This morphological picture indicates the development of intraosseous homeostasis and preservation of viability in cells of the vessel walls during restoration of the circulation in the damaged bone under favorable conditions. Restoration of the circulation of tissue fluid, it must be assumed, was facilitated by making an incision through the whole thickness of the cortical lamina in the middle of the diaphysis, for without it no such restoration of the staining properties of the vessel wall cells took place. Preservation of viability of the vascular cells was manifested as recovery not only of the staining properties, but also of their biological properties — their ability to proliferate. Recovery of these properties is probably attributable to the pericytes of the vessels forming the microcirculatory network of the vascular canals. As Rusakov [1] pointed out, the long-term viability of the vascular network in bone organs is formed in the process of phylogeny, in the struggle for existence and survival of the organism. Thus we were able to identify the beginning of accumulation of fibroblast-like cells near the cortical lamina, which was postulated by Fridenshtein et al. [2]. The model which we used enabled the beginning of mechanocyte development to be clearly traced. This was facilitated by normalization of the intraosseous circulation in the form of the appearance of tissue cysts in the medullary canal. We regard them as analogs of lymphatic microcirculatory vessels in other tissues.

LITERATURE CITED

1. A. V. Rusakov, Pathological Anatomy of Diseases of the Osseous System [in Russian], Moscow (1959).
2. A. Ya. Fridenshtein and E. A. Luriya, The Cellular Bases of the Hematopoietic Microenvironment [in Russian], Moscow (1980).
3. W. H. Knospe, S. A. Gregory, W. Fried, and F. E. Trobaugh, Blood, 41, 519 (1973).

REPARATIVE CHANGES IN LATERAL HYPOTHALAMIC NEURONS DURING FOOD DEPRIVATION

B. M. Abushov

UDC 616.831.41-091.81-003.9-02:616.393-092.9

KEY WORDS: reparative changes; food deprivation; hypothalamus; ultrastructure of the neuron.

During food deprivation polymorphic ultrastructural changes arise in neurons in the lateral hypothalamic region (LHR) of the rat brain, and the dynamics of these changes depends on the stage of the experiment [1, 2]. In various pathological states not only destructive changes, but also processes of a reparative character are observed [4, 5, 7].

Reparative changes in neurons of different parts of LHR were studied in rats during food deprivation.

EXPERIMENTAL METHOD

Experiments were carried out on male Wistar albino rats weighing initially 190-200 g. Ten animals, receiving water and food *ad lib.* served as the control group. The experimental group consisted of 20 rats, which were killed 1, 3, 5, and 7 days after the beginning of starvation (5 animals at each time). The animals were anesthetized with ether and the brain perfused through the aorta with a solution of 2.5% glutaraldehyde and 2% paraformaldehyde in phosphate buffer, pH 7.2-7.4. The anterior, middle, and posterior parts of LHR were excised and the material was treated with a 2% buffered solution of osmic acid. Pieces of brain were dehydrated and embedded in a mixture of Araldite and Epon-812. The sections were examined and photographed with the HU-600 electron microscope (Hitachi, Japan).

Laboratory of Brain Ultrastructure, Brain Institute, All-Union Mental Health Research Center, Academy of Medical Sciences of the USSR, Moscow.

(Presented by Academician of the Academy of Medical Sciences of the USSR, A. P. Avtsyn.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 101, No. 2, pp. 205-207, February, 1986.

