Effects of oral potassium supplementation on acid-base status and plasma ion concentrations of horses during endurance exercise

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Objective—To compare effects of oral supplementation with an experimental potassium-free sodium-abundant electrolyte mixture (EM-K) with that of oral supplementation with commercial potassium-rich mixtures (EM+K) on acid-base status and plasma ion concentrations in horses during an 80-km endurance ride.

Animals—46 healthy horses.

Procedure—Blood samples were collected before the ride; at 21-, 37-, 56-, and 80-km inspection points; and during recovery (ie, 30-minute period after the ride). Consumed electrolytes were recorded. Blood was analyzed for pH, Pv_{CO_2}, and Hct, and plasma was analyzed for Na^+, K^+, Cl^-, Ca^{2+}, Mg^{2+}, lactate, albumin, phosphate, and total protein concentrations. Plasma concentrations of H^+ and $HCO3^-$, the strong ion difference (SID), and osmolarity were calculated.

Results—34 (17 EM-K and 17 EM+K treated) horses finished the ride. Potassium intake was 33 g less and Na^+ intake was 36 g greater for EM-K-treated horses, compared with EM+K-treated horses. With increasing distance, plasma osmolarity; H^+, Na^+, K^+, Mg^{2+}, phosphate, lactate, total protein, and albumin concentrations; and Pv_{CO_2} and Hct were increased in all horses. Plasma $HCO3^-$, Ca^{2+}, and Cl^- concentrations were decreased. Plasma H^+ concentration was significantly lower in EM-K-treated horses, compared with EM+K-treated horses. Plasma K^+ concentrations at the 80-km inspection point and during recovery were significantly less in EM-K-treated horses, compared with EM+K-treated horses.

Conclusions and Clinical Relevance—Increases in plasma H^+ and K^+ concentrations in this endurance ride were moderate and unlikely to contribute to signs of muscle fatigue and hyperexcitability in horses. (*Am J Vet Res* 2005;66:466–473)

Electrolyte and water losses occur during prolonged exercise in horses and have been implicated in medical problems.[1,2] Many clinical signs of these problems are detected at veterinary inspection points during endurance rides and have been ascribed to an increase in neuromuscular excitability in striated and smooth muscles.[1-6] Such signs may include cardiac arrhythmias, a slower decrease in heart rate following exercise, and muscle cramps and twitches as well as an increased or decreased intestinal motility.

Neuromuscular excitability depends on **resting membrane potential (E_K)** and threshold potential. The E_K is mainly determined by the K^+ distribution across the cell membrane according to the Nernst equation[7] so that when plasma K^+ concentration increases, E_K will increase (become less negative) and more closely approach the threshold for an action potential, thereby increasing neuromuscular excitability. Plasma K^+ concentration increases in accordance with the intensity of exercise at speeds above 4 m/s in horses.[8,9]

We propose that the oral supplementation with potassium-free electrolyte during endurance exercise may moderate the increase in plasma K^+ concentration and thereby reduce the risk of increasing neuromuscular excitability. Secondarily, K is largely replaced by Na to maintain the cation equivalents in designing a potassium-free formula, and this exchange may affect the plasma H^+ concentration response to exercise. The purpose of the study reported here was to compare effects of oral supplementation with an experimental **potassium-free sodium-abundant electrolyte mixture (EM-K)** with that of oral supplementation with commercial **potassium-rich mixtures (EM+K)** on acid-base status and plasma ion concentrations in horses during an 80-km endurance ride. A companion study on antioxidant status was also conducted in this ride, and preliminary reports[a,b] have been presented.

Materials and Methods

Study conditions and design—This study was undertaken during the Middleburg Research Ride 2001, which was held on April 1 and followed American Endurance Ride

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Conference rules.[6] The ride started at 7 AM and covered 80 km of rolling hills with altitudes varying from 121 to 442 m. Rest stops were provided at 21, 37, 56, and 72 km at which time veterinary inspections of horses were performed.[6] The trail between 2 rest stops is called a loop, and this trail consisted of 5 loops. Ambient temperature ranged from 4.7° to 10.7°C and humidity from 97% to 100%. A 2 × 2 factorial design was used to compare 2 vitamin treatments (ie, vitamin E alone or with vitamin C)[a] and to compare 2 electrolyte treatments (ie, EM-K- and EM+K-treated horses). The institutional animal care and use committee approved the protocol.

Horses—Forty-six horses (45 Arabians, purebred or crossbred, and 1 Thoroughbred) with a mean age of 10.8 ± 0.6 years were included in this study. A preride survey provided a nutritional and performance history of each horse. Owners of 22 horses required use of their customary commercial electrolyte supplements containing K (ie, EM+K), and owners of 24 horses volunteered to use the novel experimental potassium-free mixture (ie, EM-K).

