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FEATURES ACID-BASE, ELECTROLYTE AND GAS EXCHANGE OF BLOOD CHILDREN BRONCHOPULMONARY DYSPLASIA   
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**Summary:** The 620 surveys of premature aged 1 month to 36 months: 491 examinations of children diagnosed with bronchopulmonary dysplasia premature and 129 observations, which were respiratory disorders, but did not form bronchopulmonary dysplasia. A characteristic feature of gas exchange in children with bronchopulmonary dysplasia were low SpO2 (KW H (n = 620) = 105.26, rank - 10.04, p = 0.0001) and PaO2 (KW H (n = 96) = 10.85; rank - 3.29; p = 0.001), reflecting a violation of blood oxygenation in the lungs. Proved correlation SpO2 (r = -0,432; p <0,05) and PaO2 (r = -0,563; p <0,05) with the presence of permanent cyanosis at rest. Cyanosis nasal triangle and peripheral cyanosis during anxiety did not correlate with SpO2 and PaO2. Proved lack of strength marker for bronchopulmonary dysplasia PetCO2 in the air, but increased PaCO2 was a characteristic feature of BPD (KW H (n = 96) = 4.04, rank - 4.04, p = 0.044). For children with BPD were characterized by violations of the acid-base balance of blood, despite the changes in the respiratory component of the regulation of respiration (PaSO2), indicating a compensation of respiratory regulation of acid-base balance metabolic component. Prognostic marker of unfavorable prognosis BPD were SpO2, PaO2, the ventilation-perfusion ratio and CO2 level of K + in the blood of patients.

**Keywords:** bronchopulmonary dysplasia, acid-base status of blood electrolyte status of blood gas exchange.

The severity of bronchopulmonary dysplasia (BPD) is defined depend of oxygen patients [1,2]. Patients with moderate and severe BPD are not able to postnatal period to the effective diffusion of gases in FiO2 21% [3]. In these children may develop chronic hypoxia organs and tissues, which usually leads to the activation phosphofructokinase, increasing the capacity of anaerobic glycolysis, accumulation of lactic acid and pyroracemic acid [4]. A typical reaction of mast cells and platelets on the development of hypoxia and acidosis considered their degranulation with excessive release of proinflammatory mediators increase the permeability of biological membranes, activation of lipid peroxidation. As a result of hypoxia occurs disruption of cytoplasmic, lysosome, mitochondrial membranes. In response to the disruption of excess activated by cytokines course of regeneration, which are able to maintain chronic inflammation in the transformation of fibrosis, such as transforming growth factor (TGFβ1) and vascular endothelial growth factor (VEGF) [5,6,7]. Thus, hypoxia and acidosis are not only indicators of disease severity may be, and factors of unfavorable prognosis of bronchopulmonary dysplasia. However, the influence of parameters of acid-base balance of blood on the remodeling of the bronchi and lung tissue and the relative weight of hypoxia and acidosis in the forecast bronchopulmonary dysplasia, are still not clearly defined.

Objective: improving diagnostic prediction of bronchopulmonary dysplasia as a marker by determining the acid-base, electrolyte and blood gas exchange conditions of children with bronchopulmonary dysplasia.

Materials and Methods: The study was conducted at the Department of Pediatrics and Neonatology №1 Kharkiv National Medical University (head of department - G.S.Senatorova) in the regional center for diagnosis and treatment of bronchopulmonary dysplasia in children Kharkiv Oblast Children's Hospital (chief doctor – G.R. Muratov). For monitoring the percentage of oxyhemoglobin in arterial blood we used pulse oximetry - a non-invasive method that meets ensure complete safety inspection, its comfort, efficiency and high reliability of measurement results. Рulse oximetry сonducted for children with bronchopulmonary dysplasia in a period of remission. Patients comparison group study was conducted in the absence of disease. In patients with mild BPD severity and examined рulse oximetry comparison group performed at rest in FiO2 21%. In children with severe and moderate severe BPD severity saturation of hemoglobin in arterial blood oxygen was measured within 5 minutes after transfer to breathing room air. Analyzed the average of several measurements. in the fall of SpO2 below 88% of a child with BPD receiving respiratory support early. An annual 620 SpO2 measurement in patients 1 to 36 months corrected age. Among them, 491 (79,1 ± 1,63%) of observation patients of the main group and 129 (20,8 ± 1,63%) children surveyed group of comparisons. The content of carbon dioxide in the air measured by carbonometry. Indicators and рulse oximetry carbonometry i were compared with the results of the study of partial pressure of oxygen and carbon dioxide in the alveolar blood. Pearson Criterion corrected Yetesa showed no difference in the distribution of the sample when compared to a sampled (χ2 = 4,04; p = 0.044). In a sample of children were not with repeated measurements. Sample structure consistent with the general population in the form (χ2 = 3,64; p = 0.034) and severity of BPD (χ2 = 3,9; p = 0.039). So prove full compliance with the general population sample. Comparison between the main group and the comparison group was performed using accounting methods independent variables. Statistical analysis of data was performed using the «Statistica-6".

