THE HEPATIC CIRCULATION IN TRAUMATIC SHOCK

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It is shown by the method of contrast x-ray cinematography that in cats with traumatic shock the linear and volume velocity of the portal blood flow in the liver is decreased and the arterial blood flow slightly increased. This is brought about by changes in resistance to the blood flow in different parts of the hepatoportal system.

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Although a number of investigators have described the typical features of the hepatic circulation in hemorrhagic [10, 11, 13, 14, 16], traumatic [4-6], and other types of experimental shock [3], many of their aspects are still unclear. Changes in the blood flow in the liver discovered in hemorrhagic shock by the method of contrast angiography [12, 13] are not applicable to traumatic shock, because there is a significant difference between them [1, 2, 6].

In this paper we describe the results of an x-ray cinematographic study of the intrahepatic circulation in experimental traumatic shock.

EXPERIMENTAL METHOD

Experiments were carried out on 11 cats weighing 3.0-4.5 g. From 7 to 10 days before the experiments with vasography, a catheter was introduced into the portal vein of the animals [7]. The aorta was catheterized under local anesthesia (0.25% procaine solution) immediately before the experiment. For the experiments with angiocinematography, the unanesthetized animals were fixed to a frame 30-40 min before it began.

A "Philips" image intensifier of an "Arriflex" camera was used in the investigation. Type RF-3 35-mm film with a sensitivity of 900-1100 reciprocal roentgens was used. The filming speed for observations on the blood flow in the portal vein was 12 frames/sec and for investigation of the blood flow in the hepatic artery 25 frames/sec. Conditions: voltage 42-45 kV, current 8-10 mA, focal length 370 mm. The contrast material (70% diodone solution) was injected in a volume of 3 ml through the appropriate catheter.

The experiments with shock were carried out 4-7 days after the controls. Shock was produced by Cannon's method. Its depth and period of development were assessed from changes in the pulse, arterial pressure, and respiration.

The kinoangiograms were analyzed by means of a "Microphot" apparatus. The linear velocity of blood flow in the main blood vessels was estimated from measurement of the distance passed by the contrast material along a particular vessel and data for the time between two corresponding frames. To calculate the volume velocity of the blood flow the formula recommended by Ardran [9] was used. In addition, the time taken to fill particular vessels, their branches, and the lobes of the liver with contrast material was determined (the phases of spread of the material).

The development of traumatic shock followed the same course as was described previously [4, 6]. By noting differences between the various periods of the torpid phase of traumatic shock, the investigation of the hepatic blood flow could be carried out in a period which we described as the period of stabilization.

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TABLE 1. Time of Spread of Contrast Material (in sec) along Portal and Arterial Systems of the Liver in Shock (M ± m)

Series of experiments	Injection into Portal vein					Injection into arterial system		
	filling of right medial lobar branch	filling of left lateral lobar branch	beginning of filling of sinusoids	passage into system of hepatic vein	liberation of sinusoids from contrast material after end of injection	beginning of filling of sinusoids	passage into system of hepatic vein	liberation of sinusoids from contrast material after end of injection
Shock	2.5 ± 0.16	4.3 ± 1.20	4.3±0.60	8.5±2.10	16.6±1.80	0.9±0.06	1.2±0.0	2.4±0.03
Control	1.8 ± 0.28	2.3 ± 0.32	2.4±0.33	6.0±1.80	9.2±1.40	1.0±0.06	1.6±0.18	2.5±0.65
P	0.05	>0.1	<0.02	>0.3	<0.01	>0.2		

TABLE 2. Volume Velocities of Blood Flow in Liver Vessels and Resistance of Different Segments of Hepatoportal System in Traumatic Shock (M ± m)

Series of experiments	Volume velocity of blood flow (in ml/min)			Fraction of arterial blood flow (in %)	Resistance of vessels in 10^3 dyn · sec · cm^{-5})			Volume velocity of blood flow (in ml/min/100 g wt. of organ)		
	portal vein	hepatic artery	through whole organ		portal vein	hepatic artery	hepato-portal system	portal vein	hepatic artery	through whole organ
Shock	10.9±1.26	15.2±2.33	26.0±3.62	58.5±1.00	32.43	340.0	202.0	10.2 ± 1.33	14.3±2.33	24.5±3.66
Control	35.8±5.48	8.3±1.64	44.6±7.56	19.6±3.56	9.4	1270.0	238.0	33.4 ± 4.85	7.7±1.20	41.7±6.24
P	<0.01	<0.05	<0.05	<0.01				<0.05	<0.05	