Electrolyte mixtures were administered orally by syringe. Riders that used EM-K were instructed to supply 2 doses/loop. Riders that used EM+K followed manufacturer's directions, which were usually 1 to 3 doses/loop. One or 2 doses were also given 2 hours before the ride started. The formula of EM-K is based on sweat composition study results,[9] except for the absence of K, and approximates the electrolytes lost in 2.5 L of sweat. Each dose consisted of 21.8 g of NaCl, 1.7 g of $CaCl_2$, 1.1 g of $MgCl_2$, and 0.13 g of NaH_2PO_4. The commercial formulas (ie, EM+K) all contained K.[c-f] Intakes of electrolytes during the ride were calculated from the formulas and amounts given, as recorded by the riders. Horses received water but no electrolytes or food from the 80-km inspection point until blood sample collection after the ride (ie, during recovery).

Horses were weighed without tack before the start of the ride, after the veterinary inspection at 56 km, and after the ride finish at 80 km (before access to water) by use of an electronic scale.[g] Horses underwent American Endurance Ride Conference regulation veterinary inspections prior to the ride, at rest stops, and at the finish.[6] Heart rate and rectal temperature were recorded immediately before blood sample collection.

Blood—Blood samples were collected via jugular venipuncture at 6 AM (before the ride); 60 to 120 seconds after arrival at 21-, 37-, 56-, and 80-km veterinary inspection points; and during recovery (20 to 30 minutes after finishing the ride). An aliquot of blood was collected into a blood gas syringe[h] and another was collected to heparinized evacuated tubes.[i] Blood was kept in ice water and the evacuated tube centrifuged 20 to 30 minutes after collection. The gas syringe aliquot was analyzed 20 to 30 minutes after collection for blood pH, $PvCO_2$, and Hct and plasma Na^+, K^+, Cl^-, Ca^{2+}, and Mg^{2+} concentrations by use of a blood-gas analyzer.[j] Within sample coefficient of variation was < 2% for all variables analyzed by the blood gas instrument, except for Hct, which had a coefficient of variation of 5%. In addition, plasma H^+ and $HCO3^-$ concentrations and osmolality were calculated.[j] Plasma was stored at –80°C for further analysis. Spectrophotometric assays were used to determine plasma lactate, albumin, phosphate, and total protein concentrations within 2 weeks of the ride.[k]

Acid-base status—Changes in acid-base status during exercise have been analyzed by use of the comprehensive physicochemical model of Stewart.[10-15] In this model,[10] concentrations of H^+ and $HCO3^-$ are dependent upon the **strong ion difference (SID)**, $PvCO_2$, and **total weak acids** (A_{tot}; mainly proteinates and phosphates). The SID was calculated as the algebraic sum of Na^+ and K^+ concentrations minus the sum of Cl^- and lactate concentrations; the algebraic sum of other strong ions was assumed to be < 1 mEq/L and to contribute negligibly to changes in the SID observed during exercise and recovery. Estimates of A_{tot} (mEq/L) were predicted from multiplying the total protein (g/L) concentration by 0.211.[15]

The partition of changes in plasma H^+ concentration into contributions from the SID, $PvCO_2$, and A_{tot} was performed by use of software designed for the Stewart system.[14] Resting values for the SID, $PvCO_2$, and A_{tot} were set in the panel for independent variables, and the predicted value of H^+ concentration was observed in the panel for dependent variables. Then the series of values of the SID at each sample collection stage were entered one at a time (without changing $PvCO_2$ and A_{tot}) to yield corresponding series of changes in H^+ concentration contributed by the changes in the SID.[13,14] The procedure was repeated for $PvCO_2$ (without changing the SID and A_{tot}) and for A_{tot} (without changing the SID and $PvCO_2$).

Calculated E_K of muscle cells—The E_K (mV) was calculated from **extracellular K^+ concentration (K_o)** and **intracellular K^+ concentration (K_i)** by use of the Nernst equation as follows[7]:

$$E_K = 61.5 \log [K_o]/[K_i] \text{ at } 37°C.$$

This equation uses the ratio of extracellular-to-intracellular concentrations of only K^+ because the permeability constant of K^+ is much higher than those of other ions. Assumptions are needed to test the possible effects of changes in plasma K^+ concentration on E_K. Measured plasma K^+ concentrations were used for K_o without the approximately equal and opposing adjustments for plasma solids and for lymph. For K_i, a mean equine middle gluteal muscle K_i of 124 mEq/L was calculated from date on equine muscular K (μM/g of wet wt) and water content (%).[16]

Data analysis—Data were summarized as least squares means (± SE). Effects of sample collection stage (before the ride; at 21-, 56-, and 80-km inspection points; and during recovery), treatments (EM-K, EM+K, vitamin E, and vitamin E with vitamin C), and their interactions were evaluated by use of an ANOVA with repeated measures in a mixed model and applied to the 34 horses that completed the 80-km ride.[k] Nonsignificant interactions were eliminated from the model, and treatments with vitamin E and vitamin E with vitamin C were eliminated because they had no interactions with EM-K and EM+K. Significance of differences between means were tested by least significant differences covered by a significant F test for the ANOVA.[l] A Fisher exact test was used to compare frequencies of EM-K- and EM+K-treated horses that did not finish the ride. For estimates of outliers, a Z value was used to denote the normal SD of the probability value.[17] Simple relationships of plasma H^+ concentration to the SID, A_{tot}, and $PvCO_2$ and that of the SID to plasma Na^+, K^+, Cl^-, and lactate concentrations were tested by linear regression.[l] Values of $P < 0.05$ were considered significant.

