NEWBORN SCREENING FOR HEARING LOSS USING TRANSIENT EVOKED OTOACOUSTIC EMISSIONS IN KENYATTA NATIONAL HOSPITAL.

A dissertation submitted in partial fulfillment of the requirements for the degree of M.Med (Ear Nose Throat Head and Neck Surgery) University of Nairobi 2004.

By

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DECLARATION

I declare that this is my original work and it has not been presented for a degree in any other university.

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This thesis has been submitted for examination with my approval.

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AKNOWLEDGEMENTS

First I would like to thank Impact East Africa for providing the TEOAE screening equipment used in this study, without which there would have been no study.

Thanks also go to Prof. Macharia for being my lecturer and providing the idea of newborn screening and also for supervising this study.

I would also wish to thank Prof. Oburra and Dr. Mugwe for their kind comments and suggestions.

I would also like to thank all those Consultants in the ENT department in Kenyatta National hospital whose input I could not have done without.

To the staff the Newborn Unit in Kenyatta National Hospital, I thank you for all the cooperation I received.

DEDICATION

This book is dedicated to my Parents Mr. Fredrick Muhoho Njoroge and Mrs. Jane Njoki Muhoho for all their love, sacrifice and devotion that has gone into my Education.

CONTENTS

ABBREVIATIONS

ABLB	Alternate Binaural Loudness Balance
ABR	Auditory Brainstem Response
CHL	Conductive Hearing Loss
CMV	Cytomegalovirus
CN	Cranial Nerve
DPOAE	Distortion Product Otoacoustic Emissions
FDA	Federal Drug Authority
HLDB	Hearing loss decibels
HZ	Hertz
ILO	Institute of Laryngology and Otology
KNH	Kenyatta National Hospital
NBU	Newborn Unit
OAE	Otoacoustic Acoustic Emissions
PTA	Pure Tone Audiometry
RMS	Root Mean Square
SFOAE	Sustained Frequency Otoacoustic Emissions
SISI	Short increment Sensitivity Index
SNHL	Sensorineural Hearing Loss
SOAE	Spontaneous Otoacoustic Emissions
SPL	Sound Pressure Level
TEOAE	Transient Evoked Otoacoustic emissions
UNHS	Universal Newborn Hearing Screening

ABSTRACT

INTRODUCTION

Hearing loss is amongst the commonest congenital abnormality occurring in one to three newborns per thousand and has a profound effect on the development of language and cognition in the affected child. The critical age for intervention has been identified as six months after which the child is disadvantaged in developing satisfactory communicative skills. Early detection of this problem has been difficult, although various screening test have been developed. Transient Evoked Otoacoustic emissions (TEOAEs), the test to be used in this study has been found to be a quick, objective, non-invasive, accurate and easy test to perform.

BROAD OBJECTIVE:

Was to identify newborns with hearing loss and document results of hearing screening at KNH using TEOAE

SPECIFIC OBJECTIVES

1. To determine the usefulness of TEOAE as a screening tool
2. To correlate results of hearing screening with TEOAE and presence of risk factors in newborns in KNH.
3. To determine the feasibility of a UNHS at the KNH

DESIGN OF STUDY

A cross sectional descriptive study.

METHODOLOGY

This was based on National UK Newborn Hearing Screening Pilot recommended test protocol, 26 November 2001.

RESULTS

291 Newborns were tested for hearing loss using TEOAE. 11 failed the screening. Risk factors for hearing loss were identified as low birth weight, prematurity, birth asphyxia, craniofacial anomalies, and bacterial meningitis. Ototoxic drugs were given universally making analysis impractical. Prolonged intubation newborns could not be tested due to technical limitations

CONCLUSIONS

Birth asphyxia as measured by a low APGAR scores is the most common risk factor to failure of TEOAE in our environment. Low birth weight and gestational immaturity are also very significant. Ototoxic medications should be used with more rationale.

LITERATURE REVIEW

INTRODUCTION.

Hearing is probably the single most important factor in the development of speech, language and cognitive ability in the human being. The reduction in acoustic input feedback for congenitally or prelingually hearing impaired child, places him/her at a considerable disadvantage for acquiring spoken language. (1)The age of detection appears to be important in determining outcome for those children suffering from profound hearing loss. (2)

Early detection of hearing impairment may lead to early intervention in the form of acoustic amplification, where applicable and exposure to educational programmes encouraging the use of residual hearing and development of language.(1) It has been implied that many of the psychological and social detrimental and debilitating effects of "deafness" can be alleviated if children are taught to utilize any residual hearing or even vibrotactile sensitivity before critical period of language acquisition thought to be in the first six months of life.(2, 3, 4)

In 1991 Ramkalawan, T.W. and Davis A.C showed that even children with milder hearing losses might suffer

detrimental effects in their development of spoken language if intervention is long delayed. This has been postulated by some of the few studies that investigated milder and fluctuating conductive losses on speech and language. (6)

Advances in acoustic and electrophysiological methods of screening for hearing loss mean congenital hearing impairment is potentially detectable from 24 hours of age. If is therefore feasible that a larger proportion of those children with mild to moderate losses could be detected at a very early age, along with the more severe losses. (5, 6, 7)

HISTORY

Audiology as a clinical entity developed in the 20th century.

Equipment for measuring the thresholds of tonal signals was introduced commercially in the 1920s but Pure Tone Audiometry came into clinical practice in the 1940's.8)

Speech Audiometry became a routine clinical application in the 1950s; it was developed in the Psychoacoustics Laboratory in Harvard and by Raymond Carhart at Northwestern University (9)

Diagnostic Site of Lesion Audiometry was the rave in the 1960s and included Tone Decay Tests, Bakesy audiometry, the Alternate Binaural Loudness Balance (ABLB) and Short Increment Sensitive Index (SISI) procedure within a battery for differential diagnosis of SNHL, site of lesion determination and the identification of retrocochlear auditory dysfunction. (10) Immitance, then termed aural impedance measurement was based on a paper published by Jerger, in 1970. This led to the recording of tympanometry and acoustic reflexes.11) Auditory brainstem response (ABR) was discovered in the early 1970's but really became incorporated into clinical practice in the 1980's. Otoacoustic emissions (OAE's) were discovered by David T. Kemp in 1978, but only came into clinical use in the 1990's, with commercially and FDA approved devices for measuring OAE's.

In the United States the average age at which hearing loss is identified is 2 years 6 month, this is way past the critical age of 6 months. In *Healthy People 2010*, a US health statement, the goal is to screen all newborns for hearing loss and provide intervention by 6 months of age. (12, 13, 14)

At least five criteria must be met so as to justify universal screening as proposed by the American Academy of pediatrics task force on newborn and infant hearing.

1. *An easy to use test that possesses a high* degree of sensitivity and specificity.
2. The condition being screened for is otherwise not detectable by clinical parameters.
3. Interventions are available to correct the conditions detected by screening.
4. Early screening detection and intervention result in improved out come.
5. The screening program is documented to be in an acceptable cost effective range.

(5,15,16,17)

TEOAE's can not be elicited in persons with hearing thresholds over 30 dBHL.

Behavioral responses (Infant distraction test) have been used in the past and involve measurements, head movements and general body movement to sound, but require very high sound levels and are more reliable only after 8 months of age.

Hearing loss screening using TEOAE has a number of advantages which include:

- It is a very simple test
- It is a very quick test and provides efficiency of time
- It is an objective test
- It is a non-invasive test
- It is accurate and reproducible.
- It is cheap (18)

The main disadvantages of using ABR over TEOAEs for screening are

- Increased test time - studies have suggested test times in the order of 21 minutes for ABR compared to 12 minutes for TEOAE
- That ABR is relatively more invasive than TEOAE and this leads to parent acceptability issues
- Increase in cost due to consumables such as disposable electrodes and increased test time compared to TEOAE
- ABR requires an audiologist perform the screening for analysis of the waveform where as in TEOAE the analysis is automated. (27)
- ABR only detects high frequency hearing loss

OTOACOUSTIC EMISSIONS

Otoacoustic emissions are sounds which can be recorded in the ear canals of normal functioning ears that are generated in the cochlear. (5, 20) These can be broadly classified into spontaneous or evoked, the latter being in response to stimuli presented to the ear. (21)

To understand Otoacoustic emission (OAE) one has to understand the physiology of hearing.

PHYSIOLOGY OF HEARING.

There are 4 regions of the auditory system that either contribute to the generation of OAEs or can influence OAE recording. These are the external ear canal, the middle ear system, the cochlear, and the efferent auditory system. (Figure 1)

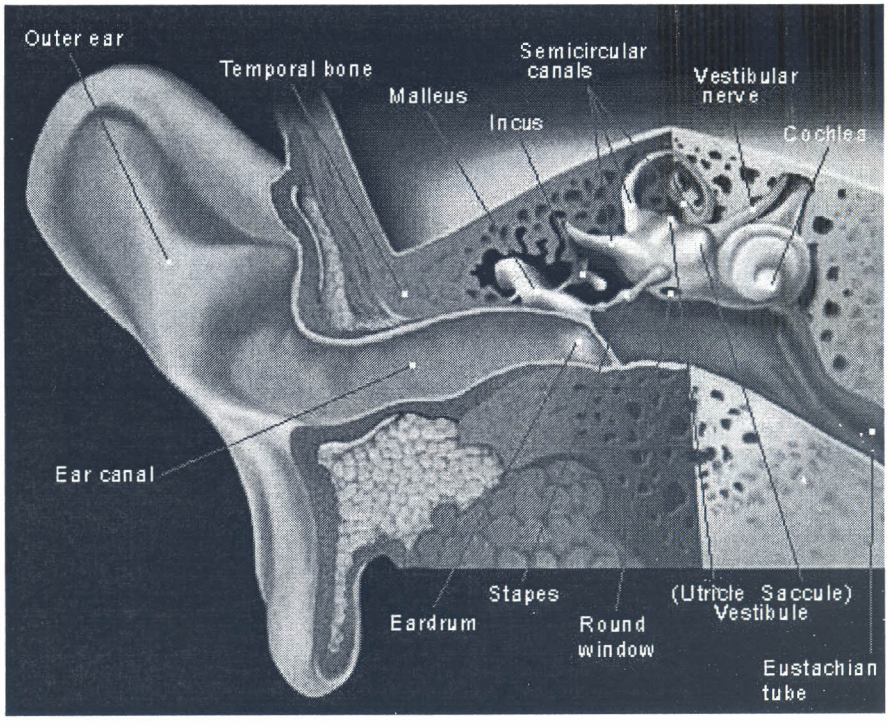


Figure 1: *A cutaway view of the Ear.*

EXTERNAL EAR

In almost all-audiologic measurement the stimulus is presented to the patient via the external auditory canal either directly via earphone or remotely via loudspeaker. The exception to this is in bone conduction studies.

