EFFECT OF WHITE NOISE ON AUDITORY CORTICAL ULTRASTRUCTURE IN RATS

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Like other stress factors, white noise is widely used in research for the simulation of experimental neuroses in animals. Neurophysiological studies have shown that prolonged exposure to noise leads to considerable functional disturbances of the CNS [5, 8]. Some of these investigations, demonstrating the pathogenic effect of chronic exposure to noise on brain morphology in animals, have been undertaken by light-microscopic methods [1, 6, 7, 11, 13]. Changes in the ultrastructure of the auditory centers following exposure to noise have been reported in only a few investigations [9, 12].

The investigation described below is part of a study of structural changes in the cerebral cortex in animals with experimental neuroses produced, in particular, by exposure to noise and also to a combination of noise and other factors (electric shock, angiotensin-II, etc.). Its aim was to study the ultrastructure of the cerebral cortex in rats following exposure to white noise alone under chronic experimental conditions.

EXPERIMENTAL METHOD

Experiments were carried out on 12 noninbred male rats weighing 200 g. The rats were exposed to noise for 14 h daily for 21 days. The source of noise was a GZ-12 low-frequency signal generator, to the output of which a 10-W column was connected.

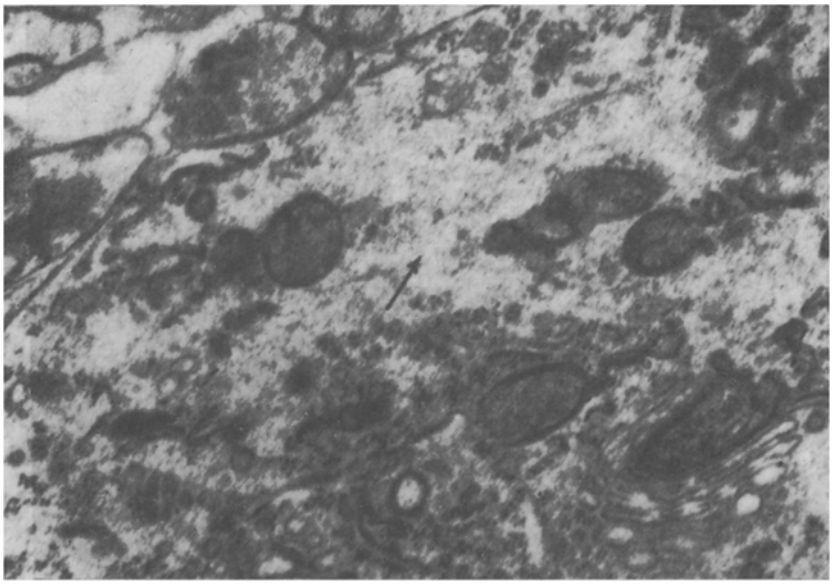

Fig. 1. Focal peripheral chromatolysis (arrow) in nerve cell from surface layers of cortex of experimental rats after exposure to noise. Reduction of ribosomes visible near cell membrane; 50,000×.

KEY WORDS: auditory cortex; ultrastructure; white noise; pigment.

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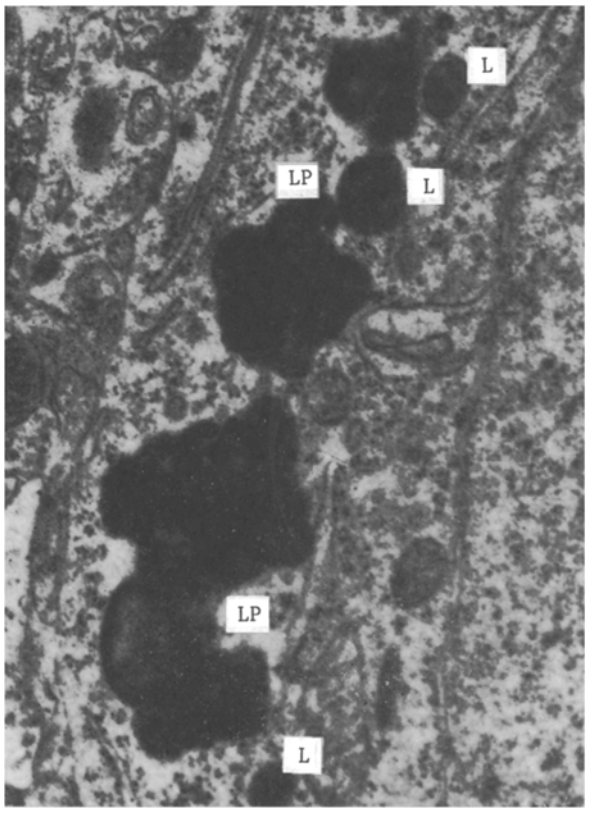

Fig. 2. Accumulation of lysosomes (L) and of lipofuscin (LP) in cytoplasm of nerve cell from surface layers of auditory cortex in experimental rats after exposure to noise, 30,000 ×.

