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# THE EFFECTS OF WATER AND SALT INTAKE UPON THE PERFORMANCE OF MEN WORKING IN HOT AND HUMID ENVIRONMENTS

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It is commonly supposed that drinking increases the sweat loss at the same time as improving the overall performance of men working in the heat. There is considerable experimental evidence supporting the latter supposition; thus Gosselin (1947) recorded that the rectal temperature rose to higher levels in dehydrated men; Bean & Eichna (1943) found that the work capacity of men was improved when they replaced their sweat losses; Gregory & Lee (1936) found thermal equilibrium was more easily maintained by men when they were taking water; Johnson and co-workers (Johnson, Belding, Consolazio & Pitts, 1942; Johnson, Pitts & Consolazio, 1944) found that in long exposures water delayed the falling off in sweat rate and gave lower final rectal temperatures; they were unable (Pitts, Johnson & Consolazio, 1944) to get men to a steady state in the heat unless they drank and in non-drinkers they found the rectal temperatures and heart rates rose to 'uncomfortably high levels'.

The effect on sweat rate is less well substantiated; Hunt (1912) recorded an increased sweat loss when his subjects took water, but he noted that dehydration itself did not stop sweating. In so far as water drinking staves off sweat gland fatigue, Johnson and his team would support Hunt's findings. Lee & Mulder (1935*a*, *b*) showed that drinking increased the sweat loss; while Pitts *et al.* reported a decreased sweat rate in men when dehydrated. On the other hand, Adolph (1947*a*) is quite definite that sweat rate is independent of water intake, and the Fort Knox team (Eichna, Bean, Ashe & Nelson, 1945) found that replacing the sweat losses did not affect the sweat rate. In some tests saline has been given in place of water; Lee, Murray Simmonds & Atherton (1941) found a reduction in sweat rate with saline, more marked in hot dry conditions; Johnson *et al.* (1944) reported the same, although the same team (Pitts *et al.* 1944) also reported the opposite in another paper. Eichna *et al.*

found no effect at all, and Gerking & Robinson (1946) in contrast to Johnson did not find that drinking of saline deferred the onset of sweat gland 'fatigue'.

In tests on heat tolerance there has been no consistent practice between different workers. Sometimes saline has been given (Bean & Eichna, 1943; Robinson & Gerking, 1947) and sometimes water (Pitts *et al.* 1944; Robinson, Gerking, Turrell & Kincaid, 1950; Robinson, Kincaid & Rhamy, 1950). Sometimes there has been full replacement (Weiner & van Heyningen, 1952*a*, *b*), and sometimes in short exposures no drinking at all (Ladell, 1951); tolerance limits have been defined for men drinking '*ad lib.*' (McArdle, Dunham, Holling, Ladell, Scott, Thompson & Weiner, 1947), although the probability of 'voluntary dehydration' (Rothstein, Adolph & Wills, 1947) occurring has been known for many years (Vernon & Warner, 1932).

The McArdle team recognized that drinking might have a considerable effect on heat tolerance; but they considered that free rather than controlled drinking was more realistic. At the same time one of the group, the present author,

TABLE 1. Sweat production and rectal temperature (R.T.) rise in two fully acclimatized subjects during 130 min exposure with mean metabolic rate of 100 kcal/m²hr in two different climates, once drinking freely and once with no water.

| | Climate | | Duration | Drinking | | Not drinking | |
|---|---|---|---|---|---|---|---|
| Subject | Wet bulb (° F) | Dry bulb (° F) | of experiment (min) | Sweat loss ml. | R.T. rise (° F) | Sweat loss (ml.) | R.T. rise (° F) |
| 1 | 95 | 90 | 130 | 3160 | 3·3 | 2727 | 3·4 |
| | 100 | 95 | 85 | 1915 | 3·4 | 2178 | 3·2 |
| 2 | 95 | 90 | 130 | 3130 | 3·5 | 3379 | 2·4 |
| | 100 | 95 | 85 | 2145 | 3·2 | 1984 | 3·1 |

carried out a number of trials to determine, for the conditions of the experiments in progress, the effect of drinking on sweat rate and performance. The results of preliminary experiments were equivocal, as in the example shown in Table 1, and confirmed what was apparent from the literature, that the effect of drinking was variable and not easy to demonstrate in simple experiments. More elaborate experiments were therefore carried out, in which the effect of saline administration as well as of pure water was investigated.

## METHODS

In series 1 and 2, four trained but not hyperacclimatized subjects were exposed for 4 hr, in series 1 to dry bulb 93° F, wet bulb 91° F, and in series 2 to dry bulb 95° F, wet bulb 93° F, air movement in both cases 100 ft./min. In both series the routine consisted of stepping on and off a stool 12 in. high 12 times a minute for 30 min at the beginning and end of the 4 hr period; the subjects began this work 10 min after entering the room, and had 170 min rest between the two bouts.

In series 1 the men wore full naval anti-flash gear and on the first day drank 0·2% saline and on the second day the same quantities of pure water. In series 2 the men wore shorts only; the tests ran for 5 days, during which each man drank on different days: water, 0·1% saline, 0·2% saline and 0·5% saline as follows: Subject SHA drank water, 0·1%, 0·5%, water and 0·2% on days 1, 2, 3, 4 and 5 respectively; subject MAC drank 0·1%, water, water, 0·5% and 0·2% on the

5 successive days; subject PAR drank 0·1%, water, 0·1%, 0·2%, and 0·5% in that order; and subject HUF drank water, 0·1%, 0·2%, 0·1% and 0·5%. (This order and distribution was determined by administrative considerations.) Each man maintained the same drinking pattern, that self-imposed on the first day of the series, throughout the five tests.

In series 3 and 4 hyperacclimatized subjects were used, two in series 3 and these two plus two more in series 4. The subjects were exposed for a shorter time, 140 min in series 3, 110–160 min in series 4, during which time they carried out a routine of alternating rest and work in cycles of either 20 (series 3) or 25 min, which had mean metabolic costs of 100 or 110 kcal/m²hr respectively, in a dry bulb temperature of 100° F and wet bulb temperature of 93·5° F with the air movement 100 ft./min. The series 3 tests lasted 4 days and compared the effects of drinking water, 0·2% saline or 0·5% saline with those of not drinking. It was arranged in two blocks of 2 days, in each of which each treatment was given once only. In series 4 there were four different regimes: (I) drinking water equal to the sweat loss (subjects approaching water balance only); (II) not drinking water but taking salt as 10% saline to equal the salt loss as estimated from the salt concentrations of the samples in the arm bags (subjects approaching salt balance only); (III) drinking water and salt to equal the estimated losses (subjects approaching both water and salt balance); (IV) taking neither water nor salt (subjects in neither water nor salt balance). Subject ELL did drink to a small extent when on regime (IV); but he always incurred a water debt greater than 1·3 l. Each treatment had a variant in which a single diuretic dose of 750 ml. of water was given after the first 15 min of exposure; also in a few experiments there was a further variant in which the diuretic dose consisted of 500 ml. of 3·5% saline, representing sea water. Only two subjects were tested on any given day, but the other two subjects also came into the hot room and assisted with the observations so that each man was exposed to the heat 5 days a week. The only statistical control possible was to ensure that on no day were both subjects on the same regime.

In all four series observations of body weight, pulse rate and rectal temperature were made before and after each bout of work and on first entering and on leaving the hot room. In series 4 the men wore arm bags for the collection of sweat for analysis; urine and vomitus (if any) was also collected and analysed for chloride by the Volhard-Harvey method. No measurements were made of skin temperature. Drinks were given at about 70° F. In the replacement routines 'floats' of water, salt or both were given to the subject before he entered the climatic chamber to cover the estimated loss in the first 15 min.

## RESULTS

### *Series* 1

It was not possible to distinguish between the effect of 'days' and the effect of a saline drink. There was, however, a statistical difference between the mean sweat losses on the second day, when water was drunk (5356 ml.), and that on the first day, when 0·2% saline was drunk (3890 ml.). From an analysis of variance, the standard error for each day from random sampling was ±337 and between days ±477; from this $t = 3{\cdot}08$ ($P < 0{\cdot}05$). These higher sweat rates on the water day were associated with higher rectal temperatures, the mean final temperatures being 102·1 and 101·8° F for days 2 and 1 respectively. From the analysis of variance $t = 3{\cdot}36$ ($P < 0{\cdot}01$). The effect on heart rates was the same: the mean rise in rate during the first bout of work, before the nature of the drink could have any effect, was 14 beats/min for the water day, and 18 on the saline day; but this difference was reversed for the second bout: the means rises were 18·25 and 12·75 beats/min for water and saline respectively. The mean maximum heart rate was also greater for water than for saline: 171 compared with 163 beats/min.

*Series* 2

Subjectively the subjects did not appear to have any preference for saline, of whatever concentration, over water, or vice versa. Complaints of 'exhaustion' were made by subjects on water, on 0·1% saline and on 0·5% saline. Rectal temperature change and heart rate were not affected by the treatment; the observed differences in final rectal temperatures could all be traced to variations in the response of the subjects during the first 30 min of exercise, before any differences in the drinking regime could have become effective.

TABLE 2. Series 2 tests: alterations in rectal temperature during work, recovery and subsequent work, maximum observed pulse rates, and sweat losses in last 2½ hr, for each exposure. An assessment of the subject's condition, part clinical part subjective, is also given; (1) indicates normal tiredness, (5) complete exhaustion and intervening numbers intervening states.

| | | | Changes in rectal temp. | | | | | |
|---|---|---|---|---|---|---|---|---|
| Regime | Subject | Day | First work rise (° F) | Recovery (° F) | Second work rise (° F) | Max. pulse rate (beats/min) | 2½ hr sweat (ml./man) | Subject condition |
| Water | SHA | 1 | 1·9 | 1·77* | | 148 | 3013* | 4 |
| | SHA | 4 | 2·1 | 1·6 | 1·2 | 144 | 3565 | 1½ |
| | MAC | 2 | 1·9 | 1·4 | 1·8 | 164 | 1820 | 1½ |
| | MAC | 3 | 1·7 | 1·2 | 1·4 | 140 | 1765 | 1 |
| | PAR | 2 | 2·0 | 1·5 | 1·6 | 180 | 2360 | 1 |
| | HUF | 1 | 2·3 | 1·6 | 1·9 | 168 | 1670 | 1 |
| 0·1% | SHA | 2 | 2·3 | 1·6 | 1·7 | 160 | 2275 | 1 |
| | MAC | 1 | 2·0 | 1·4 | 1·5 | 168 | 2370 | 2 |
| | PAR | 1 | 2·3 | 2·0 | 2·0 | 180 | 2940 | 1 |
| | PAR | 3 | 1·9 | 1·5 | 1·8 | 164 | 2955 | 1 |
| | HUF | 2 | 2·8 | 2·1 | 2·0 | 168 | 1746 | 1 |
| | HUF | 4 | 2·7 | 1·1 | 1·6 | 184 | 2765 | 4 |
| 0·2% | SHA | 5 | 1·8 | 1·3 | 1·9 | 124 | 2652 | 1 |
| | MAC | 5 | 1·8 | 1·0 | 1·6 | 136 | 1470 | 1½ |
| | PAR | 4 | 2·6 | 1·4 | 1·9 | 172 | 2800 | 1 |
| | HUF | 3 | 2·9 | 2·2 | 2·0 | 164 | 1900 | 1½ |
| 0·5% | SHA | 3 | 2·0 | 2·0 | 1·9 | 144 | 1441 | 1 |
| | MAC | 4 | 2·1 | 1·4 | 1·5 | 132 | 1630 | 1½ |
| | PAR | 5 | 1·8 | 1·1 | 1·7 | 184 | 1945 | 5 |
| | HUF | 5 | 1·1 | 1·7 | 2·2 | 176 | 1320 | 1 |

* These values calculated by the principle of minimum error.

In the second 30 min work period the mean rises in rectal temperature differed by less than 0·1° F with the four regimes; the individual variations are apparent in Table 2. There were no significant differences between the mean maximum temperatures reached in the various regimes, or between the mean maximum heart rates. The sweat losses in the last 2½ hr of each exposure when the drinking regime could be expected to show its effect, did vary with the regime. The individual values are shown in Table 2; they include an extrapolated value for one subject who failed to complete the full 4 hr calculated according to the principle of 'minimum error' (Snedecor, 1948). The mean 2½ hr sweat productions were 2365·5, 2508·5, 2205·5 and 1584·0 ml. when

drinking water, 0·1 % saline, 0·2 % saline and 0·5 % saline respectively. From the analysis of variance the standard error for the first two means was 131·1 ml., and for the third and fourth groups 160·5 ml. Comparison of the variance between groups with the error variance gave $z = 0{\cdot}9893$, which is midway between the 5 and 1 % points for $P$ with $n_1 = 3$ and $n_2 = 8$ (one degree of freedom lost due to the extrapolated value). There was therefore a statistically significant difference between the four groups; but this difference was entirely due to the group drinking 0·5 % saline. Thus, in this series the sweat production was the same whether the subjects drank water, 0·1 % saline or 0·2 % saline, but markedly diminished when they drank 0·5 % saline.

*Series* 3

The results of this series of tests are shown in Table 3; differences in sweat rate, rectal temperature change and heart rate were found with the different regimes. The mean sweat losses for the two men in the last 80 min of exposure were: when drinking water, 1571 ml.; when not drinking, 1544 ml.; when drinking 0·2 % saline, 1626 ml.; and when drinking 0·5 % saline, 1493·5 ml. In the analysis of variance into persons, treatments, blocks of days and error, comparison of the error with the treatment variance gave $z = 1{\cdot}39$; this is 0·08 greater than the value for $P = 5\%$. In this series, therefore, the sweat production was not significantly affected by the type of drink. The standard error of the means due to sampling was $\pm 29{\cdot}3$ ml.

