glucocorticoids at 0.25 to 0.5 mg/kg per os every day, every other day or intermittently as needed in dogs.

*Adapted from Plumb DC. Veterinary Drug Handbook, 3rd ed. White Bear, MN: Pharma Veterinary Publishing, 1999.

PHARYNGOSTOMY/ ESOPHAGOSTOMY/ GASTROSTOMY TUBES

Pharyngostomy and esophagostomy tubes (8 to 19 Fr.) may be placed in patients with disease or trauma to the nasal or oral cavity. The tip of the tube is placed in the caudal esophagus and the tube can be used for long-term (weeks to months) in-hospital or home feedings. **Boxes 25-6** and **25-7** describe pharyngostomy and esophagostomy tube placement, respectively.

For patients in which the pharynx and esophagus must be bypassed, gastrostomy tubes (mushroom-tipped, 16 to 28 Fr.)[e] can be placed either intraoperatively or percutaneously using an endoscope or a gastrostomy tube introduction device (Clary et al, 1996).[f] Gastrostomy tubes are also recommended for long-term feeding (weeks to months) if needed and have generally replaced pharyngostomy tubes, even when the esophagus is normal. Gastrostomy tubes are convenient and safe for in-hospital and at-home feedings.

There are two basic techniques for percutaneous placement of gastrostomy tubes. One technique uses an endoscope, whereas the other involves a "blind," nonendoscopic approach using a gastrostomy tube placement device or applicator. The advantages of percutaneous vs. surgical gastrostomy tube placement are ease and speed of placement, lower cost and less tissue trauma. **Boxes 25-8** through **25-10** describe these three methods of gastrostomy tube placement.

Any tube that has been placed into the esophagus or stomach allows for bolus or meal-type feeding schedules because the stomach acts as a food reservoir. Some patients, however, cannot tolerate bolus feeding into the stomach without vomiting. Such patients may benefit from a slow, continuous drip admin-

istration (by pump or gravity flow) of food into the stomach. Most veterinary patients tolerate bolus feedings of enteral nutritional support via nasoesophageal, esophagostomy or gastrostomy feeding tubes.

JEJUNOSTOMY TUBES

Jejunostomy tubes (J-tubes, 5 to 8 Fr.) are placed within the small intestine, ideally at the time of exploratory celiotomy, to bypass the proximal GI tract (Orton, 1986). J-tubes may also be placed by mini-laparotomy, or by threading a small feeding tube through a larger esophagostomy, pharyngostomy or gastrostomy feeding tube and placing the tip of the smaller tube in the jejunum (Crowe, 1986; Jergens et al, 2007). There is risk, however, that even a weighted-tip tube will be returned to the stomach by reverse peristalsis. Ideally, food should be administered through J-tubes at a slow, continuous drip delivered by a pump. Some patients, however, will tolerate frequent small-bolus feedings.

Amount to Feed and Feeding Schedule

Feeding plans require an understanding of the patient's metabolic state relative to changes in metabolism resulting from ongoing food deprivation. Estimating a patient's approximate caloric requirement is important because feeding more of any food than is necessary may cause metabolic complications. Overfeeding patients is possible through a feeding tube and should be avoided because it results in metabolic and mechanical complications. **Table 25-7** provides an example of using feeding guidelines to determine how much to feed and the feeding schedule.

The feeding schedule is often determined by the patient's ability to tolerate food and the logistics of feeding. Feeding an amount equal to the patient's RER during the first 24 hours of food reintroduction, if physically tolerated, is recommended. Feeding one-third of RER the first 24 hours and then increasing the amount by one-third every 24 hours until at RER is a more cautious approach to initial feeding, but is not always necessary. Foods should be warmed to room temperature, but not higher than body temperature, before feeding.

Food boluses must be infused slowly (over approximately one minute per 5 ml of food) to allow gastric expansion. Daily food dosage should be divided into several meals according to the expected stomach capacity. Gastric capacities for cats and dogs are typically 5 to 10 ml/kg body weight during initial food reintroduction. Maximum capacities as high as 45 to 90 ml/kg body weight have been measured in cats and dogs when fully re-alimented. Most often, the patient's RER can be met in volumes far less than these maximum gastric capacities. Salivating, gulping, retching and vomiting may occur when too much food has been infused or when the infusion rate is too fast.

Research in people has demonstrated that the stomach does not "shrink" during a prolonged fast, but rather the stretch receptors are more sensitive and stimulated by a smaller volume when refeeding occurs. Feeding should be stopped at the first sign of retching or salivating; then the meal size reduced by 50% for 24 hours and then increased by 25% gradually. Foods

Box 25-5. Nasoesophageal Tube Placement.

Nasoesophageal tubes are generally used for three to seven days, but are occasionally used longer (weeks if moved to the opposite side every seven days). Polyurethane tubes (6 to 8 Fr., 90 to 100 cm) with or without a weighted tip and silicone feeding tubes (3.5 to 10 Fr., 20 to 105 cm) may be placed in the caudal esophagus or stomach. The preferred placement of all tubes originating cranial to the stomach is in the caudal esophagus to minimize gastric reflux and subsequent esophagitis. An 8-Fr. tube will pass through the nasal cavity of most dogs; a 5- Fr. tube is more comfortable for cats.

The length of tube to be inserted is determined by measuring from the nasal planum along the side of the animal to the caudal margin of the last rib (**Figure 1**) and marking the tube at a point that is approximately three-fourths of the total measured length with a piece of adhesive tape or an indelible marker. This mark is how far the tube should be inserted. Tape will also provide a tab to secure the tube. The animal's nose is desensitized by placing a few drops of topical anesthetic (2% lidocaine or 0.5% proparacaine) into a nostril and tilting the head upward for a few seconds. The tip of the tube is lubricated with a water-soluble lubricant or 2 to 5% lidocaine ointment/jelly before passage.

To pass the tube, direct the tip in a caudoventral, medial direction into the ventrolateral aspect of the external nares. The head is generally held in a normal static position. As soon as the tip of the catheter reaches the medial septum at the floor of the nasal cavity in dogs, the external nares are pushed dorsally, which opens the ventral meatus, ensuring passage of the tube into the oropharynx (**Figure 2**). To aid passage, the proximal end of the tube is lifted as the nose is pushed upward (**Figure 2**). In cats, because of the lack of a well-developed alar fold, the tube can be inserted initially in a ventromedial direction and continued directly into the oropharynx. The tube is inserted until the adhesive tape tab or indelible mark is reached (**Figure 3**).

To evaluate proper tube placement, 3 to 15 ml of sterile water or saline solution may be injected through the tube and the animal evaluated for coughing (**Figure 4**). A lateral radiograph may be taken of the neck to confirm the tube is placed in the caudal esophagus (i.e., over the larynx). After confirmation of position, the tube is secured with either sutures or glue. The first tape tab is secured to the skin just lateral to the external nares. A second tape tab is secured to the skin on the dorsal nasal midline, just rostral to the level of the eyes. An Elizabethan collar is used in most animals to prevent inadvertent removal of the tube (**Figure 5**).

Complications of nasoesophageal intubation include epistaxis, lack of tolerance of the procedure and inadvertent removal of the tube by the animal. Incidence of tube removal by the animal has been reported to be as high as 50% even with use of collars. Nasoesophageal tubes should not be used in vomiting patients or those with respiratory disease.

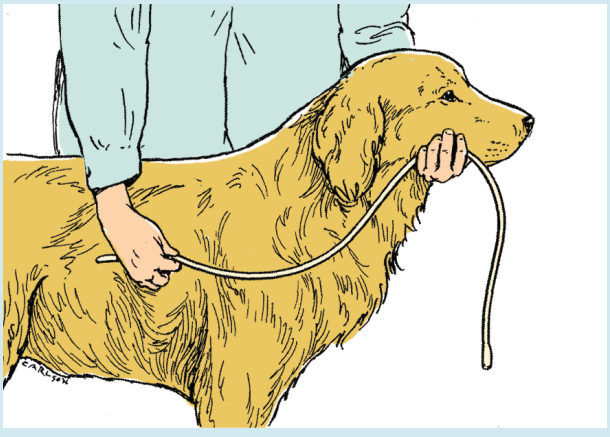

Figure 1. The length of tube to insert is determined by measuring from the nose to the last rib. Marking the tube at three-quarters of the distance between the last rib and the nose will place the end of the tube in the caudal esophagus. This location is marked with an indelible marker or a piece of adhesive tape. Tape can also serve as a suture tab to secure the tube.

Figure 2. The external nares are pushed dorsally and the proximal end of the tube is lifted to facilitate passage of the tube into the ventral nasal meatus.

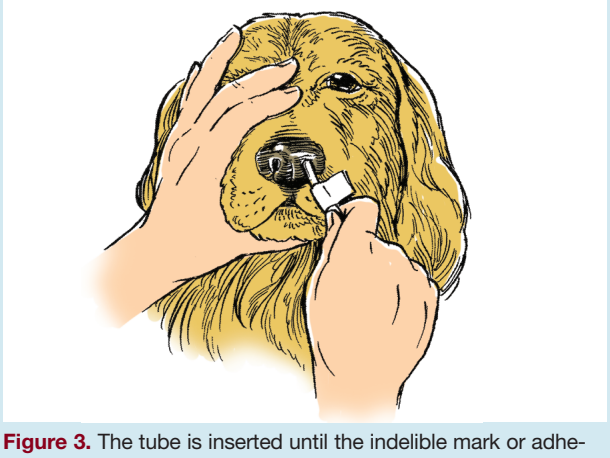

Figure 3. The tube is inserted until the indelible mark or adhesive tape tab is reached. Sutures or glue are used to secure the tape tab to the skin.

