

SLOW AND RAPID COOLING: THE DIFFERENCES IN PHYSIOLOGICAL RESPONSES

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INTRODUCTION

The response to external cooling is largely dependent on the skin thermoreceptors. It is known that the activities of the skin cold receptors are static and dynamic. The functional significance of the two types of activity remains an open question. It is known that the cold receptors show hardly, if any, dynamic activity during cooling at a rate lower than $0.01-0.02^{\circ}\text{C}\cdot\text{sec}$ whereas, at higher cooling rates, they begin to exhibit dynamic activity which increases with cooling rate (1). **An** attempt is made here to elucidate the specificity of the formation of the cold defense responses, sympathetic system activation and the trace effects of two types of external cooling: slow, when skin thermoreceptors show no dynamic activity and rapid, when they show it.

MATERIALS AND METHODS

Urethane anesthetized Wistar rats ($1.0 \text{ g}\cdot\text{kg}^{-1}$) were cooled in the area of the abdomen (25 cm^2) with a thermostat. At the first cooling, the rate was either low ($0.004-0.006^{\circ}\text{C}\cdot\text{sec}$) or high ($0.03-0.05^{\circ}\text{C}\cdot\text{sec}$). The depth of each cooling was the same, rectal temperature decreased by 1.8°C . Then, rats were warmed and, after recovery of all the resting parameters repeatedly cooled at a high cooling rate. Intracutaneous temperature of the cooled surface of the abdomen, rectal temperature, total oxygen consumption (the metabolic response), Intracutaneous temperature of the thigh were continuously recorded. Thigh temperature allowed us to estimate the changes in the tone of the skin vessels, i.e. heat loss.

In other experimental series, noradrenaline (NA) and adrenaline (A) concentrations in arterial plasma were measured by high-performance liquid chromatography with electrochemical detection (3). Blood samples (0.5 ml) were drawn from the femoral artery three times from each slowly or rapidly cooled animal: 1) before cooling, 2) at a rectal temperature decreased by 0.5°C and 3) by 3°C . Animals were given Ringer solution (0.5 ml), i.v., after each blood sampling as fluid replacement.

RESULTS

Resting parameters were $36.8\pm 0.24^{\circ}\text{C}$ for rectal temperature, $34.0\pm 0.33^{\circ}\text{C}$ for thigh and $36.8\pm 0.26^{\circ}\text{C}$ for abdomen skin temperatures, $20.4\pm 1.2 \text{ ml}\cdot\text{min}\cdot\text{kg}^{-1}$ for oxygen consumption.

At the first rapid cooling, the metabolic response was triggered before rectal temperature started to fall (Figure 1). Later, after fall in a rectal temperature,

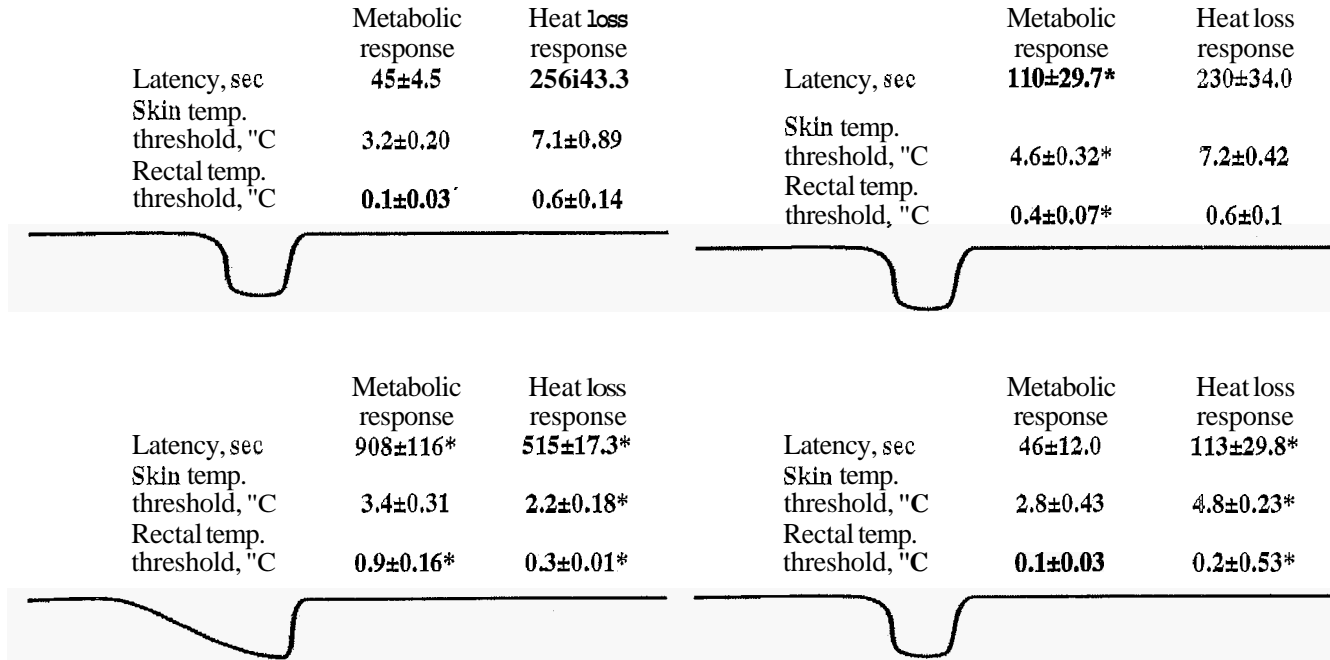


Fig.1. Latency and threshold temperatures for the metabolic (total oxygen consumption) and heat loss (skin blood flow) responses to the first rapid (top) and slow (bottom) cooling and to the repeated rapid cooling. Means are values \pm SE. * - significant differences compared to the values at the first rapid cooling, $P < 0.05$, Student's test.

heat loss started. Conversely, at slow cooling, heat loss is reduced first as a result of the response of the skin vessels, then, on the background of a considerably lowered rectal temperature, metabolic rate increases.

