Effect of oral administration of electrolyte pastes on rehydration of horses

Harold C. Schott II, DVM, PhD; Shannon M. Axiak, DVM; Kristina A. Woody, DVM; Susan W. Eberhart, BS

Objective—To determine whether the composition of electrolyte pastes formulated for oral administration influences voluntary water intake (WI) by horses recovering from furosemide-induced dehydration.

Animals—6 horses.

Procedures—Voluntary WI, body weight, and blood and urine constituents were measured before and after induction of dehydration by furosemide administration and overnight withholding of water; these same variables also were measured during a 36-hour rehydration period. Each horse was evaluated 4 times with random application of 4 treatments (electrolyte pastes) that provided 0.5 g of KCl/kg of body weight, 0.5 g of NaCl/kg, 0.25 g of NaCl and 0.25 g of KCl/kg, or no electrolytes (control treatment). Electrolyte pastes were administered 3 times (4, 8, and 12 hours after start of the rehydration period).

Results—Administration of all electrolyte pastes resulted in significantly greater voluntarily WI, compared with the control treatment, and was accompanied by significantly greater recovery of body weight when NaCl was a component of the paste. Administration of NaCl and NaCl-KCl pastes tended to produce a state of transient hyperhydration; however, electrolyte administration also resulted in significantly greater urine production and electrolyte excretion during the final 24 hours of the rehydration period. Adverse effects of oral administration of hypertonic electrolyte pastes were not observed.

Conclusions and Clinical Relevance—Oral administration of electrolyte pastes to dehydrated horses increases voluntary WI and improves rehydration during the rehydration period. Rehydration is more rapid and complete when NaCl is a component of the electrolyte paste. (Am J Vet Res 2002;63:19–27)

Food and water deprivation, gastrointestinal tract disorders, and prolonged exercise can produce substantial depletion of body stores of fluids and electrolytes. Treatment of horses dehydrated by these conditions, especially those afflicted with profuse diarrhea, typically involves IV administration of large volumes of fluids. In contrast, forced enteral rehydration with electrolyte solutions administered via nasogastric intubation or by voluntary intake (drinking) remains the mainstay of treatment of dehydration in ruminants and humans, respectively. In these species, use of IV administration of fluids is generally limited to patients in hypovolemic shock that are unable to ingest or retain fluids administered orally. Although many dehydrated horses can be effectively rehydrated by repeated administration of electrolyte solutions via a nasogastric tube, this practice is not widely used because of the limited volume (10 to 12 L) that can be administered during each treatment and safety concerns for the horses and treatment personnel associated with repeated nasogastric intubation.

Many producers have become proficient at passing semirigid esophageal tubes to administer isotonic fluids and electrolyte solutions to cattle as a practical and economical alternative for treatment of dehydration associated with diarrhea in calves. Using a slightly different approach, riders commonly provide electrolytes to horses during endurance events in the form of orally administered pastes in an attempt to stimulate drinking and attenuate depletion of body stores of fluids and electrolytes consequent to sweating. These electrolyte pastes are hypertonic in relation to body fluids, and their use has raised concerns about adverse effects on rate of gastric emptying and the potential for a transient shift of body water into the lumen of the gastrointestinal tract to initially dilute the hypertonic paste.

In comparison to IV or enteral administration of fluids, orally administered electrolyte pastes have not been studied extensively in controlled situations. Sosa-León et al reported that oral administration of a single high dose of electrolytes (176 g, an equal mixture of table salt [NaCl]:lite salt [50% NaCl and 50% KCl], on a wt:wt basis) increased voluntary water intake (WI) and enhanced recovery of body weight during a 6-hour rehydration period following furosemide-induced dehydration. A study conducted in our laboratory also documented that oral administration of a 2:1 mixture of NaCl:KCl (0.6 g of mixture/kg of body weight, divided into 3 doses) can attenuate loss of body weight by increasing voluntary WI in horses performing endurance exercise (60 km) on a treadmill. Results of these 2 studies provide support that oral administration of electrolytes can be an effective and safe method to attenuate and, perhaps, correct dehydration providing horses have access to water and are monitored to ensure that they voluntarily drink water.

To our knowledge, the effect of varying the composition of the electrolyte paste on WI has not been studied. Furthermore, although increases in plasma tonicity and hypovolemia are stimuli of thirst in mam-
Materials and Methods

Horses—Five geldings and 1 mare of various breeds and ages that ranged in body weight from 419 to 509 kg were included in the study. The horses were part of a group of horses used for research that were affected with recurrent airway obstruction and were in clinical remission while kept on pasture. Studies were performed from June to August. At the beginning of each experimental period, horses were moved from pasture to stalls in an appropriately ventilated hospital ward. Body weight of each horse was measured, and diet was changed from pasture grasses to a complete pelleted feed.