Results

Mean body weight was 421.0 ± 4.9 kg before the ride for 46 horses and 423.8 ± 5.1 kg for 34 horses that finished the ride. No significant differences were found in mean weight loss between EM-K- and EM+K-treated horses, which was greatest at the 56-km inspection point as follows: 4.52% for 17 EM-K-treated horses that finished the ride, 4.36% for 17 EM+K-treated horses that finished the ride, and 4.80% for 9 of 12 horses that did not finish the ride.

The 12 horses that did not finish the ride comprised 7 of 24 EM-K-treated horses and 5 of 22 EM+K-

treated horses. Number of horses not finishing the ride was not significantly ($P = 0.74$) different between EM-K- and EM+K-treated horses. Reasons for elimination were lameness (3 horses), exertional rhabdomyolysis (2), failure to recover a heart rate of 64 beats per minutes within 30 minutes (1), a decrease in gastrointestinal tract sounds (1), and rider option (5).

Mean speed was 3.30 ± 0.08 m/s over the distance of 80 km, with mean speeds of 3.87, 3.34, 3.29, 2.84, and 2.72 m/s during the 5 loops, respectively. Mean heart rates were 44 ± 2 beats/min, 72 ± 2 beats/min, 66 ± 2 beats/min, 70 ± 2 beats/min, 78 ± 2 beats/min, and 52 ± 2 beats/min before the ride; at the 21-, 37-, 56-, and 80-km inspection points; and during recovery, respectively. Mean rectal temperatures were 36.50°, 38.66°, 38.31°, 38.51°, and 38.72°C before the ride; at the 21-, 37-, 56-, and 80-km inspection points; and during recovery, respectively. Consumption of K^+ was zero grams in EM-K-treated horses and 33 g in EM+K-treated horses (**Table 1**). Consumption of Na was 26 g higher in EM-K-treated horses, compared with EM+K-treated horses.

Sample collection stage—All dependent variables changed during the ride, that is, with sample collection stage. No significant differences in variable measurements were found between EM-K- and EM+K-treated horses, except for plasma H^+ concentration; therefore, data were combined (**Table 2**). With increasing distance, increases were found in plasma osmolarity; Mg^{2+}, total protein, and albumin concentrations; and Hct and $PvCO_2$ and decreases were found in plasma $HCO3^-$, Ca^{2+}, and Cl^- concentrations.

Plasma K^+ concentration significantly ($P < 0.001$) increased from before the ride to the 56-km inspection point and significantly ($P < 0.001$) decreased from the 56-km inspection point to recovery. Plasma Na^+ concentration significantly ($P < 0.001$) increased from before the ride to the 37- and 56-km inspection points and significantly ($P < 0.001$) decreased from the 56-km inspection point to recovery. Plasma Cl^- concentration significantly ($P < 0.001$) decreased from before the ride to the 21-km inspection point and returned to preride values at the 37-km inspection point and thereafter. Plasma lactate concentration significantly ($P < 0.001$)

Table 1—Oral intake of electrolytes by EM-K- and EM+K-treated horses during the 80-km endurance ride.

	EM-K treated (n = 17)		EM+K treated (17)	
Oral electrolyte supplementation	**Mean**	**90% CI**	**Mean**	**90% CI**
K (g)	0	0	33.3	27.6, 39.1
Na (g)	64.3	57.8, 70.7	38.2	31.9, 44.5
Ca (g)	2.3	2.1, 2.6	8.0	5.8, 10.2
Mg (g)	0.9	0.9, 1.0	4.0	1.4, 6.7
Cl (g)	105.5	94.9, 116	98.9	87.2, 110.5
PO_4 (g)	0.21	0.19, 0.24	0	0

EM-K = An experimental potassium-free sodium-abundant electrolyte mixture. EM+K = Commercial potassium-rich mixtures. CI = Confidence interval.

Table 2—Mean and mean SE (MSE) values of plasma variables measured in 34 horses before, during, and after an 80-km endurance ride.

