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ELECTRON MICROSCOPIC OBSERVATIONS ON EXPERIMENTAL CEREBRAL ISCHEMIA IN IMMATURE AND ADULT RATS

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Glycogen accumulation in the C.N.S. is one of the earliest and most sensitive symptoms of anoxic and ischemic impairment of nervous tissue (Mossakowski et al.: J. Neuropath. Exp. Neurol. 27, 500, 1968; Mossakowski and Zelman: Postępy Astronautyki, Suppl. 1, 37, 1971). Partial brain ischemia produced by bilateral ligation of the common carotid arteries causes significant glycogen deposition with rarely occurring morphological changes.

The aim of the present work was to localize the glycogen deposits at the ultrastructural level in immature and adult brains, and to describe the remaining ultrastructural changes produced by bilateral ligation of the carotid arteries.

MATERIAL AND METHODS

Eighteen rats, 1, 4, 6, and 10 weeks old, were used. Forty-eight hours after bilateral ligation of the carotid arteries, perfusion was carried out through the left heart ventricle with 4% solution of glutaraldehyde. Samples of cortex and white matter were taken from the parietal brain region.

RESULTS AND DISCUSSION

In 1-week-old rats, the nervous tissue was characterized either by no pathological changes at all, or by extensive necrotic lesions with complete absence of glycogen grains. In the cortex of 4, 6, and 10-week-old rats, significant deposits of glycogen were noticed in astroglia (Fig. 3, × 7000), especially in the perivascular processes. In 4, and occasionally in 6-week-old rats, weak glycogen deposition was observed in small neuronal pro-

cesses and synapses, and sometimes in larger dendrites. No changes were noted in perikaryons of nerve cells. In the white matter of 4, 6, and 10-week-old rats, an increased number of glycogen grains was also visible, mainly in astrocytic processes around the vessels. In single cases in the 4-week-old rats, accumulation of glycogen was also noted in the perikaryon and processes of oligodendroglia.

In one case, in a 4-week-old rat, a small spongiose focus was observed in the cortex. The following cases were distinguished in the electron microscope picture: 1) localized between the normal or degenerated axon and the unbroken myelin sheet (Fig. 1, × 25,000), 2) surrounded by myelin sheet only, 3) bounded by neuronal and glial processes, probably also of neuronal origin, 4) bounded by a single membrane, located within the neuronal processes. In the spongiotic focus, in not fully disintegrated neuronal processes, rich proliferation of filaments and numerous sheet-like structures essentialy identical with those originally described by Hirono (J. Neuropat. Exp. Neurol., 27, 167, 1968) were observed (Fig. 2, × 30,000), as well as pathological pictures characterized by accumulation of mitochondria and occurrence of numerous dense bodies (Fig. 2), and also changes characterized by the presence of a large number of small vesicles surrounded by a single or double membrane.

In the presented case, a necrotic focus was observed in samples cut through both hemispheres, contiguously to the area from which the material for EM study was taken, the samples being stained with hematoxylin-eosin. The necrotic focus was located in the same hemisphere somewhat below the cortical area which served as a source of material for ultrastructural studies. The described changes in the ultrastructure of the cortex probably resulted from the impairment in the course of the experiment of neuronal fibers crossing the necrotic focus in the white matter (secondary degeneration).

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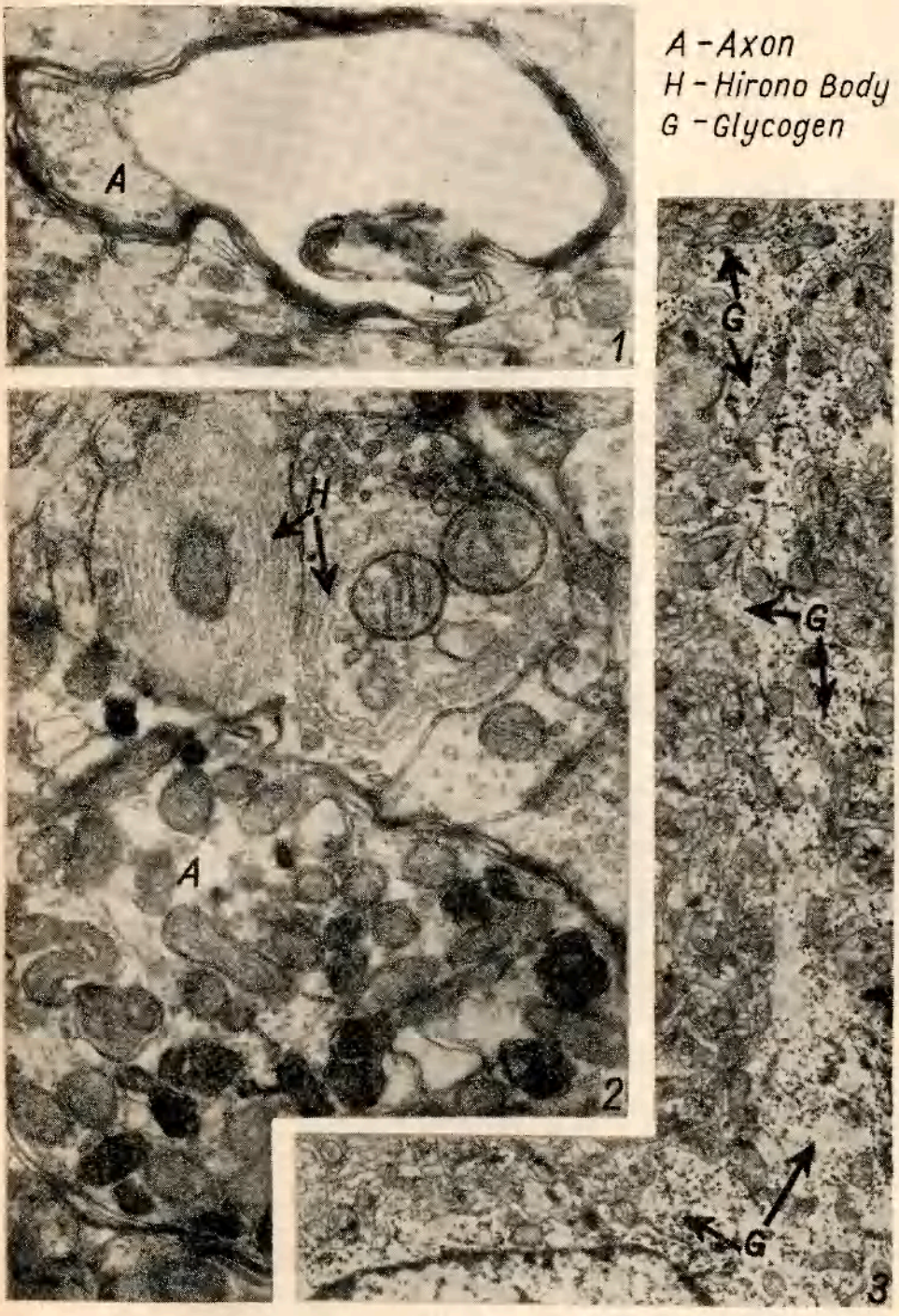

Fig. 1—3