alveolar surface area (33.3), volume of the lungs (25.2), and area of the lumen of the venules (11.8), arterioles (8.2), and bronchioles (4.7).

Exposure to gradually increasing cold, simulating the gradual transition from fall to winter temperatures in the Far North, thus leads to the formation of stable and adequate adaptation of the lungs of experimental rats, even in the case of severe hypothermia (–15, –20°C). Morphometric analysis of the acinus confirmed the general trend of structural adaptation to cold toward an increase in the alveolar and capillary surface area, the anatomical dead space, and hypervolemia of the arterial and capillary components of the pulmonary circulation, described previously both experimentally [5, 6] and in adapted inhabitants of the north [3]. The new data revealed by the present investigation are to the effect that long-term gradual cooling causes a series of stages of structural changes in the acinus: After initial functional stress definite stabilization took place, followed by adequate adaptation of the lungs of the experimental rats. These experimental data form the theoretical basis for the planning of preventive measures against lung diseases in the inhabitants of the North not only in the harsh winter season, but also at below comfortable temperatures (5 and 0°C).

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ULTRASTRUCTURAL CHANGES IN SENSOMOTOR CORTICAL NEURONS DURING PROLONGED HYPOKINESIA IN RATS

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KEY WORDS: neurons; sensomotor cortex; hypokinesia; posthypokinetic period.

Many clinical and experimental studies have demonstrated the adverse effect of restricted movement on activity of all systems of the body and, in particular, the CNS. However, the study of the structural organization of the CNS and of the brain in particular during hypokinesia is essentially only just beginning [7, 9, 13].

There is no information in the literature on reversibility of structural changes in cortical neurons after the end of long-term hypokinesia.

The aim of this investigation was to study changes in ultrastructure of sensomotor cortical neurons in rats during 90 days of hypokinesia and at different times of the posthypokinetic period.

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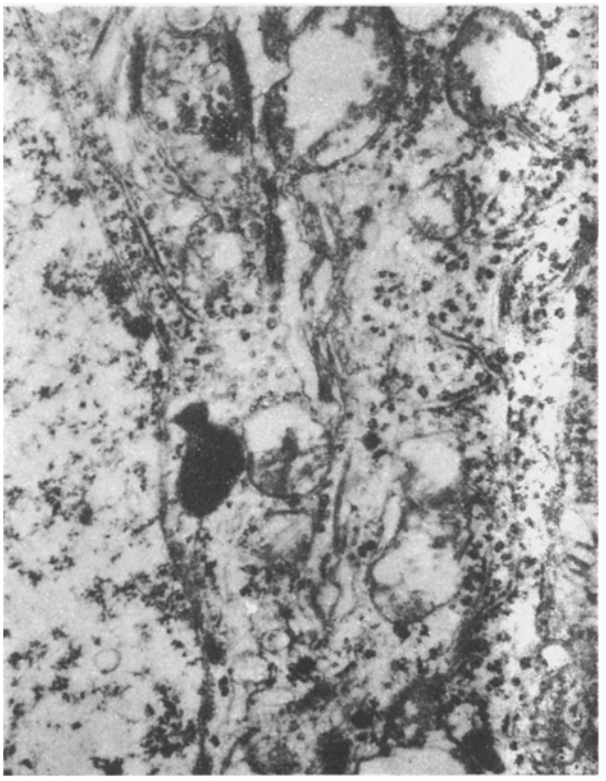

