editorial review

Temperature regulation and hypohydration: a singular view

SENAY, LEO C., JR. *Temperature regulation and hypohydration: a singular view.* J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 47(1): 1–7, 1979.—Body temperatures of exercising humans who have been denied water are elevated when compared to hydrated controls. The simplest "explanation" for the elevated temperature is a decrease in sensitivity of the sweating mechanism. This and similar "explanations" do not direct attention to basic causes but only the result(s) of more fundamental aspects of regulatory physiology. Among the items considered in this speculative presentation are influences of changes in osmolarity, specific ions, peptide hormones, fluid shifts, and muscular contractions during exercise. A hypothesis is offered for consideration in explaining elevations of body temperature in exercise with and without water replacement. In general, the hypothesis relates changes in hypothalamic osmotic pressure and/or ionic constituents with fluid and ionic events in muscle during exercise. The fluid and ionic shifts are probably proportional to the amount of lean body mass engaged in dynamic exercise. Since blood volume has also been shown to be related to lean body mass, similar relative work loads should lead to similar changes in the osmotic and/or ionic environment of the hypothalamus, thus resulting in similar increases in body temperature during exercise. Hypohydration is superimposed on this basic response. Increases in body temperature of resting hypohydrated subjects appear to be due to increases in osmotic pressure and/or specific ion concentrations. During exercise, these changes are added to those induced by muscle contraction. The focal point of all such ionic and osmotic changes is thought to be neural processes within the hypothalamus.

evaporative water loss; exercise; hypothalamus; ionic concentrations; muscle contractions; osmolarity; plasma volume

RECEPTORS THAT AFFECT the regulation of body temperature are in a variety of locations within certain animals (24). The applicability of all such experimental findings to man remains to be proven but the central role of the hypothalamus in integrated responses to environmental temperature fluctuations in both man and animals appears to be firmly established (3, 4, 18). Without entering into the debate as to whether the hypothalamus possesses a "set point" or is simply an integrator of inputs, one can reasonably claim that the regulation of body temperature can be best understood if it is considered to be a neural process which is subject to a number of modifying influences other than regional variations in body temperatures.

One of the factors that affect body temperature regulation is water balance (1, 19, 41). It is my purpose to review certain findings which emphasize the complexity of physiological responses resulting from alterations in the volume and character of body fluids particularly as they pertain to the regulation of body temperature.

With humans as subjects, a simple demonstration of this interaction can be seen in an experiment in which water is withheld. The subjects may be exercising or at rest in a variety of environments and since water loss becomes progressively greater such a treatment has been labeled "progressive dehydration" (19). As noted by Greenleaf and Sargent (16), this term lacks descriptive precision because it does not differentiate between salt or water loss and should be replaced by "hypohydration," which indicates a greater loss of water than salt. If hypohydration is produced in resting subjects in cool surroundings wherein most of the heat loss from the body is by radiation and convection little change in body temperature is noted. As the surrounding temperature is increased and responsibility for heat loss shifts toward evaporation it soon becomes evident that, for a given exposure time to a warm environment, the body temperature is higher in hypohydration than with water replacement (8, 14, 15). Clearly, the transfer of heat from body to environment is compromised and under the conditions

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of the experiment the fault may lie with either convective transfer of heat within the body or the transfer of heat from the body surface to the environment or both. In examining what does occur, a general consideration of the main effects of hypohydration upon humans when at rest and when exercising forms the basis for later suggestions as to cause and effect.

Hypohydration At Rest

For resting subjects in warm environments (≥35–40°C), hypohydration is accompanied by increases in core and skin temperatures (19). As exposure continues the core-to-skin gradient remains virtually constant, which implies that for moderate degrees of hypohydration cutaneous blood flow is not diminished (41).

Evaporative water loss. Changes in rates of whole-body water loss during progressive hypohydration of resting subjects have been difficult to detect, mainly because the instruments and methods employed were insensitive to the changes that occurred, i.e., balances with sensitivities of ±50 g for example. To detect changes in sweat rate (as reflected in rates of reduction in body weight) in resting subjects during progressive hypohydration, the results of hypohydration experiments must be compared with those of rehydration experiments (38) with care being taken as to the state of hydration of the subjects before each exposure and of equal restrictions on subject activity during exposures. With these precautions, it has been shown that during progressive hypohydration subjects do sweat slightly less than during rehydration experiments (Fig. 1). The elevation in body temperature that accompanies the slight reduction in sweating suggested to Hertzman and Ferguson (19) that the sensitivity of the sweating mechanism was reduced. The exact location of the reduced sensitivity has not been ascertained but responses to water and saline ingestion by hypohydrated subjects suggest a reduction in a central process (42).

