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Research Article

Morphological Changes in rats' Cerebral Cortex Neurons during Strangulation Asphyxia

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Abstract

Objective: To study the peculiarities of morphological changes of neurons of the occipital lobe of rats with strangulation asphyxiation.

Methods: The study was carried out on the native white rats (24 males, weighing 240+20 g), divided into a control group and three experimental groups with a strangulation asphyxia of 6 individuals in each group.

Results: Morphological changes in the occipital cortex neurons were observed in experimental animal groups during the study periods, in the form of changes in the area, shape of the neurons and intensity of cytoplasm staining.

Conclusion: It was concluded that the strangulation asphyxia caused anoxic damage to the neurons of the occipital lobe of rats, manifested in the form of changes in the size, shape of neurons, intensity of coloration of their cytoplasm.

Keywords: asphyxia; neurons; occipital cortex; brain; rats

Introduction

Acute oxygen deficiency can cause severe and often irreversible changes in brain tissue and even lead to its death [3]. Brain asphyxia occurs in many diseases, pathological conditions, and due to environmental factors. Specifically, oxygen deficit can result from impaired air passage in the respiratory tract due to external mechanical factors (mechanical asphyxia) [4]. The occipital lobe cortex, responsible for visual information perception and processing, and orientation in new environments, deserves special attention. Hypoxia in this brain region can lead to loss of visual function - cortical blindness.

Previous studies have examined morphological changes in the brain cortex under total ischemia caused by decapitation [1, 2].

To date, the specific damage to neurons in the occipital lobe cortex due to respiratory-genesis anoxia caused by external mechanical factors has not been studied.

The aim is to study the morphological changes in neurons of the occipital lobe cortex in rats with strangulation asphyxia.

Materials and methods:

The study was conducted on non-breed white male rats (24 individuals, weight 240±20 g), divided into a control and three experimental groups with strangulation asphyxia, six animals in each group.

The control group consisted of sham-operated rats without strangulation asphyxia (group 1, n=6).

Strangulation asphyxia was modeled under thiopental anesthesia (intravenous, 50 mg/kg) by applying a ligature to the trachea 1.0 cm below the cricoid cartilage for 30 minutes (group 2), 60 minutes (group 3), and 24 hours (group 4) [7].

The experiment was conducted in compliance with the requirements of the European Parliament and Council Directive No. 2010/63/EU of 22.09.2010 on the protection of animals used for scientific purposes.

The brain was extracted in the cold and fixed in Carnoy's solution. Serial frontal paraffin sections 7 µm thick were prepared and stained with thionin using the Nissl method. The location of the occipital lobe cortex was established using a stereotaxic atlas [6].

In each animal, 30 neurons of the fifth layer of the occipital lobe cortex were studied, determining their area, shape, and chromophilic degree. Changes in the area and shape (form factor, elongation factor) of neurons were assessed using the ImageWarp image analysis software (Bitflow, USA) [5].

Obtained quantitative continuous data were processed using non-parametric statistical methods and the licensed computer program

Statistica 10.0 for Windows (StatSoft, Inc., USA). Data are presented as Me(LQ;UQ), where Me – median, LQ – lower quartile value; UQ – upper quartile value. Differences between the control and experimental groups were considered significant at $p<0.05$ (Mann-Whitney U-test).

Research results: In the experimental groups, morphological changes in the neurons of the occipital lobe cortex were observed during the studied periods in terms of changes in area (table 1, fig.2), neuron shape (table 1, fig.1,3), and the intensity of their cytoplasm staining (figure. 4).

