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HYDRATION EFFECTS ON HUMAN PHYSIOLOGY AND EXERCISE-HEA PERFORMANCE

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U S ARMY RESEARCH INSTITUTE
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ENVIRONMENTAL MEDICINE
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During exercise in the heat, sweat output often exceeds water intake resulting in hypohydration, which is defined as a body fluid deficit. This fluid deficit is comprised of water loss from both the intracellular and extracellular fluid compartments. There is no evidence that hypohydration can benefit exercise performance; in addition, man cannot adapt to chronic dehydration. Exercise tasks that primarily require aerobic metabolism and that are prolonged will more likely be adversely influenced by hypohydration than exercise tasks that require anaerobic metabolism as well as muscular strength and power. Likewise, the warmer the environmental temperature, the greater the potential for hypohydration to cause decrements in all types of exercise performance. Hypohydration causes a greater heat storage and reduces endurance as well as maximal effort exercise performance in comparison to euhydration levels. The greater heat storage is mediated by a decreased sweating rate (evaporative heat loss) as well as by a decreased cutaneous blood flow (dry heat loss). These response decrements have been attributed to both a plasma hyperosmolality and a reduced blood volume. The reduced blood volume also makes it difficult to maintain an adequate cardiac					
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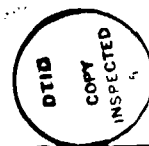
→ output during exercise-heat stress. Finally, preliminary data indicate that hypohydration does not alter muscle glycogen utilization during exercise or the glycogen resynthesis during recovery from exercise.

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Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.



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HYDRATION EFFECTS ON HUMAN PHYSIOLOGY
AND EXERCISE-HEAT PERFORMANCE

by

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ABSTRACT

During exercise in the heat, sweat output often exceeds water intake resulting in hypohydration, which is defined as a body fluid deficit. This fluid deficit is comprised of water loss from both the intracellular and extracellular fluid compartments. There is no evidence that hypohydration can benefit exercise performance; in addition, man cannot adapt to chronic dehydration. Exercise tasks that primarily require aerobic metabolism and that are prolonged will more likely be adversely influenced by hypohydration than exercise tasks that require anaerobic metabolism as well as muscular strength and power. Likewise, the warmer the environmental temperature, the greater the potential for hypohydration to cause decrements in all types of exercise performance. Hypohydration causes a greater heat storage and reduces endurance as well as maximal effort exercise performance in comparison to euhydration levels. The greater heat storage is mediated by a decreased sweating rate (evaporative heat loss) as well as by a decreased cutaneous blood flow (dry heat loss). These response decrements have been attributed to both a plasma hyperosmolality and a reduced blood volume. The reduced blood volume also makes it difficult to maintain an adequate cardiac output during exercise-heat stress. Finally, preliminary data indicate that hypohydration does not alter muscle glycogen utilization during exercise or the glycogen resynthesis during recovery from exercise.

I. INTRODUCTION

The distribution of body water and the ability to maintain blood volume as well as plasma osmolality are critical factors influencing man's ability to perform exercise in the heat. Water is necessary to support metabolism as it provides the medium for biochemical reactions and body solutes. Likewise, water is the primary component of plasma, which is essential to maintain blood volume. Muscular exercise will routinely increase metabolic rate by 5 to 15 times above resting levels and require an adequate blood volume to sustain the simultaneous perfusion of the metabolically active skeletal muscles and cutaneous vasculature. The increased cutaneous circulation enhances radiative and convective (dry) heat loss. Also, plasma supplies the precursor fluid for sweat. Therefore, sweat secretion can reduce the blood volume and make it more difficult for the cardiovascular system to provide an adequate cardiac output during the combined stress of exercise and heat.

Individuals in the military, occupational and athletic settings may have to perform physical exercise while incurring a body water deficit. Generally, the individual dehydrates during exercise because of fluid nonavailability or a mismatching between the body's sensation for thirst and the body's water requirements. In these instances, the individual begins the exercise task euhydrated but then incurs a progressive dehydration over a prolonged period of time. This scenario is common for many occupational and athletic settings; however, in the military setting, particularly combat, the individual will often start the exercise task hypohydrated (33). In the military, individuals might also dehydrate to achieve their weight standard (AR 600-9) which is evaluated prior to semi-annual physical fitness tests. There are also several sports (e.g., boxing, power lifting, wrestling) where the athlete may purposely achieve hypohydration prior to competition (139,147). These athletes desire to compete in a

lower weight class to gain a size advantage over their opponents. In these instances, hypohydration might be achieved by a combination of restricted water and food intake, exercise-heat dehydration and/or the use of diuretics (18,135). Finally, some individuals in military and occupational settings may take diuretics as medication, and as a result routinely have a body water deficit when performing physical work.

The military was the first to recognize the importance of water on man's ability to perform exercise, and major battles have been decided by the availability of water to the combatant troops. For example, dehydration induced heat stroke was believed to be responsible for 20,000 deaths among Egyptian troops during the 1967 six-day war with Israel (63). The recognition of the importance of adequate hydration has resulted in military funding of comprehensive research programs concerning this topic, first at the Harvard Fatigue Laboratory (60), then at the Armored Medical Research Laboratory (1), the Quartermaster Research Command, and now at the U.S. Army Research Institute of Environmental Medicine (43). Research from those institutions has led to the military changing their interpretation of the "water doctrine" from "withholding water for the toughening of troops" to the current "forced drinking". The genesis of this current viewpoint required the dispelling of several myths concerning the benefits of water deprivation (68). Unfortunately, myths may persist for some individuals responsible for supervising physical training. For example, during the summer of 1988, Massachusetts State Police recruits were limited access to water during training sessions, and 11 of the 50 recruits received serious heat injuries and were hospitalized; two subsequently underwent kidney dialysis, and one recruit required a liver transplant and later died (22).

This Technical Report will provide a comprehensive review concerning human hydration status and exercise performance. It will primarily address the question of the magnitude of body water deficit that adversely affects exercise performance, and the physiological mechanism(s) responsible for the performance decrements. It should become evident, upon reading this report, that the extent of performance decrement is related to how the water is lost, the magnitude of water deficit, the fluid containing space that predominantly sustains the loss, the type of exercise and the thermal environment to which the individual is exposed. For each combination of exercise and environment, there is a critical level of water deficit at which exercise performance will be adversely affected.

Throughout this report, the term "euhydration" will refer to "normal" body water content; whereas, "hypohydration" will refer to body water deficit. The more common term "dehydration" will denote the dynamic loss of body water or the transition from euhydration to hypohydration. The term "hypovolemia" will define a steady-state blood volume that is less than normal.

II. BODY WATER LOSS

Physical exercise will routinely increase total body metabolism in order to meet the increased energy demands of skeletal muscle contraction. Depending upon the exercise task, between 70 and 100% of the metabolic rate results in heat which needs to be dissipated in order to restore body heat balance. Depending on the environmental temperature, the relative contributions of evaporative and dry heat exchange to the total heat loss vary, so that the hotter the environment the greater the dependence on evaporative heat loss, and thus on

sweating (91). Therefore, in hot environments, a considerable amount of body water can be lost via eccrine sweat gland secretion which enables the evaporative cooling of the body (146).

For a given person, the sweating rate is dependent on environmental conditions (ambient temperature, dew point temperature, radiant load and air velocity), clothing (insulation and moisture permeability), and the physical activity level (1,77,125,137). Adolph and associates (1) reported that for 91 men studied during diverse military activities in the desert, the average sweating rate was 4.1 L every 24 hours, but values ranged from 1 to 11 L every 24 hours. Strydom and colleagues (137) reported that for 60 men studied during a forced field march, carrying backpacks and rifles in a warm environment, the average water requirement was 7.0 L every 24 hours. Table 1 provides a detailed description of these water requirements. These investigators noted that ~1 L of water was provided in the food, so that only 6 L of fluid needed to be provided in the form of beverage.

Table 1. Water requirements of men living and working in the heat (~32° C, 30% rh) (137).

A. SLEEPING	—————	8 HOURS	= 0.5 L
B. SEMI ACTIVE	—————	8 HOURS	= 1.0 L
C. MARCHING 6.8 kph WITH PACKS	—————	5 HOURS	= 4.6 L
D. PREPARATION ACTIVITIES	—————	3 HOURS	= 0.9 L
TOTAL		= 24 HOURS	= 7.0 L

The water requirements of soldiers on the modern battlefield may often exceed the levels suggested by earlier military studies. The threat of chemical warfare may require military personnel to wear nuclear-biological-chemical (NBC) protective clothing. Characterized by low moisture permeability and high insulating properties ($\text{clo} \sim 2.6$; $i_{\text{a}}/\text{clo} \sim 0.1$) NBC clothing, which prevents noxious agents from reaching the skin, also prevents the normal dissipation of body heat generated metabolically or gained from the environment. As a result, both core and skin temperatures can become excessively elevated and result in high levels of sweat output, which cannot evaporate within the garments. For example, during light intensity ($\sim 150\text{--}400\text{W}$) exercise in hot environments soldiers wearing NBC clothing will routinely have sweating rates of 1 to 2 $\text{L}\cdot\text{h}^{-1}$ (80,99,126).

For athletes, the highest sweating rates occur during prolonged high intensity exercise in the heat. Figure 1 provides (114) an approximation of hourly sweating rates and therefore water requirements for runners based upon metabolic rate data from several laboratories. The sweating rates were predicted by the equation developed by Shapiro *et al.* (125). The amount of body fluid lost as sweat can vary greatly, and sweating rates of 1 $\text{L}\cdot\text{h}^{-1}$ are very common. The highest sweating rate reported in the literature is 3.7 $\text{L}\cdot\text{h}^{-1}$, which was measured for Alberto Salazar during the 1984 Olympic Marathon (6).

During physical exercise in the heat, the principal problem is to closely match the volume of fluid consumed to the volume of sweat. This is a difficult problem to overcome, since thirst provides a poor index of body water requirements (1,38). Numerous investigators (1,10,98) report that ad libitum water intake results in incomplete water replacement or "voluntary" dehydration during exercise and/or heat exposure. It is common for individuals to "voluntarily" dehydrate by 2 to 8% of their body weight during exercise-heat stress, despite

adequate amounts of fluid available for rehydration (1,6,7,15,64,102). The flavoring (64,78) and cooling (7) of the ingested fluid increases its palatability, which can help to minimize "voluntary" dehydration.

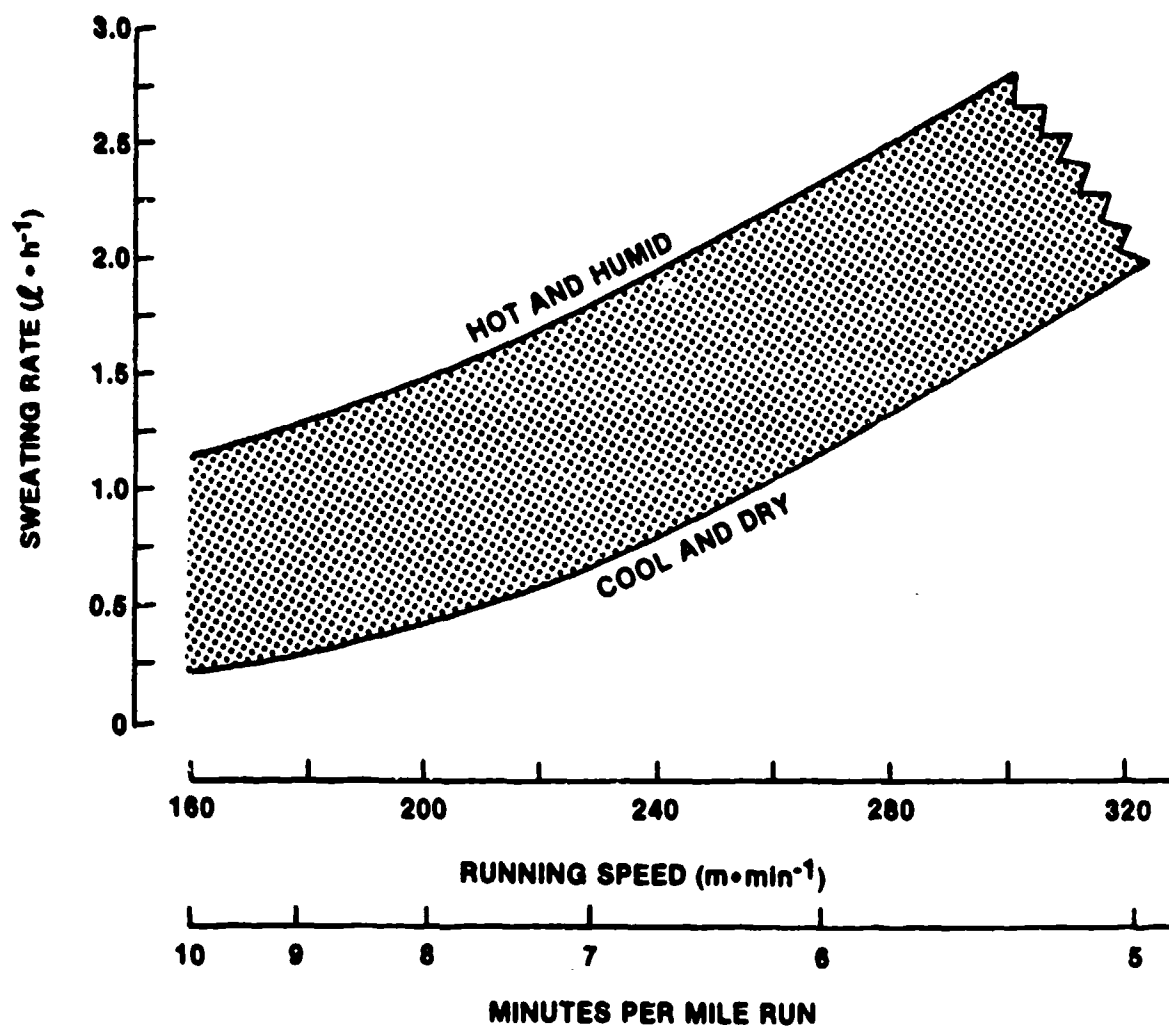


FIGURE 1. An approximation of the hourly sweating rates for runners (114).

A person's heat acclimation status may also influence the "voluntary" dehydration incurred during exercise in the heat. Figure 2 presents data redrawn from Eichna *et al.* (36) on the "voluntary" dehydration (water deficit) incurred during exercise in a cool environment and during six consecutive days in a hot environment. The water deficit was much greater during the initial exercise-heat exposures. In agreement with these findings, Greenleaf *et al.* (50) reported that water deficit during exercise-heat stress was reduced by approximately 30% after the initial four days of heat acclimation. Therefore, heat acclimation can improve the relationship between thirst and body water needs. Although heat acclimation improves the relationship between thirst and body water requirements (36,50), "voluntary" dehydration will still occur (72).

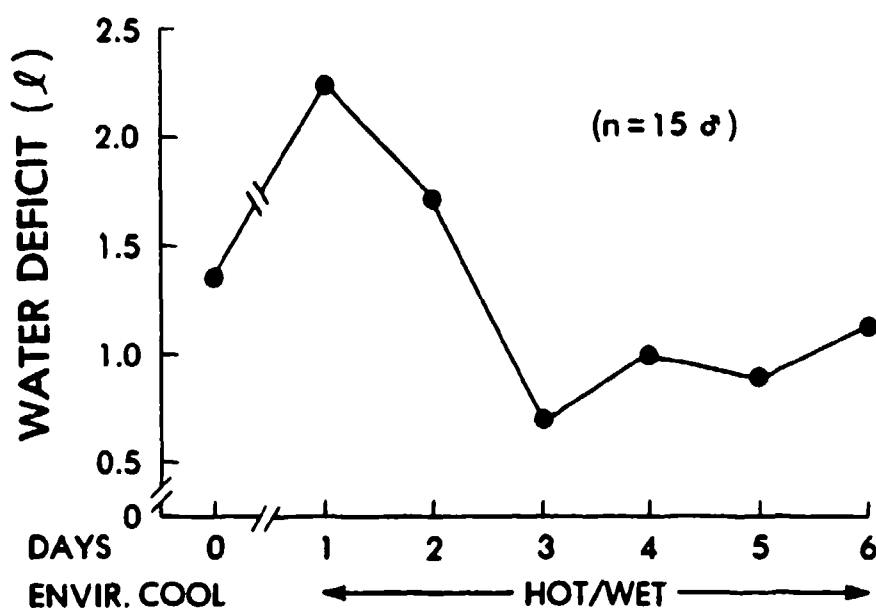


FIGURE 2. Difference between ad libitum water intake and water loss during work in the heat (data from 36 as presented in 108).

