A STUDY ON SCREENING FOR HEARING IMPAIRMENT IN AT RISK NEONATES WITH TRANSIENT EVOKED OTOACOUSTIC EMISSIONS

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BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled "A STUDY ON SCREENING FOR HEARING IMPAIRMENT IN AT RISK NEONATES WITH TRANSIENT EVOKED OTOACOUSTIC EMISSIONS" submitted by **DR. SARANYA.S** to the Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfilment of the requirement for the award of M.S Degree Branch- IV (OTO-RHINO-LARYNGOLOGY) is a bonafide research work carried out by her under my direct supervision and guidance during the tenure of her course in M.S. ENT from May 2015 to April 2018.

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This is to certify that this dissertation entitled "**A STUDY ON SCREENING FOR HEARING IMPAIRMENT IN AT RISK NEONATES WITH TRANSIENT EVOKED OTOACOUSTIC EMISSIONS"** is a bonafide and genuine research work done by **Dr. SARANYA. S** in partial fulfilment of the requirement for the degree of M.S Degree Branch- IV (OTO-RHINO-LARYNGOLOGY) under guidance of **PROF.DR. N. DHINAKARAN, M.S. ENT.,** Professor, Department of OTO-RHINO-LARYNGOLOGY .

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I, **DR. SARANYA.S** declare that, I carried out this work on, "**A STUDY ON SCREENING FOR HEARING IMPAIRMENT IN AT RISK NEONATES WITH TRANSIENT EVOKED OTOACOUSTIC EMISSIONS**" at the Department of ENT, Madurai Medical College during the period from August 2016 to July 2017. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award degree or diploma to any other University, Board , either in India or abroad.

This is submitted to The Tamil Nadu Dr.M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the MS DEGREE examination in OTO –RHINO-LARYGNGOLOGY.

Place: Madurai

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CONTENTS

LIST OF CHARTS

LIST OF TABLES

INTRODUCTION

Hearing is the birthright of every child. It is one of the five senses which help in the development of a child's speech and language skills. It is the most common prevalent deficits of all. Any hearing loss if present has to be detected at the earliest and intervention is the key. All domains including personal, social, emotional and academic achievements of a child can be adversely effected [1-3]. Society should see a hearing impaired child as a responsibility rather than a burden.

Hearing deficit prevalence is 1.5 to 2.6 per 1000 live births (WHO). Four in every 1000 children suffer from severe to profound hearing loss in India. Over 100,000 babies are born with hearing deficiency every year in our country. It can be either unilateral or bilateral. Any degree of hearing loss should be considered for further evaluation. Early intervention, as young as 6 months is the key. [4,5]

In 1994, the Joint Committee on Infant Hearing (JCIH) advocated universal detection of newborns with hearing loss and stated that all infants with hearing loss should be identified before 3 months of age and intervened by 6 months [6].

Hearing assessment is a tool to determine an individual's hearing status, while a screening is a pass/fail tool to determine whether a full assessment is warranted. Screening programs for hearing impairment may be either "universal" or "high risk" population based [7].

In the past, infant hearing screening has been attempted with behavioral audiometry tests and measurement of acoustic reflexes. But for the past 15 years, electrophysiological methods like auditory brain stem response (ABR) are most commonly used. And very recently, measurement of otoacoustic emission (OAE) has attracted much attention, which is a promising method since it is swift, inexpensive, and a noninvasive test of the cochlear function.

The prevalence of bilateral hearing loss is high, in neonates admitted to the neonatal intensive care unit (NICU) who increasingly present with risk factors for hearing loss.

Most common risk factors for hearing loss are congenital infections: In utero infections by cytomegalovirus, herpes, toxoplasmosis, rubella and syphilis

Genetic factors for hearing loss including family history of hereditary hearing loss, first degree consanguinity, head or neck malformations, polymalformation syndrome known to include hearing

loss, maternal intoxication during pregnancy with alcohol or drugs and other conditions of the neonate like gestational age <36 weeks, neonatal birth weight <1,500 g, Apgar score of 0–6 at 5 min, exchange transfusion (hyperbilirubinemia or rhesus incompatibility), medical care including neonatal intensive care unit stay >5 days, newborn ototoxic medication, assisted ventilation ≥24 h, other neurologic disease of the newborn (e.g., meningitis), endocrine disease of the newborn (e.g., thyroidal disease). The first three years of life are important for speech and language acquisition.

Studies have shown that early auditory deprivation impairs with the development of neural structures necessary for hearing. The goal of early identification and intervention is to minimize this adverse effect. Neonatal and infant screening programs using otoacoustic (OAE) emission and auditory brain stem response (ABR) have been established worldwide for this purpose.

The basic notion of newborn hearing screening is that early detection followed by early intervention maximizes the benefits for the child, the family and the society. Improved outcomes for children with congenital hearing impairment are better when confirmation and intervention is perfected by six months of age. Yet even in United Kingdom and United States with good audiology services the median age

of confirmation of congenital hearing impairment has exceeded 18 months. Universal neonatal hearing screening has effectively reduced the age at confirmation of congenital hearing impairments. It is estimated that 10 to 12% neonates have an established risk factor, and among this group with risk factors, 2.5 to 5% have sensorineural hearing impairment.

Studies show that children who received intervention have better cognition than those without. Before the implementation of hearing screening program, it was advised to only test those newborn who have significant risk factors for hearing loss. It is indeed a big challenge to provide special education, vocational training and employment to this large population. The gravity of this problem can only be tackled if available infrastructure is used to mainstream hearing impaired people in regular education, vocational training and employment, by attending to hearing loss on time and instituting appropriate remedial measures. The concept of early identification and intervention is yet to gain foothold in India.

In high risk population screening 50% of the cases will be missed. But it is preferred in our hospitals because of financial constraints and case overload. Kemp introduced Otoacoustic Emissions (OAE) measurement for newborn screening. OAE is sensitive, non-invasive, cost and time effective, making it an ideal screening method [8]. In a normally

functioning cochlea, the outer hair cell emits 'echoes' back which are measured as OAE recorded in the external ear canal by a small, sensitive microphone connected to microcomputer[9].

AIMS AND OBJECTIVES

- To determine the prevalence of hearing impairment in at risk neonates
- To determine distribution of common risk factors in newborns with hearing loss.
- Early detection leading to early intervention
- Improving speech and language acquisitions

Significant hearing loss is one of the most common health problems present at birth and, if undetected, will impede speech, language, and cognitive development. Early detection, intervention, treatment and rehabilitation prevent the consequences of neonatal hearing.

The statement of the Joint Committee on Infant Hearing (1994), supported by the American Academy of Pediatrics (AAP), endorses the goal of universal detection of hearing loss in infants before 3 months of age, with appropriate intervention no later than 6 months of age. Infant distraction test has the disadvantage that it cannot be performed until 6 months of age[10].

ANATOMY AND PHYSIOLOGY

The normal cochlea does not just receive sound. It also produces low-intensity sounds called OAEs. Otoacoustic emission is the sound emitted by the cochlea generated by motion of outer hair cell that can be recorded within the external canal although they occur spontaneously in 50% to 60% of ears (Parving, 1999). Otoacoustic emissions, though their name suggests a unity, cannot be considered to be a single phenomenon. Different types of emissions can be distinguished on the basis of the type of stimulus and of the latency onset with respect to the stimulus onset.

Classification of OAE Types

The phenomena of acoustic emission can be observed by various methods, this it can be classified into

Transient Evoked Otoacoustic Emissions (TEOAE)

Kemp (1978) used a transient excitation to measure the OAEs. He found that after 5 ms post stimulus the original excitation had decayed to a negligible level, but a slowly decaying response component was present between 5 and 20 ms post stimulus. This OAEs has been termed the transient evoked OAEs, or delayed OAE, and is commonly referred to as the cochlear echo. Clicks are the most commonly used stimuli (tone-burst stimuli may be used). Most commonly, 80- to 85-decibel (dB) SPL

stimuli are used clinically. Stimulation rate is less than 60 stimuli per second. TOAEs generally occur at frequencies between 500-4000 Hz.

Distortion Product Otoacoustic Emissions (DPOAE)

Sounds emitted in response to 2 stimulations tones of different frequencies. That is the emissions have components at a frequency, which is not present in the stimulation. The lower tone is usually the F1 and the higher tone the F2. The relative merits of TEOAEs and DPOAEs are widely discussed. Essentially, DPOAEs allow greater frequency specificity and can be used to record at higher frequencies than TEOAEs. DPOAEs has been introduced recently in hearing screening though most screening OAE machines use the transient evoked OAEs.

Stimulus Frequency Otoacoustic Emissions (SFOAE)

Emissions can be evoked at the stimulus frequency by continuous tone. In this method of observation the detailed amplitude and phase variations of the sound in the ear canal are monitored in relation to frequency stimulus. This is caused by the emission interacting with the stimulus, producing cancellation and addition with the stimulus tone. (Wilson, 1980) used a lock-in analyzer to measure the stimulus frequency OAEs from several subjects, and concluded, from measurement of the emission delay, that it must be a function of the cochlea.

Spontaneous Otoacoustic Emissions (SOAES)

Gold (1948) hypothesized that the same active mechanism in the ear, which overcame the damping of the membrane resonance, could result in a spontaneous emission if the positive feedback was too high. Such emission has been found to exist. Several investigators have shown the presence of spontaneous emissions in 30-40% of normal ears.

Only the first 2 types are currently used clinically. Transient evoked otoacoustic emissions are a major subclass of evoked OAEs, because these responses are commonly elicited by the use of brief acoustic stimuli. Commonly used transient stimuli are clicks, single sinusoids, or tone bursts. A major condition to register these emissions, elicited by different stimuli, in the outer ear canal is the reverse conductance of the vibratory energy from the cochlea, through the middle ear (ossicular chain, tympanic membrane) and the outer ear canal. In the outer ear canal, this vibratory energy is transformed to acoustic energy by using the tympanic membrane as a kind of loudspeaker. In our study we used the TEOAEs (Echocheck) using click Stimulus tone bursts.

Limitation of OAE

Spector, et al., (1991) reported that TEOAEs limitations is due to their inability to provide good frequency specific information, because the click is a wide-band signal which stimulates the entire cochlea.

Another limitation of TEOAEs is that it cannot quantify the degree of hearing loss of the subjects. It is well documented that OAE testing has a high false positive rate (up to 15.6%) in the first 24 hours of life, falling to about 4 % by 72 hours. Some of this is related to middle ear effusion and debris in the external ear canal, and it may also be related to neurological immaturity. According to the American Academy of Pediatrics, the recommended median age of testing is 48 hours, thereby eliminating any early neonatal problems (Kei et al., 1997).

Prevalence of OAE

From the first report of OAEs it was found that they were present in normal ears but were absent in cases of deafness. For otoacoustic emissions to be an effective indicator of normal physiology. Kemp, (1978), & Johnsen and Elberling (1982) found that emissions occurred for the entire subject they tested with normal ear 100%. It is apparent that OAE has a high prevalence, but not all the researchers were able to measure emissions in all normal subjects tested. Dijk et al., (1987) found emissions present in 85% of the 210 normal subjects tested. Although all these studies used transient stimuli to evoke the emissions.

Clinical Applications of OAE

The clinical applications of otoacoustic emissions are mainly focused on the identification of sensorineural losses in the auditory

periphery. Despite the fact that the otoacoustic emissions signals are affected by alterations in the sound transmission chain (outer ear to middle ear and middle ear to outer ear) there are no current applications based on the transmission loss concept. The presence of OAEs provides direct evidence of the existence of an active mechanism in the cochlea. Otoacoustic emissions have potential for the study of the detailed mechanical function of the cochlea in a noninvasive and objective manner otoacoustic emissions have potential clinical importance and will function in the near future as a supplement to other standard clinical methods. Therefore measurement of otoacoustic emissions in neonates and young infant is rapidly becoming widespread.

Role of OAE In Neonatal Hearing Screening

Rutten, (1980) concluded that physiology vulnerability of the OAEs seems important for early detection of progressive hearing loss. Kemp suggest that the potential application of OAEs is the registration of the detailed otoacoustic parameters of the patient for future use indicating early changes in the ear. The OAE test has possible application such as;

- The patients with handicaps children in special school.

- Neonatal hearing screening (targeted or universal)[26].

- Children hearing screening

- Monitoring of the course of a potentially ototoxic medications

- Noise induces hearing loss monitoring in industrial, and or military environment.

- Differential diagnoses (between OAE present and ABR altered).

Effect of The Ear Pathology of The Presence of OAE

Many studies have been performed on the occurrence of OAE in abnormal ears. Kemp, (1978) found no emissions in subjects who had best threshold of greater than 30 dB HL. (Rutten 1980) found that if an OAE was present at a given frequency, then the audiogram threshold at this same frequency was better than 15 dBHL.

Bray and Kemp, (1987) found that subjects with conductive losses, due to diseased middle ears had no measurable. Even though the cochlea may well have been functioning normally, the poor transmission of the emission, from the cochlear to the eardrum, resulted in the emission being immeasurably small. In addition, the stimulus is also attenuated as it is propagated from the ear canal to the cochlear, and as a result the cochlear receives less stimulation.

Anderson & Kemp (1979) and Johnsen & Elberling, (1982), have both investigated the effect of ototoxic drugs on the OAE. Johnsen & Elberling induced a flat sensorineural hearing loss of 25-30 dB HL using serum salicyate. They found that the emission virtually disappeared. However, after 2 days complete recovery of the emission occurred.

Anderson & Kemp used injection of both furosemide and ethacryic acid in laboratory primates to study the effect on the emission caused by these drugs. They found that administration of each drug caused a substantial reduction of the emission intensity, within minutes followed by some degree of recovery (within hours).

