



## STARVATION AND MALNUTRITION OF HORSES: RECOGNITION AND TREATMENT

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### Summary

Starvation and partial starvation lead to loss of body weight and condition, which are general clinical signs seen also with bad teeth, bad feet, intestinal parasites, cancer, and several chronic diseases. More specific signs may accompany malnutrition, but often poor performance is the only indication. Thus both starvation and malnutrition require a careful differential diagnosis from the points of view of humane investigators and veterinarians. Abrupt refeeding usually results in the death of a severely starved horse in about three days. Thus, refeeding should be gentle, beginning with water and electrolytes, then enterals, slurries and, eventually, good quality forage and concentrates. Once the horse begins to thrive, it can be built up to a growth regimen. Not all nutritional abuse is due to nature or naivete. For example, the use of pre-race "jugs" of intravenous nutrients occasionally causes fatal reactions. Also, the partial starvation of young horses confined in the dark to suppress manifestation of the wobbler trait has dubious merit, medically or ethically. Nutritional suffering may be countered therapeutically by recognition and treatment, and prophylactically by planning, education and regulation.

### Introduction

Horses suffer starvation and malnutrition for natural reasons that can be avoided or mitigated by planning, and for man-made reasons that can be prevented by education and regulation. Horse owners and veterinarians must engage in this planning, education and regulation. And, when prevention fails, they must be ready to intervene by recognizing and treating the consequences of nutritional neglect or abuse.

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Table 1. Causes of emaciation in horses

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Deprivation of food, total or partial —
bad teeth, bad feet, bad feed, bad people.
Deprivation of energy or protein or both —
abuse of supplements, abuse of wobbler candidates.
Seasonal declines of energy and nutrients in pasture.
Malnutrition — protein deficiency, fluorosis.
Malabsorption, chronic diarrhea.
Intestinal parasites, also severe lice or ticks.
Cancer — melanoma, adenoma, lymphoma, etc.
Diabetes mellitus.
Chronic disease of liver, pancreas, kidney, heart.
Chronic infectious disease — pneumonia, abscesses, EIA.
Lactation, pregnancy.
Old age, senescence and senility.

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### Recognition

Starvation is death or suffering from lack of food. The lack is usually assumed to be total unless qualified as partial. The severity is cumulative. Deprivation of energy-yielding food but not water is accommodated for weeks or months, but deprivation of water can kill in days. The word fasting is often substituted for starving but more precisely is voluntary, thus it is seldom if ever appropriate for animals.

Starvation is an extreme form of malnutrition, a more general term. Malnutrition can result from insufficient or improper food. It can involve overfeeding as well as underfeeding, and nutrient imbalances as well as deficiencies or excesses. Feeding a vitamin-mineral supplement during a period of partial starvation, intended to reduce weight or retard growth, may cause imbalances, that is, malnutrition.

Starvation is usually recognized by loss of body weight and condition in adults, or by poor growth in the young. These superficial signs are non-specific, bringing up a long list of possible causes or "rule-outs" (Table 1). A painstaking differential diagnosis by a fully qualified person is essential to establishing the fact, or should it be called the arguable proposition, that these non-specific signs are due to deprivation of food.

Clinical signs of malnutrition may be more specific or indicative of certain nutrients (Table 2).<sup>1</sup> Early signs of malnutrition are often rather general, such as poor performance, poor attitude and poor coats.

Confirmation of a diagnosis of starvation or malnutrition is established by history taking, physical examination, laboratory findings and, occasionally, post mortem examination.

*History:* Natural events, such as a flood, a drought, or winter



Table 2. Specific signs of malnutrition in horses<sup>1</sup>

Crooked bones and large joints — calcium deficiency or excess; possibly copper and zinc deficiencies, vitamin D deficiency, vitamin A deficiency or excess, manganese deficiency.

Shifting lameness and softening and enlargement of jaw bones and facial crest —phosphorus excess.