	Blood sample collection stages						
Variables	**PRE**	**21 km**	**37 km**	**56 km**	**80 km**	**REC**	**MSE (±)**
Hct (%)	39[a]	47[b]	47[b]	47[b]	46[b]	36[c]	± 0.83
Total protein (g/dL)	6.73[a]	7.21[b]	7.25[b]	7.09[b,c]	7.05[c]	6.95[c]	± 0.11
Albumin (g/dL)	3.5[a]	3.8[b]	3.8[b]	3.8[b]	3.8[b]	3.6[c]	± 0.05
Lactate (mmol/L)	0.70[a]	2.64[b]	1.13[c]	1.38[c]	1.53[c]	1.46[c]	± 0.15
Na^+ (mmol/L)	145[a,c]	146[a]	149[b]	148[b]	145[a,c]	142[d]	± 0.5
K^+ (mmol/L)	4.01[a]	4.13[b]	4.22[b]	4.42[c]	4.10[a]	3.61[d]	± 0.07
Cl^- (mmol/L)	104.4[a]	102.4[b]	103.6[a]	104.0[a]	103.9[a]	103.8[a]	± 0.50
Ca^{2+} (mmol/L)	1.53[a]	1.44[b]	1.43[b]	1.46[b]	1.35[c]	1.43[b,d]	± 0.01
Mg^{2+} (mmol/L)	0.35[a,b]	0.34[a]	0.36[b]	0.37[b,c]	0.40[d]	0.40[d]	± 0.008
PO_4^- (mmol/L)	2.56[a]	2.31[b]	2.09[c]	2.71[a,d]	3.56[e]	2.57[a,d]	± 0.12
pH	7.416[a]	7.450[b]	7.443[b]	7.432[c]	7.396[d]	7.381[d]	± 0.004
$Pvco_2$ (mmHg)	48.44[a]	45.42[b]	49.48[a]	48.25[a]	49.20[a]	49.56[a]	± 0.40
HCO_3^- (mmol/L)	31.23[a]	31.49[a,b]	33.51[c]	32.28[b,d]	30.46[a,e]	29.39[e]	± 0.41
H^+ (nEq/L)	38.43[a]	35.55[b]	36.10[b]	37.92[c]	40.60[d]	41.31[d]	± 0.34
SID (mEq/L)	44.55[a,c]	45.72[a]	48.92[b]	47.71[b]	43.41[c]	41.28[d]	± 0.46
Osmolarity (mOsm/kg)	290.63[a]	294.82[b]	299.95[c]	297.64[d]	291.19[a,e]	288.06[f]	± 0.90
A_{tot} (mEq/L)	14.17[a]	15.09[b]	15.24[b]	14.93[b,c]	14.73[c]	14.65[c]	± 0.10

[a-f]Mean values with different superscript letters differ significantly ($P < 0.05$) from each other for a given variable.
PRE = Before the 80-km ride. During = At the 21-, 37-, 56-, and 80-km inspection points during the 80-km ride. REC = During recovery from the 80-km ride. nEq/L = Nanoequivalent per liter. SID = Strong ion difference. A_{tot} = Total weak acids.

increased 4-fold by the 21-km inspection point and then significantly ($P < 0.001$) decreased by the 37-km inspection point and remained approximately twice the preride value until recovery. Plasma H^+ concentration significantly ($P < 0.001$) decreased from before the ride to the 21-km inspection point, then significantly ($P < 0.001$) increased from the 21-km inspection point to the 80-km inspection point and changed little ($P < 0.068$) during the 30-minute recovery period.

Treatment—Plasma H^+ concentration was significantly ($P = 0.024$) lower in EM-K-treated horses, compared with EM+K-treated horses (**Figure 1**). Plasma H^+ concentration was lower specifically at the 80-km inspection point ($P = 0.022$) and during recovery ($P = 0.013$) in EM-K-treated horses, compared with EM+K-treated horses.

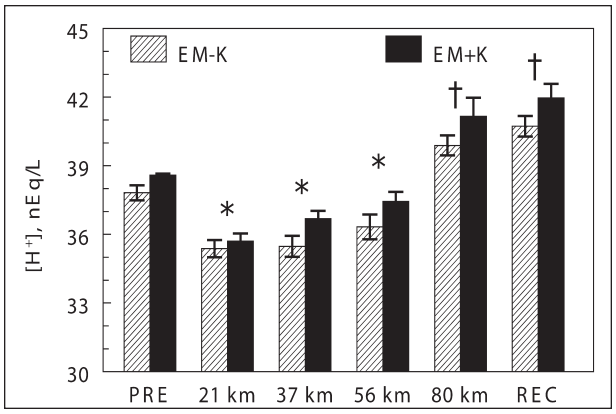

Figure 1—Mean (± SE) plasma H^+ concentrations versus stage of sample collection before (PRE), during (at the 21-, 37-, 56-, and 80-km inspection points), and after (during recovery [REC]) the 80-km endurance ride in horses treated orally with an experimental potassium-free sodium-abundant electrolyte mixture (EM-K; n = 17) or commercial potassium-rich mixtures (EM+K; 17). Notice the overall ($P < 0.05$) differences between EM-K- and EM+K-treated horses for all stages of sample collection. *Significant ($P < 0.05$) decrease from PRE values for all (EM-K- and EM+K-treated) horses. †Significant ($P < 0.05$) increase from PRE values for all horses.

