MINISTRY OF HIGHER EDUCATION, SCIENCE AND INNOVATIONS OF THE REPUBLIC OF UZBEKISTAN

MINISTRY OF HEALTH OF THE REPUBLIC OF UZBEKISTAN

SAMARKAND STATE MEDICAL UNIVERSITY

SHARIPOV RUSTAM KHAITOVICH

PECULIARITIES OF THE COURSE OF RESPIRATORY PATHOLOGY IN CHILDREN WITH PERINATAL CNS LESIONS

(monograph)

SAMARKAND 2025

UDK:

AUTHOR:

Sharipov Rustam Khaitovich - medicine sciences Doctor, Samarkand State Medical University postgraduate education faculty Pediatrics and Neonatology associate professor

REVIEWERS:

Akhmadeeva E.T. - Professor of the Department of Hospital Pediatrics, Bashkir State Medical University, Doctor of Medical Sciences.

Lim M.V. - Doctor of Sciences Department 1 of Pediatrics and Neonatology Samarkand State Medical University, Associate Professor

Since the issue of prenatal nervous system injury is becoming increasingly significant due to the rise in the prevalence of this disorder, the monograph focuses on the issue that has been garnering particular attention lately.

The most common condition affecting a newborn's neurological system is hypoxic brain alterations.

More than 60% of all pediatric nervous system disorders are caused by perinatal brain injury, which also plays a direct role in the emergence of conditions including convulsions, cerebral palsy, and minor brain dysfunction. Etiopathogenesis, categorization, clinic, diagnosis, and therapy of the effects of prenatal nervous system injury are among the topics covered in the monograph.

The facts and rules based on evidence about this disease, as well as their own views based on the results of modern research methods, are covered. The proposed monograph presents the results of many years of observation of children with perinatal damage to the Central Nervous System. The conducted analyzes suggest a diagnostic algorithm for the examination and treatment of children with the consequences of perinatal damage to the nervous system.

The monograph is intended for students of higher medical institutions, masters, clinical residents, cadets of the Faculty of Postgraduate Education and pediatricians.

The monograph was discussed and approved at the meeting of the Scientific Council of Samarkand State Medical University and recommended for publication.

«_» _____ 2025, protocol No. ____

Secretary of the Scientific Council: ***U.U. Ochilov***

TABLE OF CONTENTS

LIST OF ABBREVIATIONS

CPDNS – consequences of perinatal damage to the nervous system

CNS – central nervous system

BP – blood pressure

K – potassium

LPO – lipid peroxidation

DNA – deoxyhydronucleic acid

EA – non-esterified fatty acids

MDA – malondialdehyde when accumulated in membranes

ExoEG – echoencephalography

ARVI – acute respiratory viral infection

IDA – iron deficiency anemia

OVERVIEW

The in-depth investigation of perinatal lesions of the central nervous system has received particular attention in recent years, and this is not by chance, as the issue of perinatal nervous system lesions is still relevant, particularly given the rise in perinatal nervous system lesions. The most common condition affecting a newborn's neurological system is hypoxic brain alterations.More than 60% of all pediatric nervous system disorders are caused by perinatal brain injury, which also plays a direct role in the emergence of conditions including convulsions, cerebral palsy, and minor brain dysfunction.

Fetal asphyxia is linked to the majority of perinatal diseases. Changes in cerebral hemodynamics accompany prenatal hypoxia and asphyxia, and this is now thought to be the most compelling theory for the pathophysiology of the effects of perinatal nervous system injury in neonates. These kids need extra care because they are more prone to respiratory illnesses than kids in good health. Consequently, this results in a poor prognosis, higher treatment costs, and reduced growth and development in children.

Based on the aforementioned, antihypoxic neurotrophic treatment is required in the early posthypoxic phase to restore normal cerebral and overall blood circulation. Thus, Vincamine, a medication with vascular-regulating, antihypoxic, and neurometabolic effects, was utilized judiciously. Being a natural herb, Vincamine is safe to use over an extended period of time. Additionally, Vincamine was prescribed based on the lack of adverse effects and the drug's strong tolerance by patients who experienced no problems.

CHAPTER I: REVIEW OF LITERATURE

1.1. Etiopathogenetic treatment concepts and risk factors and hemodynamic problems in babies with repercussions of prenatal nervous system injury.

In the framework of prenatal disorders, perinatal pathologies of the central nervous system have a prominent position and have an impact on an individual's future existence (2,67,122). The incidence of children with central nervous system perinatal lesions has been rising in recent years. Numerous later psychoneurological and somatic disorders may arise, contingent on the extent and trajectory of the damage (1, 7, 87, 122, 216). Hypoxic-ischemic, metabolic, toxic, viral, and traumatic factors are some of the causes of the development of disease as a result of perinatal injury to the nervous system that occurs before, during, and after pregnancy. The main pathogenetic causes are hemodynamic abnormalities and hypoxia to varied degrees (35,79,189).

The prenatal, intranatal, and early neonatal phases are all included in the perinatal period. The start of labor marks the conclusion of the prenatal period, which starts at 22 weeks of fetal development (31, 45,129, 184). The time between the start of labor and the child's birth is referred to as the intranatal phase. The actual neonatal era is separated into two phases: early neonatal, which spans the first week of a child's life, and late neonatal, which spans the eighth to the twenty-eighth day of life. The category of children at risk of cerebral palsy includes those who have been diagnosed with hydrocephalus and hypertensive syndrome as a result of perinatal lesions of the central nervous system during their first year of life (22, 65, 129,2 11).

B.N. Klosovsky asserts that the embryo and fetus undergo three nutritional phases that are essential for the growth and creation of the brain: blood (beginning at six months of pregnancy), cerebrospinal fluid (two to three months of pregnancy), and cerebrospinal fluid and blood (four to five months of pregnancy). An examination of the central nervous system's nutritional phases reveals that the brain's oxygen saturation value rises as erythrocyte and hemoglobin function increases. Hypoxia can easily appear in the fetus beginning in the sixth month of

intrauterine development, when blood nutrition takes precedence. The two most common causes of prenatal nervous system dysfunction are suffocation and intrauterine brain trauma.

This distinction is critical because intracerebral bleeding typically occurs in conjunction with a brain oxygen shortage, whereas cerebral hypoxia is typically characterized by small-point perivascular hemorrhages. Regardless of how it develops, fetal hypoxia is the most prevalent cause of mortality in the early neonatal period and one of the main causes of preterm delivery. It should be mentioned that perinatal central nervous system injury in preterm newborns presents with unusual symptoms and an unclear clinical picture. This complicates diagnosis, particularly in the early stages of a child's life (10, 43,137,218).

There are five types of intrauterine hypoxia:

- hypoxic - occurs due to insufficient oxygen saturation of the fetus (the most common cause is placental pathology);

- hemic - leads to a decrease in oxygen in the tissues due to a decrease in hemoglobin in the blood;

- circulatory - due to a slowdown in blood flow;

- tissue type - occurs as a result of impaired oxidative processes in tissues due to enzyme deficiency;

- cellular.

The main factor leading to damage to the central nervous system of the fetus in the antenatal period is a violation of fetoplacental blood flow. Fetoplacental insufficiency leads to fetal hypoxemia, which, in turn, is accompanied by the activation of compensatory mechanisms and redistribution of blood with improved blood supply to the vital organs of the fetus. At the same time, the vessels of the fetal brain expand and its blood supply increases (5, 38,76,163).

However, if the increased blood flow cannot compensate for the lack of oxygen, brain cells undergo degenerative changes, some cells die. Vascular elements also do not remain intact: the formation of veins is disrupted, arterial anastomoses are delayed in areas with collateral blood supply, the intensity of

capillary formation decreases, they become empty and atrophy under conditions of hypoxia (14, 98, 176, 217).

In the walls of most cerebral vessels, there is an underdevelopment of the muscle layer and connective tissue, swelling of the endothelium, and increased vascular permeability. The migration of glial elements to the cortical plate is disrupted, glial cells are also damaged, as it is known that glia perform a trophic function, forming connections between capillaries and neurons. Such changes affect not only the brain, but also the spinal cord. Under conditions of hypoxia, degenerative processes occur in the neurons of the spinal cord, the capillary network decreases, and the venous network is disorganized (6, 96, 136, 177, 212).

All these changes observed in chronic intrauterine hypoxia lead to a sharp decrease in the compensatory ability of the central nervous system and subsequently become the basis for functional and morphological changes in the vessels of the central nervous system, stress during childbirth, and secondary disorders of cerebral hemodynamics in conditions of active postnatal development of the child (17, 72, 90, 212).

Childbirth complications during the intranatal period might result in hypoxic-traumatic variables that decrease cerebral blood flow and cause intracerebral hemorrhages in the baby.

All adverse aspects of the labor process that unavoidably have an impact on the kid are considered intranatal harmful factors:

The child's extended stay in the breech position

The use of stimulation during labor because the expectant lady was weak or absent; the labor act; inadequate birth canal dilatation; quick labor; manual labor techniques; cesarean section; wrapping of the fetus in the umbilical cord; and a huge fetus.

The risk group for intranatal damage includes premature babies and children with low or very large body weight. The first adaption stage, when pulmonary respiration is produced and hemodynamics are restored, is particularly crucial for the future survival of the kid. The transmitted hypoxia leads to a breach of the

adaptation of the child's body to extrauterine life. The central nervous system's blood supply deteriorates as a result of systemic hypotension, disturbed homeostasis, and injury to key organs (heart, kidneys, and lungs) caused by hypoxia and metabolic abnormalities during hypoxia (13,77, 83,159, 188).

The ability to autoregulate cerebral blood flow is also lost in children who have experienced hypoxia; in other words, cerebral blood flow passively follows changes in central hemodynamics, and these changes can result in both increased cerebral ischemia and postpartum intracerebral hemorrhage. Thus, the central nervous system's blood flow deteriorates as a result of pathological circumstances such reduced cardiac and pulmonary function, severe anemia, adrenal insufficiency, polycythemia, and sepsis that causes arterial hypotension in the postpartum period (16, 44, 97, 181, 212).

As the child grows, there is intense growth of nervous tissue, but the damaged nerve cells cannot handle the increasing load, so the recovery processes are disrupted and the number of dead cells increases again. Vasospasm, which is linked to the accumulation of calcium in the vascular endothelium during hypoxia, also contributes to a deterioration in blood flow in the central nervous system. The mechanism of disruption of recovery processes is also determined by the discrepancy between the needs of developing nerve cells and the level of blood supply to the central nervous system in conditions of underdevelopment of glia, blood vessels, and capillaries (8, 50, 96, 139,2 18).

As a result, the healing phase alternates between times when the repair or destruction processes are more prevalent. Additionally, previously injured cells take part in the destruction process, neuroglia are also harmed, and newly produced capillaries atrophy and become loose. Will systemic alterations in the central nervous system eventually recover? Windle (1971) reported that even after 8–10 years, a progressive loss of nerve cells was seen in monkeys subjected to intranatal hypoxia. Mature animals' spines also showed changes, including an increase in capillary dilatation and a decrease in the differentiation and tortuous appearance of

veins. These changes were thought to be indicators of impaired venous flow and a decline in the spinal cord's capacity to compensate (22, 54, 176, 219).

Considering the postnatal period, it can be noted that the following factors play the greatest role in the genesis of central nervous system damage:

1) neuroinfections

2) injuries

The neonatal period of human life is very rich in biological, medical, social and many other aspects of adaptation to later life, which are an indicator of the socio-biological health of society, in particular, its most active part - young people. The source of many chronic diseases of childhood and adulthood (such as bronchopulmonary, neurological and mental diseases, diseases of the gastrointestinal tract and endocrine organs) lies in the neonatal period. The establishment of evidence-based centers for the care and treatment of newborns is one of the most important medical and social problems that determine the conditions for reducing perinatal and infant mortality (6,17, 92, 174, 212).

Since the problem of perinatal damage to the nervous system has become particularly relevant due to the increase in the incidence of this pathology and the success of resuscitation of seriously ill newborns, it is not by accident that special attention has been paid in recent years to the in-depth study of the consequences of such damage. Additionally, the etiopathogenesis, morphology, and clinical symptoms of prenatal injury to the central nervous system have changed as a result of the altered ecological and socioeconomic circumstances.

1.2. Etiopathogenetic basis of the consequences of perinatal damage to the nervous system

Epidemiological studies show that 715 out of 1000 children are diagnosed with "perinatal lesions of the central nervous system" within their first year of life. Over 90% of children treated in neonatal hospitals are diagnosed with this main and concurrent illness. Intrauterine and intranatal fetal hypoxia rank first among the causes of perinatal brain injury, whereas mechanical trauma to the infant during

labor, which can result in intrauterine hypoxia, ranks second. Additionally, infectious (including viral) and toxic-metabolic forms of nervous system injury are etiopathogenetic causes of prenatal disorder (8,59, 119, 198).

Thus, among the factors that cause perinatal damage to the central nervous system, the following are distinguished:

1. Intrauterine hypoxia.
2. Intranatal fetal hypoxia.
3. Mechanical injury during childbirth.
4. Infectious (viral) factors.
5. Toxic factors.
6. Hereditary factors.
7. A combination of the listed factors.

A non-specific symptom of several pregnancy and delivery issues, chiefly pregnant women's toxicosis, is fetal hypoxia. Its duration and severity are determined by the degree and severity of toxicosis, its correlation with extragenital pathology in females, particularly cardiovascular diseases, and the length and intensity of fetal hypoxia in the central nervous system, which is most vulnerable to oxygen deficiency (46, 90, 166, 222).

Intrauterine antenatal hypoxia can occur for the following reasons:

1) maternal conditions in which hypoxemia develops (decompensated heart defects, bronchopulmonary disorders, severe anemia or decompensated acidosis, the aforementioned conditions, severe infectious diseases, intoxication, obstetrical dominance inhibition, malnutrition, one-sided nutrition, occupational hazards, and pregnant women's smoking);
2) impaired uteroplacental blood circulation as a result of the following: placenta previa, premature birth, placental calcifications, infectious placenta, prolonged toxicosis of the pregnant woman, mother endocrine diseases (decompensated or poorly compensated diabetes mellitus), and changes in the placenta with various somatic (hypertension, hypotension, nephropathy, etc.);

3) Fetal illnesses include severe neonatal hemolytic disease, widespread intrauterine infections, brain malformation-related encephalopathies, breech presentation, toxicosis, and maternal intoxication.

The formation of brain capillaries is slowed down and their permeability is increased by antenatal hypoxia. Cell membrane permeability and metabolic acidosis both rise, and cerebral ischemia occurs when intracellular acidosis occurs. Intranatal asphyxia frequently coexists with antenatal hypoxia. Five percent of cases are primary asphyxia. The rise in anaerobic glycolysis is the most significant of a set of compensatory-adaptive responses that accompany hypoxia and asphyxia (18, 47, 82, 129).

Asphyxia that occurs during childbirth is acute. The causes of acute fetal hypoxia, which leads to asphyxia of the child, are:

- acute hypoxia in the mother during childbirth (posthemorrhagic and other shock in the mother, carbon monoxide poisoning, etc.);
- acute disorders of uteroplacental blood circulation - uterine rupture, premature detachment of the placenta, its weakness or excessive activity (in drug-induced and rapid labor - less than 4 hours in primiparous women and less than 2 hours in multiple births), compression of the genital vein by the uterus (depending on the condition of the woman);
- disorders of placental-fetal (umbilical) blood circulation, due to the tight wrapping of the umbilical cord, the formation of true knots, stretching of the umbilical cord, prolapse of its loops, pressing them against the walls of the birth canal with the head;
- Depressing the respiratory center due to damage to the fetal brain (due to drugs, intoxication, infectious diseases, blood clots, etc.) and its premature activation and the onset of intrauterine breathing movements through an open vocal cord (obstetric care, fetal rotation, antenatal hypoxia, intracerebral and spinal hemorrhages, etc.).Additional factors that may contribute to a newborn's respiratory failure include intrauterine hypoxia and spinal cord damage from birth trauma; severe posthemorrhagic anemia; aplasia, or hypoplasia of the lungs;

esophageal atresia with tracheoesophageal fistula; polycythemic syndrome and certain cardiovascular system defects; prematurity (in very premature babies); metabolic disorders (hypoglycemia, etc.); and hereditary and congenital myopathies (18, 44, 97, 154).

In neonatal asphyxia, the integrated pathogenetic connection of the different links of the pathogenic process is impacted by harm to the entire organism brought on by hypoxia and hemodynamic abnormalities in the fetus. Moderate hypoxia for a brief period of time sets off compensatory processes meant to supply enough oxygen to the fetal tissues. The adrenal cortex's production of glucocorticoids also results in an increase in the quantity of erythrocytes in circulation, which raises the amount of blood in circulation. Without an increase in cardiac output, there is tachycardia and perhaps a minor rise in systolic pressure (4-8, 76, 223).

Closed vocal cords, which also serve to compensate for hemodynamic disruptions, are seen to increase fetal motor activity and respiratory rate. Extended hypoxia triggers anaerobic glycolysis, which lowers oxygenation of the body's muscles, skin, and digestive tract. The primary blood supply to the brain and heart is redistributed, and tissue acidosis—the buildup and release of lactic acid into the bloodstream—is exacerbated by the centralization of blood circulation (6, 53, 187, 210, 219).

The main symptoms of a violation of compensatory mechanisms caused by severe or protracted hypoxia include bradycardia, a decrease in cardiac output, collapse, shock, arterial hypotension, and a decline in adrenal function. Cell membranes and the vascular wall become more permeable when there is pathological acidosis. Hemoconcentration, increased erythrocyte viscosity, intravascular thrombi, the discharge of the blood's liquid component into the interstitial space, and hypovolemia follow (3-8, 75, 149).

Hujayra membranalarining o'tkazuvchanligining oshishi diselektrolitemiyani (giperkalemiya, gipomagnemiya, gipokalsemiya) keltirib chiqaradi va tarqalgan qon tomir ichidagi koagulyatsiyaning rivojlanishiga olib kelishi mumkin.

Thrombosis of venules and arterioles, impaired venous outflow due to cerebral edema, ischemia of the brain tissue (blood pressure less than 50 mm Hg when measured at the elbow), dyselectrolyteemia, and minor hemorrhages in the brain tissue and subarachnoid space are the main causes of hypoxic encephalopathy, the most severe clinical manifestation of severe asphyxia (21, 52,129, 173).

Such hemorrhages are not a significant predictor of outcome during the acute phase or for the development of neuropsychiatry later on. Hypoxia combined with hemorrhageological and tissue abnormalities causes the heart to overperfuse, which exacerbates arterial hypotension and leads to the development of ischemic necrosis of the endocardium and capillary muscles. Along with arterial hypotension, hypoxia also causes excessive resistance in the pulmonary veins, which results in pathological shunting, or the movement of blood from right to left through the foramen ovale and arterial duct. A variety of brain injury pathways are predetermined by the polyetiological character of neonatal encephalopathy (2, 94, 172, 196, 204).

One of them is a decrease in cerebral blood flow, which may be due to antenatal hypoxia, which is accompanied by a slowdown in the growth of cerebral capillaries, an increase in their permeability and fragility, and in addition, an increase in the permeability of cell membranes. Against the background of increased metabolic acidosis, cerebral ischemia occurs, which is characterized by the development of intracellular lactic acidosis and neuronal death (7, 42, 84, 148).

A decrease in cerebral blood flow is influenced by a violation of the autoregulation mechanisms of cerebral blood flow. In healthy children, cerebral blood flow and intracerebral pressure are relatively stable and do not depend on changes in blood pressure (BP). In children who have undergone hypoxia, the autoregulation mechanisms of cerebral blood flow are reduced (in moderate hypoxia) or completely absent (in severe hypoxia), and cerebral blood flow depends on changes in BP.

Children who have experienced hypoxia also have decreased cardiac stroke volume (caused by hemodynamic abnormalities and hypoxic myocardial damage),

decreased blood pressure, reduced cerebral venous flow, and increased blood vessel resistance in the brain. endothelium, causing the capillary lumen to abruptly constrict (6-9, 94, 168, 211).

Vasogenic cerebral edema develops against the backdrop of reduced cerebral blood flow and impaired ADG production (asphyxia-syndrome of excessive production, hypoxia-syndrome of insufficient production). This results in the release of "excitatory" amino acids, particularly glutamate, which reduces the supply of energy (glucose) to neurons, inhibits the synthesis of ATP and creatine phosphates, disrupts the functioning of cell membrane pumps, causes depolarization of the outer membranes, and affects neuronal receptors and opens channels that facilitate the entry of sodium and calcium into the cell (10, 69, 117, 194).

Cell edema develops when sodium pulls water with it, while cellular calcium necrosis develops when more calcium enters the cell than is necessary. J.J. Volpe outlines many chains of pathophysiology for intrauterine hypoxia-induced neonatal hypoxic-ischemic encephalopathy: intrauterine hypoxia Fetal acidosis is characterized by a drop in oxygen saturation and a rise in carbon dioxide saturation. Intracellular swelling Brain tissue enlargement, a reduction in cerebral blood flow locally, and overall cerebral edema cerebral necrosis, a generalized reduction in cerebral blood flow, and elevated intracerebral pressure (37, 66, 82, 165).

Stage I is edematous-hemorrhagic; Stage II is encephalic edema; Stage III is leukomalacia (necrosis); and Stage IV is leukomalacia with hemorrhage. These are the morphological phases of acute hypoxia alterations in the brain. Neurons die irreversibly in the next two phases, while the previous two are reversible. Neuronal degeneration, glial growth, sclerotic processes, and the development of cystic spaces in the regions of tiny foci of necrosis are all noted in prenatal (chronic) hypoxia. Therefore, metabolic abnormalities, which are based on oxygen deficit, are the primary linkages in the pathophysiology of neonatal hypoxic-ischemic

encephalopathy. The components that cause direct brain injury in this process are the result of defective metabolism (6, 15, 62,144).

One of the most significant mechanisms of the pathogenesis of hypoxic brain damage in newborns is impaired cerebral circulation, so brain damage that develops after asphyxia can more accurately be called hypoxic-ischemic. Acute severe hypoxic-ischemic encephalopathy caused by hypoxemia and hypercapnia is accompanied by metabolic acidosis, usually with base deficiency, with subsequent deposition of protein and lipid peroxidation products on the endothelium of cerebral vessels, impaired postsynaptic effects of Ca ++, which results in impaired cerebral hemodynamics (17, 68, 128, 205).