Higher rectal temperatures were found when the subjects drank water or refrained from drinking than when they drank saline. The mean for all post-work rectal temperatures taken after 60 min exposure, by which time thermal equilibrium was being approached and the drinking regime could be considered to be effective, was 102·82° F for non-drinking subjects, and for drinking water, 0·2 % saline and 0·5 % saline were 102·59, 101·69 and 101·66° F respectively. The corresponding heart rates were 36·9, 37·8, 31·6 and 33·5 beats per $\frac{1}{4}$ min. (Pulse rates are given as beats per $\frac{1}{4}$ min, as rates were often changing rapidly and only $\frac{1}{4}$ min counts were made.) The means for all pre-work (recovery or resting) values, again after the first 60 min, were: when not drinking, 102·65° F and 33·3 beats/15 sec, and for drinking water, 0·2 % saline and 0·5 % saline respectively, 102·45° F, 32·1 beats/15 sec; 101·52° F, 28·5 beats/15 sec; and 101·46° F, 28·8 beats/15 sec. Comparing the immediate post-work values for the men when drinking water or not drinking on the one hand, with those for when they were drinking saline, on the other, gave in each instance $t = 3{\cdot}97$. The rectal temperatures and pulses of these two men were therefore significantly lower when they were taking saline. The resting values were also significantly lower ($t = 4{\cdot}17$ for temperature, $t = 6{\cdot}35$ for heart rate).

In Fig. 1, the regression of the 'resting' heart rate on the 'resting' rectal temperature is plotted, pooling the values for the subjects both drinking water

TABLE 3. The effect of different drinking regimes on rectal temperature, heart rate and sweat production during exposure to heat. Regimes: A, drinking nothing; B, C and D, drinking water, 0·2% saline and 0·5% saline respectively

| Subject | Day | Regime | Minutes after entry 0 | 63 | 70* | 83 | 90* | 103 | 110* | 123 | 130* | 143 | Sweat output 63–143 min (ml.) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Rectal temperature (° F) | | | | | | | | | | |
| IKE | 1 | A | 98·4 | 102·0 | 102·55 | 102·7 | 103·0 | 103·0 | 103·0 | 103·1 | 103·0 | 102·9 | 984 |
| | 3 | B | 98·7 | 101·2 | 101·7 | 101·6 | 101·8 | 102·0 | 102·0 | 102·0 | 102·0 | 101·85 | 987 |
| | 2 | C | 98·3 | 101·6 | 101·95 | 102·0 | 102·3 | 102·2 | 102·5 | 102·4 | 102·5 | 102·5 | 1044 |
| | 4 | D | 98·0 | 100·4 | 100·6 | 100·8 | 100·75 | 100·8 | 101·0 | 100·9 | 100·9 | 100·85 | 995 |
| DON | 3 | A | 98·0 | 101·2 | 101·8 | 102·2 | 102·7 | 102·9 | 103·1 | 103·1 | 103·4 | 103·4 | 2158 |
| | 1 | B | 98·2 | 102·0 | 102·6 | 102·9 | 103·2 | 103·5 | 103·6 | 103·65 | 103·8 | 103·8 | 2101 |
| | 4 | C | 98·4 | 100·3 | 100·8 | 100·8 | 101·0 | 101·0 | 101·1 | 101·1 | 101·4 | 101·3 | 2208 |
| | 2 | D | 98·2 | 101·0 | 101·8 | 101·9 | 102·4 | 102·5 | 102·8 | 102·6 | 103·0 | 102·9 | 1992 |
| | | | Heart rates—beats per ¼ min | | | | | | | | | | |
| IKE | 1 | A | 27 | 35 | 38 | 33 | 42 | 35 | 32 | 32 | 34 | 34 | — |
| | 3 | B | 26 | 29 | 36 | 31 | 36 | 30 | 35 | 34 | 34 | 30 | — |
| | 2 | C | 24 | 32 | 34 | 29 | 33 | 30 | 29 | 29 | 29 | 29 | — |
| | 4 | D | 25 | 27 | 34 | 26 | 33 | 29 | 30 | 26 | 28 | 26 | — |
| DON | 3 | A | 23 | 31 | 38 | 32 | 41 | 34 | 35 | 35 | 35 | 32 | — |
| | 1 | B | 23 | 33 | 43 | 34 | 44 | 34 | 36 | 33 | 39 | 33 | — |
| | 4 | C | 21 | 27 | 34 | 27 | 33 | 28 | 30 | 27 | 29 | 27 | — |
| | 2 | D | 24 | 30 | 38 | 30 | 37 | 32 | 34 | 31 | 34 | 31 | — |

* Indicates end of work period.

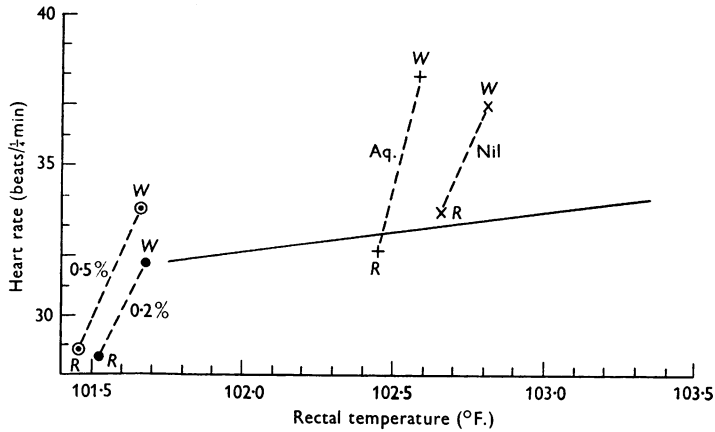


Fig. 1. Series 3 tests: the solid line shows the regression of heart rate on rectal temperature in 'recovery', with the subjects either drinking water or abstaining from drinking altogether. The regression equation is $P = 1{\cdot}3T - 80{\cdot}55$ ($P$ = heart rate, $T$ = rectal temp.), and is extrapolated one S.D. each side of the mean. The symbols and the broken lines show the mean recovery ($R$) and immediately post-work ($W$) values for the subjects taking nothing (×), drinking water (+), drinking 0·2% saline (●) and 0·5% saline (⊙) respectively. (Only values after 60 min exposure taken.)

and not drinking. On the same figure are shown the mean post-work and mean 'resting' values for each of the four regimes individually. The heart rates of the subjects taking saline were considerably lower than might have been expected if they had been governed solely by the lower rectal temperatures.

*Series* 4

Ninety tests were carried out, of which two were discarded for technical reasons. The number of tests undergone by each subject in each regime is shown in Table 4 and Table 5 (*a*) shows rectal temperatures, pulse rates and sweat losses at selected times for all tests on one subject and, for the other subjects, the mean values for each routine. Table 5 (*b*) shows the final water and salt balances in each test for all subjects. The disparity between the number of tests undergone by the different subjects* makes it difficult to apply strict statistical methods in the comparisons. However, according to Snedecor (1948), a preliminary analysis of variance ignoring the differences in cell numbers, is useful as an indicator, either of the absence of differences, or of major differences.

TABLE 4. Summary of experiments done in series 4

| Regime ... | I | II | III | IV |
|---|---|---|---|---|
| Subject | Water only | Salt only | Water and salt | No water, no salt |
| IKE | 10 | 6 | 8 | 6 |
| DON | 7 | 6 | 8 | 7 |
| ERG | 6 | 2 | 8 | 3 |
| ELL | 6 | 1 | 1 | 3* |

* ELL did drink in these tests but most inadequately and ended up in gross negative water balance (>1·3 l.). Hence these were treated as if belonging to regime IV.

In the present instance the preliminary analysis has been carried out as follows: a 4 × 4 table has been contructed, rows corresponding to individuals and columns to treatments, each cell therefore containing the results for one individual undergoing one of the four treatments (the modifying effect of the diuretic doses of water were not considered large enough to justify separate consideration, and the sea-water doses were only given in treatments (II) and (III) in which salt was already being given). For each row or column the value ($\bar{x}Sx$) was calculated. The 'sum of squares' for persons was calculated by summing the $\bar{x}Sx$ values for rows and subtracting the correction factor ($\bar{x}Sx$) for the whole series, and the sum of squares for treatments was obtained from the columns in a similar way. Some allowance was therefore made in the manner of making the calculations for the unevenness in group numbers.

The arithmetical labour involved in making complete allowances for the unevenness was not considered justified as marginal differences were not being sought for. Although the statistical comparisons were made between the unweighted means, weighted means were also calculated and are shown in the text.

* The eleven tests on subject ELL were included as it was considered that to exclude them was equivalent to suppressing data. Statistical analysis of the results, on subjects IKE, DON and ERG only, lead to the same conclusions as when all the results are considered.

TABLE 5(*a*). Rectal temperatures and heart rates at representative times during exposure and overall sweat production in the series 4 tests, for each of the regimes, (*a*) with no extra water, (*b*) with a single extra diuretic dose of water, and (*c*) with a drink of sea water. End of work periods marked by asterisks. Exposure time 160 min except as indicated otherwise by small letter at 'sweat production'. To indicate the range of individual variation all the values for one subject are shown; means only are shown for the other three. For number of tests in each group see Table 5(*b*).

