

BLOOD VOLUME OF THE LIVER AND SPLEEN DURING THE ACTION OF CERTAIN HUMORAL FACTORS

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The object of the present study was to examine simultaneously the variations in the blood volume of the liver and spleen during the action of certain humoral factors—adrenalin and histamine, and also in hypercapnia and hypoxemia caused by asphyxia.

There are reports in the literature of contraction of the spleen following injection of adrenalin and histamine into the blood stream, and also in asphyxia [3, 6, 7, 14, 21, 22]. Data concerning the changes in the blood volume of the liver are less consistent. Under the influence of adrenalin the volume of the liver is known to decrease [14, 18, 19]. A volume of blood equal to 59% of the weight of the liver itself may leave the organ for the general circulation [15]. By using the bromsulfalein method, some authors have demonstrated an increase in the blood flow through the liver associated with dilatation of the hepatic arteries [10, 13], while others have found a decrease [16]. It has been shown that histamine increases the volume of the liver [19] and increases the blood flow through that organ [13], and there is information indicating that histamine may also cause a decrease in the volume of the liver [17]. During transient asphyxia, some authors [14] have found an increase in the volume of the liver, while others [8-12] have found a decrease.

EXPERIMENTAL METHOD

Experiments were conducted on 20 dogs. Simultaneous recordings were made of the blood volume of the liver and spleen by means of plexiglas oncometers, consisting of two closely fitting hollow hemispheres containing a rubber balloon and fitted with a notch to allow passage of vessels and nerves. To record the volume of the liver, its left (the largest in the dog) lobe was placed in the oncometer. Blood from the splenic vein enters the left lobe of the liver [20].

If the dog's thorax was wide enough, the liver could be placed in the oncometer without incising the diaphragm and with preservation of natural respiration. In some experiments a thoraco-abdominal incision was made, the diaphragm and parietal pleura divided, and artificial respiration applied. Recordings were also made of the arterial pressure and respiration.

Adrenalin and histamine were injected usually into the femoral vein in doses of between 0.1 and 0.5 ml of the 0.1% solution (from 100 to 500 μ g dry weight). Asphyxia was produced by temporary compression of the trachea.

EXPERIMENTAL RESULTS

After intravenous injection of adrenalin into the animals, in agreement with data in the literature, a reduction in the volume of the spleen was always observed, proportional to the dose of the drug injected, and usually considerable in degree.

The blood volume of the liver also diminished. This combination of emptying of the liver and spleen undoubtedly plays a part in the elevation of the arterial pressure after injection of adrenalin. It is clear from Fig. 1

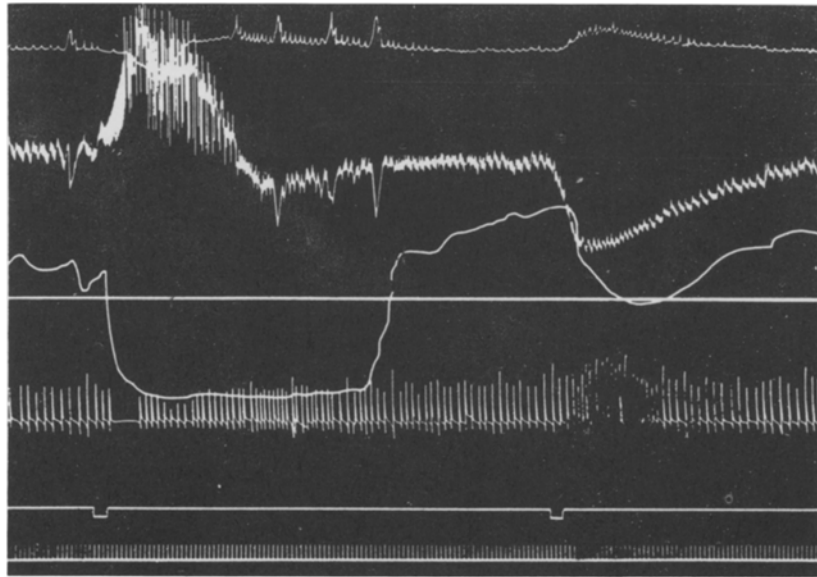


Fig. 1. Changes in volume of liver and spleen after intravenous injection of adrenalin and histamine. Significance of curves (from top to bottom): oncogram of liver; pressure in carotid artery; zero line of arterial pressure; oncogram of spleen; respiration; marker of stimulation; time marker (5 sec).