The external canal is therefore a conduit for acoustic stimuli i.e. tones, speech or clicks to the inner ear. The response is then usually recorded proximal to the ear canal e.g. Middle ear immitance measures, Inner ear - EcochG, Eighth cranial nerve and brain stem ABR or more rostral auditory regions, including the highest levels of auditory cortex or speech audiometry. However in OAEs the stimulus, a click or tone, is presented to the ear canal and the response, also a sound, which is referred to as OAE, is recorded in the ear canal.

It is important to rule out or document pathology of the external ear canal prior to OAE measurement, but non pathological conditions involving the canal e.g. vernix casseosa, foreign bodies and debris must be taken into account whenever OAE findings are not entirely normal.

Therefore, close otoscopic inspection of the external ear canal is of utmost importance prior to OAE measurement.

MIDDLE EAR.

When sound waves strike the tympanic membrane, the waves cause it to vibrate, setting off a chain of vibrations along the 3 ossicles (in order, malleus, incus, and stapes) to the membrane of the oval window at the entrance to the cochlea. (Figure 2). This process amplifies the environmental sound by approximately 18 fold. The impedance matching provided by the middle ear effectively reduces impedance mismatch between the environment and the inner ear there by enhancing hearing sensitivity.

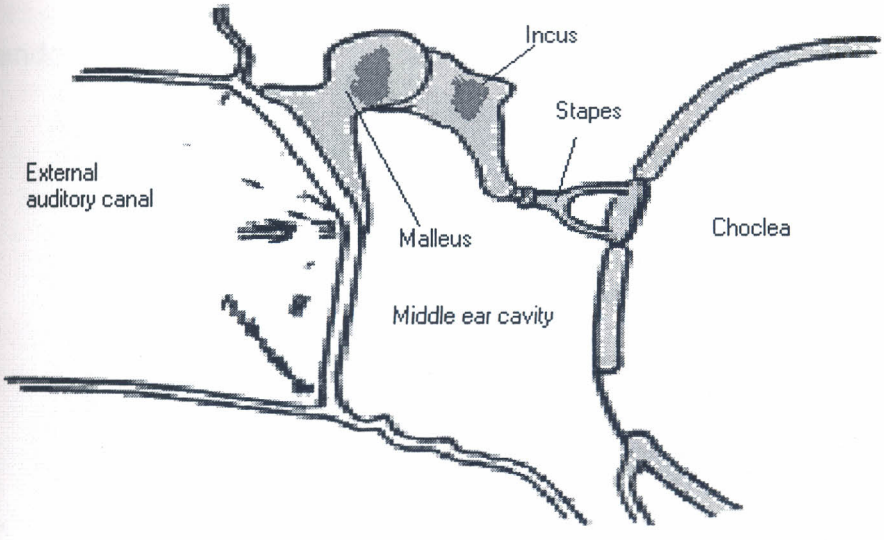


Figure 2: *The middle ear ossicles and their relation to the tympanic membrane and inner ear.*

However, in OAE measurement energy generated within the cochlear must be propagated in a retrograde fashion in order to be measured in the external ear canal. This is an uphill journey against significant physical odds and therefore, any abnormalities of the middle ear significantly affect the measurement of OAE.

COCHLEAR

The cochlear is located within the hardest bone in the body, the temporal bone. It is a coiled tube and the end organ of hearing and is shaped like a snails shell with 2 and 1/2 turns, which is subdivided into 3 compartments. (Figure 3)

The scala tympani and scala vestibuli are filled with perilymph while the scala media is filled with endolymph.

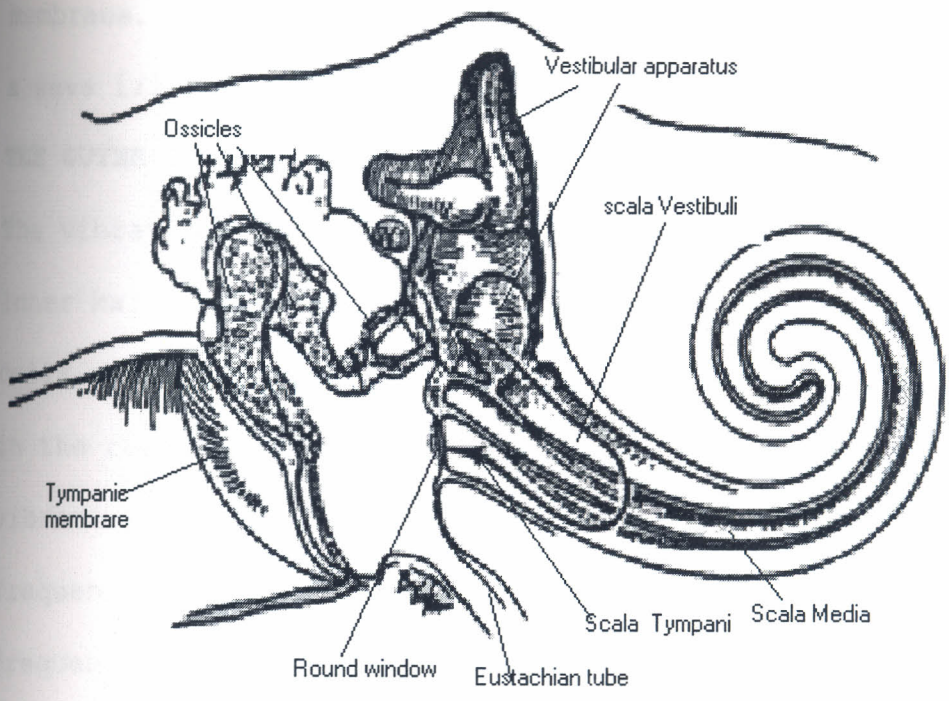


Figure 3: *The Path of sound waves*

The stapes foot plate is coupled to the scala vestibuli so mechanical energy from the middle ear reaching the stapes footplate produces piston like movements of the stapes footplate. Pressure vibrations are then transmitted from the base to the apex of cochlear through the perilymph of the scala vestibuli through a small passage way (helicotrema) at the apex to the scala tympani which ends at the round window.

Between the scalae vestibuli and tympani is the scala media which is bounded on the lower side by a wall consisting from medial to lateral of three continuous structures, the spiral limbus, the basilar membrane and spiral ligament and on the upper side by Reissner's

membrane. (Figure 4) The basilar membrane is deformed in a wave like motion by vibration in the perilymph.

THE OUTER HAIR CELLS.

The vibrations in the Perilymph movement displaces the inner hair cells which open ion channels in the hair cells, triggering an action potential, causing a nerve in the cochlea to fire to the brain. The location of the vibration within the cochlea correlates with the frequency of the sound originally produced with low frequency sounds being near the apex and high frequencies near the base. (5, 22, 23) Twenty thousand nerves representing the twenty thousand frequencies are located along the length of the cochlea, which account for our hearing range.

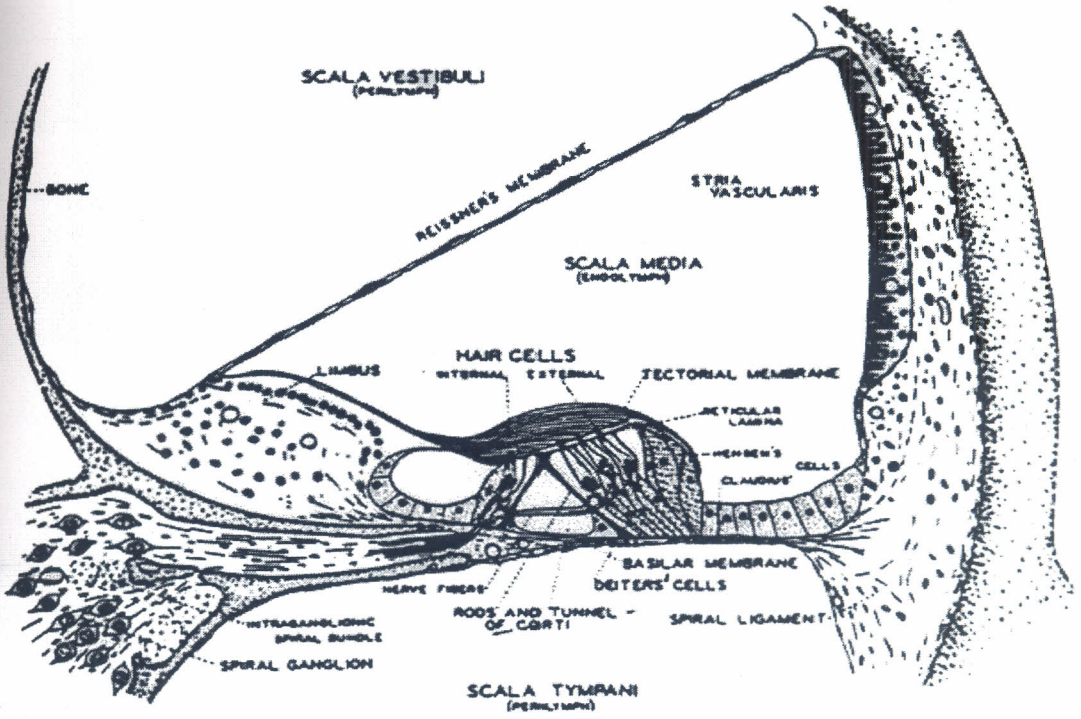


Figure 4: *Cross-section of the cochlea; the organ of Corti*

These cells have more than 100 delicate stereocilia sitting on the apical surface of the hair cells. The stereocilia, which have inter-linkages with each other, are important in triggering the opening of ion channels of the hair cells, and initiate the biochemical processes that lead to motility. (5)

