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Research Article

Effect of Hypoxia on Neurons of the Parietal Lobe Cortex of Rats Under Conditions of Acute Respiratory Failure

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Abstract

Acute respiratory failure, which can be caused by airway obstruction due to various reasons, has a negative effect on vital organs and, first of all, on the brain, reducing functional capabilities and leading to structural damage. When studying the cortex of the parietal lobe of the brain in rats under conditions of total and partial obstructive respiratory failure, structural changes were revealed after 30 and 60 minutes, which were manifested in a change in the size and shape of neurons, the degree of staining of their cytoplasm. For total obstructive respiratory failure lasting 30 minutes, a change in the shape of neurons is characteristic in the form of a loss of sphericity with an increase in elongation, and for a 60-minute period, a decrease in the area (by 35%, $p < 0,05$) of neurons is characteristic against the background of a significant increase in the number of hyperchromic wrinkled neurons in both time intervals to 75% and 80%, respectively. At the same time, partial obstructive respiratory failure in both periods studied was manifested by an increase in the area of neurons (by 24% and 45%, respectively, $p < 0,05$) without changing their shape against the background of an increase in the number of hypochromic neurons with signs of swelling and shadow cells (up to 80% and up to 95%, respectively). These differences are due to the different severity of acute respiratory failure.

Keywords: Obstruction, Hypoxia, Neurons, Parietal cortex.

Introduction

The main function of external respiration is to ensure adequate gas exchange with the environment to meet the body's metabolic needs. Various causes may lead to impaired external respiration and the development of acute respiratory failure [1]. In particular, obstruction of the airways may occur as a result of airway spasm, edema, inflammatory infiltration, obstruction by sputum, mucus, a foreign body, aspiration of gastric contents, blood, etc. (obstructive respiratory failure). Alveolar hypoventilation underlies the development of acute respiratory failure. Low blood oxygen levels negatively affect the body as a whole, but primarily the vital organs, primarily the brain, reducing functional capacity and leading to structural damage [2]. The severity of cerebral oxygen starvation depends on the degree of narrowing of the airways and the duration of the hypoxic period. The parietal cortex of the brain deserves special attention; damage to it leads to disturbances in response to sensory stimuli and spatial orientation.

To date, the literature contains data on changes in brain neurons due to acute circulatory hypoxia [3, 4, 5]. Information on the nature of morphological changes in neurons of the parietal cortex due to acute respiratory failure caused by partial airway obstruction (stenosis) is insufficient.

The aim is to study the changes in neurons of the parietal cortex of the rat brain during acute respiratory failure caused by partial airway obstruction and to compare them with the changes in neurons during total obstructive respiratory failure.

Materials and Methods

The study was conducted on outbred white rats (30 males, weight 240 ± 20 g), divided into 5 groups (n=6) in compliance with the requirements of Directive of the European Parliament and of the Council No. 2010/63/EU of 22.09.2010 on the protection of animals used for scientific purposes.

In rats of the experimental groups, obstructive respiratory failure was modeled by total (groups 2, 3) or partial (groups 4, 5) tracheal compression under intravenous thiopental anesthesia (40 mg/kg). Total obstructive respiratory failure was modeled by ligating the trachea below the cricoid cartilage of the larynx with a ligature for 30 minutes (group 2) and 60 minutes (group 3). Partial obstructive respiratory failure was modeled by placing a 1,5 mm plastic wire on the trachea below the cricoid cartilage of the larynx and ligating the trachea with a ligature in this area (narrowing of the lumen reached 65%) and then removing the wire with sampling of material after 30 minutes (group 4) and 60 minutes (group 5).

The control group consisted of sham-operated rats with all stages reproduced without tracheal stenosis (group 1).

The brain was quickly removed in the cold and fixed in Carnoy's fluid, followed by dehydration of the brain sections in increasing ethanol concentrations and embedding in paraffin. Frontal paraffin sections of the parietal lobe (7 μm thick) were then prepared and stained using the Nissl method. The location of the parietal cortex was determined using a stereotaxic atlas [6].