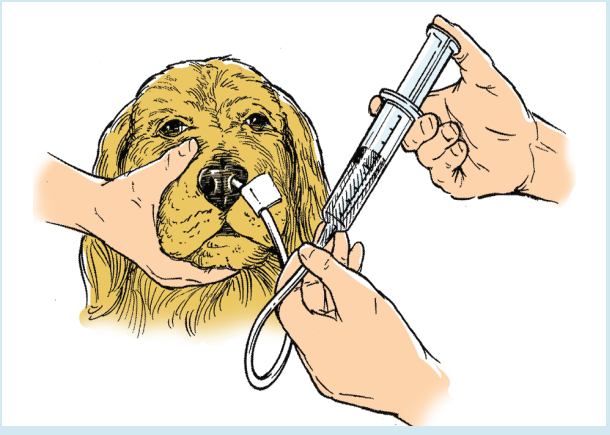

Figure 4. A test injection of sterile water or saline solution is made to ensure proper tube placement.

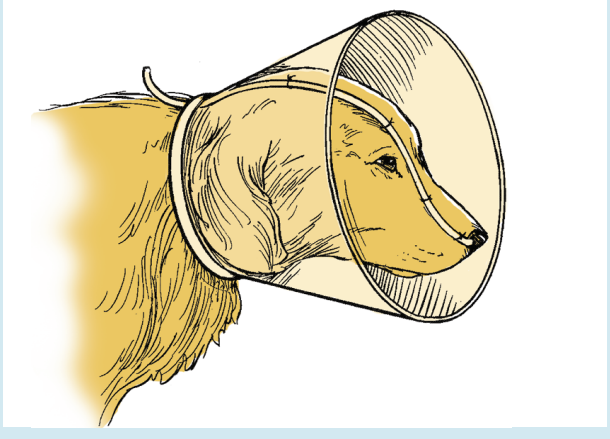

Figure 5. Securing the tube at several locations by suturing or gluing tape tabs to the skin and applying collars will help decrease inadvertent removal of the tube by the animal.

Box 25-6. Pharyngostomy Tube Placement.

In some instances, a pharyngostomy tube is used to bypass the nose and mouth of an animal requiring nutritional support (e.g., in cases of facial trauma) or when nasoesophageal tubes are not tolerated. Pharyngostomy tubes have been largely replaced by esophagostomy tubes or gastrostomy tubes placed percutaneously.

The patient is anesthetized, intubated and positioned in lateral recumbency. The area caudal to the mandible on either side is prepared for aseptic surgery. A 14- to 18-Fr. polyvinylchloride tube is premeasured as described in **Box 25-5, Figure 1**, except that the tube exit site will be caudal to the mandible.

With the mouth held open with a speculum, palpate the hyoid apparatus with one finger. The tube exit site must be carefully planned to avoid interfering with laryngeal opening and epiglottic movement. The tube should exit as far caudally and dorsally along the lateral pharyngeal wall as possible. The finger inside the mouth locates the hyoid apparatus and protrudes from the pharyngeal wall laterally at the selected exit site (**Figure 1**). Alternatively, forceps can be used to bulge the pharyngeal wall laterally. The finger locates the pulsating carotid artery, ensuring that it will be avoided, while providing a target for the tunneling forceps. A 1-cm skin incision is made over the bulging pharyngeal wall. Long, curved forceps are used to bluntly tunnel caudally through the tissues from outside to inside. Blunt dissection prevents injury to nearby nerves, carotid artery and jugular vein. Forceps are used to grasp one end of the feeding tube so it exits through the dissection site while the other end is advanced down the esophagus (**Figure 2**). The tube is then secured to the skin with tape and sutures.

Complications include airway obstruction, tube displacement, damage to cervical nerves and blood vessels and infection at the exit site. Placing the tube exit site caudal to the hyoid apparatus or use of very large diameter tubes is much more likely to result in airway obstruction or aspiration (**Figure 3**). The animal should be observed frequently for signs of respiratory embarrassment as it recovers from anesthesia. Frequent inspection and cleansing of the tube entrance/exit site help prevent skin infection. These tubes should not be used in vomiting patients or those with respiratory disease.

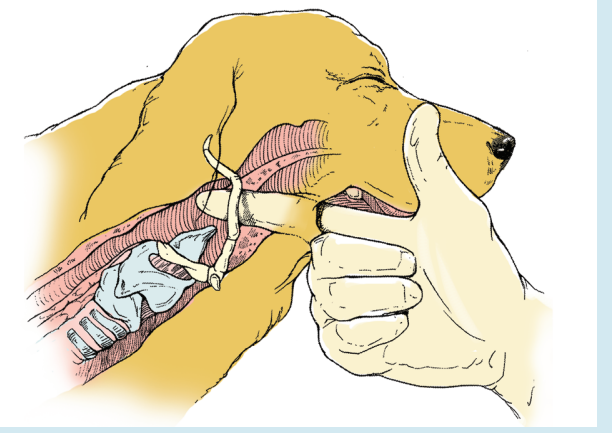

Figure 1. A finger is used to find the optimal exit site for the pharyngostomy tube. The tube should exit the pharyngeal wall as far caudally and dorsally as possible.

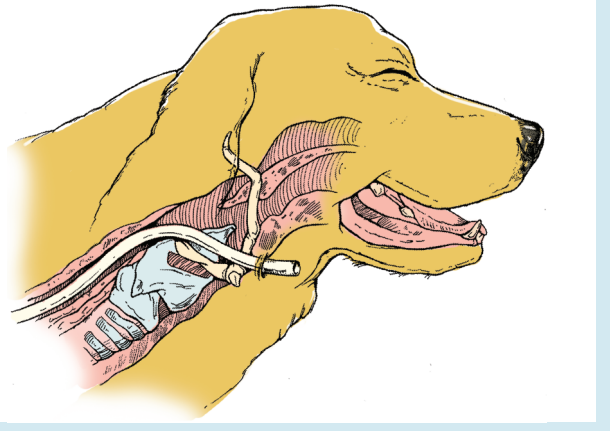

Figure 2. Proper placement of a pharyngostomy tube with the tube exiting dorsal and caudal to the larynx.

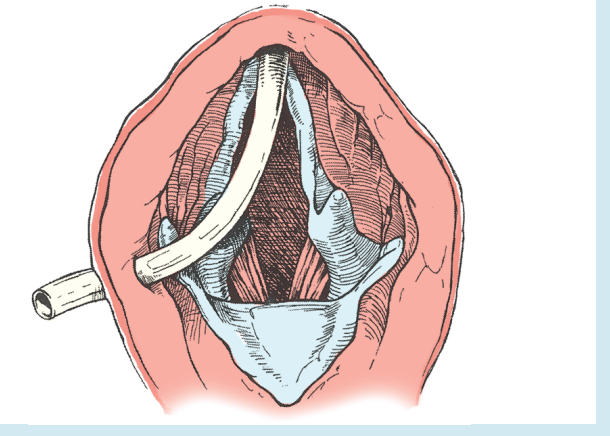

Figure 3. Inappropriate positioning of a pharyngostomy tube, as depicted here, causes the tube to course over the laryngeal opening and to interfere with movement of the epiglottis. This placement can lead to serious airway obstruction. The tube should exit the pharyngeal wall as far caudally and dorsally as possible.

Box 25-7. Esophagostomy Tube Placement.

Several techniques have been described for mid-cervical placement of esophagostomy tubes in dogs and cats. The animal receives light general anesthesia for esophagostomy tube placement. The entire lateral cervical region from the ventral midline to near the dorsal midline is clipped and aseptically prepared for surgery.

In one technique, appropriately sized, curved Kelly, Carmalt or similar forceps are inserted into the pharynx and then into the proximal cervical esophagus. The tip of the forceps is turned laterally and pressure is applied in an outward direction, thereby tenting up the cervical tissue so that the instrument tip can be seen and palpated externally. A small skin incision, just large enough to accommodate the feeding tube, is made over the tip of the forceps. In small dogs and cats, the tip of the forceps is forced bluntly through the esophagus. In larger dogs, a deeper incision is made to allow passage of the tip of the forceps through the esophagus. Tube sizes 12- to 19-Fr. are generally used. The tube is premeasured as described in **Box 25-5, Figure 1** so that the distal tip resides in the mid to caudal esophagus. The distal tip of the tube is grasped with forceps, pulled into the esophagus and out through the mouth, turned around and redirected into the esophagus. The tube is then secured with tape and sutures. A light circumferential bandage containing antibiotic-impregnated gauze is then placed at the exit site.

Another technique uses a percutaneous feeding tube applicator (ELD Gastrostomy Tube Applicator) (**Figure 1**).

Reported complications of tube esophagostomy for nutritional support include tube displacement due to vomiting or scratching by the animal and skin infection around the exit site.

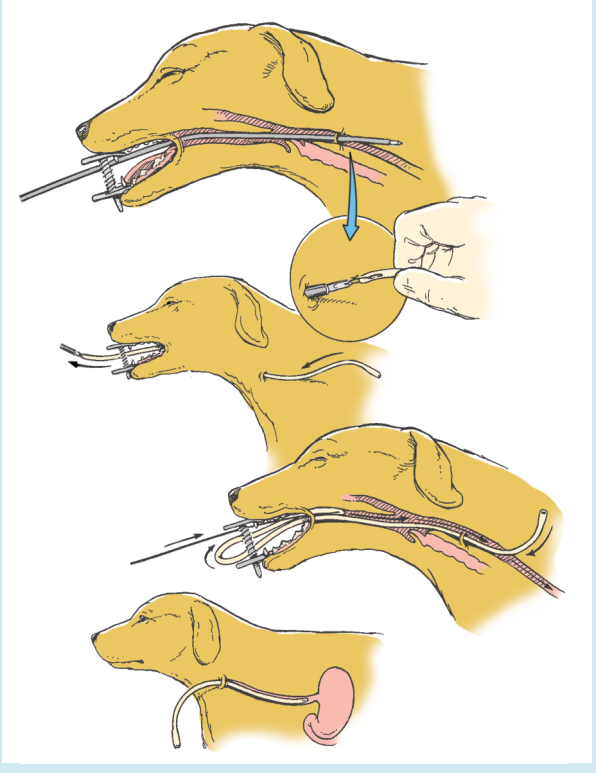

Figure 1. Insertion of a percutaneous feeding tube applicator into the mid-cervical esophagus. The distal tip is palpated and an incision is made through the skin and subcutaneous tissue over the tip of the applicator. The trocar is advanced through the esophageal wall and directed through the incision. The distal end of the feeding tube is secured to the eyelet of the trocar with suture material. The applicator and attached feeding tube are retracted into the esophagus and out the mouth. The feeding tube is redirected into the esophagus for final placement. A wire stylet can be inserted into the feeding tube if necessary to ease placement in the esophagus.

provided by J-tubes must be infused slowly and often in either very small quantities or preferably by a slow gravity drip or enteral pump with an hourly rate equal to RER/24 hours because the jejunum is volume sensitive.

