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Summary

An attempt to modulate the fever-induced changes in the blood level of endogenous arginine vasopressin (AVP) by intravenous injection of exogenous AVP in different doses, and thus to investigate the possibility of vasopressin hormonal pool to participate in the thermoregulatory mechanisms of febrile process, was made in experiments in rabbits.

According to this purpose the pattern of AVP blood concentration changes during intravenous leucocytic pyrogen fever was described. It was shown that plasma but not cisternal cerebrospinal fluid concentration of immunoreactive AVP increased with growing body temperature. A significant positive correlation between plasma level of AVP and rectal temperature was established both in febrile and normal conditions.

The second preparative series was aimed at determining «physiological» and «pharmacological» AVP doses, and characterizing their effects in non-febrile rabbits. Besides thermoregulatory indices the heart rate was recorded as far as it is known that baroreflex bradicardia in rabbits in response to intravenous AVP injection is mediated via medial preoptic area of anterior hypothalamus — the key structure of body temperature regulation. In doses of 100 pg—1 ng AVP did not influence either rectal or ear skin temperature, or heart rate in euhydrated rabbits in thermoneutral conditions. Intravenous injection of AVP in the doses of 10—100 ng led to the development of slight bradicardia but did not change the thermoregulatory indices. In the doses 1—10 ug which are higher than the blood content of endogenous AVP by 10^3—10^4 times the peptide produced deep bradicardia and simultaneously short-term contradirected temperature changes, i. e. rectal temperature increased by 0,1—0,3 °C and cutaneous temperature slightly decreased. The latter effects are likely to reflect general vasoconstriction and reduction of heat transfer from heat-producing body core to the periphery.

In the third experimental series AVP was injected intravenously during leucocytic pyrogen fever in one of following doses: 1 ng (the dose induced no changes in both thermoregulatory indices and heart rate in non-febrile animals); 100 ng («physiological» dose possessed mild bradicardiac but not hypo- or hyperthermic action) and 10 ug («pharmacological» dose influenced both deep and cutaneous temperatures, and heart rate in normal conditions). The peptide was injected in 15 min after pyrogen administration just at the time of maximal rate of increase of endogenous AVP blood level. It was shown that in doses of 1 and 100 ng vasopressin did not influence the development of fever, but in the dose of 10 ug exerted obvious fever-prolonging effect. The latter may be explained by entering brain tissue and central action of AVP.

It is suggested that hormonal pool of vasopressin does not directly participate in the body temperature regulation nor in normal nor in febrile conditions. At the same time AVP blood level follows body temperature dynamics. The reaction of vasopressin hormonal pool induced by deep body temperature elevation of febrile or non-febrile genesis is likely to be aimed at developing antidiuresis and subsequently at conserving of water which is necessary for effective heat loss. The described fever-prolonging «pharmacological» effect of AVP

is to be taken into consideration as far as the broadening of application of vasopressin as regulatory peptide in clinical practice may lead to use of non-traditional doses of AVP in pathological conditions.

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