HEMODYNAMICS, OXYGEN SUPPLY, AND GLUCOSE CONSUMPTION IN THE KIDNEY DURING ACUTE OCCLUSION OF ITS ARTERY AND IN THE EARLY POSTISCHEMIC PERIOD

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After occlusion of the renal artery the blood flow is maintained in the ischemic kidney by collateral vessels, but the oxygen and glucose utilization per unit volume of renal blood flow is considerably increased. During the 24 h immediately after removal of the occlusion to the renal artery after 4 h of ischemia of the kidney the circulation in the organ is not completely restored. The renal blood flow is maintained at a level somewhat lower than originally and the resistance of the renal vessels remains above normal.

The writers have shown previously [2] that despite its relatively small size the collateral blood flow in the kidney has a distinctly favorable effect on the outcome of acute renal ischemia.

The object of this investigation was to study some of the mechanisms possibly promoting a favorable outcome of acute ischemia of the kidney when the collateral renal circulation is preserved.

EXPERIMENTAL METHOD

Ischemia of the left kidney for 4 h was produced in 19 dogs under pentobarbital anesthesia (25 mg/kg) by applying a rubber-covered hemostat to the renal artery leaving the main sources of the collateral renal blood flow intact. The blood flow [2], vascular resistance [1], and the consumption of oxygen and glucose were determined in the test kidney, exteriorized through a lumbar incision. The pressure in the abdominal aorta was recorded with an electromanometer and the venous pressure in the left renal vein by Waldman's apparatus; the blood oxygen saturation was measured with the OSM I apparatus (Radiometer, Denmark) and the parameters of the acid-base balance with the ABC-1 micro-Astrup apparatus (Radiometer, Denmark).

Knowing the blood O_2 saturation and hemoglobin concentration the absolute O_2 content per unit volume of arterial blood and of venous blood flowing from the test kidney could be calculated [6].

Consumption of O_2 and glucose by the kidney was calculated from the arteriovenous difference and the blood flow. The blood glucose concentration was determined by an enzymic method [5]. The values of the renal blood flow and the oxygen and glucose consumption were expressed per 100 g weight of kidney tissue.

EXPERIMENTAL RESULTS

The experimental results given in Table 1 show that throughout the period of occlusion of the main renal artery the blood flow in the ischemic kidney was maintained by collateral vessels, and that O_2 was utilized by the kidney tissue. The arterio-venous difference for O_2 increased considerably under these conditions. By the end of the 4th hour of ischemia it was 5.7 times higher than initially.

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TABLE 1. Indices of Hemodynamics, Acid-Base Balance, and O_2 and Glucose Consumption in the Kidney during Acute Occlusion of Its Artery and in the Early Postischemic Period (M ± m)

Index studied	Original values	Period of ischemia		After ischemia	
		30 min	4 h	15 min	24 h
Index studied	394±24,5	7±0,9‡	6±0,9‡	262±38,6†	240±36,ft
Resistance of renal vessels (in dynes/sec·cm^{-5}·10^3)	21,2±2,74			34,0±4,85*	45,6±7,91†
Arterio-venous difference for O_2 (in vols.%)	2,2±0,28	9,0±1,59‡	12,6±1,26‡	4,5±0,96*	2,1±0,31
O_2 consumption (in μmoles/min/100 g)	360±45,1	30±7,3‡	31±4,7‡	378±69,1	212±39,0*
Arterio-venous difference for glucose (in mg %)	0,5±0,25	39,6±12,18†	52,5±8,22‡	18,3±2,97‡	0,3±0,19
Glucose consumption (in mg/min/100 g)	2,0±1,07	2,5±0,57	2,7±0,31	43,4±8,51‡	1,2±0,48
Buffer base shift (in μmeq/liter)	—2,6±1,59	—11,2±0,96‡	—10,3±1,42†	—6,3±1,26	—2,3±0,54
pH of outflowing blood	7,377±0,010	7,166±0,050†	7,154±0,047†	7,297±0,024	7,381±0,014

*P < 0.05, †P < 0.01, ‡P < 0.001 compared with initial values.