Each bolus-fed meal must be followed by a water flush to clear the feeding tube of food residue. When the patient is volume sensitive, it is important to know the minimum volume required to effectively flush the tube. The patient's daily fluid requirement must also be met and additional water may be administered through the feeding tube to meet that requirement. Liquid oral medications may also be administered easily through feeding tubes. Plugged feeding tubes can be cleared by filling the tube with water or a nonalcoholic carbonated beverage and allowing time for the food plug to dissolve. End-port tubes are usually easier to maintain because food tends to become trapped in the blind end of side-port tubes. All tubes except orogastric and nasoesophageal tubes require standard every-other-day bandage care.

Assess and Select the Food

Selecting a food for hospitalized patients requires complete knowledge of the case, and often the food needs to be individually tailored because of each patient's unique circumstances. Refeeding patients in the early phase vs. refeeding in the later phases of food deprivation dictates the proportion of fat and carbohydrate in the refeeding formula. For example, the refeeding formula for a patient that has not eaten in seven days or more should contain predominantly fat as the energy fuel, as opposed to higher levels of carbohydrate (e.g., glucose). Pancreatitis patients, in which high-fat foods are contraindicated for dogs and presumably for cats, would be exceptions. In these cases, consider foods providing lower fat, low to moderate carbohydrate and increased protein calories. The food selection process should include a comparison of the key nutritional factor content of the food to the recommended levels. **Tables 25-8** and **25-9** list selected foods for assisted feeding and compares them to the recommended levels of key nutritional factors for dogs and cats, respectively.

Pre-existing conditions requiring specific nutritional modifications (e.g., renal insufficiency) or dietary modifications (e.g., adverse reactions to foods) must be understood and incorporated into selecting a food for the patient. For example, a cat diag-

Box 25-8. Surgical Gastrostomy Tube Placement.

A limited left flank celiotomy for gastrostomy tube placement provides an alternative when endoscopic or blind gastrostomy techniques are not performed. A gastrostomy tube may also be inserted when a celiotomy is performed for other reasons. General anesthesia is administered and the left flank is aseptically prepared for surgery. The prepared left paracostal area is draped and a 2- to 3-cm incision is made through the skin and subcutaneous tissue. The incision is made just caudal and parallel to the last rib, with its dorsal limit just below the ventral edge of the paravertebral epaxial musculature. The incision should be extended ventrally so that the intraperitoneal rather than the retroperitoneal space is accessed. The incision should be long enough to permit insertion of one or two fingers and a tissue forceps.

The greater curvature of the stomach is located and an Allis or Babcock tissue forceps is used to grasp and exteriorize the stomach through the incision. A stomach tube may be passed by an assistant and the stomach dilated with 10 to 15 ml of air/kg body weight if difficulty is encountered locating the stomach. Exteriorizing the stomach through a small flank incision can be difficult, especially in larger, deep-chested canine breeds. The left lateral aspect of the gastric body or the caudal aspect of the fundus is selected for the ostomy site. Two pursestring sutures are placed around the selected ostomy site (**Figure 1**). A stab incision is made through the ostomy site, the tube is inserted into the stomach and the pursestring sutures are tied snugly. Tube sizes 14 to 28 Fr. can be inserted.

The tube may exit the body wall through a separate stab wound or the original incision. The stomach is then fixed to the abdominal wall where the tube enters the peritoneum using a continuous suture pattern circling the gastrostomy tube placement (**Figure 2**). After the gastropexy sutures are placed, gentle traction is applied to the external end of the tube to ensure the stomach is adjacent to the abdominal wall (**Figure 3**). A rubber flange, which is slid down the tube to rest lightly against the skin, is sutured to the skin to secure the tube in place.

Potential risks with this procedure are the same as with any celiotomy and include wound infection, peritonitis and dehiscence. Pressure necrosis of the stomach may also occur if excessive tension is placed on the pursestring sutures. Wrapping the intraperitoneal tube with the omentum should contain leakage to a localized site. A layer of greater omentum can also be placed over the ostomy site before the stab incision is made into the stomach.

Percutaneous gastrostomy tube placement with gastropexy using a large-bore stiff plastic stomach tube has also been described. This technique is less invasive than the technique described here and may be more convenient for some veterinary practitioners.

Figure 1. Two full-thickness pursestring sutures are placed concentrically around the selected gastrostomy site to help invert the stomach around the tube. A stab incision is made in the center of the suture pattern for tube placement.

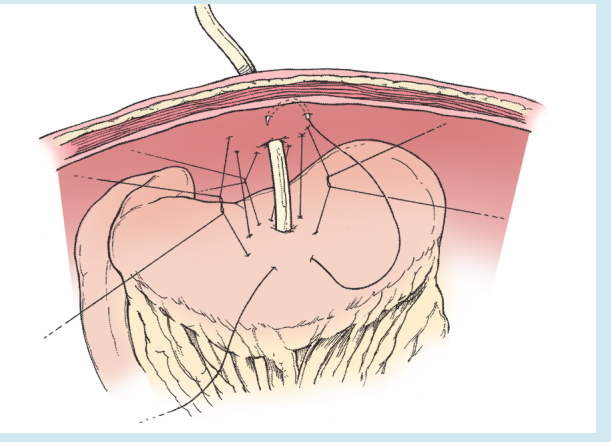

Figure 2. The stomach is sutured to the abdominal wall with four preplaced mattress sutures (or a simple continuous pattern). These sutures should include the strong abdominal fascia and the gastric submucosa. Tightening the loops brings the gastric serosa and omentum snugly in contact with the peritoneum.

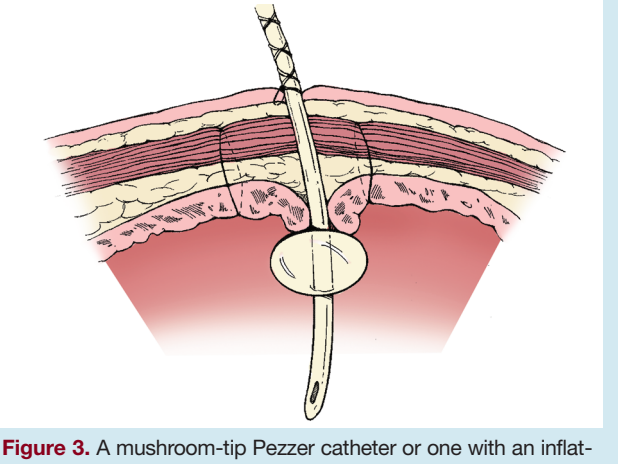

Figure 3. A mushroom-tip Pezzer catheter or one with an inflatable bulb is placed in the stomach. After the gastropexy sutures are placed, gentle traction is applied on the external end of the tube to ensure this area of the stomach is adjacent to the abdominal wall.

nosed with colitis that has a history of chronic renal insufficiency will require a feeding plan compatible with both diseases. Alternatively, the patient may be fed the food it was accustomed to eating before the injury or illness. The latter approach reduces the number of food changes that ultimately will need to be made.

Food selection will also depend on tube size and location within the GI tract, the availability and cost of products and the experience of the clinician. Commercial foods available for enteral use in veterinary patients can be divided into two major types: 1) liquid or modular products and 2) blended pet foods. Nasal and jejunostomy tubes usually have a small diameter (<8 Fr.), which requires use of liquid foods. Orogastric, pharyngostomy, esophagostomy and gastrostomy tubes have large diameters (>8 Fr.) and are suitable for blended pet foods.

Liquid Foods and Modules

Liquid foods consist of two basic types: 1) elemental or monomeric and 2) polymeric. Commercially available foods defined as "elemental" are not truly elemental, but contain nutrients in small hydrolyzed absorbable forms and are best described as monomeric. Protein requirements are usually met by free amino acids, dipeptides or tripeptides or larger hydrolyzed protein fractions. The fat source is often an oil of mixed (medium- and long-chain) fatty acids and the carbohydrate sources are mono-, di- and trisaccharides. Several liquid enteral feeding products on the human medical market are positioned as monomeric or hydrolyzed foods. They range between 270 to 550 mOsm/l and vary in protein and fat content based on the disease-specific formulation. These monomeric products are homogenized liquids that can be fed through any feeding tube including a J-tube. Monomeric foods are indicated in disease conditions such as inflammatory bowel disease, lymphangiectasia, refeeding parvoviral enteritis and pancreatitis cases and any other condition in which a patient's digestive capabilities are impaired. Most human liquid foods are adequate for adult dogs but are too low in protein for cats, puppies and adult dogs with increased protein losses (e.g., protein-losing enteropathies, drains). Most human liquid enteral products do not contain adequate concentrations of protein, taurine, arginine and arachidonic acid for long-term feeding of cats.

Polymeric products contain mixtures of more complex (less refined) nutrients. Protein is supplied in the form of large peptides (e.g., casein or whey). Carbohydrates are usually supplied as cornstarch or syrup, and fats are provided by medium-chain triglycerides (MCT) or vegetable oil. These foods require normal digestive function and are appropriate for most veterinary clinical situations, especially when a small tube (<8 Fr.) has been placed. In comparison to the rather vast selection of human polymeric diets, diets formulated for small animal veterinary patients are limited. Currently there are two liquid polymeric veterinary foods[g,h] that meet the current AAFCO nutrient allowances for adult dogs and cats. They are homogenized liquids providing between 1 to 1.25 kcal/ml, and one of these products[g] contains supplemental glutamine and carnitine.

