SOME ASPECTS OF THE PORTAL CIRCULATION IN THE LIVER IN SHOCK

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The study of the blood flow in the portal vein, especially in its intrahepatic course, is essential to the analysis of the hemodynamic disturbances in traumatic shock. A considerable volume of blood is retained in this part of the blood stream, and the terminal branches of the portal vein possess high vasomotor activity, while the portal system is a powerful reflexogenic zone [3].

There are few reports in the literature of the state of the portal blood flow in the liver during shock [6, 8, 13]. They describe a decrease in the blood flow through the portal vein in the liver, and little variation in the portal pressure in cases of shock following hemorrhage. Constriction of the portal system of vessels and the hepatic vein in this form of shock is also described [8].

The workers cited above were guided by the conception, widely held abroad, that blood loss is of decisive importance in the genesis of traumatic shock [5]. Nevertheless, it must now be regarded as firmly established that the leading role in the development of this process belongs to changes in the functional state of the central nervous system and the associated disturbances of the endocrine balance, the circulation and the respiration, and abnormalities of metabolism.

Since the most important disturbances of the bodily functions in hemorrhagic shock are due to oligemia, we undertook the study of the blood flow in the portal venous system in a form of shock characterized by a minimal decrease in the mass of the circulating blood. This requirement was satisfied by shock caused by stimulation of the sensory nerves of an animal by a nontraumatic electric current.

EXPERIMENTAL METHOD

Experiments were conducted on 18 cats of various breeds and of both sexes, weighing from 3 to 4.5 kg. Shock was induced by stimulation of the animal's right paws with a pulsed current of intensity 100 mA, the duration of the stimuli being 0.08 msec and their frequency 4 imp/sec. During the experiments the arterial pressure, pulse and respiration were recorded on a kymograph, and observations were kept on the animal's condition and its reaction to stimulation. The portal blood flow was estimated by measuring the pressure in the portal vein and the posterior vena cava at the level of the hepatic sinus, and the portocaval gradient was calculated.

The pressure was measured by means of a twin-capillary, single-scale saline manometer, through polyethylene catheters 0.8-1.0 mm in diameter, introduced under local anesthesia into the portal vein through one of the mesenteric veins, and into the posterior vena cava through the left femoral vein. In a series of experiments the portal vein was examined repeatedly by contrast roentgenography, during injection of 2 ml of a 50% solution of cardiotrast into it through the catheter. The hypotensive action of the cardiotrast, as described by some writers [8], was not observed. The depth of the shock was assessed by comparing the arterial pressure, pulse, respiration, and the animal's reaction to stimulation. Four phases of shock were distinguished: erectile, torpid (1st and 2nd), and terminal [1, 2, 4].

EXPERIMENTAL RESULTS

The pressure in the posterior vena cava and portal vein of animals fixed to the bench varied within a wide range: in the posterior vena cava from 70 to 120 mm water, and in the portal vein from 105 to 180 mm water. The mean pressure in the posterior vena cava was 94 ± 3.4 mm water, and in the portal vein 144 ± 7.6 mm water. The portocaval gradient varied from 40 to 60 mm water (mean 50 ± 4.9).