Keep in mind that the nature of morphological changes in the brain in encephalopathy is dependent not only on the etiological factor and its duration, but also on the degree of brain maturation at the time of exposure to adverse factors.

A few key considerations must be made when evaluating the condition of cerebral circulation at a particular stage of posthypoxic encephalopathy. The process that causes arteriole vasoconstriction and vasodilation, which under normal conditions produce relatively constant perfusion with appropriately large swings in systemic pressure, is first violated. This is known as cerebrovascular autoregulation (8, 27, 44, 93, 174).

It is known that during brain hypoxia, in response to the influx of Na ions and water molecules into the cell, an increase in the concentration of K + and H + is observed in the extracellular fluid of the brain, which leads to an increase in the activity of cortical neurons, an increase in the dilatation ability of blood vessels and a decrease in their constructive ability. At the same time, hypoxia leads to excessive accumulation of Ca ++ in the cells of the central nervous system, which is manifested by a simultaneous decrease in the extracellular calcium concentration and a decrease in the activity of striatal neurons, an increase in the contractile activity of cerebral vessels and a decrease in their dilatation ability. Thus, it is clear that cerebrovascular autoregulation in hypoxic-ischemic lesions largely depends on electrolyte balance (4, 18, 91,176).

Second, the hypoxic form of hemocirculation, which is characterized by a reduction in cardiac contractility and minute volume, predominates in the first few days of life in prenatal brain injuries caused by hypovolemia and/or temporary myocardial dysfunction.

Thirdly, the thrombogenic direction of hemostasis, or the structural thickening of the blood, is most obviously seen in children with severe congenital hypoxia, even though the oxygen delivery to the tissues is highly dependent on the blood's rheological characteristics.

Fourth, the premorbid background, the level of maturity of the newborn, and the tactics of the treating physician are also of great importance (48,92,116).

It is obvious that a specific amount of time must elapse between the start of the harmful factor's action and the development of structural alterations in the brain. Considering the aforementioned, it is possible to witness many successive, conditionally differentiated stages of the pathological process in the development of hypoxic-ischemic encephalopathy, even with such a polyetiological etiology.

Anatomy of pathology. Asphyxia is characterized by hemorrhages, cerebral edema, and macroscopic brain and internal organ congestion. Hypoxia is characterized by parietal vein hemorrhages, subependymal hemorrhages, and subarachnoid hemorrhages, according to Yu.A. Yakunin (1981). The length of prenatal and intranatal hypoxia is reflected in the macroscopic image. Long-term hypoxia causes glial growth, neuronal degeneration, sclerosis, and cystic gaps in the regions of tiny necrotic foci. The optic peduncle, the subcerebral nuclei, and the cerebellum—particularly the higher poorly differentiated layers—all exhibit destructive alterations (7, 31, 56, 97, 163, 198).

Significant brain structural immaturity, inadequate striatal differentiation, and immaturity of the sulci and fossae are observed in preterm newborns, in addition to generalized dystrophic alterations and glial proliferation. Focal gliosis, porencephaly, and "empty spaces" of brain nerve cells or their calcification are some of the pathological and anatomical abnormalities seen in children who died later in life and had fetal hypoxia. The morphological features of brain alterations

brought on by intrauterine hypoxia—the direct impact of hypoxia on the brain that results in a disruption of cerebral and general hemodynamics, followed by recovery and, ultimately, progressive atrophic changes in the brain—are described in the literature (11, 81, 218).

Course and outcome. The course of CPDNS is determined by a number of factors:

1. The severity and duration of antenatal hypoxia;
2. The presence of intranatal asphyxia;
3. The degree of brain maturity at the time of birth;
4. General signs of prematurity of the newborn and infant;
5. Functional features of homeostasis with immunological "deficiency" of the patient;
6. Somatic problems at 1 year of age.

Varied results and varied dynamics of syndromes during the healing phase might result from the combination of these elements. Chronic intrauterine hypoxia is the main contributing factor, and it can cause serious brain damage as well as developmental delays. Without serious brain material damage, the psychomotor retardation syndrome in preterm children is frequently a consequence of myelination abnormalities brought on by prematurity. While immunodeficiency with frequent recurrent somatic diseases, prolonged postpartum malnutrition, and myelin metabolism can cause severe diseases in children, such as cerebral palsy, these conditions are conditionally referred to as "temporal delay" and can be fully compensated in the future. The mix of symptoms and the severity of the disease during the acute phase influence the outcome (6, 83, 122, 159).

Consequence: By the age of one, the primary symptoms listed below are discernible:

1) Impaired motor development: the child's ability to hold their head, roll over, sit, crawl, stand, and walk independently is delayed compared to their physical age.
2) The development of one or more limb paralysis and paresis (monoplegia, diplegia, hemiparesis, or tetraparesis) linked to different types of cerebral palsy.

4) Psycho-speech development disorders: the emergence of babbling, screaming, the first words and phrases, the quality of spoken sounds, the time at which intentional gestures are formed, the comprehension of address speech, interest in and intentional use of surrounding objects, the type of game, the development of attention deficit hyperactivity disorder, and the concentration of attention.

5) Emotional and behavioral disorders: the development of the animation complex, the ability to distinguish between family members and strangers, emotional resonance, the degree of emotional expression, communication with adults and peers, the capacity for cooperative play, the development of neat skills, and the potential for autism spectrum disorders.

5) Hydrocephalus: excessive head circumference enlargement, head deformity, tortuosity of the direct vessels in the temporal areas, signs of hypertension, and hydrocephalic syndromes; 6) non-epileptic paroxysmal states: infantile torticollis, Sandiffer syndrome, early infantile epileptic encephalopathy (Otahara syndrome), early myoclonic encephalopathy, Dravet syndrome, West syndrome, early neonatal epileptic syndromes, and benign myoclonic epilepsy of infancy.

1.3. Features of metabolic changes in the consequences of perinatal damage to the nervous system

The autonomic nervous system and the endocrine apparatus both have regulatory systems that are violated during hypoxia, which leads to profound alterations in blood circulation and metabolism. One factor in brain preservation, or the reduction in the frequency of cerebral infarction, is a sudden rise in catecholamine concentration during hypoxia (17, 84, 113, 149). Anaerobic glycolysis and the delivery of glucose to tissues are both directly impacted by hypoxia. There is a drop in the amount of glucose in the brain itself, despite the increased flow of glucose to the brain, the activation of glycogenolysis, and glycolysis (predominance of the intensity of utilization of the incoming flow).

Soon, uric acid levels rise, lactate builds up in the blood and tissues, and acidosis occurs.

Redistributing blood flow to the central nervous system can help sustain the oxygen supply during the initial stages of oxygen shortage. High-energy phosphate molecules are broken down from cells in the second stage, which is also thought to be a symptom of the brain's self-defense, coupled with a reduction in the oxygen supply to the brain. When neurophysiological activity is disrupted, several significant alterations occur (14,73, 91, 162).

First, a reduction in the production and release of many chemicals (adrenaline, noradrenaline) that aid in the conduction of excitement causes a shift in synaptic conductivity. A reduction in Na-K-ATPase activity, the inability of the neuronal cell membrane to sustain electrical activity, and the inhibition of calcium ion outflow into the protoplasm of neurons and K ion outflow into the brain's intercellular space are all consequences of anoxia. Proteases, lipases, protein kinase C, and free radicals are all activated when the "intracellular" quantity of calcium rises, which results in cell death (9, 37, 89, 168).

Together, these elements can cause cell necrosis in a matter of minutes or hours. However, apoptosis can also result in neuronal loss in addition to necrosis. The primary distinction between apoptosis and necrosis-induced neuronal death is that the former occurs gradually. The identification of psychoneurological abnormalities during postnatal development should be viewed as a delayed phenomena of suffering, as hypoxic brain injury is gradual in nature. Thus, a crisis of cellular activity starts: water is drawn to neurons, causing edema, and the intracellular concentration of calcium and sodium rises abnormally (41, 58, 152, 218).

There are many phases of fluid retention in the brain: initially, cytotoxic effects cause intracellular edema, which happens most quickly in neuroglial cells; next, disruption to the vascular wall, or blood-brain barrier, causes vasogenic edema. Plasmorrhagia is the result of an increase in extracellular fluid in the brain, which

raises cerebral vascular resistance, increases cerebral blood volume, and alters the dynamics of the cerebrospinal fluid (12, 66, 172, 199, 223).

1.4. The role of structural and functional disorders of cell membranes in the pathogenesis of the consequences of perinatal damage to the nervous system.

There is now a belief that hypoxic problems in neonates fall within the category of free-radical illnesses. The extremely reactive chemicals known as free oxygen radicals, which build up during hypoxia, damage brain cells by attacking membrane components including mitochondria and cell membranes. Their emergence is indicative of the severity of a long-term process, and they can be regarded as harmful compounds that seriously harm nerve tissue. LPO reactions have a chain nature, contribute to the physiological repair of membranes, and are a crucial sign of homeostasis. They also continually occur in the tissues of a live organism, producing active products such as hydroperoxides, aldehydes, ketones, and free peroxide radicals (22, 86, 149, 216).

Lipid peroxidation is triggered, the equilibrium between peroxide activation and breakdown is upset, and an excessive buildup of free radicals in cell membranes alters the structural and functional characteristics of cell membranes, mainly permeability and enzymatic activity. Lecithin breaks down, cell division slows, and the cellular energy supply is reduced. Oxygen radicals' detrimental effects on tissues are linked to the overoxidation of membrane lipids' polyunsaturated fatty acids, the production of harmful aldehydes, the inactivation of enzymes, and their interactions with the cell nucleus' DNA (27, 55, 96, 177).

Systems that accelerate and prevent peroxide generation interact to control the steady-state amount of peroxides. First, lipid peroxidation cannot spread unchecked because of the membrane structure itself. Furthermore, specific membrane systems are in charge of a particular lability that is required for the precise localization of every component and the quick reaction of cells to any effect. The flow of compounds with antioxidant qualities is greatly decreased

against the backdrop of ischemia, which lowers the ischemic organs' capacity for antioxidant defense. The consequences of oxidative stress, stratification of post-hypoxic alterations, and subsequent severe damage to cell membranes and structures result from the antioxidant defense system's inability to neutralize the reactive oxygen species that are produced (14, 63, 96, 160).

During the first 72 hours of life, basic abnormalities in neonates with hypoxic-ischemic circumstances are joined by secondary lesions; post-hypoxic lesions and the impact of oxidative stress are key factors in the pathogenic process's continuance.

While active oxygen species participate in FRO events involving amino acids, proteins, and carbohydrates, the body places a great deal of weight on the oxidation of phospholipids and non-esterified fatty acids (NFAs). Malondialdehyde is the most common LPO product that results from oxidizing agents attacking non-NFAs repeatedly. Malondialdehyde builds up in membranes and combines with proteins and lipids to generate polymeric compounds that increase membrane stiffness and microviscosity while decreasing permeability and deformation. This shortens the lifetime of cells and speeds up aging (37, 88, 125).

For biological membranes, lipid peroxidation below normal levels is just as harmful as its abrupt activation. Since the quantity of metabolites relies on both the rate of breakdown and excretion of the metabolite from the cell as well as the degree of secretion, the accumulation of metabolites does not necessarily indicate the intensity of the activity.Both active metabolism and inactive cells exhibit a rise in malondialdehyde (MDA) when it accumulates in membranes. The MDA accumulation coefficient, which is the ratio of the MDA content to the degree of erythrocyte hemolysis, provides a more accurate picture of the severity of LPO. This indication shows how well the cell can withstand MDA's harmful effects (17, 65, 92, 169).

Despite certain structural and functional variations, the fundamental structure of all plasma membranes is the same. With a total surface area of tens of thousands of square meters, this enables us to view the human body as a complicated system of

individual structural components. All of this points to the possibility of shared membrane disease mechanisms. Since EyoK, the primary substrate of lipid peroxidation, is a crucial part of every biological membrane, the detrimental effects of promoting lipid peroxidation processes are evident in every cell membrane. Erythrocytes can be a useful model in clinical settings for examining the status of the organism's cell membranes (8, 44, 83, 139, 212).

Despite the unique characteristics of individual cells, erythrocyte membranes represent the structural and functional characteristics of cell membranes as well as the overall health of metabolic processes inside the body. Given these factors, it is challenging to interpret alterations in erythrocyte membrane conditions in different disorders as a result of reorganizations in other cells' plasma membranes. Erythrocyte deformability diminishes and blood viscosity increases under the hypoxic and acidotic circumstances that accompany it. These blood rheological alterations impair microcirculation, slow down transcapillary exchange, and increase tissue oxygen deprivation (27, 54, 88, 153, 162).

Increased lipid peroxidation in erythrocyte membranes and a change in their rheological characteristics—the deformation of erythrocytes decreases and the surface architecture is disturbed—have been demonstrated in newborns with perinatal nervous system damage. These changes are linked to the severity of circulatory disorders in the brain. A highly significant phenomena is seen in the form of activation of cell repair mechanisms when there is mild hypoxia, which is frequently present in newborn units and damage is either small or has not yet happened (59,93,158).

With the activation of these mechanisms, the body's resistance to subsequent hypoxic attacks increases with repeated exposure to hypoxia. Such conditions, which increase the adaptation to hypoxia, should also be considered as a very important phenomenon of the brain's self-defense (12, 44, 85, 149).

The brain of newborns is very neuroplastic and has a very large compensatory potential. The search for ways to influence the processes of restoration of damaged brain tissue opens up broad prospects and gives hope for the possibility of

restoring lost functions. A very important circumstance that should be taken into account when assessing the possibility of compensation for brain diseases that have arisen in newborns is that nerve cells of the brain that have undergone hypoxia, but have not died, do not lose their ability to further develop (5, 47, 73,172).

Additionally, the tremendous neuroplasticity of a newborn's brain explains why harm triggers healing mechanisms almost immediately.

1.5. Newborn brain compensatory capacities in relation to the effects of perinatal nervous system injury

The number of nerve cells and synapses per cubic millimeter of brain tissue in an adult is only 40% of that in children aged 1 to 7 years, and at this age the number of synapses per nerve cell is 20% less, which indicates the great plastic and reparative capabilities of the brain of young children, including the ability to compensate for defects in prenatal brain formation (55, 93, 167, 217).

This is also explained by the immaturity of its structures and their lower dependence on energy-consuming ion pumps. At the same time, an increase in glycolytic capacity allows for faster restoration of ATP and preservation of brain functions. The active recovery phase in the acute period is facilitated by neurotrophic factors injected into the extracellular space of the brain after infarction (16, 52, 93, 187).

Experimental investigations have shown that the brain can transplant immature neurons and create new neurons in response to damage. Furthermore, it can help denervated neurons regain their structural integrity. Furthermore, the detrital tree's preservation, metabolic activity, protein synthesis, and other processes—the primary determinants of the process of rebuilding nerve tissue—increase with the onset of the reinnervation process. The use of millions of synapses, which are converted into thousands of functional complexes, is linked to the vast potential for recovery. The earlier therapy may start—that is, before the ontogenetic processes

of the nervous system's development are finished—the more likely it is to restore the neuroplasticity potential that already exists (88, 152, 195, 218).

When discussing factors contributing to recovery processes, the concept of the use of alternative neuronal pathways and / or the functional awakening of synapses after their damage is expressed. It is assumed that many of these pathways are involved in the many bidirectional pathways that exist in the central nervous system and arise not only at the level of the brain stem and cerebellum, but also at the neocortical level (18, 68, 162, 202).

All neuronal systems of the central nervous system are closely interconnected. However, even in a normally functioning nervous system, the activity of some fiber structures can be in a state of slowed down and inhibited. After some kind of harmful effect on the brain (for example, due to hypoxia), their activation and partial or complete restoration of functions can be observed (61, 84, 173, 209).

Restoring the function of nerve cells and preserving their vitality are made possible by strengthening the nerve signal. It appears that the quantitative balance of neurotransmitters that have been redistributed to the remaining undamaged polysensory neurons must be restored following injury to the central nervous system. Then and only then can the healing process start. Following an acute hypoxic damage, brain functions recover in stages. Changes in brain metabolism, a fully reversible process, are linked to the quick recovery phase. At this point, intense therapy helps to minimize the pathological process's advancement or residual consequences by actively preventing more neuronal death and stimulating the pathology's reversal (33, 85, 147, 222).

The intermediate period, which can persist for many weeks or even months, is clinically equivalent to the fast recovery phase. It typically lasts nine to twelve months. The characteristics typical of the age standard can be restored in this situation.

Numerous factors influence the rehabilitative outcome, but the backdrop against which hypoxia damage occurred has a major influence.

Normal intrauterine development increases a child's compensating reserve and resilience to oxygen shortage. It is crucial to remember that the degree of primary injury also affects how well the central nervous system operates (11, 96, 136, 176). The recovery rate is just 3% in cases of diffuse cerebral infarction, while it might reach 80% in cases of mild hypoxic-ischemic encephalopathy. It is important to remember that postasphyxial encephalopathy has distinct phases of morphofunctional development and progressive course.
To get the desired result, it's critical to understand which stage and which medicine combination to utilize. In the future, stimulating treatment for injured but surviving nerve cells is preferred if the goal during the acute phase is to minimize potential harm to brain tissue (48, 76, 141, 188, 209).

It's also critical to keep in mind that brain damage extends beyond the original lesion and that, throughout the first year of life, two processes take place simultaneously: the healing of hypoxically damaged neurons and their destruction. The most effective stage of neuroplasticity should be the focus of restorative therapy to fully realize its potential.

Illness/death : The death rate is more than 50% in cases of severe perinatal nervous system injury. In the first month of life, about half of the youngsters pass away. Aspiration pneumonia and other diseases can cause the death of certain infants with serious neurological abnormalities. Among newborns who survive severe perinatal nervous system damage, cerebral palsy, epilepsy, and oligophrenia are the most prevalent causes of impairment. These youngsters need to get specialist treatment in clinics that are equipped to handle their multisystemic issues through a coordinated set of actions.

The severity of the neurological system's impairment during pregnancy determines how frequently each of its effects manifests. Up to 10% of children who survive the effects of severe prenatal nervous system damage are known to be healthy, 10% to 20% have moderately severe issues, and up to 80% have substantial ones. About 30–50% of children who survive moderately severe

prenatal nervous system damage experience major long-term difficulties, and 10–20% experience some health abnormalities (29, 83, 144, 170).

Children who suffer from mild hypoxic-ischemic encephalopathy frequently do not experience significant central nervous system consequences. Such children may not have functional difficulties later in life, even if they do not exhibit any overt neurological signs during the newborn period. Even in the absence of overt indications of brain damage, 15-20% of kids who have suffered from the effects of mild prenatal nervous system injury struggle significantly in their studies. As a result, a neurologist should continue to monitor all children who have suffered from moderate to severe prenatal nervous system impairment, just as they were throughout infancy (28,77, 143, 191, 218).

1.6. Principles of rehabilitation of the consequences of perinatal damage to the nervous system

The goal of restorative treatment is to help a kid who has been ill or damaged become well again while avoiding a chronic condition. Three primary phases exist: Children suffering intrauterine hypoxia or asphyxia during birthing are transported straight from the maternity hospital to the neonatal pathology department, which is the neurological section of a children's hospital. The second step, known as the post-hospital stage, might be completed at home, at a recovery facility, or in a hospital's rehabilitation department. A doctor working in a clinic, rehabilitation center, dispensary, or outpatient setting may oversee the third stage (4,92, 165, 200, 214).

In order to avoid or minimize irreparable brain damage, the primary objective of the treatment procedures used to restore lost or changed functions of the central nervous system is to regulate or enhance the metabolism of the injured brain, activate the preserved structures, and so on. A number of prerequisites must be met for rehabilitation therapy to be successful, taking into consideration the features of a newborn's growing brain:

1. Since delay endangers the newborn's life as well as the central nervous system,

early and objective identification of brain abnormalities enables the prompt initiation of pathogenetically based therapy.

The main strategy of therapeutic measures is to ensure that the therapeutic intervention coincides with the stage of reversible disorders, thereby stopping apoptosis, minimizing the focus of damage and, possibly, preserving more functioning nerve cells.

2. Hypoxic brain damage is characterized by a progradient course with the presence of several stages of the pathological process. In this case, in the posthypoxic period, the volume of the primary lesion increases significantly due to the involvement of other parts of the brain. Thus, dystrophy of previously altered neurons, which is prolonged over time, leads to their loss in different parts of the brain and the disruption of synaptic connections between interacting structures. At the same time, neuroglia and the vascular network of the brain are involved in the process.

3. As a result, the approach from the very beginning of life is both exciting and neuroprotective. Its goals include restoring the brain's metabolic processes, improving local absorption of oxygen and glucose, making the best use of fatty and amino acids, boosting intracellular protein synthesis, improving the metabolism of nucleic acids, eliminating harmful metabolic products, and more. Based on this, efforts are made to protect the neuroplasticity of a newborn's and a child's brain during the first year of life, employ the phenomena of self-healing, and stimulate reparative processes in the central nervous system.

Two processes occur simultaneously in the brain of a child who has been exposed to the effects of hypoxia: the repair of nerve tissue and its dystrophy. In this sense, the recuperation stage may be prolonged indefinitely. Furthermore, delayed post-hypoxic psychoneurological illnesses are a condition that can be seen. This supports the claim that recovery and neural state restoration are not the same thing. As a result, children in this group may have brain weakening, which shows up when the brain is under more stress (neuroinfections, stress, trauma). Deviant

conduct, sleep issues, convulsions, and other psychoneurological diseases are some of the ways they show up (9, 72, 119, 168).

The search for pathogenetic treatment methods appears to be highly relevant, as the current treatment methods do not satisfy practitioners due to the high percentage of residual complications. This is because hypoxic damage is the basis of all such changes, and the nervous system's reaction can be phasic.
This may be because medications having antihypoxic actions are not included in the complex of treatment medicines, even if the damage to the central nervous system is hypoxic. Consequently, we employed Vincamine, a medication that has a strong antihypoxic, selective vasoregulation impact on cerebral blood flow and helps to adjust cerebral blood flow to the brain's metabolic requirements (64,99, 172, 191).

