

# Ultrasonographic Evaluation of the Neonatal Brain in Cases of Birth Asphyxia

SUSMITA GIRI, TAPAN KUMAR JANA, ARUNABHA TAPADAR

## ABSTRACT

**Introduction:** Hypoxic ischemic encephalopathy (HIE) is a common condition in newborns as a result of birth asphyxia. Affected neonates display a number of clinical features by which the condition may be suspected.

**Aim:** To study the different varieties of gross anatomical defects of the neonatal brain in cases of HIE as seen by ultrasonography.

**Materials and Methods:** Ultrasonography (USG) of the neonatal brain was done in 100 cases of hypoxic ischemic encephalopathy (HIE) during the first and second weeks of life, through anterior, posterior and mastoid fontanelles.

The results were analysed to display the spectrum of abnormalities in the affected babies.

**Results:** A large proportion of the neonates had a normal study (47%), followed by intracranial haemorrhage (24%), cerebral oedema and infarction (17%), dilatation of ventricles (5%), and others (7%).

**Conclusion:** Intracranial haemorrhage is the commonest consequence of hypoxic ischemic encephalopathy. The large number of normal studies shows the need for combining ultrasonography with other imaging modalities in selected cases.

**Keywords:** Cerebral oedema, Fontanelle, Hypoxic ischemic encephalopathy (HIE), Periventricular leukomalacia

## INTRODUCTION

The manifestations of hypoxic and ischemic injury to the brain in a developing foetus in utero or during the birth process consist of a pattern of abnormal neurological signs occurring in sequence over a period of the first few days of life. This is known as hypoxic ischemic encephalopathy (HIE) [1]. HIE occurs in about 25% infants following severe birth asphyxia [2]. A clinical staging system has been described in the Sarnat staging system [3] [Table/Fig-1]. A clinical diagnosis of hypoxic ischemic encephalopathy automatically initiates a search for the anatomical location and extent of the brain lesions and a follow up of their progress over the next few weeks. This helps the clinician to formulate a line of management for the affected neonate and form some idea of the short and long term prognosis for the affected child.

## MATERIALS AND METHODS

The present descriptive and analytical study was conducted in the Department of Radiology, R.G. Kar Medical College and Hospital, Kolkata in collaboration with Department of Radiology, Bangur Institute of Neurology, Kolkata. The study was conducted over a period of one year from January 2006 to December 2006.

S No.	Features	Stage - 1	Stage - 2	Stage - 3
1	Consciousness	Alert	Lethargic	Comatose
2	Muscle tone	Normal	Hypotonic	Flaccid
3	Tendon reflexes	Brisk	Exaggerated	Absent
4	Myoclonus	Present	Present	Absent
5	Sucking	Active	Weak	Absent
6	Moro response	Exaggerated	Incomplete	Absent
7	Grasping	Normal	Exaggerated	Absent
8	Oculo-cephalic reflex	Normal	Over reactive	Reduced or absent
9	Pupils	Dilated and reactive	Constricted	Dilated and fixed
10	Respiration	Regular	Periodic	Apneic attack
12	Seizures	Absent	Common	Unconscious
13	EEG changes	Normal	Low voltage periodic / or paroxysmal	Periodic / Isoelectric

**[Table/Fig-1]:** Clinical staging of hypoxic-ischaemic encephalopathy

The patients were selected from the neonatal unit, Department of Paediatric Medicine, R.G. Kar Medical College and Hospital, Kolkata.

Proper informed consent was taken from the babies parents during the study and the Institutional Ethics Committee prior to the study.

A total of 100 cases of birth asphyxiated babies showing clinical features of Hypoxic Ischemic Encephalopathy (HIE) were selected for the present study. Of these, 70 babies were born at term and 30 were pre-term.

Cases included in our study were those neonates who were suspected to be affected by hypoxic ischemic encephalopathy (HIE) according to the clinical features displayed in the Sarnat staging system [Table/Fig-1].

Neonates who showed features of altered consciousness, diminished muscle tone and reflexes as a result of correctable electrolyte disturbances, neonatal infections and developmental anomalies and malformations were excluded from the study.