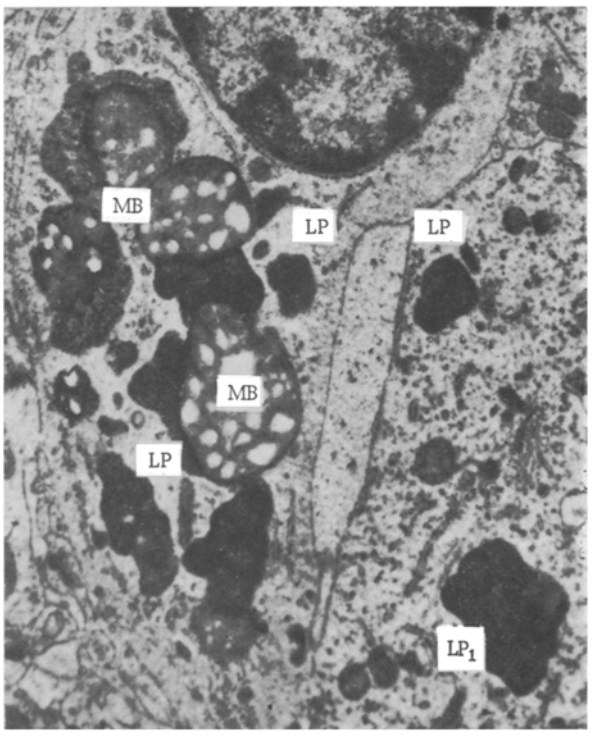

Fig. 3. Accumulation of lipofuscin granules (LP) and multivesicular bodies (MB) in cytoplasm of glial satellite cell (GL). Lipofuscin granules (LP_1) also present in cytoplasm of nerve cell, 25,000 ×.

The frequency band used was 250-3500 Hz, and the intensity 90-95 dB above the threshold of audibility of the human ear. The sound was periodically interrupted by means of a special switch. The sound—phase ratio was 2 sec:1 sec.

Rats were taken for morphological study immediately after the last exposure to noise. The animals were decapitated and the brain removed.

Pieces of auditory cortex were fixed in 2.5% cold glutaraldehyde solution and postfixed in 1% OsO_4, made up in phosphate buffer, pH 7.4. The pieces were embedded in Araldite. Sections were cut on the LKB-III Ultrotome, stained with lead citrate by Reynolds' method [15], and studied in the JEM-100B electron microscope.

The ultrastructure of nerve and glial cells and also of blood vessels was studied.

For examination of cortical preparations under the light microscope, the animals' brain was fixed in formalin and sections stained by Nissl's method.

EXPERIMENTAL RESULTS

Under the electron microscope three groups of neurons, differing in the degree of osmiophilia of the matrix of the cytoplasm and nucleus, and also in the content of organelles, could be distinguished in the auditory cortex, just as in other parts of the cerebral cortex. These were moderately osmiophilic, dark, and pale cells. The number of organelles in these groups of neurons varied considerably, depending on the type of the nerve cells and also on the area of the cytoplasm.

Long exposure to noise led to the appearance of many pale cells with different degrees of chromatolysis in the auditory cortex. The number of ribosomes and polysomes and also of tubules of the rough and smooth endoplasmic reticulum was reduced in these cells. Cells with focal peripheral chromatolysis were found most frequently (Fig. 1), and total chromatolysis

was more rarely observed. Cells with lysis of their basophilic substance were found also in Nissl preparations in different layers of the auditory cortex.

In individual cells chromatolysis was combined with changes in other organelles of the cytoplasm — swelling of the mitochondria, vacuolation of elements of the endoplasmic reticulum. The nuclei in such cells were pale, with a moderate content of chromatin located mainly near the nuclear membrane. The nucleoli also were more frequently adjacent to the nuclear membrane, without any appreciable changes in size. Chromatolysis in the auditory cortical cells and ectopia of the nucleoli after various types of exposure to noise have also been observed by other workers during light-microscopic investigations [11, 13].

