macromolecules of EM. In fact, amniotic epithelium, which is a derivative of trophoblasts, can synthesize types III, IV, and V of collagen in tissue culture [2]. Both in vitro and in vivo trophoblasts secrete fibronectin, type IV collagen, and laminin [4, 5]. It is not yet clear which of the known populations of trophoblasts [11] are responsible for the production of the various components of EM.

The cells evidently also determine the heterogeneous composition of fibrinoid. In their absence the fibrinoid masses are of predominantly hematogenous origin. On the other hand, the presence of the principal types of collagen in fibrinoid of the CTC even in the early stages of pregnancy raises the question of the existence of a physiological prototype of sclerosis in addition to the typical sclerosis of dying chorionic villi [6].

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MORPHOLOGIC CHANGES IN THE CANINE CENTRAL NERVOUS SYSTEM IN UNILATERAL CAROTID ARTERIAL LESIONS

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UDC 616.133-031.12

KEY WORDS: cerebral vessels; occlusion; morphology; analysis

Occlusive lesions of the main vessels of the brain are currently of great interest to clinicians because the problem of cerebral ischemia is closely linked with lesions of the carotid artery. In addition, surgeons are tackling the problem of preserving brain tissue when pathological changes are present in the vessels themselves and also after their surgical correction [2].

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(Presented by Academician of the Russian Academy of Medical Sciences D. S. Sarkisov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 114, No. 9, pp. 326-329, September, 1992. Original article submitted April 13, 1992.