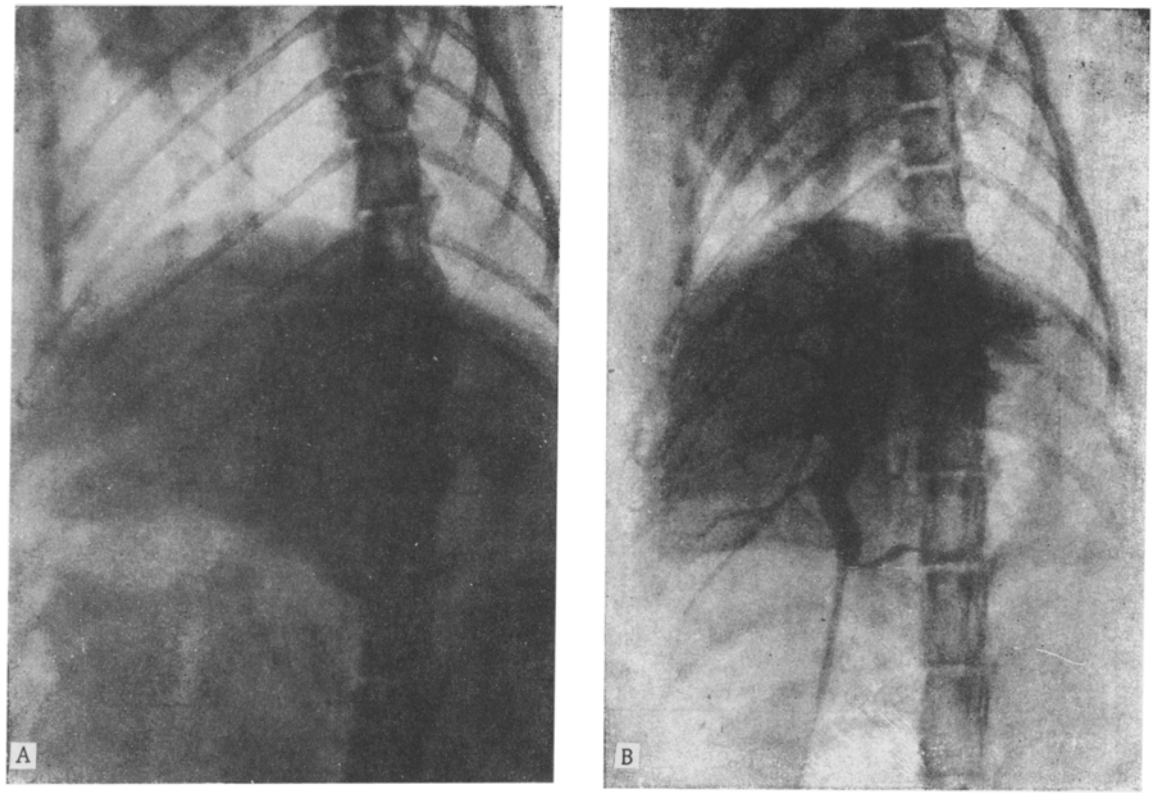

Fig. 1. Roentgenograms of the liver of a cat during injection of cardiotrast into the portal vein. A) Before shock; B) during shock (second torpid phase).

Pressure in the Portal Vein and Posterior Vena Cava and Portocaval Gradient during Shock Compared with Changes in the Arterial Pressure, Pulse, and Respiration (mean values M ± m)

Criterion	In initial state	During shock			
		erectile phase	torpid phase		terminal phase*
			1st	2nd	
Respiration (per min)	92 ± 10.6	51 ± 2.6	88 ± 9.0	34 ± 3.9	Periodic or terminal
Pulse (per min)	198 ± 3.3	246 ± 12.0	204 ± 7.0	157 ± 8.6	60 ± 8.9
Arterial pressure (in mm Hg)	103 ± 3.5	127 ± 5.4	65 ± 5.6	38 ± 1.7	20 ± 1.7
Pressure in posterior vena cava (in mm water)	94 ± 3.4	192 ± 19.0	95 ± 9.3	83 ± 10.7	74 ± 6.8
Pressure in portal vein (in mm water)	144 ± 7.6	277 ± 27.6	178 ± 19.8	156 ± 15.3	134 ± 17.6
Portocaval gradient (in mm water)	50 ± 4.9	85 ± 14.7	83 ± 15.1	73 ± 11.8	60 ± 16.1

*All the values for the terminal phase were determined at a time when the respiration had become periodic (Kussmaul's type) or terminal.

Immediately after stimulation, in the erectile phase of shock, characterized by elevation of the arterial pressure, quickening of the pulse, and slowing of respiration, a considerable increase was observed in the pressure in the posterior vena cava, to 192 ± 19 mm water (significance of difference from initial value $P < 0.001$), together with an even greater increase in the portal pressure to 277 ± 27.6 mm water (significance of difference $P < 0.001$). In this phase the value of the portocaval gradient also showed a significance increase to 85 ± 14.7 mm water ($0.02 < P < 0.05$; see table).

This sharp increase in the pressure in the portal vein, with a simultaneous increase in the portocaval gradient, which most completely [8] reflects the state of the portal blood flow, could be attributed both to an increase in the

resistance in the portal venous system and to an increase in the mass of blood contained in it on account of drainage of the blood depots. The adrenalinemia and increased tone of the sympathetic division of the nervous system, characteristic of this phase of shock, invariably [9, 10, 12] lead to an increase in the portal pressure.

In the torpid phase, especially at its beginning (first torpid phase), when the arterial pressure fell to two-thirds the initial value, the portal pressure was still higher than usual, although it varied in different animals fairly widely; the pressure in the posterior vena cava came close to its initial values, so that the portocaval gradient remained fairly high (83 ± 15.1) and significantly greater than its initial value ($P = 0.05$).