Eight kg (approx 2% of body weight) of the pelleted feed containing 0.5% sodium chloride and 1.3% potassium (on a weight basis) was fed daily as four 2-kg meals offered at 6-hour intervals (2 AM, 8 AM, 2 PM, and 8 PM). A 4-day period was allowed for acclimation to the diet change, during which the horses had a mean ± SEM weight loss of 6.5 ± 1.7 kg. Following the acclimation period, each gelding was fitted with a custom-designed urine collection harness (urine was not collected from the 1 mare in the study). The experimental protocol and all procedures were performed in accordance with guidelines of the Institutional Animal Care and Use Committee of Michigan State University.

Protocol—Water intake and urine production were measured to the nearest 0.25 L in horses in the euhydrated state during an initial 12-hour period (day 1, 8 AM to 8 PM). Body weight was measured prior to provision of feed at the beginning and end of this period, and all feces produced during the 12-hour period were collected and placed into a plastic bag. The amount of feces produced was weighed to the nearest 0.25 kg, and a representative sample was saved for determination of fecal water content by drying to a constant weight. Dehydration, with a goal of producing loss of 4 to 5% of body weight, was induced over the subsequent 12-hour period by administration of 3 doses of furosemide—1 mg/kg (body weight, IV, q 5 hours) starting at 8 PM on day 1 and accompanied by withholding of water overnight. Urine produced during the overnight dehydration period was not collected, but all feces produced were collected from the stall floor at 8 AM on day 2. At that time, body weight was measured, and urine collection harnesses were placed onto the geldings. All urine and feces produced during the subsequent 36-hour rehydration period were collected. Body weight and WI were subsequently measured every 6 hours during the rehydration period. Total WI during the 4-hour period of 8 AM to noon on day 2 after return of access to water and before administration of the first dose of paste also was recorded.

Furosemide was used 4 times during the study: horses were allowed a minimum of 1 week at pasture between each experiment. After dehydration had been induced during each of the 4 experiments, initial rehydration was accomplished by allowing horses to voluntarily drink from a large water container (plastic garbage can) that was placed in the stall at 8 AM on day 2 (time 0 for rehydration period). After 4 hours of ad libitum access to water, a randomized process was used to select which of 4 treatments would be given to each horse.

The 4 treatments included oral administration of electrolyte paste (0.5 g of KCl/kg, 0.5 g of NaCl/kg, or 0.25 g of NaCl and 0.25 g of KCl/kg) or administration of a suspension that did not contain electrolytes (control treatment). Treatments were divided into 3 equal aliquots of an oral suspension in 100 ml of water (control treatment was three 100-ml doses of water) and were administered at 4-hour intervals (noon, 4 PM, and 8 PM on day 2). To assess potential effects of stimulation of oropharyngeal receptors, electrolyte suspensions were administered by use of oral dosing syringes (60 ml) to 3 horses and via a small-diameter tube passed into the rostral portion of the esophagus in the other 3 horses.

Sample collection and analyses—Blood samples were collected by jugular venipuncture into heparin-coated and dry plastic syringes at the start of each experiment (8 AM on day 1), at the start and finish of the dehydration period (8 PM on day 1 and 8 AM on day 2), and every 6 hours during the 36-hour rehydration period. Each heparinized sample was placed on ice until analysis for pH and bicarbonate and electrolyte (sodium, potassium, and chloride) concentrations within 60 minutes after collection. The remainder of each sample was used for measurement of Hct (using a microhematocrit method), plasma protein (PP) concentration (using refractometry), and plasma osmolality (Ponset; using freezing-point depression). Changes in PP concentration were used to calculate estimated changes in plasma volume (PV), using the following equation:

$$\text{APV} = \left( \frac{\text{PP}_1}{\text{PP}_2} - 1 \right) \times 100$$

where PPpre is the baseline PP concentration, and PPtime is the PP concentration at each of the sample collection points.

Nonheparinized blood samples were transferred to glass tubes. Samples were centrifuged, and serum was harvested and stored at −20 C until measurement of creatinine (Cr) concentration.

A sample of urine produced during each 12-hour collection period was centrifuged (1,500 g for 10 minutes), and the supernatant was removed and stored at −20 C until determination of urine osmolality (Uosmo) and Cr concentration, using the same methods that were used for plasma. Urine electrolyte concentrations were measured with ion-specific electrodes after being diluted 1:2 with assay diluent or 1:10 with saline (0.9% NaCl) solution before measurement of urine sodium or potassium concentrations, respectively. These data were used to calculate endogenous Cr clearance as a measure of glomerular filtration rate (GFR), fractional clearance of sodium (FCrNa), and free water clearance (Cw). These data were used to calculate endogenous Cr clearance as a measure of glomerular filtration rate (GFR), fractional clearance of sodium (FCrNa), and free water clearance (Cw).

