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Endocrine concomitants of sweating and sweat depression

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Summary. The effect of humid heat ($T_a = 43°$ C, $P_a =$ 32 Torr) on sweat rate, plasma renin activity and plasma levels of aldosterone and antidiuretic hormone (ADH) was studied in four male subjects before and after repeated heat exposures. Oversweating and sweat drippage followed by hidromeiosis were observed in three subjects during initial heat exposure. With repeated humid heat exposures increased sweat rates were accompanied by a more intense sweat depression (hidromeiosis) in all four subjects. In our conditions, no changes in plasma levels of aldosterone and ADH or plasma renin activity were observed with hidromeiosis. Plasma renin activity was slightly depressed by repeated exposures, whereas plasma volumes were enhanced, with no significant changes in plasma Na or K. The results suggest that neither ADH nor the components of the renin-angiotensin aldosterone system are involved in the hidromeiotic phenomenon.

Key words: Hidromeiosis – Aldosterone – Antidiuretic hormone – Renin activity

Introduction

During exposure to hot humid conditions, humans increase sweat rate in order to allow evaporative skin cooling. Since the high vapor pressure in the humid environment impairs the free evaporation of excreted sweat, some drippage occurs from the skin surface and the maintenance of thermoequilibrium is only achieved by oversweating and loss of unevaporated sweat (Givoni 1963, Candas et al. 1979). This transient increase in sweat rate is observed during the 1st h of heat exposure, followed by a decline in sweat rate called hidromeiosis (Brown and Sargent 1965). Dripping sweat depression, associated with skin wetting, is apparently due to skin swelling which takes place when the epidermis is well hydrated (Randall and Peiss 1957). Thus hidromeiosis, previously considered as a reflection of failure in the temperature regulatory system, could originate from progressive narrowing of sweat duct pores which reduces the water loss and consequently saves body water content (Hénane 1972).

Vasopressin (ADH) and aldosterone, which are involved in water and salt reabsorption respectively, are expected to be released under hot humid conditions when the conservation of water is desirable. It is likely that these hormones could be involved in the control of hidromeiosis. Before and after heat acclimatization, exposure to heat induces an increase in both urinary metabolites (Streeten et al. 1960), Fletscher et al. 1961, Beisel et al. 1968) and plasma aldosterone levels (Kosunen et al. 1976, Finberg and Berlyne 1977, Collins and Few 1979). Today, it is well-known that sodium deprivation and heat acclimation potentiate the activity of mineralocorticoids (Smiles and Robinson 1971, Follenius et al. 1979, Davies et al. 1981). In the same way, heat-related rises in plasma ADH have been reported in humans (Segar and Moore 1968) and animals (Itoh 1954, Forsling et al. 1976, El Nouty et al. 1980) with dehydration leading to a rise in plasma ADH and a concomitant reduction in the volume of urinary output (Itoh 1954, Segar and Moore 1968).

Despite the numerous studies of the relationships between body water content and hormonal secretion, it appears that the variation of plasma aldosterone and of ADH have not been investigated in resting nude men exposed to hot humid surroundings capable of inducing hidromeiosis.

The aim of this study was to make simultaneous measurements of plasma levels of aldosterone and ADH and of renin activity, and to determine their

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relationships to the salt and water economy occurring during hidromeiosis.

Material and methods

Four fit men (age: 20–25 years, weight: 65–82 kg, height: 1.78–1.91 m) volunteered to participate in the experiments. Prior to the experimental period, they underwent medical examinations. All subjects were informed of potential risk and gave their written consent. During the experiments, only briefs were worn by the subjects, who were lying on a hammock consisting of seven rubber straps (5 cm-width) suspended from a balance.

For blood sampling, a small plastic catheter was inserted into an antecubital vein and blood was collected during consecutive 10-min sessions at a rate of 0.5 ml · min^{-1}. After immediate blood centrifugation, plasma samples were stored at −20° C.

Each subject was exposed from 15:00 to 17:45 h during 5 consecutive days (day 1 to day 5) to a hot humid environment: air and wall temperatures set at 43° C, air velocity at 0.3 m · s^{-1} and water vapor pressure at 32 Torr.