When sound is used to elicit an emission, it is transmitted through the outer ear, where the auditory stimulus is converted from an acoustic signal to a mechanical signal at the tympanic membrane and is transmitted through the middle ear ossicles; the stapes footplate moves at the oval window causing a traveling wave in the fluid filled cochlea. The cochlear fluid's traveling wave moves the basilar membrane; each portion of the basilar membrane is maximally sensitive to only a limited frequency range. The arrangement is a tonotopic gradient. Regions closest to the oval window are more sensitive to high-frequency stimuli. Those regions further away are most sensitive to lower-frequency stimuli for OAEs, therefore, the first responses returned and recorded by the probe microphone emanate from the highest-frequency cochlear regions, because the travel distance is shorter. Responses from the lower-frequency regions, closer to the cochlear apex, arrive later. When the basilar membrane moves, the hair cells are set into motion and an electromechanical response is elicited, while an afferent signal is

transmitted and an efferent signal is emitted. The efferent signal is transmitted back through the auditory pathway, and the signal is measured in the outer ear canal. As described above, the responses from the high-frequency region arrive first, progressively followed by responses from lower-frequency regions. Outer hair cells are located in the Organ of Corti on the basilar membrane. These hair cells are motile; an electrochemical response elicits a motoric response. The 3 rows of outer hair cells have stereocilia arranged in a 'W' formation. The stereocilia are linked to each other and, therefore, move as a unit. These are the outer hair cells believed to underlie OAEs generation. The ear canal supports (resonates or enhances) sound vibrations best at the frequencies, which the human ears hear most sharply. This resonance amplifies the variations of air pressure that make up sound waves, placing a peak pressure directly at the eardrum. For frequencies between approximately 2 KHz and 5.5 KHz, the sound pressure level at the eardrum is approximately 10 times the pressure of the sound at the auricle. There are two types of nerves at the base of the hair cells: "afferent nerve fibers" carry sensory information away from the cells to the brain while "efferent nerve fibers" bring information from the brain to the hair cells. These afferent neural pulses are then collected and sent out the internal acoustic meatus via the auditory nerve thus translating

mechanical information into neural information. Once the auditory nerve has received the neural impulses, it continues the signal through various pathways in the brainstem. From the auditory nerve, signal information sent to the cochlear nucleus, then proceeds to the superior olivary complex, to the lateral lemniscus, to the inferior colliculus, and to the medial geniculate body, until reaches its final resting place in the brain, the auditory cortex. The auditory cortex then interprets the signal into sound where, from previous experience, we are able to understand what that sound represents.

Signal Morphology of TEOAE

A typical TEOAEs signal consists of acoustic "burst spindles", the main frequencies, which decrease with increasing time distance from the stimulus. This phenomenon is caused by the tonotopic organization of the cochlea. High frequency components of the acoustic input signals stimulate the more basal parts of the cochlea and cause the earliest responses as a result of the traveling wave whereas the lower frequency components stimulate the apical hair cells[27].

Hearing Loss

The Aetiology

Hearing loss can be broadly defined as the decreased ability to receive or process acoustic stimuli. There are many causes of hearing loss in newborns. Some may be temporary and easily corrected for example, a blockage in the ear canal, or fluid in the middle ear may cause a hearing loss. Some hearing loss is permanent and may only be corrected by hearing aids or other listening devices. Maternal infections during pregnancy, such as rubella, may cause an infant's hearing loss at birth. Hearing loss may also be passed on in families. Sometimes there is no known cause for hearing loss in newborns.

Types of Hearing Loss

There are three basic types of hearing loss;

A - Conductive hearing loss

B - Sensorineural hearing loss

C - Mixed hearing loss.

Conductive Hearing Loss

Conductive hearing loss occurs when sound is not conducted efficiently through the outer and middle ears, including the ear canal, eardrum, and the tiny bones, or ossicles of the middle ear. Conductive

hearing loss usually involves a reduction in sound level, or the ability to hear faint sounds. This type of hearing loss can often be corrected through medicine or surgery. Absence or malformation of the pinna, ear canal, or ossicles can cause a conductive hearing loss. Presence of a foreign body; impacted ear wax (cerumen) fluid in the ear associated with colds, allergies, ear infections (otitis media) or a poorly functioning eustachian tube are all examples of conditions that may cause a conductive hearing loss.

Sensorineural Hearing Loss

Sensorineural hearing loss occurs when there is damage to the inner ear (cochlea) or to the nerve pathways from the inner ear (retrocochlear pathway of the acoustic nerve) to the brain. Sensorineural hearing loss not only involves a reduction in sound level or ability to hear faint sounds, but also affects speech understanding or ability to hear clearly. Sensorineural hearing loss can be caused by diseases, birth injury, drugs that are toxic to the auditory system, and genetic disorder with or without syndromes. Sensorineural hearing loss may also occur as a result of noise exposure, viruses, head trauma, aging, and tumors. Sensorineural hearing loss cannot be corrected medically or surgically, it is a permanent loss.

Mixed Hearing Loss

Sensorineural hearing loss occurs in combination with a conductive hearing loss. In other words there may be damage in the outer or middle ear and the cochlea or auditory nerve. When this occurs, the hearing loss is referred to as a mixed hearing loss.

Degree of Hearing Loss

Degree of hearing loss refers to the severity of the loss. There are 5 categories that are typically used. The numerical values are based on the average of the hearing loss at three frequencies 500 Hz, 1000 Hz, and 2000 Hz in the better ear without amplification.

Degree of hearing loss accordingly to the (WHO) classification;

1- Normal no impairment = 0 - 25 dB (better ear).

2- Mild impairment = 26- 40 dB (better ear).

3- Moderate impairment = 41- 60 dB (better ear).

4- Severe impairment = 61- 80 dB (better ear).

5- Profound impairment = 81 dB or greater (better ear).

High Risk Criteria

According to Joint Committee on Infant Hearing and American Academy of Pediatrics, followings are the high-risk criteria [10];

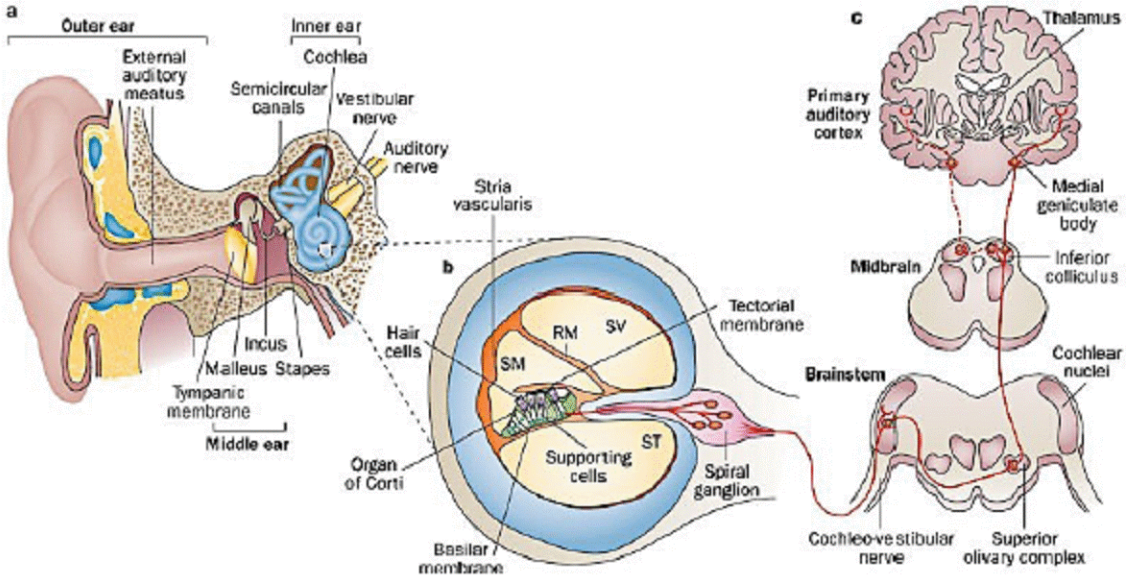
1. Family history of hereditary childhood sensorineural hearing loss
2. In utero infections such as toxoplasmosis, cytomegaly, rubella, herpes simplex and syphilis.
3. Craniofacial anomalies including those with morphologic abnormalities of the pinna and ear canal
4. Birth weight less than 1500 g.
5. Hyperbilirubinemia at serum level requiring exchange transfusion
6. Ototoxic medication
7. Bacterial meningitis
8. Postnatal asphyxia (Apgar < = 5 at 1 minute or < = 6 at 5 minutes).
9. Mechanical ventilation lasting 5 day's or longer Stigmata or other findings associated with syndrome known to include a sensorineural and or conductive hearing loss. (NIH, statement 1993).
10. Stigmata or other findings associated with syndrome known to include a sensorineural and or conductive hearing loss. (NIH, statement 1993).

Sound and its Transmission

Sound is audible disturbance in the particle density; where a particle is made of many molecules of sound propagating medium. This is triggered by a sound producing body or source. It is propagated when this disturbance travels through an elastic medium. Pure Tone: sound produced by a simple harmonic motion. Noise: complex vibration with no repetition. Impedance: the ratio of acoustic pressure to the volume velocity generated by the acoustic pressure. Resonance: When the acoustic impedance is at its lowest point— that is, at the frequency where the stiffness and mass components of the acoustic impedance cancel each other out—the system is said to be in resonance.

Propagation of sound depends on the nature of medium, irregularities or inhomogeneities it contains and on the boundaries of the medium[28]. Sound travels in all directions from the source. As sound propagates intensity (loudness) decreases. Air is the best medium because of its low impedance. Water has a higher impedance, so when sound passes from air to water; a portion of the sound gets reflected. This cuts off the amplitude of the travelling sound wave. Importance: cochlea has high impedance compared to air; so if sound travels directly to cochlea only 1% will get transmitted others being reflected back.

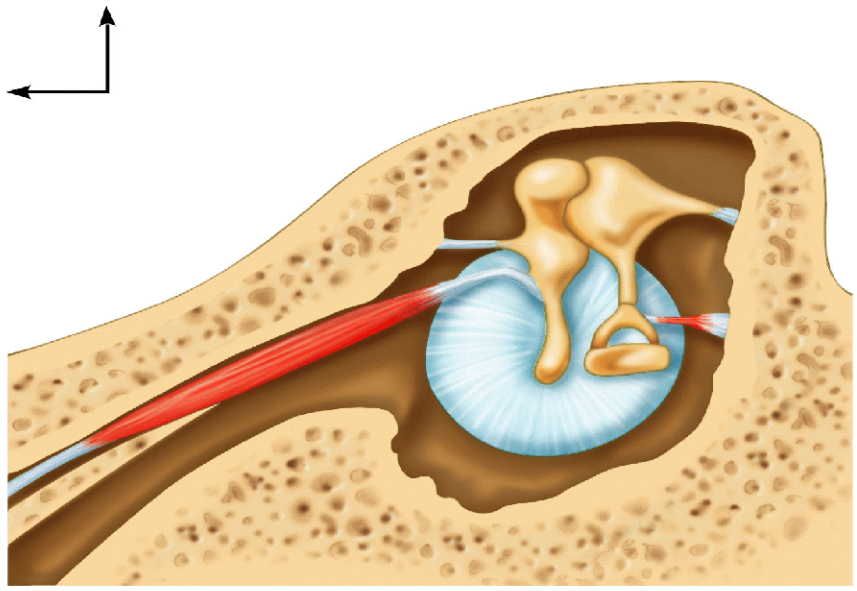
TRANSMISSION OF SOUND FROM EAR TO AUDITORY CORTEX



External ear constitutes pinna, concha and external auditory meatus. Peculiar shape of pinna and EAM gives specific resonant frequencies. Function of external auditory system: It increases the pressure at the tympanic membrane in a frequency sensitive way and helps in sound localization

Tympanic membrane holds stiff upto 2kHz. Reduction of transmitted energy occurs after 6kHz. It has two parts: pars tensa and pars flacida. Pars flacida being more mobile

Middle ear constitutes tympanic membrane, 3 ossicles- malleus, incus and stapes, 2 muscles stapedius and tensor tympani.



