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# Metabolic responses of chronically starved horses to refeeding with three isoenergetic diets

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**Objective**—To examine metabolic responses of chronically starved horses to refeeding with 3 isoenergetic diets.

**Design**—Uncontrolled clinical trial.

**Animals**—22 mature mixed-breed horses that were emaciated but otherwise clinically normal.

**Procedure**—Horses were fed 1 of 3 diets: alfalfa hay, oat hay, or a combination diet of half oat hay and half commercially prepared ration. Digestible energy of diets was gradually increased throughout the refeeding period. One pre- and 4 postprandial blood samples were obtained daily, and analyses included RBC count, Hct, and determination of hemoglobin, glucose, insulin, free fatty acid, total bilirubin, 2,3-diphosphoglyceric acid, phosphorus, magnesium, calcium, sodium, and potassium concentrations. Body weight, fecal output, and feed and water consumption were measured and recorded daily. Repeated-measures ANOVA was used to examine dietary and temporal (day) effects of the 3 dietary regimens during 10-day trials.

**Results**—19 horses survived. Three horses (2 fed alfalfa diet, 1 fed combination diet) died of metabolic or gastrointestinal problems. Increasing temporal effects in serum concentrations of glucose, insulin, magnesium, calcium, and sodium; decreasing temporal effects in serum concentrations of free fatty acid, 2,3-diphosphoglyceric acid, and phosphorus; and dietary effects in serum concentrations of glucose, insulin, magnesium, and potassium were detected in the 19 surviving horses. Serum phosphorus and free fatty acid concentrations decreased dramatically during the first 5 days of refeeding with all 3 diets. Serum magnesium concentrations increased in horses fed the alfalfa hay diet, whereas improvement was not evident in horses fed oat hay or combination diets. Horses receiving the alfalfa and oat hay diets had lower postprandial glucose and insulin concentrations than horses receiving the combination diet. Horses fed oat hay alone ate 92% of feed offered, compared with 98% feed consumption for horses fed alfalfa hay or combination diets.

**Clinical Implications**—Clinically normal emaciated horses can be successfully rehabilitated by gradual refeeding with a high forage diet. (*J Am Vet Med Assoc* 1998;212:691–696)

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Metabolic and immune responses to food deprivation in horses have been documented by means of short-term fasting studies (12 hours to 9 days), using healthy horses and ponies on regular feeding schedules prior to enrollment in these studies.[1-10] Research on emaciated or fasted human patients has provided information on physiologic changes that develop during starvation and immediately on refeeding enterally or through intravenous infusion, such as **total parenteral nutrition (TPN)**.[11-16] A broad range of interrelated metabolic derangements with potentially adverse effects, including death within 5 days of initiating TPN, have been termed the refeeding syndrome.[14] Severe hypophosphatemia, hypomagnesemia, and hypokalemia are metabolic disorders that develop most often in human beings as a result of refeeding used in the treatment of chronic starvation.[11,12,14] Hypophosphatemia contributes to depletion of phosphorylated metabolites, such as ATP and **2,3-diphosphoglyceric acid (2,3-DPG)**, and red blood cell dysfunction.[11,14]

Limited research has been conducted on physiologic responses of chronically starved horses to refeeding.[17,18] Veterinarians, nutritionists, and members of rescue organizations can benefit from information on refeeding malnourished or starved horses. The purpose of the study reported here was to examine metabolic responses of chronically starved horses to refeeding, using 3 isoenergetic diets.

