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NUTRITIONAL SUPPORT OF THE SICK ADULT HORSE

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Among hospitalized human patients, malnutrition has been associated with increased infectious morbidity, prolonged hospital stay, and increased mortality. Conversely, several studies of critically ill human patients have shown that nutritional support can attenuate body weight loss, reduce complication rates in surgical patients, and shorten the period of hospitalization. Recognition of the beneficial effects of nutritional intervention in human patients has spurred interest in veterinary clinical nutrition. Over the past 10 to 15 years, the nutritional management of hospitalized animals has received increasing attention (Donoghue, 1992).

Clinical experience suggests that nutritional intervention can improve clinical outcomes in diseased horses, particularly neonates. However, it should be noted that there have been no controlled studies of the relationship between nutritional support and clinically important endpoints (e.g. surgical complication rate, duration of hospitalization, mortality). Furthermore, there are few data on the effects of specific disease conditions on the nutritional requirements of horses. As a result, recommendations for the nutritional management of sick horses have largely been extrapolated from data in other species.

This paper considers: (1) the metabolic effects of starvation and disease; (2) clinical assessment of nutritional status and the need for nutritional intervention; (3) dietary management of certain disease conditions; and (4) methods for delivery of nutritional support to anorectic or hypophagic horses. Several other excellent reviews of equine clinical nutrition are available (Ralston, 1990; Burkholder and Thatcher, 1992; Naylor, 1992; Rooney, 1998).

Metabolic Effects of Starvation and Disease

To appreciate the importance of nutritional support in disease states, it is useful to distinguish the metabolic effects of fasting (or starvation) and severe illness. Fasting and starvation imply a lack of nutrient intake. To compensate, the body attempts to reduce energy expenditure and directs substrate stores to essential functions. The initial energy sources are stored liver glycogen and, to a lesser extent, glucose derived from amino acids. However, liver glycogen stores are quickly depleted, usually within 24 to 36 hours of the onset of a fast, necessitating an increase in gluconeogenesis (glucose synthesis in the liver) to meet the obligate needs of the central nervous system and red blood cells. The carbon skeletons from glycerol, lactate, and some amino acids (particularly alanine) are used for gluconeogenesis. Use of amino acids for glucose synthesis mandates the breakdown of body proteins (proteolysis), particularly those in muscle. Simultaneously, there is increased mobilization of fatty acids from fat stores in adipose tissue and increased utilization of fatty acids for energy (in all tissues but the central nervous system and red blood cells). Studies in man have demonstrated

that fat supplies approximately 80 to 85% of the body's energy during prolonged fasting, with the remainder derived from metabolism of protein, either direct oxidation of carbon skeletons via the Krebs cycle or utilization of glucose synthesized from amino acids.

The principal mechanism of adjustment to prolonged fasting or starvation is a change in hormone balance. In particular, there is a sharp decrease in insulin production. As well, the muscle and adipocytes become somewhat resistant to the action of insulin (i.e. whatever insulin available becomes less effective in promoting cellular uptake of nutrients for protein synthesis and lipogenesis). Decreased insulin activity, coupled with increased synthesis of counterregulatory hormones such as glucagon and cortisol, promotes fatty acid mobilization and the catabolism of muscle protein. An additional hormonal change facilitating adjustment to starvation is a decrease in synthesis of the thyroid hormone triiodothyronine (T_3), thus resulting in a lowered metabolic rate and daily energy requirement.

In several species, a further important adaptation to starvation is increased synthesis and utilization of ketones. Ketogenesis in the liver is favored under conditions of increased fat mobilization and a low insulin-to-glucagon ratio. Ketones are an important energy source during starvation and other negative energy states. In particular, the brain and other nervous tissues adapt to use of ketones and, as a result, become less dependent on glucose for energy. This reduction in glucose demand allows for a decrease in the rate of proteolysis, thus preserving lean body mass. Although plasma ketone concentrations increase during feed deprivation in horses (Rose and Sampson, 1982), the increase is small in comparison to man, suggesting that ketones are less important as an energy source in this species during prolonged fasting.