Thirst is probably not perceived until an individual achieves a water deficit of ~2% of his body weight (1). Likewise, as previously discussed, thirst is a poor index of body water requirements such that ad libitum water intake during exercise in the heat results in an incomplete replacement of body water losses (1). As a result, it is likely that unless forced hydration is stressed, some "voluntary" dehydration will occur during exercise in the heat. Neuffer et al. (85) recently reported that hypohydration will reduce the gastric emptying rate of ingested fluids during exercise in the heat. They found a 20 to 25% reduction in gastric emptying rate when their subjects were hypohydrated (5% body weight); and the reduction was related to the increased thermal strain (Figure 3). Therefore, forced hydration during the early stages of exercise-heat stress is important, not only to avoid "voluntary" dehydration, but also to maximize the bioavailability of the ingested fluids.

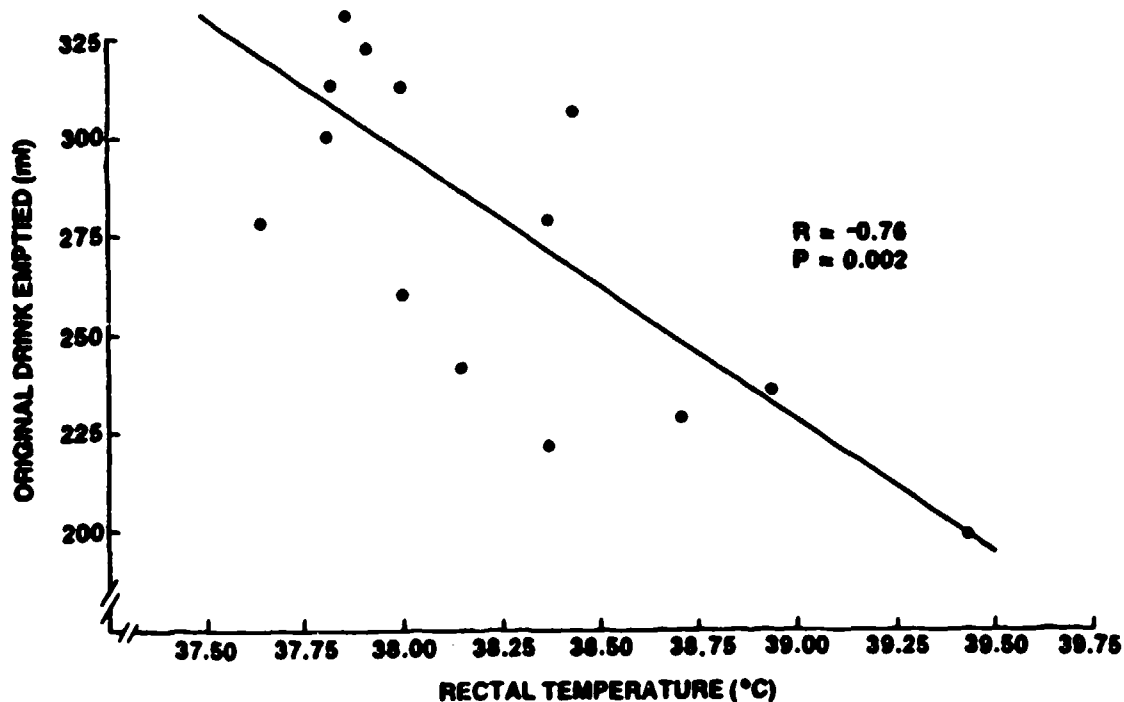


FIGURE 3. Correlation of final exercise rectal temperatures (°C) recorded vs the corresponding volume (ml) of gastric fluid emptied (original drink emptied) for each exercise session (85).

The maximal gastric emptying rate is 1 to 1.5 liters per hour (84,85,95); this rate can be reduced by high intensity exercise (84), heat stress (85,95) and hypohydration (85). Soldiers wearing NBC protective clothing and athletes performing high intensity exercise, can elicit sweating rates which exceed their gastric emptying rate. During these situations, the volume of fluid intake should not match the volume of sweat output as excessive water in the stomach might lead to gastric distress.

III. BODY WATER DISTRIBUTION AND REDISTRIBUTION

Water is the largest component of the human body and represents 45 to 70 percent of body weight. Table 2 provides the water content of various body tissues for a 75 kg man (131). As indicated in the Table 2, the composition of an average-sized man includes about 45 L of water, which corresponds to about 60 percent of his body weight. It is important to note that adipose tissue is approximately 10 percent water, while muscle tissue is approximately 75 percent water. Therefore, individual differences in total body water are primarily the result of differences in skeletal muscle or lean body mass. Athletes have a greater percentage of total body water than their sedentary counterparts by virtue of a greater skeletal muscle mass and lower percent body fat; likewise, gender and age differences in total body water are related to difference in lean body mass (27,56,119). This relationship enables an accurate estimation of total body water at approximately 72 percent of an individual's lean body mass (75).

Table 2. Water content of various body tissues for a 75 kg man (131).

TISSUE	% WATER	% OF BODY WEIGHT	LITERS OF WATER PER 75 kg
SKIN	72	18	9.72
ORGANS	76	7	3.99
SKELETON	22	15	2.47
BLOOD	83	5	3.11
ADIPOSE	10	12	0.90
MUSCLE	76	43	24.51

The water contained in these body tissues is distributed between the intracellular and extracellular fluid compartments. The intracellular fluid compartment is larger and contains about 30 L of water, whereas the extracellular fluid compartment contains about 15 L of water for an average 75 kg male. Figure 4 presents a schematic of the approximate volume of water in each compartment, and the mechanisms for the exchange of fluid between the compartments. At rest, and probably more so during exercise, there is continuous water exchange between fluid compartments, and the volumes depicted represent only net values.

Sweating reduces total body water if adequate amounts of fluid are not consumed. As a consequence of free fluid exchange, hypohydration should affect each fluid space. Figure 5 illustrates the findings of two studies (28,34) on the redistribution of water between fluid spaces when hypohydrated (106). Costill *et al.* (28) dehydrated subjects by using a

combination of cycle ergometer exercise and heat exposure. Shortly after completing cycle ergometer exercise, blood and skeletal muscle samples were obtained from their subjects. Their data may be biased by the cessation of cycle ergometer exercise immediately prior to estimation of water distribution between fluid compartments. Durkot *et al.* (34) dehydrated rats by using a passive heat exposure for several (5 to 11) hours and indicated that their data were slightly biased by the animals' appetite loss. The intent of Figure 5 is to present data trends, and not to imply that a given percent decrease of total body water is similar between man and rat. At low volumes of body water loss, the water deficit primarily comes from the extracellular space, and the body water loss increases, a proportionately greater percentage of the water deficit comes from the intracellular space.

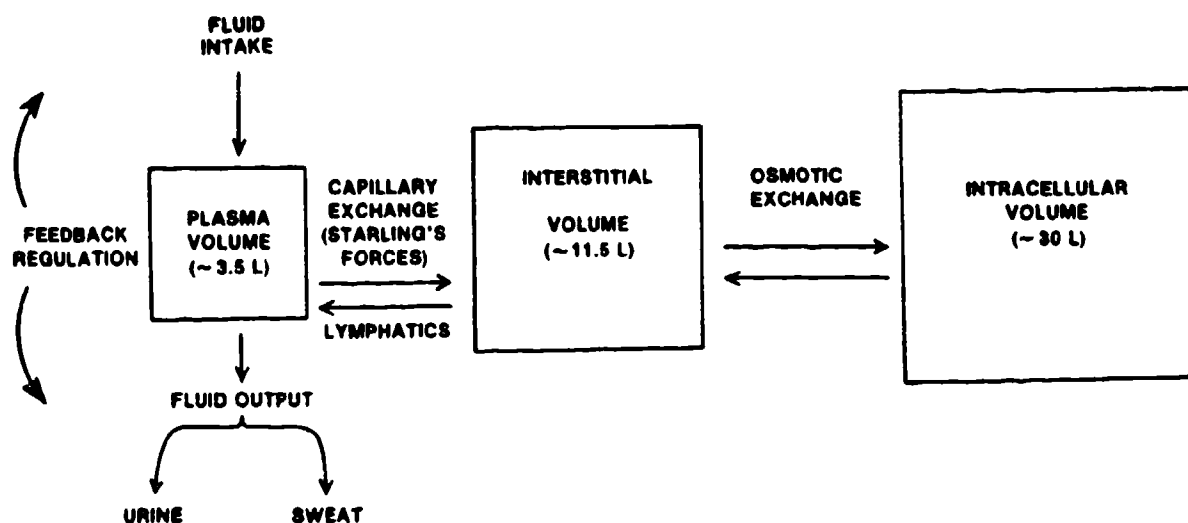


FIGURE 4. Schematic of the approximate volume of water and the mechanisms for its exchange between the fluid compartments (106).

Nose and colleagues (93) attempted to determine the distribution of body water loss not only among the fluid spaces, but also among different body organs. These investigators thermally dehydrated rats by 10% of body weight, and after the animals regained their normal core temperature, the body water measurements were obtained. Figure 6 presents a summary of their findings. The water deficit was apportioned between the intracellular (41%), and extracellular (59%) spaces; and among the organs, 40% from muscle, 30% from skin, 14% from viscera and 14% from bone. Neither the brain nor liver lost significant water content. It was concluded that hypohydration results in water redistribution largely from the intra- and extracellular spaces of muscle and skin in order to maintain blood volume. Homeostatic mechanisms seem to be present which defend the water content of organs (e.g., brain, liver) that are necessary to maintain life.

The method by which dehydration is induced may affect the partitioning of the remaining water between the body fluid spaces (71). For example, it has been estimated that 3-4 g of water is bound with each gram of glycogen (94), although this relationship is variable (127). Regardless, several papers (28,29,20) have reported a substantial decrease of intracellular water content in skeletal muscle following 1.5 to 2.5 h of exercise dehydration. These investigators suggest that the loss of intracellular water may be the result of water released with the breakdown of muscle glycogen. Thus, exercise-induced hypohydration may result in a greater intracellular water loss than thermally-induced hypohydration. Kozlowski and Saltin (71) have reported data to support this view, but Costill and Saltin (30), found no difference between exercise and thermal dehydration for the partitioning of water between the fluid spaces. Therefore, whether or not exercise and thermally induced hypohydration cause a difference in the redistribution of water between fluid spaces remains unresolved.

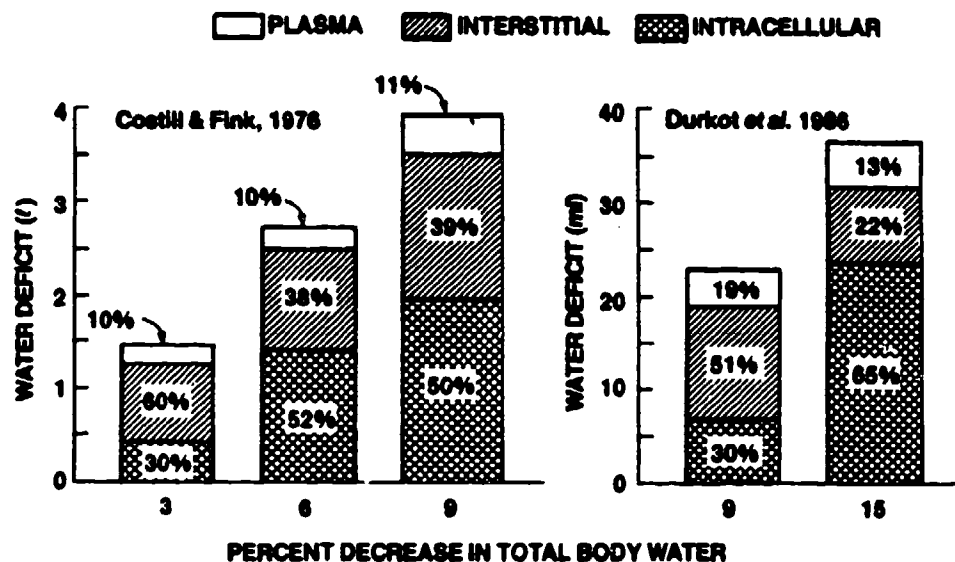


FIGURE 5. The findings of two studies (28,34) concerning the partitioning of water loss between fluid spaces during resting conditions (presented in 106).

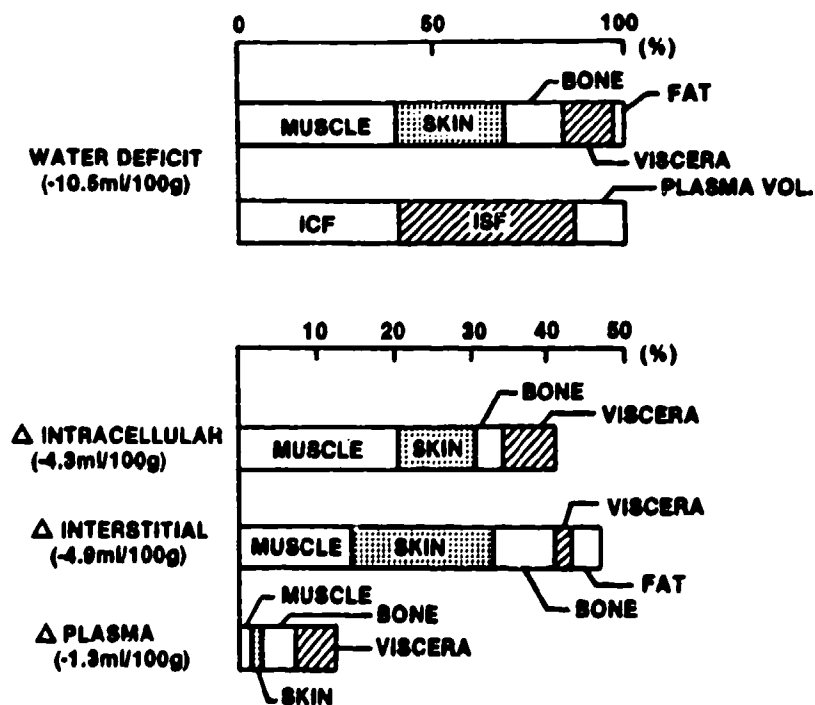


FIGURE 6. Partitioning of water loss between the body fluid spaces and different body organs (redrawn from 93).

The redistribution of water between the intracellular and the extracellular space is dependent upon the osmotic gradient between these spaces. Cell membranes are freely permeable to water but only selectively permeable to various solutes. As a result, transient alterations in the solute concentration cause water redistribution across the cell membrane until the two fluid spaces are in equilibrium with respect to osmolality. Therefore, if the methods used to attain hypohydration lead to differences in the intracellular and/or extracellular solute losses, the partitioning of water loss between the fluid spaces will vary accordingly.

It is known that exercise or heat-induced hypohydration will increase the osmotic pressure in the plasma (122). Eccrine sweat is ordinarily hypotonic relative to plasma (66); therefore, the plasma becomes hyperosmotic when dehydration is induced by sweat output (106,121). For resting subjects, plasma osmolality can increase from about 283 mosmol·kg⁻¹ when euhydrated to values exceeding 300 mosmol·kg⁻¹ when hypohydrated (Figure 7). Sodium, potassium, and their principal anions (chloride), are primarily responsible for the elevated plasma osmolality during hypohydration (73,120). As re-emphasized by Nose *et al.* (92), it is the plasma hyperosmolality which mobilizes fluid from the intracellular to the extracellular space to enable the defense of the blood (plasma) volume in hypohydrated subjects.