| | Rectal temperature (° F) Time after entry (min) | | | | | | | | Heart rate (beats/¼ min.) Time after entry (min) | | | | | | | Sweat production (ml.) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Regime | 0 | 10 | 50* | 60 | 100* | 110 | 150* | 160 | 10 | 50* | 60 | 100* | 110 | 150* | 160 | Total for expos. | 60–110 min |
| **Subject DON—observed values** | | | | | | | | | | | | | | | | | |
| I*a* | 98·5 | 98·6 | 100·8 | 101·3 | 102·6 | 102·6 | 102·8 | 103·0 | 23 | 36 | 32½ | 33½ | 31 | 36 | 34 | 4564 | 1823 |
| | 98·6 | 98·6 | 100·5 | 101·1 | 101·8 | 101·7 | 102·0 | 102·0 | 24 | 38 | 31 | 36 | 32 | 36 | 34 | 4797 | 1830 |
| | 98·4 | 98·4 | 100·8 | 101·0 | 101·7 | 101·8 | 102·0 | 102·0 | 22 | 36 | 30 | 30½ | 29½ | 31½ | 30 | 5068 | 1992 |
| | 98·8 | 98·8 | 101·2 | 101·6 | 101·8 | 101·8 | 101·8 | 101·8 | 19 | 40 | 30 | 30 | 27 | 33 | 30 | 4542 | 1766 |
| I*b* | 98·5 | 98·7 | 101·0 | 101·5 | 102·7 | 102·7 | 102·5 | 102·4 | 26½ | 42½ | 31½ | 38 | 36½ | 35½ | 38 | 3401 | 1278 |
| | 98·8 | 98·8 | 101·5 | 101·6 | 102·7 | 102·9 | 103·1 | 103·0 | 22 | 37 | 30 | 36 | 31 | 35 | 33 | 4051 | 1568 |
| | 98·2 | 98·2 | 100·6 | 101·0 | 102·5 | 102·5 | 102·5 | 102·5 | 23 | 24 | 28½ | 35 | 32 | 33½ | 31 | 6330 | 2642 |
| II*a* | 98·8 | 98·8 | 101·6 | 102·1 | 103·4 | 103·5 | — | — | 24 | 39 | 29 | 38 | 31 | — | — | 2845[a] | 1452 |
| | 98·6 | 98·6 | 101·2 | 101·6 | 103·0 | 103·2 | — | — | 24 | 39 | 34 | 39 | 36 | — | — | 2979[a] | 1633 |
| | 98·4 | 98·4 | 101·0 | 101·5 | 102·9 | 103·1 | — | — | 19 | 37 | 30 | 39 | 34 | — | — | 2754[a] | 1508 |
| II*b* | 98·6 | 98·6 | 100·8 | 101·2 | 102·3 | 102·6 | — | — | 19½ | 36½ | 30 | 35 | 31½ | — | — | 3196[a] | 1728 |
| | 98·6 | 98·6 | 100·5 | 101·0 | 102·4 | 102·4 | — | — | 22 | 35 | 33 | 36 | 30 | — | — | 2651[a] | 1489 |
| II*c* | 98·9 | 98·8 | 101·1 | 101·5 | 102·6 | 102·6 | — | — | 21 | 39 | 31 | 33 | 29 | — | — | 2080[a] | 836 |
| III*a* | 99·1 | 99·3 | 101·5 | 102·0 | 103·0 | 103·1 | 103·0 | 102·8 | 21½ | 40½ | 35½ | 39½ | 39 | 37½ | 36 | 4933 | 1801 |
| | 99·6 | 99·5 | 101·4 | 101·9 | 102·6 | 102·5 | 102·8 | 102·4 | 20½ | 40½ | 35½ | 37 | 35½ | 36 | 31 | 5442 | 2037 |
| | 99·0 | 99·0 | 101·2 | 101·6 | 102·3 | 102·0 | 102·0 | 102·0 | 23 | 39 | 32 | 36 | 28 | 36 | 30 | 4683 | 1776 |
| III*b* | 98·5 | 98·6 | 100·6 | 100·1 | 102·3 | 102·3 | 102·5 | 102·4 | 24 | 39 | 33½ | 35 | 32 | 35½ | 32½ | 4420 | 1698 |
| | 98·4 | 98·4 | 100·6 | 101·0 | 101·7 | 101·6 | 101·7 | 101·5 | 22 | 34 | 29 | 33 | 27 | 29 | 27 | 5499 | 2186 |
| | 98·4 | 98·2 | 100·7 | 101·0 | 101·6 | 101·4 | 101·1 | 101·1 | 19 | 33 | 26 | 31 | 28 | 32 | 28 | 4041 | 1577 |
| III*c* | 98·6 | 98·4 | 100·5 | 100·8 | 101·7 | 101·5 | 101·4 | 101·3 | 25 | 40 | 33 | 33 | 31 | 34 | 29 | 4333 | 1715 |
| | 98·4 | 98·4 | 100·8 | 101·2 | 101·8 | 101·8 | — | — | 23 | 38 | 32 | 35 | 30 | — | — | 2908[a] | 1645 |
| IV*a* | 98·3 | 98·3 | 100·8 | 101·5 | 103·0 | 103·1 | 103·4 | 103·6 | 21½ | 39 | 33½ | 36 | 35 | 36 | 38 | 3521 | 1254 |
| | 98·8 | 98·8 | 101·4 | 102·0 | 103·2 | 103·5 | — | — | 24 | 39 | 37 | 40 | 32½ | — | — | 3046[a] | 1582 |
| | 99·0 | 99·0 | 101·5 | 102·1 | 103·6 | 103·8 | — | — | 21 | 40 | 35 | 48 | 36 | — | — | 3231[a] | 1696 |
| | 98·2 | 98·4 | 101·4 | 101·8 | 103·1 | 103·0 | — | — | 24 | 38 | 32 | 36 | 31 | — | — | 2908[a] | 1648 |
| | 98·9 | 98·8 | 101·4 | 102·0 | 103·4 | 103·4 | — | — | 23 | 47 | 34 | 35 | 35 | — | — | 2557[a] | 1341 |
| IV*b* | 98·7 | 98·7 | 100·7 | 101·4 | 102·9 | 103·2 | 104·0 | 103·8 | 24 | 41 | 33 | 40 | 35½ | 44 | 40 | 3795 | 1522 |
| | 98·7 | 98·3 | 100·8 | 101·4 | 102·5 | 103·0 | — | — | 23 | 39 | 32 | 39 | 40 | — | — | 3520[a] | 1884 |
| **Subject IKE—mean values** | | | | | | | | | | | | | | | | | |
| I*a* | 98·5 | 98·4 | 100·9 | 101·2 | 101·5 | 101·3 | 101·3 | 101·1 | 24 | 40 | 30 | 34 | 29 | 34 | 30 | 3147 | 1139 |
| I*b* | 98·7 | 98·75 | 100·85 | 100·9 | 101·6 | 101·5 | 101·5 | 101·4 | 26 | 39 | 31 | 39 | 32 | 35½ | 31 | 2871 | 1028 |
| II*a* | 98·3 | 98·3 | 101·0 | 101·2 | 101·9 | 101·05 | — | — | 25½ | 40½ | 35½ | 39½ | 35½ | — | — | 2441[a] | 1179 |
| II*b* | 98·35 | 98·3 | 100·75 | 101·15 | 101·8 | 101·85 | — | — | 23½ | 39 | 30 | 35 | 32 | — | — | 2861[e] | 1114 |
| II*c* | 98·4 | 98·4 | 101·0 | 101·2 | 102·2 | 101·8 | — | — | 24 | 41 | 36 | 41 | 32 | — | — | 2130[a] | 1140 |
| III*a* | 98·55 | 98·6 | 101·25 | 101·35 | 101·7 | 101·5 | 101·35 | 101·15 | 24½ | 38½ | 30 | 33½ | 28 | 33 | 29½ | 3294 | 1141 |
| III*b* | 98·4 | 98·35 | 100·55 | 100·55 | 100·95 | 100·65 | 100·8 | 100·6 | 24 | 35 | 27 | 29½ | 26 | 29½ | 25 | 2925 | 1068 |
| III*c* | 98·45 | 98·5 | 101·0 | 101·25 | 101·7 | 101·45 | — | — | 26 | 41 | 35 | 34 | 31½ | — | — | 2219[a] | 1090 |
| IV*a* | 98·4 | 98·35 | 101·2 | 101·5 | 102·5 | 102·2 | — | — | 25 | 40½ | 33 | 37½ | 33½ | — | — | 2656[e] | 1012 |
| IV*b* | 98·5 | 98·55 | 100·5 | 100·7 | 101·5 | 101·35 | 101·75 | 101·65 | 25 | 38 | 30 | 36½ | 30 | 39 | 32 | 2767 | 1064 |
| **Subject ERG—mean values** | | | | | | | | | | | | | | | | | |
| I*a* | 98·8 | 98·85 | 101·25 | 101·7 | 102·35 | 102·2 | 102·1 | 102·05 | 28 | 44 | 37½ | 38½ | 34½ | 38½ | 33½ | 4565 | 1731 |
| I*b* | 98·0 | 98·2 | 100·6 | 100·9 | 101·6 | 101·3 | — | — | 27 | 41 | 34 | 33 | 34 | — | — | 3536[b] | 1641 |
| II*c* | 98·9 | 98·8 | 100·85 | 101·2 | — | (102·05[f]) | — | — | 27 | 42½ | 38 | — | (39½[f]) | | — | 2118[c] | 1539[f] |
| III*a* | 98·75 | 98·6 | 101·1 | 101·35 | 102·05 | 102·0 | 102·2 | 102·1 | 25 | 43 | 33½ | 38 | 35 | 39½ | 35½ | 4158 | 1542 |
| III*b* | 99·55 | 99·5 | 101·75 | 101·95 | 102·55 | 102·4 | — | — | 28 | 40½ | 33 | 35 | 32 | — | — | 2784[b] | 1136 |
| III*c* | 98·2 | 98·3 | 101·2 | 101·3 | 102·0 | 102·0 | — | — | 28 | 46 | 38 | 44 | 40 | — | — | 3241[a] | 1683 |
| IV*a* | 98·8 | 98·75 | 101·2 | 101·5 | 102·9 | 102·9 | — | — | 26½ | 43½ | 38 | 45 | 40½ | — | — | 3890[e] | 1562 |
| **Subject ELL—mean values** | | | | | | | | | | | | | | | | | |
| I*a* | 99·5 | 99·5 | 102·15 | 102·4 | 103·2 | 103·2 | — | — | 27 | 45 | 40½ | 43½ | 36 | — | — | 3988[b] | 1638 |
| I*b* | 99·65 | 99·5 | 102·35 | 102·8 | 103·45 | 103·3 | — | — | 27 | 42½ | 37 | 42 | 37 | — | — | 3993[b] | 1649 |
| II*a* | 99·3 | 99·5 | 102·4 | 102·9 | 104·1 | 104·1 | 104·3 | 104·1 | 21 | 43 | 37 | 44 | 40 | 45 | 34 | 4849 | 1735 |
| III*a* | 99·4 | 99·4 | 102·6 | 102·9 | 104·0 | 103·5 | 104·1 | — | 26 | 45 | 35 | 44 | 36 | 46 | — | 4296 | 1404 |
| IV*b* | 99·5 | 99·5 | 102·25 | 102·8 | 103·75 | 103·7 | — | — | 24 | 46 | 39½ | 46 | 38 | — | — | 4378[b] | 1861 |

*Notes.* 1: *a*, *b*, *c* and *d* indicate length of exposure (see notes to table below). 2: *e* indicates single value from only test in that series which lasted 160 min. 3: *f* indicates value obtained by extrapolation. 4: *g* indicates mean derived from four tests only of a series of five.

(*a*) *Endurance.* In each of the seventy-six tests on subjects IKE, DON and ERG, there was no special incentive for them to continue beyond a certain

TABLE 5 (*b*). Water and salt balances at end of exposure for all four subjects. Complete replacement of sweat losses was rarely achieved owing to the difficulty of anticipating actual losses in the last rest period. Values in square brackets are those for two experiments which were discarded.

| | Subject DON | | Subject IKE | | Subject ERG | | Subject ELL | |
|---|---|---|---|---|---|---|---|---|
| Regime | Water balance (ml.) | Salt balance (g) | Water balance (ml.) | Salt balance (g) | Water balance (ml.) | Salt balance (g) | Water balance (ml.) | Salt balance (g) |
| I*a* | −894 | −16·6* | −313 | −15·0* | −139 | −19·9 | −920[d] | −22·0 |
| | −324 | −19·4 | −145 | −17·4* | −64 | −20·5 | −859[b] | −20·7 |
| | −385 | −21·6 | +25 | −18·7 | −118 | −13·8 | −138[b] | −16·9 |
| | −488 | −25·9 | +23 | −17·8* | −256[a] | −15·5 | — | — |
| | [−319 | −16·7] | −158 | −20·1 | −214 | −20·3 | — | — |
| I*b* | +47 | −18·2 | +506 | −14·5* | +547[b] | −15·2 | +355[b] | −20·2 |
| | +305 | −18·2* | −237 | −16·4* | — | — | +182[b] | −22·1 |
| | +288 | −25·2 | +535 | −14·9 | — | — | +546[b] | −19·4 |
| | — | — | +556 | −14·7 | — | — | — | — |
| | — | — | +755 | −14·2 | — | — | — | — |
| II*a* | −2933[a] | −3·0 | −2271[a] | +2·2 | — | — | −4629 | −2·2 |
| | −3272[a] | −3·5 | −2431[a] | +1·6 | — | — | — | — |
| | −2975[a] | −3·1 | — | — | — | — | — | — |
| II*b* | −2468[a] | −4·1 | −1542[a] | −0·7 | — | — | — | — |
| | −1880[a] | −3·2 | −2078 | −0·1 | — | — | — | — |
| | — | — | −1418 | −2·7 | — | — | — | — |
| II*c* | −1665[a] | +16·7 | −1871[a] | +8·2 | −1292[c] | +18·9 | — | — |
| | — | — | — | — | −2351[c] | +1·0† | — | — |
| III*a* | −477 | −3·6 | −464 | −2·8 | −125 | −0·8 | +87 | +2·3 |
| | −497 | −3·8 | −173 | −1·2* | −356 | −1·0 | — | — |
| | −197 | −3·1 | +269 | −3·9 | −181 | −2·4 | — | — |
| | — | — | −10 | −1·0 | — | — | — | — |
| III*b* | +118 | −3·7 | +455 | −2·6 | +514[b] | −3·1 | — | — |
| | +468 | −2·6 | +398 | −1·3 | +651[b] | −2·6 | — | — |
| | −147 | −4·2 | [+225 | −1·0] | +633[b] | −2·6 | — | — |
| | — | — | — | — | +426[b] | −2·2 | — | — |
| III*c* | +28 | +15·6 | +393[a] | +16·5 | +283[a] | +16·5 | — | — |
| | +91[a] | +15·1 | +416[a] | +15·5 | — | — | — | — |
| IV*a* | −3829 | −15·2 | −2746 | −14·5 | −3961 | −21·1 | — | — |
| | −3226[a] | −12·9* | −2064[a] | −11·3 | −3001[a] | −12·7 | — | — |
| | −3418[a] | −15·2 | −2024[a] | −11·8 | −3345[a] | −14·3 | — | — |
| | −3288[a] | −16·6 | −2547[a] | −15·9 | — | — | — | — |
| | −2651[a] | −13·8 | — | — | — | — | — | — |
| IV*b* | −3193 | −15·6* | −2291 | −15·4* | — | — | −2230[d] | −20·1 |
| | −2835[a] | −16·8 | −1903 | −15·7 | — | — | −1643[b] | −22·8 |
| | — | — | — | — | — | — | −1276[b] | −20·9 |

*Notes.* 1: duration of exposure 160 min except as indicated by letter against water balance figure as follows: [a], 110 min; [b], 135 min; [c], 90 min; [d], 150 min. 2: in balances marked * the chloride losses in the urine have not been included, but these losses were negligible. 3: in experiment marked † the subject vomited most of the sea water drunk. The necessary correction was made.

stage of subjective fatigue, if they reached this stage before the end of 160 min exposure and dropped out, as they were allowed to do, they were deemed to have 'failed' in that particular test. The nine tests in which the subjects had

no water except as 10% saline or 'sea water' were all failures. When the subjects were on a regime of a single diuretic dose of water and subsequently only 10% saline, there were four failures in five tests; full replacement of water and salt, but with the addition of the diuretic dose of 'sea water' resulted in five failures in six tests. The nature of these failures when the subjects had a chloride excess but were water depleted was usually a sudden inability to carry on rather than a gradually increasing fatigue as in the other failures; the subject would say he was in excellent condition and capable of carrying on indefinitely, then 10 or 15 min later he would suddenly find he could do no more. When the subjects were taking neither water nor salt there were nine failures in twelve tests, but with a single dose of 750 ml. water at the beginning of the test the failure rate was only one in four tests. There was one failure in eleven tests in which water and salt losses were completely replaced, and no failures in six tests in which there was, in addition, the early diuretic dose of water. When only the water losses in the sweat were replaced there were no failures in the eight tests in which the diuretic water dose was given and only one failure in the fifteen tests in which it was omitted.

These differences in failure rate were statistically significant. The results were grouped into three: (1) no water but salt, or salt grossly in excess of water, 18 failures out of 20; (2) neither water nor salt, 9 failures out of 12; (3) water replacement with or without salt, 3 failures out of 41. $\chi^2$ for this distribution was 47·7 ($P < 0{\cdot}001$).

(*b*) *Rectal temperature.* In Fig. 2 the weighted means of the rectal temperatures are plotted for subjects IKE and DON, the two subjects on whom the most complete series of tests were done, for the whole course of the exposure for each of the four treatments. The rectal temperatures were the same whatever the regime for the first hour of exposure; but in the second hour it rose higher when the subjects were without water or had only as little as 10% saline than when they were allowed to drink. On the waterless regimes temperatures rose to above 102° F and the subjects could not continue for more than 110 min, but when water was allowed temperatures never reached 102° F; thermal equilibrium appeared to be established at a slightly lower level when salt was replaced as well as water.

A preliminary analysis of variance was made of the rectal temperature at 110 min. The value of $z$ for the comparison of treatment with discrepance was 1·42, whereas for the degrees of freedom concerned when $P = 0{\cdot}1\%$, $z = 0{\cdot}9$. Such a low value of $P$ even in this preliminary analysis indicates that some at least of the differences due were not likely to be due to chance. The values of the unweighted means were: treatment I, $102{\cdot}13 \pm 0{\cdot}085$° F (S.E. of mean); treatment II, $102{\cdot}47 \pm 0{\cdot}120$° F; treatment III, $101{\cdot}90 \pm 0{\cdot}093$° F; and treatment IV, $102{\cdot}86 \pm 0{\cdot}107$° F. The differences between these were significant according to the $t$ test except in the case of I/III, where $t = 1{\cdot}832$ ($0{\cdot}05 < P < 0{\cdot}1$.)

The values of the means weighted for persons were, 102·24, 102·68, 102·24 and 102·95° F for treatments I, II, III and IV respectively.