The body's response to severe stress, such as sepsis, major surgery and trauma, is much different than its response to starvation. Whereas adaptation to starvation is associated with a decrease in metabolic rate, severe stress induces a hypermetabolic state that results in rapid breakdown of the body's reserves of carbohydrate, protein and fat. The term "septic autocannabalism" has been coined to describe the metabolic response and wasting that accompanies severe sepsis in humans (Michie, 1996). Hyperglycemia with insulin resistance, hyperlipidemia, profound negative nitrogen balance, and diversion of protein (particularly glutamine) from skeletal muscle to the splanchnic tissues are prominent features. Unlike starvation, protein catabolism remains unchecked, resulting in severe wasting of lean body mass. These responses are mediated in part by marked increases in the counterregulatory or "stress" hormones (glucocorticoids, epinephrine and glucagon) and several of the inflammatory cytokines (low molecular weight peptides that evoke a number of varied reactions in the body), including interleukin-1 (IL-1) and tumor necrosis factor (TNF). This severely catabolic state quickly results in malnutrition that, in turn, impairs host defense, delays tissue repair, and increases risk of mortality.

The metabolic effects of fasting and, to a much more limited extent, illness have been studied in horses (Morris et al., 1972; Naylor et al., 1980; Sticker et al., 1995a and 1995b). As in other species, fasting results in linear increases in

the plasma concentrations of glycerol and nonesterified fatty acids (NEFA), reaching a plateau about three days after the start of food deprivation. These changes are consistent with an increase in lipolysis and suggest greater use of fat during fasting. Some of the mobilized fat is re-esterified in the liver and transported to the peripheral tissues as very low-density lipoproteins (VLDL). As a result, serum triglyceride concentrations are also increased in fasting horses. Pregnancy, lactation and obesity accentuate the effect of fasting on plasma triglyceride concentrations. Furthermore, the increase in plasma triglycerides is typically more pronounced in sick, hypophagic horses (e.g. pneumonia, diarrhea, renal failure), perhaps reflecting the increased metabolic demands associated with the primary illness.

The term hyperlipemia has been used to describe a disease syndrome in ponies, miniature horses and, more rarely, horses that is characterized by a marked increase in plasma triglycerides (often >1000 mg/dl), cloudy serum or plasma, and fatty infiltration of the liver and kidneys. Affected animals usually have a primary illness that results in reduced feed intake (hypophagia), but the clinical manifestations of hyperlipemia and fatty liver often overshadow the primary problem. Increased metabolic demands associated with the primary illness tend to exacerbate the increase in fat mobilization associated with hypophagia. Furthermore, the increased metabolic demands of pregnancy or lactation intensify lipolysis. In obese ponies and miniature horses, insulin resistance is another underlying factor. Under conditions of rapid and sustained lipolysis, the liver's capacity for lipoprotein (VLDL) synthesis is overwhelmed and fat is deposited in the liver. Affected horses and ponies require aggressive nutritional support and, even then, the prognosis for recovery is guarded to poor.

Effects of Malnutrition on Host Defenses

Ultimately, all body systems are adversely affected by undernutrition. However, organs and cells with high rates of metabolism are more rapidly impaired by nutrient imbalances. The intestinal tract is particularly susceptible to the effects of anorexia. Enterocytes (the cells that line the intestinal mucosa) have an extremely high metabolic rate and a very short lifespan, with an average turnover rate of three days. Enterocytes participate in the digestion and absorption of nutrients and also provide a physical barrier that prevents entry of bacteria into circulation. Because many of the nutrients required for synthesis of new enterocytes are taken directly from the intestinal lumen, even short periods of food deprivation result in mucosal atrophy and impaired digestion and absorption. Perhaps more importantly, the mucosal barrier is compromised and there is increased risk of sepsis as a result of bacterial translocation.

Cells of the immune system also have high metabolic demands. In several species, including the horse (Naylor and Kenyon, 1981), 3 to 5 days of complete feed deprivation severely compromises cellular and nonspecific immune function, thus rendering the animal more susceptible to infection. This decrease in immune function, together with breakdown of the intestinal mucosa barrier, helps to explain the decreased survival rate in malnourished critically ill humans.

What are the Nutrient Requirements of Sick Horses?

The short answer to this question is "we don't know." As previously indicated, data from human and animal studies have demonstrated that metabolic rate is increased with certain conditions. For example, severe trauma increases energy expenditure (metabolic rate) by a factor of 1.3 to 1.4. Similarly, the metabolic rate of patients with sepsis or a major burn can be up to 1.4- to 1.7-fold higher when compared to resting healthy humans. Indirect calorimetry has been used to measure energy balance in a group of healthy neonatal foals and a small number of foals that were either premature or diagnosed with neonatal maladjustment syndrome (NMS) (Ousey, 1992; Ousey et al., 1996). Interestingly, the metabolic rate of these compromised neonates was reduced by approximately 50% compared to healthy, age-matched counterparts. As the premature and NMS foals were recumbent during the measurement periods, the most likely cause of the low energy expenditure was inactivity. Despite their low rates of metabolism, these foals were in negative energy balance because of very low milk intake (either by bottle or nasogastric tube). Energy deficits are probably greater in foals with more severe illnesses such as septicemia, diarrhea, and pneumonia as a result of increased metabolic demands (hypermetabolic states).