The plasma electrolytes, sodium and chloride, that are lost with sweat, are primarily found in the extracellular fluid (106). The amount of electrolytes that are lost in sweat can be modified by an individual's state of heat acclimation (3,66). For example, Kirby and Convertino (66) reported that over the course of heat acclimation the sweat sodium concentration decreased, so despite a 12% increase of sweating rate the sodium losses decreased by 59%. Therefore, for a given sweating rate the heat acclimated individual loses less

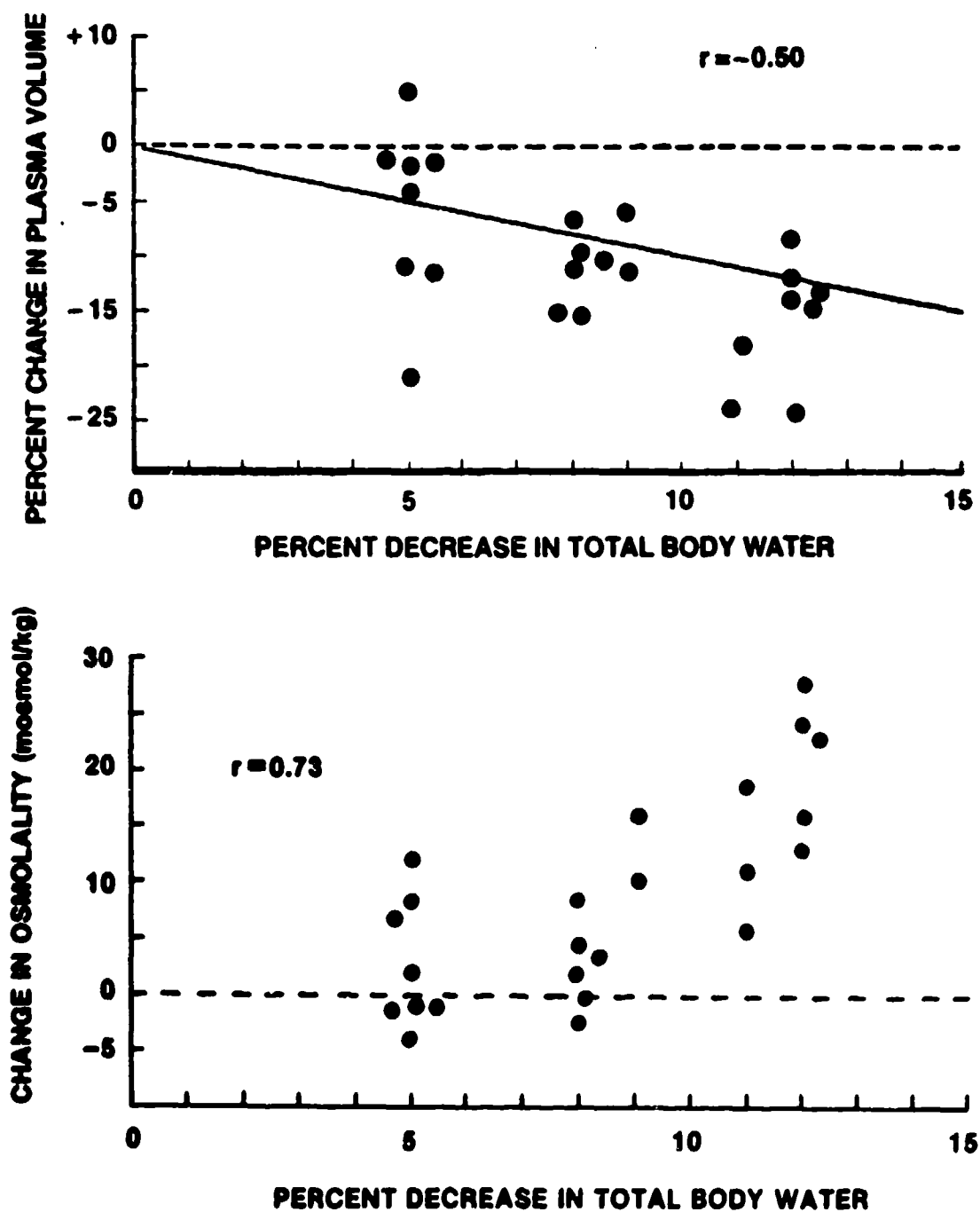


FIGURE 7. Individual data on the plasma volume reduction and increase in osmolality associated with a given level of body water loss (106). These data represent heat acclimated subjects while at rest.

solute from the plasma. It would seem logical that for a given amount of body fluid loss via sweat, in a heat acclimated individual, a greater osmotic gradient would move fluid from the intracellular to the extracellular space as compared to an unacclimated individual. The greater osmotic gradient should enhance water redistribution and perhaps enable a better defense of blood (plasma) volume in hypohydrated subjects after heat acclimation.

In the early 1930's, investigators from the Harvard Fatigue Laboratory found that for a given loss of body water, via sweating, the blood volume decreased more in winter than in summer (60). This observation was made on one subject who voluntarily dehydrated over an extended period by performing physical exercise in the desert. Figure 8 presents recent data (106) showing the magnitude of hypovolemia associated with hypohydration of 5% of body weight both before and after a 10-day heat acclimation program. These data were obtained while the individuals were resting; hypohydration was achieved by voluntary food and fluid denial combined with exercise in a hot environment. Subjects rested 15 h in a comfortable environment (while hypohydrated) prior to the measurements. An identical program of exercise-heat stress, but with full fluid replacement, was completed on the day prior to the euhydration measurements (dotted line). After the subjects were heat acclimated, there was a smaller plasma volume reduction for a given body water loss. It can be theorized that, since hypohydration was induced by exercise-heat stress, a more hypotonic sweat secretion in heat acclimated subjects resulted in a greater amount of solute remaining in the plasma that enabled redistribution of fluid from the intracellular space. Therefore, an individual's state of heat acclimation probably alters the magnitude of hypovolemia associated with hypohydration.

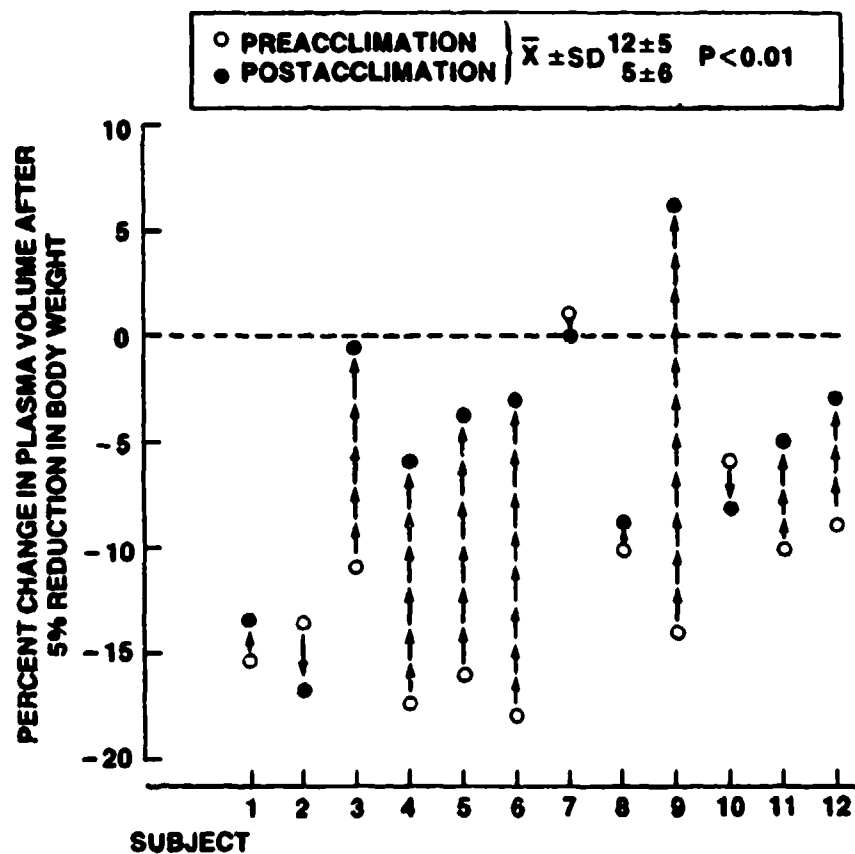


FIGURE 8. The magnitude of plasma volume reduction, while at rest, associated with a hypohydration of 5% body weight both before and after a heat acclimation program (106).

Figure 7 (upper section) illustrates individual values for the plasma volume reduction associated by a given level of body water loss for heat-acclimated subjects at rest (106). Hypohydration was achieved by voluntary food and fluid denial combined with exercise in a hot environment. A period of ~15 h was spent resting in a comfortable environment (while still hypohydrated) to provide time for the fluid compartments to equilibrate at the achieved hydration level. Plasma volume was estimated after the heat-acclimated subjects stood quietly for about 20 minutes in a comfortable environment. This same procedure of exercise-heat stress, but with full fluid replacement, was completed on the day prior to the euhydration

measurements (dotted line). The line of identity (solid line) represents a proportional loss of plasma and total body water.

The magnitude of plasma volume reduction is related to the level of total body water loss. Simply, more pronounced hypohydration results in a larger hypovolemia. At low levels of body water loss, considerable variability exists for the magnitude of hypovolemia; in fact, in one subject, plasma volume was elevated and in several other subjects plasma volume was not reduced below euhydration levels. The data from the moderate and high level of body water loss could be interpreted as showing either a continued reduction or a defense of plasma volume (similar to Figure 5). The mean values for plasma volume reduction between these levels are similar, but at the high level two subjects had a hypovolemia of greater than 20 percent.

The plasma volume responses at rest and exercise for heat acclimated subjects when euhydrated and hypohydrated by 3%, 5% and 7% of their body weight are presented in Figure 9 (118). Dehydration procedures were identical to those previously described. Note that plasma volumes were generally reduced with increased hypohydration, although there was some evidence of a plasma volume defense during the 7% hypohydration. The most important point from this figure is that the hypohydration mediated plasma volume reduction that occurs at rest continues throughout the subsequent light intensity (25% $\dot{V}_{O_{2max}}$ exercise). In fact, the differences between the euhydration and hypohydration plasma volume values are greater during exercise than rest because of the small exercise hemodilution that occurs when subjects are euhydrated but not when hypohydrated (107,117,118). These data clearly demonstrate that an exercise or heat-induced hypohydration results in hyperosmotic hypovolemia during both rest and exercise.

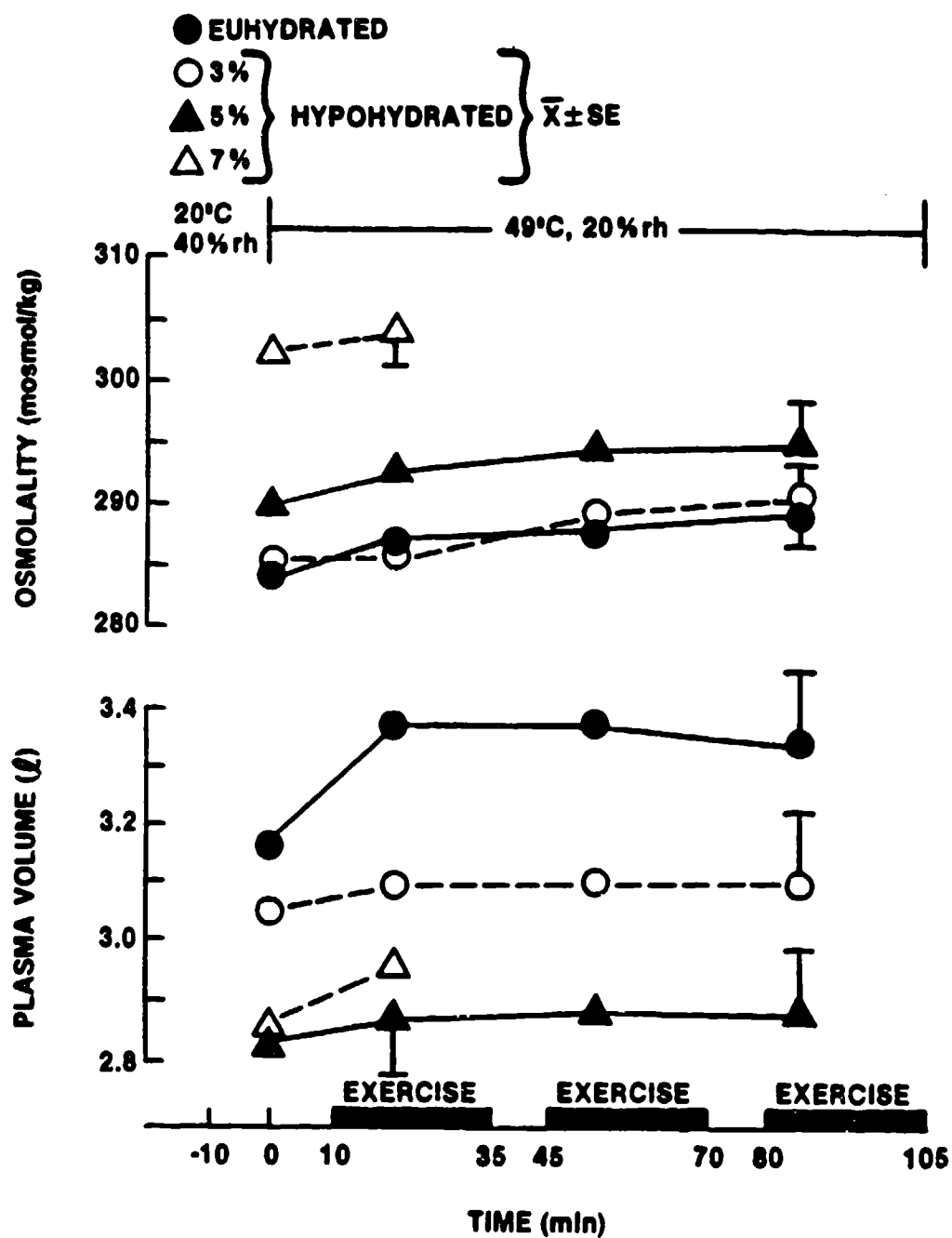


FIGURE 9. Plasma osmolality and plasma volume values ($\pm$ SE) at rest and exercise when euhydrated and hypohydrated (118).

There is evidence that plasma volume can be partially defended despite a progressive dehydration during intense (65-75% $\dot{V}O_{2max}$) running exercise (46,69,112). For example, Sawka and colleagues (112) showed that during 100 min of treadmill running the plasma volume remained stable despite a 4% reduction in body weight. Likewise, Kolka *et al.* (69) reported that during a competitive marathon race the plasma volume remained stable despite a 7% reduction in body weight. Reasons for the stable plasma volume during intense exercise, despite progressive dehydration, might include the release of water from glycogen breakdown and metabolic water (101) and the redistribution of water from inactive skeletal muscle (106). The higher the exercise intensity, the greater the use of muscle glycogen as a metabolic substrate (23) and therefore water release from glycogen breakdown. Also, the greater amount of substrate oxidation, including fat, will result in a greater amount of metabolic water release. Convertino (24) has speculated that the endocrine system could contribute to the redistribution of water into the intravascular space during intense exercise. The plasma concentration of angiotensin and vasopressin are increased in relation to the exercise intensity (25,26) and the magnitude of water deficit (14,44,45). Both of these hormones are potent vasoconstrictors, and their increased circulating concentrations cause vasoconstriction in inactive tissues (24). Vasoconstriction increases the ratio of pre- to post-capillary resistance, and favors fluid absorption from the inactive tissues (106). Therefore, during high intensity exercise while hypohydrated the elevated circulating concentrations of these hormones favors additional fluid absorption from the inactive tissues. Regardless of the physiological mechanisms, exercise intensity may influence the availability of water for redistribution to the intravascular space.

As previously discussed, some individuals may use diuretics to reduce their body weight. Diuretics are drugs that increase the rate of urine formation and generally result in

the loss of solutes (14). The commonly used thiazide (e.g. Diuril), carbonic anhydrase inhibitors (e.g. Diamox) and furosemide (e.g. Lasix) are all diuretics that result in natriuresis. Diuretic-induced hypohydration generally results in an iso-osmotic hypovolemia, with a much greater ratio of plasma loss to body water loss than either exercise or heat induced hypohydration (62). As a result, with a diuretic-mediated loss of body water, there is not a solute excess in the plasma to exert an osmotic gradient for the redistribution of body water. Therefore, diuretic-induced hypohydration results in a relatively greater loss of extracellular and thus plasma water than sweat-induced hypohydration.

