LITERATURE CITED

1. V. G. Baranov, Vestn. Akad. Med. Nauk SSSR, No. 8, 15 (1969).
2. L. N. Bogatskaya, in: Adaptive Powers of the Aging Organism [in Russian], Kiev (1968), p. 131.
3. S. V. Bogush, "Age differences in the insulin production of the body," Author's Abstract of Candidate's Dissertation, Kiev (1972).
4. G. G. Gatsko, in: Aging and the Physiological Systems of the Organism [in Russian], Kiev (1969), p. 117.
5. G. G. Gatsko and V. V. Gul'ko, Izvest. Akad. Nauk Belor. SSR, Ser. Biol. Nauk, No. 2, 93 (1969).
6. V. M. Dil'man, Aging, the Climacteric, and Cancer [in Russian], Leningrad (1968).
7. N. Ya. Dzeranova and P. B. Shlimovich, Probl. Éndokrinol., No. 3, 14 (1971).
8. N. A. Zhukov, Probl. Éndokrinol., No. 10, 13 (1964).
9. B. A. Kronrod, in: Age and Functional Morphology of the Endocrine System [in Russian], Leningrad (1964), p. 124.
10. L. P. Lisitsyna, Izvest. Akad. Nauk Belor. SSR, Seriya Biol. Nauk, No. 3, 57 (1972).
11. V. I. Puzik, Age Morphology of the Glands of Internal Secretion in Man [in Russian], Moscow (1951), p. 96.
12. I. P. Solenova-Filippova, in: The Molecular Biology of Aging [in Russian], Kiev (1969), p. 115.
13. V. Frol'kis, in: Annual Supplement to the Great Medical Encyclopedia [in Russian], Vol. 1, Moscow (1968), p. 1168.
14. S. Berger et al., New Engl. J. Med., 274, 1460 (1966).
15. W. G. Duncombe, Clin. Chim. Acta, 9, 122 (1964).
16. A. Gommers and M. Gasparo, Gerontology, 29, 139 (1974).
17. B. Lewis and B. Wexler, Gerontology, 18, 179 (1972).
18. G. Shochalf and D. Wilansky, Metabolism, 17, 928 (1968).

ROLE OF THE SPLANCHNIC NERVES IN REGULATION OF MAXIMAL GLUCOSE TRANSPORT IN THE KIDNEYS

M. I. Mavrin

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Glomerular filtration is reduced in the kidney on the side of the stimulated splanchnic nerve but maximal glucose reabsorption is unchanged. After demedullation of the adrenals, splanchnic nerve stimulation increases filtration in the contralateral kidney without changing maximal glucose transport. Adrenergic fibers of the splanchnic nerve have no direct action on maximal glucose transport in the kidneys.

KEY WORDS: splanchnic nerve; maximal glucose transport in the kidneys; demedullation of the adrenals.

Data on nervous regulation of tubular transport of glucose in the kidneys are few in number and contradictory in nature [3, 7].

In the investigation described below the role of the splanchnic nerves in the regulation of maximal glucose transport in the proximal tubules of the dog kidney was studied.

EXPERIMENTAL METHOD

The dogs used had their ureters exteriorized separately. The control group consisted of 11 animals. In the experimental group 15 dogs underwent adrenal demedullation: Six of these animals received adrenalin by intravenous injection in a dose of 0.1 mg/kg daily (except on the day of the operation) [2]. During the period

Department of Normal Physiology and Professorial Department of Surgery, S. V. Kurashov Kazan' Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR A. A. Vishnevskii [deceased].) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 82, No. 7, pp. 778-780, July, 1976. Original article submitted November 4, 1975.