By promoting glucose oxidation, Vincamine increases energy generation and stimulates brain activity in general, improving brain metabolism.
Vincamine lowers and stabilizes the cerebral vascular bed's peripheral resistance while increasing the oxygen delivery to neurons under hypoxia.
A naturally occurring neurometabolic herb is Vincamine. According to the literature, Vincamine's oxygenating effect enhances the brain tissues' capacity to bind and use oxygen. It also raises the level of carbon dioxide in venous blood, which, via a secondary vasoregulation mechanism, increases the blood supply to the affected area's collateral network. never results in "steal" syndrome.

lowers cerebral vascular resistance, particularly in the precapillaries and arterioles. fixes problems with phosphate content and glucose metabolism. The difference in the brain's capacity to absorb oxygen, respiratory rate, glucose consumption, and arteriovenous lactic acid concentration is the basis for Vincamine's beneficial metabolic impact in syndromes that develop against the backdrop of cerebral arteriopathy (55, 93, 118).

In summary, Vincamine is an activator of metabolism in brain tissues, improving impaired metabolism in noradrenergic and 5-HT receptors; increasing the activity

of noradrenergic neurons linked to behavioral reactions, attention, and memory; increasing the brain tissues' utilization of glucose; and contributing to an increase in total brain activity. Additionally, Vincamine activates phosphorylation processes in AMF and ADF, improves oxidative processes in mitochondria, raises the oxygen content in brain tissues and neurons by 23%, and has the effect of preserving metabolic activity for a considerable amount of time following hypoxia (3 months).

The drug's selective vasoregulation effect is another significant feature. It reduces artery spasm and increases venous vessel resistance, helps adjust cerebral blood flow to the brain's metabolic demands, and lowers and stabilizes the peripheral resistance of the brain's vascular bed. According to the results of the literature study, hypoxic vascular injury continues to be the most common condition affecting a newborn's neurological system, with perinatal encephalopathy being the most severe. Finding new medications to improve the lives of these children and their parents is necessary due to the emergence of conditions including cerebral palsy, epilepsy, and minor brain malfunction (49, 82, 174).

We think there may be promise in using Vincamine in this way. Vincamine's efficacy in treating adults and adolescents with a variety of brain disorders is well documented in the literature, however its application in treating children with CPDNSis not mentioned. Based on tracking improvements in the child's overall health and the ExoEG score, we made the decision to close this gap by investigating the efficacy of Vincamine in perinatal central nervous system injuries.

CHAPTER II. RESEARCH MATERIALS AND METHODS.

37 children with respiratory conditions, ages 3 months to 1 year, were admitted to Samarkand City Children's Hospital No. 1 while we were in charge of them. The primary diagnosis for all children admitted to the hospital was severe respiratory virus illness. Three sizable groups were formed from the analyzed children: Ten children make up the control group, twenty make up the major group, and

seven make up the healthy group. Twenty hospitalized children with respiratory disorders and the effects of perinatal nervous system damage were in the main group, receiving Vincamine; ten children with respiratory disorders and the effects of perinatal nervous system damage were in the control group, receiving conventional therapy.

The conditionally healthy group consisted of 7 children with respiratory diseases, but without the consequences of perinatal damage to the nervous system, who received conventional therapy.

In the distribution of patients by gender, boys predominated over girls 17 (45.9%) 20 (54%). We also divided the children into age categories.

Table 2.1.

Age distribution of patients

Children's age	Control group	Main group	Healthy group
3-6 oy	4 (40%)	8 (40%)	2 (28,57%)
6-12 oy	6 (60%)	12 (60%)	5 (71,4%)

As can be seen from Table 2.1, the total number of children aged 3 to 6 months was 14 (37.8%), and children aged 6 months to 1 year were 23 (62.2%).

The next step was to classify children with respiratory diseases according to the consequences of perinatal damage to the nervous system. When making a clinical diagnosis of the consequences of perinatal damage to the nervous system, we were based on the classification adopted by the Department of Child Neurology, TashVMOI, as well as the Pediatric Research Institute.

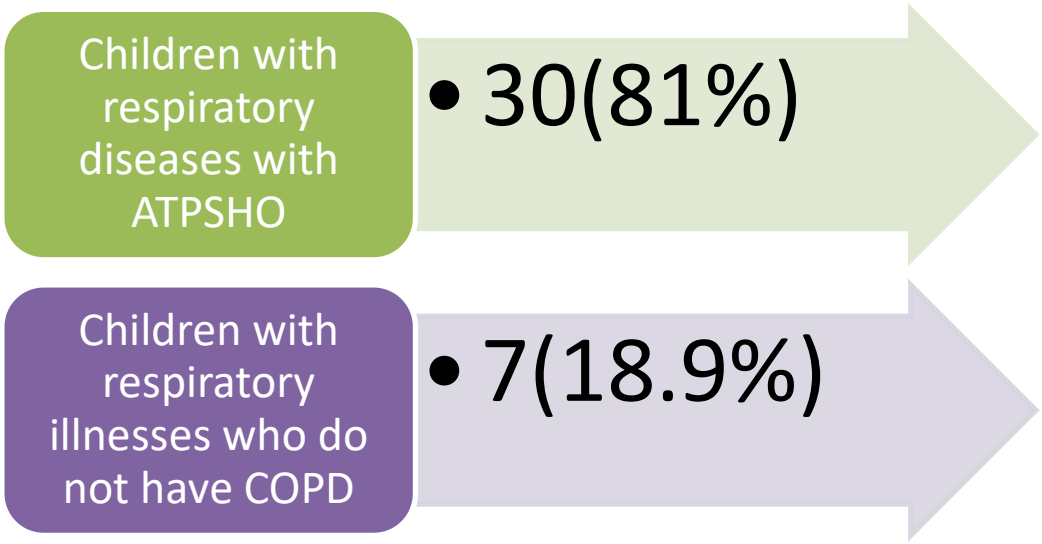


Figure 2.1. Distribution of respiratory diseases in children with the consequences of perinatal damage to the nervous system.

Respiratory diseases, In addition to the study of the neurological status of children with the consequences of perinatal damage to the nervous system, a study of the somatic condition, peripheral blood and urine composition was conducted. All children were examined by narrow specialists: ENT doctor, neurologist, pediatrician.

For diagnostic purposes, all children underwent exoencephalography of the brain to determine the growth of the cerebral ventricular complex.

In addition to assessing the neurological and somatic condition of children, we studied the following parameters characterizing the LPO processes and the intensity of oxidative processes in erythrocyte membranes: before incubation (mechanical stability of erythrocytes) and under physiological conditions (unassisted peroxide hemolysis), the degree of erythrocyte hemolysis after incubation, malondialdehyde (MDA) content before and after incubation, the intensity of MDA degradation, and the ratio of % MDA degradation/MDA content (D/MDA).

2.1. The disease's clinical manifestation in patients.

Thirty-seven children with respiratory conditions were assessed. Acute respiratory

virus infections were the primary diagnosis for all of the youngsters admitted to the hospital. Clinical, analytical, and instrumental exams revealed that 30 children had the effects of prenatal nervous system injury. All of the moms in this groupexperienced a difficult pregnancy and delivery. **Disease prevalence and pregnancy and delivery problems are shown in Table 2.1.2.**

Nosological features	Absolute number	%
A. Diseases:		
Cardiovascular system diseases	2	5.5
Kidney disease	3	8.3
Chronic inflammatory diseases of the genital organs	15	41.7
SUD	13	36.1
Total	33	91.7
B. Pregnancy complications:		
Toxicosis of the first half of pregnancy	10	47.2
Risk of miscarriage	6	16.7
Iron deficiency anemia	18	63.9
Total	34	91.8
B. Complications of childbirth:		
Rapid labor	1	13,9
Weak labor activity	6	22,2
Long dry period	3	11,1
Boot and foot delivery	2	11,1
Umbilical cord entrapment	2	13,9
Surgical delivery	3	8,3
Total	17	45,9

From Table 2.1.2, it can be seen that 33 out of 37 mothers (91.71%) had chronic diseases. Cardiovascular system pathology was detected in 2 (5.5%) mothers, urinary incontinence - in 15 (41.7%). Most often, the pregnancy process was complicated by anemia in 18 (63.9%), ARVI in 13 (36.1%). Toxicosis in the 1st half of pregnancy was observed in 10 pregnant women (47.2%), the threat of miscarriage in 6 (16.7%).

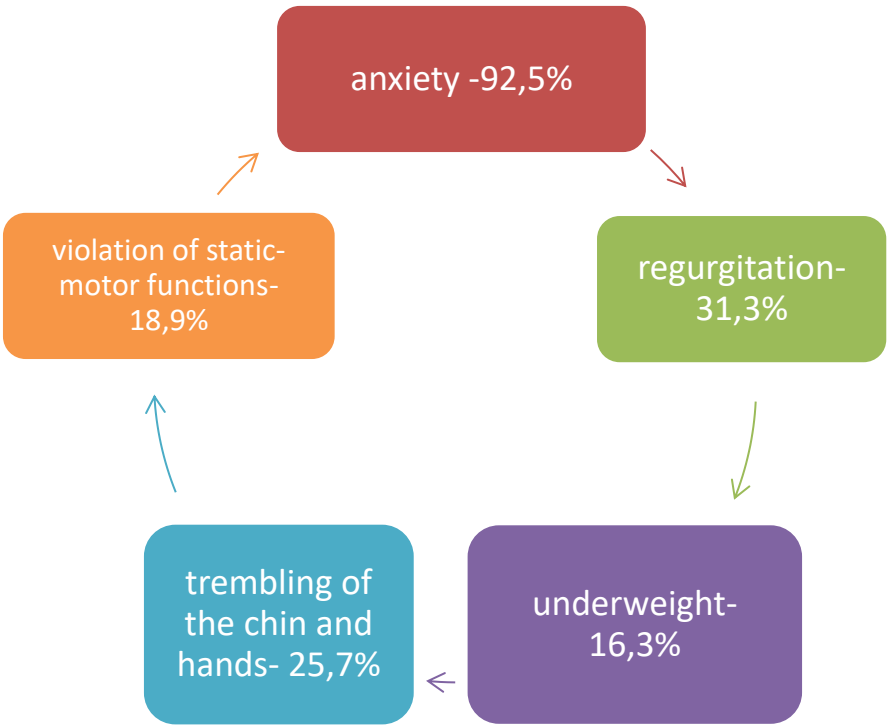
It was found that labor was complicated in 17 (45.9%) mothers. The most common causes of pathology of the labor period were weak labor activity, prolonged waterless period, entanglement in the umbilical cord system, surgical intervention, rapid labor.

Thus, most children suffering from the consequences of perinatal damage to the nervous system were born to mothers with chronic diseases of the cardiovascular system, genital organs, kidneys, and nasopharynx.

Pregnancy was difficult for all moms of children with the effects of prenatal nervous system impairment. Every kid was born to moms who underwent a difficult childbirth.

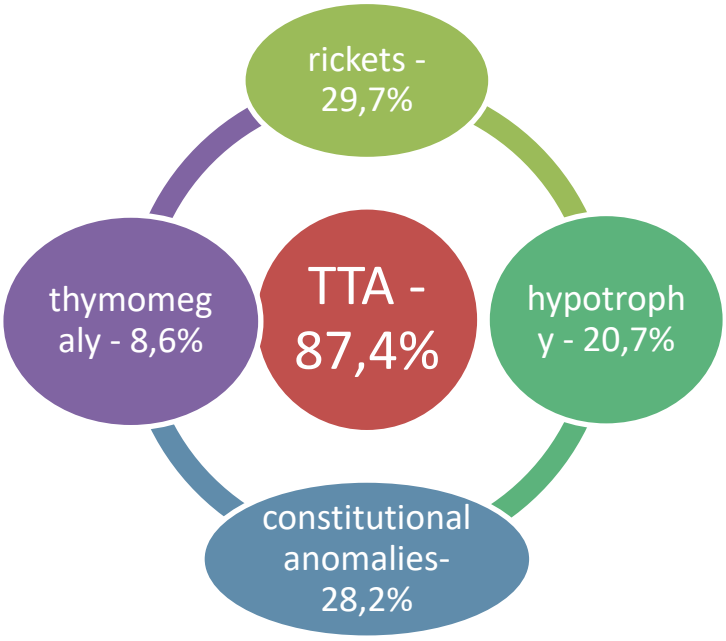
Analyzing anamnestic data revealed that just two infants experienced acute renal failure with a probable intrauterine infection, indicating that the newborn phase was unremarkable.

Only 8 (21.6%) of the 22 patients who received a first-time diagnosis of the effects of prenatal nervous system injury had previously received treatment for these conditions. The majority of parents reported early complaints of inadequate weight growth, systematic constipation, causeless malaise from birth, and occasional unexplained fever, according to a thorough analysis of the anamnesis (Fig. 2.1.2). Children received symptomatic therapy for this.



The most frequent concerns regarding children's anamnesia are shown inFigure 2.1.2.

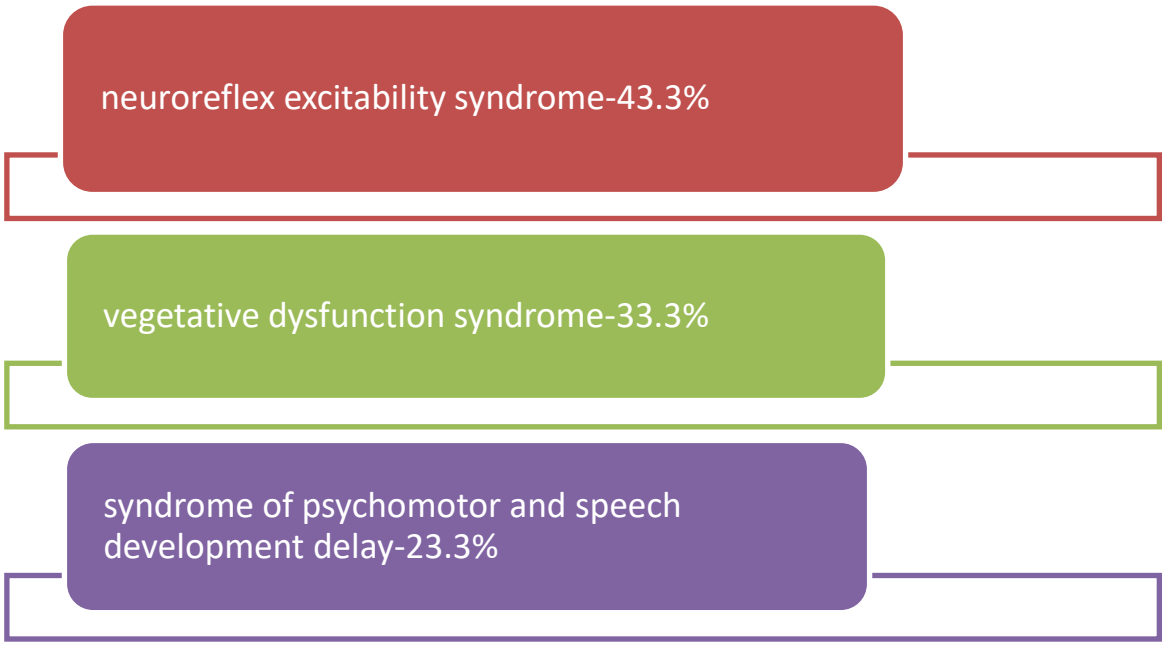
Anxiety was the mother's most frequent complaint during the child's evaluation, occurring in 92.5% of instances. Changes typical of disorders such rickets, anemia, hypotrophy, constitutional abnormalities, and thymomegaly were also found during a thorough clinical and laboratory evaluation of every kid. All children were found to have at least two background illnesses as a result (Figure 2.1.3). Sweating, loss of nape hair, pale complexion, underdeveloped subcutaneous adipose tissue, and low appetite are all signs of these kids. The development of respiratory disorders as well as the effects of prenatal nervous system injury are both accelerated by these underlying illnesses.



Background illnesses in children (Figure 2.1.3).

Figure 2.1.3 shows that iron deficiency anemia (87.4%) and rickets (29.7%) were the most prevalent background conditions that coexist with the emergence of respiratory disorders as a result of prenatal nervous system injury. Every kid in the study had an adverse premorbid history; 20 (54.5%) had primary anemia, 15 (40.5%) had secondary anemia, and 2 (5.4%) had tertiary anemia. The majority of children (33) (73%) had moderate to severe general conditions. After being admitted to the hospital with a serious condition, the remaining three (8.1%) children's condition improved to a moderate state in two to three days due to the effects of the underlying disease's therapy.

When youngsters were examined, their neurological condition was found to be abnormal. After analyzing the clinical and instrumental data, the following findings were derived: 13 children (43.3%) had neuroreflex excitability syndrome, which was the most common. Ten children (33.3%) had vegetative dysfunction syndrome, and seven children (23.3%) had psychomotor developmental delay syndrome (Fig. 2.1.4).



Frequency of different symptoms resulting from prenatal nervous system injury (Fig. 2.1.4)

Constant regurgitation, chronic hypotrophy, respiratory rhythm abnormalities, skin color, acrocyanosis, bradypnea, thermoregulation problems, gastrointestinal tract dysfunction, and temperamental dysfunction were the primary alterations in the vegetative dysfunction syndrome. A child with a syndrome of increased neuro-reflex excitability was found to have emotional lability and movement restlessness against the backdrop of normal mental and physiological development. This happens when visual, auditory, and tactile analyzers are affected by slight changes in the environment.

These kids sometimes twitched, experienced small-amplitude tremors, had trouble falling asleep, were superficially restless, and didn't sleep long enough. Reduced unconditioned congenital reflexes are a hallmark of the syndrome of delayed psychomotor development. Youngsters lacked active attention, made passive, brief noises, delayed object manipulation, and showed little active interest in toys or other items. They also did not react appropriately to their mother's presence.

Common (banal) bronchitis was characterized by a rise in body temperature, followed by a dry, severe cough that lasted for three to four days before turning into a wet cough. Percussion produces distinct lung sounds. Dry, widespread wheezing on auscultation, accompanied by labored breathing.

The department employed piracetam, medications that increase cerebral circulation (vinpocetine), and syndrome treatment as part of complicated therapy. Vitamin preparations and diuretics were employed according to the clinical signs that predominated. Simultaneously, the conditions brought on by respiratory and background illnesses were also improved.

Based on the primary symptom of the effects of prenatal nervous system impairment, we separated all of the patients we studied into three groups (Table 2.1.3).

Table 2.1.3 shows how patients with respiratory conditions are distributed according to the syndrome of the effects of prenatal nervous system injury.

Syndromes	Control group	Main group	Total
Neuro-reflex excitability syndrome	3(21%)	10 (50%)	13 (43,3%)
Vegetative dysfunction syndrome	4(28,5%)	6(30%)	10 (33,3%)
Psychomotor and speech development delay syndrome	3(21%)	4(20%)	7(23,3%)

Mothers reported that emotional instability, twitching during sleep, chin and hand tremors, difficulties falling asleep, short-term, shallow sleep, and head backwards jerking were the primary symptoms of children with neuro-reflex excitability syndrome.

Children with autonomic dysfunction syndrome primarily complained of stress, restlessness, acrocyanosis, and capriciousness. Falling behind their classmates in

mental and linguistic development was the primary complaint of children with delayed psychomotor development syndrome. Later, the kids in this group learned to sit, stand, walk, turn, and hold their heads.

In youngsters of all groups, on inspection, the general condition was moderate. The skin was clean, warm, and pale. Of the youngsters, 12 (32.4%) had acrocyanosis. Ten youngsters (27%) had a poorly formed subcutaneous fat layer. Regarding the respiratory system, every youngster complained of fever, runny nose, and cough. The sound of strained breathing was accompanied by auscultatory dry scattered wheeze. On percussion, distinct lung sounds could be detected. From the cardiovascular system, children with severe anemia had functional murmurs, but nearly all children had muted auscultatory heart sounds. The majority of kids had elevated heart rates.

From the gastrointestinal tract, in children with vegetative-visceral syndrome: regurgitation was observed in 5 (13.5%), vomiting in 3 (8.1%), and frequent constipation in 4 (10.8%). Examination revealed increased bowel movements, rumbling, and abdominal distention. The feces were unformed, without pathological impurities.

No pathological signs were detected from the gastrointestinal tract in children with neuroreflex syndrome and psychomotor developmental delay syndrome. The abdomen was soft and painless. The liver protruded 2 cm above the costal margin, the spleen was not palpable. The feces were formed without pathological impurities.

In children with neuro-reflex excitability syndrome against the background of normal mental development, the following symptoms were observed from the central nervous system: emotional lability, anxiety that occurs after minor changes in the environment, chin tremor, periodic small tremors in the hands. Increased innate reflexes were observed, spontaneous Moro reflex.

Delays in the development of cervical-tonic reflexes were noted in children with Robinson, Babkin, muscular hypertonia, and vegetative-visceral syndrome. reduced tendon reflexes and spontaneous activity. The following was seen in

children with central nervous system psychomotor developmental delay syndrome: weakened motor and static abilities. Children searched their eyes for the source of the sound without turning their heads, and in rare instances, a grin would arise without explanation. There was no response to the mother's voice, no aural attention, no humming, no vague weeping. Active attention was absent. There was a decrease in unconditioned innate responses.

2.2 Research techniques

We used exoencephalography to investigate the psychosomatic processes brought on by Vinkamin usage and to evaluate the medication's efficacy. Based on the EXO 12 ultrasound equipment, an exoelectroscopic investigation was carried out at City Children's Hospital No. 1. The degree of erythrocyte hemolysis, the role of structural and functional alterations in cell membranes, and the degree of MDA and MDA degradation in their contents were also investigated.

There were 37 studies carriedout in all (Table 2.2.4).

