equina, even in the chronic period of trauma considerable recovery of the patient's motor function can be achieved [5]. Recovery is based on the ability of voluntarily contracting muscles to take over additional motor functions. Movements controlled by the distal segment of the spinal cord can be triggered by the motor apparatus of the sound part of the body, by the formation of new motor reflexes and by the involvement of new muscles in the movement, in much the same way as was observed in spinal dogs. The second and no less important factor is the use of intensive physical thereapy and massage: the prevention of dystrophic changes in the spinal reflex apparatus [1].

LITERATURE CITED

1. T. N. Nesmeyanova, Stimulation of Recovery after Spinal Cord Trauma [in Russian], Moscow (1971).
2. T. N. Nesmeyanova, Byull. Éksp. Biol. Med., No. 10, 25 (1975).
3. A. N. Trankvillitati, in: Rehabilitation of Patients with the Sequellae of Injury to the Spine and Spinal Cord [in Russian], Kiev (1969).
4. A. N. Trankvillitati, in: Remedial Gymnastics [in Russian], Moscow (1970), pp. 305-308.
5. L. Guttman, Spinal Cord Injuries, Oxford (1976).

MECHANISMS OF THE PRIMARY RESPONSE OF THE ADRENAL CORTEX TO PAIN STRESS IN DOGS

S. A. Eremina, T. V. Kondrukh, R. F. Morozova, and E. A. Rogacheva

UDC 616-001.36:577.17

A sharp decrease in the glucocorticoid content accompanied by an increase in the free cholesterol and a decrease in the content of esterified cholesterol were observed in the adrenal cortex of dogs 10-15 sec after nociceptive stimulation. The blood concentrations of the hormones were increased, mainly due to the protein-bound hydrocortisone fraction. The next phase of the response (30-60 sec after stimulation) was marked by activation of synthetic processes, leading to considerable accumulation of hormones in the gland. The blood glucocorticoid level was doubled, the original ratio of hydrocortisone to corticosterone was restored, and the transcortin depot was replenished. The role of the adrenal and transcortin depots of glucocorticoids in the feedback mechanism during stress is discussed.

KEY WORDS: stress; adrenal cortex; glucocorticoids.

Despite extensive literature on the manifestations of stress and its pathogenetic mechanisms, the initial factors of the stress response, leading to activation of synthesis and secretion of hormones of the pituitary—adrenocortical system have not yet been adequately explained. It was therefore decided to study the earliest stages of the response of the adrenal cortex to stress.

EXPERIMENTAL METHOD

Noninbred mature male dogs were used. For several days the animals were adapted to the experimental situation. A state of pain stress was produced by one-stage mechanical injury to the thigh, not sufficient to produce shock. The animals were decapitated during the 10-15 sec after nonciceptive stimulation. The adrenals and blood from the inferior vena cava from the region where it receives the lumboadrenal veins were used as the test material.

Hydrocortisone, cortisone, costicosterone, and cholesterol and its esters in the adrenocortical tissue were investigated by thin-layer chromatography [1-2]. The levels of hydro-

Department of Pathological Physiology, Rostov Medical Institute.

(Presented by Academician of the Academy of Medical Sciences of the USSR V. K. Kulagin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 88, No. 7, pp. 18-20, July, 1979. Original article submitted November 10, 1978.

0007-4888/79/8807-0691$07.50

TABLE 1. Content of Corticosteroids and Free and Esterfied Cholesterol in the Adrenal Cortex of Dogs Before and After Nociceptive Stimulation

Group of animals	No. of experiments	Weight of adrenals, mg	Glucocorticoids: hydrocortisone μg/g tissue	hydrocortisone μg/weight of gland	cortisone μg/g tissue	cortisone μg/weight of gland	corticosterone μg/g tissue
Intact (control)	10	880,0±88,2	10.6±1,4	9,3±1,3	4,4±0,4	3,9±0,35	4,8±0,33
Experimental:							
10-15 sec after nociceptive stimulation	12	798,7±81,6	2,9±0,13*	2,3±0,1*	2,3±0,26*	1,84±0,21*	1,9±0,19*
30-60 sec after nociceptive stimulation	4	757,5±73,3	26,7±3,0*	20,2±2,3*	4,4±0,9	3,3±0,71	9,3±1,8*

Group of animals	No. of experiments	Weight of adrenals, mg	Glucocorticoids: corticosterone μg/weight of gland	Cholesterol: free μg/g tissue	free μg/weight of gland	esters μg/g tissue	esters μg/weight of gland
Intact (control)	10	880,0±88,2	4,2±0,30	7,9±0,73	6,9±0,64	47,4±3,1	41,7±2,7
Experimental:							
10-15 sec after nociceptive stimulation	12	798,7±81,6	1,52±0,15*	10,3±0,9*	8,23±0,78	36,6±2,61*	29,2±2,1*
30-60 sec after nociceptive stimulation	4	757,5±73,3	7,0±1,4	4,6±0,52*	3,5±0,4*	39,7±3,46*	30,0±2,6*

*Changes significant compared with data for intact animals ($P \leq 0.05$).

cortisone and corticosterone, in the free forms and bound with transcortin, were determined fluorometrically [3].