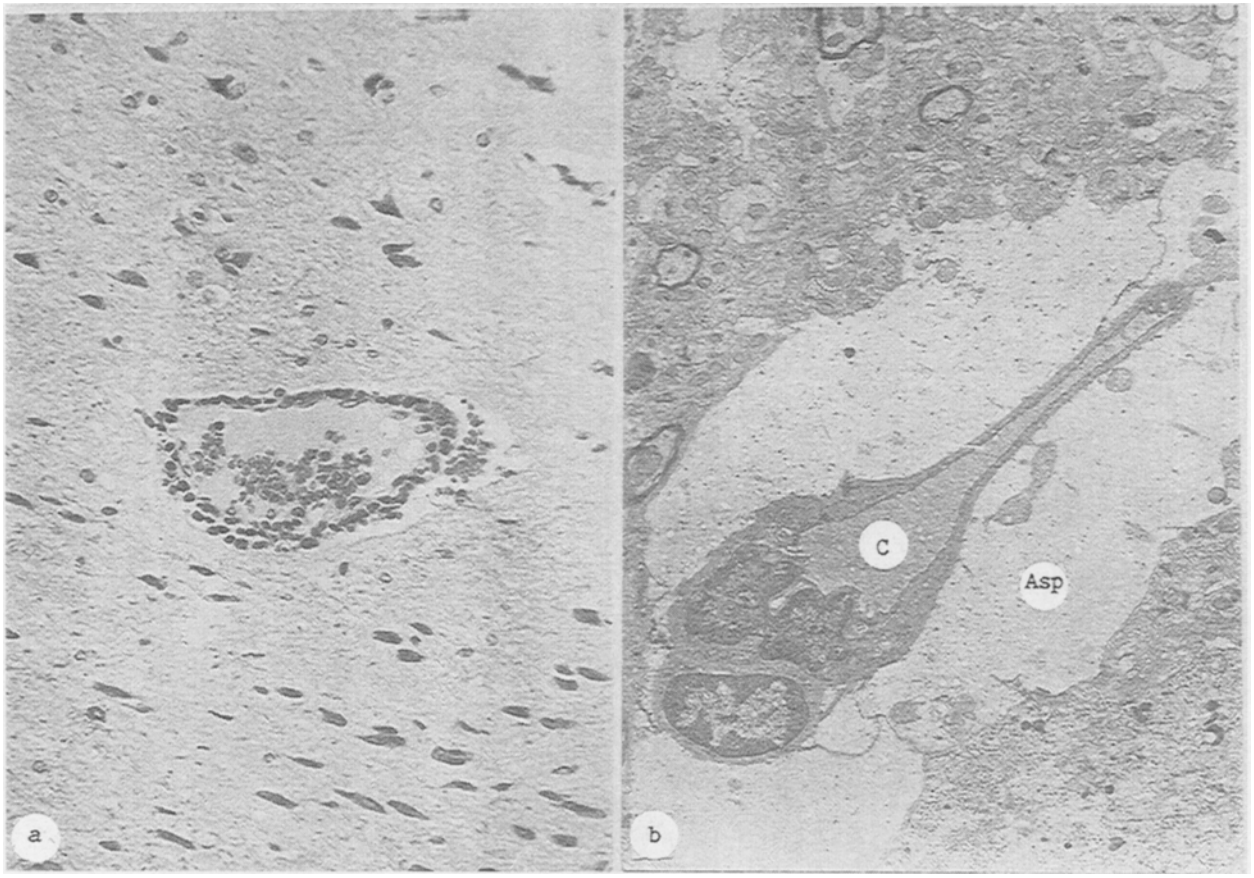

Fig. 1. Changes in microcirculatory bed of canine cerebral cortex 24 h after unilateral ligation of carotid artery: a) pericapillary edema, diapedetic hemorrhage, hematoxylin-eosin, 250×; b) compression of lumen of a capillary (C) by edematous processes of an astrocyte (ASP), 6000×.

The aim of this investigation was a morphologic analysis of changes in the ganglion cells and the microcirculatory bed of the cerebral cortex after unilateral ligation of the common carotid artery and pathological kinking of the artery. Accordingly, an original method of creating pathological kinking of the common carotid artery in experimental animals and a model of unilateral total occlusion (ligation) of the artery have been developed.

EXPERIMENTAL METHOD

Experiments were carried out on 16 mongrel dogs with angiographic control [3]. The material for analysis was the brain, removed 24 h after unilateral ligation of the common carotid artery. In the experiments of series 1 (eight dogs) perfusion was carried out under general anesthesia with 5-10% formalin solution in phosphate buffer, pH 7.4. In series 2 (eight dogs) a model of pathological kinking of the common carotid artery was created unilaterally. After 24 h the animals were withdrawn from the experiment and the brain fixed by perfusion. Material from three different regions of the cerebral cortex (frontal, parietal, and occipital), from symmetrical areas on the right and left sides, was subjected to light-optical and electron-microscopic investigation. Serial histologic and semithin sections were stained with hematoxylin and eosin and with cresyl violet by Nissl's method. Material was processed for electron microscopy in the usual way.

