Cutaneous Vascular Responses of Dehydrated Men Following Ingestion of Saline or Water[1] (36748)

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In recent years the suggestion that dilatation of the cutaneous vascular bed accompanying exposure of humans to elevated ambient temperature is determined by sweat gland activity (1) has gained rather wide acceptance (2–4). Acceptance of this "bradykinin theory" has occurred in spite of evidence that dilatation of cutaneous blood vessels can proceed independently of sweat gland activity (5, 6). One such observation was reported by Senay and Christensen and concerned 1 of their dehydrated subjects (7). When this subject ingested either tap water or 300 milliosmolar saline, an increase in sweating occurred. However, cutaneous vascular dilatation only occurred after water ingestion. With only 1 observation on 1 subject, the possibility of an abberrant result was quite real. The results from three additional subjects are presented below and these results support the concept that dilatation of the cutaneous vascular bed is independent of the activity of eccrine sweat glands.

Methods. Three male medical students whose informed consent had been obtained were instructed to forego breakfast, but not water, before reporting to the laboratory between 4 and 6 AM. Following an initial oral temperature determination, the subjects, clad only in light cotton shorts, entered the heat chamber which was maintained at a temperature of 43.3° DB and 29° WB throughout the experiments. During the first 3 hr of heat exposure observations were restricted to oral temperatures and body weights. Three 30 min observation periods began at 3, 6 and 9 hr after subjects had entered the heat chamber, while a final 40 min observation period was started 11.75 hr after entry. The subjects were not given any fluid to drink until the final observation period whereupon either water or saline was delivered to the subject (see below).

During observation periods, the subjects were in a semireclining position on a stainless steel screen-back chair. Weighted mean skin temperatures (8) were obtained by attaching copper–constantan thermocouples to the dorsal surface of the foot, medial calf, medial thigh, lateral trunk, chest (5 cm below the nipple), palm, forearm, and cheek. The thermocouples consisted of junctions of uncovered crossed wires held in firm contact with the skin by an open plastic ring in which the wires were mounted. The device permitted free convection, radiation and evaporation from skin surfaces to which the thermocouples were applied. The time necessary for a complete skin temperature monitoring cycle was 35 sec. Photoelectric devices were used to record the cutaneous volume opacity pulses (9, 10) and were placed on the anteromedial calf, medial thigh, and upper posterior forearm. Heart rates were determined from opacity pulse records. With a constant experimental ambient temperature, rapid changes of skin temperature in these studies could be taken to indicate changes in evaporation rates if cutaneous blood flow does not change. Previous findings (7) supported this view.

During the final observation period, the subjects were given tepid tap water (36–38°) or tepid 300 milliosmolar saline. The type of fluid ingested was reversed in a second exposure. Amounts given depended on the desires of the subject and it should be noted that the fluid was delivered to the subject through a plastic drinking tube from a beaker held at a level such that a siphoning action

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TABLE I. Initial Body Weights Before Heat Exposure, Total Body Weight Loss and Rate of Change of Oral Temperatures (T_O) for Subjects 7, 8, and 9 During the Present Study.[a]

Subject	Preexposure body wt (kg)	Pre-ingestion wt loss (% body wt)	Pre-ingestion ΔT_O per hr
7	77.7	5.44	.18
7_S	78.2	4.96	.12
8	85.3	4.42	.10
8_S	83.2	4.76	.09
9	79.9	4.46	.13
9_S	80.3	4.22	.12
5	65.5	4.33	.05
5_S	64.7	4.65	.04

[a] Similar measurements for subject 5 were taken during a previous study (7). s denotes saline ingestion.

occurred upon initiation of drinking. Therefore, little muscular effort was required on the part of the subject to obtain the necessary fluid. Cutaneous volume opacity pulses and skin temperature were continuously recorded throughout this final observation period. Each subject was exposed to heat at weekly intervals. The subjects knew from the beginning of each experiment which fluid they were to drink.

