5. A. S. Golovatskii and S. I. Sikora, in: The Magnetic Field in Medicine (Collection of Scientific Papers) [in Russian], Frunze (1974), p. 28.
6. K. B. Kakchinbaev, Z. M. Abdullina, and M. D. Dzhunusheev, in: Proceedings of the 3rd All-Union Symposium on the Effect of Magnetic Fields on Biological Objects [in Russian], Kaliningrad (1975), p. 101.
7. D. Purpura, in: Mechanisms of the Whole Brain [Russian translation], Moscow (1963), p. 9.
8. L. I. Roitbak, Bioelectrical Phenomena in the Cerebral Cortex [in Russian], Tbilisi (1955).
9. T. S. Sachava, in: Proceedings of the 2nd All-Union Conference on the Study of the Effect of Magnetic Fields on Biological Objects [in Russian], Moscow (1969), p. 200.
10. F. N. Serkov, Neirofiziologiya, No. 4, 349 (1970).
11. F. N. Serkov and N. V. Bratus', in: Current Problems in the Electrophysiology of the Central Nervous System [in Russian], Moscow (1967), p. 253.
12. V. V. Fanardzhyan, Fiziol. Zh. SSSR, No. 7, 823 (1962).
13. Yu. A. Kholodov, Effect of Electromagnetic and Magnetic Fields on the Central Nervous System [in Russian], Moscow (1966).
14. Yu. A. Kholodov, in: Effect of Magnetic Fields on Biological Objects [in Russian], Moscow (1971), p. 124.
15. V. A. Chigirinskii, in: Proceedings of the 2nd All-Union Conference on the Study of the Effect of Magnetic Fields on Biological Objects [in Russian], Moscow (1969), p. 251.
16. T. Gualtierotti, in: Twelfth International Astronautic Congress. Proceedings, Vol. 2, New York (1963), p. 586.
17. T. Gualtierotti and V. Capraro, in: Life Sciences and Space Research, Vol. 2 (ed. by M. Florkin and A. Dollfus), Elsevier, New York (1964), p. 311.

MECHANISM OF THE INFLUENCE OF SYMPATHETIC NERVES ON KIDNEY FUNCTION

M. I. Mavrin

UDC 612.463.8:612.89

In dogs receiving large doses of reserpine hyponatriemia, hypokaliemia, oliguria, a reduction in the renal blood flow and in the sodium and potassium excretion, together with abolition of the inhibitory effect of the splanchnic nerve on the function of the glomerular and tubular portions of the nephron are observed as the result of functional insufficiency of the sympathetic innervation of the kidneys through the development of catecholamine deficiency.

KEY WORDS: *Sympathetic nerves; reserpine; kidney function.*

Previous investigations established depression of the function of the glomerular and tubular portions of the nephron of the kidney on the side of splanchnic nerve stimulation [1]. To confirm that the inhibition of kidney function was due to activity of adrenergic fibers it was decided to use reserpine to block the action of the sympathetic innervation.

EXPERIMENTAL METHOD

Chronic experiments were performed on 12 dogs with separately exteriorized ureters. Altogether 18 experiments were performed on six intact animals (control) and 12 experiments on

Department of Normal Physiology and Faculty of Surgery, S. M. Kurashov Kazan' Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR A. A. Vishnevskii*.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 82, No. 8, pp. 910-911, August, 1976. Original article submitted November 4, 1975.

*Deceased.