Fig. 1

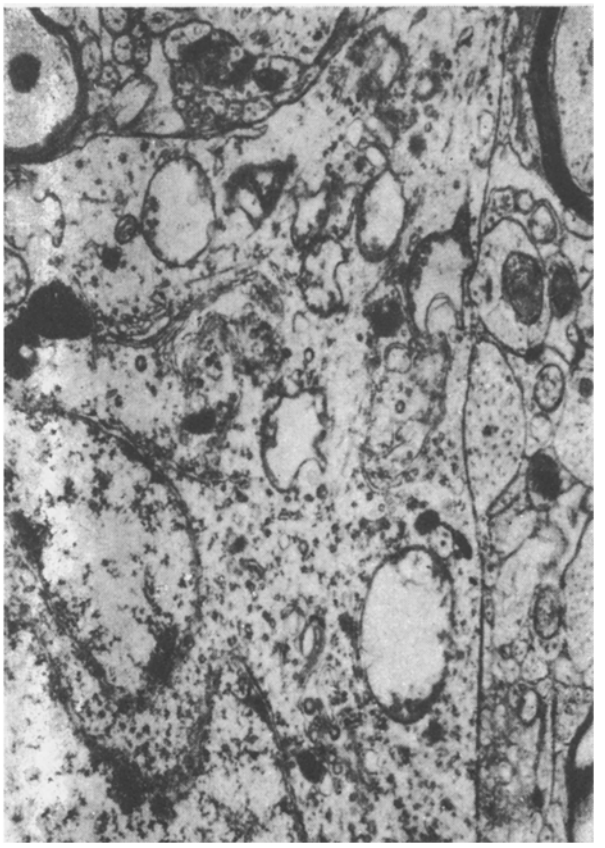

Fig. 2

Fig. 1. Rat sensomotor cortical neuron with structural changes in cytoplasmic organelles after 90 days of hypokinesia (14,000×).

Fig. 2. Ultrastructure of rat sensomotor cortical neuron 7 days after ending of hypokinesia (11,600×).

EXPERIMENTAL METHOD

To create a model of hypokinesia, month-old Wistar rats were kept in special restraining cages, the walls of which could be adjusted depending on the size of the growing animal. The rats were killed after 90 days of hypokinesia and 7, 14, and 21 days after reversion to normal animal house conditions. Intact animals of the same age served as the control. Material for electron microscopic analysis was processed in the usual way. Sections were examined in the field of vision of a "Hitachi-PE" electron microscope.

EXPERIMENTAL RESULTS

After 90 days of hypokinesia the predominant changes in the rat sensomotor cortex were dystrophic changes in the neurons: a decrease in the content of nuclear chromatin and of free cytoplasmic ribosomes, swelling of individual cisterns of the rough endoplasmic reticulum and components of the lamellar complex, sometimes with rupture of the membranes of that complex (Fig. 1). Mitochondria of nerve cells underwent significant changes. Marked swelling and clearing of their matrix, focal or total destruction of the cristae, and conversion of the organelles into vacuoles or myelin-like structures, were observed, evidence of a disturbance of energy metabolism and enzyme activity under the conditions of hypokinesia. Biochemical analysis showed that after 90 days of hypokinesia monoamine oxidase activity and the serotonin content were reduced in subfractions of cellular mitochondria isolated from the rat sensomotor cortex [5]. Similar ultrastructural changes in mitochondria of cortical neurons were observed in experimentally induced hypoxia [2, 8] and in old animals [1, 7, 8], a possible indication of the development of cerebral hypoxia during long-term hypokinesia.