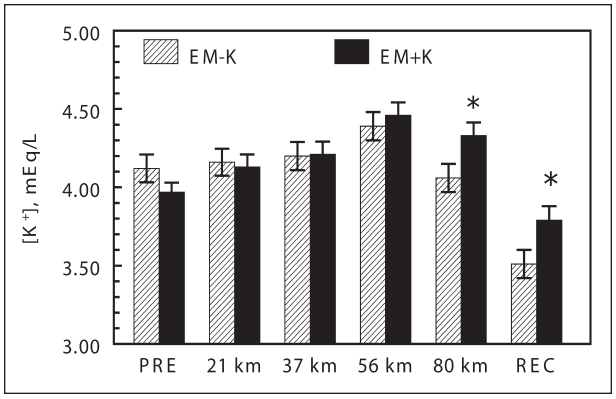

Figure 2—Mean (± SE) plasma K^+ concentrations versus stage of sample collection before, during, and after the 80-km endurance ride in EM-K- and EM+K-treated horses. Notice the increase ($P < 0.05$) with stage from before the ride to the 56-km inspection point and the decrease ($P < 0.05$) from the 80-km inspection point to during recovery for all (EM-K- and EM+K-treated) horses. *Significant ($P < 0.05$) differences between EM-K- and EM+K-treated horses at the 80-km inspection point and also during recovery.

A significant ($P = 0.048$) stage-by-treatment interaction was found for plasma K^+ concentration (**Figure 2**). Plasma K^+ concentration was significantly lower at 80 km ($P = 0.033$) and during recovery ($P = 0.021$) in EM-K-treated horses, compared with EM+K-treated horses.

Partition—Mean changes from before the ride to the 21-, 37-, 56-, and 80-km inspection points and during recovery in plasma H^+ concentration were partitioned among the 3 independent variables for EM-K- and EM+K-treated horses (**Figure 3**). In EM-K- and EM+K-treated horses, $PvCO_2$ was the dominant independent variable at the 21-km inspection point and the SID was the dominant independent variable thereafter. Actual changes in plasma H^+ concentrations from preride values were negatively related to changes in the SID for EM-K- ($r = -0.836$, $P = 0.038$) and EM+K-treated horses ($r = -0.865$, $P = 0.026$; data not shown but derived from data in Table 2). Calculated contributions of changes in the SID to changes in plasma H^+ concentration from preride values were positively related. Changes in the SID were highly correlated with corresponding changes in plasma Na^+ concentration from preride values for EM-K- ($r = 0.998$, $P < 0.001$) and EM+K-treated horses ($r = 0.906$, $P = 0.013$; **Figure 4**).

In EM-K-treated horses, the initial 3-**nanoequivalent per liter (nEq/L)** decrease in plasma H^+ concentration at the 21-km inspection point was partitioned

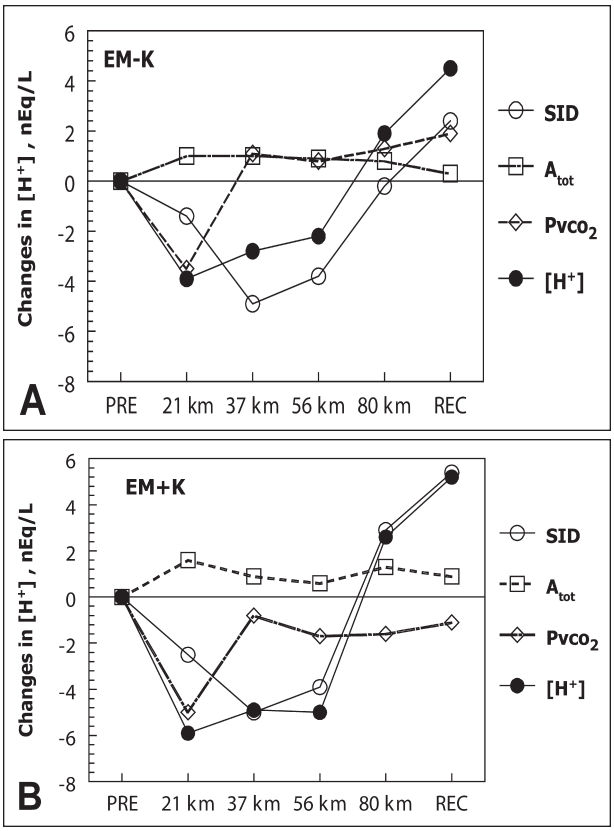

Figure 3—Partitioning of changes in plasma H^+ concentration from resting values, during the 80-km endurance ride, and during recovery into contributions from 3 independent variables: the strong ion difference (SID), total weak acids (A_{tot}), and $PvCO_2$ in EM-K- (A) and EM+K- (B) treated horses.

