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CHARACTERISTICS OF THE SODIUM ACCUMULATING CAPACITY OF THE LIVER

A. Ya. Terner and R. I. Aizman

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The liver is known to participate in regulation of water and electrolyte balance not only as a receptor zone [2, 5, 9-12, 14], but also as a depot organ capable of retaining an excess of water and certain ions [1, 3, 7, 11, 12].

This paper describes a study of the sodium-accumulating capacity of the liver during the creation of sodium concentration shifts in the portal system of different magnitude and duration, and an attempt is made to describe this capacity quantitatively.

EXPERIMENTAL METHOD

In the first part of the investigation, consisting of acute experiments on cats anesthetized with chloralose, the aim was to study the ability of the liver to retain Na^+ during the creation of a temporary but considerable shift of the Na concentration in blood from the portal vein. This shift was created by injecting 2 ml/kg body weight of 3% NaCl solution in the course of 1 min through a catheter fixed in one of the small mesenteric veins. Blood samples were taken simultaneously from the portal vein through an angiostomy cannula and from the posterior vena cava above the liver by venupuncture 30 sec after the beginning of injection of the solution. To prevent dilution of blood flowing from the liver the posterior vena cava was clamped below the liver during blood sampling.

The second stage of the work consisted of acute experiments on dogs (anesthetized with pentobarbital), in whose portal vein sodium shifts similar to those developing under normal physiological conditions during absorption of Na^+ from the alimentary tract, were created artificially [11]. The scheme of the experiments was as follows. After control blood samples had been taken from the portal and hepatic veins and a sample of liver tissue also had been removed, an infusion of 2% NaCl solution was given through a cannula introduced into a mesenteric vein, at the rate of 0.5 ml/min•kg for 15 min. During infusion of the solution at the 5th, 10th, and 15th minutes, and 5 min after the end of infusion of the solution, blood and liver tissue samples were taken. The sodium concentration in the blood plasma was determined by flame photometry, and in the liver tissue by extraction of the undefatted dried sample in 0.75 N nitric acid followed by flame photometry of the extract [8].

EXPERIMENTAL RESULTS

In series I there were 14 experiments. As Fig. 1 shows, the sodium concentration in the portal vein at the time of injection of the solution was considerably raised — up to 190 mM,

Laboratory of Histophysiology, Institute of Physiology, Siberian Branch, Academy of Medical Sciences of the USSR, Novosibirsk. Department of Anatomy and Physiology of Man and Animals, Pedagogic Institute, Novosibirsk. (Presented by Academician of the Academy of Medical Sciences of the USSR Yu. I. Borodin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 97, No. 3, pp. 263-265, March, 1984. Original article submitted December 28, 1982.

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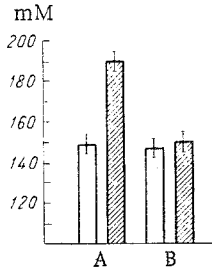

Fig. 1

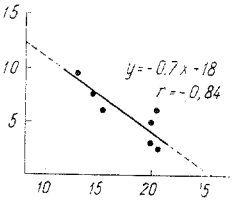

Fig. 2

Fig. 1. Sodium concentration in portal vein (A) and posterior vena cava (B) during infusion of 2 ml/kg body weight of 3% NaCl into portal blood flow of cats. Unshaded columns — before, shaded — during infusion.

