

**PREVALENCE AND RISK FACTORS OF HEARING LOSS AMONG  
PEDIATRIC PATIENTS AT A TERTIARY TEACHING HOSPITAL IN ACCRA**



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**THIS DISSERTATION IS SUBMITTED TO THE UNIVERSITY OF GHANA, LEGON IN  
PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE AWARD OF THE MASTER  
OF SCIENCE DEGREE IN AUDIOLOGY**


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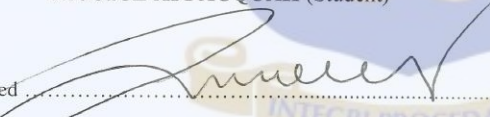
### DECLARATION


#### DECLARATION

I, **REJOICE AFI ACQUAH**, hereby declare that this dissertation which is submitted in partial fulfillment of the requirements for Masters of Science degree in Audiology is the result of my own independent research project and that, except where otherwise other sources are acknowledged with explicit references and are included in the reference list, this work has not previously been accepted in substance for any degree and neither is it being concurrently submitted in candidature for any degree.

I hereby give permission for the Department of Audiology to seek dissemination/publication of the dissertation in any appropriate format. Authorship in such circumstances to be jointly held between me as the first author and the project supervisors as subsequent authors.

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## **DEDICATION**

This dissertation is dedicated to my husband, William, and my children Benedict, Jeffrey and Olive.



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**LIST OF ABBREVIATIONS**

AAA	American Academy of Audiology
ABR	Auditory Evoked Potentials
ADP	Acoustic Distortion Product
ASHA	American Speech-Language and Hearing Association
ASSR	Auditory Steady- State Response
BSA	British Society of Audiology
CAEP	Cortical Auditory Evoke Potential
CDC	Center for Disease Control
CHL	Conductive Hearing Loss
CPA	Conditioned Play Audiometry
CSHL	Congenital Sensorineural Hearing Loss
dB	Decibel
DF	Degree of Freedom
DP	Distortion Product
DPOE	Distortion Product Otoacoustic Emission
ER	Etymotic Research
GBD	Global Burden of Disease
HAC	Hearing Assessment Center
HL	Hearing Loss
Hz	Hertz
ISO	International Standards Organization
JICH	Joint Committee on Infant Hearing

KATH	Komfo Anokye Teaching Hospital
KBTH	Korle Bu Teaching Hospital
MHL	Mixed Hearing Loss
MSAC	Medical Services Advisory Committee
NF	Noise Floor
NICU	Neonatal Intensive Care Unit
NIH	National Institute of Health
NIHL	Noise Induced Hearing Loss
OAE	Otoacoustic Emission
OHC	Out Hair Cell
PCHL	Permanent Congenital Hearing Loss
SNHL	Sensorineural Hearing Loss
SNR	Signal-to-Noise Ratio
SPL	Sound Pressure Level
SPSS	Statistical Package for Social Sciences
SVD	Spontaneous Vaginal Delivery
TDH	Telephonic Dynamic Headphone
TEOAE	Transient Evoked Otoacoustic Emission
TROCA	Tangible Reinforced Operant Conditioned Audiometry
TTS	Temporary Threshold Shift
UNICEF	United Nations Children Fund
USA	United States of America
VRA	Visual Reinforcement Audiometry

VROCA Visual Reinforced Operant Conditioned Audiometry

WHO World Health Organization

## ABSTRACT

**Background:** Hearing loss is the most frequent hearing deficit in both adult and pediatric populations, affecting more than 250 million people in the world. The consequences of hearing impairment are diverse and include severe impairment, often resulting in a reduced ability to communicate, delay in language acquisition, economic and educational disadvantage, social isolation and stigmatization. However, these consequences can be reduced by early detection together with appropriate audiological and speech therapy interventions to enhance speech, language and cognitive development in hearing impaired children.

**Aim:** This research study was aimed at identifying maternal and infant risk factors predisposing children to hearing loss as well as establishing the prevalence of hearing impairment in children aged 6 months to 10 years in a teaching hospital in Ghana.

**Methods:** Different audiological test batteries (otoscopy, tympanometry, otoacoustic emissions, behavioral audiometry) and instrumentation (GSI TYMPSTAR tympanometer, Interacoustics Paediatric PA5 and Interacoustics AD229e audiometers) were utilized to identify risk factors and determine prevalence of hearing loss in pediatric patients presenting for hearing profile evaluations at a tertiary hospital. Behavioral assessments were performed via visual reinforcement audiometry, and play audiometry

**Results:** Anaemia was the most prevalent (8.3%) maternal condition reported by the mothers during pregnancy. The prevalent infant risk factors to hearing loss were anoxia after delivery (24.0%), admission of infants at NICU for more than two days (22.9%), ear infection (21.9%), and low birth weight (8.9%). Only 6.3% had family histories of hearing loss. Overall, there were less passes (40.1%) than refers (59.9%) in the OAE tests. No significant associations between hearing loss and demographics (age and gender) in both ears as measured by the various test

batteries across the test frequencies were found. Significant associations between hearing loss and tympanograms were established in both ears across all frequencies via VRA. On the contrary, no significant association was found between hearing loss and tympanograms by play audiometry test results. The prevalence of hearing loss was relatively higher in the younger (0.5 – 2.5 years) children. Profound hearing loss was generally most prevalent across all frequencies in the various assessments, while mild and severe hearing loss were less prevalent.

**Conclusion:** Maternal and infant risk factors predisposing children to hearing loss were identified. Degrees of hearing loss were assessed and the associated prevalence was estimated. Non-significant associations between hearing loss and demographics were established using the assessment tools. The need to have a mandatory hearing screening for newborns before discharge from hospitals to ensure early detection and intervention of hearing loss is recommended.

**Keywords:** Risk factors, hearing loss, otoacoustic emission, tympanometry, prevalence.

## **CHAPTER ONE**

### **INTRODUCTION**

#### **1.1 BACKGROUND**

Hearing impairment, also known as hearing loss is the most frequent disorder in both adult and pediatric human populations, affecting more than 250 million people in the world. The consequences of hearing impairment are diverse and include inability to interpret speech sound often resulting in a reduced ability to communicate, delay in language acquisition, economic and educational disadvantage, social isolation and stigmatization (Marthers et al, 2000). These consequences could be life-long in children with permanent hearing loss and impact negatively on their families. However, these consequences can be reduced by early detection together with appropriate audiological and speech therapy interventions to enhance speech, language and cognitive development in hearing impairment in children. (Paludetti et al, 2012).

Hearing impairment in children can be classified by type (conductive, sensorineural, mixed, and central), degree, configuration, time of onset, aetiology, and finally, consequences on speech development.

##### **1.1.1 Incidence and Prevalence of Hearing Impairment**

Incidence and prevalence are terms commonly used in describing disease epidemiology. Incidence is the rate of new (or newly diagnosed) cases of the disease, and generally reported as the number of new cases occurring within a period of time (e.g., per month, per year). It is more meaningful when the incidence rate is reported as a fraction of the population at risk of developing the disease (e.g., per 100,000 or per million population).

The accuracy of incidence data depends upon the accuracy of diagnosis and reporting of the disease. In some cases it may be more appropriate to report the rate of treatment of new cases since these are known, whereas the actual incidence of untreated cases is not. Incidence rates can be further categorized according to different subsets of the population – e.g., by gender, by racial origin, by age group or by diagnostic category (Greenberg et al., 2005).

Prevalence is the actual number of cases alive; with the disease either during a period of time (period prevalence) or at a particular date in time (point prevalence). According to Greenberg et al (2005), period prevalence provides the better measure of the disease load since it includes all new cases and all deaths between two dates, whereas point prevalence only counts those alive on a particular date. Prevalence is also most meaningfully reported as the number of cases as a fraction of the total population at risk and can be further categorized according to different subsets of the population.

There are 360 million persons (5.3% of the world's population) in the world with disabling hearing loss of which 328 million (91%) are adults and 32 million (9%) are children. Geographically, hearing loss is unequally distributed across the world. The statistical distribution as reported by the World Health Organization (WHO) is presented in Table 1.1 (WHO, 2012). The prevalence of childhood permanent congenital hearing loss (PCHL) is 1.2 to 1.7 cases per 1000 live births and is essentially due to sensorineural hearing loss (SNHL). In the United States (US), the rate of PCHL ranges from 1 to 3 per 1000 live births and represents the most common neurological birth defect (Korver et al. 2010). According to the National Institute of Health, 2 to 3 children out of 1,000 in the USA are born deaf or with some loss of hearing.

**Table 1.1: WHO distribution of hearing impairment**

Geographical location	Percent distribution, %
South Asia	27.0
East Asia	22.0
Developed countries	11.0
Asia Pacific	10.0
Latin America and the Caribbean	9.0
Central and East Europe. Central Asia	9.0
Sub-Saharan African	9.0
North Africa and The Middle East	3.0
Total	100.0

Newborn hearing tests are done in some US cities for purposes of early intervention which is important for any form of identifying hearing impairment in children (NIH, 2010). A study from the United Kingdom found the prevalence of hearing loss > 40 dB hearing level (HL) to increase from 1.06/1000 at birth to 1.65-2.05/1000 at 9 years of age. This would mean that 35% to 50% of all hearing loss in 9-year-old children is postnatal. A large number of postnatal hearing losses are conductive (Billings and Kenna, 1999). It has been estimated that the cost of communication disorders to the US economy ranges between US\$176 billion and US\$ 212 billion in 1999, representing 2.5% - 3 % of the gross national product (Ruben, 2000). In Australia, it has been shown that the real financial cost of hearing impairment was approximately US\$ 9.6 billion (1.4% of gross domestic product for the country) (Access Economics, 2006).

According to the WHO (2012) report, 32 million children below the age of 15 years are affected by hearing loss worldwide. The prevalence is highest among South Asia, Asia Pacific and Sub-Saharan Africa. Smith (2003) reported an overall increase in the prevalence of hearing loss in

developing countries. Seely et al. (1995) reported a 9.1% prevalence of hearing loss among Sierra Leonean children presenting with mild or greater hearing loss. The prevalence was 9% in the 5-15 year age group, while 4.1% prevalence was been reported in Swaziland for children of the same age group (Swanepoel et al., 2009, Olusanya and Newton, 2007). In Kenya, Gambia and Tanzania, about 2.5 to 3.5 children in 1000 suffer from severe to profound hearing loss. In South Africa, infant hearing loss is the most common congenital sensory birth defect with a prevalence of 4 to 6 per 1 000 live births, while about 7.5% of school children suffer from varying degrees of hearing loss.

Causes for the development of hearing loss were established in 2007 by the Joint Committee on Infant Hearing (JCIH). Among the most prominent causes are family history, craniofacial abnormalities, in-utero infections, severe hyperbilirubinaemia, neonatal intensive care units (NICU), admission for 2 days or more, respiratory distress, prolonged mechanical ventilation and syndromes associated with hearing loss. However, in 50% of infants with PCHL there are no known causes (Joint Committee on Infant Hearing, 2007).

Hearing loss has been associated with various diseases such as meningitis, mumps, measles, and jaundice. In most bacterial infections, damage to the ear results from infiltration of the organism via the internal meatus. Viral diseases may cause histopathological damage to the organ of Corti, or complete destruction of the stria vascularis and tectorial membrane, and atrophy or destruction of the neural pathways (Northern and Downs, 1991). A recent study found hearing defects in children born of mothers who suffered from symptomatic rubella during their pregnancies (Niedzielska et al, 2000). Several medications have shown potential harmful effects on hearing. Drugs such as salicylates, aminoglycoside, antibiotics and loop diuretics have long been known

to adversely affect hearing (Hicks and Bacon, 1999). Another recent study of infants of smoking mothers showed an increase in arousal thresholds (i.e. the level of sound necessary to awake the infant) compared to those of non-smoking mothers (Lewis, 2000). There is also a genetic component to hearing loss. Genes have been linked with susceptibility to noise-induced hearing loss and as completion of the human genome project approaches more and more of these genes are being identified (Lewis, 2000; Willems, 2000).

In Ghana, studies have been conducted on the prevalence of hearing impairment among pre-school and school children (Brobby, 1988; Amedofu, Brobby and Ocansey, 1997; Amedofu, Opoku-Buabeng and Osei-Bagyina, 2003). Few studies have been conducted in Ghana on prevalence and causes of hearing loss among the general population. However, a thorough review of the literature has shown that there are no studies on prevalence and risk factors of hearing loss among children in Southern Ghana. This study therefore seeks to determine the prevalence and risk factors associated with hearing loss among children in a tertiary teaching hospital in Accra

## **1.2 PROBLEM STATEMENT**

It is well known that hearing and hearing loss are important for communication, learning, speech and language development of children. Language deficits may result in reduced academic achievements, while communication difficulties resulting from hearing loss often lead to social isolation and poor self-concept. Ultimately, these challenges may have an impact on vocational choices (ASHA (2014). The seriousness of the effects of hearing loss on the child's development is linked to how early the hearing loss occurs in the child's life. Consequently, early

identification of hearing problems and subsequent initiation or implementation of interventional strategies lessens the ultimate impact of hearing loss (ASHA, 2014).

From WHO statistics, approximately 278 million people worldwide are estimated to have a moderate or greater hearing loss, and about 20%–40% of children born with hearing loss may also present with significant additional disabilities that might prevent them from reaching their full potential (World Health Organization, 2010). Kennedy et al. (2006) reported that additional disabilities were present in 19.2% of their sample of 120 British children with hearing loss. A similar figure (of 18.6%) was reported by Berrettini et al. (2008) for an Italian sample of similar size. A review by Picard (2004) suggested higher prevalence of about 30–40%.

In Ghana, data available at the tertiary teaching hospital (Korle Bu Teaching Hospital) suggest a steady increase in pediatric cases. In 2010, a total number of 1105 children reported to the Hearing Assessment Centre for audiometric assessment. In 2011, this number grew by 51.95% resulting in a total of 1679 cases. The Centre again recorded a total of 1907 (72.58%) and 2028 (83.53%) cases in 2012 and 2013 respectively. The records available at the Centre showed that most of these cases were presented late and parents had no information on the condition and its causes. Another challenge is the associated high cost of management which is unaffordable to most parents. The economic, social, physical and psychological burden on both children and parents cannot be over emphasized.

Few studies have been done in Ghana on the prevalence and risk factors of hearing loss in children (Brobby, 1988; Amedofu, Brobby and Ocansey, 1997; Amedofu, Opoku-Buabeng and Osei-Bagyina, 2003). However, no study is currently available on the prevalence and risk factors

of hearing loss in children aged between 6 months to 10 years in Southern Ghana. Therefore there is a void in the literature on the prevalence and risk factors of hearing loss in Southern Ghana. The present study is therefore designed to fill the vacuum in the literature. In view of these challenges, this study was conducted to establish the prevalence and risk factors of hearing loss among children in Korle Bu Teaching Hospital.

### **1.3 SIGNIFICANCE OF THE STUDY**

Hearing loss is detrimental to human development at all ages. However, children are more affected as their development is impacted in several ways. By establishing the prevalence and risk factors of hearing loss in children, it is hoped that parents will be educated on how to avoid or minimize exposure of their children to hearing loss and its negative effects. The findings of this study will determine the prevalence of hearing loss at Ghana's foremost hospital. This would help stakeholders in the health care sector including policy makers to carve out policies and measures to reduce to the barest minimum the cases of hearing loss in children. The outcome of this study will also add up to the body of knowledge on issues of hearing loss in children in Ghana and to some extent the West Africa sub-region.

