AN EXPERIMENTAL STUDY OF THE PORTAL CIRCULATION IN THE AGONY PERIOD AND DURING RESUSCITATION FROM CLINICAL DEATH

Yu. M. Levin

Experimental Surgical Division (Director, Yu. M. Levin), Novosibirsk Research Institute of Traumatology and Orthopedics (Director, Docent D. P. Metelkin)
(Presented by Active Member AMN SSSR A. V. Lebedinskii)
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The importance of the study of the regional circulation in shock and terminal states has been recognized [5, 7, 15]. The circulation in various organs and tissues undergoes specific changes in pathological conditions [3, 6, 8, 12], and in some cases, for example, in agony and resuscitation from clinical death, these changes may be irreversible [14, 15, 16]. The regional circulation in severe shock and terminal states therefore continues to attract the increasing attention of research workers [2, 4, 9, 10, 14, 15, 16].

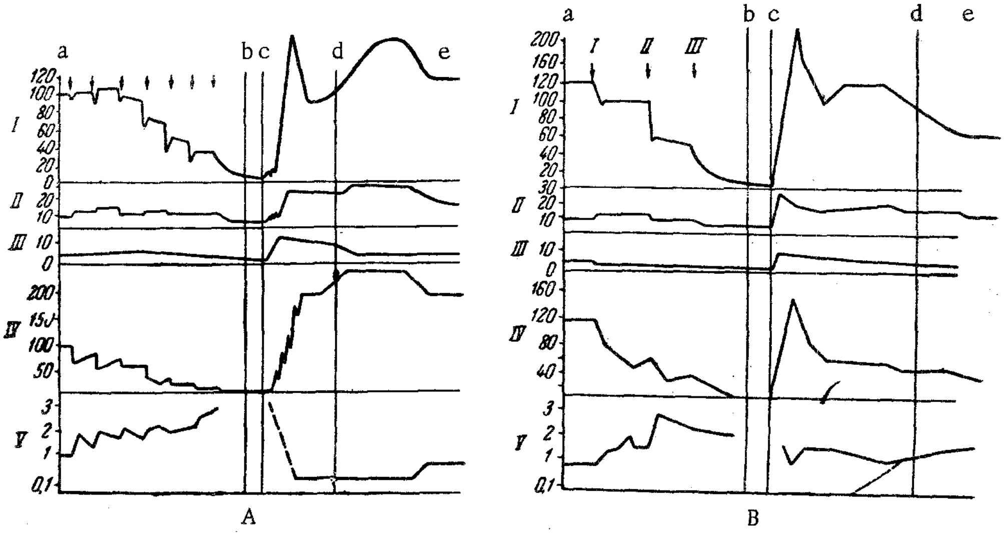
In the present research we studied the changes in the portal circulation. The importance of studying the state of this portion of the circulatory system, which can accommodate up to 60-80% of the total blood volume in circulatory disturbances, has repeatedly been stressed [6, 10-13].

EXPERIMENTAL METHOD

Changes in the portal circulation were studied in 16 dogs during lethal exsanguination and subsequent resuscitation after a period of clinical death lasting 4-5 min. Measurements were made of the aortic pressure, the pressure and velocity of the blood flow in the superior mesenteric artery, and the pressure in the portal vein and the posterior vena cava at the junction of the hepatic veins. From these figures it was possible to calculate the arterio-portal and porto-caval pressure gradients and the resistance of the mesenteric vessels, and also to assess the state of the hepatic shunt mechanism. The portal and caval pressures were recorded during the respiratory pause. Additional observations on the local circulation of the blood and the state of the blood vessels of the mesentery and liver during the periods of blood loss, clinical death, and subsequent resuscitation were made on 44 dogs, cats, and rats. Observation and photography of the mesentery were carried out by the ordinary methods, and in the case of the liver the MUF-3M luminescence source was also used [13]. To prevent the part under observation from drying, it was continuously irrigated with warm physiological saline. Blood was withdrawn in small volumes at a time for a period of 20-30 min. The animal was resuscitated by Negovskii's method after 4-5 min of clinical death, and wherever possible the use of stimulants was avoided. In some cases the heart had to be defibrillated [1].

EXPERIMENTAL RESULTS

In the different animals the effect of bleeding was to produce similar changes in the criteria examined (see figure). Usually the velocity of the blood flow fell before the arterial pressure was lowered. The resistance of the mesenteric vessels rose during this period. Despite the reduction in the volume of blood arriving, the portal pressure increased. This led to a very slight fall in the arterio-portal gradient and to a larger increase in the porto-caval gradient. The increase in the portal pressure showed that passage of the blood through the liver was difficult. Since the inflow of blood to the portal vein was thereby decreased and the resistance of the mesenteric vessels was increased, considerable activation of the sphincter mechanism of the liver could be assumed. The increased arterial pressure sometimes observed (after loss of a small volume of blood), especially in the mesenteric artery, was associated not only with an increase in the work of the heart and the mobilization of depot blood, but also with spasm of the vessels of the portal system. A significant feature was that the increase in pressure in the portal vein was not accompanied by an increase in the blood flow. During the pause after the first, and after the second bleeding, all the indices under examination showed a tendency to return to normal, except the portal pressure.



Circulation in the system of the mesenteric artery and portal vein during lethal exsanguination and subsequent resuscitation. A and B) Characteristics of groups of animals with increased (A) and decreased (B) volume blood flow after resuscitation. I) blood pressure in mesenteric artery (in mm); II) portal pressure (in mm); III) pressure in posterior vena cava (in mm); IV) volume velocity of blood flow in mesenteric artery; V) index of resistance of mesenteric vessels, calculated from the formula R = pressure gradient/volume blood flow. a-b) Period of bleeding; b-c) clinical death; c-d) period from beginning of resuscitation to appearance of spontaneous respiration; d-e) period of observation after restoration of spontaneous respiration.