Группы	Показатели		
	площадь нейронов (мкм^2)	форм-фактор (ед.)	фактор элонгации (ед.)
контроль	220,0 (175,5; 264,5)	0,9 (0,9; 0,9)	1,4 (1,2; 1,4)
СА 30 мин	190,5 (145,5; 234,5)	0,6 (0,6; 0,6)*	2,3 (2,2; 2,4)*
СА 60 мин	124,4 (123,4; 126,4)*#	0,6 (0,6; 0,6)*	2,4 (2,3; 2,4)*
СА 24 ч	111,1 (109,2; 119,3)*#&	0,6 (0,6; 0,6)*	2,6 (2,5; 2,8)*#&

Note: - * - differences are significant ($p<0.05$) compared to the control group
- # - differences are significant ($p<0.05$) compared to 30 minutes of SA
- & - differences are significant ($p<0.05$) compared to 60 minutes of SA

Table 1 - Parameters of size (area) and shape (form factor and elongation factor) of neurons in the occipital lobe cortex of rats with strangulation asphyxia (SA), Me(LQ;UQ)

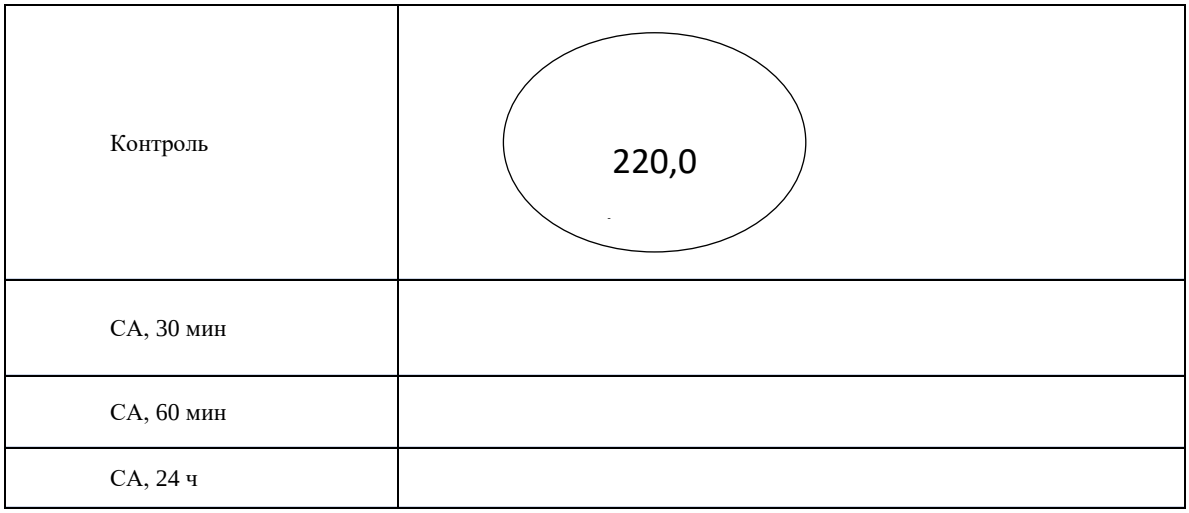


Figure 1. Graphical representation of morphometric parameters of neurons (size, μm², and shape) in the occipital lobe cortex of rats with strangulation asphyxia (SA)

