

SALURESIS IN EXPERIMENTAL MYOCARDIAL ISCHEMIA

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In experiments on unanesthetized dogs a decrease in water diuresis and an increase in sodium and potassium excretion were found in acute myocardial ischemia.

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On the basis of the transmineralization phenomenon in the ischemic myocardium a disturbance of the total water and salt balance of the body can be postulated in acute myocardial ischemia. The possibility of such a disturbance is indicated by changes in concentrations of electrolytes in the blood of the sinus venosus of the heart and in the peripheral blood cells [1]. We know of only one investigation dealing with the state of the water-excretory function of the kidneys in experimental myocardial ischemia [2].

The object of the present investigation was to study certain homeostatic functions of the kidney, principally the excretion of sodium and potassium, in experimental acute myocardial ischemia.

EXPERIMENTAL METHOD

Chronic experiments were performed on adult noninbred dogs of both sexes after preliminary exteriorization of the ureteric orifices onto the skin of the abdominal wall. Between 8 and 12 days after the first operation temporary Kapron ligatures were passed under the descending branches of the left coronary artery and vein, and their ends were brought out beneath the skin of the chest wall. Three or four days after the second operation water (50 ml/kg body weight) was introduced into the stomach of the unanesthetized animal through a tube and 2% insulin solution was injected by intravenous drip. After the collection of two control 10-min samples of urine separately from each kidney, blood was taken from the jugular vein and the ECG recorded in three standard leads. The coronary circulation was then disturbed and the indices mentioned above recorded again during the next 60-70 min.

In the experiments of series I (13 experiments on 9 dogs) the effect of temporary myocardial ischemia on kidney function was studied. Ischemia was produced by applying traction to the Kapron loop passed beneath the coronary vessels by means of a weight of between 100 and 200 g for 20 min, after which the weight was removed and the blood flow restored.

In series II (11 experiments on 11 dogs) the effect of prolonged myocardial ischemia on kidney function was studied. Ischemia was produced by drawing the temporary Kapron thread ligature tight without subsequent restoration of the blood flow.

The control series consisted of 20 experiments on 19 dogs, which included 5 experiments in which traction was applied to a ligature attached to the myocardium near the usual place of ligation of the blood vessels.

In all the experiments the inulin concentration in the urine and plasma was determined by a resorcin method and the sodium and potassium concentration by flame photometry. From the results of these measurements the inulin concentration index, reflecting the degree of reabsorption of water in the tubules, was calculated, and the inulin clearance, reflecting the glomerular filtration, and the excretion of sodium and potassium were determined.

EXPERIMENTAL RESULTS AND DISCUSSION

In the experiments of both series I and series II myocardial ischemia was accompanied by marked behavioral reactions: The dogs whimpered, moved about restlessly, sometimes vomited, and defecated

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TABLE 1. Renal Excretion of Water and Electrolytes by Dogs during Chronic Experiments before and during Acute Myocardial Ischemia Lasting 20 min ($M \pm \sigma$)

Index of renal function	Control period	During myocardial ischemia	P
Diuresis (in ml/min)	7.6 \pm 1.4	5.1 \pm 1.5	< 0.02
Inulin concentration index	12.5 \pm 3.8	19.7 \pm 4.2	< 0.02
Inulin clearance (in ml/min)	79.9 \pm 6.1	77.4 \pm 6.3	< 0.7
Sodium excretion (in μ eq/min)	100.3 \pm 22.0	145.6 \pm 27.3	< 0.01
Sodium excretion (in % of filtration charge)	1.27 \pm 0.31	2.01 \pm 0.33	< 0.01
Potassium excretion (in μ eq/min)	35.9 \pm 10.7	50.9 \pm 13.0	< 0.05

TABLE 2. Renal Excretion of Water and Electrolytes by Dogs in Chronic Experiments before and during Acute Irreversible Myocardial Ischemia ($M \pm \sigma$)

