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EFFECT OF SHORT-LASTING ANOXIA ON *IN VITRO* CULTURE OF CEREBELLUM

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Morphological evaluation of the lesions of various structural elements of the nerve tissue caused by oxygen deprivation in *in vivo* conditions is difficult, owing to overlapping influences of various systemic and local factors, which accompany anoxia or result from it. The tissue culture conditions, despite their obvious limitations, resulting from methodological problems create a convenient experimental model for studying the effects of „pure" anoxia.

In our previous studies (Kraśnicka et al., 1974) the influence of short-lasting, transient anoxia on the cellular elements of the peripheral nervous system were investigated in detail. It seemed interesting to compare those results with observations concerning the effects of the same type of anoxia on cellular elements originating from the tissues of the central nervous system.

MATERIAL AND METHODS

Experiments were carried out on *in vitro* cultures of nerve tissue from cerebellum of newborn rats (Kraśnicka, Mossakowski, 1965).

Selected cultures 2, 3 and 4 week-old were kept for a period of 30 min in an atmosphere of 100 per cent nitrogen. The detailed description of nitrogen administration was given in a previous paper of Kraśnicka et al. (1974). Following anoxia cultures were kept in standard conditions for 1, 3 and 5 days and then elaborated technically for both light- and electron-microscopic examination.

For light-microscopic examination histological (Nissl and Bodian) and histochemical (Sudan black B, PAS and PAS-dimedon) techniques were used. In addition histoenzymatic reactions, revealing activities of UDPG--glycogen transferase, glycogen-phophorylase a, lactic-, succinate-, and glucose-6-phosphate dehydrogenases were performed.

Cultures for electron-microscopic examination were prepared according to the technique described by Borowicz and Kraśnicka (1971). Ultrathin sections counterstained with uranyl acetate and lead citrate were examined in a JEM 7A electron microscope. Pictures were taken on ORWO EU 2 plates at an accelerating voltage of 80 KV.

Control material consisted of cultures of the same age as the experimental ones, kept in standard conditions for the whole period of their life.

RESULTS

Light microscopic studies

Two-week-old cultures

Obvious morphological abnormalities were noted as early as 24 hours following anoxia. They differed in their intensity depending on the type of cells and their location in the culture. Among the neuronal population the most advanced changes concerned Purkinje cells (Fig. 1). Their cytoplasm in Nissl staining was swollen and very often contained a number of small vacuoles. Tigrolysis was a very common feature in a great number of Purkinje cells. On the contrary, the granular neurons were much better preserved and the majority contained distinct tigroid granules distributed concentrically under the cell membrane. In Bodian's impregnation an abundant dense network of nerve fibres was visible (Fig. 2). No abnormalities in their morphological picture were noted. In some of the Purkinje cells intracytoplasmic neurofibrils, normal in their appearance were present. Sudan black B staining revealed significant damage to myelin sheaths. Granules of sudanophilic material varying in size and shape as well as larger „fyelin halls" were irregularly distributed along the nerve fibres (Fig. 3).

The glial cells within the explanted tissue showed moderate enlargement of their cytoplasm, its swelling and pallor. In some glial cells small intracytoplasmic vacuoles were present. The most intensive changes concerned large glial cells situated in close vicinity of the Purkinje cells: the seemed to correspond to Pergman's glia. The morphological abnormalities in the glial population from the outgrowth zone were much more advanced, although their morphological pattern was essentially the same as in the glia localized within explanted tissue. The pathological

changes consisted in considerable swelling and pallor of cellular cytoplasm, its vacuolar degeneration and generalized fragmentation of cell processes. The glial nuclei were also enlarged and stained poorly with thionine. The above presented morphological abnormalities concerning both neuronal and glial populations, as well as myelin sheaths persisted during the whole observation period, that is till the 5th day following anoxia.

Throughout the entire period of observation the content of PAS-positive granular substances in all cellular elements of the cultures was greatly increased as compared with normal conditions. This being most pronounced in large astrocytes from the outgrowth zone. Granular glycogen deposits in neurons, mostly Purkinje cells, were present already 24 hours after the anoxic episode. On the 3rd and 5th days following anoxia the amount of glycogen granules in neurons decrease progressively. On the contrary, in glial cells intensive glycogen deposition was present during the whole observation period, although the polysaccharide material was accumulated only in some glial cells, mostly astrocytes, of both explanted tissue and the outgrowth zone.

The activity of glycogen-metabolizing enzymes was significantly higher in cultures submitted to anoxia than in the control ones. High activity of both glycogen phosphorylase a and UDPG-glycogen transferase persisted from the first till the last day of observation. Neurons manifested higher activity than oligodendrocytes and astrocytes situated within the explanted tissue. Glial cells in the outgrowth zone exhibited lower activity of both enzymes than the cellular population of the explant, however, this activity was much more intensive than in the corresponding cells in normal control cultures.

The nerve cells from the experimental cultures exhibited relatively high activity of all dehydrogenases under study, except succinic dehydrogenase, the activity of which was slightly reduced, mostly in Purkinje cells (Fig. 4) The activity of all oxidative-reducing enzymes in glial cells from the explanted tissue was lower than in neurons, however, it was better preserved than in astrocytes and oligodendrocytes present within the outgrowth zone.

Three- and four-week-old cultures

Older cultures showed less intensive morphological changes as compared with 2 week-old ones. The appearance of nerve cells in cultures stained by Nissl's and Bodian's methods did not essentially differ from the normal picture. Only in some Purkinje cells there was a slight decrease of Nissl's substance content at the 24th hour following anoxia. In

the 3rd and 5th day after anoxia even these changes became less evident. However, in cultures stained with Sudan black B severe myelin damage of various degree was observed; this involved almost all myelinated nerve fibres, except a few fibres with preserved normal myelin sheaths. Nerve cells still contained great amounts of PAS-positive substances. Glycogen accumulation in the nerve cell cytoplasm was less than in 2 week-old cultures, but significantly greater as compared with normal control cultures of appropriate age (Fig. 5). So was the activity of glycogen-metabolizing enzymes. The activity of oxidative-reducing enzymes seemed to be comparable with that observed in neurons from the control cultures (Fig. 6). Only succinic dehydrogenase activity was slightly weaker.

Oligodendrocytes and astrocytes situated within the explanted tissue also revealed less advanced morphological changes as compared with those from 2-week-old cultures. Only some large astrocytes from the direct vicinity of Purkinje cells were swollen, whereas the remaining glial population of the explant did not differ significantly from that from the control cultures. On the other hand, in the cultures stained with Sudan black B a great proportion of glial cells, mostly oligodendrocytes, contained in their cytoplasm numerous small sudanophilic granules. PAS-positive substances filled the cytoplasm of a great number of glial cells, while glycogen granules were present in perikarya and processes of only some astrocytes and oligodendrocytes. Glycogen metabolizing enzymes activity was higher than in normal glial population from the control cultures. The activity of oxidative-reducing enzymes was lower than in nerve cells, and only slightly reduced as compared with that of normal glial cells in tissue cultures.