Table 2.2.4: Total number of studies carried out

Research methods	Number of studies
Exoencephalography of the brain	37
Blood analysis	37
Urinalysis	37
Intensity of hemolysis of erythrocytes and the content of MDA in them	37
Intensity of MDA degradation	37

The ExoEG method

Exoencephalography is a technique that uses ultrasonography to analyze the brain. The skull's soft tissues and brain matter reflect ultrasound to varying degrees and have varying acoustic resistances, which are employed for diagnostic purposes. During 1956–1958, Swedish researcher Leksell originally developed the so-called middle exo (M-exo), a technique for locating ultrasound signals reflected from the brain's middle structures. In addition to lesions like hydrocephalus, intracerebral pressure, and brain cancers, ExoEG enables the detection of volumetric processes in the brain, hematomas, abscesses, foreign bodies, etc. There are very few contraindications to the procedure.

One-dimensional ExoEG is performed using ultrasonic encephalographs, in particular, the Exo-11 stationary exoencephalograph and the Exo-12 portable, manufactured by domestic manufacturers. No special preparation of the patient is required for the study. ExoEG is usually performed with the patient lying down. An ultrasonic sensor, the working surface of which is treated with vaseline oil (to ensure acoustic contact), is sequentially placed on different parts of the head. Ultrasonic signals converted into electrical impulses appear on the device screen in the form of a curved line - an exoencephalogram, which is photographed and analyzed. It is considered convenient to obtain an echo signal when the sensor is located on the lateral surface of the head at a height of 4-5 cm from the external auditory canal along the biauricular line passing through the parietal region.

In the exoencephalogram, impulses from the initial complex (NK), final complex (KK), middle exo (M) and various non-medial brain structures (ET) are distinguished. Some impulses are unstable, others are relatively stable, and a number of impulses appear only when there is a pathological process in the brain. The most constant signal is the exo signal from the middle structures of the brain. This exo signal (M-exo) dominates over impulses from other structures, its base is wider, sometimes it consists of two or more impulses. Usually, the M-exo is located along the midline of the brain, and its permissible displacement should not exceed 1-2 mm. A displacement of the M-exo by more than 2 mm indicates the

presence of a volumetric process in the cerebral hemisphere opposite to the direction of displacement.

Other diagnostic criteria are different exo signals when examining the right and left halves of the head.

In the acute period, with perinatal brain damage, with exoencephalography during brain edema, an increase in the number of exo signals on both sides of the M-exo is noted. In local edema and hemorrhages, an increase in the pulsation of the exo signal up to 100% - M-exo shift up to 3 mm, an increase in the number of signals on the affected side, an increase in the number of M-exo pulsations up to 70%. In the recovery period, ExoEG shows: a shift of M-exo by 1 mm, pulsation of exo signals below 20% and above 90%, sharp, pulsating, with horizontal or chaotic elements, a ventricular index of more than 2.6 mm, a number of exo signals, a width of 3 ventricles of more than 8 mm. a decrease in the ventricular index.

All of the aforementioned syndromes can occur in children who have the effects of perinatal nervous system damage, particularly if the ExoEG shows pathology suggesting hypertensive syndrome. More significantly, if the M-Exo shift is greater than 2 mm, it indicates the presence of atrophic processes in the brain tissue. It should be mentioned, nonetheless, that in the majority of situations, stability of the process may be accomplished with early and well prescribed medication, even in cases where the ExoEG shows noticeable alterations.

Investigation of how alterations in cell membrane structure and function contribute to the pathophysiology of the effects of fetal nervous system injury.

The degree of erythrocyte hemolysis before incubation (mechanical stability of erythrocytes) and after incubation under physiological conditions (peroxide hemolysis without initiation) were among the parameters we studied in addition to evaluating the neurological and somatic status of the children. We also looked at the intensity of MDA degradation, the ratio of % MDA degradation/MDA content (D/MDA), and the degree of erythrocyte hemolysis before and after incubation.

The National Center for Organic Chemistry's clinical biochemistry lab was used to investigate the lipid peroxidation mechanisms in erythrocytes. Both before and after therapy, the LPO processes were studied. For the study, 0.5 ml of peripheral venous blood was drawn in the morning, precisely at the same time, and placed in a test tube with 3.0 ml of citrate solution. The erythrocytes were cleaned three times in saline after the blood was centrifuged for ten minutes at 1500 rpm. The investigations were conducted between 0.5 and 1 hour following blood collection.

Measurement of the erythrocytes' MDA concentration and the degree of hemolysis.

The "Bulletin of Discoveries, Inventions, Industrial Designs, Trademarks" approach was utilized to sequentially determine the level of hemolysis and the amount of MDA present in erythrocytes. Yu.A. Yurkov and V.V. Bankova, 1986. Issue 11. Page 207. Using a 5% solution of pH-7.4 mM Tris-HCI buffer, centrifugation at 1500 rpm for 10 minutes, and spectrophotometry of the supernatant at 532 nm on SF-26, the degree of hemolysis of the erythrocytes prior to incubation was assessed. By mixing 0.02 milliliters of 0.1% saponin with a parallel sample, the percentage of extinction of complete hemolysis was determined.

Following incubation, centrifugation at 1500 rpm for 10 minutes in a 5% solution in 25 mM Tris-HCI pH-7.4 buffer, incubation at 37 °C for 10 minutes, and spectrophotometry using SF-26 nm were also used to assess the degree of hemolysis of the erythrocytes. The quantity of MDA was ascertained by reacting with thiobarbituric acid after the percentage of hemolysis in the same samples was ascertained. A molar extinction coefficient of 1.56x10-5 cm-1 for MDA was used to calculate the values, which were expressed in nanomoles per 10 erythrocytes. By computing the MDA/hemolysis coefficient upon incubation, it was feasible to evaluate how the degree of hemolysis depended on the quantity of MDA present due to the full unification of the circumstances when utilizing both approaches.

The MDA/hemolysis ratio is an independent metric that indirectly describes membrane structural alterations and shows a cell's capacity to tolerate the harmful effects of MDA. The retention of MDA in non-hemolyzed erythrocytes is directly correlated with the value of this ratio, which is known as the MDA accumulation coefficient. It is well known that an increase in this coefficient subtly signals modifications to the structural and functional state of membranes that aid in MDA retention. These modifications, in turn, cause a decline in the rate of LPO, membrane metabolism, and the activity of numerous membrane enzymes.

Method for studying the intensity of MDA degradation.

Finding the degree of MDA degradation enables us to consider how LPO processes might be controlled; that is, a rise in MDA content may be caused by both a drop in the intensity of MDA degradation and an increase in the LPO rate. After hydrolyzing 1,1,3,3-tetramethoxypropane for three minutes at 40 °C with 0.1 N hydrochloric acid, it is neutralized and diluted in Tris-HCl buffer pH-7.4 pH-7.4, yielding a concentration of 10–15 nmol in 10 μl of buffer. Two parallel tubes are filled with 400 μl of buffer. The first erythrocyte sample and the control sample (850 μl of buffer) are treated with 10 μl of MDA solution, and the second sample is treated with 10 μl of buffer.

500 μl of 30% trichloroacetic acid (TCA) solution, 150 μl of 5M HCl, and 600 μl of 0.67% thiobarbituric acid (TBA) solution are added to all three samples after they have been incubated for two minutes at 37C. A centrifuge is used to measure the quantity of MDA in each of the three samples after they have been in a bath at 95 °C for 40 minutes. The following formula is used to determine the degree of degradation:

$$E0 - E1 + E2 < E0x100 = D$$

where E0 is the optical density of the control sample, E1 is the optical density of the first erythrocyte sample with MDA added, E2 is the optical density of the

second erythrocyte sample without MDA added, and D is the intensity of MDA degradation expressed as a percentage.

Before and after therapy, the erythrocytes' capacity to eliminate MDA was assessed. To ascertain the variations in these indicators' alterations under the impact of various medications, the D\M coefficient (% MDA degradation in erythrocytes \ MDA content) was computed concurrently.

CHAPTER III: RESEARCH RESULTS DISCUSSION.

Presently, symptomatic and pathogenetic effects are combined in the treatment of children with respiratory disorders that result from prenatal neural system injury. However, the prevalence of neurological disorders in young children has grown despite a decline in infant perinatal death. Thus, the creation of fresh, economical, and efficient rehabilitation techniques is still important.

The medication Vincamine is one of the most cutting-edge and promising ways to address the effects of prenatal nervous system injury. The mechanics of the psychobiological state are easily impacted by this "ecologically clean" technology. The intensity of metabolic processes, the respiratory and cardiovascular systems, brain tone, and blood circulation are all indirectly impacted by emotions, which dynamics invariably result in specific hormonal and biochemical changes. In light of this, we investigated how well Vincamine works for the restoration and rehabilitation of children with respiratory conditions brought on by the effects of prenatal neural system injury.

Twenty children (the main group) with respiratory conditions brought on by perinatal nervous system injury, ages three months to one year, were treated with Vinkamin and symptomatic medication under our care.

Ten children in the control group, who only got conventional care, had respiratory conditions brought on by perinatal nervous system injury. Traditional treatment included the use of pathogenetic medications that enhance the brain's energy supply, such as antibiotic therapy, angioprotectors that increase cerebral blood

flow (cinnarizine, cavinton), cerebroprotectors and nootropics (piracetam, nootropil, encephabol, actovegin, and cerebrolysin), and medications that decrease the production of cerebrospinal fluid (diacarb).

Traditional therapy was used to treat respiratory problems in a group of seven conditionally healthy children who did not experience the effects of prenatal nervous system impairment. The distribution of the children in the main group was as follows:

Ten children (50%) with neuroreflex excitability syndrome made up Subgroup 1.

Six children (30%) with vegetative dysfunction syndrome made up Subgroup 2.

Four children (20%) with psychomotor and verbal development delay syndrome made up subgroup 3.

The control group's kids were split up into:

Three children (21% of the total) in Subgroup 1 had neuroreflex excitability syndrome.

Four children (28.5%) in subgroup 2 had vegetative dysfunction syndrome.

3. Three children with psychomotor and verbal development delay syndrome (21%), who were part of subgroup

In other words, both groups were equivalent; all children in the control group were conditionally healthy, of the same age, and had conditions that were almost as severe as those in the main group.

According to our research, using Vincamine in conjunction with conventional therapy had a definite favorable therapeutic impact that limited the medication load and maximally stimulated neuroregulation and natural compensating mechanisms. Vincamine (7.5 mg daily during meals) was administered for 20 days to children in the first grouping who had neuro-reflex excitability syndrome, a condition caused by elevated neural excitability. Children in the second category, who had vegetative dysfunction syndrome, received 7.5 mg of Vincamine daily with meals for a month.

Vincamine was administered for one and a half months to children in the third

group, who had a condition of delayed psychomotor and verbal development. (7.5 mg daily with food.)

Table 3.5.

Dynamics of respiratory symptoms in children with neuro-reflex excitability syndrome.

Symptoms	traditional therapy	Use of Vincamine
1. emotional lability	6-7day (60%)	3-4day (100%)
2. loss of motor restlessness	9-10day (70%)	5-6day (100%)
3. loss of chin tremor	14-15 day (61%)	5-6day (80%)
4. loss of hand tremor	16-17day (72%)	7-8day (92%)
5. dry scattered wheezing on auscultatory hard breathing	7-8 day (82%)	5-4 day (88%)

As can be seen from Table 3.5, the disappearance of signs of emotional lability in children who received Vincamine occurred in 100% of cases within 3-4 days, and in the group of conventional therapy this figure was 60%. At the same time, movement disorders in children who received Vincamine disappeared within 5-6 days (100%), which is twice as fast as in patients in the control group. Children became calmer, more sociable, their respiratory rate, heart rate, and blood pressure normalized. Children began to fall asleep easily. In patients treated with Vincamine, chin tremor disappeared in 80-92% of cases within 5-6 days, while in patients with conventional therapy, these signs disappeared in only 61-72% of patients within 14-15 days. In 88% of patients, auscultatory vesicular breathing appeared on the 5-4th day of treatment.

For children with vegetative-visceral syndrome, Vincamine was used for 1 month (orally with food, 7.5 mg per day).

Table 3.6.

Dynamics of respiratory symptoms in children with vegetative dysfunction syndrome.

Symptoms	traditional therapy	Use of Vincamine
1. disappearance of regurgitation.	4-5 day (54%)	2-3day (81.6%)
2. disappearance of perioral hoarseness	4-5 day (70%)	2-3 day (95%)
3. disappearance of muscular dystonia	10-12 day (55-62%)	7-8 day (71-80%)
4. dry scattered wheezing against auscultatory hard breathing	8-9 day (80%)	5-4 day (88%)

Table 3.6 shows that the disease's symptoms have reversed by the conclusion of the period of therapy. Conventional therapy caused symptoms including regurgitation and loss of periocular crusting to go away on the fifth day for 54–70% of children, while Vincamine caused these symptoms to return to normal earlier, on the second or third day, for 81–95% of children. Muscular dystonia went away in 55–62% of the children in the control group on day 12, and in 71–80% of the children in the main group on day 8. Vegetative-vascular responses, hunger, and overall health all stabilized. 88% of children showed auscultative vesicular breathing by the fifth or fourth day.

Children with psychomotor retardation syndrome used Vincamine for 1.5 months (7.5 mg per day, orally, during meals).

Table 3.7.

Dynamics of respiratory symptoms in children with psychomotor retardation syndrome

Symptoms	traditional therapy	Use of Vincamine
1. Moderation of muscle tone	7-9day (42%)	5-6day (88%)
2. Improvement of communication with the environment (analyzer tasks)	12-15day (55%)	9-10day (72%)
3. Attempts to pronounce individual syllables	20-25day (41%)	12-15day (80%)
4. The appearance of small movements of the fingers	9-10 day(70%)	6-7day (86%)
5. Dry scattered wheezing against the background of auscultatory heavy breathing	7-8 day(82%)	5-4day (88%)

3.7. The table shows that kids who took Vincamine had better moods and started talking to people more readily, especially their mother. Muscle tone returned to normal on days five and six (88%), compared to 42% in the group of kids receiving standard treatment. Youngsters started to become interested in their surroundings, and they started to build appropriate imitation reactions. Additionally, there was an increase in the rate of speech development, as

evidenced by efforts to articulate individual syllables and active singing. Auscultation on the fifth and fourth days revealed vesicular breathing in 88% of cases. Therefore, our clinical findings demonstrate that Vincamine is a highly successful treatment for children with perinatal nervous system impairment during the recovery phase. To objectively assess the outcomes, all children had a 2-fold echoencephaloscopy both before and after using Vincamine, in addition to a neurological examination.

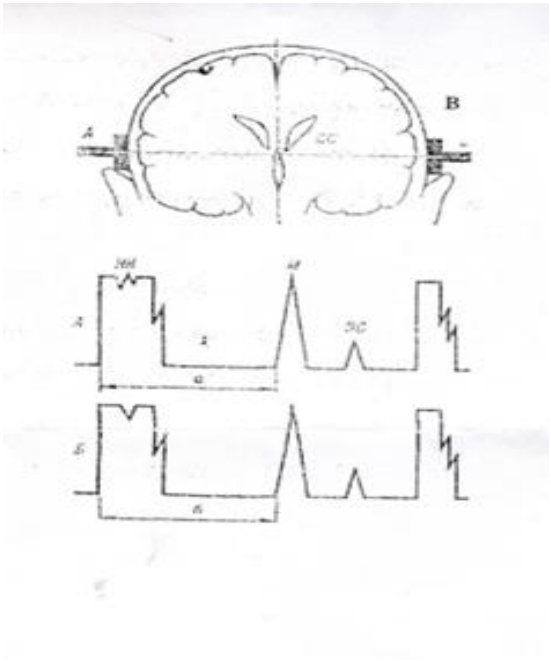
3.1. ExoEG markers of youngsters in good health

The existence of an initial complex, a final complex, an exosignal from intermediate structures, and several signals from non-intermediate brain regions are characteristics of a typical exoencephalogram.

An impulse generator and exo signals coming from the soft tissues of the head, skull bones, and superficial brain regions make up the initial complex (NK), a portion of the exoencephalogram. Exo signals from the soft tissues of the head, the inner surface of the skull bones, and the cranial-air components combine to generate the final complex; the exo signals from the inner surface of the skull bones are the most consistent.

Numerous pulses emerge between the two primary exoencephalogram complexes as a result of ultrasound reflection from different brain areas. A handful of pulses only manifest when a pathogenic condition is present in the brain, whereas others are more steady.

The echo signal from the brain's middle (M) structures (third ventricle) is the most consistent. The III ventricle typically has a width of 7-9 mm. The M-echo has a broader base, dominates other structures' impulses, and occasionally has two or more impulses. The M-echo is normally situated along the brain's midline, and it can be moved by little more than 1-2 mm.



Ultrasound sensors are positioned in the central regions (A-right, B-left) of the frontal part of the skull. O S (SS): the brain's third ventricle. BK-initial complex (NK). The intermediate structures' M-exosignal ES-different signals from the brain's non-middle regions. The KK-final complex

3.2. ExoEG markers of the primary group's children.

There were twenty kids in this cohort. The overall health, neurological status, and ExoEG nature gradually altered in children with respiratory disorders, the effects of prenatal nervous system injury, and those who underwent typical therapy, according to the research' findings. These values shifted faster in the direction of a favorable trend following the addition of Vincamine to the treatment mix. Compared to the control group, children with neuro-reflex excitability syndrome showed a substantial reduction in the number of impulses in their exogram. The third ventricle's breadth shrank by 8 mm.

In children with vegetative dysfunction syndrome, after the course of Vincamine, the wave pulsation on the exogram decreased. The number of impulses increased. The width of the III ventricle approached the normal range (7-9 mm).

In general, the results of the conducted studies allow us to conclude that complex rehabilitation with the use of the Vincamine preparation is effective for the consequences of perinatal damage to the nervous system and its concomitant diseases in patients with it. The advantages of this method are primarily due to the activation of natural mechanisms of regulation, which stimulate the regenerative

abilities of the brain. Having studied the functional changes in the central nervous system, when using the Vincamine preparation and recording exoencephalograms in children, we found that there are positive changes in the ExoEG and contribute to the faster normalization of neurological symptoms.

Rehabilitative opportunities are increased and the proportion of children with lingering effects of perinatal central nervous system injuries is considerably decreased when occipital neurological problems may be corrected. Making a diagnosis consequences of neuro-reflex excitability syndrome and neonatal nervous system injury.

(before treatment with Vincamine)

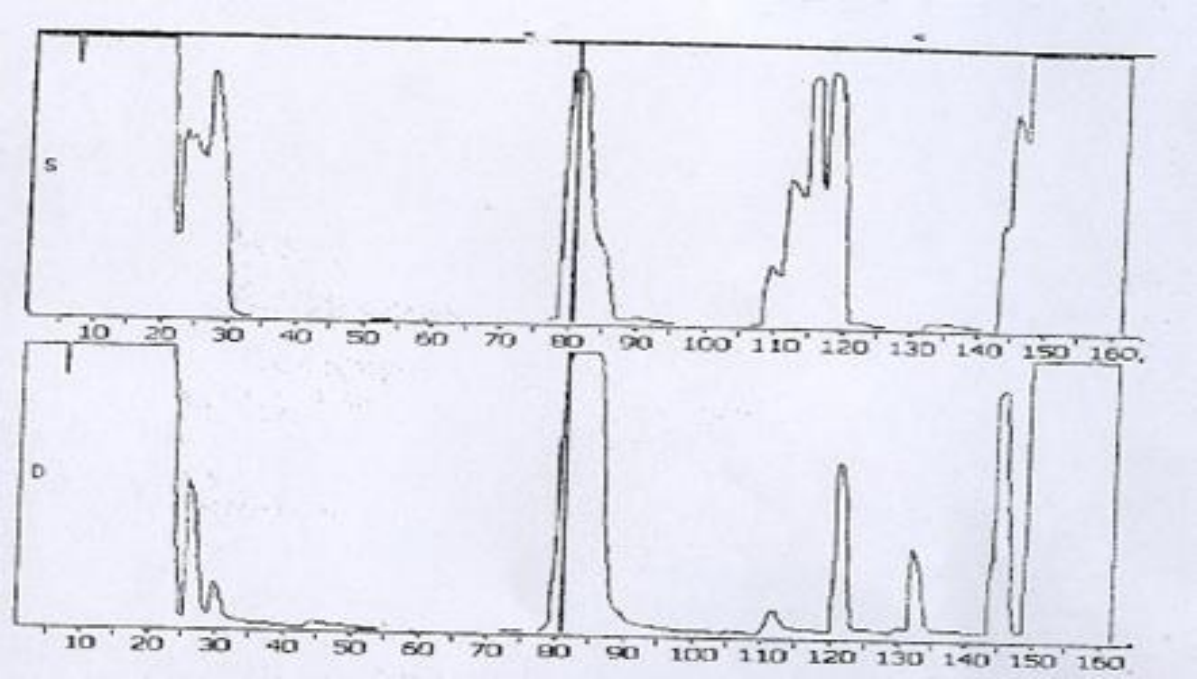


Figure 3.2.5: EXO-EG conclusion example. no medial structural displacement. V3-6 mm, SSIs 3.1 and SSId-3.0, dilatation percentages of 40% and 45%, respectively, indicate moderate dilatation of the brain's ventricular system.

