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Mini Review

What are the Differences between Birth Trauma and Hypoxia?

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Abstract

The article raises a long-standing question about the differences between birth trauma (BT) and hypoxia, which is important for making a correct diagnosis. It is proposed to isolate compression hypoxia (CH) as an independent nosological unit. The necessity of this is substantiated in the work, as well as the signs characteristic of BT and CH are presented. The small focal subarachnoid hemorrhages typical for CH are described.

Keywords: Birth Trauma; Compression (Circulatory) Hypoxia; Hypoxic - Ischemic Encephalopathy; Nosology; Causes of Hypoxia

The question indicated in the title of the article arises when a neonatologist tries to differentiate hypoxic-ischemic encephalopathy (HIE) from birth trauma (BT), when a pathologist or a forensic expert differentiate BT with hypoxia of the fetus and newborn and seek to establish the correct diagnosis. The difficulty in the differential diagnosis arises because virtually any natural birth is accompanied by a physical effect (compression) on the fetal head in the birth canal of the mother. It is difficult to distinguish which hemorrhage is traumatic and which is posthypoxic. Earlier it was suggested that point, small focal and subarachnoid hemorrhages are hypoxic, and massive, large focal and often subdural hemorrhages are traumatic [1,2]. However, to date, the question of the differences between hypoxic and traumatic lesions has not been resolved, many pathologists are not our differences between hypoxic and traumatic hemorrhages. This is due to the fact that to date, such a concept as "compression hypoxia" (CH) [3], which is identical to the concepts of "compression hypoxic encephalopathy" [4,5] and "circulatory hypoxia" [6,7], has not been clearly defined.

In clinical and experimental studies, it has been shown that compression of the head is accompanied by compression of the veins, sinuses and arteries of the brain, impaired venous outflow of blood from the head, increased intracranial pressure, hypoxia and acidosis of brain tissue, impaired oxidative phosphorylation in brain cells, bradycardia, increased blood pressure, etc. [8-10]. There is a state of brain hypoxia without hypoxia in other organs. With significant compression of the head, aspiration of amniotic fluid and fetal death occurs.

It seems that the CH should be isolated from HIE and BT, to represent a special type of nosology. It is thanks to the release of CH that clear boundaries are established between BT and hypoxia. CH is a condition when hypoxic brain damage occurs as a result of circulatory disturbance of the brain caused by compression of the skull and the brain itself. Other conditions of HIE are caused by violations of the uteroplacental and umbilical cord blood circulation, fetal pathology and diseases of the mother.

BT and CH are closely interconnected by the unity of the cause of their occurrence. CH and HIE have much in common in the nature of brain damage (hypoxic and ischemic lesions of neurons and glia, brain necrosis).

How to distinguish between BT and CH? Both brain lesions are caused by compression of the head and skull. The differences should lie precisely in the nature of compression lesions. In BT, traumatic brain lesions are significant, there are tears and fractures, subdural hemorrhages and others. In CH, hypoxic lesions of neurons dominate, but there are also signs of mechanical damage and cerebrovascular accident. It seems appropriate to list all of these lesions.

- **BT lesions:** Skull fractures; ruptures of the tentorium cerebelli, falx, bridging veins, tributaries of the vein of Galen; subdural and epidural hemorrhages; subarachnoid hematomas; large focal intracerebral hemorrhages; areas of brain compression, cephalohematomas [11].
- **Lesions characteristic of CH:** Intradural hemorrhage in the tentorium cerebelli and the falx; the region of periosteal blood congestion of the skullcup; spotty epidural hemorrhages; spotted subpial hemorrhages on the tops of the convolutions due to rupture of small vessels and capillaries between the choroid and the molecular layer of the cortex; subarachnoid hemorrhages in the areas of the cerebral hemispheres, where the bridge veins connect with the arachnoid membrane, caused by ruptures of the inflows of the bridging veins; small focal intracerebral and leptomeningeal hemorrhages in the areas of pressure on the brain of the bones of the roof of the skull coming on each other (mainly in the areas of lambdoid and coronal sutures); intraventricular hemorrhages due to rupture of blood-filled capillaries of the vascular plexuses of the lateral ventricles of the brain; some subependymal hemorrhages; hemorrhages in the region of the quadrangular lobes of the cerebellum caused by pressure of the edges of the tentorium cerebelli; ischemic changes in neurons of the cortex and stem structures, astrogliosis and others [11].

The data presented are based on the results of our own extensive sectional studies of fetuses and newborns. With BT, significant mechanical damage occurs and subdural hemorrhages are often present. With CH, there are hypoxic damage to neurons and various types of hemorrhages associated with a mechanical effect on the brain and its membranes. To prove CH, it is important to diagnose these types of hemorrhages that occur during compression of the head and expressed configuration of the skull. This allows us to prove that brain damage is due to compression of the skull, and not, for example, placental abruption or compression of the umbilical cord.

Conclusion

With compression of the head, expressed configuration, two types of diseases can occur - birth trauma and compression hypoxia. In BT, ruptures, fractures, massive intracranial hemorrhages dominate. In CH, hypoxic-ischemic lesions of the brain dominate and there are small hemorrhages due to compression of the skull. The described brain lesions will help to distinguish between BT and CH, which will interfere with overdiagnosis of BT and intrapartum hypoxia of the fetus.

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