ferential activity of their genome, thereby directing their future cytodifferentiation and adapting them to the greatest possible degree to the tasks they will have to form when the functional systems of the developing organism have been created. The time of appearance of first synaptic contacts on the neuroblastic cell can thus be regarded as the key period in its development, when a qualitative transition from the neuroblastic stage to the stage of the juvenile neuron is completed.

LITERATURE CITED

1. N. Okado, S. Kakimi, and T. Kojima, J. Comp. Neurol., 184, 491 (1979).
2. A. A. Milokhin and I. V. Chernova, Byull. Éksp. Biol. Med., No. 4, 496 (1980).
3. W. E. Windle and J. E. Fitzgerald, J. Comp. Neurol., 67, 493 (1937).
4. J. E. Fitzgerald and W. E. Windle, J. Comp. Neurol., 76, 159 (1942).

EFFECT OF ACOUSTIC STRESS ON THE MORPHOLOGY OF THE RAT SENSOMOTOR CORTEX

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KEY WORDS: acoustic stress; sensomotor cortex; hemorrhages; neurons; neroglia; lysosomes; pigment.

Prolonged exposure to white noise causes structural and functional changes in various formations of the CNS in man and animals. Prolonged exposure to noise leads to disturbance of motor functions also. There is clinical evidence that in persons exposed to noise changes arise in muscle tone and in reflex reactions: Tendon reflexes are increased, but the pharyngeal, palatal, and abdominal reflexes are inhibited. In animals rigidity of movements, motor stereotypy, convulsive seizures, and other disturbances may arise after acoustic stress [4, 6, 7]. There have been only isolated studies of the morphology of the motor cortex under the influence of noise, and these have been undertaken at the light-optical level [7]. Most studies of brain morphology following exposure to noise have been carried out on different formations of the auditory system [1, 3, 5, 8, 9]. The problem of the extent to which noise affects the structure of nonspecific (nonauditory) formations of the CNS, including the motor centers of the brain, has not yet been settled.

The object of this investigation was to study the morphology of the sensomotor cortex and to compare its structure and ultrastructure with changes in the structure of the auditory cortex of rats during acoustic stress.

EXPERIMENTAL METHOD

Noninbred sexually mature male rats weighing 180-200 g were used. The animals were exposed to noise for 14 h daily for 7, 14, and 21 days. The source of the noise was a GZ-12 generator of low-frequency signals, to the output of which columns with a power of 10 W were connected. The frequency band used was 250-3500 Hz and the intensity 80-90 dB above the threshold of audibility of the human ear. A special contact breaker periodically interrupted the noise. The ratio between noise and pause was 1-2 sec. The structure and ultrastructure of the sensomotor cortex were studied in 18 experimental and fixed control rats. Brain sections for light-optical microscopy were stained by Nissl's, Cajal's, and Hortega's methods in Aleksandrovskaya's modification. Pieces of brain for electron-microscopic study were fixed in a 5% solution of glutaraldehyde, then postfixed in 1% OsO_4 solution in phosphate buffer and embedded in Arladite. Sections were cut on the LKB-III Ultrotome, stained by the method in [10], and studied in the JEM-100B electron microscope.

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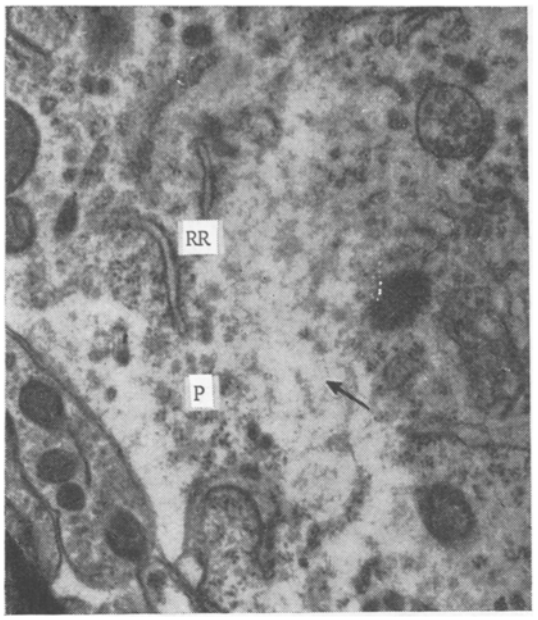

