# INCREASE IN THE EPILEPTIC REACTION OF RATS DURING ACUTE DISTURBANCE OF THE CIRCULATION IN THE ZONE SUPPLIED BY THE EXTRACRANIAL VASCULAR TRUNKS OF THE BRAIN

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Interest in the pathology of the extracranial vascular trunks of the brain has recently increased [5-8]. The neurological symptoms of apoplexy after disturbance of the cerebral circulation have long been regarded as a manifestation of disease of the intracranial vessels. However, as a result of the development of angiography [9] it has been found that cerebral disturbances may also be caused by disease in the extracranial vessels, both carotid and vertebral. The clinical picture of these vascular lesions has been clearly defined, and may consist of various neurological symptoms (hemipareses, aphasias, sensory disturbances, epileptic fits, etc.). The appearance of epileptic fits in patients with diseases of the extracranial portions of the vascular trunks of the brain has been reported by several authors [2, 5-8, 10, 12, 13].

The main pathogenetic factor in the epileptiform fits is considered by E. V. Shmidt to be the hypoxia developing because of the occlusive process in the carotid arteries.

On the assumption that disturbances of the cerebral circulation may result from pathological processes in the extracranial portions of the carotid and vertebral arteries, experiments were carried out to study the pathogenetic relationship between epliptic fits and the patency of the extracranial portion of the cerebral vessels.

## EXPERIMENTAL METHOD

Experiments were carried out on 104 albino rats (70 experimental and 34 control), sensitive to an acoustic stimulus. Epileptic fits were caused by the method of overexcitation of the central nervous system by an acoustic stimulus, developed in L. V. Krushinskii's laboratory [3]. After determination of the background reaction of the rats to acoustic stimulation (3 or 4 applications of the sound on alternate days), the following operations were carried out: ligation of the left common carotid artery (30 rats) without general anesthesia; ligation of the right common carotid artery in the same rats (in 17 of the 30 animals) without general anesthesia; one-stage ligation of both common carotid arteries (30 rats) under urethane anesthesia; one-stage ligation of both vertebral arteries (10 rats) under urethane anesthesia; pseudo-operations in the animals of the control group (34 rats).

Twenty-four hours after the operation on the common carotid arteries, the animals were again exposed to acoustic stimulation. In the case of ligation of the vertebral arteries, experiments were carried out on the rats on the 7th day after the operation. Before and after the operation, the character of the rats' reaction to the bell was determined from the length of the latent period, the intensity of the reaction and the intensity of the fit.

## EXPERIMENTAL RESULTS

In the experiments of series I (operations without anesthesia) on the rats with ligation of the left common carotid artery, the indices mentioned above were practically unchanged. After ligation of the right common carotid artery in the same animals one week after the first operation, the intensity of the reaction and the fit diminished, although these changes were not statistically significant. These indices were unchanged in the control animals. In the second group of experimental rats, in which both common carotid arteries were ligated simultaneously under general urethane anesthesia, changes took place in the main indices of the reaction: the latent period was shortened, and the intensity of the reaction and of the fit increased (see the table).

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Effect of One-Stage Ligation of Both Common Carotid Arteries on the Course of the Epileptic Fit

| Experimental conditions | Index of reaction | Before operation | After operation | t |
|---|---|---|---|---|
| One-stage ligation of common carotid arteries (30 rats) | Latent period (in sec) | 12.19±1.17 | 8.63±0.65 | 2.40 |
| | Intensity of reaction (in points) | 1.40±0.11 | 1.82±0.09 | 2.91 |
| | Intensity of fit (in points) | 1.14±0.17 | 1.84±0.13 | 3.32 |
| Control (14 rats) | Latent period (in sec) | 10.00±1.19 | 7.74±0.76 | 1.60 |
| | Intensity of reaction (in points) | 1.76±0.03 | 1.98±0.14 | 1.46 |
| | Intensity of fit (in points) | 1.61±0.21 | 1.78±0.18 | 0.59 |

In the 10 rats in which both vertebral arteries were ligated at the same time no changes could be found in the latent period and intensity of the reaction. The epileptic reaction in these rats was appreciably increased. The difference of 0.80±0.33 point was statistically significant ($t = 2.43$ ; $P < 0.02$).

The reaction of the rat to acoustic stimulation revealed changes in the fundamental nervous processes of excitation and inhibition [4]. The shortening of the latent period and increase in the intensity of the reaction and of the epileptic fit demonstrated an increase in the excitability of the nervous system. This increase in excitability, caused by preliminary ligation of the carotid arteries, in the present experiments led to an increase in the severity of the epileptiform reaction in 17 of 26 rats (4 of the 30 animals did not have epileptic fits during the period of the experiment), the reaction was unchanged in 3, and in 6 it was very slightly weaker. After stenosis of the vertebral arteries, the epileptic reaction became stronger in 4 of 5 rats and weaker in 1 animal.

In these experiments the cause of the increase in the epileptic reaction after ligation of the vessels in their extracranial portion was evidently acute cerebral hypoxia. Hypoxia is known to produce dysrhythmic disturbances of the cortical neurons [1, 11]. Conditions of "convulsive readiness" are created in the form of both structural and functional changes in the nervous system, which by themselves cannot give rise to fits. Before this happens, additional factors are necessary. In the present experiment such an additional factor was acoustic stimulation, which, against the background of the increased excitability of the brain, readily produced an epileptic discharge.

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