## Materials and Methods

**Horses and basic study design**—All phases of the study were approved by the University of California Animal Care and Use Committee. Three 10-day refeeding trials, using 22 horses, were conducted between January and May 1996. A death rate of 20% was anticipated for this study, and 6 horses/dietary treatment were estimated as necessary to satisfy statistic requirements for validity. Twenty-two horses with unknown histories were purchased in Mexcali, Mexico by a commercial buyer. After satisfying federal importation procedures, including quarantine (minimum 3 days) and serologic testing (equine infectious anemia, piroplasmosis, venereal trypanosomiasis, and glanders), horses were transported by trailer to the University of California-Davis. Horses (12 mares, 10 geldings) were of mixed breeding, were 3 to 24 years old (mean ± SD, 10 ± 6 years) as estimated by examination of teeth, and weighed between 242 and 399 kg (532 to 878 lb; mean, 307 ± 44 kg [675 ± 97 lb]). Withers height ranged from 137 to 155 cm (mean, 143 ± 6 cm). **Body condition scores (BCS)**[19] ranged from 1 to 4, using a scale with a maximum score of 9 (obese); most horses had BCS of 1 or 2. Horses in each refeeding trial (trial 1, 6; trial 2, 8; trial 3, 8) were emaciated but clinically normal as determined by physical examination. Five horses in trial 2 that had resolving submandibular abscesses were treated topically with cleansing and nitrofurazone ointment. These horses maintained good appetites. One horse had difficulty masticating oat hay but would consume the meal prior to the subsequent feeding.

Horses were housed in individual concrete stalls (6.8 × 3.5 m). Bedding was not provided, but approximately 40% of the floor was covered with rubber mats. Horses were weighed daily between 1:00 and 2:00 PM, using an electric portable scale[a] after blood sampling was complete. Temperature and humidity[b] were recorded continuously in the center of the barn. Mean daily temperature ranged from 15 to 20, 15 to 24, and 14 to 33 C (59 to 68, 59 to 75, and 57 to 92 F) during trials 1, 2, and 3, respectively. Mean daily humidity ranged from 50 to 60, 40 to 76, and 28 to 70% during trials 1, 2, and 3, respectively. Fecal samples were collected on day 5 and submitted to the veterinary medical teaching hospital for parasite identification and quantification, using flotation and McMaster's tests.[20]

**Dietary formulations and feeding protocol**—Within each trial, horses were randomly assigned the following 3 diets: 100% **oat hay** (**OH**; n = 7), 100% **alfalfa hay** (**AH**; 7), or a combination diet of **half oat hay and half commercially prepared ration**[c] (**CD**; 8). The commercial diet was an extruded complete feed containing multiple grains, ground alfalfa, and molasses. These feed types have been shown to elicit different glycemic responses when fed to healthy horses.[21] All feeds were analyzed by an approved laboratory[d] prior to the start of the first trial (Table 1). Diets were formulated according to the National Research Council's tables[22] for the composition of feeds to provide equal calories per kg of body weight. On arrival at the research unit, horses were subjectively estimated to have had a minimum body weight loss of 25%, compared with horses of similar height and breed type with BCS of 4 to 5. Therefore, the **digestible energy (DE)** requirement per horse was calculated, using the formula:

$$\text{Mcal DE/d} = 1.4 + 0.03\ \text{BW},$$

where BW = body weight of horse (kg)[23] calculated at 125% of body weight at arrival.

Table 1—Digestible energy (DE), nutrient, and moisture content on an as fed basis of 3 diets fed to 22 emaciated horses during a 10-day refeeding period

| | Diets | | |
|---|---|---|---|
| Analyte | Alfalfa hay | Oat hay | Oat hay/ commercial |
| DE (Mcal/kg) | 2.28 | 1.62 | 2.70 |
| Moisture (%) | 11.9 | 11.7 | 13.2 |
| Crude protein (%) | 21.4 | 6.8 | 14.0 |
| Crude fiber (%) | 22.1 | 27.8 | 13.8 |
| Crude fat (%) | 2.3 | 2.4 | 4.8 |
| Ash (%) | 7.5 | 5.9 | 8.7 |
| Phosphorus (%) | 0.44 | 0.11 | 0.23 |
| Magnesium (%) | 0.62 | 0.13 | 0.39 |
| Potassium (%) | 1.8 | 1.0 | 1.1 |
| Calcium (%) | 0.92 | 0.14 | 0.45 |
| Sodium (%) | 0.21 | 0.59 | 0.22 |

