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Hydration effects on physiological strain of horses during exercise-heat stress

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Geor, Raymond J., and Laura Jill McCutcheon. Hydration effects on physiological strain of horses during exercise-heat stress. *J. Appl. Physiol.* 84(6): 2042–2051, 1998.—This study examined the effects of hyperhydration, exercise-induced dehydration, and oral fluid replacement on physiological strain of horses during exercise-heat stress. On three occasions, six horses completed a 90-min exercise protocol (50% maximal O₂ uptake, 34.5°C, 48% relative humidity) divided into two 45-min periods (*exercise I* and *exercise II*) with a 15-min recovery between exercise bouts. In random order, horses received no fluid (NF), 10 liters of water (W), or a carbohydrate-electrolyte solution (CE) 2 h before exercise and between exercise bouts. Compared with NF, preexercise hyperhydration (W and CE) did not alter heart rate, cardiac output (Q), stroke volume (SV), core body temperature, sweating rate (SR), or sweating sensitivity during *exercise I*. In contrast, after *exercise II*, exercise-induced dehydration in NF (decrease in body mass: NF, 5.6 ± 0.8%; W, 1.1 ± 0.4%; CE, 1.0 ± 0.2%) resulted in greater heat storage, with core body temperature ~1.0°C higher compared with W and CE. In *exercise II*, the greater thermal strain in NF was associated with significant (*P* < 0.05) decreases in Q (10 ± 2%), SV (9 ± 3%), SR, and sweating sensitivity. We concluded that 1) preexercise hyperhydration provided no thermoregulatory advantage; 2) maintenance of euhydration by oral fluid replacement (~85% of sweat fluid loss) during exercise in the heat was reflected in higher Q, SV, and SR with decreased heat storage; and 3) W or an isotonic CE solution was equally effective in reducing physiological strain associated with exercise-induced dehydration and heat stress.

dehydration; hyperhydration; oral fluid replacement; sweating; ion losses; temperature regulation; body temperature; equine

IN HUMAN ATHLETES it is well established that hypohydration, or a reduction in total body water (TBW), impairs cardiovascular function and increases heat strain during prolonged dynamic exercise (1, 8, 28, 34, 35). Moreover, the increments in thermal and cardiovascular strain are directly related to the magnitude of hypohydration (28, 35). In horses, as in humans, cutaneous evaporative heat loss (sweating) is the primary mechanism for temperature regulation during exercise (18). Rates of sweat fluid loss in horses can exceed 10–12 l/h (25), and, even in moderate ambient conditions, dehydration equivalent to ~6% or more of body mass can result during 2–3 h of exercise (19). However, the nature of this exercise-induced dehydration and the impact of the resultant body-water deficit on thermoregulatory function differ between equine and human athletes. The ionic composition of equine sweat during exercise is isotonic to hypertonic (19, 25), with an osmolality ranging from ~290 to 320 mosmol/kgH₂O, whereas human sweat is hypotonic relative to extracel-

lular fluid (ECF). Therefore, heavy sweat losses will elicit a hypertonic hypohydration in human subjects, whereas the approximately isotonic nature of equine sweat results in an isosmotic to hyposmotic hypohydration. Because plasma hyperosmolality has been demonstrated to impair thermoregulation (12, 17, 29), an isotonic fluid loss could mitigate the effect of hypohydration on thermoregulatory mechanisms. Kingston et al. (20) reported that progressive dehydration (~6% loss of body mass) did not alter sweating rate (SR) or heat storage in horses during >3 h of low-intensity exercise [40% maximal O₂ uptake ($\dot{V}O_{2\max}$)] in moderate ambient conditions. Conversely, Naylor and co-workers (30) demonstrated that isotonic and hypertonic hypohydration (3.2–3.9% decrease in body mass) induced before exercise resulted in increased heat storage during 40 min of exercise at 40% $\dot{V}O_{2\max}$ in similar conditions. This impairment to thermoregulation was attributed to decrements in heat transfer from core to periphery rather than decreases in SR or sensitivity of the sweating response (30). Taken together, these findings suggest that, at least during low-intensity exercise in moderate ambient conditions, hydration state has no effect on sweating responses and the rate of cutaneous evaporative heat loss in horses. Importantly, however, no study has evaluated the effects of hydration state on thermoregulation in horses during exercise-heat stress, when the greater thermal strain increases reliance on sweating for heat dissipation.

Given the detrimental effects of hypohydration on thermoregulation, appropriate strategies for fluid replacement are required to minimize thermal strain and optimize exercise performance. In human subjects, fluid-replacement strategies that limit increases in plasma hyperosmolality will mitigate any impairment to thermoregulation associated with hypohydration (8, 14, 35). Several investigators have reported that pure water or hypotonic solutions are similarly effective for attenuation of thermal and circulatory strain during exercise-heat stress (2–4). In horses, however, secretion of electrolyte-rich sweat (Na⁺ concentration ~120–140 mM; Cl⁻ concentration ~135–155 mM) results in marked contraction of the ECF space but minimal change in extracellular osmolality (19, 25). In this circumstance, ingestion of water could dilute the ECF space and limit mobilization of water from the intracellular fluid (ICF) space to the ECF compartment. Conversely, ingestion of sodium-containing fluid may assist in the maintenance of ECF and blood volumes by creating an osmotic gradient between the ECF and ICF spaces (31). No study has evaluated the effects of water vs. an isotonic electrolyte solution on thermal and

circulatory regulation in horses during exercise-heat stress.

Preexercise hyperhydration (increased TBW) has been advocated for reduction of thermal strain during exercise-heat stress. However, there are conflicting results regarding the thermoregulatory effects of hyperhydration during exercise in human subjects. Some investigators have reported that hyperhydration can reduce thermal strain (16, 23), whereas other studies have indicated no thermoregulatory advantage over the maintenance of euhydration during exercise (3, 22). Given the difficulty in ensuring adequate voluntary fluid intake by horses, preexercise hyperhydration is a practical strategy for optimization of fluid balance during prolonged exercise. Sosa León et al. (37) reported no differences between hyperhydrated and control horses for cardiac output, heart rate, or rise in core body temperature (T_{core}) during 90 min of exercise despite maintenance of a higher plasma volume in the fluid-treated group. Importantly, however, these trials were conducted in moderate ambient conditions (19–20°C); in hotter conditions, when rates of sweat fluid loss and rise in T_{core} are greater, it is possible that hyperhydration will assist cardiovascular and thermoregulatory responses during exercise.

The primary purpose of this study was to examine the effects of hydration state on physiological strain of horses during exercise-heat stress. Specifically, we determined the effects of hyperhydration, and of hypohydration with or without oral fluid replacement, on thermal and circulatory responses during 90-min of moderate-intensity exercise in hot ambient conditions. We hypothesized that 1) preexercise hyperhydration would enhance thermoregulatory responses (lower T_{core} , improve sweating) compared with euhydrated subjects and 2) maintenance of euhydration by oral fluid replacement would reduce physiological strain during exercise-heat stress. We further hypothesized that addition of electrolytes to oral rehydration solutions would improve the effectiveness of oral fluid replacement. Therefore, a further objective of the study was to compare the efficacy of water vs. an isotonic electrolyte solution for attenuation of thermal and circulatory strain during exercise.

