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CIRCULATORY AND THERMOREGULATORY ACTIONS OF HYDRATION
DURING EXERCISE-HEAT STRESS(U) ARMY RESEARCH INST OF
ENVIRONMENTAL MEDICINE NATICK MA M N SAWKA ET AL

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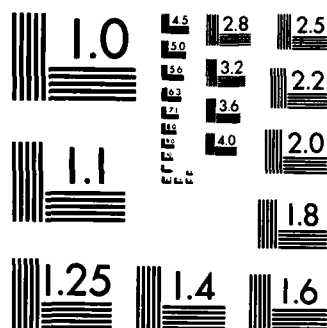
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CIRCULATORY AND THERMOREGULATORY ACTIONS OF HYDRATION DURING
EXERCISE-HEAT STRESS

Michael N. Sawka, Ralph P. Francesconi and Kent B. Pandolf

US Army Research Institute of Environmental Medicine,
Kansas Street, Natick, MA. 01760

Mailing Address: Dr. Michael N. Sawka
US Army Research Institute of Environmental Medicine
Kansas Street
Natick, MA 01760

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ABSTRACT

During exercise in the heat, sweat output often exceeds water intake resulting in hypohydration. Hypohydration during exercise causes a greater heat storage and reduces endurance in comparison with euhydration levels. The greater heat storage is attributed to a decreased sweating rate as well as decreased cutaneous blood flow. These response decrements are attributed to both plasma hyperosmolality and plasma hypovolemia. In addition, plasma hypovolemia will result in a reduced cardiac output response, relative to euhydration, during exercise-heat stress. Hyperhydration, or body fluid excess, only provides the advantage of delaying the onset of hypohydration.

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I. INTRODUCTION

Exercise performance in a hot environment has been shown to be primarily affected by state of heat acclimation (1), aerobic fitness (2), and hydration level (3). Heat acclimated individuals who are aerobically fit and fully hydrated will have less body heat storage and may achieve optimal performance during exercise-heat stress. Hydration level is particularly important during exercise in the heat because the body fluid deficit will neutralize the thermoregulatory advantages conferred by aerobic fitness (4) and heat acclimation (5, 6).

II. WATER LOSS

In hot environments, body fluid is primarily lost via eccrine sweat gland secretion which enables evaporative cooling of the body. For a given individual, sweating rate is dependent upon the environmental conditions, clothing and physical activity level (7). Hypohydration will occur if the volume of fluid ingested is less than sweat output. However, simple ad libitum water intake may not insure maintenance of euhydration. Several studies (8,9) have reported that ad libitum water intake results in incomplete fluid replacement or "voluntary" dehydration during exercise in the heat. A person's state of heat acclimation may also influence the level of "voluntary" dehydration incurred during exercise in the heat (8, 9). Eichna et al. (8), as well as Greenleaf et al. (9), reported that water deficits during exercise-heat stress were markedly reduced when subjects were heat acclimated. Therefore, heat acclimation may improve the relationship between thirst and body water needs.

If euhydration is not maintained, sweat loss will result in a reduction of total body water. Of particular importance for thermoregulation and exercise performance is the influence of hypohydration on the volume and composition of plasma fluid. Hypohydration has been consistently reported to reduce plasma

volume below euhydration levels (10, 11, 12), and the magnitude of this hypovolemia probably increases with the severity of hypohydration (10, 12). A recent study however, indicates that plasma volume is defended and does not continue to decrease at severe levels (7% reduction in body weight) of hypohydration (12).

Ecceine sweat is ordinarily hypotonic relative to plasma. Therefore, the plasma will become hyperosmotic when hypohydration is primarily mediated by sweat output. Plasma osmolality has been reported to increase from euhydrated levels of approximately 283 mosmol/kg to levels exceeding 300 mosmol/kg when hypohydration occurred by exercise-heat stress (12). Later, it will be demonstrated that the plasma hypovolemia as well as plasma hyperosmolality contribute to the less efficient thermoregulatory responses when hypohydration occurs during exercise-heat stress. The plasma hypovolemia and hyperosmolality also appear to be the triggering mechanisms for adaptive hormonal responses to conserve body fluids during dehydration. For example, our research indicates that the renin-aldosterone responses during exercise-heat stress mirror the changes in plasma volume induced by hydration levels (13, 14, 15).