(*c*) *Heart rate.* The pulse counts of the subjects standing, after 10 min rest and 110 min exposure, differed with treatment, but less so than did the rectal temperatures. The mean heart rates, unweighted for persons, were 32·6 beats/15 sec ± 0·31 (S.E. of mean), 34·03 ± 0·23, 31·42 ± 0·13 and 34·98 ± 0·17 for treatments I, II, III and IV respectively. The only significant differences found with variance analysis were between I and IV, II and III, and III

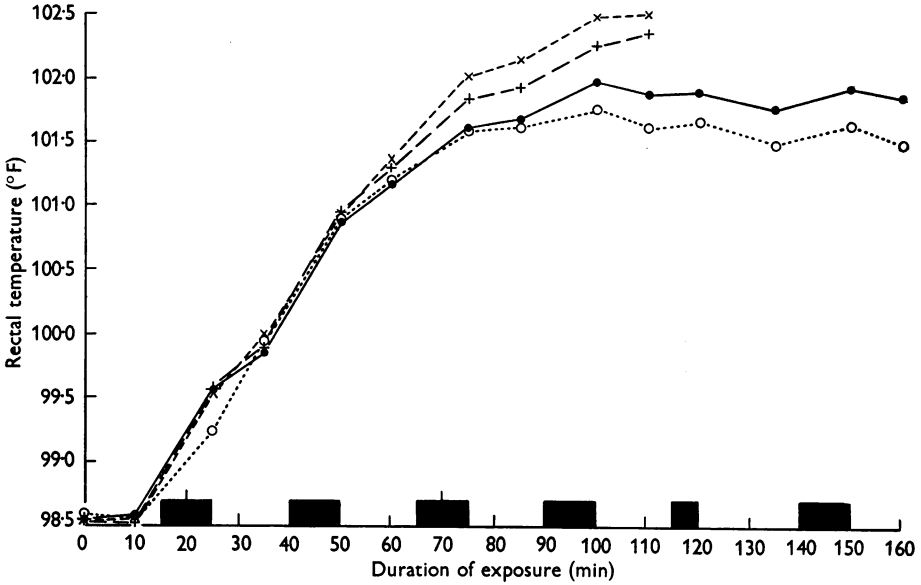


Fig. 2. Rectal temperature changes during a standard routine in a standard climate on different regimes of water and salt. Series 4 tests, weighted means for IKE and DON only. Regime I, water only: solid line and ●. Regime II, salt only: broken line and +. Regime III, water and salt: dotted line and ○. Regime IV, neither water nor salt: dashed line and ×. The solid blocks indicate the work periods.

and IV, for which $t$ was 3·62, 4·40 and 4·73 respectively (in each case $P < 0·01$). The weighted mean heart rates at 110 min were 33·1, 36·2, 32·4 and 35·2 beats/15 sec for treatments I, II, III and IV respectively.

The effect of work on the heart rate in the different regimes was tested by comparing the means for the observed rises in heart rate for all work periods after the first two. These means were 4·15 ± 0·27 (S.E. of mean), 4·84 ± 0·46, 4·03 ± 0·28 and 5·16 ± 0·60 beats/15 sec for 108 observations with treatment I, 32 with II, 88 with III and 53 with IV respectively. There were significantly smaller rises on treatments I and III when water was being taken, than on II and IV which were waterless; the pooled mean for I and III was 4·10, and

the pooled mean for II and IV was 5·05; comparison between the two gave $t=2{\cdot}376$ ($0{\cdot}01<P<0{\cdot}02$).

(*d*) *Sweat rate.* No differences attributable to the different regimes could be expected to show before 60 min exposure, and in many of the exposures on the waterless regimes the subjects did not last beyond 110 min. Sweat productions were therefore compared for the period 60–110 min. In Fig. 3 the mean sweat losses during this period are shown for each subject separately

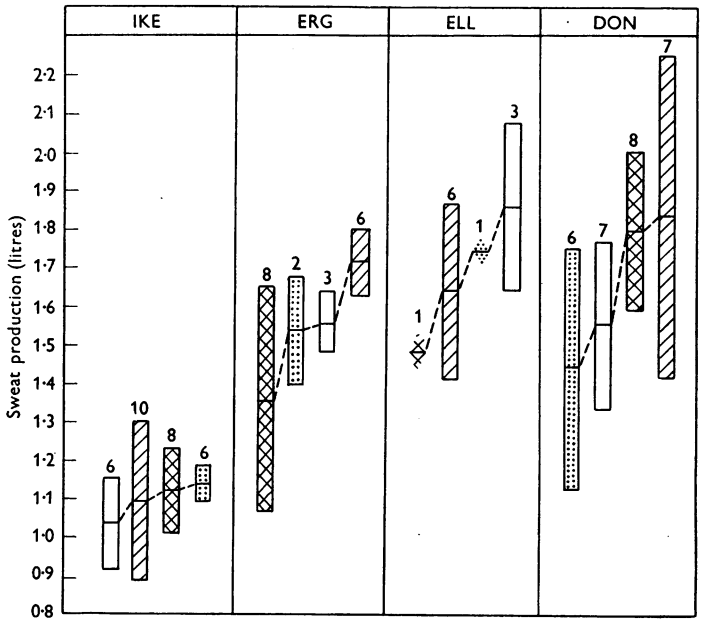


Fig. 3. Series 4 tests: the mean sweat production in the period 60–110 min, for each of the four subjects, for each of the four regimes; the subjects are arranged in ascending order of sweating capacity, and the regimes, for each subject, in ascending order of sweat production. The actual mean in each case is shown by the cross-line in the middle of each block which is drawn to include once the standard deviation each side of the mean (or, when there were only two observations, the actual range). The broken lines join the means for each of the four regimes for a given subject; the figures above the blocks indicate the number of observations made. Regime I, water only, is shown by single hatching; regime II, salt only, by dots; regime III, water and salt, by double hatching; and the empty blocks show regime IV, no intake at all.

for each regime, arranged in ascending order for each man. This diagram shows that the regime affected the sweat production differently in different individuals. The weighted means for the four regimes were for I, II, III and IV respectively 1570, 1469, 1440 and 1503 ml. There was no statistical difference between the unweighted means for the respective four regimes of $1513\pm39{\cdot}8$ ml. (S.E. of mean), $1354\pm55{\cdot}3$ ml., $1428\pm42{\cdot}8$ ml. and $1440\pm49{\cdot}1$ ml., respectively.

(*e*) *Salt loss.* The concentration of chloride in the bag samples was taken as being the same as that of mixed sweat from the whole body (Ladell, 1948). Van Heyningen & Weiner (1952) have confirmed that this is justified but only under those conditions of extreme heat and humidity which prevailed in the series 4 tests.

The overall sweat salt losses for each successive sampling period were calculated from the observed sweat loss and the chloride concentration in the sample; by summing the successive losses in the period 60–110 min and by dividing this sum by the total sweat lost during the same period, the mean chloride content of the sweat lost in this 50 min index period was estimated.

Weighted means calculated in this way were 0·477, 0·535, 0·527 and 0·509% expressed as sodium chloride for treatments I, II, III and IV respectively. The corresponding unweighted means were: 0·486 ± 0·0089% (S.E. of mean), 0·565 ± 0·0123%, 0·502 ± 0·0096% and 0·518 ± 0·0110%. Statistical comparisons were made between I and II ($t = 5·2$), I and IV ($t = 2·27$), II and III ($t = 4·01$) and II and IV ($t = 2·84$). In view of the discrepancy between the unweighted and the weighted values the only significant difference that can be accepted is probably that between I and II.

## DISCUSSION

### *Part I. The effect of drinking*

The above results suggest that the subjective effect of drinking or of not drinking is more marked than the objective effect. The chances of failure in the effort to complete a given task in the heat increase with an increasing water deficit and are further increased when there is a relative salt retention; but it is difficult to associate this increased risk of failure with any specific physiological factors. There are, however, certain changes that take place in heat-acclimatized men when not drinking which result, as Eichna *et al.* (1945) showed, in their behaving as if they were no longer acclimatized.

The effects of drinking either water or saline on rectal temperature, heart rate and sweat rate were confused. The series 4 results corroborated the observation by various workers referred to in the introduction that a man is better able to attain thermal equilibrium if he drinks. Adolph (1946) suggested that the higher rectal temperatures attained by dehydrated men exposed to heat might be protective; he has a higher skin temperature and so absorbs less heat from his surroundings, or, if the mean radiant temperature is less than the skin temperature, he radiates more to them. No skin temperatures were taken in any of the tests described here, but if Adolph is correct, very much greater sweating might have been expected by the waterless men as sweat rate is a function of the skin temperature (Robinson & Gerking, 1947). Only one subject in these tests, however, sweated most when waterless. Adolph's (1947*b*) animal experiments, moreover, did not show that an

artificially increased water intake, given by injection, had any favourable effect on heat tolerance; nor did dehydration result in any change in the evaporative loss.

In contrast to the series 4 results, in series 3 there was little difference between the rectal temperatures reached by the subjects when drinking water and that reached when they were waterless. But lower rectal temperatures were found in the men taking either 0·2 or 0·5% saline; the reverse of what had previously been found in field experiments (Ladell, Waterlow & Hudson, 1944). Series 1 results also suggest that a man reaches equilibrium at a lower rectal temperature when taking salt than when thirsting. The Fort Knox workers have reported similar differences between men on 0·1% saline and thirsting men.

In series 1, 3 and 4 when the subjects were taking salt the heart rates were slower than could be accounted for by the lower rectal temperatures. At a given rectal temperature the heart rate was lower when the men were drinking saline than when they were drinking water, and when they were taking salt than when they were taking nothing. These differences were examined statistically for the series 4 tests; for each of the four regimes of this series two regressions of heart rate on rectal temperature were calculated, one for all the observations made after 'recovery' and the other for the immediate post-work observations, in both cases only observations made after 60 min exposure being taken into account. These regressions are shown in Fig. 4(*b*) showing the 'recovery' regressions and (*a*) the immediate post-work regressions; in each case the regression lines have only been drawn over a range of standard deviation $\times 2$ about the relevant mean rectal temperature.

There were no significant differences between the regressions for regimes I (drinking) and IV (not drinking) ($n_1 = 138$, $n_2 = 71$ pairs, $t = 1{\cdot}899$); nor between regimes II (salt only) and III (full replacement of salt and water) ($n_1 = 47$ pairs, $n_2 = 112$, $t = 1{\cdot}798$). In the comparison between I and II, however, $t = 5{\cdot}099$; between I and III, $t = 4{\cdot}256$ and between II and IV, $t = 2{\cdot}984$. These three latter comparisons confirm statistically the impression given by Fig. 4 that the heart rate in recovery after exercise in the heat did not increase so rapidly with a rectal temperature rise when salt was taken; this is irrespective of whether water was taken or not. The comparison between III and IV showed no significant difference ($t = 1{\cdot}660$) possibly because the relative salt balance of the body was unchanged during heavy sweating as the sweat collected in these tests was approaching the isotonic at times. The lower 'recovery' heart rates found in the salt experiments are therefore a special manifestation of a difference which extended throughout the whole series of tests.

Except for regime III the regressions for the observations taken immediately post-work did not differ significantly from the recovery regressions; in III, however, the comparison gave $t = 2{\cdot}26$ ($n = 196$, $0{\cdot}02 < P < 0{\cdot}05$). But when

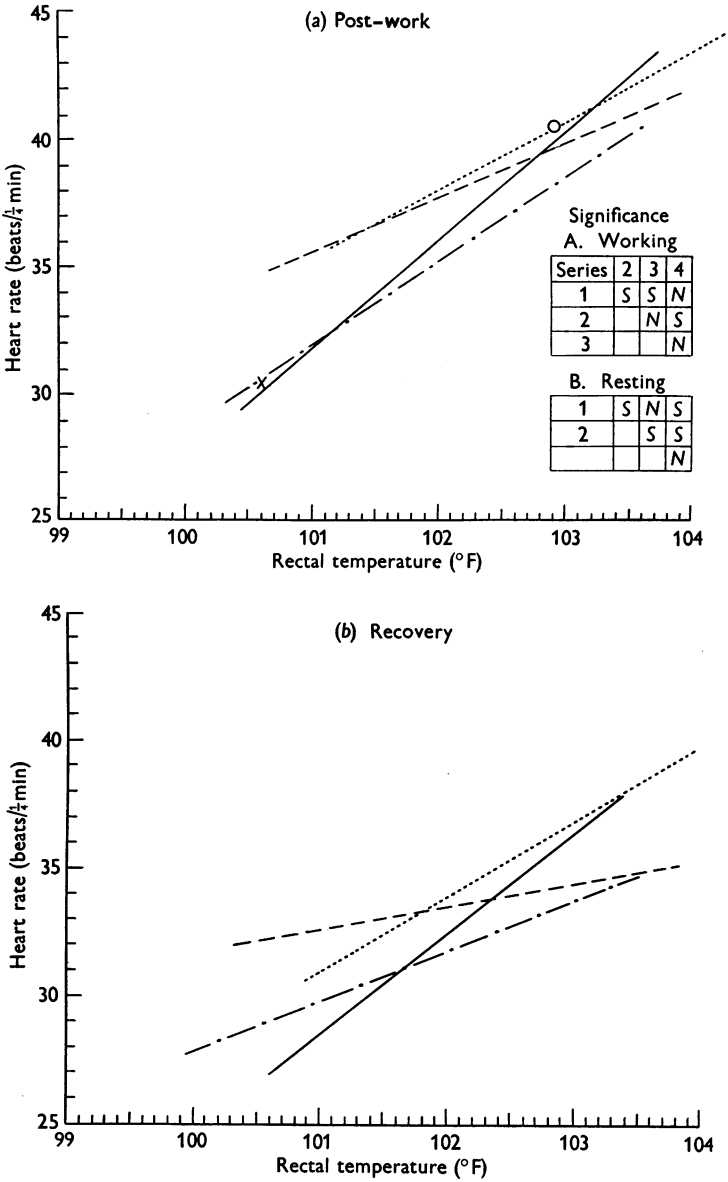


Fig. 4. Series 4 tests: the regressions of heart rate on rectal temperature, (*a*) immediately post-work, and (*b*) in recovery, for each of the four regimes. The regressions are extrapolated to twice the standard deviation of the rectal temperature each side of the respective mean. The statistical significance of the differences between the regression coefficients is shown by the table inset. Regime I, water only, solid line; regime II, salt only, broken line; regime III, water and salt, 'morse' line; regime IV, no intake, dotted line. Smith, Robinson & Pearcy's data (1952) for men working are shown as × for water-fed men, and ○ for waterless men. The regression equations are—*Regime I:* working, $P = 4{\cdot}21T - 294{\cdot}73$; resting, $P = 4{\cdot}02T - 377{\cdot}62$. *Regime II:* working, $P = 2{\cdot}11T - 177{\cdot}41$; resting, $P = 0{\cdot}95T - 63{\cdot}36$. *Regime III:* working, $P = 3{\cdot}28T - 300{\cdot}21$; resting, $P = 1{\cdot}99T - 170{\cdot}74$. *Regime IV:* working, $P = 2{\cdot}70T - 237{\cdot}06$; resting, $P = 2{\cdot}91T - 262{\cdot}88$.

the 'work' regressions for the four regimes were compared statistically the differences found were not the same as for the recovery regressions.