IV. SYMPTOMS AND EXERCISE PERFORMANCE

Adolph and associates (1) described the symptoms associated with hypohydration in the desert. Thirst was reported to occur after a 2 percent water deficit and did not increase in intensity with greater hypohydration levels. The concept of thirst as an "all or none" response has been accepted for many years, but recent data have questioned this hypothesis (38). Engell and colleagues (38) have reported that thirst and many related symptoms increase in intensity with the level of hypohydration. Adolph and associates (1) also report that a 4 to 6 percent water deficit is associated with anorexia, impatience and headache; whereas, a 6 percent to 10 percent water deficit is associated with vertigo, dyspnea, cyanosis and spasticity. A person who incurs more than a 12 percent water deficit will be unable to swallow and will need assistance with rehydration. An estimate of the lethal hypohydration range is 15 to 25 percent (1).

Individuals who incur a body water deficit may perform a variety of different exercise tasks. For example, the exercise tasks required of a soldier in combat are different from those

of competitive athletes; and the exercise tasks required of a wrestler, power lifter and marathon runner are all dissimilar. Some of these activities require primarily anaerobic metabolism and skeletal muscle strength, whereas other activities require primarily aerobic metabolism and the support of the cardiovascular and thermoregulatory systems. Since the metabolic and physiological system requirements can vary greatly, it is not surprising that hypohydration does not have homogeneous effects upon exercise performance. Environmental conditions will also influence the magnitude of the performance decrement during exercise when hypohydrated.

Table 3 presents a summary of studies investigating the influence of hypohydration on muscular strength and muscular endurance as well as anaerobic performance. These investigations were ordered to present a continuum from the lowest to most severe water deficit. Caution should be employed when comparing the results of these different investigations (within Table 3 as well as Tables 4-6) because of differences in subject populations, exercise protocols and methods to elicit hypohydration. The arrows in the table denote the direction of a significant difference between euhydration and hypohydration.

Muscular strength was examined in 12 studies, of which four demonstrated a strength reduction after hypohydration. Of the four studies which demonstrated strength reductions, hypohydration was achieved by fluid restriction in three (12,13,61) and by a combination of exercise and heat exposure in the fourth (144). Therefore, prolonged fluid restriction, perhaps accompanied by a caloric deficit (13,61) was the dehydration method which most often reduced muscular strength. The magnitude of water deficit appeared to influence the frequency with which muscular strength reductions were reported. Only one (13) of the five studies employing less than a 5% reduction in body weight reported a strength reduction; whereas, three of the seven studies employing between 5 to 8% reduction in body weight reported

Table 3. Summary of studies investigating the influence of hypohydration on muscular strength and endurance as well as anaerobic performance (114).

	STUDY		n	DEHYDRATION PROCEDURE	Δ WT.	STRENGTH METHODOLOGY	RESULTS	ANAEROBIC METHODOLOGY	RESULTS
1.	AHLMAN & KARVONEN	1981	32	SAUNA & EXERCISE	-2 kg	BACK & LEG LIFTS	NC IN STRENGTH	—	—
2.	GREENLEAF et. al.	1967	12	EXERCISE IN HEAT	-3%	ISOMETRIC	NC IN STRENGTH	—	—
3.	BOSCO & TERJUNG	1968	9	FLUID RESTRICTION	-3%	ISOMETRIC	↓ (11%) IN STRENGTH	—	—
4.	BAJLANI & SHARMA	1980	5	EXERCISE IN HEAT	-3%	ISOMETRIC	NC IN STRENGTH ↓ IN ENDURANCE	—	—
5.	NIELSEN et. al.	1981	6	DIURETIC	-3%	—	—	SUPRAMAXIMAL CYCLE	↓ (18%)
			6	SAUNA	-3%	—	—	SUPRAMAXIMAL CYCLE	↓ (35%)
			5	EXERCISE	-3%	—	—	SUPRAMAXIMAL CYCLE	↓ (44%)
6.	SALTIN	1964	4	SAUNA, EXERCISE IN HEAT, EXERCISE	-4%	ISOMETRIC	NC IN STRENGTH	—	—
7.	TORRANON et. al.	1979	20	SAUNA	-4%	ISOMETRIC	↓ (31%) IN ISOMETRIC ENDURANCE	—	—
						ISOTONIC	↓ (29%) IN ISOTONIC ENDURANCE	—	—
8.	MNATZAKAMIAN & VACCARO	1982	7	?	-4%	ISOKINETIC	NC IN ENDURANCE	—	—
9.	TUTTLE	1943	13	EXERCISE & HEAT	-5%	ISOMETRIC	NC IN STRENGTH	—	—
10.	JACOBS	1950	11	HEAT	-5%	—	—	WINGATE TEST	NC
11.	SERFASS et. al.	1984	11	?	-5%	ISOMETRIC	NC IN STRENGTH NC IN ENDURANCE	—	—
12.	WEBSTER et. al.	1988	7	EXERCISE IN HEAT, SAUNA	-5%	ISOKINETIC	NC IN LEG STRENGTH ↓ (7%) IN SHOULDER AND CHEST STRENGTH	WINGATE TEST	↓ (21%) ANAEROBIC POWER ↓ (10%) ANAEROBIC CAPACITY
13.	BOSCO et. al.	1974	21	FLUID RESTRICTION	-6%	ISOMETRIC	↓ (10%) IN STRENGTH	—	—
14.	GREENLEAF et. al.	1966	9	FLUID RESTRICTION	-7%	ISOMETRIC	NC IN STRENGTH	—	—
15.	SINGER & WEISS	1969	10	?	-7%	ISOMETRIC	NC IN STRENGTH	—	—
16.	HOUSTON et. al.	1981	4	FLUID RESTRICTION	-8%	ISOKINETIC	↓ (11%) IN STRENGTH	SUPRAMAXIMAL RUN	NC

strength reductions. Interestingly, the magnitude of water deficit did not appear to influence the magnitude of strength reduction, as decreases approximating 10% were reported for each study. Finally, it appears that the upper body muscle groups are more likely than the lower body muscle groups to show a strength reduction from hypohydration (13,144).

Muscular endurance was evaluated in four studies, of which two (11,140) demonstrated a reduction during hypohydration experiments. The two studies that found no change in muscular endurance did not report their dehydration procedures (76,124), but the other two studies used either thermal (140) or exercise and thermal (11) dehydration procedures. There were no systematic differences among these four studies in the magnitude of water deficit, muscle group tested or test methodology. Torranin et al. (140) evaluated the isometric endurance of a small muscle group (hand grip) and the isotonic endurance of large muscle groups (arm and leg). Despite the use of very diverse methodology, they found a consistent 30% reduction in muscular endurance when hypohydrated. They speculated that higher muscle temperature during the hypohydration experiments might have mediated the reduced muscular endurance. The muscular endurance experiments were conducted approximately 1 hour after the subjects had finished dehydrating in an 80°C sauna. Bijlani and Sharma (11) performed their muscular endurance experiments either immediately after or within 30 min after dehydrating their subjects in a 41°C environment. In addition, it appeared that all of their muscular testing (when eu- and hypohydrated) was conducted in a fairly warm (30-35°C) environment. They showed an inverse relationship between the control (euhydration) muscular endurance values and the dry bulb temperature. Therefore, in that study (11) an elevated muscle temperature could have mediated the reduced muscular endurance during the hypohydration experiments (21,35,97).

Anaerobic exercise performance was evaluated in four studies, of which two employed Wingate type tests (65,144) and two studies employed supramaximal endurance tests (66,90). Jacobs (65) performed a comprehensive evaluation of anaerobic exercise performance (Wingate test) in subjects when they were euhydrated and when they were hypohydrated by 2%, 4% and 5% of their body weight. This investigator found that hypohydration did not alter anaerobic exercise performance or the post-exercise blood lactate values. On the other hand, Webster and colleagues (144) found a 21% reduction in anaerobic power and a 10% reduction in anaerobic capacity when their subjects were hypohydrated (5% body weight). Both of these studies used similar methodologies so that their disparate results are not easily explained.

Nielsen et al (90) had subjects perform a supramaximal (105% of $\dot{V}O_{2\max}$) cycle ergometer test both when euhydrated and when hypohydrated (3% of body weight). Supramaximal exercise performance was decreased by 18, 35 and 44% when subjects were hypohydrated by diuretics, sauna and previous exercise, respectively. These reductions in supramaximal exercise performance were found to be related to the elevation of plasma potassium concentration as well as the increase in skeletal muscle temperature. In addition, Houston et al. (61) reported that hypohydration (8% of body weight) did not affect performance in a supramaximal (1 min) treadmill run. In these experiments, dehydration was achieved by fluid and food restriction over several days, and the subjects were not exposed to any heat stress.

Table 4 presents a summary of studies investigating the influence of hypohydration on maximal aerobic power and physical work capacity. A body water deficit of less than 3% body weight did not alter maximal aerobic power in a neutral environment. Maximal aerobic power was decreased (16,18,144) in three of the five studies when hypohydration equaled or

exceeded 3% body weight. Therefore, a critical water deficit (3% body weight) might exist before hypohydration reduces maximal aerobic power in a neutral environment. For experiments conducted in a neutral environment, the reduction in maximal aerobic power was not related to the magnitude of body water deficit or the dehydration procedures employed. In a hot environment, Craig and Cummings (31) demonstrated that small (2% body weight) to moderate (4% body weight) water deficits resulted in large reductions of maximal aerobic power. Likewise, their data indicate a disproportionately larger decrease in maximal aerobic power with an increased magnitude of body water deficit. It seems that environmental heat stress has a potentiating effect on the reduction of maximal aerobic power elicited by hypohydration.

The physical work capacity for aerobic exercise was decreased during hypohydration in all of the studies presented in Table 4. Physical work capacity was decreased by even a marginal water deficit that did not alter maximal aerobic power (5,18). The more pronounced the water deficit, the larger the reduction in physical work capacity. In addition, the environmental temperature influenced the magnitude of reduction in physical work capacity. Hypohydration resulted in a much larger reduction in physical work capacity in a hot as compared to a neutral temperature environment. Again, it appears that the thermoregulatory system, via increased body temperature, has an important role in the reduced exercise performance mediated by a body water deficit.

One investigation has examined the effects of a body water deficit on competitive distance running. In a study by Armstrong and colleagues (5) individuals competed in 1,500, 5,000 and 10,000 meter races when euhydrated and when hypohydrated. Hypohydration was achieved by administration of a diuretic (furosemide) which decreased body weight by 2% and

plasma volume by 11%. Remember, diuretics result in a disproportionately larger loss of plasma water relative to total body water loss than either exercise or thermally-mediated hypohydration. They found that running performance was degraded by hypohydration to a greater extent in longer races (~5% for the 5,000 and 10,000 m) than a shorter race (~3% for 1,500 m). These investigators speculated that hyperthermia may have provided the physiological mechanism that caused greater performance decrements in the longer races.

Table 4. Summary of studies investigating the influence of hypohydration on maximal aerobic power and physical work capacity (114).

	STUDY		n	DEHYDRATION PROCEDURE	ΔWT.	TEST ENVIRONMENT	EXERCISE MODE	MAXIMAL AEROBIC POWER	PHYSICAL WORK CAPACITY
1.	ARMSTRONG et. al.	1985	8	DIURETICS	-1%	NEUTRAL	TM	NC	↓(8%)
2.	CALDWELL et. al.	1984	16	EXERCISE	-2%	NEUTRAL	CY	NC	↓(7w)
			15	DIURETICS	-3%	NEUTRAL	CY	↓(8%)	↓(21w)
			16	SAUNA	-4%	NEUTRAL	CY	↓(4%)	↓(23w)
3.	SALTM	1984	10	SAUNA, EXERCISE, HEAT, DIURETICS	-4%	NEUTRAL	CY	NC	↓(7)
4.	CRAIG & CUMMINGS	1986	9	HEAT	-2%	HOT	TM	↓(10%)	↓(22%)
					-4%	HOT	TM	↓(27%)	↓(42%)
5.	BUSKIRK et. al.	1958	13	EXERCISE, HEAT	-8%	NEUTRAL	TM	↓(-0.22 l/min)	-
6.	WEBSTER et. al.	1988	7	EXERCISE ON HEAT, SAUNA	-8%	NEUTRAL	TM	↓(7%)	↓(12%)
7.	HERBERT & REISL	1971	8	?	-8%	NEUTRAL	CY	-	↓(17%)
8.	HOUSTON et. al.	1981	4	FLUID RESTRICTION	-8%	NEUTRAL	TM	NC	-

V. PHYSIOLOGICAL FUNCTION

Generally, body water deficits adversely influence exercise performance. The critical water deficit and magnitude of performance decrement are related to the exercise task and the environmental temperature. Exercise tasks that primarily require aerobic metabolism and are prolonged will more likely be adversely influenced by hypohydration than exercise tasks that primarily require anaerobic metabolism as well as muscular strength and power. In addition,

the warmer the environment the greater the potential for decrements in exercise performance. There is absolutely no evidence that hypohydration can benefit exercise performance. In fact, man cannot adapt to chronic dehydration (1), and most of the thermoregulatory advantages conferred by high aerobic fitness and heat acclimation are negated by hypohydration during exercise in the heat (16,17,115). Mental performance is also adversely influenced by body water deficits (1,48,74). For many complex military, industrial and athletic tasks, both the mental decision making and physiological function are closely related. As a result, hypohydration can have more profound effects on real-life tasks than on solely physiological performance measures discussed in this report.

Hypohydration does not have consistent effects on muscular strength, but muscular endurance is often reduced by hypohydration (Table 3). Reduced muscular endurance is probably mediated by the dehydration procedures which cause an elevation of skeletal muscle temperature. Increased skeletal muscle temperature markedly reduces muscular endurance, but does not affect strength (97). Clarke and colleagues (21) found that if skeletal muscle was heated beyond 32°C there was a reduction of isometric endurance. Other laboratories (35,97) have substantiated these findings. Possibly, the dehydration process rather than hypohydration per se is responsible for the decreased muscular endurance.

The effects of hypohydration on anaerobic performance are not clear-cut. Hypohydration can reduce anaerobic performance, however, there does not appear to be a distinguishable critical water deficit. Rather, the dehydration procedures employed may play an important role as to whether or not there is a decrement in anaerobic performance. Anaerobic performance (Table 3) is more likely to be reduced if the dehydration procedures of exercise and heat exposure (90,14) are employed as opposed to fluid restriction alone (61).

Electrolyte imbalances and elevated body temperature encountered during hypohydration are the physiological mechanisms responsible for reducing anaerobic performance during hypohydration experiments. Nielsen et al. (90) found that anaerobic performance was inversely related to plasma potassium concentration and muscle temperature in hypohydrated subjects. Interestingly, these investigators found no effect of hypovolemia on anaerobic performance. Since potassium is the primary intracellular cation, an increased plasma concentration may indicate an intracellular electrolyte imbalance. Sjogaard (130) has suggested that a loss of intracellular potassium might impair the membrane potential and reduce muscle contractibility. Finally, it has been suggested that high muscle temperatures might result in an elevated hydrogen ion concentration which would inhibit phosphofructokinase activity and therefore anaerobic performance (39).

As previously stated, hypohydration decreases an individual's maximal aerobic power with the critical water deficit and magnitude of reduction dependent upon the presence of environmental heat stress (Table 4). In a neutral environment, a 4 to 8% decrease in maximal aerobic power occurs after hypohydration by 3% body weight. In hot environments, a marginal water deficit (2% body weight) will decrease maximal aerobic power by a substantial amount (10%), and this performance decrement is amplified at greater hypohydration levels. Maximal aerobic power is dependent upon both central circulatory (oxygen delivery) and peripheral (oxygen extraction) factors; with cardiac output and arteriovenous oxygen difference as indices of these central and peripheral factors, respectively.

A reduced maximal cardiac output might be the physiological mechanism by which hypohydration decreases an individual's maximal aerobic power. Remember, hypohydration is associated with a decreased plasma volume during both rest and exercise. A decreased

volume can increase blood viscosity (143) as well as possibly reduce venous return. During maximal exercise, a viscosity mediated increased resistance and a reduced cardiac filling could both decrease stroke volume and cardiac output. Several investigators (4,133) have reported a tendency for reduced cardiac output for hypohydrated subjects during short-term moderate intensity exercise in a neutral environment.