Table 25-7. Example using enteral-assisted feeding guidelines.

Patient data needed	**Canine patient example**
1. Current body weight	12 kg
2. Calculate resting energy requirement (RER) as kcal/day	451 kcal/day
3. Expected daily fluid volume in ml/kg/day	60 ml/kg
4. Size (Fr.) and volume capacity of feeding tube	18-Fr., E-tube (10-ml volume)
Food information needed:	
Determine the caloric density of the food or food blend. Liquid foods have a set caloric density (kcal/ml) provided on the product information sheet. Moist foods need to be blended with a liquid (water or liquid food) to make a gruel (food blend) that can be delivered through a feeding tube or syringe.	
1. Determine gruel caloric density:	
Identify kcal per can of food	569 kcal/12.7 oz. can
Calculate ml/can (XX oz. in can x 30 ml/oz.); assumes 30 ml/weight/oz.	12.7 oz. x 30 = 383 ml
Determine ml of fluid needed to blend with canned food	100 ml warm water
Determine caloric density of fluid if not water*	–
Calculate caloric density of food blend. kcal/ml = (total kcal ÷ total ml)	569 ÷ (383 + 100) = 1.2 kcal/ml
2. Determine water provided in food or food blend:	
Calculate water in canned food (ml x % moisture); 75% moisture in canned food obtained from product information sheet	383 x 75% = 287 ml
Calculate water in liquid if not water**	–
Calculate % water in blend (ml total water ÷ total ml)	(287 + 100) ÷ 483 = 79%
3. Provide a feeding protocol:	
Method of food delivery (bolus feeding or constant rate infusion)	bolus
Beginning feeding rate (x % of RER)	25% RER
Daily caloric intake goal (kcal/day)***	113 kcal/day
Daily feeding rate	–
Calculate amount (kcal/day ÷ kcal/ml of food blend)	113 kcal ÷ 1.2 kcal/ml = 94 ml food blend/day
Determine meals/day (per 24 hr)	4
Determine feeding dosage (ml/meal/day)	94 ml ÷ 4 meals = 24 ml/meal; therefore, 24 ml q6hr
Water provided by food or food blend/day (ml)	94 ml x 79% = 74 ml water
Flush required after food delivery (ml)	4 x 10 ml flush = 40 ml water
Additional water needed to meet daily fluid volume (daily requirement = 60 ml/kg)	720 ml/day – (74 + 40) = 606 ml
Provide guidelines for residuals (see text)	–
Provide monitoring guidelines (see text)	–
Tube maintenance and removal guidelines (see text)	–

*If blending the canned food with a commercial liquid food, these foods provide a caloric density greater than 0. Determine the liquid food's caloric density and plug it into the top half of the equation. The caloric density can range between 0.8 to 1.9 kcal/ml depending on the commercial product.

**Every liquid food is part solids and part water, find this information about the product and calculate the absolute water contribution to the food blend, or assume the liquid food to be 100% water, if moisture content is 90% or greater.

***Increase this rate as tolerated by the patient or with a feeding goal to meet the patient's RER by Day 2 or 3.

Box 25-9. Percutaneous Endoscopic Gastrostomy Tubes.

Percutaneous endoscopic gastrostomy (PEG) tubes are inserted with the aid of general anesthesia. The patient is placed in right lateral recumbency and an area of the left flank extending 4 to 6 inches caudal to the last rib is surgically prepared. **Figures 1** to **7** describe tube placement technique in detail. Landmarks for feeding tube placement are usually 1 to 2 cm caudal to the last rib and one-third the distance from the ventral border of the epaxial musculature to the ventral midline. Commercial PEG catheter assembly kits, ranging in size from 16 to 28 Fr., are now available for small animal patients and provide cost-effective, convenient materials for PEG tube placement (**Figure 4**).

Following insertion, the tube is usually incorporated into a light bandage, with the free end brought to a convenient position for feeding. PEG tubes should be left in place for a minimum of five to seven days. Firm adhesions between the gastric serosa and the peritoneum have been reported to form within 36 to 48 hours of PEG tube placement in healthy dogs but do not reliably form in healthy cats. Adhesion formation may also be variable in undernourished animals.

The stomach should be empty when the tube is removed. Sedation or anesthesia is not generally required for tube extraction. Tubes are removed by exerting firm traction on the tube, while simultaneously applying counter-pressure around the exit site (**Figure 8**). An alternative method of removal, suitable for dogs weighing more than 10 kg, is to cut the catheter off flush with the skin, leaving the catheter tip to be passed in the feces. The resulting gastrocutaneous fistula usually heals rapidly.

Complications of PEG tube placement include vomiting, peristomal skin infection, cellulitis and pressure necrosis at the tube exit site.

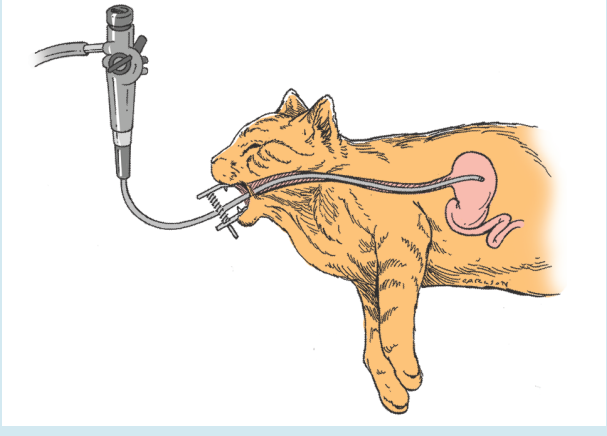

Figure 1. The animal is positioned in right lateral recumbency and an endoscope is introduced. The stomach is insufflated with air so that the gastric wall comes in contact with the body wall and the spleen is displaced caudally.

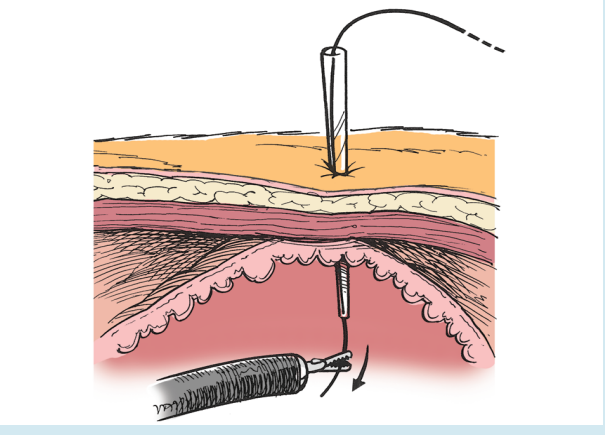

Figure 3. Nylon suture is advanced through the needle or catheter until it can be grasped with endoscopic retrieval forceps. The suture material is pulled out through the mouth as the endoscope is withdrawn.

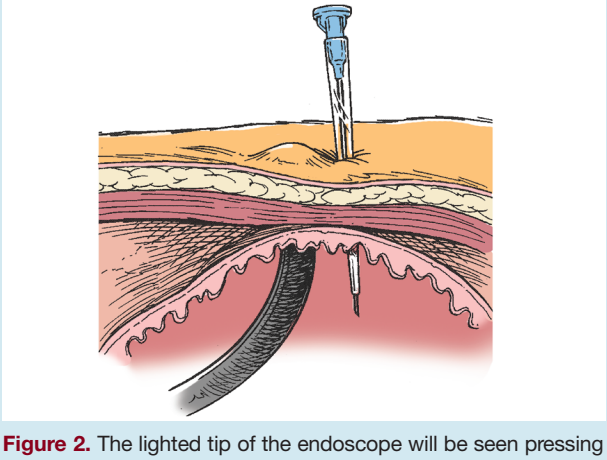

Figure 2. The lighted tip of the endoscope will be seen pressing outward against the abdominal wall. A large-bore needle or over-the-needle intravenous catheter is inserted into the stomach adjacent to the endoscope tip.

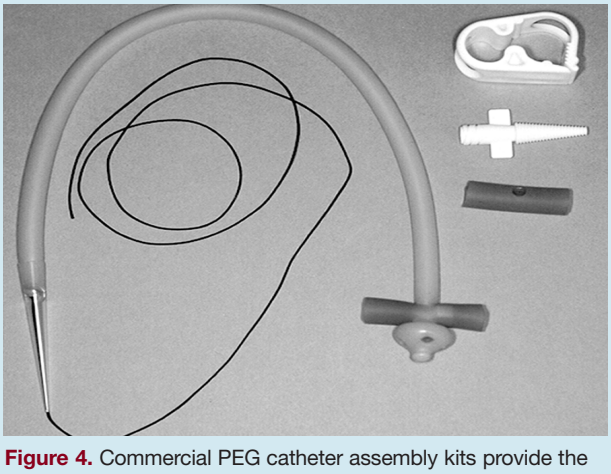

Figure 4. Commercial PEG catheter assembly kits provide the most convenient materials for PEG tube placement. The catheter guide is already secured to the free end of the feeding tube in commercial kits.

These products are usually accepted better than human liquid products containing MCT oil. In most critical care veterinary cases, these liquid foods are the best option currently available in North America when small-diameter nasogastric and jejunostomy feeding tubes have been placed, or when continuous drip feedings are necessary.

Liquid milk replacer products are generally inappropriate to feed to adult dogs and cats because they typically contain some lactose, have high (>300 mOsm/l) osmolarity and some are low in caloric density (<1.0 kcal/ml), which can result in RER constraints due to volume limitations.