The similarities were a significant difference between I and II ($n_1=107$, $n_2=32$, $t=2{\cdot}525$) and no differences between II and III ($n_1=32$, $n_2=88$ pairs, $t=1{\cdot}378$), or between III and IV ($n_1=88$, $n_2=53$, $t=0{\cdot}864$). The changes were: a significant difference now between I and IV ($t=2{\cdot}37$) and no differences between I and III ($t=1{\cdot}74$) or between II and IV ($t=1{\cdot}58$).

These differences might be interpreted as indicating that, while the effect of salt was to diminish the apparent dependency of the heart rate on the rectal temperature during 'rest' in the heat, the effect of water, either with or without salt, was to maintain the exercise tolerance; the flatter regressions with higher mean heart rates seen in waterless regimes indicating that the heart was approaching a limiting rate with exercise even at low rectal temperatures so that with higher rectal temperatures no further increases were possible.

Because 'drinking' has usually meant taking 0·1% saline, this difference between the effects of saline and water has not been described by other authors. The Fort Knox team reported lower heart rates in men when they were drinking 0·1% saline than when their fluid intake was restricted; but Adolph (1946) concluded that there was no advantage to be gained by taking salt in short exposures; Pitts *et al.* (1944) reported likewise. Smith, Robinson & Pearcy (1952) found that the heart rates and rectal temperatures were lower both at work and at rest in men fully hydrated than in men with a water deficit of from 4·56 to 6·09% of their body weight. As was to be expected, the true resting values for heart rate and rectal temperature which they reported were considerably lower than the 'recovery' figures given here; viz. for water-fed men, 99·2° F, 130 beats/min and for waterless men, 101·3° F, 98 beats/min compared with 102·4° F, 140 beats/min. But, as shown in Fig. 4, their work values fall very close to the present regression lines for 'working, fully hydrated', and 'working, waterless'. Adolph (1946) also found that the heart rate of men working in the heat was affected by the state of hydration. With an increase in water deficiency the rise in heart rate during work became greater; his charts indicated that the working heart rate rises from 122 beats to 125 beats/min as the water deficit increases from nil to 2·6% of the body weight; but a further water deficit of 0·9% increases the heart rate to 130 beats/min.

No effect of drinking on the sweat rate can be clearly distinguished in any of these tests. Opinion by other workers as to the effect of drinking or of dehydration on the sweat rate is much divided. The present results show that the effect of salt depends upon the level of intake. In the three short series of tests, when subjects took saline less concentrated than their own sweat their sweat rates increased, but when they drank saline more concentrated than their sweat the sweat rate diminished. Accurate replacement of the salt loss

as in regimes II and III did not affect the sweat rate. Other workers have reported likewise; Pitts *et al.* (1944) found that the sweat rate was better maintained by drinking 0·2% saline than by drinking water; but the same workers later reported (Johnson *et al.* 1944) that 0·2% saline lessened the rate of sweating at the beginning of work in the heat, i.e. when the body temperature and skin temperature were presumably low and hence the salt intake was probably greater than the sweat loss.

Lee & Mulder (1935*b*) noted a sudden increase in the amount of visible sweat on the skin after drinking a lot of water; the same phenomenon was probably meant by Hunt (1912) when he described increased sweating by water-deficient men in response to water drinking. But this transient increase in sweat rate is, as Adolph points out, self-regulating. With more sweat on the skin there is more evaporation in a non-limiting environment, the skin therefore becomes cooler and sweating is automatically decreased again. The actual outburst of sweat is just a manifestation of the well-known sudomotor reflex of deglutition (Bazett, 1949).

In the series 4 tests, the chloride concentration of the sweat was greater when salt was taken. The skin temperature may be the controlling factor for the chloride content both for arm sweat (Robinson, Gerking, Turrell & Kincaid, 1950) and for whole body sweat (Weiner & van Heyningen, 1952*a*). Bazett (1951) suggested that the skin temperature and rectal temperature are so closely linked that the one is the reflexion of the other. It might therefore be expected that, as the subjects had lower rectal temperatures when taking salt than when not taking salt, the chloride content of their sweat would also be lower. This has been reported by Johnson *et al.* (1944) and by Lee *et al.* (1941). But a low daily salt intake diminishes the salt loss in the sweat (McCance, 1938) and a high salt intake increases the chloride content of arm sweat (Ladell, 1945) also of whole body sweat (Weiner & van Heyningen, 1952*b*). In these tests enough salt was given to cover the salt losses in the sweat as estimated from the arm bag samples; this was probably too much rather than too little. In the tests by other authors in which salt decreased the chloride content of the sweat complete replacement was not necessarily achieved, nor, apparently, even aimed at. There are probably two factors at work; first there is the 'improvement in performance' indicated especially by the lower rectal temperature, which results in lower skin temperature and hence in lower sweat chloride; and secondly, the direct relationship between the available chloride in the body and the chloride content of the sweat. It is not surprising therefore that the results of giving salt are equivocal.

### *Part II. The underlying physiological mechanisms*

The physiologically normal sweating man is one who most nearly succeeds in maintaining his original, pre-sweating state, that is a man who is replacing

his water and salt losses by drinking. The emphasis should be not on the effects of drinking, or of taking salt, but on the effects of *not* drinking water or of *not* taking salt.

The more important ways in which abstention from water or salt can act are by means of:

(1) The direct effect of an accumulating water debt.

(2) The effect of a redistribution in body fluids due to water or salt depletion or both.

(3) Changes in adrenal cortical activity secondary to either of the other two.

(4) Variations in renal activity.

(1) *The effect of an accumulating water debt*

There was not, in any of these tests, any consistent association of deterioration in performance with the accumulation of a water debt, but body temperatures tended to be higher and heart rates to be faster in men who did not drink. Abstention from drinking also diminished the exercise tolerance. Although it seems unlikely that any man could lose a high proportion of his body water and continue to sweat at the same rate, the sweat rate, also, was not obviously affected by a water debt; this is shown by the scatter diagram (Fig. 5) on which every observed sweat rate after 60 min exposure in series 4 tests is plotted against the corresponding water debt calculated for the beginning of the sweating period (*a*) for a heavy, and (*b*) for a light sweater. This is comparable with what has been described in men who abstain from water under temperate conditions. A man can lose 5% of his body weight when thirsting without showing gross physiological changes. In the first 24 hr without water a non-sweating man excretes almost as much urine as when he is drinking (Ladell, 1947). It is only when he has lost a litre or more of urine and about the same quantity as insensible water loss through skin and lungs that his urine flow begins to fall to the basal level. Adolph (1947*a*) describes a progressive diminution in salivary flow with dehydration, but this author found little change in salivary activity until the subject had been without water for 24 hr and incurred a water debt of 2 l. or more. These observations on urine and saliva may be looked at in two ways: either the body can withstand a loss of approximately 2 l. before renal or salivary activities are affected; or the body has available for such purposes approximately 2 l. of water which, by analogy with Lusk's 'free circulating protein' could be called 'free circulating water' and, until this store is used up, bodily function continues unimpaired. If the latter concept is correct, then sweat rate might also not be affected until the 'free circulating water' has been used up and a water debt of at least 2 l. incurred.

The results of the series 4 tests were examined from this point of view. Only results in the second half of each test were considered; this was to ensure,

first that the men were sweating well and second that thermal equilibrium was being approached. Sweat rates were calculated for each 10 or 15 min

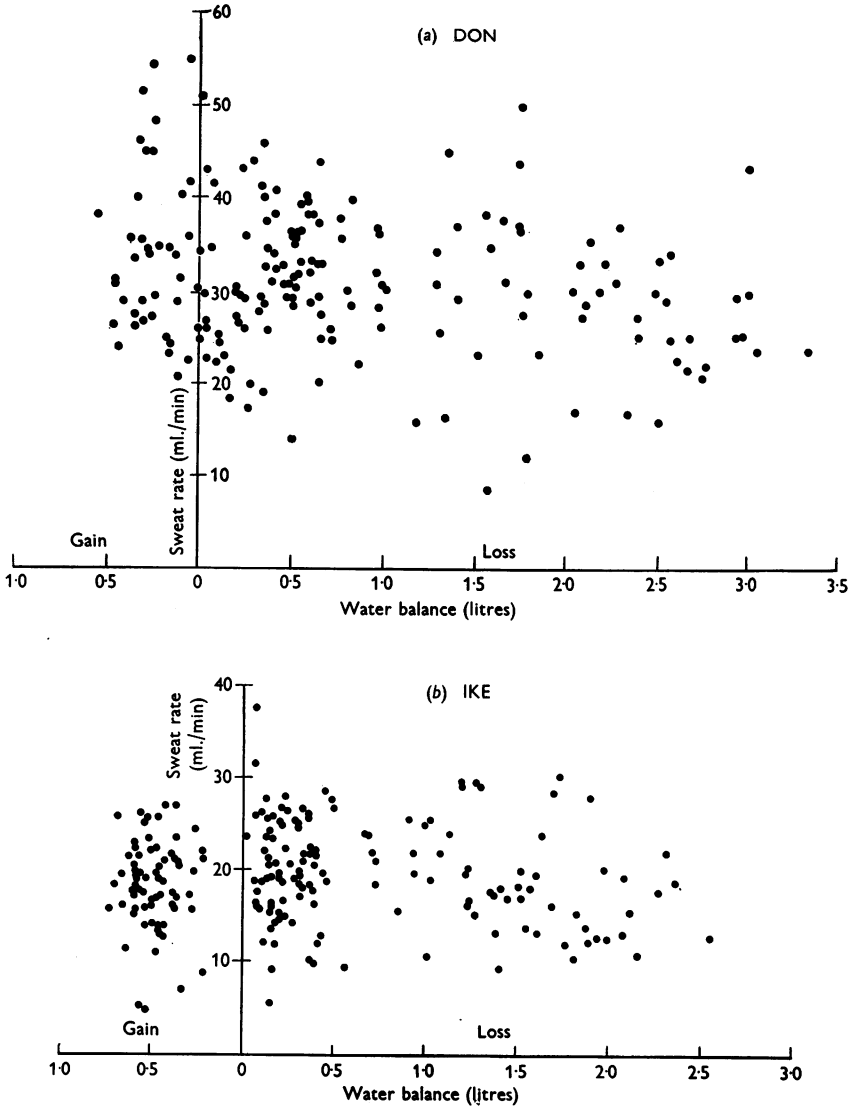


Fig. 5. Scatter diagrams showing the lack of association between sweat rate and water balance in two different subjects. DON was a heavy sweater and IKE was a light sweater. Both were fully acclimatized.

period between successive weighings and also the corresponding water debts for the beginning of each period. The average sweat rate for the period

60–110 min was calculated for each of the four subjects separately; the sweat rate/water debt figures were then classified:

(1) Into two classes according to sweat rate, either above or below the mean rate for 60–110 min for that subject.

(2) Into four classes according to water debt or water balance: (*a*) positive water balance; (*b*) water debt of not more than 1249 ml.; (*c*) water debt of 1250 and up to 2499 ml.; (*d*) water debt of 2500 ml. or more.

The results of this classification were included in a $2 \times 4$ table (Table 6).

TABLE 6. The distribution of high and low sweat rates according to water balance

| | | Water debt | | | |
|---|---|---|---|---|---|
| Sweat rate | Positive water balance | 0–1249 ml. | 1250–2499 ml. | 2500 ml. and above | Total any balance |
| Above mean for subject | 53 | 107 | 39 | 5 | 204 |
| Below mean for subject | 114 | 181 | 63 | 28 | 386 |
| Total all rates | 167 | 288 | 102 | 33 | 590 |

Means: DON, 33·5 ml./min; ELL, 34·0 ml./min; IKE, 22·0 ml./min; ERG, 30·5 ml./min.

The 590 sweat rates so classified showed a significant irregularity of distribution among the four water-debt classes: $\chi^2 = 9{\cdot}62$, $0{\cdot}02 < P < 0{\cdot}05$. The uneven distribution is almost entirely due to a preponderance of low sweat rates when the water debt exceeds 2500 ml. To exclude the possibility that this association of low sweat rates with large water debts might be due to the fact that, in general, large water debts might only have been found towards the end of long exposures in the heat when sweat rates would in any case be low owing to fatigue (Gerking & Robinson, 1946; Ladell, 1945), the number of times water debts of more than 2500 ml. were incurred within the first 110 min of exposure was counted; of the thirty-three occasions on which this large water debt was found, on eighteen occasions it had been accumulated within this period and therefore before fatigue of the sweat glands could be expected. If the low sweat rates were attributable to fatigue the ratio of low sweat rates to high among the high water-debt group would have been 15:18; the actual observed ratio was 28:5. Comparison between these two ratios gives $\chi^2 = 20{\cdot}6$, which is highly significant. The low sweat rates were not therefore a manifestation of fatigue and it appears that sweating, like two other functions of the body concerned with water as raw material, is not affected by water debt until that debt has become considerable. Any failure to show deterioration in sweat rate when subjects abstain from water is probably due to their having failed to contract a sufficiently large water debt.