Nervousness, muscle tremors and incoordination — depletion of magnesium and calcium, plus stress (transit).

Discolored teeth, bone lesions, unthriftiness — fluorine excess.

Goitre, enlarged thyroid gland, stillbirths, abnormal estrus cycles — iodine deficiency or excess in pregnant mare.

Anemia in foals —iron deficiency.

White muscle disease, possibly tying-up, possible abortion— deficiencies of selenium and vitamin E.

Blind staggers, colic, diarrhea — acute selenium intoxication.

Loss of hair, mane, tail, cracked coronets —chronic selenium intoxication.

Incoordination, slow heart, missing beats — thiamin deficiency induced by bracken fern or Amprolium (a coccidiostate).

Poor hooves —possibly biotin deficiency.

pasture decline, are usually conspicuously evident. Nevertheless, they should be quantified and recorded in detail, especially if a case has legal implications. The local county agricultural extension agent may have relevant data about regional soils, pastures and poisonous plants as well as weather.

An owner (or agent) with good intentions may provide a useful history of the availability and consumption of food and water. Epidemiologic studies, however, have shown the need for careful validation of questioning techniques for estimating food intakes of humans and companion animals, especially when memory must stretch. No such validated technique exists for horses, to my knowledge. Though ration evaluation of grazing animals is fairly standard, there are certain differences.

Most of my colleagues have a routine series of questions that they ask in an orderly manner in order to determine feeding history. This approach imposes a system, and there may have been a different one or none at all. So I always start with the simplest prompt and encourage dialogue.

If more than one person is involved in the feeding, I try to speak to them separately, because I have found that people try, consciously or unconsciously, to show themselves in a good light by saying what they think I want to hear. If possible, I obtain three feeding histories, then divine the truth by trigonometry.

Customary ration evaluation involves estimate of intakes of all available feeds, analysis of these feeds, calculation of

Table 3. Body condition scores of horses<sup>1</sup>

1. *Extreme emaciation.* No fatty tissue. Protruding bones. Wasted muscles especially noticeable over bones; flat shelf over transverse processes.
2. *Emaciation.* Slight fat cover. Prominent bones. Wasted muscles.
3. *Thin.* Fat covers transverse processes and half-way up spinous processes; tailhead prominent but individual vertebrae not seen; ribs seen sharply.
4. *Moderately thin.* Slight back ridge; ribs barely discernible.
5. *Moderate.* Back is flat (no crease or ridge), ribs easily felt but not seen.
6. *Moderately fleshy.* Fat feels spongy over ribs and around tailhead; back crease slight or absent.
7. *Fleshy.* Back crease definite. Ribs covered but individual ribs can be palpated. Fat is palpable in neck, rump.
8. *Fat.* Back creased, neck thick, fat along withers, behind shoulders, and inside thighs.
9. *Too fat.* Back crease is deep; fat bulging on neck, along withers, behind shoulders, around tailhead, inside thighs.
10. *Morbidly obese.*

daily intakes of energy and nutrients, and comparison of these estimated daily intakes to minimal standards.<sup>1,2</sup> Even under the best circumstances, any estimate of pasture intake is approximate. Under conditions in which starvation can occur, obstacles to estimating food intake may be present overtly or covertly. Nevertheless, in my view, the attempt to quantify availability and consumption of food and water, both at the time of inspection and historically for days or months, is needed to illuminate obstacles as well as to reach the best estimate and some suggestion of its precision. Premises and pastures should be inspected for hazardous materials, for example, poisonous plants, old lead batteries, and salmonella sources.

If the horse has been in a group, ration evaluation may be more correct for the group than the individual. An individual may have been starving amidst plenty, due to bad feet or teeth, or to crowding and fear, or to disease. A horse may appear to be grazing but actually be consuming little food. Genuine continuation of feed intake despite cachexia is indicative of cancer and other chronic diseases. Information is needed on the feeding opportunity for sure and the individual's actual intake whenever possible.