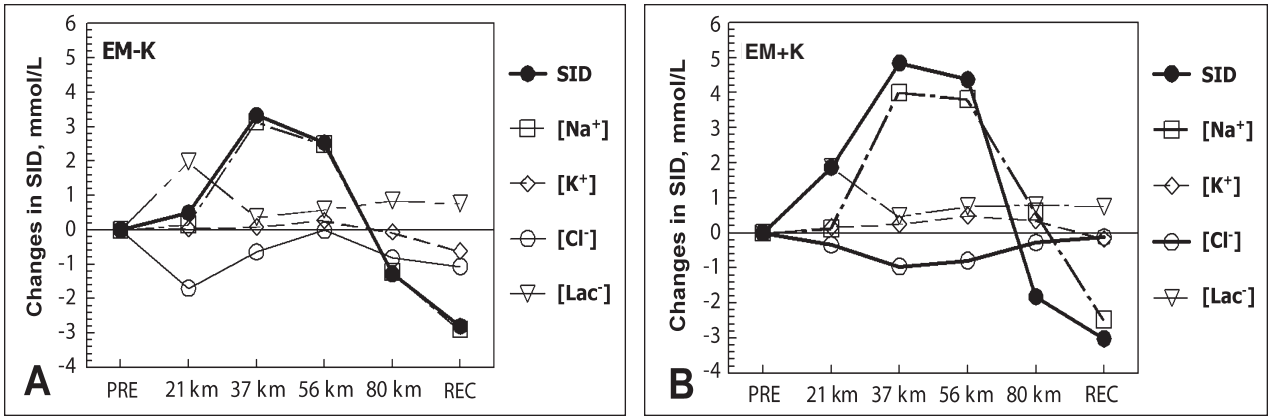

Figure 4—Changes in plasma concentrations of strong ions and their algebraic sum, the SID, from resting values, during the 80-km endurance ride, and during recovery in EM-K- (A) and EM+K- (B) treated horses. [LAC^-]= Lactate concentration.

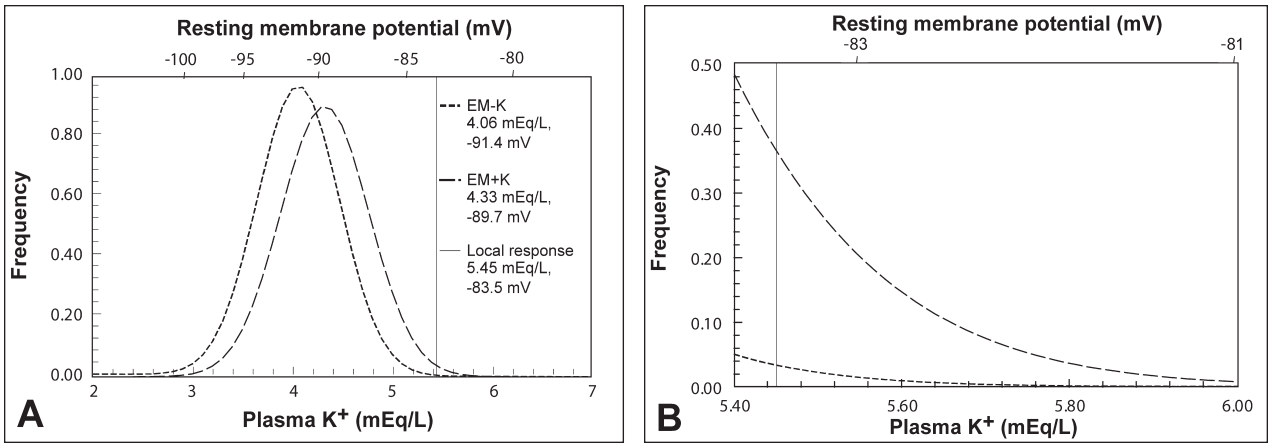

Figure 5—Frequency distributions of plasma K^+ concentration in EM-K- (short dashed curved line) and EM+K-treated (long-dashed curved line) horses are compared with a line for the start of local responses or disproportionately greater increases in excitability (solid vertical line), which corresponds to depolarization of +7 mV from the mean resting membrane potential of muscle cells at the 80-km inspection point. The +7 mV is a textbook value based on other species[7] and assumed here for the horse (A). The cutoff areas under the curves (magnified in B) are proportional to the cumulative frequencies, namely, 1 horse in approximately 170 given EM+K and 1 horse in approximately 770 given EM-K.

into –2.4-, +0.11-, and +0.9-nEq/L contributions from changes in $PvCO_2$, the SID, and A_{tot}, respectively (Figure 3). Subsequently, the predominant contribution to change in plasma H^+ concentration was from the SID at the 37-, 56-, and 80-km inspection points and during recovery. In EM+K-treated horses, the initial decrease in H^+ concentration of 2.6 nEq/L at the 21-km inspection point was partitioned into contributions of –2.1, +1.6, and +0.9 nEq/L from changes in $PvCO_2$, the SID, and A_{tot}, respectively. Subsequently, the predominant contribution to change in plasma H^+ concentration was from the SID at all stages.