Pagan and Hintz (1986) reported that the resting energy expenditure (REE) of horses in metabolism stalls could be estimated from the formula: REE = 21 kcal (BWkg) + 975 kcal. Thus, for a 500-kg horse, REE would be approximately 11.5 Mcal/day, 30% lower than the requirement for maintenance under field conditions (16.4 Mcal/day for a 500-kg horse). Although data on the energy expenditure of injured or sick adult horses are not available, it has been proposed that the multipliers used in human medicine be applied to the horse. Therefore, a stall rested 500-kg horse that has generalized infection or is recovering from major surgery would have energy requirements of 16 to 20 Mcal/day (1.5 to 1.8 x 11.5 Mcal/day). Given the large increase in protein catabolism during severe illness or injury, it follows that protein requirements are also increased in sick patients. Rooney (1998) has suggested that 5 g of protein be provided per 100 kcal, i.e. 800 g of crude protein for a diet containing 16 Mcal digestible energy. This represents about a 25% increase over the NRC (1989) maintenance protein requirement.

It is probable that the requirements for other nutrients (e.g. micro- and macrominerals) are also altered during illness. However, in the absence of data, feeding levels should be based on the horse's current body weight and the recommended maintenance requirements. It should be emphasized that for horses that are completely anorexic and in need of "involuntary" tube feeding, it is often difficult to meet maintenance requirements. Nonetheless, delivery of even 60 to 70% of maintenance requirements is likely to be beneficial in sick horses. The primary goal is to limit catabolism and further loss of body weight.

In human medicine, there is currently intense clinical interest in the therapeutic role of specific nutrients, including arginine, glutamine, omega-3 fatty acids (from fish oil), and ribonucleic acid (a nucleotide). As each of these nutrients is important in the immune response, it is proposed that administration of these

nutrients to critically ill patients will result in up-regulation of immune function and reductions in the morbidity and mortality associated with infection. A recent meta-analysis that addressed whether enteral nutrition with these "immuno-enhancing" nutrients benefits the critically ill demonstrated significant reductions in infection rate and length of hospital stay in patients receiving enteral supplements containing arginine with or without glutamine, nucleotides, and omega-3 fatty acids (Beale et al., 1999).

Of these nutrients, glutamine has been the subject of most clinical investigation. Glutamine is the most abundant amino acid in both plasma and the free intracellular amino acid pool in skeletal muscle, and is important in several key metabolic processes of immune cells and enterocytes. Because most tissues have the ability to synthesize glutamine, it is defined as a nonessential amino acid. However, marked decreases in plasma and skeletal muscle glutamine concentrations have been reported in a variety of catabolic states (Vinnars et al., 1975; Jackson et al., 1999). In horses, as in man, plasma glutamine concentrations decrease following viral infection (Routledge et al., 1999), a finding consistent with glutamine's proposed role in immune cell metabolism. The decrease in plasma glutamine concentrations during illness is temporally related to an increase in splanchnic amino acid uptake, perhaps reflecting increased intestinal demand for glutamine. Indeed, the small intestine is the principal organ of glutamine consumption (Nappert et al., 1997). These findings have led to the suggestion that glutamine may behave as a "conditionally essential" amino acid during severe illness, i.e. endogenous supply fails to meet increased demands (Jackson et al., 2000).

Several investigations in animals have indicated that oral glutamine supplementation improves growth and repair of the small intestinal mucosa and helps maintain intestinal immune function (Nappert et al., 1997). Glutamine supplementation also increases renal arginine production, another amino acid important for the body's response to injury (Welbourne, 1995). Human studies have demonstrated that glutamine supplementation (oral or intravenous) enhances in vitro measures of immune function, for example, bacterial killing function of neutrophils (Saito et al., 1999). Furthermore, in randomized clinical trials, patients receiving glutamine had reduced incidence of severe infection, decreased length of hospitalization (Jones et al., 1999), and a significant improvement in six-month survival (Griffiths, 1997). Taken together, these findings indicate that glutamine supplementation is beneficial in sick patients. Whether or not glutamine is similarly effective in the treatment of equine disease conditions is not known and requires study.