III. PHYSIOLOGICAL EFFECTS

Hypohydration reduces maximal exercise performance (3, 6, 16) and this performance reduction increases with the magnitude of fluid deficit (3, 16). In addition, these maximal exercise performance decrements are potentiated by the presence of heat stress (3). The mechanisms responsible for hypohydration and heat stress mediated decrements in exercise performance probably reside in the cardiovascular system. The hypovolemia associated with hypohydration results in a decreased effective central blood volume and this reduces venous return and cardiac output during exercise (17,18). Likewise, the presence of heat strain

results in competition between central and peripheral circulation for the limited blood volume (17,18). As body temperature increases during exercise, cutaneous vasodilation occurs, which decreases venous resistance and pressure. These cardiovascular adaptations to heat strain appear to be consistent with the potentiated exercise performance decrement mediated by hypohydration with heat stress. Figure 1 presents the cardiovascular responses of subjects performing two prolonged exercise (70% of $\dot{V}O_2$ max) bouts that were spaced by a brief rest period. During the exercise bouts, the subjects were gradually dehydrated (to a 4% reduction in body weight) and incurred rectal temperatures in excess of 40°C (18). During these exercise bouts, heart rate increased but was unable to compensate for the falling stroke volume and therefore, cardiac output decreased.

Hypohydration during exercise causes a greater heat storage which elevates core temperature above euhydration levels (5,6,12,18,19). These elevated core temperature responses do not represent a "threshold" effect (3), but increase linearly with hypohydration level (12,19). The magnitude of the core temperature increment mediated by hypohydration is specific to the conditions of exercise intensity and to the environmental conditions (20). Figure 2 provides data of a typical subject's rectal temperature response to four hydration levels during standardized regimens of exercise-heat stress. Since hypohydration does not influence metabolic rate (12), the elevated core temperatures must be the result of decreased heat dissipation. The relative contribution of evaporative and dry heat exchange depends on the specific environmental condition however, both avenues of heat dissipation are adversely affected by hypohydration (3,12,17).

Figure 3 shows sweating rate responses plotted against rectal temperatures for individuals at four hydration levels during exercise-heat stress (12). The sweating rate responses were systematically decreased at a given core temperature with increased severity of hypohydration. The singular and combined effects of plasma hypovolemia and hyperosmolality have been suggested as mediating the reduced sweating rate response when hypohydrated (3). Figure 4 presents individual relationships of the change in sweating rate from euhydration levels during exercise to the plasma volume and osmolality changes from euhydration levels when hypohydrated (12). Elevated plasma osmolality provided a strong and consistent relationship with reduced sweating rate responses. This finding is consistent with the data of Harrison *et al.* (21) who reported that plasma hyperosmolality will elevate core temperature response during exercise-heat stress despite the maintenance of euhydration. Hyperosmolality may have a direct CNS effect on the hypothalamic thermoregulatory centers or a peripheral effect on the eccrine sweat gland (3).

Plasma volume loss provides a weaker and less consistent relationship (than hyperosmolality) with the reduced sweating rate responses. Fortney *et al.* (22) have provided evidence that an isosmotic hypovolemia (~15% plasma volume reduction) caused a reduced sweating rate during exercise in the heat. Their observation is in agreement with Figure 4, which suggests that plasma volume reductions of about 10-15% are necessary for consistent reductions of sweating rate. Fortney *et al.* (22) theorized that hypovolemia may unload atrial baroreceptors which have afferent input to the hypothalamic thermoregulatory centers which control sweating rate. Similarly, Nadel *et al.* (17) reported that an isosmotic hypovolemia (~18% plasma volume reduction) will reduce cutaneous blood flow for a given core temperature and thus, the potential for dry heat

exchange. These high levels of hypovolemia induced by diuretics (17,22) appear to be greater than those levels generally observed to be elicited by exercise-heat dehydration for moderate (up to 5% reduction in body weight) hypohydration levels (6,10,12,18).

If hypohydration reduces performance, can excess body fluids improve performance beyond the levels achieved when euhydrated? If hyperhydration does improve performance during exercise-heat stress, these improvements would most likely be mediated by hypervolemia or expanded plasma volume (3). Several recent studies on the effects of artificially expanded plasma volume have indicated no differences in core temperature (17,23), sweating rate (22,23), cardiac output (17), arterial blood pressure (23), or peripheral blood flow (17) during exercise-heat stress. These data indicate that hyperhydration does not provide a thermal advantage during exercise-heat stress, but will delay the development of hypohydration.

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FIGURE LEGENDS

Figure 1. Heart rate, stroke volume, cardiac output and arteriovenous oxygen difference responses of subjects during two prolonged runs spaced by a brief rest period. During these exercise bouts the subject experienced progressive dehydration and hyperthermia. *for $P < 0.05$ and ** $P < 0.01$.

Figure 2. A representative subject's rectal temperature response to exercise-heat stress when euhydrated (0%) and hypohydrated by 3%, 5% and 7% of his body weight.

Figure 3. A plot of group mean sweating rate and rectal temperature responses to exercise-heat stress when euhydrated (0%) and hypohydrated by 3%, 5% and 7% of their body weight.

