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Clinical-neurophysiological and Morphological Studies of Children with Consequences of Hypoxic

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ABSTRACT

Objective: *The problem of perinatal brain injury of hypoxic-ischemic genesis remains a critical issue, with a focus on its high mortality rates, significant disability, and impact on neuropsychic and somatic development in children. These injuries lead to difficulties in social adaptation, diagnosis, and treatment.* ***Methods:*** *The study aims to explore current therapeutic measures applied to children with perinatal hypoxic-ischemic damage to the central nervous system (CNS), evaluating their effectiveness in addressing these challenges. Various medical and social factors are examined to understand the limitations in the treatment of such conditions.* ***Results:*** *It is found that the existing therapeutic interventions for children suffering from perinatal hypoxic-ischemic CNS damage are not always sufficiently effective. The analysis highlights the need for better approaches to diagnosis, treatment, and overall care to improve the quality of life and development for affected children.* ***Novelty:*** *This study contributes new insights into the current state of perinatal brain injury treatment and diagnosis, emphasizing the importance of developing more effective therapeutic strategies to combat the long-term effects of hypoxic-ischemic damage in children. The findings suggest areas for further research to address these challenges comprehensively.*

INTRODUCTION

The severity of the medical and social consequences of perinatal hypoxic-ischemic pathology of the central nervous system determines the possibilities of limiting the center of damage by using new approaches in the study of the properties of pathogenesis and mechanisms for restoring damaged tissue of the central nervous system, determining the degree of morphological changes at the molecular cell level and maintaining the viability of and their functional activity, as well as the development of pre-clinical, diagnostic and therapeutic measures early, including during the period of intensive maturation and development of the brain [1], [2].

However, there are certain difficulties in the fundamental research of the pathology of the central nervous system of a newborn:

1. Biological (anatomical and physiological characteristics) of children;
2. Ethical (the impossibility of conducting research and obtaining biopsy material in children, the impossibility of drug testing).

Live models can be used in vivo to solve the above problems, taking into account the characteristics of the developing brain, as well as the compatibility of animal models with human age [3], [4], [5], [6].

RESEARCH METHOD

The study of various aspects of perinatal hypoxic-ischemic damage to the central nervous system is carried out in primates, pigs, sheep, rabbits and rodents [7], [8], [9], [10], [11]. Given the enormous similarities between the blood supply to the brain and the neurobiology of rodents and high mammals, as well as the practical and economic advantages of experimental models of hypoxic-ischemic damage to the central nervous system, models using rodents (mice, rats, guinea pigs) are now more commonly used.compared to models that attract large animals [12], [13], [14].

The leading causes of Perinatal encephalopathy are fetoplasental and uteroplacental blood flow disorders, respiratory and heart failure, and pathogenesis leading joints brain hypoxia and ischemia, reperfusion and excitotoxicity phenomenon, energy balance, and cell-level oxidative stress. The result of these processes IS cell death of the central nervous system [15]. In this case, pathomorphological changes in brain tissue (periventricular leukomalacia, intraventricular and periventricular bleeding, necrosis of the brain substance, ischemic and hemorrhagic stroke) depend on the nature and duration of the pathological effect, as well as the maturity of the central nervous system.

RESULTS AND DISCUSSION

The clinical picture of perinatal hypoxic-ischemic lesions of the central nervous system in children is expressed by frequent clinical syndromes (depression, agitation, convulsive, minimal brain dysfunction) [16], but the result of perinatal encephalopathy in both humans and laboratory animals may differ from laboratory animals, despite the development of neuropsychiatric insufficiency. Therefore, when modeling hypoxic-ischemic damage of the central nervous system, it is necessary to achieve not only the similarity of clinical manifestations, but also the maximum specificity of the main mechanisms of the development of hypoxic-ischemic injury in humans and experimental animals [3].

As part of this review, the main methods for modeling global brain hypoxia-ischemia in laboratory animals were analyzed.

In general, experimental models of cerebral hypoxia-ischemia can be described as global, focal, and multifocal. Their main difference is that with global hypoxyishemia, there is a decrease in cerebral blood flow throughout the brain (total global hypoxia-ischemia) or in most of it (incomplete global hypoxia-ischemia); with focal, there is a decrease in blood flow to different but certain regions of the brain. The type and age of laboratory animals are very important in modeling hypoxia-ischemia. Thus, it is necessary to take into account that the age-related morphofunctional Organization of the brain of laboratory animals corresponds to the brain of a newborn [17].