The morphological and histochemical abnormalities of the same type involved also the glial population within the outgrowth zone, but here they were more advanced than within the explanted tissue. Generally, the pathological changes were more pronounced in cultures examined on the first day following anoxia than in later stages of the postanoxic period, with the exception of glycogen accumulation which increased in later phases of observation.

Electron-microscopic studies

Two-week-old cultures

On the first day following anoxia the pathological changes were found in all cellular elements of the cultures. In Purkinje cells, characterized by large nuclei with typical deep invaginations (Fig. 7), the amount of rough endoplasmic reticulum was distinctly reduced as compared with

their normal electron-microscopic picture. The canals of rough endoplasmic reticulum did not form any more the characteristic parallel systems. RER consisted of short fragments covered with scanty ribosomes. The number of polyribosomes was greatly reduced. Golgi apparatus was swollen and its canals and cisterns were usually distinctly dilated. Some of numerous round, oval or elongated mitochondria were swollen and contained pale matrix and shortened cristae. In peripheral areas of Purkinje cells cytoplasm numerous, irregularly distributed neurofilaments and neurotubules were present. In the cytoplasm of some Purkinje cells glycogen particles were seen.

The neurons of the cerebellar granular layer (Fig. 8) showed much less advanced pathological changes than the Purkinje cells. The rough endoplasmic reticulum of granular cells was very well developed, forming parallel systems typical for nerve cells. Slight abnormalities in the ultrastructure of the Golgi apparatus were observed, they consisted in widening of some of its canals and cisterns. Numerous mitochondria were characterized by dark matrix.

Some myelinated axons were swollen and contained scanty glycogen particles. Significant pathological changes were noted in the structure of all myelin sheaths present within the cultures (Fig. 8). Myelin laminae were split from each other at various depths of the myelin sheaths. In some places, on the contrary, they coalesced. In many fibres the laminar structure of the myelin sheaths become entirely obliterated.

Significant abnormalities were seen in the ultrastructure of the perikarya and processes of astrocytes (Figs 7, 8, 9). The pathological changes consisted of swelling of the cellular cytoplasm, decrease of rough endoplasmic reticulum, reduction of mitochondria and widening of the canals and cisterns of the Golgi apparatus. All these changes led to a watery appearance of the astrocytic cytoplasm. Almost all astrocytes showed a distinctly increased number of gliofibrils and various amounts of glycogen rosettes as well in the perikarya as in the processes (Fig 7, 9).

The most prominent changes of oligodendrocytes consisted in the presence of numerous lysosome-like structures and other structures of various size and electron density (Fig. 9) in their cytoplasm. Remarkable dilatation of Golgi apparatus canals and cisterns was a very common feature, whereas mitochondria and other subcellular organelles seemed to be unchanged. Noteworthy was the great amount of glycogen rosettes in the cytoplasm of oligodendrocytes (Fig. 10).

Three and five days following anoxia pathological changes similar to those described in the 24th hour of the postanoxic period were noted. On the 5th day after anoxia rough endoplasmic reticulum in Purkinje cells was less changed.

Three- and four-week-old cultures

In all cellular elements from older cultures the ultrastructural abnormalities were less intensive as compared with those described in 2-week-old cultures. Granular cells did not show any distinct abnormalities. Rough endoplasmic reticulum and mitochondria were well preserved and not damaged. Only in some Purkinje cells dilatation of canals and cisterns of the Golgi apparatus was noted (Figs 11, 12), with slight reduction of rough endoplasmic reticulum and free polyribosomes. Numerous myelinated axons were swollen, and characterized by watery appearance, reduction of subcellular organelles and slight swelling of their mitochondria (Fig. 12). Other axons apparently not damaged contained various amounts of glycogen particles (Fig. 13). Myelin sheaths showed

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Fig. 1. Two-week-old culture, 3 days after anoxia. Purkinje cell with complete tigrolysis. Granular cells well preserved. Swollen glia around Purkinje cell. Toluidine blue. × 200.

Ryc. 1. Hodowla 2-tygodniowa, 3 dni po niedotlenieniu. Komórka Purkinjego z całkowitą tigrolizą. Komórki ziarniste dobrze zachowane. Wokół komórki Purkinjego obrzmiały glej. Błękit toluidyny. Pow. 200 ×.

Fig. 2. Three-week-old culture, 3 days after anoxia. Nerve cells with well stained processes. Bodian's meth. × 400.

Ryc. 2. Hodowla 3-tygodniowa, 3 dni po niedotlenieniu. Komórki nerwowe z wypustkami dobrze wyimpregnowanymi. Met. Bodiana. Pow. 400 ×.

Fig. 3. Four-week-old culture, 3 days after anoxia. Prominent damage of myelin sheaths. Sudan black B. × 400.

Ryc. 3. Hodowla 4-tygodniowa, 3 dni po niedotlenieniu. Znaczne uszkodzenie osłonek mielinowych. Sudan czarny B. Pow. 400 ×.

Fig. 4. Two-week-old culture, 3 days after anoxia. Succinic dehydrogenase. Low enzyme activity in glial cells, better maintained in neurocytes. × 200.

Ryc. 4. Hodowla 2-tygodniowa, 3 dni po niedotlenieniu. Dehydrogenaza bursztynianowa. Aktywność enzymatyczna mniejsza w komórkach glejowych, lepiej zachowana w neurocytach. Pow. 200 ×.

Fig. 5. Three-week-old culture, 3 days after anoxia. Glycogen transferase. High activity of the enzyme in neurocytes, lower in glia. × 400.

Ryc. 5. Hodowla 3-tygodniowa, 3 dni po niedotlenieniu. Transferaza glikogenowa. Wysoka aktywność enzymatyczna w neurocytach, mniejsza w gleju. Pow. 400 ×.

Fig. 6. Three-week-old culture, 3 days after anoxia. Lactic dehydrogenase. Enzyme activity close to the norm. × 200.

