6. V. I. Rokityanskii and Yu. V. Yakimov, in: Some Problems in Fluid Physics [in Russian], No. 4, Kazan' (1973), p. 119.
7. T. Farrar and E. Becker, NMR Pulsed and Fourier-Spectroscopy [Russian translation], Moscow (1973).
8. N. M. Émanuél', L. I. Murza, V. I. Naidich, et al., Dokl. Akad. Nauk SSSR, 225, No. 2, 460 (1975).
9. P. A. Bottomley, Cancer Res., 39, 468 (1979).
10. R. Damadian, Science, 171, 1151 (1971).
11. M. Ferguson, E. Eriksson, and M. C. Robson, Burns, 5, 293 (1979).
12. B. M. Fung, Biochim. Biophys. Acta, 497, 317 (1977).
13. L. H. Michael, P. Seitz, J. McMillin-Wood, et al., Science, 208, 1267 (1980).
14. L. A. Saryan, D. P. Hollis, J. S. Economou, et al., J. Natl. Cancer Inst., 52, 599 (1974).

CHANGES IN THE NORADRENALIN CONCENTRATION IN THE PORTAL VEIN AND AURICLES OF RATS IN THE COURSE OF STRESS

E. B. Manukhina, E. Ya. Vorontsova, E. V. Volina, and F. Z. Meerson

UDC 612.452.018:612.123]-06:613.083

KEY WORDS: noradrenalin; portal vein; auricle; emotional-pain stress.

Strong and prolonged excitation of the adrenergic system arising immediately after severe emotional-pain stress (EPS) leads regularly to a considerable fall in the noradrenalin (NA) content in the myocardium, adrenals, hypothalamus, and other organs due to the slower rate of resynthesis than of breakdown of catecholamines [3].

Increased liberation of NA into the blood is accompanied by damage to the myocardium [4]. The degree of this damage very probably varies in different parts of the cardiovascular system, depending on the intensity of the adrenergic effect therein.

The object of this investigation was to compare the degree of lowering of the NA content in different parts of the circulatory system in EPS, and to use this criterion to assess the intensity of the adrenergic effect. For this purpose the dynamics of the NA content in the myocardium of the auricles and ventricles and in the smooth muscle of a resistive vessel was compared. As the most adequate model of a resistive vessel the portal vein was used, for it has powerful muscles and intrinsic spontaneous activity, and so it closely resembles the resistive vessels in its properties [5, 6]. This vessel also has sufficient mass to allow quantitative determination of NA.

EXPERIMENTAL METHODS

Experiments were carried out on male Wistar rats weighing 180-200 g. Two groups of rats were investigated: the control (group 1) and animals exposed to EPS (group 2). EPS was reproduced in the form of an "anxiety neurosis" by Desiderato's method [7] for 6 h. The index of effectiveness of exposure to the harmful stressor was the formation of ulcerative lesions of the gastric mucosa in the rats. The NA concentration was determined in the portal vein and auricles 2 h and 1, 2, 4, and 8 days after the end of EPS and in the control. Measurements were made individually for each animal. The rats were decapitated and the test organs removed. NA was determined by the trihydroxyindole method [2]. Specific fluorescence was measured on the MPF-4 fluorescence spectrophotometer (Hitachi, Japan), with an excitation wavelength of 390 nm and was recorded at 485 nm. The results were subjected to statistical analysis by Student's test [1].

Institute of General Pathology and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR A. M. Chernukh.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 92, No. 12, pp. 673-674, December, 1981. Original article submitted April 28, 1981.

TABLE 1. Dynamics of NA Concentration (in ng/mg) in Ventricles, Auricles, and Portal Vein of Rat (M ± m)

Test object	Control	Time after end of EPS				
		2 h	1 day	2 days	4 days (5 days for ventricles)	8 days
Ventricles*	1,117±0,048 (*n*=12)	0,841±0,031 (*n*=6)†	0,616±0,081 (*n*=6)†	0,658±0,041 (*n*=6)†	0,924±0,049 (*n*=6)†	1,173±0,190 (*n*=6)
Auricles	1,647±0,140 (*n*=17)	1,267±0,120 (*n*=10)†	1,660±0,097 (*n*=5)	1,717±0,094 (*n*=10)	1,786±0,115 (*n*=12)	1,660±0,126 (*n*=6)
Portal vein	1,385±0,085 (*n*=21)	0,224±0,057 (*n*=11)†	1,292±0,163 (*n*=9)	0,914±0,195 (*n*=11)†	1,325±0,132 (*n*=11)	1,675±0,191 (*n*=10) †

*Data on dynamics of NA concentration in rat ventricles taken from [3].
†$P < 0.05$.

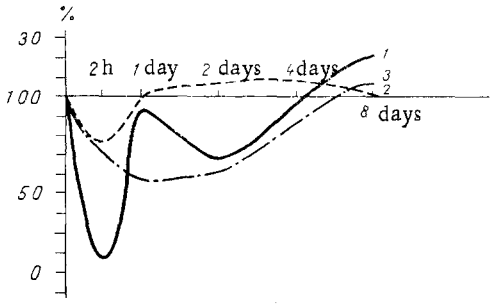

Fig. 1. Post-stress changes in NA concentration in rat organs. 1) Portal vein, 2) auricle, 3) ventricle. Abscissa, time after end of EPS; ordinate, NA concentration (in % of control).