that after the intravenous injection of 0.2 ml of 0.1% adrenalin solution (1st stimulus), besides elevation of the arterial pressure and a decrease in the amplitude of its pulse oscillations, the blood volume of the liver and spleen also fell. The blood volume of the liver was restored fairly rapidly and exceeded its initial value, and the restoration of its volume coincided with the onset of a fall of the arterial pressure. The spleen remained contracted much longer, and its volume subsequently increased.

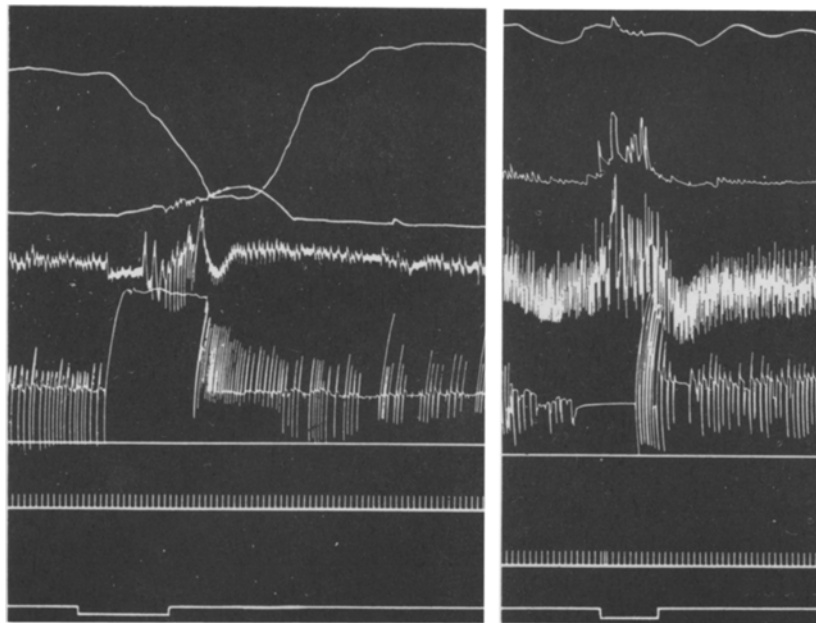


Fig. 2. Changes in volume of liver and spleen in asphyxia. Significance of curves (from top to bottom): oncogram of spleen; oncogram of liver; pressure in carotid artery; respiration; zero line of arterial pressure; time marker (5 sec); marker of stimulation.

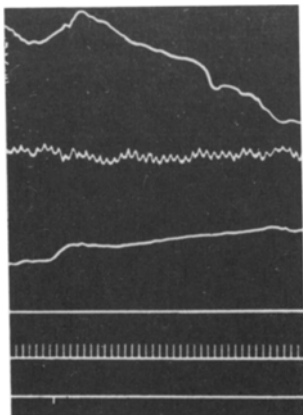


Fig. 3. Changes in volume of spleen during a reduction in the outflow of blood from the liver. Significance of curves (from top to bottom): oncomogram of spleen; pressure in carotid artery; oncomogram of liver; zero line of arterial pressure; time marker (5 sec); marker of stimulation (artificial respiration).

The relationships between the two blood depots were completely different in the same experiment after intravenous injection of 0.3 ml of 0.1% histamine solution (see Fig. 1, 2nd stimulus): a reduction in the volume of the spleen was observed (less marked than in response to injection of adrenalin), the volume of the liver began to increase before the spleen contracted, and the arterial pressure fell.