### **1.4 AIMS**

The aims of this research study were:

1. examining the prevalence of hearing loss in pediatric patients aged between 6 months to 10 years and referred to the KBTH Hearing Assessment Center
2. documenting the hearing status, possible maternal and infant risk factors for children evaluated for the first visit to the Center

## 1.5 OBJECTIVES OF THE STUDY

The specific objectives defined for achieving the desired goals of the study include:

1. determination of the prevalence of hearing impairment in children
2. identification of the predominant risk factors of hearing loss in children
3. identification of the degree of hearing loss in children
4. determination of associations between identified risk factors and hearing loss among children

## 1.6 RESEARCH QUESTIONS

The following research questions were posed:

**Research Question One:** *What are the predominant risk factors of hearing loss in children?*

**Research Question Two:** *What are the outcomes of the physiologic assessments in the children?*

**Research Question Three:** *What are the associations between the OAE results and demographics?*

**Research Question Four:** *What are the degrees and prevalence of hearing loss via VRA, play audiometry at the various test frequencies?*

**Research Question Five:** *What are the associations between hearing loss and demographics as measured via VRA and play audiometry assessments?*

**Research Question Six:** *What are the associations between hearing loss and tympanograms using VRA and play audiometry assessments?*

## 1.7 DEFINITION OF TERMS

**Audiogram:** It is a plot or a graph on which the result of pure tone audiometry are recorded.

**Cochlea:** The spiral organ of the labyrinth of the ear, which is concerned with the reception and analysis of sound.

**Conductive hearing loss (CHL)** results from interference with the mechanical transmission of sound through the external and middle ear, and is far more common in children; it can be congenital, as a consequence of anatomic abnormalities, but it can commonly be acquired following middle ear inflammatory pathologies.

**Decibel:** One tenth of a bell: a unit for comparing levels of power ratios (especially sound) on a logarithmic scale.

**Deafness:** A condition wherein the ability to detect certain frequencies of sound is completely or partially impaired.

**Hearing Impaired:** The term “hearing impaired” is a technically accurate description of someone who is hard of hearing or who has some hearing deficit.

**Hearing Loss:** Hearing loss is the total or partial inability to hear in one or both ears.

**Hertz:** The unit of frequency defined as the number of cycles per second of a periodic phenomenon. It is represented the symbol Hz

**Mastoiditis:** Inflammation of the mastoid air cell.

**Mixed hearing loss** involves a combination of conductive and sensorineural hearing loss.

**Otoscope:** An apparatus for examining the eardrum and the passage leading to it from the ear (external auditory meatus).

**Sensorineural hearing loss (SNHL)** results from failure to transduce vibrations to neural impulses in the cochlea, and can also be divided in congenital and acquired, and may otherwise be indicated on the basis of the time of onset as prenatal, neonatal or postnatal.

**Tympanometry:** A test that measure the function of the middle ear. Tympanometry works by varying the pressure within the ear canal and measuring the movement of the ear drum (the tympanic membrane).

## **CHAPTER TWO**

### **LITERATURE REVIEW**

#### **2.1 INTRODUCTION**

This Chapter presents a review of relevant literature on the subject. The literature is reviewed in accordance with the stated research questions of the study.

#### **2.2 HEARING LOSS**

According to Gelfand (2009), hearing impairment is a deviation or change for the worse in either auditory structure or auditory function, usually outside the range of normal. Hearing loss is a decrease in the hearing sense of an individual and can manifest as difficulty to hear sound, understand speech, and even deafness. Hearing loss can have several different causes and can occur instantaneously or over a period of time (Sataloff and Sataloff, 2006).

There are two main types of hearing loss differentiated by the specific anatomical areas of the ear affected. Conductive hearing loss (CHL) is caused by interruption of sound waves prior to entering the cochlea. This type of hearing loss does not involve the neurological components of hearing; instead sound is prevented from reaching the nerve cells of the ear. Conductive hearing loss can be caused by a buildup of wax in the ear canal, fluid in the in the middle ear, ruptured eardrum, or trauma to the ossicles in the middle ear. According to Sataloff and Sataloff (2006), sensorineural hearing loss (SNHL) results from the disruption of the neural components of hearing, either the nerve pathways or the sensory receptors in the cochlea (hair cells). Hearing

dysfunctions in children can be classified by type, degree, configuration, time of onset, aetiology, and finally, consequences on speech development (Kral and O'Donoghue, 2010).

### **2.3 PREVALENCE OF HEARING LOSS IN CHILDREN**

The prevalence of permanent hearing loss in children ranges from 1.2 to 6 per 1,000 live births worldwide. According to Cunningham and Cox (2003), Fortnum et al, (2001), Kemper and Downs (2000), and Mason and Herrmann (1998), this statistic rises by ten-fold in children who receive intensive care at birth due to prolonged exposure to noise, resulting in permanent hearing loss. The global prevalence of hearing impairment  $\geq 35$  dB HL among children less than 15 years of age was 1.2% (Global Burden of Disease, 2010). In developed countries, the incidence of congenital hearing loss was estimated at 2 to 4 per 1000 live births by White (2004). Kansherla et al., (2013) reported a hearing loss prevalence of 1.3 per 1000 among 8-year-olds in Atlanta (USA). In a cross sectional study conducted by Sue et al., (1998) involving 6166 children aged 6 to 19 years in the USA, 14.9% of them had low-frequency or high-frequency hearing loss at  $\geq 16$  dB HL, while 7.1% and 12.7% of them had hearing loss at 16 dB HL in the low- and high frequency regimes respectively. Most hearing loss was unilateral and low in severity (16 dB HL to 25 dB HL), and 10.8% were reported to have current hearing loss during the interview. The WHO reports a hearing loss prevalence of 11% among children of high income countries and 9% in Central/East Europe and Central Asia (WHO, 2012).

A retrospective cohort study conducted by Mytton and Mackenzie (2005) in Oldham in the UK reported a prevalence of permanent childhood hearing impairment in the non-Asian community (1.34/1000 live births) equal to published national rates (1.33/1000 live births). In Australia, congenital hearing loss of greater than 35 dB in both ears is reported to occur in 1.3 per 1,000

live births while acquired hearing loss occurs in 3.2 per 1,000 infants (Medical Services Advisory Committee, 2007). In developing countries, 798,000 out of 133 million annual live births are likely to have permanent congenital and early onset hearing loss (Olusanya et al., 2007),

Epidemiological data on early childhood (0-5 years) hearing impairment in developing countries is scarce but the information available is helpful (Bolajoko, 2008). Higher prevalence has been found in developing countries (Olusanya et al., 2006). In a study done in Nigeria, a prevalence of 13.9% of hearing loss was reported among pupils. The study also reported that 8.9% experienced mild hearing loss while 2.8% and 0.8% respectively, suffered from moderate and severe hearing loss (Olusanya et al., 2000). It is estimated that in countries below the Sahara, more than 1.2 million children aged between 5 to 14 years suffer from moderate to severe hearing loss in both ears. In a survey of hearing loss in some African countries, Sahi (2011) reported that about 2.5 to 3.5 children in 1000 suffered from severe to profound hearing loss in Kenya, Gambia and Tanzania, and about 7.5% and 4.1% of the school children aged between 5 and 15 years suffered varying degrees of hearing loss in South Africa and Swaziland respectively. The same reported indicated a higher prevalence of 9.0% in Sierra Leone for children of the same age group.

In Ghana, studies on hearing loss focused on the adults. Hence, there is very little information on prevalence of hearing impairment specific to children. In particular, Awuah et al. (2012) reported a 72.6% prevalence of hearing loss among out-patients both adult and children attending a hearing clinic. In a prospective study by Amedofu et al. (2006), a prevalence of 89.9% was reported among 6,428 patients with hearing problems. In a relatively older study involving 128 children aged between 1-5 years Amedofu et al., (1997) reported that 51.5% presented with

congenital SNHL (CSHL), while the remaining 48.5% suffered acquired SNH (ASHL) at a hearing clinic.

### **2.3.1 Impact of Hearing Loss in Children**

Sharma et al, (2005) found that the outcome of children with long term hearing loss was cross modal re-organization of the brain (re-assignment of auditory brain cells to other functions such as vision). This, according to the literature (Petitto *et al.*, 2000; Nishimura *et al.*, 2000; Lee *et al.*, 2001; Neville and Bavelier, 2002; Roder *et al.*, 2002) limited the neuroplastic adaptation of the cortex to auditory input. The impact on the auditory brain activity of a child with hearing loss deprived of sound for a long period is more significant than the impact for a child deprived for a short period. According to Thompson *et al.*, (2001) a bilateral severe or profound loss will cause significant effects on speech and language development, particularly if not detected early. Hearing loss also negatively affects the development of reading abilities because phonological processing, one of the fundamental prerequisites for reading takes place in the auditory areas of the brain (Werker and Tees, 2005).

Hearing loss in a child may also impact negatively on the child's family (Young and Tattersall, 2007). The negative effects of a child's hearing loss on the family are initially centered on the parent's response to the diagnosis, and adapting to the needs of the child (Feher-Prout, 1996). In the literature, several studies (Calderon and Greenberg, 1999; Feher-Prout, 1996; Lederberg and Golbach, 2002) have reported that the long-term impact on such families includes chronic parental grief and stress, emotional sensitivity, depression, feelings of disgrace and denial and the need to change their lives in order to meet the ongoing challenges.

It has been established that paediatric hearing loss adversely affects development of auditory skills (Sininger, 1999), and speech and language (Geers and Moog, 1994). A bilateral severe or profound loss will cause significant effects on speech and language development, particularly if not detected early (Erenberg et al, 1999; Geers and Moog, 1989; Thompson et al, 2001). Even a unilateral, mild or moderate hearing loss may impact on speech and language development (Moeller, 2000; Tharp and Bess, 1991; Yoshinaga-Itano et al, 1998).

Neural imaging has shown a strong relationship between phonological processing and reading skills (Strickland & Shanahan, 2004). Related to this, childhood hearing loss can also restrict writing ability and overall educational achievements (Wray, Hazlet, and Flexer, 1988). Trawler (2000) reported that 4,804 children with severe to profound hearing loss, on average, completed the twelfth grade with a third to fourth grade reading level (language levels of a nine to ten-year old child with normal hearing), and also had severely compromised mathematics ability. Behaviour is also adversely affected in some individuals (Prizant and Meyer, 1993).

## **2.4 CAUSES OF HEARING LOSS BY ANATOMICAL SITES**

### **2.4.1 External Auditory Canal**

Conductive hearing loss can simply be due to obstruction of the external auditory canal, as by an occluding ceruminous plug or by canal atresia. A 60 dB HL of hearing loss is caused by the latter. Per the proposal of Dai and Gan (2008), bone-conduction hearing aids should be provided in the first two to three months to newborn babies if both external auditory canals are atretic to enable normal development of hearing and speech

### 2.4.2 Middle Ear

According to Pau et al, (2010), closure of the Eustachian tube reduces the pressure in the tympanic cavity to 16 mm Ws (daPa) within two hours and thereby lessens the vibration of the tympanic membrane, mainly in the lower frequencies. If the Eustachian tube is blocked for months, amucoserous tympanic effusion arises, leading to conductive losses of up to 40 dB over the entire frequency range (Zahnert, 2011). Tubal blockage is more common in children than in adults because they have narrower Eustachian tubes and are more susceptible to middle ear infection. About 10% to 30% of all children have a tympanic effusion leading to conductive hearing loss at some time before the third birthday (Karosi and Sziklai, 2010). This problem is much more likely to occur in children with anatomical malformations of the palate and Eustachian tubes, including cleft lip, maxilla, and palate, and in those with Down or Turners syndrome. A tympanic effusion that has been present for three months or longer should be treated with tympanic drainage, as well as adenotomy if necessary in order to prevent a disturbance of speech development (Leitlinie, 2005).

Permanent conductive hearing loss is generally caused by chronic bacterial infection of the middle ear, affecting either the mucosa (otitis media mesotympanalis) or the bone (cholesteatoma). Hearing is impaired as a result of muffling of sound by granulations or cholesteatoma as well as enzymatic destruction or inflammatory fixation of the tympanic membrane and ossicular chain (Probst, 2008). The degree of hearing impairment (in the 30dB to 60dB range) is poorly correlated with the extent of tissue destruction because inflammatory tissue in the tympanic cavity can itself conduct acoustic vibrations and thus partly compensate for a pathological deficit. The treatment of choice is surgery, with the twin goals of eradicating infection and reconstructing the ossicular chain (Zahnert, 2011).

### **2.4.3 Congenital Causes (Pre-natal)**

Congenital hearing loss implies that the hearing loss is present at birth. It can include hereditary hearing loss or hearing loss due to other factors present either in utero (prenatal) or at birth. Genetic factors are thought to cause more than 50% of all incidents of congenital hearing loss in children (Canalis, 2000). Genetic hearing loss may be autosomal dominant, autosomal recessive, or X-linked (related to the sex chromosome).

In autosomal dominant hearing loss, one parent who carries the dominant gene for hearing loss and typically has a hearing loss and passes it on to the child. In this case there is at least a 50% probability that the child will also have a hearing loss. The probability is higher if both parents have the dominant gene (and typically both have a hearing loss) or if both grandparents on one side of the family have hearing loss due to genetic causes. At least if one parent usually has a hearing loss, there is prior expectation that the child may have a hearing loss (Canalis, 2000).

In autosomal recessive hearing loss, both parents who typically have normal hearing, carry a recessive gene. In this case the probability of the child having a hearing loss is 25%. Because both parents usually have normal hearing, and because no other family members have hearing loss, there is no prior expectation that the child may have a hearing loss (Dhooge, 2003).

In X-linked hearing loss, the mother carries the recessive trait for hearing loss on the sex chromosome and passes it on to males, but not to females. There are some genetic syndromes for which hearing loss is one of the known characteristics. These include Down syndrome (abnormality on a gene), Usher syndrome (autosomal recessive), Treacher Collins and syndrome (autosomal dominant), Crouzon syndrome (autosomal dominant), and Alport syndrome (X-linked).

#### **2.4.4 Peri-natal hearing loss**

Peri-natal hearing loss accounts for 5-10% of the cases of congenital hearing loss and include infections during pregnancy, such as rubella, cytomegalovirus, herpes or syphilis, toxins consumed by the mother during pregnancy or other conditions occurring at the time of birth or shortly thereafter. Premature babies also have an increased risk of becoming hard-of-hearing.

