EFFECT OF PITUITARY SOMATOTROPIC HORMONE ON ADRENAL FUNCTION IN RABBITS TRANSFUSED WITH DEXTRAN AFTER ACUTE BLOOD LOSS

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Dextran transfusion of rabbits after acute blood loss stimulates adrenocortical function. This activation is more marked if the animals are injected with growth hormone.

The writers have shown previously that injection of STH into rabbits after acute blood loss stimulates restoration of the serum proteins after dextran transfusion. STH stimulated metabolism, including that of the polysaccharide dextran, stimulated protein synthesis in the animal, and thus accelerated the repair processes after blood loss.

The problem of the role of the adrenals in the stimulant action of STH on repair processes after blood loss has arisen, for the adrenal hormones participate directly in these processes [1, 6, 7, 9].

EXPERIMENTAL METHOD

Rabbits weighing 3-3.5 kg were bled without anesthesia by introduction of a waxed cannula into the femoral artery until the blood ceased to flow. The mean blood loss was 20 ml/kg body weight. Immediately after bleeding a rapid transfusion of dextran was given into the femoral vein in a volume equal to that of the blood lost. The rabbits were then divided into two groups with ten animals in each group: group 1 was the control, while the rabbits of group 2 received daily injections of STH in a dose of 0.5 mg/kg body weight for 6 days. The animals were decapitated on the 6th-7th day. Intact rabbits were used as the control for group 1. The weight of the right and left adrenals and their ascorbic acid content were determined in the animals of all groups.

EXPERIMENTAL RESULTS

Dextran is widely used in clinical practice as a blood substitute. The physiological mechanisms of its action have been studied in detail by Koziner [2]. However, the role of the adrenals in the mechanism of action of dextran has not been investigated.

The study of the change in weight of the adrenals gave the following results. In intact rabbits the weight of the left adrenal was 104 ± 7.3 mg and of the right 103 ± 8.1 mg, in the rabbits after blood replacement by dextran the weight of the left adrenal was 153 ± 14 mg ($P < 0.01$) and of the right 132 ± 9.7 mg ($P < 0.05$), while in the animals after blood replacement by dextran and injections of STH the left adrenal weighed 154 ± 11.2 mg ($P < 0.01$) and the right 147 ± 9.4 mg ($P < 0.01$). Dextran transfusion in rabbits with acute blood loss thus led to an increase in the weight of the adrenals by more than 50% ($P < 0.01$) in comparison with their weight in intact rabbits, while after administration of STH the increase in weight of the glands was somewhat smaller.

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The ascorbic acid content in the intact rabbits was 341 ± 13 mg%, after acute blood loss it was 312 ± 9.6 mg% ($P > 0.05$*), while after dextran transfusion it was 287 ± 17 mg% ($P < 0.02$, $P_1 < 0.05$). In the rabbits receiving daily injections of STH the ascorbic acid content in the adrenals fell still more, on the average to 158 ± 22 mg% ($P < 0.001$, $P_1 < 0.001$). A decrease in the ascorbic acid content of the adrenals has been shown to reflect the increased formation of corticosteroid hormones in the adrenals [4, 8]. The work of Chattopadhyay et al. [5] has shown that in rats with spontaneous hemorrhage a decrease in the ascorbic acid content of the adrenals took place simultaneously with an increase in the corticosterone concentration in the blood serum.

Dextran transfusion after acute blood loss is evidently followed by increased adrenal function and the more rapid formation of corticosteroid hormones. It can accordingly be assumed that increased secretion of adrenocortical hormones play an important role in the mechanism of the physiological action of dextran in acute blood loss.

Besides stimulating metabolism, including the metabolism of dextran, and besides stimulating protein synthesis (as the writers' previous investigation showed), administration of STH also stimulates adrenocortical function. This must have a definite role in the activation of repair processes after blood loss. Retention of sodium and water in the body after blood replacement and administration of STH may be connected with increased secretion of aldosterone. This is shown by results indicating intensification of the mineralocorticoid function of the adrenals after STH administration [6, 7]. As the work of Palmore et al. [9] has shown STH inhibits the elimination of sodium and water from the circulating blood, a matter of great importance for maintenance of the circulating blood volume after blood replacement. Injection of STH after acute blood loss thus has a multiple action aimed at restoring the circulating blood volume. This action is manifested by stimulation of protein synthesis, leading to rapid restoration of the plasma proteins, and also by retention of sodium and water; this last effect of STH may be either direct or indirect, brought about through activation of adrenocortical function.

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*Here and subsequently: P is the criterion of significance of differences between the experimental and intact rabbits, P_1 the same between that group and the preceding group of animals.