THE EFFECT OF THE ADRENALS ON SHOCK

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Recently a great deal of attention has been paid to the effect of disturbed adrenocortical function in the development of shock [1,4]. It is known that when adrenocortical function is impaired the degree of shock required to elicit trauma is reduced, and the shock is more severe. However,there is need for further information concerning the separate adrenocortical functions.

The aim of the present study has been to determine the influence of changes in the mineral and glucocorticoid adrenal components on the course of shock.

We used autografted adrenals, because from experiments which we carried out previously with D. Ya. Shurygin [3], it was found that after transplanting the adrenals, mineral corticoid function was restored within two weeks, whereas glucocorticoid function did not recover for two months. Therefore, during the first month after autografting, the two principal adrenocortical functions are dissociated.

METHOD

The experiments were carried out on 30 rats weighing 150–200 g. The adrenals were isolated, and then transplanted into the region of the superior pole of the kidneys. Shock was induced by applying special clamps for four hours to compress the soft tissue of both thighs. A study was made of the behavior of the animals, their survival time, body and rectal temperatures, the condition of the blood (red cells, leucocytes and number of eosinophils, by Dunger's method). Tests were made before applying the clamps, one hour after their application, and immediately after their removal. Three experiments were performed: 1) the control; 2) one which was carried out 12 days after autografting; and 3) a third carried out 30 days after autografting.

RESULTS

In the control group, only one rat died, 10 days after the trauma had been inflicted. Rats traumatized 12 days after the adrenals had been autografted developed severe shock and died within two days. Of the ten rats which were traumatized 30 days after autografting, three survived, and the remainder died within two days. The resistance of the autografted animals was therefore reduced, particularly in the group which were traumatized 12 days after operation. It had been shown previously that the laparotomy by itself increases resistance to trauma.

The reduced resistance must therefore be due to an impairment of adrenocortical function resulting from the autografting.

Twelve days after autografting, the wound from the operation had healed completely. The animals could be distinguished from the control group only by their behavior. They showed lassitude and reduced responses to external stimuli; these stimuli included reapplication of the clamps.

In the autografted animals, as can be seen from the table, after the trauma had been inflicted there was a marked hypothermia, particularly in the group in which the trauma was inflicted 12 days after the autografting. The difference between the control group and the normal was highly significant.

By itself, autografting caused an increase in the number of erythrocytes, evidently as a result of the blood becoming thicker. After trauma, the number in the autografted animals rose to 8,000,000 or more per mm^3.

In the control group, in many cases a leucocytosis developed after the trauma, but sometimes, on the contrary, the number of leucocytes was reduced. The change depended on the original number. Where it was originally

Changes in Body Temperature and in the Blood after Trauma in Animals with Transplanted Adrenals and in a Control Group (mean figures)

Quantity measured	Group of animals	Before traumatizing	While clamps were applied	After removing clamps
Temperature	Control	37.2± 0.70°	35.2± 1.2°	34.8± 1.3°
	Autografted, trauma after 12 days	36.6± 0.50°	29.6± 2.9°	27.7± 1.9°
	Autografted, trauma after 30 days	38.1± 0.54°	34.3± 1.1°	31.6± 1.1°
Number of erythrocytes (in millions)	Control	6.67± 0.81	6.98± 1.10	7.46± 1.17
	Autografted, trauma after 12 days	7.64± 1.30	7.75± 1.00	8.44± 1.20
	Autografted, trauma after 30 days	7.50± 0.80	8.80± 1.07	8.37± 0.87
Number of leucocytes (in thousands)	Control	18.7± 8.3	17.4± 5.4	17.2± 5.9
	Autografted, trauma after 12 days	24.5± 7.9	34.5± 9.4	25.6± 5.8
	Autografted, trauma after 30 days	18.5± 3.6	37.6± 13.6	37.3± 10.3
Number of eosinophils (median)	Control	55	44	0
	Autografted, trauma after 12 days	1240	980	460
	Autografted, trauma after 30 days	220	1890	198

relatively high, it was reduced after trauma, and vice versa. In the autografted animals, independently of what the original level was, in all the experiments there was a marked leucocytosis and the number of white cells increased to 25,000–37,000 per mm^3. The increase was best shown in the animals in which the trauma was inflicted 30 days after autografting.

In the autografted animals, the initial number of eosinophils was considerably higher than in the control group, showing that the trauma had not caused any marked reduction in their number. In the control animals, after the clamps had been removed, the eosinophils dissappeared almost entirely from the blood. Apparently, in the animals with transplanted adrenals, the adrenal cortex is functionally deficient.

The facts described above show that during the month following autografting, despite the structural recovery of the adrenals [3], the animals still showed a reduced resistance to trauma, on account of functional adrenal cortical insufficiency. Indeed, in these animals the trauma did not produce such a marked drop in the number of eosinophils as it did in the control experiments.

As we showed in our work with D. Ya. Shurygin [3], there had been an almost complete restoration of the mineral corticoid function of the adrenal cortex by the time the trauma had been inflicted, as was shown by the return of the salt content of the urine and plasma to normal. The reduced resistance to trauma must therefore be associated with insufficiency of the glucocorticoid adrenocortical function. The same conclusion is indicated by results obtained in experiments on the effect on shock of mineral and glucocorticoids. In practice it was found that treatment of shock with deoxycorticosterone acetate is without effect, whereas the hypophyseal adrenocorticotropic hormone and cortisone have a markedly favorable effect [2].

It should be noted that in animals with transplanted adrenals, during shock there is a marked hypothermia, a thickening of the blood, and an intense leucocytosis.

SUMMARY

The effect of mineral and glucocorticoid adrenocortical function on the pathology of trauma was studied in rats with autotransplanted glands.

Studies were made of behavior, survival time, temperature and blood.

It was found that during the first month after autografting, despite the recovery of the cortical structure of the

adrenals, the resistance of the animals to trauma was reduced; this effect may be attributed to adrenocortical glucosteroid failure.

The shocked autografted animals also showed pronounced hypothermia, a thickening of the blood, and an intense leucocytosis.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
