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Original Research Article

# A Study of Deafness in Term Infants with Birth Asphyxia by Otoacoustic Emissions and Brainstem Evoked Response Audiometry Tests

**Subodh Kumar[1], Abhinav Kumar[2]**

[1]Senior Resident, Department of ENT, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India

[2]Senior Resident, Department of ENT, Bhagwan Mahavir Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India

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**Corresponding author: Dr. Abhinav Kumar**

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**Abstract**

**Background:** Birth asphyxia is the leading cause of neonatal morbidity and mortality and results in substantial neurological sequelae. Hypoxic-ischemic damage can involve both peripheral and central auditory pathways, which may make these infants at high risk for hearing loss. The early detection of hearing loss is crucial in preventing resultant speech, language, and cognitive developmental delays.

**Methods:** This was a prospective observational study done in the Neonatal Intensive Care Unit (NICU) of a tertiary care hospital for the duration of 1 year from January 2025 to January 2026. One hundred fifty full-term neonates (≥37 weeks' gestation) who were diagnosed with birth asphyxia were recruited. All neonates had at least one Otoacoustic emissions (OAE) screen before either discharge or 1 month of age. Patients with a "Refer" response on the screening test had been submitted for confirmatory Brainstem evoked response audiometry (BERA), testing to evaluate auditory pathway integrity and measurement of hearing thresholds. The severity of asphyxia was graded as per Sarnat stage of hypoxic-ischemic encephalopathy. Analysis was performed in SPSS, and the relationship of severity to hearing loss was evaluated by the chi-square test.

**Results:** The referral rate of the OAE screening was 20%. BERA confirmed hearing loss was found in 12 (rate 8%). The prevalence of hearing loss is designed with the severity of asphyxia, being highest in infants with severe hypoxic-ischemic encephalopathy. There was a significant relationship between the severity of asphyxia and hearing loss ($p < 0.05$).

**Conclusion:** Birth asphyxia is an important risk factor of neonatal hearing impairment, in particular for severe cases. Single-step screening for bilaterally impaired hearing with OAE and confirmatory BERA helps in early detection and definitive diagnosis. Regular hearing screening for asphyxiated neonates is necessary in order to enable prompt intervention and to optimize developmental outcome.

**Keywords:** Auditory Neuropathy**,** Birth Asphyxia**,** Brainstem Evoked Response Audiometry**,** Neonatal Hearing Loss**,** Otoacoustic Emissions**,** Term Neonates.

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## Introduction

Neonatal hearing loss is a vital global public health problem, which adversely influences speech, language, cognitive and social development when early diagnosed [1]. The World Health Organization reports that millions of children are affected by disabling hearing loss with a significant number occurring in the neonatal period.

Prevalence of congenital or early-onset hearing impairment is 1 to 3 per 1000 live births in the general population and is much higher in high-risk newborns born preterm or those admitted to neonatal intensive care units (NICUs) [2]. In developing countries, this is compounded by the absence of screening programs as well as delayed diagnosis and follow-up. Early detection of hearing loss at the critical period of brain development is crucial for early intervention and best developmental outcomes.

Early hearing detection and intervention (EHDI) programs promote screening by 1 month of age, diagnosis confirmation by 3 months, and enrollment in services by 6 months [3]. This 1-3-6 regulation has proved effective in enhancing language learning and academic performance enormously. But the application of a newborn screen for this condition is not universal across

settings and high-risk infants might go undiagnosed [4]. Of diverse perinatal risk factors, birth asphyxia is a leading cause of neonatal morbidity and mortality, but it has been recently acknowledged to be an important contributor to auditory dysfunction.

Birth asphyxia, which is the failure to provide adequate gas exchange at birth resulting in hypoxemia, hypercapnia and metabolic acidosis, may lead to hypoxic-ischemic encephalopathy (HIE) [5]. The hypoxic-ischemic insult can have a deleterious effect on various organ systems, especially the central nervous system as well as that for hearing. The cochlear micro-environment, especially the outer hair cells, is energetically very active and susceptible to hypoxia. Physiologically, the effect of the functional structure on cell damage in organ of Corti. Hypoxia is generally known as sensorineural hearing loss [6]. Ischemic damage to the auditory nerve and brainstem nuclei resulting from ischemia may also disrupt neural transmission along the auditory pathway. This involvement may cause auditory neuropathy spectrum disorder, where cochlear function is intact but neural conduction is pathological. Consequently, the peripheral and central parts of the auditory system can be adversely affected among infants with birth asphyxia [7].

