STRUCTURAL CHANGES IN THE CNS OF DOGS WITH BILATERAL LESIONS OF THE CAROTID ARTERIES

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One of the commonest causes of acute disturbance of the cerebral circulation is a combined bilateral lesion of the carotid arteries. By examination of clinical material, it is possible to some extent to assess the various forms of pathology in the brachiocephalic vessels [1, 7, 8]. However, in the accessible literature we were unable to find the results of experimental morphological studies of a combined lesion of the extracranial portions of the carotid arteries to confirm the clinical evidence.

The aim of this investigation was a morphological analysis of changes taking place in the cerebral cortex after pathological kinking of the common carotid artery and occlusion or stenosis of the internal carotid artery on the opposite side.

EXPERIMENTAL METHOD

An original method of producing pathological kinking of the common carotid artery in the neck in dogs with the aid of a model shaping device, with an angle of kinking of each model of 30° and 45°, and with a model of complete occlusion (ligation) of the internal carotid artery, causing stenosis of the artery with the aid of clamps reducing the lumen to two-thirds of its diameter [6] was developed. Experiments were carried out on 20 mongrel dogs weighing 10-12 kg under general anestheia, and with angiographic and radio-isotopic control. The angiographic investigation of the animals' carotid arteries was carried out on an ARD-2 apparatus with "VARKT" attachment, the radio-isotopic investigation with the use of albumin-I^{131}. Altogether 48 informative angiograms and the results of 24-radio-isotopic investigations were studied to determine the linear velocity of the blood flow in the heart–brain segment. The material used consisted of the brain, taken 24 h after creation of the bilateral lesion of the carotid arteries.

Series 1 consisted of eight animals in which pathological kinking of the right common carotid artery was reproduced during the operation with an angle of kinking of 45° and with occlusion of the left internal carotid artery at the point of bifurcation.

Series 2 consisted of eight dogs in which pathological kinking of the right common carotid artery with an angle of 30° and occlusion of the left internal carotid-artery distally to the bifurcation were created (Fig. 1).

Series 3 was the control and consisted of four animals.

Under general anesthesia perfusion was carried out with 5-10% formalin solution in phosphate buffer, pH 7.4. Pathological kinking of the right common carotid artery was not chosen by accident, for because of its anatomical structure (arising from the brachiocephalic trunk), it undergoes deviation much more frequently. For the same reason, it undergoes greater deformation in the presence of hemodynamic disturbances, in particular in arterial hypertension. The animals were taken from the experiment after 24 h and the brain subsequently fixed by perfusion. Material from three regions of the cerebral cortex (frontal, parietal, occipital) from symmetrical areas on the right

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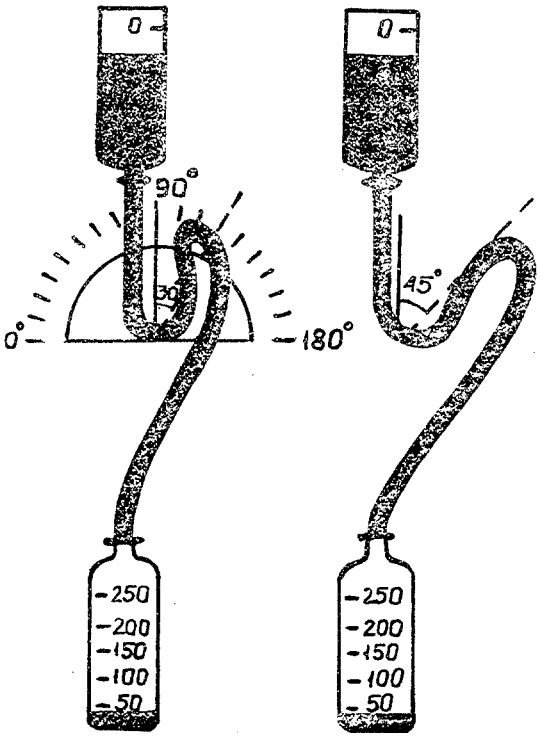