Statistical analysis—All values were reported as mean ± SEM. Data were analyzed by use of a 2-way repeated-measures ANOVA1 to assess effects of time and treatment. When F ratios were significant (P < 0.05), a Student-Newman-Keuls post-hoc test was performed to detect specific differences. In some instances (eg, total WI from 4 to 18 hours of the rehydration period), data also were analyzed by use of a 1-way ANOVA to assess effects of treatment. Water intake of the 3 horses administered electrolytes via oral dosing syringes was compared with that of the 3 horses administered electrolytes via the small-diameter tube, using a nonpaired t-test. Finally, Pearson product-moment correlations were performed to examine relationships between selected measurements and WI.

Results

Experiments were successfully completed for all 4 treatments in 5 horses; however, experiments were successfully completed for only 2 treatments in the sixth horse, because it developed signs of recurrent air-
respectively). Feces collected during the 12-hour peri-
dium for KCl, NaCl, NaCl-KCl, and control treatments,
differences were not detected among treatments.

64.1

horses drank similar (

administration of electrolyte pastes were not observed.

sumed the entire amount of pellets fed during the

were included in the statistical analysis. All horses con-

way obstruction, which resulted in an early return to

pasture during experiments involving the other 2 treat-
ments. Data for all successfully completed experiments
were included in the statistical analysis. All horses con-

tained the entire amount of pellets fed during the

experiments, and adverse effects of dehydration or

administration of electrolyte pastes were not observed.

change in body weight, WI, and fecal and urine

production before and during induction of dehydration and during the subsequent 36-
hour rehydration period in 6 horses orally administered electrolyte pastes (KCl, NaCl, and NaCl-KCl) or water (control treatments) at 4, 8, and 12 hours of the rehydration period.

Table 1—Change in body weight and voluntary water intake before and during induction of dehydration and during the subsequent 36-hour rehydration period in 6 horses orally administered electrolyte pastes (KCl, NaCl, and NaCl-KCl) or water (control treatments) at 4, 8, and 12 hours of the rehydration period.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Treatment</th>
<th>Before dehydration</th>
<th>Dehydration</th>
<th>Rehydration (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>0 to 6</td>
<td>6 to 12</td>
</tr>
<tr>
<td>Weight change (kg)</td>
<td>KCl</td>
<td>3.9 ± 1.1</td>
<td>-22.2 ± 1.3</td>
<td>15.9 ± 1.9</td>
</tr>
<tr>
<td></td>
<td>NaCl</td>
<td>2.3 ± 1.1</td>
<td>-19.8 ± 1.9</td>
<td>14.6 ± 1.5</td>
</tr>
<tr>
<td></td>
<td>NaCl-KCl</td>
<td>4.8 ± 1.1</td>
<td>-22.2 ± 0.9</td>
<td>16.1 ± 1.0</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>5.6 ± 1.6</td>
<td>-24.1 ± 1.4</td>
<td>16.1 ± 1.9</td>
</tr>
</tbody>
</table>

Water intake (L)

| KCl | 16.0 ± 1.7 | NA | 20.4 ± 1.9 | 13.0 ± 1.1* | 8.8 ± 0.6* | 4.1 ± 1.2 | 6.8 ± 0.3 | 7.3 ± 1.0 |
| NaCl | 17.2 ± 1.2 | NA | 18.5 ± 1.9 | 14.5 ± 1.4* | 9.8 ± 1.0* | 3.6 ± 0.6 | 7.0 ± 1.3 | 6.2 ± 1.5 |
| NaCl-KCl | 15.2 ± 1.4 | NA | 20.3 ± 1.1 | 14.2 ± 1.3* | 9.5 ± 0.9* | 3.8 ± 0.5 | 6.5 ± 0.4 | 7.3 ± 0.6 |
| Control | 16.2 ± 2.2 | NA | 19.0 ± 1.9 | 7.8 ± 0.8* | 5.8 ± 0.4* | 2.9 ± 0.7 | 7.7 ± 0.7 | 6.9 ± 1.0 |

Values represent mean ± SEM.

*Within a column, values with different superscript letters differ significantly (P < 0.05).

NA = Not applicable.