*Physical Examination:* The first signs are behavioral and often missed. Humans experience hunger from 3-4 hours to 3-4 days after their last meal. A horse's response to offered feed may suggest hunger or its abatement. Spontaneous activity becomes depressed in line with metabolic rate. Reactivity to



external stimuli may also be depressed, though this depression may take longer to become evident.

In a week or two, loss of body substance may become noticeable, especially if stress is superimposed on starvation. Quite often the loss of body condition is so slow and progressive that it escapes the attention of somebody who sees the horse every day but is noticed by a person who has not seen the horse for a week. ("The horse got thin during my vacation" is probably genuine and shifts the blame.)

For humane investigations, one needs to quantify body weight and body condition score (Table 3), and to conduct a thorough physical examination with special attention to the conditions that must be ruled out (Table 1). The body condition scores of other horses in the group, on the same farm, or in the vicinity, also needs to be recorded.

Body weight of horses is often predicted from heart girth measured by a tape. Now that portable scales are more readily available, actual weight should be determined whenever possible. Care should be taken to avoid use of the word weight when only a tape estimate is available.

Body condition scores follow a system developed by Potter and Associates.<sup>1</sup> Optimal is scored as 5 or 6, severe emaciation as 1, morbidly obese as 10 (Table 3). Attention is given to superficial body fat, especially at certain sites — the rump, shoulder, neck and flank. Muscle wasting is most likely to be evident in muscles covering bones, such as the temporals and masseters, the rump, and the loin above the transverse processes of the vertebrae. Prominence of bones may be most evident in the rib cage, hip points, top of skull and spinous processes.

Dehydration may be detected by increased skin turgor, perhaps sunken eyes, and perhaps delayed capillary refill. A dehydrated horse will almost certainly drink as soon as water is made available, unless it has lost relatively more sodium than water and has a low serum sodium concentration. Occasionally a horse admitted to veterinary hospital will fail to drink from a bucket about a yard off the floor. Also an occasional horse used to a bucket or trough may not drink from a stream. So care is taken to offer water in a way acceptable to the horse.

*Laboratory Aids:* Dehydration usually affects the extracellular space first, thereby increasing the hematocrit, serum protein (total solids), and serum sodium concentration. Starved horses also drink less and undergo hemoconcentration.

In addition, starved horses mobilize fat from adipose tissue, thereby increasing plasma concentrations of glycerol and free fatty acids. These substances are taken up by the liver and converted respectively to glycogen and triglycerides. The latter increase in blood and account for most of the

characteristic increase in blood total lipids of starved horses.<sup>3</sup> When serum separates, hyperlipemia may be evident as an opalescence or creamy appearance. If a horse has been starved for a long time, so that its fat reserves are entirely depleted, it may no longer exhibit hyperlipemia.

Food deprivation also causes an increase in serum bilirubin in horses.<sup>4</sup> The increase in bilirubin regresses significantly on the increases in triglycerides and free fatty acids. Any increase in serum bilirubin above the predicted value may indicate liver disease, which itself may contribute to anorexia and weight loss.

Glucose concentration in blood tends to fall as hepatic production declines during food deprivation. On the other hand, metabolic adaptations to starving tend to conserve glucose utilization and maintain or elevate blood glucose concentration. A mare in the last trimester may develop a pregnancy toxemia-like condition, with impaired regulation of blood glucose concentration.

Low serum albumin is probably not as good a guide to protein insufficiency in horses as in some other species. It might reflect losses of albumin in the kidney or digestive tract as well as reduced production in the liver. Once established, hypoalbuminemia might cause diarrhea.

Immune competence declines significantly within 3 or 4 days of food deprivation.<sup>5</sup> Indications are given by several tests, including neutrophil phagocytosis, lymphocyte transformation, and delayed skin hypersensitivity. Such tests are not specific for starvation, but positive results should be considered consistent.