(after receiving Vincamine therapy)

EXOENCEPHALOSCOPY

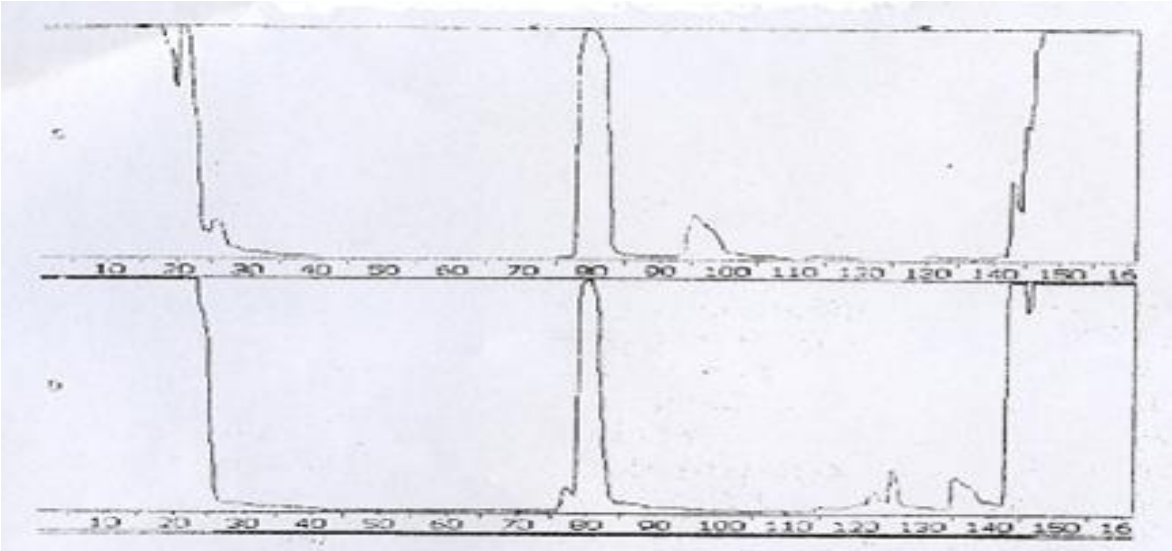


Figure 3.2.6. Example of EXO-EG conclusion. Displacement in the medial structures. The ventricular system of the brain is not dilated.

1.3. Children in the control group had their ExoEG markers evaluated.

Ten children in this group received antibiotic therapy and medication rehabilitation for respiratory conditions brought on by perinatal nervous system impairment. The exoencephalogram of children with neuroreflex excitability syndrome displays a large number of high-pitched, double impulses between the two major complexes. These impulses are created by ultrasonic reflection from different brain areas. The median M-exo is double and broad. Angiospasm causes a rise in pulse, whereas spasms cause a reduction. Following a 20-day medication regimen, intraventricular blood flow dropped to 9.5–10 mm. There was also a reduction in the quantity of impulses.

There was a modest suppression of the background rhythm in children with vegetative dysfunction syndrome. The frontal and superior directions (areas of greater vascular responsiveness and excitability) showed hypersynchronization of the β rhythm, indicating interzonal variations. Age-appropriate rhythms emerged during therapy, and hypersynchronization vanished. Oblique, sporadic waves were observed on the exoencephalogram. The pulse slowed. A handful of impulses. The III ventricle's breadth fell within the typical range (7-9). Positive dynamics of the exoEG image were seen following a 20-day course of therapy, albeit these changes

were negligible. It was discovered during these patients' surveillance that the metrics only fully returned to normal after the third month of therapy.

There was a residual backdrop of broad general brain alterations in children with a diagnosis of delayed psychomotor and verbal development. There are few waves and a mild pulse on the echoencephalogram. There were no more echo signals found.

Diagnosis: Syndrome of delayed psychomotor development, resulting from prenatal nervous system injury.

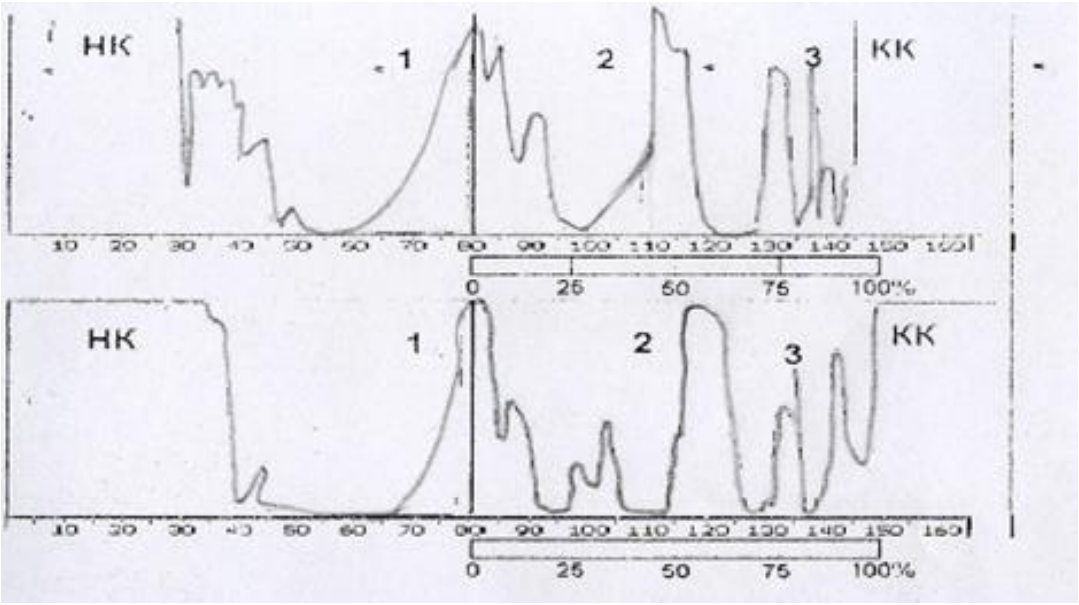


Figure 3.2.7: Hydrocephalic-hypertensive condition in an exoencephalogram 25% of the lateral ventricles are out of limits, the middle complex is expanded (1), the lateral ventricle peaks are clearly expressed (2), and the lateral peaks of the interphases of the deep sulci of the hemispheres are raised (3).

Intensive drug rehabilitation produced a very gradual improvement in this illness. Only four months following the initiation of therapy was full normalization observed.

3.4. Characteristics of the state of lipid peroxidation in erythrocytes in healthy children.

We examined the characteristics of LPO processes in seven youngsters in good health. The children in this group were born to moms who were in good health and who had gone through a normal pregnancy and delivery process. The youngsters were in acceptable overall health both at delivery and in their early days of life. The Apgar score was 8-10 points.At birth, the children in this group weighed between 3000 and 3800 grams. The internal organs did not exhibit any abnormal alterations. The neonates' physiological responses were adequate. Within three to four days, the umbilical chord in nearly every infant came off. On the first day of life, every infant was placed to the breast and aggressively sucked. The majority of the six children were breastfed at the time of the research.

Five of these children started receiving supplemental foods after the age of six months, whereas two of them were nursed exclusively until that time. One youngster was therefore receiving mixed feedings. All of the children in this group were active and showed an interest in their surroundings, which was indicative of their psychomotor development. Two youngsters between the ages of four and five months made "bumbling" noises, whereas children in their later years spoke in single syllables. The development of motor skills conformed to the age standard. On schedule, all youngsters got their prophylactic vaccines. Table 3.4.8 presents the findings from the analysis of LPO processes.

Table 3.4.8

Indicators of lipid peroxidation processes in healthy children.

№	Indicator	Difference
1.	Hemolysis of erythrocytes before incubation, %	1,1±0,09
2.	Hemolysis of erythrocytes after incubation, %	2,21±0,44
3.	Increase in hemolysis%	100
4.	MDA before incubation of erythrocytes, nmol\10^6	0,86±0,12
5.	MDA after incubation of erythrocytes, nmol\10^6	1,4±0,16

6.	MDA after incubation\hemolysis	0,5±0,1
7.	MDA degradation, %	1,3±0,1
8.	Degradation/MDA ratio	2,02±0,4

The table shows that the hemolysis of erythrocytes in this age group is 1.1 ± 0.09% before incubation and 2.21 ± 0.44% after incubation, which indicates the strength of metabolic activities in the cell. Peroxide hemolysis is therefore two times more than mechanical hemolysis, which supports the facts from the literature. Hemolysis has increased by 100 percent. The MDA content in healthy youngsters is 0.86 ± 0.12 nmol < 10^6 erythrocytes prior to incubation and 1.6 times higher (1.4 ± 0.16 nmol < 10^6 erythrocytes) following incubation.

The MDA to hemolysis ratio, which is 0.5±0.1% at a specific age, shows how well a cell can withstand the harmful effects of MDA. The strength of MDA degradation, which is 1.3 ± 0.1% in babies, and the breakdown ratio to MDA, which is 2.02 ± 0.4, are indicators of the control of lipid peroxidation processes. Our research on lipid peroxidation processes in healthy newborns demonstrates that this process has unique properties that may be employed as a control indication in sick children with a variety of diseases, including perinatal central nervous system lesions.

3.5. The state of lipid peroxidation in erythrocytes in children in the control group.

This group included 10 infants with the consequences of perinatal damage to the nervous system who received conventional therapy.

The results of the study are presented in Table 3.5.9.

Table 3.5.9.

LPO indicators in children in the control group

№	**Indications**	**When to Apply**	**Within 7-10 days of**

			treatment
1.	Hemolysis of erythrocytes before incubation, %	2,0±0,24	1,4±0,05* **
2.	Hemolysis of erythrocytes after incubation, %	3,2±0,4	2,3±0,03**
3.	Increase in hemolysis%	60	64
4.	MDA before incubation of erythrocytes, nmol\10^6	3,3±0,33	2,8±0,1* **
5.	MDA after incubation of erythrocytes, nmol\10^6	2,8±0,17	2,0±0,17* **
6.	MDA after incubation\hemolysis	1,6±0,39	1,0±0,07* **
7.	MDA degradation, %	0,87±0,16	1,28±0,28**
8.	Degradation/MDA ratio	1,6±0,44	1,96±0,5**

* Reliability indicator compared to data from healthy children (R<0.05)

** Reliability indicator compared to data from the first study (R<0.05)

The table shows that the hemolysis of erythrocytes prior to incubation in this group of children did not normalize (1.4 ± 0.05% against 1.1 ± 0.09% in healthy children), while being much lower than pre-treatment data (2.0 ± 0.24% versus 1.4 ± 0.05%, respectively).

Following incubation, the hemolysis of erythrocytes essentially matched the markers in children in good health (2.3 ± 0.03% and 2.21 ± 0.44%, respectively). Therefore, the hemolysis of erythrocytes after incubation did not differ from that of healthy children, even though the value of erythrocyte hemolysis was higher before incubation. In this sense, children in this group saw a substantially lower percentage increase in erythrocyte hemolysis than children in the healthy group (64 and 100%, respectively).

Prior to incubation, MDA levels were still high (0.86 ± 0.12 nmol / 10^6 erythrocytes against 2.8 ± 0.1 nmol / 10^6 erythrocytes in healthy youngsters),

although they tended to drop. When compared to pre-treatment data, the indicator indicated that treatment significantly reduced the MDA levels in this group of children, which were significantly higher than normal after incubation (2.0 ± 0.17 nmol < 10^6 erythrocytes compared to 1.4 ± 0.16 nmol < 10^6 erythrocytes in healthy children).
Following incubation, the MDA/hemolysis ratio was much lower than baseline (1.6 ± 0.39 vs 1.0 ± 0.07), although it was still higher than in healthy children (1.0 ± 0.07 versus 0.5 ± 0.1).

The degree of MDA degradation was not different from the indicators of the children in the healthy group, although it was substantially higher than baseline (1.28±0.28% and 0.87±0.16%). The degradation/MDA ratio showed the similar alterations before and after treatment: 1.6±0.44 and 1.96±0.5, respectively. Therefore, there is a notable positive dynamic in the indicators of LPO processes in children with the effects of prenatal nervous system injury who underwent standard therapy, even if the majority of them fall short of the norm.

In terms of clinical symptoms, positive dynamics were also observed: the children's sleep returned to normal, their intracranial pressure progressively dropped, and their internal organ functions stabilized. The kids started gaining weight as their appetites increased. There was a keen interest in the environment. Generally, after 10 to 15 days of therapy, a clinically meaningful improvement was seen.

3.6. The state of lipid peroxidation processes in erythrocytes in children of the main group

Twenty children with the effects of perinatal nervous system injury were in this group. The condition of lipid peroxidation in erythrocytes was examined in the City Children's Hospital No. 1 department, and Vincamine, which was given at a dose of 7.5 mg daily, was used as part of a treatment plan. Additionally, medications that raise intracranial pressure and decrease the formation of cerebrospinal fluid were employed.

Vincamine is a neurometabolic substance that occurs naturally. Vincamine's oxygen saturation action enhances brain tissue's capacity to bind and use oxygen; venous blood's carbon dioxide concentration rises, increasing blood flow to the afflicted area's collateral network via a secondary vasoregulation process. never results in "steal" syndrome. lowers cerebral vascular resistance, especially in the precapillary and arterial regions. fixes problems with phosphate content and glucose metabolism.

Analyzing the data collected before to therapy revealed alterations that point to a serious lipid peroxidation imbalance and, therefore, profound metabolic abnormalities at the cellular level. Table 6 illustrates how the indicators of mechanical and peroxide hemolysis, the amount of MDA in cells, and a decline in the cells' capacity to tolerate the harmful effects of MDA dramatically rise in children who suffer from the aftereffects of perinatal nervous system damage. This is reflected in an increase in the hemolysis coefficient following MDA incubation. Simultaneously, a notable reduction in the severity of MDA degradation and a rise in the hemolysis % were observed.

The study of changes in LPO processes was carried out after the end of the course of treatment with Vincamine. The results of the study are presented in Table 3.6.10.

Table 3.6.10

Indicators of lipid peroxidation processes after the use of Vincamine.

№	**Indicators**	**Before treatment**	**After treatment**
1.	Hemolysis of erythrocytes before incubation, %	2,0±0,24*	1,17±0,12***
2.	Hemolysis of erythrocytes after incubation, %	3,2±0,4*	2,37±0,16**
3.	Increase in hemolysis%	60	99,7

4.	MDA before incubation of erythrocytes, nmol\10^6	3,3±0,33*	2,7±0,24* **
5.	MDA after incubation of erythrocytes, nmol\10^6	2,8±0,17*	1,6±0,18** ***
6.	MDA after incubation\hemolysis	1,6±0,39*	0,9±0,2* **
7.	MDA degradation, %	0,87±0,16*	1,24±0,13**
8.	Degradation/MDA ratio	1,6±0,44*	2,08±0,5**

* Reliability index compared to data from healthy children (R<0.05)

** Reliability index compared to the original study data (R<0.05)

*** Reliability index compared to data from the control group (R<0.05)

The table shows that the hemolysis of erythrocytes before incubation in this group was significantly lower than in the control group and did not differ from the data obtained in healthy children (1.4₱0.05% and 1.17±0.12%, respectively). There was no statistically significant difference between the hemolysis of erythrocytes after incubation in children with the consequences of perinatal damage to the nervous system treated with Vincamine and the data obtained in healthy children and children of the control group (2.37±0.16%, 2.21±0.44%, and 2.3±0.03%).

The proportion of enhanced hemolysis brought on by the normalization of peroxide and mechanical hemolysis was much greater than that of the control group (99.7% and 64%, respectively) and did not deviate from the results of children in good health. Children in the main group had the same MDA content prior to incubation as those in the control group. In other words, even after receiving Vincamine treatment, children with PEP had a considerably greater MDA content prior to incubation than children in good health (2.7^{i}0.24 nmol<10^6 erythrocytes as opposed to 0.86^{i}0.12 nmol<10^6 erythrocytes). This indicator does, however, show a notable decline from the original data (2.7₱0.24 nmol\10^6 erythrocytes vs 3.3±0.33 nmol\10^6 erythrocytes).

Following incubation, Vincamine had a favorable impact on the MDA content. There was no statistically significant difference between this indicator and the norm (1.6±0.18 nmol<10^6 erythrocytes and 1.4i0.16 nmol<10^6 erythrocytes). Simultaneously, the MDA content in the control group (2.0±0.17 nmol‡10^6 erythrocytes and 1.6i0.18 nmol‡10^6 erythrocytes, respectively) was substantially higher than that in the main group of children following incubation. Following incubation, the MDA/hemolysis ratio was substantially higher than normal values (0.9±0.2, 1.0±0.07, and 0.5±0.1, respectively) and did not vary from the findings of the control group.

The main group's children's MDA degradation intensity was 1.24²0.13%, 1.2810.28%, and 1.3±0.1%, respectively, and did not vary significantly from the results collected in the control group. Comparing the degradation/MDA ratios in these groups (2.08±0.5, 1.96±0.5, and 2.02±0.4, respectively) clearly reveals this. Therefore, Vincamine therapy helps to correct the majority of lipid peroxidation markers in infants with perinatal central nervous system injury.

This is demonstrated by a notable reduction in erythrocyte hemolysis following incubation, a normalization of the hemolysis percentage increase, the maintenance of a normal level of MDA degradation intensity, and a notable decrease in MDA following incubation (albeit without its normalization). However, we also discovered that the MDA content before to incubation was unaffected by Vincamine treatment.

Additionally, there was a definite upward trend in clinical signs: kids grew more relaxed and energetic. Normalized sleep. Intracranial pressure dropped significantly more quickly against the backdrop of Vincamine usage (7–10 days versus 10-15 days in the control, depending on the beginning growth rate). Children started gaining weight, the gastrointestinal tract's function improved, and the pulse and breathing rate normalized.

Regardless of the administration of antipyretic medications, the temperature dropped or returned to normal in children who had previously had an inexplicable fever. Youngsters started to actively engage with their environment, and they

tended to acquire motor skills. The sixth through tenth days of therapy were often when the most noticeable clinical impact was seen.

FINAL RESULTS

In the framework of prenatal disorders, perinatal abnormalities of the central nervous system have a prominent position and impact an individual's entire future existence. The incidence of children with central nervous system perinatal lesions has been rising in recent years. Numerous ensuing somatic and psychoneurological disorders may arise, contingent on the extent and trajectory of the damage. Hypoxic-ischemic, metabolic, toxic, viral, and traumatic events are among the causes of perinatal diseases of the central nervous system that occur throughout the prenatal, intranatal, and postnatal stages. The main pathogenetic causes are hemodynamic abnormalities and hypoxia to varied degrees.

In children, almost 60% of all nervous system disorders are caused by perinatal brain injury. Depending on the intensity and course of the injury, a wide range of future psychoneurological and somatic illnesses might occur. The prenatal, intranatal, and early neonatal phases are all included in the perinatal period. The start of labor marks the conclusion of the prenatal period, which starts at 22 weeks of fetal development. The time between the start of labor and the child's birth is referred to as the intranatal phase. Early neonatal (the first week of the child's life) and late neonatal (the eighth to the twenty-eighth day of life) are the two divisions of the neonatal period.

Fetal asphyxia is linked to the majority of perinatal diseases. Changes in cerebral hemodynamics accompany perinatal hypoxia and asphyxia, and this is presently the most widely accepted theory on the pathophysiology of CPDNS in neonates. Ischemia of the brain tissue (arterial hypotension with a pressure of less than 50 mm Hg at the elbow, thrombosis of venules and arterioles, impaired

venous outflow due to cerebral edema), dyselectrolyteemia, and minor hemorrhages in the brain tissue and subarachnoid space are the main causes of hypoxic encephalopathy, the most severe clinical manifestation of severe asphyxia.

Such hemorrhages are not a significant predictor of outcome during the acute phase or for the psychoneurological development that follows. In addition to causing hyperperfusion of the heart, hemorrhagic and tissue illnesses that are accompanied by hypoxia also contribute to the development of ischemic necrosis of the endocardium and capillary muscles, which raises arterial hypotension. Along with arterial hypotension, hypoxia also keeps the pulmonary arteries' resistance high, which causes pathological shunting (from right to left through the arterial tract and the foramen ovale). Different pathways of brain injury are predetermined by the polyetiology of encephalopathy in neonates.

One of these is a reduction in cerebral blood flow, which might be brought on by prenatal hypoxia. Slower cerebral capillary formation, increased permeability and fragility, and an increase in cell membrane permeability are also associated with this condition. The development of intracellular lactic acidosis and neuronal death are hallmarks of cerebral ischemia, which takes place against the backdrop of growing metabolic acidosis.

The decrease in cerebral blood flow is influenced by the disruption of the autoregulatory mechanisms of cerebral blood flow. In healthy children, cerebral blood flow and intracerebral pressure are relatively stable and do not depend on changes in blood pressure (BP). In children who have experienced hypoxia, the autoregulatory mechanisms of cerebral blood flow are reduced (moderate hypoxia) or completely disappear (severe hypoxia), and cerebral blood flow becomes dependent on fluctuations in BP. In addition, in children who have experienced hypoxia, the cardiac output decreases (hemodynamic disturbances and hypoxic damage to the myocardium), blood pressure decreases, venous flow in the brain is impaired, and due to hypoxic damage to the endothelium, the resistance of blood vessels in the brain itself increases, which leads to a sharp narrowing of the capillary lumen.

Vasogenic cerebral edema arises in the context of decreased cerebral blood flow and impaired ADG production (asphyxia-excess production, hypoxia-(secretory)production deficit syndrome). Vasogenic cerebral edema develops as a result of decreased cerebral blood flow, and cytotoxic edema is brought on by the release of "excitatory" amino acids, mainly glutamate.

Reduced glucose supply to neurons; inhibition of ATP and creatine phosphate synthesis; disruption of cell membrane pump function; depolarization of outer membranes; excessive glutamate release into the interstitium and inadequate neuronal absorption of it impact neuronal receptors, opening channels that allow sodium and calcium to enter the cell. Cell edema develops when sodium pulls water with it, while cellular calcium necrosis develops when calcium enters cells too readily.

The buildup of hydroperoxides, aggressive free radicals that damage neuronal membranes, interferes with lipid peroxidation under hypoxia (9, 19). Extremely reactive, free oxygen radicals harm DNA, mitochondria, and cell membranes. Their presence is indicative of the severity of a long-standing process, and they might be regarded as destructive agents that cause overall harm to nerve tissue. The detrimental effects of promoting lipid peroxidation events mainly impact the condition of all cell membranes, without exception, as NYK, the primary substrate of lipid peroxidation, is an essential part of every biological membrane.

Since erythrocytes' membranes differ in some ways and have a lot of similarities with other biological membranes in the body, it is most practical to examine changes in the structural and functional characteristics of cell membranes at the erythrocyte level in clinical settings. Based on a rise in the quantity of lipid peroxidation products, some studies make inferences on the severity of lipid peroxidation in neonatal cerebral circulation problems. According to Shilyaev, erythrocyte membranes with cerebral circulation disorders exhibit increased lipid peroxidation and altered rheological characteristics, including decreased erythrocyte deformability and a breach of surface architectonics, which are linked to the severity of the disorders.