It is not surprising that environmental heat stress potentiates the hypohydration mediated reduction in maximal aerobic power. For euhydrated individuals, environmental heat stress alone decreases maximal aerobic power by ~7% (116). In the heat, the superficial skin veins reflexively dilate to increase the cutaneous blood flow and volume. This displacement of blood to the cutaneous vasculature could decrease maximal aerobic power by: (a) reducing the portion of cardiac output perfusing the contracting musculature or, (b) decreasing the effective central blood volume and thus reduce venous return and cardiac output. If an individual was hypohydrated and encountered environmental heat stress, he/she would be hypovolemic and still have to simultaneously perfuse the cutaneous vasculature and contracting skeletal muscles. As a result, both environmental heat stress and hypohydration could act independently to limit cardiac output and therefore oxygen delivery during maximal exercise.

Hypohydration has its most adverse effects on the performance of prolonged aerobic exercise (5), and acts through an impairment of the thermoregulatory and cardiovascular systems. Hypohydration has little effect on respiratory and acid-base responses during rest and exercise in the heat (112,123). In comparison to euhydration, hypohydration results in an increased core temperature during exercise in comfortable (17,49,85,112) as well as in hot (20,96,100,110,120,132) environments. The critical water deficit of 1% body weight elevates core temperature during exercise (37). As the magnitude of water deficit increases, there is

a concomitant graded elevation of core temperature during exercise. Tables 5 and 6 present summaries of selected investigations examining the thermoregulatory effects of hypohydration during rest and exercise. These investigations were selected to represent a continuum from a marginal to the greatest level of fluid deficit reported during the conditions of rest and exercise in the heat. A comparison of results from investigators who examined a single level of body water deficit during rest or exercise-heat stress (Tables 5 and 6), however, does not support this concept of graded core temperature responses with hypohydration (108). However, conclusions based on inter-investigation comparisons, can be tenuous because of differences in subject populations, environmental conditions, exercise intensities and test methodologies (108).

Two studies examined core temperature responses to exercise while hypohydration levels were varied during independent tests in the same subjects. Strydom and Holdsworth (136) studied two miners at two hypohydration levels (low, 3%-5%, and high, 5%-8% weight loss) and found higher core temperatures at the greater hypohydration level. Sawka *et al.* (118) reported that hypohydration linearly increased the core temperature by 0.15°C during exercise in the heat for each percent decrease in body weight. Other investigators, reporting a gradation of elevated core temperature with increased water deficits, have interpolated from a single hypohydration level (51) and/or employed prolonged exercise-heat exposure to elicit a progressive dehydration (1,47). Reanalyzing the data of Adolph and associates (1), we find that their subjects had an elevated core temperature of 0.20°C for each percent decrease in body weight. Their data represents progressive dehydration under a variety of field and laboratory conditions. Greenleaf and Castle (51) reported that core temperature was elevated 0.10°C for each percent decrease in body weight during exercise (49% $\dot{V}O_{2\max}$) in a moderate

Table 5. Summary of studies investigating the effects of hypohydration during rest in the heat (108).

STUDY	YEAR	% Δ WT	HR	T_{C}	$\dot{M}_{sw}$	PBF
SWAMY et al	1981	-1%	=	=	=	
SENAY & CHRISTENSEN	1965	-2%			=	=
HERTZMAN & FERGUSON	1960	-3%	↑ 8 bpm	↑ 0.6°C	=	
MYRE & ROBINSON	1977	-3%		=	=	
SWAMY et al	1981	-3%	↑ 17 bpm	↑ 0.4°C	↓ 22%	
HORSTMAN & HORVATH	1972	-4%		↑ 1.0°C	↓ 21%	↓
HERTZMAN & FERGUSON	1960	-6%	↑ 17 bpm	↑ 1.2°C	=	
SENAY & CHRISTENSEN	1965	-6%			=	=

Table 6. Summary of studies investigating the effects of hypohydration during exercise in the heat (108).

STUDY	YEAR	% Δ WT	HR	T_{C}	$\dot{M}_{sw}$	PBF
MOROFF & BASS	1965	-2%	↑ 10 bpm	↑ 0.5°C	↓ 3%	
CLAREMONT et al	1976	-3%	↑ 20 bpm	↑ 0.6°C	=	↓
NADEL et al	1980	-3%	↑ 8 bpm	↑ 0.5°C		↓
STRYDOM et al	1966	-3%		=	=	
SWAMY et al	1981	-3%	↑ 40 bpm	=	=	
SENAY	1968	-4%			↓ 17%	
STRYDOM et al	1968	-4%	↑ 19 bpm	↑ 0.3°C	↓ 33%	
SAWKA et al	1983	-5%	↑ 13 bpm	↑ 0.5	↓ 13%	
		-5%	↑ 16 bpm	↑ 0.8	↓ 13%	
SWAMY et al	1981	-6%	↑ 60 bpm	↑ 0.8°C	=	
STRYDOM et al	1968	-7%	↑ 29 bpm	↑ 0.7°C	↓ 33%	

environment. This relationship was based on interpolation from a single hypohydration (5% body weight loss) level. Gisolfi and Copping (47) reported that core temperature is elevated by 0.40°C for each percent decrease in body weight after a weight loss of greater than 2% during intense exercise ($74\% \dot{V}\text{O}_{2\text{max}}$) in a hot environment. Figure 10 presents the relationships between hypohydration and the elevation in core temperature (above euhydration levels) during exercise (1,47,51,118,136). It seems that exercise intensity and environmental conditions probably modify the magnitude of core temperature elevation associated with hypohydration.

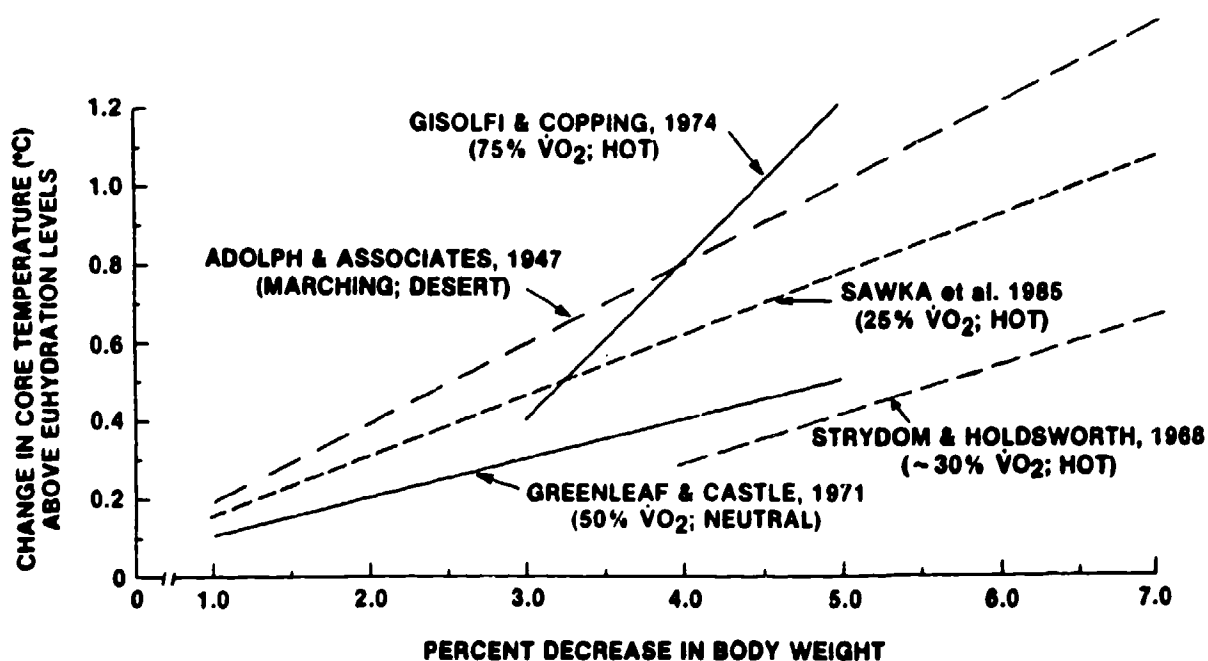


FIGURE 10. Relationships for the elevation in core temperature (above euhydration) at a given magnitude of water deficit during exercise conditions in different environments (114).

An increase in heat storage is the result of either an increase in metabolic heat production or a decrease in heat loss. Hypohydration probably does not influence the rate of aerobic or anaerobic metabolism during exercise (51,105,110,111,115,118) and as a result does not cause greater metabolic heat production. It should be noted, however, that an increased aerobic (100) and anaerobic (87) metabolism during exercise when hypohydrated have been reported. However, since most studies indicate that metabolic heat production is not increased, a decreased heat dissipation is most likely responsible for the hypohydration-mediated increase in heat storage during exercise. The relative contributions of evaporative and dry heat exchange during exercise depend on the specific environmental conditions, but both avenues of heat loss are adversely affected by hypohydration (Tables 5 and 6).

Hypohydration is associated with both reduced or unchanged sweating rates at a given metabolic rate during exercise in the heat (108). Those investigators who report no change in sweating rate, usually still observe an elevated core temperature (Tables 5 and 6). Therefore, during hypohydration the sweating rate is lower for a given core temperature, and the potential for heat dissipation via sweat evaporation is reduced. Figure 11 presents data showing that total body sweating rate for a given core temperature is systematically reduced with increased hypohydration levels during exercise in the heat (118). Likewise, Figure 12 presents data showing a reduced local sweating rate with hypohydration (5% body weight) during exercise in the heat (113). Since core temperature provides about 90% of the drive for thermoregulatory effector responses, these figures are depicting information indicating a change in the control of sweating. Both of these figures (11 and 12) suggest an increased

threshold temperature for thermoregulatory sweating during hypohydration, which indicates a reduced sweating rate for a given thermal drive.

The physiological mechanisms mediating the reduced sweating rate during hypohydration are not clearly defined. Figure 13 presents individual data for the reduction (3 to 4% (from euhydration)) in total body sweating rate plotted against the change in osmolality and plasma volume during exercise-heat stress (118). Both the singular and combined effects of plasma hyperosmolality (19,42,55,109,121) and hypovolemia (41,58,109,118) have been suggested as mediating this reduced sweating response. Plasma osmolality changes may relate to tonicity changes in the extracellular fluid bathing the hypothalamic neurons (70,89). Silva and Boulant (129) have demonstrated that in rat brain slices, there are preoptic-anterior hypothalamic neurons which are both thermosensitive and osmosensitive. Nakashima and colleagues (83) have reported similar results for preoptic medial hypothalamic neurons in rats. Such data suggests a central interaction between thermoregulation and body water regulation. Numerous other animal studies have demonstrated that the intravascular (8,9,52,70) or intracranial (32,141) infusion of hypertonic solutions will elevate core temperature during rest and exercise in the heat. Tonicity could also exert a peripheral effect via a high interstitial osmotic pressure inhibiting the fluid availability to the eccrine sweat gland (51,89).

Several human studies have demonstrated that the ingestion of hypertonic fluid will elevate core temperature responses in the heat, despite the maintenance of euhydration (55,88,89). Consistent with this, in humans, a significant relationship ($r = 0.62$ to 0.85) between changes in plasma osmolality and changes in thermoregulatory sweating responses during exercise-heat exposure has been reported (109,118,121). Likewise, Fortney *et al.* (42)

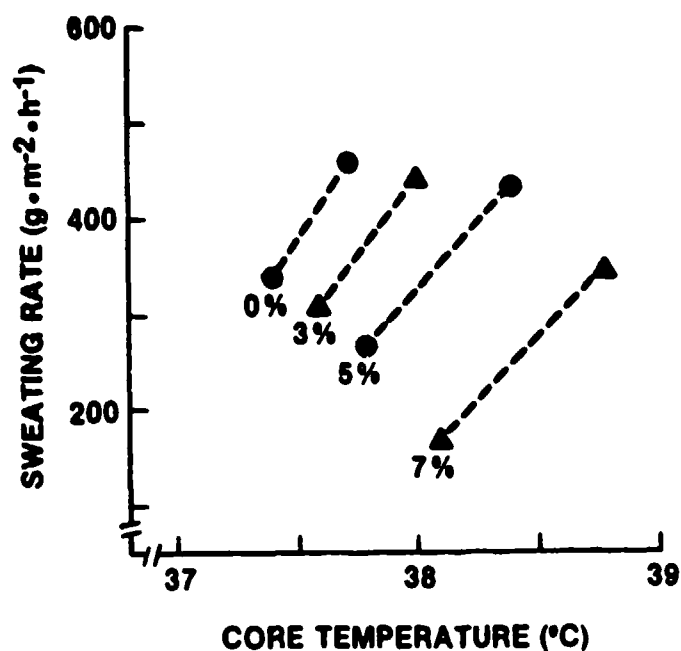


FIGURE 11. Plot of mean total body sweating rate and core temperature during exercise when euhydrated (0%) and hypohydrated by 3%, 5% and 7% of body weight (118).

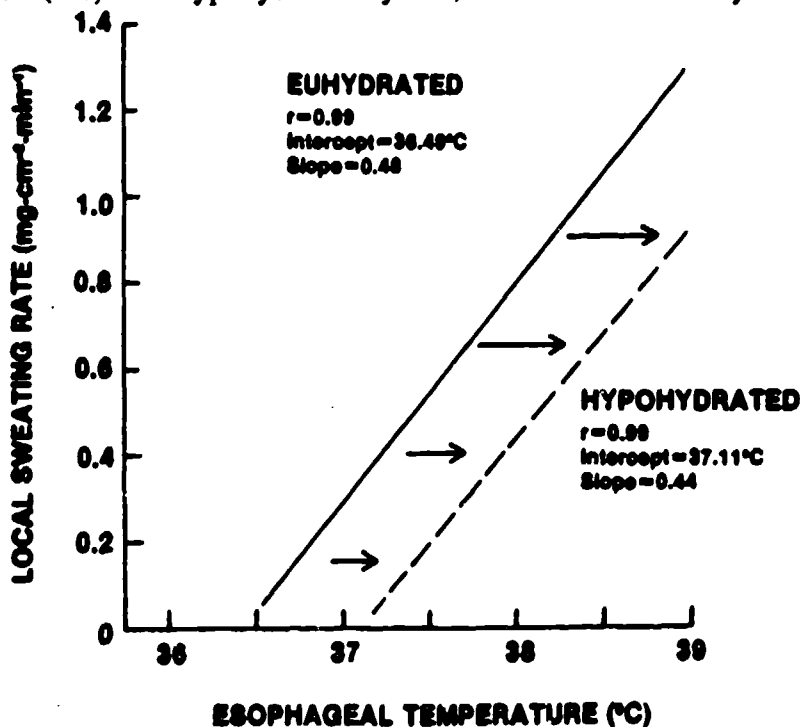


FIGURE 12. The local sweating rate (dew point hygrometry) for a given core temperature during exercise-heat stress for three subjects when euhydrated and hypohydrated by 5% of body weight (113).

have reported that hyperosmolality will increase the threshold temperatures for sweating and cutaneous vasodilation even without a blood volume reduction, during exercise in the heat. The combined results of these studies indicate that plasma hyperosmolality exerts a powerful influence on thermoregulatory sweating and body temperature responses to exercise and heat stress.

