

THE REACTION TO BLOOD LOSS OF VARYING DEGREE DURING EXPERIMENTAL NONINFECTIOUS PERITONITIS

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Inflammation of the peritoneum occupies an important place in clinical practice; it is especially common to observe a combination of peritonitis with blood loss. The complex of defensive and adaptive reactions, developing in response both to peritonitis and to blood loss, occasionally run their course against a background of modification of reactivity caused by each of these processes.

This provides an explanation of the influence of blood loss on the course of inflammatory processes, which is known from clinical and experimental observations [1-5].

In the present research we attempted to examine the development of certain compensatory defensive reactions in peritonitis complicated by blood loss.

EXPERIMENTAL METHOD

Experiments were carried out on 50 rabbits, which were divided into five groups, with ten animals in each group.

The first group consisted of healthy animals, and were controls; the second group, of rabbits with peritonitis but without blood loss; the third, of rabbits with peritonitis and with loss of 5% of blood; the fourth, of rabbits with peritonitis and a 10 % blood loss, and the fifth, of rabbits with peritonitis and a 15 % blood loss. Before the experiment the animals were kept for two days under identical conditions and on the same diet. In order to produce peritonitis, under aseptic conditions the rabbits were given an intraperitoneal injection of a 20 % peptone solution, in a dose of 4 ml/kg body weight. In the experiments with bleeding, the blood was taken from the chamber of the heart, and the total blood mass was taken to be one thirteenth of the body weight. Indications of the development of peritonitis were the body temperature, leukocytosis in the blood, the presence of an exudate in the abdominal cavity and, finally, pathohistological examination of the peritoneum.

The consequences of blood loss were assessed by the decrease in the hemoglobin concentration of the blood and in the red cell count.

The animals thus prepared were examined repeatedly for one or two days; they were then subjected to the acute experiment, in the course of which we studied the reaction of the animals to the intravenous injection of the following substances: 1) a 20 % peptone solution; 2) transudate taken from patients with cirrhosis of the liver; 3) exudate taken from patients with exudative pleurisy, and 4) the rabbits' own exudate. All these substances were injected in a dose of 0.5 ml/kg body weight. Changes in the blood pressure were recorded on paper on a kymograph.

EXPERIMENTAL RESULTS

The experimental results are shown in the table.

It may be seen from the results in the table that, in the animals in which peritonitis was accompanied by blood loss, the febrile reaction was weakened in proportion to the degree of blood loss. The leukocyte reaction was stimulated by moderate blood loss, but loss of a larger volume of blood (15 %) did not have this

Indices in All Groups of Experimental Animals
(mean figures)

Group	Body temperature	Leukocyte count in the blood (thousands/mm ³)	Red cell count in the blood (thousands/mm ³)	Hemoglobin (Sahli units)	Initial systolic pressure (in mm Hg)
First	38.3°	8.1	4 546	75	93
Second	39.5° (+1.2°)	15.0 (+6.9)	3 758 (-788)	63 (-12)	90 (-3)
Third	39° (+0.7°)	18.9 (+10.8)	3 078 (-1468)	61 (-14)	77 (-16)
Fourth	38.7° (+0.4°)	16.7 (+8.6)	3 051 (-1495)	60 (-15)	74 (-22)
Fifth	38° (-0.3°)	14.3 (+6.2)	2 954 (-1 592)	58 (-17)	73 (-20)

effect. Analysis of the kymograms obtained showed that, in the group of control animals, the injection of peptone lowered the systolic pressure; the same phenomenon, but to a less pronounced degree, was observed after injection of the transudate and exudate from the patients, and injection of the rabbit exudate led to the greatest fall in pressure. In the rabbits with peritonitis but without blood loss, these reactions were less pronounced; in animals with peritonitis and blood loss a greater fall in pressure—both the initial pressure and that obtained in response to the injection of the various substances—was observed. The development of peritonitis evidently prevented the creation of compensatory pressor mechanisms, for the peritonitis itself caused no essential fall in pressure.

SUMMARY

An investigation was made into the development of some protective-compensatory reactions in peritonitis

complicated by blood loss of various degrees (5, 10, and 15 % of the total mass). The febrile reaction in non-infectious peritonitis was reduced by blood loss. A slight blood loss stimulated leukocytic reaction; the latter was not observed in response to considerable bleeding. The pressor reactions in peritonitis were weakened by blood loss; this was especially noticeable when certain hypotensive substances (20 % peptone solution, transudate and exudate obtained from the patients, and exudate of rabbits) were injected intravenously.

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