Water was available to horses ad libitum prior to transport and throughout the study. Feed was withheld from horses on arrival (day 0), and horses were fed their first meal on the following morning at 9:00 AM (day 1). Amount of feed given was gradually increased from 50 to 100% of the calculated DE/d throughout the 10-day refeeding period. On days 1 through 3, feed providing 50% of horses' calculated daily DE requirement was divided into 6 equal feedings and offered every 4 hours (1:00, 5:00, and 9:00 AM and 1:00, 5:00, and 9:00 PM). During days 4 and 5, total daily amount fed was increased to that which would provide 75% of horses' DE requirement and was offered, using the same schedule. During days 6 through 10, feed providing 100% of horses' DE requirement was fed in 3 equal feedings every 8 hours (1:00 and 9:00 AM and 5:00 PM). Meals were individually weighed for each horse. All refused feed was collected, weighed, recorded, and discarded prior to the next meal. Water intake for each horse was recorded daily, and feces were collected and weighed prior to each feeding.

**Blood collection**—On day 0, a 14-gauge indwelling catheter was placed aseptically in the left jugular vein of each horse. Catheters were replaced when swelling or functional problems developed and several hours prior to obtaining the next blood sample to avoid effects of stress responses resulting from catheter placement. Preprandial blood samples (30 ml) were collected at 8:45 AM prior to the first meal (9:00 AM). Postprandial samples (25 ml each) were drawn every hour between 10:00 AM and 1:00 PM for a total of 4 samples.

Blood samples to be analyzed for concentrations of electrolytes, **free fatty acid (FFA)**, insulin, and glucose were immediately placed on ice and allowed to clot. Serum was obtained from these samples and frozen at –56 C (–68.8 F). Complete blood counts were performed each morning on preprandial samples collected in tubes containing EDTA. Samples for which 2,3-DPG concentration was to be determined were collected into evacuated tubes containing potassium oxalate and sodium fluoride. One milliliter of this blood was immediately added to 3 ml of cold trichloroacetic acid and centrifuged, and the supernatant was then frozen for storage and measurement of 2,3-DPG concentration.

**Blood analyses**—Glucose concentration was determined by use of an autoanalyzer.[e] Insulin was measured, using a commercially available radioimmunoassay kit.[f] Free fatty acid[g] and 2,3-DPG[h] concentrations were quantified, using enzymatic colorimetric assays. Electrolyte (calcium, potassium, magnesium, phosphorus, and sodium) concentrations were measured, using an automated spectrophotometry system.[i] Red blood cell count, **hemoglobin (Hgb)** concentration, and Hct were determined by means of an automated cell counter,[j] using the impedance principle.[24]

**Statistical analyses**—Repeated-measures ANOVA was used to examine effects of day and diet on daily blood biochemical responses of each horse to its 9:00 AM meal. The magnitude of the daily postprandial increment change from the preprandial (8:45 AM) value for glucose, insulin, FFA, and total bilirubin concentrations was calculated as the area under the curve, using the trapezoidal method. For 2,3-DPG concentration, RBC count, Hct, and Hgb and electrolyte concentrations, repeated-measures ANOVA was based on log-transformed data, using a weighted geometric mean. These variables were measured once or twice daily, and postprandial peaks were not anticipated. Univariate results include a Huynh-Feldt adjustment for the possibility of pseudoreplication. Post hoc analyses were conducted, using pairwise contrast tests (for mean effect of diet) or contrast decomposition (for day × diet interactions). A value of $P < 0.05$ was considered significant.