Module products are concentrated powdered or liquid forms of nutrients and are primarily supplemental (**Table 25-10**). These products may be added to a liquid product to increase the concentration of a specific nutrient. Protein, fat and carbohydrate modules (e.g., casein powder, vegetable oil or corn syrup) are available. For example, a modular protein product may be added to a human liquid product for a patient with a high protein requirement. A vegetable oil or menhaden-fish oil can be added to increase omega-6 and/or omega-3 fatty acids. Soluble fiber (e.g., psyllium husk fiber or pectin) can be added to modular products, but requires greater than an 8-Fr. tube due to the increased viscosity of the food.

Blended Pet Foods

The term blended pet foods refers to commercial products that are nutritionally complete and balanced according to AAFCO allowances for dogs and cats. Moist veterinary therapeutic foods are available with nutrient profiles that assist in the management of various disease conditions in dogs and cats. Requirements for all other nutrients need not be calculated when the food contains non-energy nutrients properly balanced to the caloric density of the product. When the patient consumes the proper amount of a balanced food, all other

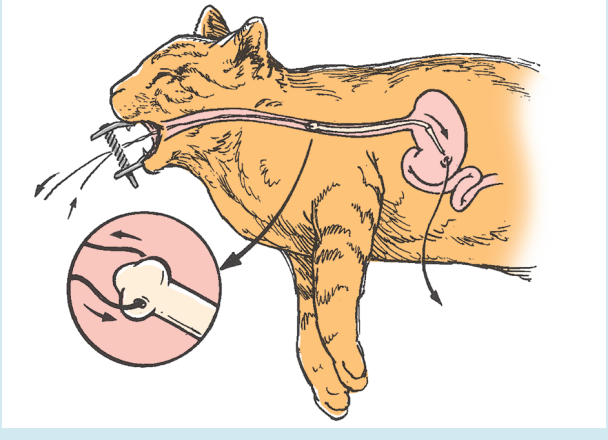

Figure 5. The lubricated catheter is drawn down the esophagus as the suture exiting the body wall is pulled. A second "safety" suture is placed through the openings in the mushroom-tip feeding tube (insert) and exits the mouth. This safety suture is used to retrieve the feeding tube from the stomach if problems occur during the placement procedure.

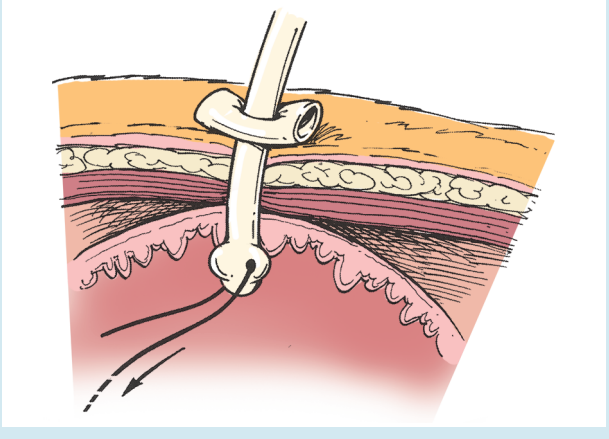

Figure 7. Gentle traction is used to bring the stomach and abdominal wall into loose contact. A rubber flange is fitted down the tube and a piece of tape attached to prevent tube slippage. The tube is not usually sutured or glued to the skin. The safety suture is removed via the mouth (arrow) after the feeding tube is secured.

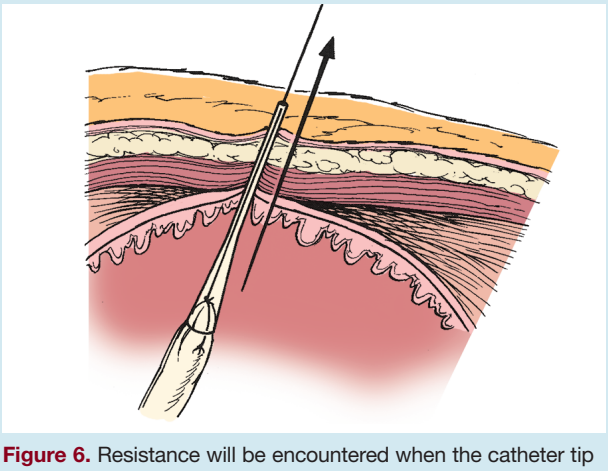

Figure 6. Resistance will be encountered when the catheter tip guide contacts the body wall. Steady traction and firm application of counter-pressure to the body wall will allow the guide tip to emerge through the skin (arrow). A small skin incision (2 to 3 mm) at the point of exit may help.

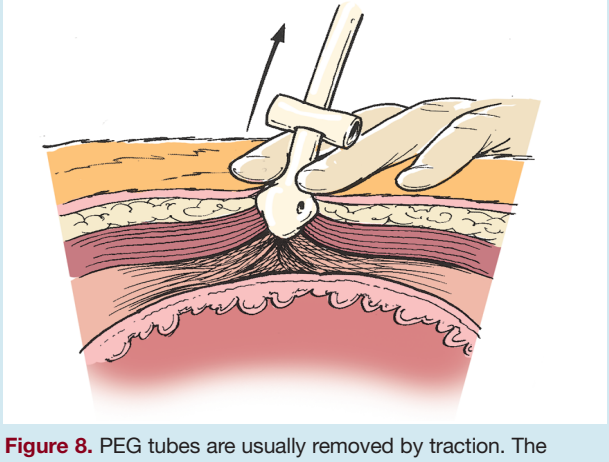

Figure 8. PEG tubes are usually removed by traction. The mushroom tip will usually collapse as it pulls through the abdominal wall. The resulting gastrocutaneous fistula usually heals rapidly.

Box 25-10. Percutaneous Nonendoscopic Gastrostomy Tubes.

Percutaneous gastrostomy techniques have been developed to allow convenient, cost-effective placement of feeding tubes without relying on availability of relatively expensive endoscopes. One nonendoscopic technique uses a commercial feeding tube applicator device (**Figure 1**) as described in **Box 25-7**. The other nonendoscopic technique uses a commercial gastrostomy tube placement device (**Figure 2**) pressed against the stomach wall. Use of either device allows suture material to be placed through the body wall into the stomach and retrieved through the mouth, and a gastrostomy tube to be inserted as described for PEG tube placement.

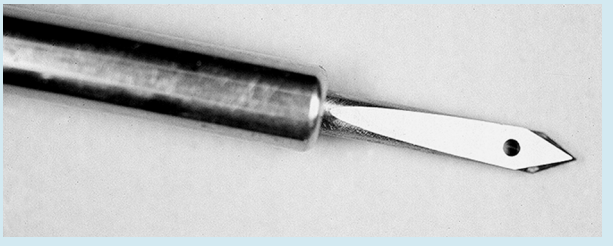

Figure 1. A commercial gastrostomy tube applicator can be used for percutaneous nonendoscopic gastrostomy tube placement in dogs and cats. The rigid outer tube encloses a trocar that can be pushed through the stomach and abdominal wall. A suture is placed through the small hole in the trocar tip, pulled into the stomach and then pulled antegrade out through the mouth. See **Box 25-7**, **Figure 1** for use of this device in esophagostomy tube placement.

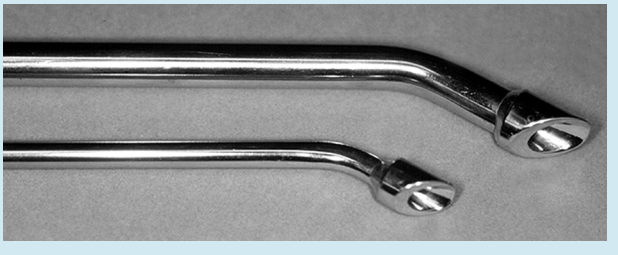

Figure 2. Commercial gastrostomy tube placement devices in various lengths and diameters can be used for percutaneous nonendoscopic gastrostomy tube placement in dogs and cats.

nutrient needs have been met, except when known losses of particular nutrients occur (e.g., protein and electrolytes).

Commercial products used as blended pet foods should provide complete and balanced nutrition for dogs or cats and should have passed AAFCO or equivalent feeding trials. These products are more readily available, better tolerated and less expensive than the human liquid foods. These pet food products contain essential amino acids and micronutrients properly balanced to the caloric density of the food. Fewer medical complications (e.g., diarrhea, hyperglycemia) are likely to result with blended pet foods. However, blended products are more likely to plug the feeding tube if the tube is not properly flushed after feeding and/or the blend is not of appropriate consistency to flow through the feeding tube. Patients may later consume the pet food orally, eliminating a food change when the patient's appetite returns and the tube has been removed. Blended pet foods, particularly the "recovery," "growth" or "performance" type foods, are appropriate for patients in catabolic states that are using fat and protein substrates from body stores.

These foods can be blended with a liquid to form a diet with a consistency that flows easily through a feeding tube. Some products have a blended texture, a high water content and very small particle size and may not need to be mixed with water or can easily be mixed with water depending on the size of the feeding tube. Most moist pet foods must be mechanically blenderized with water (or an appropriate liquid food) and strained to produce slurries or gruels that are administered through medium- and large-bore feeding tubes (i.e., 14 Fr. or larger). **Table 25-11** summarizes food blends commonly used for critical care tube feeding of feline and canine patients. Examples of patients that may benefit from these blended therapeutic formulas include those with renal or hepatic insufficiencies, diabetes mellitus, hyperlipidemia, pancreatitis, congestive heart disease and severe trauma. Appropriate moist foods are listed in the respective disease chapters.

Human Baby Foods

Some canine and feline patients voluntarily eat human baby foods packed in jars. In general, the meat and/or egg baby foods are high in protein (30 to 70% DM) and fat (20 to 60% DM), which, at the lower end of these ranges compares favorably with blended pet food products used for patients with increased protein and calorie needs. However, baby foods that provide upwards of 45% DM protein and 35% DM fat exceed the upper limits of veterinary critical care foods and would have little clinical value. Additionally, baby foods contain only one or two food types (protein, protein/grain) and do not contain a balanced mixture of other essential nutrients (amino acids, vitamins and minerals). For example, these products contain only 10% of the calcium required by dogs and cats and, therefore, have a large inverse calcium-phosphorus ratio. Some products contain onion powder, which can cause Heinz body formation in cats (Robertson et al, 1997). The human and veterinary liquid products have a better nutritional profile for feline and canine patients than do the human baby food products.