The single most important contributor to OAE production is the motility of the outer hair cells. (5, 20)

The outer hair cell consists of 3 different distinct cylindrical components from out side to the innermost. These are the Plasma membrane, Cortical lattice, Sub surface cisternae (SCC) (Figure 5)

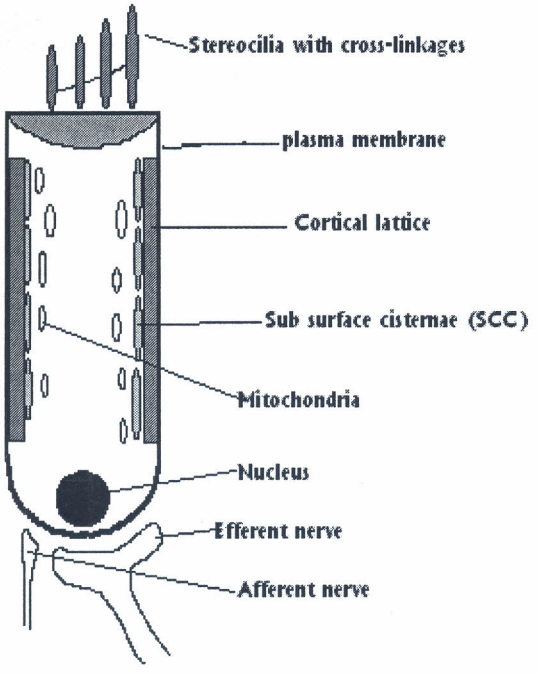


Figure 5: *Schematic of the outer hair cell*

The wall has critical mechanical properties of Stiffeners, resistance to bending and elasticity. The

cortical lattices on the outer portion of the lateral cisternae (part of the endoplasmic reticulum) have chains of rigid segments and molecular substances with elastic properties and are capable of length and shape changes. (5)

Movement of the basilar membrane, secondary to oscillations in outer hair cell length, is transmitted radially from the lateral to the medial portion of the basilar membrane. The result is an enhancement in the inner hair cell function and improved hearing sensitivity and frequency selection. (18)

The residual energy from these movements is transmitted through the perilymph and into the middle ear from the stapes foot plate in retrograde manner and can be measured in the ear canal as Otoacoustic emissions. (5, 18)

The normal cochlea therefore, does not just receive sound; it also produces low-intensity sounds called spontaneous OAEs. The presence of cochlear emissions was hypothesized in the 1940s based on mathematical models of cochlear nonlinearity. (5, 18)

TYPES OF OTOACOUSTIC EMISSIONS

The 4 types of Otoacoustic emissions are as follows:

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

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

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

4. Sustained-frequency Otoacoustic emissions (SFOAEs) - Sounds emitted in response to a continuous tone.(24)

In this study, Transient Evoked Otoacoustic Emissions (TEOAEs) were used. In Transient Otoacoustic emissions, Clicks are the most commonly used stimuli although, tone-burst stimuli may be used. Most commonly, 80- to 85-dB SPL stimuli are used clinically. The stimulation rate is less than 60 stimuli per second. TEOAEs are generally recorded in the time domain over approximately 20 milliseconds. Alternating responses are stored in

alternating computer memory banks, A and B. Data that correlate between the 2 memory banks are considered a response. Data that do not correlate are considered noise. When present, TEOAEs generally occur at frequencies of 500-4000 Hz. Data in the time domain then are converted to the frequency domain, usually in octave band analysis. (5, 7)

Pure-tone (PT) audiometry measures the outer ear, middle ear, cochlea, cranial nerve (CN) VIII, and central auditory system. However, OAEs measure only the peripheral auditory system, which includes the outer ear, middle ear, and cochlea. The response only emanates from the cochlea, but the outer and middle ear must be able to transmit the emitted sound back to the recording microphone.

OAE testing often is used as a screening tool to determine the presence or absence of cochlear function, although analysis can be performed for individual cochlear frequency regions. OAEs cannot be used to fully describe an individual's auditory thresholds, but they can help question or validate other threshold measures (e.g., in suspected functional [feigned] hearing loss), or they can provide information about lesion sites. TEOAE cannot be elicited in persons with hearing threshold over 30 dBHL. Using current technology, most researchers and clinicians find a correlation between

frequency-specific analysis of TEOAEs/DPOAEs and cochlear hearing loss. (15, 16, 17, 22) Transient Evoked Otoacoustic Emissions have a sensitivity of 100% and specificity 93.7%. (28)

An important feature of Otoacoustic emissions is that their synchronization to the stimulus. (Figure 6). This frequency specificity permits the use of powerful signal extraction and processing techniques which lie at the heart of Otoacoustic emission instrumentation. OAE can therefore provide highly specific information about the cochlear response; however they cannot be used to detect the audiometric threshold of a patient. OAEs are therefore a valuable addition to the diagnostic process and not a replacement of the previously trusted methods. (17)

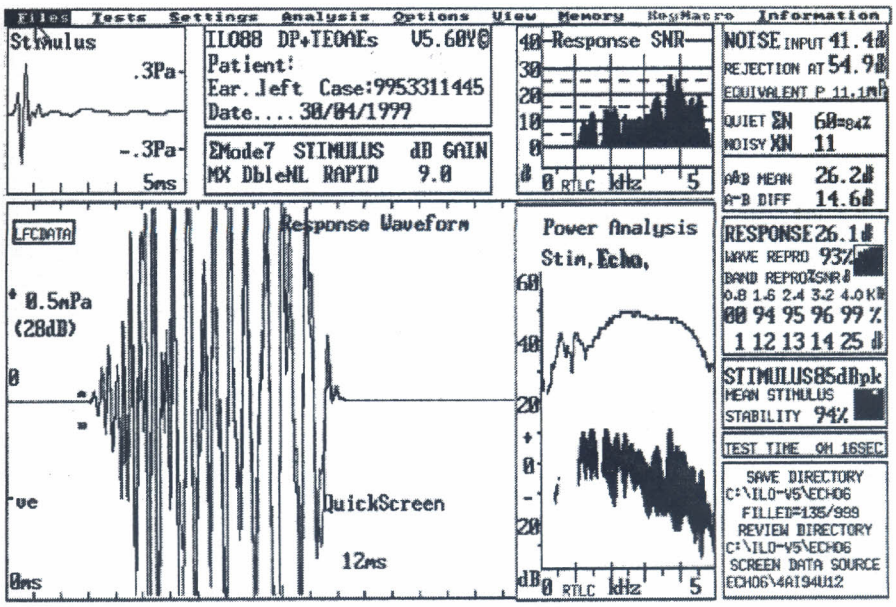


Figure 6: An *example of a TEOAE recording showing the delay between stimulus, presence of Emissions in all frequencies, noise rejection and response confidence and reproducibility at various frequencies.*

CLASSIFICATION OF HEARING LOSS

CONDUCTIVE HEARING LOSS (CHL) results from anything preventing sound from being transmitted from the outside world to the cochlea. Causes range from simple cerumen impaction to middle ear effusions or ossicular chain dysfunction. Sounds perceived by the brain are diminished but generally are not distorted. The bone conduction thresholds are normal, less than 20 dB, but air conduction results suggest a decrease in hearing sensitivity, with an air bone gap greater than 15 dB averaged over 0.5, 1 and 2 kHz. (3, 25)

The patient with a conductive hearing loss typically demonstrates decreased sensitivity across all frequencies. Sometimes hearing is better for the higher frequencies than it is for the lower ones.

Patients with a conductive hearing loss complain of tinnitus frequently. The tinnitus may be localized in one ear, perceived in both ears, or unlocalized within the head. In the case of a conductive impairment, the tinnitus tends to be of relatively low pitch. (17)

SENSORINEURAL HEARING LOSS (SNHL) may result from disruptions in cochlear or neural lesion. These may be a result of hair cell destruction in the cochlea or damage to the eighth cranial nerve. Sounds perceived by the brain, are both diminished and distorted. The degree of distortion is independent of the degree of hearing loss (e.g., it is possible to have a very mild hearing loss but very poor speech discrimination). (17)

The term sensorineural includes both cochlear and retrocochlear disorders. A pure sensorineural impairment exists when the sound-conducting mechanism (outer and middle ear) is normal in every respect, but a disorder is present in the cochlea or auditory nerve.

Audiometrically a hearing loss with an air-bone conduction gap less than 15 dB averaged over 0.5, 1, and 2 kHz frequencies is considered to be a SNHL.