Intestinal absorption decreases during lapses of oral intake of nutrients. This has been demonstrated by the xylose absorption test.<sup>6</sup> A delayed peak or flattened response of plasma xylose concentration following an oral dose is consistent with but not specific of food deprivation.

All too often, the response of a starved horse to refeeding is terminal collapse after about three days.<sup>7</sup> Likely reasons are impaired digestive function and imbalances among metabolic rates developing upon the reintroduction of food. The metabolic refeeding syndrome, characterized by hypophosphatemia and hypokalemia,<sup>8</sup> has not been described in horses but is so likely to occur that serum concentrations of potassium and inorganic phosphate should be monitored.

*Necropsy:* Upon food deprivation, fat is mobilized before protein. Fat depots undergo serous atrophy which may be observed postmortem as the first sign of starving. Depleted usually first are the coronary and perirenal adipose tissues, then subcutaneous fat, last omental fat. Eventually, serous atrophy disappears and there is little or no trace of fat depots. Wasting of muscle also indicates prolonged and severe starvation.



Muscle atrophy is more rapid during catabolic stress than simple starvation. Hyperactivity of the adrenal gland may be accompanied by increases in size and blood flow.

Post-mortem examination affords an opportunity to rule out cancer, intestinal parasites, diseases of the liver and kidney, and other causes of emaciation (Table 1). The pituitary should be examined for an adenoma (cholesteroloma). Lymphoma may be found in the thymus as well as lymph nodes. Tissues taken for histologic examination should include liver, kidney and, when appropriate, lymph nodes, thymus, pancreas, pituitary and gut wall. Parasites need to be identified expertly.

*Occurrence:* Natural disasters are usually overt, except perhaps for toxins, and partially predicable. Veterinarians and humane societies, especially those with hospitals and other facilities, should cooperate in the planning for animals with their regional Emergency Management Agency. These agencies maintain records of all emergency activities in the region and state.

Seasonal decline of pastures in provision of energy, protein, phosphorus and beta-carotene (precursor of vitamin A) is well known to farmers but not, sadly, to amateur horse owners. Pastures for horses should be stocked for their lowest carrying capacity, unless supplements of conserved feeds are available for use when needed. Otherwise, horses will enjoy an abundance during late spring and early summer, and a paucity for the remaining 7-9 months of the year. Pastures for horses, the most notoriously selective grazers, must be managed with non-selective grazers, such as sheep or bush-hogs, otherwise a horse may fast amidst an abundance of overgrown and unpalatable herbaceous material, for example, overgrown fescue.

The backyard pony may suffer because its owners do not know about the seasonal decline of pasture. Worse, advertising may lead people to believe that nutritional supplements will improve efficiency of utilization of feed by substantial amounts. The horse starves for energy and protein while fed a surfeit of vitamins and trace minerals, probiotics and wondrous pseudo-scientific concoctions.

Not all starvation and malnutrition is due to neglect. Two medically and ethically questionable practices are being carried out by veterinarians.

*"Starving in the dark builds character"*. This Alaskan aphorism describes a dubious treatment of selected young horses. It was developed in two stages.

First, young horses showing signs of developmental orthopedic disease (DOD) were subjected to partial starvation.<sup>9</sup> The rationale was that abnormal conversion of cartilage to bone (osteochondrosis, OCD) may have genetic component that is manifested only in response to abundant

feeding. It follows, in this theory, that underfeeding will prevent manifestation of this genetic predisposition. So young Thoroughbreds and Quarter horses were fed only limited amounts of poor hay. This partial starvation stunted growth and roughened coats, the "Colorado Syndrome."

Second, initial restriction of energy and protein followed by "paced" growth, has been used to prevent the clinical manifestation of an inherited predisposition for cervical malformation.<sup>10</sup> So far, affected youngsters have been from certain strains of stakes winning Thoroughbreds owned by famous names. Candidates were chosen by their breeding and by radiographic demonstration of a narrow cervical canal at 2-4 months of age, the putative familial trait.

