MICROCIRCULATORY CHANGES IN THE MUCOUS MEMBRANE OF THE RETROBUCCAL POUCH DURING EXPERIMENTAL TRAUMATIC SHOCK IN HAMSTERS

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Substantial disturbances of the microcirculation in the mucous membrane of the retrobuccal pouch were observed in acute experiments on hamsters in which severe shock was induced by standardized mechanical trauma to the thigh. All the animals died within 24 h of such trauma. If the animals died not less than 1 h after trauma the microcirculatory changes were definitely phasic in character; in particular, a phase of temporary relative adaptation and stabilization of the peripheral circulation was observed in these animals, followed inevitably by a phase of decompensation and a terminal phase ending in death. In shock proceeding rapidly to a lethal termination within 1 h, no definite phasic pattern of the microcirculatory changes was observed, but all the indices worsened more or less rapidly. Unlike most other investigators, the authors saw no marked intravascular aggregation of red cells in these experiments.

KEY WORDS: shock; microcirculation; retrobuccal pouch of hamsters.

This paper gives results of a study of the microcirculation in the course of traumatic shock from its origin until death of the unanesthetized animals, i.e., under conditions as close as possible to those encountered in clinical practice.

EXPERIMENTAL METHOD

Experiments were carried out on 65 hamsters weighing 100-150 g. Shock was produced by Cannon's method in the writers' modification by means of an electromagnetic hammer that they designed and produced [8]: A series of blows, strictly graded in strength (35 kg/cm^2) and frequency (100/min) were applied to the hind limbs of the unanesthetized animals. The criteria of shock were the general condition of the animals, a fall in blood pressure (BP) in the carotid artery to 40-50 mm Hg, and an increase in the respiration and pulse rates. The trauma was discontinued when the blood pressure had fallen to 40-50 mm Hg.

The state of the microcirculation was observed on an apparatus mounted on an MBI-3 microscope in the mucous membrane of the retrobuccal pouch, previously enclosed in a transparent chamber made of plastic. By means of this method of intravital microscopy, observations could be kept for a long time (2-3 weeks) on the state of the nutritive circulation without any effects on the object observed [5]. All the indices studied were observed before trauma and continuously thereafter in the various phases of shock until the animal's death. Changes in the state of the terminal vascular network and of the blood flow in it were recorded photographically by means of an MFN-11 photomicrographic attachment.

EXPERIMENTAL RESULTS AND DISCUSSION

As a result of trauma all the animals developed shock. All the hamsters died within 24 h. However, despite the standard conditions of reproduction of shock, the state of the animals, the actual character of the process, and the changes in the indices studied differed in different animals. Depending on the character of the course of the shock and the dynamics of the changes in the indices studied the animals were divided into two principal groups: 1) with a distinct phasic course of shock, with a marked period of temporary compensa-