Sensorineural impairment can be congenital or acquired. Congenital sensorineural hearing loss may result from hereditary factors, malformation of the cochlea, intrauterine viral infections, or birth trauma. The etiology of most sensorineural hearing loss is unknown. Acquired sensorineural hearing loss may be caused by noise exposure, acoustic tumor, head injury, infection, toxic drug effects, vascular disease, or presbycusis. (17)

The configuration of the audiogram demonstrating a sensorineural hearing loss may vary significantly and in some instances may suggest the etiology of the loss. Many people with sensorineural losses experience a loss only in the high frequency region. These individuals may have no difficulty understanding speech at normal intensities in a quiet environment since low-frequency hearing is unimpaired. However, they do experience difficulty in understanding speech in a noisy environment. Generally, the low frequencies are defined as the range from 250 Hz to 750 Hz, the middle frequencies as 1,000 Hz to 3,000 Hz, and the high frequencies as 4,000 Hz to 8,000 Hz on the standard audiogram.

Loudness recruitment is usually associated with sensory loss of cochlear origin, which constitutes the majority of sensorineural losses. Recruitment is an abnormally rapid growth of loudness with an increase in sound intensity. The recruiting patient with sensory loss will not hear low-intensity sounds at all, and may just barely hear sounds of moderate intensity, but a small or moderate increase in sound intensity is perceived as uncomfortably loud. This disruption of normal loudness function may be painful to the individual and require hearing aids with the utilization of variable

compression circuitry should the patient pursue hearing aid use.

The patient with sensorineural hearing loss is usually subject to tinnitus of a somewhat different sort from that associated with conductive hearing loss. Generally, the patient with sensorineural loss reports a constant ringing or buzzing noise, which may be localized in either ear or may not be localized. In general, the pitch of tinnitus tends to be higher in sensorineural impairment than in conductive impairment. In addition, the patient may report that tinnitus is only present at night or when background noise is minimal, when in fact it is always present but the patient's perception is only in quiet environments.

In sensorineural losses, the audiometric Weber test is expected to lateralize to the better hearing ear. Audiometrically, sensorineural loss is characterized by overlapping air and bone conduction thresholds. The tympanogram is typically normal, and acoustic reflexes may be present, elevated, or absent.

Contrary to a commonly held misconception, sensorineural hearing loss may be improved by the use of hearing aids. Current hearing aid technology utilizes full dynamic range compression to significantly increase the effectiveness of amplification. (17, 21, 26)

MIXED HEARING LOSS has components of both conductive and sensorineural hearing loss. With a mixed loss, both air and bone conduction thresholds are elevated but bone conduction thresholds are better than air conduction thresholds. The difference between the two thresholds is referred to as the air-bone gap and represents the amount of the conductive component present. The bone and air conduction are raised and the bone-air conduction gap of 15 dB plus bone conduction Thresholds of greater than 20 dB. (17)

Regardless of the type, hearing loss is defined by the American National Standards Institute (1969), in terms of decibels (dB) lost in the following categories:

Slight: 16-25 dB

Mild: 26-40 dB

Moderate: 41-55 dB

Moderately severe: 56-70 dB

Severe: 71-90 dB

Profound: >90 dB. (5, 18, 21)

Most hearing loss in children is congenital or acquired perinatally; however, hearing loss may occur at any age. Approximately 10-20% of all deafness is acquired postnatally, although some genetic causes of deafness

result in hearing loss that begins during childhood or adolescence.

CAUSES OF HEARING LOSS:

Genetic (30-50%): These can be divided into syndromic and nonsyndromic causes. As with all genetic syndromes, genetic causes of hearing loss may be autosomal dominant, autosomal recessive, X-linked, or sporadic.

Nonsyndromic deafness accounts for slightly more than half of genetic deafness. It probably accounts for the majority of those cases classified as "unknown." Children with nonsyndromic deafness are deaf or hard of hearing without any other physical abnormalities, no particular risk to other organ systems, and no increased risk of mental deficiency. Some children have a family history of deafness in a close or distant family member. Others have new mutations or manifest an autosomal recessive gene with no known proband. Subsequent siblings and progeny may help to distinguish a genetic cause from a developmental arrest or prenatal insult. (25, 27)

Exciting new developments in genetic mapping have identified approximately 2 dozen-different abnormal genes that lead to deafness. In some, the molecular or structural defect has been identified (e.g., collagen in

the basilar membrane, or structural defect in a membrane-gating protein). These genes have been classified according to their mode of inheritance, autosomal dominants (*DFNA1* through *DFNA11*), autosomal recessives (*DFNB1* through *DFNB12*), X-linked recessives (*DFN1* through *DFN6*), or mitochondrial (12sr RNA and tRNA-Ser UCN). Some of the genes exhibit variable penetrance. (17, 27)

Syndromic deafness accounts for the remainder of genetic deafness. Some syndromes have a particular inheritance pattern (e.g., Rosenberg syndrome is X-linked) others are sporadic (e.g., Turner syndrome, Klinefelter syndrome). Findings on physical examination usually indicate the presence of a syndrome; however, children with some syndromes develop the associated physical findings later in childhood. In other syndromes, the children present with either deafness or the sequelae of some other biochemical or metabolic derangement. As noted earlier, syndromes may affect any single organ or multiple organ systems. (25)

Prenatal (5-10%): Congenital infections (e.g., cytomegalovirus [CMV], herpes, rubella, syphilis, toxoplasmosis, and varicella) can result in SNHL. Similarly, fetal exposure to teratogens (e.g., alcohol,

cocaine, methyl mercury, thalidomide) also may result in SNHL. All of these perinatal insults result in physical abnormalities, which should prompt recognition of the diagnosis and confirmatory evaluation. (25) Even if these children pass the neonatal screen, careful follow-up concerning their hearing is necessary.

Perinatal (5-15%): A history of prematurity and/or low birth weight, anoxia and/or low APGAR scores, Hyperbilirubinaemia, or sepsis should prompt an evaluation of hearing as these conditions also may result in SNHL. (25, 27)

Postnatal (10-20%): Childhood infections, such as meningitis or mumps, may result in SNHL. Treatment with Ototoxic medications, such as aminoglycosides or furosemide, can result in SNHL. Otitis media or major head injury may result in SNHL or CHL. (25, 27)
Between 20-30% of deaf children have **no certain etiology**. (25) These likely result from a maldevelopment of the ear or the neurological system, which may have been a developmental accident or may have been due to an undiagnosed infectious or teratogenic agent. However, many are likely due to previously undiagnosed genetic defects that may represent new mutations or a genetic recessive trait. Parents frequently implicate causes

including high fevers, seizures, or minor traumas. These have no merit as they do not result in hearing loss.

HIGH-RISK CRITERIA FOR NEONATES (birth to 28 days)

Family history of congenital or early SNHL

Congenital infection known to be associated with SNHL

Craniofacial anomalies

Birth weight less than 1500 g (<3.3 lb)

Hyperbilirubinaemia over exchange level

Ototoxic medications

Bacterial meningitis

Low APGAR scores at birth 0-4 at one minute and 0-6 at 5 minutes

Prolonged mechanical ventilation

Findings of a syndrome associated with SNHL. (18)

HIGH-RISK CRITERIA FOR INFANTS (29 days to 2 years)

Concern about hearing, speech, language, and developmental delay

Bacterial meningitis

Neonatal risk factors associated with SNHL

Head trauma, especially with fracture of the temporal bone

Findings of a syndrome associated with SNHL

Ototoxic medications

Neurodegenerative disorders

Infectious diseases associated with SNHL. (18)

JUSTIFICATION OF THE STUDY

Significant hearing loss is one of the most common abnormalities present at birth and may be present in 3-5 per 1000 children in the United states of America and 9 to 27 per 1000 children internationally 29. This numbers rise in graduates of the Newborn Intensive care units to 10 to 40 per 1000.(29) Screening by high-risk registry can only identify 50% newborns with significant congenital hearing loss and reliance on physician observation and/or parental recognition has not been successful in detecting significant hearing loss in the first year of life. (7, 17, 22)

No universal screening for hearing loss has been established in our region or environment, and consequently delays in intervention leads to poor outcome in children with impaired hearing in terms of language and cognitive development, which is a further strain on our limited resources. This can be overcome by a universal hearing screening program, such as this study plans to establish, and early intervention by the Otolaryngologist and audiologist. Most of the intervention modalities mentioned later are available locally with the exception of cochlear implants. The use of TEOAEs provides a cheap and easy to use test that possesses a high degree of sensitivity and specificity

and hearing loss in newborns is otherwise not accurately detectable by clinical parameters.

Data obtained from this study will help Pediatricians and Otolaryngologist identify risk factors associated with hearing loss. The data will also be useful to other similar studies in the future.

THE STUDY HYPOTHESES

STUDY HYPOTHESIS

TEOAE can be used as a screening tool for universal neonatal hearing loss in a third world set up.

NULL HYPOTHESIS

TEOAE cannot be used as a screening tool for universal neonatal hearing loss in a third world set up.

OBJECTIVES OF THE STUDY

BROAD OBJECTIVE:

Was to identify newborns with hearing loss and document results of hearing screening at KNH using TEOAE

SPECIFIC OBJECTIVES

I. To determine the usefulness of TEOAE as a screening tool

II. To correlate results of hearing screening with TEOAE and presence of risk factors in newborns in KNH.

III. To determine the feasibility of a UNHS at the KNH

MATERIALS AND METHODOLOGY

SETTING

The Kenyatta National Hospital in Nairobi, Kenya.

DESIGN OF STUDY

A cross sectional descriptive study

DURATION OF THE STUDY

This was six months starting in April 2004 and ending in October 2004.

SAMPLE SIZE

The following formula by Kisch and Leslie was used to calculate the sample size n:.

$$n = \frac{Z^2_{1-\alpha/2}\, P(1-P)}{d^2}$$

$$= \frac{1.96^2 \times 0.22 \times 0.78}{0.0025} = 263.68$$

Where:

$Z^2_{1-\alpha/2}$ is the standard normal deviation corresponding to the level of significance of $\alpha = 0.05$.