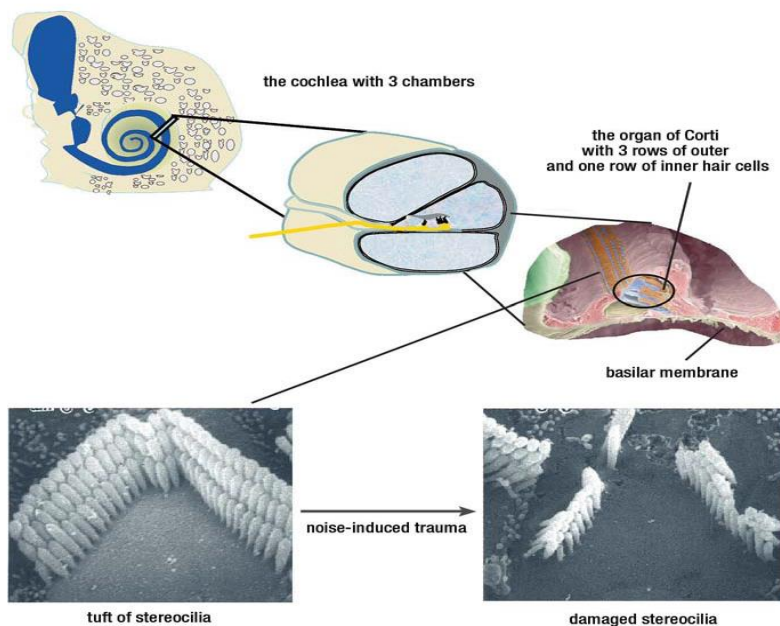
#### **2.4.5 Post-natal hearing loss**

Worldwide, 10% to 30% of hearing loss occurrences is due to postnatal causes. Most studies on the causes of hearing loss in NICU graduates have identified hypoxia to be a factor. The different definitions of hypoxia include apnoea, difficulty in breathing (Rovert *et al.*, 1996), a history of duration of ventilation (Borradori, 1997), a lower Apgar score at 5 minutes, or a more frequently documented oxygen saturation (PO<sub>2</sub>) of less than 50mm Hg. Bergman *et al.*, (1985) identified 36 children with SNHL of less than 55 dB bilaterally in their NICU follow-up programme, which included all infants of less than 1500 g birth weight or with neonatal seizures. Significant neonatal predictors of hearing loss in high-risk premature infants were prolonged respirator care, high serum bilirubin concentration, and hyponatremia. Childhood infections such as meningitis, encephalitis, and head injury can also cause hearing loss (Eavey *et al.*, 1995).

#### **2.4.6 Perceptual Hearing Impairment**

The organ of Corti is the functional unit that transduces perilymphatic vibrations into neural signals. Vibration of the basilar membrane leads to mechanical deflection of the stereocilia and thereby to electrolyte influx into the hair cells, causing depolarization. Motor- proteins in the cell walls of the outer hair cells bring about a non-linear amplification of this mechano-electrical transduction process in a particular dynamic range (Zahnert, 2011). Sensory hearing loss is due,

to dysfunction of the organ of Corti. This problem is most commonly located in the outer hair cells and manifests itself in such cases as a shift of the bone conduction threshold by up to 50 dB, suggesting a loss of nonlinear reinforcement (the so-called recruitment phenomenon), and impaired frequency selectivity (distortions)(Zahnert, 2011).



**Figure 2.1: Organ of Corti with outer and inner hair cells and the attached stereocilia before and after noise-induced trauma (chinchilla). Source: Zahnert, 2011**

#### 2.4.7 Permanent Sensorineural Hearing Loss in Childhood

The prevalence of congenital, bilateral, permanent SNHL of 35 dB or more is estimated at 1.2 per 1000 live births in Germany (Gross et al, 2000). About 25% of these cases were of genetic origin, while 18% are acquired, and 57% via indeterminate causes. Congenital hearing loss worsens between the ages of 2 to 5 years in 30% of affected children. Mild hearing loss in non-syndromic autosomal recessive hearing loss (the most common type) is often due to a genetic mutation that impairs the synthesis of the trans membrane proteins connexin 26 and 30, which in

turn affects the ion transport mechanism in the hair cells(gap junction protein). Whenever hearing impairment of genetic origin is suspected, connexin 26 and 30 mutations should be sought (Zahnert, 2011). Genetic hearing impairment of recessive inheritance is usually severe; it is usually due to a sporadic mutation, and therefore hard to diagnose (Ptok and Ptok, 2001). Infectious, toxic, and traumatic processes frequently cause acquired hearing impairment in the pre-natal, peri-natal or post-natal period and can also cause progressive or newly acquired hearing loss later in childhood.

#### **2.4.8 Hearing Impairment Due to Acute Toxic Damage to the Inner Ear**

Several medications have shown potential harmful effects on hearing drugs such as salicylates, aminoglycoside antibiotics, cytostatic agents, quinine and loop diuretics have long been known to adversely affect hearing (Hicks and Bacon, 1999). Medications used for prophylaxis of this disease include anti-inflammatory steroids such as prednisolone and inhaled bronchodilators such as the glucocorticoids. Axelsson et al., (1996) showed that inflammatory tissue alterations (elicited by cellular damage, tissue hypoxia and ischemia) occur in various structures of the cochlea following noise exposure. These bacterial and viral toxins reach the inner ear by way of the membrane of the round window, the cerebrospinal fluid, or the bloodstream and irreversibly damage the hair cells. The risk of hair-cell damage from medications can be reduced by careful monitoring of serum concentrations (Feldman, 2006). Bacterial toxins and inflammatory mediators called forth by viral infection can also have a toxic effect on the inner ear (labyrinthitis). Systemic viral infections (mumps, measles, rubella, cytomegalovirus and HIV) reach the labyrinth by way of the bloodstream (Probst, 2008). A synopsis of the causes and clinical features of hearing impairment, with differential diagnoses for each hearing impairment syndrome is illustrated in Table 2.1.

**Table 2.1: Synopsis of causes, clinical features of hearing loss, and differential diagnoses**

Conductive Hearing Loss	
Cause(s)	Acoustic-mechanical disturbance of sound conduction in the external auditory canal, across the tympanic membrane, or in the ossicular chain
Clinical features	If the cause is in the external auditory canal: reduced sound intensity. If the cause is in the tympanic membrane or ossicular chain: altered sound frequency and intensity
Differential Diagnosis	<b>Acute:</b> blockage by cerumen, tubular catarrh, tympanic effusion, traumatic eardrum perforation, acute otitis media or externa <b>Permanent:</b> Canal stenosis /atresia, defect of eardrum or ossicular chain due to chronic purulent infection of the mucosa, cholesteatoma, malformation, otosclerosis, tympanosclerosis
Audiological testing	Tuning-fork, pure-tone audiogram, impedance
Sensorineural Hearing Loss	
Cause(s)	Dysfunction of the hair cells or their synaptic connections to the cochlear nerve; if the outer hair cells are affected, loss of cochlear amplification and that of recruitment of intermediate intensities, blurring of frequency resolution, reduction of temporal resolution
Clinical features	Loss of intensity and dynamics: soft noises or speech may be perceived as either too soft or too loud, often, distorted perception
Differential Diagnosis	Acute: idiopathic sudden sensorineural hearing loss, acute noise-induced trauma blast trauma, explosion trauma, bacterial/viral labyrinthitis Hereditary/permanent: hereditary hearing impairment, presbycusis, noise induced hearing impairment, toxic, idiopathic chronic progressive hearing, impairment, drug side effects, lasting sequelae of infections
Audiological testing	Tuning-fork test: Pure-tone audiogram, speech audiogram, otoacoustic emissions

**Table 2.1: Synopsis of causes, clinical features of hearing loss, and differential diagnoses**

Neural Hearing Loss	
Cause(s)	Cochlear nerve dysfunction: delayed impulse conduction, disturbed neural encoding of the acoustic signal
Clinical features	Similar to sensory hearing loss, but usually unilateral; speech perception worse than tone perception
Differential Diagnosis	Acoustic neuroma (= vestibular schwannoma), other tumors of the petrous bone or cerebellopontine angle (meningioma, chordoma, chondrosarcoma), compression syndrome
Audiological testing	Pure-tone audiogram: speech audiogram, supraliminal tests, auditory fatigue tests, electric response audiometry
Central hearing loss	
Cause(s)	Dysfunction of the auditory pathway or auditory cortex (processing of bilateral auditory stimuli, synchronization, signal modulation, recognition, noise)
Clinical features	There may be no disturbance of tone perception: impaired rapid speech processing, impairment of poor sound localization
Differential Diagnosis	Infarction: hemorrhage, tumor, multiple sclerosis, auditory processing disorder
Audiological testing	Pure-tone audiogram, speech audiogram, supraliminal tests, auditory fatigue tests, electric response audiometry.

**Source:** Zahnert, 2011

## 2.5 RISK FACTORS OF HEARING LOSS

### 2.5.1 Infections

Hearing loss has been associated with various diseases such as meningitis, mumps, measles, and jaundice. In most bacterial infections, damage to the ear results from infiltration of the organism via the internal meatus. Viral diseases may cause histopathological damage to the organ of Corti, damage or complete destruction of the stria vascularis and tectorial membrane, and atrophy or destruction of the neural pathways (Northern and Downs, 1991). These diseases can affect

children of all ages, including foetuses, newborn and pre-term infants. Studies on premature babies (Marlow et al, 2000), especially those who spent time in a NICUs (Billings and Kenna, 1999), showed an increased risk of sensorineural hearing loss in these infants compared to full-term infants. Maternal diseases during pregnancy may also pose a risk of auditory damage to the unborn child. Congenital infections such as syphilis are known to cause several central nervous system abnormalities but often result in foetal death and miscarriage. A recent study found hearing defects in children born of mothers who had suffered from symptomatic rubella during their pregnancy (Niedzielska et al., 2000).

### **2.5.2 Physical Barriers**

Hearing loss may also be associated with a build-up of ear wax in the external auditory meatus. Ear wax is produced by the apocrine and sebaceous glands of the ear canal and is normally removed from the canal by migratory movements of the epithelium. However, some people may produce excessive ear wax or have an inadequate cleaning mechanism. Such accumulation of wax blocks the ear canal and can subsequently cause hearing loss (Northern and Downs, 1991)

### **2.5.3 Low Birth Weight**

Low birth weight has also been associated with hearing loss in children (Northern and Downs, 1991). Other factors influencing birth weight may also directly contribute to hearing loss, including drinking and smoking by the mother during pregnancy. A recent study of infants of smoking mothers showed an increase in arousal thresholds (i.e. the level of sound necessary to awake the infant) of those children with smoking mothers compared to those of non-smoking mothers (Lewis, 2000).

#### **2.5.4 Genetics**

There is also a genetic component to hearing loss. Genes have been linked with susceptibility to noise-induced hearing loss and as (Willems, 2000) completion of the human genome project approaches more and more of these genes are being identified (Lewis, 2000).

### **2.6 AUDIOLOGICAL ASSESSMENT**

Advancements in technology have forged a new era in the diagnosis and treatment of paediatric hearing loss, which have created unprecedented potential for listening and spoken language for children with hearing loss (Yoshinaga-Itano, 2004). Current attempts to limit the burden of hearing impairment conditions through primary prevention are inadequate and sometimes ineffective (Smith, 2003). About 50% of the burden of hearing impairment is believed to be preventable or avoidable (Smith, 2003). Before the advent of new hearing technology, such as cochlear implants, congenital hearing loss in a child prevented sound stimulation from reaching auditory brain centre, resulting in lack of auditory brain development, particularly if not detected early (Gilley, Sharma, and Dorman, 2008). Auditory stimulation influences the organization and maturation of auditory brain pathways, allowing a child to make meaning out of what they hear (Boothroyd, 1997; Chermack, Bellis, and Musiek, 2007).

#### **2.6.1 Otoacoustic Emissions**

Otoacoustic emissions (OAEs) are sounds given off by the inner ear when the cochlea is stimulated by a sound. 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 (ASHA, 2010). Otoacoustic emission is a test that checks the status of the outer hair cell. Because this test does not rely on a person's

response, the person being tested can be sound asleep during testing (CDC, 2010). 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 (ASHA, 2010). An otoacoustic emission (OAE) is a low-level sound emitted by the cochlea either spontaneously or evoked by an auditory stimulus.

Specifically, OAEs provide information related to the function of the outer hair cells (OHC) (Stach, 2003). Over the past 20 years, their use in routine audiological assessments has increased significantly. Today, OAEs are used commonly in the audiological assessment of difficult to test patients, such as persons who cannot or will not volunteer reliable behavioral responses. OAEs are routinely used in the pediatric population to verify behavioral responses and obtain additional frequency-specific information. In addition, they are routinely used in newborn hearing screening programs across the world. OAEs have many benefits: they are easy to obtain, non-invasive, and provide reliable information regarding cochlear status in a relatively short time (AAA, 2011).

The presence of OAEs is consistent with normal or near-normal hearing thresholds in a given frequency region. Although relations exist between OAEs and behavioral thresholds (Gorga, Neely, and Dorn, 2002; Gorga Stover, and Neely, 1996; Martin, Ohlms, Franklin, Harris, and Lonsbury-Martin, 1990) and there has been improvement in strategies for predicting thresholds using OAEs (Boege and Janssen, 2002; Gorga, Neely, Dorn and Hoover, 2003), variability among individuals suggests that caution should be exercised when attempting to predict behavioral thresholds from OAEs.

### **2.6.2 Behavioural Audiometry Evaluation**

Behavioral audiometry evaluations test the function of all parts of the ear and how a person responds to sound overall. The person being tested must be awake and actively respond to sounds heard during the test. The high inter- and intra-subject variability in responses has not completely ruled out behavioral observation procedures for estimating hearing thresholds in infants 4 months of age and younger but supplemented by other measures of physiological tests (Hicks, Tharpe, and Ashmead, 2000; Thompson and Weber, 1974). There is some benefit, however, in observing auditory behaviors of young infants, because behavioral responses provide information on how infants respond to auditory input. Any behavioral observation assessment is intended for corroboration of patient/caregiver report of the child's auditory behavior rather than for threshold estimation.

According to ASHA (2004), audiologic assessment of infants and young children includes a thorough case history, otoscopy, and behavioral and physiologic measures. Children undergo rapid sensory, motor, and cognitive development, and because some children present with multiple developmental problems, it is vital that assessment tools are appropriate for the neuro-developmental state of the young child. In addition to the assessment of peripheral hearing status, it is essential for audiologists working with infants and young children to consider the functional implications of hearing loss. Assessments of speech perception ability, and screening for communication skills, cognitive development and social-emotional status should be included as part of the pediatric test battery. Such assessments and screenings are consistent with the objective of formulating recommendations and additional referrals as needed (ASHA, 2004).

Ear-specific assessment is the goal for both behavioral and physiologic procedures. Determining hearing sensitivity for each ear is important for establishing supportive evidence for medical/surgical diagnosis and treatment, selecting amplification when appropriate, establishing baseline function, and monitoring auditory status when progressive, fluctuating, or late-onset hearing loss is suspected. Acoustic stimuli used for behavioral assessment should provide frequency-specific information regarding auditory sensitivity. Therefore, responses to pure tones, FM tones, or narrow bands of noise should be obtained in behavioral testing of children regardless of the response levels obtained to broadband signals (Pediatric Working Group, 1996). Several audiologic assessment procedures require the insertion of a probe into the external auditory canal. Thus, a visual inspection of the outer ear canal should be conducted to verify that there is no contraindication to placing a probe in the ear canal (e.g., drainage, foreign objects, occluding cerumen, atretic canal) (ASHA, 2004).