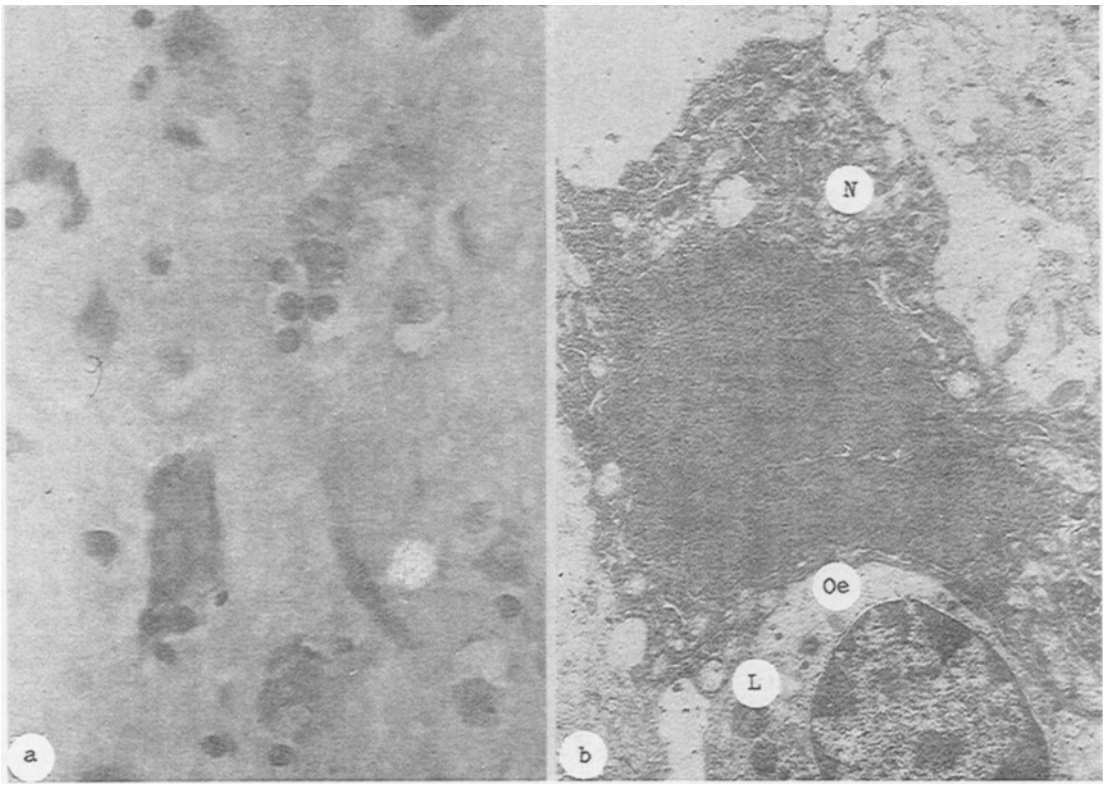

Fig. 2. Relations between ganglion and glial cells in cerebral cortex 24 h after unilateral ligation of carotid artery: a) satellitosis and neuronophagy of a destroyed neuron, Nissl's stain, 400×; b) satellitosis of pycnomorphic neuron (N) in cytoplasm of an oligodendrocyte (Ol), with lysosome (L) formation, 8000×.

EXPERIMENTAL RESULTS

Histologic investigation of material from the side of ligation of the right common carotid artery (series 1) revealed changes in the blood vessels and neurons in all three regions of the cortex. However, the severity and extent of the changes differed somewhat in each part.

In the first place dilatation and congestion of the vessels of a moderate to marked degree were observed. The microcirculatory bed was predominantly affected by hyperemia. The hyperemia was more marked in the frontal lobe, where marked capillary dilatation was present, sometimes with extravasation (Fig. 1a).

Lesions of the neurons were found in all parts of the cortex, and were of mild, moderately severe, and severe degree. Changes in the neurons were more marked in the parietal lobe.

The mild lesions consisted of edema and swelling of single neurons, whose cytoplasm appeared pale blue and homogeneous when stained by Nissl's method. Intensive staining of the cell body while the processes stained palely was frequently observed. The nucleus was situated eccentrically. Moderately severe lesions consisted of swelling, deformation, and hyperchromatosis of the neurons. Swelling was sometimes combined with vacuolation of the cytoplasm of the nerve cells. As a rule pericellular edema was found. These processes also were accompanied by satellitosis (Fig. 2a, b).

