MORPHOLOGICAL CHANGES IN THE BRAIN OF THE CAT AS THE RESULT OF ACUTE CLOSED INJURY TO THE SKULL

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The study of closed injuries of the skull and brain goes back many centuries but many aspects of this important subject still remain debatable and undecided. This applies, above all, to the explanation of the degree and character of the morphological changes in the brain. The development of the morphological changes as well as their pathogenesis have received little study. Some workers [1, 3, 9] believe that, in closed trauma, changes in the nerve cells occur primarily. Others [8, 11] regard disorders of the circulation as primary and the cell changes as secondary, developing after several hours [10] or even days [2] following injury. The situation of the major lesions is unexplained. A.L. Fisanovich [6] points out that in injuries of the convex surface of the skull, the morphological changes are shown most severely at the site of the blow. According to L.I. Smirnova [4] and P.E. Snesarev [5], severe changes are found in the brainstem. There are also differences of opinion about the origin of the hemorrhages. B.I. Sharapov [7] believes the appearance of these to be due to rupture and stretching of the vessel walls, whereas L.I. Smirnov and P.E. Snesarev [4, 5] confer the leading role on diapedesis.

The conflicting nature of the available experimental evidence impelled us to study the character and the course of the morphological changes in the brain after closed injury to the skull.

EXPERIMENTAL METHOD

Experiments were carried out on 32 healthy cats weighing from 1900 to 4100 g. A closed injury to the skull and brain of constant magnitude was inflicted on 22 animals by means of a 1 kg weight, falling through a tube of diameter 7 cm from a height of 2 m. The area of the blow was 2 × 2 cm. The injury was localized to the right frontoparietal region. Ten cats acted as controls. Of the 22 animals, three, weighing less than 2 kg died immediately from the injury. The animals weighing more than 2 kg survived. The cats were killed by air emboilism after 24 hours and 2, 3, 5, 8, 12, 15, 18, 21 and 45 days. Autopsy of the animals was performed immediately after death. The brain was fixed in 10% formalin. Histological examinations were made of the frontal segments of the brain, together with the subcortical ganglia, the brainstem and the cerebellum. Part of the material was taken through celloidin, and the rest cut into sections on the freezing microtome. Sections were stained with the routine examination methods (hematoxylin-eosin, Van Gieson) and various neurohistological and special methods (Nissl's, Spielmeyer's, Gol'tser's, Hortega's, Aleksandrovskaya's modification of Miagawa's method, Cajal's gold-mercuric chloride method, Mallory's method and Sudan III).

EXPERIMENTAL RESULTS

Immediately after injury, the cats showed transient immobility, accompanied by a lowering of muscle tone and failure to react to painful stimulation. In some cases, spasms of the limbs occurred, with tachycardia, slowing of the respiration, dilatation of the pupil on the side of injury, lacrimation, hemiparesis on the side opposite the injury and involuntary micturition. These signs disappeared in the course of the first three days, and the animals outwardly appeared healthy.

