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Compression Circulatory Hypoxia of the Brain as a Type of Intrapartum Hypoxia

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Abstract

Among the 5 types of intrapartum hypoxia of the fetus is proposed to allocate compression circulatory hypoxia of the brain (CCHB). The relationship between the CCHB and the stages of the fetal head configuration is considered. When a fetal head is pressed, three successively developing stages are distinguished: 1) Stage of the physiological configuration of the head, 2) Stage of the CCHB and 3) Stage of the birth trauma (BT) of the skull.

Keywords: Intrapartum Hypoxia; Compression of the Head; Configuration (Molding) of the Head; Compression Circulatory Hypoxia of the Brain; Birth Trauma

Abbreviations: CCHB: Compression Circulatory Hypoxia of the Brain; BT: Birth Trauma; IH: Intrapartum Hypoxia; ICP: Intracranial Pressure; pH: Pondus Hydrogenii

Mini Review

The results of our own research and literature data allow us to distinguish the following 5 types of acute intrapartum hypoxia (IH), and each species includes a group of causes.

- **Uteroplacental hypoxia (due to pathology of the placenta and uterus):** 1) Placental abruption, 2) Placental presentation, 3) Placental infarction, 4) Placental tumors (chorioangioma, teratoma, etc.), 5) Placental developmental defect, 6) Uterine rupture, 7) Intensive contractions of the uterus caused by the administration of oxytocin.
- **Funicular hypoxia (due to pathology of the umbilical cord)**: 1) True cord knot, 2) Cord entanglement around the neck and extremities, 3) Umbilical cord prolapse, 4) Cord compression, 5) Cord compression in the process of delivery during pelvic presentation, 6) Absolutely short cord 7) Aplasia of the umbilical artery, 8) Rupture of the vessels during the umbilical cord attachment (vasa praevia), 9) Thrombosis of the umbilical vessels.
- **Maternal hypoxia (diseases of the mother):** 1) Blood loss during obstetric bleeding (with placental abruption, placenta previa, uterine rupture), 2) Pre-eclampsia and eclampsia, 3) Haemorrhagic shock,, 4) Amniotic fluid embolism, pulmonary thromboembolism, 5) Thrombosis and hemorrhagic syndrome, 6) Diseases of the cardiovascular system (congenital and acquired heart defects with hemodynamic disturbances, cardiac arrhythmia), 7) Severe asthma, 8) Cardiac and respiratory arrest, 9) Generalized seizures, 10) Circulatory disoders (sudden arterial hypotension, hypertension), 11) Aorto-caval compression, 12) Severe intoxication.

- **Fetal hypoxia (hypoxia caused by own fetal diseases):** 1) Hemolytic disease of the newborn, 2) Congenital defects of the cardiovascular system, 3) Fetal infection, 4) Fetal abnormalities, 5) Skull abnormalities.
- **Compression circulatory hypoxia of the brain (due to circulatory disorders in the fetal brain during labor):** 1) Fast and rapid delivery, 2) Uterine hypercontractility 3) Long delivery and weakness of labor activity, 4) Discoordinated labor activity, 5) Wrong presentation and insertion of the head, 6) Pelvic presentation, 7) Transverse presentation, 8) Excessive and asymmetrical configuration of the head, 9) Shoulder dystocia, 10) Discrepancy between the sizes of the fetal head and pelvis of the mother, 11) Use of obstetric forceps and vacuum extractor, benefits, 12) Squeezing out of the fetus.

The first four types of hypoxia are well known, but compression circulatory hypoxia of the brain (CCHB) is the least studied, in which hypoxia occurs primarily in the brain due to impaired blood circulation in it in the pathology of the skull configuration and compression of the head during labor. The main part of the listed causes of hypoxia of the fifth type are given in the International Classification of Diseases and Causes of Death 10 revision under the heading P03 "Damage to the fetus and newborn, caused by other complications of childbirth and delivery". This type of hypoxia under the name “circulatory hypoxia” in 1935 may have identified Gutner MD [1] for the first time, considering it the main and most frequent. It was isolated by other authors [2]. However, this name requires clarification, since it duplicates one of the types of hypoxia studied by general physiology (along with hypoxic, hemic and tissue hypoxia). Therefore, I propose the concept of CCHB, which reflects the cause and scope of the action of hypoxia.

Many write about hypoxic brain damage in case of compression of the fetal head; relevant experimental animal studies were conducted. Thus, in one of these studies, the heads of dogs in the uterus of a fetus were squeezed with intracranial pressure (ICP) control. With compression of the head of the fetuses, an increase in ICP was observed, after 1-2 seconds bradycardia appeared (gradually disappeared), oxygen tension decreased in various parts of the brain, oxidative phosphorylation was disturbed in the brain tissue and pronounced ultrastructural changes in neurocytes occurred (mitochondria, endoplasmic reticulum, Golgi apparatus). In the jugular vein, the content of the partial pressure of oxygen dropped sharply in the fetuses, the pH (acidosis) decreased, and in the umbilical arteries and the vein, slight changes in the gas composition and blood pH were observed; at excess ICP above 18 mm Hg the fetus could not withstand repeated manipulation with a head compression and died [3-6]. Compared with the model of induction of asphyxia by ligation of umbilical cord vessels, with the model with compression of the head, the described changes in the fetus were more pronounced. It follows that cerebral hypoxia can occur without impairment of the uteroplacental and umbilical cord blood circulation.

