SPECIAL FEATURES OF THE MICROCIRCULATION IN THE LIVER IN ANAPHYLACTIC SHOCK

V. G. Ovsyannikov and S. M. Vashetina

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Changes in the microcirculation in the liver during anaphylactic shock were studied in experiments on rats and dogs. After injection of the reacting dose of horse serum the systemic blood pressure rose on the average by 20 mm Hg, spasm of the sinusoids and hepatic venules was observed, and the blood flow in them was accelerated. During the subsequent fall of the arterial pressure to 60-40 mm Hg congestion of the blood vessels, slowing of the blood flow, intravascular aggregation of the blood cells, and hemorrhages together with ischemic disturbances were observed in the liver. Restoration of the systemic arterial pressure and microcirculation took place parallel, but the normal microcirculation in the liver was not restored under these circumstances.

Key words: microcirculation; liver; anaphylaxis.

The problem of anaphylactic shock has recently attracted particular attention because of mass allergization of the population [3]. The role of the liver in the disturbance of the hemodynamics in anaphylaxis is well known. For that reason, the elucidation of the mechanisms of formation of anaphylactic shock is impossible without a study of the characteristics of the terminal circulation in this organ.

EXPERIMENTAL METHOD

The microcirculation in the liver was studied in 23 rats and 10 dogs. The rats were sensitized by 3 injections (intraperitoneal, subcutaneous, followed 2 days later by another subcutaneous) with a mixture consisting of 0.2 g lanolin, 0.25 g mineral oil, 6×10^9 cells of a whooping cough vaccine, and 0.7 ml normal horse serum. The dogs were sensitized by 3 injections, each of 0.3 ml/mg horse serum. The reacting dose (0.5 ml/100 g for rats, 2 ml/kg body weight for dogs) was injected into the jugular vein. The investigations were carried out under pentobarbital anesthesia, by means of a microscope with dark-field contact objective (ocular 10, objective 10) and with a type MFN-12 camera attachment for photomicrography. The microcirculation was assessed on the basis of visual observations and photomicrographs.

In the terminal vascular system of the liver in rats and dogs the portal and hepatic venules and the sinusoids were accessible to observation. The portal venules were seen much less often than the hepatic, and mainly at the periphery of the lobes. The chief difference between these vessels was the opposite direction of movement of the blood in them: in the portal vessels from venules into sinusoids, but in the hepatic (central) venules from sinusoids into venules.

EXPERIMENTAL RESULTS AND DISCUSSION

After injection of the reacting dose of horse serum the blood pressure of the rats rose sharply by 10-30 mm Hg (to 110-130 mm Hg); at this time spasm of the sinusoid and of the portal and hepatic venules and acceleration of the blood flow along them were observed in the liver.

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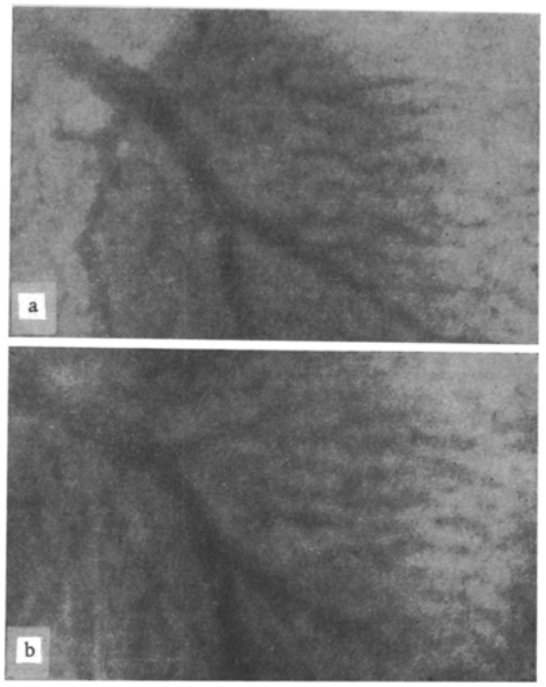

Fig. 1. Effect of anaphylactic shock on microcirculation in the rat liver: a) original appearance (blood pressure 100 mm Hg): sinusoids and a hepatic venule can be seen: b) anaphylactic shock (blood pressure 30 mm Hg): sharp dilatation of sinusoids (same part of the liver). Here and in Fig. 2: ocular 10, objective 10.

During the subsequent fall of blood pressure to 65-40 mm Hg the blood flow in the hepatic venules was slowed and sometimes became intermittent in character. In most sinusoids and hepatic venules congestion and statis developed, with petechial hemorrhages into the liver tissue, evidently resulting from the increased permeability of the microvessels. At this level of the arterial pressure, aggregation of blood cells was frequently seen in the hepatic venules and sinusoids, and this condition progressed with the development of the process (Fig. 1).