Behavioral assessment of hearing sensitivity in children is complicated by developmental and maturational factors. Several studies have shown that once an infant reaches a developmental age of 5–6 months, it is possible to elicit reliable conditioned auditory responses using an operant, visually reinforced behavioral response technique (Primus and Thompson, 1985; Thompson and Wilson, 1984; Thompson, Wilson, and Moore, 1979; Widen, 1993; Wilson, 1978). Typically, developing children as young as 5 months of age may be conditioned to produce a motor response contingent on the presence of an auditory stimulus (Wilson and Thompson, 1984). The behaviour, usually a head turn, is reinforced by an appealing visual display.

More recent studies confirm that frequency-specific thresholds may be obtained from infants, at developmental levels of 5–6 months, enabling accurate evaluation of hearing sensitivity

regardless of type, degree, or audiometric configuration (Bernstein and Gravel, 1990; Gravel and Wallace, 1999; Widen et al., 2000). The basic paradigm used in the tangible reinforcement operant conditioning audiometry (TROCA) or visually reinforced operant conditioning audiometry (VROCA) procedure involves a bar press response coupled with either tangible or visual reinforcement. TROCA or VROCA has been shown to be most effective with children between 2 and 4 years of age developmentally, and also is effective with children with mental challenges (Diefendorf, 1988; Wilson and Thompson, 1984). In conditioned play audiometry (CPA), children learn to engage in an activity each time they hear the test signal. When children are taught to perform play audiometry, it is usually not difficult to select a response behavior that they are capable of performing. The challenge in play audiometry is teaching the child to wait, listen, and respond with the play activity only when the auditory signal is audible. From 25 to 30 months, CPA is sometimes possible within the time constraints of clinical activity (Thompson and Vethivelu, 1989)

## **2.7 RESEARCH GAP**

Few studies on the prevalence and causes of hearing loss in adult and pediatric populations have been reported in Ghana. Currently, no research study has been performed or is available on the prevalence and risk factors of hearing loss specific to children aged between 6 months to 10 years in Ghana. There is therefore a gap in the literature as well as in inadequacy in respect of research on the prevalence, and risk factors of hearing loss in pediatric patients in Ghana. No research has also been conducted on the impact of hearing loss on pediatric patients and their families. On these bases, this research study was conducted to address some of the research gaps by establishing the prevalence and identifying risk factors of hearing loss among pediatric patients in a tertiary hospital in Accra, Ghana.

## **CHAPTER THREE**

### **METHODOLOGY**

#### **3.1 INTRODUCTION**

This Chapter presents the various methods, techniques and tools used in data collection. It describes the study design, study location, variables to be measured sampling plan and analytical tools for data analysis. The ethical considerations of the study are also presented herein.

#### **3.2 STUDY DESIGN**

In health and social research studies, quantitative design is described as the best method for collecting large information in social and health research and is regarded as an efficient way for investigating health issues in the communities as well as the analysis of data collected from a population, or a representative subset, at one specific point in time .Cross-sectional surveys are studies (neither longitudinal nor experimental) which allow for one time collection of data and may be used to describe some features of a population, such as prevalence of an illness, or they may support inferences of cause and effect. They are also often used to assess the prevalence of acute or chronic conditions, or to answer questions about the causes of disease or the results of intervention. This design is fast, cheap and less complex and does not require much expertise. For these reasons, a descriptive quantitative design with a cross-sectional survey was employed for this study.

#### **3.3 STUDY SITE**

The study was conducted at the Hearing Assessment Centre (HAC) of the Korle Bu Teaching Hospital (KBTH), the premier and leading national referral hospital in Ghana. The study site was

chosen because the Centre is better equipped than other hospitals to manage all audiological referral cases.

### **3.4 STUDY POPULATION**

According to Cardwell (1999), a population is a group of people who constitute the focus of a research study and to whom the results would be applied. The target population of this study consisted of paediatric clients aged between 6 months to 10 years reporting at the Hearing Assessment Centre for hearing evaluations. This age group was chosen because available information at the HAC shows that about 92% of paediatric cases presenting with hearing challenges fall within the age bracket. Furthermore, anecdotal evidence from Hearing Assessment Centre has also shown that treatment of hearing loss and its outcome are more difficult at 10 years and above, which is late though to begin treatment.

### **3.5 SAMPLE SIZE AND SAMPLING TECHNIQUE**

Non-probability purposive sampling techniques are usually applied for investigating units or systems based on the peculiar characteristics of a population or group of participants in relation to a study. For this reason, a non-probability purposive sampling method was adopted to select participants for the study for which limited work on prevalence and risk factors of hearing loss in children has been either done or reported in Ghana. The sample size was estimated via Eqn. (3.1)

$$N = Z^2 \left( \frac{pq}{E^2} \right) \quad 3.1$$

where

$N$ =minimum sample size,  $Z$ = z-score for 95% confidence interval,

$E$ = margin of error of the study (0.1)

$p$ =proportion of people with hearing loss in the population (0.5)

$q$ =proportion of people without hearing loss in the population (0.5)

Using the assumed values, a minimum sample size of 96 participants was required for the study.

### **3.6 INCLUSION AND EXCLUSION CRITERIA**

#### **3.6.1 Inclusion Criteria**

All subjects between 6 months and 10 years whose parents consented to the study were included.

Those children within the age bracket whose parents did not consent to the study were excluded.

#### **3.6.2 Exclusion Criteria**

The following criteria were used:

- Children aged below 6 months and above 10 years
- Children whose parents declined consent

### **3.7 INSTRUMENTATION**

The different types of tests instrumentation used in otoscopy, tympanometry, OAE, pure-tone audiometry and questionnaire assessments employed are described below.

#### **3.7.1 Otoscope**

A Welch Allyn otoscope was used to examine the external auditory canal for wax impaction, discharging ears and perforation.

#### **3.7.2 Tympanometer**

A GSI TYMPSTAR with a low frequency probe tone of 226Hz was used to measure the tympanic membrane mobility and determine middle ear status. This device measures the peak

compliance and peak pressure of the ear at the probe tone frequency. The probe has four tubes for insertion into the ear canal and consists of probe tone loud speaker, monitor microphone, pressure pump manometer and ipsilateral acoustic reflex loud speaker. These tubes measure acoustic admittance of the ear.

### 3.7.3 Otoacoustic Emission

Otoacoustic emission (OAE) is sound generated by the cochlea in the ear and is measured using microphones placed in the auditory canal (Gelfand 2009). Clinically, the two types of OAEs are the Transient Evoked OAE (TEOAE) and Distorted-Product OAE (DPOAEs). In particular, TEOAEs are produced by acoustic stimuli such as a click or tone burst following which a response occurs after a brief delay in time. It is recorded at a frequency range of 500 to 4000 Hz. They are measured following the presentation of transient stimulus and are accomplished using time synchronous averaging and often evaluated in terms of amplitude, percentage reproducibility, and amplitude/noise or signal-to noise ratio (SNR) (Prieve and Fitzgerald, 2002). The DPOAEs sometimes referred to as acoustic distortion products (ADP) are one result of non-linear behaviour in the cochlear. According to Prieve and Fitzgerald, (2002), they result from inter-modulation distortion products that undergo reverse transduction via the middle ear and are converted to measurable acoustic energy in the ear canal. They are produced when two pure tone stimuli at frequency  $f_1$  and  $f_2$  are presented to the ear simultaneously. DPOAE occurs at expected primary frequencies and can be simultaneously determined via the mathematical expressions

$$f_2 - f_1, 2f_1 - f_2, 3f_1 - 2f_2, 2f_2 - f_1 \quad (3.1)$$

OAE is measured in quiet environment, and does not require behavioural response and sedation. A probe is placed in the ear canal which contains one or more miniature loud speakers to generate stimulus and a microphone to record the sound in the ear. The AuDx PRO Bio-Logic

System was used in the study. In this study the DPOAE preset test protocol was used for determining hearing sensitivity which resulted in an automated pass or refer. Table 3.1 indicates the automated OAE test protocols and test parameters.

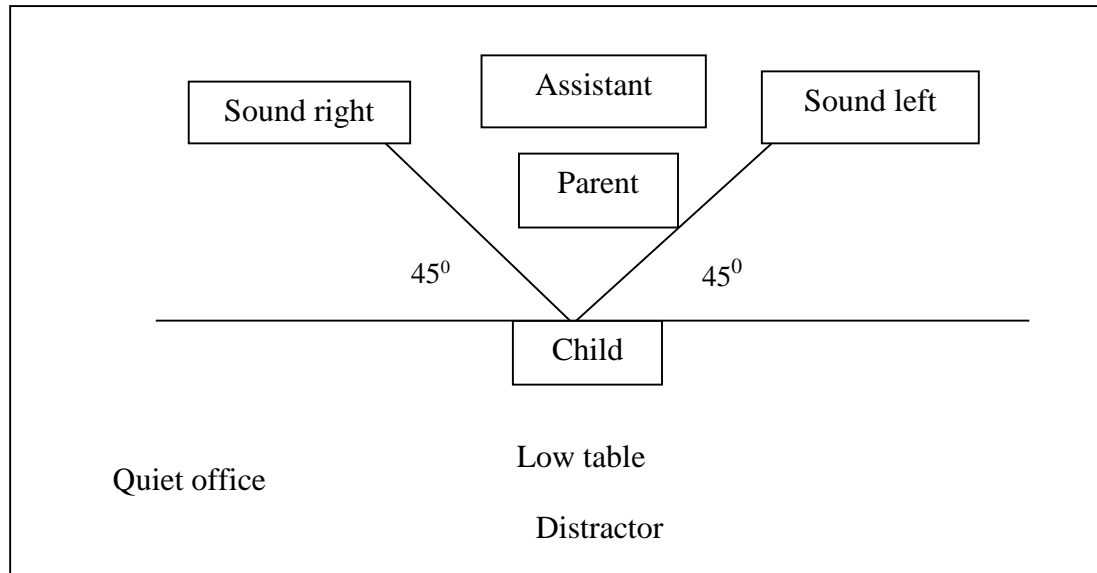
**Table 3.1: OAE test protocol and setup parameters**

	Level	Sound pressure levels
Recording/Stimulus Parameters	L1	65 dB SPL
	L2	55 dB SPL
	f2/f1 ratio	1.2
	Sample size	1024 points (20 ms sample)
Stopping rules	Minimum DP amplitude	-5 dB SPL
	Minimum DP-NF amplitude	8 dB SPL
	or Minimum Noise Floor Amplitude	-17 dB SPL
	Or Time Out	20 sec 20 sec (3 kHz and below)
Pass/Refer Criteria	Minimum DP amplitude:	
	F2 frequencies DP amplitude for pass (dB SPL)	
	5014	-6
	3983	-5
	2999	-8
	2015	-7
	Number of frequencies to pass test	3
	Minimum DP-NF amplitude	6Db
Number of frequencies for Pass	2-5 kHz, 3 of 4 for Pass	

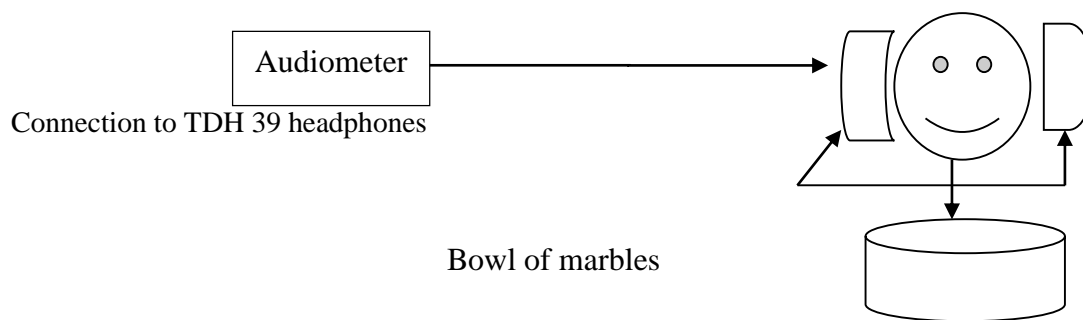
### 3.7.4 Audiometer

An audiometer is a device used for evaluating hearing loss. Behavioural assessment was done using the Interacoustics Paediatric PA5 and Interacoustics AD229e audiometers at test frequencies of 500Hz, 1000Hz and 2000Hz to determine the minimum response level at which a

child could respond to sound stimuli. The Interacoustics AD229e and Telephonic TDH-39 supra-aural earphone were calibrated for visual reinforcement audiometry (VRA) and play audiometry test (Figure 3.1-3.3).



**Fig. 3.1: Schematic diagram of distraction test**



**Fig. 3.2: Schematic flowchart of play audiometry**

The hand-held Interacoustics Paediatric PA5 audiometer was used for distraction tests on 16 respondents who could not perform VRA (with insert ear phones) and play audiometry (with TDH 39 supra-aural earphone), while the Interacoustics AD229e audiometer was used on 44 respondents aged 6 months to 30 and above months for VRA, and 36 respondents play audiometry 3 years to ten 10 years.