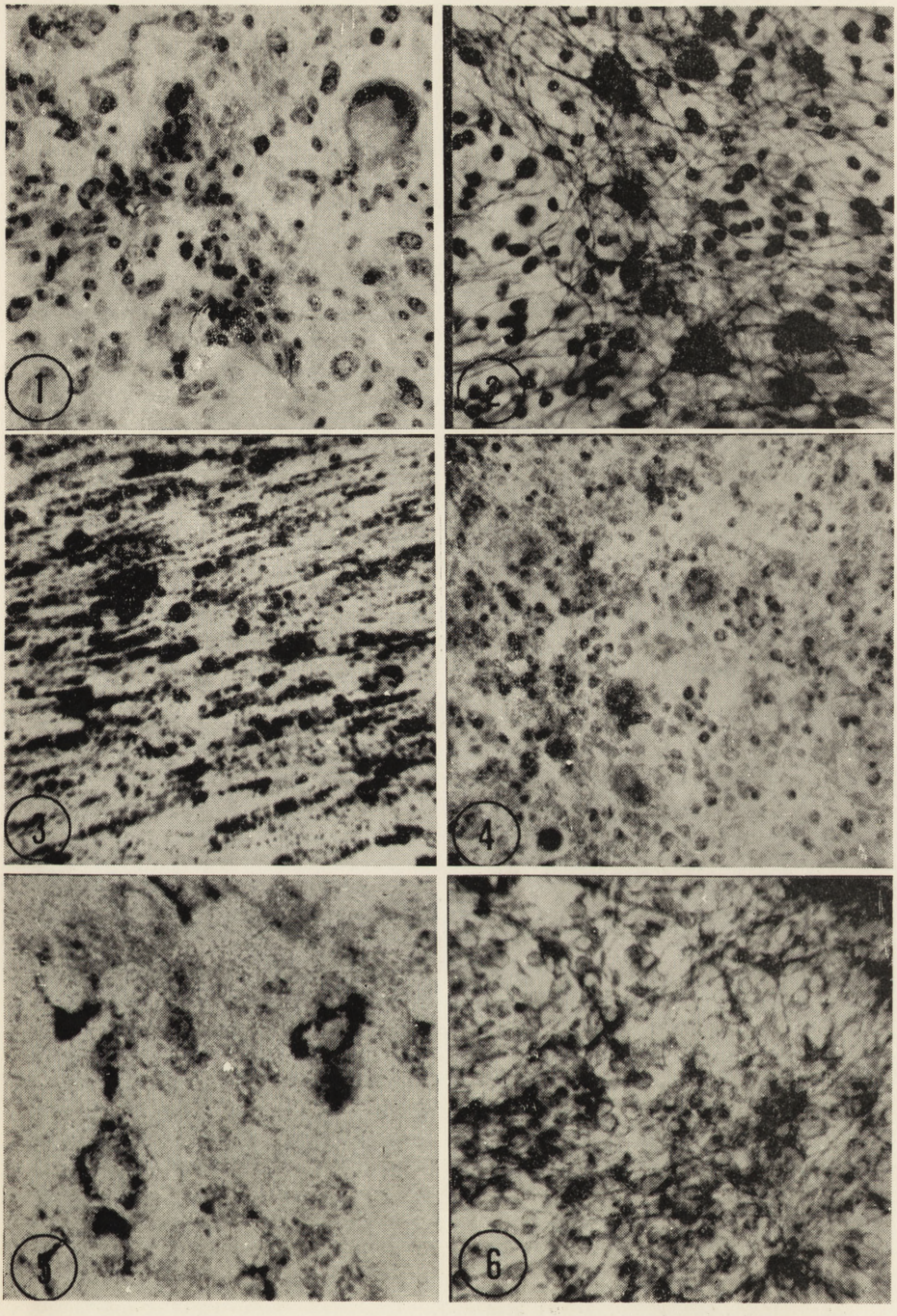
Ryc. 6. Hodowla 3-tygodniowa, 3 dni po niedotlenieniu. Dehydrogenaza mleczanowa. Aktywność zbliżona do normy. Pow. 200 ×.

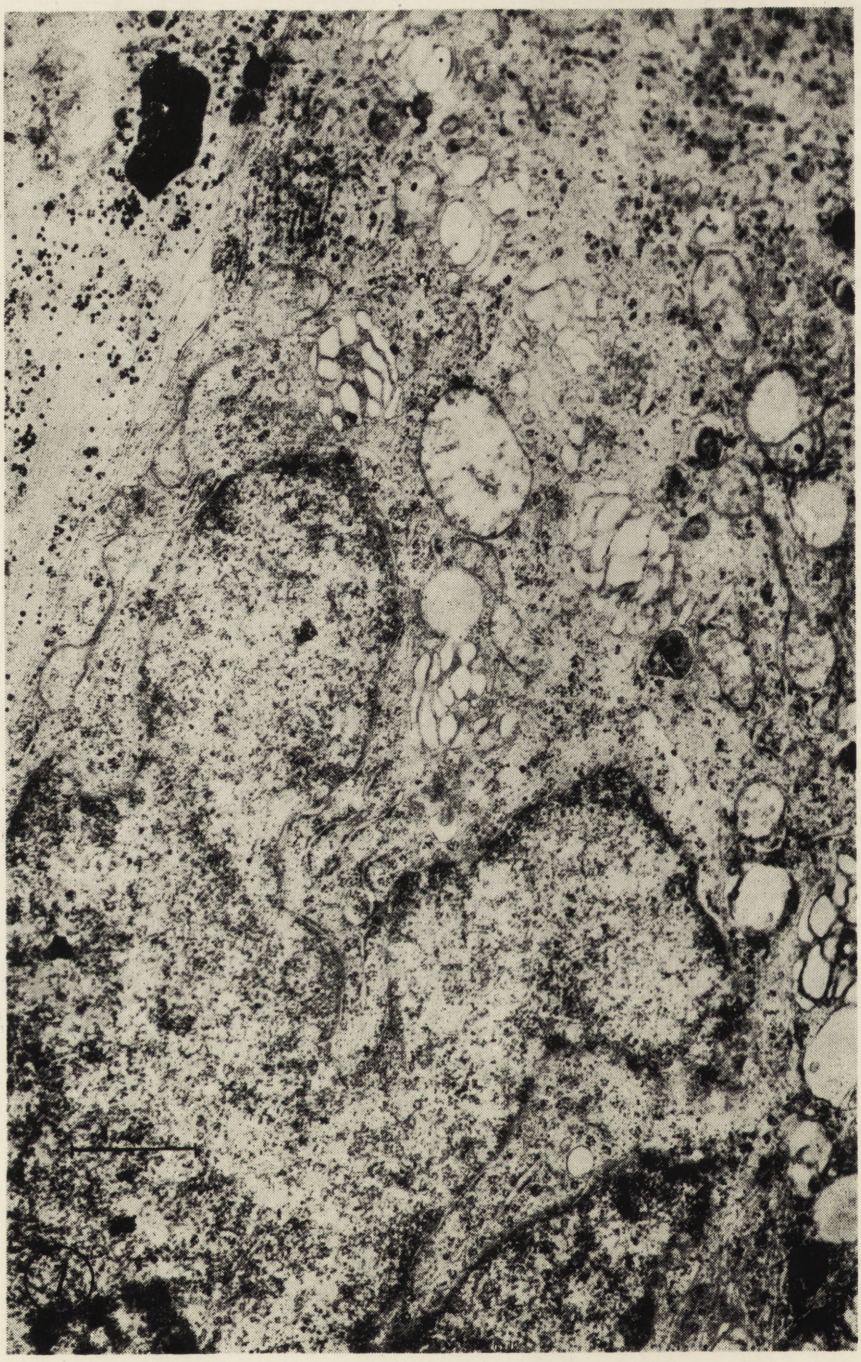
Fig. 7. Two-week-old culture, 3 days after anoxia. Fragment of Purkinje cell. Nucleus with deep invaginations. Well developed Golgi apparatus with dilated canals, some mitochondria are swollen. Rough endoplasmic reticulum fragmented. Fragment of astrocyte with numerous glycogen granules.