Middle ear provides physical protection for cochlea, serves as an acoustic transformer to match the impedance of the air to that of the cochlear fluids. It couples sound preferentially to only one window of the cochlea, thus producing a differential pressure between the windows, required for the movement of the cochlear fluids

Ear ossicles

Malleus: largest of the three. The handle of malleus runs downwards medially & backwards between the fibrous & mucosal layer of tympanic membrane. The tip of the handle of malleus attaches to the membrane at the umbo. Incus articulate with head of malleus Stapes looks like stirrup. The head of stapes articulates with incus & the oval foot plate contact the oval window of the cochlea. The function of ear ossicles is to magnify the intensity of sound by 1.2 to 1.3 times by lever action. The malleus, which is coupled to the TM, vibrates in response to the motion of the TM which causes the entire ossicular chain to vibrate and results in sound transmission to the inner ear via the stapes footplate. This pathway of sound transmission is referred to as ***ossicular coupling.*** The pathway of sound transmission to the inner ear in the absence of the ossicular system is referred to as ***acoustic coupling***. Difference between ossicular coupling and acoustic coupling is about 60 dB

Middle ear muscles: tensor tympani and stapedius increase the stiffness of the middle ear cavity. In effect both controls excessive movement of ossicles and reduce intensity of sound transmission by 30 – 40 dB (↓ 1000 cycles/second)

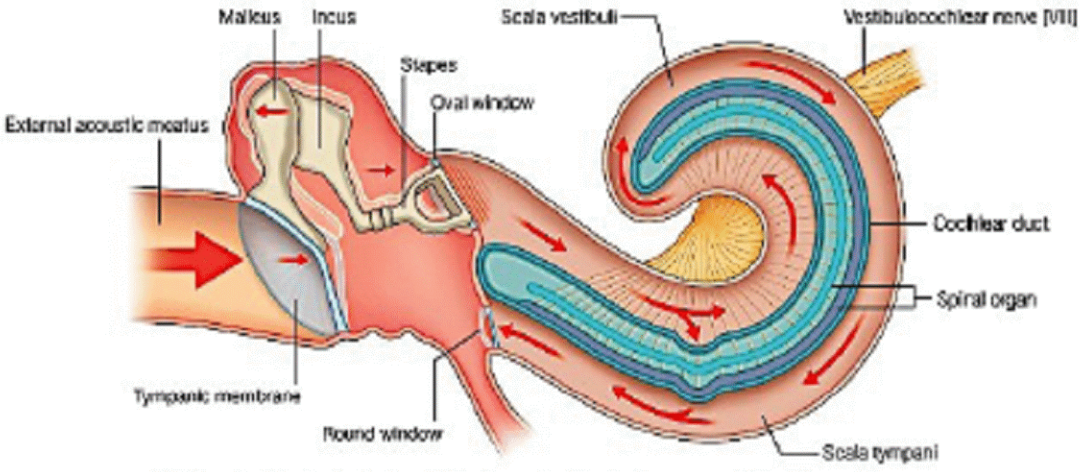
Inner ear

The cochlea is shaped like a snail and has a spiral configuration with two and a half turns. The center portion of the spiral is called modiolus. The portion of the cochlea that is closest to the oval window is the base, whereas the farthest away from the oval window is the apex. The cochlea is a fluid-filled space with 3 compartments known as the scala tympani, scala media, and scala vestibule.

The scala vestibuli and scala tympani are filled with perilymph, which has a composition similar to that of the extracellular fluid (high in sodium, and low in potassium). The scala media is filled with endolymph, which has a similar composition to the intracellular fluid (low in sodium, high in potassium). The unique electrolyte composition of the scala media sets up a large electrochemical gradient, called the endocochlear potential, which is +60 to +100 mV relative to the perilymph. The pressure in the scala vestibuli is higher than the pressure in the scala tympani. A pressure gradient is generated, which causes the cochlear partition to vibrate[29]. It sets up a traveling wave on the basilar membrane, which travels from the base of the cochlea to its apex.

Basilar membrane is tonotopically tuned. The basilar membrane varies in its stiffness along its length, with higher stiffness near the base and lower stiffness near the apex. Higher frequency

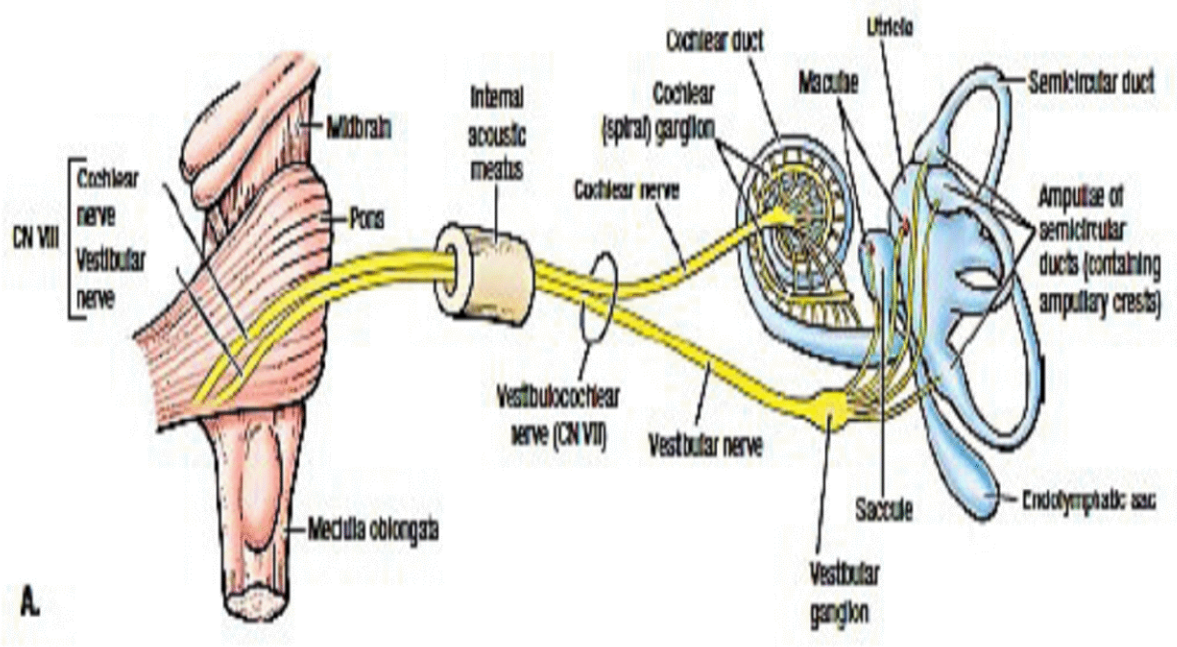
concentrated in the base and lower frequency at apex. This property of the basilar membrane allows it to respond to various frequencies differently.



Cochlear hair cells: Inner hair cells are flask shaped which rests on the side of inner pillar cells. 3000 in number arranged in a single row from base to apex. Outer hair cells are cylindrical shaped and rests on the side of outer pillar cells. 12000 in number arranged in 3 to 4 rows. As the stereocilia are deflected away from the tallest row, the tip links relax and thereby decrease the probability of the ion channel opening; this leads to hyperpolarization of the hair cell. This ion flow converts mechanical signal (inner ear fluid wave) into an electrochemical signal. Once inner hair cells are depolarized, voltage-gated calcium channels open which are concentrated in the basolateral surface of inner hair cells.

These voltage-gated calcium channels trigger release of neurotransmitter; activating the auditory nerve fibers. Outer hair cell can change its length in response to voltage changes[30]. It contracts with depolarization and elongates with hyperpolarization. This change in length is due to the presence of a voltage-dependent integral membrane protein called *prestin*. This adds energy to the basilar membrane motion through a mechanical feedback scheme. Thus outer hair cell acts as a cochlear "amplifier" that augments the signals transmitted into the inner ear by the stapes vibration.

Peripheral auditory system



Central Pathway

Cochlear nucleus is 2nd order with first relay station for all ascending auditory information originating in the ear. It is tonotopically organized and located in the pontomedullary junction of the dorsolateral brainstem. The spatial representation of frequency-specific information in the cochlea is preserved in the cochlear nucleus. From the cochlear nucleus, three fiber tracts project auditory information to the contralateral inferior colliculus. The dorsal stria, also called the stria of Monaco; the intermediate stria, also called the stria of Held; the ventral stria, also known as the trapezoid body. These fiber tracts collectively form the lateral lemniscus.

Superior olivary complex located medial to the cochlear nucleus in the caudal portion of the pons. This region plays an important role in sound localization by analyzing interaural time and amplitude differences. It helps to enhance auditory perception by two additional mechanisms, binaural squelch and summation. Lateral lemniscus is one of the most prominent fiber tracts in the auditory pathway; formed by the three fiber tracts from the cochlear nucleus. It terminates in contralateral inferior colliculus.

Inferior colliculus is located in the midbrain just caudal to the superior colliculus. It processes frequency-specific information and

receives auditory inputs from the lateral lemniscus, cochlear nucleus and the superior olivary complex and projections from the somatosensory system, visual and vestibular systems. It projects on to medial geniculate body.

Medial geniculate body is the thalamic auditory relay station. It has 3 divisions: ventral-projects to primary auditory cortex, dorsal-projects t auditory association area, and medial. Helps in sound localization and processing of complex vocal communications.

Auditory part of cerebral cortex lies in temporal one near sylvian fissure. Primary auditory cortex; area A1; Brodmann area 41. On the superior surface of temporal lobe (Heschl gyrus). It is tonotopically tuned with high frequency medially and involved in language comprehension. Auditory association cortex; area A2; Brodmann area 22 & 42. Location is lateral to the primary auditory cortex. It is a part of Wernickes area and involved in speech perception

Auditory messages are conveyed to the brain via two types of pathway:

• primary auditory pathway which exclusively carries messages from the cochlea

• non-primary pathway (also called the reticular sensory pathway) which carries all types of sensory messages

The primary auditory cortex is located in the temporal area within the lateral sulcus.

Primary auditory cortex

- The major cortical target of the neurons in the medial geniculate nucleus.
- It receives point-to-point input from the ventral division of the medial geniculate complex.
- It contains an accurate tonotopic map.
- The belt areas of the auditory cortex receive more diffuse input from the belt areas of the medial geniculate complex which are less precise in their tonotopic organization.
- The primary auditory cortex has a topographical map of the cochlea.

- The cochlea has decomposed the acoustical stimulus so that it is arrayed tonotopically along the length of the basial membrane.
- The neurons in one stripe are excited by both ears: EE cells.
- The neurons in the next stripe are excited by one ear and inhibited by the other ear: EI cells. This EE and EI stripes alternate.

Primary auditory pathway

- The first relay of the primary auditory pathway occurs in the cochlear nuclei situated in the brainstem (type I spiral ganglion axons). At this level an important decoding of the basic signal occurs according to duration, intensity and frequency.
- The second major relay in the brain stem is in the superior olivary complex. The majority of the auditory fibres synapse there having already crossed the midline.
- A third neuron carries the message up to the level of the superior colliculus; situated in the mesencephalus.
- These two relays have an essential role in the localization of sound. A last relay, before the cortex, occurs in the medial geniculate body (thalamus).
- An important integration occurs: preparation of a motor response (vocal response).

Non-primary pathways

- The final neuron of the primary auditory pathway links the thalamus to the auditory cortex.
- The message, already decoded, is recognized, memorized and perhaps integrated into a voluntary response. The main function of these pathways is to select the type of sensory message to be treated first.
- These pathways are connected to wake and motivation centers and hormonal systems. The first relay is located in the cochlear nuclei (brainstem). From here, the small fibers rejoin the ascending reticular pathway. In the reticular pathway of the brainstem and the mesencephalus, several neural synapses occur. After the reticular formation: the non-primary pathway leads to the non-specific thalamus and then to the polysensory cortex. Conscious perception requires the integrity of both types of pathways.

Cochlear echoes/microphonics

It was discovered by KEMP. In response to a click applied to the speaker in the EAC, there was a peak of pressure at EAC followed by a smaller peak much later. The second wave was a complex wave with a form different for different individuals. If a high frequency

wave was applied, 2nd wave appeared soon and if low frequency it appeared later. It is suggested that the outer hair cells contribute to this.

Auditory neuropathy (AN) or auditory dys-synchrony

Auditory neuropathy (AN) or auditory dys-synchrony is a pattern of sensorineural hearing loss (SNHL). It is defined by a profile of absent or severely distorted auditory brainstem responses (ABR) with preserved otoacoustic emissions (OAE) and cochlear microphonics (CM). Acoustic reflexes are also absent[31]. This electrophysiologic profile suggests an abnormality of the hearing apparatus proximal to the outer hair cell that include the synapses between the cochlear inner hair cells, neurons of the spiral ganglion and the entire auditory nerve. Clinically, the hearing loss is variable with an unsynchronized auditory signal, and often fluctuating with particular difficulty in speech perception in the presence of background noise disturbances.

Many of the risk factors associated with AN are the same as for SNHL. These risk factors include positive family history, hyperbilirubinemia, exposure to ototoxic medications, hypoxia, birth asphyxia and intracranial hemorrhages, including intraventricular

hemorrhage[32-37].. As such, there are no specific risk factors to distinguish AN from cochlear hearing loss.

The overall rate for SNHL in newborns is approximately 4 to 5 per 1000. There is a range of prevalence rates reported in neonates, varying from 1 per 1000 for SNHL in normal newborns to 10 per 1000 in neonates from the high-risk nursery. Among children with diagnosed hearing loss, the prevalence rate of AN is very high, ranging between 5.1 and 14.6 per 100.

AN was first defined by Starr *et al* in 1996; even though there has been case reports of patients presenting with symptoms consistent with AN since 1970s. Thus, there is a paucity of information regarding prevalence rates of AN among neonates. Berg *et al*[39] showed that infants in the intensive care nursery are at significantly higher risk for the development of AN. Many of the infants in their nursery had been transported back to the birth hospital and were not included in the study, skewing their data toward an increased AN incidence.

Infants with AN are at high risk for impaired speech and language development. Management protocols are not well outlined and require a multidisciplinary approach. Early institution of visual modes of communication is extremely important. Sound amplification

may or may not be of benefit, likely due to the lack of temporal synchrony of signal or to the fluctuating nature of the hearing loss. In selected individuals, cochlear implantation may improve auditory, speech and language outcomes. Unless diagnosed early, infants from the intensive care nursery with AN are at risk for adverse language and cognitive outcomes.

REVIEW OF LITERATURE

In 1948, it was predicted that outer hair cells of cochlea was able to produce energy by an active mechanical process by Professor Gold. In 1978, David Kemp proved experimentally that the cochlea is able to produce low intensity recordable echoes called Otoacoustic emissions (OAE). First commercially available instrument to record OAE was designed in 1986. [11]

Otoacoustic emissions (OAE) are low intensity acoustic signals emitted from the cochlea to the external ear canal through middle ear, where they can be recorded. They are generated by active mechanical contraction of the outer hair cells, spontaneously or in response to sounds. They travel in a reverse direction from outer hair cells to basilar membrane, perilymph, oval window, ossicles, tympanic membrane and lastly ear canal. There are four types of OAE-1) Spontaneous OAE (SOEA) 2) Transient OAE (TEOAE) 3) Distortion product OAE (DPOAE) 4) Stimulus frequency OAE. [12]

Spontaneous OAEs: are present in normal hearing healthy persons with hearing loss less than 30 dB. They can be absent in 50% of normal people.