Fig. 1

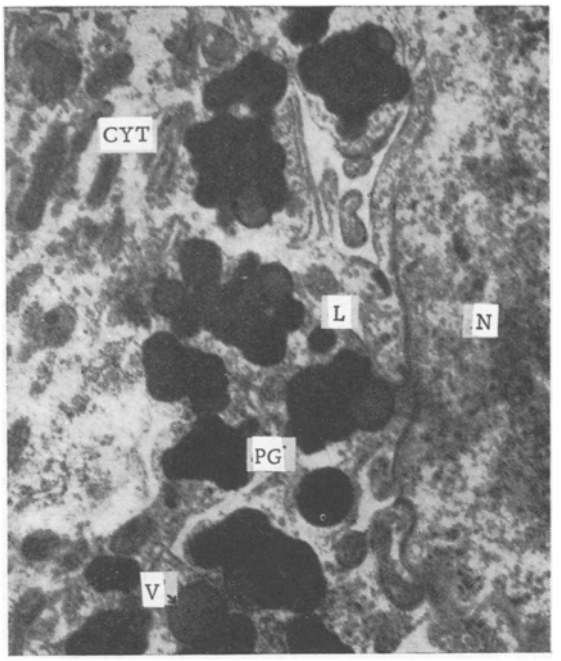

Fig. 2

Fig. 1. Area of cytoplasm of nerve cell from deep layers of sensomotor cortex of rat after exposure for 21 days to acoustic stress. Focal peripheral chromatolysis. Decrease in number of channels of rough endoplasmic reticulum (RR) and of free ribosomes and polysomes (P); focus indicated by arrow. 300,000 ×.

Fig. 2. Area of nucleus (N) and cytoplasm (CYT) of nerve cell from deep layers of sensomotor cortex of rat after 21 days of acoustic stress. Accumulation of lysosomes (L), filled with pigment granules (PG) and vacuoles (V), visible in cytoplasm. 20,000 ×.

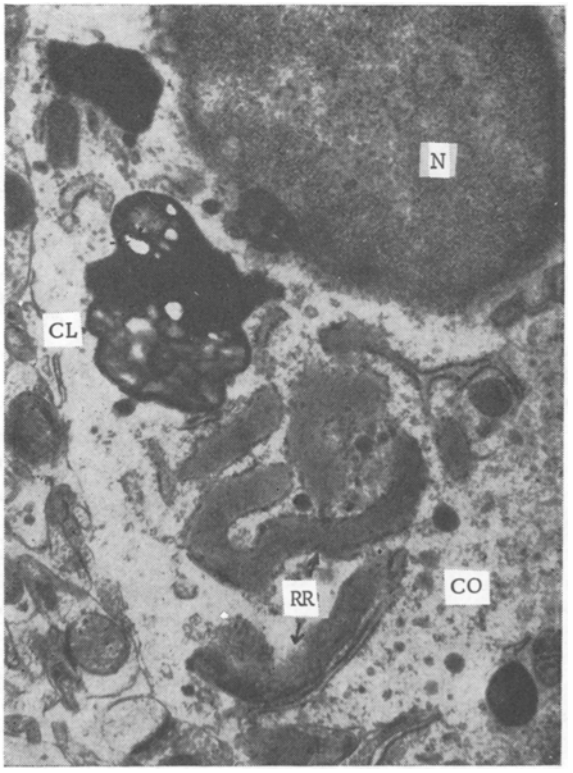

Fig. 3. Area of nucleus (N) and cytoplasm of satellite oligodendrogliocyte (CO) from surface layers of sensomotor cortex of rat after 21 days of acoustic stress. **Cytolysome** (CL) present in cytoplasm. Channels of rough endoplasmic reticulum (RR) dilated and filled with turbid secretion. 20,000 ×.