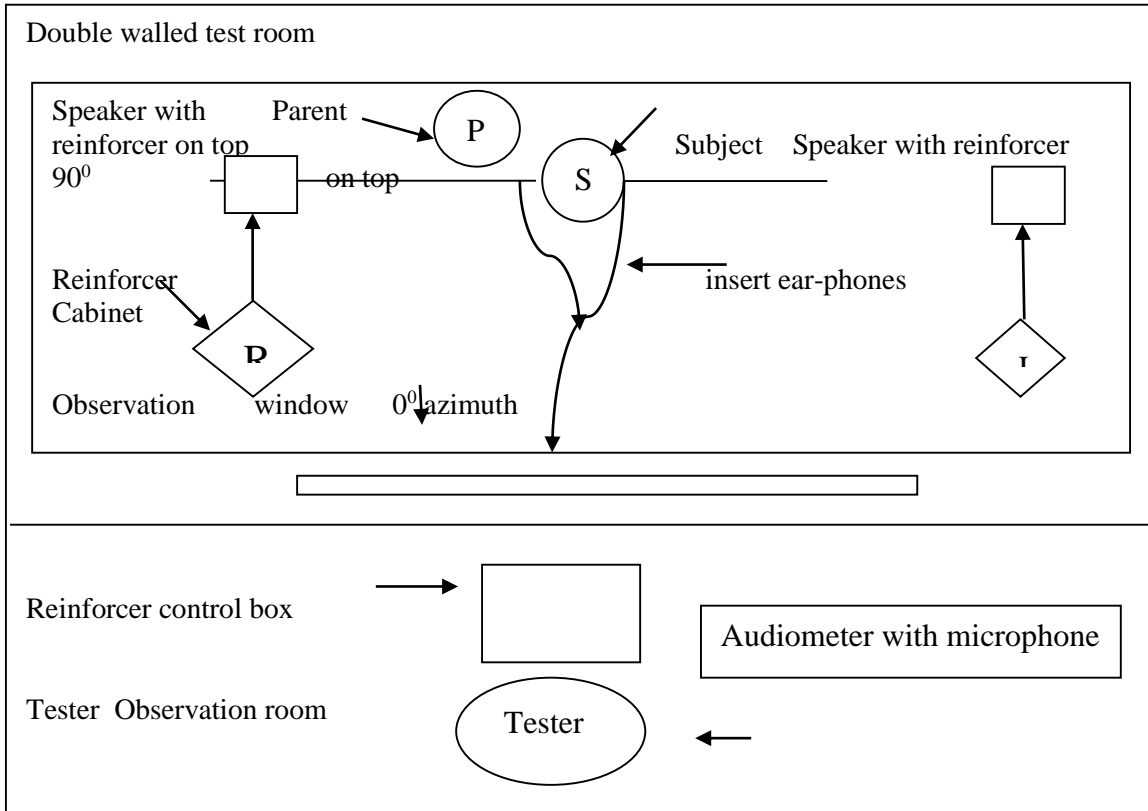


Fig.3.3: Block diagram showing VRA set-up

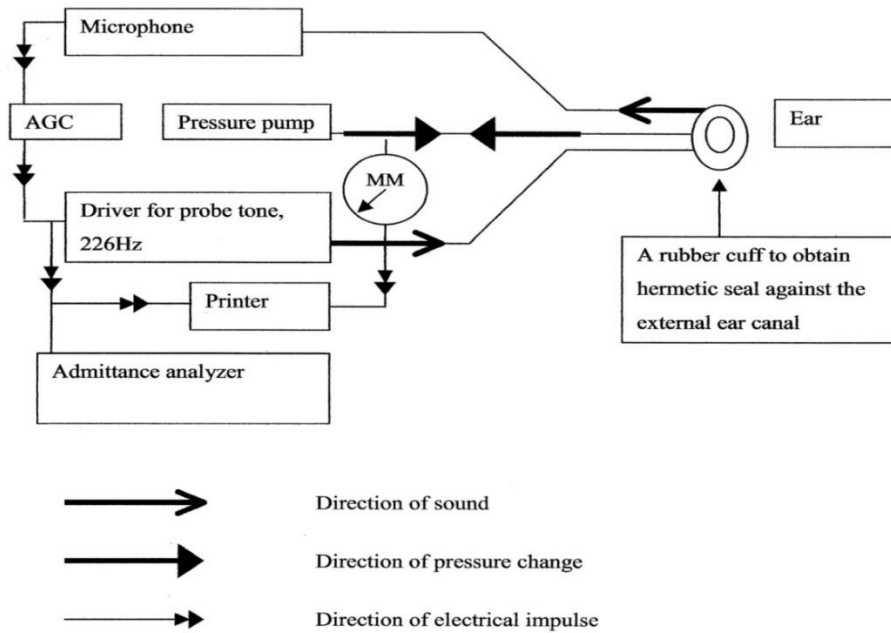


Fig. 3.4: Schematic diagram of tympanometer

### **3.8 PROCEDURE FOR DATA COLLECTION**

The procedure for data collection included questionnaire, physical examination of the ear lobe, otoscopy, tympanometry, otoacoustic emissions, and behavioural (distraction test, VRA and play audiometry) assessment with the use of an audiometer. Data was collected from 96 participants using a self-administered and structured questionnaire comprising close-ended and open ended questions. Parents were given time to answer the questions on the questionnaire after the purpose of the study had been adequately explained and informed consent signed.

#### **3.8.1 Questionnaire and Physical Examination**

The questionnaire was extracted from Northern and Downs (Hearing in Children 1991) modified to suit the objectives of the study. The questionnaire consisted of information on mothers and child demographic data (coded name, gender, age) with particular reference to when hearing loss was suspected. Other aspects included family history on hearing loss, maternal factors, delivery or labour history, infant new born history and infant childhood history. Otoscopy was performed to identify impacted wax in the ear canal, discharging ear, foreign body.

#### **3.8.2 Objective Assessment Techniques**

##### **3.8.2.1 Tympanometry**

Middle ear pathologies among the paediatric population were examined with 226 Hz probe tone tympanometry. Jerger's (1972) classifications were used to classify the types of tympanograms into Type A, B, C, and As. Type A tympanogram was consistent with normal middle ear pressure and defined by ear canal volume of 0.2-1.8ml, peak pressure between -150 +100 daPa and a peak compliance of 0.2ml-1.8ml. Types B, C, and As tympanograms represents "no middle ear pressure", "negative middle ear pressure" and shallow peak compliance" respectively.

### **3.8.2.2 Otoacoustic Emission**

The status of outer hair cells of participants was assessed with the distortion product otoacoustic emissions test (DPOAE). Test outcome were categorized into a pass or refer based on fore mentioned criteria.

### **3.8.2.3 Behavioural Assessment**

Behavioural assessments were used to assess participant's hearing. The distraction test, VRA with insert ear phones and play audiometry with supra-aural ear phones were used in the assessment.

### **3.8.2.4 Distraction Test**

This test was used to assess subjects who could not be tested with VRA. The Interacoustics PA5 was used for the test. The test was done in a quiet room with an average ambient noise level of  $37.1 \pm 4.0$  dB. A tester, distractor and parent of the child were involved in the process. The distractor engaged the child with various activities while the tester simultaneously presented narrow band noise within 2-3 seconds with the PA5 audiometer at various intensities at a distance of 1 m from the subject. The procedure was repeated until consistent results were obtained. Test results were categorized into "response/pass" and "no response/fail".

### **3.8.2.5 Visual Reinforcement Audiometry**

Visual reinforcement audiometry was administered to children between 6 months to 2.52 years in a sound treated booth. The test was also applicable to children more than 2.5 years with development delay. This procedure allowed for hearing thresholds to be determined with the presentation of a sound stimulus. Randomized frequency-modulated pure tones at 500, 1000, and

2000 Hz was presented through insert ear phones calibrated to an Interacoustics AD299e audiometer. Participants sat on an adult's (parent) lap on a chair facing the observation window at the calibration mark. The following instruction was given to the adult after placing ear phones in the child's ear:

*“Sounds are going to be presented through the ear phones and my job is to get the child to look for the sound. Your job is just to sit on the chair. Please do not help locate the moving toys”*

In the observation room, stimulus was initially presented through the right ear phone at the same time activating the reinforcer. This was intended to condition the child to localize the sound stimulus. The procedure was repeated to the left ear. The preliminary conditioning stimulus was a 1 kHz frequency modulated sound at 60 dBHL at maximum intensity (100 dB). The intensity of conditioning/test stimulus was increased by 10 dB steps until reliable responses were obtained. If a no response was obtained at the initial frequency, the procedure was repeated at 2 kHz, and 500 Hz. No responses were confirmed with live voicing presented via the ear phones at maximum intensity. Findings were appropriately marked on an audiogram.

Successful conditioning was followed by a testing phase to obtain minimum response levels. Responses to sound stimulus were followed by an activation of the reinforcer. The conventional ‘Up 10 down 5’ was used to establish minimum response levels. Minimum response levels were marked appropriately on an audiogram.

### 3.8.2.6 Play Audiometry

This test was administered to children aged between 3.5-10 years, successfully conditioned to a play response to sound stimuli. The test was conducted in a sound treated booth with TDH 39 ear phones to obtain minimum response levels. A portable Interacoustics AD 299e calibrated to TDH 39 supra aural earphones were utilized. Subjects sat on a chair facing away from the portable audiometer (with ear phones) in the sound treated booth. A container of balls was given to the test child while their parents were instructed to sit near the child with an empty box. The following instructions were given to participants:

*“When you hear a beep in the earphone put the ball in the empty box”.*

The preliminary phase aimed to condition the child to respond to sound by putting a ball in the box. The audiometer was set at 55 dBHL with a 1 kHz frequency modulated test tone in the right earphone. The child was conditioned by an assistant by helping the child to hold the response object (ball) to the right earphone while presenting the test stimulus to the right ear for about 2 seconds. In the course of presenting the test tone, the child’s hand was moved to the box to drop the response object (ball). The process was repeated until the child could respond to the sound stimulus with the ball independently. The intensity of sound (test) stimulus was increased in 10 dB steps to for children who did not respond at the initial level. A no response at maximum output was followed by a presentation of sound intensity at maximum output at 1 kHz, 2k and 500 Hz. No responses were marked appropriately on an audiogram. The procedure was repeated for the left ear. Successful conditioning was followed by a testing phase to obtain minimum response levels. The conventional ‘Up 10 down 5’ was used to established minimum response levels which were marked appropriately on an audiogram. The process was also repeated for the left ear.

At the study site, responses at 25 dB or better are categorized as normal for VRA and play audiometry hearing tests. The validity of each test was ranked by the audiologist on a scale of 1 to 5 (1= poor validity, 5= excellent validity) based on the child's state (e.g., cooperativeness, interest in reinforcers). Auditory assessment lasted for 3 months. Sessions with validity rankings of 1 (non-reliable) were deferred for re-testing in the subsequent week.

### **3.9 INFECTION CONTROL**

The following measures were taken to avoid cross infection

- Disposable ER 3A insert earphones were utilized in VRA testing.
- The Germ-X hand sanitizing wipes were used to clean the TDH 39 supra-aural ear phones before and after testing a participant with the play audiometry.
- Clean probe tips were utilized in assessing each participant with the OAE test and tympanometer.

### **3.10 DATA MANAGEMENT PLAN**

The quantitative data generated from the study was coded and entered into the Statistical Package for Social Sciences (SPSS) version 20.0 Software.

### **3.11 DATA ANALYSIS**

The following criteria were used in analyzing data: A response to narrow band noise, warble tone and speech noise at any level was denoted as a pass while the converse was a fail for distraction test. In addition, hearing loss was determined by a minimum response level < 25 dB for VRA and play audiometry tests. Audiograms are often classified by categories based on the degree of hearing loss. The frequencies used for this purpose are usually 500, 1000 and 2000 Hz, (Katz et

al., 2002). Northern and Downs (2002) suggest using 15 dBHL as the upper limit for normal hearing for children between 2 and 18 years of age and a higher limit for adults. However, the KBTH upper limit (>25 dB) was adopted in classifying hearing loss into degrees (Table 3.2).

**Table 3.2: Classification of hearing loss**

Hearing level (dB)	Classification
0 to 25	Normal hearing
26 to 40	Mild hearing loss
41 to 55	Moderate hearing loss
56 to 70	Moderately severe hearing loss
71 to 90	Severe hearing loss
> 90	Profound hearing loss

The statistical software known as a Statistical Package for Social Sciences (SPSS) (version 20.0) was used to process and analyze Data obtained from the study. Data was summarized as frequencies, percentages means and standard deviations. The Pearson's chi-square was used to test for significant associations at 0.05 confidence level.

### **3.12 ETHICAL CONSIDERATIONS**

Ethical approval was sought from the Ethics and Protocol Review Committee of the School of Biomedical and Allied Health Sciences and Permission requested from the authorities of the Hearing Assessment Centre. Consent to participate in the research was sought from parents/guardians of the participants. In accordance with ethical considerations, all information obtained from participants was treated as confidential. The purpose of the study was fully explained to both parents and children were made aware of the fact that the study was voluntary and they were free to opt out at any time without any effect to their normal care. The researcher

acknowledged that this research would cause some psychological discomfort, during the answering of some parts of the questionnaire. However, the level of psychological distress would be minimal. The study is envisaged, to be beneficial to the study population, health workers, physicians and the general public. No compensation was given to persons with hearing loss for participating in the research; however their inputs were recognized and appreciated.

### **3.13 DISSEMINATION OF RESULTS**

The outcome of the study will be disseminated via submission of an MSc Audiology dissertation to the University of Ghana, School of Biomedical and Allied Health Sciences. The results will be published in a peer reviewed journal.

## CHAPTER FOUR

### RESULTS

#### 4.1 INTRODUCTION

This Chapter presents the results of the study. The results are presented in accordance with the stated study objectives and research questions. The results are summarized as means and standard deviations for continuous variables and as frequencies and percentages for categorical variables. The results are presented in charts and tables where appropriate.

#### 4.2 DEMOGRAPHIC CHARACTERISTICS

##### 4.2.1 Age Distributions of Children and Mothers

The gender and age distributions of the 96 children and their mothers surveyed in the study are presented in Table 4.1.

**Table 4.1: Age distributions of children and mothers**

		Frequency	Percent, %
<b>Gender</b>	Male	53	55.2
	Female	43	44.8
Age group (years)			
<b>Children</b>	0.5 – 3.5	78	81.3
	3.6 – 6.5	13	13.5
	6.6 – 10.0	5	5.2
	Total	96	100
<b>Mothers</b>	20 – 24	8	8.3
	25 – 29	20	20.8
	30 – 34	29	30.2
	35 – 39	35	36.5
	40 – 43	4	4.2
	Total	96	100.0

There were more male children ( $n=53$ , 55.2%) than females ( $n=43$ , 44.8%). The calculated mean ages of the children and mothers were  $2.74 \pm 2.01$  years and  $32.3 \pm 4.92$  years respectively. Majority of the children ( $n=78$ , 81.3%) were less than 3.5 years, while most mothers ( $n=29$ , 36.5%) were aged 35 – 39 years.

#### 4.2.2 Age of Children Suspected with Loss of Hearing

The age distribution of children suspected of hearing loss by their mothers is shown in Table 4.2.

**Table 4.2: Age distribution of suspected hearing loss**

Age at hearing loss (years)	Children suspected of hearing loss		Children not suspected of hearing loss		Total	
	Frequency	Percent, %	Frequency	Percent, %	Frequency	Percent, %
0.0 – 2.5	66	68.75	17	17.70	83	86.5
2.6 – 4.5	3	3.13	5	5.20	8	8.3
4.6 – 6.5	3	3.13	1	1.04	4	4.2
6.6 – 8.5	0	0.00	0	0.00	0	0.0
8.6 - 10.0	0	0.00	1	1.04	1	1.0
Total	72	75.0	24	25.0	96	100.0

Seventy-two children suspected of hearing loss by their mothers were aged 0.0 to 6.5 years. The majority ( $n=66$ , 68.8%) were aged less than 2.5 years. No mothers reported of hearing loss in children older than 6.5 years. However, 24 children were not suspected of hearing loss but rather presented with developmental milestone relating to hearing and speech delay. Most of such children ( $n=17$ , 17.7%) were also aged under 2.5 years.

### 4.3 RISK FACTORS

**Research Question One:** *What are the predominant risk factors for hearing loss in children?*

In order to address this question, medical histories were obtained from the parents. These included family histories, maternal, delivery and labour histories, infant birth and childhood histories factors. The detailed results are illustrated in Tables 4.3 – 4.7.

#### 4.3.1 Family History

Table 4.3 shows the congenital (family histories) of children presenting with hearing loss.

**Table 4.3: Family histories of children suspected of presenting with hearing loss**

Family history	Yes	No	Total
Are parents' blood relations?	3 (3.1%)	93 (96.9%)	96 (100.0%)
Is there a family history of hearing loss?	6 (6.3%)	90 (93.8%)	96 (100.0%)
Are other siblings affected with hearing loss	5 (5.2%)	91 (94.8%)	96 (100.0%)

Most children with hearing loss did not show any family history of hearing loss. Whereas only 3.1% of children had blood related parents, 6.3% had family histories of hearing loss. Also, only 5.2% of the children with hearing loss had other siblings affected with hearing loss.

#### 4.3.2 Maternal Factors

The results of studies on the prenatal factors during pregnancy which could affect hearing loss (Table 4.4) showed that anaemia was the most prevalent condition of mothers as reported by 8.3% of the respondents. Only 1% presented with diabetes and preeclampsia respectively.