Evoked OAEs: They are further divided into two types depending on the sound stimulus used to elicit them. Distortion product Otoacoustic Emissions (DPOAE) is generated by frequency specific region of the cochlea & has the potential to test micromechanical properties of outer hair cells in frequency specific regions. [13]

Gorga *et al* reported that DPOAE level, noise levels and signal noise ratio were similar in NICU babies, well babies with and without risk factors. DPOAE measurements in infants result in robust responses in majority of ears for frequencies f2 at 2, 3, 4kHz. [14]

Studies conducted at Rapid City Regional Hospital which screened 1002 infants tested with first screening OAE, showed refer of 111 infants which reduced to 2 at second OAE screen. On further evaluation with brainstem evoked responses (BERA) of these referred cases only 1 baby was diagnosed to have sensorineural

hearing loss. They concluded that OAE testing can be accomplished in a normal newborn nursery with acceptable false positive rates when two stage approach is used. [15]

Studies done at CMC Vellore on 500 infants reported 6.4% and 1.6% hearing loss at initial and repeat screen with OAE. Thus concluded that screening done by OAE followed by an BERA, would minimize referral rates.[15]

Study conducted on sensitivity and specificity of portable transient otoacoustic emission (TEOAE) in newborn hearing screening on July 2004 at Department of Otorhinolaryngology, Medical Faculty, University Kebangsaan Malaysia, Kuala Lumpur showed that TEOAE is sensitive and moderately specific screening tool for hearing impairment as compared to the other studies. At second and third stage screening test, the sensitivity was 100% for both groups. Whereas the specificity was 68% and 74.1% for post natal and NICU patients respectively. Although the test is quick, noninvasive, easy and does not require skilled personnel to perform, further study is needed to improve the specificity.

MATERIALS AND METHODS

This study was a hospital based prospective study undertaken in the department of Otorhinolaryngology, Government Rajaji Hospital, Madurai; a tertiary care referral hospital. The study was done over a period of one year from August 2016 to July 2017. Ethical committee clearance was given by the hospital ethical committee. Since my study was hearing assessment in neonates, seeked help from Department of Paediatrics, Government Rajaji Hospital, Madurai. Informed consent was taken from the parents and the guardians after explaining to them, the purpose of the study.

SAMPLING FRAME

The sample size of the study group comprised of the 110 neonates between the age of 1 and 28 days selected by consecutive sampling method who were considered to be high risk patients as described below for hearing loss:

- Prematurity
- Perinatal asphyxia determined by APGAR less than 6 at 5 minutes
- Birth weight (<1500gm)
- Hyperbilirubinemia requiring exchange transfusion
- Culture positive neonatal sepsis

- Family History of hereditary childhood sensorineural hearing loss

Neonates with malformed ear, middle ear pathology and birth injuries were excluded from the study.

METHODOLOGY

The case history and clinical examination including otoscopy was done thoroughly for all 110 cases. After getting informed and written consent from parents, neonates are subjected to primary screening with transient evoked otoacoustic emission testing before discharge from hospital.

If initial screening result falls under refer criteria, repeat TEOAE after 4 weeks is suggested.

If the repeat TEOAE result falls under refer criteria, confirmative evaluation with BERA was done.

The neonatal hearing screening was carried out by portable transient evoked automated otoacoustic emissions (OAE) before discharge from the hospital. OAE was carried out in both ears using a portable device which uses click stimuli involving frequency bands between 1,500 Hz and 3,800 Hz. The click is presented at an intensity of 75 to 83 dBpeSPS. The response was considered positive (passed) when the otoacoustic emissions captured were 6 dB higher than the noise. No

further test are to be done for neonates who have their otoacoustic emissions under pass criteria. The parents of newborns who had met the pass criteria were informed regarding the delayed-onset hearing impairment and follow-up of their children regarding hearing loss was recommended. They were not called back for screening again as this implementation did not exist in our screening program.

If there was no clear emission in either ear, infants undergo secondary testing with the OAE. In absence of emissions in secondary OAE, infants were advised to have brainstem evoked response audiometry(BERA) tested.

Measurement of BERA produced by a series of clicks at 45, 90 and 110 dBNHL was made via three scalp electrodes, an averaging computer, and a printer.

The presence and latency of the ABR were determined by a trained observer. Screening was done by two trained audiologists under the supervision of an otorhinolaryngologist.

INFORMATIONS COLLECTED DURING THE STUDY

- Gestational age at birth
- Birth weight
- APGAR Score at 5'
- Blood investigations including Total, direct and indirect Bilirubin, C reactive protein
- Otoscopic examination
- Tympanometry
- Transient evoked otoacoustic emission testing
- Brainstem Evoked Auditory Response

❖ Gestational age at birth

This study included preterm babies according to WHO definition[17]. World Health Organization has defined preterm as babies born alive before 37 weeks of pregnancy are completed. There are sub-categories of preterm birth, based on gestational age:

extremely preterm (<28 weeks)

very preterm (28 to <32 weeks)

moderate to late preterm (32 to <37 weeks).

❖ **Neonatal birth weight**

Birth weight of less than 1500g was considered as the risk factor in the study. Low birth weight (LBW) is defined by the World Health Organisation as a birth of a live born infant of 2,499 g or less, regardless of gestational age. Very low birth weight (VLBW), is birth weight less than 1500 g (3 pounds 5 ounces), extremely low birth weight (ELBW), birth weight is less than 1000 g (2 pounds 3 ounces). Normal weight at term delivery is 2500–4200 g (5 pounds 8 ounces – 9 pounds 4 ounces).

❖ **Perinatal Asphyxia**

Perinatal asphyxia, neonatal asphyxia or birth asphyxia is the medical condition resulting from deprivation of oxygen to a newborn infant that lasts long enough during the birth process to cause physical harm, usually to the brain. Hypoxic damage can occur to most of the infant's organs (heart, lungs, liver, gut, kidneys), but brain damage is of most concern and perhaps the least likely to quickly or completely heal. In more pronounced cases, an infant will survive, but with damage to the brain manifested as either mental, such as developmental delay or intellectual disability, or physical, such as spasticity.

It results most commonly from a drop in maternal blood pressure or some other substantial interference with blood flow to the infant's brain during delivery.

This can occur due to inadequate circulation or perfusion, impaired respiratory effort, or inadequate ventilation. Perinatal asphyxia happens in 2 to 10 per 1000 newborns that are born at term, and more for those that are born prematurely. WHO estimates that 4 million neonatal deaths occur yearly due to birth asphyxia, representing 38% of deaths of children under 5 years of age.[18]

Neonates with Apgar score of less than 4 at one minute or less than 6 at fifth minute were taken for this study.

Apgar score is a method to quickly summarize the health of newborn children. Dr. Virginia Apgar, anesthesiologist at NewYork–Presbyterian Hospital, developed the score in 1952 in order to quantify the effects of obstetric anesthesia on babies[16].

The Apgar scale is determined by evaluating the newborn baby on five simple criteria on a scale from zero to two, then summing up the five values thus obtained. The resulting Apgar score ranges from zero to 10. The five criteria are summarized using words chosen to form a acronym (Appearance, Pulse, Grimace, Activity, Respiration)

The test is generally done at one and five minutes after birth, and may be repeated later if the score is and remains low. Scores 7 and above are generally normal, 4 to 6 fairly low, and 3 and below are generally regarded as critically low.

A low score on the one-minute test may show that the neonate requires medical attention[4] but does not necessarily indicate a long-term problem, particularly if the score improves at the five-minute test. An Apgar score that remains below 3 at later times—such as 10, 15, or 30 minutes—may indicate longer-term neurological damage, including a small but significant increase in the risk of cerebral palsy. However, the Apgar test's purpose is to determine quickly whether a newborn needs immediate medical care.

APGAR SCORING SYSTEM

	0 Points	1 Point	2 Points	Points totaled
Activity (muscle tone)	Absent	Arms and legs flexed	Active movement	
Pulse	Absent	Below 100 bpm	Over 100 bpm	
Grimace (reflex irritability)	Flaccid	Some flexion of Extremities	Active motion (sneeze, cough, pull away)	
Appearance (skin color)	Blue, pale	Body pink, Extremities blue	Completely pink	
Respiration	Absent	Slow, irregular	Vigorous cry	

Severely depressed	0-3
Moderately depressed	4-6
Excellent condition	7-10

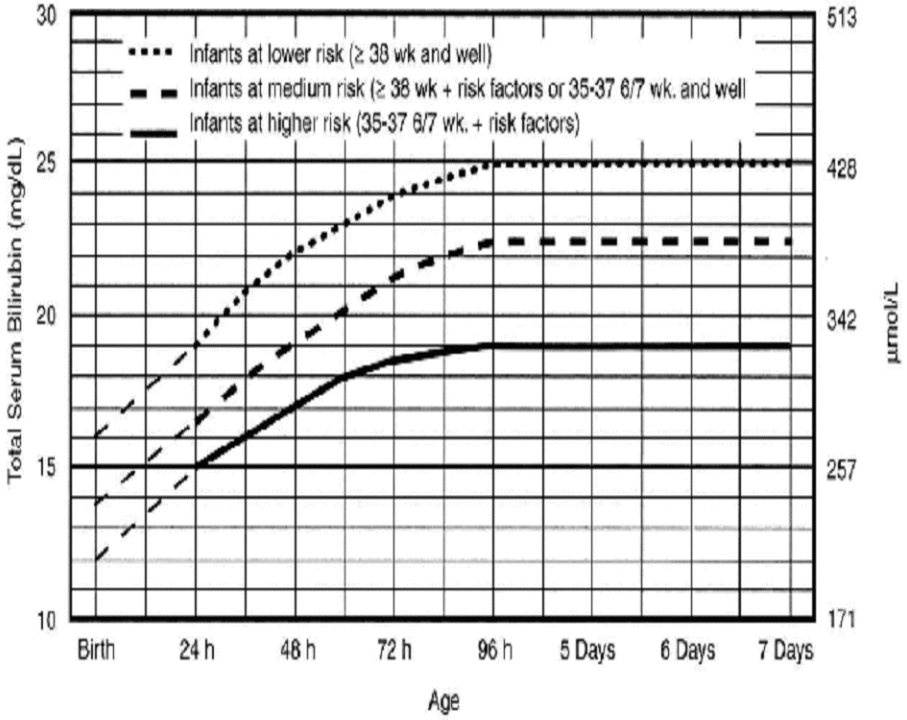
❖ Neonates with hyperbilirubinemia at a serum level requiring exchange transfusion

An exchange transfusion involves removing aliquots of patient blood and replacing with donor blood in order to remove abnormal blood components and circulating toxins whilst maintaining adequate circulating blood volume. It is primarily performed to remove antibodies and excess bilirubin in isoimmune disease, the incidence of exchange transfusion is decreasing secondary to the prevention, and improved prenatal management of alloimmune haemolytic disease and improvements in the management of neonatal hyperbilirubinemia.

A total serum bilirubin level at or above the exchange transfusion level should be considered a medical emergency and intensive phototherapy should be commenced immediately[19].

NOMOGRAM FOR EXCHANGE TRANSFUSION IN INFANTS' ≥35 WEEKS GESTATION.

AAP Subcommittee on Neonatal Hyperbilirubinaemia. Management of Hyperbilirubinaemia in the Infant ≥35 Gestation. Clinical Practice Guideline. *Pediatrics. 114(1):297-316, 2004 July.*



- The dashed lines for the first 24 hours indicate uncertainty due to a wide range of clinical circumstances and a range of responses to phototherapy.
- Immediate exchange transfusion is recommended if infant shows signs of acute bilirubin encephalopathy (hypertonia, arching, retrocollis, opisthotonos, fever, high pitched cry) or if TSB is ≥5 mg/dL (85 µmol/L) above these lines.
- Risk factors - isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis.
- Measure serum albumin and calculate B/A ratio (See legend)
- Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin
- If infant is well and 35-37 6/7 wk (median risk) can individualize TSB levels for exchange based on actual gestational age.

The following guidelines for exchange transfusion levels are based on the American Academy of Pediatric Guidelines and are adapted from the Department of Human Services (Victoria) Neonatal Handbook.

GUIDELINES FOR EXCHANGE TRANSFUSION IN INFANTS 35 OR MORE WEEKS OF GESTATION

Age (hrs)	**Infants at higher risk** 35-37^{+6} weeks + risk factors	**Infants at medium risk** ≥38 weeks + risk factors or 35-37^{+6} weeks and well	**Infants at lower risk** 38 weeks and well
	SBR (micromol/L)	**SBR (micromol/L)**	**SBR (micromol/L)**
Birth	200	235	270
12 hours	230	255	295
24 hours	255	280	320
48 hours	290	320	375
72 hours	315	360	405
96 hours	320	380	425
5 days	320	380	425
6 days	320	380	425
7 days	320	380	425

❖ Otoscopic examination

The otoscopic exam is performed by gently pulling the auricle upward and backward. In children, the auricle should be pulled downward and backward. This process will move the acoustic meatus in line with the canal. Hold the otoscope like a pen/pencil and use the little finger area as a fulcrum. This prevents injury if the patient turn suddenly.

