

CONTENT OF ELECTROLYTES AND WATER IN THE VASCULAR  
WALL AND THE INTERNAL ORGANS OF DOGS  
WITH EXPERIMENTAL RENAL HYPERTENSION

M. I. Gurevich  
and N. G. Kochemasova

UDC 616.12-008.331.1-02:616.61-092.9-008.92-074

More and more reports are appearing in the literature suggesting that the vascular tone is largely dependent on changes in the content of electrolytes and water in the walls of the blood vessels [3, 4, 11, 17, 18, 23]. From this point of view the study of the composition of the electrolytes in the vascular wall in experimental arterial hypertension may be very important. However, despite the fact that several investigations in this direction have been conducted [6, 7, 9, 10, 12, 13, 19-23], the character of distribution of electrolytes and water in the vascular wall and in the internal organs in arterial hypertension is not yet clear.

Most investigations of electrolytes in experimental renal hypertension have been carried out on rats [6, 7, 13, 15, 20-23], and only a few studies have been made on the content of water and electrolytes in the vascular wall [9, 12] and skeletal muscle [8] of dogs with experimental renal hypertension lasting up to 3 months.

The object of the present investigation was to study simultaneously the changes in the electrolyte composition of the vascular wall, the skeletal muscles, and the heart, liver, and kidneys of dogs with experimental renal hypertension.

EXPERIMENTAL METHOD

Experiments were carried out on mongrel male dogs weighing 14-22 kg of middle age. Altogether 8 normal animals and 8 dogs with experimental renal hypertension caused by two-stage application of silver rings to the renal arteries [1, 2], were investigated. The arterial pressure was measured by puncture of the femoral artery. Six months after the second stage of the operation the arterial pressure of the animals was elevated on the average by 80 mm Hg. The dogs were sacrificed by exsanguination under Nembutal anesthesia (30 mg/kg). Blood vessels (without adventitia) from the following segments of the vascular system were taken for investigation: arch of the aorta (1 cm above the origin of the brachiocephalic artery), the thoracic aorta (at the level of the third intercostal space), the abdominal aorta (2 cm below the origin of the renal arteries), and the carotid artery. Other tissues taken for analysis included the skeletal muscle of the thigh, the heart muscle (right and left ventricles), the liver, and the kidney (cortex and medulla). The tissues were dissected at 5°, weighed on torsion scales, dried for 4 days to constant weight at 105°, and again weighed. The total water content was estimated from the difference in weight. The dry residue was defatted by extraction twice with ethyl ether [16]. The electrolytes were extracted with a 1 N solution of nitric acid for 48 h at room temperature. The chloride in the supernatant fluid after centrifugation was determined by Volhard's method, and the potassium and sodium, after suitable dilution, by a flame photometer. The serum chloride was determined by Rusniak's method. The concentration of electrolytes in the tissues was expressed in milliequivalents, and the water content in grams/100 g dry, defatted residue. The intracellular concentration of electrolytes in the heart and skeletal muscle was calculated by Benson's formula [5], and expressed in milliequivalents/kg of intracellular water.

EXPERIMENTAL RESULTS

Figures for the total content of electrolytes and water in the serum and tissues are given in Table 1. They show that no significant difference was found between the levels of potassium, sodium, and water in the blood serum, the skeletal muscle, and the liver of normal dogs and animals with renal hypertension. In the heart, a decrease in

---

Laboratory of the Physiology of the Circulation, A. A. Bogomolets Institute of Physiology, Academy of Sciences of the UkrSSR, Kiev (Presented by Active Member of the Academy of Medical Sciences of the USSR V. V. Parin). Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 62, No. 10, pp. 53-56, October, 1966. Original article submitted February 1, 1965.