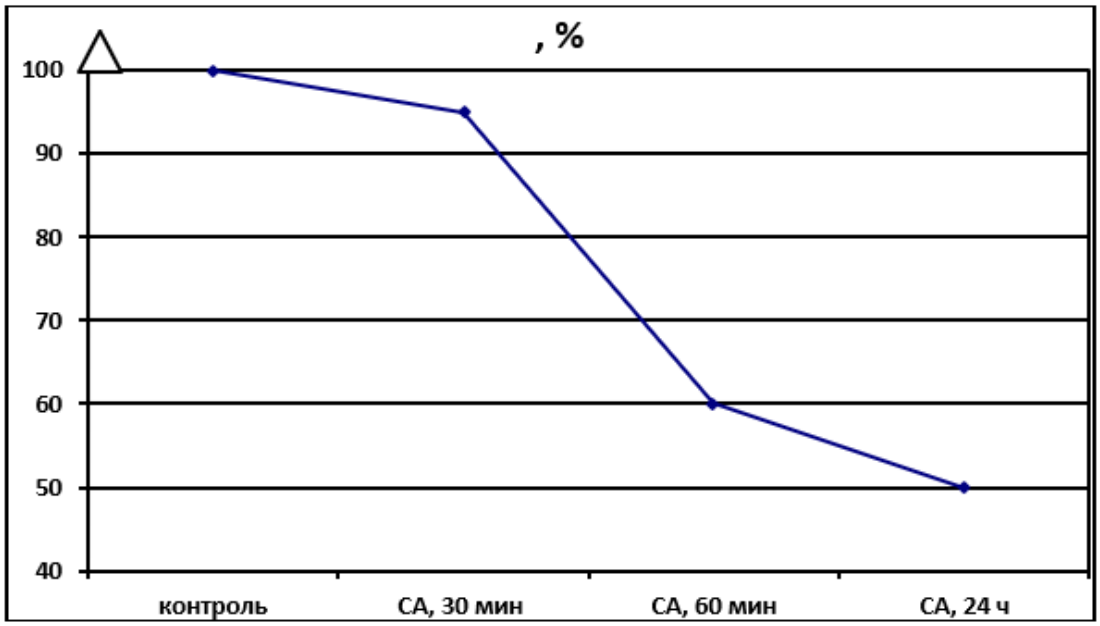
After 30 minutes of asphyxia, there were no changes in the area of neurons compared to the control group, but a decrease in the form factor by 29% ($p<0.05$) and an increase in the elongation factor by 68% ($p<0.05$) were noted, indicating a change in the shape of neurons in terms of loss of sphericity and increased elongation.

After 60 minutes of asphyxia, the area of neurons decreased by 40% compared to the control group ($p<0.05$), while the form factor decreased by 32% ($p<0.05$), and the elongation factor, conversely, increased by 74% ($p<0.05$).

Additionally, in rats with 60 minutes of asphyxia, there was a 35% decrease in neuron area compared to the 30-minute asphyxia period ($p<0.05$), but no changes in the shape of neurons were observed ($p>0.05$).

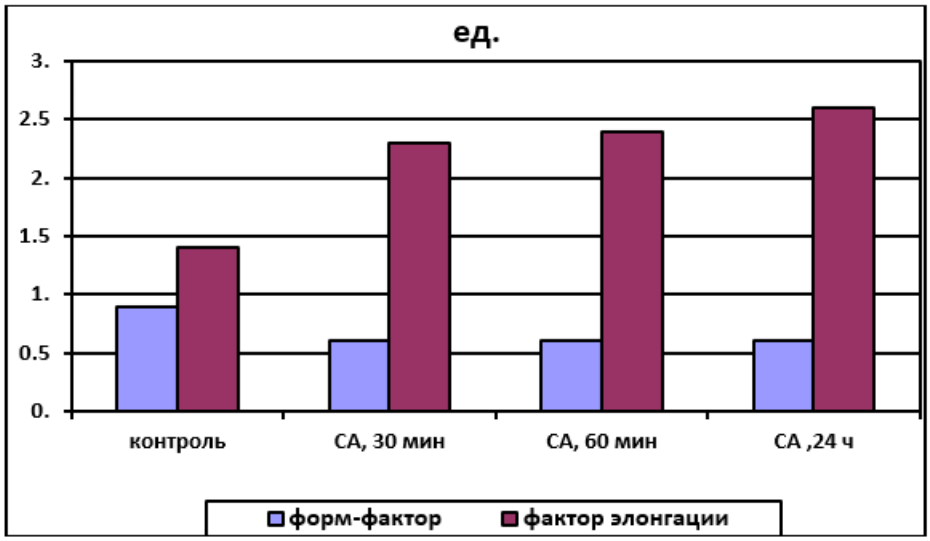
By 24 hours of asphyxia, the area of neurons decreased by 50% compared to the control group ($p<0.05$), the form factor decreased by 32% ($p<0.05$), and the elongation factor increased by 88% ($p<0.05$), reflecting an increase in the elongation of perikarya.