These two-fold vulnerabilities, unbiased and accurate methods are required to assess hearing in newborn infants. OAE testing evaluates the functional status of cochlear outer hair cells and is a fast, non-invasive screening assessment [8,9]. However, OAE test cannot determine neural pathway lesion. BERA or Auditory Brainstem Response (ABR), a test to measure the integrity of the auditory nerve and pathways in the brainstem by recording responses to electrical stimulation [10]. With the complementation of OAE and BERA in clinics, it is possible to make comprehensive evaluation of cochlear and neural function, and thus increase diagnostic reliability for at-risk infants.

Various studies have investigated hearing outcomes in high-risk neonates, but information specifically on term newborns with birth asphyxia is still lacking and even scarce in resource-limited environments [11]. Differences in methodology and sample size, as well as length of follow-up, have produced an inconsistent picture of the incidence.

The present study, therefore, was addressed with an intent to report the rate of hearing impairment in term neonates with birth asphyxia using OAE and BERA and to find out any correlation between the severity of asphyxia and auditory dysfunction.

**Objective**

- To calculate the prevalence of hearing loss in full-term babies with Birth asphyxia by using OAE and BERA.
- To determine if the degree of asphyxia is related to the type of hearing loss
- To evaluate the nature of hearing loss observed in affected neonates.

**Materials and Methods**

This was a prospective observational study completed in NICU of tertiary care hospital over a duration of one year from January 2025 to January 2026. The data of all full term neonates with diagnosis of birth asphyxia and admitted in NICU during the study were entered into the study. One hundred and fifty term neonates were recruited consecutively after fulfilling the inclusion criteria. Sample size was calculated taking the expected prevalence of hearing impairment among high-risk neonates of similar studies and feasibility within the study period.

**Inclusion Criteria**

- Term neonates (gestational age 37+ weeks).
- Birth asphyxia if Apgar score <7 at 5 min.
- Admission to the NICU for observation or care.

**Exclusion Criteria**

- Preterm infants (<37 weeks of gestation).
- Neonates with congenital malformation in the ear or family history of congenital deafness.
- Infants with known intrauterine infections (example, TORCH), chromosomal anomalies or recognized genetic syndromes that are also linked to hearing impairment.

**Classification of Severity**

The severity of birth asphyxia was determined on clinical and Hypoxic Ischaemic encephalopathy (HIE) - Staging by Sarnat and Sarnat stages. Mild asphyxia (HIE Stage I): This consisted of babies who were either hyperalert, irritable or had normal or increased muscle tone. Moderate asphyxia (HIE Stage II) was characterized by lethargy, hypotonia, seizures and abnormal reflexes. HIE stage III was defined as deep stupor or coma, flaccidity, no reflexes and prolonged seizures. Correlation with hearing results was analyzed according to this classification.

**Study Procedure:** All enrolled infants underwent hearing assessment in two stages.

**Step 1: Otoacoustic Emissions (OAE) Screening**

The testing with OAE screening was performed at the time of discharge or when the infants were around 1 month old in quiet room condition, which used portable OAE equipment. Transient evoked otoacoustic emissions (TEOAE) were subjected to

analysis. Where possible, findings were described as "Pass" or "Refer" using the automated device criteria. OAE in both ears was a pass and these infants were considered to have normal cochlear outer hair cell function on screening.

**Step 2: Brainstem Evoked Response Audiometry (BERA)**

Those neonates with a "Refer" on OAE screening underwent confirmatory BERA. Sleep-stage development BERA was recorded with surface electrodes in natural sleep, to click stimuli at various intensities.

Wave V latency and replicability were measured as thresholds of hearing. The hearing loss was categorized as mild, moderate or severe according to thresholds. Infants who had abnormal or absent BERA and normal OAE data underwent testing to diagnose auditory neuropathy spectrum disorder.

**Outcome Measures:** Outcomes studied was the prevalence of hearing loss in term neonates with birth asphyxia on OAE and BERA. Correlation between the severity of asphyxia and hearing loss, and determination of the nature of the hearing deficit (sensorineural or auditory neuropathy) were secondary outcomes.