EXPERIMENTAL RESULTS

In rats exposed for 7 days to acoustic stress considerable fluctuations of arterial blood pressure were observed in the course of the 24-h period. In the sensomotor cortex, just as elsewhere in the cortex, the local cerebral blood flow was increased on average by 40%. If exposure to noise was prolonged for 14 days and, in particular, for 21 days, the rise of blood pressure became more permanent, whereas the local cerebral blood flow decreased. The animals developed a motor stereotype, and some of them developed adynamia, which became more marked and enduring after 21 days of acoustic stress.

Morphological examination of the brain of the rats after exposure for 7 days to noise showed petechial pericapillary hemorrhages in the sensomotor cortex, just as in the auditory and other areas of the brain. The response of the astrocytic glia and microglia was productive-focal in character. Microglial cells and fibrous astrocytes accumulated in particularly large numbers near the hemorrhages. After 14 and 21 days of acoustic stress no fresh hemorrhages appeared but the old ones were absorbed and replaced by glial scar tissue. Cells of the microglia were seen in the form of small clusters after exposure to noise for 14 days, and as single cells located near the capillaries after exposure for 21 days. The first marked features of morphological changes in the neurons and neuroglia were found in the animals after exposure to noise for 14 days, when cells with peripheral tigrolysis appeared in the cortex. Under the electron microscope a decrease in the quantity of rough endoplasmic reticulum and, in particular, of free ribosomes and polysomes near the cell membrane could be seen in the nerve cells (Fig. 1). These foci of chromatolysis became more extensive in the animals after exposure to noise for 21 days, but unlike in the auditory cortex, total chromatolysis of the cells was not found there. In some cells in the surface and deep layers of the sensomotor cortex the number of lysosomes, containing pigment granules of lipofuscin and vacuoles, was increased after exposure to noise for 14 days. After exposure for 21 days the number of lysosomes continued to increase. They joined together to form cytolysomes (Fig. 2). Many cells were overloaded with lysosomes containing pigment, just as in the auditory cortex after acoustic stress [2].

Ultrastructural changes in the sensomotor cortex similar to those in the auditory cortex were found also in the glial cells. In cells of the satellite, free, pericapillary glia lysosomes loaded with pigment also accumulated and merged with one another and with multivesicular bodies to form cytolysomes. Accumulation ofopaque (turbid) secretion in the cavities of the reticulum with secretion of this type were greatly dilated, to form cyst-like structures in some cases. The most marked destructive changes were observed in the satellite and pericapillary glia. Changes in the free neuroglia (oligoglia) were less marked.

The ultrastructural changes found in the neurons and neuroglia of the sensomotor cortex, although less diffuse, nevertheless had qualitative similarity to changes described by the writers in the auditory cortex after exposure to noise [2]. The results are evidence that acoustic stress has a direct influence on the sensomotor cortex of animals, and this could be one cause of the disturbance of their motor functions.

Analysis of the results obtained previously and those of the present investigation leads to the conclusion that exposure to noise under chronic conditions gives rise to serious dystrophic changes in neurons and glial cells not only in the auditory cortex, but also in the sensomotor cortex. In addition, noise in young animals causes morphological changes in the neurons and glia characteristics of aging, evidence that acoustic stress has some influence on the process of premature aging.