Table 2—Fecal and urine production before and during induction of dehydration and during the subsequent 36-hour rehydration period in 6 horses orally administered electrolyte pastes (KCl, NaCl, and NaCl-KCl) or water (control treatments) at 4, 8, and 12 hours of the rehydration period.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Treatment</th>
<th>Before dehydration</th>
<th>Dehydration</th>
<th>Rehydration (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>0 to 12</td>
<td>12 to 24</td>
</tr>
<tr>
<td>Feces (kg)</td>
<td>KCl</td>
<td>1.6 ± 0.4</td>
<td>0.9 ± 0.2</td>
<td>2.8 ± 0.7</td>
</tr>
<tr>
<td></td>
<td>NaCl</td>
<td>2.0 ± 0.7</td>
<td>1.2 ± 0.3</td>
<td>1.8 ± 0.1</td>
</tr>
<tr>
<td></td>
<td>NaCl-KCl</td>
<td>2.3 ± 0.4</td>
<td>1.3 ± 0.3</td>
<td>2.0 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>2.6 ± 0.4</td>
<td>1.0 ± 0.2</td>
<td>1.4 ± 0.3</td>
</tr>
<tr>
<td>Urine (L)*</td>
<td>KCl</td>
<td>5.2 ± 1.5</td>
<td>NA</td>
<td>3.6 ± 0.6*</td>
</tr>
<tr>
<td></td>
<td>NaCl</td>
<td>4.2 ± 0.8</td>
<td>NA</td>
<td>2.5 ± 0.6*</td>
</tr>
<tr>
<td></td>
<td>NaCl-KCl</td>
<td>4.0 ± 0.6</td>
<td>NA</td>
<td>4.2 ± 0.3*</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>3.3 ± 0.7</td>
<td>NA</td>
<td>2.3 ± 0.6*</td>
</tr>
</tbody>
</table>

Values represent mean ± SEM.

*Urine production was only measured in 5 horses.

See Table 1 for key.

administration of electrolyte pastes resulted in greater voluntary WI, compared with the control treatment, and it was accompanied by greater recovery of body weight between 6 and 12 hours when NaCl was a component of

Figure 1—Mean ± SEM voluntary water intake from 4 to 18 hours of the rehydration period in horses orally administered electrolyte pastes or water (control treatments). **Values with different letters differ significantly (P < 0.05).
the paste (Fig 1; Table 1). As a consequence, administration of NaCl and NaCl-KCl pastes tended to produce a state of transient hyperhydration after 18 hours of the rehydration period, compared with body weight before dehydration. However, electrolyte administration also resulted in greater urine production, compared with the control treatment, during the final 24 hours of the rehydration period. As a result, body weight at the end of the 36-hour rehydration period was not significantly different from body weight before dehydration for any treatment. Nevertheless, body weight remained 0.7 to 0.8% lower than body weight before dehydration for the control treatment at the end of the rehydration period, but this value did not differ significantly ($P = 0.06$). Water intake by the 3 horses administered treatments via the small-diameter tube was not significantly different from that of the 3 horses administered treatments via oral dosing syringes at any time point, except for 1 (2 PM to 8 PM on day 2; Table 3).

Values for Hct, PP concentration, $P_{osm}$, pH, and bicarbonate concentration were determined (Table 4). On the basis of changes in PP concentration, dehydration produced similar ($P = 0.48$) decreases in estimated PV of $-13.7 \pm 6.0$, $-11.0 \pm 1.0$, $-11.9 \pm 1.3$, and $-11.7 \pm 1.3$% for KCl, NaCl, NaCl-KCl, and control treatments, respectively. After 18 hours of the rehydration period,
treatment with NaCl and NaCl-KCl resulted in significant (P = 0.01) PV expansion (9.3 ± 2.0 and 5.4 ± 2.2%, respectively), compared with a lack of change with KCl or the control treatment (–0.2 ± 1.7 and –1.1 ± 0.8%, respectively). Despite these large changes in estimated PV, significant changes in Fosm were not detected, except for the control treatment, which had a decrease from the value before dehydration until after 18 hours of the rehydration period. Furosemide administration and withholding of water overnight resulted in increases in venous pH and bicarbonate concentration for all treatments, and these increases persisted throughout the rehydration period for the control treatment.

Plasma electrolyte concentrations were determined (Fig 2). Furosemide administration and withholding water overnight produced a decrease in plasma chloride concentration for all treatments, and this change persisted throughout the rehydration period for the control treatment. During the rehydration period, plasma sodium and potassium concentrations changed in a manner consistent with what would be expected for supplementation with sodium and potassium salts (ie, sodium concentration tended to be greater with NaCl and NaCl-KCl treatments, and potassium concentration tended to be greater with KCl and NaCl-KCl treatments). In contrast, sodium concentration remained decreased from the concentration determined before dehydration throughout the entire rehydration period for the control treatment.

Plasma electrolyte concentrations were determined (Fig 2). Furosemide administration and withholding water overnight resulted in increases in venous pH and bicarbonate concentration for all treatments, and these increases persisted throughout the rehydration period for the control treatment.