MATERIALS AND METHODS

The care and use of animals followed the “Guide to the Care and Use of Experimental Animals” (Canadian Council on Animal Care, Ottawa, ON). All animal experiments were conducted after approval by the Animal Care Committee of the University of Guelph and performed in compliance with their recommendations.

Experimental animals. Six Thoroughbred horses (4 geldings and 2 mares), ranging in age from 3 to 6 yr, and weighing 420–475 kg [455 ± 10.0 (SE) kg], were used. Each horse's right carotid artery was surgically relocated to a subcutaneous position at least 6 mo before the study. All horses were housed indoors during the experimental period and fed a diet of timothy grass-alfalfa hay and mixed grain supplemented with 100 g NaCl and 50 g KCl daily. The diet was designed to meet the National Research Council guidelines for the nutritional requirements of horses performing regular exercise

and training. All horses were conditioned and undertaking regular treadmill exercise but were not heat acclimated. The $\dot{V}O_{2\text{max}}$ of each horse was determined twice 7–10 days before the start of the experiments by use of an incremental step test and an open-circuit calorimeter (13). The mean $\dot{V}O_{2\text{max}}$ was $140 \pm 8 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$. From linear regression analysis, the running speed that elicited 50% $\dot{V}O_{2\text{max}}$ was calculated for each horse.

Experimental protocols. The study was designed as a crossover, with each horse completing standardized exercise tests after each of the following treatments: 1) no fluid (NF); no oral fluid during the experimental period; 2) water (W): 20 liters of water, administered via nasogastric tube in 10-liter doses, with the first dose given 2 h before exercise and the second dose given after 45 min of exercise; and 3) carbohydrate-electrolyte solution (CE): 20 liters of CE solution administered using the same protocol as for the W treatment. The CE was isotonic (305 mosmol/kgH₂O) and contained 105 mM Na⁺, 15 mM K⁺, 132 mM Cl⁻, and 5% carbohydrate (as sucrose). This volume of fluid was chosen on the basis of previous studies in our laboratory in which the rate of sweat fluid loss in trained Thoroughbred horses was measured under similar exercise and environmental conditions (25). The temperature of the orally administered W or CE was 25°C. The order of treatments was randomized (but balanced), with a minimum of 7 days between experiments for each horse.

Food and water were withheld for 3 h before administration of the first fluid treatment and until after completion of the experimental protocol. Catheters for collection of mixed venous and arterial blood samples were placed in the pulmonary (via the left jugular vein; PE-240, Becton Dickinson, Parsippany, NJ) and carotid (Intracath 21 gauge, 3 in.; Deseret, Sandy, UT) arteries, respectively, after aseptic preparation and local anesthesia of the overlying skin. The position of the catheter in the pulmonary artery was verified by observation of characteristic pressure traces with an oscilloscope monitor (Tektronics 401; Spacelabs Medical Products, Mississauga, ON) and a pressure transducer (DTX model T36AD-R; Viggo-Spectromed, Oxnard, CA). After measurement of body mass (± 0.5 kg; KSL Scales, Kitchener, ON), collection of baseline blood samples, and administration of the first dose of fluid, horses were kept in a holding area (room temperature 20°C) until 15 min before exercise. At that time, horses were moved to a temperature-controlled exercise laboratory and positioned on a high-speed treadmill (Sato, Sweden). A thermohygrometer (model 3309-60, Cole-Palmer Instruments, Chicago, IL) was used to monitor ambient conditions during all trials. Ambient conditions were similar for all trials; mean values for room temperature and relative humidity during the experiments were $34.5 \pm 0.3^\circ\text{C}$ (SE) and $48 \pm 5\%$, respectively.

With the treadmill set at a 5% slope, horses exercised at a speed equivalent to 50% of their predetermined $\dot{V}O_{2\text{max}}$ (range 4.4–4.6 m/s) for two 45-min periods (*exercise I* and *exercise II*) with a 15-min rest between phases, during which the second dose of oral fluid was administered. A fan mounted above and 0.5 m in front of the treadmill was used to maintain an air velocity of 3.5–4 m/s over the anterior and dorsal aspects of the horse. Air velocity was measured with an anemometer (Davis Instruments, Hayward, CA) positioned at three sites: lateral midcervical region, lateral and dorsal thorax, and dorsal to the gluteal region of the hindquarters.

During each phase of exercise, heart rate (HR), pulmonary artery temperature (T_{pa}), rectal temperature (T_{re}), SR, hematocrit, and plasma osmolality and total solids concentration (TS) were measured at 5-min intervals. Middle gluteal muscle

temperature (T_{mus}) was measured before and after each phase of exercise. O_2 uptake ($\dot{V}\text{O}_2$), CO_2 production ($\dot{V}\text{CO}_2$), respiratory exchange ratio (RER), cardiac output (\dot{Q}), and stroke volume (SV) were measured at 0, 5, 15, 30, and 45 min of exercise in each phase. Body mass was measured immediately before and after completion of each exercise phase.

Analytic methods. T_{pa} was measured by inserting a copper-constantan thermocouple (IT-14, Physitemp Instruments, Clifton, NJ) into the pulmonary artery within an 8-Fr polyethylene catheter. For measurement of T_{re} , a thermocouple (T-180, Physitemp Instruments) was inserted 25–30 cm proximal to the anal sphincter. T_{mus} was measured by inserting a needle thermocouple (MT-23, Physitemp Instruments) ~4 cm into the muscle through the lumen of an 18-gauge 37-mm needle. All thermocouples had response times of ~1°C/s and were calibrated in a heated water bath with a precision thermometer (Fisher Scientific, Mississauga, ON). All measurements of T_{mus} were performed after aseptic preparation and local analgesia of the skin.

Local SR was measured on the left lateral thorax by use of a direct sweat-collection method, as described elsewhere (20, 25). In brief, a sealed polyethylene pouch enclosing a 150-cm² area of skin was attached to an area of shaved skin with a dermal adhesive. The edges of the pouch were further sealed by dermal tape that covered the pouch-skin margin. A ventral reservoir, formed by a deep fold in the polyethylene, separated accumulating sweat from the skin surface and facilitated the removal of all collected sweat through polyethylene tubing (ID 1.67 mm, Intramedic; Becton Dickinson) incorporated into the lateral margin of the pouch. Sweat samples were collected every 5 min throughout each phase of exercise and during the 15 min rest between *exercise I* and *exercise II*. SR, expressed as milliliters per square meter per minute, was calculated on the basis of the volume of sweat collected at the end of each time interval from the measured skin area within the pouch. Therefore, the measured SR represents the average rate of sweat production over a 5-min period. Total sweat fluid loss was calculated from the change in body mass after accounting for fecal and estimated respiratory water losses (19, 20). No horse voided urine during any of the trials. Sweating sensitivity was defined as the slope of the regression line representing the individual 5-min local SR and T_{pa} values obtained during the linear phase of the exercise transient (up to 20–25 min in each bout of exercise) (29). Sweating sensitivity was expressed as milliliters per minute per square meter per degree Celsius. Percent dehydration was estimated from the difference in body mass after each phase of exercise compared with the body weight recorded 2 h before exercise.

