CHANGES IN THE MICROCIRCULATION IN THE RAT MESENTERY AFTER REPEATED SKIN BURNS

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Acute experiments on rats showed that repeated extensive burns inflicted on animals convalescing from previous burns are tolerated better, do not cause death, and are accompanied by moderate changes in the microcirculation. Similar burns in intact rats lead to severe, lethal shock, intravascular aggregation of erythrocytes, and substantial disturbances of the microcirculation, so that tissue nutrition is impaired. It is postulated that these results could be indirect evidence that toxemia plays an essential role in the genesis of intravascul aggregation of erythrocytes in burns.

KEY WORDS: burn shock; toxemia; microcirculation.

Repeated experimental burns in animals are known to be tolerated better than the original burn: the mortality falls significantly [3, 7, 10], the functions of the heart, liver, and kidneys are less severely disturbed [3, 6, 7], and the toxemic manifestations are less marked; these differences are attributed to the existence of active antiburn antitoxic immunity in these animals [8, 10].

It might be expected that after repeated burns the other functional disturbances and injuries due to toxemia would also be less well marked. It was postulated previously that intravascular aggregation of erythrocytes and other disturbances of the microcirculation observed in the acute phase of burns are due mainly to changes in the relative proportions of the plasma proteins and to the toxemia which follows burns [9, 12, 13].

The object of this investigation was to test this hypothesis.

EXPERIMENTAL METHOD

Experiments were carried out on August rats weighing 200-280 g. The clinical state and microcirculation were compared in the animals of two groups. Group 1 consisted of 28 rats, investigated 24 h after a primary fourth-degree burn covering 25-35% of the body surface (on the back and the sides of the trunk); group 2 consisted of eight rats investigated 24 h after a similar burn had been inflicted during convalescence after a less severe skin burn inflicted 2-4 months before the experiment. The control group consisted of 20 healthy rats of the same strain and age. A burn of the assigned degree of depth of tissue injury was obtained by exposure of the shaved skin for an empirically determined time (45 sec) to a spirit flame, to produce the necessary heating of the skin, subcutaneous cellular tissue, and muscles. The temperature of heating was monitored during the burning by means of a contact electrothermometer. The depth and degree of injury to the burned tissues were additionally verified later at autopsy. The burn trauma was inflicted on unanesthetized animals. Observation on the photographic recording to the microcirculation in the mesentery were carried out under urethane anesthesia on an apparatus for intravital biomicroscopy, in accordance with the method described previously [9]. Each animal was used for a single observation.

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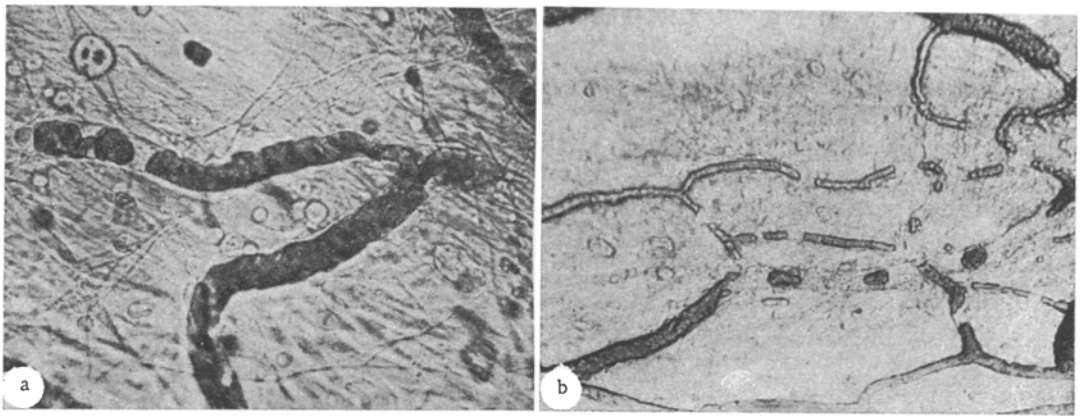

Fig. 1. Photomicrographs of vascular network of mesentery of a rat in the terminal phase of burn shock (24 h after an extensive skin burn): a) aggregates of erythrocytes and stasis in venules, 520 ×; b) stasis, 260 ×. Explanation in text.