Depending on the approach to modeling cerebral hypoxia-ischemia, the classification of experimental models can be expressed as follows.

1. Global (general) cerebral hypoxia-ischemia.
2. Complete.

3. Put a tourniquet or cuff around the neck.
4. Decapitation.
5. Cardiac arrest (ventricular fibrillation).
6. Blockage of the aorta or vena cava.
7. Hypoxic anoxia of newborns.
8. Intrauterine ischemia of the fetus (anoxia).
9. Incomplete cerebral hypoxia-ischemia.
10. Hypoxic ischemia.
11. Bipolar occlusion and hypotension of the common carotid arteries.
12. Four-vein occlusion (common carotid and vertebral arteries).
13. Focal cerebral hypoxia-ischemia.
14. Occlusion of the middle cerebral artery.
15. Endothelin-1-induced narrowing of arteries and veins.
16. Embolization (macrospheres).
17. Multifocal brain hypoxia-ischemia.
18. Thromboembolism.
19. Embolization (microspheres).
20. Photo thrombosis.

Given the etiopathogenetic properties of perinatal damage to the central nervous system, the following are key models of global cerebral ischemia live in vivo.

Put a tourniquet or cuff around the neck. One of the first models of Global ischemia is the use of neck cuff in large animals. On the basis of this model, the consequences of acute short-term cessation of blood circulation in the brain, the characteristics of animal behavioral reactions and neurological disorders during the early and late recovery period, as well as the effects of long-term hypoxia (more than 8 minutes) were studied [18]. Some time later, the technique of applying a neck cuff with modifications (arterial hypotension) was used in primates [19] and pigs. In general, this model is best suited for studying the features of circulatory disorders of the hippocampus and brain stem. The main disadvantage of this technique is that the vertebral arteries do not undergo complete occlusion because they pass within the vertebrae. Thus, in order to achieve a complete cessation of blood flow, it is necessary to surgically separate the arteries and then connect them. In small laboratory animals, this method is practically not used due to the anatomical and physiological characteristics of their structure and the development of various complications (vertebrae, spinal cord damage, compression of the vagus nerve).

Decapitation. Hypoxic-ischemic lesion model based on decapitation proposal in the 60s of the XX century qilingan.va it is now rarely used, primarily to study the pathogenesis of global ischemia, as well as the mechanisms of action of various fast-acting drugs under development [20], [21], [22]. However, it is impossible to study the effects of various drug modulators during decapitation, as well as the characteristics of local blood supply disorders and reperfusion effects. Nevertheless, after decapitation, the

brain can be stored for a long time in a frozen or homogeneous state for further biochemical studies.

Cardiac arrest (ventricular fibrillation). This method is usually used to mimic the clinical condition of cardiac arrest. It was he who was used by many researchers to study the effectiveness of cardiovascular resuscitation and its effects on the central nervous system [23], [24], [25], [26], [27]. According to the results of studies in this model, the rules of cardiopulmonary resuscitation are applied to practical medicine, including the reception of a trip three times. The Model is considered technically very complex, as well as economically expensive, as a result of which it is mainly used in large animals. However, mice have techniques to model global hypoxia-ischemia through cardiac arrest, which is acceptable for studying changes in the hippocampus, anterior and posterior shells [28]. Using this model, research continues on the molecular properties of the pathogenesis of hypoxic-ischemic damage to the central nervous system, as well as new drugs are being tested to treat post-toxic encephalopathy [29], [29], [30], [31].

Blockage of the aorta or vena cava. As a result of cardiac arrest, ischemia of the whole organism and its involvement in the pathological process of all organs and systems is observed, which aggravates brain damage and does not allow assessing the effect of hypoxia-ischemia on the central nervous system. Various variants of the global hypoxia-ischemia model based on the occlusion of large vessels, primarily the aorta and/or vena cava, have been proposed, allowing the non-cerebral appearance of ischemia to be minimized [32], [33], [34], [35], [36], [37], [38], [39], [40].

Using the technique of large vessel occlusion, hyperbaric oxygen therapy performed in the early postoperative period was found to have a positive therapeutic effect: accelerating neurological recovery and improving survival even after fifteen minutes of global ischemia. The main disadvantage of the occlusion of large vessels is the operational complexity and high mortality of experimental animals [41], [42], [43], [44], [45], [46], [47], [48], [49].