Plasma water loss. There has been a revival of interest in body fluid changes brought about either singly or in a combination of conditions including exercise, heat exposure, and hypohydration (40). One of the main controversies concerns the reduction of plasma volume during progressive hypohydration. Adolph and associates (1) claimed that water was lost from the plasma at a rate 2.5 times that of the body. Lee and Mulder (23) had indicated some 12 years earlier that the ratio was 2. Depending on experimental conditions, ratios of plasma to total body water loss of up to 5 have been reported (39). My own findings indicate that the plasma of resting subjects in the heat loses water at a rate equal to that of the rest of the body (39). There is no argument as to the accuracy of the observations but there is a serious question as to the state of hydration of subjects at the start of the experiments. For example, the plasma osmolarity of hydrated individuals has been found to be between 280 and 288 mosmol/l plasma. In a recent study, which was designed to examine the effects of hypohydration on body fluids, mean control plasma osmolarities in excess of 290 mosmol/l were reported (2). Because the state of hydration of the control subjects will influence the results and conclusions in such studies, more attention must be paid to the control conditions before starting the experiments (see below and Ref. 17).

More disturbing, however, are those experimental find-

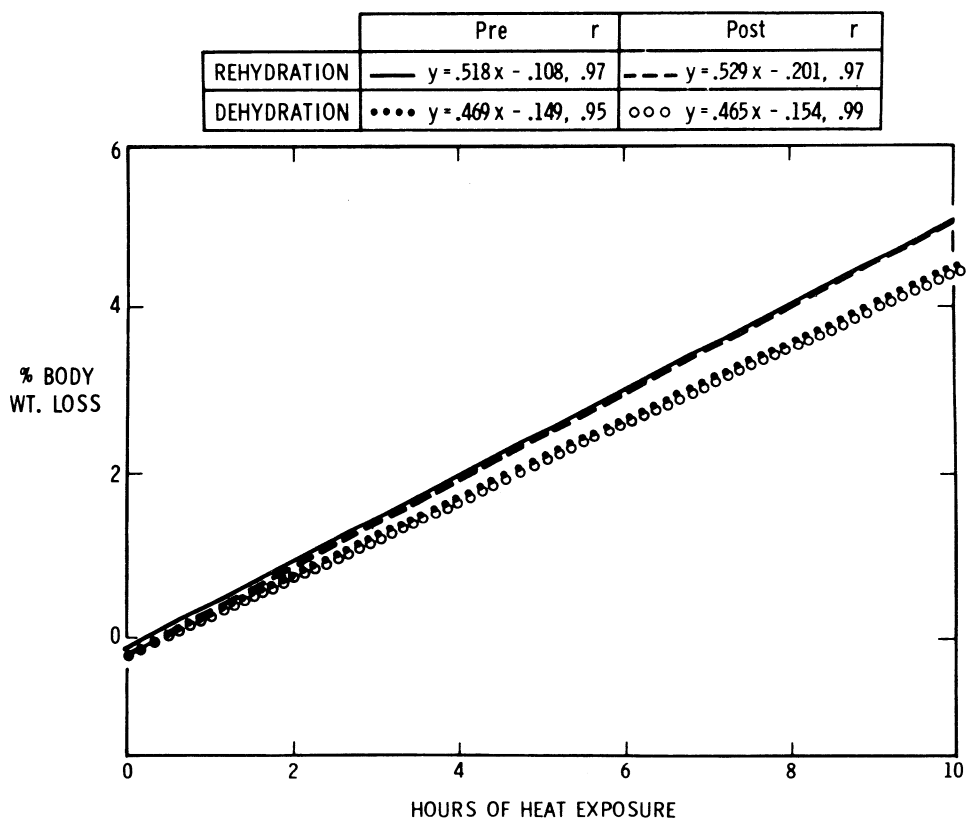

FIG. 1. Comparison of rates of weight loss for 5 women with and without rehydration before (Pre) and after (Post) ovulation. Subjects were at rest; 43.3°C db, 28–29°C wb. (Data taken from Ref. 38.)

ings wherein progressive hypohydration led to inequitable losses of plasma volume (see Ref. 39 for summary). Clearly, protein osmotic pressure must increase and, given equal permeation of extracellular space by inorganic ions, water must diffuse out of cells into the extracellular volume. Water does so move in other species (20, 22) and why Fick's law is apparently not obeyed in certain human subjects has not been explained.