TABLE 1. Content of Electrolytes and Water in the Blood Serum and Tissues of Healthy Dogs and Dogs with Experimental Renal Hypertension

	Water		Sodium		Potassium		Chloride	
	nor- mal	hyper- tension	nor- mal	hyper- tension	nor- mal	hyper- tension	nor- mal	hyper- tension
Serum	952 ±11,5	948 ±19,0	168 ±5,3	169 ±3,1	4,8 ±0,68	4,9 ±0,43	121 ±4,0	123 ±3,7
Aorta Arch	294,0 ±13,0 $P<0,001$	329,0 ±19,0	37,0 ±2,5 $P<0,01$	42,1 ±3,7	22,5 ±2,0 $P<0,01$	25,5 ±1,9	26,0 ±1,8 $P<0,1$	28,4 ±3,1
Thoracic	261,0 ±22,0 $P<0,05$	286,0 ±17,0	34,5 ±3,0 $P<0,05$	39,4 ±6,0	20,0 ±2,4 $P<0,01$	20,8 ±2,0	25,0 ±2,1 $P<0,01$	25,5 ±2,7
Abdominal	226 ±22,0 $P<0,05$	253 ±25,0	31,4 ±0,6 $P<0,001$	35,2 ±1,0	15,8 ±1,4 $P<0,5$	16,3 ±2,0	25,6 ±2,4 $P<0,01$	25,0 ±2,8
Carotid artery	204 ±17 $P<0,001$	250 ±20	32,6 ±1,5 $P<0,001$	38,1 ±3,0	15,1 ±2,6 $P<0,5$	16,3 ±2,6	23,9 ±1,6 $P<0,01$	24,2 ±2,0
Skeletal muscle	320 ±14 $P<0,3$	311 ±17	8,0 ±0,8 $P<0,001$	7,09 ±1,0	45,4 ±2,2 $P<0,5$	45,5 ±2,5	4,3 ±0,96 $P<0,01$	4,3 ±0,97
Heart								
Right ventricle	359 ±17,2 $P<0,3$	351 ±17,0	14,6 ±1,7 $P<0,5$	15,1 ±1,4	42,8 ±1,2 $P<0,01$	42,5 ±2,3	11,0 ±0,64 $P<0,02$	10,0 ±0,57
Left ven- tricle	355 ±9,0 $P<0,02$	343 ±9,6	14,7 ±1,76 $P<0,3$	15,4 ±1,26	41,7 ±2,4 $P<0,01$	41,8 ±2,8	11,1 ±0,80 $P<0,02$	9,8 ±0,88
Liver	239 ±14,0 $P<0,001$	237 ±9,0	11,3 ±1,0 $P<0,5$	12,5 ±2,6	28,2 ±2,2 $P<0,5$	28,7 ±1,6	9,4 ±0,5 $P<0,01$	9,5 ±1,4
Kidneys								
Cortex	330 ±25,0 $P<0,001$	330 ±24,0	32,0 ±5,0 $P<0,001$	33,0 ±5,5	31,0 ±2,2 $P<0,5$	31,0 ±1,0	23,0 ±2,4 $P<0,01$	23,0 ±3,1
Medulla	600 ±67 $P<0,001$	497 ±84	110 ±15,5 $P<0,001$	80 ±21,5	32,0 ±3,0 $P<0,5$	30,0 ±2,2	95,0 ±21 $P<0,01$	69,0 ±21

Note. Water in grams, sodium, potassium, and chloride in milliequivalents (in tissues, per 100 g dry, defatted residue, in serum per liter).

the total content of water was found in the left ventricle, and a decrease in the level of chlorides in the right and left ventricles, whereas the total sodium and potassium contents in the heart muscle were unchanged. The most marked changes in the total electrolyte and water contents were found in the vascular wall and the kidneys. In the vascular wall in all the investigated segments of the aorta and the carotid artery, an increase in the content of water and sodium was observed. The concentration of chloride remained essentially unchanged. The increase in the potassium concentration was statistically significant only in the arch of the aorta. In the kidneys changes were found only in the medulla; they took the form of a decrease in the water, sodium, and chloride contents whereas the potassium concentration remained within normal limits. In the kidney cortex the levels of potassium, sodium, chloride, and water were essentially indistinguishable from normal.

