# ULTRASOUND DIAGNOSIS AND CORRELATION BETWEEN OBSTETRICAL TRAUMA AND NEUROLOGICAL DISORDERS

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**Abstract**

*Introduction* Obstetric traumas are neonatal disorders of multiple causes, often unpredictable, with implications for the clinical development of the newborn. Incidence is estimated at 2-7 ‰ live births, decreasing due to improved health care. Mortality 2-3% of all neonatal deaths, 5-8 / 100,000 through physical trauma, 25 / 100,000 through hypoxia. *Objectives* The authors propose a review of severe forms of brain injury resulting from birth traumas, correlated with ultrasound monitoring and clinical evolution. *Results* Depending on the degree of somatic and neurological maturity, respectively gestation age and birth weight, the conditions observed or caused by perinatal insult may be different. Also, the type of mechanical insult (physical or hypoxic) can cause different injuries depending on the intensity or degree of neurological immaturity. Both short-term prognosis and long-term neurological prognosis depend on the early diagnosis of these lesions (subarachnoid hemorrhage, subdural hemorrhage, massive cerebral infarction, perinatal hypoxic-ischemic encephalopathy, grade IV intraventricular hemorrhage), the early establishment of the treatment and, if appropriate, the integration of the baby into the follow-up program and neurological dispensary. *Conclusions* Cerebral trauma due to obstetric causes is still an important cause of infant morbidity and mortality. The most serious brain injuries secondary to severe perinatal insult are: subdural hemorrhage, massive cerebral infarction and severe perinatal hypoxic-ischemic encephalopathy, grade IV intraventricular hemorrhage.

**Keywords:** ultrasound diagnosis, neurological lesions

## Introduction

*Obstetrical traumas* - mechanical, hypoxic, ischemic lesions to which the newborn was subjected in the labor and delivery process, are the major cause of death in the neonatal period. The incidence is 2-7 ‰ of living newborns and mortality - 2-3% of all neonatal deaths, 5-8 / 100,000 through obstetrical trauma and 25 / 100,000 through hypoxia. [1]

Brief history

In ancient Greece, Hippocrates (460-377 B.C.) wrote: “no head injury is not too severe to despair, or too trivial to ignore ", making a distinction between epilepsy and neonatal seizures.

A link between obstetrical trauma and neonatal neurological disorders was made in 19th century by Little (in 1843) that considered cerebral diplegia was associated with a traumatic birth and prematurity and asphyxia to be predisposing factors. Gowers in 1888 reported as etiologic factors asphyxia, transversal presentation and primiparity. In 1931, Ehrenfest noticed that 35% of newborns with laborious birth presented nystagmus, and 12% had retinal hemorrhage.

Risk factors: macrosomia, mechanic/dynamic dystocia, prolonged expulsion, abnormal presentation, prematurity, applying the forceps at birth, using vacuum extraction.

Classification of posttraumatic neurological lesions

In the premature infant, the posttraumatic neurological lesions are hypoxic-ischemic lesions of the periventricular white matter (focal and diffuse cerebral ischemic lesions, haemorrhagic necrosis of basal nuclei and thalamus) and the peri/intraventricular haemorrhage.

In term newborn, mechanical injuries during labor and birth induce asphyxious necrosis of the gray matter and cerebral haemorrhage (epidural, subdural, subarachnoid haemorrhage; periventricular and intraventricular haemorrhage; cerebellum hemorrhage).

The role of transfontanellar US

The transfontanellar ultrasounds are effective in evaluating term and premature newborns with brain lesions and allows early diagnosis and monitoring of brain lesions as long as the anterior fontanelle allows. Echographic signs are closely correlated with the anatomo-pathological data.