EXPERIMENTAL RESULTS

Under normal conditions the NA concentration in the ventricles was 1.117 ± 0.048 ng/ml, in the auricles 1.647 ± 0.140 ng/ml, and in the portal vein 1.385 ± 0.120 ng/ml. It will be clear from Table 1 that exposure to EPS caused a significant fall in the NA content in the ventricles, auricles, and portal vein. The greatest fall in NA occurred in the vein (by 84%), in the ventricles the NA concentration fell by 54%, but in the auricles by only 23%. These maximal changes were observed in the ventricles 1 day, and the vein and auricles as early as 2 h after the end of exposure to EPS. After a sharp fall in the NA concentration in these tissues it began to recover rapidly; recovery was slower in the ventricles than in the auricles and vein (Fig. 1). The character of recovery of the NA concentration differed somewhat in different parts of the cardiovascular system. In the tissues of the auricles and vein the NA concentration was restored quickly. In the auricles it reached normal after only 1 day. In tissues of the vein, recovery of NA followed a fluctuating course. On the 4th day the NA level was back to normal or even above, although as late as on the 8th day normalization was not yet complete. The NA concentration was significantly above the normal level, i.e., recovery processes in the vein were characterized by considerable but gradually diminishing fluctuations in the NA level. The recovery process in the tissues of the ventricle was somewhat delayed. Normalization was not observed until the 8th day and the recovery curve in this case was smooth in character, without any fluctuations. By the 8th day the NA concentration was a little higher than normal, just as in the auricles (on the 4th day) and the smooth muscle of the vein (on the 8th day). Although this excess was not significant for the myocardium of the auricles and ventricles, it nevertheless points to the existence of a definite tendency.

After exposure to EPS more profound and prolonged changes in the NA concentration were thus observed in the portal vein than in the auricles and ventricles, and the recovery period was characterized by fluctuating changes. It will be noted that the smooth muscles of the vein is evidently a very labile structure, as shown by its strong reaction to the stressor,

the considerable fluctuations in NA concentration during the recovery period, and the longer recovery period than for the myocardium. It can be tentatively suggested that the differences in the response of the vein to the stressor are due to the fact that the apparatus for NA resynthesis is weaker in the smooth muscle of the vessels than in the myocardium of the ventricles and, in particular, of the auricles. It is also possible that the smooth muscle of the vessel has lower resistance than the myocardium to stress.

With all these explanations it seems probable that stress injury to the cardiovascular system may be more severe in the vascular muscles, where the most profound changes in NA content after stress were discovered.

LITERATURE CITED

1. B. S. Bessmertnyi, Mathematical Statistics in Clinical, Preventive, and Experimental Medicine [in Russian], Moscow (1967).
2. B. N. Manukhin, L. V. Berdysheva, and E. V. Volina, Vop. Med. Khim., No. 3, 317 (1975).
3. B. N. Manukhin, V. I. Pavlova, T. G. Putintseva, et al., Fiziol. Zh. SSSR (1981) (in press).
4. F. Z. Meerson, Adaptation, Stress, and Prophylaxis [in Russian], Moscow (1981).
5. B. Folkow and E. Neil, The Circulation [Russian translation], Moscow (1976).
6. I. Axelsson, B. Wahlström, B. Johansson, et al., Clin. Res., 21, 609 (1967).
7. O. Desiderato, I. R. MacKinnon, and H. Hisson, J. Comp. Physiol. Psychol., 84, 208 (1974).

STATE OF THE SMALL INTESTINE IN RABBITS INFECTED WITH *Salmonella*

M. I. Semashko, E. D. Bobkova, and N. D. Yushchuk

UDC 616.98:579.842.14]-092.9-07:616.341-008.931.074

KEY WORDS: salmonellosis; small intestine; morphology; enzymes.

The degree of involvement of different parts of the small intestine in salmonellosis has not yet been adequately studied. Yet the results of such investigations could tell us more about the mechanism of development of the fundamental functional and morphological disturbances in this infection.

The object of this investigation was to study the role of different parts of the small intestine in rabbits in the genesis of disturbances of digestive function in experimental salmonellosis. Activity of hydrolytic enzymes (lactase, maltase, alkaline phosphatase) was accordingly determined in the mucosa of the duodenum, jejunum, and ileum of rabbits infected with *Salmonella typhimurium*, with differences in the severity of the morphological changes in these respective zones. The effect of morphological and biochemical changes in the small intestine on the development of diarrhea also was studied.

EXPERIMENTAL METHODS

Salmonellosis was produced in 110 male chinchilla rabbits weighing 1000-1200 g. All the animals had previously been quarantined and tested bacteriologically for salmonellas. After starvation for 48 h the rabbits were given 2 ml of 5% soda solution. Next, 84 rabbits were infected perorally with 5 billion *S. typhimurium* cells in suspension with milk, and 26 control rabbits were given milk only. Experimental salmonellosis was confirmed by clinical, bacteriological, and serological tests. The disease in the rabbits was mild, moderately severe, or severe. Some of the animals with the severe form of the illness died at various times after infection. Experimental rabbits were killed by air embolism 1, 3, 5, 7, 9, 14,

Central Research Institute of Epidemiology, Ministry of Health of the USSR, Moscow.

(Presented by Academician of the Academy of Medical Sciences of the USSR K. V. Bunin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 92, No. 12, pp. 674-677, December, 1981.