CHIEF FEATURES OF THE MICROCIRCULATION IN THE LIVER IN TRAUMATIC SHOCK

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The microcirculation in the liver was studied in rats with experimental traumatic shock by intravital microscopy and transillumination. In the initial periods of shock (the erectile phase) restriction of the circulation in both the arterial and venous systems was found. In deep shock the hepatic sinusoids were congested with blood, the blood flow in the portal vein was severely disturbed, while that in the hepatic artery was also disturbed but to a somewhat lesser degree, and shunting of the blood flow was observed. Aggregation of the blood cells developed in the hepatic vessels in shock.

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The only description of the microcirculation in the rat liver during shock produced by crushing of the limbs is that given by Senevirente [13]. Other studies have dealt with shock resulting from blood loss and operation trauma [12], anaphylactic shock [8], and clinical death from blood loss [1, 2]. In the present investigation some of the features of the liver microcirculation in traumatic shock were studied.

EXPERIMENTAL METHOD

Experiments to investigate the microcirculation in the liver by intravital microscopy were carried out on 33 rats lightly anesthetized with thiopental (10-15 mg/100 g body weight).

Shock was produced by traumatizing the soft tissues of the thigh with a small hammer weighing 130 g (180-250 blows were applied in the course of 1.5-2 min). The severity of the shock was estimated from the arterial pressure in the carotid artery and changes in respiration. Polyethylene catheters were introduced into the carotid artery and femoral vein, and in the course of the experiments a suspension of ink was injected into the latter.

Intravital microscopy of the liver was carried out by means of the MBS-1 microscope after laparotomy. To transilluminate the border of the liver, as well as quartz light conductors recommended in the literature [7, 9, 10], conical light conductors made of methyl methacrylate, 500 μ in length, 30 mm in diameter at the end nearest to the source of light, and 2 mm in diameter at the end placed beneath the test object, were used. The light conductors were mounted en bloc with the source of light (a 30-50 W incandescent lamp or an ultraviolet source). During the observations the liver was continuously irrigated with Ringer's solution warmed to 38°.

In the course of the observations the hepatic vessels were measured from time to time with an ocular micrometer, and drawn or photographed with a "Zenit-3m" miniature camera. As a rule the magnification used in the investigation was 56×. A general impression of the microcirculation was obtained from observations conducted with a magnification of 32×, and the details were examined at a magnification of 122×.

EXPERIMENTAL RESULTS

Traumatic shock in the rats was characterized by a distinctive motor response, a cry, and elevation of the arterial pressure in the erectile phase, which was of very short duration. Later arterial hypotension quickly developed, the pulse became stabilized, the respiration rate gradually fell, the animal ceased to react to weak or even strong stimuli, and the torpid phase of shock developed. As the shock became more severe the arterial hypotension progressed, and respiration became slow and sometimes periodic (Table 1).

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TABLE 1. Arterial Pressure, Pulse Rate, and Respiration at Various Periods of Traumatic Shock in Rats (M ± m)

Index	Before shock	Shock			
		Torpid phase			Terminal phase
		Beginning	Period of stabilization	End	
Arterial pressure (in mm Hg)	117 ± 3.1	74 ± 9.6	87 ± 7.0	44 ± 2.9	24 ± 2.5
Pulse rate (per min)	192 ± 8.1	202 ± 3.5	208 ± 6.8	180 ± 13.8	Not determined
Respiration (per min)	83 ± 9.8	84 ± 11.2	68 ± 7.5	25 ± 3.3	Agonal state

The mean duration of shock (until the terminal phase) was 60 min. Sometimes chronic forms of shock lasting up to 240 min developed, but in other cases fulminating forms developed, the terminal phase arising after 5-15 min. As a rule the shock was lethal.

Traumatic shock in rats in the experiments described was similar in its chief features to that in other animals, so that the corresponding phases and periods could be distinguished in its course [5].

Microscopic study of the hepatic vessels showed that the outlines of the hepatic lobules are largely dependent on the state of function of the vessels: the lobules themselves have no sharp borders and they are identified from the integral pattern of their blood vessels. The hepatic venules were particularly clearly distinguishable. The blood flow in them was usually so rapid that it was difficult to identify the cells. The portal venules were less clearly outlined.

The hepatic capillaries (sinusoids) appeared as delicate, thin vessels, converging on the central venules, with a comparatively slow blood flow. Usually not all the sinusoids were functioning, but only some of them, although within 2-5 sec the blood flow in some sinusoids stopped and restarted in others, previously in an inactive state.

Hepatic arterioles were observed extremely rarely as thin vessels close to the portal venules. The results of our observations of the normal microcirculation in the liver were similar with those described previously [1, 2, 7, 13].

Immediately after trauma, pallor of the liver was observed, as a result of marked contraction of the sinusoids. Many of them lost their clear outline and became interrupted. The portal and hepatic venules were constricted.

With the development of the torpid phase of shock, characterized by the symptoms described previously, the blood flow in both the portal and hepatic venules was activated. The lumen of these vessels increased. The sinusoids were filled with blood and they became more numerous than in the initial state. Hepatic arterioles began to be visible more frequently than is usual.