Results and discussion: In children, the main group SpO2 was 93,23 ± 0,2%, which was lower than the comparison group of children (95,48 ± 0,7%; p = 0.01). In the general population SpO2 level was a marker in relation to bronchopulmonary dysplasia (KW H (n = 620) = 105.26, rank - 10.04, p = 0.0001). It is proved that SpO2 dependent on disease severity (r = 0,811; p <0,05). Low SpO2 was a predictor of mortality from bronchopulmonary dysplasia (KW H (n = 491) = 65.25, rank - 7.9, p = 0.0001).

Results of the analysis of respiratory gases in the alveolar blood of patients of the main group and the comparison group are presented in table.

Table   
Results of the study of gases, acid-base status and electrolytes alveolar blood in children with BPD (study group; n = 85) and patients who were born prematurely, had respiratory disorders in the early neonatal period, but not formed BPD (comparison group; n = 11 )

|  |  |  |
| --- | --- | --- |
| Indicators | Main group  n=85 | Group of comparisons n=11 |
| Ме [Lq; Uq] | Ме [Lq; Uq] |
| РaO2, мм Hg | 34 [24; 40] | 45 [40; 48] |
| РaСO2, мм Hg | 42 [35,1; 53,7] | 34,5 [33,5; 37,3] |
| pH, од. | 7,38 [7,31; 7,42] | 7,4 [38; 42] |
| BE, mmol/l | -2,1 [-3,6; -0,7] | -1,1 [-1,65; +0,25] |
| Ht, % | 35 [29; 40] | 40 [29; 40] |
| Na+, mmol/l | 132,4 [122,1; 135] | 132 [132; 133] |
| К+, mmol/l | 4,2 [3,9; 5,3] | 4 [3,8; 4] |
| Cl -, mmol/l | 101 [98,1; 112] | 100 [99; 106] |
| Са++, mmol/l | 1,1 [1,0; 1,2] | 1,1 [0,93; 1,04] |
| Mg++, mmol/l | 1,08 [0,96; 1,25] | 1,25 [1,0; 1,4] |
| P +, mmol/l | 1,06 [1,0; 1,2] | 1,02 [0,95; 1,15] |

Me [Lq; Uq] \* - Median and Quartile within the same group

Low partial pressure of oxygen in alveolar blood in the main group of children was a characteristic feature of bronchopulmonary dysplasia (KW H (n = 96) = 10.85, rank - 3.29, p = 0.001). RaO2 depended on the severity of BPD (KW H (n = 96) = 26.46, p = 0.001) and did not depend on the form of BPD (KW H (n = 96) = 3.51, p = 0.173). Low partial pressure of oxygen in the blood of patients with BPD is likely due to diffusion disorders due to thickening of the membrane alveole on a background of fibrosis, shunting of blood in the lungs, which is important for understanding the pathological processes in the respiratory and cardiovascular with BPD.

Because clinical sign of low partial pressure of oxygen in the blood was cyanosis us Correlation analysis of parameters of cyanosis content of O2 in arterial blood measured by рulse oximetry and analysis of gas exchange in arterial blood. Proven SpO2 and PaO2 correlated with the presence of permanent cyanosis at rest (r = -, 432; r = - 0,563; p <0,05). However, cyanosis nasal triangle and peripheral cyanosis during anxiety did not correlate with SpO2 (r = 0,02; r = - 0,247; p> 0,05). PaO2 was independent of the presence of cyanosis nasal triangle (r = -0,096; p> 0,05).

This could be due to the presence in patients of stages of hypoxia, when vision is not recorded cyanosis (cyanosis subjective evaluation), or due to dysregulation of skin microcirculation vessel (peripheral vasoconstriction), which is characterized by children who were born prematurely. Inconsistency SpO2 levels affect by peripheral cyanosis in children with bronchopulmonary dysplasia suggests the possibility of microcirculation disorders in this category of patients. Thus, the findings confirm the need to study the cardiovascular system, all patients with BPD who have an imbalance of color, discovered during the inspection and performance SpO2 and PaO2.