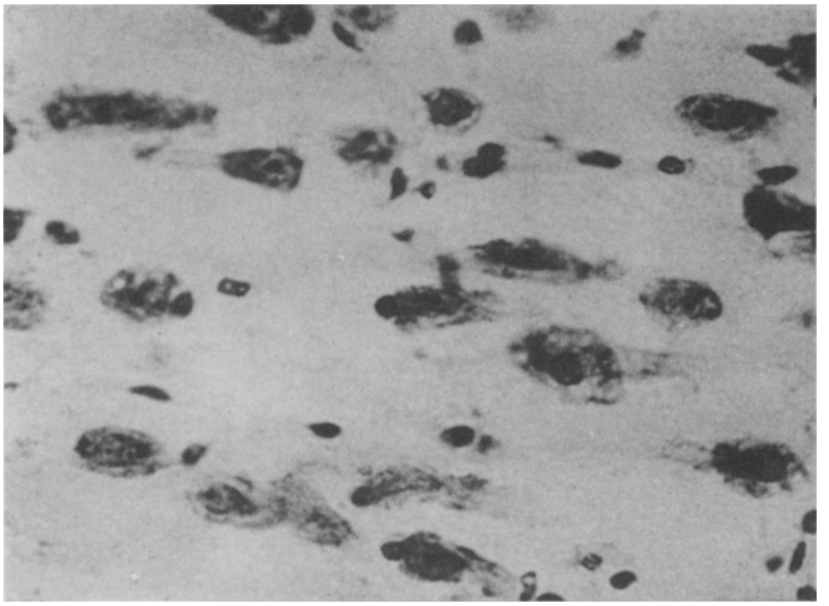

Fig. 1. Changes in the nerve cells in the deep layers of the cortex 24 hours after injury. Vacuolation, chromatolysis, "cell-shadows" and solitary pyknomorphic cells. Microphotograph. Objective 40, ocular 10. Stained with thionine.

Fig. 2. Hemorrhages in the substance of the pons variolii on the 21st day after injury. Microphotograph. Objective 40, ocular 10. Spielmeyer's stain.

In the cats which were subjected to trauma and killed at different times during the experiment, no macroscopic changes were found in the substance of the brain. On histological examination at various times during the experiment, however, considerable micorscopic abnormalities were seen.

An even hyperemia of the pia mater and the brain substance was observed 24 hours after injury. In the basal areas of the pons variolii and the medulla oblongata, small hemorrhages were observed near the vessels.

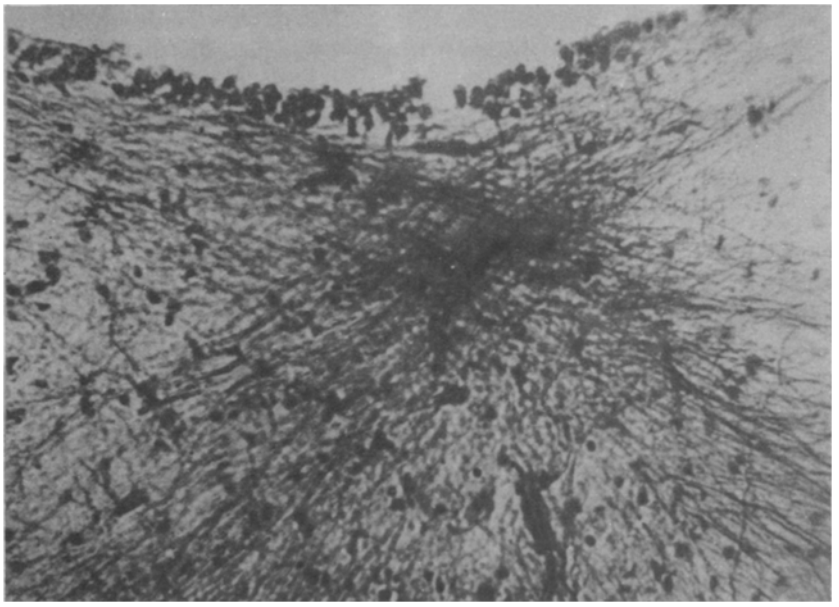

Fig. 3. Signs of fibrous gliosis in the subependymal layer of the aqueduct of Sylvius on the 45th day after injury. Microphotograph. Objective 40, ocular 10. Gol'tser's stain.

The white matter of the cerebral hemispheres, the brainstem and the cerebellum were edematous and permeated by round and fairly large vacuoles, displacing the nerve fibers. In areas containing cells of the fascicular oligodendroglia, the dematous changes were expressed as the formation of a zone of translucency aroung the "drainage" cells. In the cerebral cortex, individual nerve cells and groups underwent pyknomorphic changes (shrinking of the nucleus and protoplasm, diffuse staining with thionine). Starting with the third layer of cortical cells, hydropic changes were predominant, in the form of vacuolation of the protoplasm and signs of chromatolysis of the Nissl substance, with conversion of individual cells into "shadows" (Fig. 1). Signs of neuronophagia were observed aroung the affected cells.

In the subcortical ganglia, the brainstem and the cerebellum the changes in the nerve cells were much more strongly expressed. The macro- and microglial cells were not particularly affected. No essential difference between the right and left cerebral hemispheres, from the point of view of circulatory distrubances or cell changes, could be observed.