Selection of Patients Requiring Nutritional Support

Assessment of body condition and consideration of clinical history are important in identifying horses in need of nutritional support (Donoghue, 1992). Well-conditioned horses that are not pregnant (last trimester) or lactating can likely withstand a short period (2 to 4 days) of partial or complete anorexia without adverse affects. On the other hand, regardless of the duration of illness, thin

horses (condition scores of 1 to 3) or those that have sustained a substantial loss of condition (10% or more of body weight) are candidates for immediate nutritional support. Conditions of adult horses associated with rapid weight loss include sepsis/endotoxemia, deep-seated bacterial infections (e.g. pulmonary abscess, pleuropneumonia, abdominal abscesses), diarrhea, severe trauma, surgery (e.g. colic surgery involving bowel resection), and intestinal disorders characterized by protein loss and nutrient malabsorption. Over-conditioned (scores of 7 to 9) horses, ponies, and miniature horses, when sick, can be poorly tolerant of even short periods of anorexia; in these animals, plasma or serum should be visually appraised and triglyceride concentrations measured. Hypertriglyceridemia (>500 mg/dl) and/or recognition of cloudy, opaque plasma or serum suggest hyperlipemia and risk of development of liver dysfunction from hepatic lipidosis. Neonates are also poorly tolerant of short periods of undernutrition. Particularly during the first week of life, neonates have negligible energy stores, and negative energy balance can quickly result in hypoglycemia, generalized weakness and death.

For many disease conditions, most horses maintain a reasonably good appetite. However, there are some circumstances in which some change in diet is indicated. These include inflammatory airway disease, chronic obstructive pulmonary disease (COPD), laminitis, renal failure, hepatic disease, hyperadrenocorticism (Cushing's syndrome), chronic diarrhea, and small intestinal malabsorption syndromes. Horses that have had portions of the small or large intestine removed also may require dietary adjustments. For athletic horses that have sustained minor injuries or are recovering from elective orthopedic surgery or bacterial or viral infections, a major consideration is a reduction in energy intake. During convalescence, and until resumption of training, most of these horses can be maintained on a diet composed primarily of hay (fed at 1.5 to 2.0% of body weight per day), a vitamin/mineral supplement, and loose salt (or access to a salt block). To facilitate the return to full grain feeding upon return to training, it may be desirable to continue feeding small amounts of grain (1 to 2 kg per day).

Nutritional Management of Selected Conditions

Respiratory diseases:

In horses with inflammatory airway disease and COPD, disease exacerbations are related to exposure to airborne allergens (dusts, molds, fungi) and toxins (endotoxin). Changes in feeding and housing management are essential for long-term control of these conditions. Ideally, affected horses are housed outside or in well-ventilated barns with ample daily turnout. If housed indoors, bedding must be dust-free, e.g. shredded paper, wood chips. Hay is a primary source of allergens, including molds and fungi. The hay should either be removed from the diet or only fed after thorough soaking (completely immersed in a tub of water for a minimum of 5 minutes). Hay cubes are an alternate fiber source, but these must also be soaked in water prior to feeding.

Gastrointestinal disorders:
Gastrointestinal disorders can be categorized into those affecting the small intestine (protein-losing enteropathies/malabsorption syndromes; "short-bowel syndrome" following surgical resection of intestine) and those involving the large intestine (colitis/diarrhea; colonic or cecal impaction; colon resection). With small intestinal diseases, the primary goal is to optimize large bowel digestive function. This can be achieved by feeding highly digestible fiber sources such as leafy alfalfa, beet pulp or soybean hulls, with a reduction in grain feeding. Offering small grain meals (i.e. <1 kg) may minimize the risk of passage of undigested starch into the hindgut. Ponies fed a complete pelleted feed were able to maintain body condition following resection of less than 50% of the small intestine (Tate et al., 1983). However, ponies fed the same diet after a more extensive resection (60 to 80% of the small intestine) suffered rapid and substantial weight loss. Clinical experience has also indicated difficulty in maintaining body condition of horses following resection of 50% or more of the small intestine (and horses with malabsorption syndromes). However, some horses can be maintained on a diet of highly fermentable fiber (beet pulp and alfalfa) and fat (rice bran and corn oil).

