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Pituitary-Adrenal and Sympathetic-Adrenal Systems in Patients with Complicated Spinal Trauma

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Plasma contents of catecholamines and cortisol are measured in patients with spinal trauma and spinal cord injury by high-performance liquid chromatography and radioimmunoassay. Compared with healthy donors and patients with skull injury, the epinephrine content in spinal patients is increased significantly, while the cortisol content shows a tendency toward an increase. This suggests that disturbances both in the transmitter and hormonal systems persist 2-3 weeks after trauma, complicating its treatment.

Key Words: *blood cortisol and catecholamines; spinal and spinal cord trauma*

Generalization of stress reaction may have adverse consequences and complicate the primary disease. The ratio between the transmitter and hormonal regulation determines the nature of nonspecific adaptation syndrome. In light of this, it was interesting to assess the functional state of the adrenals and the activity of the sympathetic-adrenal system. In the present study we investigated plasma contents of catecholamines (transmitter component) and cortisol (hormonal component) in stress reaction.

MATERIALS AND METHODS

The study included patients with complicated spinal trauma (1.5-2 months after trauma, group 1), patients no longer than 1 week (group 2) and 2-3 weeks (group 3) after craniocerebral trauma (CCT), and healthy donors (control, group 4). Plasma samples were stored at -50°C. The content of glucocorticoids was determined by radioimmunoassay using highly specific antiserum [4]. Catecholamines (epinephrine, norepinephrine, and dopamine) were assayed by high-performance liquid chromatography in a Beckman apparatus with a BAS electrochemical detector. The data were analyzed using the Student's t test.

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RESULTS

In patients with complicated spinal trauma (group 1), plasma epinephrine content was significantly increased, while the norepinephrine and dopamine contents were close to normal (Table 1). The cortisol content was higher than in healthy donors (group 4) and patients with CCT (groups 2 and 3). In patients with CCT, plasma cortisol concentration decreased, the decrease being statistically significant in group 3 patients. In patients with the central nervous system (CNS) trauma, changes in the hormonal and transmitter components were related to each other, which was not observed in patients with CCT and spinal patients at the same period after trauma (2 weeks).

It is known that in patients with the CNS trauma blood levels of catecholamines depend on the time

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TABLE 1. Blood Content of Catecholamine and Cortisol in Patients with CNS Trauma

Group (trauma)	Epinephrine	Norepinephrine	Dopamine	Cortisol (nmol/l)
		pg/ml		
Spinal trauma (1.5-2 months)	913±230 (9)**	290±48.8 (9)	76.2±30.3 (9).	534±73 (5)
CCT (less than 1 week)	270.7±57.3 (8)*	355.6±35.9 (9)*	106±33.5 (8)	326±68 (5)
CCT (2-3 weeks)	108.8±24.6 (9)	170.3±56.4 (9)	39.6±12.9 (9)	179±68 (8)
Control (donors)	131.1±25.3 (9)	225.5±36.4 (9)	97.7±25.8 (9)	444±34 (11)

Note. Number of patients is shown in parentheses. *$p<0.05$, **$p<0.01$ compared with the control.

elapsed after the trauma. Previously, we showed that on day 1 after trauma of spinal column and spinal cord the epinephrine content was several times higher than in healthy subjects and considerably surpassed that in spinal patients at later periods of observation [1]. The same relationships between epinephrine levels were observed in patients with CCT (groups 2 and 3). The epinephrine content returned to normal on the 2nd week after trauma. With normalization of the epinephrine content in CCT patients blood cortisol decreased, the decrease being significant on the 2nd week. Two weeks after trauma, in spinal patients the cortisol level remained increased against the background of high epinephrine level. The role of catecholamines in stress reaction induced by the CNS trauma has been described in detail [1]. In the present study, we evaluated the functional state of the adrenal glands in the reaction to a damaging factor.

It has been demonstrated that the adrenal cortex reacts to stress factors by a rapid rise (within several minutes) of blood cortisol concentration [2]. Changes in blood concentration of corticosteroids (CS) caused by spinal and spinal cord trauma induce biochemical shifts which trigger plastic rearrangements in many systems of the organism. Under conditions of traumatic edema and disturbed blood supply in the cerebral tissue glucocorticoids up-regulate glycolytic processes. Elevation of CS, a family of catabolic hormones, inevitably affects protein synthesis, which in spinal patients usually manifests itself as the loss of muscular mass. Animal experiments showed that the dynamics of blood cortisol concentration depends on the nature of stress factor: it may drop either on day 4 of adaptation [2] or much later, for example, on day 130 [3]. An increase in blood CS concentration leads to changes in monoamines. Corticosteroids act through the CNS or directly by regulating the activity of enzymes involved in the catecholamine metabolism. In any case, the development of stress reaction is regulated by the feedback mechanism, the transmitter mechanisms being triggered via hypothalamic and extrahypothalamic structures. Disorders of brain vascularization and hemorrhages are important pathogenetic factors in CCT. Cerebral trauma may lead to disturbances in vascular innervation, which results in venous congestion and impaired blood drainage from the sinuses. Disturbances in the coordinating vascular mechanisms may interfere with normal sequence of the adaptation process, suppressing its second (glucocorticoid) phase [5]. This agrees with the observation that blood CS level does not always depend on the intensity and duration of stress. In our studies, plasma cortisol content was slightly decreased in patients with CCT (group 2) in comparison with the control. This decrease became statistically significant 2-3 weeks after trauma.

An increase in plasma cortisol concentration after spinal trauma may be indicative of generalized stress reaction. Hyperactivation of nonspecific adaptation mechanisms is known to induce a number of disorders. Within 2-3 months after spinal cord trauma, multiple complications such as pneumonia, cystitis, decubitus, general intoxication, and sepsis may develop in the patients. Long-term hyperactivation of nonspecific mechanisms of adaptation may be one of the causes of these disorders. This hyperactivation is confirmed by elevated blood cortisol and epinephrine levels. Hyperproduction of CS by the adrenal cortex is associated with various disorders, including those in the immune system.

Our study revealed some peculiarities of adaptation mechanisms in patients with brain and spinal cord traumatic disease.

An increase in blood cortisol and epinephrine contents in spinal patients suggests that the disturbances both in the transmitter and hormonal systems persist for 2-3 week after trauma, complicating the treatment of trauma and secondary diseases.

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