**Statistical Analysis:** Data were analysed by Microsoft Excel and SPSS. Demographic and clinical characteristics were summarized using descriptive statistics, including means, standard deviations, frequencies, and percentages. Severity of asphyxia and the hearing impairment relationship were tested by Chi-square. A value of $p < 0.05$ was considered statistically significant.

**Ethical Considerations:** The protocol of this study was approved by the Institutional Ethics Committee of hospital before initiation. Parents or legal guardians provided written informed consent prior to enrollment. Confidentiality of patients' information was strictly upheld; all procedures were carried out in accordance with the ethical considerations as stated in the declaration of Helsinki.

**Results**

The sample size was calculated to be 150 term neonates with birth asphyxia from January 2025 until January 2026. All the infants who were included underwent OAE screening and those who had "Refer" on OAE also had a BERA.

**Demographic Data**

From 150 neonates, 88 (58.7%) were males and 62 (41.3%) were females. Average birth weight of the study population was 2.82 ± 0.41 kg. Regarding mode of delivery, 92 infants (61.3%) were delivered by LSCS and 58 babies (38.7%) were delivered vaginally. The demographic features are described in Table 1.

**Table 1**: **Demographic Characteristics (n = 150)**

| Variable | Number (%) / Mean ± SD |
|---|---|
| Male | 88 (58.7%) |
| Female | 62 (41.3%) |
| Mean Birth Weight (kg) | 2.82 ± 0.41 |
| Vaginal Delivery | 58 (38.7%) |
| LSCS | 92 (61.3%) |

**Severity Distribution:** According to Sarnat staging of hypoxic-ischemic encephalopathy (HIE), 54 infants (36%) were mild asphyxia (stage I), 63 infants (42%) were moderate asphyxia (stage II) and 33 infants (22%) were severe asphyxia (stage III). Moderate asphyxia was the commonest presentation. The distribution is presented in Table 2.

**Table 2**: **Severity of Birth Asphyxia**

| Severity (HIE Stage) | Number (n) | Percentage (%) |
|---|---|---|
| Mild (Stage I) | 54 | 36% |
| Moderate (Stage II) | 63 | 42% |
| Severe (Stage III) | 33 | 22% |
| **Total** | 150 | 100% |

**OAE Results:** OAE screening demonstrated that, 120 infants (80%) had "Pass" result in both ears whereas 30 infants (20%) were found to have a hearing problem and had a concession of either or both of the ear and these subjects were submitted to BERA test for further evaluation. These results are presented in Table 3.

**Table 3**: **OAE Screening Results**

| OAE Result | Number (n) | Percentage (%) |
|---|---|---|
| Pass | 120 | 80% |
| Refer | 30 | 20% |
| **Total** | 150 | 100% |

**BERA Results:** BERA was done for all 30 infants with a “Refer” OAE finding. Of the above, 18 infants showed normal hearing thresholds in BERA (12% of the total sample) suggestive of false-positive OAE screening results. A total of 12 infants (8% of the overall sample) were confirmed to have hearing loss. Of these, 5 infants (3.3%) had mild hearing loss, 4 infants (2.7%) had moderate hearing loss and 3 infants (2%) had severe hearing loss. No cases of auditory neuropathy isolated were seen in the current study. The distribution is presented in Table 4.

**Table 4**: **BERA Findings**

| BERA Result | Number (n) | Percentage (%) (Total n=150) |
|---|---|---|
| Normal Hearing | 18 | 12% |
| Mild Hearing Loss | 5 | 3.3% |
| Moderate Hearing Loss | 4 | 2.7% |
| Severe Hearing Loss | 3 | 2% |
| **Total with Hearing Loss** | 12 | 8% |

**Correlation between Severity and Hearing Loss:** Deafness was more common in those infants who suffered from severe asphyxia. Among the 12 infants with hearing loss, 2 (3.7%) were in the mild group, 4 (6.3%) were in the moderate asphyxia group, and 6 (18.2%) were in the severe asphyxia group. On statistical analysis by Chi-square test the severity of birth asphyxia was found to be significantly associated with hearing loss ($\chi^2$ = 8.76, P=0.013), indicating the risk for development of hearing loss increased with increasing severity of birth asphyxia.