Diarrhea in the adult horse is usually due to large intestinal dysfunction (e.g. colitis secondary to *Salmonella* spp. or clostridial infection). In the acute phase, many affected horses are hypophagic and in need of enteral nutritional support (see below). Small intestinal digestive and absorptive function is probably well-maintained. Therefore, low residue diets with highly digestible sources of carbohydrate, protein and fat should be fed. As appetite improves, the diet should initially consist of hay and other sources of fermentable fiber. Yogurt cultures or commercially available bacterial preparations are often administered to aid reestablishment of normal microbial flora. There should be a gradual increase in grain feeding over a 1 to 2 week period.

Laminitis:
A common cause of laminitis is overconsumption of starch (grains and lush pasture). In this circumstance, a large amount of undigested substrate passes into the hindgut and undergoes bacterial fermentation. Therefore, a primary goal in the prevention of recurrences is elimination of grain from the diet. These animals should be fed hay, a vitamin-mineral supplement and, if needed, a fat supplement such as rice bran or corn oil. However, in many ponies and some horses, chronic active laminitis is associated with obesity and gradual weight loss is necessary. Older horses with hyperadrenocorticism (Cushing's syndrome) are also prone to laminitis. In addition, persistent hyperglycemia and insulin resistance are common features. Accordingly, nutritional management of affected horses should also emphasize restriction of grain intake.

Hepatic disease:
With severe liver dysfunction, hypoglycemia may develop because of depressed gluconeogenesis. Therefore, the diet should contain highly digestible starches to decrease reliance on hepatic gluconeogenesis for maintenance of glucose homeostasis. Abnormalities of hepatic liver protein metabolism contribute to the

neurologic signs (hepatoencephalopathy) that frequently occur with acute hepatic failure. In particular, there is a decrease in the conversion of ammonia to urea. The resultant hyperammonemia contributes to dysfunction of the central nervous system. The body's ammonia load comes from two major sources: 1) bacterial synthesis in the large intestine; and 2) catabolism of amino acids. Therefore, to minimize colonic ammonia production, a low-to-moderate protein diet should be fed. In addition, maintaining positive energy balance will help to minimize protein catabolism. Ideally, the protein source should have a high branched chain to aromatic amino acid ratio. A 50:50 mix of ground corn cobs and sorghum meets this need and provides adequate protein to meet protein requirements. Processed corn and molasses can be added to provide glucose. Small amounts of this concentrate should be fed several times daily. Frequent feedings may facilitate maintenance of glucose homeostasis and prevent surges in colonic ammonia production. Legume hays, oats, and soybeans should be avoided because they contain a low branched chain to aromatic amino acid ratio. Grass hay should be fed as the fiber source.

Renal failure:
Chronic renal failure in horses is associated with hypercalcemia and azotemia (accumulation of urea), both the result of inadequate renal excretion. Phosphorus excretion can also be impaired and sodium deficits can develop because of poor renal conservation. In general, feedstuffs high in protein (legumes, soybeans), phosphorus (wheat bran), and calcium (legumes, calcium-containing supplements) should be avoided. On the other hand, hypoproteinemia can develop with chronic renal failure, necessitating an increase in the level of protein feeding (e.g. soybean meal). These animals also benefit from an increase in the energy density of the diet (fat supplementation).

Delivery of Nutritional Support to Anorectic or Hypophagic Horses

The method of nutritional support provided is primarily dependent on (1) whether or not the horse will voluntarily consume feed; (2) the nature and duration of the illness; and (3) economics. Adult horses can tolerate 2 to 3 days of feed deprivation providing hydration is maintained (either by water consumption or by intravenous administration of fluids). However, for illness of longer duration, some form of nutritional support is required. Nutritional support can be delivered by the enteral or parenteral (intravenous feeding) routes, and enteral feeding is either voluntary or involuntary. Voluntary feeding is by far the least invasive and should be encouraged. Donoghue (1992) has suggested that if an animal consumes at least 85% of its optimal intake, no other form of nutritional support is required.

Voluntary feeding can be encouraged by aggressive management of the primary problem, provision of a selection of feedstuffs (the "cafeteria" approach), and perhaps use of drugs purported to stimulate appetite (Naylor, 1999). In many sick horses, a reduction in appetite, at least in part, can be attributed to fever, pain and/or endotoxemia. In this regard, treatment of the primary problem

is essential for restoration of normal appetite. Relief of pain and fever by administration of nonsteroidal anti-inflammatory drugs (e.g. phenylbutazone, flunixin meglumine) may improve feed intake in some horses. Although hospitalized horses should be offered feeds similar to those fed at home, it is often necessary to provide a variety of feedstuffs for encouragement of intake. Highly palatable feeds such as fresh grass, leafy hays, and small amounts of grain or bran mash containing some grain can be offered. Only small amounts of these feeds should be offered initially to avoid problems such as diarrhea and laminitis (founder); it is generally recommended that increases in grains or concentrates should be limited to < 0.5 kg per day for a 500-kg horse. In healthy horses, addition of molasses to grains improves voluntary intake and this approach has been successful in some sick horses. The feeding of oils will increase energy intake in the face of suboptimal feed consumption. Naylor (1999) has reported that the administration of diazepam (10 mg IV for a 500-kg horse) can stimulate appetite in some sick horses. This author's clinical impression is that diazepam is ineffective as an appetite stimulant.