**Table 4.4: Distributions for maternal illness and exposure during pregnancy**

Prenatal (Maternal) factors		Yes	No	Total
Maternal illness	Bleeding	4 (4.2%)	92 (95.8%)	96 (100.0%)
	Anaemia	8 (8.3%)	88 (91.7%)	96 (100.0%)
	Diabetes	1 (1.0%)	95 (99.0%)	96 (100.0%)
	Preeclampsia	1 (1.0%)	95 (99.0%)	96 (100.0%)
	Measles	3 (3.1%)	93 (95.8%)	96 (100.0%)
	Mumps	2 (2.1%)	94 (97.9%)	96 (100.0%)
	Chicken pox	1 (1.0%)	95 (99.0%)	96 (100.0%)
	German measles	4 (4.1%)	92 (96.9%)	96 (100.0%)
	Syphilis	1 (1.0%)	95 (99.0%)	96 (100.0%)
	Herpes virus	0 (0.0%)	96 (100.0%)	96 (100.0%)
Others	Alcohol consumption	2 (2.1%)	94 (97.9%)	96 (100.0%)
	Excessive noise	2 (2.1%)	94 (97.9%)	96 (100.0%)

Measles and German measles were the most prevalent exposure conditions as reported by 3.1% respectively. Other major exposures were mumps and excessive noise (2.1%) respectively. None of the mothers in the study 0% was diagnosed with herpes or cytomegalous virus. However, syphilis was diagnosed in only 1.0% of the maternal population. Only 2.1% ( $n=2$ ) of the respondents admitted to alcohol consumption during their pregnancy.

### 4.3.3 Delivery and Labour Histories

Modes of maternal delivery histories were studied. The results (Table 4.5) showed that the most prevalent labour histories were full term ( $n=82$ , 85.4%) and spontaneous vaginal deliveries (SVD) while deliveries associated with bleeding labour and forceps modes were least prevalent at 3.1% (3) and 4.2% (4) respectively.

**Table 4.5: Distributions for mothers' delivery/labour history**

Delivery history		Yes	No	Total
Term	Full term (37 - 40 weeks)	82 (85.4%)	14 (14.6%)	96 (100.0%)
	Pre-term (< 37 weeks)	14 (14.6%)	82 (85.4%)	96 (100.0%)
Spontaneous vaginal delivery (SVD)		71 (74.0%)	25 (26.0%)	96 (100.0%)
Labour induced		8 (8.3%)	88 (91.7%)	96 (100.0%)
Labour in 24hrs		18 (18.8%)	78 (81.3%)	96 (100.0%)
Mode of delivery	Premature rupture	8 (8.3%)	88 (91.7%)	96 (100.0%)
	Bleeding labour	3 (3.1%)	93 (96.9%)	96 (100.0%)
	Forceps	4 (4.2%)	92 (95.8%)	96 (100.0%)
	Caesarian	22 (22.9%)	74 (77.1%)	96 (100.0%)

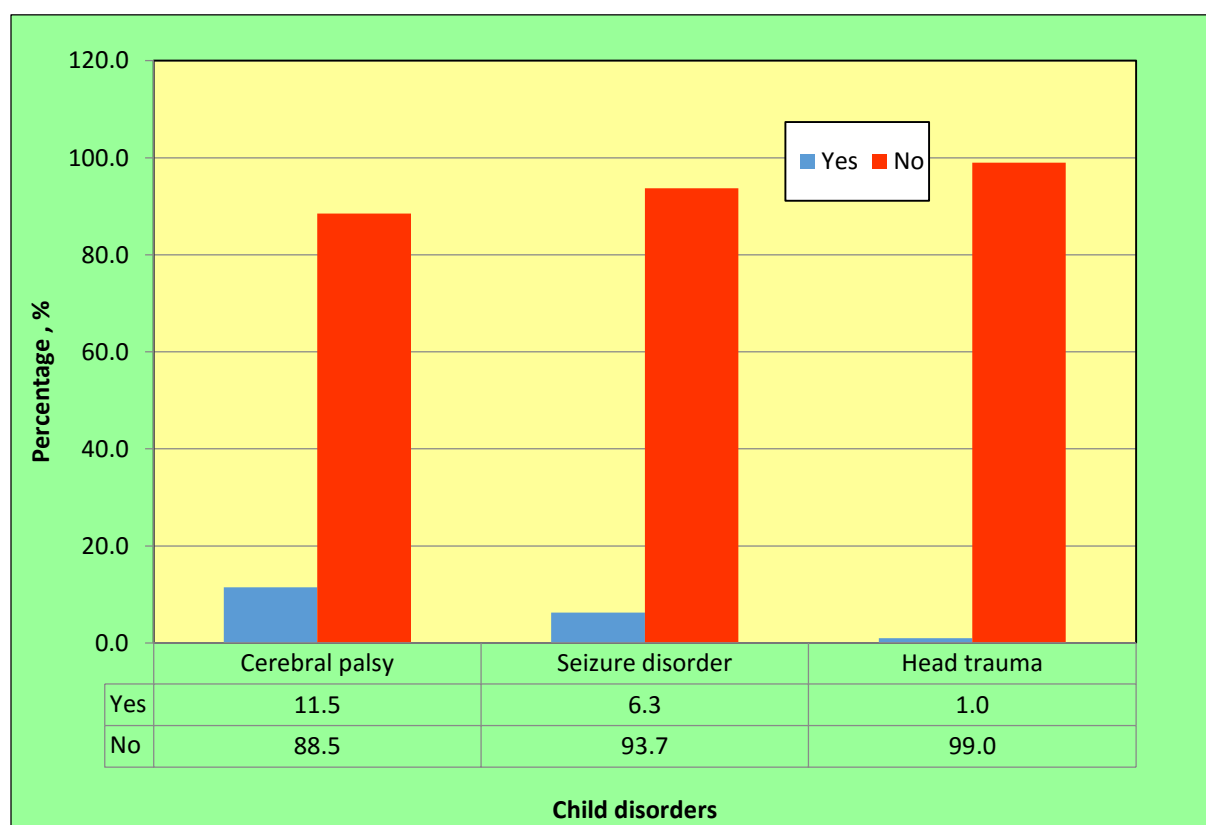
#### 4.3.4 Infant and Childhood Histories.

The results of the identified neonatal and postnatal factors are presented in Table 4.6 and Figure 4.1. Twenty three (24.0%) of the infants had anoxia, while 22.9% were either admitted to neonatal intensive care units (NICUs) or had breathing problems.

Low birth weight (8.9%), congenital heart disease and septicaemia (1.0%) were recorded as least infant or newborn factors among the children. No Apgar scores were recorded due to difficulty in assessing relevant data. Approximately 11.5% of the infants had cerebral palsy and 6.3% had seizure disorders.

**Table 4.6: Distribution of infant/newborn factors**

Infant/newborn factors	Yes	No	Total
Low birth weight	8 (8.3%)	88 (91.7%)	96 (100.0%)
Intensive care unit	22 (22.9%)	74 (77.1%)	96 (100.0%)
Problem breathing	22 (22.9%)	74 (77.1%)	96 (100.0%)
Anoxia	23 (24.0%)	73 (76.0%)	96 (100.0%)
Congenital heart disease	5 (5.2%)	91 (94.8%)	96 (100.0%)
Seizures	10 (10.4%)	86 (89.6%)	96 (100.0%)
Septicaemia	1 (1.0%)	95 (99.0%)	96 (100.0%)
Neonatal jaundice	14 (14.6%)	82 (85.4%)	96 (100.0%)

**Figure 4.1: Distribution of childhood risk factors**

Risk factors to pediatric hearing loss could arise from hospitalization. A distribution of the various conditions that necessitated hospitalization as well as the clinical diagnoses of the subjects is shown in Table 4.7.

**Table 4.7: Distribution of hospitalized and diagnosed conditions**

Clinical Risk Factors		Yes	No	Total
Hospitalized conditions	Meningitis	7 (7.3%)	89 (92.7%)	96 (100.0%)
	Cerebral malaria	3 (3.1%)	93 (96.1%)	96 (100.0%)
	Encephalitis	0 (0.0%)	96 (100.0%)	96 (100.0%)
	Measles	4 (4.2%)	92 (95.8%)	96 (100.0%)
Diagnoses	Rubella	0 (0.0%)	96 (100.0%)	96 (100.0%)
	Mumps	1 (1.0%)	95 (99.0%)	96 (100.0%)
	Chicken pox	4 (4.2%)	92 (95.8%)	96 (100.0%)
	Sickle cell	2 (2.1%)	1 (1.0%)	96 (100.0%)
	Diabetes	1 (1.0%)	95 (99.0%)	96 (100.0%)
	Ear Infection	21 (21.9%)	75 (78.1%)	96 (100.0%)

The results showed that the prevalent conditions for hospitalization were meningitis 7.3% ( $n=7$ , 7.3%) and measles ( $n=4$ , 4.2%). No case for hospitalization due to encephalitis was recorded. Ear infection ( $n=21$ , 21.9%) was the most reported diagnosed condition among the children. More than 4.0% of subjects were diagnosed with chicken pox, while none was diagnosed with rubella.

## 4.4 AUDIOLOGICAL TESTING

The results of the various audiological tests (OAE, VRA and play audiometry) are presented in this Section. Inferential statistics was applied to analyze the results for purposes of establishing any associations of hearing loss with age and gender using these assessment tools.

### 4.4.1 Otoacoustic Emission Results

**Research Question Two:** *What are the outcomes of the physiologic assessments in the children?*

In addressing this question, OAE tests were conducted to perform physiologic assessments of the children. The OAE test results as shown in Table 4.8 indicated a marginal difference in passes between the right (40.6%) and left (39.6%) ears. The converse was true for failures (right ear: 59.4%; left ear: 60.4%). Overall, there were less passes (40.1%) than refers (59.9%).

**Research Question Three:** *What are the associations between the OAE results and demographics?*

Inferential statistics were employed to investigate any association between the OAE outcomes and demographics (age and gender). The results are depicted in Table 4.8 and 4.9. The 0.5 – 2.5 years children presented with most passes ( $n=41/77$ , 53.2%) as well as refers ( $n=67/115$ , 58.3%) while the 6.6–8.5 year group was least presented (passes:  $n=2/77$ , 2.6%; refers:  $n=61/115$ , 5.2%). In general, the comparisons of the OAE test results with age ((right ears:  $[\chi^2(4, N= 96) = 2.68, p=0.61 > 0.05]$ , left ears:  $\chi^2 [(4, N= 96) = 2.89, p=0.58 > 0.05]$ ) revealed non-significant associations in right and left ears (Table 4.8). Similarly, from Table 4.9, no significant associations between the OAE test results and gender were established for both ears (right ears:  $[\chi^2(1, N= 96) = 1.06, p=0.30 > 0.05]$ , left ears:  $\chi^2 [(1, N= 96) = 0.00, p=0.99 > 0.05]$ ).

**Table4.8: Association between OAE test results and age for right and left ears**

Age (years)	Right Ear				Left Ear				Combined	
	Pass	Refer	$\chi^2$ (df= 4)	<i>p</i> -value	Pass	Refer	$\chi^2$ (df= 4)	<i>p</i> -value	Pass	Refer
0.0 – 2.5	20	34	2.68	0.61	21	33	2.89	0.58	41	67
2.6 – 4.5	13	13			12	14			25	27
4.6 – 6.5	3	6			2	7			5	13
6.6 – 8.5	1	3			1	3			2	6
8.6 - 10.0	2	1			2	1			4	2
Total	39	57			38	58			77	115

 $\alpha = 0.05$

**Table 4.9: Association between OAE test results and gender for both ears**

	Male			Female			Combined			$X^2$ $df=1$	$p$ - value
	Pass	Refer	Total	Pass	Refer	Total	Pass	Refer	Total		
Right ear											
Number	24	29	53	15	28	43	39	57	96	1.06	0.30
Percent	45.3	54.7	100.0	34.9	65.1	100.0	40.6	59.4	100.0		
Left ear											
Number	21	32	53	17	26	43	38	58	96	0.00	0.99
Percent	39.6	60.4	100.0	39.5	60.5	100.0	39.6	60.4	100.0		

$\alpha = 0.05$

#### 4.4.2 Visual Reinforcement Audiometry and Play Audiometry

**Research Question Four:** *What are the degrees and prevalence of hearing loss via VRA, play audiometry at the various test frequencies?*

In addressing this question, VRA and play audiometry assessments were performed on the subjects. In this study, 44 respondents qualified for the VRA assessments. Hearing loss was found in majority of them in both ears across all frequencies (Table 4.10).

**Table 4.10: Distributions for VRA audiometric results for both ears**

Degree of hearing loss	Right Ear			Left Ear		
	500 Hz No. (%)	1 kHz No. (%)	2 kHz No. (%)	500 Hz No. (%)	1 kHz No. (%)	2 kHz No. (%)
Normal hearing Loss	2 (4.5)	1 (2.3)	2 (4.5)	5 (11.4)	2 (4.5)	2 (4.5)
Mild hearing loss	13 (29.5)	12 (27.3)	10 (22.7)	9 (20.5)	13 (29.5)	13 (29.5)
Moderate hearing loss	6 (13.6)	7 (15.9)	8 (18.2)	7 (15.9)	6 (13.6)	5 (11.4)
Moderate to severe hearing loss	3 (6.8)	4 (9.1)	4 (9.1)	3 (6.8)	3 (6.8)	4 (9.1)
Severe hearing loss	9 (20.5)	8 (18.2)	6 (13.6)	9 (20.5)	7 (15.9)	8 (18.2)
Profound hearing loss	11 (25.0)	12 (27.3)	14 (31.8)	11 (25.0)	13 (29.5)	12 (27.3)
Total	44 (100)	44 (100.)	44 (100)	44 (100)	44 (100)	44 (100)

Normal hearing was least prevalent across all test frequencies. The most prevalent degrees of hearing loss at low frequency (500 Hz) were severe hearing loss ( $n=9$ , 20.5% for both ears), profound hearing loss ( $n=11$ , 25.0% for both ears) and mild hearing loss (right ear:  $n=13$ , 29.5%; left ear:  $n=9$ , 20.5%). At higher frequencies (1 kHz – 2 kHz), mild ( $n=13$ , 29.5% for both ears) and profound (right ear:  $n=13$ , 29.5%; left ear:  $n=12$ , 27.3%) hearing losses were most prevalent.

In respect of play audiometry, 36 children were tested. The results of the test (Table 4.11) showed that hearing loss was almost equal in both ears at all frequencies.

**Table 4.11: Distributions for play audiometric results for both ears**

Play audiometry	Right Ear			Left Ear		
	500Hz No. (%)	1KHz No. (%)	2KHz No. (%)	500Hz No. (%)	1KHz No. (%)	2KHz No. (%)
Normal Hearing Loss	6 (16.7)	5 (13.9)	4 (11.1)	4 (11.1)	6 (16.7)	6 (16.7)
Mild hearing loss	4 (11.1)	4 (11.1)	6 (16.7)	4 (11.1)	2 (5.6)	3 (8.3)
Moderate hearing loss	4 (11.1)	4 (11.1)	3 (8.3)	4 (11.1)	4 (11.1)	3 (8.3)
Moderate to severe hearing loss	4 (10.5)	3 (8.3)	3 (8.3)	6 (16.7)	5 (13.9)	5 (13.9)
Severe hearing loss	8 (22.2)	8 (22.2)	8 (22.2)	9 (25.0)	9 (25.0)	9 (25.0)
Profound hearing loss	10 (27.8)	12 (33.3)	12 (33.3)	10 (27.8)	10 (27.8)	10 (27.8)
Total	36 (100)	36 (100)	36 (100)	36 (100)	36 (100)	36 (100)

Severe and profound hearing losses were most prevalent across the test frequencies. The prevalence of severe hearing loss were 22.2% for right ear and 25% for left ear, while the prevalence of profound hearing loss was estimated at 27.8% -33.3% for right ear and 27.8% for left ear respectively at both low and high frequencies.