Blood expelled from the spleen into the portal vein causes an increase in the intraportal pressure [4]. However, the liver plays the role of a shunt in the path of the outflow of blood from the portal system, and retention of blood in the liver depot is evidently one of the factors contributing to the fall in arterial pressure after injection of histamine into the blood stream.

During asphyxia, the amplitude of the pulse oscillations of the arterial pressure increased and the arterial pressure rose. In most experiments the spleen contracted, while the volume of the liver increased. After artificial arrest of respiration in one of the experiments (Fig. 2) an increase in the amplitude of the pulse oscillations and a slight elevation of the arterial pressure were observed; the blood volume of the liver increased and the spleen contracted. It is interesting to note that the greatest increase in the volume of the liver coincided with the time of maximal emptying of the spleen and of a fall of the previously elevated arterial pressure (the latter began to fall just before restoration of respiration).

To determine the causes of the increased blood volume of the liver after injection of histamine into the blood stream and during asphyxia, it must be remembered that blood from the contracting spleen passes along the splenic veins into the portal vein. S. P. Botkin originally associated the increase in the volume of the liver arising during faradization of the spleen with the expulsion of blood from the spleen into the portal vein. However, this factor is not decisive. It is clear from Fig. 1, for instance, that the enlargement of the liver after injection of histamine took place before the spleen began to empty. In the experiments with asphyxia an increase in the volume of the liver was observed in the absence of contraction of the spleen (Fig. 2). The liver increased in volume while the spleen was still slightly enlarged and not during emptying of the spleen.

Changes in the lumen of the efferent and afferent vessels and changes in the resistance of the intrahepatic vascular network play an important role in the mechanism of the changes in the volume of the liver. Likewise, when discussing the depot function of the liver, the role of its lymphatic system cannot be completely excluded [1].

There is no direct evidence in the literature of the effect of variations in the blood volume of the liver on the volume of the spleen. In the experiments of S. P. Botkin cited above, faradization of the liver caused no changes in the volume of the spleen. Clinically, in patients with hepatomegaly (resulting from decompensation of the circulation) no enlargement of the spleen is observed [1].

To investigate this problem experimentally, stasis of the blood in the liver was produced by artificially impeding the blood flow from the organ (by partial compression of the hepatic veins). Under these circumstances, after a transient dilatation, the spleen contracted, and while the stasis in the liver was maintained, this contraction continued to increase (Fig. 3). The arterial pressure fell slightly (in this case artificial respiration was applied). Besides the hemodynamic factors, reflex influences from the hepatic vessels on the spleen cannot be excluded here.

The results of these experiments show that the two blood depots investigated—the liver and spleen—may differ in their behavior. The changes in the blood volume of the liver and spleen may occur in the same direction (after injection of adrenalin) or in opposite directions (after injection of histamine and during asphyxia). A disturbance of the outflow of blood from the liver leads to contraction of the spleen.

The relationships between these depots are reflected in the state of the circulation and must be taken into consideration when the depot function of the splanchnic region as a whole is being evaluated.

SUMMARY

In acute experiments on dogs a simultaneous study was made of the changes occurring in the liver and spleen congestion following intravenous administration of adrenalin and histamine and in hypoxemia and hypercapnia caused by asphyxia.

Experimental results demonstrated that under the mentioned actions the liver and spleen congestion could change both in one direction (with the adrenalin administration, histamine and in asphyxia). Reduction of blood outflow from the liver leads to the reduction of the spleen size. Interrelations of the aforementioned blood depots influence the state of circulation and should be taken into consideration in evaluating the depot function of the splanchnic region as a whole is being evaluated.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. Some or all of this periodical literature may well be available in English translation. A complete list of the cover-to-cover English translations appears at the back of this issue.