During the 2 experimental days with blood sampling (day 1 and day 5) the subjects, before being exposed to humid heat, were resting in the climatic chamber in a thermoneutral environment (28° C of air and wall temperature, 0.3 m · s^{-1} of air velocity and 10.5 Torr of ambient vapor pressure). Therefore, blood collection lasted from 13:00 to 19:30 h.

Esophageal temperature (T_{es}), 10 local skin temperatures and heart rate were continuously recorded. The body weight loss was recorded by the balance suspending the hammock, and the weight loss was assumed to be equal to the total body sweat rate. The amount of dripping sweat was collected in an oil pan attached to a second balance placed under the subject. Each balance had 2 g sensitivity and 1 g precision. Body sweat rate (m_{sw}) and dripping sweat rate (m_{dr}) were calculated at 1-min intervals with linear regression analysis performed over 19 consecutive min (the slope of each regression gave the instantaneous sweat rate at the 10th min of the regression). Evaporative sweat rate was then calculated from ($m_{sw} - m_{dr}$).

Plasma aldosterone (Gianotti et al. 1974) and ADH (Lutz-Bucher et al. 1977) levels were determined by radioimmunoassays. Plasma renin activity was measured by radioimmunoassay of angiotensin 1 (Haber et al. 1969). Na and K concentrations in plasma were measured by flame photometry. Changes in plasma volume were calculated from changes in hemoglobin and hematocrit (Dill and Costill 1974).

Results

Sweat rates

Typical examples of sweat rate pattern in one subject are given in Fig. 1. This figure shows that after 1 h of heat exposure, the dripping sweat rate decreased while the evaporative rate remained constant. For this particular subject the sweat depression was observed before (day 1) and after repeated humid heat exposures (day 5). The repeated exposures induced an increase of sweating, especially of drippage intensity and a more intense hidromeiosis. These results, already described from our previous studies (Candas et al. 1980) confirm that hidromeiosis corresponds to an adaptative mechanism of water economy. The subject maintains constant skin evaporation and constant body cooling, indicating that the decrease in sweat rate occurs only in the areas where sweat excretion is greater than skin evaporation. This hidromeiosis phenomenon was observed in three of the four subjects before (B) and after (A) repeated exposures (Table 1). One subject (S_1) showed less oversweating in condition B and did not decrease his sweat rate in this condition, but after the exposures A, hidromeiosis occurred in all subjects. The individual range of body dehydration at the onset of hidromeiosis ranged between 0.13 and 0.55% before repeated heat exposure, and between 0.17 and 0.45% after heat treatment.

Hormonal responses

During the 1st day of heat exposure, plasma aldosterone and ADH levels in the four subjects did not

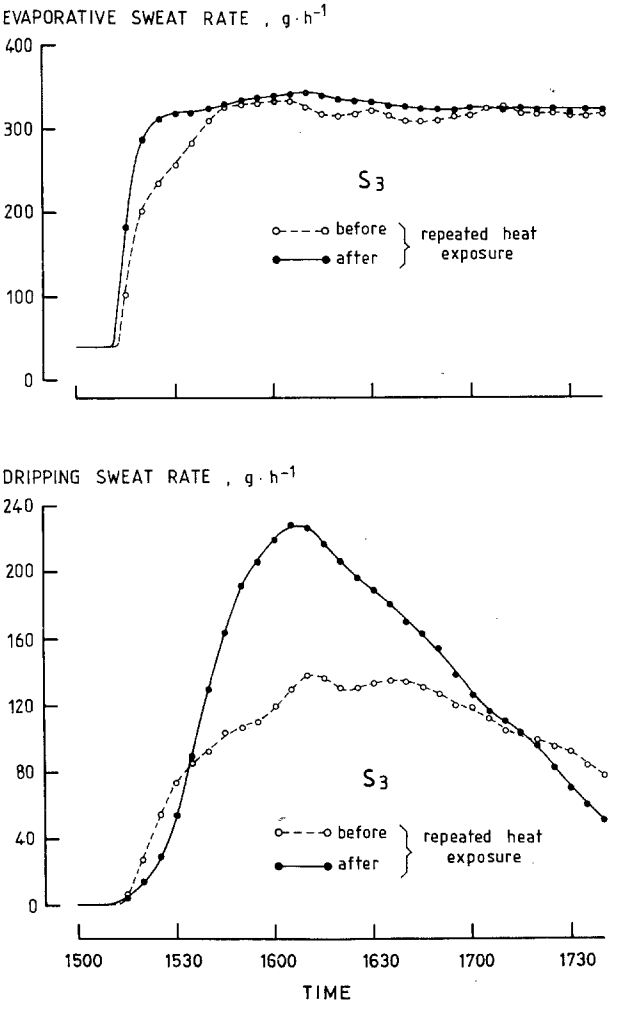

Fig. 1. Evaporative and dripping sweat rates as a function of time during the humid heat exposures