## Results

**Mortality**—Three of 22 horses were euthanatized for humane reasons prior to the end of the 10-day study period. The first of these horses (trial 1, CD; BCS = 1) became recumbent and rapidly deteriorated with muscle fasciculations and neurologic manifestations of head banging, tonic rigidity, and inability to rise at 1:00 AM on day 6. Analyses of a blood sample obtained on the morning prior to euthanasia revealed the following low values: calcium concentration,

Table 2—Body weight, daily feed and water intake, and daily fecal output for surviving horses (n = 19) in each dietary group for days 1 through 3 (50% DE requirement fed), 4 and 5 (75% DE requirement fed), and 6 through 10 (100% DE requirement fed) of the study period

| | Diets | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Alfalfa hay | | | Oat hay | | | Oat hay/commercial | | |
| | n = 5 | | | n = 7 | | | n = 7 | | |
| Initial body weight (kg)* | 314 ± 35 | | | 304 ± 48 | | | 292 ± 42 | | |
| Wither height (cm) | 145 ± 5 | | | 143 ± 8 | | | 142 ± 5 | | |
| Weight gain or loss (kg)* | 2.6 ± 4.3 | | | 14.7 ± 20.6 | | | 3.1 ± 17.0 | | |
| | Days | | | Days | | | Days | | |
| | 1–3 | 4–5 | 6–10 | 1–3 | 4–5 | 6–10 | 1–3 | 4–5 | 6–10 |
| Body weight (kg)*,† | 298 ± 30 | 306 ± 35 | 317 ± 35 | 302 ± 43 | 304 ± 50 | 319 ± 57 | 280 ± 49 | 282 ± 52 | 295 ± 57 |
| Feed consumption | | | | | | | | | |
| Daily amount offered (kg)* | 2.9 ± 0.3 | 4.3 ± 0.4 | 5.8 ± 0.6 | 3.97 ± 0.6 | 5.96 ± 0.9 | 7.9 ± 1.1 | 3.1 ± 0.4 | 4.6 ± 0.6 | 6.1 ± 0.8 |
| % consumed | 93 ± 17 | 100 | 100 | 93 ± 16 | 95 ± 12 | 90 ± 8 | 98 ± 6 | 100 | 98 ± 5 |
| Daily water intake (L) | 19 ± 8 | 25 ± 8 | 31 ± 4 | 20 ± 6 | 22.5 ± 10 | 28 ± 7 | 20 ± 8 | 23 ± 12 | 29.5 ± 9.5 |
| Daily fecal ooutput (kg)* | 3 ± 1 | 9 ± 3 | 10 ± 7 | 5 ± 1 | 9 ± 1 | 13 ± 4 | 3 ± 0.6 | 5 ± 1 | 8 ± 3 |

*To convert to lb, multiply by 2.2. †Mean body weight ± SD, days 1 to 3, 4 to 5, and 6 to 10 after feeding.



10.6 mg/dl; magnesium concentration, 1.2 mg/dl; phosphorus concentration, 2.4 mg/dl; and 2,3-DPG concentration, 1.38 mol/ml. Gross or histopathologic lesions were not detected at necropsy. A second horse (trial 2, AH; BCS = 2) was inappetent after the first meal, developed profuse watery diarrhea on day 2, had positive results on a bacteriologic culture for *Salmonella saint-paul*, and was euthanatized on day 4. The third horse (trial 3, AH; BCS = 2.5) developed diarrhea on day 5 but continued to eat and drink. On the afternoon of day 8, increasing signs of abdominal pain and a depressed appetite were evident. Euthanasia was elected when signs of abdominal pain became uncontrollable and recumbency was prolonged. Volvulus of the left large colon at the diaphragmatic flexure was found on necropsy. Blood samples obtained on day 8 revealed low calcium (10.2 mg/dl), phosphorus (1.9 mg/dl), magnesium (2.1 mg/dl), and 2,3-DPG (1.68 mol/ml) concentrations. Only data from the 19 horses that survived during the 10-day study period were included in statistical analyses.