With a further increase in the severity of shock the sinusoids became increasingly congested. The blood flow in them gradually became slower, and in some sinusoids it stopped altogether. Movement of blood in the hepatic and, in particular, the portal venules became sluggish. The cells became ill-defined, not because of the rapidity of the blood flow, but because of homogenization and concentration of the blood. Agglomeration of blood cells was observed in the vessels.

Gradually, as the period of stabilization of the torpid phase grew near, the outlines of the blood vessels, especially the sinusoids and portal venules, became indistinct, and their outlines were hard to make out against the general dark red background. Disappearance of the sharp outlines of the blood vessels can be regarded as the result of filling of the perisinusoidal spaces of Desse with fluid and the formation of aggregates of erythrocytes in the vessels themselves.

Injection of a suspension of India ink into the arterial system (sometimes necessary in order to determine the type of blood vessel) under normal conditions led to the rapid appearance of ink particles in the sinusoids, to their passage through the sinusoids, and to the accumulation of a certain number of ink particles near their initial segments. In shock ink particles did not appear in the sinusoids but were found in the branches of the hepatic vein, having reached them apparently through shunt vessels.

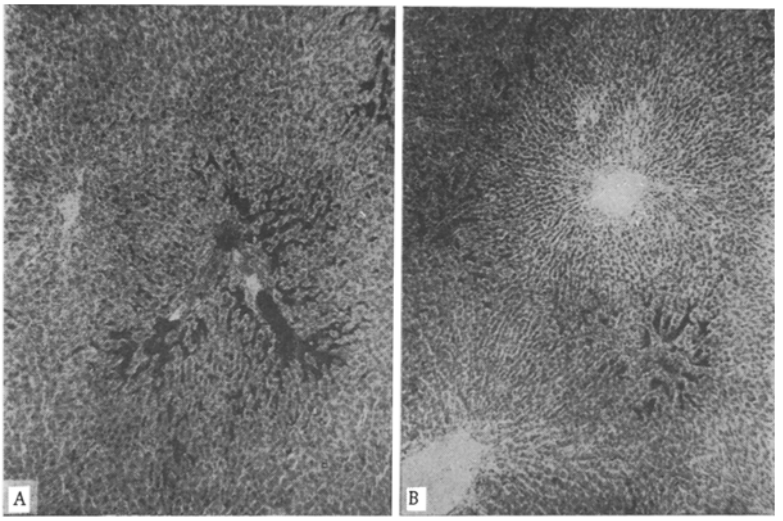

Fig. 1. Histological picture of the rat liver following injection of ink suspension into the portal vein. On the left: in the torpid phase of traumatic shock; on the right: control. 75×.

When ink suspension was injected into the portal venous system before the development of shock the picture observed was similar to that seen after injection of ink into the arterial system: ink particles also passed along the sinusoids and gradually accumulated in the region of their initial segments, but in small numbers. Injection of ink into the portal system in shock, on the other hand, was followed by its retention in the terminal segments of the portal venules, beyond which it did not progress. This was seen especially clearly in histological sections of the liver (Fig. 1). Retention of ink in the region of the terminal segments of the portal vein confirmed our previous conclusion regarding the role of an increase in their tone in disturbances of the portal blood flow in shock [4, 6].

The changes discovered in the microcirculation of the liver in traumatic shock are similar to those described previously in tourniquet shock [13] and in shock produced by blood loss and operation trauma [12].

On the basis of data in the literature [13], spasm of the hepatic vessels in the initial period of shock can be explained by an increase in tone of the sympathetic nervous system and by hyperadrenalinemia, present at this period. Disorders of the hepatic microcirculation arising with the development of the torpid phase of traumatic shock are complex in nature and are due to many different nervous and humoral factors, but a definite role in their genesis must belong to previous ischemia combined with shunting of the blood flow [3].

In the Soviet Union, Levin and co-workers [1, 2] have studied the microcirculation in the liver in clinical death caused by blood loss, and have observed changes similar to those described above in shock.

LITERATURE CITED

1. A. Ya. Evtushenko, in: Proceedings of a Scientific Conference on the Physiology and Pathology of Cortico-Visceral Relationships and Functional Systems of the Organism [in Russian], Vol. 1, Ivanovo (1965), p. 371.
2. Yu. M. Levin, Byull. Éksperim. Biol. i Med., No. 5, 33 (1963).
3. G. I. Mchedlishvili, The Capillary Circulation [in Russian], Tbilisi (1958).
4. S. A. Seleznev, Pat. Fiziol., No. 3, 21 (1963).
5. S. A. Seleznev, Byull. Éksperim. Biol. i Med., No. 8, 46 (1963).
6. S. A. Seleznev, Pat. Fiziol., No. 6, 25 (1964).
7. E. Bloch, Angiology, 6, 311 (1955).
8. W. S. Burrage and J. W. Irwin, Ann. Allergy, 11, 137 (1953).
9. M. H. Knisely, in: Handbook of Microscopical Technique, London (1937), p. 632.
10. M. H. Knisely, Anat. Rec., 120, 265 (1954).
11. M. H. Knisely, E. Bloch, and N. Warne, cited by Block [7].

12. A. G. Michel and W. C. Shormeker, Biorheology, 2, 11 (1964).
13. R. D. Senevirente, Quart. J. Exp. Physiol., 35, 77 (1949).