By combining the findings of several writers, we can say that MDA levels rise as erythrocyte membranes' antioxidant defenses deteriorate. However, as the quantity of metabolites relies on both the rate of breakdown and excretion of the metabolite from the cell as well as the degree of secretion, their accumulation does not necessarily indicate the intensity of the activity. In this instance, a more accurate measure of the severity of LPO may be the MDA accumulation coefficient, which is the ratio of the MDA content to the degree of erythrocyte hemolysis. The only way to achieve a more thorough evaluation of pathogenic alterations in plasma membranes is to use an integrated strategy, which combines many research techniques.

It is well recognized that LPO indications describe the degree of the diseased process as well as, to a lesser degree, how well the compromised metabolism is being corrected. Numerous medications are utilized to treat metabolic and microcirculatory abnormalities in perinatal central nervous system injuries, with the goal of restoring the functional condition of cells.

Vincamine is a novel herbal medication that has shown promise in treating metabolic and circulatory brain diseases in a number of recent experimental and clinical investigations. Nevertheless, there are no research examining how this medication affects perinatal nervous system lesions in infants under a year old, and the best dosage and usage period have not been established.

Vasodilator medications have long been used to try to increase the blood flow to the brain tissue in conditions linked to cerebrovascular lesions, but this frequently results in a deadly blood circulation imbalance. In actuality, the ischemia area here causes an unnecessary increase in blood supply since the pharmacological impact was more pronounced in a healthy area. It is important to remember that improving cerebral blood circulation is largely a metabolic process as well as a hemodynamic one; selective vasodilation must be used to enhance neurons' capacity to both generate glucose and absorb oxygen. This dual pharmacodynamic action provides Vincamine's utility.

Vincamine, a medication with vascular modulating, antihypoxic, and neurometabolic effects, was therefore utilized selectively. Being a natural herb, Vincamine is safe to use over an extended period of time. Additionally, Vincamine was chosen due to its high absorption and lack of negative effects. By promoting glucose oxidation, Vincamine increases energy generation and stimulates brain activity in general, improving brain metabolism. Vincamine lowers and stabilizes the peripheral resistance of cerebral blood arteries while increasing the oxygen delivery to neurons under hypoxia.

In nature, Vincamine is a neurometabolic plant. The literature claims that Vincamine's oxygenating effect enhances the brain tissues' capacity to bind and use oxygen. It also raises the amount of carbon dioxide in venous blood, which, via a secondary vasoregulation mechanism, increases the blood supply to the affected area's collateral network never results in "steal" syndrome. lowers cerebral vascular resistance, especially in the precapillary and arterial regions. fixes problems with phosphate content and glucose metabolism. Based on variations in the brain's capacity to absorb oxygen, the respiratory coefficient, glucose consumption, and the amount of arteriovenous lactic acid, Vincamine has a beneficial metabolic impact in syndromes that arise against the backdrop of cerebral arteriopathy.

According to the data, Vincamine is an activator of metabolism in brain tissues. It improves impaired metabolism in noradrenergic and 5-HT receptors; it increases the activity of noradrenergic neurons linked to behavioral reactions, attention, and memory; it increases the brain tissues' utilization of glucose; and it helps to raise brain activity levels overall. Additionally, Vincamine activates phosphorylation processes in AMF and ADF, improves oxidative processes in mitochondria, raises the oxygen content in brain tissues and neurons by 23%, and protects metabolic activity for a long period of time following hypoxia (3 months).

An equally significant characteristic is the drug's selective vasoregulation effect, which reduces and stabilizes the brain's peripheral vascular resistance, improves vascular tone, and relieves artery spasm. It also helps to adjust cerebral blood flow to the brain's metabolic requirements. According to the results of the literature

study, hypoxic vascular injury continues to be the most common cause of disorders affecting the newborn's nervous system, with perinatal encephalopathy being the most severe.

Accordingly, the emergence of conditions like cerebral palsy, epilepsy, and minor brain malfunction calls for the creation of novel medications to improve the lives of these kids and their parents. We think there may be promise in using Vincamine in this way.

Vincamine's efficacy in treating adults and adolescents with a variety of brain disorders is well documented in the literature, but its application in treating children with CPDNS is little discussed. Based on tracking improvements in the child's overall health and the ExoEG score, we made the decision to close this gap by investigating the efficacy of Vincamine in perinatal nervous system injuries.

37 children with respiratory conditions, ages 3 months to 1 year, were admitted to Samarkand City Children's Hospital No. 1 while we were in charge of them. The primary diagnosis for all children admitted to the hospital was severe respiratory virus infections. Three sizable groups were formed from the analyzed children: Ten children make up the control group, twenty make up the major group, and seven make up the healthy group.

Ten children with respiratory disorders with CPDNS who got conventional therapy made up the control group, whereas twenty children with CPDNS respiratory diseases who received Vincamine made up the main group. Seven children with respiratory conditions who did not have CPDNS and were treated with traditional methods made up the conditionally healthy group.

Boys outnumbered girls in the patient distribution by gender, with 17 (45.9%) compared to 20 (54%).

Our clinical diagnosis of CPDNS was based on the categorization used by the Research Institute of Pediatrics, TashVMOI, and the Department of Child Neurology. The somatic status, peripheral blood, and urine composition of children with perinatal brain damage were studied in addition to the neurological status. Instrumental techniques (ECG, chest X-ray, fundus examination) were also

employed. A pediatrician, neuropathologist, ophthalmologist, and ENT expert evaluated each kid.

To assess the rise in the intraventricular complex, all children had their brains exoencephalogrammed for diagnostic purposes. Along with evaluating the children's neurological and physical conditions, we investigated the following parameters that describe the LPO processes and the intensity of oxidative processes in the erythrocyte membranes: the degree of hemolysis of the erythrocytes prior to incubation (mechanical stability of erythrocytes) and following incubation under physiological conditions (initiated peroxide hemolysis); the amount of malondialdehyde (MDA) before and after incubation; the degree of MDA degradation; and the ratio of the percentage of MDA degradation to MDA content (D/MDA).

Anxiety was the most frequent complaint made by moms during the child's evaluation, occurring in 92.5% of instances. When respiratory illnesses with CPDNS developed, iron deficiency anemia (87.4%) and rickets (29.7%) were common background conditions. Every kid in the study had an adverse premorbid history; 20 (54.5%) had primary anemia, 15 (40.5%) had secondary anemia, and 2 (5.4%) had tertiary anemia.

Most children's overall health (33) 73% was rated as moderate to severe. Within two to three days, the general health of the three remaining (8.1%) children who were admitted to the hospital improved to a moderate level due to the effects of the underlying illness therapy. When youngsters were examined, their neurological condition was found to be abnormal. The following findings were derived from a summary of clinical and instrumental data: neuroreflex excitability syndrome was the most prevalent, occurring in 13 children (43.3%), followed by vegetative dysfunction syndrome in 10 children (33.3%) and psychomotor development delay syndrome in 7 children(23,3 %). Constant regurgitation, chronic hypotrophy, respiratory rhythm abnormalities, skin color, acrocyanosis, bradypnea, gastrointestinal dysfunction, thermoregulation issues, and occipital hair loss were the primary alterations associated with vegetative dysfunction syndrome.

A youngster with a condition of enhanced neuro-reflex excitability was shown to have emotional lability and movement abnormalities that arise with slight changes in the environment when visual, auditory, and tactile analyzers are disturbed, all against the backdrop of normal mental and physiological development. Periodically, these kids had twitches, small-amplitude tremors, trouble falling asleep, and shallow, restless, brief sleep.

The symptoms of ordinary (banal) bronchitis included an increase in body temperature, a dry, painful cough that lasted for three to four days before changing to a wet cough, clear lung sounds on percussion, and dry, diffuse wheezing against the background of heavy breathing. Psychomotor developmental delay syndrome is characterized by a decrease in unconditioned innate reflexes, children who did not show active interest in toys and surrounding objects, children who did not respond adequately to their mother's presence, children who were unable to make sufficient and sustained noise, manipulation with objects was delayed, and there was no active attention to objects.

Vitamin preparations and diuretics were employed according to the clinical signs that predominated. Treatment of conditions brought on by background illnesses and respiratory conditions was also done concurrently. Presently, symptomatic and pathogenetic effects are combined in CPDNS therapy methods for pediatric respiratory disorders. However, the prevalence of neurological disorders in young children has grown despite a decline in infant perinatal death. Thus, the creation of fresh, economical, and efficient rehabilitation techniques is still important.

The medication Vinkamin is one of the most cutting-edge and promising treatments for CPDNS. The mechanics of the psychobiological state are easily impacted by this "ecologically clean" technology. The intensity of metabolic processes, the respiratory and cardiovascular systems, brain tone, and blood circulation are all indirectly impacted by emotions, which dynamics invariably result in specific hormonal and biochemical changes. Twenty children (the main

group) with respiratory conditions and CPDNS, ages three months to one year, underwent courses of Vinkamin and symptomatic therapy under our supervision.

Ten children with CPDNS-related respiratory disorders who simply received standard treatment made up the control group. Pathogenetic medications that enhance the brain's energy supply, such as cerebroprotectors and nootropics (piracetam, nootropil, encephabol, actovegin, and cerebrolysin), angioprotectors that enhance cerebral blood flow (cinnarizine, cavinton), medications that decrease fluid production (diacarb), and antibiotic therapy were all part of conventional therapy.

Seven children with respiratory issues who were otherwise healthy but did not have CPDNS were treated with traditional respiratory therapy.

The distribution of the children in the main group was as follows: 10 children (50%) in subgroup 1 had neuroreflex excitability syndrome, 6 children (33.3%) had vegetative dysfunction syndrome, and 4 children (20%) had psychomotor and speech development delay syndrome.

Three children (21%) in Subgroup 1 of the control group were found to have neuroreflex excitability syndrome. Four children (28.5%) in subgroup 2 had vegetative dysfunction syndrome. Three kids (21%) in subgroup 3 exhibited psychomotor and verbal development delay syndrome. Mothers' most common complaints during the child's examination were hand tremor in 25.7% of instances, chin tremor in 31.3% of cases, and restlessness, which manifested as regurgitation in 92.5% of cases.

Mothers report that emotional instability, twitching while sleeping, chin and hand tremor, trouble falling asleep, short-term, shallow sleep, and frequent head tilts back are the primary symptoms of children with neuroreflex excitability syndrome. IVH: up to 10 mm. Children with autonomic dysfunction syndrome typically complain of acrocyanosis, erratic behavior, tension, anxiety, and IVH more than 10 mm.

Lagged behind their classmates in mental and verbal development were the primary concerns of children with psychomotor developmental delay syndrome.

Later, the kids in this group started to sit, stand, walk, turn around, and hold their heads.

In youngsters of all groups, on inspection, the overall condition was of moderate severity. The skin was clean, warm, and pale. Of the youngsters, 12 (32.4%) had acrocyanosis. Ten youngsters (27%) had a poorly formed subcutaneous fat layer. During auscultation, dry scattered wheezing was noted against the background of forceful breathing. Children with severe anemia were found to have functional murmurs, and nearly all children had muffled heart sounds from the cardiovascular system. The majority of kids had elevated heart rates.

Regarding the gastrointestinal tract, among the children in the second group, five (13.5%) had regurgitation, three (8.1%) experienced vomiting, and four (10.8%) experienced regular constipation. On inspection, increased bowel motions, rumbling and heaviness were detected. The stool had no pathological contaminants, but it was asymmetrical.

No gastrointestinal tract pathological symptoms were found in the other kids. There was no discomfort and the abdomen was soft. The spleen was not palpable, and the liver protruded 2 cm above the costal border. Unaltered, the feces produced.

Against the backdrop of typical mental development, the central nervous system of children with neuro-reflex excitability syndrome showed the following symptoms: chin tremor, periodic, small-amplitude hand tremor, emotional lability, and restlessness of movement that happens with slight environmental changes. There was an increase in spontaneous Moro reflexes and innate reflexes.

There was a delay in the development of Robinson, Babkin, and cervical-tonic reflexes in children with vegetative-visceral dysfunction syndrome, as well as muscular hypertonia. There was a reduction in tendon reflexes and spontaneous activity.

Impaired static-motor capabilities were seen in children with a diagnosis of delayed psychomotor development originating from the central nervous system. There was no response to the mother's voice, no aural attention, no sobbing or

screaming, youngsters searched their eyes for the source of the sound without moving their heads, and occasionally a meaningless smile was seen. Active attention was absent. There was a decrease in unconditioned innate responses.

We used exoencephalography to investigate the psychosomatic processes brought on by Vincamine usage and to evaluate the medication's efficacy. The EXO 12 ultrasound equipment was used to conduct an exoelectroscopic examination based on City Children's Hospital No. 1. The patient doesn't need to prepare in any way for the study. The patient often lies down during an exoEG procedure.

The ultrasonic sensor's working surface is applied to various areas of the child's head in succession after being coated with vaseline oil to guarantee acoustic contact. After being transformed into electrical impulses, the ultrasound signals show up as a curve on the device's screen. This is known as an exoencephalogram, and it is captured on camera and examined. In an area where the best circumstances are formed for receiving an exo signal, the sensor is positioned on the lateral surface of the skull, four to five centimeters above the external auditory canal along the biauricular line that passes across the temporal region. According to our research, using Vincamine in conjunction with conventional therapy has a clear favorable therapeutic impact that helps to reduce the medication load and optimally promote natural compensation mechanisms and neuroregulation processes.

Vincamine (7.5 mg daily between meals) was taken for 20 days by children in the first subgroup, neuro-reflex excitability syndrome, which is caused by increased neurological excitability. Children in the second category get 7.5 mg of Vincamine daily with meals for one month in order to treat vegetative dysfunction syndrome.

Vincamine was administered for one and a half months to children in the third group, who had a condition of delayed psychomotor and verbal development. (7.5 mg daily with food.) Following Vincamine's addition to the treatment combination, these parameters showed a quicker upward trend. The exogram showed a substantial reduction in impulses in children with neuro-reflex

excitability syndrome as compared to the control group. The third ventricle's breadth shrank by 8 mm.

Following Vincamine, the wave pulsation on the exogram in children with vegetative dysfunction syndrome diminished. There were more pulses now. The III ventricle's breadth (7-9 mm) was within acceptable bounds.

Following Vincamine treatment, the exogram of children with psychomotor developmental delay syndrome displayed better pulsation and more frequent waves. There were big waves. The III ventricle's breadth (7-9 mm) was within acceptable bounds. Hemolysis of erythrocytes before incubation is 1.1 ± 0.09% in this age group, whereas hemolysis of erythrocytes after incubation indicates the strength of metabolic processes in the cell and is 2.21 ± 0.44%, according to the results of the research of LPO processes in healthy youngsters. Accordingly, peroxide hemolysis is twice as high as mechanical hemolysis, a conclusion supported by evidence from the literature. Hemolysis has increased by 100 percent.

The MDA concentration in healthy youngsters is 0.86±0.12 nmol\10^6 erythrocytes before to incubation and 1.6 times higher (1.4±0.16 nmol\10^6 erythrocytes) following incubation. The MDA to hemolysis ratio, which is 0.5±0.1% at a specific age, shows how well a cell can withstand the harmful effects of MDA. The degree of MDA degradation in babies is 1.3 ± 0.1%, and the ratio of MDA to hemolysis is 2.02 ± 0.4, which both demonstrate the control of lipid peroxidation processes.

The information gathered from the investigation of lipid peroxidation processes in healthy newborns demonstrates that this process has unique properties that may be utilized as a control in ill children with a variety of diseases, including those who have perinatal central nervous system lesions.
The findings from the control group, which consisted of kids with CPDNS symptoms getting traditional treatment, was especially intriguing. Although the majority of LPO processes fall short of the standard, it was discovered that there is a notable positive dynamic. Erythrocyte hemolysis prior to incubation did not normalize (1.1±0.09% against 1.4±0.05% in healthy children), despite a

considerable drop from pre-treatment results (2.0±0.24% versus 1.4±0.05%, respectively).

Children in this group had a substantially lower proportion of erythrocyte hemolysis than children in the healthy group (64 and 100%, respectively). The MDA level was high prior to incubation (2.8±0.1 nmol\10^6 erythrocytes, compared to 0.86±0.12 nmol\10^6 erythrocytes in healthy children), however it tended to drop before incubation. Children in this group had considerably greater MDA after incubation than normal (2.0±0.17 nmol\10^6 erythrocytes compared to 1.4±0.16 nmol\10^6 erythrocytes in healthy children), although therapy helped to dramatically lower this indication when compared to pre-treatment data.

Following incubation, the MDA/hemolysis ratio was much lower than the initial results (1.6 ± 0.39 vs 1.0 ± 0.07), although it was still higher than in healthy children (1.0 ± 0.07 versus 0.5 ± 0.1). The degradation/MDA ratio showed similar changes before and after treatment: 1.6 ± 0.44 and 1.96 ± 0.5, respectively. The medication Vinkamin was prescribed to normalize LPO values based on the data that was collected. Most lipid peroxidation markers returned to normal after receiving Vinkamin treatment for children with perinatal central nervous system injuries.

This is demonstrated by a notable reduction in erythrocyte hemolysis following incubation, a normalization of the hemolysis percentage increase, the preservation of the MDA degradation intensity at a normal level, and a notable decrease in MDA following incubation (albeit without its normalization). However, we also discovered that the MDA content before to incubation was unaffected by Vincamine treatment.

Additionally, there was a definite upward trend in clinical signs: kids grew more relaxed and energetic. Normalized sleep. Intracranial pressure dropped significantly more quickly (7–10 days versus 10-15 days in the control group, depending on the starting development rate) against the backdrop of Vinkamin usage. Children started to gain weight, their respiration and pulse became regular, and their gastrointestinal system activity normalized. Regardless of the

administration of antipyretic medications, a drop in temperature or return to normal was noted in children with a history of unexplained fever. Youngsters started to actively engage with their environment, and a propensity to acquire motor skills emerged. Generally speaking, the sixth to tenth day of therapy showed a noticeable clinical improvement.

Overall, the findings of the research enable us to draw the conclusion that patients with CPDNS and its effects on concurrent respiratory disorders benefit from intensive rehabilitation using the medication Vinkamin. The benefits of this approach are mostly attained by triggering the brain's innate regulatory processes, which promote brain regeneration.

Using the medication Vincamine and recording children's exoencephalograms, we examined the functional alterations in the central nervous system and discovered that ExoEG shows favorable changes and helps neurological symptoms return to normal more quickly. Research has indicated that both the normalization of lipid peroxidation parameters and the administration of Vincamine to children with perinatal central nervous system injuries help to hasten the normalization of neurological symptoms. The percentage of children with lingering issues from perinatal central nervous system injuries is greatly decreased because to Vincamine's capacity to treat neurological illnesses.

References.

1. Azimova, X. M. Delayed psychomotor development in young children / X. M. Azimova // Neurology. —2006. — No. 4. — Pp. 56—57.
2. Aleksandrov M. V. The effect of hypoxic hypoxia on spontaneous activity of the human brain / M. V. Aleksandrov, A. O. Ivanov, N. I. Kosenkov // Human Physiology. — 2001. — Vol. 27, No. 6. — P. 58
3. Antibacterial agents in pediatrics / L. Karen et al. // Infect. Dis. Clin. N. Am. -2004. Vol. 18. - P. 513-531.
4. Astasheva I.B. Fulminant retinopathy of prematurity ("plus-disease"): incidence, risk factors, diagnostic criteria, and variations in course / I.B. Astasheva, E.I. Sidorenko // Vestn Oftalmol. 2002. - Vol. 118(6). - P. 5-9.
5. Aleksandrova V. A. Perinatal damage to the central nervous system and its consequences in children. Textbook / V. A. Aleksandrova. — St. Petersburg: Publishing House of St. Petersburg State Medical Academy, 2003. — 48 p.
6. Alifanova L. A. Implementation of a systematic approach to health preservation and harmonious development of schoolchildren / L. A. Alifanova // Pediatrics. — 2007.—No. 2. —Pp. 106—110.
7. Akinina Z.F. Long-term consequences of perinatal damage to the central nervous system in children: dissertation for the degree of Candidate of Medical Sciences. Barnaul, 2004. - 125 p.
8. Aylamazyan E.K. Molecular neuroimmunendocrinology: role and significance in the regulation of reproductive function / E.K. Aylamazyan, I.M. Kvetnoy, // Journal of Obstetrics and Gynecology. 2003. Vol. 1-2, No. 4. pp. 4-11.
9. Assessment of the effectiveness of treatment of children in the early recovery period of perinatal hypoxic brain damage / E.A. Bobrova et al. // Bulletin of New Medical Technologies. 2005. - No. 2. - P. 31-33.
10. Arunkumar, R.S. Acute pneumonia in children in southern India: abstract of thesis for the degree of Candidate of Medical Sciences. Moscow, 2006: 17 p.

11. Autenchlus A.I. On the sensitivity of lymphocytes to thymus hormones in children with pathologies of the central nervous system of various origins / A.I. Autenchlus, O.V. Ivanova; V.G. Degtyareva, A.A. Kosacheva // Immunology. 2002. - No. 3. - P. 181-185.
12. Barashnev Yu. I. Perinatal neurology / Yu. I. Barashnev. — Moscow: Triada-Kh, 2001. — 638 p.
13. Barker R. Visual Neurology / R. Barker, S. Barazi, M. Neil. — Moscow: GEOT AR-Media, 2005. — 140 p.
14. Bakanov M.I. New biochemical criteria for the diagnosis and prognosis of perinatal CNS damage in newborns / M.I. Bakanov, V.V. Alatyrtsev, V.N. Podkopaev // Medical = scientific and educational journal. -2001. No. 4. -P. 126.-141.
15. Balabolkin I.I., Smirnov I.E., Yukhtina N.V., et al. Inflammation of the respiratory tract as the pathogenetic basis of bronchial asthma in children // Russian Pediatric Journal. 2000.No. 5. Pp. 17–20.
16. Behrman E B et all (Ed) «Nelson Textbook of Pediatrics» / E.B. Behrman // 2000.- 2414 p.
17. Bjorn A.M. Extra pulmonary complications of Mycoplasma pneumonia infections / A.M. Bjorn, A.M. Lebech // Ugeskr Laeger. 2002. - Vol. 164(13). -P. 1805-1809.
18. Belkin A. A. Transcranial Doppler ultrasound in intensive care: a method, manual for doctors / A. A. Belkin, A. M. Alasheev, S. N. Inyushin. — Petrozavodsk: IntelTek, 2006. — 103 p.
19. Belikova M.E. Infectious and inflammatory pathology in newborns with perinatal lesions of the central nervous system: immunological mechanisms of its development, prognosis, prevention, correction: doctoral dissertation in medical sciences. Ivanovo, 2008. - 278 p.
20. Beskrovnaya E. V. Clinical, neurological, and cerebral hemodynamic features of cervical spine pathology in children: abstract of dissertation for

the degree of Candidate of Medical Sciences / E. V. Beskrovnaya. — Novosibirsk, 2006. — 19 p.