Hypoxia can be exposed to the brain in the absence of significant systemic hypoxia of the fetus. At the same time, brain hypoxia can be combined with the general hypoxia of the fetus. There are numerous evidences that the fetal heart rate slows the fetal heart rate, increases ICP, decreases cerebral blood flow, oxygenation of the brain blood decreases, but in some cases the head configuration did not lead to impaired cerebral circulation and systemic fetal hypoxia [7-11]. This can be explained by the existence of compensatory mechanisms for compression of the head and the possibility of hypoxic brain damage without systemic hypoxic changes in the body of the fetus and newborn.

CCHB is associated with complications of childbirth leading to impaired cerebral circulation and hypoxia of the brain. The reasons leading to CCHB, other authors refer to the group of "mechanical effects" on the fetus in childbirth, to the birth trauma injuries [12]. Many authors consider mechanical effects on the fetus as the cause of IG. The condition of newborns with the presence of excessive compression of the head and disorders of cerebral circulation is called the "syndrome of compression of the head of the fetus" in childbirth [13]. However, many of the cases cited in the manual relate to typical birth trauma (BT). L. Lindgren [11] suggests “cerebral compression ischemic encephalopathy” (cerebral compression ischemic encephalopathy, CCIE), and BS Schifrin, et al. [14] - “cranial compressive ischemic encephalopathy”. These data indicate the need for medicine in the concept, which would explain the state of hypoxia due to mechanical compression of the head of the fetus during childbirth. It seems appropriate to single out a single concept of CCHB and differentiate it from the birth trauma of the skull. In case of CCHB, brain damage is dominant due to impaired cerebral circulation and hypoxia of the brain tissue. In BT, there may be signs of CCHB, but lesions caused by ruptures, fractures, massive hemorrhages and other injuries of a mechanical nature are dominant. CCHB and BT can be combined diseases.

CCHB is directly related to the configuration (molding) of the fetal head. The configuration of the fetal head is a compensatory-adaptive process that ensures the adaptation of its size and shape to the mechanical forces acting on it as it passes through the birth canal. Thanks to the configuration is the promotion of the head and in the end the birth of a child. This process is carried out thanks to the seams and springs, the elasticity of the bones of the skull, and most importantly - the ability of the bones to shift towards each other, to go over each other. The property of the head to adapt to the size of the birth canal is called configurability [15]. Three grades of head configuration can be distinguished: 1–overlapping of bones over one of sutures, 2-overlapping of bones over 2-3 sutures, 3-overlapping of bones over 4-5 sutures [16]. The second degree is moderate, and the third - expressed. Clinical studies have shown that with the 2nd degree configuration, early decelerations appear (disclosure 9.0 cm or more), with a 3rd degree configuration, early and sporadic decelerations appear (disclosure of uterine throat 5.0 cm or more) [17].

What happens when a head is configured when it becomes abnormal? There are disorders of cerebral circulation. Two phases can be distinguished: 1) without significant pressure of the bones of the skull on the surface of the brain and 2) with the presence of pressure of the inner surface of the bones covered with dura mater on the hemisphere of the brain.

First phase: Moderate configuration of the head, → Overlapping of skullcap bones, → Tension of the tentorium cerebelli, falx and bridge veins, → Disruption of blood flow to the sinuses and subdural veins, → Constriction of the sinuses, tension of the great cerebral vein, → Increased venous pressure in the internal cerebral veins and sinuses, → Disruption of the venous outflow from the brain, → Hypoxia and acidosis of brain tissue.

Second phase: Severe configuration of the head, → Moderate pressure of the inner surface of the bones of the skull on the arachnoid membrane of the brain, → Changing the shape of the brain, → Displacement of the brain mass, → Sliding (moving) of the arachnoid membrane along the dura mater, → Tension of the tentorium cerebelli, falx and bridge veins, intradural hemorrhages, little laceration of tentorium cerebelli, → Compression of the superficial cerebral veins and basal veins, → Increased intracranial pressure, → Obstruction of the outflow of blood in the superficial veins of the head, → Constriction and compression of the sinuses, the tension of the vein of Galen, → Obstruction of blood outflow into the sinuses and through the vein of Galen, → Venous stasis in the hemispheres of the brain tissue, subcortical nuclei, cerebellum and brain stem, → Little subarachnoidal, subpial and subependimal hemorrhages → Hypoxia and acidosis brain tissue.