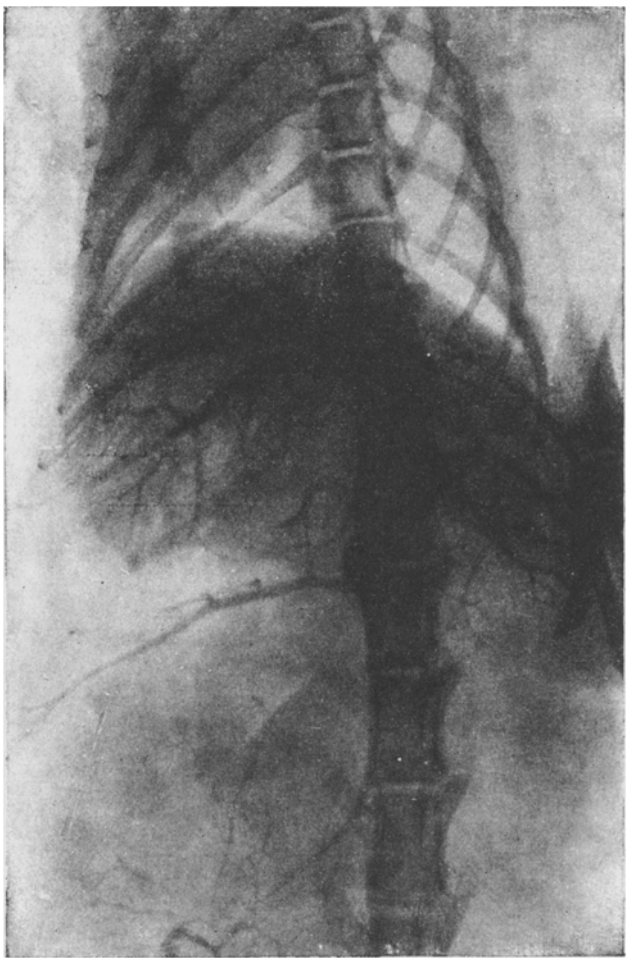

Fig. 2. Angiogram of the portal vein of a cat in the terminal phase of shock. Retrograde filling of the extrahepatic branches of the portal vein can be seen.

At the end of the torpid phase of shock, when the arterial pressure was low (about one third the initial value), the pressure in both the posterior vena cava and the portal vein remained high, and was not significantly different from the control figures; the portocaval gradient was also high (73 ± 11.8), although the excess over the usual value was not quite significant ($0.05 < P < 0.01$). The high values of the portal pressure and the portocaval gradient in this period of shock could only be explained by an increase in the resistance of the blood flow in the intrahepatic portion of the portal vein. Roentgenograms of the portal vein in the liver taken after injection of cardiotrast into the vein in the living animal showed that whereas in normal conditions all branches of the portal system as far as the tiniest vessels are filled immediately after injection of the contrast medium, so that theliver has the appearance of a homogeneous shadow, in profound shock only the larger branches of the portal vein were filled during the same time interval (Fig. 1). These angiograms may be explained by the considerable resistance to the blood flow in the portal vein and to the slowing of the blood flow, despite the high portal pressure [7].

In the terminal phase of shock, with the appearance of periodic (Kussmaul's type) or terminal respiration the arterial pressure was extremely low. At this period a significant lowering of the pressure in the posterior vena cava to 74 ± 6.8 mm was observed ($0.02 < P < 0.05$). The portal pressure remained at a high level and the portocaval gradient was fairly high (see table).

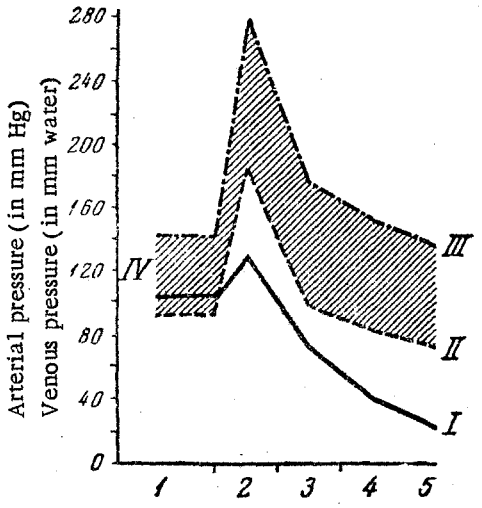

Fig. 3. Scheme of the relationships between the arterial, venous, and portal pressures. 1) Before shock; 2) in the erectile phase; 3) in the 1st torpid phase; 4) in the 2nd torpid phase; 5) in the terminal phase; I) arterial pressure; II) pressure in the vena cava; III) pressure in the portal vein; IV) portocaval gradient.

Other evidence in favor of the high resistance to the blood flow in the terminal ramifications of the portal system in shock of this depth was the fact that in some experiments the angiograms after injection of cardiotrast into the portal vein revealed retrograde movement of the dye and filling of the extrahepatic branches of the vein (Fig. 2). A similar phenomenon has been observed in hemorrhagic shock [8]. The explanation of these facts must be sought in those experiments [11] in which a reflex spasm of the hepatic vessels was observed during a lowering of the blood pressure in the region of the carotid sinuses, which may also take place in shock. The observed variations in the pressure in the portal vein and inferior vena cava and in their relationship to the changes in the arterial pressure are illustrated schematically in Fig. 3.

Shock caused by a pulsed electric current was thus characterized by a high portal pressure and a considerable portocaval gradient, resulting from the high resistance in the intrahepatic division of the portal blood stream.

SUMMARY

A study was made of the circulation in the portal vein of cats during shock resulting from current. To assess the circulation the author measured the blood pressure in the portal and posterior vena cavas, estimated the portocaval gradient and took repeated roentgenograms of the portal vein with the cardiotrast contrasting thereof. As revealed, the pressure in the portal vein and in the portocaval gradients was rather high even at the late phases of shock, when the arterial pressure had already undergone a considerable drop. This may be attributed to a marked resistance to the blood flow in the intrahepatic portion of the portal circulation, which is confirmed by the roentgenograms.

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