Studies of the microphysiological changes in the vessels of the mesentery and liver during bleeding are reasonably adequate [13, 15]. Suffice it to say that the degree of constriction of the mesenteric and hepatic vessels at the beginning of bleeding did not follow a parallel course.

Continued bleeding led to a fall in the arterial pressure, to an even greater fall in the velocity of the blood flow, and to an increase in the resistance of the mesenteric vessels (see figure, A, B). The pressure in the posterior vena cava rose very slightly or remained the same. Characteristically, even a sudden reduction in the arterial pressure and volume velocity of the blood flow caused no significant lowering of the portal pressure. The comparatively high portal pressure in association with the low arterial pressure was reflected in the pressure gradient. In contrast to the preceding period of blood loss, during the intervals between successive bleedings the tendency for the indices to return to normal was weakened. Between bleedings, the changes caused by bleeding were intensified. The velocity of the blood flow in the mesenteric artery fell to very low values, although the arterial pressure remained adequate. During this period of exsanguination the resistance of the mesenteric vessels rose more than two- or three-fold.

During the agony period the velocity of the blood flow continued to decrease or, in some cases, to stop completely. At the same time the portal pressure and the pressure in the posterior vena cava (the latter usually slightly) also fell. The resistance of the mesenteric vessels reached its highest level.

Microscopic examination showed that the vessels of the mesentery and liver were contracted. The former contained many empty capillaries; the latter, constricted or closed sinusoids. Sometimes the slowly moving blood stream contained transparent, luminescent drops, most probably lipoid in nature (similar drops were seen in our earlier investigation of the cerebral circulation [2] during microscopic examination of the pial vessels of an animal during the agony period). The possibility of fat embolism, associated with acute blood loss and hypoxia, is an extremely important matter requiring a special study. The number of visible erythrocytes diminished. With the development of stasis the erythrocytes began to aggregate, and this tendency reached its maximum in the period of clinical death. Besides the comparatively large aggregates of erythrocytes there were many smaller clusters of a few cells. The mechanism of aggregation of the erythrocytes during circulatory arrest has been studied previously [4]. It is important to remember the possibility of occlusion of vessels of small caliber by these aggregates, and of ensuing local circulatory disturbances in the initial period of restoration of the blood flow [2, 10]. The hematocrit readings, total protein, and specific gravity all demonstrated hemoconcentration of the portal blood.

The changes in our criteria in the initial period of resuscitation from clinical death were less constant than during bleeding. Nevertheless, we were able to distinguish two main groups. In the first group of animals (see figure, A), immediately after the reappearance of cardiac activity a high volume velocity of the blood flow was established, sometimes from 50 to 100% greater than the initial value. The considerable fluctuations in arterial pressure characteristic of the initial period of recovery of the circulation, were frequently reflected in the velocity of the blood flow. The portal pressure was more than twice or three times its initial value. The pressure in the posterior vena cava also was increased during the first minutes of resuscitation. Calculation of the resistance of the mesenteric vessels showed that it had fallen to 1/2 - 1/3 the normal value.

The high volume velocity in the anterior mesenteric artery was evidently dependent on the increased cardiac activity, on the one hand, and the diminished tone of the mesenteric vessels on the other. The increased pressure in the portal vein and the distension of its tributaries with blood indicate that the liver at this period retained part of the incoming blood. This discrepancy between the tone of the blood vessels of the liver and intestine evidently led in some cases to the accumulation of large volumes of blood in the portal system.

In the other group of animals (see figure, B) the arterial pressure and the velocity of the blood flow fell after a brief rise. The fall in the velocity of the blood flow was more marked than the fall in the arterial pressure. It was significant that in these, as in the preceding experiments, the portal pressure remained high. The resistance of the mesenteric vessels (in contrast to the first group) was increased. It is interesting that in these experiments, notwithstanding the reduced inflow of blood and the increased resistance of the portal vessels, the hepatic shunts retained part of the blood in the portal system. This demonstrates that the retention of blood in the portal system in the initial period of resuscitation after clinical death is dependent not only on the increased inflow of blood, but also, and to a greater degree, on the activation of the vascular sphincters of the liver.

If the course of resuscitation was satisfactory, normalization of our criteria began between 40 and 90 min after it commenced. Normalization was more difficult in the animals of the second group.

The study of the local circulation in the initial period of resuscitation showed that, even when the volume velocity of the blood flow was high, there were areas in the mesentery and in the liver with slowing or complete stasis of the movement of blood. We gained the impression that spasm of the liver vessels was more marked and longer in duration than spasm of the mesenteric vessels. If no other procedures were carried out and if the course of resuscitation was smooth, after 20-30 min the pattern of the capillary blood flow and the blood flow in the small vessels began to return to normal. Nevertheless, the diameter of the blood vessels, the velocity of the blood flow, the ratio between the fluid and cellular fractions of the blood, the permeability to protein, and the other indices which we studied remained abnormal for a considerable time.

SUMMARY

An inquiry was made into the regularities of the portal system of animals after fatal blood loss and resuscitation following clinical death. The mechanisms capable of providing blood accumulation in the portal circulation at the initial period of revivial were revealed. The following factors which could prevent normalization of the vital activity were noted: a) aggregation of erythrocytes obstructing some small blood vessels, b) possibility of fat drops penetrating into the circulation during agony, c) blood thickening in the portal circulation, inhibiting its movement along the capillary bed and oxygen exchange.

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