REASSESSMENT

Regular reassessment is a critical step in successful nutritional management of hospitalized patients, regardless of whether the enteral route, the parenteral route or both are used. Malnutrition in the form of insufficient nutrient intake to support tissue metabolism undermines medical and/or surgical management of a case. Malnutrition is far more common in veterinary patients than is currently recognized. Patients resting in a cage have been mistakenly assumed to require little or no nutrition when, in fact, the nutrient costs of tissue repair,

Table 25-8. Key nutritional factor content of selected commercial veterinary liquid foods, human liquid foods and moist veterinary foods used for enteral-assisted feeding of critically ill dogs compared to key nutritional factor recommended levels.*

Factors	Osmolarity (mOsm/l)	Energy density (kcal/ml)	CHO (g)	Protein (g)	Arginine (mg)	Glutamine (mg)	Fat (g)
Recommended levels	**250-400****	**1-2*****	**2-4**	**5-12**	**≥146**	**≥500**	**5-7.5**
Liquid veterinary products							
Abbott CliniCare Canine/Feline Liquid Diet	315	1.0	6.8	8.2	350	815	5.1
Abbott CliniCare RF Liquid Diet	235	1.0	5.9	6.3	350	615	6.8
PetAg Formula V Enteral Care HLP	312	1.2	4.2	8.5	413	na	4.8
PetAg Formula V Enteral Care MLP	256	1.1	5.8	7.5	392.6	na	5.7
Liquid human products							
Glucerna Shakes	355	1.0	9.6	4.2	na	na	5.4
Nestlé Impact Advanced Recovery	375	1.0	13.2	5.6	1,250	na	2.7
Nestlé Peptamen AF	390	1.2	8.9	6.3	na	na	4.6
Novartis Resource Diabetic	300	1.1	10.0	6.3	na	na	4.7
Moist veterinary foods†							
Hill's Prescription Diet a/d Canine/Feline	-	1.2	3.2	9.2	495	1,077	6.3
Hill's Prescription Diet n/d Canine	-	1.6	3.7	7.0	544	na	6.1
Iams Veterinary Formula Maximum-Calorie Canine & Feline	-	2.1	2.2	7.2	534	940	6.4
Purina Veterinary Diets Cardiovascular (CV) Feline Formula	-	1.4	4.7	8.8	469	1,169	5.5
Purina Veterinary Diets Dietetic Management (DM) Feline Formula	-	1.2	1.7	11.9	568	1,825	5.0
Royal Canin Veterinary Diet Feline and Canine Recovery RS	-	1.0	1.9	12.3	683	na	7.7

Key: CHO = digestible carbohydrate, na = information not available from the manufacturer.
*Liquid and moist veterinary foods in this table are formulated to meet minimum requirements of the Association of American Feed Control Officials; all nutrient values = units/100 kcal, unless otherwise stated; to convert kcal to kJ, multiply kcal by 4.184.
**250 is optimal.
***Energy density as fed basis.
†**Table 25-11** contains recipes for blending these foods for tube feeding.

immunocompetence and drug metabolism are significant. Therefore, reassessment of nutritional status is important whether the patient remains in the hospital or recovers at home.

Monitoring Parameters

Food intake or administration of nutritional support for hospitalized patients should be reviewed at least daily. Body weight should be recorded daily. Body condition should be noted; however, a patient's BCS is unlikely to change during the course of a hospital stay. Laboratory assessments specific for patients receiving nutritional support are generally not necessary beyond those tests already routinely performed for critically ill patients. The most common alterations that occur in laboratory parameters associated with nutrient administration are decreases in serum potassium, phosphate and magnesium levels, increases in serum glucose concentrations and hyperlipidemia. Even apparently stable patients might develop metabolic complications as a result of ongoing disease processes or from undiagnosed subclinical disease states. However, most patients show subjective improvement in attitude within 36 hours of refeeding.

Most parameters used to assess the nutritional status of patients will not change as a result of assisted feeding during the course of hospitalization. Laboratory parameters (e.g., albumin and total protein concentrations, RBC count and hemoglobin content) are unlikely to change in less than two weeks. Perhaps laboratory parameters that change during a hospital stay as a result of assisted feeding may be detected when acute-phase proteins with half-lives between two and 12 hours can be measured reliably in dogs and cats. The patient's body weight and condition and some laboratory parameters (albumin and total protein concentrations) should improve over the course of weeks (McAdams et al, 1996).

Residual Volume

Despite the numerous benefits of initiating enteral nutrition to hospitalized patients soon after admission, many critically ill patients are frequently intolerant of intragastric feeding due to GI motility dysfunction. The incidence of intolerance in human ICU settings is 43 to 63%, with the development of high gastric residual volumes accounting for 30 to 51% of cases (MacLaren et al, 2008). Although undocumented for veterinary patients, high gastric residual volume due to decreased GI motility is commonly encountered when refeeding. Adverse consequences for the patient include underfeeding needed calories, possible aspiration, increased mortality and prolonged hospital stay. Current therapeutic approaches for managing elevated gastric residual volumes involve administration of a prokinetic agent, alteration of the enteral feeding regimen (i.e., decreased volume, increased frequency, switch from intragastric to postpyloric site), or initiation of parenteral nutrition support. To maximize enteral support and limit complications associated with gastric residuals, measure the residual volume before each bolus tube feeding and intermittently during CRI feeding, then adjust the feeding schedule accordingly. Based on clinical experience, the authors suggest these

Table 25-9. Key nutritional factor content of selected commercial veterinary liquid foods, human liquid foods and moist veterinary foods used for enteral-assisted feeding of critically ill cats compared to key nutritional factor recommended levels.*

Factors	Osmolarity (mOsm/l)	Energy density (kcal/ml)***	CHO (g)	Protein (g)	Arginine (mg)	Glutamine (mg)	Fat (g)
Recommended levels	**250-400****	**1-2**	**2-4**	**7.5-12**	**≥250**	**≥500**	**5-7.5**
Liquid veterinary foods							
Abbott CliniCare Canine/Feline Liquid Diet	315	1.0	6.8	8.2	350	815	5.1
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Royal Canin Veterinary Diet Feline and Canine Recovery RS	-	1.0	1.9	12.3	683	na	7.7

Key: CHO = digestible carbohydrate, na = information not available from the manufacturer.
*Liquid and moist veterinary foods in this table are formulated to meet minimum requirements of the Association of American Feed Control Officials; all nutrient values = units/100 kcal, unless otherwise stated; to convert kcal to kJ, multiply kcal by 4.184.
**250 is optimal.
***Energy density as fed basis.
†**Table 25-11** contains recipes for blending these foods for tube feeding.

Table 25-10. Modules for augmenting foods.

Products	Key features
Arginine (various)	Available OTC as 500-mg capsules in most pharmacies and health food stores
Corn syrup (various)	Mostly maltose, 2.9 kcal/ml
Glutamine (various)	Available as powder from chemical catalogs and most pharmacies and health food stores; check label for concentration
Medium-chain triglyceride oil (Mead Johnson)	Fractionated coconut oil, 8.3 kcal/ml
Pectin (various)	Available OTC as a powder containing <1% crude protein and ~90% soluble fiber
ProMod (Ross Laboratories)	23.6 g protein/100 kcal, 18.2 g glutamine/100 g powder, 1.48 kcal/ml reconstituted
Psyllium fiber (FiberAll Regular, Rydelle Labs)	Available OTC as a powder containing 8% crude protein, 85% total dietary fiber, 72% soluble fiber
Psyllium fiber (Metamucil Regular, Searle)	Available OTC as a powder containing 17% crude protein, 53% total dietary fiber, 44% soluble fiber
Taurine (various)	Available OTC as 250-mg and 500-mg tablets in most pharmacies and health food stores

Key: OTC = over the counter; to convert kcal to kJ, multiply kcal by 4.184.

guidelines for monitoring residual fluid volumes:

1. Before a scheduled food delivery, attach an appropriate size syringe to the end of the feeding tube. Gently aspirate with the syringe. If more than 20 ml/kg body weight is aspirated, discard that fluid and/or place some portion of that fluid back into the tube and skip that scheduled tube feeding. If less than 20 ml/kg body weight is aspirated, proceed with the scheduled feeding.
2. If a feeding needs to be skipped due to high residual fluid volume, recheck residual volume just before the next scheduled feeding. If less than 20 ml/kg body weight is aspirated, proceed with food delivery at the predetermined volume or at a lesser volume. Slowly work back up to the daily RER feeding amount.
3. If a feeding needs to be skipped due to high residual fluid volume and the next recheck for residual volume yields more than 20 ml/kg body weight, consider diagnostics to evaluate and/or prokinetic agents to manage the GI dysmotility. Consider alternative approaches to nutritional support for the patient.
4. Check the residual fluid volume every 12 to 24 hours when using CRI.

Changing Foods

Sometimes, the patient only needs a specific therapeutic formula for a short time and then may be fed its regular food. Changing from a therapeutic formula to an over-the-counter brand may also be done according to the short schedule in Table 1-1. Should a problem such as vomiting, diarrhea or food refusal occur, the last successful food mixture should be

Table 25-11. Recipes for blending selected commercial moist veterinary therapeutic foods in **Tables 25-8** and **25-9** for use with feeding tubes.