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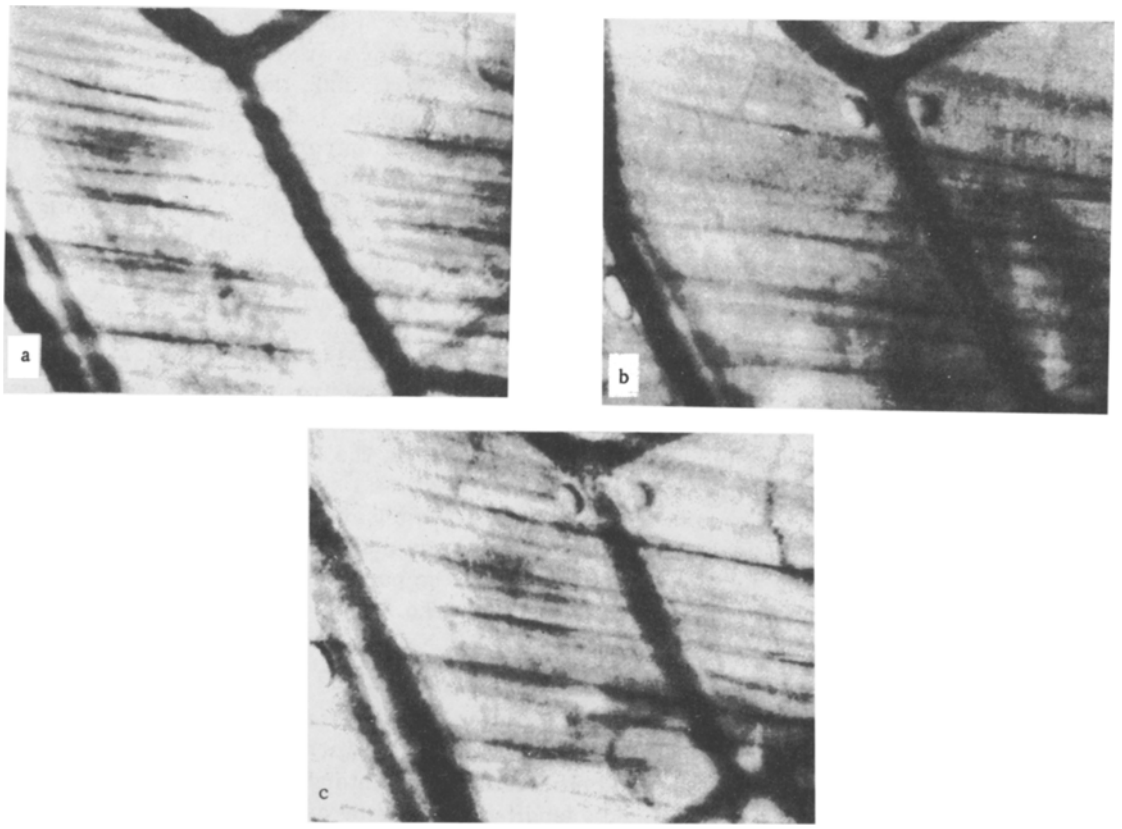

Fig. 1. Photomicrograph of small vessels in mucous membrane of hamster retrobuccal pouch: a) after trauma to the thigh; b) spasm of arteries and venules in erectile phase of shock (3 min after end of trauma to thigh); c) vasodilatation and slowed blood flow in venous and arterial microvessels in terminal phase of shock (27 min before death of animal); evidence of slowing of blood flow is ability to distinguish individual blood cells in photograph, which is impossible on filming under these conditions with the usual velocity of blood flow; 250×.

tion (stabilization) of the disturbed functions. The animals of this group (35 hamsters) died between 1.5 and 24 h after trauma (most of them before 6 h); 2) with rapidly progressive shock, with no clear phase of temporary compensation. All the animals of this group (30 hamsters) died during the first hour after trauma.

After the end of trauma all the animals of both experimental groups were in a state of shock: They were motionless and did not respond to nociceptive or acoustic stimulation. The pulse and respiration rates were raised and BP was low. Microcirculatory changes immediately after trauma and during the next 5-20 min were clearly defined and similar in type in practically all animals of both experimental groups: Severe spasms of most vessels, especially arterial, were constant and at times led to such a degree of vasoconstriction that the passage of blood cells was prevented; there was a virtually complete absence of functioning capillaries (Fig. 1a, b), slowing of the blood flow in both venous and arterial vessels, with a to-and-fro or jerky movement interspersed with stopping of the blood flow in some of them; plasmatization of the blood flow occured in some venules and small veins receiving blood from arteries in a region where they were in a state of extreme spasm.

In the animals of experimental group 1, a period of temporary partial compensation (stabilization) lasting for between a few minutes and a few hours developed 5-20 min after the end of trauma; its duration determined the length of survival of the experimental animal after trauma. During this period of shock BP rose a little (to 70-80 mm Hg) and respiration became slower, more regular, and deeper. Meanwhile the blood flow improved appreciably in the terminal vascular network: The angiospasm was reduced or abolished altogether; movement of the blood in many of the small arteries, veins, and some of the capillaries was resumed, where it had ceased immediately after trauma. However, most of the blood flow did not pass through the true capillaries, the number of which was several times smaller, but through arteriolovenular anastomoses, the num-

