DYNAMICS OF POSTMORTEM CHANGES IN ADRENERGIC NERVE FIBERS OF BRAIN ARTERIES

P. A. Motavkin, V. M. Chertok, and Yu. I. Pigolkin

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KEY WORDS: brain arteries; adrenergic innervation; postmortem changes.

The study of the adrenergic innervation of arteries of the human brain, as opposed to the animal brain, has not been undertaken on a wide scale. According to some workers [2, 3], comparable results can be achieved only if material taken a relatively short time after death is used. Not long ago the present writers [1] described the possibility of significantly increasing the duration of luminescence of adrenergic fibers in man after preliminary use of pharmacologic agents.

The object of this investigation was to study the number and intensity of luminescence of adrenergic nerve fibers in brain arteries of the rat and man in the late periods after death, in cadavers preserved at different temperatures.

EXPERIMENTAL METHOD

The adrenergic innervation was studied by the method of Furness and Costa [4] in branches of the middle cerebral artery $300-200~\mu$ in diameter. The work was done on four groups of male albino rats aged 8-12 months. The brain of the animals of group 1, which was used as the control, was removed immediately after death, caused by exsanguination. The pia mater was removed together with the blood vessels, stretched on a slide, dried, and treated with a 2% solution of glyoxylic acid. The preparations were examined under the LM-2 luminescence microscope, using DS-1 and SZS-7 filters. The intensity of luminescence was estimated by means of the FMÉL-1A photometric attachment with a 0.1-mm probe under immersion. To estimate the density of the adrenergic nerve plexuses quantitatively the number of nerve fibers per mm² arterial wall was counted. The results were subjected to statistical analysis. Cadayers of the animals of group 2 were kept after death at 18-20°C, those of group 3 at 10°C, and those of group 4 at 4°C. The material was studied every 2 h until the appearance of significant differences in density of the nerve fibers. Relative humidity (50-55%) was checked against readings of the M-21 one-day hygrograph.

To study adrenergic nerve plexuses of branches of the middle cerebral artery in man, material obtained at forensic medical autopsies from cadavers of persons aged 18-45 years dying as a result of road accidents was used. For postmortem detection of the adrenergic nervous apparatus vessels of the pia mater of persons whose cadavers were kept after death either at 18-20°C (group 1) or in a cold room at 4°C (group 2) were studied.

EXPERIMENTAL RESULTS

A dense network composed of small loops, formed by interweaving of transverse, longitudinal, and oblique adrenergic nerve fibers, was discovered on arteries from the animals of group 1 (Fig. la). Multiple varicosities, giving emerald green luminescence, were clearly visible. The largest number of nerve fibers and the most intense fluorescence were observed on the yessels in this group of rats (Table 1).

In all groups of experimental animals 2 h after death a very small decrease in the brightness of fluorescence of the adrenergic fibers was observed (P > 0.5) without any appreciable change in density of the nervous plexuses.

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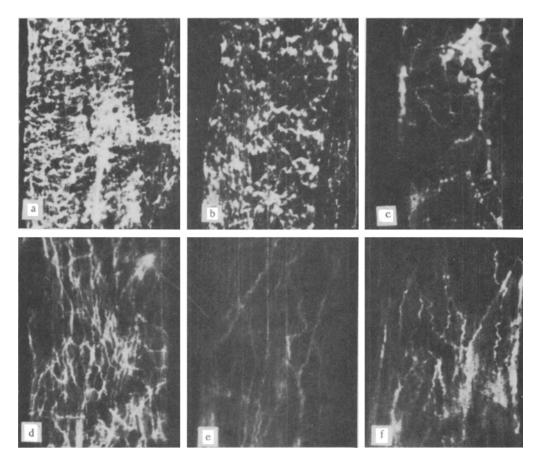


Fig. 1. Postmortem changes in adrenergic innervation of brain arteries: a) rat, group 1 (control); b) rat, group 2 (4 h after death); c) rat, group 2 (6 h after death); d) man, group 1 (2 h after death); e) man, group 1 (6 h after death); f) man, group 2 (10 h after death). Method of Furness and Costa. $200 \times$.