Feeding tube size			20 Fr.		18 Fr.		16 Fr.		14 Fr.	
Moist veterinary foods	Can size (oz.)	No. of cans	Water added (ml)	Energy density (kcal/ml)*	Water added (ml)	Energy density (kcal/ml)*	Water added (ml)	Energy density (kcal/ml)*	Water added (ml)	Energy density (kcal/ml)*
Hill's Prescription Diet a/d Canine/Feline	5.5	2	30	1.00	40	0.97	45	0.96	50	0.95
Hill's Prescription Diet n/d Canine	12.7	1	95	1.20	100	1.18	110	1.16	120	1.14
Iams Veterinary Formula Maximum-Calorie Canine & Feline	6	2	30	1.74	35	1.72	40	1.70	45	1.68
Purina Veterinary Diets Feline CV	5.5	2	100	1.04	105	1.03	110	1.01	120	0.99
Purina Veterinary Diets Feline DM	5.5	2	55	1.01	60	1.00	70	0.97	75	0.96
Royal Canin Veterinary Diet Feline and Canine Recovery RS	6	2	30	0.88	32.5	0.88	35	0.87	37.5	0.87

*Predicted as fed energy density of blended mixture.

offered for several more days before proceeding with the food change. Most pets undergo food changes with few or no detectable GI disturbances.

SUMMARY

- The major consequences of malnutrition in all patients are decreased immunocompetence, decreased tissue synthesis and repair and altered drug metabolism.
- A nutritional assessment includes a patient history, a diet history, a physical examination with special attention given to certain risk factors, body condition scoring and laboratory tests.
- Patients with a history of nausea, vomiting and diarrhea are at increased risk for malnutrition because nutrient intake and/or usage has been suboptimal for some time before admission.
- Animals use body carbohydrate, fat and protein stores to maintain blood glucose concentrations throughout the course of food deprivation, trying to maintain vital functions for as long as possible. The proportion of each stored component used varies over the course of food deprivation.
- The adaptation from the fed to the fasting state is one in which fuel use by the patient shifts from primarily a mixture of fuels to one in which the primary fuels are glycerol and fatty acids (fat).
- An understanding of the metabolic changes that occur during simple starvation is essential to understanding the underlying metabolic alterations present during anorexia with concurrent illness.
- Major electrolyte and acid-base abnormalities and blood glucose levels should be corrected or near normal before instituting either enteral or parenteral nutritional support.
- A practical goal is to begin nutritional support within 24 hours of hospitalization for the injury or illness.
- Patients with a suspected or documented food intake less than their calculated daily RER for more than three days are candidates for assisted feeding.
- The optimal target feeding for hospitalized patients is their calculated RER, realizing their actual energy requirement is likely to change over the course of the disease process through the recovery period.
- Nutritional support by an enteral, parenteral or a combination method should initially deliver sufficient calories to meet the patient's RER at its current weight, adjusted for protein and body condition. To begin feeding patients at RER is a rational and safe estimate that decreases the probability of metabolic complications.

ACKNOWLEDGMENTS

The authors and editors acknowledge the contributions of Drs. P. Jane Armstrong and Deborah J. Davenport in the previous edition of Small Animal Clinical Nutrition.

ENDNOTES

a. Impact, Novartis, Minneapolis, MN, USA.
b. Sovereign Feeding Tube. Sherwood Medical, St. Louis, MO, USA.
c. Kangaroo Enteral Feeding Tube. Sherwood Medical, St. Louis, MO, USA. KeoFeed II Feeding Tube. IVAC Corp., San Diego, CA, USA.
d. Feeding Tube. Cook Veterinary Products, Bloomington, IN, USA.
e. Pezzar Model Catheter, C.R. Bard, Inc., Covington, GA, USA.
f. Gastrostomy Tube Introduction Set. Cook Veterinary Products, Bloomington, IN, USA.
g. CliniCare. Abbott Laboratories, North Chicago, IL, USA.
h. Formula V EnteralCare, PetAg, Hampshire,IL, USA.

REFERENCES

The references for **Chapter 25** can be found at www.markmorris.org.

CASE 25-1

Gastric Tube Feeding in a Cat

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Patient Assessment

A two-year-old castrated male Persian cat was examined for evaluation and treatment of suspected septic peritonitis secondary to dehiscence of an intestinal anastomosis. Eight days before, the cat had been diagnosed with an intestinal intussusception. A jejunal resection (6 cm) and anastomosis had been performed. After surgery, the cat remained depressed, weak and was intermittently febrile with rectal temperatures spiking at 41.6°C (107°F).

When examined, the cat was thin (body weight 3.5 kg, body condition score 2/5), febrile (41.2°C [106.2°F]), depressed and showed signs of circulatory shock. Ten ml of purulent fluid were recovered by abdominocentesis. Microscopically, the effusion contained 99% degenerative neutrophils. Bacteria were present in large numbers.

Assess the Food and Feeding Method

The owners reported that the cat was normally fed a commercial grocery brand dry food free choice and was a hearty eater before the intussusception occurred. Except for a small meal three days after surgery, the cat had not eaten for nine days.

Questions

1. What is an appropriate treatment plan for this patient?
2. What are the key nutritional factors to consider in this anorectic cat with sepsis?
3. What feeding techniques should be considered to support this patient?

Answers and Discussion

1. Septic shock should be managed very aggressively and management should precede surgical exploration of the abdomen. Intravenous fluid therapy helps maintain cardiac output and prevents further decline in cardiopulmonary function. Vasoactive drugs may also be needed to maintain cardiac output. Electrolyte imbalances and hypoglycemia are common in patients with peritonitis and should be corrected in addition to providing intravenous fluids. Standard shock therapy with corticosteroids and bicarbonate is usually indicated. To combat sepsis, antimicrobial therapy should be started while awaiting the results of specific culture and antimicrobial sensitivity testing from samples obtained by prior centesis of the peritoneal cavity. After the patient has been stabilized, exploratory surgery is indicated to drain and lavage the abdomen, find the cause of the sepsis (probably dehiscence of the previous anastomosis) and repair the defect. Aggressive nutritional support is also indicated in an underweight, septic patient recovering from major surgery. Nutritional support will help reverse the catabolic process associated with sepsis, improve the immune response and optimize healing.
2. Key nutritional factors in this patient include energy, carbohydrate, protein, arginine, glutamine and fat. Providing these nutrients in an energy-dense formula will aid in sparing lean body mass and maintain host defenses. Palatability is another key factor in anorectic patients; foods with high concentrations of protein, fat and water are usually palatable.
3. Intestinal function should be normal unless a large portion of the intestinal tract is removed during the second surgery. Therefore, assisted feeding using enteral techniques is recommended for this patient during the postoperative period. Nasoesophageal tube feeding is a short-term option (five to 10 days), does not require sedation or general anesthesia, takes less than 10 minutes to complete and is less expensive than placing other tubes. Nasoesophageal tubes could also be used if the patient is unable to tolerate anesthesia or if surgery had to be postponed. Enteral tube placement (i.e., esophagostomy, pharyngostomy or gastrostomy tubes) during surgery would be easy, convenient and allow enteral feeding to begin early in the recovery period. A gastrostomy tube would be large enough to handle a variety of commercial foods specifically formulated for cats. The daily energy requirement (DER) in the hospital should be equal to at least resting energy requirement (RER) at the patient's current weight. The amount of food provided daily should be divided into multiple small meals. Assisted feeding should be continued until the cat is eating at least 50% of DER voluntarily for two to three days.

Progress Notes

The cat was initially treated for septic shock. An exploratory celiotomy was performed after the cat's physiologic parameters stabilized. A small dehiscence at the anastomosis site and severe secondary generalized peritonitis were found. A partial omentectomy was performed, the affected portion of small intestine was resected and healthy bowel was anastomosed. A mushroom-tipped, 18-Fr. Pezzer gastrostomy tube was placed intraoperatively and the abdomen was copiously lavaged and closed routinely.

The cat's RER was calculated to be 180 kcal/day (753 kJ/day) at its current weight of 3.5 kg (RER = $70[3.5]^{0.75}$). Feeding was

begun via the gastrostomy tube six hours postsurgery. A commercial moist homogenized recovery formula (Prescription Diet a/d Canine/Feline[a]) was chosen because it is complete and balanced for cats and can be administered through a gastrostomy tube. The food was made into a gruel by blending two 5.5-oz. cans with 50 ml water. The total volume was approximately 300 ml of liquid gruel that contained approximately 300 kcal (1.26 MJ) metabolizable energy. The feeding protocol for the first 24 hours of hospitalization included six 30-ml feedings of the gruel (i.e., every four hours). The gruel was given through the gastrostomy tube over three to five minutes while the cat was monitored for signs of intolerance (i.e., nausea, discomfort and vomiting). Although no signs of intolerance were noted, the appropriate strategy to follow should they occur is to discontinue feeding and attempt to feed the patient again 60 minutes later. Ten ml of water were flushed through the tube at the end of each bolus feeding. The cat's maintenance fluid requirement was approximately 210 ml/day (60 ml/kg body weight/day). The gruel provided approximately 180 ml of fluid plus six 10-ml water flushes through the gastrostomy tube per day for a total of 240 ml of fluid per day, which adequately maintained hydration.

The feeding protocol was modified to five 35-ml feedings of the same gruel during Day 2 of hospitalization. On Day 3, the feeding protocol was modified to four 45-ml feedings of the same gruel (i.e., every six hours). No problems with intolerance occurred. Fresh food (dry recovery formula cat food) and water were offered and the cat's voluntary food intake was monitored. Gastrostomy tube feedings were continued for eight days, but were gradually reduced as the cat voluntarily ate more dry food. The gastrostomy tube was removed 14 days after surgery, the patient's previous maintenance food was reintroduced gradually over several days and the cat made an uneventful recovery.

Endnote

a. Hill's Pet Nutrition, Inc., Topeka, KS, USA.

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CASE 25-2

Jejunostomy-Tube Feeding in a Dog

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Patient Assessment

An 11-year-old neutered male cocker spaniel was presented with a six-month history of recurrent episodes of regurgitation upon eating solid food. The dog was able to drink and eat moist food blended with water, but ingestion of dry food or solid moist food consistently resulted in regurgitation. Three years earlier, the patient had been diagnosed with an esophageal stricture and secondary megaesophagus, which had been successfully managed with esophageal dilatation, medications (sucralfate[a], cimetidine[b], metoclopramide[c]), a sequence of dietary changes (see below) and elevated feeding for two and one-half years. At that time, a gastric mass 1 cm in diameter was detected. The owner elected to monitor the mass via endoscopy every three to four months rather than to have it resected.