The existence of different degrees of chromatolysis can be taken as an indication that exposure to noise affects auditory cortical nerve cells differently. These differences may arise because different numbers of afferent fibers carrying acoustic impulses terminate on auditory cortical neurons.

Another characteristic type of change in the nerve cells after exposure to noise was the response of the lysosomes and destructive changes in them. The action of noise led to a marked increase in the number of small primary lysosomes. Parallel with proliferation, fusion of the lysosomes took place, with the appearance of pigmented lipofuscin granules and vacuoles inside them (Fig. 2).

Many glial cells were overloaded with pigment. Besides lipofuscin, lipid drops were found in some cells. Changes of this sort are an indication of a unique type of metabolic disturbances in nerve and glial cells during acoustic overloading.

The lysosomal reaction revealed by this investigation has been described by other workers after exposure to sound and also to other factors [4, 12].

It is also reported in the literature that after chronic exposure to noise activity of tissue respiratory enzymes (cytochrome oxidase and succinate dehydrogenase) and also the ATP level in the brain are reduced [10].

According to Bogolepov [4], the response and destruction of the lysosomes during experimental hypoxia, which is accompanied by pericapillary edema of the glia, is not a primary effect but results from changes in the mitochondria and the rough and smooth endoplasmic reticulum.

No significant changes in the mitochondria or in the endoplasmic reticulum could be found in cells with a marked response and destruction of the lysosomes on the 21st day of exposure to noise. No changes which could lead to the development of tissue hypoxia likewise could be discovered in the blood vessels. In addition, in a study of the effect of angiotensin-II on cortical ultrastructure in the rat brain, marked edema of the pericapillary glia was observed; this led to compression of the capillary network and to the development of hypoxia of the brain tissue. However, in the nerve and glial cells of these animals nothing resembling a lysosomal reaction took place [3]. That is why there were no apparent grounds for associating the lysosomal response and destruction of the lysosomes in nerve and glial cells under the influence of noise with hypoxia.

Rossi et al. [12] attribute the increase in the number of lysosomes in cells of the ganglion of Corti to the fact that under the influence of noise nerve cells must interact with qualitatively unusual metabolites or with a larger quantity of metabolites than usual, as a result of the increased functional demands.

It is impossible to deduce from the facts described above an unequivocal interpretation of the mechanism of development of ultrastructural changes in the nerve cells and, in particular, in the glial cells in response to noise. However, it can be concluded that exposure to noise under chronic conditions is a pathogenic factor leading to disturbance of metabolic processes and to the development of destructive changes in the nerve and glial cells of the auditory cortex. These changes can explain the prolonged disturbances of higher nervous activity observed in animals after the end of exposure to noise.

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SYNTHESIS OF RIBOSOMAL RNA IN PALE AND DARK CEREBRAL CORTICAL NEURONS OF RATS AFTER THERMAL TRAUMA

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Many publications have been devoted to the problem of dark and pale cells (see the surveys [2, 6]), and give comparative data on enzyme activity, content of organelles, proteins, lipids, mucopolysaccharides, glycogen, and certain other substances in them [9]. These data, it must be pointed out, give only an indirect idea of the functional difference between pale and dark cells. Direct measurement of functional activity (the velocity of protein synthesis) by dark and pale neurons was undertaken by Meitner et al. [11], who showed autoradiographically that pale cells in normal rats are functionally more active (incorporate labeled leucine more rapidly).

Having chosen dark and pale neurons as the test objects, the authors attempted to analyze how one of the most important processes — RNA synthesis, ultimately responsible for the specific function of the neuron and regeneration of structures necessary for the performance of this function, takes place in these cells in the course of a disease. Previously a significant increase in the rate of synthesis of nucleolar RNA was found in neurons of burned animals [3].

This paper gives the results of an electron-autoradiographic study of the development of this response in dark and pale neurons separately.

EXPERIMENTAL METHOD

Under ether anesthesia a burn of the IIIb-IV degree (20% of the body surface) was inflicted on noninbred albino rats weighing 180 g. RNA synthesis was investigated in intact animals (control) and 1, 12, 72, and 144 h after burns (five animals at each time). Altogether 25 animals were used.

For the electron-autoradiographic investigation of RNA synthesis a special needle was introduced into the animals' brain (cerebral cortex, cutaneous-motor area, area PA^m), and

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