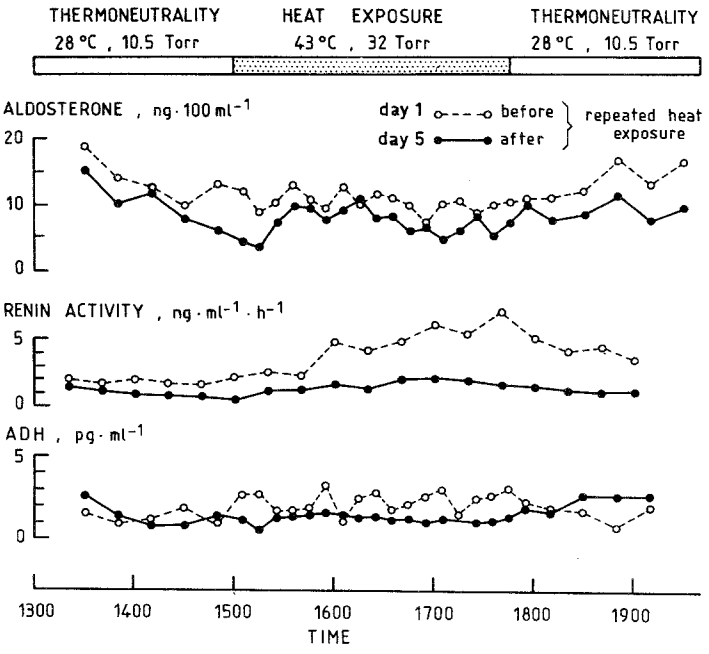

Fig. 2. Plasma aldosterone, renin activity and ADH levels in subject S_3, before, during, and after humid heat exposure

Table 1. Sweat rates during heat exposure. B: before any heat exposure (day 1) and A: after repeated heat exposure (day 5)

Subjects		Maximal sweat rate ($g \cdot h^{-1}$)	Time of maximal sweat rate (min)	Sweat rate after 2.5 h of heat exposure ($g \cdot h^{-1}$)
S_1	B	398	–	398
	A	428	90	377
S_2	B	554	100	521
	A	626	80	585
S_3	B	484	70	418
	A	573	65	403
S_4	B	432	55	376
	A	492	65	410

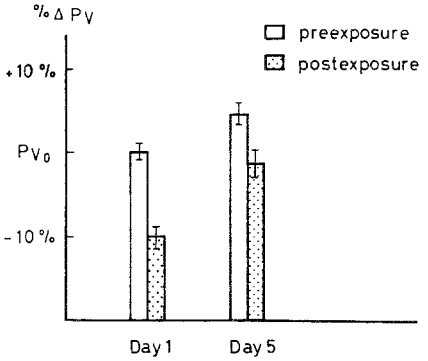

Fig. 3. Percentage of plasma volume variation. The results were calculated with respect to plasma volume which was observed during the resting thermocomfort condition before the first humid heat exposure (on day 1). Preexposure and postexposure mean, respectively, before and after humid heat exposure on the same day

vary in response to heat, whereas renin activity showed a slight increasing trend throughout the whole exposure period (mean values ± SE increasing from 1.6 ± 0.4 to 5.7 ± 1.0 $ng \cdot ml^{-1} \cdot h^{-1}$).

After repeated exposures, subjects showed non-modified patterns (day 5), but the mean plasma renin activity was slightly depressed as compared with day 1, whereas aldosterone and ADH levels were not significantly altered by repeated exposures. The mean plasma renin activity remained at constant levels during the experiment. These results are illustrated by the simultaneous variations in these hormones observed in subject S_3 (Fig. 2).

Thus the curves obtained on the four subjects did not give evidence of any hormonal change concomitant with the initiation of sweat depression (hidromeiosis).