- Inspect the external auditory canal.
- Evaluate tympanic membrane
    - Note the color (red, white, yellow) and translucency (transparent, opaque) and position (retracted, neutral or bulging) of the drum
    - Identify the pars tensa with its cone of light, the handle and short process of malleus, and the anterior and posterior folds of the pars flaccida and position of the malleus handle.

Since an intact tympanic membrane is necessary for conducting cochlear echoes, otoscopic examination is a must before OAE testing.

❖ **Tympanometry**

Tympanometry is an examination used to test the condition of the middle ear and mobility of the tympanic membrane and the conduction bones by creating variations of air pressure in the ear canal.

Tympanometry is an objective test of middle-ear function.

❖ **Otoacoustic emission**

An otoacoustic emission (OAE) is a low-level sound emitted by the cochlea either spontaneously or evoked by an auditory stimulus.

When sound stimulates the cochlea, the outer hair cells vibrate. The vibration produces a nearly inaudible sound that echoes back into

the middle ear. The sound can be measured with a small probe inserted into the ear canal.

There are 4 types of otoacoustic emissions

Spontaneous otoacoustic emissions (SOAEs) - Sounds emitted without an acoustic stimulus (i.e., spontaneously)

Transient otoacoustic emissions (TOAEs) or transient evoked otoacoustic emissions (TEOAEs) - Sounds emitted in response to an acoustic stimuli of very short duration; usually clicks but can be tone-bursts

Distortion product otoacoustic emissions (DPOAEs) - Sounds emitted in response to 2 simultaneous tones of different frequencies

Sustained-frequency otoacoustic emissions (SFOAEs) - Sounds emitted in response to a continuous tone

People with normal hearing produce emissions. Those with hearing loss greater than 25–30 decibels (dB) do not produce these very soft sounds.

This test can detect blockage in the outer ear canal, as well as the presence of middle ear fluid and damage to the outer hair cells in the cochlea.

- **Auditory brainstem response (ABR) / brainstem evoked response audiometry**

BERA is an objective way of eliciting brain stem potentials in response to audiological click stimuli. These waves are recorded by electrodes placed over the scalp. This investigation was first described by Jewett and Williston in 1971.

Even though BERA provides information regarding auditory function and sensitivity, it is not a substitute for other methods of audiological evaluation. It should be always viewed in conjunction with other audiological investigations.

Procedure: The stimulus either in the form of click or tone pip is transmitted to the ear via a transducer placed in the insert ear phone or head phone. The wave froms of impulses generated at the level of brain stem are recorded by the placement of electrodes over the scalp.

Electrode placement: Since the electrodes should be placed over the head, the hair must be oil free. The patient should be instructed to have shampoo bath before coming for investigation. The standard

electrode configuration for BERA involves placing a non inverting electrode over the vertex of the head, and inverting electrodes placed over the ear lobe or mastoid prominence. One more earthing electrode is placed over the forehead. This earthing electrode is important for proper functioning of preamplifier.

Since the potentials recorded are in far field, well displaced from the site of impulse generation, the wave forms recorded are very weak and they need to be amplified. This amplification is achieved by improving the signal : noise ratio.

To improve signal to noise ratio: Three parallel approaches are designed to achieve this goal.

Filtering: This is employed to reduce the recording bandwidth so that only the important components of the signal generated are recorded.

Repeated stimulation: This is done with synchronous time domain averaging to increase the amplitude of the components of the signal. In real time situations these two can be achieved by connecting the recording electrodes to a preamplifier, with appropriate filter settings.

Polarity alteration: By altering the polarity of impulses recorded, the artifacts are cancelled making the brain stem waves stand out.

In auditory brain stem evoked response audiometry, the impulses are generated by the brain stem. These impulses when recorded contain a series of peaks and troughs. The measured recording is a series of six to seven vertex positive peaks (vortex positive) are referred to by the Roman numerals I - VII.

Wave I originates from the dendrites of acoustic nerve fibers,

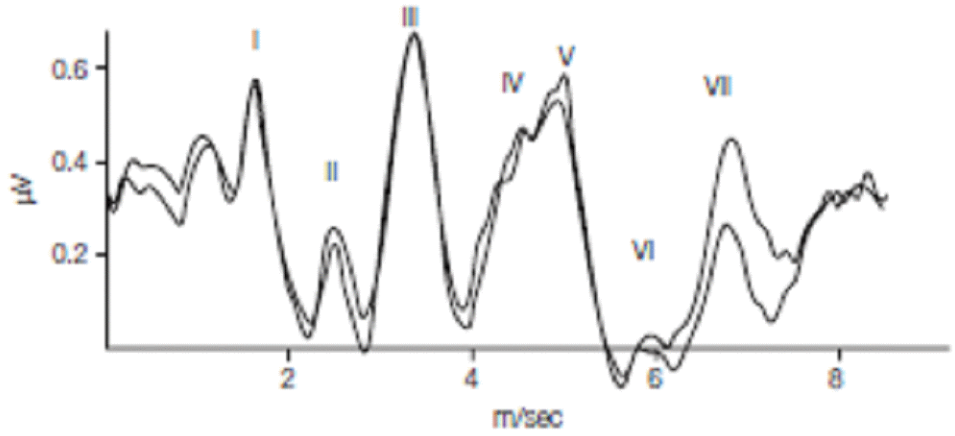
Wave II from the cochlear nucleus,

Wave III showing activity in superior olivary complex,

Wave IV-V associated with the lateral lemniscus

Wave IV and V – generated by the upper brainstem

Normal waveform obtained in BERA



STATISTICAL ANALYSIS

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using SPSS 16 and Sigma Stat 3.5 version.

Using this software range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated by One way ANOVA and Chi-square test was used to test the significance of difference between quantitative variables.

A 'p' value less than 0.05 is taken to denote significant relationship.

OBSERVATIONS AND RESULTS

A total of 110 cases comprising 39 males (35.5%) and 71 females (64.5%) were enrolled and studied.

CHART - 1

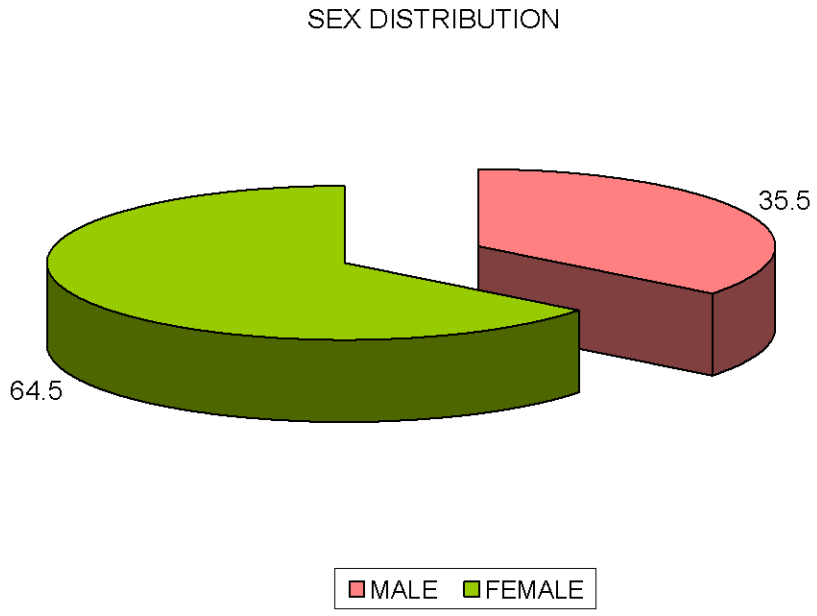
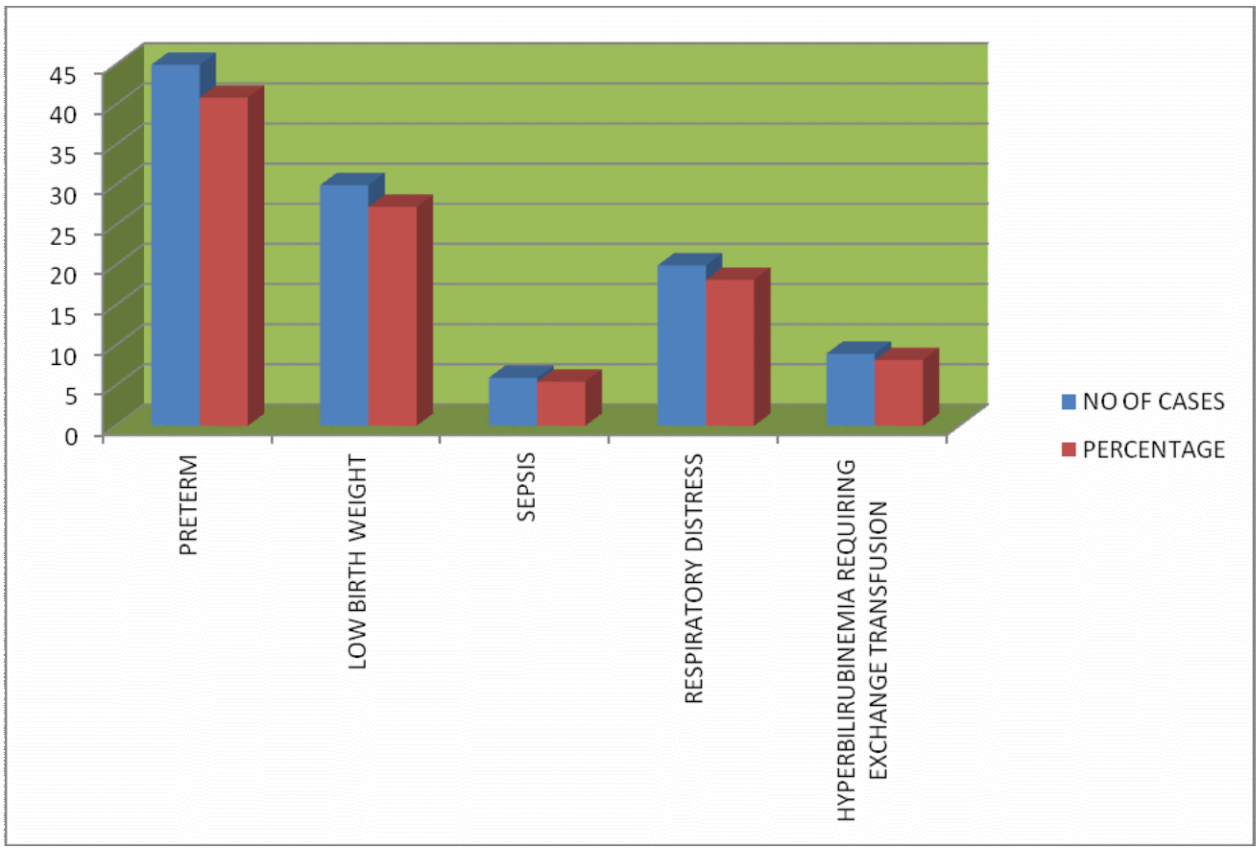


TABLE - 1

SEX	Percentage
MALE	39
FEMALE	71
TOTAL	100.0

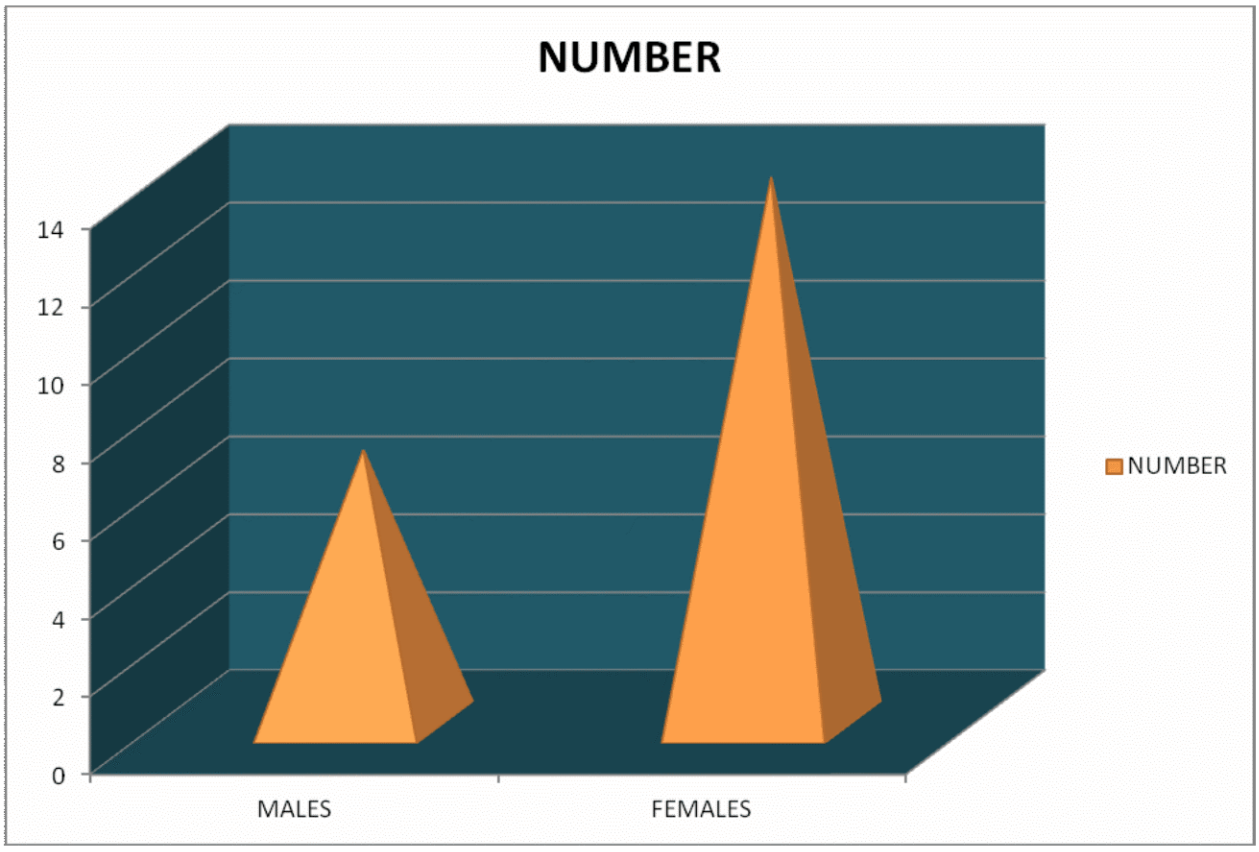
Out of 110 cases, 45 cases (40.9%) were preterm, 30 cases (27.3 %) were low birth weight, 20 cases (18.2%) were having perinatal asphyxia with Apgar score of less than 6 at fifth minute, 9 cases (8.2%) had hyperbilirubinemia requiring exchange transfusion, 6 cases (5.5%) had sepsis.