In rats with 24-hour asphyxia, a decrease in neuron area was observed compared to the 30 and 60-minute asphyxia periods (a 42% decrease ($p<0.05$) and 11% decrease ($p<0.05$), respectively), as well as an increase in neuron elongation (an 11% increase in the elongation factor ($p<0.05$) and 8% increase ($p<0.05$), respectively, while the form factor remained unchanged ($p>0.05$)).



Note: - * - differences are significant ($p<0.05$) compared to the control group
- # - differences are significant ($p<0.05$) compared to 30 minutes of SA
- & - differences are significant ($p<0.05$) compared to 60 minutes of SA

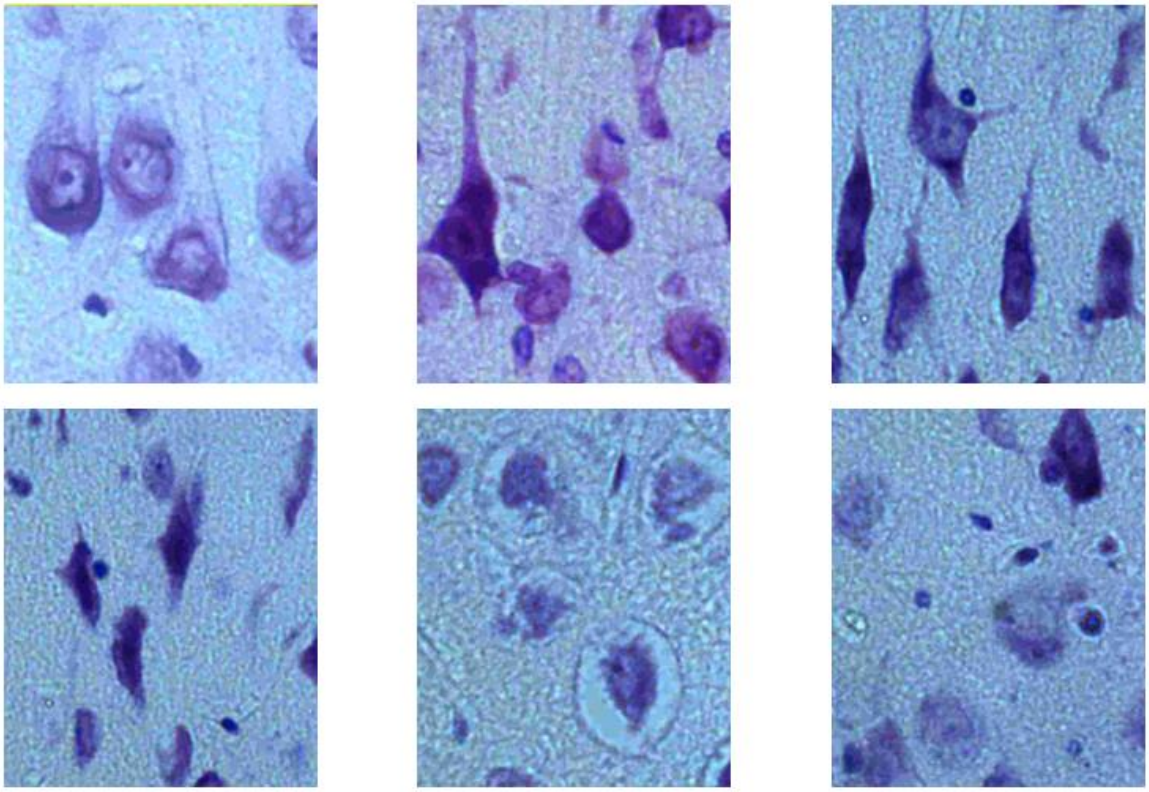
Figure 2: Changes in the area of neurons in the occipital lobe cortex of the brain during strangulation asphyxia (SA) of varying durations.



Note: - * - differences are significant ($p<0.05$) compared to the control group
- # - differences are significant ($p<0.05$) compared to 30 minutes of SA
- & - differences are significant ($p<0.05$) compared to 60 minutes of SA

Figure 3: Changes in form factor and elongation factor of neurons in the occipital lobe cortex of the brain during strangulation asphyxia (SA) of varying durations.