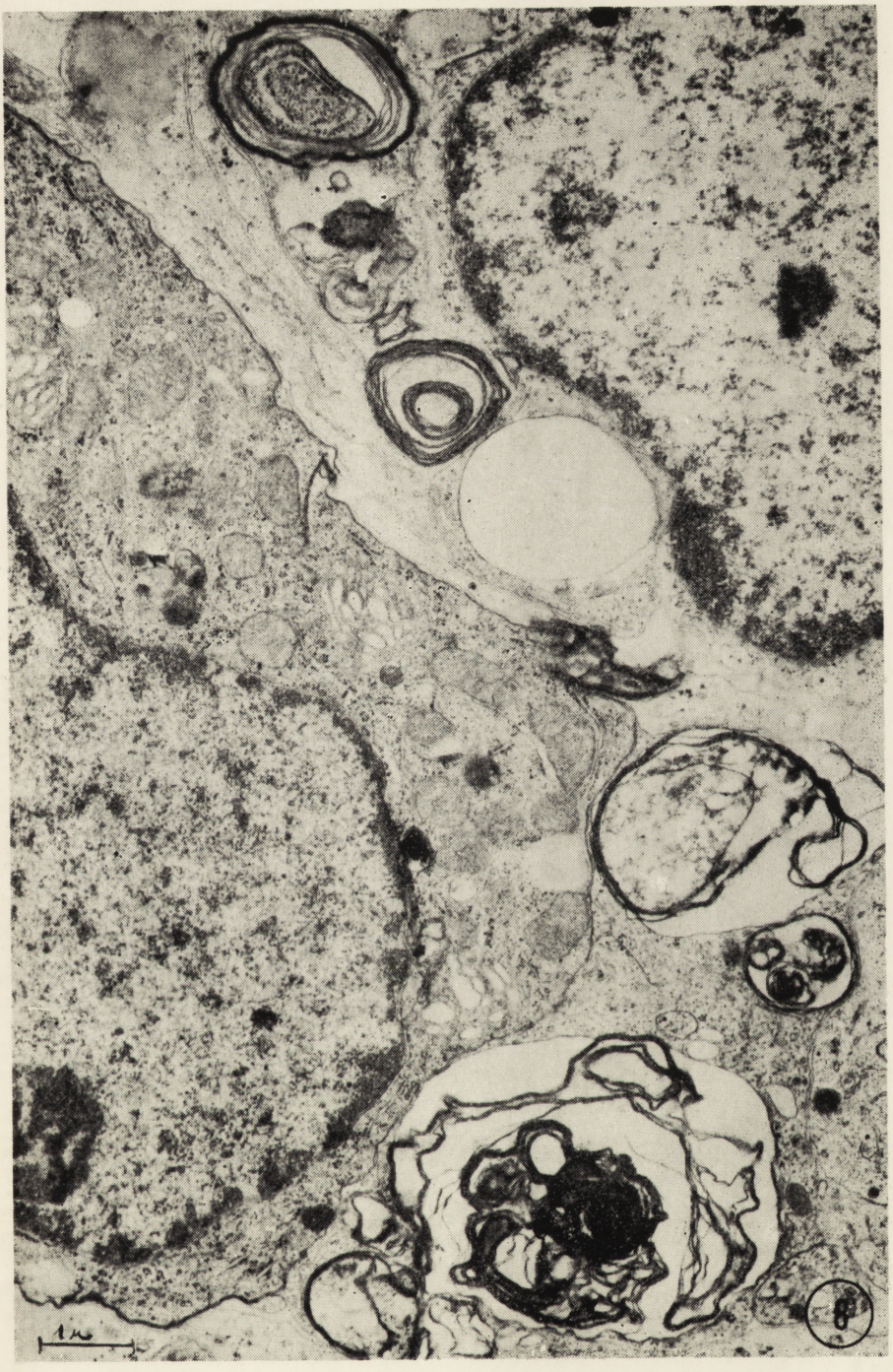
Ryc. 7. Hodowla 2-tygodniowa, 3 dni po niedotlenieniu. Fragment komórki Purkijnego. Jądro z głębokimi inwaginacjami. Rozbudowany aparat Golgiego z poszerzonymi kanałami, niektóre mitochondria obrzmiałe. Siatka śródplazmatyczna szorstka zbudowana z krótkich odcinków. Powyżej fragment astrocyta z licznymi ziarnami glikogenu.

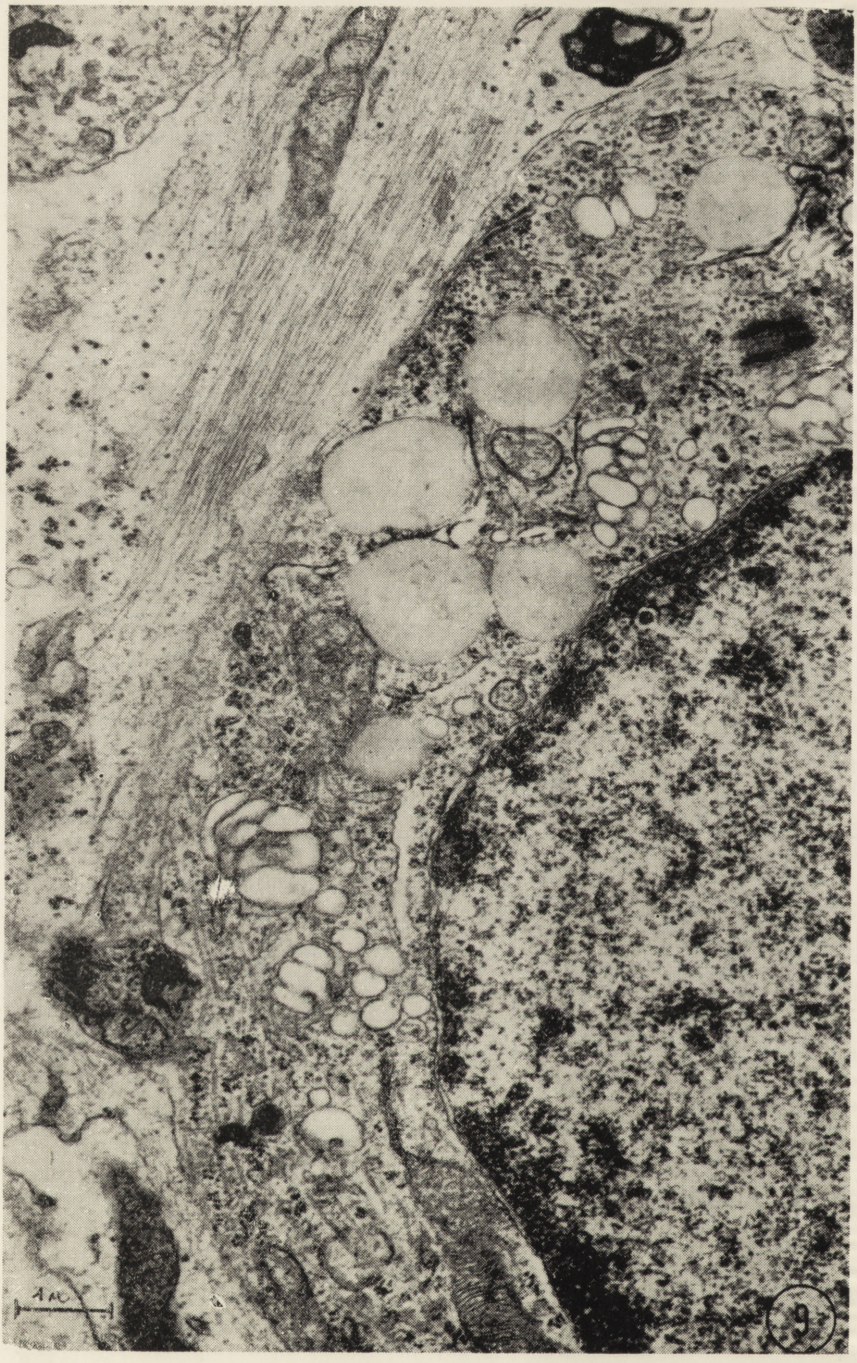
Fig. 8. Two-week-old culture, 1 day after anoxia. Fragment of granular cell with slightly dilated canals of the Golgi apparatus. Fragments of glial cells and numerous myelinated fibers displayning various degree of damage