TABLE 1. Effect of Stimulation of Peripheral End of Splanchnic Nerve on Kidney Function in Dogs (n = 11)

Index of kidney function	Kidney	Before stimulation	After stimulation (in min) 5	15	25	35
Diuresis (in ml/min · m^2)	S	4,55	—1,77±0,33 $P<0,001$	—0,32±0,39	—0,53±0,40	—0,74±0,46
	C	4,53	—0,14±0,26	—0,25±0,40	—0,63±0,45	—0,87±0,44
Filtration (in ml/min · m^2)	S	43	—11±2,22 $P<0,001$	—2,38±1,40	—0,14±1,35	+1,15±2,05
	C	43	+0,65±1,50	—1,20±0,80	+0,33±1,06	—0,39±1,11
Maximal reabsorption of glucose (in mg/min · m^2)	S	141	+1,55±2,13	+2,01±4,68	—3,50±1,45	—1,99±3,26
	C	143	+8,57±4,50	+0,50±2,36	+0,05±2,95	—0,03±2,68

Legend. Here and in Table 2: S) kidney on side of stimulation; C) kidney on contralateral side (control).

TABLE 2. Effect of Stimulation of Peripheral End of Splanchnic Nerve on Kidney Function in Dogs with Demedullated Adrenals (n = 9)

Index of kidney function	Kidney	Before stimulation	After stimulation (min) 5	15	25	35
Diuresis (in ml/min · m^2)	S	4,57	+0,20±0,12	+0,56±0,91	+0,04±0,27	—0,10±0,23
	C	4,90	+1,18±0,19 $<P$ 0,001	+1,00±0,17 $P<0,001$	—0,18±0,33	—0,32±0,22
Filtration (in ml/min · m^2)	S	44	0,00±0,95	—0,04±0,45	—0,43±0,81	—2,03±1,14
	C	46	+8,09±1,04 $P<0,001$	+7,74±1,30 $P<0,001$	—0,47±1,30	—2,62±1,70
Maximal reabsorption of glucose (in mg/min · m^2)	S	142	—2,18±2,76	—3,14±3,71	+0,40±3,49	+0,35±3,35
	C	145	+6,82±4,97	+7,35±4,50	+4,94±4,49	+3,46±4,50

of the experiment 0.6-0.7% inulin solution (0.5 mg/kg/min) and 25-30% glucose solution (40 mg/kg/min) were injected intravenously. When constant blood levels of inulin (15-20 mg%) and glucose (about 500 mg %) had been reached, the renal filtration (for inulin) [12] and the maximal reabsorption (for glucose [8]) were determined. The splanchnic nerves were stimulated by means of an ISÉ-01 electronic pulse generator in the course of the 5-min clearance period at intervals of 15 sec every 45 sec of stimulation [4]. Altogether 55 experiments were carried out.

EXPERIMENTAL RESULTS AND DISCUSSION

Stimulation of the peripheral end of the splanchnic nerve had no effect on the velocity of maximal glucose transport in the kidney on the side of stimulation despite a reduction in the rate of glomerular filtration by 25% and in the diuresis by 40% (Table 1). In the opposite kidney all the parameters of function remained as before.

The constancy of the maximal glucose reabsorption during a decrease in the rate of glomerular filtration is evidence of complete saturation of the active glucose transport system in the proximal renal tubules. In the view of Thompson et al. [15], a decrease in the maximal glucose reabsorption can arise only when filtration is reduced by 40-50% as the result of a decrease in the number of functioning nephrons, inadequate saturation of the transport system with glucose, and the heterogeneity of the individual nephrons [13]. Some workers [6, 10] were unable to determine the value of the maximal glucose transport in dogs on the grounds that the absolute reabsorption of glucose in the kidneys is directly dependent on the rate of glomerular filtration and the filtration charge of glucose [1, 9], and inversely proportional to the blood glucose concentration [6]. However, during incomplete saturation of the system responsible for active glucose transport in the proximal renal tubules, maximal reabsorption of glucose cannot be attained [16]; its value is constant and is independent on the rate of glomerular filtration [5, 11, 14].