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Intracranial haemorrhage (ICH)

The traumatic injury of the newborn's brain is conditioned by mechanical trauma during the process of delivery, with the production of tentorial and vascular ruptures with extensive supra or subtentorial haemorrhages. The incidence of intracranial haemorrhage in newborns is directly correlated with weight loss:

- 50-60% in those with BW under 1000g,
- 10-20% in those with BW 1000-1500g. [1]

ICH are rarely present at birth: 50% occur on the first day after birth, 80-90% occur up to the third day after birth and 20-40% progress during the first week of life. [2]

Epidural hemorrhage are rare in the newborn, up to 2% of total ICH, but are difficult to diagnose, the symptoms are developed later. They are often associated with linear fractures of the cranial bones that occur after the application of forceps or vacuum extractor in a laborious labor. [3,4]

More frequent in term newborn, in fetal disproportions, when pelvic structures are extremely rigid, are the subdural haemorrhages. When the duration of labor is too short, it does not allow sufficient expansion of the pelvic structure, and when is too long, the fetal head is subject to prolonged compression.

Clinical variants of subdural haemorrhage: injury of the cerebellar tentorium with the inferior sagittal sinus rupture, hematoma in the posterior cranial fossa due to the tentorium laceration with the right, transverse sinus or Galen vein rupture, or rupture of superficial cerebral connecting veins.

Clinical signs occur within the first 24 hours after birth: focal or generalized seizures, disturbance of consciousness, irritability, neurological focal signs (hemiparesis, tonic convulsions, pupil dilatation on the same side as the hematoma). Evolution is severe, so diagnosis needs to be established quickly.

Transfontanelar ultrasound reveals large, hyperechogenic formation adjacent to the fronto-parietal lobes (large hematomas of convexity), echodense image between the two hemispheres (interhemispherical, subdural fluid collections), hyperechogenic subtentorial image located above the cerebellar hemispheres – (hematomas in the posterior subdural fossa - rarely encountered). [5]

Subdural haemorrage complications are severe: cerebellum and brainstem compression, obstruction of prepontine cisterns, IV ventricle and aqueduct.

More common in premature newborns than in term newborns (29% in premature under 2000g) [3] is subarachnoid haemorrhage. If the haemorrhage is limited, it is asymptomatic. The extended form has a severe evolution: seizures, precoma or coma, hydrocephalus.

Transfontanelar ultrasound has low sensibility for diagnosis: the brain has an increased echogenity to the periphery or, it can reveal, hyperechogenic enlargement of the Sylvius aqueduct in coronary incidence. [5].

Peri/intraventricular haemorrhage, more frequently in premature babies, ocazional in term newborns, can determine severe neurologic sequels. The incidence is 50% in GA < 30 weeks and BW < 1500 g, and frequency, inversely proportional with BW.

There are 4 degrees of severity:

- First degree (I)– subependymal haemorrhage;
- Second degree (II)– intraventricular (iv) haemorrhage - less than 50% of the lateral ventricle (LV) volume;
- Third degree (III)– iv haemorrhage - over 50% of the LV volume- Fig. 1 [5];
- Fourth degree (IV)– iv and intraparenchymal haemorrhage- Fig. 2 [5].

Obstetrical trauma is associated with ~ 10% of cases with peri/iv haemorrhage of third or forth degree. Fourth degree IV Haemorrhage is the most sever form. In 80% of newborn babies, the haemorrhage occurs in the first 72 hours after birth [2].

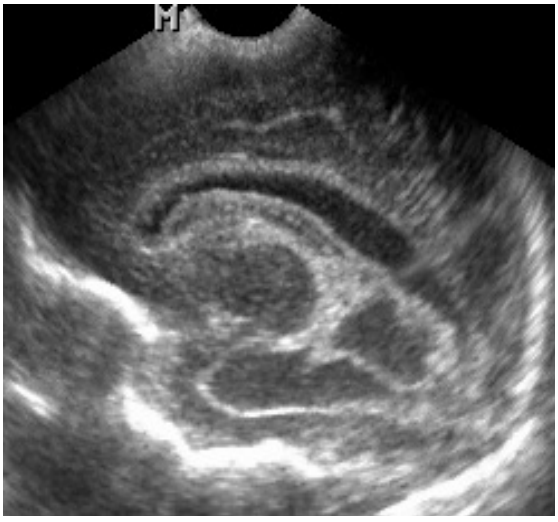


Fig. 1. Dilated lateral ventricles, the blood ocupies more then 50 % of the LV volume. The anterior horns, and especially the posterior horns are dilated and so the aspect of colpocephaly can be observed.