Urine variables were calculated (Fig 3; Table 5). Urine flow and GFR were lower for the NaCl and control treatments, compared with KCl and NaCl-KCl treatments, during the first 12 hours of the rehydration period. Subsequently, urine flow increased from 12 to 36 hours of the rehydration period for the NaCl and NaCl-KCl treatments. These increases in urine flow were accompanied by a return of GFR to values obtained before dehydration. In contrast, GFR remained decreased throughout the rehydration period for the control treatment. Despite induction of dehydration, Uosm tended to decrease during the initial 12 hours of the rehydration period for all treatments and remained decreased from values obtained before dehydration for the NaCl and control treatments throughout the rehydration period. Similarly, Cposm tended to decrease, and C\text{\textsubscript{H}2O} tended to increase, during the ini-
Table 5—Mean ± SEM values for urine flow, urine osmolality (Uosm), urine concentrations of sodium and potassium, fractional clearance of sodium (FClNa), and fractional clearance of potassium (FClK) before induction of dehydration and during the subsequent 36-hour rehydration period in 5 horses orally administered electrolyte pastes (KCl, NaCl, or NaCl-KCl) or water (control treatments) at 4, 8, and 12 hours of the rehydration period.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Treatment</th>
<th>Before dehydration</th>
<th>Rehydration (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0 to 12</td>
<td>12 to 24</td>
</tr>
<tr>
<td>Urine flow (ml/min)</td>
<td>KCl</td>
<td>7.3 ± 2.1</td>
<td>5.0 ± 0.8**</td>
</tr>
<tr>
<td></td>
<td>NaCl</td>
<td>5.8 ± 1.1</td>
<td>3.5 ± 0.6**</td>
</tr>
<tr>
<td></td>
<td>NaCl-KCl</td>
<td>5.6 ± 0.8</td>
<td>5.9 ± 0.5*</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>4.5 ± 1.0</td>
<td>3.2 ± 0.8*</td>
</tr>
<tr>
<td>Urine osmolality (mOsm/kg)</td>
<td>KCl</td>
<td>836 ± 194</td>
<td>682 ± 82</td>
</tr>
<tr>
<td></td>
<td>NaCl</td>
<td>925 ± 118</td>
<td>623 ± 49*</td>
</tr>
<tr>
<td></td>
<td>NaCl-KCl</td>
<td>462 ± 122</td>
<td>658 ± 56</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>1003 ± 105</td>
<td>613 ± 71*</td>
</tr>
<tr>
<td>Urine sodium (mmol/L)</td>
<td>KCl</td>
<td>38.0 ± 14.3</td>
<td>11.8 ± 6.1</td>
</tr>
<tr>
<td></td>
<td>NaCl</td>
<td>13.1 ± 6.4</td>
<td>18.7 ± 12.2</td>
</tr>
<tr>
<td></td>
<td>NaCl-KCl</td>
<td>28.0 ± 12.0</td>
<td>12.6 ± 5.7</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>26.0 ± 12.8</td>
<td>12.2 ± 8.4</td>
</tr>
<tr>
<td>Fractional clearance (Na)</td>
<td>KCl</td>
<td>0.270 ± 0.100</td>
<td>0.067 ± 0.037</td>
</tr>
<tr>
<td></td>
<td>NaCl</td>
<td>0.080 ± 0.040</td>
<td>0.102 ± 0.080</td>
</tr>
<tr>
<td></td>
<td>NaCl-KCl</td>
<td>0.142 ± 0.071</td>
<td>0.110 ± 0.093</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>0.102 ± 0.081</td>
<td>0.122 ± 0.093</td>
</tr>
<tr>
<td>Urine potassium (mmol/L)</td>
<td>KCl</td>
<td>143.0 ± 34.3</td>
<td>135.1 ± 19.2</td>
</tr>
<tr>
<td></td>
<td>NaCl</td>
<td>185.0 ± 22.9</td>
<td>91.5 ± 11.0*</td>
</tr>
<tr>
<td></td>
<td>NaCl-KCl</td>
<td>165.6 ± 38.8</td>
<td>117.9 ± 10.8</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>172.3 ± 26.4</td>
<td>96.8 ± 11.4*</td>
</tr>
<tr>
<td>Fractional clearance (K)</td>
<td>KCl</td>
<td>37.3 ± 6.7</td>
<td>26.9 ± 4.4</td>
</tr>
<tr>
<td></td>
<td>NaCl</td>
<td>37.0 ± 5.0</td>
<td>21.8 ± 3.9*</td>
</tr>
<tr>
<td></td>
<td>NaCl-KCl</td>
<td>38.5 ± 5.3</td>
<td>34.6 ± 4.3</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>34.3 ± 6.6</td>
<td>18.7 ± 2.8*</td>
</tr>
</tbody>
</table>

See Table 4 for key.

Figure 4—Correlation between voluntary water intake and change in body weight (BW) from 0 to 4 hours of the rehydration period (top) and between voluntary water intake from 6 to 24 hours of the rehydration period and the change in plasma sodium concentration from 12 to 24 hours of the rehydration period (bottom) for horses administered electrolyte pastes or water (control treatments). Each symbol represents results for 1 horse. See Figure 2 for key.