The increase in the extraction of oxygen per unit volume of inflowing blood is evidently an important adaptive response aimed at reducing renal hypoxia. As a result of this response the O_2 consumption of the ischemic kidney was reduced much less proportionately than the volume velocity of the renal blood flow. It must be borne in mind that during occlusion of the renal artery the oxygen consumption of the ischemic kidney was reduced because of cessation of the active sodium reabsorption in its tubules, a process requiring a considerable expenditure of energy in the intact organ [12]. In the absence of active sodium reabsorption (basal respiration) the oxygen demand of the kidney is about 100 μmoles/min/100 g tissue [10].

The present experiments showed that the respiration of the kidney at the end of 4 h of ischemia was about one-third of the basal O_2 consumption.

The presence of a collateral circulation, the increase in the utilization of oxygen and glucose by the ischemic kidney per unit volume of the blood flow, and also the decreased energy requirements of the kidney are evidently together largely responsible for the favorable results of operative treatment of thrombosis and embolism of the renal artery, which as a rule is carried out several hours after the onset of acute vascular occlusion [7-9, 11].

At the same time, total ischemia of the kidney arising during transplantation of the organ is known to lead to very serious functional and structural changes in the ischemic kidney after only 1 h at the normal body temperature [4].

Consequently, the level of energy metabolism maintained in the kidney tissue by the collateral circulation and by increased utilization of oxygen and glucose per unit volume of inflowing blood is sufficient to substantially delay the development of changes caused by ischemia in the kidney when the main artery is occluded. However, the collateral blood flow can only partly satisfy the kidney's requirements of O_2, and for this reason occlusion of the renal artery must inevitably lead to the development of metabolic acidosis in the organ.

After removal of the occlusion to the renal artery the acid-base balance in the blood flowing from the ischemic kidney was restored to normal and gradual recovery of the arterio-venous difference for oxygen and glucose was observed. Although in the early postischemic period the renal circulation was not fully restored (the volume velocity of the blood flow remained below its initial level and the resistance of the renal vessels remained increased), nevertheless the resulting circulating blood volume satisfied the necessary conditions for the development of repair processes in the ischemic organ. Previous experiments by the writers [2, 3] showed that the function of the ischemic kidney is largely restored to normal after occlusion for 4 h; the compensatory powers of the kidney are restored.

LITERATURE CITED

1. E. B. Berkhin and Yu. I. Ivanov, Methods of Experimental Investigation of the Kidneys and of Water and Mineral Metabolism [in Russian], Barnaul (1972).
2. V. S. Makarenko and V. M. Gapanovich, Byull. Eksperim. Biol. i Med., No. 9, 22 (1972).

3. V. S. Makarenko and V. M. Gapanovich, in: Mechanisms of Regulation of Vital Activities in Disease, Proceedings of the 5th All-Union Conference of Pathophysiologists [in Russian], Baku (1970), p. 88.
4. B. V. Petrovskii, G. M. Solov'ev, V. I. Govallo, et al., Transplantation of the Kidney [in Russian], Moscow–Warsaw (1969).
5. M. I. Prokhorova and Z. N. Tupikova, Large Textbook of Practical Carbohydrate and Lipid Metabolism [in Russian], Leningrad (1965).
6. S. A. Seleznev, The Liver in the Dynamics of Traumatic Shock [in Russian], Leningrad (1971).
7. A. Brest, R. Bower, and C. Heider, J. Am. Med. Assn., 187, 540 (1964).
8. J. M. Erskine and F. W. Blaisdell, Arch. Surg., 90, 247 (1965).
9. E. I. Goldsmith, F. W. Fuller, C. T. Lambrew, et al., J. Urol. (Balitmore), 99, 336 (1968).
10. N. A. Lassen, O. Munck, and J. H. Thaysen, Acta Physiol. Scand., 51, 369 (1961).
11. N. E. Peterson and D. F. McDonald, J. Urol. (Baltimore), 100, 140 (1968).
12. G. Torelli, A. Milla, A. Faelli, et al., Am. J. Physiol., 211, 576 (1966).