Enteral/Parenteral Nutrition in Foals and Adult Horses Practical Guidelines for the Practitioner

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KEYWORDS

• Enteral nutrition • parenteral nutrition • Equine • Critical illness

KEY POINTS

- The metabolic response to starvation in healthy individuals includes a decrease in metabolic rate and sparing of body proteins.
- The catabolic response to injury or inflammation may increase metabolic rate and protein breakdown despite a decrease in nutritional intake.
- Enteral nutrition provides intraluminal nutrition to the gut and can improve gut barrier integrity, mass, protein content, motility, and function.
- Parenteral nutrition provides nutritional support when the enteral route is unavailable.
- Parenteral nutrition must be handled with strict aseptic techniques and should be gradually introduced and discontinued to prevent metabolic consequences.

INTRODUCTION

Protein/Calorie Malnutrition

The average, healthy adult horse can apparently tolerate food deprivation (protein/calorie malnutrition [PCM] or simple starvation) for 72 hours with few systemic effects. During the first hours to days of starvation, glycogen stores are used from various tissues (liver, kidney, muscle) for glucose production. As glucose becomes limited, many body tissues begin to rely on fatty acid oxidation and the production of ketone bodies as energy sources. Glycerol produced from lipid degradation, lactate from the Krebs cycle, and amino acids provided from muscle tissue breakdown continue to be used for gluconeogenesis to provide energy to glucose-dependent tissues (central nervous system and red blood cells). This response to starvation correlates with an increase in circulating levels of growth hormone, glucagon, epinephrine, leptin, and cortisol and a

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decrease in insulin and thyroid hormones. During PCM, there is an increased drive to eat and a decrease in energy expenditure. Metabolism slows in an effort to conserve body fuels, and the body survives primarily on fat stores, sparing lean tissue until such a time as refeeding occurs.[1]

Although similar adaptive responses to PCM can occur in the healthy older foal, the neonate has limited body reserves. The healthy newborn foal should have enough liver glycogen to support energy needs for several hours of life. However, glycogen stores at birth can vary significantly with illness or prematurity. Lack of nutritional reserves can result in hypoglycemia and hypothermia and quickly affect the ability to maintain normal function and behavior. A weak suck response often develops, leading to an increased risk of aspiration pneumonia, which can start a vicious cycle of deterioration. Consequently, when treating an inappetent neonate, institution of nutritional support is recommended as quickly as possible.

Catabolic Response to Injury

During illness or after trauma, food intake frequently decreases. However, despite this decline in intake, the adaptive responses to starvation do not occur. In contrast to PCM in humans and laboratory animals, the metabolic response to injury (eg, critical illness, sepsis, trauma, surgical manipulation) is characterized by an increased metabolism and the onset of a catabolic process leading to excessive breakdown of tissue proteins, which are used as a metabolic fuel. Insulin resistance develops and hyperglycemia may occur despite the absence of food intake. This metabolic state is the result of a complex interaction of inflammatory cytokines, circulating hormones, and neurotransmitters and is designed to provide endogenous substrates for gluconeogenesis, wound healing, immune cell replication, and synthesis of acute phase.[2] Although this response is beneficial, long-term muscle breakdown results in loss of muscle strength, visceral organ dysfunction secondary to loss of structural and enzymatic proteins, impaired wound healing (caused by loss of precursors for wound healing), immunosuppression, and compromise to the patient's overall health.

Food deprivation during this hypermetabolic/catabolic state results in a much greater loss of lean muscle mass and visceral protein than would be expected during simple starvation.

Nutritional supplementation will reverse the catabolic processes occurring during simple starvation but will not completely reverse those occurring during metabolic stress because as long as tissue injury persists, catabolic processes are maintained. The goals of nutritional support in critical illness should be to save life, maintain muscle mass, preserve and improve cellular and tissue function, and speed recovery.[3] More recently, research in nutritional therapy has focused on attenuating the metabolic response to stress, preventing oxidative cellular injury, and favorably modulating the immune response with the ultimate goals being to reduce disease severity, complications, and length of stay and improve outcome.

The purpose of this article is to review methods and goals for nutritional support of equids in the field, not to make recommendations for treating critically ill patients in an intensive care setting. However, it is worth noting that there continues to be significant debate in human medicine regarding the value of early nutritional support in critical illness.[4–8]

Indications for Nutritional Support

Individuals with pre-existing PCM are at a disadvantage when intake is restricted because of illness. In both humans and laboratory animals, nutritional supplementation has been shown to positively influence both survival and morbidity during