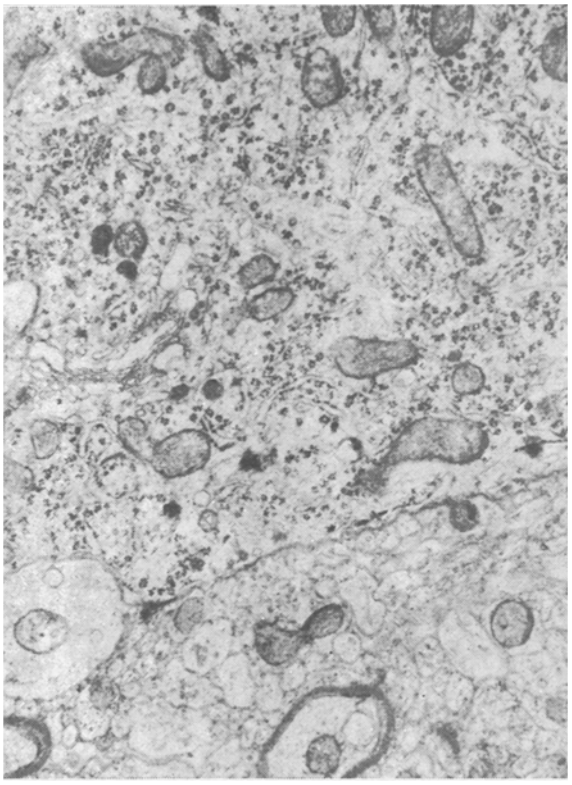

Fig. 3. Structure of cytoplasm of rat sensomotor cortical neuron on 21st day of posthypokinetic period (11,600×).

Vacuolation of the cytoplasm was noted in many nerve cells; large vacuoles were formed not only from mitochondria, but also from cisterns of the rough endoplasmic reticulum and components of the lamellar complex. Vacuolation of the cytoplasm, accompanied by preservation of the normal structure of the nucleus and nucleolus, was completely reversible and corresponded to a sharp decline in functional activity of the neuron [10]. According to physiological observations, during long-term experimental hypokinesia the electrical activity of various parts of the brain is in fact sharply depressed [3, 6] and this is accompanied by a sharp decrease in functional activity of sensomotor cortical neuron populations in rats with respect to parameters of evoked potentials and excitability recovery cycles [14].

Marked activation of the nucleus was clearly observed in most sensomotor cortical neurons 7 days after the ending of motor deprivation: increased folding of the nuclear membrane, accumulation of chromatin masses near the inner nuclear membrane and their apparent outflow into the cytoplasm, and displacement of the nucleolus toward the periphery of the nucleus. At this stage of the experiment the number and extent of cisterns of the rough endoplasmic reticulum and lamellar complex were increased in the cytoplasm of the nerve cells, accompanied by proliferation of the vesicular component and of electron-dense inclusions and lysosomes of various sizes, i.e., signs of intracellular reparative regeneration were apparent [11, 12].

A distinguishing feature of the reparative changes in the mitochondria 1 week after restoration of motor function was that quite large organelles, with a translucent matrix and virtually free from cristae, became curiously shaped because of bending and invagination of their limiting membranes. Often the mitochondria in such cases appeared to take up part of the cytoplasm, sometimes with a vesicle (Fig. 2). Similar changes in mitochondria have been found in sensomotor cortical neurons of cats after anoxia lasting 6 min, and they are regarded as a manifestation of "endocytosis," aimed at replenishing nutrients or intensifying biochemical processes on the enlarged surface [4].

The intensity of repair processes in most cortical neurons was increased 14 days after the end of motor deprivation, as shown by an increase in the number of ribosomes and polysomes, in the number and extent of cisterns of the rough endoplasmic reticulum and lamellar complex, hyperplasia of its structures, and the appearance of multiple invaginations of the nuclear membrane. During this period the mitochondria decreased in size and their cristae increased in number and were arranged more regularly in the electron-dense matrix.

After 21 days of recovery of motor activity neurons with slight ultrastructural changes and with signs of reparative changes affecting the nucleus and cytoplasmic organelles were found in the sensomotor cortex of the animals, together with nerve cells with an almost completely restored ultrastructure and neurons with hyperplasia of their organelles (Fig. 3). Repair processes during this period after the end of motor deprivation took place in accordance with the general rule [11] and were expressed as normalization of degeneratively changed neurons through intracellular regeneration, and as compensatory hyperplasia of ultrastructures in residual cells after death of the others.

These results are evidence of considerable disturbances in neuronal ultrastructure in the rat sensomotor cortex during prolonged motor deprivation and of the protracted nature of repair processes after the end of hypokinesia. These factors must be taken into account during the planning of measures aimed at abolishing the after-effects of hypokinesia.

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