Be that as it may, during progressive hypohydration, the water content of the body is reduced, the plasma volume sharing in this reduction (39). For an individual at rest, this does not pose a problem, but the reduced circulating blood volume probably contributes to orthostatic hypotension if the subject attempts to stand. Reduction in blood volume also leads to further reduction in kidney blood flow and probable reductions of blood flows to other splanchnic areas (9, 29, 45).

This reduction in body water is mainly due to sweating but respiratory water loss also contributes. The net result is that water is lost at a greater rate than are the ionic constituents, and thus body fluid osmolarity rises. Because of the reduction in kidney blood flow and the increased presence of certain hormones, particularly antidiuretic hormone (ADH), urine output is considerably reduced and although high urine osmolarity and ionic concentrations are attained early during progressive hypohydration, neither the plasma osmolarity nor the individual ionic constituents are effectively controlled by the kidney.

Finally, in resting man, progressive hypohydration is accompanied by increases in heart rate and reductions in stroke volume such that though blood volume is reduced cardiac output remains almost constant (1, 33).

Hypohydration and Exercise

Turning our attention to exercising man, we note that progressive or maintained hypohydration does cause increased heart rates and reduced stroke volumes at submaximal exercise intensities (5, 33, 34). Endurance times at all exercise levels are reduced: the more severe the hypohydration, the shorter is the work time. Internal body temperature is higher at any stated exercise intensity when compared with that prevailing for the same subject when fully hydrated (12). Also, hypohydrated subjects have been shown to suffer a reduction in sweat output (15–20%) in spite of the comparatively elevated internal body temperature (32). It would appear that the problems of the hypohydrated exercising man are exaggerations of those occurring during rest.

A Closer View of Hypohydration

The influence of changes in body fluids on body temperature regulation during hypohydration will now be considered more closely in order to particularly emphasize deficits in knowledge that accompany broad pronouncements.

Rest: sweating. For individuals at rest, the progressive loss of water and rise in body temperature are accompanied by a sweat rate that remains rather constant (1, 19, 41). This has been taken to mean that the sweating mechanism is less sensitive to increases in body temperature (19). The statement that moderate degrees of hypohydration do not greatly affect sweat rate is generally correct (31), but this does not explain why the body temperature rises. However, deficits in sweat rates that account for the rise in body temperature are small and, with weight loss taken to equal sweat loss, are of the order of 10 $g \cdot h^{-1} \cdot m^{-2}$ when db = 43.3°C, wb = 27–29°C. Clearly then, the rise in body temperature of a resting subject hypohydrating in the heat appears to be the consequence of a reduction in the rate of evaporative heat loss. What is the cause of this event? Several items need be considered.

First, an increase in the osmolarity of body fluids accompanies progressive hypophydration and, this increase is the sum of the changes in concentration of constituent ions (Fig. 2). This relationship appears obvious, but occasionally results are published that are internally inconsistent. By comparing sweat rates in rehydrated and dehydrated subjects, I have shown that a significant inverse relationship existed between osmolarity and sweat rate (assuming that weight loss = sweat) during progressive hypohydration of resting man (Fig. 3). There was also a significant relationship between Na^+ concentration and sweat rate (Fig. 4). It is likely that the osmotic effect is primarily due to changes in Na^+ concentration, but this remains to be proven.

Hypohydration, osmolarity, and ions. Indeed, it is doubtful that the events seen in progressive hypohydration can be ascribed to a single factor, for water deprivation alters many body functions. Thus, observations limited to measurements of body temperature and selected body parameters automatically bias the interpretation of results. Using evidence at hand in the literature, assume that we have placed human subjects in a warm environment without water and we make certain measurements on these subjects as they become hypohydrated. The usual measures show a rise in skin temperature, a rise in core temperature, a slight fall in sweat rate, an increased body fluid osmolarity, and a decrease in circulating blood volume. So, let us select from this limited group of observations a particular measurement, i.e., increased body fluid osmolarity. Is it the change in osmolarity per se that causes body temperature to rise, or is it perhaps one of the constituent ions that is more directly responsible for changes in body temperature? Evidence from experiments wherein the concentrations of Na^+ and of Ca^{2+} have been altered in the cerebrospinal fluid (26) seems to indicate that body temperature regulation may depend on the ratio of Na^+ to Ca^{2+} in the vicinity of neurons within the hypothalamus. Changes in body temperatures of human subjects upon ingestion of concentrated Ca^{2+} or Na^+ solutions (27) appears generally to support the animal studies, and both types of studies indicate that increases in Na^+ concentration may be the cause of increases in body temeprature. But is this really so? Consider the progressively hypohydrating subject: in the resting subject the increase in body fluid osmotic pressure is also accompanied by a gradual increase in blood pH (44). The decrease in hydrogen ion concentration, accompanied by an increase in the relative concentrations of all proteins in the body, must certainly