The content of carbon dioxide in the air of children of main group (n = 85) was 38 [30.4; 49.4] mm Hg. Patients comparison group (n = 11) had PetCO2 equal to 38 [38; 39] mm Hg. The analysis demonstrated the absence of marker forces partial pressure of carbon dioxide in the air for bronchopulmonary dysplasia (KW H (n = 96) = 1.36, p = 0.253). The average level PetCO2 in both groups entered the threshold requirements (36-43mm Hg). The high partial pressure of carbon dioxide in the alveolar blood marker was a sign regarding BPD (KW H (n = 96) = 4.04, rank - 4.04, p = 0.044), but with less weight than low rank PaO2.

The difference PaCO2 of PetCO2 core group of children was 0.2 [-11.1; 11.3] in the comparison group - -3.5 [-4.0; -1.5] That the two groups did not exceed 5 mm and allowed adequate to assume the ventilation-perfusion ratio. However, attention is drawn to significant differences in values ​​ quartiles of main group and significant dispersion parameters D [239], indicating heterogeneity ratio PaSO2 to PetCO2 in patients with BPD.

In children, the main group the ventilation-perfusion ratio of CO 2 was significantly more 0,8 (KW H (n = 96) = 8.27, rank - 2.1, p = 0.044). Most likely, this phenomenon is caused by increased alveolar dead space and possible shunting of blood through the redistribution of ventilation and discharge of venous blood during increased pressure in the pulmonary artery in patients with bronchopulmonary dysplasia. Differences between perfusion ratio of CO2 in children with various forms of BPD have been identified (KW H (n = 96) = 3.34, p = 0.067). However, increasing the ventilation-perfusion ratio of CO2 was correlated with the severity of BPD (KW H (n = 96) = 7.95, p = 0.01) and was predictive of mortality of patients (KW H (n = 96) = 9.1, p = 0.027). Results of arterial blood pH did not differ in the intervention group and the comparison group (KW H (n = 96) = 0.0144, rank - 0.38, p = 0.78). Index in both groups was within the normal range (-3 to 2.5) and was not a characteristic feature of BPD (KW H (n = 96) = 1.1, rank - 1.0, p = 0.029).

In the examined fluid and electrolyte status was assessed by determining the blood hematocrit and blood electrolytes (Table.). The distributions of fluid and electrolyte status venous blood marker of the degree of significance in relation to bronchopulmonary dysplasia, the results of analysis of variance for Kraksel Wallis presented in fig. 2.

Fig. 2. The distributions of fluid and electrolyte status of venous blood in the degree of significance in relation to bronchopulmonary dysplasia, the results of analysis of variance for Kraksel Wallis (n = 96)

The relatively high K + was a characteristic feature of bronchopulmonary dysplasia KW H (n = 96) = 6.6; rank - 2.57; p = 0.01, we explain the redistribution of potassium between the intra- and extracellular sectors, against hypoxia. This proven correlation level of K + Blood РaO2 (r = 0,315; p <0,05). The level of potassium in the blood is also dependent on the severity of bronchopulmonary dysplasia (r = 0,443; p <0,05).

Conclusions:   
1. A characteristic feature of gas exchange in children with bronchopulmonary dysplasia have a low percentage of oxyhemoglobin and partial pressure of oxygen in arterial blood, reflecting violations of blood oxygenation in the lungs.   
2. proven correlation between SpO2 and PaO2 with the presence of permanent cyanosis at rest. In addition, in patients with bronchopulmonary dysplasia cyanosis nasal triangle and peripheral cyanosis during anxiety did not correlate with SpO2. PaO2, which we believe was due to dysregulation of microcirculation vessel of the skin. Dissonance indicators SpO2, PaO2 and cyanosis suggests the probability microcirculation disorders in patients with BPD.

3. The lack of proven strength marker for bronchopulmonary dysplasia partial pressure of carbon dioxide in the air that vydyhalosya. However, the increased partial pressure of carbon dioxide in the alveolar blood was a characteristic feature of BPD, which is possible due to the greater sensitivity of measurement of partial pressure of carbon dioxide in the blood than in the air that vydyhalosya in patients with bronchopulmonary dysplasia.   
4. For children with bronchopulmonary dysplasia were characterized by violations of the acid-base balance of blood, despite the changes in the respiratory component of the regulation of respiration (RaSO2), indicating a compensation of respiratory regulation of acid-base balance metabolic component

5. The fluid and electrolyte indicators marker was increasing K + blood, which we regard as a redistribution of potassium between the intra- and outcells sectors, against hypoxia.   
6. With respect to prognosis important parameters percentage of oxyhemoglobin, the partial pressure of oxygen in arterial blood, the ventilation-perfusion ratio of CO2 and potassium levels in the blood of patients with bronchopulmonary dysplasia..