P= is the proportion of TEOAE screening failing found in similar studies. (29)

d = the width of the confidence interval.

In this study a sample size of 291 was used, which is a higher number than that recommended by the calculation above.

STUDY POPULATION

INCLUSION CRITERIA

All babies born in Kenyatta National Hospital Maternity, in the newborn unit or admitted to the newborn unit after birth elsewhere and are less than 4 weeks old.

EXCLUSION CRITERIA

Any neonate noted to have middle ear infections, congenital meatal atresia or any disease entity that may cause conductive hearing loss, which may interfere with the sensitivity or make the recording of TEOAE not possible.

Any neonate whose parent or guardian refused to give consent.

INTERVENTIONS

A number of interventions are available to these children that fail the TEOAE test. Location of the lesion within the auditory pathway will determine the most appropriate intervention. Options available include Amplification or hearing aids, Corrective surgery depending on cause of hearing loss, Parent support groups, Parental counseling, follow up and later as the child grows older School placement Lip reading or oralism, Sign language and Speech therapy. Cochlear implants are however not yet available locally.

METHODOLOGY

CRITERIA FOR SCREENING

For the purposes of this protocol, it is necessary to set pass criteria for screening such that there is a negligible probability that moderate or greater bilateral hearing impairment, present at birth, will be missed consistent with an acceptable screen pass rate. This will be based on National UK Newborn Hearing Screening Pilot recommended test protocol, 26 November 2001. (15)

Three conditions need to be met before an ear is judged to have passed an OAE test. In this study these are

already programmed into the hardware used (Figure 7), which conforms to the IL088 standard.

- Firstly there must be a high probability that the 'response' seen is a true cochlear response and not due to artifact.
- Secondly, there must be a high probability that a response-like signal is present at the frequency expected. This is usually determined by the degree of reproducibility or the signal to noise ratio although other statistical methods can be applied.
- Thirdly, the intensity of the validated response obtained must be large enough to be within the normal physiological range.

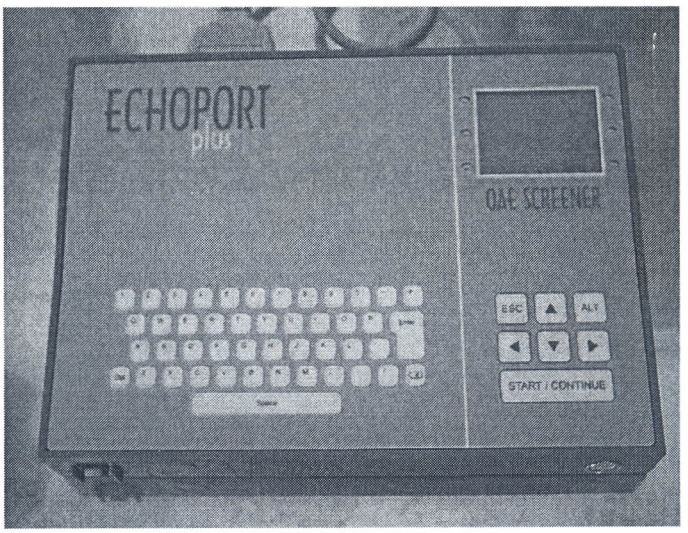


Figure 7: The Echoport OAE screener.

OTHER CRITERIA

In addition, there shall be a minimum amount of good data (below the reject level) of 240 sweeps at the low

stimulus level in the non-linear mode. Stimuli are often presented in packets e.g. groups of 8 (2 stimuli at the high level to 6 at the low level), in this example, the equivalent figure to be used is 40 sets of good data).

MAXIMUM TEST TIME

The maximum test time is 6 minutes for each child. If more than 6 minutes actual testing time is required:

a) The baby is usually too unsettled to test; continuing to test may lead to parental anxiety

b) The testing conditions are unsatisfactory for successful testing

DATA REJECTION LEVEL

The data rejection level will be set as low as possible and not above 55 dB peak SPL. (Figure 6)

DIAGNOSTIC TESTING

The results were analyzed in half octave bands centered at 1, 1.5,2,3 and 4 kHz. A response was reported as present within a particular half octave band if the signal to noise ratio is >=6dB. This was done automatically by the software in the testing apparatus. (Figure 6)

RECORDING DETAILS

ENVIRONMENT

OAE screening in noisy environments is time consuming and inefficient. Every effort was made to screen in a room without continuous background noise such as air-conditioning, ventilation or road traffic noise. Occasional voices and other noises are less of a problem since they are rejected by the instruments artifact rejection system.

STATE OF BABY AND PROBE FITTING

Fitting the probe need not disturb the baby who should be quiet during the test. The baby was fed a few minutes before testing. The probe should be the appropriate size for each child tested. (Figure 8)

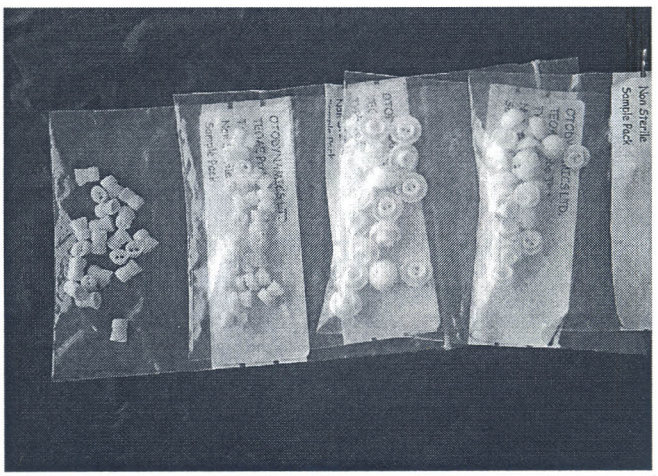


Figure 8: *Probes of various sizes*

STIMULATION AND IN THE EAR CALIBRATION

Unlike audiometry and ABR the level of stimulation is not critical to the interpretation of OAEs. The recommended stimulus level for the TEOAE click stimulus (lower level) is 80 to 88dB peak equivalent sound pressure level (pe SPL) as measured in the neonatal ear canal or an equivalent sized cavity. This provides a means of achieving the target stimulation levels in the ear. (Figure 6)

SIGNAL PROCESSING

TEOAE systems use signal averaging and frequency analysis to enhance and display the response. The instrument, in this study, uses a numerical assessment of the confidence that a true OAE response has been observed. (Figure 6)

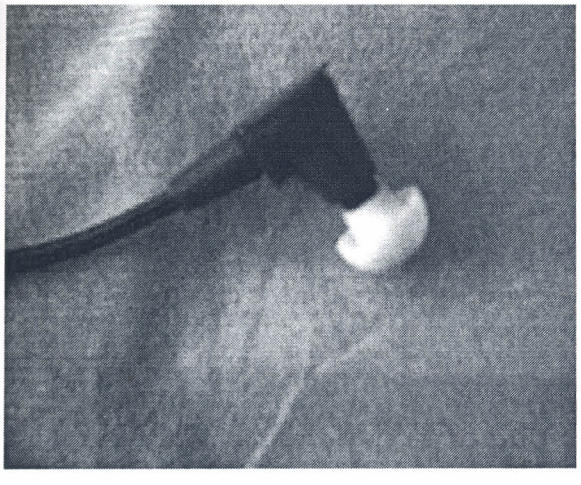


Figure 9: *The Probe*

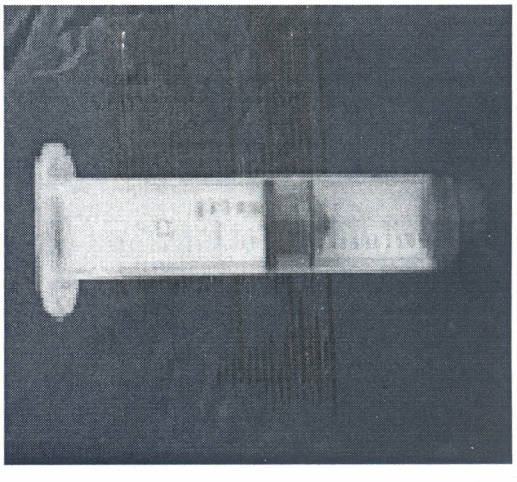


Figure 10: *Cavity tester*

PROBE CHECKS

The probe (Figure 9) was regularly checked for sound output and microphone sensitivity at least:

a) Every 50 babies

b) Once per week

c) After any changes are made to the probe.

After performing an acoustic loop back test with the probe in a cavity tester (figure 8), a biological TEOAE check was carried out on the tester prior to each testing session.

FAILURE TO OBSERVE AN OAE

A recordable OAE indicates the presence of a normal cochlear function at or near the frequencies present in the emission. Its absence could be for one of many reasons e.g. poor recording conditions, bad probe fitting, the presence of outer ear or middle ear disease, or an absent cochlear response or one of too small amplitude to record. Normally hearing ears produce a wide range of TEOAE intensity and waveforms. Some healthy ears may only produce emissions strong enough to be visible above the infant and background noise in an only narrow range of emission frequencies whilst others will produce a broad range of emission frequencies. (Figure 6)

DATA MANAGEMENT

All data emanating from this study was entered into the questionnaire (appendix ii) and then into a computer data base, cleaned and verified and analyzed using the Statistical Package for Social Sciences® software, Release 11.0.0.

Data was analyzed into means and rays and is presented in the form of tables, pie charts and graphs. Any associations will be considered statistically significant at a P value of less than or equal to 0.05.