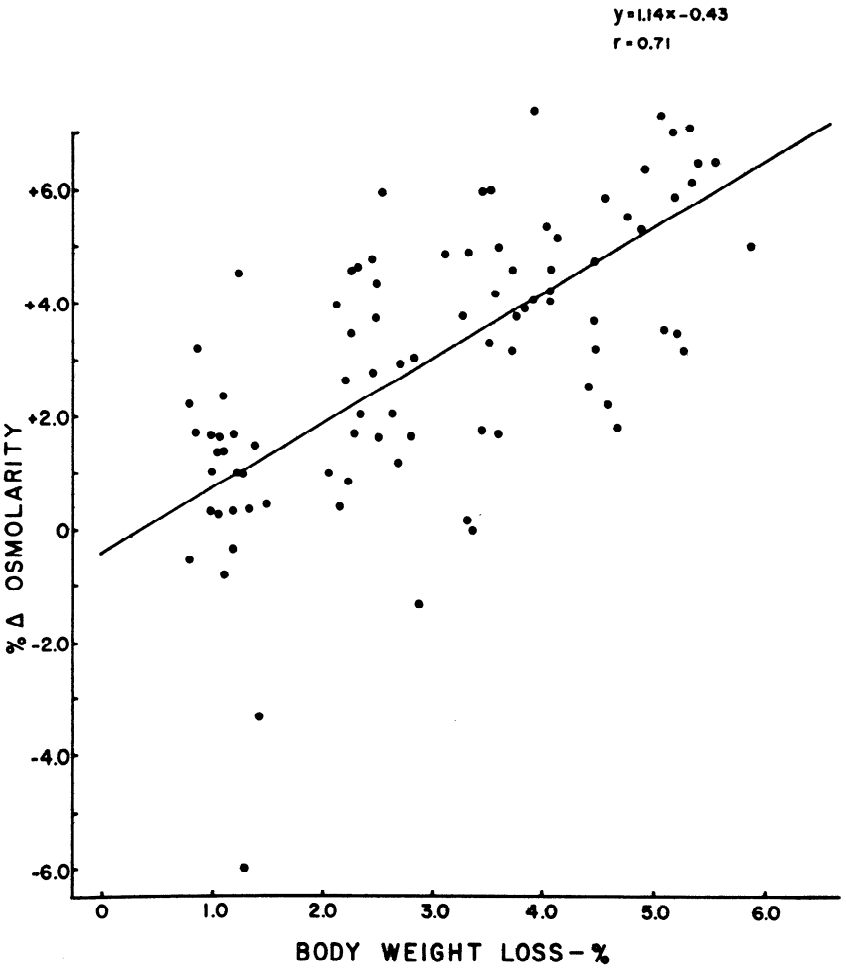

FIG. 2. Relationship of changes in osmolarity to body weight loss of 12 subjects at 43.3°C db, 29°C wb. (Taken from Ref. 43.)

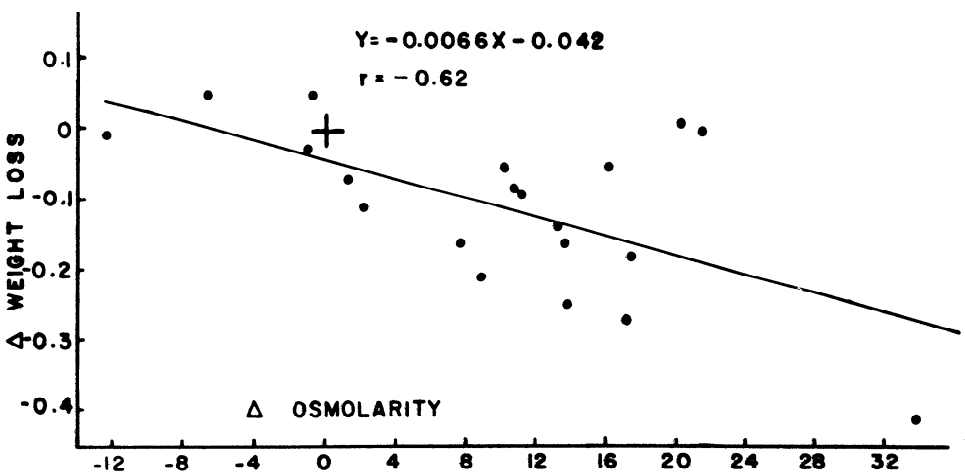

FIG. 3. Relationships of changes in serum osmolarity (mosmol) to changes in rates of evaporative weight loss (% body wt) between paired rehydration and progressive hypohydration exposures for 5 subjects at 43.3°C db, 29°C wb. (Taken from Ref. 36.)

lead to changes in the equilibrium constant $K = [Ca^{2+}] \cdot [\text{protein}]/[Ca^{2+} \text{ proteinate}]$.