A significant decrease in the intensity of fluorescence of the nerve fibers was found for the first time 4 h after death in the rats of group 2 (P < 0.05). Despite some widening of the loops of the nerve net as a result of disappearance of fluorescence in the thinnest nerve fibrils (Fig. 1b) no significant differences in the density of innervation of the vessels compared with the control could be detected at this period (P > 0.5). In the remaining groups of animals the number and brightness of luminescence of the adrenergic nerve fibers on branches of the middle cerebral artery showed no significant change.

A significant decrease in the number of adrenergic fibers in the vessels (P < 0.001) was observed in animals of group 2 6 h after death and it was accompanied by a marked fall in the intensity of fluorescence of the nerve fibers (Fig. lc). By this time a marked decrease in the brightness of luminescence of the adrenergic fibers (P < 0.01) was observed in the arteries of the rats of group 3, but there were no significant differences from the control. Despite the reduction in brightness of luminescence of the fibers, the density of the plexuses showed very little change. Not until 8 h after death were significant (P < 0.01) differences from the control in the number of adrenergic fibers observed in the animals of group 3. In the rats of group 4 at this time a significant decrease (P < 0.01) in the intensity of luminescence of the nerve fibers in the vessels was observed for the first time, but was not accompanied by any significant fall in the number of adrenergic fibers (P > 0.5). Marked differences from the control in the density of innervation of the arteries were not found until 12 h after death (P < 0.01), although the density of the nervous plexuses still remained quite high (Fig. lc).

The study of postmortem material from the subjects of groups 1 and 2 showed that 2 h after death a dense network of fluorescent nerve fibers (Fig. 1d), the number and intensity of luminescence of which were virtually identical in the two groups (P > 0.5; Table 1), was observed on the arteries irrespective of the temperature at which the cadayers were kept.

TABLE 1. Intensity of Luminescence and Number of Nerve Fibers on Brain Arteries (x \pm s $_x^-$)

	Control		At various times after death				
			2 h		4 h		6 h
Group investigated	number of fibers/mm²	intensity of luminescence, conventional units	number of fibers/mm²	intensity of luminescence, conventional units	number of fibers/mm ²	intensity of luminescence, conventional units	number of fibers/mm ²
1	45,6±2,60	21,8±0,79		_			
2	— n=		44,1±1,90	20,8±0,85	$39,4 \pm 3,38$	14,4±1,40	$29,6\pm 2,90$
3			45.8 ± 2.56	=5 21,0±1,96	$43,2\pm 2,37$	$19,6\pm2,01$	$n=5$ $40,4\pm2,93$
4	***************************************		45,6±2,82	$121,0\pm1,43$	44.8 ± 2.29 $n = n = 1$	=5 20,4±0,91 =5	$n = 7$ $42,5 \pm 3,10$ $n = 6$
1	7						
1		MINISTER .	46,2±2,35	27,6±2,51	41,4±2,90	14,9±2,27	$31,4\pm1,90$
2	<u> </u>		$48,3\pm 5,1$	$28,4\pm3,38$	$44,6 \pm 3,60$	$ 22,4\pm2,70$	$n=6 \\ 42,5\pm3,01 \\ n=6$
	investigated 1 2 3 4	Group number of fibers/mm ²	Group number of fibers/mm² intensity of luminescence, conventional units	Group number of intensity of number of fibers/mm²	Group number of intensity of luminescence, conventional units intensity of luminescence, conventional units	Group investigated number of fibers/mm² intensity of luminescence, conventional units number of fibers/mm² $= 1$ $= $	Group investigated number of fibers/mm² intensity of luminescence, conventional units number of fibers

Test object	Group investigated	At various times after death								
		6 h	8 h		10 h		12 h			
		intensity of luminescence, conventional units	number of fibers/mm²	intensity of luminescence, conventional units	number of fibers/mm²	intensity of luminescence, conventional units	number of fibers/mm²	intensity of luminescence, conventional units		
Rat	1 2	9,4±0,83 n=5					_			
	3	14.9 ± 1.10 n=7	$36,4\pm3,10$ $n=$	10.0 ± 1.54		_	_	_		
	4	$18,6\pm1,27$ $n=6$	41.6 ± 3.16 17.3 ± 1.09 $n=7$		$\begin{array}{c c} 39.8 \pm 2.90 & 14.3 \pm 1.30 \\ n = 8 & \end{array}$		$\begin{array}{c ccccccccccccccccccccccccccccccccccc$			
Man	1	9.6 ± 1.47 $n=6$		_	_		_	_		
Man	2	$ \begin{array}{c c} n = 0 \\ 19,1 \pm 1,30 \\ n = 6 \end{array} $	39,4±1,90	$\begin{vmatrix} 14,5\pm1,90 \\ = 8 \end{vmatrix}$	29,4±3,20 n=	$9,6\pm1,80$		_		