**Feed and water intake and fecal output**—Horses consumed 91.7 ± 16.2, 97.5 ± 5.0, and 98.2 ± 4.0% of total amounts offered of OH, AH, and CD diets, respectively, with a significant difference in consumption between horses fed OH and CD diets (Table 2; $P = 0.02$). A significant difference was not detected between diet fed and water consumed. Fecal output was significantly different between all pairings of diets.

**Weight gain**—Mean arrival weight of surviving horses fed AH was 314 ± 35 kg (691 ± 77 lb), horses fed OH was 304 ± 48 kg (669 ± 106 lb), and horses fed CD was 292 ± 42 kg (642 ± 92 lb; Table 2). Mean weight gains during the 10-day study period for surviving horses fed AH, OH, and CD diets were 2.6 ± 4.3, 14.7 ± 20.6, and 3.1 ± 17.0 kg (5.7 ± 9.5, 32.3 ± 45.3, and 6.8 ± 37.4 lb), respectively. A significant difference between diets with respect to weight gain was not detected.

**Parasite load**—*Strongyle* sp was detected by means of McMaster's test in all fecal samples collected. The number of **eggs per gram (EPG)** ranged from < 50 to 3,550, with a mean of 742 ± 811 EPG. Six horses had 100 EPG, which was classified as subclinical parasitic infection. *Parascaris* sp was identified in fecal samples from 3 horses, which had 50, 750, and 950 EPG. *Dictyocaulus filaria* was detected on flotation of a fecal sample from 1 horse.

**RBC count, Hct, and Hgb concentration**—Significant differences were not found among horses in the 3 diet groups or between values for horses on days 1 and 10 for RBC count, Hct, or Hgb concentrations.

**Total bilirubin concentration**—Mean total serum bilirubin concentrations (0.88 ± 0.11 mg/dl) were within the reference range (0.1 to 2.5 mg/dl) for adult horses throughout the study.[25] Significant differences in bilirubin concentrations were not detected among horses with respect to day or diet.

**Glucose concentration**—Mean serum glucose concentrations (81.9 ± 3.4 mg/dl) for horses fed the 3 diets were within the reference range for healthy horses (71 to 104 mg/dl).[25] A maximum in serum glucose concentration was reached 2 to 3 hours after horses ingested their morning meal. Serum glucose concentrations increased significantly ($P = 0.0001$) over time in horses fed all diets during the trial. Postprandial serum glucose concentrations were significantly ($P = 0.006$) higher in horses fed the CD diet than in those fed the AH diet. Dietary effects on serum glucose concentration were not detected between horses fed AH and OH diets ($P = 0.06$) or between horses fed OH and CD diets ($P = 0.17$).

**Insulin concentration**—Changes in serum insulin concentration reflected changes in serum glucose concentrations in horses fed all 3 diets. Maximum postprandial insulin concentrations were reached 3 to 4 hours after meals and were generally higher than reported resting concentrations (4.7 ± 1.0 U/ml).[21] Serum insulin concentrations increased significantly ($P = 0.0003$) over time in horses fed all diets during the trial. Significant differences were detected in serum

insulin concentrations between horses fed AH and CD diets and between horses fed OH and CD diets ($P$ = 0.005 and 0.0003, respectively). Horses fed the CD diet had higher insulin concentrations than horses fed AH or OH diets. This difference was particularly evident on day 6, when the amount fed was increased to 100% of the calculated DE requirement.

**Free fatty acid concentration**—Mean preprandial concentration of FFA in serum of healthy horses has been reported to be 0.25 ± 0.12 mmol/L.[26] For horses consuming all 3 diets, there was a rapid temporal decrease ($P$ = 0.0001) in serum FFA concentrations during the study period. Mean preprandial serum FFA concentration on day 1 was 0.334 ± 0.003 mmol/L, and by day 10, it had decreased to 0.041 ± 0.008 mmol/L. Significant differences related to diet consumed were not detected.