While all neonates suspected of having HIE were thoroughly investigated and treated, only the first 100 cases referred to the Department of Radiology for neonatal USG after being diagnosed with suspected hypoxic ischemic encephalopathy were included in our study.

In all cases, USG of the brain was done in the 1<sup>st</sup> and 2<sup>nd</sup> weeks of post natal life.

USG was done with Image point HX ultrasonography machine with 3.5 MHz, 5MHz and 7.5 MHz sector and linear transducers. All the images were recorded in a paper print.

Ultrasonographic scans were done through the anterior, posterior and mastoid fontanelles.

**Anterior fontanelle scans** – The sonographic examination of neonatal brain was performed through anterior fontanelle in both the coronal and sagittal planes.

At first coronal images were obtained by placing the transducers transversely across anterior fontanelle. The plane of the ultrasound beam was then made to sweep in an anterior to posterior direction completely through the brain. Six standard frozen images were obtained during this anterior to posterior scan [4,5].

The sagittal images were obtained by placing transducers longitudinally across the anterior fontanelle and angling it to each side. At first the sagittal section was taken in the midline. Then the second parasagittal section was angled about 150 away from the midline to visualise the full sweep of the lateral ventricle. The third parasagittal section was angled outward about 30°, lateral to ventricle and through Sylvian fissure to demonstrate the peripheral aspect of ventricles and more lateral cerebral hemispheres.

**Posterior fontanelle scans** – The posterior fontanelle scan is a very useful view to evaluate the occipital horns for the diagnosis of intraventricular haemorrhage. So this technique

was adopted in the present study for better visualisation of occipital horns.

**Mastoid fontanelle scans** – Scan through mastoid fontanelle allows assessment of the brain stem and posterior fossa that are not well demonstrated in the standard planes through the anterior fontanelle. The ultrasound transducer was placed about 1 cm. behind the helix of the ear and 1 cm. above the tragus. This technique was also included in our study.

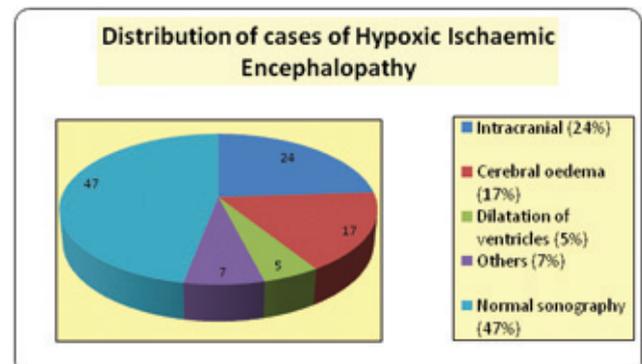
## RESULTS

In the current study, 100 babies were asphyxiated at birth including term and preterm births. Among these 100 babies, intracranial haemorrhage (ICH) was found in 24 babies (24%), cerebral oedema in 17 babies (17%), various degrees of dilatation of ventricles in 5 babies (5%), other abnormalities in 3 babies and no abnormality (normal study) was seen in 51 cases (51%). These findings are displayed in [Table/Fig-2].

Intracranial haemorrhage (24%) included cases of subependymal haemorrhage, intraventricular haemorrhage and intra parenchymal haemorrhage.

The cases of intra parenchymal haemorrhage included cases of periventricular and subcortical haemorrhage. Intraventricular and periventricular haemorrhages may be considered together as Germinal Matrix Haemorrhages (GMH).

In the present study out of 100 neonates, 30 were preterm and 70 were mature (term). The distribution of lesions according to maturity is displayed in [Table/Fig-3].



[Table/Fig-2]: Distribution of cases of Hypoxic Ischaemic Encephalopathy

51 cases of clinically diagnosed hypoxic ischaemic encephalopathy (HIE) showed a normal sonographic scan. Of these, 18 neonates were in Grade I, 18 neonates in Grade II and 15 neonates were in Grade III, according to Sarnat's clinical grading system.