Calculated E_K of muscle cells—Estimated mean E_K before the ride was –91.0 and –92.2 mV for EM-K- and EM+K-treated horses, respectively (**Figure 5**). Assuming no change in K_o during the ride, estimated mean changes in E_K from before the ride to the 80-km inspection point were –0.392 and +2.52 mV for EM-K- and EM+K-treated horses, respectively.

Discussion

Results of our study are consistent with previous observations on endurance horses.[1,18-25] A salient finding is that plasma K^+ concentration increases during prolonged exercise at only 3.4 m/s over hilly terrain. New findings include a biphasic response of plasma H^+ concentration, which decreased initially, then increased during prolonged exercise and increased further during recovery. Also new is the finding that changes in plasma H^+ and K^+ concentrations in the final stage were moderated by supplementation with EM-K. Moreover, application of the Stewart[10] comprehensive model revealed the major impact of the SID on plasma H^+ concentration and the major impact of plasma Na^+ concentration on the SID. Thus, the lower plasma K^+ and H^+ concentrations in the last stage may be attributable to the absence of K and the higher amount of Na, respectively, in the EM-K formula.

The critical differences in plasma K^+ and H^+ concentrations between EM-K- and EM+K-treated horses were not evident until the last stage, perhaps because this ride was at the start of the season. Riders agreed that our ride was less challenging than most in mid season. Also, weather conditions were mild. Horses were eliminated for reasons similar to those in previous reports.[22-25] Our elimination rate of 26% was smaller than previous rates of 40% or more.[26,27] Weight losses of

only 5% at the 56- and 80-km inspection points are similar to a 5% weight loss in another 80-km ride[19] and smaller than 7% losses at a 80-km inspection point during 160-km rides.[19,26] The 5% weight losses are consistent with mild ambient conditions, footing, and trail difficulty in our study.[28]

Increases in plasma Na^+ concentration during exercise presumably helped to maintain hyperosmolality, and hence thirst, during the ride. Effects of increased plasma Na^+ concentration at the 37- and 56-km inspection points would contribute to the decrease in plasma H^+ concentration via the SID and hyperosmolality. The decrease in plasma Na^+ concentration that was observed during recovery reflects fluid redistribution after further drinking of water. Plasma osmolality was also increased by 5% in a 62-km ride in horses supplemented with salt paste and saline solution.[29]

The biphasic plasma H^+ concentration response was evaluated by the Stewart model,[10] which revealed that $PvCO_2$ was the predominant contributing factor to the decrease in plasma H^+ concentration at the 21-km inspection point and that the SID was the predominant contributing factor to the subsequent increase in plasma H^+ concentration (Figure 3). Respiratory alkalosis has been observed previously in horses running at 40% of maximum oxygen consumption on a treadmill.[30] In our study, hemoconcentration and a decrease in plasma Cl^- concentration also contributed to the decrease in plasma H^+ concentration at the 21-km inspection point. Alkalosis in endurance rides has previously been attributed mainly to the loss of Cl in sweat,[1,9,31-33] but the impact of the SID on the change in plasma H^+ concentration at the 21-km inspection point was slight in EM+K-treated horses and negligible in EM-K-treated horses in our study.

Use of the model of Stewart[10] reveals the dominance of the SID contribution to the increasing plasma H^+ concentration from the 21-km inspection point to recovery (Figure 3). This dominance was also indicated by the overall negative regression of changes in plasma H^+ concentration on changes in the SID and by the partition specifically at the 80-km inspection point and during recovery, where the increase in plasma H^+ concentration mainly reflected a 3.3-mEq/L decrease in the SID. This dominance of the SID agrees with previous results of a study[11] on human athletes subjected to maximal exercise but does not agree with the results of studies[12,13] on horses during repeated sprints in which $PvCO_2$ predominated. The progressive increase in plasma H^+ concentration from the 21-km inspection point to recovery occurred despite hemoconcentration from the 37-km inspection point on, which has a propensity to increase the SID and hence to moderate acidosis.[10]

The only significant difference in ions contributing to the SID between EM-K- and EM+K-treated horses was the lower plasma K^+ concentration in EM-K-treated horses at the 80-km inspection point and during recovery, which opposed the lower plasma H^+ concentration in EM-K-treated horses. Thus, changes in other strong ions that were not significant must have accounted for the greater impact of the SID in EM-K-treated horses.

The high correlations of plasma H^+ concentration with the SID and the SID with plasma Na^+ concentration illustrate how the comprehensive physicochemical model of Stewart[10] can extract information that is not evident in routine analysis of variance. The model results suggest that the higher Na intake of 26 g, rather than the lower K intake of 33 g, in EM-K-treated horses, compared with EM+K-treated horses, may explain the lower plasma H^+ concentration in EM-K-treated horses at the 80-km inspection point and during recovery.