Thirty neurons in layer fifth of the parietal cortex of each animal were examined, determining their size and shape. Changes in neuron area and shape (form factor, elongation factor) were assessed using ImageWarp image analysis software (Bitflow, USA). Different neuron types were identified in histological preparations based on the degree of cytoplasmic staining and their percentage content.

The obtained quantitative continuous data were processed using nonparametric statistical methods, using the licensed computer program Statistica 10.0 for Windows (StatSoft, Inc., USA). The data are presented as Me (LQ; UQ), where Me is the median, LQ is the lower quartile value; UQ is the upper quartile value. Differences between the parameters of the control and experimental groups were considered significant at $p<0,05$ (Mann-Whitney U-test with Bonferroni correction) [7].

Results and Discussion

In the control group, the area of neurons in the parietal cortex of the rat brain was 186,6 (180,2; 191,0) μm2. They had a rounded shape (form factor – 0,9 (0,9; 09) units, elongation factor – 1,2 (1,2; 1,3) units), with distinct, smooth contours of the cellular and nuclear membranes.

In rats of the experimental groups, changes occurred in the size and shape of neurons (Table 1), as well as the degree of staining of their cytoplasm.

Groups	Indicators		
	area (μm²)	form factor (units)	elongation factor (units)
control	186,6 (180,2; 191,0)	0,9 (0,9; 0,9)	1,3 (1,2; 1,3)
total obstruction 30 min	180,6 (167,5; 188,7)	0,7* (0,7; 0,7)	2,1* (2,0; 2,2)
total obstruction 60 min	121,5*# (118,6; 133,4)	0,6* (0,6; 0,7)	2,2* (2,1; 2,3)
partial obstruction 30 min	231,4* (221,2; 240,5)	0,9 (0,9; 0,9)	1,2 (1,2; 1,3)
partial obstruction 60 min	270,6 *& (261,3; 280,4)	0,9 (0,9; 0,9)	1,2 (1,2; 1,3)

Note: – * – the differences are significant compared to the control group ($p<0,05$);
– # – the differences are significant compared to the "total obstruction 30 minutes" group ($p<0,05$);
– & – the differences are significant compared to the "partial obstruction 30 minutes" group ($p<0,05$).