## DISCUSSION

Cranial ultrasound is used as the first imaging modality for newborns [6]. Selection of neonates for ultrasonographic screening of the brain to determine the pattern of hypoxic

Intracranial Lesion	Term Babies (70)	Pre-term Babies (30)
<b>(1) Intracranial haemorrhage</b>		
Subependymal haemorrhage (SEH)	0	06 (20%)
Intraventricular haemorrhage (IVH)	04 (5.71%)	08 (26.67%)
Intraparenchymal haemorrhage (IPH)	02 (2.86%)	2 (6.67%)
Subarachnoid haemorrhage (SAH)	02 (2.86%)	0
<b>(2) Cerebral oedema and infarction</b>		
Diffuse oedema	14 (20%)	01 (3.33%)
Infarction	02 (2.86%)	0
<b>(3) Dilatation of Ventricles</b>		
	03 (4.29%)	02 (6.67%)
<b>(4) PVL</b>		
	0	04 (13.33%)
<b>(5) Thalamic lesion</b>		
	03 (4.29%)	--
<b>(6) Normal Sonography</b>		
	40 (57.14%)	07 (23.33%)

**[Table/Fig-3]:** USG findings of intracranial lesions in term and pre-term babies.

injury requires a high index of suspicion. Patterns of hypoxic ischemic brain injury detected by sonography have been shown to correlate very well with autopsy findings [7].

### Features of Hypoxic Ischemic Encephalopathy

Preterm neonates exhibit very few clinical features suggestive of hypoxic-ischemic brain injury. Detection needs meticulous observation of subtle signs and symptoms. In all neonates the problem may be suspected by the following features –

Impairment or of consciousness or irritability, impairment of muscle tone, tendon reflexes, sucking, Moro response, grasping and oculo cephalic reflex; abnormal pupils, respiration, heart rate; seizures and EEG changes.

### Summary of Human Brain Development

Brain development starts from the epiblast cells of the germ disc of the embryo. The epiblast further differentiates into surface ectoderm and neuro ectoderm. The neuro ectoderm in turn, undergoes a folding to form a neural tube. The cephalic expanded part of the neural tube forms the forebrain, midbrain and hindbrain vesicles.

The cells of the neural tube differentiate in three layers – from inside outwards ependymal, mantle and marginal layers. Cells of the ependymal layer migrate outwards and differentiate into neuroblasts and spongioblasts forming nerve cells and glial cells respectively [8].

### Maturational Changes in the Human Brain in the 3<sup>rd</sup> Trimester

It is generally agreed that the dividing line between the immature and the mature brain is around 34 weeks.

The germinal matrix is a fine network of blood vessels and primitive neural tissue that lines the ventricular system in the subependymal layer during fetal life. As the fetus matures,

the germinal matrix regresses toward the foramen of Monro so that by full term only a small amount of germinal matrix is present in the caudothalamic groove. These delicate vessels are highly susceptible to pressure and metabolic changes, which can lead to rupture of the vessels.

Premature infants are therefore at risk of spontaneous hemorrhage in and around the ventricles [9]. Full-term infants rarely experience this type of hemorrhage [10-12]. There is a high degree of vascular supply to the basal ganglia and the germinal matrix on one side and numerous small penetrating branches from the leptomeningeal vessels to a thin cortex on the other side. A watershed area, therefore, exists between these two vascular territories in the periventricular white matter. HIE related pathological lesions in the immature brain are germinal matrix hemorrhage and periventricular leukomalacia (PVL) which are highly specific and are observed in this watershed area.

After 34 weeks of gestation, the cerebral vascular architecture matures with less chance of haemorrhage [13].

In a nutshell, it may be said that in full term babies, parasagittal cerebral injury occurs as a result of generalised reduction in the cerebral blood flow. In preterm babies, the area of infarction involves the deeper periventricular white matter [14].

In the present study 24% of the neonates showed features of intracranial hemorrhage. This is detected on USG by increased echogenic masses occupying ventricles, separating and thickening the sulci.

Among the hypoxic preterm babies, the greatest incidence was of intracranial hemorrhage while among the full term hypoxic babies the maximum number of cases had a normal scan [Table/Fig-4]. This in general highlights the lability of intracranial blood vessels in the developing foetus as well as the discrepancy between structure and function, between gross anatomical impressions and pathological conditions in full term hypoxic babies.