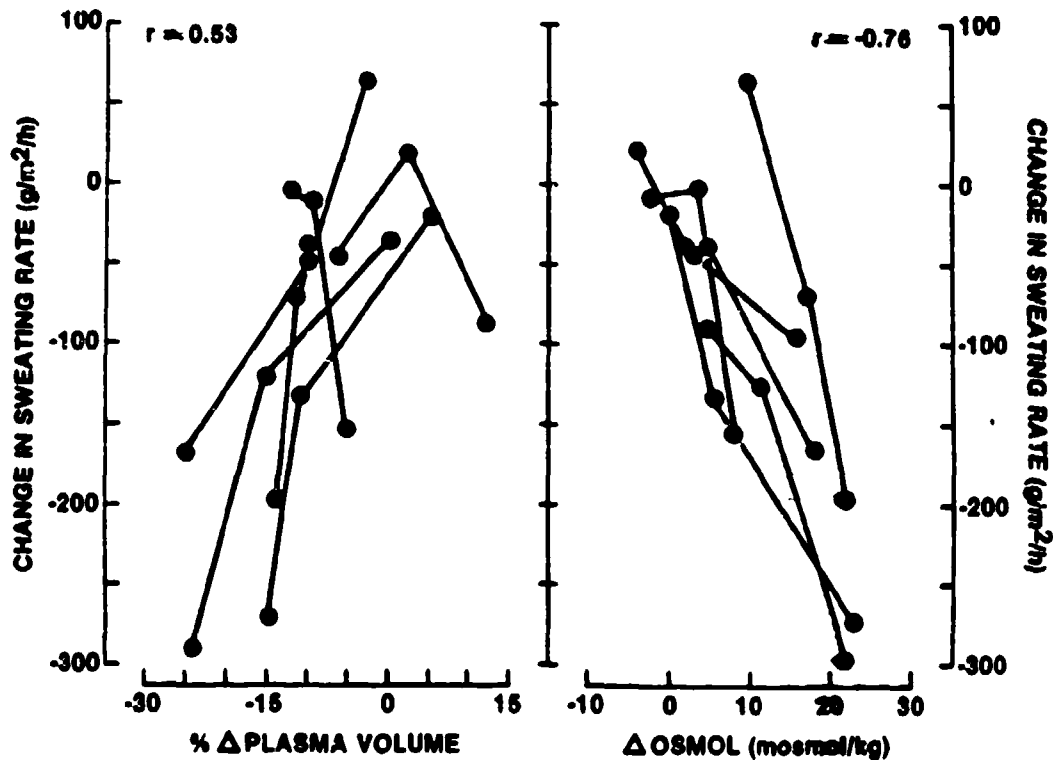


FIGURE 13. Individual relationships for the change in exercise sweating rate from euhydration with changes in plasma volume and changes in osmolality from euhydration (118).

Hypovolemia can also mediate a decreased sweating rate during exercise in the heat (42,42,109,118). The thermoregulatory disadvantages of hypohydration can also be partially reversed by the re-establishment of the normal blood volume during exercise in the heat (134). Fortney *et al.* (41) have provided strong evidence that a large iso-osmotic hypovolemia can

cause a reduced sweating rate and elevated core temperature response during exercise. They theorize that hypovolemia may alter the activity of atrial baroreceptors that have afferent input to the hypothalamus. Therefore, a reduced atrial filling pressure might modify neural information to the hypothalamic thermoregulatory centers which control sweating rate. Consistent with these findings, a significant relationship ($r = 0.53$ to 0.75) between changes in blood (or plasma) volume and changes in thermoregulatory sweating responses during exercise-heat exposure have been reported (109,118).

The effects of hypohydration on cardiovascular responses to exercise have been investigated (4,82,105,111,133). During submaximal exercise with little thermal strain, hypohydration elicits an increase in heart rate and decrease in stroke volume, and usually no change in cardiac output relative to euhydration levels (4,105,133). Apparently, during hypohydration, a decreased blood volume reduces central venous pressure (67) and cardiac filling which reduces stroke volume and requires a compensatory increase of heart rate. Figure 14 shows the relationship between stroke volume and the reduction in plasma volume for eu- and hypohydrated subjects during exercise-heat stress (82). During submaximal exercise with moderate (82) or severe (111) thermal strain, hypohydration (3 to 4% body weight) leads to an increase in heart rate, decrease in stroke volume and a decrease in cardiac output relative to euhydration levels. Figure 15 presents the cardiovascular responses of subjects performing two prolonged exercise bouts ($70\% \dot{V}_{O_{2max}}$) that were spaced by a brief rest period. The subjects were progressively dehydrated (4% body weight) and incurred core temperatures in excess of 40°C . Note that the increased heart rate does not compensate for the decreased stroke volume and results in a decreased cardiac output. Likewise, Sproles *et al.* (133)

demonstrated that a severe water deficit (7% body weight) in the absence of thermal strain, can also reduce cardiac output during submaximal exercise.

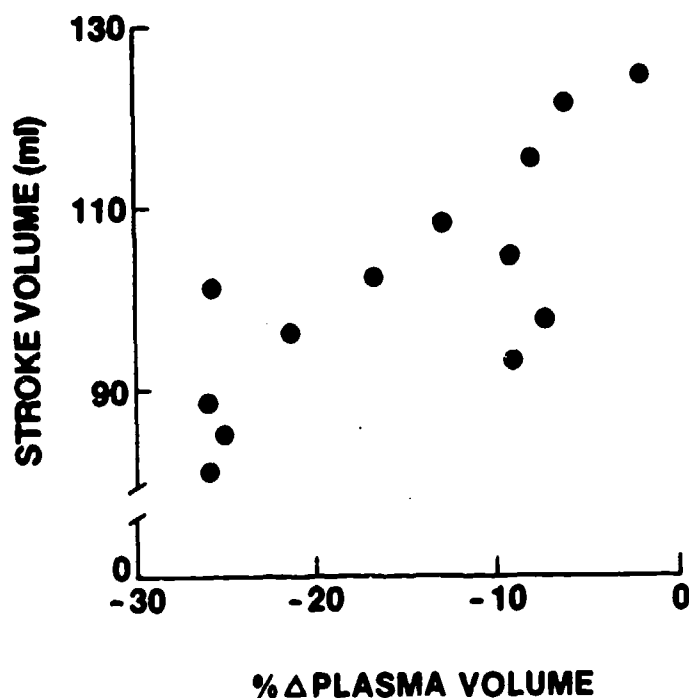


FIGURE 14. Relationship between cardiac stroke volume and reduction in plasma volume for eu- and hypohydrated subjects during exercise-heat stress (redrawn from 82).

The combination of exercise and heat strain results in competition between the central and peripheral circulation for a limited blood volume (103). As body temperature increases during exercise, cutaneous vasodilation occurs and the superficial veins become more compliant, thus decreasing venous resistance and pressure. As a result of decreased blood volume and increased blood displacement to cutaneous vascular beds; venous return, and thus, cardiac output will be decreased below euhydration values (82,111). Several investigators (40,42,82) report that these conditions also reduce cutaneous blood flow for a given core

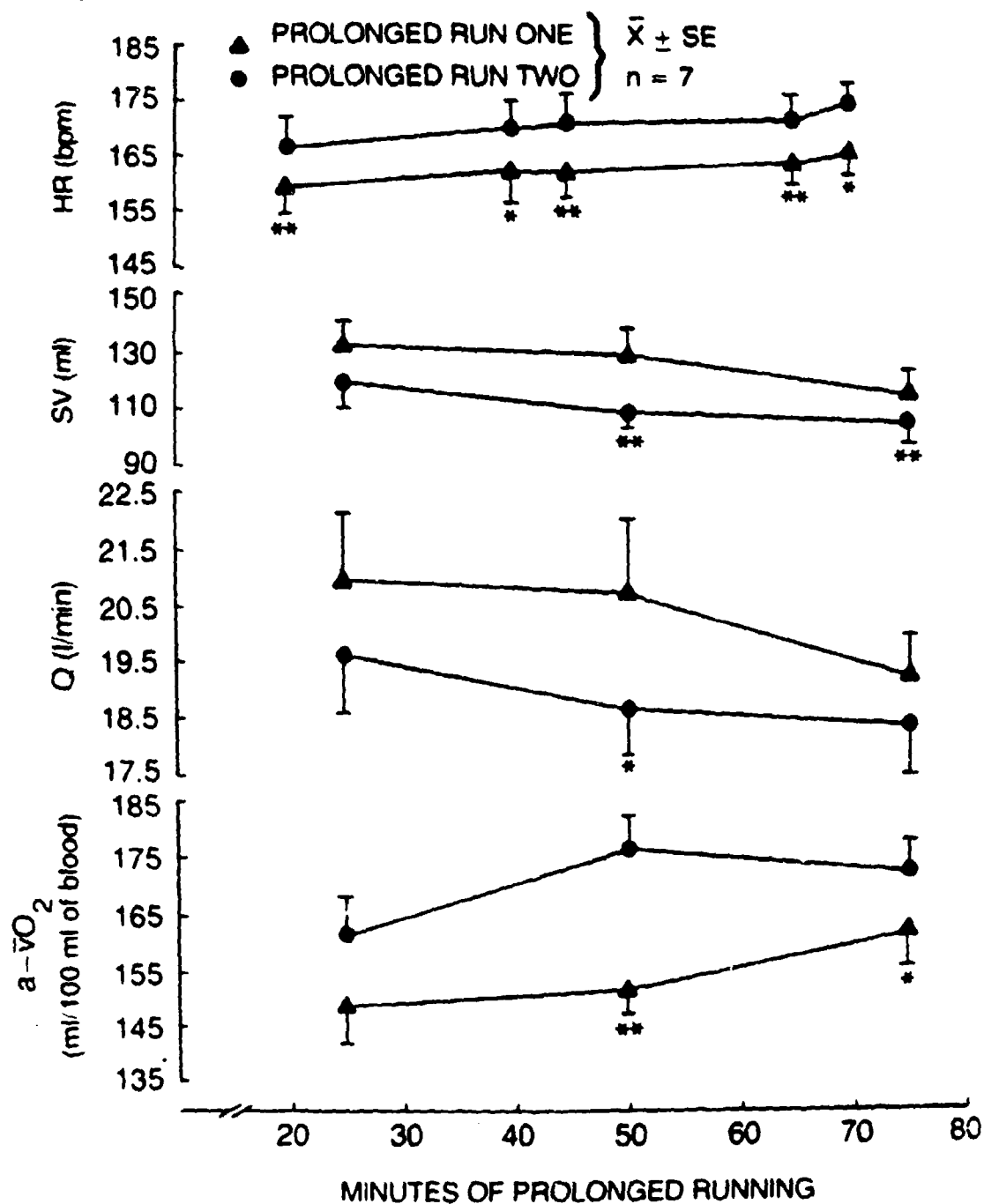


FIGURE 15. Heart rate, stroke volume, cardiac output and arteriovenous oxygen difference responses of subjects during two prolonged runs spaced by a rest period (111). During this experiment, the subjects were progressively dehydrated. *P<0.05, **P<0.01.

temperature and therefore the potential for dry heat exchange. Figure 16 presents forearm (cutaneous) blood flow for eu- and hypohydrated individuals during exercise-heat stress (82). The diuretic induced hypohydration (3% body weight) delayed the onset of cutaneous vasodilation and reduced the maximal cutaneous blood flow. Likewise, hyperosmolality, in the absence of hypovolemia, can also reduce the cutaneous blood flow response during exercise-heat stress (40).

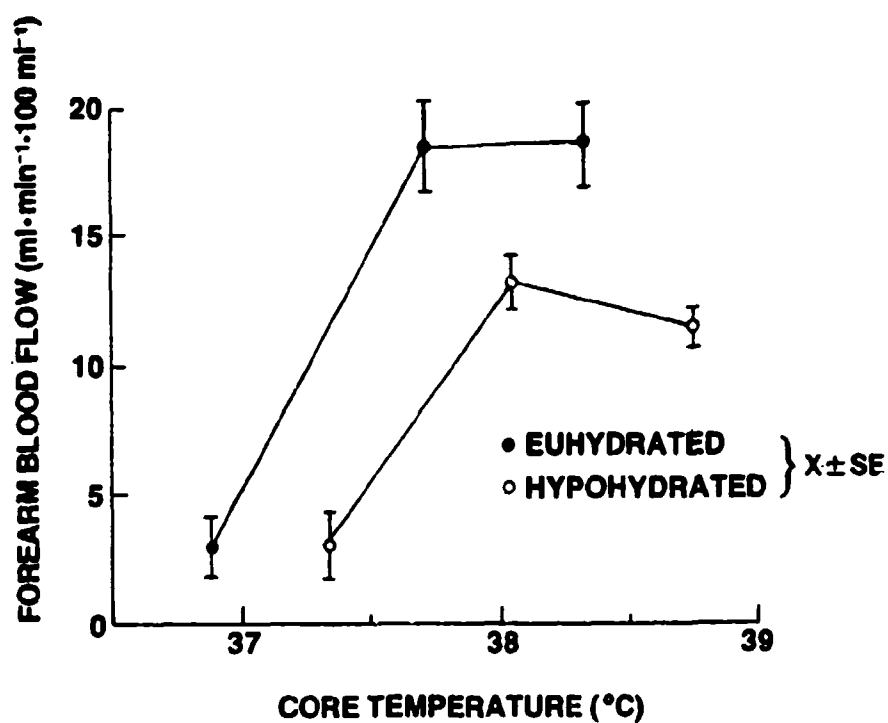


FIGURE 16. Forearm blood flow as a function of core temperature for eu- and hypohydrated subjects during exercise-heat stress (redrawn from 82).

Another physiological mechanism by which hypohydration might limit submaximal exercise performance is by altering skeletal muscle metabolism. Skeletal muscle glycogen concentration has been demonstrated to be related to submaximal exercise performance (23). In preliminary work, Neuffer *et al.* (87) found no significant difference in muscle glycogen

utilization between experiments during 1 h of cycle ergometer exercise (50% $\dot{V}_{O_{2\max}}$ 18°C, 30% rh) when subjects were hypohydrated (-5% body weight) as compared with euhydrated. Although muscle glycogen depletion did not differ between the experiments, evaluation and interpretation of these data are not conclusive due to subnormal muscle glycogen and hydration levels before exercise in the control "euhydration" experiments. Since 3-4 grams of water are bound to each gram of glycogen, as much as 400-500 ml of water might be needed for glycogen resynthesis after heavy exercise. Neufer and colleagues (86) also hypothesized that hypohydration might reduce glycogen resynthesis because of reduced body water availability, despite the intake of an adequate carbohydrate diet. Preliminary findings by Neufer *et al.* (86) indicate that, despite reduced muscle and body water availability, muscle glycogen resynthesis is not altered by hypohydration during the first 14 hours after heavy exercise. Figure 17 presents muscle glycogen levels during control (euhydrated, rested and fed) conditions and immediately (0 h) and 14 h after completion of a Heat Stress Test (113).

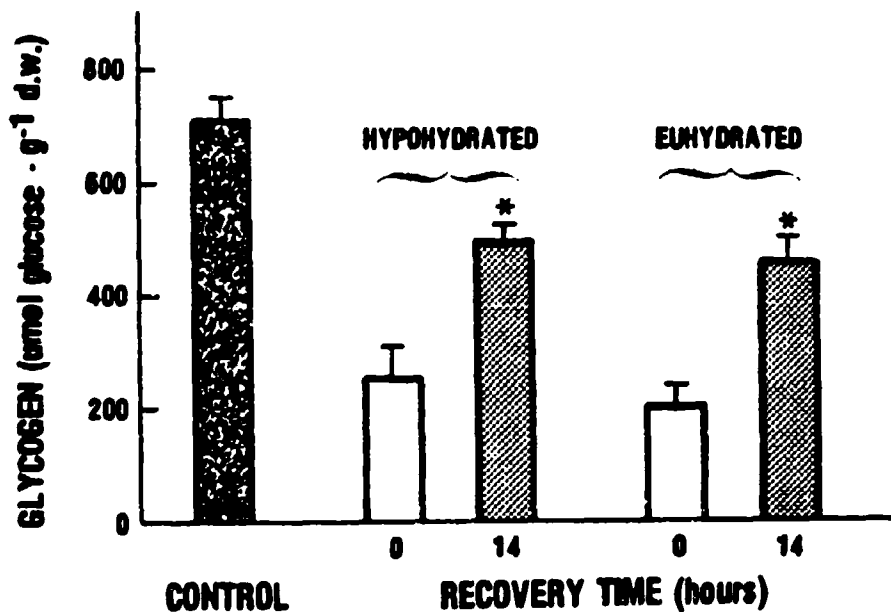


FIGURE 17. Skeletal muscle glycogen resynthesis after exercise when euhydrated and hypohydrated by 5% of body weight (113). *Significantly different (* $P < 0.05$) from time 0.