0007-4888/86/0002-0240$12.50

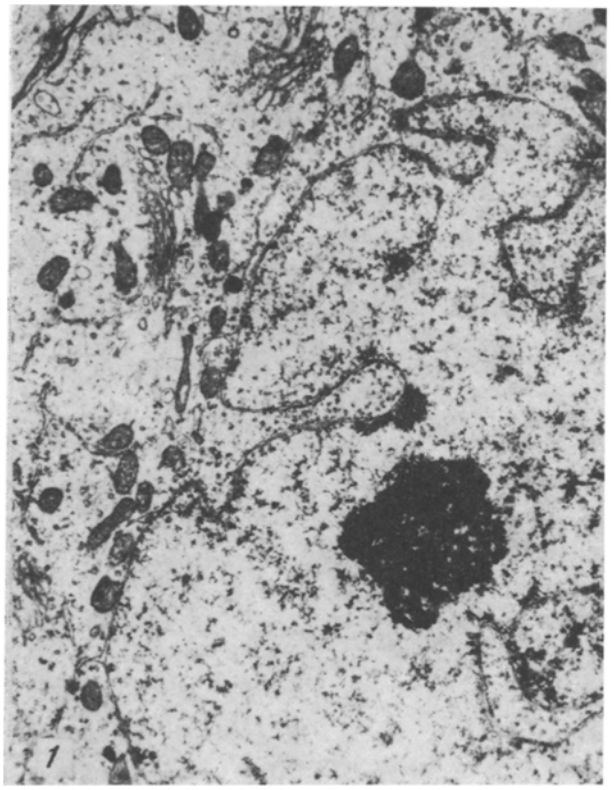

Fig. 1

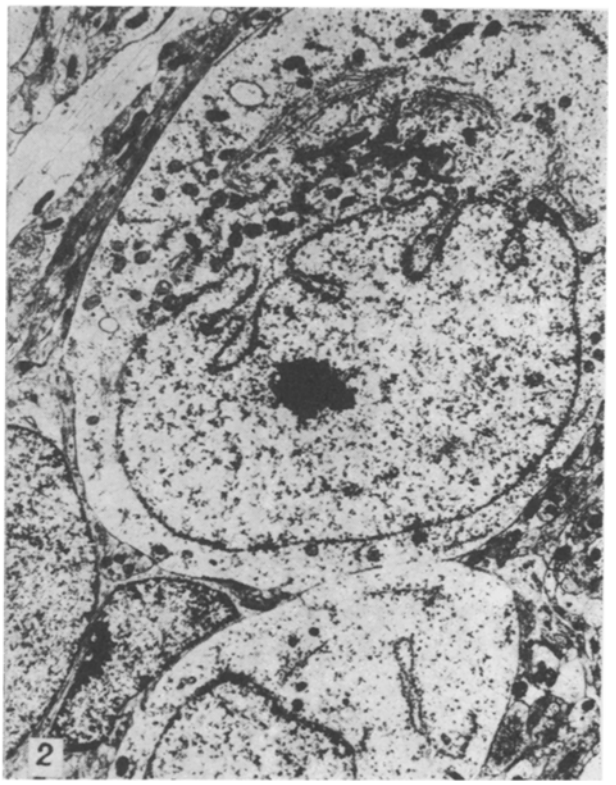

Fig. 2

Fig. 1. Deep invaginations of nuclear membrane in large neuron of anterior part of LHR of rat brain 3 days after beginning of food deprivation (9000 ×).

Fig. 2. Formation of assemblages of cells with the appearance of "paired" neurons in anterior part of LHR of rat brain on 3rd day of starvation (4000 ×).

EXPERIMENTAL RESULTS

Signs of reparative processes, in the form of ectopia of the nucleolus and nucleus, and an increase in the number of invaginations of the nuclear membrane to 6-7 compared with 2-3 in intact animals, with the folds also becoming deeper (Fig. 1), were observed in LHR neurons of the rat brain 3 days after the beginning of food deprivation. Proliferation of the vesicular component of the endoplasmic reticulum, hyperplasia of the mitochondria and lysosomes, and hypertropy of individual mitochondria were observed in the cytoplasm. It must be emphasized that reparative structural changes were clearly defined in certain neurons of LHR, especially those of a large size, and they were observed more frequently in its posterior part.

During this period of food deprivation glial cells, together with medium-sized and small neurons, formed assemblages around large nerve cells in the anterior and posterior part of LHR. The number of cells involved in these assemblages varied from three to five, but in some cases they were larger. Often two neighboring neurons or a nerve cell and a glial cell were in close apposition in these assemblages. At sites where cells were in contact, local destruction of the cytoplasmic membranes was observed with the cytoplasm of one cell communicating with the cytoplasm of the other. Under these circumstances activation of the nucleus and signs of intracellular hyperplasia of the cytoplasmic organnelles were clearly visible in the large neurons (Fig. 2).

The number of "paired" neurons in LHR (especially in its posterior part) was increased 5 days after the beginning of food deprivation of the animals, but on the whole, destructive intracellular changes predominated over regenerative. After 7 days of food deprivation dystrophic changes in the neurons became massive in character, and single nerve cells died. At the same time, cells without visible ultrastructural changes could be identified, as well as neurons (most frequently in the middle part of LHR) with manifestations of reparative changes, expressed as hypertrophy of the nucleolus, reduction and redistribution of the nuclear chrom-