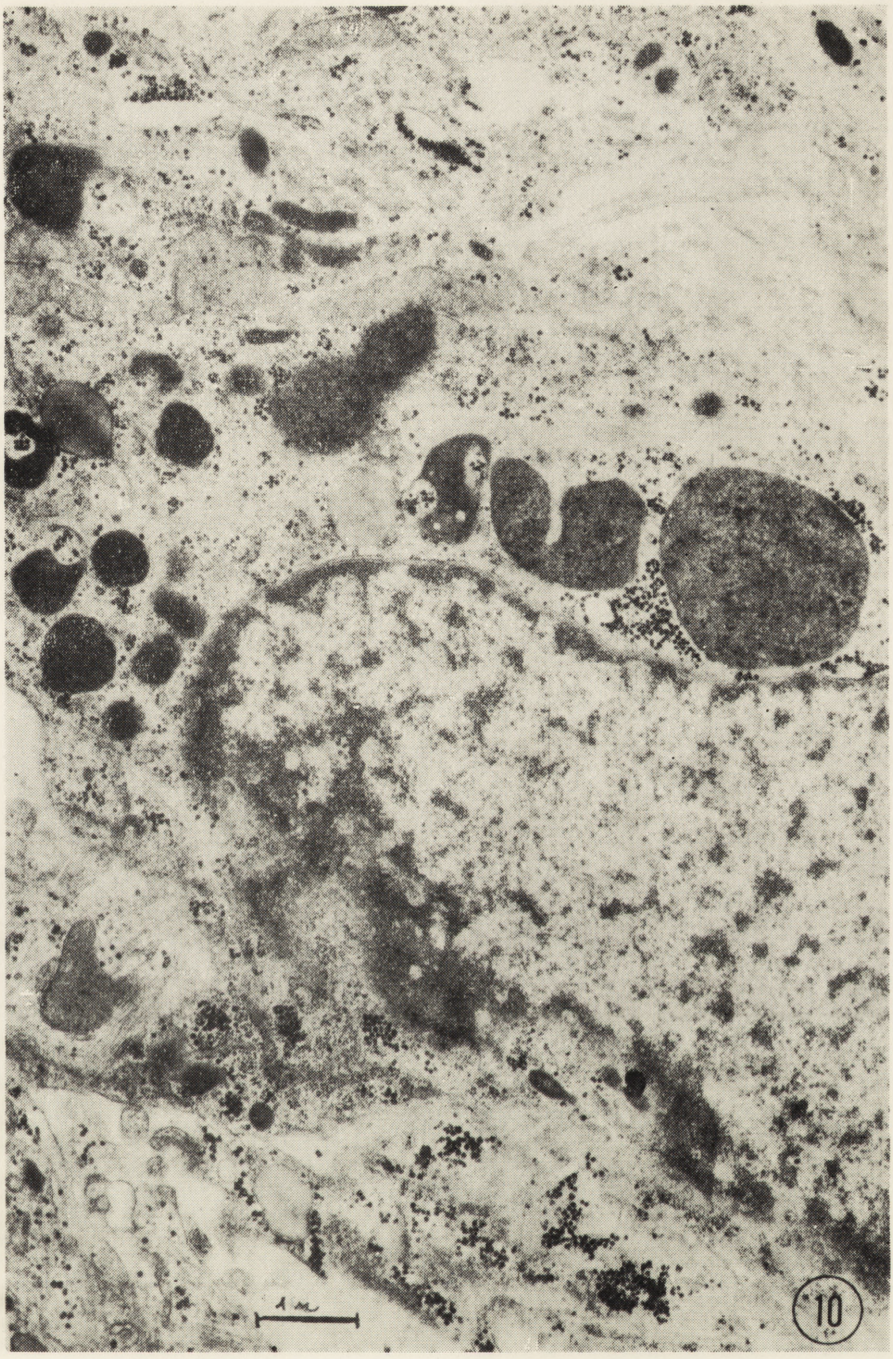
Ryc. 8. Hodowla 2-tygodniowa, 1 dzień po niedotlenieniu. Fragment komórki ziarnistej z nieznacznie poszerzonymi kanałami aparatu Golgiego. Obok fragmenty komórek glejowych i liczne zmielinizowane włókna o różnym stopniu uszkodzenia.