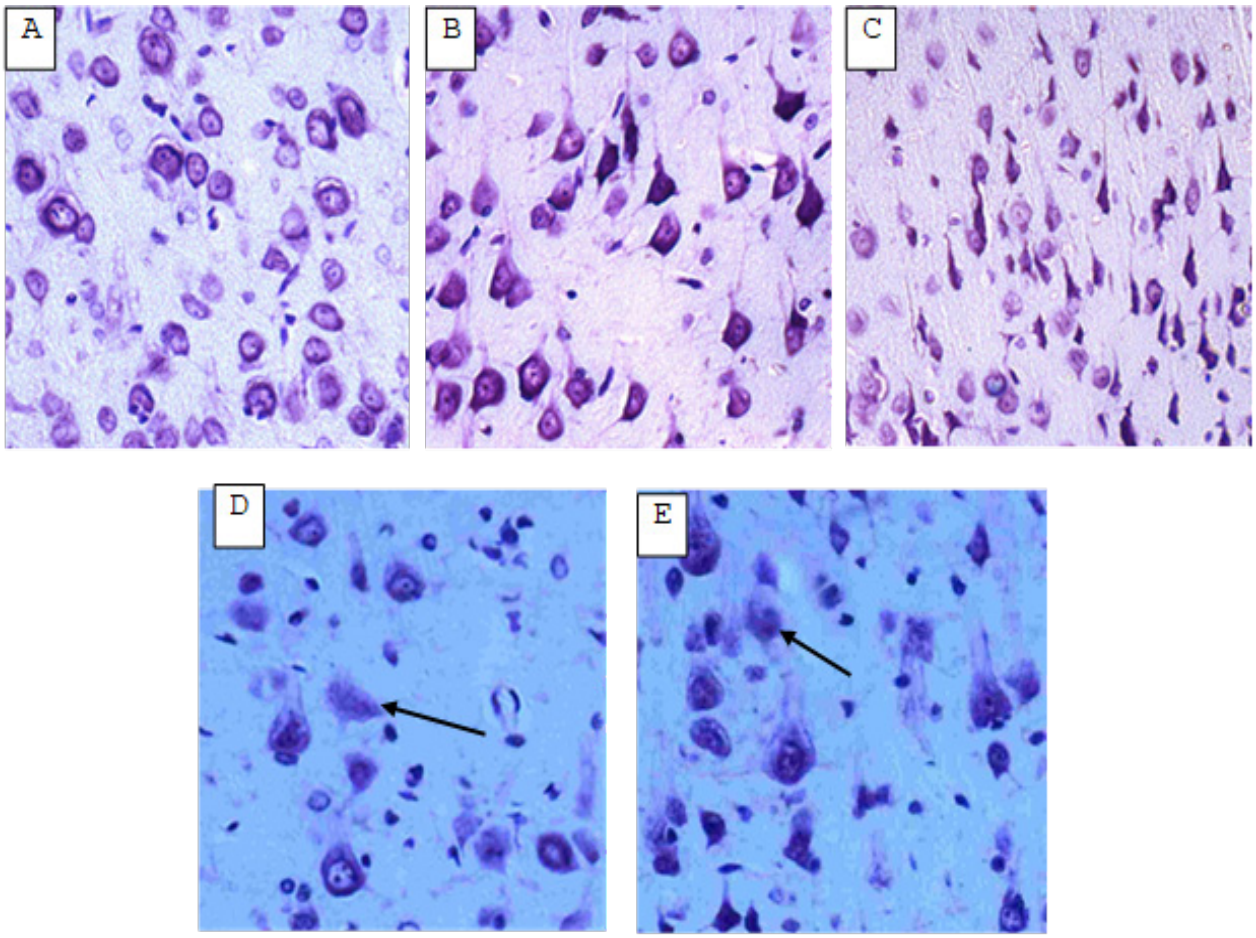
Table 1: Indicators of the size and shape of neurons in the parietal cortex of the brain of rats with total and partial obstructive respiratory failure (Me (LQ; UQ))

After 30 minutes of total tracheal obstruction, there was no change in the area of neurons in the parietal cortex ($p>0,05$). However, a change in neuronal shape was observed, with a loss of sphericity and increased perikarya elongation. This is demonstrated by a 23% decrease in form factor ($p<0,05$) and a 70% increase in elongation factor ($p<0,05$) compared to the control group.

In rats with partial tracheal obstruction, a 24% increase in neuronal area ($p<0,05$) was observed after 30 minutes of hypoxia compared to the control group. However, the neuronal form factor and elongation factor remained unchanged ($p>0,05$), indicating preservation of neuronal shape, in contrast to changes seen with total tracheal obstruction.

By the 60-minute period of total tracheal obstruction, the area of neurons in the parietal lobe cortex decreased by 35% compared to the control group ($p<0,05$) and by 33% compared to 30 minutes of total tracheal obstruction ($p<0,05$). At the same time, the form factor decreased by 31% ($p<0,05$), and the elongation factor, on the contrary, increased by 75% ($p<0,05$) compared to the control group, which reflects the presence of changes in the shape of neurons, which are similar to changes during 30 minutes of total tracheal obstruction

(p>0,05).
After 60 minutes of partial tracheal obstruction, the area of neurons increased by 45% compared to the control group (p<0,05), which is 17% more than 30 minutes of partial tracheal obstruction (p<0,05), and there was no change in the shape of neurons, as evidenced by the absence of changes in the form factor (p>0,05) and elongation factor (p>0,05).
In the control group, up to 95% of the population of neurons in the parietal cortex of the brain were normochromic cells, and the remaining neurons were hypochromic (4%) and hyperchromic (1%) cells.
In contrast to the control group, in the experimental groups with total tracheal obstruction in both study periods, hyperchromic shrunken neurons predominated: up to 75% in the group of rats with 30-minute obstruction (p<0,05) and up to 80% in the group of rats with 60-minute obstruction (p<0,05), and the remaining neurons were represented by normochromic cells - up to 25% and 20%, respectively (Fig. 1).



