# **BERA in Newborns with Hyperbilirubinemia**

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Type of the Paper: Research Paper. Type of Review: Peer Reviewed. Indexed in: OpenAIRE. DOI: <u>http://dx.doi.org/10.5281/zenodo.1012344</u>. Google Scholar Citation: IJHSP

### How to Cite this Paper:

Dias, Edwin. (2017). BERA in Newborns with Hyperbilirubinemia. *International Journal of Health Sciences and Pharmacy (IJHSP)*, 1(2), 31-35. DOI: http://dx.doi.org/10.5281/zenodo.1012344.

**International Journal of Health Sciences and Pharmacy (IJHSP)** A Refereed International Journal of Srinivas University, India.

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# **BERA** in Newborns with Hyperbilirubinemia

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#### ABSTRACT

#### **BACKGROUND:**

Neonatal hyperbilirubinemia is a common problem seen in the newborn period, is usually mild and transient without long-lasting sequelae. Bilirubin induced neurologic damage may occur and auditory pathway is the most sensitive past to bilirubin toxicity. Auditory brainstem response (BERA) provides an electro physiologic means of assessing ascending auditory pathway and to localise the lesion.

#### AIMS:

To assess the effect of bilirubin toxicity on brainstem auditory pathway among neonates with hyperbilirubinemia using BERA and compare with control of normal newborns.

# MATERIAL AND METHODS:

BERA was recorded in twenty five term newborns admitted to Tertiary care hospital, with hyperbilirubinemia at level exceeding exchange transfusion (mean bilirubin level 25.4+/-4.66 mg/dl). They were compared with 25 term normal newborns without any risk factor for hearing impairment. The results were analysed by Gaussian test (Z), student unpaired 't' test, chi square test and Mann-Whitney U test.

#### **RESULTS:**

At least one of waves I, III and V was absent in 8% of newborn with hyperbilirubinemia. There was statistically significant prolongation of mean latencies of waves III and V and mean I-V interwave latency in hyperbilirubinemia newborn compared to normal neonates. Auditory threshold was elevated in 6 out of 25 jaundiced newborn. Significant positive correlation between BERA abnormalities and bilirubin levels was found with respect to presence of waves I, III and V and auditory threshold. No significant positive correlation was found between bilirubin levels and BERA latencies.

#### **CONCLUSION:**

BERA abnormalities were noted in form of absence of waves I, III, V,prolongation of latency and interwave latencies and increased auditory threshold in newborns with hyperbilirubinemia as compared to normal neonates. BERA abnormality was also found to be transient and was normal in most of these patients during follow up.

Keywords: BERA, Hyperbilirubinemia, Bilirubin toxicity.

#### **1. INTRODUCTION :**

Neonatal hyperbilirubinemia is responsible for neonatal morbidity. Hyperbilirubinemia which is commonly seen in newborn period can cause bilirubin encephalopathy leading to hearing loss [1]. BERA (Brainstem Evoked Response Audiometry) is a non-invasive, effective and objective means of assessing the integrity of central auditory pathway [2, 3]. ABR [Auditory Brainstem Response] is recorded asfive to seven waves. Waves I, III and V can be obtained consistently in all age groups, II and IV waves appear less consistently. Latency of each wave increases and amplitude decreases with reduction in stimulus intensity [4]. The generator sites of waves I-V are auditory nerves, cochlear nucleus, superior olivary complex, lateral leminiscus and inferior colliculus in brainstem [5, 6]. Damage to auditory system has far reaching consequences for affected children as language development is tied to auditory function [7]. The present study was done to assess the effect of bilirubin toxicity on brainstem auditory pathway among newborns with severe hyperbilirubinemia requiring exchange transfusion using BERA and compare results with a control group of normal term newborns. The aim the present work is to assess effect of bilirubin toxicity on brainstem auditory pathway among neonates with hyperbilirubinemia using BERA and compare with control of normal newborns.

#### 2. MATERIAL AND METHODS :

A case control study was conducted in a secondary care hospital in Mangalore.Twenty five term newborns admitted in NICU with hyperbilirubinemia at level exceeding exchange transfusion as per Cokington chart [8] were selected for the study and mean bilirubin level was 24.40 (+/- 4.66) mg/dl. They were compared with twenty five term normal newborns with uneventful perinatal and postnatal period (control group).The test was performed only after patients were stabilized and shifted out of NICU.