CHART - 2



Out of 110, 10 males and 16 females had hearing loss (initial OAE testing). A total of 21 neonates, 7 males and 14 females had hearing loss on follow up OAE and BERA. Hearing loss had no statistical relationship with gender (p=0.978).

CHART - 3



75 (68.2%) neonates in the age group of 11 to 20 days, 23 (20.9%) neonates in the age group of more than 20 days, 12 (10.9%) neonates in the age group of less than 10 days were tested for OAE.

CHART - 4

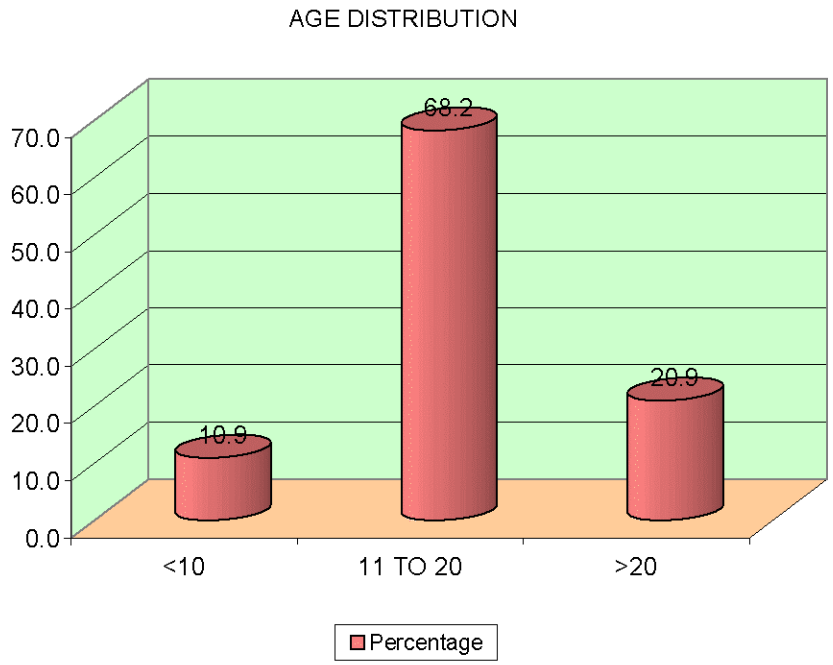
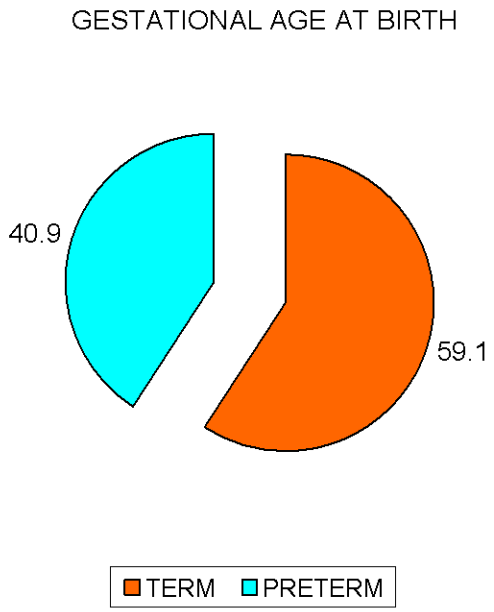


CHART - 5



45 out of 110 (59.1%) neonates were preterm and 65 out of 110 (40.9%) neonates were term.

30 neonates (27.3%) had birth weight less than 1500g, 80 neonates (72.7%) had normal birth weight.

CHART - 6

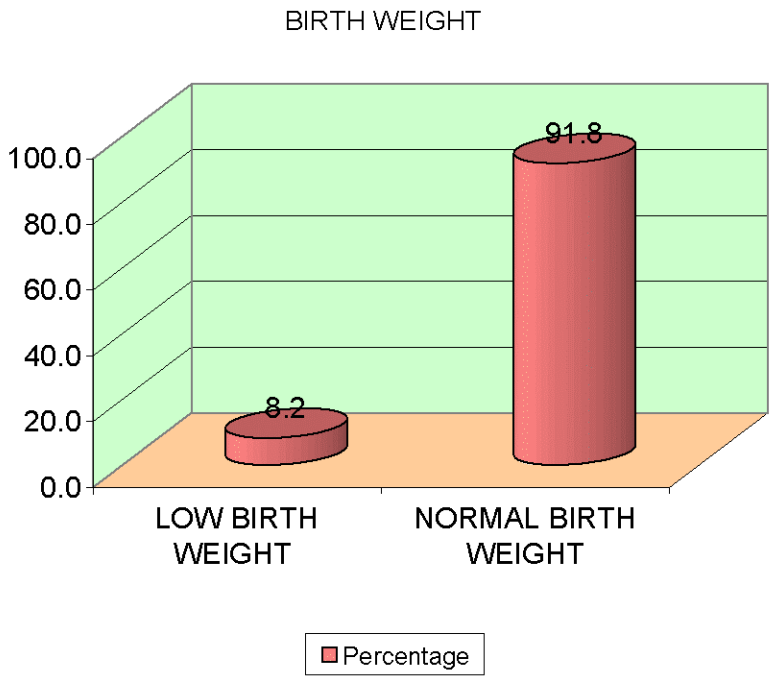


TABLE - 2

BIRTH WEIGHT	No.of cases
LOW BIRTH WEIGHT	30
NORMAL BIRTH WEIGHT	80
Total	110

20 neonates (18.2%) had history of perinatal asphyxia and 90 neonates had no history of perinatal asphyxia.

CHART - 7

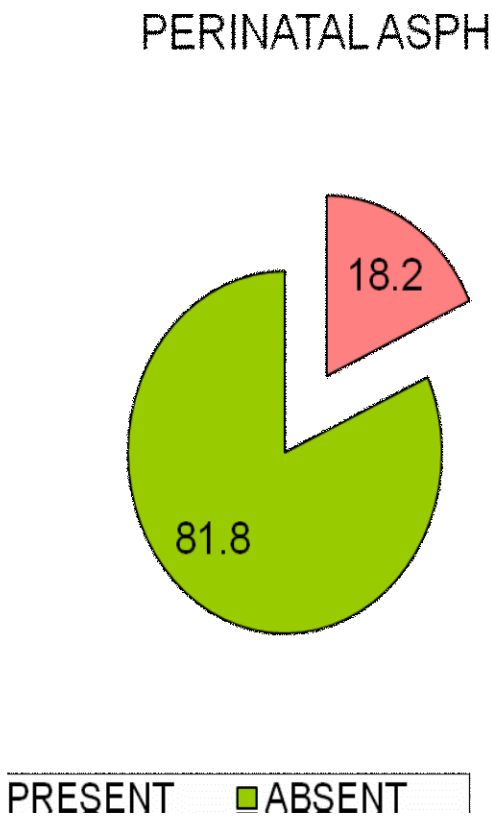


TABLE - 3

PERINATAL ASPHYXIA	No.of cases
PRESENT	20
ABSENT	90
TOTAL	110

9 neonates (8.2%) had received exchange transfusion for hyperbilirubinemia; 101 neonates had normal serum bilirubin levels and did not any transfusion.

CHART - 8

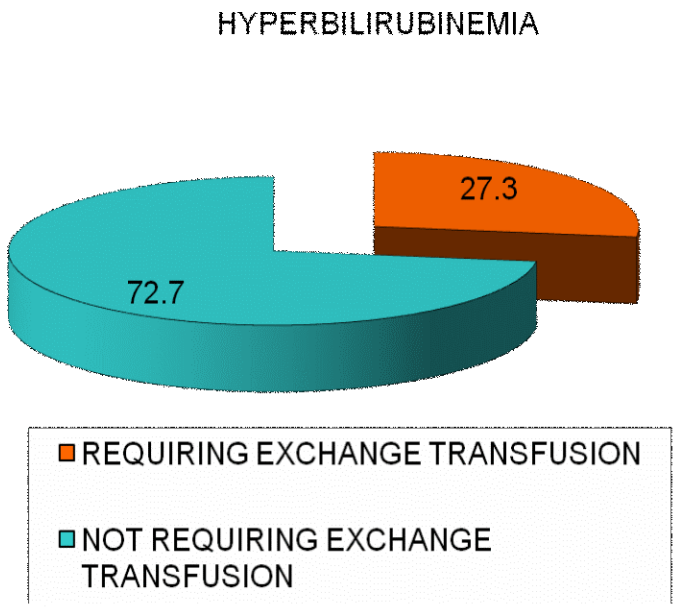
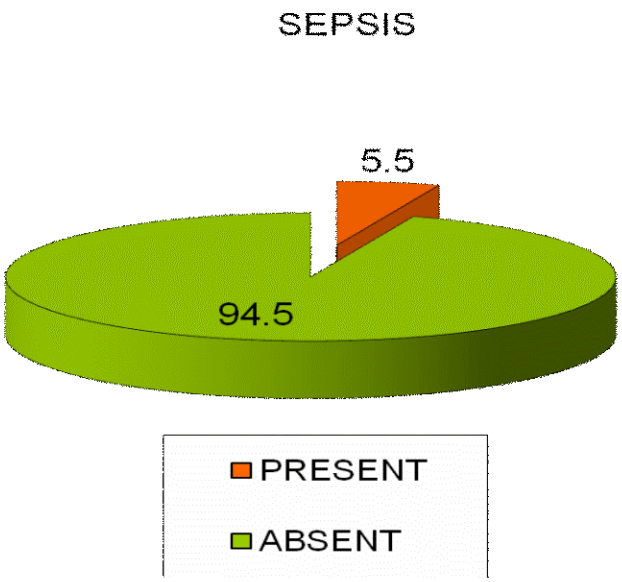


TABLE - 4

HYPERBILIRUBINEMIA	No.of cases
REQUIRING EXCHANGE TRANSFUSION	9
NOT REQUIRING EXCHANGE TRANSFUSION	101
Total	110

CHART - 9



6 neonates (5.5%) had sepsis and raised C reactive protein.

TABLE - 5

SEPSIS	No.of cases
PRESENT	6
ABSENT	104
TOTAL	110

Preterm, low birth weight, perinatal asphyxia were the major risk factors occurring in 40.9%, 27.3% and 18.2% at risk neonates respectively. None of the study neonates had family history of hearing loss.

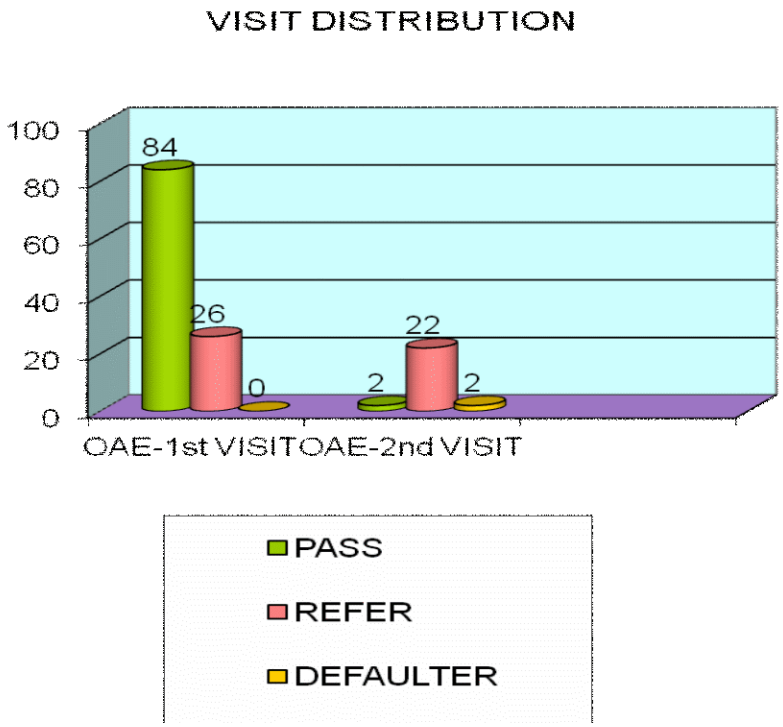
TABLE – 6

OAE RESPONSES

VISIT	OAE-1st VISIT	OAE-2nd VISIT
PASS	84	2
REFER	26	22
DEFAULTER	0	2

In the first visit, with 110 neonates and 220 test ears, OAE was tested. 84 neonates (76.36%) had both ears pass response, while 26 neonates (23.63%) had refer response. 2 neonates did not appear for follow up visit. 2nd OAE was tested after 4 weeks of initial testing. Out of 24 neonates who visited the second time, 22 had refer response and 2 neonates had pass response.