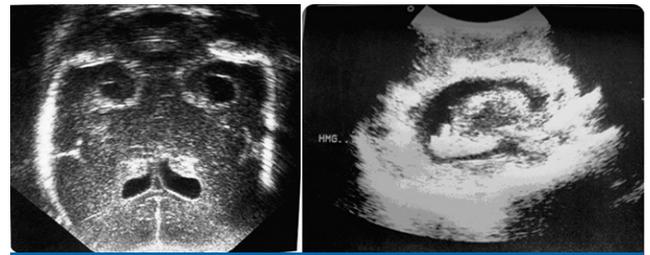


**[Table/Fig-4]:** USG of normal neonatal brain

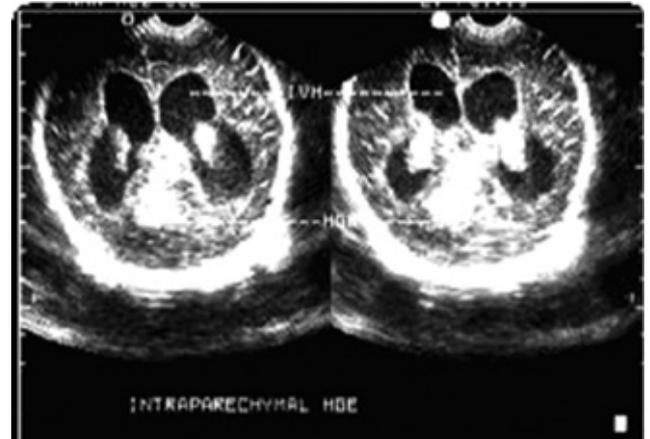
The general features of the lesions found in hypoxic neonates by ultrasonography are summarised in [Table/Fig-5-10].

Lesion	Sonographic Features
Subependymal Haemorrhage (SEH)	A homogeneous, echogenic mass. The echogenic clot often causes focal haemorrhage in the caudothalamic groove or a bulge in the choroid plexus. As the haematoma ages, the clot becomes less echogenic with its centre becoming sonolucent [9,15].
Intraventricular Haemorrhage (IVH)	It appears sonographically as hyper echoic material that fills a portion of the ventricular system or all of a ventricle when the clot forms a cast of the ventricle. The clot itself may obscure the ventricle due to complete filling of the lumen. IVH may or may not be associated with an intra parenchymal haemorrhage [16].
Intraparenchymal haemorrhage (IPH)	Complications of IPH are permanent areas of damaged brain that can become necrotic. Such necrotic areas may degenerate to form a porencephalic cyst [17,18].
Subarachnoid haemorrhage (SAH)	The presence of enlarged interhemispheric and Sylvian fissure with thickened sulci and increased echogenicity can suggest the diagnosis of subarachnoid haemorrhage [19]. Complications of subependymal haemorrhage and intraventricular haemorrhage are intraventricular obstructive hydrocephalus (usually at the foramen of Monro or the Sylvian aqueduct) and extra ventricular obstructive hydrocephalus (at the arachnoid granulations).
Cerebral oedema and infarction	Brain oedema will cause slit-like ventricles in a diffusely echogenic brain with poorly defined sulci. This may cause silhouetting of the sulci, so that the sulci seem to disappear. Infarction shows diffuse brain volume loss with ventricular enlargement secondary to atrophy [20]. The area of infarction may be better defined and increasingly reflective, before gradually changing into echo-poor or echo-free area of encephalomalacia.
Dilatation of Ventricles	Dilatation of ventricles or ventriculomegaly in hypoxic ischemic injury is due to destruction of brain tissue due to infarction. Ventriculomegaly is diagnosed when the lateral ventricles are dilated to $\geq 10$ mm at the level of the atrium at any gestational age [21,22].
Periventricular leukomalacia (PVL)	Periventricular white matter undergoes coagulative necrosis followed by phagocytosis of the necrotic tissue. It shows Periventricular echodense areas evolving into multiple cysts in the parieto-occipital white matter.
Thalamic lesions	Hypoxic ischemic thalamic lesions are relatively uncommon. Such lesions are suggested by normal cranial nerve function with muscle hypotonia and weak sucking and swallowing [17]. Ultrasound examination of the brain may be normal or show increased echogenicity in the thalamic region with evidence of haemorrhage. There may also be dilatation of the lateral and third ventricles [23,24].

