STATE OF ADRENAL CORTICAL FUNCTION IN EXPERIMENTAL BLOOD TRANSFUSION COMPLICATIONS

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Experiments on dogs have shown that if heterotransfusion is not accompanied by the development of acute renal failure, the increased intensity of adrenal cortical function is observed only for the first 1-2 days, and the functional reserves of the organ are indistinguishable from those present initially. In acute renal failure with a favorable outcome, the increased concentration of 17-hydroxycorticosteroids in the blood returns to normal on the 4th-5th day and the functional reserves of the adrenal cortex are slightly lower than initially. In acute renal failure with a lethal outcome, the increased level of adrenal cortical function persists until death of the animals. However, loading with ACTH causes no further increase in the level of 17-hydroxycorticosteroids in the blood, indicating maximum stress on the organ and exhaustion of its functional reserves.

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The writers' clinical observations have shown that significant activation of adrenal cortical function takes place in patients with blood-transfusion complications, including acute renal failure [2, 3]. However, the nature of the clinical material is such that this problem cannot be studied in sufficient detail.

It was accordingly decided to investigate the state of adrenal cortical function and the functional reserves of the gland in experimental blood-transfusion complications.

EXPERIMENTAL METHOD

Experiments were carried out on 30 mongrel dogs of both sexes, weighing from 10-20 kg, of which 3 were control and 27 experimental animals. To reproduce blood-transfusion complications with lesions of the kidney, large doses of heterologous blood (human blood, 25-30 ml/kg) were transfused after massive blood loss (25-30 ml/kg) [1, 5]. The blood concentration of 17-hydroxycorticosteroids was determined by the method of Silber and Porter as modified by N. A. Yudaev and Yu. A. Pankov. The state of the functional reserves of the adrenal cortex was judged from changes in the blood level of 17-hydroxycorticosteroids after intravenous injection of 20 units ACTH by Usvatova's method [4]. In addition, the blood urea was determined by Kovarskii's method, the potassium and sodium levels in the blood plasma by flame photometry, the blood alkaline reserves by Nevodov's method, and the daily diuresis was estimated.

EXPERIMENTAL RESULTS

Control observations showed that in intact dogs no significant changes in the blood level of 17-hydroxycorticosteroids took place in the course of three days.

Of the 27 experimental animals four died on the day of heterologous transfusion. The remaining animals were combined into groups based on the outcome of the blood transfusion complications.

In nine dogs (group 1) transient oliguria occurred (down to 610 ml urine per diem), accompanied by intravascular hemolysis without uremia, and by disturbance of the electrolyte composition and the acid-base balance of the blood.

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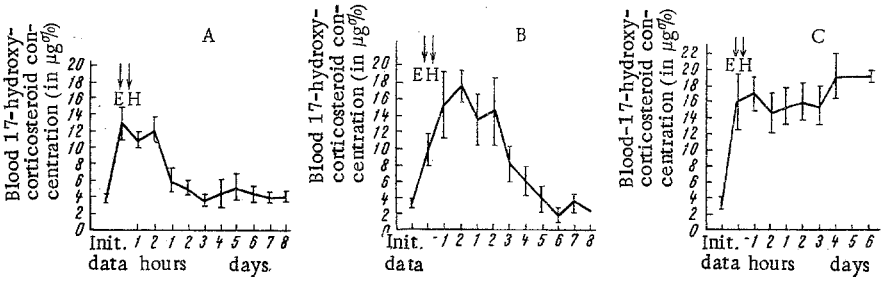

Fig. 1. Dynamics of blood 17-hydroxycorticosteroid concentration in dogs with blood transfusion complications. A) Dogs without acute renal failure; B) dogs surviving after renal failure; C) dogs dying from renal failure; E) exsanguination; H) heterotransfusion.

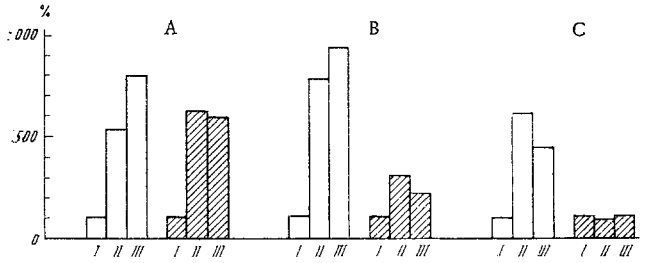

Fig. 2. Functional reserves of adrenal cortex in experimental blood transfusion complications (initial blood concentration of 17-hydroxycorticosteroids in animals before ACTH loading taken as 100%). Unshaded columns, before; shaded columns, after transfusion of heterologous blood; I) before; II) 1.5 h after; III) 4 h after injection of ACTH. Remainder of legend as in Fig. 1.