At later periods in the experiment, on the 2nd, 3rd and 5th days, disturbances of the circulation in the form of hyperemia of the vessels of the brain substance with hemorrhages around them, signs of edema and changes in the cells (vacuolation, chromatolysis, ischemic changes) continued to dominate the histological picture, reaching their maximum on the 8th day. On the 12-18th day after injury, the character of the changes in the nerve cells of the cerebral cortex, the subcortical ganglia, the nuclei of the brainstem and the cerebellum remained as before, but the degree of intensity of the cell changes, like the circulatory disturbances and signs of edema of the brain substance, was diminished. At the same time there was increased hyperplasia of the macroglial cells, with growth of the marginal layer of glial fibers and also of the layer of fibers beneath the ependyma of the ventricles and the aqueduct of Sylvius.

On the 21st day after injury, circulatory distrubances were present in the form of slight hyperemia of the brain substance and the membranes, with solitary, small hemorrhages (Fig. 2). The edema of the white matter persisted, although to a much less degree than in the preceding times of the experiment. Hydropic changes in the nerve cells of the cortex, subcortical ganglia, brainstem nuclei and cerebellum, at this time, took the form of slight vacuolation. Less commonly, cells were found in a state of chromatolysis and "shadow" conversion. As before, signs of neuronophagia were observed. Side by side with the diminution of the pathological disturbances in the brain, especially the destructive changes in the nerve cells, the reactive gliosis perceptibly progressed: the hyperplasia of the macroglial cells was accompanied by the development of marginal and subependymal gliosis.

The macroglial cells became hypertrophied. 45 days after injury the circulatory disturbances in the brain disappeared and the ischemic changes in the nerve cells were also absent. Edema of the white matter, at this period, was very slight. Only solitary nerve cells of the subcortical ganglia, brainstem nuclei and cerebellum were affected by hydropic changes, signs of chromatolysis of the Nissl's granules and conversion of the cells into "shadows". Signs of neuronophagia still remained fairly pronounced in the subcortical ganglia. By comparison with the 21st day of the experiment, there was a perceptible increase in the signs of marginal and subependymal gliosis (Fig. 3), due to hyperplasia of the astrocytic glia.

At autopsy of the cats which died at the time of injury, a fracture of the base of the skull with massive hemorrhages in the basal areas of the brain (brainstem, base of the temporal lobes) were found. On both naked-eye and microscopic examination of the brain, no signs of pulping of the brain substance were found. On the convex surface of the cerebral hemispheres, in the cortex near to the vessels were solitary small hemorrhages. In the subcortical ganglia and the cerebellum, these were found somewhat more often, and in the brainstem, they were far more numerous, while in the basal areas, they acquired a confluent and massive character.

Thus, in the cats with fatal injuries, as in animals killed at various times during the experiment, the greatest number of hemorrhages was observed not at the site of the blow but at the base of the brain.

It is a noteworthy fact that the character of the changes in the brain was no different from that found in the course of our experiment, although the degree of both the circulatory and the edematous and degenerative (in the nerve cells) changes in the brain was very severe, corresponding roughly to that which we observed in cats killed on the 8th day after injury.

SUMMARY

Experiments were conducted on 32 healthy cats. Closed trauma of the cranium was inflicted in 22 of them, 10 served as control. The animals were sacrificed at intervals ranging from 1 to 45 days. Investigations of the brain were conducted by various neurohistological methods.

It has been established that following a closed trauma of the skull the cat's brain shows different pathomorphological changes, disturbances of the blood and cerebrospinal fluid dynamics (hyperemia, stasis, hemorrhages, edema of the white matter) and degenerative-regressive changes in the nerve cells (hydropic, pyknomorphous, chromatolysis of Nissl's bodies, transformation of cells into "shadows").

These changes rise until the eighth day and then begin to decline. Simultaneously, a progressive reaction of the glia (hypertrophy of microglia, hyperplasia of macroglia) becomes apparent, resulting in the development of fibrous gliosis in the marginal and subependymal layers.

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