What role then does such a change in ionized $[Ca^{2+}]$ play in the elevation of body temperature during progressive hypohydration? Is the rise in body temperature due to respiratory-induced alteration in the ratio $[Na^+]/[Ca^{2+}]$?

Hypohydration and respiration. The increase in body temperature that accompanies progressive hypohydration is accompanied by hyperventilation, thus accounting for an upward shift in body fluid pH. Now, if body temperature rises in a well-hydrated man, an increased sensitivity to inhaled CO_2 can be shown (10). The same maneuver during progressive hypohydration does not lead to the same result; i.e., during progressive hypohydration, there is no increase in sensitivity to inhaled CO_2 (37). Is the lack of increased sensitivity to CO_2 and the rise in body temperature during progressive hypohydration simply a reflection of a decreased neural sensitivity, and is this decrease caused by increased osmolarity, increased $[Na^+]$, or are there other factors that our simple experiments overlooked?

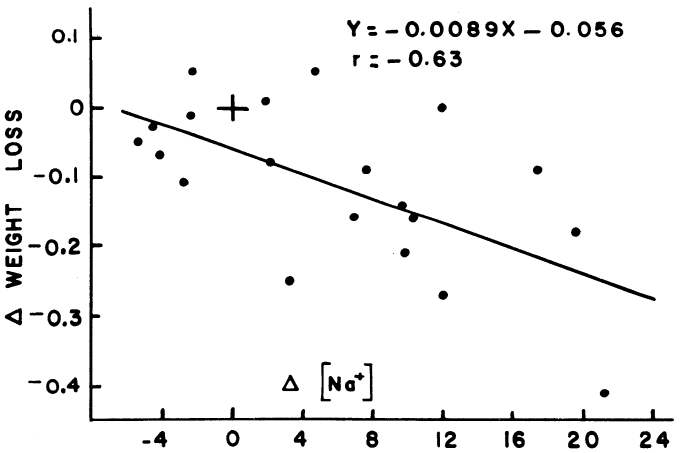

FIG. 4. Relationship of changes in serum [Na^+] (meq) to changes in rates of evaporative weight loss (% body wt) between paired rehydration and progressive hypohydration exposures for 5 subjects at 43.3°C db, 29°C wb. (Taken from Ref. 36.)

Peptides and hypohydration. With progressive hypohydration, free water clearance diminishes and soon becomes negative due to increased levels of circulating ADH. Does ADH have any other influence(s) upon the body during progressive hypohydration? Perhaps—for ADH has been identified at various places within the brain other than the supraoptic and paraventricular nuclei (7, 13) and ADH has been shown to affect the behavior of experimental animals, with a particular influence on memory (11, 21). Does ADH play a role in the regulation of body temperature? Does it possibly play a role in the reduced sensitivity of the regulatory processes?

Prolactin may also be implicated in fluid balance (25). In man, the plasma levels of prolactin have been shown to be inversely related to the state of the subject's hydration (6). It appears that an appreciation of an effect of peptides on temperature responses is beginning to develop.

Neuronal involvement: generalizations. The focus of our considerations is the neurons and their interconnections within the central nervous system (CNS). The answer to the influence of progressive dehydration on temperature regulation in resting man most likely resides in this matrix. The evidence at hand suggests to me that the decreased responsiveness of central processes is due to increased resistance to the passage of information from one neuron to the next. Both pre- and postsynaptic events are probably involved. The events that occur during progressive hypohydration suggest further that we are dealing with alterations in membrane excitability that is probably based on changes in osmolarity, [Na^+], [Ca^{2+}], [H^+], neurotransmitters, and certain fundamental changes in the membrane itself—possibly an alteration from a more fluid to a less fluid matrix.

Exercise. Turning now from our considerations of the effect of progressive hypohydration on resting man, I ask, What of exercising man? Are his responses to reductions in body water simply the result of exaggerated resting responses or does the imposition of exercise introduce new elements into our consideration of temperature regulation and dehydration?