To determine the mechanism of action of the adrenergic fibers of the splanchnic nerve concerned in the regulation of tubular activity [4] experiments were carried out on animals undergoing adrenal demedullation in order to disturb catecholamine synthesis [2]. On the fifth-10th day after the operation, when the most severe disturbances took place in metabolism of the adrenergic mediator, splanchnic nerve stimulation likewise had no significant effect on the dynamics of maximal glucose transport in the two kidneys, although the diuresis in the opposite kidney increased by 18-21% and the filtration by 16-18% (Table 2). Systematic injection of adrenalin into the demedullated animals [2] prevented these disturbances of urine secretion and filtration activity of the

contralateral kidney during splanchnic nerve stimulation without altering the value of the maximal glucose reabsorption in the two kidneys.

In the kidney on the side of splanchnic nerve stimulation maximal reabsorption of glucose was thus unchanged although the rate of the filtration process was reduced. After adrenal demedullation splanchnic nerve stimulation increased filtration in the contralateral kidney but had no effect on maximal glucose transport in the two kidneys. Consequently, maximal glucose reabsorption was unchanged during small fluctuations in glomerular filtration. The adrenergic fibers of the splanchnic nerve evidently have no direct influence on the velocity of maximal glucose transport in the proximal tubules of the kidneys.

LITERATURE CITED

1. V. F. Vasil'eva, Fiziol. Zh. SSSR, No. 3, 236 (1958).
2. I. N. Volkova and A. V. Kibyakov, Fiziol. Zh. SSSR, No. 1, 130 (1946).
3. V. F. Lysov, Byull. Éksp. Biol. Med., No. 7, 29 (1968).
4. M. I. Mavrin, Fiziol. Zh. SSSR, No. 10, 1602 (1973).
5. P. Balint, Normale und pathologische Physiologie der Nieren, Berlin (1969).
6. R. H. Bowman and T. Maack, Am. J. Physiol., 222, 1499 (1972).
7. P. Gömori, M. Foldi, and G. Szabo, Acta Med. Acad. Hung., 17, 99 (1961).
8. H. Hagedorn and B. Jensen, Biochem. Z., 135, 235 (1923).
9. L. Keyes and R. Swanson, Am. J. Physiol., 221, 1 (1971).
10. N. A. Kurtzman, M. White, P. Rodgers, et al., J. Clin. Invest., 51, 127 (1972).
11. J. A. Shannon, S. Farber, and L. Troast, Am. J. Physiol., 133, 752 (1941).
12. G. E. Schreiner, Proc. Soc. Exp. Biol. (New York), 74, 117 (1950).
13. R. G. Schultze and H. Berger, Kidney Int., 3, 291 (1973).
14. H. W. Smith, Principles of Renal Physiology, New York (1956).
15. D. Thompson, M. Barrett, and R. Pitts, Am. J. Physiol., 167, 546 (1951).
16. B. M. Tune and M. B. Burg, Am. J. Physiol., 221, 580 (1971).

POTENTIATION AND RESTITUTION OF HEART MUSCLE CONTRACTION IN RATS ADAPTED TO EXERCISE

C. Pfeiffer, V. I. Kapel'ko, and F. Z. Meerson

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The original amplitude of contraction of strips of myocardium determined the inotropic response to paired stimulation. The higher the initial amplitude, the lower the degree of potentiation and the higher the degree of restitution of contraction. For equal amplitude, the degree of potentiation of myocardial contraction of exercise-adapted rats was greater and the degree of restitution smaller than in the control. These changes probably reflect changes in the ion transport system of the myocardial cells.

KEY WORDS: isolated papillary muscles; paired stimulation; restitution and potentiation of contractions; adaptation to exercise.

The dynamics of the amplitude of premature and potentiated contractions of a muscle during paired stimulation with different intertrial intervals can be used to assess the rate of restitution and subsequent potentiation of contraction. The central role of Ca^{2+} as a regulator of the contraction process suggests that the rate of

Institute of General Pathology and Pathophysiology, Academy of Medical Sciences of the USSR, Moscow. Institute of Physiology, East German Academy of Sciences.

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