ETHICAL CONSIDERATIONS

All patients had a signed informed consent to be included in the study. The consent was in all cases given by either the parent or an appropriate guardian as the case may be. The study was carried out after approval by the Ethical and Research committee of Kenyatta National Hospital.

The results of the hearing screening are confidential and available to the parents.

All patients were managed according to the current and conventional treatment for their illnesses.

The results of the study are to be published and are available to the medical fraternity.

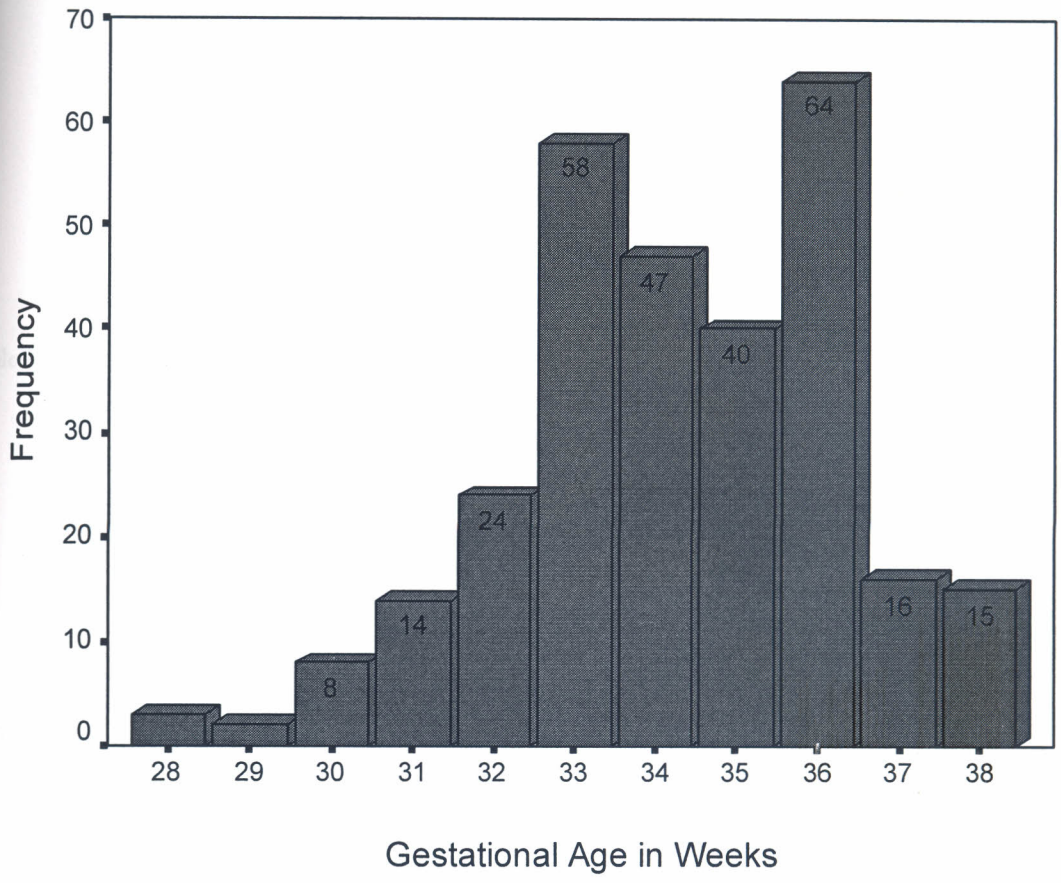
RESULTS

In this study 291 newborns were screened using TEOAE. 11 of them (3.8%) failed the screening test. (Table 1)

	Frequency	Percent
Fail	11	3.8
Pass	280	96.2
Total	291	100.0

Table 1: *percentages of Pass and fail rates in this study*

The distribution of gestational age of the newborns in the study is shown in Graph 1 and was between 28 and 38 weeks with a mean of 34.23 and mode of 36 weeks. (Table 2)



Graph 1: *Graph Frequencies of Gestational*

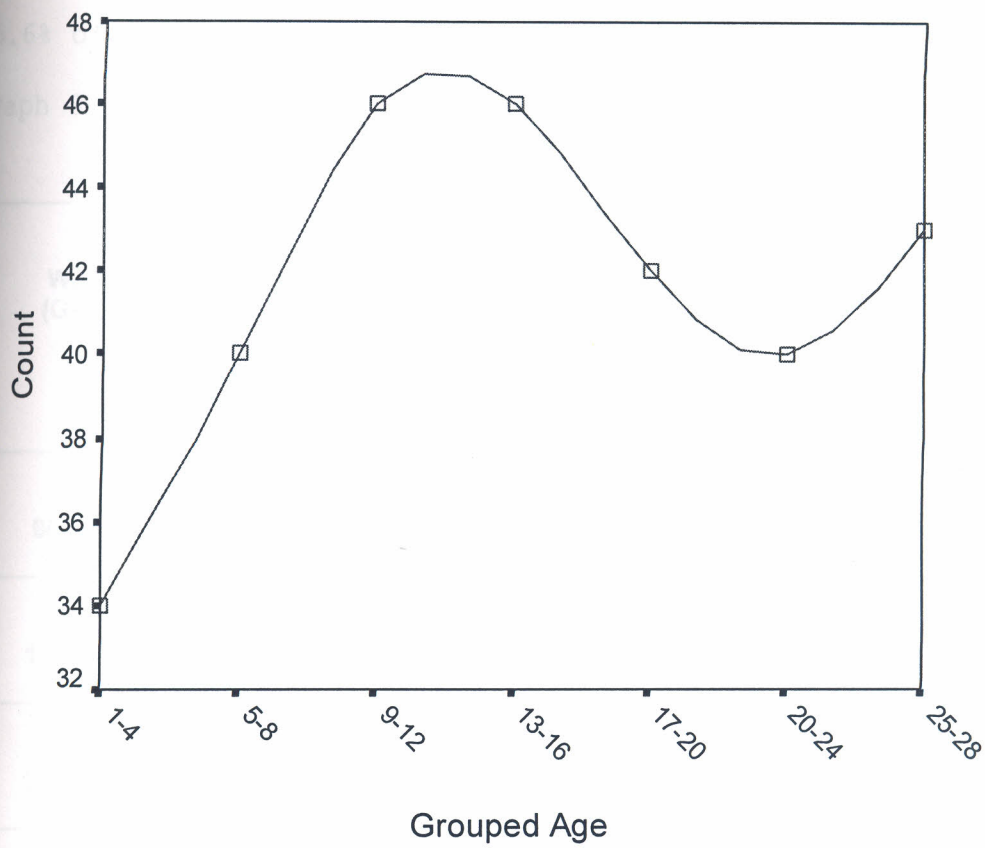
N	291
Mean	34.23
Median	34.00
Mode	36
Minimum/ Maximum	28/ 38

Table 2: *Statistics of Gestational Age*

The age in days after birth was between 1 and 28 day as per the study design and was distributed as is shown in table 3 and Graph 2

AGE (DAYS)	Frequency	Percent	Valid Percent	Cumulative Percent
1-4	34	11.7	11.7	11.7
5-8	40	13.7	13.7	25.4
9-12	46	15.8	15.8	41.2
13-16	46	15.8	15.8	57.0
17-20	42	14.4	14.4	71.5
20-24	40	13.7	13.7	85.2
25-28	43	14.8	14.8	100.0
Total	291	100.0	100.0	

Table 3: *Frequencies grouped age.*

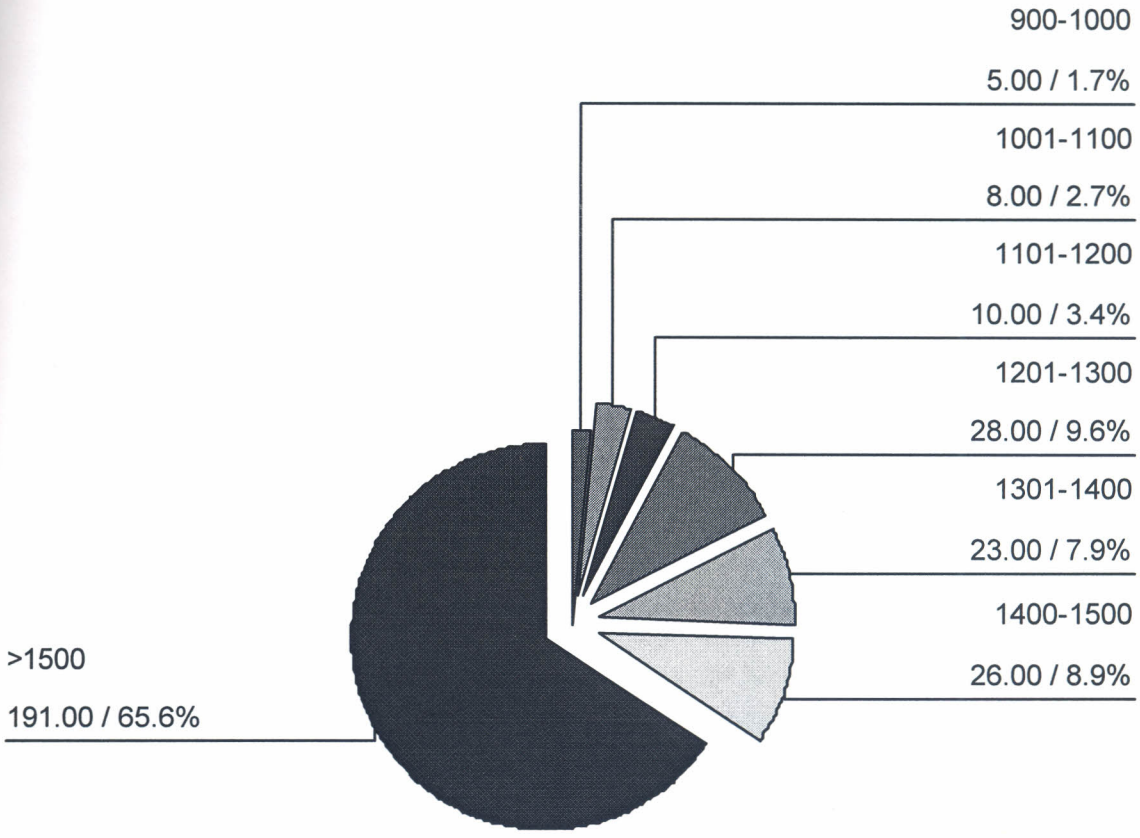


Graph 2: *Distribution of age in the study.*

The newborn tested with the lowest weight weighed 920g, 65.6% of those tested weighed over 1500g. (Table 4, Graph 3)