**Research Question Five:** *What are the associations between hearing loss and demographics as measured via VRA and play audiometry assessments?*

In answering the above question, inferential statistics via chi-square tests and  $p$ -value calculations were performed to establish any statistical associations between the VRA test results and play audiometry test batteries, and age and gender at the various test frequencies.

### **Associations of Hearing Loss with Age**

At 500 Hz (low frequency), a non-significant association between hearing loss as measured via VRA and age was established for right ears  $\{[\chi^2 (10, N= 44) = 5.99, p=0.31 > 0.05]\}$ , and left ears  $\{[\chi^2 (10, N= 44) = 5.13, p=0.40 > 0.05]\}$  as shown in Table 4.12. More children presented with severe ( $n=9, 20.5\%$ ), mild ( $n=10, 22.7\%$ ) and profound ( $n=11, 25\%$ ) hearing losses for the 0.5 – 2.5 year olds in the right ear compared to the 2.6 – 4.5 years group (severe:  $n=0, 0.0\%$ ; mild:  $n=3, 6.8\%$ ; profound:  $n=0, 0.0\%$ ) respectively. The same results were repeated in the left ear for severe and profound hearing losses. The numbers for mild hearing loss were however reduced to 7 (15.9%) and 2 (4.5%) for the 0.5 - 2.5 years group and the 2.6 – 4.5 years group respectively.

At 1000 Hz frequency (Table 4.13), no significant association between hearing loss and age via VRA was found among the children in both ears as established by the inferential statistics (right ears:  $[\chi^2 (10, N= 44) = 6.40, p=0.27 > 0.05]$ , left ears:  $[\chi^2 (10, N= 44) = 5.99, p=0.31 > 0.05]$ ). Normal hearing was least prevalent (right ear:  $n=1, 2.3\%$ ; left ear:  $n=2, 4.5\%$ ) for the younger age group. No prevalence was found for the older age group. Mild and profound hearing losses were most prevalent at 1000 Hz. For both degrees of hearing loss, the differences in the number

of children presenting with hearing loss in the right ear ( $n=12$ , 27.3%) were marginally lower compared to the left ear ( $n=13$ , 29.6%).

**Table 4.12: Association between hearing status and age for both ears: VRA at 500Hz**

Hearing status	Age (years)		Total	$\chi^2(df= 10)$	<i>p-value</i>
	0.0 - 2.5	2.6 - 4.5			
Right Ear					
Normal hearing	2	0	2	5.99	0.31
Mild H/L	10	3	13		
Moderate H/L	5	1	6		
Moderate-Severe H/L	3	0	3		
Severe H/L	9	0	9		
Profound	11	0	11		
Total	40	4	44		
Left Ear					
Normal hearing	4	1	5	5.13	0.40
Mild H/L	7	2	9		
Moderate H/L	6	1	7		
Moderate-Severe H/L	3	0	3		
Severe H/L	9	0	9		
Profound H/L	11	0	11		
Total	40	4	44		

$\alpha=0.05$

**Table 4.13: Association between hearing status and age for both ears: VRA at 1000 Hz**

Hearing status	Age (years)		Total	$\chi^2(df= 10)$	<i>p-value</i>
	0.0 - 2.5	2.6 - 4.5			
Right Ear					
Normal Hearing	1	0	1	6.40	0.27
Mild H/L	9	3	12		
Moderate H/L	6	1	7		
Moderate-Severe H/L	4	0	4		
Severe H/L	8	0	8		
Profound	12	0	12		
Total	40	4	44		
Left Ear					
Normal Hearing	2	0	2	5.99	0.31
Mild H/L	10	3	13		
Moderate H/L	5	1	6		
Moderate-Severe H/L	3	0	3		
Severe H/L	7	0	7		
Profound H/L	13	0	13		
Total	40	4	44		

$\alpha=0.05$

From Table 4.14 (2000 Hz), no significant association between hearing loss via VRA and age was found among the children in both ears (right ears: [ $\chi^2(10, N= 44) = 6.49, p=0.26 > 0.05$ ], left ears:  $\chi^2 [(10, N= 44) = 6.38, p=0.27 > 0.05]$ ). In particular, mild and moderate hearing losses were noticeable in the 2.6 – 4.5 years children. A prevalence of 4.6% was estimated for this group of children in the right ear, while prevalence of 6.9% and 2.3 % were estimated respectively in the left ear for same types of hearing loss.

**Table 4.14: Association between hearing status and age for both ears: VRA at 2000 Hz**

Hearing status	Age (years)		Total	$\chi^2(df= 10)$	<i>p-value</i>
	0.0 - 2.5	2.6 - 4.5			
Right Ear					
Normal Hearing	2	0	2	6.49	0.26
Mild H/L	8	2	10		
Moderate H/L	6	2	8		
Moderate-Severe H/L	4	0	4		
Severe H/L	6	0	6		
Profound	14	0	14		
Total	40	4	44		
Left Ear					
Normal Hearing	2	0	2	6.38	0.27
Mild H/L	10	3	13		
Moderate H/L	4	1	5		
Moderate-Severe H/L	4	0	4		
Severe H/L	8	0	8		
Profound H/L	12	0	12		
Total	40	4	44		

$\alpha=0.05$

As observed earlier, prevalence of hearing loss was relatively higher in the younger (0.5 – 2.5 years) group. Normal hearing was again least prevalent (4.6%) at 2000 Hz for the younger group, while moderate-severe hearing loss was 9.1% prevalent in both ears. The prevalence of profound hearing loss was highest for both ears (right ear:  $n=14$ , 32.8%; left ear:  $n=12$ , 27.3%). However, mild hearing loss more prevalent in the left ear ( $n=13$ , 29.6%).

The results of investigating associations between hearing loss and age by play audiometry are shown in Tables 4.15 – 4.17. The chi-square and  $p$ -value at 500 Hz were right ears: [ $\chi^2$  (15,  $N=36$ ) = 13.24,  $p=0.59 > 0.05$ ], and left ears: [ $\chi^2$  ((15,  $N=36$ ) = 22.03,  $p=0.11 > 0.05$ )] (Table 4. 15).

**Table4.15: Association between hearing loss and age using play audiometry: 500 Hz**

Hearing status	Age (years)				Total	$\chi^2$ ( $df=15$ )	$p$ -value
	2.6-4.5	4.6 - 6.5	6.6 - 8.5	8.6- 10.0			
Right ear							
Normal hearing	2	2	0	2	6	13.24	0.59
Mild H/L	3	1	0	0	4		
Moderate H/L	2	1	1	0	4		
Moderate-Severe H/L	2	1	0	1	4		
Severe H/L	5	2	1	0	8		
Profound H/L	7	1	2	0	10		
Total	21	8	4	3	36		
Left ear							
Normal hearing	0	2	0	2	4	22.03	0.11
Mild H/L	3	1	0	0	4		
Moderate H/L	3	0	0	1	4		
Moderate-Severe H/L	4	2	0	0	6		
Severe H/L	5	2	2	0	9		
Profound H/L	6	1	2	0	9		
Total	21	8	4	3	36		

Hearing loss was most common (58.3%) among the 0.5 – 2.5 years group of children for both ears, of which severe (right ear:  $n=5$ , 13.9%); left ear:  $n=5$ , 13.9%) and profound (right ear:  $n=7$ , 19.4%); left ear:  $n=6$ , 16.7%) hearing losses were the most prevalent.

At 1000 Hz (Table 4.16), no significant association between hearing loss and age by (right ears: [ $\chi^2$  (15, N= 36) = 13.99,  $p=0.53 > 0.05$ ], left ears: [ $\chi^2$  [(15, N= 36) = 15.63,  $p=0.41 > 0.05$ ]) was found using play audiometry. The 0.5–2.5 year olds presented with most degrees of hearing loss.

**Table4.16: Association between hearing loss and age using play audiometry: 1000 Hz**

Hearing status	Age (years)				Total	$\chi^2$ (df=15)	p-value
	2.6-4.5	4.6 - 6.5	6.6 - 8.5	8.6- 10.0			
Right ear							
Normal hearing	1	2	0	2	5	13.99	0.53
Mild H/L	3	1	0	0	4		
Moderate H/L	2	1	1	0	4		
Moderate-Severe H/L	3	0	0	0	3		
Severe H/L	5	2	1	0	8		
Profound H/L	7	2	2	1	12		
Total	21	8	4	3	36		
Left ear							
Normal hearing	2	2	0	2	6	15.63	0.41
Mild H/L	1	1	0	0	2		
Moderate H/L	4	0	0	0	4		
Moderate-Severe H/L	3	2	0	0	5		
Severe H/L	4	2	2	1	9		
Profound H/L	7	1	2	0	10		
Total	21	8	4	3	36		

$\alpha = 0.05$

Again, no significant association between hearing loss and age was found (right ears: [ $\chi^2$  (15, N= 36) = 21.63,  $p=0.12 > 0.05$ ], left ears:  $\chi^2$  [(15, N= 36) = 14.59,  $p=0.48 > 0.05$ ]) at 2000 Hz (Tables 4.17).

**Table 4.17: Association between hearing loss and age using play audiometry: 2000 Hz**

Hearing status	Age (years)					$\chi^2$ ( $df=15$ )	$p$ -value
	2.6-4.5	4.6 - 6.5	6.6 - 8.5	8.6 - 10.0	Total		
Right ear							
Normal hearing	0	2	0	2	4	21.63	0.12
Mild H/L	4	2	0	0	6		
Moderate H/L	3	0	0	0	3		
Moderate-Severe H/L	2	0	1	0	3		
Severe H/L	4	3	1	0	8		
Profound H/L	8	1	2	1	12		
Total	21	8	4	3	36		
Left ear							
Normal hearing	2	2	0	2	6	14.59	0.48
Mild H/L	2	1	0	0	3		
Moderate H/L	3	0	0	0	3		
Moderate-Severe H/L	3	2	0	0	5		
Severe H/L	4	2	2	1	9		
Profound H/L	7	1	2	0	10		
Total	21	8	4	3	36		

$\alpha=0.05$

### Association of Hearing Loss with Gender

Associations between hearing loss and gender as measured by VRA and play audiometry were investigated. For the VRA, more males ( $n=26$ , 59.1%) than females ( $n=18$ , 40.9%) underwent the VRA tests. The results at 500 Hz is shown in Table 4.18.

**Table 4.18: Association between hearing status and gender for both ears: VRA at 500 Hz**

Hearing status	Male	Female	Total	$\chi^2(df= 10)$	<i>p-value</i>
Right Ear					
Normal hearing	2	0	2	4.78	0.44
Mild H/L	9	4	13		
Moderate H/L	3	3	6		
Moderate-Severe H/L	2	1	3		
Severe H/L	6	3	9		
Profound	4	7	11		
Total	26	18	44		
Left Ear					
Normal hearing	4	1	5	5.60	0.35
Mild H/L	7	2	9		
Moderate H/L	3	4	7		
Moderate-Severe H/L	2	1	3		
Severe H/L	3	6	9		
Profound H/L	7	4	11		
Total	26	18	44		

$\alpha=0.05$

The statistical analysis established no significant association between hearing loss and gender among the children in both ears (right ears:  $\chi^2 (10, N= 44) = 4.78, p=0.44 > 0.05$ ], left ears:  $\chi^2 [(10, N= 44) = 5.60, p=0.35 > 0.05]$ ) by VRA. Mild hearing loss was the most prevalent degree of hearing loss in both ears (right ear:  $n=13, 29.6\%$ ); left ear:  $n=9, 20.5\%$ ) and higher in males (right ear:  $n=9, 20.5\%$ ; left ear:  $n=7, 15.9\%$ ) than in females (right ear:  $n=4, 9.1\%$ ; left ear:  $n=2, 4.5\%$ ). Generally, hearing loss was more prevalent in males in all the degrees of hearing loss in both right and left ears. However, a reverse trend was observed in profound hearing loss in the right ear (males:  $n=4, 9.1\%$ ; females: ear:  $n=7, 15.9\%$ ) and for severe hearing loss in the left ear (males:  $n=3, 6.8\%$ ; females: ear:  $n=6, 13.6\%$ ).

From Table 4.19 no significant association between hearing loss and gender was established among children in both ears at 1000 Hz via VRA (right ears:  $\chi^2 (10, N= 44) = 4.68, p=0.46 > 0.05$ ], left ears:  $\chi^2 [(10, N= 44) = 5.63, p=0.34 > 0.05]$ ). Mild and profound hearing losses were most prevalent in both ears (right ear:  $n=12, 27.3\%$ ); left ear:  $n=13, 29.6\%$ ). Although mild hearing loss was higher in males ( $n=9, 20.5\%$ ) compared to females ( $n=3, 6.8\%$ ) in the right ear, the reverse trend was true for profound hearing loss where the prevalence in males ( $n=5, 11.4\%$ ) was lower than in females ( $n=7, 15.9\%$ ).

The same trend was observed for severe hearing loss (male right ear:  $n=5, 11.4\%$ ; female: right ear:  $n=3, 6.8\%$ ; male left ear:  $n=2, 4.5\%$ ; female left ear:  $n=5, 11.4\%$ ).

**Table 4.19: Association between hearing status and gender for both ears: VRA at 1000 Hz**

Hearing status	Male	Female	Total	$\chi^2(df= 10)$	<i>p-value</i>
<b>Right Ear</b>					
Normal hearing	1	0	1	4.68	0.46
Mild H/L	9	3	12		
Moderate H/L	3	4	7		
Moderate-Severe H/L	3	1	4		
Severe H/L	5	3	8		
Profound	5	7	12		
Total	26	18	44		
<b>Left Ear</b>					
Normal hearing	2	0	2	5.63	0.34
Mild H/L	9	4	13		
Moderate H/L	4	2	6		
Moderate-Severe H/L	1	2	3		
Severe H/L	2	5	7		
Profound H/L	8	5	13		
Total	26	18	44		

$\alpha=0.05$

At 2000 Hz (Table 4.20), the statistical analysis further established no significant association between hearing loss and gender among the children in both ears (right ears: [ $\chi^2(10, N= 44) = 4.24, p=0.52 > 0.05$ ], left ears: [ $\chi^2(10, N= 44) = 6.21, p=0.29 > 0.05$ ]) via VRA. In particular, hearing loss was generally more prevalent in males in all the degrees of hearing loss in both right and left ears with the exception profound hearing loss in the right ear (males:  $n=6, 13.6\%$ ;

females:  $n=8$ , 18.2%) and severe hearing loss in the left ear (males:  $n=2$ , 4.5%; females:  $n=6$ , 13.6%).