In five animals (group 2) marked oliguria was present (down to 250 ml urine per diem), accompanied by uremia (blood urea 122 mg%), an increase in the blood potassium level (17.8 mg%), and a decrease in the alkaline reserve of the blood to 353 mg%, i.e., by the development of acute renal failure. However, on the 4th-5th day the diuresis of these dogs began to recover, the blood urea fell, and the alkaline reserve of the blood returned to normal. All the animals of this group survived.

In nine dogs (group 3) oliguria developed, followed by anuria, the blood urea concentration rose sharply (to 389 mg%), as also did the plasma potassium concentration (to 25.3 mg%), the plasma sodium concentration fell to 292 mg%, and a considerable decrease in the blood alkaline reserve was observed (to 280 mg%). The severe acute renal failure which developed caused death of all the animals within a week.

The blood level of 17-hydroxycorticosteroids in the dogs of all three groups immediately after blood loss and 1-2 h after heterotransfusion rose sharply (Fig. 1). In the animals of group 1 the blood level of 17-hydroxycorticosteroids returned to its initial value on the 3rd day. In the animals of groups 2 and 3, developing acute renal failure, the subsequent changes in the blood 17-hydroxycorticosteroid level depended on the outcome of the developing acute renal failure.

In acute renal failure terminating in recovery (Fig. 1B), a high level of 17-hydroxycorticosteroids in the blood occurred during four days. The normalization of these indices took place parallel to the improvement in the state of the animals, the decrease in the blood urea, and normalization of the blood electrolyte composition and acid-base balance.

In animals dying subsequently from acute renal failure, a high blood 17-hydroxycorticosteroid level was observed until death (Fig. 1C). Parallel with this, the general condition of the dog deteriorated, the diuresis diminished sometimes to anuria, the blood urea and potassium increased, and the blood alkaline reserve decreased.

To obtain a more complete picture of the state of adrenal cortical function in the animals of all three groups, the functional reserves of this organ were investigated before heterotransfusion and on the 3rd-4th day thereafter (Fig. 2).

ACTH loading in the animals of group 1 revealed the existence of functional reserves of the adrenal cortex, which did not differ substantially from those before heterotransfusion (Fig. 2A).

In the dogs of group 2 the functional reserves of the adrenal cortex were preserved, but were quantitatively less than initially (Fig. 2B). In the initial state, for instance, the level of 17-hydroxycorticosteroids in the animals after ACTH loading was increased by 6-8 times after 1.5 and 4 h. Injection of the same doses of ACTH in the period of acute renal failure caused an increase in the blood concentration of 17-hydroxycorticosteroids by only 2-3 times.

In the animals of group 3, ACTH loading at the height of uremia (on the 3rd-4th day after heterotransfusion) was not followed by elevation of the blood 17-hydroxycorticosteroid level (Fig. 2C), indicating maximal intensity of adrenal cortical function and exhaustion of the functional reserves of the gland. After injection of the same dose of ACTH into animals in the initial state the blood 17-hydroxycorticosteroid level increased by 4.3-6 times.

The results of these experiments show that in the course of blood transfusion complications, including acute renal failure, the functions of the adrenal cortex are intensified.

LITERATURE CITED

1. B. A. Agranenko and R. V. Nedoshivina, Pat. Fiziol., No. 1, 71 (1963).
2. R. M. Glants, Vrach.Delo, No. 9, 921 (1956).
3. R. M. Glants and G. P. Vovk, Urol. i Nefrol., No. 6, 23 (1969).
4. I. Ya. Usvatova, in: Investigation of the Functional State of the Adrenal Cortex and Sympatho-Adrenal System Under Clinical and Experimental Conditions [in Russian], Moscow (1963), p. 58.
5. M. Atik, B. Manale, and J. Pearson, J. Amer. Med. Assn., 183, 455 (1963).