The rationale was that even mild OCD in the cervical vertebrae may lead to harmful pressure on the spinal nerves if the canal is small initially. Further, the thinking was that exercise may exacerbate the damage. Thus, these selected youngsters have been confined in small, dark stalls and partially starved.

The starvation regimen has been tailored to the individual by a veterinarian using a computer program to reduce energy and protein to 50-60% of the recommendations of the National Research Council.<sup>1</sup> After several months, the ration was increased in a "paced" manner, so that full expected growth was attained by about 24 months.<sup>10</sup> This procedure has been described as 100% effective, but no comparative trial has been published.

*Is a Jug a Drug?* Another questionable nutritional practice is far removed from starvation in the dark — it might be regarded as supercharging in the open.

The expectation that the metabolic processes of athletes might be supercharged by large doses of certain vitamins and trace elements has been largely dismissed by studies on experimental animals and human athletes.<sup>11</sup> Contrarily, it persists in the backstretch, where intravenous "jugs" are commonplace on race-day. Jugs are not generally regarded as drugs by the racing fraternity; more commonly, jugs are thought to round out an optimal nutritional program. A nutrient becomes a drug, however, when that is the intention of its use. Thus, if jugs are intended to promote racing performance, then their use should be addressed by the racing authorities.

The sudden deaths of Pleasant State, the 1992 champion two-year old filly, and Dr. Somerville, another well-known Thoroughbred, following intravenous injections of B-vitamins have been described as "unexpected" and the injections as "routine."<sup>12</sup> Whether these injections should be routine is arguable, but the prospect of anaphylactic reactions to vitamin-mineral injections is not, for these are well-known in humans. The occasional anaphylactic responses might be



Table 4. Oral electrolyte mixture, suitable for provision in 15-20 liters of drinking water.<sup>13</sup>

Substance	Amount, grams	Amount, teasp*
Sodium chloride	10	2
Sodium bicarbonate	15	3
Potassium chloride	75	15
Dipotassium phosphate	60	12
Calcium chloride	45	9
Magnesium oxide	25	5

\*Approximately 5 grams per teaspoonful.

“unexpected” only in the sense that they are not well documented in horses and likely to remain so. (My letter on the subject was thought unusual and unsuitable for publication by *The Blood-Horse*.)

A veterinarian giving a jug should have a vial of epinephrine handy to combat, when necessary, any anaphylactic reaction. The reaction may range from mild to severe. Bronchospasm and swelling of respiratory tract linings may choke the horse, making it lunge and flounder, a hard target for the epinephrine injection. Cardiovascular collapse and hypotension may slow the horse down but make raising a vein difficult.

A milder clinical reaction may involve sufficiently prolonged lowering of blood pressure to lead to a compensatory shut down of the blood supply to the kidney. The horse must be watched carefully for signs of depression and altered urine flow. Acute kidney failure most often becomes manifest about the third day after the injection.

### Treatment

The treatment for starvation and malnutrition is to offer first shelter and comfort if possible, then a little water, and finally a little suitable sustenance.

If starvation is severe, refeeding is likely to be fatal and must be approached cautiously, starting with water and electrolytes then enterals, as described below. In most cases, however, about 2 lb of good hay is suitable for the start of refeeding. After a few such meals a few hours apart, the hay is allowed at about half-maintenance (8-10 lb/day) split into four meals for two days before being made available free choice. Refeeding may be that simple in most cases, but a severely debilitated horse will require more attention.

The discussion that follows should be regarded as a general guide, based on a general knowledge of nutrition, a few experimental studies of food deprivation of horses, field experience in the refeeding of partially starved young horses, and clinical experience in nutrition support of sick horses that

have refused feed. The general principle is to start slowly — avoid overloading digestive and metabolic systems that have reduced capacities in adaptation to starvation. These debilitated systems should be restored gently. If all goes well, the horse is built up to a growth regimen within about two weeks.