Horses that remain anorexic (or intake is persistently inadequate) require enteral or parenteral feeding. Although total parenteral nutrition is frequently used in sick neonatal foals, it can be cost prohibitive in mature horses (see below). Therefore, "involuntary" enteral feeding is the most cost-effective means for delivery of nutritional support in anorexic horses. It should also be noted that, as with feed deprivation and starvation, a prolonged period of total parenteral nutrition is associated with atrophy of the intestinal mucosa. Thus, another advantage of enteral feeding is maintenance of intestinal health and function. Besides anorexic horses, enteral feeding is indicated in horses that have a good appetite, but where the presence of oral, pharyngeal or esophageal problems precludes voluntary feed consumption e.g. a severely fractured jaw; soft tissue injuries involving the pharynx or esophagus (although some of these horses will do well if fed a sloppy gruel of ground complete feed or alfalfa meal). Horses with pharyngeal paralysis or similar neurologic dysfunction should not be allowed to eat because of the risk of feed aspiration.

Enteral feeding can be delivered via a nasogastric tube or a surgically-positioned esophagostomy tube. Use of esophagostomy tubes is reserved for cases in which the nasal passages, pharynx, or proximal esophagus must be bypassed, e.g. injuries to the head and neck. This approach is associated with a high complication rate and should only be used when tube feeding is anticipated for 10 days or more (Stick et al., 1981). Use of nasogastric tubes for force feeding can also be problematic. Repeated passage of a tube is traumatic and, in most cases, it is preferable to leave the tube indwelling. The tube is secured by placement of tape butterfly sutures between the tube and the false nostril, and by taping the tube to the cheek and gullet straps of the horse's halter. Between feedings the tube should be closed with a plastic syringe case or rubber stopper to prevent gastric distention from air. Indwelling tubes are also traumatic and can cause rhinitis, pharyngitis, and ulceration of the esophageal mucosa. To minimize these complications, a smooth pliable tube should be used. Even then, tubes tend to stiffen when exposed to digestive fluids and I recommend use of a new tube every 3 to 4 days

during prolonged tube feeding. Large-bore tubes (14F or ~12.5 mm internal diameter) are required for passage of most enteral diets, although slightly smaller tubes are suitable for administration of commercially available liquid diets (e.g. Osmolite-HN, Ross Laboratories).

Enteral Diets

Several enteral diets have been fed (via nasogastric tube) to sick horses, ranging from slurries of pelletized feeds or alfalfa meal to commercially available liquid diets designed for use in human patients. These liquid diets have been classified into three categories: 1) Blender diets - finely ground whole food suspended in water; 2) Composition diets - composed of highly digestible protein, carbohydrates, and fats; and 3) Elemental diets - diets containing small peptides and/or free amino acids rather than whole protein. Elemental diets are very expensive and rarely used in horses. Table 1 shows the ingredients for preparation of two enteral diets that can be used for nutritional support of horses. Note the number of batches required to meet the daily energy requirements of a 500-kg adult horse. Table 2 shows the calculated nutrient profiles of these diets as well as a commercial human diet that has been used in horses (Sweeney and Hansen, 1990). Currently, there is no commercially available enteral diet specifically designed for use in horses.

Table 1. Ingredients for two enteral diets, one made of alfalfa meal-casein-dextrose, the other a slurry of ground complete feed pellets, oil and water.

Alfalfa/casein/dextrose slurry**	Pellet-vegetable oil slurry**
454 g alfalfa meal	454 g pelleted complete feed†
204 g casein*	46 g (50 ml) corn oil
204 g dextrose	2-3 L water
52 g electrolyte mixture¶	DE per recipe = 1.76 Mcal
5 L water	
DE per recipe = 2.77 Mcal	
DE = digestible energy	

* Casein (Sigma Chemical Co.) or dehydrated cottage cheese (American Nutritional)

¶ Electrolyte mixture - 10 g NaCl, 15 g $NaHCO_3$, 75 g KCl, 60 g K_2HPO_4, 45 g $CaCl_2$, 25 g MgO

† e.g. Phase IV, Kentucky Equine Research, Inc.