The calculated data (Table 2) showed that in the heart, in both the right and left ventricles, the volume of extracellular water and the extracellular sodium content fell whereas the intracellular water content and the intracellular sodium concentration rose. Since the total sodium content in the heart was unchanged these results demonstrate the transfer of sodium from the extracellular to the intracellular space. In the skeletal muscle no such changes were found. The potassium concentration in the intracellular and extracellular space remained unchanged both in the heart and in the skeletal muscle.

Comparison between the results obtained and data in the literature showed that in dogs with experimental renal hypertension 6 months in duration, the changes in the electrolytes and water in the vascular wall were similar to those discovered in earlier stages of renal hypertension [12]. However, in the present experiment the increase in

TABLE 2. Intracellular and Extracellular Contents of Water and Electrolytes in the Skeletal Muscle and Heart of Healthy Dogs and Dogs with Experimental Renal Hypertension

	Water		Sodium			Potassium		
	(H <sub>2</sub> O) <sub>e</sub>	(H <sub>2</sub> O) <sub>I</sub>	(Na) <sub>e</sub>	(Na) <sub>I</sub>	(Na) <sub>i</sub>	(K) <sub>e</sub>	(K) <sub>I</sub>	(K) <sub>i</sub>
Skeletal muscles								
Normal . . . . .	226 ± 39	3 027 ± 194	38,0 ± 11	45,0 ± 11	14,8 ± 2,8	1,0 ± 0,39	460 ± 25	154 ± 12,5
Hypertension . . . . .	237 ± 34	2 878 ± 189	39,0 ± 11,5	41,0 ± 13,1	14,0 ± 4,0	1,0 ± 0,28	450 ± 23	158 ± 13
Heart								
Right ventricle								
Normal . . . . .	740 ± 66	2 854 ± 190	127 ± 10,0	24,9 ± 12,0	8,5 ± 3,8	3,6 ± 0,6	424 ± 12,0	150 ± 11,0
Hypertension . . . . .	658 ± 61	2 830 ± 191	114 ± 12,0	37,9 ± 8,7	13,5 ± 3,4	3,1 ± 0,47	422 ± 25,0	150 ± 14,5
	P < 0,05		P < 0,05	P < 0,05	P < 0,02	P < 0,1		
Left ventricle								
Normal . . . . .	748 ± 78	2 820 ± 120	127 ± 12,5	28,0 ± 13,0	9,5 ± 4,9	3,6 ± 0,64	415 ± 25	148 ± 14
Hypertension . . . . .	635 ± 65	2 790 ± 170	110 ± 11,0	43,0 ± 9,5	15,5 ± 3,0	3,0 ± 0,40	415 ± 28,5	149 ± 11,5
	P < 0,02		P < 0,02	P < 0,05	P < 0,05	P < 0,1		

Legend: (H<sub>2</sub>O)<sub>I</sub>, (H<sub>2</sub>O)<sub>e</sub> — intracellular and extracellular water (in g/kg dry, defatted residue); (Na)<sub>I</sub>, (Na)<sub>e</sub> — intracellular and extracellular sodium (in meq/kg dry, defatted residue); (Na)<sub>i</sub> — intracellular sodium (in meq/kg intracellular water).

the content of sodium and water in the aorta was less marked than in the experiments of Jones and co-workers [12], despite the fact that they investigated dogs in which an elevated arterial pressure had been observed for only 4 weeks. In addition, no changes were found in the content of water and electrolytes in the skeletal muscle, whereas in a study by Eichelberger [8] it was found that in dogs with experimental renal hypertension lasting 3 months, the content of extracellular water in the skeletal muscle was increased by 65% and the total sodium and chloride level was increased while the potassium level was decreased.