Adolph and associates (1) indicated that increases in body temperature were related to the amount of water lost from the body. The ratio $\Delta T/\Delta$body wt appears to depend on a number of items, but particularly the metabolic rate while water deficits accumulate. In an attempt to separate the effects of hypohydration from exercise intensity, various experiments have been carried out in which subjects were first hypohydrated or put on a limited water intake, and the effect of the pretreatment on such things as $\dot{V}O_{2\,max}$, exercise duration, sweating, and so forth are assessed. Interpretation of results has the same problems in working man as in resting man; i.e., it is doubtful that a critical experiment has ever been run.

Exercise, body temperature, and hypohydration. Certain studies do frame the problem of interpretation. For example, Strydom and Holdsworth (47) hypohydrated two men on one day, and by regulating food and fluid intake maintained the weight deficit throughout a second day's exercise and heat exposure. The reported data are interesting because the subjects responded to water deficits on the second day by maintaining a constant body temperature during exercise, but the absolute level of the exercise body temperature was higher than when the subjects were hydrated. Was the increase in exercise body temperature due to sweat deficits only or was the decrease in sweating and an increase in body temperature related to some other factor? If the work of M. Nielsen (28) is considered, perhaps what is being altered is the so-called "work factor" that appears to operate in the prescriptive zone (5–30°C) of environmental temperature. Indeed, body temperatures of exercising hypohydrated men have been shown to be shifted upward even when they are working at environmental temperatures well within the prescriptive zone. Experiments such as those of Grande et al. (14, 15) and Buskirk and co-workers (8) indicate that the rise in body temperature during work can be manipulated by water intake. As noted above, similar results can be obtained by ingestion of Na^+- or Ca^{2+}-rich fluids (27), thus indicating that body temperature of working man may not only depend on osmolarity but also may depend on specific ionic species.

At this point, the differing views as to why body temperature rises during exercise should be mentioned. They appear to fall into two general categories: *1*) that the "set point" is adjusted upward and *2*) that "the increase in body temperature is a physical consequence of the internal heat load generated by exercise itself" (46). My own view is somewhat less dogmatic and starts with the generality that events occur within the body during onset of exercise that permits the body temperature to rise to a certain level. Using this generality as a base, I offer a simple hypothesis that may account for much of the increased body temperature in hypohydrated working man. I feel the hypothesis is general enough to include normally hydrated men and may also be usefully applied to comparisons of exercising males and females.

Temperature Regulation in Exercise

When a man exercises in the prescriptive zone of environmental temperatures, the level to which his body

temperature rises is related to the exercise intensity. Similar body temperatures are reached when individuals of different aerobic capacities perform at the same percentage of their maximum oxygen consumption (35).

Muscle contraction and fluid shifts. From the information at hand, the following appears to be a reasonable explanation. If a muscle contracts, there is an increase in the number of osmotically active particles within the muscle. What seems not to be appreciated by many is the fact that concentration gradients not only cause osmotically active particles to move out of muscle into the interstitial space and blood, but also that water must move in the opposite direction. Thus, blood moving through contracting muscle not only picks up osmotically active particles, but also loses water to both the muscle and the interstitial space. Clearly, the net amount of water movement depends on a number of items, chief of which is the number of contracting fibers. Blood moving through active muscle not only has had its water content reduced but it also has suffered an increase in concentration of diffusible ions as well as a relative increase in concentration of plasma proteins.

Osmolarity and ions in CNS. What happens when this blood circulates through other portions of the body including the CNS? The osmotic gradient in these areas will favor water movement from tissue to blood while the gradient for certain ions is in the opposite direction. At steady state, it is clearly possible to end up with water loss into the region of working muscle and a net loss of water from tissues such as the hypothalamus, thus increasing both osmolarity and specific ionic concentrations in noncontracting tissues.

$\%\dot{V}O_{2\,max}$, fluid, and ions. To account for uniform changes in body temperature at similar relative exercise intensities (35), the general relationship between lean body mass and blood volume should be included in our considerations. Because water loss from plasma will probably be proportional to the number of contracting fibers, a connection between level of exercise and body temperature appears quite possible.

Superimposed hypohydration. Now, if a man is hypohydrated while exercising or if he is hypohydrated before exercising (for a given exercise intensity), he will suffer a relatively greater water loss from blood than if he were hydrated. Thus, the effects of fluid shifts out of the vascular volume during dehydration would be magnified and body temperature would be at a higher equilibrium level. This reasoning, which I will now label a hypothesis, is capable of explaining a number of results resident in the literature, only one of which happens to be a rise in body temperature of exercising man during hypohydration. The change in sensitivity of neurons in the CNS brought on by moving water about the body is, in turn, coupled with sweating, which also serves to increase body fluid osmolarity and contributes to the rise in body temperature. Such a hypothesis appears reasonable and also appears to be testable. However, if Figs. 1–4 are taken as the "state of the art," then we are a long way from a reasonable assessment of the influence of hypohydration on temperature regulation in man.