A variant of hypoxic ischemia is the double occlusion of the common carotid arteries and the way rats are placed in a low-oxygen atmosphere [50], [51]. In general, the hypoxic ischemia model combines elements of hypoxia and ischemia, consistent with the etiopathogenesis of perinatal hypoxic-ischemic injury of the central nervous system. The model is easy to obtain, has good reproducibility of morphological changes in brain tissue. Typically, hypoxic ischemia is used for short-term experiments to study the effectiveness of neuroprotectors in the early stages after hypoxic-ischemic exposure.

However, this model does not replicate temporal cerebral circulatory disorders, as humans do not have the midbrain artery damage observed in perinatal hypoxic-ischemic injury of the central nervous system, and therefore are not fully adequate [52]. In addition, the peculiarities of blood supply to the brain of rats (the presence of two vertebral arteries) do not allow effective initiation of pathological changes, and surgery itself can lead to partial brain damage. Another disadvantage of the model is the large

volume of brain damage and high animal mortality in the first hours of modeling pathology.

Bilateral occlusion and hypotension of the common carotid arteries. The peculiarities of the technique consist in the implementation of a double occlusion of the common carotid arteries, simultaneously with a decrease in blood pressure in different ways (controlled bleeding or medical hypotension) [53], [54], [55]. In this case, damage to the hippocampus is mainly observed. Double-vein occlusion with hypotension is optimal for studying the consequences of previous hypoxia-ischemia, the effects of reperfusion and the characteristics of the recovery period. Using this model, the properties of phospholipid and energy metabolism, the effects of neurotransmitters, morphological changes due to ischemia, the influence of various temperature regimes on the results of perinatal hypoxic-ischemic damage of the central nervous system were studied [20], [56]. The difficulty is determined by the need to accurately control systemic blood pressure, the duration of ischemia, body temperature, the use of a certain anesthesia regimen, as well as proper postoperative care. An important drawback of double-vein occlusion with hypotension is that with systemic hypotension, general metabolic disorders develop in vital organs, including the brain.

Four-vein occlusion (common carotid and vertebral) arteries. The technique of performing this model consists in the simultaneous clogging of vertebrates and common carotid arteries on both sides. Occlusion can be permanent or temporary. Typically, this model is implemented in two stages and is technically more complex than double-vein occlusion. Four-vein occlusion is best suited for studying damage to the cortex, trunk, hippocampus, brain striatum, metabolic properties when performing anesthesia, as well as the effects of medicinal substances. The Model allows you to check long results, behavioral reactions and memory impairment. The main disadvantages of four-vein occlusion are access through deep traumatic surgery, two steps in model execution, the possibility of incomplete occlusion of the vertebral arteries, high risk of bleeding and high animal mortality, and low recurrence of results [57], [58], [59].

The new model has a number of advantages: the modeling procedure is simpler, cheaper and faster, less invasive and less traumatic for animals. In this case, the main clinical, biochemical, morphological and histological changes correspond to hypoxic-ischemic lesions of the central nervous system in humans.

CONCLUSION

Fundamental Finding : The review highlights various modifications of models used to create global brain hypoxia-ischemia, each characterized by its complexity, result repetition, and the selective damage to specific brain areas. These models are critical for exploring mechanisms of disease development, repair, neuroprotection, prevention, diagnostics, and treatment methods, including molecular and genetic approaches. **Implication :** The findings emphasize the importance of choosing the most suitable experimental model for studying perinatal hypoxic-ischemic brain damage. This has

significant implications for advancing research on potential therapies, diagnostics, and neuroprotection methods in the treatment of central nervous system damage caused by perinatal hypoxia-ischemia. **Limitation :** A key limitation noted is that while in vivo data offers valuable insights into neuroprotection and therapy, these findings cannot be directly extrapolated to human conditions. The current models do not perfectly replicate human pathology, which limits the applicability of results to real-world clinical scenarios. **Future Research :** Future research should focus on improving the existing experimental models to better mimic human brain pathology, thereby enhancing the translational value of the findings. Additionally, exploring new therapeutic approaches and further refining diagnostics and neuroprotective methods will be essential to improving outcomes for individuals affected by perinatal hypoxic-ischemic brain damage.

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