With a further decrease in the systemic blood pressure to 30-20 mm Hg most of the sinusoids were virtually nonfunctioning. They were grossly dilated, marked aggregation of blood cells with the formation of larger intravascular conglomerations was observed, and some sinusoids and hepatic venules were club-shaped. The mosaic pattern of the changes in the microcirculation was a conspicuous feature: side by side with satisfactorily functioning sinusoids there were vessels which functioned periodically or not at all. Often areas of hyperemia alternated with areas of ischemia (Fig. 2), where the vascular structure of the liver was imprecise because of the marked vascular spasm or the perisinusoidal edema.

After recovery of the blood pressure to 70-80 mm the microcirculation in the liver improved considerably, the blood flow was accelerated in all the vessels, the phenomena of aggregation of blood cells were reduced, the congestion of the sinusoids and the hepatic venules was less marked, but the normal hemodynamics was still not restored. In addition, changes in the circulation unconnected with the animal's respiration and movements could be observed. When the mean level of the blood pressure was 80 mm periodic congestion of the sinusoids with blood was observed, sometimes with aggregation of the blood cells; this could be quickly followed by acceleration of the blood flow and spasm of the sinusoids. When the arterial pressure was 80 mm Hg the interval between two periods of vascular spasm was 20-21 sec, when the pressure was 70 mm Hg the interval increased to 26-27 sec, when it was 50 mm Hg it was longer still, and when the blood pressure fell still lower this periodic alternation in functional activity of the individual vessels of the liver could no longer be observed.

Comparison of the periodic changes in the hemodynamics in the liver with fluctuations in the systemic blood pressure showed that at times of dilatation and congestion of the sinusoids and hepatic venules and of an increased tendency toward aggregation of the blood cells the arterial pressure was increased by 5-10 mm Hg, whereas during the period of expulsion of blood and of vasospasm it fell by approximately the same amount. The periodic nature of the changes in the microcirculation in the liver in anaphylactic shock was thus connected with small (not exceeding 10 mm Hg) fluctuations in the blood pressure in the main arteries. Dependence of the blood flow in the liver sinusoids on the arterial blood pressure has also been found by other workers [4, 7].

In dogs, after injection of the reacting dose of horse serum and the subsequent lowering of the blood pressure to 60 mm Hg, aggregation of blood cells in the hepatic venules and sinusoids and congestion of these structures were observed. The mosaic pattern of function of the hepatic vessels was clearly revealed. As the blood pressure returned to normal there was a tendency toward an improvement of the microcirculation. However, even after the arterial pressure had fallen to its level before shock, areas of ischemia and hyperemia still remained in the liver, evidence of incomplete recovery of the microcirculation and of its dependence on other factors than the level of the systemic blood pressure.

When the results of these experiments are compared with data in the literature it must be noted that in other animals (rabbits, guinea pigs) the deposition of blood in the sinusoids and central venules of the liver is increased in active and passive anaphylaxis, the blood cells undergo aggregation, and microemboli

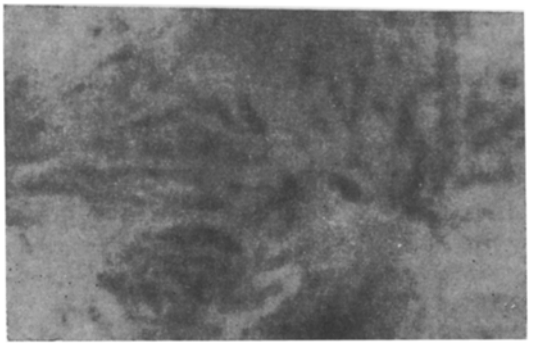

Fig. 2. Areas of hyperemia and ischemia in liver of rats with anaphylactic shock (arterial pressure 30 mm Hg).

are formed [6, 8, 10]. After injection of guinea pig serum directly into the hepatic artery of rabbits [9] necrotic foci appeared in the parenchyma of the organ, followed by degeneration of the liver cells. Pathomorphological investigations of the liver and other organs of persons dying from anaphylactic shock revealed blood stasis in all the organs, increased vascular permeability, hemorrhages, and microthromboses [5]. Well-marked aggregation blocking of the capillary blood flow and dilatation of the blood vessels of the venous microcirculation of the mesentery have been found in dogs, rabbits, and rats with anaphylactic shock [1, 2]. The results of macroscopic and microscopic investigations of the liver in dogs with anaphylaxis confirm the special features of the circulation in this organ revealed in vivo by the experiments described above.

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