VI. SUMMARY

Individuals in military, occupational and athletic settings may perform physical exercise while incurring a body water deficit. Usually, the individual dehydrates because of fluid nonavailability or a mismatching between thirst and body water requirements. This fluid deficit is comprised of water loss from both the intracellular and extracellular fluid spaces, and will result in a decreased plasma volume and increased plasma osmolality. If diuretics are employed to dehydrate an individual, there will be a relatively greater loss of extracellular water and therefore plasma water, but tonicity will not be as high as compared to dehydration elicited from exercise-heat exposure.

Generally, the loss of body water adversely influences exercise performance. There is absolutely no evidence that hypohydration can benefit exercise performance; in addition, man cannot adapt to chronic dehydration. Exercise tasks that primarily require aerobic metabolism and are prolonged are more likely to be adversely influenced by hypohydration than exercise tasks that require anaerobic metabolism as well as muscular strength and power. Likewise, the warmer the environmental temperature, the greater the potential for hypohydration to cause decrements in exercise performance.

Hypohydration will reduce exercise performance primarily by acting through the thermoregulatory and cardiovascular systems; however, intracellular electrolyte imbalances might also contribute to performance decrements. Hypohydration causes a greater body heat storage (elevated core temperature) during exercise in a neutral or a hot environment. The greater the water deficit, the greater the elevation in core temperature during exercise; but the exercise intensity and environmental conditions probably modify the magnitude of core

temperature elevation. The greater heat storage is mediated by a decreased sweating rate as well as by a decreased cutaneous blood flow. These thermoregulatory response decrements are attributed to both a plasma hyperosmolality and a decreased blood volume. The decreased blood volume as well as the displacement of blood to the cutaneous vasculature will also make it more difficult to maintain cardiac output during exercise. The inability to maintain cardiac output during exercise in the heat when hypohydrated can result in reduced oxygen delivery to the active muscles and perhaps syncope. As summarized by Ladell (1955), during exercise in the heat "Fatigue, usually sudden in onset, was more pronounced when the water debt was high".

REFERENCES

1. Adolph, E.F. and associates. Physiology of Man in the Desert. New York: Interscience, 1947.
2. Ahlman, K. and M.J. Karvonen. Weight reduction by sweating in wrestlers, and its effect on physical fitness. J. Sports Med. Phys. Fitness 1:58-62, 1961.
3. Allan, J.R. and C.G. Wilson. Influence of acclimatization on sweat sodium concentration. J. Appl. Physiol. 30:708-712, 1971.
4. Allen, T.E., D.P. Smith and D.K. Miller. Hemodynamic response to submaximal exercise after dehydration and rehydration in high school wrestlers. Med. Sci. Sports Exerc. 9:159-163, 1977.
5. Armstrong, L.E., D.L. Costill and W.J. Fink. Influence of diuretic-induced dehydration on competitive running performance. Med. Sci. Sports Exerc. 17:456-461, 1985.
6. Armstrong, L.E., R.W. Hubbard, B.H. Jones and J.T. Daniels. Preparing Alberto Salazar for the Heat of the 1984 Olympic Marathon. Physician Sportsmed. 14:73-81, 1986.
7. Armstrong, L.E., R.W. Hubbard, P.C. Szlyk, W.T. Matthew, I.V. Sills. Voluntary dehydration and electrolyte losses during prolonged exercise in the heat. Aviat. Space. Environ. Med. 56:765-770, 1985.
8. Baker, M.A. and P.A. Doris. Control of evaporative heat loss during changes in plasma osmolality in the cat. J. Physiol. (Lond.). 328:535-545, 1982.
9. Baker, M.A., P.A. Doris and M.J. Hawkins. Effect of dehydration and hyperosmolality on thermoregulatory water loss in exercising dogs. Am. J. Physiol. 244: R516-R521, 1983.
10. Bar-Or, O., R. Dothan, O. Inbar, A. Rotshtein and H. Zonder. Voluntary hypohydration in 10- to 12-year old boys. J. Appl. Physiol. 48:104-108, 1980.
11. Bijlani, R.L. and K.N. Sharma. Effect of dehydration and a few regimes of rehydration on human performance. Indian J. Physiol. Pharmacol. 24:255-266, 1980.
12. Bosco, J.S., J.E. Greenleaf, E.M. Bernauer and D.H. Card. Effects of acute dehydration and starvation on muscular strength and endurance. Acta Physiol. Pol. 25:411-421, 1974.
13. Bosco, J.S. and R.L. Terjung. Effects of progressive hypohydration on maximal isometric muscular strength. J. Sports Med. Phys. Fitness 8:81-86, 1968.
14. Brandenberger, G., V. Candas, M. Follenius, J.P. Libert and J.M. Kahn. Vascular fluid shifts and endocrine responses to exercise in the heat. Eur. J. Appl. Physiol. 55:123-129, 1986.

15. Buskirk, E.R. and W.P. Beetham. Dehydration and body temperature as a result of marathon running. Med. Sport. 37:493-506, 1960.
16. Buskirk, E.R., P.F. Iampietro and D.E. Bass. Work performance after dehydration: effects of physical conditioning and heat acclimation. J. Appl. Physiol. 12:189-194, 1958.
17. Cadarette, B.S., M.N. Sawka, M.M. Toner and K.B. Pandolf. Aerobic fitness and the hypohydration response to exercise-heat stress. Aviat. Space Environ. Med. 55:507-512, 1984.
18. Caldwell, J.E., E. Ahonen and U. Nousiainen. Differential effects of sauna diuretic-, and exercise-induced hypohydration. J. Appl. Physiol. 57:1018-1023, 1984.
19. Candas, V., J.P. Libert, G. Brandenberger, J.C. Sagot, C. Amoros and J.M. Kahn. Hydration during exercise: effects on thermal and cardiovascular adjustments. Eur. J. Appl. Physiol. 55:113-122, 1986.
20. Claremont, A.D., D.L. Costill, W. Fink and P. Van Handel. Heat tolerance following diuretic induced dehydration. Med. Sci. Sports 8:239-243, 1976.
21. Clarke, R.S. J., R.F. Hellon and A.R. Lind. The duration of sustained contractions of the human forearm at different muscle temperatures. J. Physiol. (Lond). 143:454-473, 1958.
22. Commonwealth of Massachusetts. The Report of the Investigation of Attorney General James M. Shannon of the Class 12 Experience at the Edward W. Connelly Criminal Justice Training Center, Agawam, Massachusetts. Department of the Attorney General, October 28, 1988.
23. Conlee, R.K. Muscle glycogen and exercise endurance: a twenty year perspective. In: K.B Pandolf (ed.). Exercise and Sport Sciences Reviews, Vol. 15. New York:Macmillan Publishing Co., 1987, pp. 1-28.
24. Convertino, V.A. Fluid shifts and hydration state: effects of long-term exercise. Can. J. Sports Sci. 12:1365-1395, 1987.
25. Convertino, V.A., L.C. Keil, E.M. Bernauer and J.E. Greenleaf. Plasma volume, osmolality, vasopressin, and renin activity during graded exercise in man. J. Appl. Physiol. 50:123-128, 1981.
26. Convertino, V.A., L.C. Keil and J.E. Greenleaf. Plasma volume, renin and vasopressin responses to graded exercise after training. J. Appl. Physiol. 54:508-514, 1983.
27. Cook, D.R., W.S. Gualtiere and S.J. Galla. Body fluid volumes of college athletes and non-athletes. Med. Sci. Sports 1:217-220, 1969.

28. Costill, D.L., R. Cote' and W. Fink. Muscle water and electrolytes following varied levels of dehydration in man. J. Appl. Physiol. 40:6-11, 1976.
29. Costill, D.L., R. Cote', W.J. Fink and P. Van Handel. Muscle water and electrolyte distribution during prolonged exercise. Int. J. Sports Med. 2:130-134, 1981.
30. Costill, D.L. and B. Saltin. Muscle glycogen and electrolytes following exercise and thermal dehydration. In: H. Howald and J.R. Poortmans (eds.). Metabolic Adaptation to Prolonged Physical Exercise. Basel: Birkhauser Verlag 1975, pp. 352-360.
31. Craig, F.N. and E.G. Cummings. Dehydration and muscular work. J. Appl. Physiol. 21:670-674, 1966.
32. Doris, P.A. Osmotic regulation of evaporative water loss and body temperature by intracranial receptors in the heat-stressed cat. Pflugers Arch. 398:337-340, 1983.
33. Draper, E.S. and J.J. Lombardi. Combined Arms in a Nuclear/Chemical Environment: Force Development Testing and Experimentation. Summary Evaluation Report Phase I. Ft. McClellan, AL: U.S. Army Chemical School, 1986.
34. Durkot, M.J., O. Martinez, D. McQuade and R. Francesconi. Simultaneous determination of fluid shifts during thermal stress in a small-animal model. J. Appl. Physiol. 61:1031-1034, 1986.
35. Edwards, R.H.T. and O.C.J. Lippold. Effect of temperature on muscle energy metabolism and endurance during successive isometric contractions sustained to fatigue of the quadriceps muscle in man. J. Physiol. (Lond.) 220:335-352, 1972.
36. Eichna, L.W., W.B. Bean, W.F. Ashe and N. Nelson. Performance in relation to environmental temperature. Bull. John Hopkins Hosp. 76:25-58, 1945.
37. Ekblom, B., C.J. Greenleaf, J.E. Greenleaf and L. Hermansen. Temperature regulation during exercise dehydration in man. Acta Physiol. Scand. 79:475-583, 1970.
38. Engell, D.B., O. Maller, M.N. Sawka, R.P. Francesconi, L. Drolet and A.J. Young. Thirst and fluid intake following graded hypohydration levels in humans. Physiol. Behav. 40:226-236, 1987.
39. Faulkner, J.A. Heat and contractile properties of skeletal muscle. In: S.M. Horvath and M.K. Yousf (eds.). Environmental Physiology: Aging, Heat and Altitude. New York: Elsevier/North Holland, 1980, pp. 191-203.
40. Fortney, S.M., E.R. Nadel, C.B. Wenger and J.R. Bove. Effect of acute alterations of blood volume on circulatory performance in humans. J. Appl. Physiol. 50:292-298, 1981.

41. Fortney, S.M., E.R. Nadel, C.B. Wenger and J.R. Bove. Effect of blood volume on sweating rate and body fluids in exercising humans. J. Appl. Physiol. 51:1594-1600, 1981.
42. Fortney, S.M., C.B. Wenger, J.R. Bove and E.R. Nadel. Effect of hyperosmolality on control of blood flow and sweating. J. Appl. Physiol. 57:1688-1695, 1984.
43. Francesconi, R.F., R. Byrom and M. Mager. United States Army Research Institute of Environmental Medicine: First Quarter Century. The Physiologist. 29:58-62, 1986.
44. Francesconi, R.P., M.N. Sawka and K.B. Pandolf. Hypohydration and heat acclimation: plasma renin and aldosterone during exercise. J. Appl. Physiol. 55:1790-1794, 1983.
45. Francesconi, R.P., M.N. Sawka, K.B. Pandolf, R.W. Hubbard, A.J. Young and S. Muza. Plasma hormonal responses at graded hypohydration levels during exercise-heat stress. J. Appl. Physiol. 59:1855-1860, 1985.
46. Gass, G.C., E.M. Camp, J. Watson, D. Eager, L. Wicks and A. Ng. Prolonged exercise in highly trained female endurance runners. Int. J. Sports Med. 4:241-246, 1983.
47. Gisolfi, C.V. and J.R. Copping. Thermal effects of prolonged treadmill exercise in the heat. Med. Sci. Sports 6:108-113, 1974.
48. Gopinathan, P.M., G. Pichan and V.M. Sharma. Role of dehydration in heat stress-induced variations in mental performance. Arch. Environ. Health. 43:15-17, 1988.
49. Grande, F., J.E. Monagle, E.R. Buskirk and H.L. Taylor. Body temperature responses to exercise in man on restricted food and water intake. J. Appl. Physiol. 14:194-198, 1959.
50. Greenleaf, J.E., P.J. Brock, L.C. Keil and J.T. Morse. Drinking and water balance during exercise and heat acclimation. J. Appl. Physiol. 54:414-419, 1983.
51. Greenleaf, J.E. and B.L. Castle. Exercise temperature regulation in man during hypohydration and hyperhydration. J. Appl. Physiol. 30:847-853, 1971.
52. Greenleaf, J.E., S. Kozlowski, K. Nazar, H. Kaciuba-Uscilko, Z. Brzezinska and A. Ziemba. Ion-osmotic hyperthermia during exercise in dogs. Am. J. Physiol. 230:74-79, 1976.
53. Greenleaf, J.E., M. Matter, J.S. Bosco, L.G. Douglas and E.G. Averkin. Effects of hypohydration on tolerance to +G_z acceleration in man. Aerospace Med. 37:34-39, 1966.
54. Greenleaf, J.E., E.M. Prange and E.G. Averkin. Physical performance of women following heat-exercise hypohydration. J. Appl. Physiol. 22:55-60, 1967.
55. Harrison, M.H., R.J. Edwards and P.A. Fennessy. Intravascular volume and tonicity as factors in the regulation of body temperature. J. Appl. Physiol. 44:69-75, 1978.

56. Hays, R.M. Dynamics of body water and electrolytes. In: Clinical Disorders of Fluid and Electrolyte Metabolism, edited by M.X. Maxwell and C.R. Kleeman. New York: McGraw-Hill, 1980, p.1-6.
57. Herbert, W.G. and P.M. Ribisl. Effects of dehydration upon physical working capacity of wrestlers under competitive conditions. Res. Quart. 43:416-422, 1971.
58. Hertzman, A.B. and I.D. Ferguson. Failure in temperature regulation during progressive dehydration. U.S. Armed Forces Med. J. 11:542-560, 1960.
59. Horstman, D.H. and S.M. Horvath. Cardiovascular and temperature regulatory changes during progressive dehydration and euhydration. J. Appl. Physiol. 33:446-450, 1972.
60. Horvath, S.M. and E.C. Horvath. The Harvard Fatigue Laboratory: Its History and Contributions. Englewood Cliffs, NJ: Prentice-Hall Inc., 1973.
61. Houston, M.E., D.A. Marrin, H.J. Green and J.A. Thomson. The effect of rapid weight loss on physiological functions in wrestlers. Physician Sportsmed. 9:73-78, 1981.
62. Hubbard, R.W. and L.E. Armstrong. The heat illnesses: biochemical, ultrastructural, and fluid-electrolyte considerations. In: K.B. Pandolf, M.N. Sawka and R.R. Gonzalez, (eds). Human Performance Physiology and Environmental Medicine at Terrestrial Extremes. Indianapolis: Benchmark Press, 1988, pp. 305-359.
63. Hubbard, R., M. Mager and M. Kerstein. Water as a tactical weapon: A doctrine for preventing heat casualties. Army Sci. Conf. Proc. 125-139, 1982.
64. Hubbard, R.W., B.L. Sandick, W.T. Matthew, R.P. Francesconi, J.B. Sampson, M.J. Durkot, O. Maller and D.B. Engell. Voluntary dehydration and alliesthesia for water. J. Appl. Physiol. 57:868-875, 1984.
65. Jacobs, I. The effects of thermal dehydration on performance of the Wingate Anaerobic Test. Int. J. Sports Med. 1:21-24, 1980.
66. Kirby, C.R. and V.A. Convertino. Plasma aldosterone and sweat sodium concentrations after exercise and heat acclimation. J. Appl. Physiol. 61:967-970, 1986.
67. Kirsch, K.A., H. von Ameln and H.J. Wicke. Fluid control mechanisms after exercise dehydration. Eur. J. Appl. Physiol. 47:191-196, 1981.
68. Knochel, J.P. Dog days and Siriasis: How to kill a football player. J. Am. Med. Assoc. 233:513-515, 1975.
69. Kolka, M.A., L.A. Stephenson and J.E. Wilkerson. Erythrocyte indices during a competitive marathon. J. Appl. Physiol. 52:168-172, 1982.