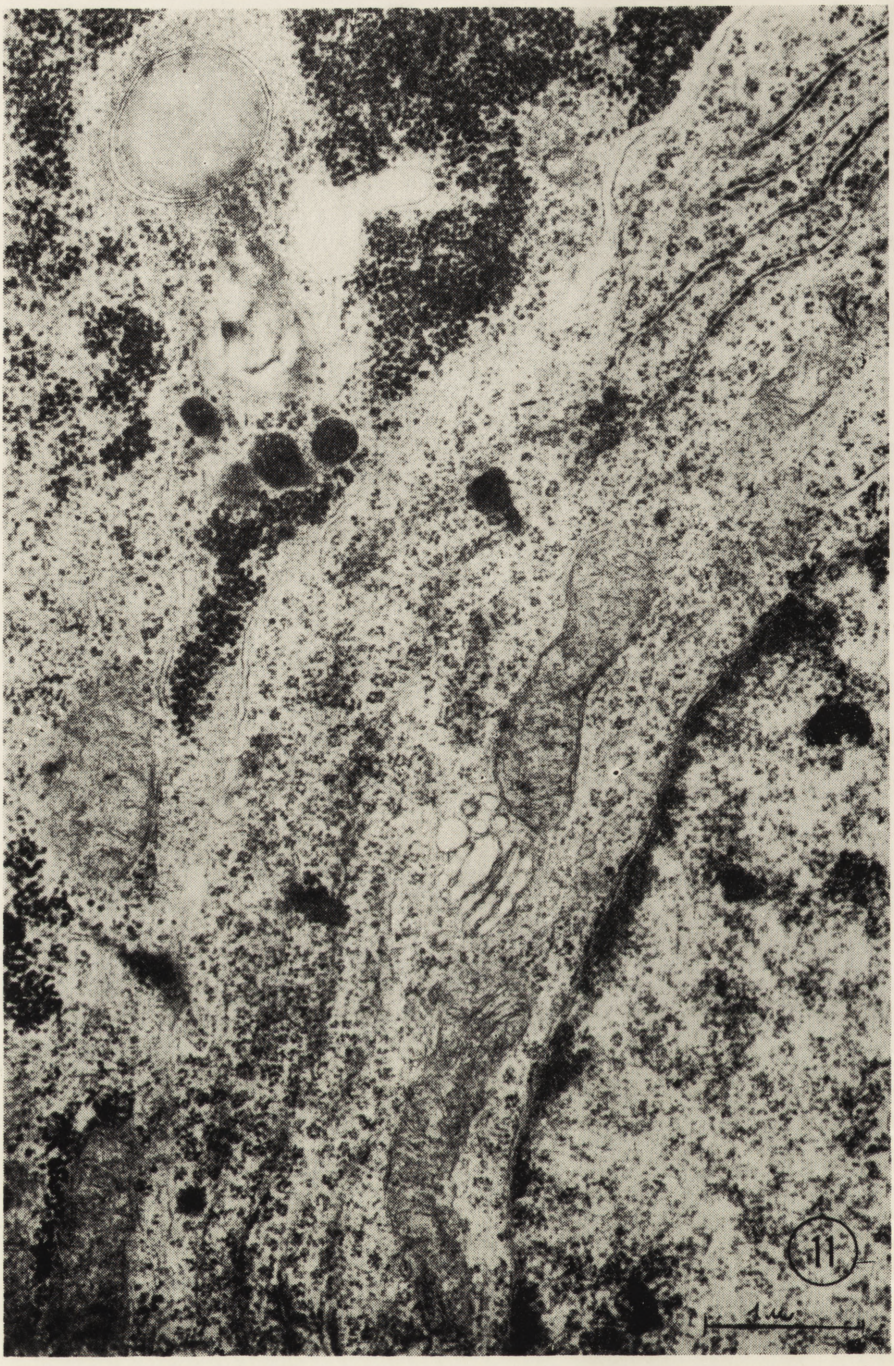


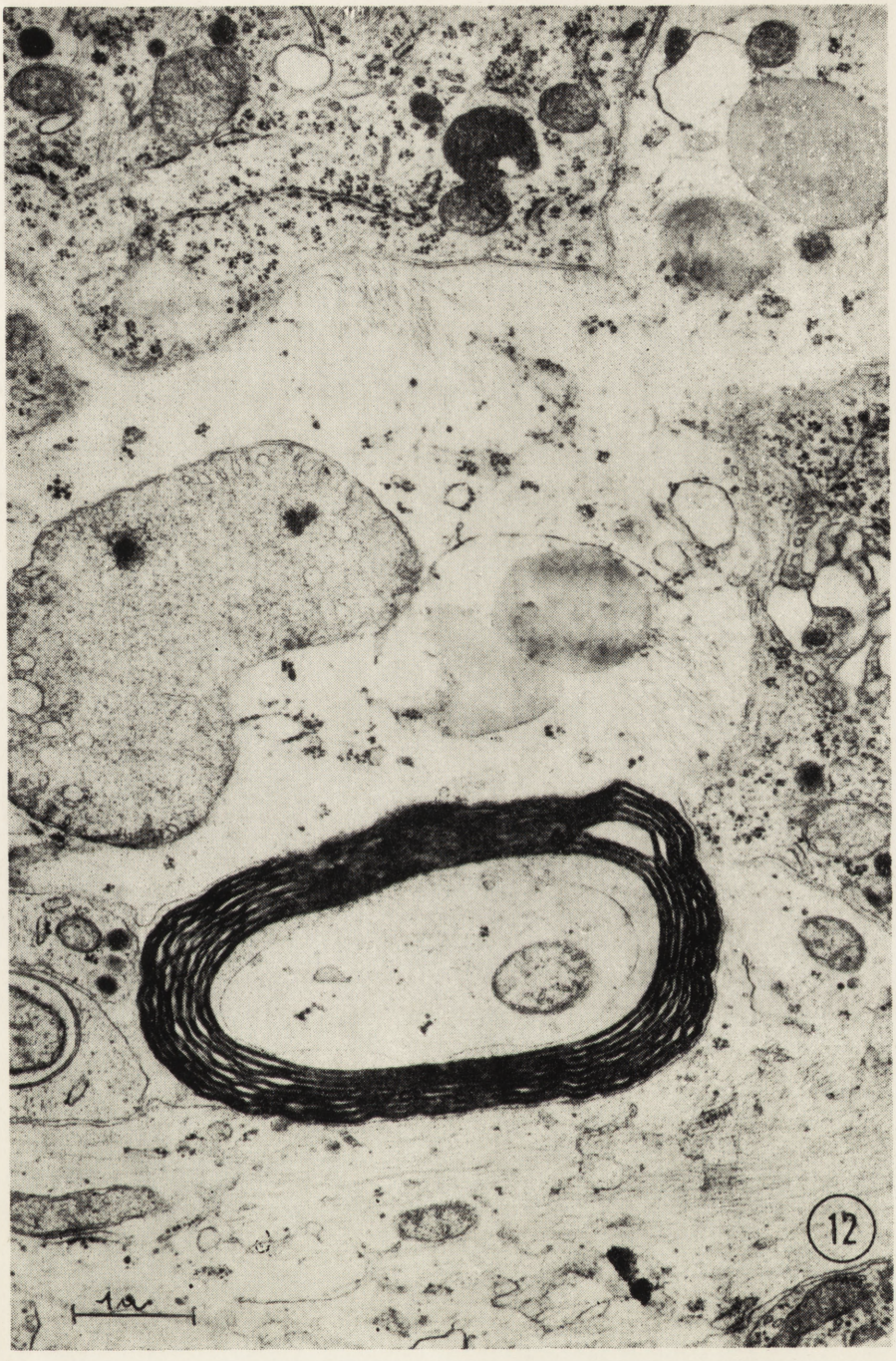


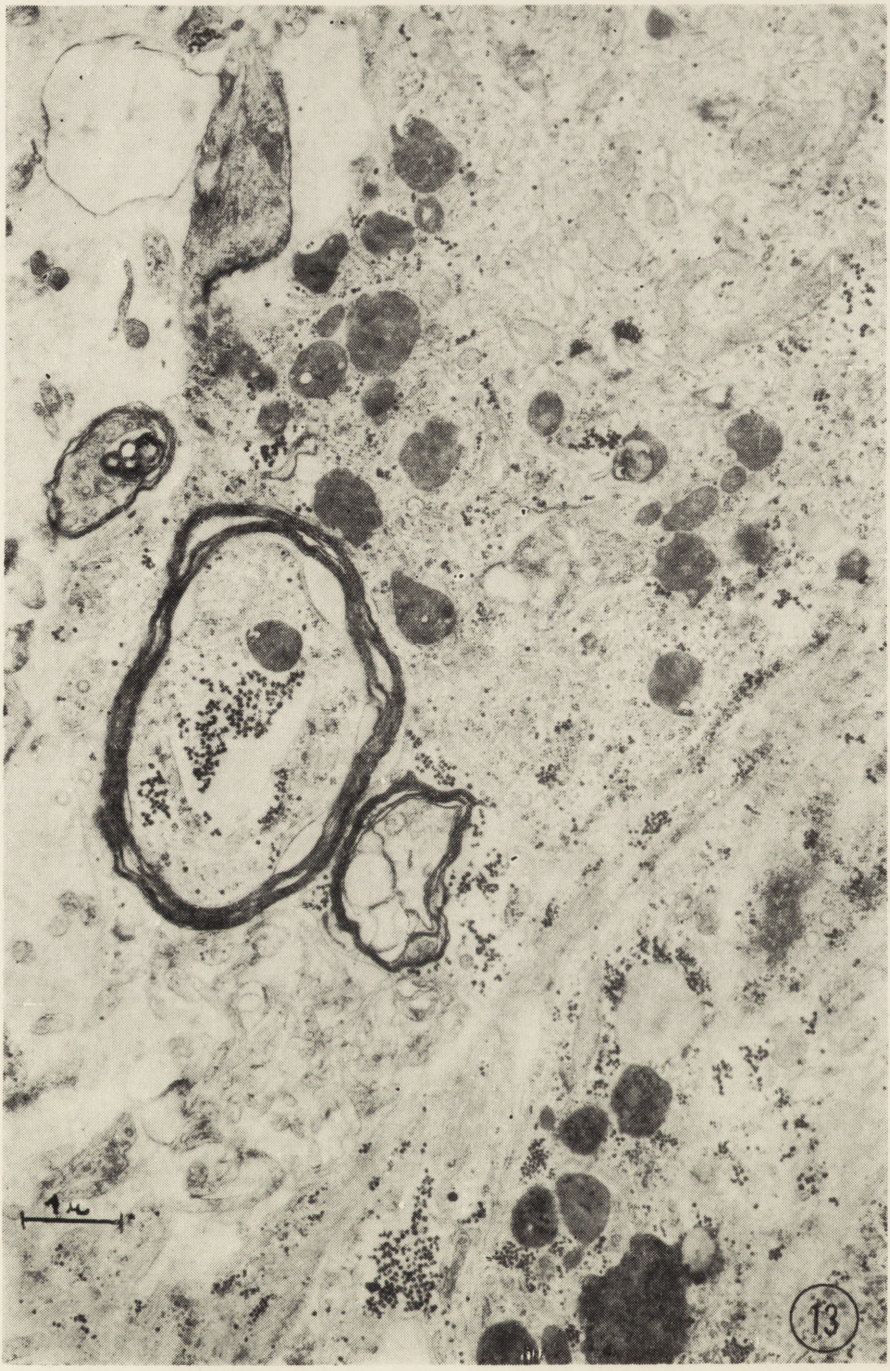












also lesser degree of damage than those in younger cultures (Figs 12, 13). Only a small number of fibres manifested disturbed laminar structure of myelin sheaths, owing to splitting and/or condensation of myelin laminae leading to abnormal shape of the fibres. Usually the myelinated nerve fibres were of normal size and shape, and were coated by myelin sheaths with regular, laminar structure.

Swelling of astrocytic cytoplasm was a very common feature, but its degree changed from cell to cell. The most intensive swelling involved cellular processes (Fig. 12). Astrocytes with less pronounced cytoplasmic swelling contained abundant glycogen granules (Fig. 11).

Oligodendrocytes from 3- and 4-week-old cultures reacted to anoxia in an essentially similar way as those from 2-week-old culture. Accumulation of various structures, characterized by varying electron density and presence of numerous lysosome-like structures in their cytoplasm was the leading ultrastructural abnormality. Various amounts of glyco-

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Fig. 9. Two-week-old culture, 3 days after anoxia. Fragment of astrocyte cytoplasm with numerous gliofibrills and glycogen rosettes. Oligodendrocyte with numerous structures of various electron density and with a dilated canals of the Golgi apparatus.

Ryc. 9. Hodowla 2-tygodniowa, 3 dni po niedotlenieniu. Fragment cytoplazmy astrocyta z dużą ilością gliofibryli i glikogenu, pozostałe organelle nieliczne. Obok oligodendrocyt z dużą ilością tworów o różnej gęstości elektronooptycznej oraz z poszerzonym aparatem Golgiego.

Fig. 10. Two-week-old culture, 3 days after anoxia. Fragment of oligodendrocyte with numerous lysosome-like structures and structures containing lipids. Numerous glycogen particles and gliofibrills are also seen.

Ryc. 10. Hodowla 2-tygodniowa, 3 dni po niedotlenieniu. Fragment komórki oligodendrocyta z licznymi ciałami lizosomopodobnymi i tworami z zawartością lipidową; widoczne również liczne ziarna glikogenu i gliofibryle.