Results. Observations on rates of weight loss and changes in oral temperature prior to the final observation period are listed in Table I. The observational procedures at 3, 6 and 9 hr after entry into the heat chamber were intended to accustom the subject to being connected to the various measuring devices, to minimize potentially emotional responses and thereby to establish whether the preingestion control values obtained during the final observation period were consistent with the earlier results.

Mean skin temperatures. The initial course and magnitude of the reductions in mean skin temperature were similar following ingestion of either water or saline (Fig. 1). Differences in responses to the type of fluid ingested became evident, however, within 5 min of the start of fluid ingestion. Whereas subject 8 (Fig. 1) had a greater decrease in mean skin temperature following water ingestion than that following ingestion of saline, subject 7 (Fig. 1) showed the opposite of this during the initial 4 min following fluid ingestion. The mean skin temperature results for subject 9 (Fig. 1) are somewhat unusual, though similar behavior has been seen before (7). Following an increase in evaporative heat loss from the skin surface (and hence, increased sweat secretion) there was a rapid rise in skin temperature commencing about the time that water intake was initially halted. A secondary decrease in mean skin temperature of subject 9 started just prior to injestion of a final 90 ml of water but it was not until some 18 min after initial water ingestion that a difference between saline and water ingestion became readily apparent for this subject.

In addition to the greater duration of the sweating response following water ingestion, the temperature data of subjects 7 and 8 (Fig. 1) indicated that increases in skin temperature that would normally follow cutaneous dilatation (Fig. 2) were more than offset by early increases in evaporation. Hence, evaporative rates in subjects 7 and 8 were greater following water ingestion than after saline ingestion. However, this was not true for subjects 9 and 5.

In all cases the final results were similar; at the end of the observation period, mean skin temperatures following water ingestion were consistently less than preingestion levels while after saline ingestion mean skin temperature were of the same magnitude or greater than preingestion levels. The results for subjects 7, 8 and 9 were similar to those obtained earlier (Subject 5, Fig. 1). Mean skin temperature data have not been previously presented for this subject (Fig. 1).

Cutaneous opacity pulses. Though the courses of mean skin temperatures were qualitatively similar for at least 15 min following ingestion of water and saline, this similarity did not extend to the cutaneous vascular responses. Following water ingestion a progressive vasodilatation was noted for all monitored cutaneous areas. For subject 7 (Fig. 2) the forearm and calf cutaneous opacity pulses increased 100% over preingestion values while thigh cutaneous perfusion increased