Fig. 1

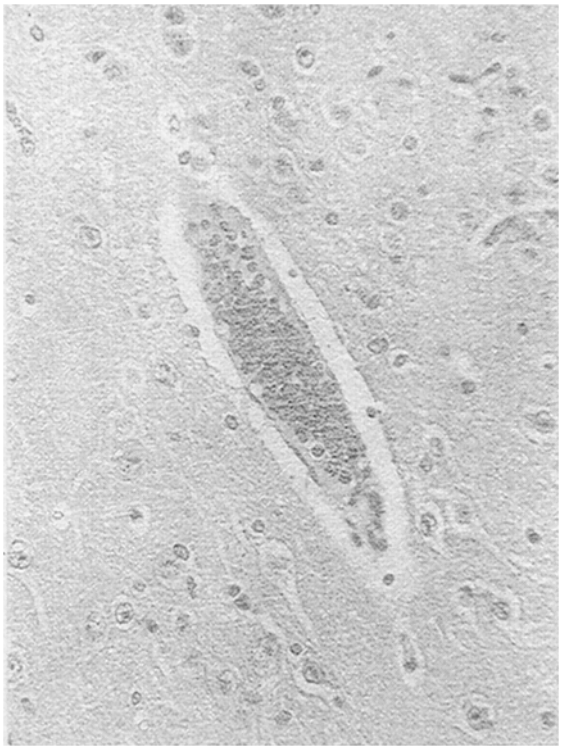

Fig. 2

Fig. 1. Diagram showing method of obtaining pathological kinking with an angle of 30° and 45°.

Fig. 2. Congestion and pericapillary edema in cerebral cortex of dog 24 h after operation to create pathological kinking of carotid artery with angle of 45°, Hematoxylin-eosin 260×.

and left sides was subjected to light-optical and electron-microscopic study. Serial histologic and semithin sections were stained with hematoxylin and eosin and with cresyl violet by Nissl's method. Processing of the material for electron microscopy was carried out by the usual method.

EXPERIMENTAL RESULTS

The results of the experimental morphological studies of series 1 showed that as early as 24 h after creation of the bilateral lesion, smoothing out of the fissures and swelling of the gyri were observed macroscopically in both cerebral hemispheres compared with the brain of the intact control animals. Vessels of the pia mater were congested in both hemispheres. Boundaries of the cortex and white matter of the brain in sections were quite clearly distinguished. No focal destructive changes could be found. On microscopic investigation of symmetrical areas of the brain in both hemispheres marked dilatation and congestion of the vessels and marked perivascular edema were observed compared with the brain of the control animals (Fig. 2). Widening of the intercellular spaces could be seen around individual neurons. The cytoplasm of these neurons was translucent, palely stained, and the nuclei were converted into pale vesicles. Meanwhile the neurons showed a severe degree of injury. Vacuolation of the bodies of the nerve cells was observed more often, as also was the case with focal destruction of the nerve cells and a disturbance of

