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Thermoregulation in Exercising Man During Dehydration and Hyperhydration with Water and Saline

by
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The plateau level of deep body temperature during exercise is affected by the water balance of the subject. A dehydration, due for instance, to a sweat loss in hot conditions, results in an increased core temperature compared to that obtained during work at the same metabolic level in the normally hydrated state. Further, a hyperhydration has been shown to cause a lowering of the exercise temperature level compared to normal (Moroff and Bass, 1965).

Snellen (1966) stated that the change in body temperature which occurs during exercise can be divided into a change due to exercise and a change proportional to the degree of dehydration.

The effect of the state of hydration on the body temperature is usually thought to be due to circulatory failures: after water loss less blood is available for the heat transport between core and shell. Consequently the gradient must increase in order to maintain the heat balance, since heat flow is equal to or proportional to blood flow x ($T_{core} - T_{skin}$) differential.

Recently Snellen (1971) reported experiments in which body temperature during rest was changed by drinking tap water and saline. The change could be divided into:

(1) a change due to temperature of the fluid drunk;
(2) a change due to the volume drunk and
(3) a change due to the osmotic change produced in the body.

Our experiments deal with the last two factors mentioned above.

We studied deep esophageal temperature (T_{es}) during a 60-min exercise on a Krogh bicycle ergometer at 20°C T_a. In the normal condition an exercise plateau temperature was reached in 20-30 min. The work load was about 50% of the subject's maximal aerobic capacity, 540 kpm/min, $\dot{V}_{O_2}$ = 1.5 l/min.

By letting the subject drink water before the experiment we increased his body's fluid contents and decreased the osmolality. By letting him drink saline we increased his fluid contents and increased osmolality, while by dehydrating him we decreased body fluid content and increased the osmotic pressure. The volumes gained were $1\frac{1}{2}$ liter water and 1 liter 2% NaCl, and the dehydration was 1 kg sweat loss during exercise or in a sauna.

The dilution or concentration of the blood was evaluated in 2 ways, by measuring colloid osmotic pressure (Hansen Osmometer) and by the hematocrit. The latter was corrected for the osmotic swelling or shrinkage due to changes in blood osmolality from the normal average 287 mOsmol/l. There was a linear relationship between the two expressions of plasma volume.

The osmolality of the plasma was determined by a freezing point depression method (Ramsay and Brown, 1955).

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Figure 1 shows the time relations of T_{es} at the 5 experimental conditions, normal, hyperhydrated with H_2O ($1\frac{1}{2}$ l), hyperhydrated with 1 liter 2 % sodium chloride, dehydrated 1 kg by sweating in a sauna, and by exercise.

A plateau temperature is reached in all conditions, but the temperature level is lower than normal when hyperhydrated with water, while higher than normal in the 3 other conditions.

Figure 2 shows the esophageal temperature plotted against an expression of plasma volume, the colloid osmotic pressure. It appears that no relationship exists. The same deep temperature can be found for the whole range of colloid osmotic pressures.

However, a linear relationship is found between plateau temperature and plasma osmolality (Fig. 3). At high osmotic pressures high T_{es} were observed, both after saline ingestion where the plasma volume is above normal, and after the two types of dehydration with a lower than normal plasma volume.

The heart rate during exercise was higher than normal in the dehydrated states, and also slightly after salt water drinking, while considerably lower than normal after drinking pure tap water (Fig. 4).

Measurements of cardiac output gave nearly identical values in all 5 conditions (Fig. 5). This means that the changes in plasma volume, and its effect on stroke volume is fully compensated by the changes in heart rate.

In Fig. 6 stroke volume is plotted against plasma volume expressed by colloid osmotic pressure. Increase in colloid osmotic pressure is followed by a decrease in plasma volume in these conditions. The expected negative relationship between stroke volume and plasma volume is demonstrated.

In conclusion: it seems that the observed changes in esophageal temperature during work cannot be ascribed to central circulatory failures. Further, the observed plateau temperature does not seem to correlate with plasma volume changes (colloid osmotic pressure). However, there is a strong relationship between the osmolality of the plasma and the plateau temperatures.

The effect of osmolality on the temperature regulation may be a direct effect of plasma osmolality on the activity of cells in hypothalamic temperature centers, via a reflex from adjacent osmorecepters in the anterior hypothalamus, or the osmolality effect may be the result of a peripheral inhibition of the sweat gland function, due to the high osmotic concentration in the intercellular fluid, which must occur after dehydration or NaCl ingestion.