A further increase in the degree of configuration and the development of pathological variants of this configuration leads to various breaks, compression of the brain tissue by the edges of the bones, intracranial hemorrhages and, consequently, BT of the skull. First of all, there are ruptures cerebellar tentorium, then ruptures in the bridge veins and the falx cerebri. Thus, common mechanisms of pathogenesis exist between CCHB and BT. Therefore; there are cases of BT with small tears tentorium cerebelli and hemorrhages, which can't explain the cause of death. At the same time, there are cases with clear morphological signs of hypoxia, in which scant signs of BT are detected. In addition, many authors write about the close relationship of BT and asphyxia, and also highlight the type of asphyxia as traumatic [1,2]. It seems inappropriate to allocate this type of hypoxia. If there are birth traumatic injuries - tears, intracranial hemorrhages caused by them and signs of brain compression, then BT should be noted. In the absence of these signs and the presence of various dyscirculatory lesions of the brain, it is possible to make a diagnosis of fetal CCHB. There are cases when CCHB is the main disease, and generic traumatic injuries are associated lesions. If generic traumatic injuries are pronounced, accompanied by significant hemorrhages, then BT is often the main disease.

When a fetal head is compressed, three successively developing stages can be distinguished: 1) Stage of the physiological configuration of the head, 2) Stage of the CCHB and 3) Stage of the BT of the skull. At each stage, functional and morphological changes are different. At the first stage, compensatory-adaptive mechanisms come into effect, at the second stage, changes characteristic of CCHB occur, and at the third stage BT occurs. The transition of one stage to another occurs when the action of physical factors increases, the cranial compression increases and the head configuration increases. However, staging can be disturbed or occur very quickly, when the head of the fetus immediately or quickly begins to experience excessive physical effects, compensatory-adaptive mechanisms and CCHB do not have time to develop, structures breaks and subdural hemorrhages occur immediately. Formed BT. It should be borne in mind that the BT of the skull can occur against the background of various types of IH. Each case of fetal death is unique in its own way, which is reflected in the diagnoses.

It should be noted that not every compression of the head is accompanied by a disorders of cerebral circulation and hypoxia of the fetal brain. That is why not all the researchers found this disorders during compression of the fetal skull [9]. The fruit is well adapted to the effects of physical factors, forms a number of compensatory-adaptive mechanisms. Pathology begins when compensation mechanisms are violated. Compensatory-adaptive mechanisms include: 1) displacement of the bones of the skull roof when the head is configured and placed over each other, 2) formation of a subdural exudate in the subdural space, ensuring pressure equalization on the brain surface when the head is configured and the hemispheres of the brain slide along the dura mater, 3) the presence of long bridge veins, which ensure the outflow of blood from the surface of the brain into the sinuses during movements of the cerebral hemispheres, 4) the presence of a cross-sectional passage of the bones, which prevents immense displacement of the bones of the skull with the configuration of the head, 5) immaturity of the cortex and the lack of myelination of the white matter of the brain hemispheres, ensuring plasticity of the brain and changing its shape when the configuration of the head, 6) the presence of Schlesinger's long anastomosing veins, providing outflow of venous blood from the cortex and superficial cerebral veins into the system of internal cerebral veins (with narrowing down of the upper sagittal sinus, with pressure of the bones of the skull on the brain hemispheres), 7) the presence of well-developed epidural venous plexuses spinal cord, ensuring the outflow of blood from the brain to the lower and upper vena cava.

These structural devices level the pathological effects on the brain of certain disorders of cerebral circulation associated with the action of physical forces on the skull and the configuration of the head. Thanks to these mechanisms, the child can be born without hypoxia during prolonged labor, with dystocia, with the use of obstetric forceps and with other physical effects on the head. However, all adaptive mechanisms have their own limits of compensation, beyond which damage and hypoxia of brain tissue occurs. These injuries are of 2 types: 1) ruptures of the veins, cerebellar tentorium, sinuses, the appearance of deep brain squeezing and hemorrhages are signs of birth trauma of the skull; 2) the absence of these lesions with the presence of intradural hemorrhages, subarachnoidal and subpial hemorrhages, perivascular hemorrhages and cerebral edema are signs of CCHB. CCHB is a stage prior to BT, in which little birth traumatic injuries that have no tanatogenetic value may be present.

Thus, CCHB is ischemia, hypoxia and acidosis of the brain and spinal cord tissue, which occur during the pathological configuration of the fetal head during childbirth, leading to impaired respiratory and cardiac functions, as well as to the possible death of the child. At the same time disorders of the placental and umbilical cord circulation may not be. In CCHB, the central nervous system, the brain and the spinal cord, suffers from hypoxia, while other organs can get enough oxygen from the blood. The accumulation of carbon dioxide in the brain and spinal cord causes irritation of the respiratory center. The fetus begins to breathe through the open glottis and aspirates the amniotic fluid, mucus, and blood. A child may die in childbirth or after birth.

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