**Phosphorus concentration**—Although serum phosphorus concentrations for all horses were within the reference range (3.1 to 5.6 mg/dl) at the beginning of the study,[27] there was a slow but steady decrease ($P$ = 0.03) in serum phosphorus concentrations for horses fed all diets during the trial. Significant differences related to diet were not identified. Horses fed the AH diet had serum phosphorus concentrations (mean, 2.1 ± 0.6 mg/dl) lower than reference values by day 4, whereas horses fed the OH and CD diets had concentrations less than reference values (mean, 2.4 ± 0.7 and 2.2 ± 0.8 mg/dl) by days 5 and 7, respectively.

**2,3-DPG concentration**—The reference mean resting concentration of 2,3-DPG in healthy horses is 2.1 mol/ml.[28] Concentrations of 2,3-DPG for horses in this study were low, ranging from 1 to 2 mol/ml, with a mean of 1.4 ± 0.2 mol/ml. The temporal decline in 2,3-DPG concentrations for all horses during the study was significant ($P$ = 0.0001). Significant effects related to diet type were not identified.

**Magnesium concentration**—The reference range for serum magnesium concentration in healthy horses is 1.3 to 2.5 mg/dl.[25] Mean serum magnesium concentration on day 1 in horses fed the AH diet (1.4 ± 0.3 mg/dl) was at the low end of the reference range but steadily increased to a mean value at the high end of the reference range (2.6 ± 0.4 mg/dl) by day 8. In contrast, mean serum magnesium concentrations in horses fed OH and CD diets remained low (1.6 ± 0.1 and 1.5 ± 0.1 mg/dl, respectively) throughout the study period. Temporal ($P$ = 0.0001) and dietary effects ($P$ = 0.005) on serum magnesium concentrations were evident for all horses; however, concentrations in horses fed the AH diet were significantly higher than concentrations in horses fed OH ($P$ = 0.005) or CD ($P$ = 0.002) diets.

**Calcium, sodium, and potassium concentrations**—Mean calcium concentration on day 1 (10.8 ± 0.3 mg/dl) in horses fed all 3 diets was slightly below reported[25] reference range values (10.9 to 12.8 mg/dl) for healthy horses. Serum calcium concentrations increased significantly ($P$ = 0.04) over time, but significant differences related to diet were not detected. The reference range for serum sodium concentrations in healthy horses has been reported[25] as 132 to 146 mmol/L. In general, significant ($P$ = 0.0002) temporal effects were evident across all dietary regimens, with serum sodium concentrations declining between days 1 and 5 and increasing between days 6 and 10. Minimal changes in mean serum potassium concentrations were detected between days 1 and 10 (mean, 3.9 ± 0.1 mmol/L) for horses fed the various diets, and values were within the reference range[25] (2.4 to 4.7 mmol/L) for healthy resting horses. Throughout the 10-day study period, mean serum potassium concentrations in horses fed the OH diet (3.6 ± 0.1 mmol/L) were significantly lower than those in horses fed AH (3.9 ± 0.2 mmol/L; $P$ = 0.02) and CD (3.98 ± 0.2 mmol/L; $P$ = 0.04) diets.

## Discussion

Starvation or malnutrition in horses can be caused by intentional neglect, ignorance, economic hardship of owner, or seasonal variation in availability of pasture feed. Investigations of starvation in 36 of 52 California counties involved 2,177 horses during 1994 and 1995.[k] When weight loss is 50% of normal body weight or more, prognosis for survival is poor.[17] Dietary regimens developed for refeeding chronically malnourished horses must take into account physiologic and physical changes that take place during starvation, such as delay in gastric emptying and slower absorption of nutrients. These changes have been observed in horses from which food has been withheld for 96 hours[29] and in human beings who have fasted.[12] Diets of hay,[18] grain and oil,[17] and hay and grain[30] have been recommended for refeeding malnourished horses. In the United States, certain nonprofit rescue organizations feed free-choice alfalfa hay or a combination of grass hay and pelleted complete feed.[k] Successful nutritional rehabilitation is achieved when chronically malnourished horses have regained normal body weight, which usually takes 3 to 10 months.[30]