Unlike the control group, where 95% of neurons are normochromic cells, in the experimental groups there was a significant decrease in normochromic neurons and an increase in hyperchromic shrunken neurons: up to 75% in the group of rats with 30 minutes of strangulation asphyxia ($p<0.05$), up to 80% in the group with 60 minutes of strangulation asphyxia ($p<0.05$), and up to 64% in the group with 24 hours of strangulation asphyxia ($p<0.05$). These are known markers of anoxic damage to nerve tissue [2, 3]. By 24 hours of asphyxia, there was a complete disappearance of normochromic neurons and the appearance of, along with hyperchromic shrunken neurons, hypochromic cells with pericellular edema (11%, $p<0.05$), and shadow cells (25%, $p<0.05$).



1 - control group (normochromic neurons);
2 - 30 min strangulation asphyxia (hyperchromic shrunken neurons);
3 - 60 min strangulation asphyxia (hyperchromic shrunken neurons);
4 - 24 hr strangulation asphyxia (a - hyperchromic shrunken neurons; b - hypochromic neurons with pericellular edema; c - shadow cells).

Figure. 4 Neurons of the fifth layer of the occipital lobe cortex. Digital microphotography. Nissl staining. Magnification objective x 40.

Conclusion:

Thus, strangulation asphyxia led to anoxic damage to neurons in the occipital lobe cortex of rats, manifested in changes in the size, shape of neurons, intensity of staining of their cytoplasm. With 30 minutes of asphyxia, neurons acquired an elongated shape, losing roundness, and their sizes did not change. At 60 minutes of asphyxia, there was a significant decrease in neuron area, which worsened by 24 hours of asphyxia, while no change in neuron shape was observed. During all studied periods of asphyxia, there was an increase in the number of hyperchromic shrunken neurons with a decrease in the number of normochromic neurons and their complete disappearance by 24 hours of asphyxia.

References:

1. Bon', E. I. (2019). Dinamika morfologicheskih izmenenij piramidnyh nejronov filogeneticheski raznyh otdelov kory golovnogo mozga krys pri total'noj cerebral'noj ishemii. Dynamics of morphological changes of pyramid neurons phylogenetically of different sections of the rat cerebral cortex in total cerebral ischemia / N.E. Maksimovich, S.M. Zimatkin, N.A. Val'ko, V.N. Kot, O.V. Mosin, A.G. Sul'zhickij // Vestnik Smolenskoj gosudarstvennoj medicinskoj akademii.-– N2. – P. 5-13. (in Russian)
2. Bon', E. I. (2020). Gistologicheskie izmenenija nejronov temennoj kory golovnogo mozga krys s subtotal'noj i total'noj ishemiej. Histological changes in the neurons of the parietal cortex of rats with subtotal and total ischemia / N. E. Maksimovich, S. M. Zimatkin // Vestnik Smolenskoj gosudarstvennoj medicinskoj akademii. – V.19, N2. – P.17-23. (in Russian)
3. Maksimovich, N. E. (2020). Golovnoj mozg krysy i ego reakcija na ishemiju. Rat brain and its reaction to ischemia. – Grodno: GrSMU,– 240p. (in Russian)
4. Hohlov, V. V. (2010). Sudebnaja medicina: Rukovodstvo. Forensic medicine: Manual. – Smolensk. (in Russian)]
5. Brown BM, Newcombe RG. (2009). Non-null semi-parametric inference for the Mann-Whitney measure. Journal of Nonparametric Statistics, 743 p.
6. Paxinos, G. (1998). The Rat Brain in stereotaxis coordinates / C. Watson // Academic Press, Australia. 242 p.
7. Feduto, M.A. (2023). Modeling of Cerebral Anoxia of Respiratory Genesis in Rats / Maksimovich N.Ye., Bon E.I., Zimatkin S.M., et al. // Archives of Urology and Nephrology. 2(1). – P.1-4.

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