These differences may perhaps be attributed to differences in the duration of the experimental renal hypertension. The problem of the content of electrolytes in the blood serum and the tissues in the late stages of renal hypertension has been inadequately studied. The only relevant reference which could be found in the literature was that to the work of Daniel and Dawkins [7], who reported that in the late stages of development of renal hypertension in rats, they were unable to find those changes in the electrolyte composition in the aorta, skeletal muscles, and heart which are characteristic of the early stages of renal hypertension.

## SUMMARY

The object of study was the content of sodium, potassium, chlorine, and water in the vessel-wall (various parts of the aorta and carotid artery), the skeletal muscle, the heart (right and left ventricles), the liver and the kidneys (cortical and cerebral layer) in 8 dogs with experimental liver hypertension caused by stenosis of the renal arteries and in 8 normal dogs.

Six months after an operation of narrowing the lumen of the renal arteries there was an increase in the arterial pressure by 80 mm Hg on the average. The blood serum, skeletal muscle, liver and cortical layer of the kidneys were found to have no substantial difference in the content of electrolytes and water as compared to normal. In the vessel-wall (in all parts of the aorta and in the carotid artery) one could observe an increase in the sodium and water level. The potassium level was significant only in the aortic arch. In the cerebral layer of the kidneys the water, sodium, and chlorine content drastically decreased, whereas the potassium content remained unchanged. The heart was found to have a decrease in the level of extracellular water and transition of sodium from the extracellular into the intracellular space.

## LITERATURE CITED

1. N. N. Gorev, Outlines of the Study of Hypertension [in Russian], Kiev (1959).
2. M. I. Gurevich, Investigation of the Pathogenesis of Arterial Hypertension [in Russian], Kiev (1960).

3. V. V. Parin and F. Z. Meerson, *Outlines of the Clinical Physiology of the Circulation* [in Russian], Moscow (1960).
4. V. V. Parin and S. M. Shenderov, *Cor et Vasa*, No. 4, Praha (1962), p. 1.
5. E. Benson et al., *Am. J. Physiol.*, Vol. 187 (1956), p. 483.
6. C. Briot and J. Baillet, *Rev. Franc. Etud. Clin. Biol.*, Vol. 8 (1963), p. 165.
7. E. E. Daniel and O. A. Dawkins, *Am. J. Physiol.*, Vol. 182 (1955), p. 567.
8. L. Eichelberger, *J. Exp. Med.*, Vol. 77 (1943), p. 205.
9. E. O. Feigl et al., *J. Clin. Invest.*, Vol. 42 (1963), p. 1640.
10. S. C. Freed, S. S. George, and R. H. Rosenman, *Circulat. Res.*, Vol. 7 (1959), p. 219.
11. S. Freedman, *Trans. Roy. Soc. Canada, Sect. 3*, Vol. 56 (1962), p. 161.
12. A. W. Jones et al., *Circulat. Res.*, Vol. 15 (1964), p. 386.
13. S. Koletsky et al., *Proc. Soc. Exp. Biol.*, Vol. 102, New York (1959), p. 12.
14. D. L. Laramore and A. Grollman, *Am. J. Physiol.*, Vol. 161 (1950), p. 278.
15. L. M. Ledingham, *Clin. Sci.*, Vol. 12 (1953), p. 337.
16. O. H. Lowry and A. B. Hastings, *J. Biol. Chem.*, Vol. 143 (1942), p. 257.
17. L. H. Peterson, *Physiol. Rev.*, Vol. 42 (1962), p. 499.
18. W. Rabb, In the book: *Current Problems in Cardiology* [Russian translation], Moscow (1960), p. 69.
19. L. Tobian and J. Binion, *Circulation*, Vol. 5 (1952), p. 754.
20. Idem., *J. Clin. Invest.*, Vol. 33 (1954), p. 1407.
21. L. Tobian, *Circulat. Res.*, Vol. 4 (1956), p. 671.
22. L. Tobian and P. D. Redleaf, *Am. J. Physiol.*, Vol. 192 (1958), p. 325.
23. L. Tobian, *Physiol. Rev.*, Vol. 40 (1960), p. 94.