Discussion

Results of the study reported here, similar to those of Sosa Leon et al.,12 documented that oral administration of electrolyte pastes to dehydrated horses increased voluntary WI and improved rehydration during the rehydration period. The horses in that other study12 received approximately 0.35 g of electrolytes/kg and remained dehydrated (approx 2%) at the end of a 6-hour rehydration period, which was the end of their experiment. In contrast, our horses treated with NaCl and NaCl-KCl pastes had a transient period of hyperhydration and 12 hours of the rehydration period for all treatments. However, these changes persisted throughout the rehydration period only for the control treatment.

Urine sodium concentration and FClNa decreased during the final 24 hours of the rehydration period for the KCl and control treatments and increased for the NaCl treatment (Table 5). Urine potassium concentration and FClK tended to decrease during the initial 12 hours of the rehydration period for all treatments; significant decreases in urine potassium concentration and FClK were evident for the NaCl and control treatments. Although urine potassium concentration remained decreased throughout the rehydration period for the NaCl and control treatments, FClK remained decreased for the control treatment alone. In contrast, FClK increased from 12 to 36 hours of the rehydration period for the NaCl-KCl treatment.

Water intake from 8 AM to noon on day 2 (after dehydration but before electrolyte administration) was significantly correlated (r = -0.55; P = 0.008) with loss of body weight, but it was not significantly correlated (r = 0.07; P = 0.43) with the small increase in sodium concentration that accompanied dehydration (Fig 4). However, WI from 6 to 24 hours of the rehydration period was significantly correlated (r = 0.70; P = 0.001) with the increase in sodium concentration from 12 to 24 hours of the rehydration period.
apparent PV expansion that lasted for approximately 12 hours after administration of the last dose of electrolytes. Although excess electrolytes were subsequently eliminated through increased urinary excretion, they would likely have been of benefit to horses afflicted with diseases that produce ongoing fluid losses (eg, diarrhea).

Each dose of electrolytes administered consisted of 1,000 to 1,300 mmol of cation and 1,000 to 1,300 mmol of chloride in 100 ml of water. For the 3 doses, 200 to 250 g of electrolytes (3,000 to 4,000 mmol of cation and 3,000 to 4,000 mmol of chloride) was administered during an 8-hour period. Compared with the control treatment, administration of KCl was accompanied by additional WI of approximately 8 L between 4 and 18 hours of the rehydration period. Compared with the control treatment, administration of KCl was accompanied by additional WI of approximately 8 L between 4 and 18 hours of the rehydration period (Fig 1). The combined result was addition of approximately 8 L of a 2- to 3-times-normal KCl solution (approx 750 mOsm/kg) to the body fluids of the horses (comparable in electrolyte content to approx 20 L of an isotonic KCl solution). However, during the rehydration period, KCl-treated horses excreted approximately 4 L more urine, which contained essentially the same amount of sodium but approximately 1,000 mmol more potassium than for the control treatment. Administration of NaCl or NaCl-KCl was accompanied by additional WI of approximately 13 L between 4 and 18 hours of the rehydration period and addition of approximately 13 L of a twice-normal solution (approx 600 mOsm/kg) to body fluids of the horses (comparable to approx 25 L of isotonic fluid). During the rehydration period, horses that received NaCl excreted approximately 7 L more urine that contained approximately 700 more mmol of sodium and 850 more mmol of potassium, compared with the control treatment. Horses that received NaCl-KCl also excreted approximately 7 L more urine that contained approximately 250 more mmol of sodium and 1,500 more mmol of potassium, compared with the control treatment. Thus, the net effect for all electrolyte treatments was retention of 4 to 6 L of water and 1,500 to 2,000 mmol of cation (comparable in electrolyte content to 10 to 15 L of isotonic fluids). However, retention of water and electrolytes was greater when NaCl was a component of the paste.

In the study by Sosa León et al,12 administration of furosemide (1 mg/kg, IM) produced a loss of body weight of approximately 4%, and urine electrolyte losses were approximately 1,750, 570, and 2,150 mmol for sodium, potassium, and chloride, respectively. Assuming similar furosemide-induced urinary electrolyte losses in the horses of our study, the NaCl-KCl treatment would have replaced these losses almost completely, although a deficit of sodium of approximately 300 mmol would have remained ([10.25 mg/kg X 300 kg X 17.1 mmol of sodium/g] -700 mmol of urinary sodium loss = 1,440 mmol retained). Administration of KCl alone adequately replaced the potassium and chloride losses but failed to address the sodium deficit. In contrast, although administration of NaCl alone adequately replaced the sodium and chloride losses, this treatment failed to address the potassium deficit and also exacerbated it by promoting additional urinary excretion of approximately 850 mmol of potassium. These varying effects of the composition of the electrolyte pastes on urinary electrolyte excretion illustrate the importance of considering relative fluid losses from extracellular (principal cation, sodium) and intracellular (principal cation, potassium) stores when developing a plan for fluid therapy of dehydrated horses.13