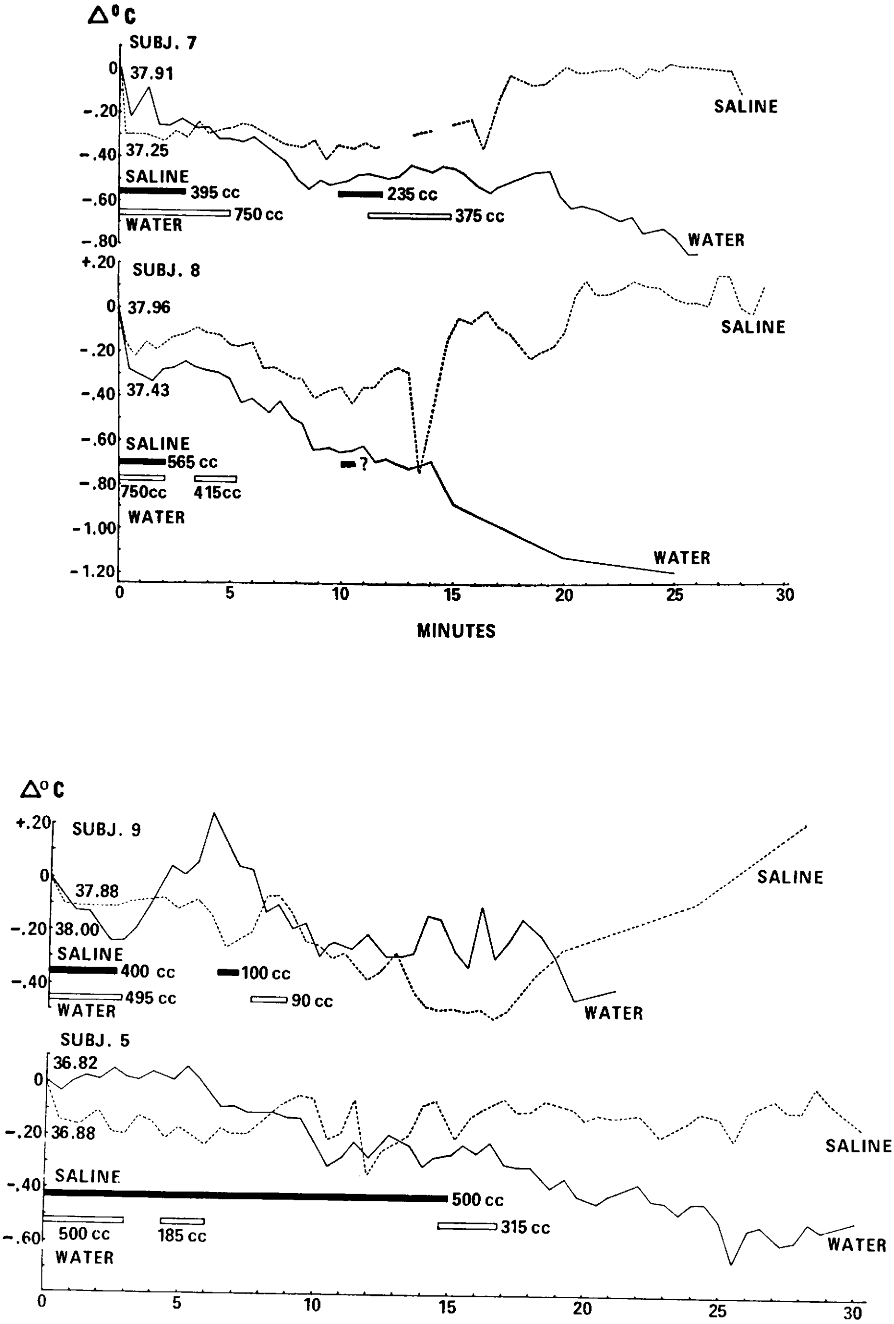

FIG. 1. Weighted average skin temperatures during and after ingestion of saline or water for subjects (7–9) of the present experiments and for 1 subject from a previous study (subject 5). Values are expressed as changes from averages obtained during a 5–7 min period preceding fluid ingestion. These absolute numbers are shown to the left of the figures. Amount of fluid and time of intake is indicated by the horizontal bars. For subject 8, the time and amount of the second draught of saline was not recorded.

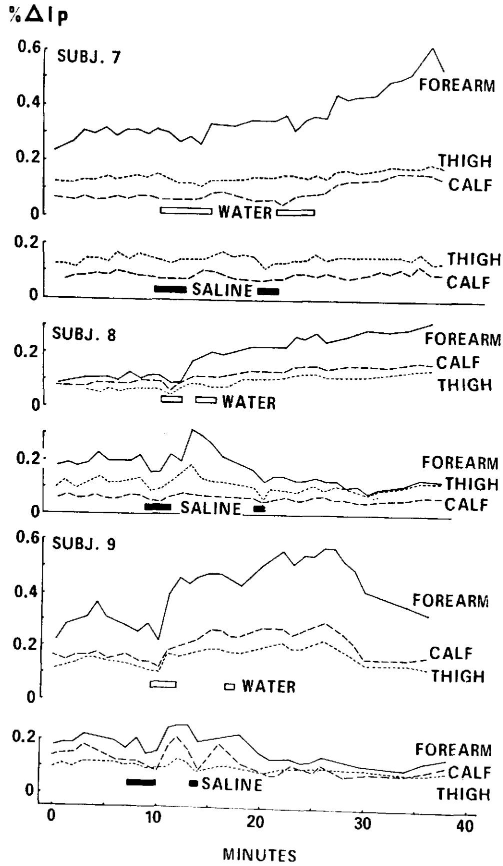

FIG. 2. Cutaneous volume opacity pulse amplitudes expressed as percentage change in photoelectric current (I_p) for subjects 7, 8 and 9 for the entire observation period. Fluid ingestion as in Fig. 1.

