# EFFECT OF PORTALIZATION OF BLOOD DRAINING FROM THE ADRENALS ON THE DEVELOPMENT OF SECONDARY HYPERALDOSTERONISM AND ASCITES

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UDC 616.453-008.61+616.381-003.217/-092:616.149-018.5

Constriction of the inferior vena cava above the diaphragm, accompanied by portocaval venous transposition, did not cause the formation of edema and ascites. At the same time, normal osmoregulatory responses to injection of hypertonic sodium chloride solution into the hepatic circulation were observed. The normal course of osmoregulatory processes may be attributed to correction of the secondary hyperaldosteronism by portalization of blood draining from the adrenals and by metabolization of the aldosterone in the liver.

It was shown previously [1] that depression of the sodium-diuretic component of the hepatic osmoregulatory reflex in ascites is due to blocking of the sodium-diuretic action of the neurohypophyseal hormones by competitive influences of aldosterone on the tubular reabsorption of sodium.

The object of the present investigation was to study whether the osmoregulatory reflexes can be restored by abolishing the secondary hyperaldosteronism as the result of portalization of blood draining from the adrenals.

## EXPERIMENTAL METHOD

Chronic experiments were preceded by three-stage operative preparation of the dogs. In the first stage portocaval venous transposition was carried out. The portal vein was anastomosed with the subhepatic portion of the inferior vena cava and the proximal part of the portal vein was connected to the distal end of the inferior vena cava. Blood from both adrenals, together with blood from the inferior vena cava, was thus directed into the liver. In the second stage, fistulae were formed into the stomach and urinary bladder. In the third stage, the inferior vena cava was constricted above the diaphragm to half its lumen in order to produce ascites. At the same time, or during the second stage of preparation, angiostomy of the inferior vena cava was performed by introducing a catheter through the femoral vein. The chronic experiments were began after 7-10 days. The dog was fixed to a frame and a stable diuresis established by administration of water into the stomach (through the fistula). Against the background of this diuresis, 5 ml of 3-5% sodium chloride solution was injected over a period of 30 sec through the catheter inserted into the inferior vena cava, thus stimulating the osmoreceptor system of the liver. Before and after injection of the hypertonic sodium chloride solution, the diuresis was measured every 5 min, and the concentrations of sodium and endogenous creatinine determined in these samples of urine. Similar tests were carried out on the blood plasma taken at the beginning of the experiment. From the results obtained, the glomerular filtration and tubular reabsorption, the sodium load on the nephron, and sodium reabsorption were calculated. Similar experiments on healthy dogs and dogs with experimental ascites were used as the controls.

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TABLE 1. Changes in Diuresis and Sodium Excretion in Response to Osmotic Stimulation of the Liver (M ± m)

| Index | Normal (n = 20) | Ascites (n = 36) | Transposition + ascites |
|---|---|---|---|
| Duration of antidiuretic reflexes (in min) . . . . . | 54.2 ± 2.8 | 47.0 ± 3.2 | 45.9 ± 2.3 |
| Maximal decrease in diuresis (in % of initial) . . . . . | 61.2 ± 5.4 | 49.0 ± 3.9 | 64.5 ± 3.4 |
| Change in glomerular filtration (in % of initial) . . . . | 87 | 79 | 94 |
| Duration of sodium-excretory responses (in min) . . . | 40 ± 9 | – | 52 ± 1.7 |
| Sodium excretion (in % of initial) . . . . . | 306 ± 18.0 | –* | 240 ± 39 |

*In five of the 36 experiments, this index had the value of 110 ± 6.0%.

TABLE 2. Aldosterone Concentration (in μg/100 ml) in Blood Plasma from Sub- and Suprahepatic Portions of Inferior Vena Cava

| Series of experiments | Dog No. | Inferior vena cava | |
|---|---|---|---|
| | | below liver | above liver |
| Normal dogs | 1 | 10.0 | 6.0 |
| | 2 | 5.0 | 3.0 |
| Portocaval transposition | 3 | 10.0* | Less than 1.0 |
| | 4 | 11.0* | Less than 1.0 |
| | 5 | 9.0 | Less than 1.0 |
| Reversed Eck–Pavlov fistula | 6 | 4.0 | 2.0 |
| | 7 | 25.0 | 4.0 |

*Investigation carried out after constriction of inferior vena cava above diaphragm.