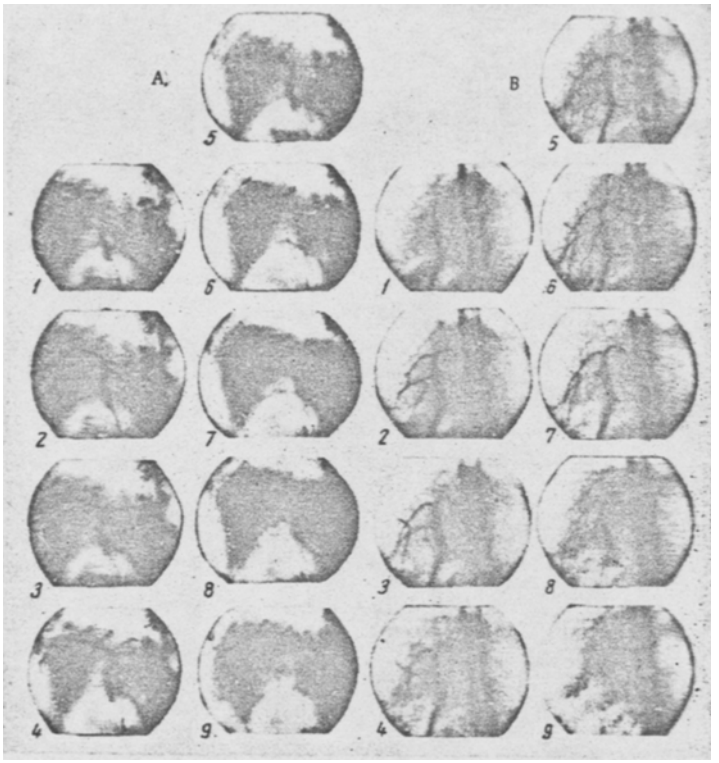

Fig. 1. Spread of contrast material through the vascular system of the liver when injected into the portal vein (in seconds from beginning of injection). A) Control experiments; B) experiments with shock. Frames 1–4) 0.6-3 sec; frames 5–6) 3.7-5.3 sec; frames 7-9) 5.9-9 sec (control) and 5.9-21 sec (during shock).

EXPERIMENTAL RESULTS

In the torpid phase of traumatic shock a decrease in linear velocity of the blood flow in the portal vein was found. Development of the sinusoidal (parenchymatous) phase of filling after injection of contrast material to the portal vein was protracted, and it was not so clearly defined as in the control animals (Fig. 1). Meanwhile, the appearance of contrast material in vessels of the portal venous system took place at times not significantly different from those in the control experiments. It was concluded from these facts that contrast material passed from the system of the portal vein into that of the hepatic vein along vascular shunts.

The linear velocity of the blood flow in the hepatic artery differed only slightly during shock from its value in the control experiments. The parenchymatous phase after injection of contrast material into the arterial system under conditions of shock developed after approximately the same time intervals as in the control (Table 1). The liver shadow in this phase of shock was less dense than in the initial experiments.

The total volume of blood flowing through the liver in the torpid phase of shock fell by more than 40% of its usual value, while the arterial fraction of the blood flow increased appreciably. Whereas in the control experiments only about 20% of the blood flowing through the liver entered it through the hepatic artery, in shock the arterial fraction of the blood flow increased to almost 60% (Table 2). Meanwhile, analysis of the kinoangiograms and calculation of the resistance to the blood flow demonstrate that shunting of the blood flow assumed an important place in the system of hepatic artery – hepatic vein (Fig. 2).

From data for the volume velocities of the blood flow and mean values of the arterial pressure (67 ± 8.2 mm Hg), the pressure in the portal vein (112 ± 9.7 mm water) and in the venae cavae (50 ± 3.9 mm water) in the period of stabilization of the torpid phase of shock, the resistance to the blood flow was

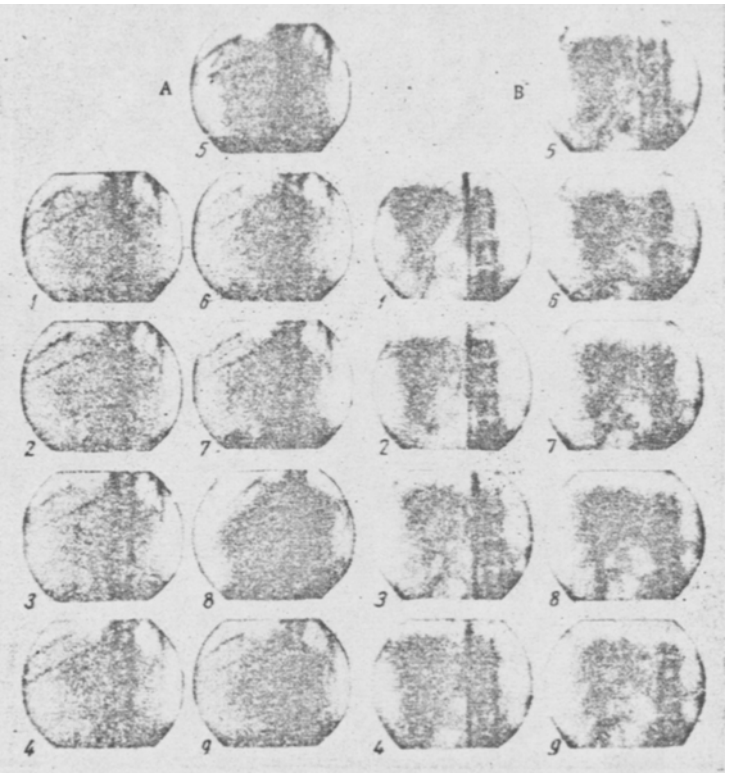

Fig. 2. Spread of contrast material throughout vascular system of liver when injected into aorta (in seconds from beginning of injection). A) control experiment; B) experiments with shock. Frames 1-3) 0.4-0.9 sec; frames 4-5) 1.2-1.6 sec; frames 6-7) 2.0-2.3 sec; frames 8-9) 2.5-2.9 sec.

calculated in vessels of different parts of the hepatoportal system. The resistance of the terminal portion of the portal vein was found to be more than 3 times higher, whereas that in the hepatic artery system was reduced (Table 2).

The results of the present experiments confirmed the hypothesis previously expressed on the basis of indirect evidence, that the arterial fraction of the blood supply to the liver is increased during shock and that the relative resistance of different parts of the hepatoportal system plays an important role in these circulatory changes [6].

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