Temperature thresholds of the cold defense responses to test repeated cooling depended on the type of preceding cooling, slow or rapid (Fig. 1). Rapid cooling in contrast the slow one resulted in the increase of the temperature thresholds for the metabolic response to repeated cooling. Slow cooling had no effect on the development of the metabolic response, but decreased the thresholds of blood vessel response to repeated cooling.

The resting mean values for catecholamine concentration in plasma were $0.620.079 \text{ ng/ml}$ for NA and $1.090.203 \text{ ng/ml}$ for A. A decrease in rectal temperature by 0.5°C at rapid cooling produced a 2.6-fold increase in NA and a 2.8-fold in A in plasma (Fig. 2). At a rectal temperature decreased by 0.5°C after slow cooling, plasma catecholamines did not change. When rectal temperature was lowered by 3°C , the increase in plasma NA was virtually the same at both cooling rates and only plasma A increased greater after deep rapid than slow cooling.

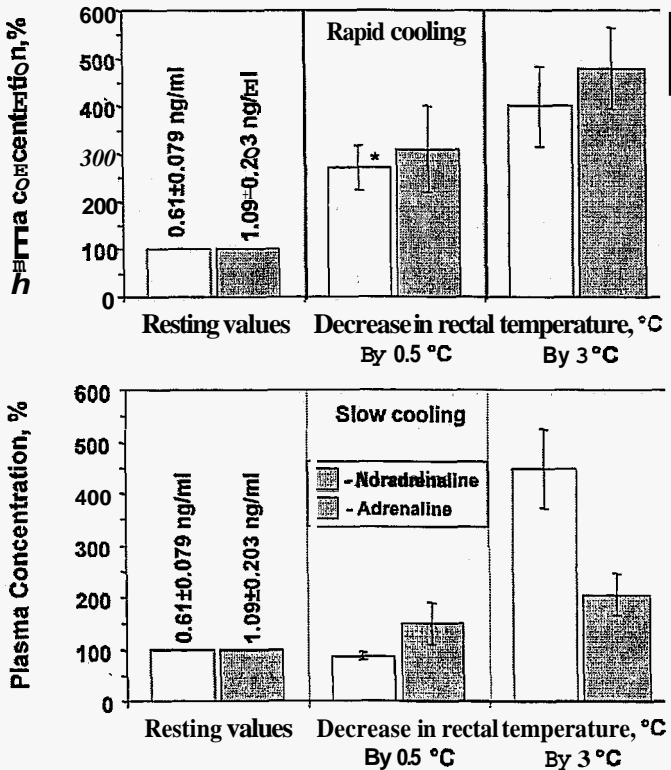


Figure 2. Changes in noradrenaline and adrenaline values in arterial plasma produced by rapid and slow cooling of rats. Bars denote the standard errors of means. * - significant differences compared to resting values. $P < 0.05$.

CONCLUSIONS

Thus, we demonstrated the importance of the cooling rate in the formation of the cold defense responses. Experimental data indicated that at rapid cooling, i.e. in the presence of the dynamic activity of the peripheral cold receptors, the metabolic response can be initiated with a very short latency without any decrease **in** deep body temperature. **This is** consistent with the results obtained in humans **during** sudden cold water immersion (2). Moreover, the formation of the thermoregulatory response to repeated cooling may be different and it depends on the **type of** the preceding slow or rapid cooling. Rapid cooling primarily delays the metabolic response and slow cooling lowers the threshold for heat loss decrease. The results support and extend the idea that the sympatho-adrenal system is activated by an external cold stimulus and, furthermore, reveal that the activation may be different depending on the cooling rate. The difference was more apparent at the early steps, when rapid cooling was accompanied by a considerable rise in plasma catecholamines and slow cooling by an unaltered catecholamine level in plasma.

To conclude, the dynamic activity of the skin cold receptors may change the development of the thermoregulatory responses to both the first and repeated cooling and provide the conditions for an earlier activation of the sympatho-adrenal system.

REFERENCES

1. Davies S.N., Goldsmitt G.E., Hellon R.F., Mitchell D. 1983. Facial sensitivity to rates of temperature change: Neurophysiological and psychophysical evidence from cats and humans. *Journal of Physiology*, 344, 161-175.
2. Mekjavic I.B., LaPrairie A., Burke A., Lindborg B. 1987. Respiratory drive during sudden cold water immersion. *Respiratory Physiology*, 70, 121-130.
3. Tcvetovskaja G.A., Naumenko S.E., Knjazkova L.C., Pyko T.N., Gilinsky M.A., Latysheva T.V. 1996. Sympatho-adrenal system at correction of mitral defect in conditions of unperfusion hypothermia. *Anesthesiology and reanimation*, 2, 35-38.