A change in maximal aerobic capacity due to the high plasma osmolarity might also influence the temperature levels, since it has been found the exercise temperature level is best correlated with the relative load, % max $\dot{V}_{O_2}$ (Åstrand, 1960; Saltin and Hermansen, 1966). Preliminary experiments indicate, however, that max $\dot{V}_{O_2}$ is unchanged by water and salt water ingestion.

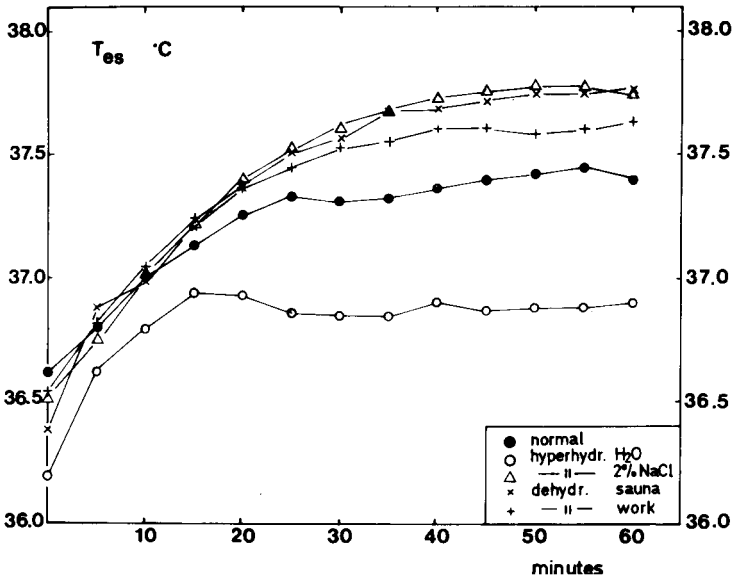

Fig. 1. Esophageal temperature (T_{es}) during 60 min exercise 540 kpm/min in 5 experimental conditions.

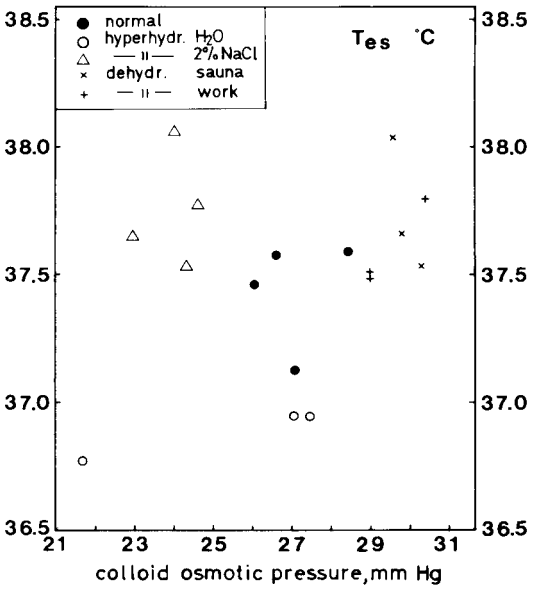

Fig. 2. Plateau values of esophageal temperature (T_{es}) plotted against plasma colloid osmotic pressure.

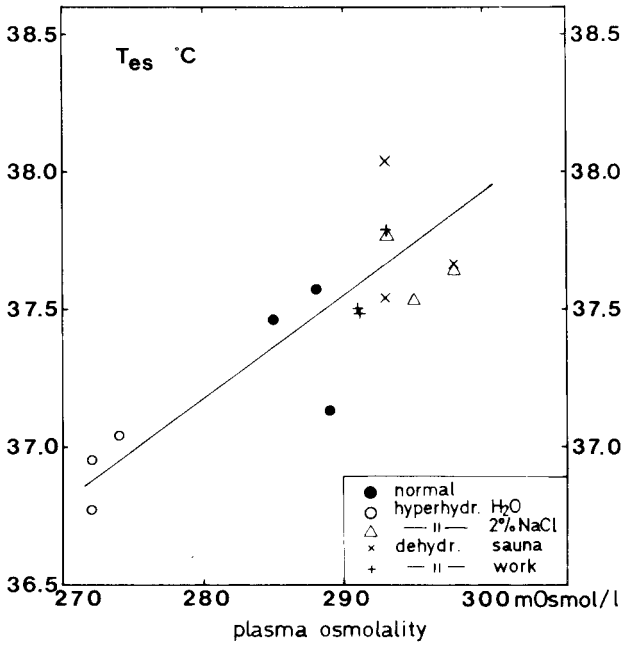

Fig. 3. Plateau values of esophageal temperature (T_{es}) plotted against plasma osmolality.

