Electrolyte Supplementation for Endurance Horses: Effects on Fluid Losses and Performance

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Administering oral electrolyte supplements to endurance horses is a well-accepted, yet poorly validated, practice. In horses supplemented with either a high-dose (HD) or low-dose (LD) of electrolytes in a 80-km ride, we found no difference in body-mass loss or competition time, yet estimated water intake and rider assessment of performance were improved with HD. However, hypernatremia and hyperchloremia developed in some horses receiving HD, which suggests that electrolyte supplementation may not be completely innocuous. Authors' addresses: Via del Mandriato, 22 Pietrasanta, 5504 Italy (Sampieri), Faculty of Veterinary Science, University of Melbourne, 250 Princes Highway, Werribee, Victoria, Australia, 3030 (Hinchcliff); Large Animal Clinical Sciences, D202 Veterinary Medical Center, Michigan State University, East Lansing, MI 48824 (Schott); Middleburg Agricultural Research and Extension Center, Virginia Tech, Middleburg, VA 20117 (Geor); and Cliffe Veterinary Group, Lewes, East Sussex, BN8 5QX, United Kingdom (Jose-Cunilleras); e-mail: fsampierivet@hotmail.com. © 2007 AAEP.

1. Introduction

Endurance horses may produce sweat at rates of 5–10 l/h, which leads to substantial depletion of body water and electrolyte stores. As a consequence, body mass (BM) losses of 3–7% routinely develop during endurance competitions, despite frequent stops for drinking, and these deficits often require >24 h to be replaced. Although poorly substantiated, it is generally accepted that a greater loss of body fluid (more likely caused by decreased voluntary fluid replacement than an increased sweating rate) is an important risk factor for "metabolic pulls" and the development of medical problems during and after endurance rides. Thus, it is

currently a common practice for endurance riders to provide additional electrolytes, such as oral pastes or in-grain meals, to their horses with both the intent of replacing lost electrolytes and stimulating water intake.⁴

Previous studies have shown that the magnitude of water and electrolyte deficits induced by endurance exercise can be attenuated by administration of oral electrolyte pastes, and recovery can be hastened when rehydration solutions containing electrolytes, compared with plain water, are provided.^{5,6} In these studies, BM losses were attenuated by greater voluntary drinking, and the amount of fluid imbibed correlated to the increase in plasma tonicity.⁵ However, to

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date, there is no direct evidence that these apparent benefits of electrolyte supplementation either affect performance or decrease the risk of "metabolic failure" in competing endurance horses. Thus, the purpose of this study, presented to the 7th International Conference of Equine Exercise Physiology in 2006, was to determine whether or not supplementation with a high dose of electrolytes, estimated to completely replace expected sweat losses, affected ride time or owner-assessed performance compared with supplementation with a lower dose of electrolytes, estimated to replace about one-third of expected sweat losses.

2. Materials and Methods

Eight horses were studied during competition in two 80-km rides: the Arabian Horse Association of Michigan Ride (July 3, 2004) and the Wolverine Ride (July 31, 2004), both held on the same course in the countryside surrounding Petoskey, MI. Because both rides were in the middle of the Michigan ride season, fitness of the horses was similar, and weather conditions were nearly identical for each ride. The horses studied included 6 Arabians, 1 Quarter Horse, and 1 Mustang and ranged in age from 9 to 17 yr (mean = 11.8 yr) and in BM from 383to 529 kg (mean = 441.5 kg). Experienced horse and rider teams were recruited for participation in the study and agreed not to provide any electrolyte supplementation for the duration of the study, including 2 days before each competition.

The study was a randomized crossover design in which horses received one of two doses. The high dose (HD) was 0.4 g/kg NaCl and 0.13 g/kg KCl (equivalent to 180 g of NaCl and 60 g of KCl in a 450-kg horse) that was estimated to replace losses of ~30 l of sweat. The low dose (LD) was 0.13 g/kg NaCl and 0.04 g/kg KCl (equivalent to 60 g of NaCl and 20 g of KCl in a 450-kg horse) that was estimated to replace losses in ~ 10 l of sweat. During the first ride, four horses received HD, and the other four horses received LD. Treatments were reversed for the second ride. Riders and official ride veterinarians were blinded to the treatments. One-half of the electrolyte dose was administered as a slurry in applesauce with an oral-dose syringe ~30 min before the ride start. The second one-half of the dose was administered during the rest period after completion of the first 40 km.

Body mass was determined on a load-bar scale^a during the 1 h before the ride start (pre-ride), after completion of the ride (80 km), and after the first 2 h of recovery. Blood samples (heparinized and whole blood) were collected through jugular venipuncture each time horses were weighed as well as during the rest period after completion of the first 40 km. Official ride veterinarians examined the horses before, during, and after the ride, and findings were recorded on American Endurance Ride Conference (AERC) ride cards. In addition, an interviewer (veterinary student) was assigned to each horse and

rider team for the duration of the ride. Before, during (at each checkpoint), and after ,the ride interviewers queried riders to complete a form that detailed riders' perception of their horses performance during the ride, estimated water and feed intake, and urine and fecal output. Race times were obtained from the official ride timers. Heparinized blood samples were placed in iced water immediately after collection and were analyzed within 30 min from collection in the field laboratory with NOVA Biomedical^b and I-STAT^c analyzers. Whole-blood samples were centrifuged and transferred to plastic tubes; they were frozen on dry ice (until transferred to a -20°C freezer) to subsequently undergo complete biochemical analysis^d within 2 wk of collection.

Two-way analysis of variance for repeated measures was employed to analyze differences for time, treatment, and interaction between time and treatment's effects. Post-hoc testing was performed using Holm-Sidak or Dunnett's tests. Significance was set at p < 0.05 for time and treatment effects and at p < 0.1 for interaction effects. The values provided in the text are means and standard error of the means.

3. Results

All eight horse and rider teams completed both rides and were declared metabolically fit to continue at the veterinary examination 30 min after the end of the ride, although one horse was excluded from completion during the first ride and two were excluded in the second race because of lameness. There were no differences in performance times (430 \pm 26 min for HD and 422 ± 21 min for LD), finishing place, or BM loss after 80 km ($-3.8 \pm 0.3\%$ for HD and $-4.0 \pm 0.3\%$ for LD) between the two treatments. Similarly, no differences in heart rate or other clinical findings recorded on AERC ride cards were detected between treatments at any time point. In contrast, information recorded on the interview forms indicated that rider perception of performance was subtly better with HD, and estimated water intake was significantly greater with HD. However, the riders did not report any difference between treatments for estimated urine or fecal production.

At the end of the ride, serum sodium and chloride concentrations were significantly higher with HD, and peak measurements were 159 and 121 mmol/l for HD and LD, respectively. No difference in serum-potassium concentration was found between treatments. Also, at the end of the ride, venous blood pH was significantly lower with HD, whereas serum bicarbonate concentration was significantly lower throughout the entire race when the horses were treated with HD. Those results were observed throughout the early phases of recovery after the race. No effect of either time or treatment was found for serum total-protein concentration,

whereas albumin concentration was lower at all time points after the start with HD.

4. Discussion

In this study, supplementation with HD compared with LD had no effect on ride time, BM loss, or any of the clinical measures assessed by ride veterinarians at checkpoints during or after the ride. Administration of HD induced horses to drink more water during and after the ride and resulted in a higher score of rider assessment of performance. However, HD also produced substantial increases in sodium and chloride concentrations, and some values were above the upper limit of commonly accepted reference ranges.

It is well recognized that endurance athletes do not completely replace their sweat fluid losses during exercise or the initial few hours of the recovery period, despite having free access to water or other rehydration solutions. This incomplete restoration of body fluids has been termed both "voluntary" and "involuntary dehydration," and it has been ascribed to blunting of thirst because of loss of both water and electrolytes in sweat.8 Thus, along with replacement of electrolytes lost in sweat, the goal of electrolyte supplementation in this study was to stimulate drinking by increasing plasma tonicity, the primary stimulus for thirst.⁹ Treatment with HD seemed to accomplish this goal, because water intake was reported by the riders to be greater with this treatment. However, a significant difference in BM loss between treatments was not found. A possible explanation for this discrepancy would be that greater water intake resulted in increased urine production. The riders did not report the latter, but these data were assimilated from an interview form that relied on rider recall after each loop and as such, may not have been entirely accurate. Furthermore, the relatively low number of horse and rider teams may have precluded finding differences that were small in magnitude.

Despite greater water intake with HD, some of the horses developed hypernatremia and hyperchloremia by the end of the ride. Even with LD, serum sodium and chloride concentrations tended to be higher than the pre-ride values after 40 km of competition. Thus, oral electrolyte supplementation tended to produce a relative water deficit in extracellular fluid. This could have been a consequence of either inadequate water intake or a shift of water into the lumen of the intestinal tract. Because serum sodium and chloride concentrations decreased to the pre-ride values by 2 h of recovery, inadequate water intake during the ride would seem a more likely explanation for development of hypernatremia and hyperchloremia. Furthermore, the decrease in albumin concentration with HD both during and after the ride provides further support that hypernatremia and hyperchloremia were not the result of shift of water into the lumen of the intestinal tract. The results obtained in regard to venous blood pH and bicarbonate concentration are in support of the electrolyte shifts caused by the HD supplementation, because it reduced the ground for metabolic alkalosis and the complications derived from it that are typical of endurance horses.

Although this study is novel in that it was the first interventional study to be completed in horses competing in actual endurance rides, there were several limitations that warrant mention. First, because of relatively mild ambient conditions expected at these rides, we anticipated that horses would lose $\sim 30 \, \mathrm{l}$ of sweat (or ~5 l/h of exercise). However, ride times were somewhat longer than expected (~9 h) such that we may have underestimated total sweat losses and incompletely replaced sweat electrolyte losses with HD. Nevertheless, development of hypernatremia and hyperchloremia in some horses supports that electrolyte replacement with HD was not inadequate. Furthermore, it is probably unnecessary, and perhaps even unwise, to attempt to replace the entire estimated electrolyte deficit during the ride, because a substantial reserve of water and electrolytes is likely available in ingesta within the large intestine. Taking this reservoir of water and electrolytes into consideration, it had been our recommendation before this study to attempt to replace no more than 50% of the amount of electrolytes expected to be lost during the course of the ride. Second, a more important limitation of our study was that treatment with LD provided a substantial amount of electrolyte replacement, nearly 60 g NaCl and 20 g KCl. This amount is not dissimilar to the one used by many endurance riders during the course of a 80 km ride, and it may have precluded our ability to detect additional effects of HD. Ideally, we should have compared HD (or both HD and LD) with a placebo treatment that contained no electrolytes. We initially attempted to recruit riders for a study in which HD was to be compared with such a placebo treatment; however, we found that riders were unwilling to participate if they would have to compete in one ride without electrolyte supplementation. A final limitation of this investigation is that we studied a group of non-elite 80-km endurance horses, and findings in these horses may not be applicable to elite endurance horses competing in 160-km races in which electrolyte supplementation may be of greater importance.

In conclusion, despite greater water intake with HD, this treatment did not result in faster competition time or provide any detectable competitive advantage in the horse and rider teams that participated in this study. Although BM loss was not attenuated by greater water intake with HD, a reason for this discrepancy was not clearly determined. Next, HD resulted in hypernatremia and hyperchloremia, yet at the same time, produced a subtle improvement in owner-perceived performance; it was also associated with detectable improvements in bicarbonate concentration and blood pH. However, development of hypernatremia and

hyperchloremia with HD, in these endurance horses, remains a concern, and until more definitive data becomes available, it has led us to decrease our current recommendation for electrolyte replacement to no more than one-third of the estimated deficits incurred by the exercise bout.

This project was funded by the Equine Research Fund of The Ohio State University. We thank Mr. J. Stern, Mrs. S. W. Eberhart, the Ohio State University and Michigan State University veterinary students, Mr. and Mrs. Dake, the Arabian Horse Association of Michigan, the Wolverine Ride Group, the official time keepers, the ride veterinarians, the riders, and the horses who participated in this field study.

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- ^dHitachi 911, Automatic Analyzer, Roche Diagnostics Corp., Indianapolis, IN 46256.