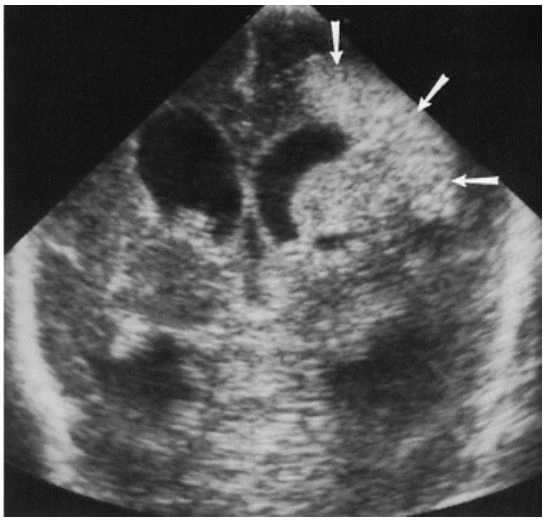


Fig. 2. Fourth degree intraventricular haemorrhage. Extended blood lesion inside frontal and occipital lobs on the same side with the intraventricular haemorhage.

Hidrocephalus is the most sever complication of sever peri/intraventricular haemorrhages (75% of survivors). First ultrasound sign is dilatation of the trigone and occipital horns. At round 2 weeks after the haemorrhage, appears small, hyperechogenic areas - particles with a low amount of proteins floating in the CSF (cerebralspinal fluid) – Fig 3,4 [5]. Obstruction, on several levels: LV near Monro's foramen, in the cerebral aqueduct of Luschka and Magendie, in the pericerebral cisterns.

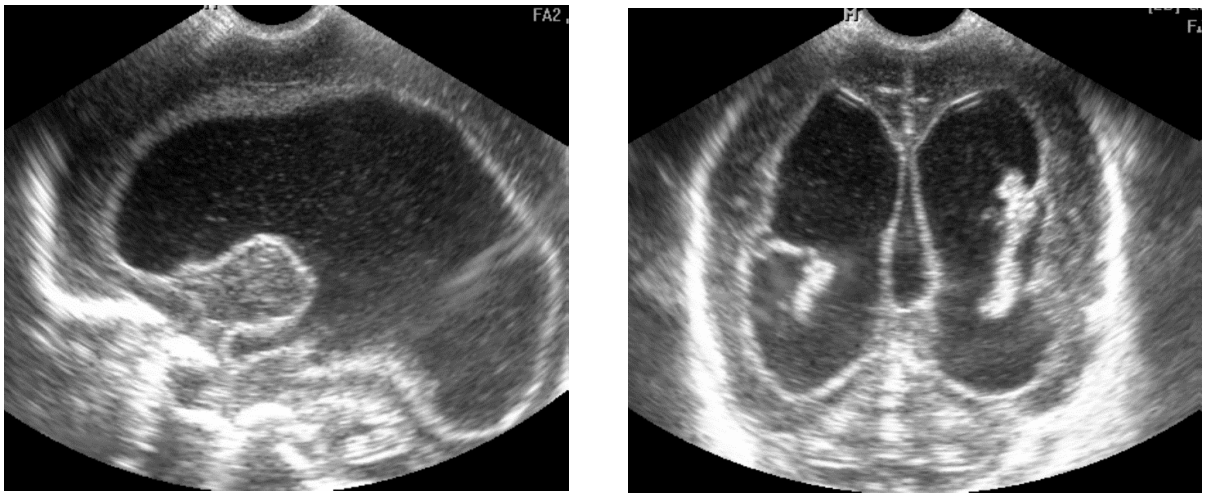


Fig. 3,4. Sever Hidrocephalus with small, hyperechogenic areas - particles with a low amount of proteins floating in the CSF.