Plasma K^+ concentration increased progressively to approximately 10% above preride values at the 56-km inspection point when speed was 3.4 m/s over hilly terrain. This result compares with a previously estimated minimal speed of 4 m/s on the flat at which plasma K^+ concentration increased during prolonged exercise in horses.[9] An impression may have been given in some previous reports[9,34,35] that plasma K^+ concentration decreases during endurance exercise in horses because blood samples were taken 3 to 60 minutes after exercise when plasma K^+ concentration is falling rapidly as K moves into muscle cells and urine.

Despite K losses in sweat,[22,28] plasma K^+ concentration may increase in proportion to exercise intensity[8] as K moves out of working muscle cells. Increasing plasma K^+ concentration initially facilitates exercise by dilating arterioles in muscle. Higher plasma K^+ concentration, however, will exert catelectrotonic effects on E_K and may exacerbate neuromuscular excitability.[7] Eventually, high plasma K^+ concentration reaches a critical level and inhibits action potentials, so muscles and nerves become unable to respond.[7,36] To evaluate these possible adverse effects in relation to increases in plasma K^+ concentration observed in our study, it is necessary to make reasonable assumptions on the basis of previous research in horses and other species, such as a K_i concentration of 124 mEq/L.[16] No relevant data have been found in horses, but a textbook value of approximately +7 mV of depolarization leads to a zone of local responses in which cathodal stimuli are facilitated, that is, in which increases in excitability are greater up to the firing threshold.[7] In the present context, a zone of 7 to 15 mV of depolarization may represent hyperexcitability and persistent depolarization of approximately > 15 mV may represent prolonged refractory periods and decreased muscle response.[7,36]

The mean calculated E_K of muscle cells in our study is –90.5 mV, so estimates of –83.5 and –75.5 mV may be predicted for catelectrotonic local responses and firing thresholds, respectively. The Nernst equation yields corresponding estimates of 5.45 and 7.35 mEq/L for K_o concentration and plasma K^+ concentration. Such values have been recorded, albeit for faster speeds and briefer periods, without clinical manifestations.[8,9]

Although the mean increases in plasma K^+ concentration at the 56- and 80-km inspection points are well below the predicted mean plasma K^+ concentration of 5.45 mEq/L that corresponds to a depolarization of +7 mV, an evaluation of the frequency distributions reveals a difference between EM-K- and EM+K-treated horses (Figure 5). The SDs of mean plasma K^+ concentrations are 0.414 and 0.442 mEq/L for EM-K- and EM+K-treated horses at the 80-km inspection point, respectively. Dividing these SDs into the respective mean plasma K^+ concentrations of 4.06 and 4.33 mEq/L at

80 km and 5.45 mEq/L (+7 mV depolarization) will yield respective estimates of 3.354 and 2.522 for the Z value, with corresponding probabilities of 1 horse in 171 or 1 in 769 reaching the zone of local catelectrotonic responses in EM+K- or EM-K-treated horses, respectively.[17]

The sensitivity of these results to the critical assumption of K_i concentration of 124 mEq/L may be tested with the alternative assumption of 150 mEq/L, which may be regarded as a population mean for many muscles in many species.[17] It gives corresponding probabilities of 1 horse in 53 or 495 in EM+K- or EM-K-treated horses reaching a zone of hyperexcitability. Regression of the means of the equine samples toward the general population mean suggests that the alternative estimates indicate respective ranges of 1 horse in 53 to 171 given EM+K and 1 horse in 495 to 769 given EM-K. The conclusions drawn from these estimates are that hyperexcitability caused by increased plasma K^+ concentration in this mild ride would be uncommon, but nevertheless, chances would be lower at the end of the ride in horses given EM-K, compared with those given EM+K.

No clinical effects of treatment were evident in our ride, which was not considered a challenging ride. Thus, the potential clinical impact of an increase in plasma K^+ concentration remains in question for faster and harder endurance rides. The likelihood of clinical manifestations of neuromuscular excitability should increase with the work intensity of a horse because increases in plasma K^+ concentration are proportional to work intensities.[8,9] Therefore, a potassium-free electrolyte mixture should be more beneficial in faster horses and in more competitive rides. More strenuous conditions are also conductive to greater sweat losses and to a greater need to replace K during slower sections of the ride and immediately after exercise by the administration of a potassium-rich electrolyte-glucose mixture, as recommended previously.[9]

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e. Lyte-Now, Pro-formula Labs, Ft Lauderdale, Fla
f. Ora-Lyte, The Butler Co, Columbus, Ohio.
g. Tyrel Platform, Model TC-105, Allweights Hamilton Scale Corp, Richmond, Va.
h. Dryhep Plus Kit, Becton-Dickinson, Franklin Lakes, NJ.
i. Vacutainer Green, Becton-Dickinson, Franklin Lakes, NJ.
j. Stat Profile M, Nova Biochemical, Waltham, Mass.
k. Beckman Instruments Inc, Brea, Calif.
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