Although not accompanied by a significant increase in serum Cr concentration (data not shown), furosemide administration and withholding water overnight resulted in a mild decrease in renal function (ie, GFR) from 0 to 12 hours of the rehydration period. The decrease in GFR was likely a consequence of hypovolemia and decreased renal perfusion. Administration of all electrolyte pastes, but not the control treatment, corrected the decrease in GFR from 12 to 36 hours of the rehydration period. Of interest, Uosm from 0 to 12 hours of the rehydration period was lower for all treatments. Although an increase in Uosm to near maximal values may be expected in the face of hypovolemia, ingestion of large volumes of water from 0 to 4 hours of the rehydration period would have led to rapid decreases in plasma tonicity and plasma vasopressin concentration. From 12 to 36 hours of the rehydration period, Uosm remained essentially unchanged despite increases in urine flow for all electrolyte pastes. Excretion of an osmotic load, as evidenced by a greater Cosem, typically produces an increase in urine flow that is greater than the increase in Uosm. In a clinical setting, treatment of anorectic horses with enteral or IV administration of solutions containing sodium as the major cation can similarly increase urine flow and Cosem. Without adequate replacement of potassium, total body depletion of potassium can actually be exacerbated because of the obligate loss of potassium in the urine (values for FClK are rarely < 15% because of the poorer renal reabsorption of potassium, compared with renal absorption of sodium, which was observed for the control treatment).

Administration of 0.25 g of NaCl-KCl/kg should have adequately replaced all furosemide-induced urinary electrolyte losses; however, the increase in urinary excretion of sodium prevented complete correction of the sodium loss. Although the increase in urinary excretion of sodium could be considered an inappropriate renal response, the aforementioned renal functional responses to dehydration and rehydration were appropriate. An alternate explanation may be that initial replacement of furosemide-induced losses of fluid and electrolytes was by translocation of fluid and electrolytes from transcellular fluid (within the lumen of the large intestine) to the interstitial and plasma spaces (movement of fluid within compartments of the extracellular fluid). In horses fed a hay diet, ingesta in the gastrointestinal tract accounts for approximately 13% of body weight, and the large intestine has been estimated to contain a reservoir of approximately 15 L of water that contains 2,000 to 2,500 mmol of sodium, potassium, and chloride, which can be used to replace losses of electrolytes in other compartments of body fluid.8,9 The diet change from pasture to a pelleted
feed in the horses reported here resulted in a mean loss of 16.5 kg and a decrease in fecal water content, compared with a value of approximately 75% for horses fed hay. A decrease in large-intestine contents was likely responsible for > 90% of this loss of body weight; nevertheless, the increase in urinary excretion of sodium for the NaCl-KCl treatment suggests that our horses may still have had a reservoir of water and electrolytes that was still considerable. In another study,26 we reported a similar increase in urinary excretion of sodium during the rehydration period. In another study,26 we reported a similar increase in urinary excretion of sodium during the rehydration period after horses that were involved in endurance exercise were orally administered electrolytes in an amount estimated to replace electrolytes lost through sweating. Although absorption of water and electrolytes from intestinal contents was also a potentially important factor in the study, it warrants mention that neuroendocrine responses, including increased intrarenal production of prostaglandins during renal hypoperfusion and release of atrial natriuretic peptides with PV expansion, may also have contributed to the increase in urinary excretion of sodium during the rehydration period.22,23 Finally, although not a factor in anorectic patients, feed intake by our horses provided another source of electrolytes. For every 24-hour period, our horses consumed 684 mmol of sodium and chloride (0.5% NaCl = 40 g in 8 kg of pellets) and 1,390 mmol of potassium (1.3% K = 104 g in 8 kg of pellets), which may also have contributed to the increased urinary excretion of electrolytes during the rehydration period.

All of the electrolyte pastes administered to the horses in the study reported here were hypertonic. However, adverse treatment effects were not observed. In fact, PP concentrations decreased for all electrolyte treatments during the initial 24 hours of the rehydration period. Thus, movement of water from plasma or interstitial fluid into the gastrointestinal tract would have been transient and was not apparent in our 6-hour blood collections. Although it has been proposed that gastric emptying rate (GER) for liquids in humans is slowed with increasing tonicity of the rehydration solution, it is now recognized that GER is primarily influenced by the volume of fluid in the stomach and its energy density (carbohydrate content).23 There has been little study of the effects of fluid tonicity on GER in horses. In 1 study,23 there was not a difference in GER when 8 L of isotonic or hypertonic solutions (628 mOsm/kg) were administered via a nasogastric tube. In fact, 90% of the fluid administered was emptied from the stomach within 15 minutes after administration, regardless of the composition of the rehydration fluid. It is important to reiterate that the amount of electrolytes administered to our horses during an 8-hour period was substantial (200 to 250 g). Despite these high doses, the only evidence supporting a potential overdose was the increase in urinary excretion of electrolytes. Thus, as long as renal function remains adequate and water is provided, there appears to be little risk associated with oral administration of large amounts of electrolytes to dehydrated horses.