A – control group (normochromic neurons);
B – after 30 minutes of total obstruction (hyperchromic shrunken neurons);
C – after 60 minutes of total obstruction (hyperchromic shrunken neurons);
D – after 30 minutes of partial obstruction (hypochromic neurons with signs of swelling and shadow cells (indicated by arrow));
E – after 60 minutes of partial obstruction (hypochromic neurons with signs of swelling and shadow cells (indicated by arrow)).

Figure 1: Neurons of the parietal cortex of rats with total and partial obstructive respiratory failure. Digital micrograph. Nissl staining. Magnifying lens x20.

By 30 minutes of partial tracheal obstruction, a sharp decrease in the number of normochromic neurons (up to 10%), a slight increase in the number of hyperchromic neurons (up to 10%), as well as the appearance of a significant number of hypochromic neurons with signs of swelling (about 65%) and shadow cells (about 15%), which are known to be markers of acute oxygen deficiency of the nervous tissue, were detected.
After 60 minutes of partial tracheal obstruction, normochromic neurons were not detected, and the majority of cells were hypochromic neurons with signs of swelling (75%) and shadow cells (20%), while the number of hyperchromic neu-

rons decreased slightly (to 5%), in contrast to rats with 30 minutes of obstruction.

A study of the consequences of respiratory failure caused by complete and partial tracheal obstruction revealed changes in the size and shape of neurons in the parietal cortex, as well as the degree of cytoplasmic staining in opposite directions. These differences, we believe, are due to the different rates of progression of acute oxygen deficiency.

As a result of oxygen deficiency, the energy supply of cells is disrupted (reduced), which is accompanied, in particular, by damage to cell membranes with an increase in their permeability, as well as a violation of the transmembrane ratio of ions (K+, Ca2+, Na+, Cl-, Mg2+), which, in turn, leads to hypohydration or hyperhydration of cells as one of the mechanisms of change in neuronal chromatophilia.

In total obstructive respiratory failure, anoxic neuronal damage develops, accompanied by rapid energy deficit. As a result, neuronal shape changes manifest as loss of sphericity and increased elongation, as well as a decrease in their area (within a 60-minute anoxic period). This may be due to the release of cellular contents through the damaged plasma membrane, decreased osmotic pressure in the cytosol, and hypohydration with shrinkage of the nucleus and cell body. The above-described changes in neuronal shape and size, accompanied by cytoplasmic hyperchromia, are characteristic of coagulative necrosis.

At the same time, in partial obstructive respiratory failure, neuronal changes were manifested by an increase in their size without changes in shape in both study periods, with these changes worsening as the hypoxic period was extended to 60 minutes. This is due to a gradual decrease in cellular energy supply, the accumulation of ions (primarily Ca2+ and Na+) and products of incomplete oxidation of organic matter in the cytosol, followed by hyperosmolarity and the development of cytotoxic edema, which is characteristic of liquefactive necrosis.

Our experimental results for total airway obstruction are consistent with published data [3-7] on hypoxic damage to neurons of circulatory origin. However, our study of partial airway obstruction revealed new findings not previously described. These findings relate to neuronal swelling and hypochromia resulting from cytotoxic edema.

The obtained experimental data, when extrapolated to humans, will help assess the nature of brain damage in respiratory failure caused by airway obstruction of varying severity. This is important for developing and determining the nature of treatments for brain disorders associated with this pathology.

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