Index of renal function	Control period	During myocardial ischemia	P
Diuresis (in ml/min)	5.8 \pm 1.7	2.5 \pm 1.0	0.01
Inulin concentration index	15.2 \pm 6.9	32.3 \pm 9.4	0.01
Sodium excretion (in μ eq/min)	102.9 \pm 20.7	151.7 \pm 23.2	0.01
Inulin clearance (in ml/min)	84.1 \pm 12.6	77.7 \pm 10.1	0.4
Sodium excretion (in % of filtration charge)	0.94 \pm 0.28	1.71 \pm 0.36	0.01
Potassium excretion (in μ eq/min)	33.5 \pm 6.4	46.8 \pm 7.4	0.02

involuntarily. Four dogs died during the experiment 20–30 min after disturbance of the coronary blood flow. In all experiments ECG changes characteristic of coronary insufficiency were observed.

Temporary myocardial ischemia was accompanied by inhibition of water diuresis on account of an increase in the reabsorption of water in the tubules, as shown by an increase in the inulin concentration index (Table 1).

Characteristically the rate of glomerular filtration, as shown by the inulin clearance, did not change significantly. A particularly noteworthy fact was that the excretion of electrolytes – both sodium and potassium – increased during the oliguric reaction. The increase in the loss of sodium through the kidneys took place on account of the tubular component, because the sodium filtration charge was unchanged. In irreversible myocardial ischemia the inhibition of diuresis was still more marked (Table 2).

The retention of water and increased sodium excretion also took place on account of tubular effects, because the inulin clearance showed no significant change although it had a tendency to diminish.

No significant changes were found in the sodium/potassium ratio in the urine and plasma or in the concentrations of these electrolytes in the plasma. In control experiments no significant changes were found in the kidney functions tested.

The results show that the changes in kidney function were similar in type in both series of experiments although the degree of the changes in some indices were slightly lower during temporary and during prolonged myocardial ischemia. This difference may be attributed most probably to the more severe ischemia of the myocardium in the experiments of series II. In most experiments the most severe changes in kidney function were observed in the period from the 10th to the 30th minute after disturbance of the coronary blood flow. After 50–70 min of observation the kidney function indices were nearly always restored to their initial level.

In the experiments terminating in death of the animals, myocardial ischemia was accompanied by a very sharp rise in excretion of electrolytes (Fig. 1). In many experiments a relationship was observed between the severity of the ECG changes and the increase in saluresis.

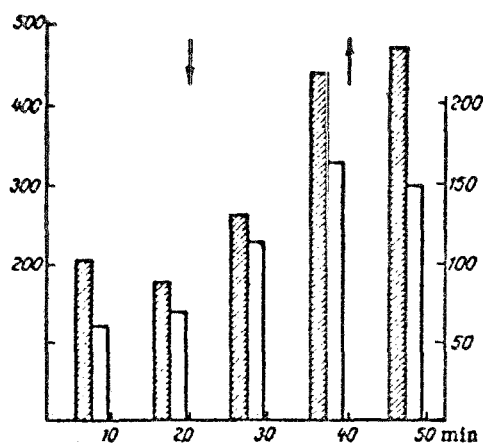


Fig. 1. Renal excretion of sodium (shaded columns) and potassium (unshaded columns) during temporary (marked by arrows) myocardial ischemia. Abscissa: time (in min). Ordinate: excretion of sodium (left) and potassium (right) in $\mu\text{eq}/\text{min}$.

The decrease in diuresis during myocardial ischemia is the result of increased tubular reabsorption of water, as shown by the increase in the inulin concentration index. In my opinion the most interesting discovery is the decrease of the tubular reabsorption of sodium and potassium, one of the most important functions of the kidney in connection with retention of electrolytes in the body, found in experimental myocardial ischemia. This phenomenon could hardly be the result of a disturbance of the distribution of electrolytes in the infarcted heart muscle or of a change in aldosterone secretion during myocardial ischemia, for in these cases we did not observe a simultaneous and rapid increase in the excretion of both these ions.

LITERATURE CITED

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