Figure 4. Individual relationships between the change in sweating rate from euhydration levels, with the change in plasma volume and change in osmolality from euhydration levels.

Fig. 1.

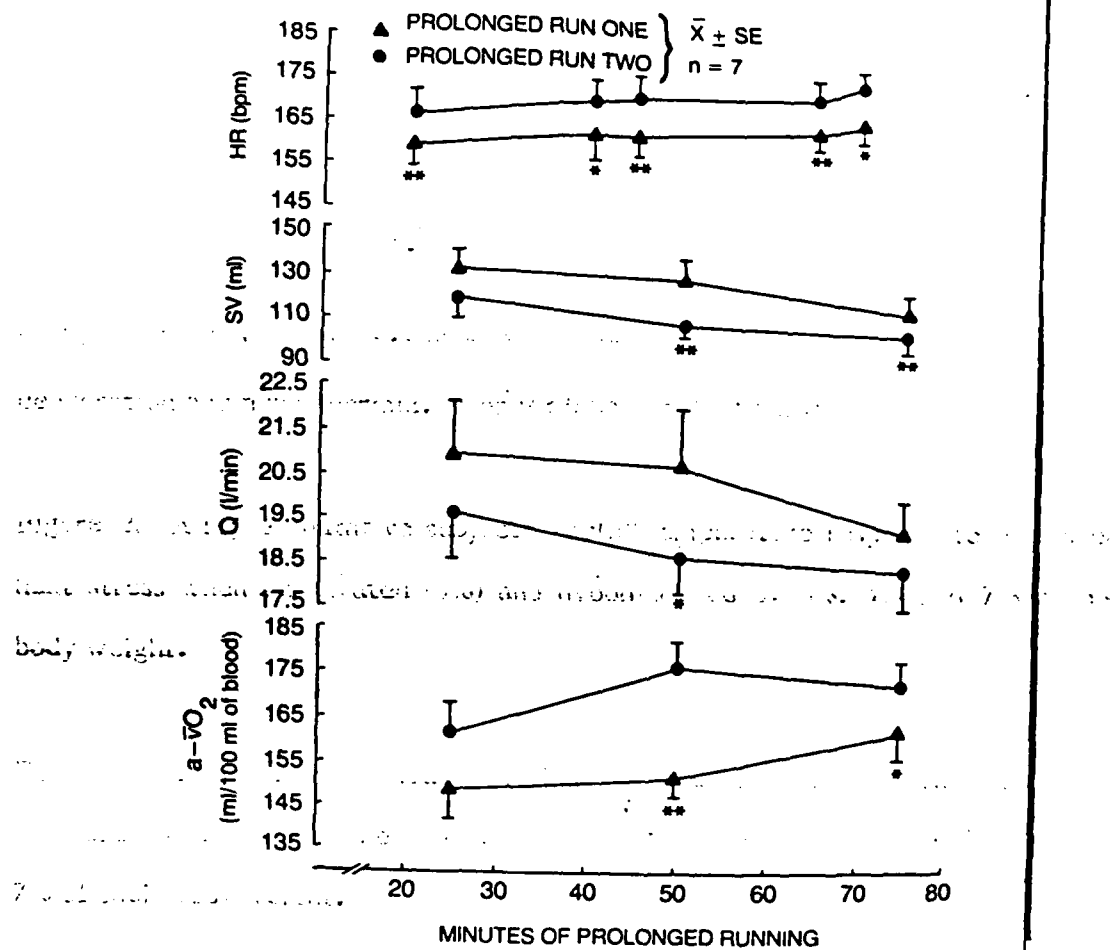


Fig. 2