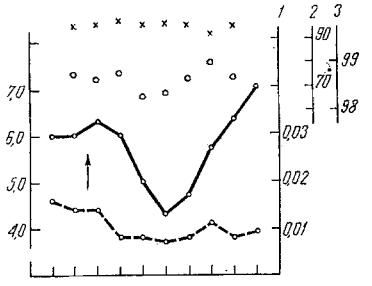


Fig. 1

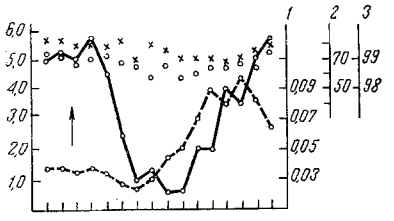


Fig. 2

Fig. 1. Effect of osmotic stimulation of the liver on diuresis and sodium excretion in experimental ascites. Depression of sodium-excretory component of osmoregulatory reflex from liver. Abscissa, time in 5-min intervals. Ordinate, on the left: diuresis (in ml/m²/min). Ordinate on the right: 1) sodium excretion (in meq/m²/min); 2) glomerular filtration (in ml/m²/min, circles); 3) tubular reabsorption of sodium (in percent of sodium load on nephron, crosses). Continuous line shows dynamics of diuresis, broken line dynamics of sodium excretion. Arrow indicates time of osmotic stimulation of the liver.

Fig. 2. Effect of osmotic stimulation of the liver on diuresis and sodium excretion after portocaval venous transposition and constriction of inferior vena cava above the diaphragm. Sodium-excretory response is clearly visible. Legend as in Fig. 1.

To judge the effectiveness of portalization of the blood draining from the adrenals, venous catheterization was carried out on healthy dogs, on dogs with portocaval transposition, and dogs with a reversed Eck–Pavlov fistula. Under x-ray control, in dogs anesthetized with hexobarbital blood was taken from the inferior vena cava at the level of its anastomosis with its portal vein and from the supradiaphragmatic portion of the inferior vena cava. The aldosterone concentration in the blood samples thus obtained was determined by thin-layer chromatography on silica gel. In the process of venous catheterization the pressure in the portal vein, in the abdominal part of the inferior vena cava, and also above and below the stenosing ring applied to the supradiaphragmatic portion of the inferior vena cava was measured.

## EXPERIMENTAL RESULTS AND DISCUSSION

Osmotic stimulation of the liver during experimental ascites and also after portocaval transposition with constriction of the inferior vena cava above the diaphragm evoked antidiuretic responses essentially indistinguishable from those observed in healthy dogs (Table 1).

Analysis of the dynamics of the sodium excretion showed that portocaval venous transposition, if performed before constriction of the inferior vena cava above the diaphragm, enabled the osmoregulatory reactions from the liver to be maintained, with respect to two components: diuresis and sodium excretion. The maintenance of normal osmoregulatory reflexes from the liver was accompanied by absence of ascites or edema in all four dogs in which the attempt to produce ascites was made after creation of the portocaval transposition. Graphs of typical experiments demonstrating the depression of sodium excretion in ascites and the distinct sodium excretory reaction after portalization of blood draining from the adrenals are illustrated in Figs. 1 and 2.

The pressure in the portal vein of the dog with constriction of the inferior vena cava of the diaphragm and with portocaval transposition was increased by 3-6 mm Hg, and the pressure in the abdominal part of the inferior vena cava was increased by 1-4 mm Hg. This indicated the presence of postsinusoidal hypertension in the experimental dogs – an essential condition for the development of ascites [4, 5]. In addition, a pressure gradient could be detected below and above the ring stenosing the inferior vena cava, amounting to 3-4 mm Hg.

Preservation of the normal osmoregulatory reflexes in the dogs with the model of experimental ascites described above was due to portalization of blood draining from the adrenals and metabolization of aldosterone in the liver. It is clear from the results given in Table 2 that when blood draining from the adrenals was directed into the liver by portocaval venous transposition or by the reversed Eck–Pavlov fistula, the aldosterone concentration in blood taken from the thoracic portion of the inferior vena cava was considerably reduced. In this way the competitive effect of aldosterone on the tubular reabsorption of sodium, which is reduced by the action of vasopressin and oxytoxin, liberated by the neurohypophesis in response to osmotic stimulation of the liver [2], were abolished. The role of aldosterone in the osmoregulatory reflexes is to create the necessary background for the action of neuropeptides on sodium transport in the renal tubules. Exclusion of this background [3], or its intensification (hyperaldosteronism), depresses the sodium-excretory component of the osmoregulatory reflexes.

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