LITERATURE CITED

1. M. M. Aleskandrovskaya and R. Chizhenkova, Fiziol. Zh. SSR, No. 6, 870 (1973).
2. N. I. Artyukhina, O. F. Kuvaeva, and I. P. Levshina, "Effect of white noise on the ultrastructure of the rat auditory cortex," Byull. Éksp. Biol. Med., No. 3 (1981).
3. G. N. Krivitskaya, The Action of Loud Noise on the Brain [in Russian], Moscow (1964).
4. L. V. Krushinskii and L. N. Molodkina, Usp. Sovrem. Biol., 44, No. 2(5), 220 (1958).
5. V. N. Larina and I. P. Levshina, in: Systemic Properties of Tissue Organizations [in Russian], Moscow (1977), pp. 145-146.
6. A. A. Manina, M. M. Khananashvili, and N. N. Lazuko, Zh. Vyssh. Nerv. Deyat., No. 4, 686 (1971).
7. S. Nichkov and G. N. Krivitskaya, Acoustic Stress and Cerebrovisceral Disturbances [in Russian], Moscow (1969).

8. G. Rossi, M. Robecci, and M. Penna, Arkh. Anat., No. 8, 14 (1978).
9. C. A. Hamberger and H. Hyden, Acta Oto-Laryng., 61, Suppl., pp. 81-89 (1945).
10. R. S. Reynolds, J. Cell Biol., 17, 208 (1963).

ADRENERGIC INNERVATION OF VENOUS AND LYMPHATIC MICROVESSELS

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KEY WORDS: microvessels; arteries, veins; lymphatics; adrenergic innervation; mesentery.

Venous and lymphatic microvessels, like arterial, are essential components of the structural and functional units of the microcirculation [1-3, 5, 6, 11]. Their integrative activity at tissue and organ levels is largely determined by neurotransmitter control mechanisms, especially sympathetic vasomotor and trophic influences [4, 7, 9, 10, 13]. This accounts for the interest shown by research workers in the adrenergic (sympathetic) innervation of vessels of the microcirculation. Numerous studies using the fluorescence-microscopic technique of Falck and Hillarp have substantially widened and clarified our present knowledge of the morphological substrate of the adrenergic component of autonomic vascular innervation. However, not all structural components of the microcirculatory syytem have been studied in equal measure. Definite preference has been shown for vessels of arterial type, as is shown by the quite complete description of their adrenergic innervation over their whole extent as far as the precapillary arterioles. Much less attention has been paid to venous microvessels. Usually it is stated as a fact that innervation structures in the arterial walls predominate quantitatively over those in the veins, and the sparseness of the adrenergic innervation of the veins is emphasized [8, 11]. So far as the lymphatic microvessels are concerned, the question of their adrenergic innervation still remains largely unexplored. There is nothing more than the simple mention, en passant, that adrenergic nerves make contact with the walls of lymphatic microvessels [8].

The writers have studied the adrenergic innervation of microvessels of the mesentery and have paid special attention to the innervation connections of venous and lymphatic microvessels.

EXPERIMENTAL METHOD

The mesentery of the small and large intestines of noninbred rats of both sexes weighing 180-220 g was studied. Immediately after decapitation (under ether anesthesia) laparotomy was performed, the mesentery removed and cut into separate areas, and stretched out on slides. After drying in a current of air from a room fan (3-5 min) the specimens were placed in a chamber containing paraform (humidity 50-51%) and kept there for 3 h in an incubator at 37°C. The specimens were examined and photographed on the LYUMAM-IZ luminescence microscope. In some cases the preparations were stained during microscopy with an aqueous solution of acridine orange to reveal the cell composition of the tissue substrate of the mesentery.

EXPERIMENTAL RESULTS

Microscopic examination of total preparations of the mesentery stained histochemically for catecholamines showed most clearly the presence of periarterial adrenergic plexuses accompanying all stages of the arterial system as far as precapillary arterioles. A conspicuous feature was the numerous side branches of the periarterial plexuses, which ran for a varied distance in the tissue of the membrane and joined the wall of the adjacent vessels. In some cases these vessels were tiny collecting veins and postcapillary venules, in others they were lymphatic microvessels. The latter were revealed sufficiently completely because

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