PORTAL BLOOD FLOW IN THE EARLY PERIOD OF REVITALIZATION AFTER LETHAL BLOOD LOSS IN CATS

A. Ya. Evtushenko

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Seventeen male and female cats were anesthetized with pentobarbital and heparinized. The portal blood flow was studied by the local thermodilution method, and the systemic arterial pressure and the pressure in the portal vein and inferior vena cava where it receives the hepatic veins were recorded for 2 h during the recovery period of resuscitation after lethal blood loss. An increase in the portal blood flow was found during the first minutes of revitalization, followed by a decrease. Changes in the portal blood flow corresponded largely to changes in the cardiac output. The passage of blood through the liver was embarrassed to some degree at the end of the first and during the second hour of the recovery period.

Disturbances of liver function have recently been discovered after recovery from terminal states [8, 10] and the beneficial effect of liver perfusion with oxygenated blood during clinical death on revitalization processes has been demonstrated [11]. It has been shown that the blood flow in the superior mesenteric artery and the microcirculation of the liver can be disturbed in the initial state of recovery of vital functions [3, 4, 7]. No data on the portal blood supply of the liver in the recovery period of resuscitation after clinical death could be found in the accessible literature although disturbance of this blood flow is ascribed an important role in the pathogenesis of the hemodynamic disorders in terminal states [9, 13-15].

The object of the present investigation was to study the blood flow in the portal vein during 2 h of the recovery period of resuscitation after lethal blood loss.

EXPERIMENTAL

Experiments were carried out on 17 male and female cats weighting 2-3.5 kg, anesthetized with pentobarbital (40-45 mg/kg intraperitoneally). The blood flow in the portal vein was studied by the local thermodilution method [16] with certain modifications of design. The measuring catheter was passed into the portal vein through one branch of the superior mesenteric vein. The blood flow was measured after closure of the abdomen. Isotonic sodium chloride solution 4-8°C below the blood temperature was used as the indicator. The thermodilution curves were recorded on a suitably modified EPP-09 apparatus [1, 2]. The blood flow was calculated by Fegler's formula [12] and the area of the curves was determined by a gravimetric method. The portal pressure, the pressure in the inferior vena cava at the point of entry of the hepatic veins, and the systemic arterial pressure were recorded simultaneously; the arterio-portal and portocaval pressure gradients were calculated. To prevent the blood from clotting heparin was injected into the vein (400-500 units/kg). In the experiments of series I the portal hemodynamics were studied in the initial period and during revitalization in 10 animals. Clinical death (4 min) was induced by exsanguination, and resuscitation was carried out by Negovskii's method without the use of stimulants. In the experiments of series II (control) the same indices were studied for 2 h in 7 animals which were not exsanguinated. The animals' body temperature was maintained throughout the experiment close to its initial level.

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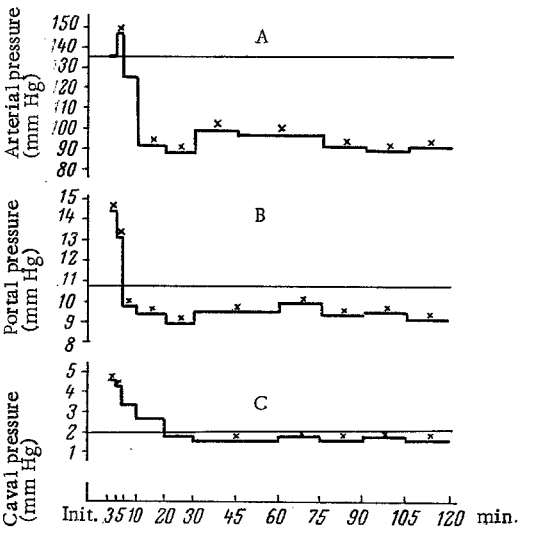

Fig. 1

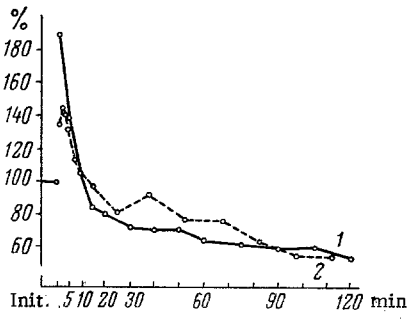

Fig. 2

Fig. 1. Arterial pressure (A), portal venous pressure (B), and pressure in inferior vena cava (C) during resuscitation. Initial levels of indices marked by horizontal lines; X) significant ($P < 0.05$) difference from initial values.

Fig. 2. Cardiac output and portal blood flow during revitalization (percent of initial level): 1) cardiac output; 2) portal blood flow.

EXPERIMENTAL RESULTS

The results of the study of the portal hemodynamics are given in Figs. 1 and 2. In the first few minutes after resumption of the cardiac contractions the portal blood flow was significantly higher than its initial level of 19.4 ± 1.2 ml/kg/min, being 26.4 ± 1.9 after 2 min, 28.4 ± 1.9 after 3 min, and 27.1 ± 3.3 ml/kg/min after 4 min. This change was accompanied by a relatively slight increase in the arterio-portal (by 8%) and porto-caval (by 13%) pressure gradients and a decrease in the resistance of the mesenteric vessels and the portal circulation of the liver. The portal blood flow then began to decrease gradually, regaining the control level after 10-20 min, and thereafter continued to fall, despite maintenance of a relatively steady level of the arterio-portal and porto-caval pressure gradients. By the end of the period of observation (2 h) the blood flow in the portal vein was 10.6 ± 0.8 ml/kg/min, i.e., 55% of the initial level, and it differed significantly from the value of this index in the control series, in which it had fallen to 79% of the initial level (initially 18.4 ± 0.8, after 2 h of the experiment, 14.5 ± 1.9 ml/kg/min).

Comparison of the results of this investigation with those obtained previously for the cardiac output [5] shows that the changes in portal blood flow were mainly parallel to changes in the minute volume (Fig. 2). Only during the period from 30 to 45 min was the blood flow in the portal vein significantly higher than previously for a constant minute volume. Bearing in mind that the portal blood flow is determined largely by vascular responses of the gastro-intestinal tract and spleen [13, 14], presumably its transient increase depends on the rise of arterio-portal pressure gradient.

The subsequent decrease in portal blood flow was probably connected not only with the decrease in cardiac output, but also with embarrassment to the passage of blood through the liver. Evidence of this embarrassment is given by the fact that the portal pressure remained constant while the blood flow was reduced. Calculation of the resistance of the portal circulation of the liver showed an increase by the end of the first hour of observation, whereas the resistance of the mesenteric vessels was still low at that time. Besides a constrictor response, microcirculatory disturbances connected with aggregation of the blood cells, swelling of the endothelium, and, in some cases, with fat embolism, may also play a role in this increased resistance to the blood flow [3, 4, 6].

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