21. Blotta M.H. Corticosteroids inhibit IL-12 production in human monocytes and enhance their capacity to induce IL-4 synthesis in CD4+lymphocytes / M.H. Blotta, R.H. DeKruyff, D.T. Umedsu // J.Immunol. 2008. - Vol. 158, №. 12. -P. 5589-5595.
22. Blasi F. Guidelines for empiric antimicrobial prescribing in community-acquired pneumonia / F. Blasi , J.R. Lonks // Chest. 2004. - Vol. 125, - P. 1888-1901.
23. Buckley R.H Pulmonary complications of primary immunodeficiency / R.H .Buckley // Paed Resp Rev 2003. - V. 83, - P. 239-247.
24. Brown J.K. Fetal and Neonatal Neurology and Neurosurgery / J. K. Brown, R. A. Minns / M.J. Eds, V.J. Levene, J.I. Bennett Punt. Edinburgh, 2005. - P 487-516.
25. Bogdanova, A.V. System for providing assistance to children with bronchopulmonary dysplasia at various stages of patient management /Bogdanova A.V., Boitsova E.V., Starevskaya E.V. - St. Petersburg, 2004. - 16 p.
26. Brin I. JI. Elkar in the complex therapy of neuropsychiatric disorders in children / I. JI. Brin, M. JI. Dunaikin, O. G. Sheinkman // Modern technologies in pediatrics and pediatric surgery: proceedings of the III Russian Congress. — Moscow, 2004. — P. 32.
27. Vatolin K. V. Ultrasound diagnosis of brain diseases in children / K. V. Vatolin. — Moscow: Vidar-M, 2000. — 129 p.
28. Vasilyeva O.A. Strategy and tactics of immunorehabilitation of patients with encephalopathies of various origins / O.A. Vasilyeva et al. // Collection of abstracts from the 10th anniversary conference “Neuroimmunology.” St. Petersburg, 2001.

29. Vegetative Disorders: Clinic. Diagnosis. Treatment: A Handbook for Physicians / A. M. Vein et al.; edited by A. M. Vein. — Moscow: MIA, 2003. — 749 p
30. Volodin N. N. Perinatal encephalopathy and its consequences / N. N. Volodin, M. I. Medvedev, S. O. Rogatkin // Ross, Pediatrician, journal. — 2001.— No. 1. — P. A—8
31. Volkova JI.C. Immunocorrection in pediatrics / L.S. Volkova, V.L. Arion, M.I. Martynov // Practical guide for doctors. 2nd edition. Moscow, 2000. pp. 105-109.
32. Garaev V. R. Amplitude-integrated electroencephalography in neonatology / V. R. Garaev et al. // Pediatrics. — 2008. — Vol. 87, No. 1. — P. 59-66.
33. Goreva E.A. Features of the neuro-immune-endocrine system in infants during their first year of life who suffered hypoxia during the perinatal period: dissertation. , Candidate of Medical Sciences / E.A. Goreva. Chelyabinsk, 2000. - 182 p.
34. Greshilov A.A. Neuroimmunological criteria for the diagnosis and prognosis of perinatal lesions of the central nervous system in newborns and young children: dissertation. Candidate of Medical Sciences. Ufa, 2004. 114 p.
35. Gulyaev S.P. Clinical and immunological features of the course of perinatal damage to the central nervous system of hypoxic origin in children in their first year of life: dissertation for the degree of Candidate of Medical Sciences. Yekaterinburg, 2006. - 141 p.
36. Goldsby R, Kindt T, The immune system / R.Goldsby, T.Kindt // Virchows Arch. B. 2003. - Vol.16. -P. 249-301.
37. Gonzalez-Ariki S. Children immunodeficiency associated with protein-energy malnutrition / S. Gonzalez-Ariki, A.J. Husband // J. Neuroimm. 2003. -Vol.99.-P. 97-104.
38. Grant C.C. Pneumonia in children: becoming harder to ignore / C.C. Grant // N.Z. Med. J. 2001. - №10. - P.345-347.

39. E. A. Gorelikova, M. A. Kornushin // Pediatrics. — 2002. — No. 1. — P. 40— 44.
40. Guzeva V. I. Epilepsy and non-epileptic paroxysmal states in children / V. P. Guzeva. — Moscow: MIA, 2007. — 568 p.
41. Dementieva G. M. Prevention of adaptation disorders and diseases in newborns / G. M. Dementieva, Yu. E. Veltishchev // Ross, Bulletin of Perinatology and Pediatrics. — 2004. — Appendix. — P. 76.
42. Evidence-based medicine: Annual reference book. Part 3. — Moscow: Media Sfera, 2003.—332 p.
43. Evseyenko D.A. Changes in the fetoplacental complex during acute and chronic intrauterine hypoxia / D.A. Evseyenko, Yu.V. Eschenko // Pediatrics. 2002. - No. 1. - P. 5-9.
44. Evsyukova I.I. Pathogenesis of perinatal pathology in newborns who developed under conditions of chronic hypoxia with placental insufficiency. / I.I. Evsyukova // Journal of Obstetrics and Gynecology. — 2010. Vol. 53, No. 2. — P. 26-29.
45. Dolgikh G. B. Early diagnosis of circulatory disorders in the vertebrobasilar system in children: abstract of dissertation for the degree of Candidate of Medical Sciences / G. B. Dolgikh. — Moscow, 2004. — 24 p.
46. Fan L. L. Pediatric interstitial lung disease: children are not small adults / L. L. Fan, C. Langston // Am. J. Respir. Cri:t Care Med. 2002. - Vol. 165. - №11. -P. 1466-1467.
47. Felten D.L. Neural influence on immune responses: underlying suppositions and basic principles of neural-immune signaling / D.L Felten // Prog. Brain Res. 2000. - V. 122. - P. 381-389.
48. Zhulev N. M. Cervical osteochondrosis. Vertebral artery syndrome. Vertebral-basilar insufficiency / N. M. Zhulev, D. V. Kandyba, N. A. Yakovlev. — St. Petersburg: Lan, 2002. — 676 p.

49. Zabramnaya S. D. Psychological and pedagogical diagnosis of mental development in children: Textbook for students of speech therapy departments of pedagogical colleges and universities / 2004, - 55 p.
50. Zavadenko N. N. Disorders of oral and written speech in children. Possibilities for their medical correction: Method, manual for doctors / N. N. Zavadenko. Moscow: Severo-Press, 2003. — 63 p.
51. Zarakovsky G. M. Psychological and physiological manifestations of the process of adaptation of the Russian population to new socio-economic conditions / G. M. Zarakovsky, V. I. Medvedev, E. K. Kazakova // Human Physiology. — 2007. —Vol. 33, No. 1.—Pp. 5—14.
52. Zaitseva O.V. Mucolytic* drugs in the treatment of respiratory diseases in children: a modern view of the problem. / O.V. Zaitseva // Pediatric pulmonology: problems and solutions. — Moscow, 2004. Issue No. 4: pp. 71-80:
53. Zenkov L. R. Clinical electroencephalography: (With elements of epileptology): Handbook for doctors / L. R. Zenkov. — Moscow: MEDpress-inform, 2004. — 367 p.
54. Ivanov D. A. On the question of differential diagnosis of cervicogenic vertigo / D. A. Ivanov // VITT All-Russian Congress of Neurologists: Collection of abstracts and articles. — Kazan, 2001. — P. 358.
55. Ivanov L. B. Lectures on Clinical Rheography / L. B. Ivanov, V. A. Makarov. — Moscow: Scientific and Medical Firm “MBN,” 2000. — 319 p.
56. Ivanova E. L. Birth injuries to the spinal cord. Diagnosis of nervous system damage using unconditioned reflexes in newborns and assessment of muscle tone: (A guide for doctors) / E. L. Ivanova. — Moscow, 2000. — 22 pp.
57. Izmailova T. D. et al. Changes in adaptation and their correction in infants with post-hypoxic changes in the central nervous system / T. D. Izmailova et al. // Pediatrics. — 2002. — No. 1. — Pp. 27–30.

58. Ismailov K.I. Features of immunological reactivity in children with iron deficiency anemia. / K.I. Ismailov, A.M. Mirakilova, N.N. Khodzhaeva // Health Care in Tajikistan. - Dushanbe, 2008. No. 3. - pp. 161-164.
59. Ilyukhina V. A. The human brain in the mechanisms of information-control interactions between the organism and the environment (on the 20th anniversary of the Laboratory of Physiology of States) / V. A. Ilyukhina. — St. Petersburg: Published by the Institute of Human Brain, Russian Academy of Medical Sciences, 2004. — 326 p.
60. Ilyukhina V. A. Principles of complementarity and commensurability as the basis for new research technologies in the field of neurophysiology of functional states / V. A. Ilyukhina // Human Physiology. — 1999. — Vol. 25, No. 2. — P. 14—24.
61. Ilyukhina V. A. Transcranial micropolarizations in physiology and clinical practice / V. A. Ilyukhina, Yu. K. Matveev, E. M. Chernysheva. — St. Petersburg: Institute of the Human Brain, Russian Academy of Medical Sciences, 2006. — 192 p.
62. Kalamkarov G. R. Molecular Mechanisms of Visual Reception / G. R. Kalamkarov, M. A. Ostrovsky. — Moscow: Nauka, 2002. — 279 p.
63. Kaganov S.Y. Pulmonology in children and its pressing problems / S.Y. Kaganov, H.H. Rozinova // Ross, vestnik perinatologii i pediatrii (Russian Journal of Perinatology and Pediatrics). 2000. No. 6. pp. 6-11.
64. Classification of perinatal lesions of the nervous system and their consequences in children in their first year of life: method, recommendations of the Russian Association of Perinatal Medicine Specialists / A. S. Burkova et al. // Questions of Practical Pediatrics. 2006. - No. 5. - P. 38-70.
65. Karlov V. A. Bulbar syncope? / V. A. Karlov, T. S. Sologubova // Neurological Journal. — 2004. — No. 3. — Pp. 19—22
66. Clinical Physiology of Vision: Essays, Reviews, Original Articles / Edited by A. M. Shamshinova. — Moscow: Scientific and Medical Firm MBN: T. M. Andreeva, 2006. — 956 pp.

67. Casanovas J L Primary immunodeficiency diseases and update allergy. / J. L. Casanova // Clin Immunol -2004. V. 114 - P. 677-687.
68. Kozhushko N. Yu. Age-related features of the formation of bioelectrical brain activity in children with long-term consequences of perinatal CNS damage / N. Yu. Kozhushko // Human Physiology. 2005. —Vol. 31, No. 1. —Pp. 5—14.
69. Chang A.B. Cough throughout life: children, adults and the senile / A.B. Chang, J.G. Widdicombe // Pulm Pharmacol Ther. 2007. - Vol. 20(4). - P. 371-382.
70. Comprehensive multiparametric study of systemic reactions of the human body under dosed hypoxic exposure / S. I. Soroko et al. // Human Physiology. — 2005. — Vol. 31, No. 5. — P. 1— 22.
71. Clinical guidelines based on evidence-based medicine / edited by I.N. Denisov; V.I. Kulakov, P.M. Khaitov; translated from English. Moscow: GEOTARMED, 2001. - 1248 p.
72. Konovalov A. N. Magnetic resonance imaging in neurosurgery / 2005, - 38 p.
73. Kozlov P.C. Ways to optimize monitoring, prevention, and pharmacotherapy of pneumococcal infections: abstract of doctoral dissertation in medical sciences. Smolensk, 2004. - 47 p.
74. Korsakova N. K. Underachieving children: neuropsychological diagnosis of learning difficulties in primary school pupils / N. K. Korsakova, Yu. V. Mikadze, E. Yu. Balashova. — Moscow: Pedagogical Society of Russia, 2001. —160 p.
75. Kozlova*JI.B. Errors in the diagnosis and treatment of community-acquired pneumonia in children / JI.B. Kozlova // Pediatric Pulmonology: Problems and Solutions. Moscow, 2004. - Issue No. 4. - Pp. 84-85.
76. Kolosova M.V. Acute pneumonia in children / M.V. Kolosova: method, recommendations / Siberian Medical University. Tomsk, 2002. - 70 p.

77. Korneva E.A. Main stages in the development of immunophysiology // Neuroimmunology. 2005. - Vol. 3. - No. 1. - Pp. 4-10.
78. Kornev, V.G. Pneumonia in young children / B.F. Kornev, M.A. Skachkova, N.M. Lapteva // Pulmonology in children: problems and solutions. Moscow, 2004. - Issue No. 4f. - Pp. 82-84.
79. Korovina H.A. Rational < antibacterial therapy for community-acquired pneumonia in children / H.A. Korovina, A.L. Zaplatnikov // Pediatrics. 2003. - No. 1. - P. 11-14.
80. Kryzhanovsky G.N. Neuroimmunopathology: A Guide / G.N. Kryzhanovsky, S.B. Magaeva, S.B. Makarov, R.I. Sepiashvili. Moscow, 2003. 438 p.
81. Kuzmenko L.G. Immunocorrection with T-activin in the complex treatment of frequently ill children / L.G. Kuzmenko, Yu.M. Lopukhin, V.Ya. Arion // Handbook for doctors. Moscow, 2005. - 47 p.
82. Krivoshapova M. N. Age-related features of activation levels in the frontal and temporal-parietal cortex in children aged 3–7 years / M. N. Krivoshapova, B. A. Ilyukhina // Human Physiology. — 2006. — Vol. 32, No. 1. — P. 56–67.
83. Krivoshapova M. N. Ultra-slow biopotentials in the study of the characteristics of the formation of activation levels of the frontal and temporal-parietal cortex in children aged 4–7 years: abstract of dissertation. . Candidate of Biological Sciences / M. N. Krivoshapova. — St. Petersburg, 2005. — 16 p.
84. Kryzhanovsky G.N. Dysregulatory pathology / G.N. Kryzhanovsky. Moscow: Medicine, 2002.- 632 p.
85. Kurshakova I.V. Hypoxia as a trigger for the development of encephalopathy complicating the course of severe intracranial trauma / I.V. Kurshakova // 2006. -98 p.
86. Khaydarov B.M. Children's Diseases / B.M. Khaydarov. Dushanbe: Irfon, 2009. - 636 p.

87. Comprehensive rehabilitation of children with perinatal damage to the central nervous system: a textbook for postgraduate professional education of pediatricians* / T.S. Krivonogova et al. Tomsk: Pechatnaya Manufaktura, 2007. - 68 p.
88. Contemporary features of acute pneumonia in children / A.A. Lebedenko et al. // Pulmonology in Children. 2003. - No. 1. - P. 42-43.
89. Pharmacotherapy of hypoxia and its consequences in critical conditions: proceedings of the All-Russian conference. St. Petersburg, October 7-8, 2004. — St. Petersburg, 2004. — P. 136-139.
90. Therapeutic effects of central and peripheral electrical stimulation: proceedings of the All-Russian scientific and practical conference. St. Petersburg, June 5–6, 2001. — St. Petersburg: Published by St. Petersburg State University, 2001. — 65 p.
91. Lokhov M. I. Stuttering and logoneurosis. Diagnosis and treatment / M. I. Lokhov, Yu. A. Fesenko. — St. Petersburg: Sotis, 2000. — 288 p.
92. Lewin A.S . Bronchi ecstasies / A.S. Lewin, B.C. Hilman Pediatric respiratory disease. Philadelphia: W.B. Saunders Company, 2003. - 220 p.
93. Lekstron-Himes JA. Immunodeficiency diseases caused by defects in phagocytes./ J.A. Lekstron-Himes // Engl J Med 2000. - V. 343 - P. 17031714.
94. Louis M. Bell. Pediatric Pulmonologist / M. Bell. Louis // 2005. 560 p.
95. Lubovsky, V. I. Psychodiagnostics of developmental disorders in children / V. I. Lubovsky // Special Psychology: Textbook / V. I. Lubovsky, V. G. Petrova, T. V. Rozanova, et al.; edited by V. I. Lubovsky. — Moscow: Akademika, 2003. — P. 416—438.
96. Machinskaya R. I. Formation of neurophysiological mechanisms of voluntary selective attention in primary school children: dissertation for the degree of Doctor of Biological Sciences / R. I. Machinskaya. — Moscow, 2001. — 352 p.

97. Maksakova T.B. Rehabilitation: children with bronchopulmonary pathology / T.E. Maksakova; R.N. Bychkova, G.M. Rezvitsko II Pulmonology: childhood: problems and solutions. Moscow, 2004. - Issue No. 4. - Pp. 109-110.
98. Medvedeva: T.Y.: Immunological reactivity and blood groups* in acute pneumonia in young children / T.Y. Medvedeva // Pediatrics. -2007.-Vol. 86.-No. 5.-Pp. 144-145.
99. Medvedeva T.Ya.: Etiological aspects of acute pneumonia in young children / T.Ya. Medvedeva // Pediatrics. 2008. - Vol. 87. - No. 1. - P. 143-145:
100. Mizernitsky Yu.L. Bronchial obstructive syndrome in acute respiratory viral infections in young children: differential diagnosis in pediatric practice / Yu.L. Mizernitsky // Pulmonology in children. 2004. - No. 4. - P. 37-41.
101. Magayeva S.V. Neuroimmunophysiology / S.V. Magayeva, S.G. Morozov: Moscow: Published by the Research Institute of Biomedical Chemistry, 2005. 160 p.
102. Metabolic therapy of post-hypoxic cardiopathy in newborns / M. Ya. Ledyayev et al. // Modern technologies in pediatrics and pediatric surgery: materials of the III Russian Congress. — Moscow, 2004. — P. 185.
103. Research Methods in Pediatric Neurology: Textbook / V. P. Zykov, D. Ch. Shiretorova, V. N. Shadrin et al. ; Edited by D. Ch. Zykov.Moscow: Triada-Kh, 2004. — 112 p.
104. Research Methods in Neurology and Neurosurgery: A Handbook for Physicians / Compiled by E. I. Gusev et al. — Moscow: Nolij, 2000. — 330 p.
105. Mitish, M. D. Long-term consequences of perinatal brain damage in children: abstract of doctoral dissertation in medical sciences / M. D. Mitish. — Moscow, 2004. — 48 p.

106. Mizernitsky Yu.L. Differential diagnosis and differentiated therapy for acute bronchial obstruction in acute respiratory viral infections in young children. Practical Medicine, 2014, 9(85): 82-89

107. Madden K.S. Alterations in T-lymphocyte activity following chemical sympathectomy in young and old Fisher 344 rats / K.S. Madden, S.Y. Stevens, D.L. Felten // J. Neuroimmunol. 2000. - Vol. 103, N 2. - P. 131-145.

108. Markova T.P. Khaitov R.M. Methodological approaches to immunological diagnosis. Immunol letters, 2005, -Vol. 56, №1-3, P. 332-349.

109. Mcintosh,K. Community-Acquired Pneumonia in Children / K. Mcintosh // N. Engl. J. Med. 2002. - Vol. 346, №6. - P. 429-437.

110. Michalowska-Wender G. Hypoxic-ischemic encephalitis in newborns / G. Michalowska-Wender // Folia NeuropathoL 2009. - Vol. 37, N 6. - P. 273276.

111. Morozova L. V. Features of visual perception and functional state of the brain in children aged 5 and 7 / L. V. Morozova // Bulletin of Pomor University. — 2002. — No. 1 (3). — P. 46.

112. Morozova JI. V. The level of development of structural components of visual perception in children as an indicator of psychophysiological maturity / L. V. Morozova, N. V. Zvyagina // Vestnik Pomorskogo Universiteta. — 2003. — No. 3 (4). — P. 48.

113. Mosin I. M. Diseases of the visual pathways in early childhood: etiology, clinical manifestations, topical and differential diagnosis, aspects of rehabilitation: abstract of dissertation for the degree of Doctor of Medical Sciences / I. M. Mosin. — Moscow, 2002. — 44 p.

114. Morton R.E. Respiratory tract infections due to direct and reflux aspiration children with severe neurodisability / R.E. Morton, R. Wheatley, J. Minford // Dev. Med. ChildlNeurol. 2003. - №8. - P: 329-334.

115. Mycoplasma pneumonia. infection, meningoencephalitis, and hemophagocytosis / L.A. Bruch et al. // Pediatr Neurol. 2001. - Vol. 25(1). - P: 67-70.

116. Mukhin K. Yu. Idiopathic forms of epilepsy: systematics, diagnosis, therapy / K. Yu. Mukhin, A. S. Petrukhin. — Moscow: Art-Business-Center, 2000. — 319 p.

117. Hereditary and Congenital Diseases of the Retina and Optic Nerve / Edited by A. M. Shamshinova. — Moscow: Medicine, 2001. — 527 p.

118. Nagibina, N.S. Risk factors and hemodynamic disorders in perinatal damage to the central nervous system in newborns / N.S. Nagibina, L.G. Gorbik, M.V. Narogan // Medical Care. 2001. - No. 2. - P. 52-54.

119. Heiskanen-Kosma Etiology of childhood pneumonia: serologic results of a prospective, population-based study / Heiskanen-Kosma et al. // Pediatr. Infect. Dis. J. -2003. Vol. 17. - P. 986-991.

120. Hendricson K.J. Viral pneumonia in children / K J. Hendricson // Seminar in Pediatric infectious Diseases. 2004. - Vol. 9. - P. 217-233.

121. Nefedovskaya L. V. Prevalence of disability caused by eye diseases / L. V. Nefedovskaya, R. N. Terletskaya // Issues of Modern Pediatrics. — 2008. — Vol. 7, No. 2. — Pp. 9—12.

