# EFFECT OF VENOUS PRESSURE ON HEPATIC MICROCIRCULATION AND LYMPH FLOW

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The direction and volume of transcapillary movement of fluid are determined by the capillary hydrostatic pressure and the coefficient of capillary filtration [1, 2, 6, 11]. The values of these parameters have been established for the brain, limbs, and intestine [2, 6, 11], but their values for the liver, whose vascular bed plays an important role in the maintenance of homeostasis in the cardiovascular system, have received little mention in the literature.

The aim of this investigation was to determine values of the capillary hydrostatic pressure (the intrasinusoidal hydrostatic pressure — IHP), the coefficient of sinusoidal filtration (CSF), and the lymph flow (LF) in the liver. Since, according to data in the literature, the pressure in the hepatic veins is 1.5-3.5 mm Hg [1, 9], parameters of the micro- and macrohepatic veins is 1.5-3.5 mm Hg [1, 9], parameters of the micro- and macrocirculation and of the lymph flow in the liver were studied when the venous pressure (VP) was 0, 2, and 4 mm Hg.

## EXPERIMENTAL METHOD

Experiments were carried out on 17 cats weighing 3-4 kg, anesthetized with urethane (1 g/kg). To prevent the blood from clotting heparin (1500 U/kg) was used. Hemodynamic isolation and perfusion of the liver (mass 110 ± 4 g), preserving its sympathetic innervation, were carried out under conditions of stabilized blood flow along the hepatic artery (25.6 ± 0.9 ml/min·100 g) and portal vein (42.2 ± 1.1 ml/min·100 g) by the method described previously [4]. IHP and CSF were determined by the method in [2]. In view of the absence of information in the literature on the venous load during determination of CSF in the liver, whatever the initial level of VP it was increased by 1, 2, or 3 mm Hg and the value of the sinusoidal filtration was calculated for 90-120 sec after the beginning of elevation of VP. The perfusion arterial pressure (PAP) and perfusion portal pressure (PPP) also were recorded during the experiments.

## EXPERIMENTAL RESULTS

In the experiments when the pressure in the hepatic vein was 0 mm Hg, PAP and PPP were 120 ± 3 and 13.0 ± 0.7 mm Hg respectively. The value of IHP in this case was 1.4 ± 0.1 mm Hg, and LF from the liver was 22.8 ± 3.5 μl/min·100 g. At this level of VP during venous loading of 1, 2, and 3 mm Hg, the corresponding values of CSF were 0.466 ± 0.055, 0.398 ± 0.044, and 0.399 ± 0.047 ml/min·mm Hg·100 g, with a mean value of 0.421 ± 0.029 ml/min·mm Hg·100 g. When VP was 2 mm Hg, PAP and PPP were 119 ± 4 and 13.9 ± 0.9 mm Hg respectively and did not differ significantly from their values when VP was 0 mm Hg. IHP was 3.3 ± 0.1 mm Hg ($p < 0.001$) and LF was 41.8 ± 5.7 μl/min·100 g ($p < 0.05$). Under these conditions, when the venous pressure load was 1, 2, and 3 mm Hg, CSF was 0.480 ± 0.052, 0.497 ± 0.042, and 0.442 ± 0.043 ml/min·mm Hg·100 g respectively, not significantly different from its values at the initial VP = 0 mm Hg.

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When the pressure in the hepatic veins was 4.0 mm Hg, PAP was 115 ± 6 mm Hg whereas PPP was 14.9 ± 1.2 mm Hg (in both cases $p > 0.05$ when compared with values observed when VP = 0 and VP = 2 mm Hg). The value of IHP was 5.4 ± 0.1 mm Hg ($p < 0.001$), whereas LF was 57.6 ± 8.6 μl/min·100 g ($p > 0.05$ when compared with the value of LF when VP = 2 mm Hg, and $p < 0.01$ when compared with the value of LF when VP = 0 mm Hg). At this level of VP, CSF was 0.461 ± 0.070, 0.506 ± 0.060, and 0.454 ± 0.053 ml/min·mm Hg·100 g (on average 0.474 ± 0.034 ml/min·mm Hg·100 g) with a venous pressure load of 1, 2, and 3 mm Hg respectively, i.e., not significantly different from values of CSF at VP = 0 and VP = 2 mm Hg.

The results show that CSF was dependent neigther on the initial level of VP (0-4 mm Hg) nor on the venous pressure load (1-3 mm Hg).

These experiments showed that IHP is directly proportional to the VP level in the liver: the transmission factor of VP to IHP was 100%, evidence of the decision role of the postcapillary resistance in the formation of the intrasinusoidal pressure level.

Comparison of the rates of LF, IHP, and CSF in the liver at different levels of VP shows that the principal parameter determining LF and, consequently, the volume of trans-sinusoidal movement of fluid in the liver, is IHP, and this is confirmed by data in the literature [8]. The area of the exchange surface and permeability of the microvascular walls in the liver had no effect, under these circumstances, on the lymph flow, as is shown by the constancy of CSF at different levels of VP. This fact reinforces the previous conclusion that permeability of the walls of the exchange microvessels in the liver is unchanged in the pressure of hemodyamic shifts [3].

An increase in pressure in the hepatic veins not only leads to an increase in IHP, but is also reflected in PPP; the increase in the portal pressure was 45-50% (0.9-1.0 mm Hg) of the venous load. Meanwhile, there are data in the literature showing 80% transmission of a change in VP to PPP [5]. Reduction of PAP observed experimentally in response to an increase in the initial level of VP from 0 to 4 mm Hg is evidently a manifestation of a venoarterial reflex; some workers, moreover, observed a considerable increase (up to 200%) of PAP in response to elevation of the VP level above 5 mm mm Hg [5, 10]. The character of changes in PAP and PPP in response to elevation of the VP level does not rule out the possibility of self-regulation in the hepatic vessels, for which evidence has been obtained [7].

Thus within the limits of physiological levels of VP (0-4 mm Hg) the hydrostatic pressure in the sinusoids, by contrast with CSF, depends on the pressure in the hepatic veins and determines the rate of lymph formation and the velocity of the lymph flow in the liver.

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