ber of which was appreciably greater than before trauma. Movement of the blood in this way evidently implied worsening of the nutritive blood flow, and this is now regarded by many workers as the main pathophysiological disturbance in shock [11, 14, 15]. The nutritional deficit and, in particular, the increase in the oxygen debt gradually led to severe uncompensated disturbances of metabolism of the body cell and, in particular, cells of the vitally important organs, thereby bringing to an end the period of temporary relative compensation which was in effect a period of temporary adaptation of the organism to its new conditions of existence. A second period of decompensation followed, and this led into the terminal phase of shock. Under these circumstances BP fell relatively rapidly and respiration as a rule became fast and superficial with occasional deep inspirations. The functioning capillaries again disappeared virtually completely and the blood flow in those small vessels in which it still continued was much slower; the blood flow gradually ceased in some venules and veins, and later in the arterial vessels also, and the blood in them was appreciably thickened. The most characteristic and constant features of the microcirculatory changes in the terminal phase of traumatic shock, indicating that a lethal outcome was near, were vasodilatation and sequestration of the red cells, connected with it and with the hemoconcentration (Fig. 1c). Pathological dilatation of all the blood vessels, especially venous, occurred; they were filled with blood and, for a few minutes, the impression of dilatation of the network of functioning microvessels and improvement of perfusion of the tissues with blood was created. Very soon, however, the motion of the blood ceased and total stasis was observed in all the dilated and congested vessels. Respiration became pathological, the cardiac rhythm was disturbed, tachycardia was replaced by bradycardia, BP fell to zero, and the animal died. Throughout the period of development of shock the animal remained motionless or nearly so, so that no fixation was required, and it responded weakly or not at all to nociceptive and other forms of stimulation.

The course of shock in the animals of experimental group 2 differed from that described above in the absence of any clearly marked period of relative compensation and stabilization. BP did not rise after trauma but continued to fall gradually, the tachypnea changed into pathological forms of respiration, and tachycardia was replaced by bradycardia. The angiospasm and other microcirculatory disturbances observed immediately after trauma were quickly replaced by generalized vasodilatation and by congestion of the dilated vessels with blood; meanwhile BP fell to zero, the heart stopped beating, and the animal died. The survival period of the animals in this group varied from 10 to 60 min.

The microcirculatory changes described above in traumatic shock agree on the whole with observations by other workers. Admittedly, in this case it was possible to make these observations without the use of anesthesia and continuously throughout the period of shock, without the risk of side effects on the test object itself. This may partly explain one of the significant differences between the present results and observations by certain other workers [4, 13]: The generalized intravascular aggregation of red cells which the writers constantly found in burn shock [6, 7] was not observed in any phase of traumatic shock if it lasted less than 24 h. Since anesthesia itself has a marked effect on the microcirculation [9] the present investigations of the peripheral hemodynamics in the course of shock without the use of general anesthetics evidently must reflect more adequately its state in shock such as is observed in clinical practice.

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CHANGES IN MASS OF THE RIGHT VENTRICLE AND ITS RNA CONTENT DURING REGRESSION OF HYPERTROPHY

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Noninbred male rats weighing 250-300 g were used. Adaptation to high-altitude hypoxia was produced by keeping the animals once a week for 5 h in a pressure chamber at an "altitude" of 6000 m. The degree of hypertrophy of the right ventricle and its RNA content were studied after 20 days of adaptation and also 2, 10, 20, and 40 days after the end of exposure to hypoxia. The mass of the right ventricle and its RNA concentration and content were significantly increased 20 days after the beginning of adaptation. After the end of exposure to hypoxia half of the increase in mass of the right ventricle was lost during the next 10 days and half of the increase in RNA during the next 2 days. Forty days after the end of exposure to hypoxia the mass of the right ventricle and its RNA content in the adapted animals were indistinguishable from those in the controls.

KEY WORDS: adaptation; hypoxia; hypertrophy of the right ventricle; regression of hypertrophy; RNA.

With an increase in the load on the heart the synthesis of nucleic acids and protein in the myocardial cells is activated and hypertrophy develops. A reduction in the load on the heart leads to a decrease in the intensity of nucleic acid and protein synthesis and in the mass of the organ [2, 3, 7]. Correspondingly, it has been shown that hypertrophy of the heart is a reversible process: After removal of aortic stenosis, creating a load on the heart, the mass of the myocardium decreases fairly rapidly [8, 10]. However, the extent to which the dynamics of regression of hypertrophy depends on the decrease in myocardial function is not quite clear, for aortic stenosis and its removal necessitated a surgical operation, and repeated operations, like changes in the region of the aorta to which the stenosing ring was applied, could affect the state of the heart and the regression of its hypertrophy.

To rule out the action of these factors, in the investigation described below hypertrophy of the right ventricles was produced by adaptation to high-altitude hypoxia; the mass of the right ventricle and its RNA content were measured during regression of the hypertrophy.

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