Horses enrolled in this study were representative of chronically starved horses that may be taken to equine veterinarians or rescue organizations for nutritional rehabilitation. These horses were the product of inadequate nutrition, economic hardship, and lack of preventive veterinary care. The horses in this study were emaciated but clinically normal with good appetites. They were standing, alert, quiet, and had no clinically important signs of lameness. Mean age was 10 years, and only 5 of the 22 horses were 15 years or older. All horses had total bilirubin concentrations within the reference range, a finding consistent with normal liver function. Six horses, including 2 horses that died, had subclinical parasite burdens, and 6 had parasite burdens of > 1,000 EPG. During days 2 through 5, most horses developed a transient increase in fecal water content and a decrease in fecal ball formation, which did not compromise their appetite or hydration status. All horses were clinically anemic with low RBC counts, Hct, and Hgb concentrations that continued to decrease throughout the study. Anemia was most likely caused by chronic lack of nutrients that resulted in decreased viability of erythrocytes and an inability to replace aged erythrocytes. Marked

improvement in these values could not be expected within a study period of only 10 days.

Three horses died during this refeeding trial. One horse was euthanatized because it had salmonellosis. Necropsy of a second horse revealed no pathologic lesions as cause for observed acute neurologic signs and recumbency. A metabolic disorder was suspected, because this horse had developed hypophosphatemia, hypocalcemia, and hypomagnesemia 3 days before necropsy. A third horse was euthanatized because of a torsion of the left large colon at the diaphragmatic flexure. This horse also developed severe hypophosphatemia and hypocalcemia. The second and third horses had low daily 2,3-DPG concentrations prior to euthanasia.

Mean body weight of 19 surviving horses was 307 ± 44 kg (675 ± 97 lb) on enrollment in the study. On day 10, mean body weight of these horses was 310 ± 52 kg (682 ± 114 lb). On days 1 through 5, horses were fed 50 to 75% of their calculated daily DE requirement based on 125% of their enrollment body weight, and in general, horses fed AH and CD diets lost weight (Table 2). Between days 1 and 5, weight change for horses fed the OH diet was not evident. Weight loss was probably caused by insufficient caloric intake but may also have been attributed to losses through feces, fecal water, urine, or heat production. When 100% of the daily DE requirement was fed to these horses, body weight increased. The large variability in weight gain between horses fed OH and CD diets (Table 2) was most likely caused by filling of the gastrointestinal tract with feed and water; the OH diet was extremely bulky, compared with the AH diet. Increase in muscle mass or deposition of fat would not be expected at this early stage of rehabilitation; however, the high survival rate along with the gradual small increase in body weight after initial weight loss was encouraging.

Refeeding syndrome was first recognized in the 1940s with the release of survivors from Nazi and Japanese concentration camps during and after World War II.[14-16] These survivors were emaciated but appeared clinically normal. When increases in the incidences of hypertension, cardiac insufficiency and failure, and neurologic complications in these survivors were observed, the Minnesota Experiment[15] was conducted to study the effects of 6 months of starvation and subsequent oral refeeding in volunteers. Testing of volunteers did not reveal evidence of cardiac or respiratory dysfunction when refeeding was initiated; however, several individuals developed insufficiencies or failure during the recovery phase.[14,15] In the 1970s and 1980s, the syndrome was recognized as affecting emaciated patients who had serum electrolyte values within the reference range at the start of refeeding, using TPN. These patients suffered from a variety of illnesses, such as anorexia nervosa, malabsorption syndrome, cachexia caused by cancer, and chronic gastrointestinal obstruction.[11,12,14] Refeeding syndrome arises when emaciated patients are given concentrated calories, primarily in the form of glucose, parenterally or enterally. Initial clinical signs include peripheral edema, cardiac insufficiency, myocardial infarction, cardiac, respiratory, or hepatic failure, coma, convulsions, and acute death within 3 to 5 days.[12,14]