A severely starved or dehydrated horse should first be offered relatively small volumes of water or a dilute solution of electrolytes, about 0.5-1.0% salt, and dextrose (D-glucose), about 2%. A solution containing sodium and glucose is preferable to plain water, because it promotes stomach emptying and rapid absorption. It also avoids hyponatremia, which would discourage further voluntary drinking. Prudence suggests a series of 2-4 liter drinks about 20-30 minutes apart until avid thirst has abated, when it becomes safe to offer water free choice. If all goes well, then the next step may be to offer good hay, perhaps one small flake (2-4 lb) at first.

If the horse will not drink well voluntarily, it becomes a candidate for parenteral or enteral fluids. Parenteral administration of water, electrolytes and nutrients might be needed only in the most severe cases, in which the horse is extremely weak or dehydrated, with hypovolemia and hypotension. It must be supervised by an experienced veterinarian.

The basal intravenous solution should be Ringer lactate or a multiple balanced electrolytes with 5% dextrose. Dextrose releases much less insulin when given intravenously compared to orally, and it is not as well utilized without insulin. Persistent hyperglycemia may inhibit fatty acid release from adipose tissue, hence production of beta-hydroxybutyrate, a preferred endogenous substrate of enterocytes. Thus intravenous dextrose does not help reestablish digestive functions.

Enteral administration of water, sodium and glucose restores the extracellular fluid volume almost as fast as the intravenous route in humans and, presumably, in horses. It requires a nasogastric tube (stomach tube). Suitable for horses are most proprietary mixtures of oral rehydration salts, such as those made for treating dehydration due to diarrhea in children and calves (Table 4).<sup>a</sup> Few of these salt mixtures contain phosphate, and an extra supply of dipotassium phosphate ( $K_2HPO_4$ ) might be needed to control hypokalemia and hyperphosphatemia if these supervene during refeeding.<sup>8</sup>

If the horse has been starved for a week or more, its

<sup>a</sup>Oral rehydration salts (sodium chloride, 3.5g; potassium chloride, 1.5g; sodium bicarbonate, 2.5g; glucose, 20g; dissolve in one liter of drinking water). Interstate Drug Exchange, Inc., Amityville, NY.

<sup>b</sup>Osmolite, Ross Laboratories, Columbus, OH.

<sup>c</sup>NutraPrime and NutraFoal, KenVet, Ashland, OH.

<sup>d</sup>Ensure, Ross Laboratories, Columbus, OH.



absorptive capabilities may have diminished in line with partial atrophy of enterocytes and flattening of villi. This debilitated condition may respond best to enteral fluids containing electrolytes and simple nutrients that do not require digestion and are readily absorbed. Some veterinary hospitals may have enterals in 1000 ml liquid packs<sup>b</sup> or powders with instructions for dilution.<sup>c</sup> Now that enterals in 400 ml cans<sup>d</sup> for humans are relatively inexpensive and readily available in supermarkets, their use should be considered for the initiation of refeeding. Two liters of enterals repeated after 60 minutes would provide about 4 Mcal or one-quarter of maintenance energy, a substantial start of refeeding a starved horse.

Next in line would be the use of a slurry via nasogastric tube, about 4-6 liters given 3-4 times a day, with a total of 16-24 liters per day. Energy intake is started at about half the maintenance level and built up gradually, according to the response of the individual horse.<sup>13</sup> In addition to electrolytes (Table 4), suitable solids include glucose, dehydrated cottage cheese and alfalfa meal. Other suitable ingredients are glucose-polymers, brewers yeast, alpha-cellulose, methyl-cellulose (psyllium fiber) and, perhaps on the third or fourth day, corn oil.

In slurries, total electrolyte concentration remains constant about 1% (weight/volume). The concentration of each other ingredient starts at about 1% and builds up to about 5% in 5 days. These guides are deliberately general because detailed comparative trials have not been reported on the refeeding of starved horses. The amounts of solids (but not water) should be diminished if the horse shows signs of diarrhea, colic or founder.