On physical examination, the dog was alert with a normal temperature, pulse and respiratory rate. Results of serum biochemistry analysis, complete blood count and urinalysis were normal. Although mild right-heart enlargement was noted radiographically, the pulmonary parenchyma was normal and no esophageal abnormalities were detected. The dog weighed 8.6 kg and had a body condition score of 2/5 and had lost 3 kg over the last six months. An endoscopic examination revealed pyloric hypertrophy and a marked enlargement of the gastric mass. Two gastroesophageal masses were removed during an exploratory celiotomy. Biopsy specimens were also obtained from the pylorus. The histologic diagnosis of the gastric masses was leiomyoma; however, the pylorus was histologically normal. A 5-Fr. jejunostomy tube was placed intraoperatively.

Assess the Food and Feeding Method

After the initial esophageal dilatation procedure, chronic regurgitation in this patient was initially managed using a moist slurry made from 454 g of Prescription Diet i/d Canine[d] blenderized with 340-ml water and fed in an elevated position. The dog was transitioned from the slurry to moist food, then water-soaked kibbles and finally to small dry kibbles (Iams Mini-Chunks[e], 1 to 1.5 cups/day). Neither regurgitation nor vomiting was observed for 2.5 years after esophageal dilatation.

Jejunostomy-tube feeding was initiated 12 hours postoperatively using a polymeric canine liquid food (Canine CliniCare[f]), which supplied 1.0 kcal/ml (4.2 kJ/ml) with a nutrient profile of 27.2% protein, 30.8% fat, 33.4% carbohydrate and 4.8% ash on a dry matter basis. This food was delivered via an enteral pump system for a continuous rate infusion. In addition, fluid therapy was maintained through a peripheral venous catheter.

Questions

1. After the esophageal dilatation, chronic regurgitation was initially managed in this patient using a highly digestible moist slurry fed in an elevated position. How might the feeding schedule, form and nutrient composition of the dog's food have facilitated gastric emptying during this time?
2. A polymeric liquid food supplying 1 kcal/ml (4.2 kJ/ml) with an osmolality of 230 mOsm/kg was administered through the jejunostomy tube. Calculate the dog's resting energy requirement (RER) and maintenance fluid requirement. Write the feeding orders for the continuous rate infusion of a liquid enteral food and concurrent crystalloid intravenous fluid administration to meet the patient's fluid requirement and RER.
3. Potential complications of enteral feeding include vomiting, abdominal discomfort and diarrhea. How might the general characteristics, administration and infusion rate of polymeric foods have reduced these complications?

Answers and Discussion

1. In general, smaller meals have a faster rate of gastric emptying than larger meals. Increasing the moisture content of foods increases the rate of gastric emptying, suggesting that a moist food will leave the stomach faster than dry kibble. Increasing the fat content of the food slows gastric emptying. Therefore, feeding multiple small meals of a highly digestible, moderate-fat, low-fiber moist product facilitated gastric emptying when the dog was initially presented three years ago. An elevated feeding position is indicated in the dietary management of megaesophagus to allow gravitational forces to enhance passage of food into the stomach.
2. This patient's RER was 352 kcal/day (1,473 kJ/day) (RER = $70[BW_{kg}]^{0.75}$ or $[8.6]^{0.75}$ x 70). The caloric density of the liquid diet was 1 kcal/ml (4.184 kJ/ml). To meet the daily RER, the patient must be fed 352 ml of the food every 24 hours. On Day 1, 176 ml or 50% of RER were delivered at a continuous infusion rate of 7 ml/hour. This amount supplied approximately 170 ml of the patient's daily water requirement. The volume of liquid food was increased to supply 100% of the RER on Day 2 using an infusion rate of 14 ml/hour, which supplied 352 ml of the daily water requirement. Because the maintenance fluid requirement for this patient was 516 ml/day (516 ml = 8.6 kg x 60 ml/kg body weight/day), the infusion of crystalloid intravenous fluid was reduced to 340 ml/day and 180 ml/day on Days 1 and 2 of jejunostomy-tube feeding, respectively.
3. Food digestion begins in the oral cavity as the particle size of the meal is reduced through mastication and salivary enzyme secretions. Subsequently, gastric and pancreatic secretions further breakdown dietary protein and carbohydrate to dipeptides and tripeptides and monosaccharides and disaccharides, respectively. Bile salts, phospholipids and cholesterol from the gallbladder and liver solubilize dietary fat within the intestine. Water moves into the duodenum diluting the chyme and reducing the osmolarity from 1,200 to 1,500 mOsm/l to 300 to 350 mOsm/l. Peristalsis and segmentation in the duodenum deliver small volumes of an isosmolar, water-soluble chyme to the jejunum for further digestion and absorption. The isosmolar polymeric food administered to this patient was composed of small peptides, saccharides and emulsified long-chain triglycerides. Continuous infusion of small volumes of liquid food (14 ml/hour [0.23 ml/min.]) mimicked normal physiology of the jejunum, fostering nutrient absorption and lessening the likelihood of abdominal cramping and diarrhea.

Progress Notes

Jejunostomy-tube feeding was continued for four days postoperatively. On Day 2, the dog was offered, and drank, small amounts of water. On Day 3, one tablespoon of a moderate-fat, low-fiber moist food (Prescription Diet i/d Canine) was offered every four hours. No vomiting occurred. The jejunostomy-tube infusion rate was reduced by 50% on Day 4 as the dog ate increasing amounts of the moist food. Tube feeding was discontinued and the tube removed on Day 5 postoperatively. The dog was maintained on two-thirds of a 15-oz. can of Prescription Diet i/d Canine (supplying 362 kcal/day [1,515 kJ/day]; can size at the time the case was written), divided between four meals per day until it was discharged on Day 7. The owners were instructed to feed one can (544 kcal/day [2,276 kJ/day]) of food, divided equally between three daily meals to exceed the dog's daily energy requirement (DER) of 492 kcal/day (2,058 kJ/day) (DER = $1.4 \times 70[8.6]^{0.75}$). When the patient returned for suture removal 14 days later, the owners reported that regurgitation had not occurred and the patient had gained 0.8 kg. The owners were encouraged to continue feeding the moist food until the dog had returned to its ideal body condition.

Endnotes

a. Carafate. Marion Merrell Dow, Kansas City, MO, USA.
b. Tagamet. SmithKline Beecham, Philadelphia, PA, USA.
c. Reglan. A.H. Robins Co., Richmond, VA, USA.
d. Hill's Pet Nutrition, Inc., Topeka, KS, USA.
e. The Iams Co., Dayton, OH, USA.
f. Abbott Laboratories, North Chicago, IL, USA.

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CASE 25-3

Gastric Tube and Parenteral Feeding in a Cat

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Patient Assessment

A 13-year-old spayed female domestic longhaired cat was referred to the oncology service for radiation therapy of a nasal lymphoma. The owner traveled to the U.S. from Canada with the understanding that the cat would remain in the hospital for two weeks to receive daily radiation therapy. When admitted, the cat was thin (body weight 3.1 kg, body condition score 1/5) and depressed. The cat had a low albumin 2.6 g/dl (normal = 3.1 to 4.1), hemoglobin 9.4 g/dl (normal = 10.7 to 16.6) and hematocrit 27.2% (normal = 30.6 to 48.5).

Assess the Food and Feeding Method

The cat presented with a gastrostomy tube already in place. The owners reported that the cat had been receiving 30 ml of CliniCare[a] liquid food six times daily followed by 10-ml water flushes. The feeding protocol was changed to 30 ml of Prescription Diet a/d Canine/Feline[b] slurry six times daily and radiation therapy was initiated. The a/d slurry was made by mixing 30 ml of water with one can of a/d to make a 1 kcal/ml solution.

Questions

1. Was the G-tube feeding plan appropriate for this patient using the liquid food?
2. What are the key nutritional factors to consider in this anorectic cat with cancer?
3. What particular features of the a/d slurry were advantageous to the cat's progress?

Answers and Discussion

1. The cat's resting energy requirement (RER) was 163 kcal (682 kJ). The feeding protocol of 30 ml of CliniCare (1 kcal/ml) six times daily provided the cat with 180 kcal. The initial feeding protocol was appropriate.
2. Key nutritional factors in this patient include energy, carbohydrate, protein, arginine, glutamine and fat. The provision of food should help spare lean body mass while supporting repair of tissue damage due to the tumor and radiation therapy.
3. The Prescription Diet a/d product contained appropriate key nutritional factors and omega-3 fatty acids and antioxidants to facilitate tissue repair, inhibit tumor growth and reduce tissue damage due to the oxidizing effects of radiation therapy.

Progress Notes

A nutrition consult was requested on Day 10 of therapy because although the cat had been tolerating the G-tube feedings, it was losing weight. The cat now weighed 2.1 kg. A review of the medical record revealed that although six feedings a day had been ordered, on most days the patient only received four feedings due to daily anesthesia for radiation therapy. The cat was receiving

120 kcal per day through the G-tube. At 2.1 kg, the cat's RER = 122 kcal. Therefore, the patient was understandably losing weight due to insufficient daily caloric intake.

Further Question

How should this caloric deficiency be corrected while not interfering with the anesthetic and radiation procedures?

Answer and Discussion

The daily caloric deficiency of 50 kcal was corrected by administering 20% lipid solution intravenously over the 18 hours the cat was not undergoing therapy. The dose of lipids was initiated to ensure the cat received a total of 180 kcal per day from the combined enteral and parenteral feedings until discharge.

Endnotes

a. Abbott Laboratories, North Chicago, IL, USA.
b. Hill's Pet Nutrition, Inc., Topeka, KS, USA.