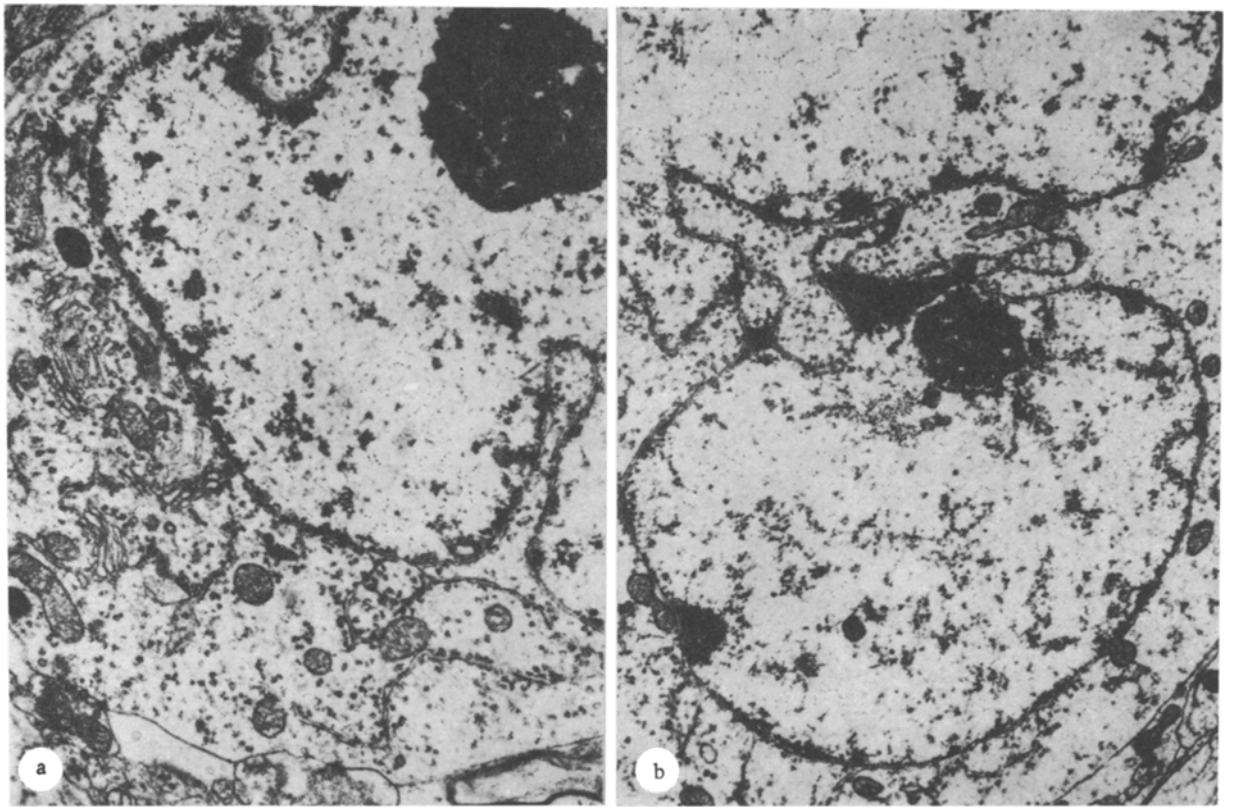

Fig. 3. Reparative changes in large neurons of LHR in rats after 7 days of food deprivation. *a*) Hypertrophy of nucleolus, redistribution of nuclear chromatin, hypertrophy and hyperplasia of individual cytoplasmic organelles in neuron in middle part of LHR; b) sharp increase in nucleo-cytoplasmic ratios in a neuron in posterior part of LHR (9000 ×).

atin, an increase in the number of cytoplasmic organelles, hypertrophy of individual mitochondria, and hyperplasia of the components of the Golgi complex (Fig. 3a). In some cells invaginations of the nuclear membrane were so deep that they gave the impression that the cells were binuclear (Fig. 3b).

During food deprivation, besides dystrophic changes in the neurons, intracellular regenerative changes also are observed, and are evidently aimed at abolishing the pathological changes arising during food deprivation; these observations confirm the view that parallel development of destructive changes in cells and the repair processes which abolish these changes takes place.

It must be pointed out that the intensity of repair processes differed at different stages of starvation in neurons of the anterior, middle, and posterior parts of LHR. In the early stages of the experiment (until 3 days) signs of reparative structural changes were most marked in cells of the posterior part of LHR, but in the late stages (after 7 days) they were most marked in neurons in the middle part; this fact demonstrates the heterogeneity of reparative structural changes in neurons of different parts of LHR in response to low blood nutrient levels in the experimental animals.

The appearance of "paired" neurons in the anterior and, in particular, in the posterior part of LHR 3 days after the beginning of food deprivation, and the number of which increased as food deprivation continued, is of definite interest. This union of neurons is evidently a unique mechanism of compensatory change in response to the deficiency of nutrients, required to maintain the activity of the damaged cells. The absence of a cytolemma at the point of contact between two cells presupposes a more active exchange of biological substances between the two neurons. Similar unions of nerve cells have been observed in hypoxia and alcoholic intoxication, and they constitute a visible manifestation of a method of maintaining function of damaged neurons [3, 6].

In the course of starvation, despite the presence of signs of reparative changes in neuronal ultrastructure in LHR (especially in its posterior part) the intensity of the dystrophic processes increases, leading to death of some of the cells, which reflects collapse of compensatory mechanism.

LITERATURE CITED

1. B. M. Abushov and L. B. Verbitskaya, Arkh. Anat., No. 1, 40 (1984).
2. B. M. Abushov and L. B. Verbitskaya, in: Current Problems in Epidemiology, Clinical Aspects, Treatment, and Prevention of Tropical Diseases [in Russian], Moscow (1984), p. 62.
3. N. N. Bogolepov, N. I. Pavlovskaya, and N. I. Yakovleva, Arkh. Anat., No. 9, 15 (1980).
4. G. N. Krivitskaya, V. B. Gel'fand, and É. N. Popova, Destructive and Reparative Processes in Local Brain Lesions [in Russian], Moscow (1980).
5. S. K. Lapin, in: Compensatory and Adaptive Processes in the Central Nervous System [in Russian], Irkutsk (1977), p. 72.
6. É. N. Popova, Byull. Éksp. Biol. Med., No. 12, 743 (1984).
7. A. S. Pushkin and S. K. Lapin, in: Problems in Experimental and Clinical Pathology [in Russian], Erevan (1980), p. 95.