Weight (Grams)	Frequency	Percent	Cumulative Percent
900-1000	5	1.7	1.7
1001-1100	8	2.7	4.5
1101-1200	10	3.4	7.9
1201-1300	28	9.6	17.5
1301-1400	23	7.9	25.4
1400-1500	26	8.9	34.4
>1500	191	65.6	100.0
Total	291	100.0	

Table 4: *Frequencies Grouped weight.*

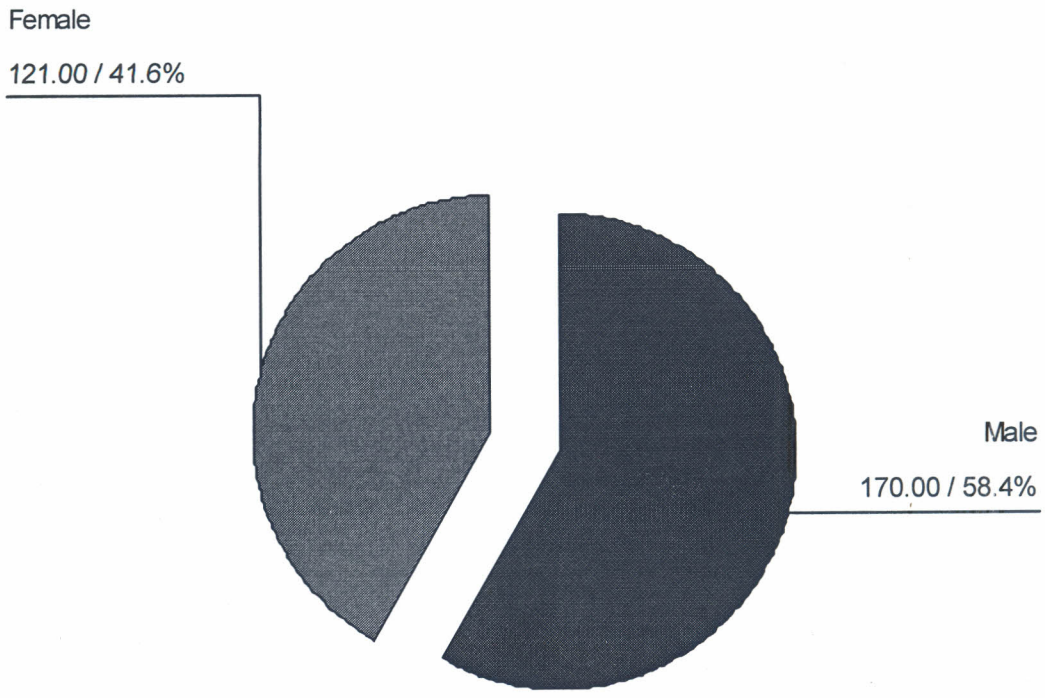


Graph 3: *pie-chart showing distribution among the newborns tested*

The female to male ratio was 1: 1.403. (Table 5, Graph 4)

	Frequency	Percent
Male	170	58.4
Female	121	41.6
Total	291	100.0

Table 5: *sex distribution in the study*



Graph 4 : Sex distribution

Only 13.1% of those tested had been delivered by Caesarean section. (Table 6)

Mode of Delivery	Frequency	Percent
] SVD	253	86.9
C/S	38	13.1
Total	291	100.0

Table 6: *Mode of Delivery*

Of the 11 neonates that failed the TEOAE screening 4 had craniofacial anomalies, when this was tested using Chi-Square Tests, it proved to be a highly significant association. (Table 7, 8) These included cleft lip/palates, Pierre-Robin syndrome and Downs Syndrome.

			TEOAE		Total
			Fail	Pass	
Cranio-facial abnormalities	Yes	Count	4	6	10
		% within Cranio-facial abnormalities	40.0%	60.0%	100.0%
	No	Count	7	274	281
		% within Cranio-facial abnormalities	2.5%	97.5%	100.0%
Total		Count	11	280	291
		% within Cranio-facial abnormalities	3.8%	96.2%	100.0%

Table 7: *Cross tabs of Cranio-facial abnormalities vs. TEOAE.*

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	37.352	1	.000		
N of Valid Cases	291				

Table8:*Chi-Square Tests for Cranio-facial anomalies versus TEOAE Failure*

Analysis of the data obtained for group weight against failure of the TEOAE screening showed a very significant correlation between the two. 34.4% of the neonates tested were below 1500g, most being between 1200-1300g making 9.6% of those tested (Table 4). The lower the birth weight the higher the probability of failing the screening, as is demonstrated (Tables 9, 10) below.

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	31.812	6	.000
N of Valid Cases	291		

Table 9: *Chi-Square Tests Grouped weight Vs TEOAE.*

			TEOAE		Total
			Fail	Pass	
Grouped Weight (In Grams)	900-1000	Count	1	4	5
		% within Group	20.0%	80.0%	100.0%
		% of Total	.3%	1.4%	1.7%
	1001-1100	Count	1	7	8
		% within Group	12.5%	87.5%	100.0%
		% of Total	.3%	2.4%	2.7%
	1101-1200	Count	1	9	10
		% within Group	10.0%	90.0%	100.0%
		% of Total	.3%	3.1%	3.4%
	1201-1300	Count	2	26	28
		% within Group	7.1%	92.9%	100.0%
		% of Total	.7%	8.9%	9.6%
	1301-1400	Count	1	22	23
		% within Group	4.3%	95.7%	100.0%
		% of Total	.3%	7.6%	7.9%
	1400-1500	Count	5	21	26
		% within Group	19.2%	80.8%	100.0%
		% of Total	1.7%	7.2%	8.9%
	>1500	Count	0	191	191
		% within Group	.0%	100.0%	100.0%
		% of Total	.0%	65.6%	65.6%
Total		**Count**	**11**	**280**	**291**
		% within Group	**3.8%**	**96.2%**	**100.0%**
		% of Total	**3.8%**	**96.2%**	**100.0%**

Table 10: *Cross tabs for Grouped weight Vs TEOAE.*

Another correlation was also obtained between Gestational age and failure of the TEOAE screening. The lower the birth weight the more likely the baby to fail the screening test. (Tables 11, 12)

			TEOAE		
			Fail	Pass	Total
grouped gestation Age (in Weeks)	28-30	Count	2	11	13
		% within grouped gestation Age	15.4%	84.6%	100.0%
		% of Total	.7%	3.8%	4.5%
	31-33	Count	7	89	96
		% within grouped gestation Age	7.3%	92.7%	100.0%
		% of Total	2.4%	30.6%	33.0%
	34-36	Count	2	149	151
		% within grouped gestation Age	1.3%	98.7%	100.0%
		% of Total	.7%	51.2%	51.9%
	37-38	Count	0	31	31
		% within grouped gestation Age	.0%	100.0%	100.0%
		% of Total	.0%	10.7%	10.7%
Total		Count	11	280	291
		% within grouped gestation Age	3.8%	96.2%	100.0%
		% of Total	3.8%	96.2%	100.0%

Table 11: *Cross tabs of Gestation vs. TEOAE.*

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	11.789	3	.008

Table12: *Chi-Square Tests for Gestation vs. TEOAE.*

83 neonates tested had low APGAR Scores (i.e. APGAR scores of less than 5), 6 of them failed the screening. This was statistically very significant. After Chi test this was a significant association as it surpassed the cut-off criterion of 0.05. (Table 13, 14)

		LOW APGAR SCORE		Total
		Yes	No	
Fail	Count	6	5	11
	%	54.5%	45.5%	100.0%
Pass	Count	77	203	280
	%	27.5%	72.5%	100.0%
Total	Count	83	208	291
	%	28.5%	71.5%	100.0%

Table 13: *Crosstabs low APGAR score vs. TEOAE.*

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	37.352	6	.000

Table 14:Chi-Square Tests

Only one neonate tested was known to have had meningitis and failed the screening.

Analysis of sex (Table 15, 16), mode of delivery (Table17, 18), hyperbilirubinaemia did not show any significant correlation in this study. Although prolonged intubation is a known risk factor, no statistical significance could be obtained firstly because of the relatively few newborns intubated and the screening machine rejected the ambient noise levels making testing no possible.

Family history of deafness and prenatal risk factors were unknown in this study.

Exposure to ototoxic drug: All the babies in the newborn unit were given an aminogylcoside cover and so analysis of this parameter was not possible even though this is a well recognized risk factor in neonates as in all inner ears.

The sex of the neonate did not appear to be stastically significant.

			Fail	Pass	Total
Sex of the child	Male	Count	7	163	170
		% within Sex	4.1%	95.9%	100.0%
		% of Total	2.4%	56.0%	58.4%
	Female	Count	4	117	121
		% within Sex	3.3%	96.7%	100.0%
		% of Total	1.4%	40.2%	41.6%
Total		Count	11	280	291
		% within Sex	3.8%	96.2%	100.0%
		% of Total	3.8%	96.2%	100.0%

Table 15: *Cross tabs sex Vs TEOAE.*

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.128	1	.720
N of Valid Cases	291		

Table 16: *Chi-Square Tests for sex Vs TEOAE.*

Mode of delivery was no shown to have any statistical significance on screening outcome.