TABLE 1. Effect of Stimulation of Peripheral End of Divided Splanchnic Nerve on Kidney Function in Reserpinized Animals (M±m; n=6)

Index of kidney function	Kidney*	Normally	Before stimulation	After stimulation for 5 min	
				5 min	25 min
Diuresis, ml/min·m²	S	5,08±1,03	3,01±0,42	3,29±0,45	3,18±0,51
	C	4,91±0,86	3,13±0,49	3,29±0,49	2,89±0,35
Filtration, ml/min·m²	S	41±1,01	39±3,17	39±3,33	40±3,50
	C	40±0,64	38±3,64	40±4,92	40±4,38
Plasma flow ml/min·m²	S	193±8,90	160±7,95	163±8,12	173±6,90
	C	191±3,08	152±6,29	151±5,77	147±7,99
Secretion, mg/min·m²	S	13,2±0,41	10,1±1,04	9,3±0,60	10,1±0,82
	C	12,8±0,27	9,9±0,60	13,0±1,87	11,0±2,15
Sodium excretion, μeq/min·m²	S	169±53,71	35±14,96	100±21,17	120±42,59
	C	174±49,05	82±10,35	106±18,83	107±31,53
Sodium reabsorption, meq/min	S	3,89±0,79	2,46±0,31	2,50±0,38	2,56±0,47
	C	3,95±0,80	2,33±0,34	2,55±0,51	2,59±0,45
Potassium excretion, μeq/min·m²	S	77±16,06	34±5,94	30±3,91	37±7,24
	C	82±16,72	37±6,86	33±7,09	39±10,09

*S) kidney on side of stimulation; C) kidney on opposite side (control).

six dogs receiving reserpine (0.18-0.20 mg/kg) for 2-3 days before the experiment [3]. To create adequate diuresis, water warmed to a temperature of 20°C in a volume of 3% of the body weight was injected through a tube into the stomach of all the animals before the experiment; the filtration (for inulin), plasma flow, and secretion (for diodone), sodium reabsorption, and excretion of sodium and potassium [1] were determined. The splanchnic nerves were stimulated by means of an ISÉ-01 pulsed electronic stimulator (frequency of stimulation 10-20 pulses/sec, pulse duration 1-2 sec, voltage 3-10 V).

EXPERIMENTAL RESULTS

Compared with the control animals, in the reserpinized animals the sodium ion concentration in the blood plasma was reduced from 142.04±7.35 to 135.62±5.40 meq/liter ($P<0.05$), whereas the potassium excretion, on the other hand, was increased from 4.22±0.25 to 5.36±0.88 meq/liter ($P<0.05$). The hyponatriemia was accompanied by a decrease in sodium and potassium excretion (Table 1). The maximal tubular secretion was not significantly changed. The decrease in diuresis and in the renal circulation was evidently connected with dehydration in the experimental animals. The oliguric effect was due mainly to an increase in the tubular reabsorption of water, for the filtration process was almost undistrubed. The concentration index of inulin rose from 10.5±2.24 to 18±3.45 ($P<0.02$) in the right kidney and from 10.3±1.83 to 16±4.98 ($P<0.05$) in the left kidney. In the reserpinized dogs, against the background of considerable exhaustion of the catecholamine reserves, mainly of noradrenalin [2], the inhibitory action of the splanchnic nerve on the rate of urine excretion, tubular filtration, maximal secretion, and potassium secretion in the kidney on the side of stimulation and on the cortical blood flow in both kidneys was totally abolished. Stimulation of the peripheral end of the divided splanchnic nerve likewise had no significant effect on the sodium excretion or on its absolute reabsorption in both kidneys. This was due to a severe disturbance of the functional state of the sympathetic nervous system, responsible for the crossed innervation of the vascular and tubular portions of the kidney nephron.

The results thus confirm the previous conclusion [1] that the inhibitory action of the splanchnic nerve on the renal hemodynamics, the maximal tubular secretion, and the potassium excretion is connected with the activity of adrenergic fibers and their mediator.

LITERATURE CITED

1. M. I. Mavrin, Fiziol. Zh. SSSR, No. 10, 1602 (1973).
2. R. S. Orlov, The Physiology of Smooth Muscle [in Russian], Moscow (1967).
3. J. Disalvo and F. Colin. Proc. Soc. Exp. Biol. Med. (New York), 136, 150 (1971).