70. Kozlowski, S., J.E. Greenleaf, E. Turlejska and K. Nazar. Extracellular hyperosmolality and body temperature during physical exercise in dogs. Am. J. Physiol. 239 R180-R183, 1980.
71. Kozlowski, S. and B. Saltin. Effect of sweat loss on body fluids. J. Appl. Physiol. 19:1119-1124, 1964.
72. Kristal-Boneh, E., J.G. Glusman, C. Chaemovitz and Y. Cassuto. Improved thermoregulation caused by forced water intake in human desert dwellers. Eur. J. Appl. Physiol. 57:220-224, 1988.
73. Kubica, R., B. Nielsen, A. Bonnesen, I.B. Rasmussen, J. Stoklosa and B. Wilk. Relationship between plasma volume reduction and plasma electrolyte changes after prolonged bicycle exercise, passive heating and diuretic dehydration. Acta. Physiol. Pol. 34:569-579, 1983.
74. Ladell, W.S.S. The effects of water and salt intake upon the performance of men working in hot and humid environments. J. Physiol. (Lond.) 127:11-46, 1955.
75. Loeppky, J.A., L.G. Myhre, M.D. Venters and U.C. Luft. Total body water and lean body mass estimated by ethanol dilution. J. Appl. Physiol. 42:803-808, 1977.
76. Mnatzakian, P.A. and P. Vaccaro. Effects of 4% dehydration and rehydration on hematological profiles, urinary profiles and muscular endurance of college wrestlers. Med. Sci. Sports Exerc. (Abst.) 14:117, 1982.
77. Molnar, G.W., E.J. Towbin, R.E. Gosselin, A.H. Brown and E.F. Adolph. A comparative study of water, salt and heat exchanges of men in tropical and desert environments. Am. J. Hyg. 44:411-433, 1946.
78. Morimoto, T.K., K. Miki, H. Nose, S. Yamada, K. Hirakawa and C. Matsubara. Changes in body fluid volume and its composition during heavy sweating and the effects of fluid and electrolyte replacement. Jpn. J. Biometeorol. 18:31-39, 1981.
79. Moroff, S.V. and D.E. Bass. Effects of overhydration on man's physiological responses to work in the heat. J. Appl. Physiol. 20:267-270, 1965.
80. Muza, S.R., N.A. Pimental, H.M. Cosimini and M.N. Sawka. Portable ambient air microclimate cooling in simulated desert and tropic conditions. Aviat. Space Environ. Med. 59:553-558, 1988.
81. Myhre, L.G. and S. Robinson. Fluid shifts during thermal stress with and without fluid replacement. J. Appl. Physiol. 42:252-256, 1977.
82. Nadel, E.R., S.M. Fortney and C.B. Wenger. Effect of hydration on circulatory and thermal regulation. J. Appl. Physiol. 49:715-721, 1981.

83. Nakashima, T., T. Hori, T. Kiyohara and M. Shibata. Effects of local osmolality changes on medial preoptic thermosensitive neurons in hypothalamic slices In Vitro. Thermal Physiology 9:133-137, 1984.
84. Neuffer, P.D., A.J. Young and M.N. Sawka. Gastric emptying during walking and running: effects of varied exercise intensity. Eur. J. Appl. Physiol. 58:440-445, 1989.
85. Neuffer, P.D., A.J. Young and M.N. Sawka. Gastric emptying during exercise: effects of heat stress and hypohydration. Eur. J. Appl. Physiol. 58:433-439, 1989.
86. Neuffer, P.D., A.J. Young, M.N. Sawka, M.D. Quigley, L. Levine and W.A. Latzka. Hypohydration and muscle glycogen resynthesis. Med. Sci. Sports Exerc. (Abstract), 21:S19, 1989.
87. Neuffer, P.D., A.J. Young, M.N. Sawka, M.D. Quigley, L. Levine and W.A. Latzka. Substrate levels and muscle metabolism while hypohydrated. FASEB Journal (Abstract), 3:A990, 1989.
88. Nielsen, B. Effects of changes in plasma volume and osmolality on thermoregulation during exercise. Acta Physiol. Scand. 90:725-730, 1974.
89. Nielsen, B., G. Hansen, S.O. Jorgensen and E. Nielsen. Thermoregulation in exercising man during dehydration and hyperhydration with water and saline. Int. J. Biometeorol. 15:195-200, 1971.
90. Nielsen, B., R. Kubica, A. Bonnesen, I.B. Rassmussen, J. Stoklosa and B. Wilk. Physical work capacity after dehydration and hyperthermia: a comparison of the effect of exercise versus passive heating and sauna and diuretic dehydration. Scand. J. Sport Sci. 3:2-10, 1981.
91. Nielsen, M. Die Regulation der Korpertemperatur bei Muskelarbeit. Scand. Arch. Physiol. 79:193-230, 1938.
92. Nose, H., G.W. Mack, X. Shi and E.R. Nadel. Shift in body fluid compartments after dehydration in humans. J. Appl. Physiol. 65:318-324, 1988.
93. Nose, H., T. Morimoto and K. Ogura. Distribution of water losses among fluid compartments of tissues under thermal dehydration in the rat. Jpn. J. Physiol. 33:1019-1029, 1983.
94. Olsson, K.-E. and B. Saltin. Variation in total body water with muscle glycogen changes in man. Acta Physiol. Scand. 80:11-18, 1970.
95. Owen, M.S., K.C. Kregel, P.T. Wall and C.V. Gisolfi. Effects of ingesting carbohydrate beverages during exercise in the heat. Med. Sci. Sports Exerc. 18:568-575, 1986.

96. Percy, M., S. Robinson, D.I. Miller, J.T. Thomas and J. De Brota. Effects of dehydration, salt depletion and pitressin on sweat rate and urine flow. J. Appl. Physiol. 8:621-626, 1956.
97. Petrofsky, J.S. and A.R. Lind. The relationship of body fat content to deep muscle temperature and isometric endurance in man. Clin. Sci. Mol. Med. 48:405-412, 1975.
98. Phillips, P.A., B.J. Rolls, J.G.G. Ledingham, M.I. Forsling, J.J. Morton, M.J. Crowe and L. Wollner. Reduced thirst after water deprivation in healthy elderly men. N. Engl. J. Med. 311:753-759, 1984.
99. Pimental, N.A., H.M. Cosimini, M.N. Sawka and C.B. Wenger. Effectiveness of an air-cooled vest using selected air temperature and humidity combinations. Aviat. Space Environ. Med. 58:119-124, 1987.
100. Pitts, G.C., R.E. Johnson, F.C. Consolazio. Work in the heat as affected by intake of water, salt and glucose. Am. J. Physiol. 142:253-259, 1956.
101. Pivarnik, J.M., E.M. Leeds and J.E. Wilkerson. Effects of endurance exercise on metabolic water production and plasma volume. J. Appl. Physiol. 56:613-618, 1984.
102. Pugh, L.G.C.E., J.L. Corbett and R.H. Johnson. Rectal temperatures, weight losses and sweat rates in marathon running. J. Appl. Physiol. 23:347-352, 1967.
103. Rowell, L.B. Cardiovascular aspects of human thermoregulation. Circ. Res. 52:367-379, 1983.
104. Saltin, B. Aerobic and anaerobic work capacity after dehydration. J. Appl. Physiol. 19:1114-1118, 1964.
105. Saltin, B. Circulatory response to submaximal and maximal exercise after thermal dehydration. J. Appl. Physiol. 19:1125-1132, 1964.
106. Sawka, M.N. Body fluid responses and hypohydration during exercise-heat stress. In: K.B. Pandolf, M.N. Sawka and R.R. Gonzalez, (eds). Human Performance Physiology and Environmental Medicine at Terrestrial Extremes. Indianapolis: Benchmark Press, 1988, pp. 227-266.
107. Sawka, M.N., R.P. Francesconi, N.A. Pimental and K.B. Pandolf. Hydration and vascular fluid shifts during exercise in the heat. J. Appl. Physiol. 56:91-96, 1984.
108. Sawka, M.N., R.P. Francesconi, A.J. Young and K.B. Pandolf. Influence of hydration level and body fluids on exercise performance in the heat. J. Am. Med. Assoc. 252:1165-1169, 1984.

109. Sawka, M.N., R.R. Gonzalez, A.J. Young, R.C. Dennis, C.R. Valeri and K.B. Pandolf. Control of thermoregulatory sweating during exercise in the heat. Am. J. Physiol. 257: R311-R316, 1989.
110. Sawka, M.N., R.R. Gonzalez, A.J. Young, S.R. Muza, K.B. Pandolf, W.A. Latzka, R.C. Dennis and C.R. Valeri. Polycythemia and hydration: effects on thermoregulation and blood volume during exercise-heat stress. Am. J. Physiol. 255:R456-R463, 1988.
111. Sawka, M.N., R.G. Knowlton and J.B. Critz. Thermal and circulatory responses to repeated bouts of prolonged running. Med. Sci. Sports. 11:177-180, 1979.
112. Sawka, M.N., R.G. Knowlton and R.G. Glaser. Body temperature, respiration and acid-base equilibrium during prolonged running. Med. Sci. Sports Exerc. 12:370-374, 1980.
113. Sawka, M.N. and P.D. Neuffer. Interaction of water bioavailability, thermoregulation and exercise performance. In: Fluid Replacement and Heat Stress. E.R. Buskirk and S. Berkow, Eds., Washington, DC: National Academy of Sciences, (In Press), 1989.
114. Sawka, M.N. and K.B. Pandolf. Effects of body water loss on exercise performance and physiological functions. In: Perspectives in Exercise Science and Sports Medicine: Volume 3. Fluid Homeostasis During Exercise. C.V. Gisolfi and D.R. Lamb, Eds., Indianapolis, IN: Benchmark Press, (In Press), 1990.
115. Sawka, M.N., M.M. Toner, R.P. Francesconi and K.B. Pandolf. Hypohydration and exercise: effects of heat acclimation, gender and environment. J. Appl. Physiol. 55:1147-1153, 1983.
116. Sawka, M.N., A.J. Young, B.S. Cadarette, L. Levine and K.B. Pandolf. Influence of heat stress and acclimation on maximal aerobic power. Eur. J. Appl. Physiol. 53:294-298, 1985.
117. Sawka, M.N., A.J. Young, R.C. Dennis, R.R. Gonzalez, K.B. Pandolf and C.R. Valeri. Human intravascular immunoglobulin responses to exercise-heat and hypohydration. Aviat. Space Environ. Med. 60:634-638, 1989.
118. Sawka, M.N., A.J. Young, R.P. Francesconi, S.R. Muza and K.B. Pandolf. Thermoregulatory and blood responses during exercise at graded hypohydration levels. J. Appl. Physiol. 59:1394-1401, 1985.
119. Schloerb, P.R., B.J. Friis-Hansen, I.S. Edelman, A.K. Solomon and F.D. Moore. The measurement of total body water in the human subject by deuterium oxide dilution with a consideration of the dynamics of deuterium distribution. J. Clin. Invest. 29:1296-1310, 1950.
120. Senay, L.C. Relationship of evaporative rates to serum Na⁺, K⁺ and osmolarity in acute heat stress. J. Appl. Physiol. 25:149-152, 1968.

121. Senay, L.C. Temperature regulation and hypohydration: a singular view. J. Appl. Physiol. 47:1-7, 1979.
122. Senay, L.C. and M.L. Christensen. Changes in blood plasma during progressive dehydration. J. Appl. Physiol. 20:1136-1140, 1965.
123. Senay, L.C. and M.L. Christensen. Respiration of dehydrating men undergoing heat stress. J. Appl. Physiol. 22:282-286, 1967.
124. Serfass, R.C., G.A. Stull, J.F. Alexander and J.L. Ewing. The effects of rapid weight loss and attempted rehydration on strength and endurance of the handgripping muscles in college wrestlers. Res. Quart. 55:46-52, 1984.
125. Shapiro, Y., K.B. Pandolf, R.F. Goldman. Predicting sweat loss response to exercise, environment and clothing. Eur. J. Appl. Physiol. 48:83-96, 1982.
126. Shapiro, Y., K.B. Pandolf, M.N. Sawka, M.M. Toner, F.R. Winsmann and R.F. Goldman. Auxilliary cooling: Comparison of air-cooled vs. water-cooled vests in hot-dry and hot-wet environments. Aviat. Space Environ. Med. 53:785-789, 1982.
127. Sherman, W.M., M.J. Plyley, R.L. Sharp, P.J. Van Handle, R.M. McAllister, W.J. Fink and D.L. Costill. Muscle glycogen storage and its relationship with water. Int. J. Sports Med. 3:22-24, 1982.
128. Singer, R.N. and S.A. Weiss. Effects of weight reduction on selected anthropometric, physical, and performance measures in wrestlers. Res. Quart. 39:361-369, 1968.
129. Silva, N.L. and J.A. Boulant. Effects of osmotic pressure, glucose and temperature on neurons in preoptic tissue slices. Am. J. Physiol. 247:R335-R345, 1984.
130. Sjogaard, G. Water and electrolyte fluxes during exercise and their relation to muscle fatigue. Acta Physiol. Scand. 128 (Suppl. 556):129-136, 1986.
131. Skelton, H. The storage of water by various tissues of the body. Arch. Int. Med. 40:140-152, 1927.
132. Smith, J.H., S. Robinson and M. Percy. Renal responses to exercise, heat and dehydration. J. Appl. Physiol. 4:659-665, 1952.
133. Sproles, C.B., D.P. Smith, R.J. Byrd and T.E. Allen. Circulatory responses to submaximal exercise after dehydration and rehydration. J. Sports Med. Phys. Fit. 16:98-105, 1976.
134. Stephenson, L.A., A. Tripathi, C.B. Wenger, J.R. Bove and E.R. Nadel. Plasma volume expansion during hypovolemic exercise (Abst.). Fed. Proc. 42:585, 1983.

135. Strauss, R.H., J.E. Wright, G.A.M. Finerman and D.H. Catlin. Side effects of anabolic steroids in weight-trained men. Physician Sportsmed. 11:87-96, 1983.
136. Strydom, N.B. and L.D. Holdsworth. The effects at different levels of water deficit on physiological responses during heat stress. Int. Z. Angew. Physiol. 26:95-102, 1968.
137. Strydom, N.B. and C.H. Wyndham and C.H. van Graan et al. The influence of water restriction on the performance of men during a prolonged march. S. Afr. Med. J. 40:539-544, 1966.
138. Swamy, W., G. Pichan and H.S. Nayar. Effects of body dehydration on soldiers during military operations in heat. Proceedings: Thirteenth Commonwealth Defence Conference on Operational Clothing and Combat Equipment 1-8, 1981.
139. Tipton, C.M. and T.K. Tchong. Iowa wrestling study: weight loss in high school students. J. Am. Med. Assoc. 214:1269-1274, 1970.
140. Torranin, C., D.P. Smith and R.J. Byrd. The effect of acute thermal dehydration and rapid rehydration on isometric and isotonic endurance. J. Sports Med. Phys. Fitness 19:1-9, 1979.
141. Turlejska, E. and M.A. Baker. Elevated CSF osmolality inhibits thermoregulatory heat loss responses. Am. J. Physiol. 251:R749-R754, 1986.
142. Tuttle, W.W. The effect of weight loss by dehydration and the withholding of food on the physiological responses of wrestlers. Res. Quart. 14:158-166, 1943.
143. Vanderwalle, H., C. Lacombe, J.c. Lelievre and C. Poirot. Blood viscosity after a 1-h submaximal exercise with and without drinking. Int. J. Sports Med. 9:104-107, 1988.
144. Webster, S.F., R.A. Rutt and A. Weltman. Effects of typical dehydration practices on performance. Med. Sci. Sports Exerc. (Abst.) 20:S 20, 1988.
145. Weiner, I.M. and G.H. Mudge. Diuretics and other agents employed in the mobilization of edema fluid. In: A.G. Gilman, L.S. Goodman, T.W. Rall and F. Murad (eds.) The Pharmacological Basis of Therapeutics. New York: MacMillan Publishing Co., 1985, pp. 887-907.
146. Wenger, C.B. Heat evaporation of sweat: thermodynamic considerations. J. Appl. Physiol. 32:456-459, 1972.
147. Zambraski, E.J., D.T. Foster, P.M. Gross and C.M. Tipton. Iowa wrestling study: weight loss and urinary profiles of collegiate wrestlers. Med. Sci. Sports 8:105-108, 1976.

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