However, in the subjects of group 1 a sharp decrease in the level of fluorescence of the adrenergic fibers (P < 0.001) took place after a further 2 h, although the decrease in density of the nervous plexuses did not reach the level of significance (P > 0.5). Marked reduction (P < 0.01) of nerve fibers in the vessels was observed 6 h after death (Fig. le). At that time, significant differences in the intensity of luminescence of the adrenergic fibers (P < 0.01) were observed on the arteries of the subjects of group 2, but there were no significant differences in the concentration of the nerve fibers (P > 0.5). A marked decrease (P < 0.001) in the number of nerve fibers in the vessels of the subjects of group 2 was observed 10 h after death (Fig. 1f).

It can accordingly be concluded that the temperature at which the cadaver is kept has a significant effect on preservation of adrenergic fibers in the arteries of the brain. The optimal time after which full demonstration of the adrenergic fibers can be guaranteed is 4 h in human cadavers kept at $18-20^{\circ}\text{C}$ and 8 h in those kept at 4°C . In both cases the relative humidity must not exceed 50-55%.

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TIME COURSE OF MORPHOLOGICAL CHANGES IN THE SPINAL CORD AFTER EXPOSURE TO NONIONIZING MICROWAVE RADIATION

V. S. Belokrinitskii

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KEY WORDS: nonionizing microwave radiation (NIMR); morphological processes; spinal cord.

According to data in the literature nonionizing microwave radiation (NIMR) causes extensive neurological disorders and disturbs many functions of the body, including visceral functions [2-6, 8, 9]. Nevertheless, the pathogenesis of these disturbances and, more especially, of structural changes in the nervous system itself under the influence of NIMR has been inadequately studied. Only isolated investigations have been carried out in this direction so far as the brain is concerned [1, 7, 10]. No work devoted specially to the study of spinal cord structure under the influence of NIMR has been undertaken.

The object of this investigation was to study the character and time course of development of morphological changes arising in different segments of the spinal cord under the influence of NIMR.

EXPERIMENTAL METHOD

Experiments were carried out on cats and dogs (50 animals altogether), which were exposed to NIMR (wavelength 12.6 cm, intensity $400-500 \text{ mW/cm}^2$) for 1 h (cats) or 4 h (dogs). A number of intact animals constituted the control group. During irradiation the animals were kept in special cages, located in a screened anechoic chamber. The "Luch-58" treatment apparatus, operating on a continuous schedule, was used as the generator.

The spinal cord was studied 1, 10, 20, and 30 days after the end of exposure to NIMR, and for this purpose the animals were anesthetized with ether, decapitated in groups, the spinal cord was removed and pieces of tissue from it, of the necessary size, were fixed in 12% neutral formalin solution and alcohol. Paraffin sections or frozen sections were stained with hematoxylin and eosin and by Yan Gieson's, Nissl's, Zimmermann's, and Cajal's methods,

Segments of the spinal cord were identified by their roots, and also by the shape of the gray and white matter, depicted on photographs of unstained sections. An atlas of the spinal cord [11], in which the author distinguishes 10 regions in each segment and conventionally calls them laminae, was used for this purpose. In each lamina neurons of various types were present and formed the nuclei of the spinal cord, with specific functions.

EXPERIMENTAL RESULTS

Changes in structure of the spinal cord tissue were found by the first day after the end of irradiation in animals exposed to NIMR. The changes increased to the 10th, 20th, and 30th days. The character of the changes was identical, but their intensity differed in neurons located in different regions (laminae) of the spinal cord. The changes were similar in corresponding regions of the cord in different individuals of the same species (cats) and also in animals of the other species (dogs), so that a general description is in order.

A. N. Marzeev Kiev Research Institute of General and Communal Hygiene. (Presented by Academician of the Academy of Medical Sciences of the USSR N. A. Kraevskiî.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 95, No. 5, pp. 98-100, May, 1983. Original article submitted April 13, 1982.