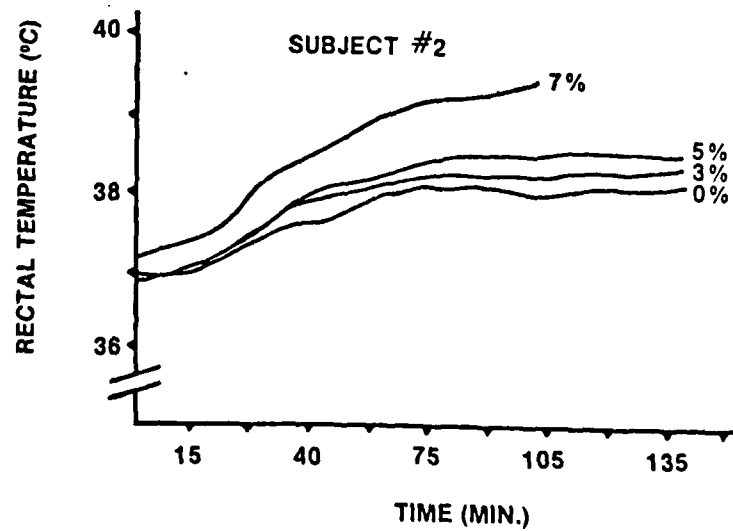


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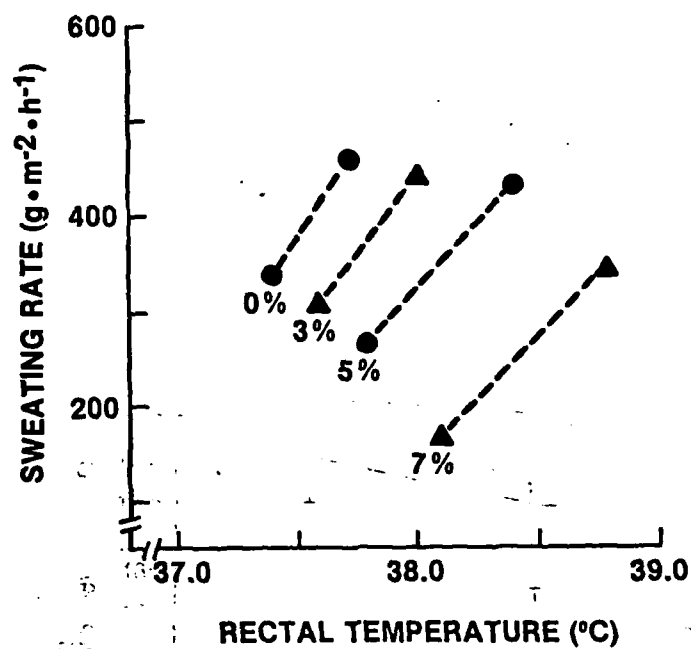
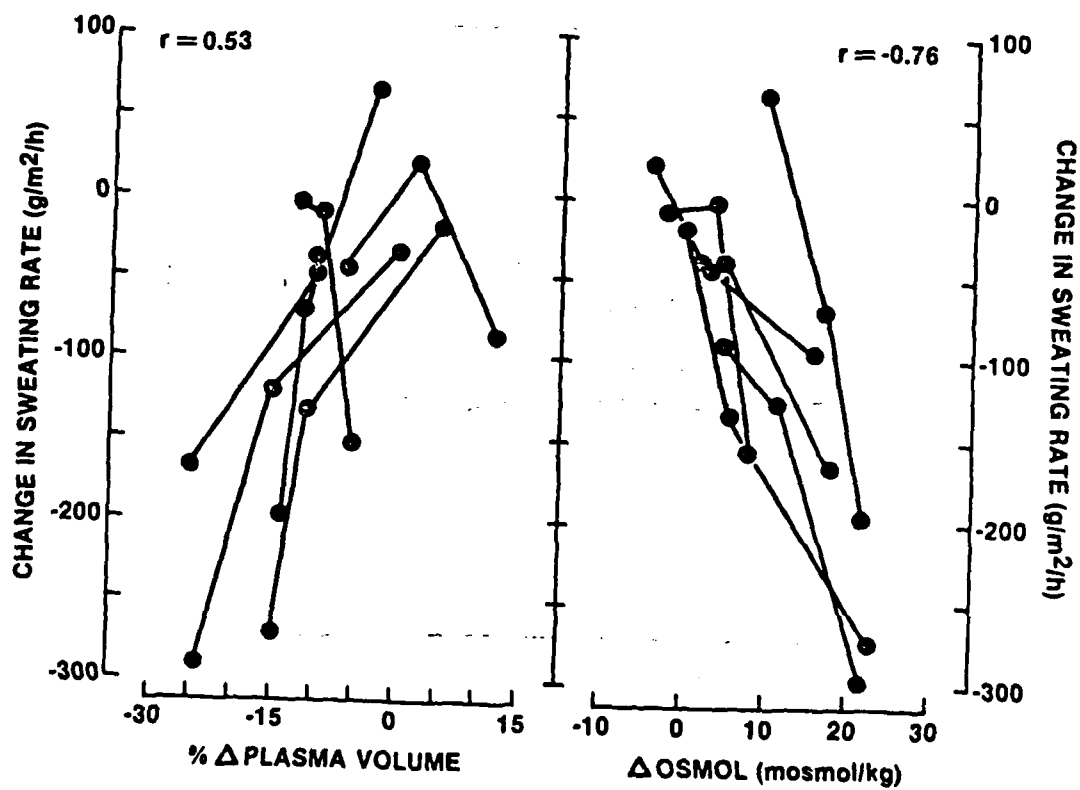


Fig. 4.



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