Cerebellar haemorrhage

Cerebellar haemorrhage is a severe form of haemorrhage. 15-25% in premature babies with GA <32 weeks and BW <1500g, strongly associated with severe intraventricular haemorrhage [3].

Clinical signs: repeated apnea episodes, severe bradycardia (through bone marrow compression by cerebellar mass), decreasing hematocrit and hemorrhagic CSF. In prematures, onset in 1-21 days of life, with rapid evolution towards death in the first 12-36 hours.

Transfontanelar ultrasound reveals echogenic increase of the cerebellum, inhomogeneous echogenity, asymmetrical, that can cover the entire cerebellar mass in severe forms.

Hypoxic ischemic encephalopathy

Frequent disease in neonatal medical practice with major implications concerning short term and long term health conditio. US is efficient in the evaluation of newborns with hypoxic-ischemic post-traumatic cerebral lesions. The basis of clinical sings are major neuropathological aspects: focal and diffuse ischemic cerebral lesions and the haemorrhagic necrosis of the basal nuclei and thalamus.

Focal and diffuse ischemic lesions are more frequently in term newborn than premature babies and they are responsible for 10-15% of neonatal seizures. The prevalence is 15-20% in the newborn with hypoxic ischemic encephalopathy [6].

Ischemic lesions in the distribution territory of one of the major cerebral arteries, especially middle cerebral artery (over 50%), determinates cerebral infarcts.

Ultrasound investigations (Fig 5,6) reveals hyperechogenic zones of different dimensions well outlined, diffuse in the cerebral cortex, the absence of arterial pulse, the absence or poor delimitation between gyrus and suculus. Cerebral infarction cause neurological sequaels or death in 90% of patients with echographic modifications of the cerebral parenchyma.

Complications of cerebral infarction are:

- cerebral atrophy - echographic: the increase of the interhemispheric fissure, the increase of the distance between gyruses (increased dimension of the suculus);
- ventriculomegaly - in 19-20% of cases;
- parenchymal calcifications;
- multicystic encephalopathy [2].

Hemorrhagic lesion of the basal nuclei and thalamus within a perinatal hypoxic-ischemic encephalopathy are rarely encountered in medical practice. It appears both in preterm newborns as well as in term newborns. Typical echographic signs (Fig 7,8) are: hyperechogenity in the area of the basal nuclei, especially at the level of the caudate nucleus and thalamus, and decrease of arterial pulse.

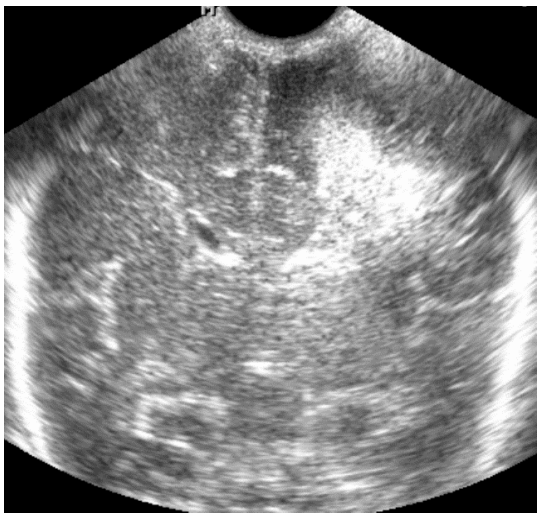


Fig. 5. Severe form of cerebral infarction in the distribution territory of the anterior cerebral artery.