CHART - 10


VISIT DISTRIBUTION
100
80
60
40
20
0
84
26
0
2
22
2
OAE-1st VISIT
OAE-2nd VISIT
PASS
REFER
DEFAULTER

Out of 24 neonates with refer response in second visit, 7 were preterm neonates, 6 had prenatal asphyxia, 5 had received exchange transfusion for hyperilirubinemia, 2 had low birth weight and 2 had sepsis. The parents of neonates who had pass response in the first visit, were educated about late onset hearing impairment. BERA was done for neonates with refer response even in the second visit and hearing impairment was confirmed.

DISTRIBUTION OF CASES WITH REFER CRITERIA IN SECOND VISIT

CHART - 11

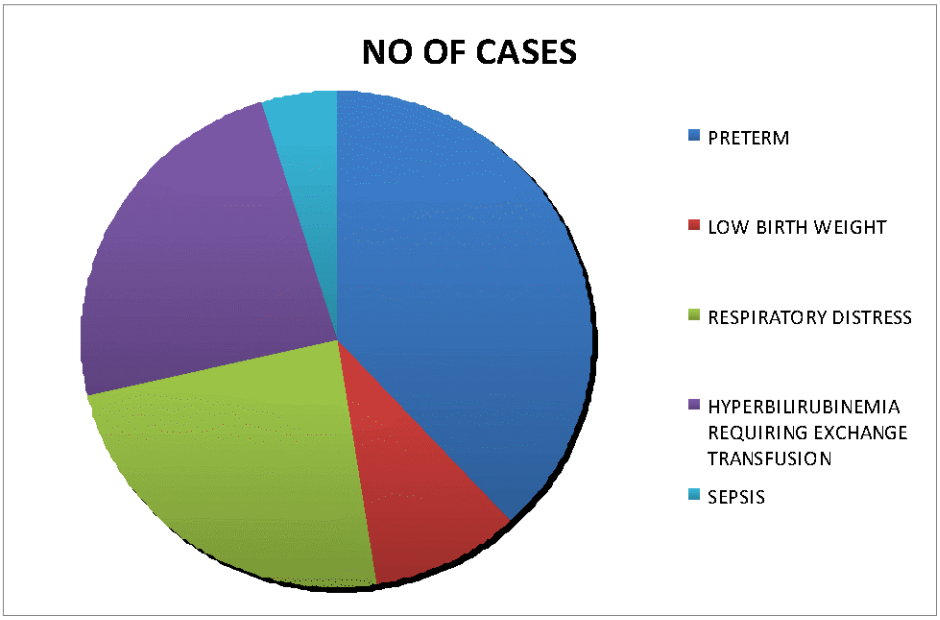


TABLE - 7

DISTRIBUTION OF CASES WITH REFER CRITERIA IN 2ND VISIT	NO OF CASES
PRETERM	8
LOW BIRTH WEIGHT	2
RESPIRATORY DISTRESS	5
HYPERBILIRUBINEMIA REQUIRING EXCHANGE TRANSFUSION	5
SEPSIS	1

In this study, prevalence of risk factors in neonates are preterm, low birth weight, perinatal asphyxia, hyperbilirubinemia requiring exchange transfusion and sepsis in decreasing order. The prevalence of hearing impairment was more in neonates with hyperbilirubinemia requiring exchange transfusion followed in order by perinatal asphyxia, preterm, low birth weight and sepsis.

TABLE - 8

DISTRIBUTION OF CASES	**NO OF CASES**	**PERCENTAGE**
PRETERM	45	40.9
LOW BIRTH WEIGHT	30	27.3
PERINATAL ASPHYXIA	20	18.2
HYPERBILIRUBINEMIA REQUIRING EXCHANGE TRANSFUSION	9	8.2
SEPSIS	6	5.5

CHART - 12

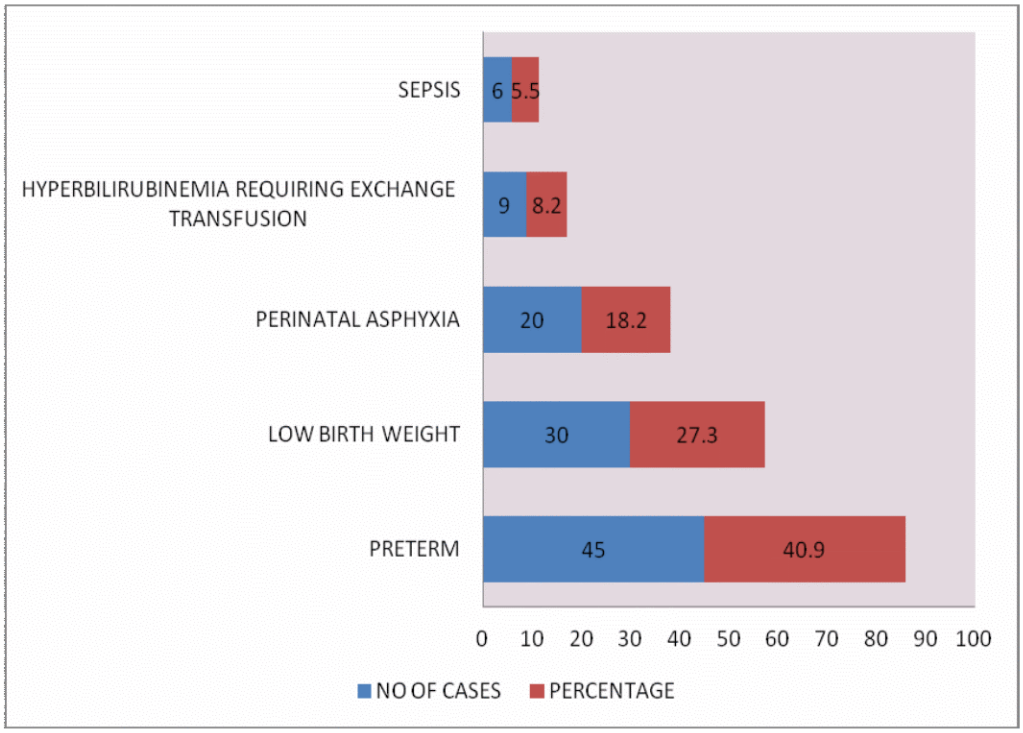
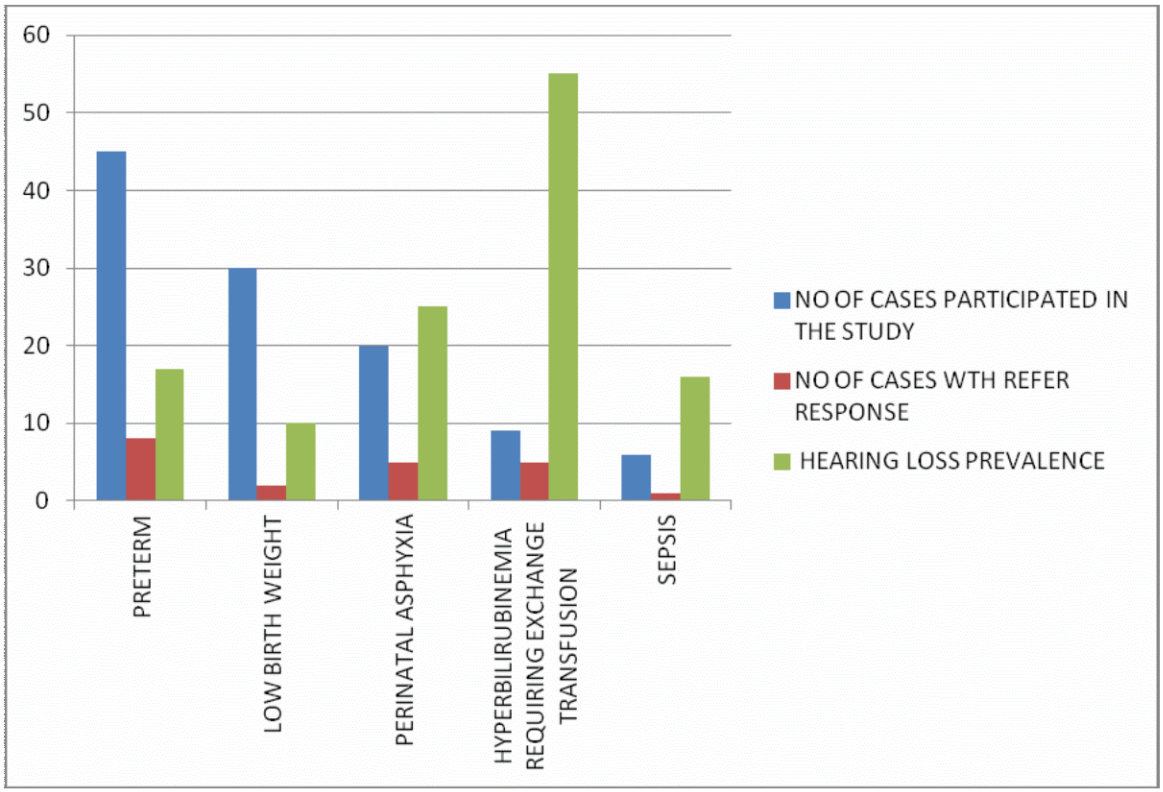

SEPSIS
6
5.5
HYPERBILIRUBINEMIA REQUIRING EXCHANGE TRANSFUSION
9
8.2
PERINATAL ASPHYXIA
20
18.2
LOW BIRTH WEIGHT
30
27.3
PRETERM
45
40.9
0 10 20 30 40 50 60 70 80 90 100
NO OF CASES
PERCENTAGE

TABLE – 9

HEARING LOSS PREVALENCE IN INDIVIDUAL RISK FACTORS

RISK FACTORS	NO OF CASES PARTICIPATED IN THE STUDY	NO OF CASES WTH REFER RESPONSE	HEARING LOSS PREVALENCE
PRETERM	45	8	17
LOW BIRTH WEIGHT	30	2	10
PERINATAL ASPHYXIA	20	5	25
HYPERBILIRUBINEMIA REQUIRING EXCHANGE TRANSFUSION	9	5	55
SEPSIS	6	1	16

CHART - 13



DISCUSSION

This study represents an initial attempt for implementing new-born hearing screening program in our hospital. In this study 110 at risk neonates were screened for hearing loss using OAE. 26 neonates tested abnormal in the initial screening procedure, which could be confirmed in 22 infants (20%) on follow-up. This implies a significant increase in hearing impairment in high risk neonates. Similar results have been obtained in the studies done by A Zamani et al., (8%) and Alwan M Maisoun et al., (13.5%) [20]. However, Christiane Meyer et al., found hearing impairment in 5.3% [21].The higher incidence in our study could be due to smaller sample size or because of severity of illness in our study population.

M AL-Harbi et al., found sepsis/meningitis and intraventricular haemorrhage as significant risk factors for hearing impairment [23]. Christiane Meyer et al., reported craniofacial anomalies, familial hearing disorders and bacterial meningitis as significant factors associated with pathologic BAER [21]. Similar findings were reported by AL-Harbi M et al., and KY Chan et al., [23,24].

In our study use of preterm, perinatal asphyxia and hyperbilirubinemia were the major risk factors in study neonates, which

is consistent with the study conducted by A Zamani et al., [20]. In the study by Christiane Meyer et al., ototoxic medication and birth weight <1500gm were the major risk factors[21]. The difference can be attributed to the relatively low survival rate of low birth infants in our set-up. Higher percentage of hyperbilirubinemia requiring exchange transfusion in our study is due to poor follow-up of neonates with blood group incompatibilities.

In our study, two neonates had initial refer response turned into pass response on follow up studies. This transient abnormality can be due to the presence of middle ear effusion seen in ventilated babies. Similar findings have been reported by Hulya Bilgen et al. [22].

In our study 5 out of 9 neonates with hyperbilirubinemia requiring exchange transfusion had refer in 2nd OAE and abnormal BERA which is supported by the study conducted by A Zamani et al.,[20], hyperbilirubinemia was the main cause of hearing loss. The transient BAER abnormalities in infants with hyperbilirubinemia has been earlier reported by VK Agrawal et al., [25].

Infant hearing screening was started in the USA more than 30 year's ago by (Downs and Sterritt, 1964) using behavioral audiometric 'arousal' technique. High rates of false positives and false negatives were detected, according to the Joint Committee On Infant Hearing, and recommended the alternative use of audiometry tests for infants with high-risk criteria. Low sensitivity and specificity in conventional screening procedures such as the arousal technique apparatus render the technique suitable for screening only and not for diagnostic procedures. In 1988 Screening for hearing impairment in infancy in most districts in the United Kingdom was done with infant distraction test (IDT) at 7 to 8 months of age, a targeted high risk babies Johnson et al., (1990) reported the distraction test was sensitive (91%) but non-specific (82%) in the high-risk population. The effectiveness of the screening program was limited. Recently, the use of TEOAEs together with ABR was shown to be reliable and high sensitivity and specificity in universal hearing screening programs. The two techniques (TEOAEs and ABR) showing maximal promise as universal screening tools for the newborn, each has its unique advantages and disadvantages (Geert De Ceulaer et al., 1999).

LIMITATIONS OF THE STUDY

Small sample size is one of the limitations in our study. Further studies are needed with larger sample size to more accurately highlight the importance of hearing assessment in high risk newborn babies. Furthermore, other risk factors like duration of NICU stay, meningitis, malformations, ototoxic drug administration could not be evaluated.