Plasma K and Na

Na and K plasma concentrations were not affected by the repeated exposures. Moreover, all subjects showed unchanged levels throughout the heat exposure as well as before and after repeated exposures.

Plasma volume

As shown in Fig. 3, plasma volume (PV) was significantly reduced by the exposure period (two-tailed t test, $P < 0.01$). The mean values for the

four subjects were reduced by 10.0% during the heat exposure on day 1 and by 5.5% after the 5-day period. Resting PV before heat exposure was elevated 4.3% above the level observed on day 1. Post exposure PV showed large differences between these 2 days, because of the less important PV decrease occurring on day 5.

Discussion

Hidromeiosis, previously considered as sweat gland fatigue, is now known to be a regulatory mechanism of water economy which does not alter the cooling efficiency of sweating. Some observations from our previous work (Candas et al. 1983) led us to think that hidromeiosis, which originates from local skin hydration, would also reflect some mechanism induced by the salt or water-retaining activity of aldosterone or ADH. Nevertheless, this suggestion remained hypothetical, as long as the plasma levels of these hormones were unknown for human subjects exposed to conditions of whole body thermal oversweating.

The present results, however, failed to demonstrate that either aldosterone or ADH or renin activity varied concomitantly with hidromeiosis.

The action of injected aldosterone on sweat gland function has been described, but the long latency and the weak effect of the exogenous hormone action cannot support the hypothesis of a role of endogenous aldosterone in stimulating short-term sweating reduction (Collins 1966). In our study, heat-exposure did not produce changes in plasma aldosterone level, contrary to what has been described previously for sitting subjects in other climates (Morimoto 1978, Follenius et al. 1979). Nevertheless, an unchanged plasma aldosterone level cannot entirely exclude the possibility that this hormone may intervene in these hot-humid conditions. In fact, slightly enhanced secretion could be masked by an increased metabolic clearance rate in supine subjects exposed to heat.

Plasma renin activity showed a small increasing trend during the exposure period in experiments of day 1, whereas a nearly constant level was observed in the repeatedly exposed subjects (day 5). Furthermore, the basal level was slightly reduced on day 5 compared to day 1. In this study, subjects were on an unrestricted salt intake, and therefore it is unlikely that the reduction in renin basal level in response to heat after repeated exposures resulted from changes in Na or K balance. Indeed, if sodium depletion occurred, it would induce enhanced levels of aldosterone and renin activity (Smiles and Robinson 1971, Follenius et al. 1979, Davies et al. 1981). These results agree with those of Finberg and Berlyne (1977), who related this effect partly to the increased plasma volume, resulting in a reduced renin secretion.

Our results also agree with the findings of Senay et al. (1976), clearly demonstrating that repeated heat exposures lead to an increase in plasma volume. During the exposure period PV decreased in all subjects, because sweat losses were not immediately replaced by water ingestion. This decrease is important during the 1st day of exposure, unlike what happened after the exposure period, to the enhanced PV observed with the acclimation process. This observation is surprising, because total sweat losses were increased by repeated exposures despite a more intense hidromeiosis, as shown by Fig. 1. This effect could be attributed to an increased secretion of ADH in response to acclimation, as already reported by Morimoto 1978. However our results did not give any evidence of an increase in plasma ADH on day 5 over the preexposure level.

The primary controlling factor in the release of ADH is the osmolarity of the plasma. Several reports have suggested that an interaction occurs between osmolar and blood volume stimuli, so that alterations in volume modified the threshold and slope of the relationship between plasma osmolarity and plasma ADH (Dunn et al. 1973, Robertson and Athar 1976, Hammer et al. 1979, Weitzman et al. 1980). In our study, plasma Na and K remained constant, and hypovolemia accompanying heat exposure and resulting from dehydration seems not to be a sufficient stimulus to produce measurable changes in plasma ADH levels.

Thus, these results suggest that neither ADH, nor the components of the renin angiotensin-aldosterone system are involved in the phenomenon of hidromeiosis occurring in hot-humid conditions. While long-term adaptative changes in hormonal levels play an important physiological role in maintaining volume homeostasis and composition of body fluids in counteracting the effect of important sweat losses, it clearly appears that these hormones do not play a stimulating role in the hidromeiosis phenomenon.

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