Fig. 11. Three-week-old culture, 3 days after anoxia. Fragment of neurocyte with well developed rough endoplasmic reticulum. Some canals of the Golgi apparatus are slightly dilated. Elongated and normal mitochondria. Fragment of glial cell with great amount of glycogen.

Ryc. 11. Hodowla 3-tygodniowa, 3 dni po niedotlenieniu. Fragment neurocyta z dobrze wykształconą siatką śródplazmatyczną szorstką. Niektóre kanały aparatu Golgiego nieznacznie poszerzone. Mitochondria wydłużone. Obok fragment komórki glejowej z dużą ilością glikogenu.

Fig. 12 Three-week-old culture, 3 days after anoxia. Fragment of swollen astrocytic process and fragments of nerve cells and axon with slightly damaged myelin.

Ryc. 12. Hodowla 3-tygodniowa, 3 dni po niedotlenieniu. Widoczny fragment obrzmiałej wypustki astrocyta, oraz fragmenty komórek nerwowych i akson z nieznacznie uszkodzoną mieliną.

Fig. 13. Four-week-old culture, 3 days after anoxia. Fragments of 2 oligodendrocytes with numerous structures of various electron density. Glycogen rosettes are present in their cytoplasm. Slight degree of myelin damage. Glycogen in cytoplasm in axon.

Ryc. 13. Hodowla 4-tygodniowa, 3 dni po niedotlenieniu. Fragmenty 2 oligodendrocytów z widocznymi tworami o różnej gęstości elektronooptycznej. W ich cytoplazmie obecny również glikogen. Nieznaczny stopień uszkodzenia mieliny. W cytoplazmie aksonu widoczny glikogen.

gen granules, accumulated among unchanged subcellular cytoplasmic organelles were also found (Fig. 13).

The above described changes in various types of cells in 3- and 4-week-old cultures were present in all the periods of observation. Their intensity did not undergo essential changes in the 1st, 3rd and 5th day following anoxia.

DISCUSSION

On the basis of our observation it can be assumed that short-lasting, transient anoxia causes structural and metabolic changes in all cellular elements of the examined portion of the central nervous systems. The character and the intensity of the described abonormalities depend on the age of the culture, type of cells and their location within the cultures.