Increases in plasma tonicity and hypovolemia are recognized stimuli of thirst in mammals. Stimulation of drinking is associated with activation of systemic and central renin-angiotensin systems.28 Studies26,27 of healthy ponies and donkeys have revealed that an increase of approximately 3% in plasma tonicity produced by water deprivation or IV administration of hypertonic saline solution is required to stimulate drinking. In ponies and donkeys, furosemide administration that induced hypovolemia also resulted in voluntary WI without an increase in plasma tonicity. However, similar to the findings in our horses from 0 to 4 hours of the rehydration period, the volume of urine imbibed was only about two-thirds of the volume of urine produced, leading to incomplete restoration of body weight. Intravenous infusion of angiotensin II can induce drinking in horses, but supraphysiologic plasma concentrations of angiotensin II must be produced before thirst is stimulated.28

To examine factors that may have stimulated thirst during dehydration and rehydration in the horses reported here, changes in body weight (reflecting the magnitude of water loss) and plasma tonicity (plasma sodium concentration) were compared with WI after dehydration and after oral administration of electrolyte pastes. Because furosemide administration and withholding of water overnight caused loss of water and electrolytes, only a modest increase in plasma sodium concentration was produced (approx 2 mmol/L [1.5%]). In contrast, loss of body weight was 3.4 to 6.1% and was accompanied by estimated decreases in PV of 7 to 16%. Thus, hypovolemia appeared to be the most important stimulus for thirst in our model of dehydration (Fig 4). In contrast, in the face of an increase in body weight for all treatments, WI after administration of electrolyte pastes was strongly correlated with the increase in plasma sodium concentration from 12 to 24 hours of the rehydration period (period of greatest change in response to electrolyte administration). Thus, the increase in plasma sodium concentration was likely an important stimulus for thirst during this time period when transient hyperhydration and PV expansion were produced.

A final factor that may have affected WI could have been stimulation of oropharyngeal receptors by administration of the concentrated electrolyte pastes. In fact, the 3 horses given the electrolyte paste through a small-diameter tube passed into the rostral portion of the esophagus drank 3 to 4 L more water from 2 PM to 8 PM on day 2. Although not determined in this study, this finding could have been attributed to partial loss of the electrolytes administered via a dosing syringe (spillage from the mouth) or a negative effect of the concentrated electrolytes on voluntary WI as a result of a salty taste. Although individual responses were highly variable, Randall et al23 reported that healthy weanling foals were equally willing to drink tap water or a dilute (≤ 0.6%) NaCl solution. However, as the concentration of the NaCl solution was increased to 5%, a strong rejection response was observed. These findings would support our clinical observations after oral administration of electrolyte pastes to research horses and dehydrated patients. Concentrated electrolyte pastes are not always accepted and may delay interest in eating and drinking for a few minutes after administration. Although this initial response could be consid-
ered an adverse effect, it has generally been transient (< 5 minutes) and is more than offset by a greater voluntary WI during the next few hours.

Although we acknowledge the fact that use of enteral administration of fluids and electrolytes for correction of mild to moderate dehydration in horses is not a new concept, most reports have described repeated administration of isotonic electrolyte solutions via a nasogastric tube. Repeated administration requires multiple daily farm visits by a veterinarian, client education on the use of an indwelling nasogastric tube, or hospitalization of a patient. Although frequent reassessment by a veterinarian is indicated for horses being treated for disorders that initially produce severe depletion of fluid and electrolytes, our findings are similar to those of Sosa León et al and support the contention that mild to moderate dehydration accompanying less serious medical disorders could be corrected by oral administration of electrolyte pastes by clients, providing horses are observed to ensure that they drink water. In addition, hospitalized horses receiving continuous IV infusion of fluids can also benefit from concurrent oral administration of electrolyte pastes. The latter may provide electrolytes that may not have been adequately replaced by IV administration of fluids (ie, KCl or KHCO3) and could allow decreases in the rate of administration as well as duration of IV administration of fluids.

1. Hurley Chow No. 100, Purina Mills Inc, St Louis, Mo.
2. Furosemide injection 5%, Hoechst Roussel Vet, Warren, NJ.
4. Procedure No. 555-A, Sigma Diagnostics Inc, St Louis, Mo.

References