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REFERENCES

1. ADOLPH, E. G., AND ASSOCIATES. *Physiology of Man in the Desert,* edited by E. F. Adolph. New York: Interscience, 1947.
2. BANNER, R. M., M. H. HARRISON, C. J. HALL, AND R. J. EDWARDS. Effect of heat acclimatization on intervascular responses to acute heat stress in man. *J. Appl. Physiol.* 41: 708–713, 1976.
3. BAZETT, H. C. The regulation of body temperatures. In: *Physioloy of Heat Regulation and the Science of Clothing,* edited by L. H. Newburgh. Philadelphia, PA: Saunders, 1949, chapt. 4.
4. BENZINGER, T. H. Heat regulation: homeostasis of central temperature in man. *Physiol. Rev.* 49: 671–759, 1969.
5. BLYTH, C. S., AND J. J. BURT. Effect of water balance on ability to perform in high ambient temperatures. *Res. Q. Am. Assoc. Health Phys. Ed. Recreat.* 32: 301–307, 1961.
6. BUCKMAN, M. T., AND G. T. PEAKE. Osmolar control of prolactin secretion in man. *Science* 181: 755–757, 1973.
7. BUIJS, R. M., D. F. SWAAB, J. DOGTERON, AND F. W. VAN LEEUWEN. Intra- and extrahypothalamic vasopressin and oxytocin pathways in the rat. *Cell Tissue Res.* 186: 423–433, 1973.
8. BUSKIRK, E. R., P. F. IAMPIETRO, AND D. E. BASS. Work performance after dehydration: effects of physical conditioning and heat acclimatization. *J. Appl. Physiol.* 12: 189–194, 1958.
9. CASTENFORS, J. Renal function during exercise. *Acta Physiol. Scand. Suppl.* 293, 1967.
10. CUNNINGHAM, D. J. C., AND J. L. H. O'RIORDAN. The effect of a rise in the temperature of the body on the respiratory response to carbon dioxide at rest. *Q. J. Exp. Physiol.* 42: 329–345, 1957.
11. DE WIED, D. Peptides and behavior. *Life Sci.* 20: 195–204, 1977.
12. EKBLOM, B., C. L. GREENLEAF, J. E. GREENLEAF, AND L. HERMANSEN. Temperature regulation during exercise dehydration in man. *Acta Physiol. Scand.* 79: 475–483, 1970.
13. GEORGE, J. M. Immunoreactive vasopressin and oxytocin: concentration in individual human hypothalamic nuclei. *Science* 200: 342–343, 1978.
14. 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.
15. GRANDE, F., H. L. TAYLOR, J. ANDERSEN, E. BUSKIRK, AND A. KEYS. Water exchange in men on a restricted water intake and a low calorie carbohydrate diet accompanied by physical work. *J. Appl. Physiol.* 12: 202–210, 1958.
16. GREENLEAF, J. E., AND F. SARGENT II. Voluntary dehydration in man. *J. Appl. Physiol.* 20: 719–724, 1965.
17. HAGEN, R. D., F. J. DIAZ, AND S. M. HORVATH. Plasma volume changes with movement to supine and standing position. *J. Appl. Physiol.: Respirat. Environ. Exercise Physiol.* 45: 414–418, 1978.
18. HARDY, J. D. Physiology of temperature regulation. *Physiol. Rev.* 41: 521–606, 1961.
19. HERTZMAN, A. B., AND I. D. FERGUSON. Failure in temperature regulation during progressive dehydration. *US Armed Forces Med. J.* 11: 542–560, 1960.
20. HOROWITZ, M., S. SAMUELOFF, AND J. H. ADLER. Acute dehydration: body water distribution in acclimated and nonacclimated *Psammomys obesus. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol.* 44: 585–588, 1978.
21. HOSTETTER, G., S. L. JUBB, AND G. P. KOZLOWSKI. Vasopressin affects behaviour of rats in a positively-rewarded discrimination task. *Life Sci.* 21: 1323–1328, 1977.
22. KIRSCH, K., K. HRYNYSCHYN, H. V. AMELN, L. ROCKER, AND H. J. WICKE. Extracellular fluid volume and central circulation after long lasting exercise and dehydration in conscious dogs. *Pfluegers Arch.* 368: 209–215, 1977.
23. LEE, D. H. K., AND A. G. MULDER. Some immediate effects of reduced cooling powers upon the water balance and related effects in the human subject. *J. Physiol. London* 84: 410–432, 1935.
24. MERCER, J. B., AND C. JESSEN. Central thermosensitivity in conscious goats: hypothalamus and spinal cord versus residual inner