(2) *Changes in fluid distribution*

In a previous communication (Ladell, 1949*a*) the alterations in fluid distribution calculated from the observed changes in water and salt balance and

in chloride concentration of the plasma were reported for twenty-seven of the eighty-eight tests of series 4. The mean alteration in intracellular fluid volume, $\Delta I$, in those tests in which full replacement was attempted and hence in which conditions were approximately normal was +330 ml.; the twenty-seven tests were, therefore, divided into two groups, A, in which $\Delta I$ was +330 ml. or more (sixteen tests), and B, those with $\Delta I$ less than +330 ml. or negative. Statistical comparisons were then made between the mean rectal temperatures and between the mean heart rates of the two groups, all values after the first 60 min exposure being included, but the working and the resting values being considered separately. The resting rectal temperatures were approximately the same for each group: group A, 71 observations, mean 102·37° F ± 0·095 (S.E. of mean), and B, 46 observations mean 102·31° F ± 0·094 ($t = 0{\cdot}3873$). The corresponding mean heart rates did however differ: for A, 35·0 beats/15 sec ± 0·40 and for B, 37·6 beats/15 sec ± 0·66 ($t = 2{\cdot}363$, $P = 0{\cdot}02$). Both temperatures and heart rates measured immediately post-work differed between the two groups; the mean of the 53 observations on temperature in A was 102·53° F ± 0·092, and of 37 observations in B was 102·97° F ± 0·147 ($t = 2{\cdot}666$, $P = 0{\cdot}01$). The corresponding mean heart rates were: for A 39·2 beats/15 sec ± 0·503, and for B 41·9 beats/15 sec ± 0·575 ($t = 3{\cdot}387$, $P = 0{\cdot}01$). The regressions of heart rate on rectal temperature were calculated for each group for both working and resting conditions; the lines are shown in Fig. 6. There is a significant difference between the two 'working' regressions, $t = 4{\cdot}397$; heart rates are higher in B but the rise with temperature is less. There was no difference between the 'resting' regressions. In A there was a significant difference between the 'working' and the 'resting' regressions, $t = 2{\cdot}909$; but no such difference between the two in B. The lack of difference between the working and resting regressions in B and the high level of the resting heart rates in this group may be interpreted as indicative of poor recovery after exercise; the heart rate when $\Delta I$ is negative (or below 'normal') becoming high and remaining high.

The association of $\Delta I$ values of +330 ml. and over with low heart rates and low rectal temperatures, and of $\Delta I$ values of less than +330 ml. or negative with high heart rates and rectal temperatures is further suggested in the scatter diagrams shown in Fig. 7. Fig. 7*a* shows the final rectal temperatures and Fig. 7*b* the final heart rates; if the distributions of high and low rectal temperatures (relative to the mean for the whole 27 of 102·7° F), and of high and low pulse rates (relative to a mean of 33·5 beats/15 sec) between tests in which $\Delta I$ was above or below +330, the distributions are as follows:

Rectal temperature below mean: $\Delta I$ above +330, 11; $\Delta I$ below +330, 2.
Rectal temperature above mean: $\Delta I$ above +330, 5; $\Delta I$ below +330, 9.

And for heart rates:

Rate below mean: $\Delta I$ above +330, 9; $\Delta I$ below +330, 2.
Rate above mean: $\Delta I$ above +330, 7; $\Delta I$ below +330, 9.

Applying Yates's correction for continuity, $\chi$ for the rectal temperature distribution was 2·19, giving $0{\cdot}005 < P < 0{\cdot}025$ (Fisher & Yates, 1948) (these values of $P$ refer to one tail of the distribution only). The relative infrequency of low rectal temperatures when $\Delta I$ is less than +330 ml. or is negative is therefore

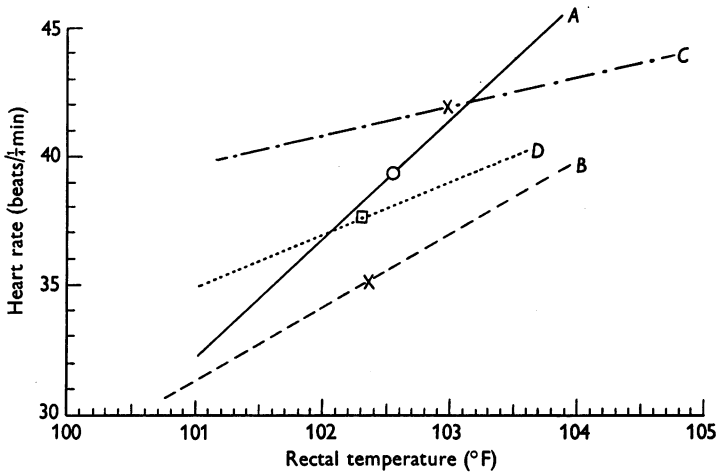


Fig. 6. Regressions of heart rate on rectal temperature in recovery and immediately post-work. Lines *A* and *B* show the regressions when $\Delta I$ is above 330 ml., for work and recovery respectively; lines *C* and *D* show the regressions for when $\Delta I$ is less than 330 ml. or is negative, again for work and recovery respectively. Each regression is extrapolated to twice the standard deviation of the rectal temperature on each side of the mean which is also indicated. *A:* shown by solid line, mean as ○; equation $P = 4{\cdot}61T - 433{\cdot}2$. *B:* shown by broken line, mean as ×; equation $P = 2{\cdot}75T - 246{\cdot}2$. *C:* shown as 'morse' line, mean as ×; equation $P = 1{\cdot}11T - 72{\cdot}5$. *D:* shown as dotted line, mean as ⊡; equation $P = 2{\cdot}03T - 170{\cdot}37$.

unlikely to be due to chance. In the case of the heart rates, analysis of the distribution as shown gives $\chi = 1{\cdot}58$ (corrected for continuity) which is not significant ($P = 0{\cdot}05$). But of the two occasions when low heart rates were associated with $\Delta I$ below +330, in one case $\Delta I$ was still above +320 ml.; if the demarcation line between 'high' and 'low' $\Delta I$ be redrawn at +320 ml. instead of +330 ml., $\chi$ for this distribution, corrected for continuity, is 2·09 ($0{\cdot}005 < P < 0{\cdot}025$).

There was no association in these twenty-seven tests between $\Delta I$ and either sweat rate (see Fig. 7*c*) or chloride loss. But in view of the significant association of $\Delta I$ with heart rate and rectal temperature it was thought that, if all tests could be considered in terms of $\Delta I$, certain of the apparent anomalies

described in this paper might be accounted for, as comparatively small changes in intake can sometimes change a positive into a negative $\Delta I$. The data for the twenty-seven tests for which $\Delta I$ had been calculated were, therefore, examined

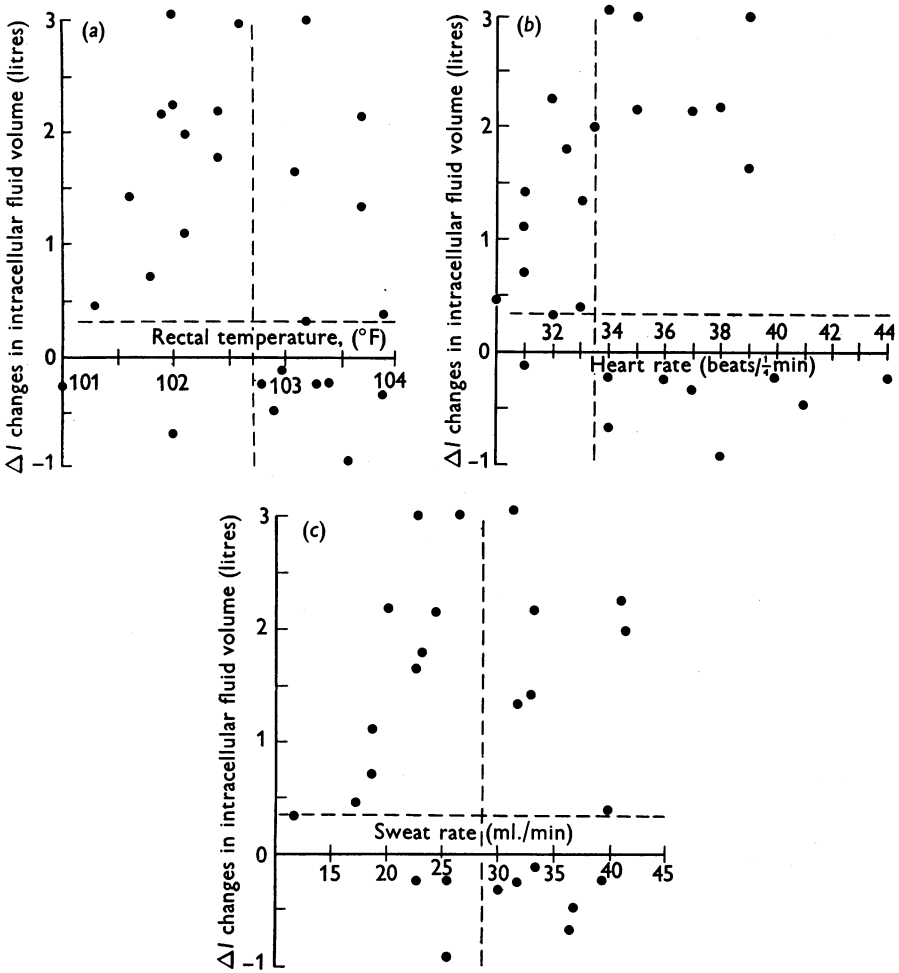


Fig. 7. Scatter diagrams showing the association of (*a*) final rectal temperatures, and of (*b*) final heart rates with $\Delta I$. The broken cross-lines are drawn at $\Delta I = +330$ ml. and at the means for rectal temperature, heart and sweat rate respectively. For comparison in (*c*) the sweat rates in the last 15 min of exposure are also plotted against $\Delta I$; here there is no association. Figures from Ladell (1949*a*). For convenience the points for $\Delta I = -3{\cdot}85$ l. have been omitted; the corresponding values were: for rectal temperature, 104·1° F; for heart rate, 34 beats/15 sec; and for sweat rate 28 ml./min.

to see whether any general expression could be derived, giving $\Delta I$ in terms of body-weight changes and sweat losses, the only relevant factors commonly observed.

If the initial chloride space in litres is taken to be numerically equal to one-fourth of the body weight in kg (Keith, 1953) and, for the purpose of these calculations, actually equal to the extracellular fluid volume, then with a change in chloride content of the fluid from $C_1$ to $C_2$ g/l. (expressed as sodium chloride) a loss of $b$ g of sodium chloride results in a change in chloride space, $\Delta E$, in a man of initial weight $M_1$, where

$$\Delta E = \left(\frac{M_1 C_1}{4} - b\right)\frac{1}{C_2} - \frac{M_1}{4}. \tag{1}$$

From this equation and from the changes in the water balance, $\Delta W$, $\Delta I$ may be calculated as $\Delta W = \Delta E + \Delta I$. In essence equation (1) is based on consideration of the changes in the chloride content of the body, $\Delta S$, which is equal, but opposite in sign, to the salt loss $b$, and

$$\Delta S = E_1 \Delta C + (C_1 + \Delta C)\Delta E,$$

where $E_1$ = initial extracellular fluid volume and $\Delta C = C_2 - C_1$, but $E_1 = \frac{1}{4}M_1$, and the small product $\Delta C \Delta E$ is negligible, whence:

$$\Delta S = \tfrac{1}{4}M_1 \Delta C + C_1 \Delta E = \tfrac{1}{4}M_1 \Delta C + C_1(\Delta W - \Delta I) = -b,$$

and so

$$\Delta I = \frac{1}{C_1}(b + C_1 \Delta W + \tfrac{1}{4}M_1 \Delta C). \tag{2}$$

If the chloride concentrations of the extracellular fluid and of the plasma are taken as equal, then the changes will also be equal, i.e. $\Delta C = \Delta P$. In the twenty-seven tests in which $\Delta P$ was measured significant correlation ($r = 0{\cdot}82$, $t = 7{\cdot}09$) was found between it and a quantity, $B$, the 'functional salt balance' (cf. Black, 1952), where $B = -(b + \Delta W\, C_1)$. When $B$ is expressed in g NaCl and $\Delta P$ in m.equiv/l. the regression equation is

$$\Delta P = 0{\cdot}313B - 0{\cdot}73.$$

But from the scatter diagram in Fig. 8 it is apparent that the constant term may be ignored, then $\Delta C = \Delta P = aB$, and if $C_1$ is in g/l. $a = 0{\cdot}01831$. Substituting this value for $\Delta C$ in equation (2) gives

$$\Delta I = \frac{1}{C_1}[b + C_1 \Delta W - \tfrac{1}{4}M_1 a(b + \Delta W\, C_1)]$$

$$= \left(\frac{b}{C_1} + \Delta W\right)(1 - 0{\cdot}00458\, M_1), \tag{3}$$

or

$$= -\frac{B}{C_1}(1 - 0{\cdot}00458\, M_1). \tag{4}$$

The change in intracellular fluid volume is therefore a function of the quantity $B$ and may be predicted, using equation (3), from the initial body weight and chloride concentration in the plasma (assuming this is equal to the extracellular

chloride concentration) and from the changes in water and salt balance. The correlation coefficient of values 'predicted' from equation (3) with the 'true' values, derived from equation (1) is 0·95, which is highly significant ($t = 14{\cdot}9$), and the regression coefficient of the predicted on the true value, calculated so as to pass through the origin is 0·933, which does not differ significantly from

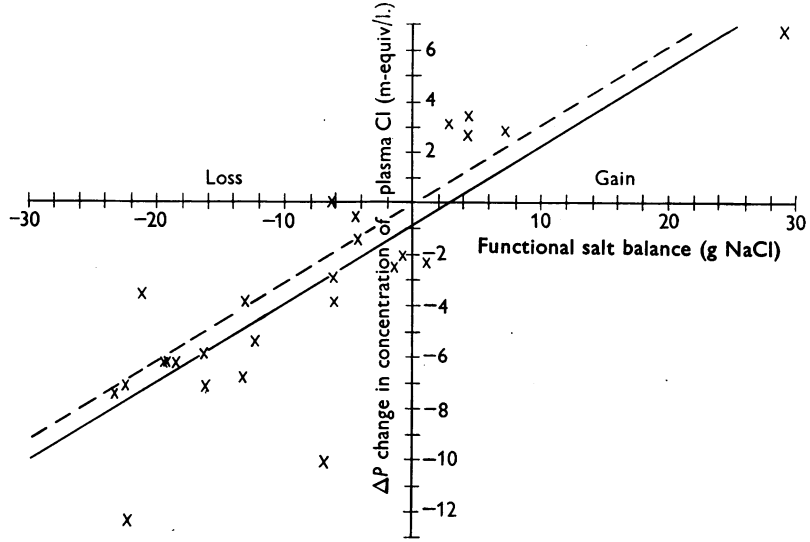


Fig. 8. Scatter diagram plotting 'functional salt balance' against changes in plasma chloride concentration; for explanation see text. The solid line shows the regression of plasma chloride change ($\Delta P$) on the 'functional salt balance' ($B$). The broken line is parallel to this regression line but passes through the origin, and shows the effect of ignoring the last term in the regression equation (see text).

unity ($t = 0{\cdot}37$); in fact, as Fig. 9 shows, $\Delta I$ may be calculated almost as well from equation (3) as from (1). Equation (3) may be further simplified by inserting a mean value of 100 m.equiv/l. (5·85 g/l.) for $C_1$; this reduces the correlation to 0·92, but reduces the mean difference between the predicted and the true values from +112 ml. to +82 ml.; finally the substitution of a standard value of 60 kg for $M_1$ gives the simple relationship

$$\Delta I = 0{\cdot}725\Delta W + 0{\cdot}123b. \tag{5}$$

Fig. 10 shows $\Delta I$, calculated from equation (5) for all combinations of $\Delta W$ and $b$. Using this chart $\Delta I$ at 110 min was determined for all the eighty-eight tests in series 4; Table 7 shows these values, together with the corresponding rectal temperatures and heart rates, and the sweat rates for period 000–110 min. From these data 2 × 2 tables were constructed (Table 8), showing the distribution of high and low rectal temperatures, heart rates and sweat rates among tests in which $\Delta I$ was negative (or unchanged) or positive. Statistically

significant irregularities of distribution were found for all three functions; heart rates below 120 beats/min, rectal temperatures below 102° F and sweat rates above 32 ml./min were rare in tests in which $\Delta I$ was negative. The probabilities of the various $\chi$ values occurring by chance were below 0·025 in every case. Thus when there was a fall in the intracellular fluid during exposure to and exercise in the heat, there were statistically significant tendencies for heart rates and rectal temperatures to be high rather than low and for sweat rates to be slow rather than fast; that is, the condition of the subject was poor.

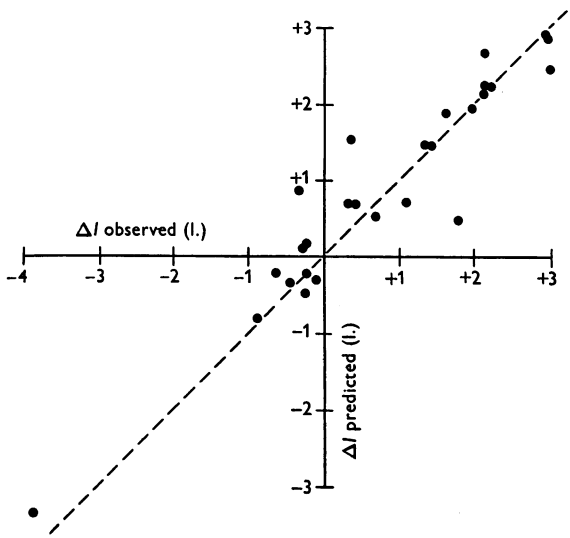


Fig. 9. Scatter diagram plotting $\Delta I$ as predicted from the formula given in the text with that calculated from the observed changes in water and salt balance and the changes in the chloride content of the plasma (Ladell, 1949*a*). The line is drawn to the equation $\Delta I$ (predicted) $= \Delta I$ (calculated).

Using the chart in Fig. 10, certain generalizations can be made about the effect of varying the water and salt intake upon the intracellular fluid volume and hence on the basis of the statistical associations found, on the performance of the subject in the heat. The lines $A$, $A'$, $B$, $B'$, $C$, $C'$ indicate some of the more important possibilities.

(1) No change in $\Delta I$ should result with full replacement of water and salt (point 0); but small miscalculations, especially in the salt requirement, would quickly give a negative $\Delta I$.

(2) The replacement of water but not of salt is represented by the lines $A$ and $A'$; $A'$ shows the unlikely situation of overhydration and $A$ the more likely state of slight underhydration. In both cases $\Delta I$ remains positive; the subject's condition is good, sweat rate is high, heart rate and rectal temperature are low, but there is danger of heat cramp (Ladell, 1949*b*).

(3) Line $B$ shows the effect of replacement of salt. Over replacement would give conditions below the line; in both cases $\Delta I$ is negative with the resultant poor condition.

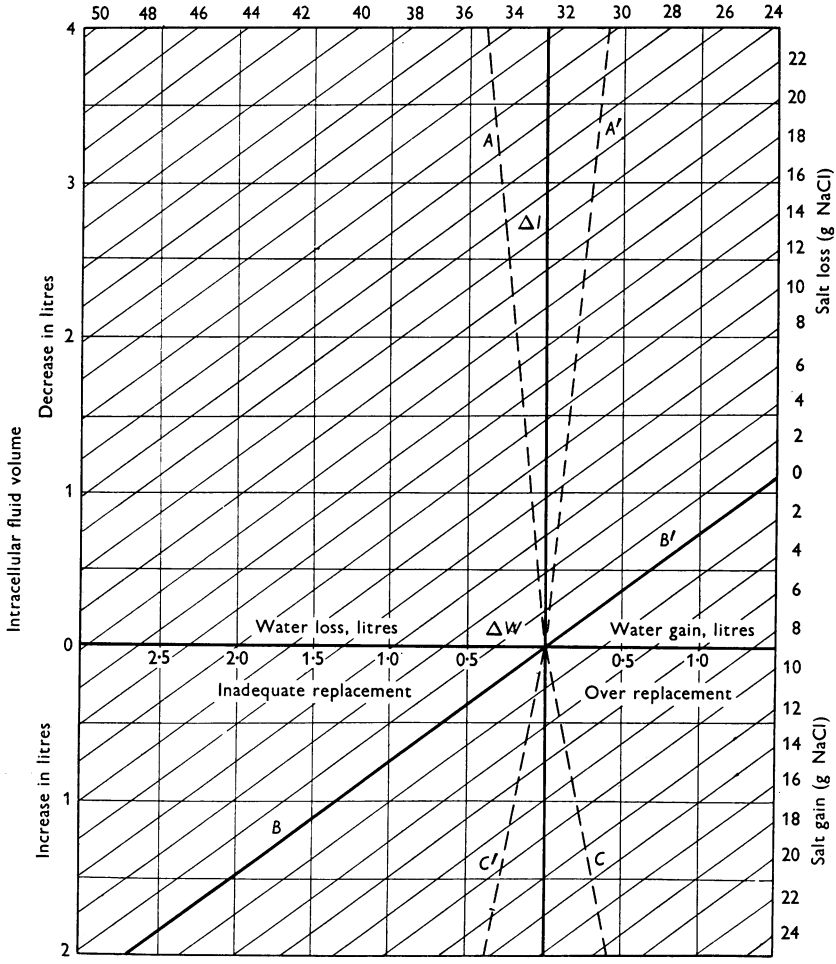


Fig. 10. The relation between $\Delta I$ (changes in intracellular fluid volume), $\Delta W$ (water balance) and $b$ (salt loss) during exposure to heat of a 60 kg man. For explanation see text. This chart may be used to predict $\Delta I$ from $\Delta W$ and $b$ only, both of which are readily estimated. Note that the salt loss is shown by the lines extending obliquely downwards from right to left, parallel with the line $B$–0–$B'$ and above it; positive salt balance, i.e. salt gain, is below this line. For body weights other than 60 kg multiply $\Delta I$ estimated from this chart by $(1 - 0{\cdot}00632x)$, where $x$ is excess weight over 60 kg; thus for an 80 kg man multiply by 0·874.

(4) Replacement of salt or water, either inadequately or not at all places the subject somewhere between $B$ and the abscissa. $\Delta I$ will always be negative unless

$$0{\cdot}123b > 0{\cdot}725\Delta W,$$

TABLE 7. Changes in intracellular fluid volume in different regimes with rectal temperatures, heart rates and sweat rates after 110 minutes exposure.

| | Subject DON | | | | Subject IKE | | | | Subject ERG | | | | Subject ELL | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Regime | ΔI (ml.) | Rectal temp. (° F) | Heart rate (beats/¼ min) | Sweat rate (ml./min) | ΔI (ml.) | Rectal temp. (° F) | Heart rate (beats/¼ min) | Sweat rate (ml./min) | ΔI (ml.) | Rectal temp. (° F) | Heart rate (beats/¼ min) | Sweat rate (ml./min) | ΔI (ml.) | Rectal temp. (° F) | Heart rate (beats/¼ min) | Sweat rate (ml./min) |
| I*a* | +850 | 102·6 | 31 | 32·1 | +1750 | 101·2 | 29 | 17·1 | +1700 | 102·1 | 37 | 28·0 | +1150 | 102·6 | 34½ | 41·2 |
| | +1400 | 101·7 | 32 | 36·1 | +1600 | 100·95 | 31 | 15·3 | +1700 | 102·3 | 35 | 29·4 | +1600 | 103·9 | 37 | 35·2 |
| | +1550 | 101·8 | 29½ | 39·8 | +1150 | 102·0 | 31 | 9·9 | +1100 | 102·0 | 34 | 31·3 | +1650 | 103·4 | 39½ | 22·6 |
| | +1550 | 101·8 | 27 | 32·9 | +1450 | 101·3 | 27½ | 19·7 | +1750 | 102·8 | 35 | 32·9 | — | — | — | — |
| | — | — | — | — | +1550 | 101·0 | 27 | 25·9 | +1600 | 101·8 | 32 | 35·2 | — | — | — | — |
| I*b* | +1600 | 102·7 | 36½ | 22·5 | +1650 | 101·9 | 33½ | 12·7 | +1950 | 101·3 | 34 | 24·3 | +2400 | 103·0 | 37 | 34·0 |
| | +1750 | 102·9 | 31 | 40·0 | +1550 | 101·4 | 27½ | 15·8 | — | — | — | — | +2100 | 103·2 | 36 | 26·5 |
| | +2400 | 102·5 | 32 | 51·0 | +1700 | 102·0 | 31½ | 20·8 | — | — | — | — | +2450 | 103·5 | 37 | 24·4 |
| | — | — | — | — | +1700 | 99·9 | 27 | 25·8 | — | — | — | — | — | — | — | — |
| | — | — | — | — | +1750 | 102·4 | 32 | 19·8 | — | — | — | — | — | — | — | — |
| II*a* | −1800 | 103·5 | 31 | 21·2 | −1450 | 102·1 | 32 | 10·9 | — | — | — | — | −2260 | 104·1 | 40 | 31·8 |
| | −2300 | 103·2 | 36 | 24·2 | −1650 | 102·0 | 34 | 17·6 | — | — | — | — | — | — | — | — |
| | −1800 | 103·1 | 34 | 22·4 | — | — | — | — | — | — | — | — | — | — | — | — |
| II*b* | −1350 | 102·6 | 31½ | 30·4 | −1100 | 102·4 | 32 | 17·6 | — | — | — | — | — | — | — | — |
| | −1050 | 102·4 | 30 | 12·5 | −750 | 101·4 | 35 | 16·6 | — | — | — | — | — | — | — | — |
| | — | — | — | — | −800 | 101·8 | 30 | 15·2 | — | — | — | — | — | — | — | — |
| II*c* | −3300 | 102·6 | 29 | 9·0 | −2450 | 101·8 | 32 | 23·8 | −3050 | 102·0 | 40 | 20·4 | — | — | — | — |
| | — | — | — | — | — | — | — | — | −1900 | 102·1 | 39 | 29·7 | — | — | — | — |
| III*a* | +100 | 103·1 | 39 | 37·9 | +50 | 102·2 | 31 | 26·8 | 0 | 103·1 | 34 | 29·2 | +200 | 103·5 | 36 | 28·0 |
| | +200 | 102·5 | 35½ | 33·1 | +150 | 101·3 | 28½ | 19·2 | +50 | 101·6 | 34 | 20·1 | — | — | — | — |
| | +400 | 102·0 | 28 | 30·1 | +200 | 101·0 | 25 | 24·8 | +200 | 101·3 | 37 | 39·0 | — | — | — | — |
| | — | — | — | — | +300 | 101·5 | 28 | 12·0 | — | — | — | — | — | — | — | — |
| III*b* | +600 | 102·3 | 32 | 31·3 | +700 | 101·3 | 27½ | 25·8 | +700 | 102·1 | 30 | 17·3 | — | — | — | — |
| | +700 | 101·6 | 27 | 41·6 | +650 | 100·0 | 24 | 16·0 | +700 | 102·4 | 33 | 18·7 | — | — | — | — |
| | +550 | 101·4 | 28 | 26·7 | — | — | — | — | +650 | 103·2 | 35 | 11·9 | — | — | — | — |
| | — | — | — | — | — | — | — | — | +600 | 101·8 | 31 | 18·8 | — | — | — | — |
| III*c* | −1850 | 101·5 | 31 | 29·5 | −1800 | 101·8 | 30 | 20·6 | −1900 | 102·0 | 40 | 31·1 | — | — | — | — |
| | −1850 | 101·8 | 30 | 17·5 | −1650 | 101·1 | 33 | 17·2 | — | — | — | — | — | — | — | — |
| IV*a* | −700 | 103·1 | 35 | 16·6 | −100 | 102·5 | 31 | 15·4 | −200 | 102·3 | 37 | 21·7 | — | — | — | — |
| | −850 | 103·5 | 32½ | 30·0 | −100 | 102·3 | 35½ | 12·6 | −650 | 103·4 | 44 | 28·3 | — | — | — | — |
| | −800 | 103·8 | 36 | 43·8 | −50 | 102·2 | 34 | 12·3 | −700 | 102·9 | 41 | 29·1 | — | — | — | — |
| | −400 | 103·0 | 31 | 30·3 | +100 | 101·8 | 33 | 21·9 | — | — | — | — | — | — | — | — |
| | −300 | 103·4 | 35 | 25·3 | — | — | — | — | — | — | — | — | — | — | — | — |
| IV*b* | −200 | 103·2 | 35½ | 27·7 | +300 | 102·0 | 32 | 20·4 | — | — | — | — | +1170 | 103·5 | 37 | 33·9 |
| | 0 | 103·0 | 40 | 33·9 | +550 | 100·7 | 28 | 18·9 | — | — | — | — | +600 | 104·0 | 39 | 39·8 |
| | — | — | — | — | — | — | — | — | — | — | — | — | +1250 | 103·7 | 33 | 31·9 |

i.e. if no replacement is taking place, $\Delta I$ remains negative unless the salt content of the sweat exceeds 5·85 g/l. i.e. it becomes hypertonic.