The greatest intensity of pathological changes in all types of cells in 2-week-old cultures indicates the higher sensitivity of maturing nerve and glial cells to oxygen deficiency, than that of the mature ones. This phenomenon has been already pointed out in our previous studies concerning the reaction of glial cells to anoxia (Kraśnicka, Renkawek, 1972; Kraśnicka et al., 1973). The difference in the intensity of cellular damage between less severely altered glial cells from the explanted tissue and the more deeply impaired glial population from the outgrowth zone, observed in the present studies once more confirms this dependence. In interpretation of differences in the degree of structural and metabolic disturbances due to anoxia between nerve cells from younger and older cultures one additional factor has to be taken into consideration. Kraśnicka (1969) showed that neurons from the spinal root ganglia, when cultured *in vitro*, undergo during the two first weeks axonal degeneration, owing to their damage at the time of starting the cultures. This process finds its morphological expression in central tigrolysis and abundant glycogen accumulation. The same phenomenon may take place also when culturing tissue from the central nervous system. In that case the abnormalities seen in neurons from 2 week-old cultures may result from two superimposed processes — axonal reaction and influence of anoxia, whereas in older cultures they indicate a reaction of neurons to oxygen deficiency alone.

The pathological changes in neurons were generally less severe than in glial cells of both astrocytic and oligodendrocytic lines. The same phenomenon indicating lesser sensitivity of neurons as compared with that of glial cells to oxygen deficiency has been already described by Dolivo et al. (1967) and Rouiller et al. (1971) in their *in vitro* studies, as well as by Hills (1964), Bakay and Lee (1968) and Brown and Brierley (1971, 1973) in their experiments on animals. Essentially the same diffe-

rences in the reactivity of neurons, satellite cells and Schwann's cells were found in our previous studies on the influence of anoxia on root ganglia cultured *in vitro* (Kraśnicka et al., 1974).

The nature of the pathological changes varied to a great extent. depending on the type of cells. Among neuronal population the most prominent abnormalities concerned the Purkinje cells. They consisted in swelling of mitochondria, decrease of rough endoplasmic reticulum, reduction of the amount of free ribosomes and enlargement of canals and cisterns of the Golgi apparatus. The above mentioned ultrastructural abnormalities correspond well with the cellular alterations observed in light microscopy. Their nature was similar to the changes, described by numerous authors (Hager et al., 1960; Hager, 1963; Hills, 1964; Brown, Brierley, 1968, 1973) in various types of experimental anoxia in *in vivo* conditions. They were essentially the same, as neuronal impairments in our previous experiments on the peripheral nervous system (Kraśnicka et al., 1974). On the other hand, the morphological and ultrastructural picture of granular layer neurons does not differ from that in normal conditions, except for the slight dilatation of the Golgi apparatus canals in some of them.

Astrocytes displayed remarkable swelling of cytoplasm, involving mostly cellular processes, and reduction of all subcellular cytoplasmic organelles. Moreover, an increased amount of gliofibrils in the cytoplasm of some of astroglial cells was noticed. The degree of damage to the astrocytes observed in the studied material was less than that described in our previous papers (Kraśnicka, Renkawek, 1972; Kraśnicka et al., 1973).

The significant changes observed in oligodendrocytes consisted in an excessive accumulation in the cytoplasm of lysosome-like structures as well as structures, varying in electron density and containing lipid substances.

The abnormality common for all cellular elements was an extensive glycogen accumulation in the perikarya and processes, accompanied by a significant increase of glycogen—metabolizing enzyme activity. This phenomenon, indicating disturbances in intracellular glucose metabolism was identical with that observed in various types of oxygen insufficiency in experimental animals (Mossakowski et al., 1968; Pronaszko et al., 1972; Long et al., 1972) and in tissue culture conditions (Kraśnicka et al., 1974). Noteworthy is the accumulation of glycogen in those cells which showed less advanced structural impairment.

Comparison of the present observations, concerning cultures from the central nervous system, with the results of our previous studies on the

influence of anoxia on cultures from the peripheral nervous system (Kraśnicka et al., 1974) points out the identity of pathological changes involving oligodendrocytes and Schwann cells. It seems that short-lasting, transient anoxia damages most severely the cells responsible for myelin formation and maintenance. Concomitance of severe degenerative changes in oligodendroglia with prominent myelin sheaths abnormalities is strongly suggestive that in conditions of anoxia myelin damage results from the metabolic and structural alterations of the cells involved in its formation.

CONCLUSIONS

1. Short-lasting, transient anoxia causes metabolic and structural changes in all the cellular elements of the cerebellum cultured *in vitro*. The nature and intensity of pathological changes depend on the degree of tissue maturity and type of cells.
2. The impairment of glial cells is more intensive than that of neurons. The most prominent pathological changes concern oligodendroglial cells; this leading to damage of myelin sheats.
3. The nature of pathological changes due to anoxia tissue culture conditions is essentially similar in tissues from the central and peripheral nervous system.

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WPŁYW KRÓTKOTRWAŁEGO NIEDOTLENIENIA NA TKANKĘ NERWOWĄ HODOWANĄ *IN VITRO* (OBSERWACJE MÓŻDŻKU W MIKROSKOPIE ŚWIETLNYM I ELEKTRONOWYM)

Streszczenie

Przebadano wpływ krótkotrwałej anoksji na elementy komórkowe ośrodkowego układu nerwowego. Badania prowadzono na tkance nerwowej pobieranej z móżdżku noworodków szczurzych i przetrzymywanej w warunkach hodowli tkankowej. Hodowle w wieku 2, 3 i 4 tygodni poddawano działaniu anoksji przez 30 minut. Okres obserwacji po niedotlenieniu wynosił od 1 do 5 dni.

Stwierdzono, że krótkotrwałe, przejściowe niedotlenienie powoduje zmiany morfologiczne w neuronach i komórkach glejowych, przy czym intensywność obserwowanych zmian jest mniejsza w neuronach niż w gleju. Największe zmiany zaobserwowano w oligodendrocytach, występowały one we wszystkich grupach wieku. W hodowlach 2-tygodniowych stwierdzano znaczne i rozległe uszkodzenie mieliny, natomiast w hodowlach starszych nie wszystkie włókna wykazywały ten sam stopień demielinizacji. We wszystkich komórkach móżdżku i we wszystkich badanych grupach stwierdzono nadmierne gromadzenie się glikogenu.

З. Красьницка, Б. Гайковска, М. Я. Моссаковски

ВЛИЯНИЕ КРАТКОВРЕМЕННОЙ ГИПОКСИИ НА КУЛЬТУРУ НЕРВНОЙ ТКАНИ *IN VITRO* (ИЗУЧЕНИЕ МОЗЖЕЧКА В ОПТИЧЕСКОМ И ЭЛЕКТРОННОМ МИКРОСКОПАХ)

Резюме

Было исследовано влияние кратковременной аноксии на клеточные элементы центральной нервной системы. Исследования проводились на нервной ткани, взятой из мозжечка новорожденных крысят и содержащейся в условиях тканевой культуры. Культуры в возрасте 2, 3 и 4 недели подвергались действию 30 минутной аноксии. Период наблюдения после гипоксии составлял от 1 до 5 дней.

Было обнаружено, что кратковременная, преходящая гипоксия вызывает морфологические изменения в нейронах и глиальных клетках. Интенсивность наблюдаемых изменений в нейронах была меньше, чем в глии. Самые большие изменения были обнаружены в олигодендроцитах и выступали во всех группах культуры. В 2 недельных культурах были заметны значительные и обширные повреждения миелина, в то время как в старших культурах не все волокна проявляли ту же степень демиелинизации. Во всех клетках мозжечка всех исследуемых групп было обнаружено чрезмерное скопление гликогена.

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