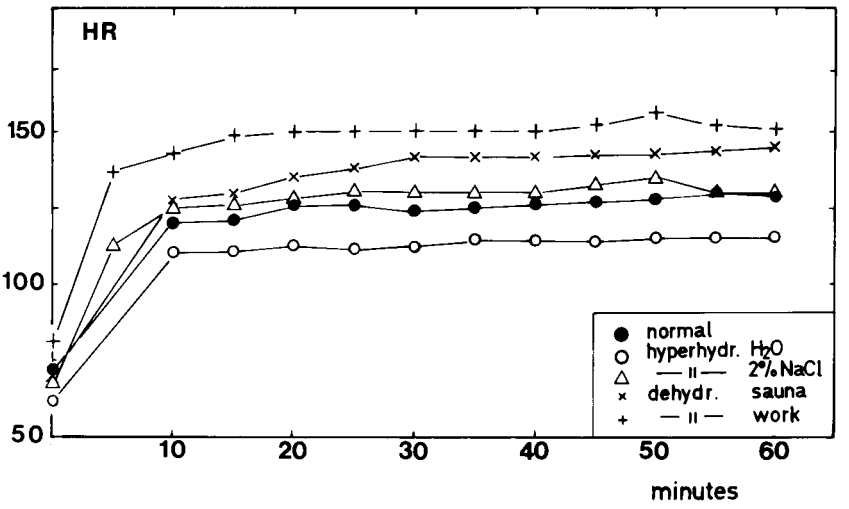

Fig. 4. Heart rate during 60-min exercise 540 kpm/min in 5 experimental conditions.

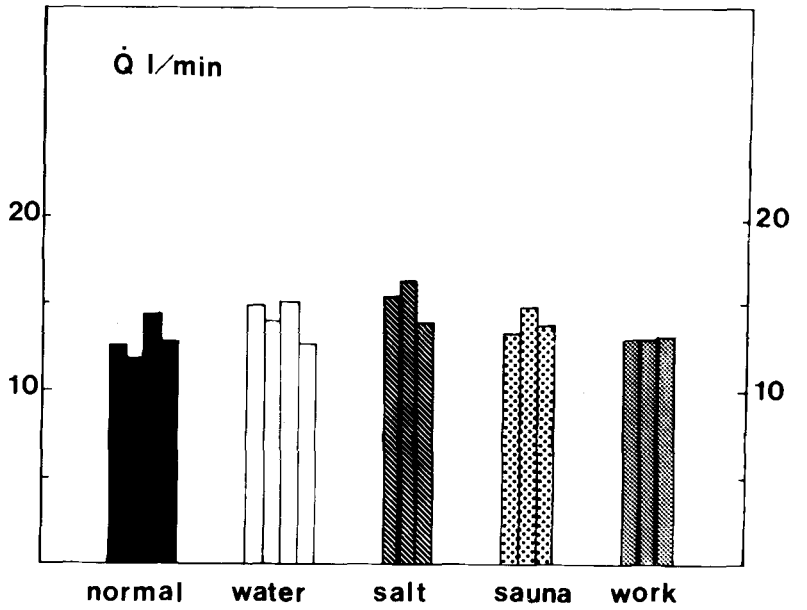

Fig. 5. Cardiac output in steady state of exercise, 540 kmp/min in 5 experimental conditions.

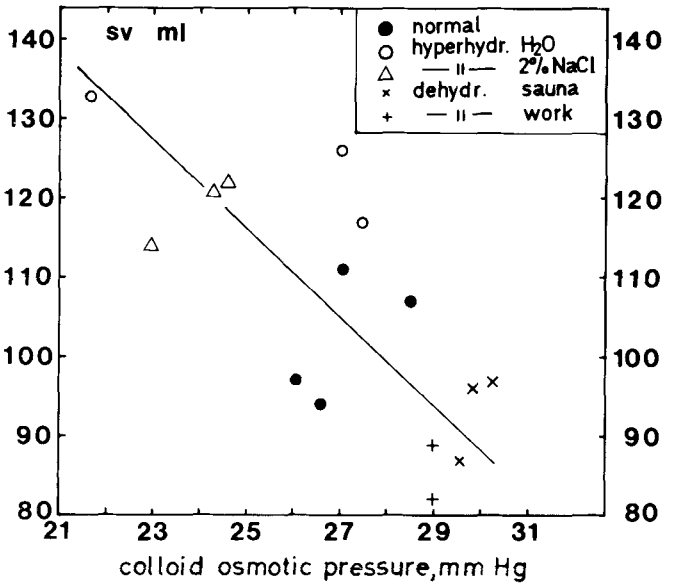

Fig. 6. Stroke volume in the 5 experimental conditions in steady state plotted against plasma colloid pressure.

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