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# TRENDS OF PLASMA CORTICOSTERONE LEVELS IN RABBITS AFTER EXPERIMENTAL CONCUSSION

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The role of the hypophyseo-adrenocortical system (HAS) in the formation of the stress reaction to various stimuli, including photic, acoustic, electrical, and also expectancy, is currently under intensive study [5, 11]. Tissue damage, pain, surgical operations, and anesthesiologic procedures are also known to activate protective and adaptive reactions that determine the course of recovery and repair, and which are expressed as increased synthesis and secretion of ACTH and corticosteroids, processes that can be regarded as the result of activation of the HAS [11, 13].

Since mild head injury (concussion) accounts for the greatest relative proportion of cases of head injury and leads to a significant increase in the degree of disability among the victims, the aim of the present investigation was to determine the plasma corticosterone levels in rabbits at different times after experimental concussion.

## EXPERIMENTAL METHOD

Experiments were carried out on 64 male "Gray Giant" rabbits weighing 2-2.5 kg. The animals were kept on the standard animal house diet. Graded trauma was inflicted by the method described previously [9].

Blood plasma was obtained from the unanesthetized animals from 9 a.m. until noon, during decapitation of the animals.

The plasma corticosterone concentration was determined by the method of De Moor et at. [15], with certain modifications. To 50-100 $\mu$l of plasma 300 $\mu$l of water and 1 ml of hexane were added, the mixture was shaken for 20 sec, centrifuged for 5 min at 1000g, after which the hexane was removed. The residue was treated with 1.2 ml of methylene chloride, shaken, and centrifuged under the same conditions, The extract was washed with 100 $\mu$l of 0.1 N NaOH, and then with 100 $\mu$l of water, after

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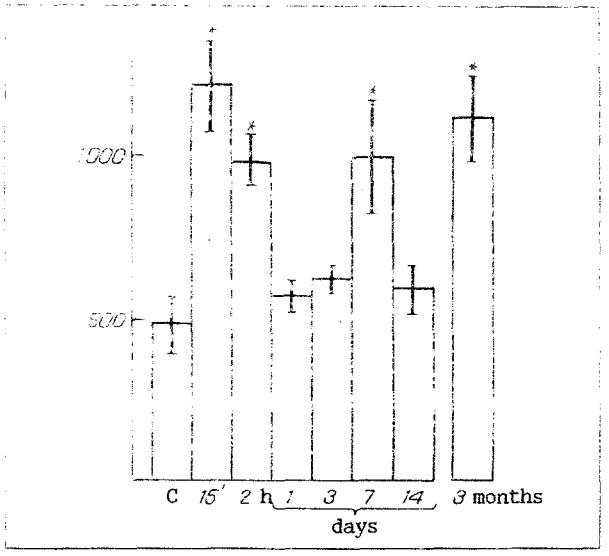


Fig. 1. Plasma corticosterone concentration (in nmoles/liter) of rabbits with experimental concussion. C) Control. Asterisks indicate values for which $p < 0.05$ compared with control values.

which a sample of 1 ml of the methylene chloride extract was withdrawn add transferred into test tubes each containing 0.5 ml of a mixture of concentrated $H_2SO_4$ and ethyl alcohol (3:1). The contents of the tube were shaken and centrifuged for 5 min at 1000*g*, after which the methylene chloride was removed and the fluorescence of the samples was measured 90 min later on an MPF-4 fluorometer ("Hitachi," Japan) in cuvettes with a capacity of 0.4 ml ($\lambda$ excitation 470 nm, $\lambda$ fluorescence 530 nm).

The numerical results were subjected to statistical analysis by Student's test [12].

## EXPERIMENTAL RESULTS

The experimental results are shown in Fig. 1. The plasma corticosterone concentration in the control animals agrees with data in the literature [14]. The plasma corticosterone level in rabbits 15 min after concussion was 2.5 times higher, and after 2 h it was twice as high as in the control, whereas after 24 h it had virtually returned to its initial value. The first flash reflecting an increase in the corticosterone concentration was probably due to activation of the limbic structures of the brain, which are responsible for regulating the functional intensity of the HAS system, through specific and nonspecific conducting systems running from the analyzers, and it can be regarded as a stress reaction to trauma. In this case, the stress-inducing factors were pain and fear. The corticosterone concentration 3 days after trauma remained within the limits recorded in the control animals. However, 7 days after concussion, a second burst reflecting an increase in the corticosterone concentration in the rabbits' blood plasma was observed. Its level was almost doubled, possibly due to metabolic changes arising in the experimental animals towards the 7th day after trauma [2, 4, 6, 7] and requiring correction with mobilization of the adaptation system of the HAS. Quite possibly, activation of the HAS system is effected by particular metabolites synthesized in traumatized structures. Another possibility is that the increase in the corticosterone concentration on the 7th day was due to the development of morphological changes in the rabbit brain [9]. Thus the second rise in the corticosterone concentration may have been due both to development of the adaptation reaction of the animal and a disturbance of integrative interactions between structures of the limbic system responsible for regulating the HAS [10].

It was shown that by the 14th day after trauma the corticosterone level returned to values observed in the control animals.

In a high proportion of cases (more than 50%) so-called late sequelae may develop after head injury. An urgent problem at the present time is how to predict the probability and character of post-traumatic complications. We postulated that the development of such complications may be connected with persistent disturbance of the neurohumeral status, due to the need to compensate the negative changes taking place as a result of trauma. Investigation of the corticosterone concentration 3 months after trauma showed that the corticosterone level was 2.5 times higher than in the control. The increased corticosterone concen-

tration after such a long period points to long-term stress of the HAS, possibly leading to the development of pathological changes in the cardiovascular and other systems [1, 3, 8].

Thus the results of this investigation suggest that the traumatic factor leads to more profound changes in hormonal homeostasis than stress. Evaluation of the functional activity of the HAS adaptation system relative to the glucocorticoid level can evidently be used as a diagnostic and prognostic test, indicating the state of the body in the late period after trauma.

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