Arterial and mixed venous blood samples for measurement of hemoglobin concentration, oxygen content, mixed-venous hematocrit, and plasma TS and osmolality were collected before the first fluid treatment, immediately before *exercise I* and *exercise II*, and at 5-min intervals during each phase of exercise. Blood samples were collected anaerobically into 7.5-ml syringes containing 100 IU of heparin lithium (Sarstedt, Nümbrecht, Germany); placed in an ice-water bath; and analyzed for hemoglobin concentration, oxygen saturation, and oxygen content within 30 min of collection (OSM 3 Hemoximeter; Radiometer, Copenhagen, Denmark). Hematocrit was determined by the microhematocrit method. Plasma was then separated from the cells by high-speed centrifugation (14,000 rpm for 5 min) and analyzed for TS by refractometry (model SPR-T2, Atago). The percent change in plasma volume (PV) was calculated by using the change in plasma TS (26). This calculation assumes there is no net movement of protein either to or from the vascular compartment during

exercise. Plasma osmolality was determined by freezing-point depression (model 3MO Plus, Advanced Instruments, Needham, MA). All analyses were performed in duplicate.

$\dot{V}\text{O}_2$ and $\dot{V}\text{CO}_2$ were measured with an open-flow respiratory gas-collection system consisting of a lightweight loose-fitting mask; flexible tubing; a flow-controlling baffle; a section of rigid tubing containing a venturi and a pressure transducer; a vacuum motor; and a multiport gas-sampling system. During exercise, flow rates of ~6,000–7,000 l/min were used. Within 15 min of the start of exercise, the O_2 (Ametek model S-3A/1, Thermox Instruments Division, Pittsburgh, PA) and CO_2 (Ametek model CD-3A, Thermox Instruments Division) analyzers were calibrated against a certified gas mixture (Canox, Mississauga, ON). A differential pressure transducer (Validyne, Northridge, CA) was used to monitor flow rate, and the system was calibrated before and after each experiment by use of the nitrogen-dilution technique (10). Both gas analyzers and the pressure transducer were interfaced with a laboratory computer (Apple IIe) and processed by using a scientific software package (Superscope II, GW Instruments, Somerville, MA). Standard equations were used to calculate $\dot{V}\text{O}_2$ and $\dot{V}\text{CO}_2$. At each measurement period, gas samples for determination of $\dot{V}\text{O}_2$ and $\dot{V}\text{CO}_2$ were collected at 10-s intervals for 2 min; the data reported represent mean values for $\dot{V}\text{O}_2$ and $\dot{V}\text{CO}_2$ during each measurement period. The RER values were calculated by dividing $\dot{V}\text{CO}_2$ by $\dot{V}\text{O}_2$. A cardiota-chometer (Equistat model HR-8A, Equine Exercise Physiology and Biomechanics, Unionville, PA) was applied on the horse's chest to record HR. \dot{Q} was calculated, by using the Fick principle, from measured $\dot{V}\text{O}_2$ and the arteriovenous O_2 content differences at each time point. SV was derived from the quotient of \dot{Q} and HR.

Statistical analyses. Data were analyzed by using two-way analysis of variance for repeated measures, with main effects of fluid treatment and time. After a significant *F*-test, Student-Newman-Keuls (to detect differences between fluid treatments) and Dunnett's (to detect differences within a treatment group) tests were used to identify pairwise differences. Regression analyses were utilized to examine effects of hydration state and plasma osmolality on sweating responses. Differences were considered significant when $P < 0.05$. All data are expressed as means \pm SE.

RESULTS

Effects of preexercise fluid administration. Significant ($P < 0.01$) decreases in hematocrit and plasma TS were evident 2 h after fluid administration (i.e., immediately before exercise) (Fig. 1, *A* and *B*), indicating hyperhydration in W and CE conditions before exercise compared with NF. Values for hematocrit and plasma TS in NF were unchanged at the end of the 2-h period before exercise. PV immediately before *exercise I*, as determined by changes in plasma TS, was increased by 8.7 and 12.1% for the W and CE treatments, respectively. Administration of W resulted in a significant ($P < 0.05$) decrease in plasma osmolality (Fig. 1*C*), whereas plasma osmolality was unchanged in NF and CE.

Metabolic responses. One horse in the NF treatment was unable to complete the exercise protocol because of fatigue. At the point of fatigue (30 min in *exercise II*), T_{pa} in this horse was 42.5°C. As a result, data from the end of exercise in NF represent means \pm SE for five horses. There was no effect of treatment on $\dot{V}\text{O}_2$, $\dot{V}\text{CO}_2$,

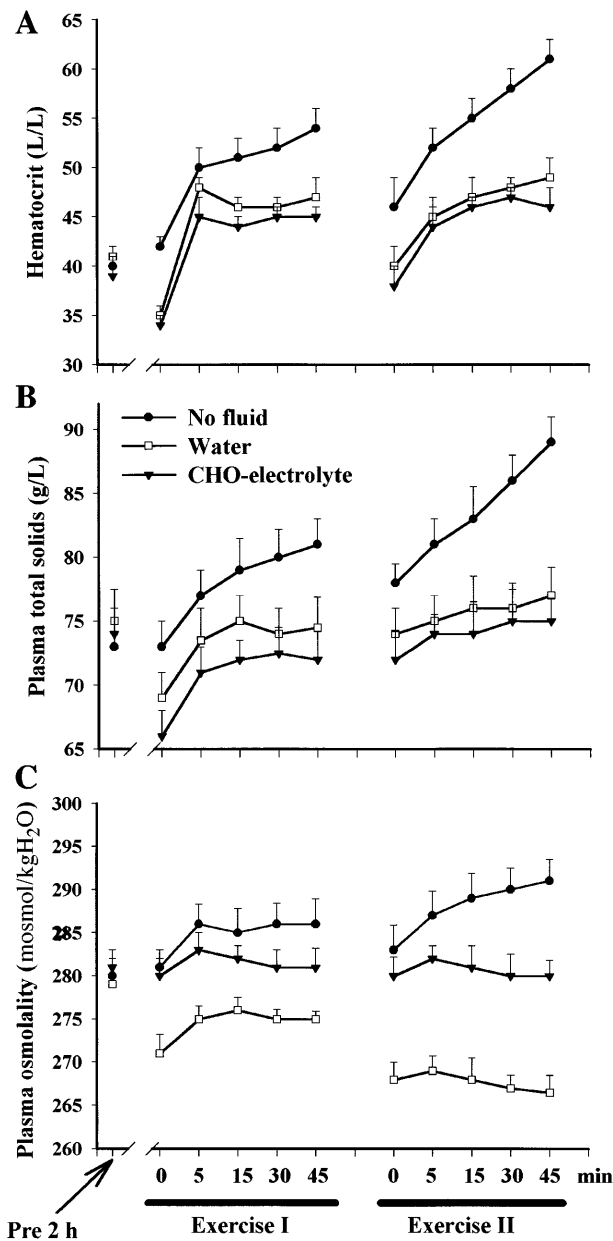


Fig. 1. Hematocrit (A), plasma total solids concentration (B), and plasma osmolality (C) in 6 horses before the first fluid treatment (pre 2 h) and during exercise at 50% of maximal $\dot{V}O_{2\max}$. Horses received either no fluid (NF), water (W), or an isotonic carbohydrate (CHO)-electrolyte solution (CE). Values are means \pm SE.

and RER during exercise (Table 1). Aerobic heat production was estimated from the $\dot{V}O_2$ data. It was assumed that each liter of O_2 consumed had an energy equivalent of 21 kJ and that 80% of the calorific value of O_2 was released as heat (18). When normalized for body surface area [body surface area = $1.09 + 0.008 \times$ body mass (kg)], aerobic heat production ranged from $1,629 \pm 95$ to $1,795 \pm 77$ W/m², with no significant between-trial differences.

Changes in body mass and blood variables in response to exercise. Loss of body weight in the W and CE treatments, as a result of the 20 liters of fluid adminis-

tered, was significantly ($P < 0.01$) lower compared with NF (Table 2). In NF, body mass was decreased by 24.1 ± 2.5 kg after exercise ($5.6 \pm 0.8\%$ of preexercise body mass). When expressed as a percentage of preexercise TBW ($0.66 \times$ body mass; Ref. 5), this decrease represented an $\sim 8\%$ reduction in TBW. In contrast, in W and CE treatments, the net decreases in body mass and TBW were ~ 1.0 and 1.5% , respectively, of pretrial body weight.

In all treatments there was a significant ($P < 0.001$) increase in hematocrit after 5 min of exercise (Fig. 1A). In NF, hematocrit progressively increased throughout *exercise I* and was significantly ($P < 0.01$) higher compared with W and CE between 15 and 45 min. Similarly, hematocrit was significantly ($P < 0.01$) higher in NF than in W and CE throughout *exercise II*. In all treatments, there was a significant ($P < 0.01$) increase in plasma TS after 5 min in *exercise I*. Thereafter, there was minimal change in plasma TS in the W and CE treatments. In contrast, in NF there was a progressive increase in plasma TS throughout both phases of exercise, and values were significantly ($P < 0.05$) higher compared with the other treatments at all time points except at 5 min of *exercise I* (Fig. 1B).

In the first 5 min of *exercise I*, PV decreased by ~ 5.5 – 6.0% in all treatments (Fig. 2). In W and CE, PV remained relatively constant at the reduced level until the end of *exercise I*. In contrast, PV in NF decreased progressively and was significantly lower compared with W and CE after 30 min of exercise. In all treatments, there was no significant restoration of PV during the 15-min period between exercise phases. In NF, PV was significantly ($P < 0.001$) lower than in W and CE throughout *exercise II*. At the end of exercise, PV was decreased by 8.1 ± 0.7 , 11.5 ± 1.1 , and $23.0 \pm 1.2\%$ in CE, W, and NF, respectively. The PV decrease in W was significantly ($P < 0.05$) greater than in CE only at the end of *exercise II*.

Coincident with the initial decrease in PV, plasma osmolality increased by ~ 3 – 4 mosmol/kgH₂O in all treatments during the first 5 min of exercise, with no further changes throughout the remainder of *exercise I* (Fig. 1C). In W and CE treatment, plasma osmolality was stable throughout *exercise II*. In contrast, in NF treatment, plasma osmolality increased progressively and was significantly ($P < 0.05$) higher than with the other treatments from 5 min until the end of this bout of exercise. Plasma osmolality in W was significantly ($P < 0.01$) lower compared with NF and CE throughout the exercise trial.

Temperature responses. Figure 3, A and B, shows T_{pa} and T_{re} during exercise. The rise in T_{pa} and T_{re} during *exercise I* (~ 3.2 and 2.5°C , respectively) was not significantly different between the three trials. During the final 20 min of *exercise II*, T_{pa} and T_{re} were significantly ($P < 0.01$) higher in NF compared with W and CE. At the end of exercise T_{pa} and T_{re} were ~ 1.0 and $\sim 0.8^\circ\text{C}$, respectively, higher in NF than in W and CE. In all treatments, T_{mus} increased by $\sim 4^\circ\text{C}$ during *exercise I*, with no significant difference between trials (Table 2). In contrast, the increase in T_{mus} during *exercise II* was

Table 1. Middle gluteal muscle temperature and metabolic variables at rest and during exercise at 50% of $\dot{V}O_{2max}$ with no fluid, water, or an isotonic carbohydrate-electrolyte solution

Variable	Group	Exercise I, min				Exercise II, min			
		0	15	30	45	0	15	30	45
T_{mus} , °C	NF	36.8 ± 0.1	ND	ND	41.0 ± 0.3	39.9 ± 0.2	ND	ND	42.2 ± 0.1*
	W	36.9 ± 0.1	ND	ND	40.8 ± 0.3	39.8 ± 0.1	ND	ND	41.1 ± 0.2
	CE	36.8 ± 0.1	ND	ND	40.8 ± 0.4	40.0 ± 0.1	ND	ND	41.2 ± 0.3
$\dot{V}O_2$, ml · kg ⁻¹ · min ⁻¹	NF	4.8 ± 1.4	63.2 ± 4.2	65.1 ± 5.1	67.8 ± 4.0	8.1 ± 1.8	66.8 ± 6.1	67.3 ± 5.4	68.1 ± 5.8
	W	5.0 ± 1.7	66.4 ± 5.0	68.1 ± 4.8	67.3 ± 6.1	7.6 ± 0.5	67.4 ± 4.2	69.1 ± 3.9	67.2 ± 5.5
	CE	4.9 ± 1.1	66.7 ± 4.6	67.2 ± 4.3	69.2 ± 7.4	8.0 ± 1.5	69.3 ± 5.5	67.9 ± 4.9	69.8 ± 5.9
$\dot{V}CO_2$, ml · kg ⁻¹ · min ⁻¹	NF	4.1 ± 0.8	57.2 ± 4.7	54.9 ± 4.6	53.3 ± 5.1	6.9 ± 0.8	53.8 ± 3.7	53.1 ± 5.9	52.9 ± 4.6
	W	4.5 ± 1.1	58.4 ± 6.6	55.1 ± 4.8	53.8 ± 4.8	6.6 ± 1.2	54.2 ± 5.5	52.5 ± 4.9	54.5 ± 6.0
	CE	4.3 ± 1.2	56.6 ± 5.5	54.8 ± 5.6	54.1 ± 5.7	7.2 ± 0.9	54.9 ± 6.5	53.6 ± 4.9	53.3 ± 4.9
RER	NF	0.85 ± 0.04	0.87 ± 0.07	0.83 ± 0.06	0.80 ± 0.06	0.85 ± 0.05	0.82 ± 0.07	0.81 ± 0.05	0.79 ± 0.06
	W	0.86 ± 0.03	0.87 ± 0.06	0.81 ± 0.04	0.82 ± 0.04	0.87 ± 0.06	0.80 ± 0.04	0.81 ± 0.05	0.80 ± 0.03
	CE	0.82 ± 0.05	0.84 ± 0.05	0.81 ± 0.05	0.80 ± 0.05	0.89 ± 0.04	0.81 ± 0.06	0.79 ± 0.04	0.78 ± 0.05

Values are means ± SE for 6 horses. $\dot{V}O_{2max}$, maximal O₂ consumption; NF, no fluid; W, water; CE, isotonic carbohydrate-electrolyte solution; $\dot{V}O_2$, O₂ uptake; $\dot{V}CO_2$, CO₂ production; RER, respiratory exchange ratio. T_{mus} , middle gluteal muscle temperature; ND, measurements not taken. *Significant difference from 0 min, exercise I, $P < 0.05$.

significantly ($P < 0.01$) greater in NF compared with W and CE. At the end of exercise, T_{mus} was ~1°C higher in NF than in W and CE.

Sweating responses. SRs during exercise, as determined by the volume of sweat collected from the sealed pouch on the thorax, are presented in Fig. 4. In all treatments, SRs increased from the start of exercise and continued to increase until the end of exercise I; SRs were similar among the three trials. Although SRs declined during the 15-min period between exercise phases, SRs at the start of exercise II were ~16–20 ml · m⁻² · min⁻¹. In all treatments, SRs increased rapidly during the initial 20–25 min of exercise II. SR then remained relatively constant for the remainder of exercise in W and CE, with values for SR significantly ($P < 0.05$) higher compared with the corresponding time points in exercise I. In contrast, after the first 15–20 min of exercise II, there was a decline in SR in NF such that values were significantly ($P < 0.05$) lower compared with W and CE during the final 20 min of exercise II. Consistent with this decrease in local SR, whole body sweat fluid loss during exercise II was significantly ($P < 0.05$) lower in NF (11.4 ± 0.6 liters) than in W (13.5 ± 1.0 liters) and CE (13.8 ± 0.7 liters). In W and CE, the volume of fluid administered before and during the exercise trial replaced ~85% of sweat losses (Table 2). Mean values for sweating sensitivity during exer-

cise are presented in Fig. 5. There were no between-group differences for sweating sensitivity during exercise I. However, in exercise II, sweating sensitivity in NF was significantly ($P < 0.05$) lower compared with W and CE. Figure 6 depicts the relationship between sweating sensitivity during exercise II and both total body mass loss ($n = 18$ observations) and final plasma osmolality ($n = 18$ observations). Sweating sensitivity decreased as total body mass loss ($P < 0.001$) and plasma osmolality ($P < 0.005$) increased.

Cardiovascular responses to exercise. In W and CE, HR, Q, and SV were stable throughout exercise (Fig. 7, A–C). In NF, the cardiovascular responses were similar

Table 2. Body weight and sweat fluid loss in response to 90 min of exercise at 50% of $\dot{V}O_{2max}$ with no fluid, water, or an isotonic carbohydrate-electrolyte solution

	NF	W	CE
Pre-trial weight, kg	454 ± 9	455 ± 8	452 ± 8
Weight loss, kg	24.1 ± 2.5	5 ± 1.0*	5.1 ± 1.1*
Δ Body weight, %	5.6 ± 0.6	1.0 ± 0.3*	1.1 ± 0.2*
Sweat loss, liters			
Exercise I	9.6 ± 0.7	10.1 ± 0.6	9.8 ± 0.9
Exercise II	11.4 ± 0.6	13.5 ± 1.0†	13.9 ± 0.7†
Total	21.9 ± 1.1	23.8 ± 0.7	23.9 ± 1.0
Sweat loss replaced, %		85.4 ± 2.2	84.3 ± 3.3

Values are means ± SE for 6 horses. Δ, Change. Significant difference from NF: * $P < 0.01$; † $P < 0.05$.

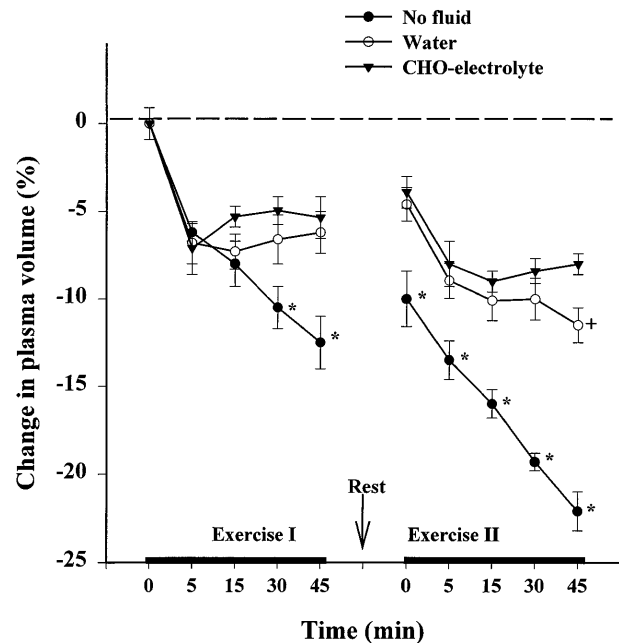


Fig. 2. Percent change in plasma volume in 6 horses during exercise at 50% of $\dot{V}O_{2max}$ with NF, W, or CE. The 0 value on y-axis represents the value just before start of exercise (2 h after administration of first dose of fluid in W and CE treatments). Values are means ± SE. * $P < 0.05$ from all other treatments at the same time point; † $P < 0.05$, W vs. CE.

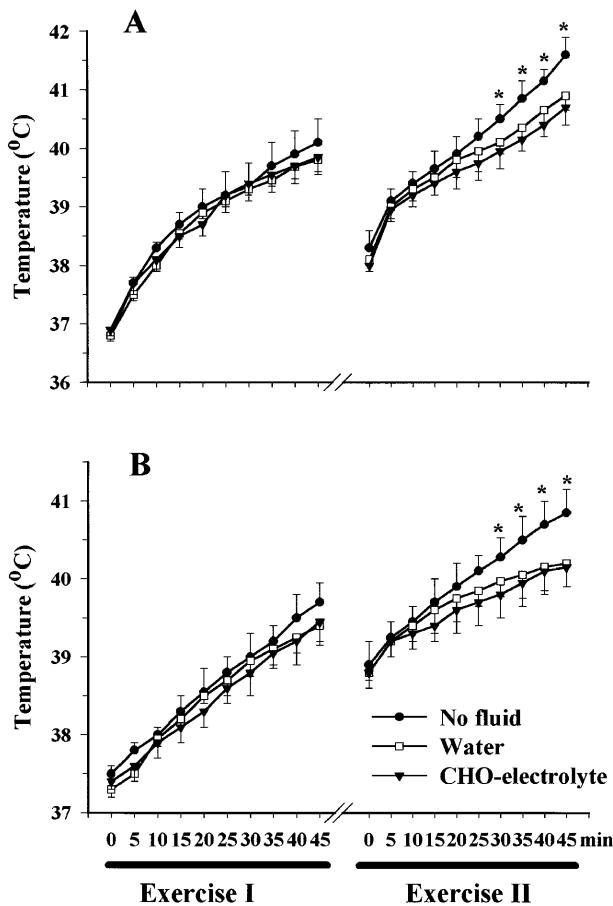


Fig. 3. Pulmonary artery blood (A) and rectal (B) temperature responses in 6 horses during 2 phases of exercise at 50% $\dot{V}O_{2max}$ with NF, W, or CE. Values are means \pm SE. * P < 0.05 from all other treatments at the same time point.

to the other treatments during *exercise I*. However, there were progressive decreases in \dot{Q} and SV during the final 30 min of *exercise II* such that values were significantly (P < 0.05) lower than in W and CE at 30 and 45 min of exercise in this bout.

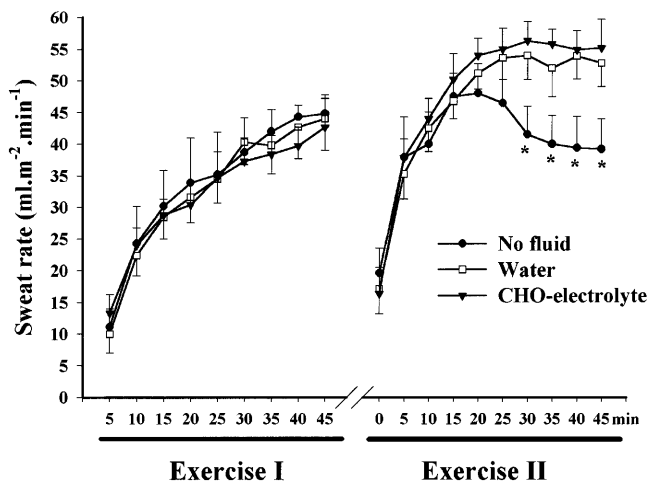


Fig. 4. Mean sweating rate on lateral thorax in 6 horses during exercise at 50% $\dot{V}O_{2max}$ with NF, W, or CE. Values are means \pm SE. * P < 0.05 from all other treatments at the same time point.

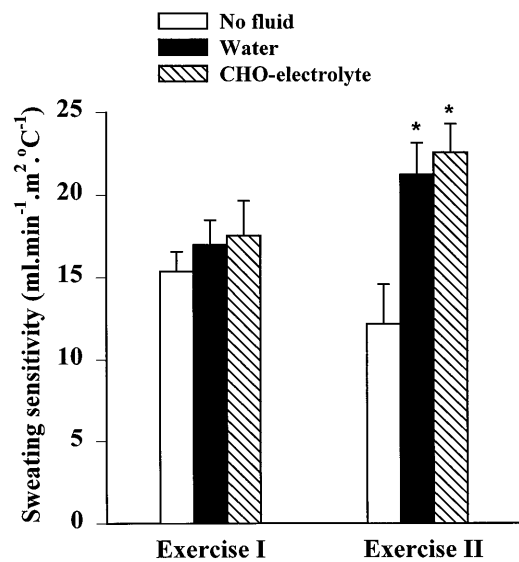


Fig. 5. Sweating sensitivity for the two 45-min bouts of exercise in 6 horses administered NF, W, or CE. * P < 0.05 vs. NF treatment in *exercise II*.

DISCUSSION

The major question addressed in this study was whether preexercise hyperhydration or attenuation of progressive dehydration would improve thermal and circulatory regulation in horses during moderate-intensity exercise in hot conditions. To answer this question, we have measured thermal, sweating, and cardiovascular responses during 90 min of exercise, with subjects receiving NF (control condition), W, or an isotonic CE solution before and at the midpoint of the trial. Under the moderately hot environmental conditions (34.5 \pm 0.3°C, 48 \pm 5% relative humidity), we hypothesized that evaporative heat loss (sweating) would be the primary mechanism for heat dissipation and, in subjects not provided fluid, the large sweat losses would result in substantial dehydration. Furthermore, we hypothesized that decrements in internal and peripheral heat transfer associated with progressive dehydration would exacerbate heat storage during exercise.

The most significant findings of the present study were 1) preexercise hyperhydration (W and CE in *exercise I*) provided no thermoregulatory advantage compared with NF; 2) maintenance of euhydration by oral administration of fluid (W and CE in *exercise II*) at a volume equivalent to \sim 85% of sweat fluid loss mitigated heat storage, as reflected by lower T_{pa} , T_{re} , and T_{mus} at the end of exercise; 3) maintenance of euhydration prevented decrements in local SR, sweating sensitivity, \dot{Q} , and SV associated with dehydration in the NF condition; and 4) W or an isotonic CE solution was equally effective in reducing physiological strain associated with exercise-induced dehydration and heat stress.

Few studies have examined the influence of preexercise fluid administration (hyperhydration) on thermoregulatory and cardiovascular responses in the horse during prolonged exercise. Sosa León and colleagues (37) administered fluid equivalent to 4% of body weight

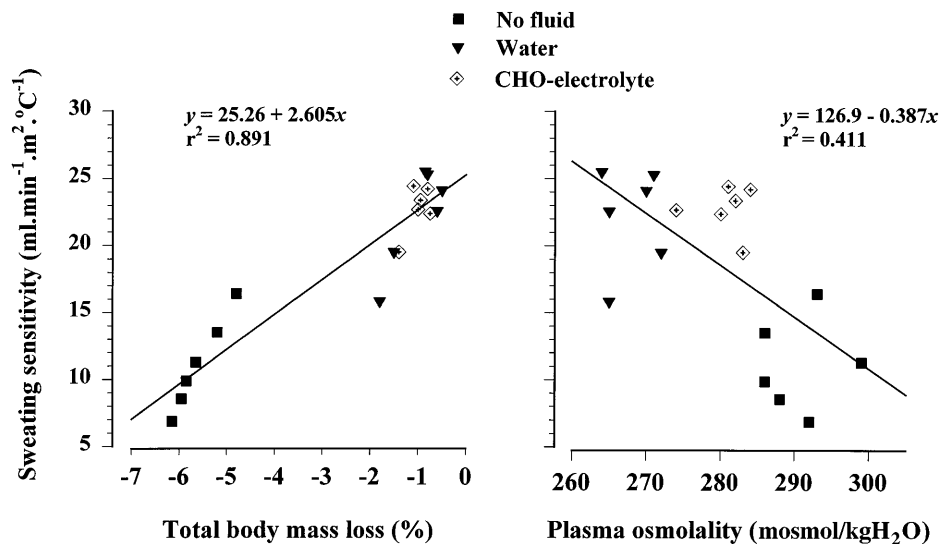


Fig. 6. Relationships between sweating sensitivity and both total body mass loss and final plasma osmolality during the second bout of exercise. Both linear regression equations were significant at $P < 0.005$.

90 min before an exercise test consisting of 90 min of low-intensity exercise (30% $\dot{V}O_{2\max}$). In that study, fluid-treated horses maintained higher PV during exercise compared with untreated controls, but there were no differences between treatments for cardiovascular and thermal variables. The work intensity and ambient conditions employed in the present study represented a much higher thermoregulatory strain, as reflected by end-exercise core temperatures (T_{pa}) that were ~ 1.5 – 2.5°C higher than those reported in the study by Sosa León et al. for work of similar duration. Despite the higher workload and more severe environmental conditions we found no evidence that preexercise hyperhydration reduced physiological strain. Similarly, Latzka et al. (22) reported that preexercise hyperhydration (water or glycerol) in human subjects did not provide any additional thermoregulatory advantage over maintenance of euhydration during compensable exercise-heat stress.

In human subjects, numerous investigations have demonstrated that even small amounts of dehydration (1–2% reduction in body weight) during moderate-intensity exercise can augment core temperature (4, 28, 34, 35). Furthermore, the magnitude of hyperthermia is directly related to the amount of dehydration accrued during moderate-intensity exercise (28). It also has been demonstrated that the optimal rate of fluid ingestion to attenuate hyperthermia and decrements in cardiovascular function is the rate that most closely matches the extent of fluid loss (8, 28, 34). While the dose of fluid administered in this study did not represent total fluid losses, when compared with NF, replacement of $\sim 85\%$ of sweat fluid losses was effective at attenuating hyperthermia at the exercise intensity and environmental conditions chosen for this experimental protocol.

Our results indicate that the fluid administered was absorbed before exercise and prevented the secondary hemoconcentration that occurred in NF after the initial decrease in PV after the onset of exercise. Furthermore, W or an isotonic CE solution were similarly effective for

attenuation of progressive dehydration during exercise. When no fluid was administered, hemoconcentration during exercise was reflected by progressive increases in hematocrit and plasma TS and osmolality during both bouts of exercise (Fig. 1, A and B). In contrast, there was little change in plasma TS and PV (Fig. 2) in W and CE between 5 and 45 min of exercise in the two exercise bouts, indicating that the administered fluid was retained in the vascular compartment. Although there was only a period of ~ 10 min between administration of the second dose of fluid and the onset of *exercise II*, it is likely that at least some of this fluid was absorbed. This likelihood is reflected in the further decrease in plasma osmolality in W at the onset of *exercise II* and minimal change in plasma total solids in W and CE throughout this bout of exercise. Considerable work has been undertaken in exercising human subjects to determine rates of gastric emptying and intestinal absorption of rehydration fluids of various formulations (7, 14, 21, 36), whereas there is little published information on these two processes in horses during exercise (24).

Although our horses were exercising at a moderate intensity, given the environmental conditions the subjects were unable to achieve a balance between heat production and dissipation during the first 45 min of exercise, as reflected by almost linear increases in T_{pa} and T_{re} . However, no difference in heat storage was evident between groups at the end of *exercise I*. In contrast, at the end of *exercise II*, the increase in T_{pa} and T_{re} was ~ 0.8 to 1.0°C greater in NF compared with W and CE. Evidence for greater heat storage in NF was also provided by the $\sim 1.0^\circ\text{C}$ higher value for T_{mus} at the end of exercise. A portion of the greater elevation in T_{core} in NF during *exercise II* could be attributed to a direct cooling effect of the fluid administered between *exercise I* and *exercise II* or a decrease in dissipation of heat (aerobic heat production was similar between trials). If we assume that the specific heat capacity of the horse is the same as for humans ($3.48 \text{ kJ}\cdot\text{kg}^{-1}\cdot^\circ\text{C}^{-1}$), the cooling effect of the administered fluid (tempera-

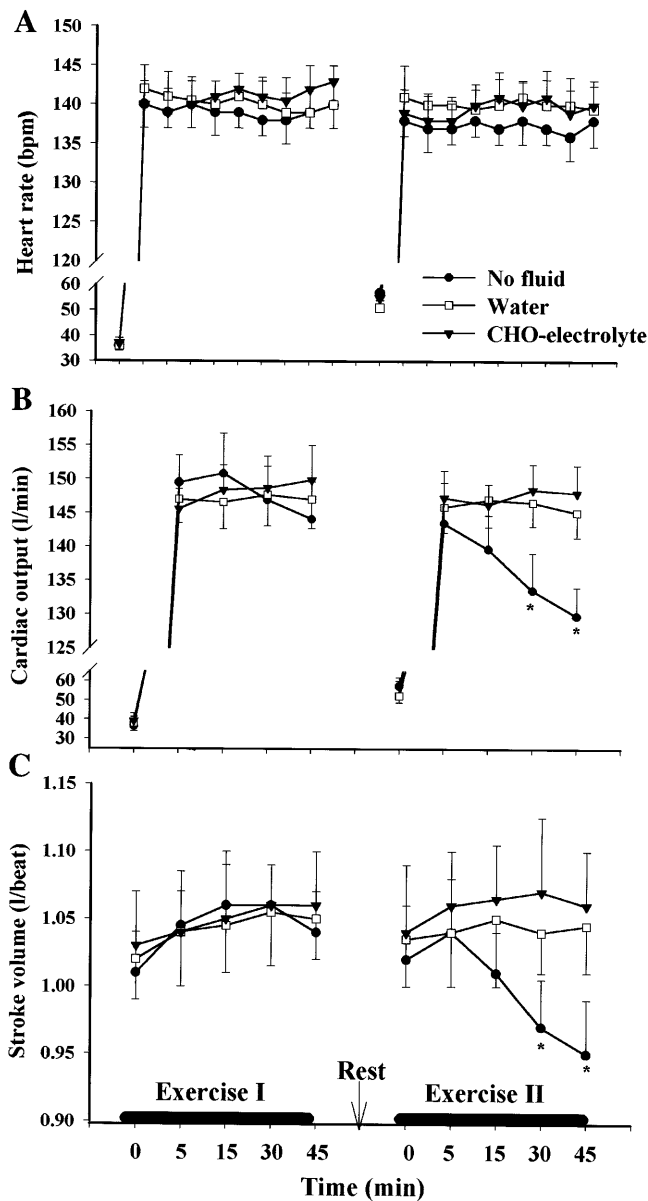


Fig. 7. Heart rate (A), cardiac output (B), and stroke volume (C) responses in 6 horses during exercise at 50% $\dot{V}O_{2\max}$ with NF, W, or CE. Values are means \pm SE. bpm, Beats/min. * $P < 0.05$ from all other treatments at the same time point.

ture $\sim 25^{\circ}\text{C}$) can be estimated on the basis of the specific heat capacity of water ($4.18 \text{ kJ} \cdot \text{kg}^{-1} \cdot ^{\circ}\text{C}^{-1}$) and body temperature at the time of fluid administration (T_{re} at the end of *exercise I*; Fig. 3B). From these calculations, we estimate that the cooling effect from the administered fluid would result in, at most, an $\sim 0.3^{\circ}\text{C}$ decrease in T_{core} . Given the $\sim 1^{\circ}\text{C}$ higher values for T_{pa} and T_{re} at the end of exercise in NF compared with W and CE, the greater heat storage must be partially due to a difference in the rate of heat dissipation between the NF and fluid treatment trials.

Studies in human subjects and other species have established two principal mechanisms by which dehydration results in a decrease in heat dissipatory capacity: a reduction in internal transfer of heat from the

body core to the periphery and decreased evaporative heat loss by decrements in SR and/or sweating sensitivity (1, 3, 8, 17, 28, 29, 34). In the present study, both factors may have contributed to a decrease in the rate of heat dissipation in NF during the latter stages of the exercise protocol. Notably, local SR and whole body sweat fluid loss were significantly lower in NF compared with W and CE during *exercise II* (Fig. 4, Table 2). Whereas local SR of $\sim 50\text{--}55 \text{ ml} \cdot \text{m}^{-2} \cdot \text{min}^{-1}$ were sustained by the horses in the fluid treatment trials, SR in NF declined during the last 30 min of *exercise II*. This decline in SR (and rate of cutaneous evaporative heat loss) was temporally related to the exacerbation of hyperthermia in the NF trial. These observations suggest that, at high thermal demands, preservation of euhydration is necessary for maintenance of high SR.

Sweating sensitivity also was significantly lower in NF than in W and CE during *exercise II* (Fig. 5). These data imply an alteration in sweating control, presumably as a result of the marked dehydration in the NF trial. In human subjects, both threshold temperature for onset of thermoregulatory sweating and sweating sensitivity are altered in a graded manner with hypohydration (29). Both extracellular hyperosmolality (12, 17, 29) and hypovolemia (11) have been implicated as mechanisms for altered sweat sensitivity in the hypohydrated state. In the present study, plasma osmolality increased only in NF and was inversely related to sweating sensitivity in *exercise II* ($P < 0.005$; Fig. 6, right). This observation agrees with previous reports in human subjects that have demonstrated an inverse relationship between the extent of plasma hyperosmolality and SR (12, 29, 35). Given the significantly greater PV loss in NF compared with W and CE (Fig. 2), hypovolemia also may have contributed to an alteration in sweating control. However, studies of exercising humans (11, 29, 34) and horses (19) have not demonstrated a consistent relationship between PV and sweating responses.

The altered sweating responses evident in the present study are in contrast to previous reports in exercising horses in which neither preexisting hypohydration ($\sim 3.5\%$ decrease in TBW) (30) nor progressive dehydration during prolonged exercise ($\sim 6\%$ of body mass) (19) altered local SR. However, in the study by Kingston et al. (19), horses were partially rehydrated during the exercise; partial replacement of body water losses may have mitigated any effect of the dehydration on thermoregulatory responses. Other factors in the aforementioned studies that may have contributed to the difference in sweating responses compared with the present study include a lower ambient temperature ($22\text{--}23^{\circ}\text{C}$) and a slightly lower rate of work ($40\% \dot{V}O_{2\max}$), which resulted in a slower rate of rise in T_{core} , and associated lower SR. The lower ambient temperature in the two previous studies would have reduced dependency on high SR and skin blood flow for heat dissipation; higher convective heat loss could alter the relationship between dehydration and hyperthermia. In cooler environmental conditions, in which there is less dependence on high SR and more potential for convective heat loss,

dehydration may occur without altering the magnitude of the hyperthermia accrued during exercise.

The greater heat storage in NF could also be attributed to dehydration-induced alterations in cardiovascular responses. In W and CE, maintenance of euhydration by fluid administration mitigated the ~10% decrease in \dot{Q} evident in NF during the last 30 min of exercise. Similar to the findings of a previous report describing cardiovascular responses of hypohydrated horses during moderate-intensity exercise (30), in the present study the decline in \dot{Q} in NF was attributed to a decrease in SV with minimal change in HR (Fig. 7, A–C). Alterations in skin blood flow and a dehydration-induced decrease in PV may, in part, explain the reduction in SV (15, 30, 33). Although we had no measures of skin blood flow or total peripheral (vascular) resistance, it is possible that the decline in \dot{Q} in NF was accompanied by a decrease in blood flow to the cutaneous circulation. In this circumstance, the decreases in \dot{Q} and skin blood flow would exacerbate hyperthermia via a reduction in transfer of heat from core to periphery. The decreases in \dot{Q} and SV evident in the NF trial are consistent with data from hypohydrated human subjects during exercise-heat stress (16, 28, 33). For example, Montain and Coyle (28) reported that the decline in SV with graded magnitudes of dehydration during exercise was proportional to the percent body weight loss and the reduction in forearm skin blood flow. Moreover, ingestion of fluid sufficient to replace 50–80% of the exercise-induced sweat loss attenuated hyperthermia by maintaining higher skin blood flow compared with trials in which either no or only a small amount of fluid was ingested (27, 28).

An interesting feature of the cardiovascular response to exercise-heat stress in the horses of the present study was the absence of a progressive increase in HR (“cardiovascular drift”). In human subjects, a progressive decrease in SV during submaximal exercise is accompanied by a compensatory increase in HR that serves to maintain or increase \dot{Q} (33). However, superimposition of dehydration on hyperthermia during exercise-heat stress causes greater reductions in SV and cardiovascular function, with an eventual decline in \dot{Q} despite a progressive increase in HR (16). By comparison, in the W and CE trials, HR, \dot{Q} , and SV were stable throughout the 90 min of exercise, whereas in NF the decline in SV during *exercise II* was not associated with an increase in HR (Fig. 7, A–C). Previous studies of horses during prolonged submaximal exercise have also demonstrated remarkable cardiovascular stability (9, 32), and it has been suggested that postural differences between horses (quadrupeds) and humans allow for better maintenance of “central” blood volume and cardiac filling pressure (9). Nonetheless, the decrease in SV observed in NF would be expected to invoke a compensatory increase in HR in an attempt to maintain \dot{Q} . In the absence of data for other cardiovascular variables, such as central venous and mean arterial pressures and skin blood flow, we are unable to offer a suitable explanation for this finding.

This study also compared the efficacy of water and an isotonic electrolyte solution for maintenance of euhydration and reduction of physiological strain during exercise-heat stress. On the basis of measured fluid intake and losses, we observed no difference in hydration between W and CE. Similarly, both fluid treatments were equally effective in mitigating hemoconcentration during exercise (Fig. 1, A and B). In human subjects, there is a strong association between plasma osmolality and thermoregulatory responses (8, 12, 17, 29). However, this relationship has not been demonstrated in the horse (18, 19). In the present study, although there were significant differences in plasma osmolality between the W and CE trials, both before and during exercise, there were no differences between fluid treatments in terms of thermoregulatory or cardiovascular responses. Some (3, 6), but not all (2), studies of human subjects have reported that PV is better maintained with the ingestion of isotonic electrolyte or carbohydrate-electrolyte solutions compared with water during exercise lasting 2–3 h. In the present study, although the CE fluid maintained PV at a higher level than did W during exercise, this difference was significant only at the end of *exercise II*.

In summary, we observed that 1) preexercise hyperhydration provided no thermoregulatory advantage compared with trials in which no fluid was administered; 2) maintenance of euhydration by oral administration of fluid at a volume equivalent to ~85% of sweat fluid loss mitigated heat storage, as reflected by lower T_{pa} , T_{re} , and T_{mus} at the end of exercise; 3) maintenance of euhydration prevented decrements in local SR, sweating sensitivity, \dot{Q} , and SV associated with progressive dehydration during exercise; and 4) oral administration of W or an isotonic CE solution was equally effective in reducing physiological strain associated with exercise-induced dehydration and heat stress.

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