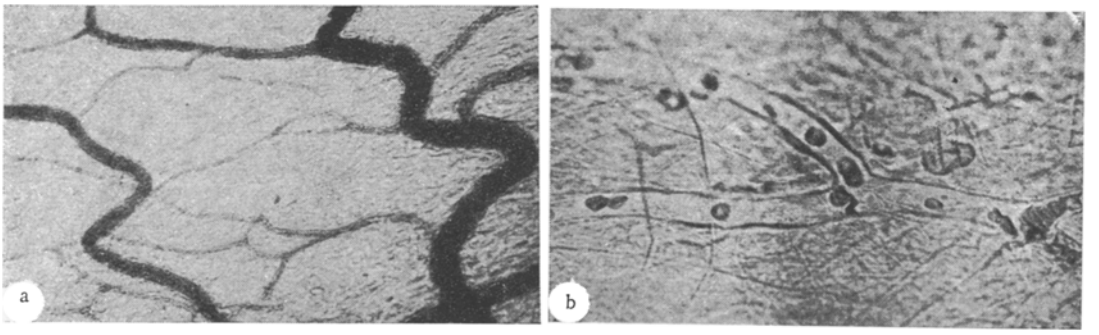

Fig. 2. Photomicrographs of vascular network of mesentery of a rat 24 h after extensive repeated burn inflicted during convalescence: a) network of microvessels with slight disturbances of structure of blood flow, 260 ×; b) venules with rarefied structure of blood flow and small, unstable aggregates, 520 ×. Explanation in text.

EXPERIMENTAL RESULTS AND DISCUSSION

All the animals of group 1 developed severe shock immediately after the burn and died in the course of 2-3 days; half of the rats died during the first day. The animals still surviving after 24 h, whose microcirculation was observed, were lethargic, their defensive reflex was weak, and they took no food or water. Most of them were in the terminal phase of shock.

The state of the animals in group 2 was characterized by a milder and, as a rule, brief period of shock which did not end in death. Usually the defensive reflex, aggressiveness, and conditioned-reflex movements of the animals were fully restored after 1-3 h; these animals took food and water.

The severe burn shock in the animals of group 1 was accompanied by marked changes in the microcirculation observable 30-60 min after trauma. The pattern of the microcirculation after 24 h in all these animals was determined by generalized aggregation of the blood cells, extensive areas of stasis, and a disturbed structure of the blood flow where it still continued (Fig. 1a). The number of functioning capillaries was greatly reduced, the blood flow was directed chiefly, not along the usual channels (arteries—arterioles—true capillaries—venules—collector veins), but along arteriovenous anastomoses, bypassing the empty capillaries. Despite the considerable plasma loss and general hemoconcentration [2], no appreciable clotting of blood in the functioning vessels of the terminal vascular network could be observed. On the contrary, the impression was created of a "rarefied" blood flow in the region of the microcirculation with a large number of almost purely "plasmatic" vessels, among which only plasma with a very few cells flowed. A few vessels apparently stuffed with erythrocytes were the exception. In these vessels movement was either to-and-fro or absent altogether, i.e., complete erythrostasis was observed. After prolonged stasis, the erythrocytes in these vessels were compressed into a solid, amorphous mass (Fig. 1b). As a result of all these events the nutrition of the tissues was disturbed and they

developed severe hypoxia [17]. Evidently as a result of the hypoxia the tissue of the mesentery itself was altered at this time. It appeared thickened, coarser, and less translucent than in the control rats or during the first few hours after burning.

In the animals of group 2 the picture of the microcirculation was very varied. Although there were far fewer functioning capillaries and the network of anastomoses was thicker than normally, the gross disturbances of the microcirculation found in group 1 were not observed. In many fields of vision the microcirculation differed only a little from that in the normal animals (Fig. 2a). However, in most cases some slowing of the blood flow and disturbances of its structure, such as partial plasmatization or, on the other hand, a paste-like movement of the thickened blood were observed, especially in the veins. However, aggregation of erythrocytes was found in by no means all the animals of this group and, moreover, the aggregates were not the large spheroidal or shapeless clumps found in the animals with severe shock, but were mainly in the form of fragile "rouleaux" (Fig. 2b). On the whole the state of the microcirculation in the mesentery of the rats after repeated burning resembled the picture described previously [9] and observed after less severe burns unaccompanied by shock.

The results of these experiments show that repeated burns inflicted on convalescent rats are tolerated better by the animals, they do not lead to severe shock terminating in death, and they are accompanied by only moderate changes in the microcirculation without any generalized, irreversible intravascular aggregation of the blood cells. A similar primary burn in intact rats is accompanied by severe shock, terminating in death, by intravascular aggregation of erythrocytes, and by substantial disturbances of the microcirculation leading to impairment of nutrition and hypoxia of the tissues. This conclusion is confirmed by earlier observations [9] showing that the state of the microocirculation corresponds to the severity of the course of burns in the acute phase and may perhaps be a determining factor of it. Meanwhile the severity of the course of burns is known to depend largely on the degree of toxemia [4, 7, 15]. Since burn convalescent serum is known to have specific antibodies against burn toxin [5, 7, 11, 14, 16] and since repeated burns inflicted on convalescents are accompanied by less severe toxemia [1, 6], the results of the present experiments may serve as indirect proof that the intravascular aggregation of erythrocytes and other disturbances of the microcirculation in burn shock may be attributable to burn toxemia. However, this claim needs to be supported by additional and, as far as possible, more direct evidence.

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