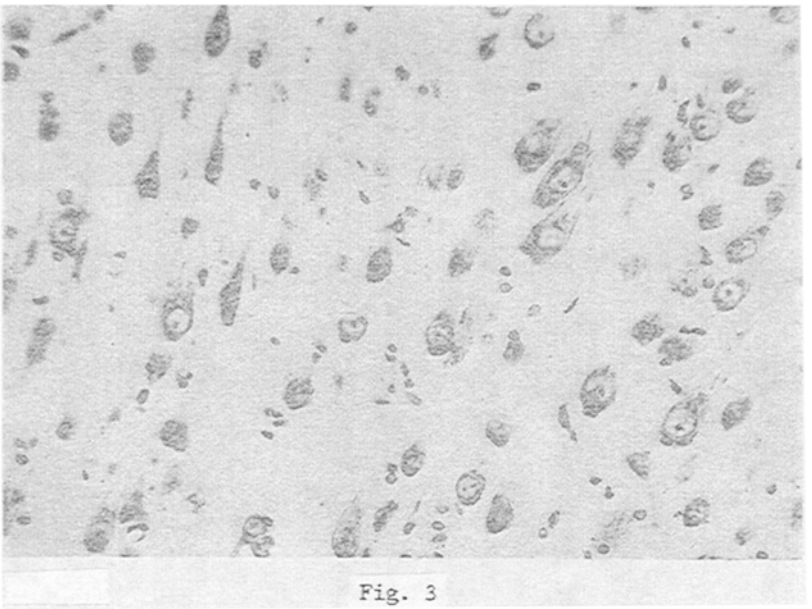

Fig. 3

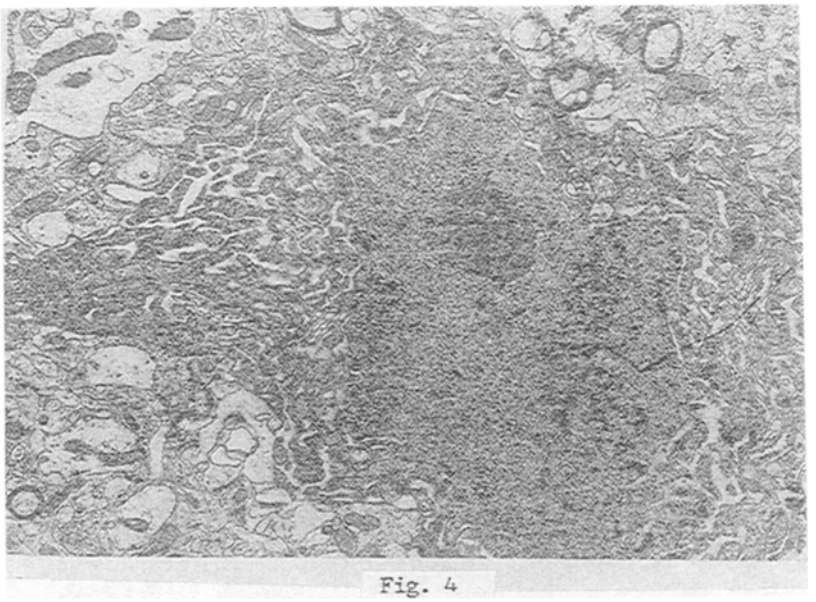

Fig. 4

Fig. 3. Cytoarchitectonic picture of layer 5 of cerebral cortex of dog with pathological kinking of carotid arteries at an angle of 30°, 24 h after operation. Hyperchromatosis of pyramidal neurons and zone of devastation, swelling of neurons and apical processes in right part, with ectopia of nucleus and nucleoli. Nissl's stain, 200×.

Fig. 4. Pycnomorphic neuron from layer 5 of cerebral cortex of dog 24 h after operation (angle of kinking 30°). Condensation of ribosomes and polysomes of rough endoplasmic reticulum, nucleus, and nucleolus. 15,000×.

the cytoarchitectonics of the cerebral cortex. Changes of this kind were less marked in both hemispheres on the side of experimental kinking with an angle of 45°.

In the experiments of series 2 the same morphological changes were observed as in the study of the cerebral cortex in the frontal, parietal, and occipital regions. At the same time, however, a certain difference must be pointed out. To begin with, there were disturbances of the cytoarchitectonics of the cortex (Fig. 3), which were more marked than in series but we were unable to find areas of ischemic cerebral infarction, although it was evidently possible to speak of the appearance of a Penumbra zone (ischemic half-shade). This tendency can be explained by a deficiency of the material blood flow supplying the brain, and it is associated with a more acute angle of kinking (30°), giving rise to critical stenosis of the common carotid artery and leading to hypoxia of brain tissue on the side of the lesion. These morphological destructive changes in the brain were caused by the inadequate cerebral blood flow, for an angle of kinking of 30° reduces it by 72% and an angle of kinking of 45° by 58% of the expected value. Furthermore, the results of electron-microscopy revealed pycnomorphic neurons in layer 5 of the cerebral cortex, which corresponded at the light-optical level, on staining by Nissl's method, to hyperchromic ganglion cells. This zone of devastation in the cortex, where the cytoarchitectonic pattern was disturbed, is evidently connected with the presence of hyperchromic cells (at the light-optical level) and of pycnomorphic cells (the ultrastructural level) [4] (Fig. 4).

On the basis of these results it is possible to determine the morphological substrate of the clinical manifestation of the disease Acute ischemic changes discovered in neurons of the cerebral cortex are evidently reversible in character, and after timely surgical correction of the pathological changes in the carotid arteries they give a positive result with regression of neurologic symptoms even with a bilateral lesion.

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