A starved horse being fed enterals or slurries by stomach tube should at the same time be allowed access to good hay (no more than half-maintenance or 8-10 lb/day at first). Its voluntary daily intake of hay should be recorded, and the involuntary (tube) feeding should be reduced as voluntary feeding improves.

After a horse has reached a maintenance intake of hay, 16 lb/day, then it may be offered a small amount of high-quality concentrate to ensure adequate amounts of protein, vitamins and minerals, starting about 2 lb twice a day and increasing to about 5 lb twice a day. However, a horse can easily eat 24-30 lb/day of hay, and this may be all that is necessary, especially if one-quarter or more is alfalfa hay. This would provide an abundant supply of good-quality protein, which is needed for replenishment of tissue proteins.

Eventually, once the initial reintroduction of feed has been accomplished, the recovering horse should be converted gradually to a growth regimen. Once it starts to thrive, the formerly starved horse is fed like a yearling.

If pasture is available, the starved horse, which is undergoing careful reintroduction to food, should be allowed access only for 2-4 hours a day at first. Take it gently. Imagine that the pasture is spring grass, that your horse has been in the barn all winter, and that you wish to avoid founder.

If a young horse has been starved partially, it must be refeed cautiously. Refeeding is complicated, because compensatory growth induces unpredictable flexure deformities (contracted tendons). One day the horse is fine, the next up on the toes. We have seen this phenomenon in young horses raised on the sandy soils of southern New Jersey when given good alfalfa hay, and in youngsters deliberately underfed to prevent manifestation of a genetic predisposition to OCD or narrow cervical canal.<sup>9,10</sup>

The latter practice, in my view, is ethically questionable in three regards. First, the combination of partial starvation and confinement in the dark is unkind to a young horse. Second, if the manifestation of the wobbler syndrome is only partially suppressed, the horse may retain a greater than average predisposition to stumble, hence pose a more than average risk for a rider. Third, if undisclosed to breeders, the practice threatens the breed. The toleration of undesirable genes incidental to selection for precocious speed could spread the wobbler trait as fast as, say, the slanting vulva and the bleeding lung. My point is that not all man-made disasters, including the malnutrition and malgenetics of our horses, are due to neglect by the naive, some manifest the self-interest of the cognoscente.

## References

1. National Research Council. *Nutrient Requirements of Horses*, ed 5. Washington: National Academy Press, 1989.
2. Kronfeld DS. Feeding on horse breeding farms. *Proc Am Assoc Equine Pract* 1978; 24: 461-464.
3. Naylor JM, Kronfeld DS, Aclamnd H. Hyperlipemia in horses: effects of undernutrition and disease. *Am J Vet Res* 1980; 41: 899-905
4. Naylor JM, Kronfeld DS, Johnson K. Fasting hyperbilirubinemia and its relationship to free fatty acids and triglycerides in the horse. *Proc Soc Exp Biol Med*. 1980; 165 86-90.
5. Naylor JM, Kenyon SJ. Effect of total calorific deprivation on host defence in the horse. *Res Vet Sci* 1981; 31: 369-372.
6. Freeman DE, Ferrante PL, Kronfeld DS, Chalupa W. The effect of food deprivation on the D-xylose absorption test results in mares. *Am J Vet Res* 1989; 50: 1609-1612.
7. Hungerford TG. *Diseases of Livestock*, ed 9. Sydney: McGraw-Hill, 1990.
8. Solomon SM, Kirby DF. The refeeding syndrome: a review. *J Parent Ent Nutr* 1990; 14: 90-97.
9. Lewis LD. *Feeding and Care of the Horse*. Philadelphia: Lea & Febiger, 1982.
10. Donawick WJ, Mayhew IG, Galligan DT, et al. Early diagnosis of cervical vertebral malformation in young Thoroughbred



horses, and successful treatment with restricted diet and confinement. *Proc Am Assoc Equine Pract* 1989; 35: 525-528.

