MICROCIRCULATION IN THE LIVER DURING LETHAL ASPHYXIA AND SUBSEQUENT RESUSCITATION

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Considerable disturbance of the hemodynamics and capillary circulation of the liver take place in shock and terminal states [1, 3-6, 8-10, and others].

Dynamics of the microcirculation of the liver during lethal mechanical asphyxia and subsequent resuscitation is considered in this paper.

EXPERIMENTAL METHOD

The experiments (including controls) were carried out on 54 albino rats of both sexes weighing 150-280 g under Nembutal anesthesia (5-6 mg/100 g intraperitoneally). The microcirculation of the liver was studied by vital transillumination microscopy [2, 7]. Still photographs and motion pictures were taken in the course of the experiments. The arterial pressure and respiration were recorded at the same time on a kymograph. Asphyxia was produced by clamping the airway. The animals were resuscitated after clinical death (2-3 min) by indirect cardiac message and mechanical artificial respiration. In some experiments stimulant doses of adrenalin were injected into the carotid artery toward the heart.

EXPERIMENTAL RESULTS

Approximately 20 sec after the beginning of asphyxia, when the arterial pressure was falling sharply (Fig. 1A), the liver sinusoids contracted: at first spasm of the inlet sphincter appeared, followed by a narrowing of the lumen of the sinusoid tube and constriction of the outlet sphincter. This vascular reaction, directed toward expelling blood from the liver, was completed at the end of the first minute (Fig. 1A) by exclusion of most sinusoids from the circulation, by spasm of the arterioles, by a decrease in the lumen of the central and collecting venules, and by a decrease in the cell concentration of the blood flowing in them (Fig. 2b). Respiration ceased also at this time.

During the terminal pause, against a background of gradually rising arterial pressure (Fig. 1A) the liver vessels became filled with blood (Fig. 2c). The blood flow in the central and collecting venules was slowed, and then intermittent and to-and-fro. At this time movement of blood in the portal venules was slowed, but still remained continuous. This obstruction to the outflow led to overfilling of the sinusoids with blood. Their outlet sphincters and small sluices of Deysach were greatly constricted or completely closed. As a result of this, an avascular band equal to one or two diameters of sinusoids often appeared around the collecting and hepatic venules.

At the height of elevation of the arterial pressure (Fig. 1A) agonal respiration appeared and, at the same time, the blood flow in the liver was resumed. If the increase of arterial pressure was considerable and breathing prolonged and deep, the total vascular reaction was completed by expulsion of blood from the liver, as at the beginning of asphyxia. In some experiments no stimulation of the blood flow took place.

After respiratory arrest, the liver again became gradually filled with blood, which ceased to move before the heart stopped beating, when the arterial pressure was 40-60 mm. In the agonal period aggregation of the blood cells was frequently observed.

Resuscitation measures (indirect cardiac massage and artificial respiration) led to the appearance of to-and-fro movement of the blood in the vessels; movement of blood through the liver took place only slightly, however. With the appearance of cardiac activity the sinusoids contracted and, at the same time

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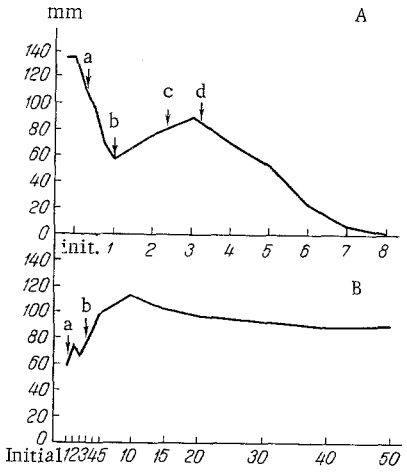

Fig. 1. Dynamics of arterial pressure. A) During lethal asphyxia; B) during resuscitation after clinical death (mean data of 9 experiments). Explanation in text.

as the arterial pressure rose (Fig. 1B), the blood stored in the liver was expelled. Only after this had taken place, at about the end of the first minute, was blood observed to flow in the arterioles and sinusoids receiving arterial anastomoses. After 1.5-2 min blood began to move in the portal venules. By this time the velocity of the blood flow in the arterioles was comparable with that observed initially. In the central and collecting veins, movement of blood was still intermittent in character, dependent on the phases of the respiratory cycle. Gradually more and more sinusoids became involved in the circulation. After emptying itself in the ejection period, the liver again began to fill with blood. In most experiments, by the time of appearance of the first spontaneous inspirations, the arterial pressure had increased (Fig. 1B) and the volume of blood in the liver had again decreased. The normal appearance of the hepatic microcirculation was restored by the 10-15th minute of resuscitation, although even in experiments in which the arterial pressure remained close to its initial level, the movement of blood was appreciably slower than in the control period.