(5) Line $B'$ shows the effect of replacing salt and of overdrinking. If a man continues to drink, he can take in salt in excess of his losses in the sweat up to 5·85 g/l. of extra water drunk and still have a positive $\Delta I$. This situation is represented by the area between $B'$ and the abscissa. It shows why a man drinking and taking plenty of salt nevertheless may sometimes get heat cramp.

(6) A man who replaces or nearly replaces his water while taking in salt faster than he is losing it is represented by the lines $C$ and $C'$. $\Delta I$ is negative and the final condition is poor.

TABLE 8. Distribution of high and low pulse rates, rectal temperatures and sweat rates according to the estimated changes in the intracellular fluid volume.

| | $\Delta I$ negative or unchanged | $\Delta I$ positive | $\chi^2$ | $\chi$ | $P$ |
|---|---|---|---|---|---|
| Pulse rates | | | | | |
| 120 beats/min and above | 33 | 38 | 7·89 | 2·80 | <0·01 |
| less than 120 beats/min | 1 | 16 | | | |
| Rectal temperatures | | | | | |
| 102° F and above | 27 | 30 | 4·21 | 2·05 | 0·025 |
| less than 102° F | 7 | 24 | | | |
| Sweat rates | | | | | |
| 32 ml./min and above | 2 | 17 | 6·63 | 2·57 | 0·01 |
| less than 32 ml./min | 32 | 37 | | | |

*Note.* The correction for continuity has been made in each case by adding 0·5 to the value, in each case less than expectation, in the cell with the smallest expectation and then making the other corrections accordingly. The values of $P$ refer to the chance of obtaining a deviation *in the observed direction* as great as or greater than that observed.

The majority of the results found in the series 4 tests may be predicted from the chart; e.g. the 'failures' associated with a high salt intake (line $C$); the high rectal temperature associated with replacement of salt alone (line $B$) or with no replacement at all (area above $B$) and the lower rectal temperatures and heart rates found with replacement of water with or without salt (line $A$ and point O). Most of the anomalous results that have been reported relate to the drinking of saline of a fixed concentration. They are no longer anomalies if it is appreciated that a man drinking 'strong' saline (say 0·5%, as in the series 2 tests) is in the condition represented by lines $C$ and $C'$. The more nearly the salt content of the sweat approaches that of the saline being drunk the less rapidly will the salt load increase and so the slower will be the change in $\Delta I$. The result of a test of this nature, therefore, will vary between individuals according to the salt content of their sweat. This holds even when the subject is not replacing all or any of his water; the greater the rate of salt loss in the sweat the slower the rate of diminution of the intracellular fluid volume and the longer he remains in good condition. As the same individual may secrete sweat of different composition at different times the same drinking regime may effect his performance variously.

Certain of the observations on heart rate, however, could not be predicted on the basis of alterations in $\Delta I$. The heart rates of men taking salt tend to be lower at a given rectal temperature than are those of men not taking salt. From the regression lines shown in Fig. 4 the resting heart rates at the mean rectal temperature for the whole eighty-eight observations, 102·7° F, for the man drinking water is 35·3 beats/15 sec; with water and salt it is 33·3 beats/15 sec; for no replacement it is 36·0 beats/15 sec; and when taking salt only it is 34·2 beats/15 sec. The lower heart rate with full replacement is to be expected as in this condition the subject is most nearly 'normal', but the $\Delta I$ changes would suggest that a man taking salt but no water would be worse off than a man taking nothing. If, however, the corresponding changes in extracellular fluid volume are considered, it is possible to see how taking salt could improve the circulation:

$$\Delta W = \Delta I + \Delta E,$$
$$\Delta E = \Delta W - \Delta I,$$

whence from equation (5), $\Delta E = 0{\cdot}275\Delta W - 0{\cdot}123b$. If, therefore, salt is taken in excess of the sweat loss so that $b$ is negative, then the extracellular fluid volume may increase even though the water balance is negative. For this to occur it is only necessary for the saline being drunk to be 2·3 g/l. stronger than sweat. When this is the case the negative $\Delta I$ that results is reflected by the high rectal temperature and the low sweat rate, but due to the maintenance or even expansion of the extracellular fluid volume, the circulating plasma volume is high and the circulation keeps in good condition with the heart rate slower than otherwise it might be; this could be the cause of the spurious sense of well-being often experienced by men who are over-salted.

The transient improvement in circulation seen with a high salt intake is the corollary of the earlier observation by Taylor, Henschel, Mickelsen & Keys (1943) that men exposed to heat had impaired cardio-vascular responses when on a salt intake of only 5 g per day. As predictable from Fig. 10 and pointed out by many workers, salt depletion gives rise to intracellular hydration, hence a fall in circulating fluid volume.

The association of changes of intracellular fluid volume with low sweat rate reported here and by Adolph (1946) accords with the observation that very large water debts must be incurred before there is any fall in sweat rate. It has been shown (Ladell, 1949*a*) that, when subjects were not taking salt, the water debt would have to exceed 2·5 l. before $\Delta I$ became negative; there is, in fact, 2·5 l. of water available to the body without drawing upon the intracellular compartment, which may be taken as another manifestation of the 'free circulating' water referred to earlier. Adolph placed the limiting water loss for efficiency at the same level—between 2 and 3 l.

It is not known why when $\Delta I$ is negative, the rectal temperature should be

high and the sweat rate low. The diminished sweating is not due to normally developing fatigue as it may be seen before there has been time for fatigue to develop. On the other hand, if the secreting cells are affected by the loss of intracellular fluid, their osmotic pressure will be higher, hence the amount of osmotic work required to produce hypotonic sweat will be greater and fatigue might develop more quickly. The high rectal temperatures cannot be attributed to the diminished sweating as, even at the reduced rate under the conditions of most of the tests described, there is more than enough sweat available for maximum evaporative cooling; these raised temperatures are analogous with those seen in the terminal stages of death by thirst (King, 1878; McGee, 1906) and with dehydration fever (Du Bois, 1948). Gamble (1944) and others (Elkinton & Taffel, 1942) have shown that in water deficiency there is intracellular dehydration. Black, McCance & Young (1944) call attention to the generalized rise in the osmotic pressure of the body fluids during gradual dehydration and suggest this is the actual cause of death: while Winkler, Elkinton, Hopper & Hoff (1944) definitely blame intracellular dehydration, pointing out that there 'certainly must be some lower limit below which (it) cannot be tolerated; since the complex metabolism of the cells cannot continue in the desiccated state'. This would also account for the low sweat rate under these conditions.

(3) *Alterations in endocrine activity*

Barnicot (personal communication) found that a fall in circulating eosinophils frequently occurred during exposure to heat in European subjects, suggesting some adrenal cortical stimulation in heat stress. Changes in fluid distribution might also stimulate adrenal activity; Nagareda & Gaunt (Gaunt, 1952) reported that, in a rat with a water load equivalent to 5% of its body weight, or given high doses of Pitressin, the ascorbic acid content of the adrenal was reduced, indicating that either a large water load, or simple extracellular dilution stimulates the adrenal. A comparable superhydration is difficult to achieve voluntarily in man; a 65 kg man would have to drink 3¼ l. in a few minutes. Nevertheless, it has been done a few times in this laboratory and a fall in the number of circulating eosinophils was seen in most cases, especially with the further administration of Pitressin. In the climatic chamber superhydration to a very much greater extent has, however, been occasionally achieved; usually intense heat cramps resulted (Ladell, 1949*b*) but when this did not occur the subject would show the puffy appearance and the sausage fingers typical of overdosage of one of the synthetic cortical hormones, e.g. DOCA. There is therefore reason to believe that in some cases at least overhydration stimulates the human adrenal cortex.

The body's response to heat would certainly be affected by variations in adrenal cortical activity. Douglas & Paton (1952) reported that both pyrexia

and the normal body temperature in rabbits were reduced by the administration of adrenocorticotrophic hormone. In man there is indirect evidence that adrenal cortical activity reduces body temperature irrespective of thermal conditions. One of the criteria of acclimatization to heat is a smaller rise in body temperature during exposure; Conn (1949; Conn, Johnston & Louis, 1946) has shown that acclimatization is an adrenal cortical function. The natural histories of Type II Heat Exhaustion (Ladell, Waterlow & Hudson, 1944) and of the post-prickly heat syndrome suggest endocrine fatigue. In both disorders the rise in body temperature under given conditions of work and heat is greater than normal even though the amount of sweat produced is more than enough to provide full evaporative cooling (Ladell, 1951); here a lack of cortical hormone may be contributing to the higher temperatures. Rectal temperatures were slightly lower when the subjects were taking salt rather than nothing, or were taking saline rather than water. This is contrary to all immediate expectations; taking salt would depress $\Delta I$ and on general considerations would inhibit adrenal activity, either of which effects would be expected to result in an increased rectal temperature. Taking salt in large amounts, however, might either be in itself a stress, or lead, as shown above, to an expansion of the extracellular fluid volume, which could stimulate the adrenal.

A direct effect of adrenal cortical hormones cannot be altogether ruled out as a contributory cause of any improvements of circulation seen. Administration of ACTH will protect animals (Britton, personal communication) and men (Conn, Louis, Wark & Springer, 1950) against fainting on a tilt table. Horvath & Botelho (1949) have shown that the primary factor in fainting could be a 'running away' of the pulse; if this is where the adrenal acts it could act in the same way on men in the heat, keeping their heart rates down.

Salt depletion in dogs stimulates the release of ACTH (Streeten & Vaughan Williams, 1952), though it has not yet been shown in man (Doughaday & McBryde, 1950). This is not in accord with the present suggestion that excess of salt also stimulates the cortex. The difference lies in the mode of action. Too much salt could result in a temporary expansion of the extracellular fluid and a transient alteration in the osmotic pressure, the probable stimulus. Salt depletion results in a permanent and greater fall in the extracellular osmotic pressure independent of its volume. The difference is really between an acute temporary change and a chronic more permanent one. It is this chronic change, a long continued stimulus to the adrenal cortex, which is responsible, according to Conn (1949; Conn *et al.* 1950), for the development of full acclimatization to heat and for the fall in the chloride content of the sweat when men are kept on a low salt intake. Whatever the mechanism may be, however, the 'good' effects of a high salt intake, even in the presence of water, may be transient and deceptive, and to this extent unlimited salt is dangerous.

(4) *Variations in renal activity*

Weiner (1944) reported a suppression of diuresis during exposure to heat; there may also be a suppression of urine production in subjects not given a diuretic load. This effect is by no means constant, it is commoner among incompletely acclimatized subjects (personal observation) and there is considerable individual variation (Smith *et al.* 1952). If urine production is slowed up the rate of water loss is diminished; but if urine continues to be excreted the already difficult task of keeping in water balance while sweating heavily may become impossible; a man's maximum sustained sweat rate is usually greater than his maximum sustained rate of drinking without vomiting (Eichna *et al.* 1945; personal observations). A subject who drinks merely to equal his sweat loss will remain in water balance only if urine production is minimal; if urine output remains high, as it sometimes does for reasons not always understood, the subject will go into water debt with the usual consequences when it reaches the critical value.

SUMMARY

1. The effects of drinking water or saline, of not drinking at all and of taking salt alone, on fully acclimatized men working in a hot and humid environment were investigated in a number of experiments; in some tests the amount of water drunk, and/or of salt taken, was equated to the amounts of these substances lost in the sweat; in others saline of fixed strength was given in varying amounts.

2. Subjective effects were more marked than objective effects. The chances of failure to complete a given task in the heat increased with increasing water deficit. Fatigue, usually sudden in onset, was more pronounced when the water debt was high.

3. Sweat rate tended to be lower in men drinking saline. Abstention from water had no effect on the sweat rate, until water debts of more than 2·5 l. had been incurred. In this respect sweat secretion behaves similarly to urine excretion and salivation during dehydration.

4. Thermal equilibrium was established at a higher level in men who abstained from drinking than in those who did drink, and in those not taking salt than in those taking salt.

5. The heart rate in recovery increased with rectal temperature less rapidly when the subjects were taking salt or saline than when they were not drinking or drinking only water. In those taking salt the heart rates were faster at low rectal temperatures, and slower at high rectal temperatures, than in those taking water only. Exercise tolerance was better maintained by subjects when they drank water or saline than when they did not drink or took salt only.

6. The chloride content of the sweat was higher when salt was taken.

7. The changes in thermal equilibrium and in heart rates may be predicted from the changes in intracellular fluid volume ($\Delta I$) which can themselves be predicted with reasonable accuracy from the water and salt losses using the equation given.

8. Apparently contradictory responses to given conditions by different men at the same time, or even by the same man at different times, can be traced to variations in sweat rate and especially to differences in the sodium chloride losses in the sweat, which shift the water and salt balance in different ways.

9. Further modifications of the response to heat may be the result of alterations in adreno-cortical activity or of changes in renal function.

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