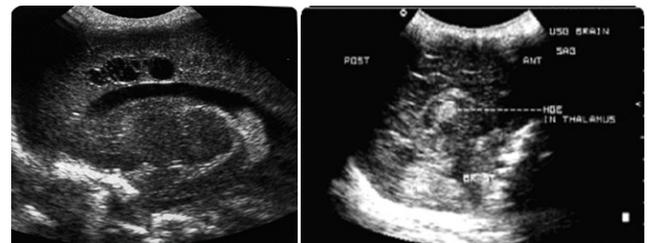
[Table/Fig-5]: Sonographic features of lesions found in hypoxic neonates.



[Table/Fig-6]: USG of neonatal subependymal haemorrhage  
[Table/Fig-7]: USG of neonatal intraventricular haemorrhage



[Table/Fig-8]: Intraparenchymal haemorrhage with hydrocephalus in neonate



[Table/Fig-9]: Periventricular leukomalacia in neonate  
[Table/Fig-10]: Thalamic haemorrhage in neonate

The findings of the present study are displayed with results obtained by other investigators in [Table/Fig-11]. However, the sample size and study conditions have wide variations in most available data.

### Pitfalls of USG Study

Over half of the total number of cases showed a normal USG scan despite having features of Hypoxic Ischemic Encephalopathy (51%). This may be due to several reasons –

- (i) USG may not be the optimal investigation for all cases of HIE. Although USG is invaluable as a screening tool, the level of differentiation and clarity may be insufficient for minor alterations in cerebral structure.

- (ii) Some cases of small subependymal haemorrhages resolve quickly and are not visible by one week.

Lesion	Present Study	Other Studies
Intracranial haemorrhage - Subependymal haemorrhage (SEH) Intraventricular haemorrhage (IVH) Intraparenchymal haemorrhage (IPH) Subarachnoid haemorrhage (SAH)	08 out of 70 term and 16 out of 30 preterm neonates	02 out of 46 term and 32 out of 54 preterm and infants (Imran et al.) [25].
Cerebral oedema and infarction	17 out of 100 neonates	07 out of 32 asphyxiated newborns had cerebral oedema, detected by CT scans [Lupton]. 04 out of 12 neonates suffering from HIE detected with cerebral infarction (Cowan et al.) [26].
Ventricular dilatation	5 neonates out of 100 (02 out of 30 preterm)	66 out of 699 preterm neonates (Vollmer et al.) [27].
Periventricular leukomalacia (PVL)	4 out of 30 premature neonates.	2% to 25 % of premature neonates (Hernandez et al.) [28].
Thalamic lesion	3 out of 100 cases of HIE	12 out of 19 cases of intraventricular haemorrhage (Roland et al.) [29].

**[Table/Fig-11]:** Comparison of present study with those of other investigators.

(iii) Some cases of periventricular leukomalacia and hydrocephalus manifest beyond two weeks.

(iv) Many cases of HIE may have functional abnormalities, but may not have gross structural anomalies.

(v) Alterations in cerebral blood flow and effect of metabolic toxins may not be detectable by routine USG.

A more detailed analysis of the neonatal brain may be obtained by combining USG with cerebral Doppler studies, CT scan and functional imaging methods. However, the portability, availability, ease of application, low cost and lack of side effects and radiation exposure make USG the most desirable initial investigation for neonatal hypoxia.

## CONCLUSION

The term birth asphyxia denotes a constellation of signs and symptoms resulting from inadequate perfusion and oxygenation of the foetus during the process of childbirth. This is known as hypoxic ischemic encephalopathy (HIE). It results in several forms of pathology of the brain characterized by hemorrhage and softening of brain tissue around a periventricular watershed zone. Although, USG is easy to perform and cost effective, functional hypoxic damage to the cerebral neurons

may not always be evident on ultrasonography. Therefore, USG studies may be supplemented with MRI and functional imaging of the brain for better analysis of hypoxic ischemic encephalopathy in neonates.

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