**Table 5**: **Correlation between Severity of Asphyxia and Hearing Loss**

| Severity | Total Cases | Hearing Loss (n) | Percentage (%) |
|---|---|---|---|
| Mild | 54 | 2 | 3.7% |
| Moderate | 63 | 4 | 6.3% |
| Severe | 33 | 6 | 18.2% |
| **Total** | 150 | 12 | 8% |

Such results indicate that a significant relationship exists between the degree of birth asphyxia and the development of hearing loss in term neonates.

**Discussion**

The current prospective observational research was conducted to determine the frequency of hearing loss in 150 neonates with birth asphyxia, using OAE and BERA. The prevalence of confirmed hearing loss in the study population was 8%. There was OAE screening refer rate of 20%; however, permanent hearing loss was confirmed in 12 babies by BERA requiring the need for electrophysiological confirmation. There was a significant relationship between the severity of birth asphyxia and developing hearing loss ($p < 0.05$). The prevalence of hearing impairment rose from lower to higher grades and was highest in infants with HIE stage III. These results contribute further support for hypoxic insult as an important independent risk factor for neonatal hearing loss.

**Comparison with Previous Studies:** The prevalence of hearing loss in high-risk newborns mentioned in national studies ranges from 5 to 12%, depending on the selection criteria and method used [12]. Several Indian studies on NICU graduates have found hearing loss rates of around 6–10%, which is similar to the current study's incidence rate of 8%. To this effect, [13] studies that target birth asphyxia have also reported an increased rate of hearing impairment among those with moderate to severe hypoxic-ischemic encephalopathy.

Auditory impairment is also found at an increased rate among asphyxiated neonates. Studies conducted in industrialized countries have documented hearing loss prevalence of 4% to 15% among infants with severe perinatal hypoxia [14]. Any differences in incidence could be due to variations in screening programs, timing of diagnostic testing, presence or absence of therapeutic hypothermia and follow-up practice. In populations where universal newborn hearing screening is prevalent, early diagnoses could be overestimated because of a diagnostic follow-up procedure.

The progressive increase in hearing loss with increased severity of asphyxia observed is not new to earlier reports. Severe HIE has been repeatedly linked to increased neurological morbidity in the form of auditory pathway damage [15]. The concordance of results between national and international data further supports the association between hypoxic insult and auditory impairment.

**Pathophysiological Explanation:** The auditory system is especially susceptible to hypoxic-

ischemic damage because of its high metabolic requirement.

The cochlea particularly the outer hair cells of the organ of Corti, depends on an adequate supply of oxygen for its normal functioning. Hypoxia can cause cellular edema, oxidative stress and subsequent hair cell injury with sensorineural hearing loss. Furthermore, stria vascularis can be damaged by ischemic insult leading to endolymph production and ionic cochlear balance disturbance.

Apart from peripheral injurious effects, hypoxia may change function in central audiological structures. Cochlear, cochlear nucleus, superior olive and inferior colliculus of the auditory pathways could be damaged in severe asphyxia. Central participation can bring about elongated wave latencies or absent responses in BERA. When cochlear function is maintained and neural processing of sound is disturbed, this may result in Auditory Neuropathy Spectrum Disorder (ANSD). ANSD was not identified in our study, but has been observed in other high-risk neonatal populations.

**Strengths:** This study has several strengths. It was prospective allowing the elimination of recall bias and for systematic assessment. The small study period and large sample size of 150 term neonates is sufficient to be representative. Concurrent application of OAE and BERA made it possible to evaluate cochlear as well as neural hearing, lending the total diagnosis greater credibility.

**Limitations:** Although the study has potential, there are some limitations. It was a single center study performed in the tertiary care set up and hence may not be generalizable to other population profiles.

The duration of follow-up was short without long-term hearing and neurodevelopmental outcomes. The study also did not examine the effects of interventions, such as therapeutic hypothermia on hearing status.

## Conclusion

Birth asphyxia is a major cause of HI among term neonates. This current study revealed an 8% prevalence of hearing loss in asphyxiated newborns, with a distinct risk that gradually increases with the severity of hypoxic-ischemic encephalopathy. OAE can only be used as a highly effective initial screening method for identifying cochlear abnormalities, but it is BERA that must be used to confirm diagnosis and evaluate the integrity of the neural tract. Use of these objective tests together allows for early and reliable detection of hearing loss. Early recognition facilitates the implementation of adequate rehabilitative measures and can improve language development as well as general neurodevelopmental outcomes. Routine assessment should be made of the auditory function in neonates with birth asphyxia.

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