CONCLUSION

To conclude, prevalence of hearing impairment in at risk neonates are preterm neonates (40.3%), low birth weight (27.3%), perinatal asphyxia (18.2%), hyperbilirubinemia requiring exchange transfusion (8.2%), sepsis (5.5%). Hence emphasizes the importance of screening for hearing impairment in such high risk newborns. The prevalence of hearing impairment was more in neonates with hyperbilirubinemia requiring exchange transfusion than other risk factors. The study highlights that although universal hearing screening programs are warranted; most newborns with a detected hearing loss can be identified based on the risk factors. Thus, a targeted approach for hearing screening may be more feasible in resource limited settings.

PROFORMA

Name : Age in days: Sex

IP No. Address:

DOA: DOE:

H/o Present illness:

H/o Ear discharge

H/o Response to sound

H/o Nasal discharge

H/o incessant cry

H/o yellowish discolouration of body/ eyes/ palms/ soles

H/o fever

H/o respiratory distress

H/o Low birth weight

H/o phototherapy/ exchange transfusion

H/o Neonatal Intensive care unit stay

H/o Seizures

H/o bleeding episodes

NATAL HISTORY

Full term/ preterm

Mode of delivery: Vaginal / Vacuum assisted/ Forceps/ Cesarean

H/o birth trauma

H/o respiratory distress/ seizure at birth

H/o neonatal jaundice

H/o bleeding diathesis

TREATMENT HISTORY

NICU/ neonatal well baby clinic admission:

Mode of treatment:

Duration of treatment:

PERSONAL HISTORY

Sleep and appetite:

Bowel and Bladder habits:

Feeding habits:

FAMILY HISTORY

H/o congenital deafness in family:

GENERAL EXAMINATION

Patients active, alert, afebrile

Anemic +/-

Cyanosis +/-

Icterus +/-

Lymphadenopathy +/-

Edema +/-

RS - NVBS, added sounds

CVS – S1 S2

EXAMINATION OF EAR	RIGHT	LEFT
Preauricular area		
Pinna		
External Auditory Canal		
Tympanic membrane		
Post auricular area		
Facial nerve		

Gestational age at birth

Birth weight

APGAR Score at 5'

Total Bilirubin

Direct Bilirubin

Indirect Bilirubin

C reactive protein

	RIGHT EAR	LEFT EAR
Transient evoked otoacoustic emission		
1ST visit :		
2nd visit :		
Brainstem Evoked Auditory Response		

ABBREVIATIONS

OAE	: OTOACOUSTIC EMISSIONS
TEOAE	: TRANSIENT EVOKED OTOACOUSTIC EMISSIONS
SOAE	: SPONTANEOUS OTOACOUSTIC EMISSIONS
DPOAE	: DISTORTON PRODUCT OTOACOUSTIC EMISSIONS
BERA	: BRAINSTEM EVOKED RESPONSE AUDIOMETRY
AN	: AUDITORY NEUROPATHY
EAC	: EXTERNAL AUDITORY CANAL
NICU	: NEONATAL INTENSIVE CARE UNIT
SNHL	: SENSORINEURAL HEARING LOSS
CM	: COCHLEAR MICROPHONICS

MASTER CHART

SL NO.	NAME	AGE	SEX	GESTATIONAL AGE AT BIRTH	BIRTH WEIGHT	PERINATAL ASPHYXIA	HYPERBILIRUBINEMIA	SEPSIS	OAE-1st VISIT	OAE-2nd VISIT	BERA
1	B/o shobanadevi	20	F	Preterm	2	2	2	2	REFER	PASS	
2	B/o Selvapandiyammal	8	M	Term	1	2	2	2	PASS		
3	suriyapandi	25	M	Preterm	2	2	2	2	REFER	DEFAULTER	
4	B/o valliswari	9	F	Term	2	1	2	2	PASS		
5	B/o kaleeswari	14	F	Term	1	2	2	2	PASS		
6	B/o Anandi	5	M	Preterm	2	2	2	2	REFER	PASS	NORMAL
7	B/o valarmadi	18	F	Term	1	2	2	2	PASS		
8	B/o sindhya	26	M	Preterm	2	2	2	2	PASS		
9	B/o krishnaveni	20	F	Term	1	2	2	2	PASS		
10	B/o sathya	14	F	Term	2	2	1	2	PASS		
11	B/o guruchandrika	16	M	Term	2	1	2	2	REFER	REFER	ABNORMAL
12	B/o chithra	12	F	Term	1	2	2	2	PASS		
13	B/o mariammal	28	M	Preterm	2	2	2	2	REFER	REFER	ABNORMAL
14	B/o saranya	11	M	Term	2	1	2	2	PASS		
15	B/o muneeswari	14	F	Term	1	2	2	2	REFER	REFER	ABNORMAL
16	B/o prema	21	M	Preterm	2	2	2	2	PASS		
17	B/o muthumari	22	F	Preterm	2	2	2	2	PASS		
18	sabari	12	M	Term	1	2	2	2	REFER	REFER	ABNORMAL
19	B/o thotturmalliga	14	F	Term	2	2	1	2	PASS		
20	B/o chithra	15	M	Term	2	1	2	2	REFER	REFER	ABNORMAL
21	B/o panchavarnam	17	F	Preterm	2	2	2	2	PASS		
22	B/o prema	17	F	Term	1	2	2	2	PASS		
23	B/o prema	17	F	Term	2	1	2	2	PASS		
24	B/o subiammal	21	F	Term	2	2	2	1	PASS		
25	B/o kousaliya	25	M	Preterm	2	2	2	2	REFER	REFER	ABNORMAL

26	B/o reena	22	F	Term	1	2	2	2	PASS		
27	B/o jeyalakshmi	20	M	Term	2	2	1	2	REFER	REFER	ABNORMAL
28	B/o angaleswari	19	F	Preterm	2	2	2	2	PASS		
29	B/o rajasree	15	F	Term	1	2	2	2	PASS		
30	B/o veeralakshmi	14	M	Preterm	2	2	2	2	PASS		
31	B/o Anneswari	19	M	Term	2	2	2	1	PASS		
32	B/o Eswari	19	F	Preterm	2	2	2	2	REFER	REFER	ABNORMAL
33	B/o sharmila	25	F	Preterm	2	2	2	2	PASS		
34	B/o malarkodi	9	F	Term	2	1	2	2	REFER	REFER	ABNORMAL
35	B/o parameswari	14	F	Term	1	2	2	2	PASS		
36	B/o mehraj	5	M	Term	1	2	2	2	PASS		
37	B/o deepa	18	F	Preterm	2	2	2	2	REFER	REFER	ABNORMAL
38	B/o abirami	26	F	Term	2	2	2	1	PASS		
39	B/o durgadevi	20	F	Term	1	2	2	2	PASS		
40	B/o jayalakshmi	20	M	Preterm	2	2	2	2	PASS		
41	B/o kaviyammal	14	F	Term	2	2	1	2	PASS		
42	B/o krithika	16	F	Term	1	2	2	2	PASS		
43	B/o lakshmi	12	M	Preterm	2	2	2	2	PASS		
44	B/o karpagasri	18	M	Term	1	2	2	2	PASS		
45	B/o jayagowri	26	F	Term	2	2	2	1	REFER	REFER	ABNORMAL
46	B/o jayalakshmi	22	F	Preterm	2	2	2	2	REFER	REFER	ABNORMAL
47	B/o jayalakshmi	21	F	Term	1	2	2	2	PASS		
48	B/o shobana	18	F	Term	2	1	2	2	PASS		
49	B/o vanitha	27	M	Preterm	2	2	2	2	REFER	REFER	ABNORMAL
50	B/o nagalakshmi	14	F	Preterm	2	2	2	2	PASS		
51	B/o abhinaya	15	F	Term	1	2	2	2	REFER	REFER	ABNORMAL
52	B/o sivagami	17	M	Preterm	2	2	2	2	PASS		
53	B/o pavithra	18	F	Term	2	1	2	2	PASS		
54	B/o veeralakshmi	26	F	Preterm	2	2	2	2	PASS		
55	B/o kunthigamselvi	20	F	Preterm	2	2	2	2	PASS		
56	B/o muneeswari	14	M	Term	1	2	2	2	PASS		
57	B/o murugeswari	16	F	Term	2	1	2	2	PASS		
58	B/o rema	12	M	Term	1	2	2	2	PASS		
59	B/o sivaranjini	20	M	Preterm	2	2	2	2	REFER	DEFAULTER	

60	B/o punitha	19	M	Preterm	2	2	2	2	PASS		
61	B/o jasmin	15	F	Term	2	1	2	2	PASS		
62	B/o pandiammal	14	F	Preterm	2	2	2	2	PASS		
63	B/o hemalatha	19	F	Term	2	1	2	2	PASS		
64	B/o suryaprabha	19	M	Term	1	2	2	2	PASS		
65	B/o ganesa	25	F	Preterm	2	2	2	2	PASS		
66	B/o podhumani	18	F	Term	2	2	1	2	REFER	REFER	ABNORMAL
67	B/o veeralakshmi	26	M	Term	1	2	2	2	PASS		
68	B/o durgai	20	F	Preterm	2	2	2	2	PASS		
69	B/o murugeswari	14	F	Preterm	2	2	2	2	PASS		
70	B/o alagi	17	F	Term	1	2	2	2	REFER	REFER	ABNORMAL
71	gurumurthi	14	M	Preterm	2	2	2	2	PASS		
72	B/o geethu	5	F	Term	2	1	2	2	PASS		
73	murugan	18	M	Term	2	2	2	2	PASS		
74	B/o kuppammal	26	F	Preterm	1	2	2	2	REFER	REFER	ABNORMAL
75	vasanth	20	M	Preterm	2	2	2	2	PASS		
76	B/o maniswari	14	F	Term	2	1	2	2	PASS		
77	B/o veditha	17	F	Term	1	2	2	2	PASS		
78	B/o jothi	20	F	Preterm	2	2	2	2	PASS		
79	podumponnu	14	F	Term	2	1	2	2	PASS		
80	B/o chinnadakki	17	M	Preterm	2	2	2	2	PASS		
81	B/o muthuselvi	14	M	Preterm	2	2	2	2	PASS		
82	B/o pandiswari	5	F	Term	2	2	1	2	PASS		
83	B/o nirmala	8	F	Term	2	1	2	2	PASS		
84	B/o revathi	25	F	Preterm	2	2	2	2	REFER	REFER	ABNORMAL
85	B/o shanmughapriya	13	F	Term	1	2	2	2	PASS		
86	B/o indumathi	14	M	Term	2	1	2	2	PASS		
87	B/o sudhu	15	F	Preterm	2	2	2	2	PASS		
88	B/o priyadarshini	17	F	Term	2	2	1	2	PASS		
89	B/o annalakshmi	18	F	Term	1	2	2	2	PASS		
90	B/o perumayi	26	M	Preterm	2	2	2	2	PASS		
91	B/o aadhilakshmi	20	F	Term	2	1	2	2	REFER	REFER	NORMAL
92	B/o kousaliya	14	F	Preterm	2	2	2	2	PASS		
93	B/o chandra	17	M	Term	2	2	2	1	PASS		

94	B/o gowri	25	F	Term	1	2	2	2	PASS		
95	muthamilselvi	9	F	Preterm	2	2	2	2	PASS		
96	B/o azhaguponnu	14	F	Term	1	2	2	2	PASS		
97	pradeep	15	M	Term	2	2	1	2	PASS		
98	B/o jancy	16	F	Preterm	2	2	2	2	PASS		
99	B/o angel	9	F	Term	2	1	2	2	REFER	REFER	ABNORMAL
100	pandiswaran	14	M	Term	2	1	2	2	PASS		
101	B/o renuka	5	F	Term	1	2	2	2	PASS		
102	B/o podumponnu	18	F	Preterm	2	2	2	2	PASS		
103	B/o syed ali fathima	26	F	Term	2	2	2	1	PASS		
104	B/o dhivya	20	M	Preterm	2	2	2	2	PASS		
105	B/o revathi	20	F	Term	2	2	1	2	PASS		
106	B/o latha	14	M	Preterm	2	2	2	2	PASS		
107	B/o sneha	9	F	Term	2	1	2	2	REFER	REFER	ABNORMAL
108	B/o karupayi	14	F	Term	1	2	2	2	REFER	REFER	ABNORMAL
109	B/o meenakshi	15	F	Preterm	2	2	2	2	PASS		
110	vishnu	27	M	Preterm	2	2	2	2	PASS		

HYPERBILIRUBINEMIA

REQUIRING EXCHANGE TRANSFUSION : 1

NOT REQUIRING EXCHANGE TRANSFUSION : 2

PERINATAL ASPHYXIA

PRESENT: 1

ABSENT : 2

SEPSIS

PRESENT: 1

ABSENT : 2

GESTATIONAL AGE AT BIRTH

<37 WEEKS : PRETERM

>37 WEEKS : TERM

BIRTH WEIGHT

LOW BIRTH WEIGHT : 1

NORMAL BIRTH WEIGHT : 2

MADURAI MEDICAL COLLEGE
MADURAI, TAMILNADU, INDIA -625 020
(Affiliated to The Tamilnadu Dr.MGR Medical University,
Chennai, Tamil Nadu)

ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.Saranya S.
Course	:	PG in MS., Otorhinolaryngology
Period of Study	:	**2015-2018**
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	A study on screening for hearing impairment in at risk neonates with transient evoked otoacoustic emissions
Ethical Committee as on	:	21.04.2017

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.,(Neuro), Dsc (H
CHAIRMAN
IEC - Madurai Medical College
Madurai

DEAN
Madurai Medical College
Madurai-20

URKUND

Urkund Analysis Result

Analysed Document: Dr SARANYA.docx (D31303290)
Submitted: 10/13/2017 6:58:00 PM
Submitted By: ssaranyasavi@gmail.com
Significance: 3 %

Sources included in the report:

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9