Fig. 2. Dependence of sodium accumulation on initial sodium content in liver. Abscissa, initial sodium content in liver (in millimoles/100 g dry weight); ordinate, increase in sodium concentration in liver after infusion of 2% NaCl for 15 min (in the same units). Equation calculated by method of least squares [4].

whereas in blood flowing from the liver its concentration did not change significantly. The small increase in the sodium concentration in blood from the hepatic veins, at a time of considerable concentration shift in the system of the portal vein can be explained as follows: 1) by the ability of the liver to retain the excess of sodium; 2) by dilution of blood in the portal vein with blood supplied by the hepatic artery. To rule out the role of this last factor calculations were carried out using the Fick principle, according to which the magnitude of the portal blood flow is given by

$$V_p = \frac{C_s \cdot V_s - C_{p_2} \cdot V_s}{C_{p_2} - C_{p_1}},$$

where V_p is the velocity of the portal flow, in ml/min, C_s the sodium concentration in the injected solution, in mM, V_s the rate of injection of the solution, in ml/min, C_{p_1} the sodium concentration in the portal vein before injection of the solution, and C_{p_2} its concentration in the portal vein during injection of the solution, in mM. Substituting numerical values of the parameters in the formula we obtain: V_p = (513•2 − 190•2)/(190 − 148) = 15.4 ml/min. The arterial blood flow in the liver is about one-quarter of the venous flow [6] or, in this case, 3.85 ml/min. The sodium concentration after mixing of venous and arterial blood ought to be:

$$C_{v.c} = \frac{C_a \cdot V_a + C_{p_2} \cdot V_p}{V_a + V_p},$$

where $C_{v.c.}$ is the sodium concentration in blood from the posterior vena cava in mM, C_a the sodium concentration in the arterial blood, in mM, and V_a the velocity of the arterial blood flow in ml/min. Consequently: $C_{v.c.}$ = (146•3.85 + 190•15.4)/(15.4 + 3.85) = 181 mM. The dilution factor thus plays a negligible role in damping the Na concentration shift.

To estimate the retention capacity of the liver quantitatively the experiments of series II were carried out, and their results are given in Table 1. It will be clear that during prolonged infusion of hypertonic NaCl solution, despite an increase in sodium concentration in blood of the hepatic veins, effective retention of the cation takes place in the liver. Calculations showed that during the 15 min of infusion the animals received 18.5 millimoles sodium per 100 g dry weight of liver tissue (assuming that the weight of the liver is about 50 g/kg body weight [13] and the water content is 72% [11]). Toward the end of infusion of the solution, on average 4.1 millimoles sodium per 100 g was retained in the liver, or 22% of the injected dose. In some experiments, however, the quantity of retained cation varied between 2.5 and 9.4 mmoles/100 g.

To determine the causes of these fluctuations, dependence of the increase in sodium concentration in the liver on its initial content in the organ was analyzed. It will be clear from Fig. 2 that negative correlation exists between these values, evidence that the quantity

TABLE 1. Changes in Sodium Concentration in Blood of Portal and Hepatic Veins and in Liver Tissue during Infusion of 2% NaCl Solution into Portal Blood Flow of Dogs (M ± m, n = 7)

Time, min	Sodium concentration in portal vein, mmoles/liter	P	Sodium concentration in hepatic veins, mmoles/liter	P	Increase in sodium concentration in liver tissue, mmoles 100 g	P
Control	146±1	—	143±2	—	—	—
5	155±2	<0,01	148±2	<0,1	1,5±0,7	<0,05
10	155±2	<0,01	149±2	<0,05	3,5±1,4	<0,05
15	155±2	<0,01	151±2	<0,01	4,1±1,1	<0,01
20	—	—	149±1	<0,02	4,0±1,1	<0,02

of sodium accumulated in the liver is determined by its original concentration in that organ. Liver tissue can retain up to 62% of the injected load if the original concentration of the cation is about 10 mmoles/100 g, but virtually no sodium is retained if its original concentration is 25.6 mmoles/100 g body weight.

Thus, on the one hand, the liver plays the role of damper, preventing the development of hyperosmia and hypernatriemia in the general circulation during absorption of sodium from the alimentary tract; on the other hand, the mechanism limiting sodium accumulation in the liver is evidently based on the fact that sodium, having accumulated in the liver up to a definite concentration, induces reflex stimulation of sodium excretion in the urine [9, 11], and to prevent hyponatriemia, the cation passes from the liver into the general circulation.

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