** Approximately 6 batches of the alfalfa/casein/dextrose mixture and 9 batches of the pellet and vegetable oil slurry are required to meet the maintenance energy requirements of a 500-kg adult horse (DE = 16.4 Mcal/day).

Table 2. Nutrient profiles for three enteral diets supplying maintenance energy requirements for a 500-kg adult horse.

Nutrient	Require-ments*	Naylor Diet**	Pellet Slurry Diet¶	Osmolite-HN†
Energy (DE), Mcal	16.4	16.4	16.4	16.4
Protein, g	656	1710	505	688
Calcium, g	20	81	25	11.7
Phosphorus, g	14	41	23	11.7
Sodium, g	8.2	16.2	10	14.4
Potassium, g	25	159	37	24.2
Magnesium, g	7.5	97	8	4.7
Copper, mg	82	35	193	23.6
Zinc, mg	328	100	535	264
Iron, mg	328	984	518	211
Selenium, mg	0.82	0.9	2.1	-

* From Nutrient Requirements of Horses, ed 5, National Research Council, 1989
** Diet developed by Dr. Jonathan Naylor and composed of alfalfa meal, casein, and dextrose
¶ A slurry of complete feed pellets (Phase IV, Kentucky Equine Research, Inc.) and corn oil
† Ross Products Division, Abbott Laboratories, Columbus, Ohio

Use of pelleted complete horse feeds allows the clinician to select the diet based on the age and physiologic state of the animal. In addition, these feeds are nutritionally balanced (providing protein, energy, fiber, vitamins and minerals), easily procured, and less expensive than other hand-prepared enteral diets. Pellets must be pulverized in a kitchen blender. Just prior to feeding, the ground feed is mixed with water. Addition of corn oil is also recommended to increase the energy density of the diet. Soybean meal or whey protein can be added if a higher protein diet is needed. A disadvantage of this approach is that large volumes of water are required to reduce the viscosity of the slurry mixture, thus reducing the energy density of the diet. In most cases, 2 to 3 liters of water is mixed with each 1 lb of ground pellet. Even then, this mixture is difficult to administer by gravity flow and tube blockage is a frequent occurrence.

An alternative diet that has been used for enteral nutritional support of horses is composed of alfalfa meal, casein or dehydrated cottage cheese, dextrose, and an electrolyte mixture [the "Naylor diet"](Naylor, 1977; Naylor, 1999). Although the individual components of this diet are relatively inexpensive, these costs do not account for the labor input required for procurement and preparation of the mixture. One advantage of this diet is the provision of fiber (alfalfa meal) which helps to support large colon function. Osmolite-HN (Ross Products Division, Abbott Laboratories, Columbus, Ohio), a liquid diet designed for use in hospitalized human patients, has been used as the sole source of nutritional support in sick horses (Sweeney and Hansen, 1990). Primary components of this diet include soy protein, starch hydrolysates (maltodextrins, oligosaccharides), and vegetable oils. Each liter contains approximately 1 Mcal of energy, of which

64% is derived from carbohydrate sources, 9% from fat, and the remainder from protein. The fat is predominantly medium-chain triglycerides (MCT) prepared from the fractionation of coconut oil. MCTs do not require micelle formation for absorption in the small intestine. Furthermore, the medium length fatty acids (6 to 10 fatty acids) are water soluble and gain direct access to the portal circulation, thus providing a readily available energy source. Use of starch hydrolysates reduces the osmolality of the diet. In fact, Osmolite-HN is iso-osmolar (~300 mOsmol/L), whereas preparations containing dextrose or glucose tend to be hyperosmolar, a factor that can contribute to digestive upsets. The main disadvantage of this diet is expense; to meet the maintenance energy requirements of a 500-kg horse, the cost per day of treatment is $70 to $80. As an alternative, 60 to 70% of energy requirements could be met by this human enteral diet, with the remainder provided by slurries of alfalfa meal (e.g. 3 to 4 feedings of 1 lb alfalfa meal mixed with 2 to 3 L of water).