Approximately 30 min after resuscitation, or 1.5 h after administration of Nembutal (i.e., when the anestetic was wearing off, as the 10 control experiments showed), new changes were observed: the vessels of the peripheral portions of the liver became empty while the central vessels, on the contrary, were filled with blood. The question arose whether these changes were associated with the severe hypoxia or whether they were the result of the end of the anesthetic action of Nembutal. To answer this question, the liver microcirculation was observed in 4 intact rats anesthetized with Nembutal. The blood flow was observed to become slower 1.5-2 h after administration of the anesthetic, and the central portions of the hepatic globules were filled with blood while the peripheral portions were emptied. On the other hand, studies of the hepatic microcirculation in rats under local anesthesia (5 experiments) revealed the same picture as in the animals recovering from the general anesthetic. Administration of Nembutal to the animals caused an appreciable decrease in the blood volume in the liver, while the velocity of movement of the blood increased and the differences between the circulation in the central and peripheral portions of the lobules disappeared. It may therefore be considered that the redistribution of the blood flow within the lobules developing in the recovery period of resuscitation is the result of ending of the anesthetic action of Nembutal.

In only 31% of cases was the recovery of the microcirculation completed almost to its initial state. More frequently in the resuscitation period, after the phase of ejection, the liver gradually filled with blood.

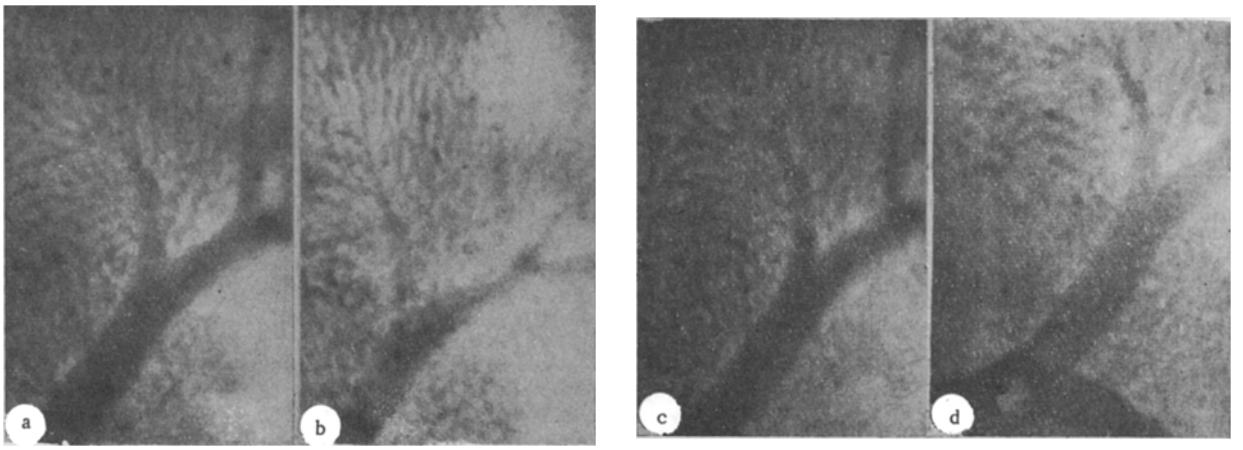

Fig. 2. Liver of a rat (sinusoids, central and collecting veins) during lethal asphyxia and subsequent resuscitation. a) Initial state; b) during expulsion of blood (30 sec of asphyxia); c) in the period of filling with blood (1.5 min of asphyxia); d) 26 min after resuscitation (arrow indicates a fat globule). Photomicrograph, 8 × 7.

The blood flow was active only in the arterial vessels and sinusoids receiving arterial anastomoses. The vessels of many of the lobules played no part in the circulation. These experiments also were characterized by a low level of arterial pressure and by the appearance of numerous fat lobules and emboli (Fig. 2d), causing still further disturbances to the circulation.

Hence, the reaction of the liver vessels to asphyxia is dynamic in character and consists of alternating phases of emptying (with an initial fall of pressure and during agonal respiration) and of deposition (during the terminal pause and after respiratory arrest) of blood. In the resuscitation period the blood flow through the liver appears after the phase of blood expulsion, and is seen initially in the vessels of the arterial system, evidently in connection with the relatively greater arterio-caval pressure gradient; movement of blood in the portal vessels is resumed later. The normal blood flow is restored in comparatively fewer cases, and more frequently the passage of blood is obstructed as the vessels are overfilled with blood. This last variant is observed if the arterial pressure falls considerably and respiration is disturbed, and it is evidently related pathogenetically to these factors.

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