Exclusion criteria : Prematurity (<37 weeks), low birth weight (<1500gms), low APGAR scores (0-4 at 1 minute and 0-6 at 5 minute) babies on mechanical ventilation, craniofacial anomalies, exposure to ototoxic medications, history of hereditary childhood sensory neural hearing loss, history suggestive of intrauterine infections or bacterial meningitis.Informed consent was taken from the parents. BERA was performed during their natural sleep after adequate feeds in a partially sound treated room with minimal mechanical and electrical interference. The inverting electrode was placed on the mastoid behind the test ear, noninverting electrode on the forehead and common electrode on the mastoid of the nontest ear. To obtain reliable BERA impedance was kept less than 5000ohms. Bio-Logic AEP VERSION 2.1was used and the stimulus was presented through TDH-39 type earphone. Both ears were tested separately with rarefaction clicks of 0.1ms duration administered at the rate of 13/second. 1024 responses were averaged with filter setting of 100-3000Hz and amplifier gain of 1,00,000. Minimum of two tests were performed for reproducibility. Initially a high intensity stimulus of 75 dBnHL was administered and then the intensity of stimulus was reduced in steps of 15dBnHL till 30dBnHL click. The parameter analysed were:

I ne parameter analysed were:

- Presence of waves I, III and V at 75 dBnHL.
- Absolute latencies of individual waves.
- Interwave latencies I-III, III-V and I-V and
- Wave V response at 30 dBnHL which was taken as pass per fail criteria.

An infant was considered to have passed the test if wave V was present at 30 dBnHL in both ears.

The neonates who passed the initial test were not asked to return for follow-up. Those who failed the initial test were retested after aperiod of 3 months.

#### 3. DATA ANALYSIS :

The results were analysed by Gaussian test (Z), student unpaired 't' test, chi square ( $X^2$ ) test and Mann – Whitney U test, using computer statistical packages SPSS/PC VERSION 7.0.

#### 4. RESULTS :

This study was conducted after the jaundiced infants were treated appropriately with phototherapy and exchange transfusion. ABR abnormalities were observed in hyperbilirubinemic newborns even after therapy with respect to-

- Presence of waves I, III, and V at 75 dBnHL.
- Mean latencies of waves III and V and also mean I-V interwave latency.
- Presence of wave V at 30 dBnHL ( auditory threshold)

In normal newborns at 75 dBnHL, wave I was present in 98% of recordings and waves III and V were present in 100% recordings. Wave V was present at 30 BnHI in all newborns and absent in 6 out of 25 jaundiced newborns.

There was significant correlation between BERA abnormalities and bilirubin levels with respect to presence of waves I, III and V and auditory threshold. No significant correlation was found between bilirubin levels and mean latencies of waves I, III, V and mean I -V

interwave. Among 6 newborns with hyperbilirubinemia who failed 30 dBnHl latency, 5 returned for followup at 3 months, one infant had persistent abnormality and there was significant reduction in absolute latencies of waves I, III and V among other 4 patients. Re-evaluation of the one patient at 6 months and one and half years showed normal hearing but had psychomotor retardation.

#### 5. DISCUSSION :

In hyperbilirubinemic newborns wave I, III and V were present in 68%, 88% and 88% respectively. Study by Soni A showed that 33.3% cases had absent wave forms (at 90 dB) [9]. Wave V was absent at 30 dBnHl in 24% of newborns with hyperbilirubinemia. A study by Agarwal et al showed raised threshold of waveV in 40% of neonates [10]. Our studies showed that the latencies of waves III and V and interwave latency were significantly prolonged incases which have been consistent with study conducted by Soni A where 13.3 % had prolonged latencies and significant interpeak latencies [9]. This study also showed that there was significant correlation between BERA abnormalities and bilirubin levels only with respect to presence of waves I, III, and V and auditory threshold and met with mean latencies. A study by Jadhav et al also showed that BERA abnormalities were significantly more among cases with serum bilirubin levels >15mg/dl [11]. Our study showed that 16.6% of newborns had persistent abnormality at 3 months of age. Similar findings have been observed by Sharma et al where abnormal records persisted in 23.3% of study group [12]. Another study also showed persistence of abnormality in 135 of patients at 3 months [1]. Most of previous studies have shown transient nature of bilirubin encephalopathy were BERA abnormalities were reverted to normal during follow up studies [1, 10, 13]. Our study also demonstrated psychomotor retardation in a child with persistent BERA abnormality during follow up at 6 months and 1 <sup>1</sup>/<sub>2</sub> years of but behavioural audiometry age. and impedance test hearing was normal. Studies have also demonstrated disorders of speech and language in children with auditory neuropathy spectrum disorder [7].

#### 6. CONCLUSION :

Abnormalities of BERA in form of absence of

waves I, III and V, prolongation of latencies of waves III and V and interwave latency, absence of wave V at 30 dBnHl (24%) was noted in neonates with hyperbilirubinemia in comparison to significant correlation between BERA abnormalities and bilirubin levels was found with respect to presence of waves I, III and V and auditory threshold. Hearing abnormality was found to transient and was found to be reverted to normal in most of these patients during follow up.

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