Feeding Regimens and Clinical Monitoring

Regardless of the diet chosen, the most important consideration is a gradual increase in the rate of feeding. For horses that have been anorectic for five days or more, a reasonable goal for the first day of nutritional support is provision of 25% of the target feeding volume. The amount fed can then be gradually increased over the next 2 to 4 days. As mentioned, it may not be necessary to feed 100% of requirements and, in many horses, delivery of 75-80% of needs will maintain body weight. Repeated clinical assessment will provide the best indication of tolerance to tube feeding. Vital parameters (temperature, pulse and respiration), gastrointestinal motility, and fecal volume and character should be evaluated two to three times daily. Horses on liquid diets commonly develop low-grade diarrhea, although the volume of feces produced depends on the diet. On the other hand, signs of colic, abdominal distention, poor gastric emptying (residual feed can be easily evacuated through the nasogastric tube at the time of the next feeding) and gastrointestinal motility indicate intolerance to the diet and the need for a reduction in the level of feeding. Given the high carbohydrate content of these enteral diets, laminitis is another potential complication. The character and strength of digital pulses should be assessed regularly, particularly during the early phase of feeding.

In general, tolerance to liquid diets is best when small feedings are delivered frequently. When feeding slurry diets to an adult horse, a reasonable target is to feed 2 to 4 liters every 2 to 4 hours. If the horse tolerates this feeding regimen, the volume can be gradually increased and the feeding frequency decreased. However, a 500-kg horse has an average stomach capacity of 7 to 9 liters and single feedings should not exceed ~6 liters. Even then, this volume should be administered over a 10- to 15-minute period. Slurry diets can be administered using a large funnel and gravity or by use of a pump. Individual feedings of commercial liquid diets (e.g. Osmolite-HN) can be administered through gravity feeding sets (and a small bore tube), with or without use of an infusion pump. With these diets, it is feasible to administer feedings over a 30- to 60-minute

period. In all circumstances, the proper position of the tube should be verified before each feeding. The tube must be flushed with warm water before and after each feeding. Recumbent horses should only be fed if kept in sternal position. Tube obstruction is common with the use of slurry diets. One method for clearing obstructions involves passage of a polyethylene tube inside the feeding tube to the site of obstruction. A 60-ml syringe is attached to the free end of the polyethylene tube and the obstruction is vigorously flushed with water.

The horse's daily water requirements should be calculated. If needed, additional water can be administered via the feeding tube. Even with a nasogastric tube in place, horses are able to safely consume water. Therefore, good-quality water should be available at all times. Horses with pharyngeal or esophageal dysfunction are the exception to this rule; their water requirements should be administered via the feeding tube.

The duration of enteral nutritional support will depend on the nature of the primary illness. Horses with pharyngeal or esophageal injury or reversible neurologic dysfunction may require a prolonged period of tube feeding. If possible, body weight should be measured every two to three days during the period of nutritional support. Maintenance of body weight will give the best guide as to the effectiveness of enteral nutrition. An increase in appetite will accompany improvement in clinical condition. When voluntary consumption is not precluded by the primary condition, the horse's appetite should be assessed regularly by offering palatable feeds such as fresh cut grass or leafy alfalfa hay. Even when the horse is willing to eat, it is best not to abruptly stop enteral nutritional support. Rather, the nasogastric tube can be removed to allow voluntary feed consumption, but replaced two to three times daily to allow administration of the enteral diet. The volume and frequency of tube feeding can be gradually reduced as voluntary intake increases, with discontinuation of enteral support when feed intake reaches 70 to 80% of maintenance requirements.

Parenteral Nutrition

Enteral feeding is contraindicated in horses with severe small intestinal diseases, such as anterior enteritis or following surgical correction of a strangulating obstruction. In these cases, intravenous feeding (parenteral nutrition) is the only option available for nutritional support. A complete discussion of parenteral nutrition is beyond the scope of this article. Furthermore, the expense of total parenteral nutrition often precludes its use in horses. Nonetheless, it should be emphasized that the administration of glucose-containing fluids can provide considerable energy. One to two liters of a 5% dextrose solution will provide 50 to 100 g of glucose (170 to 340 kcal). Over a 24-hour period, a 5% dextrose solution administered at a rate of 2 liters per hour will provide approximately 8 Mcal, or ~50% of maintenance energy requirements. Depending on requirements for other types of IV fluids, it may be possible to administer a larger quantity of dextrose. However, even at 1 to 2 liters per hour of 5% dextrose, some sick horses develop hyperglycemia and glucosuria, necessitating a decrease

in the rate of glucose administration. This hyperglycemic state possibly reflects decreased peripheral glucose uptake as a result of mild insulin resistance. As a general rule, IV glucose should be used as the sole source of nutrition for no more than 2 to 3 days.

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