some 30%. Saline ingestion by this subject was not followed by an increase in perfusion of the cutaneous vascular bed. The results for subjects 8 and 9 were similar to those for subject 7. There did appear to be *small* increments in cutaneous opacity pulses recorded from the forearm and thigh of subjects 8 and 9 (Fig. 2) immediately after saline ingestion. However, the general response in both subjects was one of a *reduction* in cutaneous opacity pulses after saline ingestion. Simultaneous comparison of mean skin temperature and cutaneous opacity pulses indicated that the reductions in cutaneous blood flow occurred in spite of a continuation of the sweating response brought on by the initial ingestion of saline. This is particularly well marked in subject 9 (Figs. 1 and 2).

Heart rates. A typical heart rate response to fluid ingestion is presented in Fig. 3. The magnitude of the increase upon ingestion of water greatly exceeded that observed after saline ingestion, but eventually heart rates following water ingestion became less than those following saline ingestion. Apparently, the increase in heart rates occurred as rapidly as did the sweating response to fluid ingestion. Cutaneous opacity pulse recordings indicated that a transient vasoconstriction accompanied both the increased sweating and heart rates. At the end of the observation period both systolic and diastolic blood pressure values were less than values obtained before water ingestion. Blood pressure values were similar prior to and at the end of the observation periods wherein saline was ingested.

Discussion. Increased activity of eccrine sweat glands, in these experiments, cannot be assigned a causative role in cutaneous vasodilatation (1). Both ingestion of saline and water triggered a generalized increase in sweating. Eventually, the increased sweating following water ingestion was accompanied by a progressive cutaneous vasodilatation. Even here, following water ingestion, increased eccrine sweat gland activity and decreases in cutaneous vasomotor tone have been temporaly separated. Most telling, however, was the almost complete lack of change in cutaneous vasomotor tone following saline ingestion. Indeed in two subjects (8 and 9)

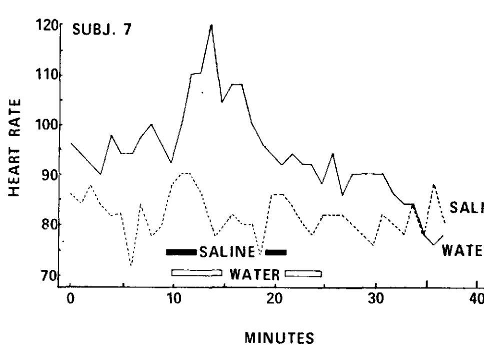

FIG. 3. Typical heart rate response to ingestion of water and of saline.

there was an eventual increase in vasomotor tone.

The ingestion of 300 milliosmolar saline would little alter the osmotic pressure of body fluids and therefore the dilatation of the cutaneous vascular bed following water ingestion can be ascribed to a lowering of the osmotic pressure of body fluids. Two subjects (7 and 8) drank approximately 1.1 liter of water but the same results were obtained from subject 9 after he drank approximately 1/2 this amount. Based on approximations of total body water, a lowering of the osmotic pressure of body fluids by less than 1% probably triggered the observed cutaneous vasodilatation. Considering how rapidly cutaneous vasodilatation begins upon drinking of water (subjects 8 and 9, Fig. 2) the effective amount of water absorbed would be a good deal less than the total eventually ingested. The receptors for this response (*i.e.*, initiation and continuation of cutaneous vasodilatation) do not appear to correspond to those implicated in the general sweating response following fluid ingestion (11). Whether these osmoreceptors are in the central nervous system or elsewhere remains to be determined.

Summary. In 3 dehydrated male subjects, ingestion of water and saline were equally effective at triggering immediate increases in sweating. Sustained increases in cutaneous volume opacity pulse amplitudes were observed only after water ingestion. Since both saline and water ingestion induced increased sweating while cutaneous vasodilatation only occurred after water intake, support was indicated for the concept that dilatation of the cutaneous vascular bed is independent of the activity of eccrine sweat glands.

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