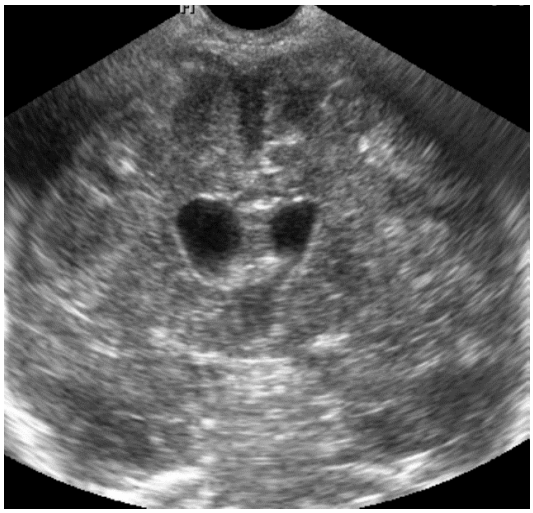
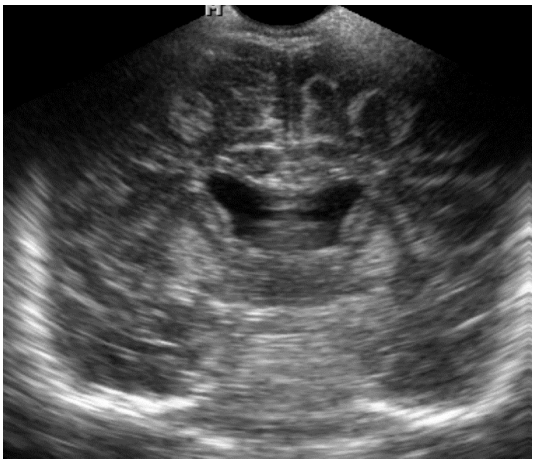


Fig. 6. Cerebral atrophy –characterized by the increase of the interhemispheric fissure, the increase of the distance between gyruses and ventriculomegaly.



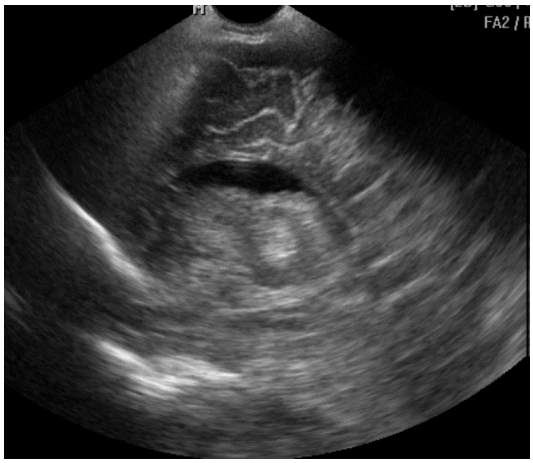


Fig.7,8. Hemorrhagic lesion of the basal nuclei and thalamus.

**Conclusions**

Obstetrical brain injury represent an important cause of infant morbidity and mortality. In the premature infant, the hypoxic-ischemic lesions of the periventricular white matter and peri/iv haemorrhage are the main brain injuries, while in term newborn, mechanical injuries during labor and birth with asphyxious necrosis of the gray matter and cerebral haemorrhage.

Term prognosis and neurological long-term prognosis depend on the early diagnosis of these lesions, on the early establishment of treatment and, if necessary, the integration of the child in the follow-up program and neurological monitoring.

**References**

1. Nicolas Nunes R., Hamilton M., Cardeal D., Traumatic brain lesions in newborns, Arq. Neuro-Psiquiatr. Sao Paulo 2017; 75:170-188
2. T. Michael O'Shea, Karl C.K. Kuban et al., Neonatal Cranial Ultrasound Lesions and Developmental Delays at 2 Years of Age Among Extremely Low Gestational Age Children; Pediatrics 2008; 122: 662-669
3. Volpe JJ. Injuries of extracranial, cranial, intracranial, spinal cord and peripheral nervous system structures. In

Volpe JJ – Neurology of the newborn, 2008, Elsevier Health Sciences: 959-984
4. Kokolakis M., Koutelekos I.; Birth related traumatic brain injury –nursing interventions; Perioperativ nursing 2015; 4:121-136
5. Boia M, Ghid practic de ecografie transfontanelara, Editura Eurobit, Timisoara, 2015.
6. De Vries L., Groenendaal F., Patterns of neonatal hypoxic–ischaemic brain injury; Neuroradiology 2010 Jun; 52(6): 555–566.

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