During starvation, patients are in a catabolic state that depletes body stores of fat, muscle, and electrolytes. Renal adaptation enables the body to maintain serum electrolyte concentrations that are within the reference range. Free fatty acids from adipose tissue are the primary source of energy metabolites[31] during fasting or starvation, and high serum concentrations of FFA would, therefore, be expected. Introduction of carbohydrates, specifically glucose, to starved patients results in release of insulin, the primary hormonal regulator of glucose. Insulin prevents release of FFA and causes an intracellular influx of glucose and selected electrolytes, which, in turn, decreases serum concentrations of those substances. Availability or solubility of carbohydrate sources determines the intensity of the glycemic response.[31] Alfalfa and oat hay are low in starch (< 3%) and high in the insoluble carbohydrate cellulose (25 to 28%),[22] whereas the commercial ration fed to horses in this study contained 18.3% starch.[1] Insulin stimulates anabolic protein synthesis, which further depletes the body's marginal mineral and electrolyte stores often leading to severe extracellular hypophosphatemia, hypomagnesemia, and hypokalemia.[11,14] This response can cause depletion of phosphorylated metabolites, especially ATP and 2,3-DPG, and results in RBC dysfunction, including decreased viability, reduced ability to pass through capillary beds, and inability to release oxygen to tissues.[11] Cardiac and respiratory failure may then develop.[11,14,31,32]

Responses of horses in this study to refeeding were similar to responses reported for human beings affected by refeeding syndrome. Free fatty acid and serum phosphorus concentrations were initially high but decreased dramatically during the first 5 days of refeeding. This was true for all horses fed all diets. In addition, horses fed the CD diet that contained 18.3% available starch in the commercial ration portion of the diet had higher postprandial glucose and insulin concentrations than horses fed the AH or OH diets. Serum magnesium concentrations improved in horses consuming the AH diet, which contained the most magnesium (Table 1). Concentrations of 2,3-DPG and RBC values were less than reference values when horses were enrolled in the study and did not improve significantly for horses fed any of the diets during the study.

Small frequent meals were offered initially in this study, because gastric volume and emptying time and digestive and absorptive capabilities are often greatly reduced in starved human beings and horses.[12,28,30] Oat or grass hay may be too bulky to provide horses with adequate quantities and quality of required calories and nutrients. Early in the refeeding regimen, high-quality, leafy alfalfa hay may induce gastrointestinal problems in horses not previously adapted to alfalfa hay. Concentrated pelleted feed may elicit an exaggerated insulin response. The AH diet was well accepted in amounts offered and supplied the highest concentration of magnesium. Actual amounts fed depended on the health status, body weight, height, and history of individual malnourished horses.

Monitoring of electrolytes, especially phosphorus and magnesium, may assist in evaluating progress and detecting deficiencies. Deworming and correction of

dental problems will further improve prognosis for recovery.

[a]Model H90-3042, Fairbanks, St Johnsbury, Vt.
[b]Serdex, Bacharack, Pittsburgh, Pa.
[c]EquineSenior, Purina Mills Inc, St Louis, Mo.
[d]Association of Official Analytical Chemists, JL Analytical Service, Modesto, Calif.
[e]YSI 2300 STAT Plus, Yellow Springs, Ohio.
[f]Kit No. D1804, Micromedic Systems, Horsham, Pa.
[g]Procedures 541 and 35-UV, Sigma Diagnostics, St Louis, Mo.
[h]Procedure 337, Boehringer-Mannheim, Indianapolis, Ind.
[i]Ektachem DT-60, Eastman Kodak Co, Rochester, NY.
[j]System 9000, Serono-Baker Diagnostics Inc, Allentown, Pa.
[k]Questionnaire responses from California animal control and equine rescue personnel.
[l]Purina Mills Inc, St Louis, Mo.

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