EXPERIMENTAL RESULTS

Nociceptive stimulation led to a sharp decrease in all fractions of glucocorticoids in the adrenal cortex: hydrocortisone by 72%, cortisone by 50%, and corticosterone by 60%. Depletion of the adrenocortical hormone reserves, in the writers' view, was the result of mobilization of steroid secretion before a corresponding increase in their synthesis. The consequent greater decrease in esterified cholesterol (by 30%) compared with the increase in its free fraction (by 25%) is indirect evidence in support of the view that the rate of glucocorticoid synthesis during this period remained within its previous limits whereas the sharp increase in cholesterol esterase activity led to accumulation of the precursor (Table 1). The assumption that steroid production may have been inhibited seems less logical for, on the contrary, that process is usually accompanied by an increase in the cholesterol ester pool. The state of synthesis in the adrenal gland during the first moment of action of the stressor perhaps reflects a latent period in the switch of steroid production from the basal to the high, stressor level.

Meanwhile the total plasma glucocorticoid concentration immediately after trauma fell sharply because of a decrease (by 56%) in the concentration of hydrocortisone and, in particular, of its protein-bound form, whereas the levels of free and transcortin-bound corticosterone remained virtually unchanged. As a result the physiological ratio of hydrocortisone to corticosterone, which in dogs is 2:1, was significantly changed to 1:1 (Fig. 1).

In some experiments, for technical reasons material for investigation was obtained later — from 30 to 60 sec after stimulation. This group of experiments reflected the next phase of formation of the corticosteroid response to stress, which is characterized by hypercompensation of synthesis and secretion in the adrenal (Table 1). The hydrocortisone level in the cortical tissue was increased by 2.5 times compared with the control group and by 9 times compared with the results of the previous series of experiments. The corticosterone concentration was twice and 6 times higher respectively, as a result of which the original ratio between the hormones in the gland was approximately restored. A further decrease was ob-

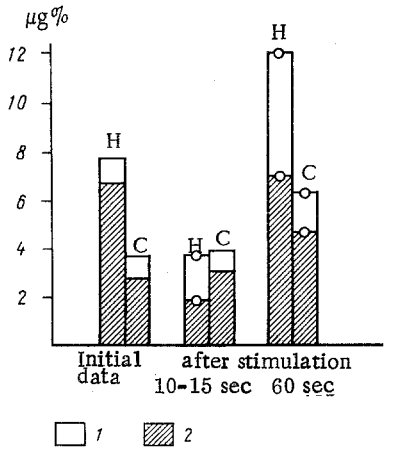

Fig. 1. Concentrations and relative proportions of free and transcortin-bound fractions of hydrocortisone (H) and corticosterone (C) in blood of dogs before and after nociceptive stimulation. 1) Free fractions; 2) transcortin-bound. Circle indicates significance of difference from initial values: $P<0.05$.

served in the total cholesterol level as a result of a decrease in the concentration not only of its esters, but also of the free form, evidence of intensification of the initial reactions of steroid formation. The original ratio between total hydrocortisone and corticosterone in the blood was restored, but their absolute concentrations were double the control values mainly on account of an increase in the free, biologically active form of the hormones. Characteristically at this stage, just as during the primary response, the most marked changes were found in the hydrocortisone fraction (Fig. 1). Analysis of the results suggested that the primary response of the adrenal cortex to stressor stimulation is probably one factor in the regulatory mechanism of feedback, and is characterized by the following features: 1) deprivation of the hormone reserves in the adrenal cortex through mobilization of secretory processes in the gland; 2) a fall in the blood glucocorticoid concentration and utilization of the transcortin depot, evidently as a result of increased elimination of hormones by the tissues; the changes were more marked in the case of the hydrocortisone fraction than of cortisterone; 3) accumulation of precursors in the gland in preparation for the subsequent intensification of hormone synthesis, confirmed by the development of the hypercompensation phase.