body. *Pfluegers Arch.* 374: 179–186, 1978.
25. MILLER, M., AND M. VAN GEMERT. Antidiuretic action of prolactin in the rat with diabetes insipidus. *Hormone Res.* 7: 319–332, 1976.
26. MYERS, R. D., AND W. L. VEALE. The role of sodium and calcium ions in the hypothalamus in the control of body temperature of the unanesthetized cat. *J. Physiol. London* 212: 411–430, 1971.
27. NIELSEN, B. Effect of change in plasma Na^+ and Ca^{++} ion concentration on body temperature during exercise. *Acta Physiol. Scand.* 91: 123–129, 1974.
28. NIELSEN, M. Die Regulation der Korpertemperature bei Muskelarbeit. *Scand. Arch. Physiol.* 79: 193–230, 1938.
29. RADIGAN, L. R., AND S. ROBINSON. Effects of environmental heat stress and exercise on renal blood blow and filtration rate. *J. Appl. Physiol.* 2: 185–191, 1949.
30. ROBERTSON, G. L., R. L. SHELTON, AND S. ATHAR. The osmoregulation of vasopressin. *Kidney Int.* 10: 25–37, 1976.
31. ROBINSON, S. Physiological adjustments to heat. In: *Physiology of Heat Regulation and the Science of Clothing*, edited by L. H. Newbury. Philadelphia, PA: Saunders, 1949, p. 215.
32. ROBINSON, S., R. T. MALETICH, W. S. ROBINSON, B. B. RÖHRER, AND A. L. KUNZ. Output of Na Cl by sweat glands and kidneys in relation to dehydration and salt depletion. *J. Appl. Physiol.* 8: 615–620, 1956.
33. SALTIN, B. Aerobic and anaerobic work capacity after dehydration. *J. Appl. Physiol.* 19: 1114–1118, 1964.
34. SALTIN, B. Circulatory responses to submaximal and maximal exercise after thermal dehydration. *J. Appl. Physiol.* 19: 1125–1132, 1964.
35. SALTIN, B., AND L. HERMANSEN. Esophageal, rectal and muscle temperature during exercise. *J. Appl. Physiol.* 21: 1757–1762, 1966.
36. SENAY, L. C., JR. Relationship of evaporative rates to serum $[Na^+]$ $[K^+]$, and osmolarity in acute heat stress. *J. Appl. Physiol.* 25: 149–152, 1968.
37. SENAY, L. C., JR. Increased blood osmolarity and its effect on respiration of dehydrating men. *Pfluegers Arch.* 309: 167–175, 1969.
38. SENAY, L. C., JR. Body fluids and temperature responses of heat-exposed women before and after ovulation with and without rehydration. *J. Physiol. London* 232: 209–219, 1973.
39. SENAY, L. C., JR. Plasma volumes and constituents of heat exposed men before and after acclimatization. *J. Appl. Physiol.* 38: 570–575, 1975.
40. SENAY, L. C., JR. Effects of exercise in the heat on body fluid distribution. *Med. Sci. Sports* In press.
41. SENAY, L. C., JR., AND M. L. CHRISTENSEN. Cutaneous circulation during dehydration and heat stress. *J. Appl. Physiol.* 20: 278–282, 1965.
42. SENAY, L. C., JR., AND M. L. CHRISTENSEN. Cardiovascular and sweating responses to water ingestion during dehydration. *J. Appl. Physiol.* 20: 975–979, 1965.
43. SENAY, L. C., JR., AND M. L. CHRISTENSEN. Changes in blood plasma during progressive dehydration. *J. Appl. Physiol.* 20: 1136–1140, 1965.
44. SENAY, L. C., JR., AND M. L. CHRISTENSEN. Respiration of dehydrating men undergoing heat stress. *J. Appl. Physiol.* 22: 282–286, 1967.
45. SMITH, J. H., S. ROBINSON, AND M. PEARCY. Renal responses to exercise, heat and dehydration. *J. Appl. Physiol.* 4: 659–665, 1952.
46. STITT, J. T. Fever versus hyperthermia. *Federation Proc.* 38: 39–43, 1979.
47. STRYDOM, N. B., AND L. D. HOLDSWORTH. The effects of different levels of water deficit on physiological responses during heat stress. *Int. Z. Angew. Physiol. Einschl. Arbeitsphysiol.* 26: 95–102, 1968.

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