11. Clarkson PM. Vitamin and trace minerals. In: *Perspectives in Exercise and Sports Medicine. Volume 4: Ergogenics—Enhancement of Performance in Exercise and Sport*. Eds. DR Lamb, MH Williams. Carmel, IN: Brown & Benchmark, 1991; 4: 123-182.

12. *The Blood-Horse* 1990; 116 (42): 5533 and 1992; 118 (36): 3906.

13. Naylor JM, Freeman DE, Kronfeld DS. Alimentionation of hypophagic horses. *Compend Cont Ed Vet Practnr* 1984; 6:S93-S99.

## REHABILITATION OF HORSES SUFFERING FROM MALNUTRITION

D.B. Poupard

### SUMMARY

A condition classification system is described for malnourished horses, the categories being very poor, poor, underweight, moderate, and acceptable. A ration for undernourished horses is described using a concentrate with 12% protein, and mineral supplementation. Feed quantities and schedules are described for the various condition categories.

### INTRODUCTION

The SPCA National Horse Care Unit, on the eastern coast of South Africa, has the charge to care for horses which have been neglected and are malnourished. In 1984 it was determined that there was a need for a diet specifically for horses suffering from malnutrition; one which would ensure a safe progression from malnutrition to health with the least amount of stress, colic and organic dysfunction. The eventual goal being to allow the horse to reach an acceptable weight condition.

Early on, in dealing with large numbers of malnourished horses, it was learned that a thorough veterinary examination with specific blood tests was extremely helpful. If the horse did not show an initial improvement with good quality feed of

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**Acknowledgements:** D.A. Daniel, for support during research; Dr. J.P. Fleming, for advice and support during research and for veterinary services; S.R.P. Poupard, for support during research; Dr. A.L. Pringle, for support during research; EPOL Animal Feeds, and Romix Animal Feeds.

Table 1. Progression of improvement in body condition.

<u>initial condition</u>	<u>time</u>	<u>eventual condition</u>
very poor	2-3 mon.	poor
poor	2-3 mon.	underweight
underweight	1-2 mon.	moderate
moderate	1-2 mon.	acceptable

adequate quantity, certain veterinary clinical tests were done to determine if the horse had an underlying organic dysfunction. These included 1) liver function test; 2) kidney function test; 3) gastrointestinal dysfunction test; and 4) complete blood count and differential. All horses initially were wormed and the teeth were floated.

The diet is conservative in the amount of protein (12%), with a Ca:Ph ratio of 1.8: to 1. It is a grain/molasses meal, containing a balanced ration, with supplemental vitamins and minerals, fed with a good quality hay such as eragrostis, veldt, red grass, teff or other "bulk" hay, free choice. Lucerne/alfalfa hay is not included at first, because it is believed that the higher protein content causes too high an energy level in a malnourished horse (in a condition classified as "underweight"). Once the horse has reached an acceptable weight condition, lucerne/alfalfa can then be introduced with care taken regarding the freedom of lush grazing in order to prevent scouring.

Meal has been found to be preferable to cubes or pellets as it has been noted that horses suffering from malnutrition tend to bolt the food given to them. When the kukuya grazing is lush and available, faster improvement is observed in the malnourished horse.

### Stages of Weight Improvement

The length of time between the different stages of weight improvement has been quite consistent on those horses with no organic dysfunction or dental problem. With the feeding program used, it has generally taken from 6 to 10 months for a horse to progress from "very poor" to "acceptable." Table 1 shows the typical time needed for progression from one classification to another. From 1984 to 1992, 6,490 horses were classified visually by an estimate of weight and body condition. The most extreme stage is "very poor" and is described below. There are three classifications between very poor and "acceptable." These are poor, underweight, and moderate. Each of the conditions described for "very poor" are slightly better as the condition improves, until an "acceptable" condition exists. The "acceptable" condition is also described below. Therefore, the condition classification