The results are all the more interesting because the role of the peripheral corticosteroid component in the "trigger" mechanisms of activation of the hypothalamic-hypophyseo-adrenal system in acute stress has not hitherto been proved in the literature. The primary decrease in the circulating blood corticosteroid level in stress, stimulating ACTH secretion by a feedback principle, although postulated theoretically [6], was not confirmed experimentally [4, 5]. The results now obtained shed definite light on the role of the initial decrease in the blood steroid concentration immediately after stressor stimulation in the feedback mechanisms in the pituitary–adrenal cortex system.

In conclusion, the characteristics of the initial corticosteroid response to pain stress revealed by these experiments indicate the role of the adrenal and transcortin depots in meeting the primary demands of the body for increased quantities of these hormones.

LITERATURE CITED

1. T. I. Ivanenko, N. R. Shchedrina, and M. I. Kondor, Probl. Éndokrinol., No. 1, 72 (1968).
2. M. A. Krekhova and M. K. Chekhranova, Vopr. Med. Khim., No. 1, 93 (1971).
3. L. N. Negrei and E. B. Sopotsinskaya, Lab. Delo, No. 1, 15 (1975).

4. G. Franksson and C. A. Gemzell, Acta Chir. Scand., 106, 24 (1953).
5. A. A. Dandberg, D. M. Neilson, J. Palmer, et al., J. Clin. Endocrinol., 13, 629 (1953).
6. G. Sayers and M. Sayers, Endocrinology, 40, 265 (1947).

A PHENOMENON IN VESTIBULAR COMPENSATION

G. I. Gorgiladze

UDC 616.282.3-003.96

The role of spinal afferentation from the lower half of the body in compensation of the sequelae of unilateral loss of vestibular function was studied in experiments on guinea pigs. Division of the spinal cord at the thoracic level under local anesthesia had no appreciable effect on the development of compensation after simultaneous or subsequent destruction of the labyrinth and did not disturb compensation in previously labyrinthectomized animals. Division of the spinal cord in labyrinthectomized animals under ether or chloroform anesthesia was accompanied by a sharp disturbance of compensation. These substances evoked a similar picture of decompensation in unilaterally labyrinthectomized animals with an intact spinal cord also. The results indicate that the disturbance of vestibular compensation discribed in the literature after division of the spinal cord under ether anesthesia is not the result of removal of spinal afferentation from the lower half of the body, but is due to the direct effect of inhalational anesthetics on compensation mechanisms.

KEY WORDS: vestibular system; compensation of disturbed functions; effects of general anesthesia.

The role of spinal afferentation in compensation of the effects of unilateral labyrinthectomy has been studied in experiments on animals: Division of the spinal cord in guinea pigs at the thoracic level under ether anesthesia was accompanied by a significant disturbance of compensation mechanisms [3]. However, in earlier experiments on rabbits the present writer found that processes of vestibular compensation are very sensitive to the action of ether and chloroform. Inhalation of these anesthetics led to a marked disturbance of compensation for a long time after unilateral labyrinthectomy [1].

The object of the present investigation was to verify whether removal of spinal afferentation from the lower half of the body does in fact weaken compensation of the effects of unilateral loss of vestibular function.

EXPERIMENTAL METHOD

Experiments were carried out on 85 adult guinea pigs of both sexes. The animals were divided into several groups. In group 1 (six animals) the spinal cord was divided 10 days before unilateral labyrinthectomy, in group 2 (seven animals) the spinal cord was divided simultaneously with destruction of the labyrinth, in group 3 (27 animals) it was divided at various times after labyrinthectomy: 1 week in six animals, 1 month in eight, 2 months in seven, and 2.5 months in six animals. Groups 4 and 5 were controls: In the 31 animals of group 4 labyrinthectomy alone was performed, and in those of group 5 (nine animals) only the spinal cord was divided. A further five intact guinea pigs (group 6) also were used. The labyrinth (usually the right) was destroyed mechanically through the middle ear under local anesthesia. Postoperative nystagmus was recorded in the horizontal derivation by an electrooculographic method on a Mingograph [2] apparatus and the animals also were photographed on motion picture film. The spinal cord was divided with a spatula after exposure at the level of segments T5-7 and preliminary infiltration of the surrounding tissues with 1% procaine. The animals of all groups inhaled ether and chloroform until a state of complete anesthesia developed: The unilaterally labyrinthectomized animals inhaled the anesthetics 1 week and 1, 2, and 6 months after destruction of the labyrinth.

(Presented by Academician of the Academy of Medical Sciences of the USSR O. G. Gazenko.)
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 88, No. 7, pp. 21-23, July, 1979. Original article submitted May 12, 1978.

 0007-4888/79/8807-0694$07.50