122. Novikova G. R. The state of higher mental functions in children entering the first grade of general education schools (based on the results of neuropsychological examination) / G. R. Novikova // Defectology. — 2000. — No. 2. — pp. 51–55.

123. Nance D.M. Brain regulation-immune response in rats after hypoxemia / D.M Nance, D. Rayson // Brain Behav. Immuniti. 2000. - №11 - P. 292-305.

124. Narita M. Pathogenesis of neurologic manifestations of Mycoplasma pneumonia infection / M. Narita // Pediatric Neurol. 2009. - Vol. 41(3). - P. 159-166.

125. Nikol'skii I.S. Clinical-immunological' studies on4 the therapeutic efficacy of Immune, in acute bronchitis in children, frequently and persistently ill / I.S. Nikol'skii, V.V. Nikol'skaia, L. Kushko, N.E. Vikhot // Lik-Sprava. 2006. -N6-P. 128-131.

126. High-risk newborns / Edited by V. I. Kulakov, Yu. I. Barashnev. — Moscow: GEOTAR-Media, 2006. — 528 pp. — 256151. General issues of neonatology and perinatology: textbook /

127. M. Ya. Ledyayev et al. — Volgograd: Volgograd State Medical University, 2003. — 56 pp.

128. Okovity, S. V. Clinical pharmacology of antihypoxants. Part 1 / S. V. Okovity // PHARMindex-Practic. — 2004. — Issue 6. — P. 30—39.

129. Okovity S. V. Clinical pharmacology of antihypoxants. Part 2 / S. V. Okovity // PHARMindex-Practic. — 2005. — Issue 7. — Pp. 48—63.

130. Ovsyannikov, D.Yu. Bronchopulmonary dysplasia: issues of terminology and classification /D.Yu. Ovsyannikov, I.V. Davydova //Russian Pediatric Journal. - 2008. - No. 2. - Pp. 18-23.

131. Ovsyannikov, D.Yu. Bronchopulmonary dysplasia in children / D.Yu. Ovsyannikov, N.I. Petruk, L.G. Kuzmenko // Pediatrics. - 2004. - No. 1. - P. 91-94.

132. Olimova K.G. Dynamics of manifestations and long-term consequences of perinatal damage to the central nervous system in children: abstract of dissertation for the degree of Doctor of Medical Sciences. Moscow, 2004. 44 p.

133. Determination of the functional capabilities of schoolchildren's bodies using various methods of physical exercise dosing / V. D. Sonkin et al. // New Research. Almanac. — 2004. — No. 1–2. — P. 360.

134. Experience in the use of evoked potentials in clinical practice / Edited by V. V. Gnezditsky, A. M. Shamshinova. — Moscow: Antidor, 2001. — 472 p.

135. Main factors reducing stress resistance in children aged 6–8 years with long-term consequences of perinatal CNS pathology during the transition to school age / 2007.- 88 p.

136. B. A. Ilyukhina et al. // Human Physiology. — 2002. — Vol. 28, No. 3. —P. 5— 15.

137. Features of the course of hypertensive-hydrocephalic syndrome in • newborns / A. V. Kovalenko et al. // Russian Pediatric Journal. — 2007.4. — P. 54—55.

138. From Neuron to Brain / J. G. Nicolle, A. R. Martin, B. J. Wallace, et al. — Moscow: UOSS, 2003. —672 p.

139. Palchik, A. B. Hypoxic-Ischemic Encephalopathy in Newborns: A Handbook for Physicians / A. B. Palchik, N. P. Shabalov. — St. Petersburg: Piter, 2000. — 219 p.

140. Palchik A. B. Modern concepts of perinatal encephalopathy / A. B. Palchik, N. P. Shabalov, A. P. Shumilina // Russian Pediatric Journal. — 2001. — No. 1. — Pp. 31—34.

141. Practical Guide to Anti-Infective Chemotherapy: edited by JI.C. Strazhunsky, M.N. Belousov, S.N. Kozlov. Moscow, 2002. 288 pp.

142. Palchik A. B. Evolutionary neurology / A. B. Palchik. — St. Petersburg: Piter, 2002. —383 p.

143. Papadopoulos NG, Arakawa H, Karlsen KH,Kustovich A, Gern JE, Lemanske R, et al.International Consensus on (ICON) Pediatric Asthma. Allergy, 2012, 67(8): 976-997

144. Pinchuk D. Yu. Transcranial micro-polarizations of the brain: clinical features, physiology (20 years of clinical experience) / D. Yu. Pinchuk. — St. Petersburg, 2007. — 713 p.

145. Principles of managing newborns with respiratory distress syndrome: method. recommendations /edited by N.N. Volodin. - Moscow, 2008.

146. Polonskaya N. N. Neuropsychological Characteristics of Children with Different Levels of Academic Success / N. N. Polonskaya // A. R.

Luria and Psychology of the 21st Century: Proceedings of the Second International Conference Dedicated to the 100th Anniversary of the Birth of A. R. Luria. — Moscow, 2003. — P. 206.

147. Program for education and training of preschoolers with intellectual disabilities / L. B. Baryayeva et al. — St. Petersburg: Soyuz, 2001. — 320 p.

148. Psychological diagnosis of developmental disorders in primary school children: method, manual / A. D. Vinogradova, N. L. Konovalova, S. T. Posokhova et al.; edited by L. M. Shipitsyna. — St. Petersburg: Rech, 2004. — 48 p.

149. Pinna G.S. The significance of Urea plasma-urea lithium.as a pathogenic agent in the pediatric population / G.S. Pinna, C.L. Skevaki, D.A. Kafetzis // Curr OpinInfect Dis. 2006. - P. 283-289.

150. Panacek E.A. Fetal adaptive responses to hypoxemia / E.A. Panacek, V. Dobos, P. Temesvari et al:. // Pediatrics and Neonate. 2001. - Vol. 74, №6. -P. 445-450.

151. Pallapies D. Trends in childhood disease // Mutat Res. 2006. Inflammation / D. Pallapies, D. Quarcoo, S. Pavlovic, R.A. Joachim. // Neuroimmunomodulation. - 2009: - Vol. 16(5). - P. 318-324.

152. Pandemic influenza: clinical issues / M. Boyd et al. // Med. J. Aust. 2006. -Vol. 185(10 Suppl). - P. 44-47.

153. Rozhkov V. P. Formation of interaction between wave components of basic EEG rhythms in children during the first five years of life / V. P. Rozhkov, S. I. Soroko // Human Physiology. — 2001. — Vol. 26, No. 6.1. P. 5—19.

154. Rozhkova JI.A. Spectral power of EEG in primary school children with perinatal CNS pathology // Human Physiology. — 2008.1. Vol. 34, No. 1. —Pp. 28-38.

155. Rakhimova H.H. Features of the development and health status of infants and preschool children who have suffered perinatal damage to the

central nervous system: dissertation. Candidate of Medical Sciences. Dushanbe, 2009. -123 p.

156. Romanenko E.S. Clinical and immunological features of acute obstructive bronchitis and pneumonia in young children with perinatal damage to the central nervous system: abstract of thesis for the degree of Candidate of Medical Sciences. Chelyabinsk, 2003. - 124 p.

157. Rosin Yu. A. Dopplerography of cerebral vessels in children. Method, manual / Yu. A. Rosin. — St. Petersburg: Med. Center “Prognosis,” 2000. — 60 pp.

158. Russian Statistical Yearbook: Statistical Compilation. Moscow: State Statistics Committee of Russia, 2005. 819 pp.

159. Russian Statistical Yearbook: Statistical Compilation. Moscow: State Statistics Committee of Russia, 2006. 806 pp.

160. Russia: Trends and Prospects for Development. — Moscow: RAGS, 2005.

161. Rumyantseva S. A. Free radical processes and their correction in hemorrhagic stroke / S. A. Rumyantseva, A. I. Fedin, S. B. Bolevich // Neurological Journal. — Vol. 12, No. 5. 2003. — Pp. 51-56.

162. Svyatogor I. A. Temporal organization of EEG structure in anxiety-phobic disorders / I. A. Svyatogor, I. A. Mokhovikova // Journal of Neurology and Psychiatry named after S. S. Korsakov. — 2005. — Vol. 105, No. 9. — Pp. 30–35.

163. Stolyarov I.D. Neuroimmunology: some theoretical and applied clinical aspects / Stolyarov I.D. et al. // Neuroimmunology. 2005. - Vol. 3, No. 3-4. - P. 11-17.

164. Samsygina G.A. Pneumonia in children / G.A. Samsyagina // Pediatrics. 2002. - pp. 37-41.

165. Semenova O. A. Formation of voluntary regulation of activity and its brain mechanisms in ontogenesis / O. A. Semenova // Human Physiology. — 2007. — Vol. 33, No. 3. — P. 115—127.

166. Semenova, O. A. Formation of regulatory and control functions in younger schoolchildren: abstract of dissertation for the degree of Candidate of Psychological Sciences / O. A. Semenova. — Moscow, 2005. — 23 p.

167. Simonova Yu. V. Correction of metabolic syndrome in patients with multiple sclerosis using mexidol / Yu. V. Simonova, V. I. Golovkin, Yu. F. Kamynin // Bulletin of Experimental Biology and Medicine. Appendix 1. Neurology — 2006. — P. 82—85.

168. Skvortsov, A. S. Development of the nervous system in children: normal and pathological / A. S. Skvortsov, N. A. Ermolenko. — Moscow: MEDpress-inform, 2003. — 368 p.

169. Skvortsov I. A. Disorders of psychoneurological development of hereditary and non-hereditary origin: Textbook and teaching aid /

170. Smirnov V. M. Neurophysiology and Higher Nervous Activity in Children and Adolescents / V. M. Smirnov. — Moscow: ASA-OEM1A, 2000. — 400 p.

171. Contemporary aspects of diagnosis and treatment of attention deficit hyperactivity disorder in children. Method, manual / Edited by T. A. Lazebnik et al. — St. Petersburg, 2002. — 48 p.

172. Sokolov A. N. Diagnosis of the level of cognitive development in older preschool children with cerebral-organic developmental delays. Method, manual / A. N. Sokolov, N. F. Berezhnaya, V. A. Ilyukhina. — St. Petersburg, 2004. —50 p.

173. Sokolov, A. N. Psychological effects of corrective and developmental activities during transcranial micro-polarization sessions in children with mental retardation: abstract of dissertation for the degree of Candidate of Psychological Sciences / A. N. Sokolov. — St. Petersburg, 2005. — 22 p.

174. Solodkov, A. S. Human physiology. General. Sports. Age-related. Textbook / A. S. Solodkov, E. B. Sologubov. — Moscow: Olympia Press, 2005. — 528 p.

175. Soroko S. I. Comprehensive multiparametric study of systemic reactions of the human body under dosed hypoxic exposure / S. I. Soroko et al. // Human Physiology. — 2005. — Vol. 31, No. 5. — P. 1—22.

176. Special Psychology: Textbook: Textbook for students of the Faculty of Defectology of Higher Pedagogical Educational Institutions / V. I. Lubovsky, V. G. Petrova, T. V. Rozanova et al.; edited by V. I. Lubovsky. — Moscow: Akademika, 2003. — 461 p.

177. Starshov, A. M. Rheography for Professionals. Methods of Researching the Vascular System / A. M. Starshov, I. V. Smirnov. — Moscow: Poznavatelnaya Kniga Press, 2003. — 80 p.

178. Strachunsky L.S. Antibacterial therapy for pneumonia in children: Guidelines for pharmacotherapy in pediatrics and pediatric surgery / L.S. Strachunsky: edited by S.Yu. Kaganov. Vol. 1. - Moscow: Medpraktika, 2002. -103 p.

179. Suslova G. A. Scientific justification for the organization of medical care for children with psychosocial development disorders (based on the example of MMD): abstract of doctoral dissertation in medical sciences / G. A. Suslova. — St. Petersburg, 2001. — 39 p.

180. Tatchenko V.K. Pneumonia in children: diagnosis and treatment / V.K. Tatchenko // Attending Physician: Medical Scientific and Practical Journal. 2008. - No. 8. - P. 510.

181. Tatchenko V.K. Pneumonia in children: scientific publication / V.K. Tatchenko // Pediatrician's Handbook: scientific and practical journal. - 2006. - No. 7. - Pp. 5-29.

182. Tatchenko V.K. Practical Pulmonology in Children. Moscow, 2001. 255 p.

183. Tsarkova JI.H. Chronic pneumonia / Diseases of the respiratory system / JI.H. Tsarkova: edited by N.R. Paleev. -M.: Medicine,.2000. Pp. 441-449.

184. Tozlyan E. V. Clinical significance of mitochondrial disorders in children with undifferentiated forms of neuropsychiatric developmental delay: abstract of dissertation for the degree of Candidate of Medical Sciences / E. V. Tozlyan. — Moscow, 2003. — 32 p.

185. Tereshchenko S.Y. Idiopathic interstitial pneumonia in children / S.Y. Tereshchenko, M.B. Vlasova // Pulmonology in children: problems and solutions. Moscow, 2004. - No. 4. - Pp. 23-34.

186. Tereshchenko Yu.A. Idiopathic interstitial pneumonia in adults and children (literature review) / Yu.A. Tereshchenko, S.Yu. Tereshchenko, Yu.A. Vlasova // Pulmonology: scientific and practical journal. 2005. - No. 2. - P. 119-125

187. T-lymphocyte content and thymogen antibody levels in children with central nervous system damage / A.I. Autenshlius et al. // Immunology. 2003. - Vol. 24. - No. 4. - P. 231-237.

188. Ulaschik V. S. General physiotherapy / V. S. Ulaschik, I. V. Lukomsky. — Minsk: Knizhny Dom, 2005. — 512 p.

189. Farber, D. A. Development of visual perception in ontogenesis. Psychophysiological analysis / D. A. Farber // World of Psychology. — 2003. — No. 2(34). — p. 114.

190. Farber D. A. Formation of the visual perception system in ontogenesis / D. A. Farber, T. G. Beteleva // Human Physiology. — 2005. — Vol. 31, No. 5. —P. 26.

191. Flimova X. M. Factors of mental dysontogenesis / X. M. Flimova // Neurology. 2007. No. 2. P. 60-63.

192. Fokin V. F. Energy physiology of the brain / V. F. Fokin, N. V. Ponomareva. — Moscow: Antidor, 2003. — 288 p.

193. Fotekova T. A. Test methodology for oral speech of primary school pupils / T. A. Fotekova. — Moscow: ARKTI, 2000. — 56 p.

194. Functional states of the main life support systems of newborns / I. A. Belyaeva et al. // Russian Pediatric Journal. — 2007. — No. 3. — Pp. 49–54.

195. Kholin A. V. Magnetic resonance imaging in diseases of the central nervous system / A. V. Kholin. — St. Petersburg: Hippocrates, 2000. — 191 p.

196. Tsvetkova L. S. Methods of Neuropsychological Diagnosis of Children / L. S. Tsvetkova. — Moscow, 2000. — 128 p.

197. ThagaRajan S. Effects of L-deprenyl- treatment on noradrenergic innervate and immune reactivity in lymphoid organs of young F344' rats / S. Thaga-Rajan, K.S. Madden, S.Y. Stevens // J. Neuroimmunol. 2005. - Vol. 96, №1. - P. 57-65.

198. Tosini G. Melatonin circadian rhythm in the retina of mammals / G. Tosini // Chronobiol. Int. 2000. - Vol. 17. - P. 599-612.

199. The X-linked hyper-IgM syndrome: clinical and immunologic features of 79 patients / J.A. Winkelstein et al. // Medicine (Baltimore). 2003. - Vol. 82(6). - P. 373-384.

200. Twenty year surveillance of invasive pneumococcal disease in Nottingham-servo groups responsible and implications for immunization / P. Ispahani et al. //Arch. Dis. Child: 2004. - Vol. 89(8). - P. 757-762.

201. Raboei E. Neonatal cricopharyngeal achalasia—a case report /E. Raboei, R. Luoma // Eur. J. Pediatr Surg. 2000. - Vol. 10(2). - P. 130-132.

202. Rhythms is human bone marrow and blood cells / R. Smaaland, R.B. Sothem, etal. // Chronobiol. Int. 2002. - Vol. 19. - P. 101-127.

203. Shakhnovich V. A. Cerebral ischemia. Neurosonology / V. A. Shakhnovich. — Moscow: ACT, 2002. — 305 p.

204. Shelyakin A. M. Micropolarization of the brain (effectiveness, physiological analysis): abstract of dissertation for the degree of Doctor of Biological Sciences / A. M. Shelyakin. 1. St. Petersburg, 2003. — 32 p.

205. Shelyakin A. M. Micropolarization of the brain / A. M. Shelyakin, G. N. Ponomarenko; edited by O. V. Bogdanov. — St. Petersburg: IIC Baltika, 2006. 223 p.

206. Seo S.H. Cytology T lymphocyte responses to infectious bronchitis virus infection / S.H. Seo, E.W. Collisson // Adv. Exp. Med. Biol. J. 2001. - №440. - P. 455-460.

207. Sermet-Gaudelus. La mesure de la temperature en pratuque pediatrique quotidienne. / Sermet-Gaudelus, I. Chadelat, G. Lenoir // Archives de pediatric. -2005.-№12.-P. 1292-1300.

208. Stalder A.K. LPS-induced LI-12 expression in the CNS and cultured atrocities and microglia / A.K. Stalder, A. Pagenstecher, N.C. Yu // J. Immunol. 2000. -Vol. 159, №3.-P. 1344-1351.

209. Stead A. Humeral immunity and bronchiectasis./ A. Stead , E .Herriot // Clin Exp Immunol.- 2002.- №130.- P. 325-330.

210. Savino W. Immune neuron endocrines connectivity: the paradigm of the thymus- hypothalamus/pituitary axis / W. Savino, E. Arzt, M. Dardenne // NeuroimmunomoduK 2005. - Vol. 8, №3-4. - P. 126-136.

211. Sakoulas G. Brainstem and striate encephalitis complicating Mycoplasma pneumonia: possible benefit of intravenous immunoglobulin / G. Sakoulas // Dev Pediatric Infect Dis J. 2001. - Vol. 20(5). - P. 543-545.

212. Shelyakin A. M. Application of local direct current in experiments and clinical practice / A. M. Shelyakin, I. G. Preobrazhenskaya, Bogdanov O. V. // Journal of Neurology and Psychiatry named after S. S. Korsakov. —2001. —No. 8. — P. 62—64.

213. Shiretov R. T. The state of cerebral hemodynamics in neurological manifestations of craniocervical anomalies: abstract of dissertation for the degree of Candidate of Medical Sciences / R. T. Shiretov. — Perm, 2001. — 20 p.

214. Shkolnikova M. A. Physiology and pathology of the cardiovascular system in children in their first year of life / M. A. Shkolnikova, JI. A. Kravtsova. — Moscow: Medpraktika—M, 2002. — P. 58—69.

215. Shabalov, N.P. Fundamentals of Perinatology: Textbook / N.P. Shabalov, Yu.V. Tsveleva. Moscow: MED Press-Inform, 2002. - 2nd ed., revised and expanded. - 576 p.

216. Shabalov N.P. Neonatology: Textbook: Vol. 1,2. - 4th ed., rev. and expanded. - Mi: MED Press-Inform, 2006. - 608 p.

217. Shurygin I. A. Respiratory monitoring: Pulse oximetry, capnography, oximetry / I. A. Shurygin. — Moscow: Binom; St. Petersburg: Nev. Dialect, 2000 — 300 p.

218. Yakovlev N. A. Vertebral-basilar insufficiency / N. A. Yakovlev.1. Moscow, 2001. —400 p.

219. Yatsyk G. V. Staged rehabilitation of newborns with perinatal pathology — prevention of delayed health disorders in adolescents / G. V. Yatsyk, A. A. Stepanov, E. P. Bombardirova // Russian Pediatric Journal. — 2007.—No. 2. —Pp. 33-35.

220. Velissariou IM. Pneumonia in children: recent patents and advances / I.M. Velissariou // Recent Pat Antiinfect Drug Discov. 2007. - Vol. 2(1). - P. 7377.

221. Van-Aalderen W.M. / W.M. Van-Aalderen, P.L. Brand, M.O. Hoekstra // Pediatr. Allergy Immunol J. 2006. - №9. - P. 42-47.

222. Wong G.W. Severe acute respiratory syndrome in children / G.W. Wong, T.F. Folk // Pediatric Pulmonol. 2003. - №36. - P. 261 -265.

223. Warren E. Roentgen graphic features of common pediatric viral respiratory tract infections / E. Warren, T. Chonmaitree, L.E. Swischuk // Am. J. Dis. Child. 2002. - Vol. 142. - P.43-46.

Classification of the consequences of perinatal damage to the nervous system of young children (Pediatric Research Institute, attached to the Department of Child Neurology, Tashkent State Medical University)

Etiopathogenesis	Periods	Severity	Clinical syndromes	Consequences
Hypoxia Transient cerebral ischemia By ischemic type of MQE By hemorrhagic type of MQE	1. Period of neurological deficit formation (1-3 months) 2. Recovery period (3-12 months) Up to 24 months in premature infants.	light moderate Severity	**A. Clinical syndromes of the "formation" period** **- cerebral excitability syndrome** **- cerebral depression syndrome** **- vegetative-visceral disorders syndrome** **- cerebrospinal fluid distension syndrome (intracerebral hypertension)** **- convulsive syndrome** **- muscle tone and movement disorders syndrome** **B. Clinical syndromes of the recovery period** **- delayed PMO stages syndrome** **- emotional-behavioral disorders** **- psychoverbal development disorders** **- movement disorder syndrome;** **- hydrocephalus (acquired)** **- convulsive syndrome** **- nonconvulsive paroxysms** **- vegetative dysfunction** **sleep disorders (parasomnia)**	1. Recovery 2. Functional impairment 3. Organic sequelae 4. Death
Birth trauma Of the brain Of the spinal cord Of the peripheral nerves				
Metabolic disorders Of carbohydrate metabolism Of calcium and magnesium metabolism Kernel jaundice Vitamin K hypovitaminosis				
NT toxic effects				
Infections and parasitic diseases Viral Bacterial sepsis Parasitic				