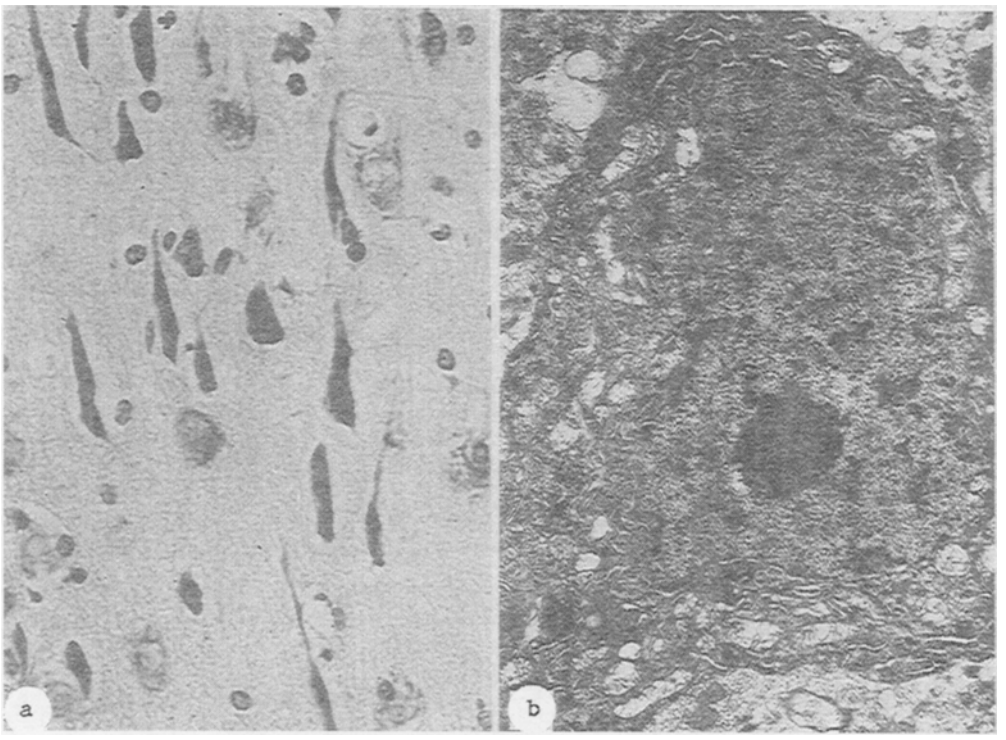

Fig. 3. Morphologic changes in ganglion cells of canine cerebral cortex 24 h after unilateral ligation of carotid artery: a) marked hyperchromatosis and pycnosis of neurons with pericellular edema, Nissl's stain, 200×; b) pycnomorphic neuron, condensation of ergastoplasm and karyoplasm, destruction of mitochondrial cristae. 9000×.

Severe damage took the form of a varied degree of hyperchromatosis and pycnosis of the nerve cell nuclei. Electron-microscopy revealed destruction of the ultrastructure, vacuolation, and marked osmiophilia of the nucleus and cytoplasm of the pycnomorphic cells (Fig. 3a, b). Their outlines were blurred, the cell membrane destroyed, and the cytoplasm became foamlike and underwent lysis. Frequently signs of gliosis (neuronophagy) were found in the cytoplasm of the altered cells. The cytoarchitectonics of the cortex was disturbed, with the appearance of empty spaces and zones of cell depopulation in place of dying neurons.

Histologic study of the cerebral cortex on the side of pathological kinking of the carotid artery (series 2), just as with total occlusion (series 1), revealed changes in the neurons and microcirculatory bed, which could be detected in all three regions studied. However, the changes were less severe than after occlusion of the carotid artery (series 1).

The blood vessels of the cerebral cortex of the experimental animals (series 2) were slightly dilated and congested in some places. Perivascular edema could be seen around individual pyramidal neurons, whose cytoplasm was translucent and stained palely by Nissl's method, and the nuclei were converted into pale vesicles. Neighboring neurons had the ordinary shape and staining properties, or the latter were even enhanced to some degree. Vacuolation of the body of the nerve cells was rare, as also was focal destruction and loss of individual neurons, i.e., the cytoarchitectonics of the cortex was undisturbed and its stratification preserved.

The degenerative changes in the nerve cells described above were found in all regions of the cerebral cortex. However, it is impossible to state in which region the changes were more marked, because they were found comparatively rarely and were mosaic in character. Furthermore, in the symmetrical areas of the contralateral lobes the

changes observed were almost the same, basically mild in severity, and characterized by pericellular edema and swelling of the cytoplasm of the altered neurons.

Electron-microscopic study of the material in series 1 and 2 of the experiment in zones most affected by ischemia in the cerebral cortex as shown by light microscopy, revealed changes in the microcirculatory bed. These changes were manifested mainly in the capillary wall: swelling of the endothelial cells and their nuclei, widening of the basal layer of the capillaries, fenestrations, and junctions between processes of endothelial cells. All these changes are characteristic features of increased vascular permeability. As regards the pericapillary edema, it has a number of special features in the cerebral cortex. Under the light microscope the typical picture of this pathological process was seen in the form of the appearance of a lucid interval between the capillary wall and the surrounding brain tissue (Fig. 1a). On electron microscopy, however, the lucid interval was found to contain edematous and swollen processes of glial cells (astrocytes), whose outer membranes extend, as they do normally, in firm contact with the basal layer of the capillary (Fig. 1b). This is evidence that the so-called pericapillary edema in the brain is more likely to be edema and swelling of processes of glial cells surrounding the capillary, so that true pericapillary edema does not arise here.

Destructive ischemic changes in cerebral cortical neurons were thus observed 24 h after ligation of the common carotid artery, both on the side of occlusion and on the contralateral side. No foci of ischemic infarction of the cortex were found. Destructive changes in ganglion cells were mainly mild or only moderately severe. Mosaic changes also were found in the ultrastructure of the microcirculatory bed. The structural disturbances were more marked on the side of occlusion than on the control side. This proves that a zone of ischemic "penumbra" is formed during occlusive changes in the brachiocephalic arteries, the existence of which is still a matter for debate.

The morphologic study of the cerebral cortex of dogs with pathological kinking of the common carotid artery gives a more precise picture of the ischemic changes taking place in the neurons and can help clinicians to determine their tactics for correcting neurological symptoms.

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