			TEOAE		Total
			Fail	Pass	
Mode of Delivery	SVD	Count	10	243	253
		% within Mode of Delivery	4.0%	96.0%	100.0%
	C/S	Count	1	37	38
		% within Mode of Delivery	2.6%	97.4%	100.0%
Total		Count	11	280	291
		% within Mode of Delivery	3.8%	96.2%	100.0%

Table 17: *Cross-tabulation Mode of Delivery Vs TEOAE*

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	.159	1	.691
N of Valid Cases	291		

Table 18: *Chi-Square Tests Mode of Delivery Vs TEOAE*

DISCUSSION

1. The present study confirms the great accuracy of neonatal screening for congenital hearing loss by means of TEOAE analysis, despite the fact that the possibility of false negatives (hearing neuropathy) must always be considered. The reduced frequency of TEOAEs detectable in the high-risk subpopulation confirms data in the literature. 11 of the 291 newborns tested failed the TEOAE screen. The prevalence of congenital loss of hearing was 17.2 times higher in the high-risk subpopulation compared to the control group i.e. 38 per 1,000 live births vs. 2.2 per 1,000 live births. (30) Data in the literature have reported a prevalence of sensorineural hearing defects from 4.4-7.1 up to 50 times greater in premature infants in NIC. (13, 16, 22) Emissions are not produced in an ear when there is hearing loss of more than 30 to 40 dB, but unfortunately a small number of data points, a weak stimulus, low reproducibility, poor probe stability, a whimpering or crying infant, debris in the external ear canal, or performance of the test at too early a juncture are other factors that may cause absence of a response, in addition to a hearing defect.

2. Sex:

There was a slightly higher number of male neonates as compared to females with a female to male ratio of

1:1.404 (Graph 4). This did not however affect significantly the outcome of the screening test. 95.9% of males passed compared to 96.7% of females.

3. Ototoxic medication:

The results stress the importance of audiological risk factors, in particular of, the administration of ototoxic drugs (100%), severe birth asphyxia (28.5%) and craniofacial anomalies (1.37%). Analysis of clinical significance of ototoxic medication could not be done as all the newborns had received an aminogylcoside at some point in time before the screening. Despite this it is a well documented fact that such drugs are injurious to hearing in all ages and more so in the newborn.

4. Birth asphyxia:

83 neonates tested had low APGAR Scores (i.e. APGAR scores of less than 5), 11 Neonates who failed the TEOAE screen 6(54.5%) had a low APGAR score. (Table 13) This was statistically very significant. Birth asphyxia was a common indication for admission to the NBU. A low APGAR score was also the most frequent indication for intubation. However it was difficult to test these intubated Neonates as the ambient sound was rejected by the screening equipment. This does not dispute the fact that both factors are of major clinical and statistical significance.

5. Gestational age;

There was a higher chance of failure of TEOAE screening with reducing gestational age and birth weight. Failure rates in screening before discharge from a NBU have been highest in infants with the lowest Birth Weights (19). A similar trend was observed in this study. The Groped age with the highest failure was 28-30 weeks with 13 babies tested of whom 2 (15.4%) failed the screen; this failure rate shows a steady decline with increase in the gestational age at birth. (Table11)

6. Birth weight:

Of the neonates with lowest weight (900-1000g) screened one (20%) failed out of 5 Babies screened this failure figure appeared to decrease with increase in birth weight (Table 9).

7. Multiple risk Factors

Many of the subjects that failed had multiple risk factors; this made analysis of the data rather difficulty, and the question as to which risk factor had a greater contribution to the failure of the screening in a particular newborn. The high rate of sensorineural hearing damage in these risk categories has been attributed to a combination of cochlear immaturity, hypoxia-acidosis, prolonged exposure to acoustic trauma and ototoxic drugs. (29).

8. Cranio-facial abnormalities:

It was noted that newborns with cleft lip/palates failed the screen, this could be attributed to the fact they are more prone to otitis media with effusion (OME) but cleft lips/ palates are not a risk factor to SNHL per se. one child was Identified to have Pierre-Robin Syndrome and failed the screen.

9. Sensitivity And Specificity

Unfortunately in this study it was not possible to calculate the sensitivity or specificity of TEOAE because only one measuring tool was available and this was not one of the objectives of the study. Universal TEOAE screenings of newborn infants, within the Rhode Island Hearing Assessment Program and elsewhere, have achieved sensitivities of 95-100% and specificities of 90-95% for the prediction of hearing loss. Because of the high failure rates among preterm and other high-risk infants a sensitivity of 100% with a specificity of 50% has been reported for such series. (23) The gold standard determination of permanent hearing impairment for validating results of screening tests is a combination of otolaryngological and audiological consultation, diagnostic ABR testing, and other electrophysiological testing.

10. Overall, the data confirm the feasibility of universal screening for congenital hearing loss. The study also demonstrates that important data may be obtained via TEOAEs in high-risk infants. The initial cost of the equipment may seem prohibitive but the long term management cost of children with pre-lingual deafness that has not had any intervention may be far greater. The initiative is now for the hospital to consider implementing a screening program. A high volume of testing expected in this area and it is therefore important that attention and support be given to the development personnel to be involved in the identification, diagnosis, and follow-up of congenital hearing loss.

LIMITATIONS

1. Inability to test babies that were receiving O_2 by mask as the screening TEOAE machine kept rejecting the ambient noise threshold.

2. A number of parents were apprehensive to give consent to the screening test in the study despite being counseled extensively. This may be an indicator for congenital hearing loss as well as other defects information inclusion in the Ante Natal Clinic education program in this country.

3. Inability to ascertain history of prenatal risk exposure in the mother.

CONCLUSIONS

1. Birth asphyxia as measured by a low APGAR scores is the most common risk factor to failure of TEOAE in our environment.

2. Low birth weight and gestational immaturity are also very significant.

3. Ototoxic medications should be used with more rationale. Despite the fact that this parameter could not be analyzed there is no doubt of its significance in contribution to cochlear damage as shown by numerous studies all over the world.

4. Craniofacial Anomalies, meningitis were shown to be of significance.

RECOMMENDATIONS.

1. A study to establish the effect ototoxic drugs on the cochlear would appear warranted in view of the extensive use of aminoglycosides, especially, in this institution. Issues to be addressed in such a study should include indication, dosage, duration, plasma levels and effects on DPOAE's (as these are more accurate and standard for such a study.

2. Establishment of a permanent hearing screening program using already established protocols e.g. the Rhode Island or National UK Newborn Hearing Screening Pilot recommended test protocol as was used in this study.

3. A feasibility assessment study for the establishment of a cochlear implant programme should be carried out. This should in due course lead to an Early Intervention and Rehabilitation program that is coupled to the Newborn Screening programme.

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APPENDIX I

INFORMED CONSENT

I_____of_____The mother/father/guardian of_____
IP NO_____
STUDY ID _____
Hereby consent that my child be included in the Newborn hearing screening study. This study shall include clinical examination and Measurement of Otoacoustic Emissions (TEOAE)
The TEOAE measurement is a safe test. The benefits of the study include early identification of any hearing loss, which will mean early intervention or treatment.
It is NOT a MUST to be included in this study.
One can always withdraw from the study if they changed their mind, however they would still be eligible to enjoy the normal health facilities and treatment like any other routine patient.

I understand all the above as it has been explained to me by Dr Njoroge:

Signature of parent/next of kin_____
Date_____/_____2003

Signature of doctor _____Date ____/_____/2004

KUKUBALI KWA MCHUNGANJI KWA UTAFITI WA UPUNGUFU WA KUHISI SAUTI

Mimi_____Kutoka_____
Mama/baba/mchungaji wa_____
IPNO_____
Nambari ya utafiti_____
Ninakubali mtoto wangu kuwa mojawapo wa wagonjwa watakao husika na utafiti wa upungufu wa kuhisi sauti.
Nimeelezwa ya kwaba kuhusika kwa utafiti huu ni kwa HIARI YANGU mwenyewe na si kwa LAZIMA.
Na weza kujitoa kwenye utafiti wakati wowote na bado ni tapata matibabu kama wagonjwa wengine. Nimeelezwa na nimeelewa hii yote na Daktari Njoroge
Sahihi ya mzazi/mchungaji_____
Sahihi ya daktari_____
Tarehe:_____

APPENDIX II

STUDY PROFORMA SHEET

CONSENT STUDY No: WARD:

NAME: MOTHER/GUARDIAN:

P.O. BOX TELEPHONE:

AGE: SEX: GESTATIONAL AGE :
DATE OF BIRTH:

APGAR SCORES AT BIRTH: MODE OF DELIVERY:

	YES, Please specify	NO
BIRTH WEIGHT LESS THAN 1500 G		
CRANIOFACIAL ANOMALIES		
OTOTOXIC MEDICATIONS		
BACTERIAL MENINGITIS		
HYPERBILIRUBINEMIA REQUIRING EXCHANGE TRANSFUSION, ie Over (170 x Weight) mmol/ml		
PROLONGED MECHANICAL VENTILATION, over 5 days.		
FINDINGS OF A SYNDROME ASSOCIATED WITH SNHL		
FAMILY HISTORY OF DEAFNESS		
PRENATAL RISK FACTORS		

	RIGHT EAR	LEFT EAR
OTOSCOPY		
TEOAE PASS OR FAIL		

DISCHARGED/FOLLOW UP RECOMMENDED: