ROLE OF LOCAL BLOOD AND PLASMA LOSS IN THE PATHOGENESIS OF HYPOTENSION IN TRAUMATIC SHOCK

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In traumatic shock the blood and plasma loss in the injured limb does not exceed on the average 2% of the body weight, and its maximum coincides with the erectile stage of shock. Loss of blood equivalent to 2% of the body weight is not accompanied by the development of a serious state or by marked hypotension; only transient dyspnea and tachycardia are observed. The results of the investigation indicate that local blood and plasma loss is not the main pathogenetic factor in the mechanism of the primary hypotension accompanying traumatic shock.

Despite the abundant literature on traumatic shock, there is still no general agreement regarding the role of local blood and plasma loss in its pathogenesis. Some workers [1, 2] see no significant difference between acute massive blood loss and severe traumatic shock. These mistaken conclusions are in part due to similarity between the courses of these essentially different pathological processes.

In this investigation an attempt was made to examine the role of local blood and plasma loss in the pathogenesis of primary hypotension in experimental traumatic shock in dogs.

EXPERIMENTAL METHOD

Altogether, 21 experiments were carried out on sexually mature male dogs weighing 16-24 kg. The experiments were divided into three series. In series I severe trauma was inflicted on the animals without, however, causing the development of hypotension (eight dogs). In series II severe traumatic shock with hypotension (mean blood pressure 62 mm Hg) was produced in seven dogs. In the experiments of series III (six dogs) bleeding was carried out in a volume equivalent to 2% of the body weight over the same period of time as was occupied by the trauma in the experiments with severe shock (3 min).

Traumatic shock was produced by Cannon's method: acute blood loss by repeated small bleedings from the right femoral artery. Traumatization of the dogs in the experiments of series I continued until the sharp rises of blood pressure (BP) disappeared and it was stabilized at the normal level (124 ± 3 mm). By this time the animals' vocal response had also subsided, indicating the end of the erectile phase of shock and the beginning of its transition into the torpid phase. Traumatization of the animals in the experiments of series II continued until the development of a general serious state and of persistent hypotension. The BP in the left femoral artery was recorded on a kymograph.

In the experiments of series I and II the animals were killed 10 min after trauma by intravenous injection of potassium chloride solution; the hind limbs were immediately amputated and weighted by the method developed by O. S. Nasonkin in the authors' department. An incision was made over Poupart's ligaments symmetrically on both sides, and the hip joints were disarticulated. The difference in weight between

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TABLE 1. Blood Pressure and Magnitude of Trauma and Local Blood and Plasma Loss (M ± m)

Index studied	Expts. of series I (eight dogs)	Expts. of series of II (seven dogs)
Ny	124±28	237±54
BP_{10}	124±3	62±5
$\Delta P\%$	2,06±0,24	2,01±0,27
Coeff. of correlation between BP_{10} and $\Delta P\%$		
BP_{10} and $\Delta P\%$	0,867	0,674
BP_{10} and Ny	0,870	0,330
$P\%$ and Ny	0,892	—0,115

the traumatized and intact limbs, expressed as a percentage of body weight, was used as the index of the degree of local blood and plasma loss. Special experiments showed that when this method was used to amputate the dogs' hind limbs the experimental error was very small, not more than 25-30 g.

The chief characteristics of the severity of the process used in these experiments were the value of BP 10 min after trauma and blood loss (BP_{10}) and the magnitude of the trauma itself (Ny).

EXPERIMENTAL RESULTS AND DISCUSSION

In the original state the animals' BP varied within wide limits (126-200 mm Hg) with a mean value of 158 ± 10 mm (M ± m).

In the initial period of trauma the state of general excitation of the animals was accompanied by a sharp rise of BP (on the average to 204 ± 23 mm Hg). Soon after, while trauma continued, the BP fell slightly and became stabilized at the level of 124 ± 3 mm Hg, after which the vocal response quickly disappeared. At this stage the trauma to the dogs in series I was discontinued. In series II, in which the trauma was continued, the BP began to fall rapidly after 1-2 min, reaching 62 ± 5 mm Hg.

The magnitude of the trauma in the animals which developed marked hypotension averaged 124 blows, whereas in the dogs with severe shock it averaged 237 blows.

Analysis of the results showed that the magnitude of the local blood and plasma loss in experiments with trauma of different degrees of severity did not differ significantly ($P < 0.05$) and it averaged 2% of the body weight. Meanwhile, bleeding by an amount of 2% of the body weight did not cause any serious disturbances of the bodily functions of the dogs; only transient dyspnea and tachycardia were observed, and the BP showed only a certain tendency (also temporary) to fall a little – by 20-30 mm Hg ($P > 0.05$). After the acute experiment, all the animals in the experiments of series III were in good condition and were kept in the animal house. Next day these dogs were outwardly indistinguishable from healthy animals. Meanwhile the animals with severe shock, having undergone blood and plasma loss of the same magnitude but purely locally, usually did not survive longer than 3-4 h.

Statistical analysis (Table 1) showed no significant correlation between the change in the magnitude of the local blood and plasma loss ($\Delta P\%$) and the severity of the shock as assessed by the value of BP_{10}, in the experiments of series II. However, in the experiments with less severe trauma, direct correlation was found between the magnitude of trauma and $\Delta P\%$. The greatest blood and plasma losses are thus observed in the first stage of trauma. These results suggest that the specific character of traumatic shock cannot be reduced simply to local blood and plasma loss as the decisive factor in the mechanism of primary hypotension.

LITERATURE CITED

1. A. Blalock, Arch. Surg., 15, 762 (1927).
2. A. Blalock, Arch. Surg., 20, 959 (1930).