#### 4.20: Association between hearing status and gender for both ears: VRA at 2000Hz

Hearing status	Male	Female	Total	$\chi^2(df=10)$	$p$ -value
Right Ear					
Normal hearing	2	0	2	4.24	0.52
Mild H/L	7	3	10		
Moderate H/L	4	4	8		
Moderate-Severe H/L	3	1	4		
Severe H/L	4	2	6		
Profound	6	8	14		
Total	26	18	44		
Left Ear					
Normal hearing	2	0	2	6.21	0.29
Mild H/L	9	4	13		
Moderate H/L	3	2	5		
Moderate-Severe H/L	2	2	4		
Severe H/L	2	6	8		
Profound H/L	8	4	12		
Total	26	18	44		

$\alpha=0.05$

From play audiometry measurements (Tables 4.21 -4.23), the chi-square and  $p$ -value calculations showed non-significant associations between hearing loss and gender at 500 Hz (right ears:  $[\chi^2(5, N=36) = 4.17, p=0.53 > 0.05]$ , left ears:  $[\chi^2(5, N=36) = 2.68, p=0.75 > 0.05]$ ), at 1000 Hz (right ears:  $[\chi^2(5, N=36) = 1.76, p=0.88 > 0.05]$ , left ears:  $[\chi^2(5, N=36) = 4.37, p=0.50 > 0.05]$ ),

and 2000 Hz (right ears: [ $\chi^2 (5, N= 36) = 2.40, p=0.79 > 0.05$ ], left ears: $\chi^2 [(5, N= 36) = 5.64, p=0.34 > 0.05]$ ).

**Table 4.21: Association between hearing loss and gender (play audiometry): 500 Hz**

Hearing status	Gender			$\chi^2 (df= 5)$	<i>p</i> -value
	Male	Female	Total		
Right ear					
Normal hearing	2	4	6	4.17	0.53
Mild H/L	3	1	4		
Moderate H/L	3	1	4		
Moderate-Severe H/L	2	2	4		
Severe H/L	4	4	8		
Profound H/L	3	7	10		
Total	17	19	36		
Left ear					
Normal hearing	3	1	4	2.68	0.75
Mild H/L	2	2	4		
Moderate H/L	2	2	4		
Moderate-Severe H/L	2	4	6		
Severe H/L	5	4	9		
Profound H/L	3	6	9		
Total	17	19	36		

$\alpha=0.05$

**Table 4.22: Association between hearing loss and gender (play audiometry): 1000 Hz**

Hearing status	Gender			$\chi^2$ (df= 5)	<i>p</i> -value
	Male	Female	Total		
Right ear					
Normal hearing	2	3	5	1.76	0.88
Mild H/L	2	2	4		
Moderate H/L	3	1	4		
Moderate-Severe H/L	1	2	3		
Severe H/L	4	4	8		
Profound H/L	5	7	12		
Total	17	19	36		
Left ear					
Normal hearing	4	2	6	4.37	0.50
Mild H/L	1	1	2		
Moderate H/L	1	3	4		
Moderate-Severe H/L	2	3	5		
Severe H/L	6	3	9		
Profound H/L	3	7	10		
Total	17	19	36		

 $\alpha=0.05$

**Table 4.23: Association between hearing loss and gender (play audiometry): 2000 Hz**

Hearing status	Gender			$\chi^2$ (df= 5)	<i>p</i> -value
	Male	Female	Total		
Right ear					
Normal hearing	2	2	4	2.40	0.79
Mild H/L	3	3	6		
Moderate H/L	2	1	3		
Moderate-Severe H/L	1	2	3		
Severe H/L	5	3	8		
Profound H/L	4	8	12		
Total	17	19	36		
Left ear					
Normal hearing	4	2	6	5.64	0.34
Mild H/L	2	1	3		
Moderate H/L	1	2	3		
Moderate-Severe H/L	1	4	5		
Severe H/L	6	3	9		
Profound H/L	3	7	10		
Total	17	19	36		

 $\alpha=0.05$

### 4.4.3 Distraction Test

Only 16 respondents qualified for distraction assessment. The results (Table 4.24) showed most failures ( $n=13$ , 81.2%) in the distraction test.

**Table 4.24: Distributions for distraction test results**

Distraction test	Frequency	Percent, %
Pass	3	18.8
Refer	13	81.2
Total	16	100.0

Gender	Distraction test			$\chi^2$ (df=2)	p-value
	Pass	Fail	Total		
Male	2	8	10	0.02	0.87
Female	1	5	6		
Total	3	13	16		

$\alpha=0.05$

There was no significant association between pass/refer and gender [ $\chi^2$  (2, N= 16) = 0.02,  $p=0.87>0.05$ ] as measured by distraction tests.

### 4.4.4. Tympanograms

**Research Question Six:** *What are the associations between the tympanograms, and test results from the test batteries?*

In answering this question, the following assessments were done. Tympanometry was performed to assess the middle ear function of the 96 children. Significant associations between

tympanograms and OAE test results were established for right: [ $\chi^2$  (2, N= 96) = 12.52,  $p=0.00 < 0.05$ ] and left ears: [ $\chi^2$  (2, N= 96) = 12.95,  $p=0.00 < 0.05$ ], as shown in Table 4.25.

**Table 4.25: Association between tympanograms and OAE test for both ears**

OAE	Tympanogram				$\chi^2$ (df = 2)	<i>p</i> -value
	Type A	Type B	Type C	Total		
Right ear						
Pass	37	0	0	39	12.52	0.00
Refer	37	11	11	57		
Total	74	11	11	96		
Left ear						
Pass	37	0	1	38	12.95	0.00
Refer	39	12	7	58		
Total	76	12	8	96		

$\alpha=0.05$

Out of 74 right ears with Type A tympanograms, 37 passed and 37 were referred. All ears ( $n=11$ , 100.0%) with Type B tympanograms referred. All right ears with Type C tympanograms (11) referred in the OAE test. For the left ear, the number of passes ( $n=37$ , 48.7%) was marginally lower compared to the referrals ( $n=39$ , 51.37%) with Type A tympanograms ( $n=76$ ). All left ears with type B tympanograms (12) referred in the OAE tests, while only 12.5% passed with Type C tympanograms ( $n=8$ ).

Statistical associations between hearing loss and tympanograms as measured by VRA and play audiometry at the three test frequencies (Table 4.26 -4.29) were analyzed.

From the VRA measurements, the calculated chi-square and  $p$ -values were [ $\chi^2$  (10, N= 44) = 23.79,  $p=0.01 < 0.05$ ] at 500 Hz. At higher frequencies of 1000 Hz and 2000 Hz, the statistics indicated [ $\chi^2$  (10, N= 44) = 20.82,  $p=0.02 < 0.05$ ], and [ $\chi^2$  (10, N= 44) = 20.18,  $p=0.03 < 0.05$ ] respectively. Majority of the right ears with Type A tympanograms presented with a hearing loss ranging from mild to profound (Table 4.26).

Again, from Table 4.27, there was a significant association between hearing loss and tympanograms via VRA for left ears at 1000 Hz [ $\chi^2$  (10, N= 44) = 22.30,  $p=0.01 < 0.05$ ] and 2000 Hz [ $\chi^2$  (10, N= 44) = 20.63,  $p=0.02 < 0.05$ ] kHz except at 500 Hz [ $\chi^2$  (10, N= 44) = 17.64,  $p=0.06 > 0.05$ ]. Most left ears with Type A tympanograms presented with hearing loss ranging from mild to profound.

Similarly, majority of the ears with Type A tympanograms presented with hearing loss ranging from mild to profound (Table 4.28 and 4.29) for both ears at 500 Hz (right ear:  $n=23$ ; left ear:  $n=24$ ), 1000 Hz (right ear:  $n=23$ ; left ear:  $n=22$ ) and 2000 Hz (right ear:  $n=24$ ; left ear:  $n=22$ ) from play audiometry measurements respectively.

There was no significant association between hearing loss and tympanograms via play audiometry at all tested frequencies for right ears [500 Hz:  $\chi^2$  (10, N= 36) = 8.66,  $p=0.57 > 0.05$ , 1000 Hz:  $\chi^2$  (10, N= 36) = 7.07,  $p=0.72 > 0.05$ , 2000 Hz:  $\chi^2$  (10, N= 36) = 6.21,  $p=0.80 > 0.05$ ] and left ears [500 Hz:  $\chi^2$  (10, N= 36) = 11.54,  $p=0.32 > 0.05$ , 1000 Hz:  $\chi^2$  (10, N= 36) = 9.33,  $p=0.50 > 0.05$ , 2000 Hz:  $\chi^2$  (10, N= 36) = 8.63,  $p=0.57 > 0.05$ ].

**Table 4.26: Association between hearing loss and tympanograms (VRA right ears)**

Frequency (Hz)	Tympanogram (Right)	Moderate-						Total	$\chi^2$ df=10	p-value
		Normal	Mild	Moderate	Severe	Severe	Profound			
500	Type A	2	13	6	2	7	3	33	23.79	0.01
	Type B	0	0	0	0	2	4	6		
	Type C	0	0	0	1	0	4	5		
	Total	2	13	6	3	9	11	44		
1000	Type A	1	12	7	3	6	4	33	20.82	0.02
	Type B	0	0	0	0	2	4	6		
	Type C	0	0	0	1	0	4	5		
	Total	1	12	7	4	8	12	44		
2000	Type A	2	10	8	3	5	5	33	20.18	0.03
	Type B	0	0	0	0	1	5	6		
	Type C	0	0	0	1	0	4	5		
	Total	2	10	8	4	6	14	44		

**Table 4.27: Association between hearing loss and tympanograms (VRA left ears)**

Frequency (Hz)	Tympanogram (Right)	Moderate-						Total	$\chi^2$ df=10	p-value
		Normal	Mild	Moderate	Severe	Severe	Profound			
500	Type A	5	9	6	3	4	7	34	17.64	0.06
	Type B	0	0	0	0	5	3	8		
	Type C	0	0	1	0	0	1	2		
	Total	5	9	7	3	9	11	44		
1000	Type A	2	13	5	3	2	9	34	22.30	0.01
	Type B	0	0	0	0	5	3	8		
	Type C	0	0	1	0	0	1	2		
	Total	2	13	6	3	7	13	44		
2000	Type A	2	13	4	4	3	8	34	20.63	0.02
	Type B	0	0	0	0	5	3	8		
	Type C	0	0	1	0	0	1	2		
	Total	2	13	5	4	8	12	44		

**Table 4.28: Association between hearing loss and tympanograms for ears (play audiometry)**

Frequency (Hz)	Tympanogram (Right)	Moderate-						Total	$\chi^2$ (df=10)	p-value
		Normal	Mild	Moderate	Severe	Severe	Profound			
500	Type A	5	4	4	4	4	7	28	8.66	0.57
	Type B	0	0	0	0	2	2	4		
	Type C	1	0	0	0	2	1	4		
	Total	6	4	4	4	8	10	36		
1000	Type A	5	3	4	3	5	8	28	7.07	0.72
	Type B	0	0	0	0	2	2	4		
	Type C	0	1	0	0	1	2	4		
	Total	5	4	4	3	8	12	36		
2000	Type A	4	5	3	3	5	8	28	6.21	0.80
	Type B	0	0	0	0	2	2	4		
	Type C	0	1	0	0	1	2	4		
	Total	4	6	3	3	8	12	36		

**Table 4.29: Association between hearing loss and tympanograms for left ears (play audiometry)**

Frequency (Hz)	Tympanogram (Right)	Moderate-						Total	$\chi^2$ (df=10)	p-value
		Normal	Mild	Moderate	Severe	Severe	Profound			
500	Type A	4	3	3	6	4	8	28	11.54	0.32
	Type B	0	0	0	0	2	1	3		
	Type C	0	1	1	0	3	0	5		
	Total	4	4	4	6	9	9	36		
1000	Type A	6	1	3	5	5	8	28	9.33	0.50
	Type B	0	0	0	0	2	1	3		
	Type C	0	1	1	0	2	1	5		
	Total	6	2	4	5	9	10	36		
2000	Type A	6	2	2	5	5	8	28	8.63	0.57
	Type B	0	0	0	0	2	1	3		
	Type C	0	1	1	0	2	1	5		
	Total	6	3	3	5	9	10	36		

## CHAPTER FIVE

### DISCUSSIONS

#### 5.1 INTRODUCTION

This Chapter discusses the results of the present study in relation to the relevant literature, taking into consideration the defined objectives and research questions of the study.

#### 5.2 DEMOGRAPHIC CHARACTERISTICS

In this study, 96 respondents of ages 6 months to 10 years participated in the study. The average age of the respondents was  $2.74 \pm 2.00$  years. Of this number, a greater majority of 72 children aged 0.0 to 6.5 were suspected of hearing loss by their mothers while the remaining 24 presented with developmental milestone relating to hearing and speech delay. Most of the children ( $n=17$ , 17.7%) presenting with developmental challenges were relatively younger (aged under 2.5 years) compared to those suspected of hearing loss. Over 81% of all the children ( $n=78$ ) were aged less than 3.5 years. The population of male children ( $n=53$ ) was higher than females ( $n=43$ ). Statistically, the mode of the maternal age distribution was 35 – 39 years (range: 20 years – 43 years), and an average maternal age of  $32.3 \pm 4.92$  years was indicative of reproductive age.

The age range of children considered in this study as can be noted above is consistent with Kansherla et al., (2013) study where a hearing loss prevalence of 1.3 per 1000 was reported among 8-year-olds in Atlanta, USA. According to accepted conventions in clinical audiology, an average of 2.74 years as obtained in this study may be considered as quite late for detection of hearing loss in children. In this study also, 66% of the children suspected of hearing loss were

aged 2.5 years (younger than the overall average age) while 78% of all the children were aged from 6 months to 3.5 years. This is suggestive that most of the children were sent to the clinic for late intervention and not early detection of hearing loss. The late efforts at addressing the suspected hearing loss problems could be attributed to parents' inability and inefficient methods at detecting early signs of hearing impairments at birth in their children. Early detection makes it easier for early intervention thereby reducing the impact of hearing impairment among children (Paludetti et al, 2012).

### **5.3 RISK FACTORS**

#### **5.3.1 Maternal Factors**

The medical histories of the children and their mothers were investigated via studies on children's family histories, maternal factors, delivery and labour histories, infant birth and childhood histories.

Hearing loss has been associated with various maternal factors or diseases such as meningitis, mumps, measles, and jaundice. According to Northern and Downs (1991), damage to the ear results from infiltration of the organism via the internal meatus in most bacterial infections, and further indicated that viral diseases may cause histopathological damage to the organ of Corti, damage or complete destruction of the striavascularis and tectorial membrane, and atrophy or destruction of the neural pathways. In this study, anaemia was identified as the most prevalent (8.3%) maternal condition while diabetes and preeclampsia were least prevalent (1.0%). Exposure factors such as measles, German measles (rubella infection), mumps and which has been associated with hearing loss were also less prevalent (0.0 – 3.1%). Again, the findings of this study are consistent with the work of Niedzielska et al, (2000) where hearing defects in

children born of mothers who had suffered from symptomatic rubella during their pregnancy have been reported. In particular, rubella infections are transferable and hence could induce congenital conditions in unborn children from pregnant mothers. This may also pose a risk of auditory damage to unborn children as revealed by Niedzielska et al, (2000).

As seen from the results of this study, mild, severe and profound hearing losses were the most prevalent degrees of hearing impairment identified in the cross-sectional study of 96 paediatric patients using several test batteries (OAE, VRA, and play audiometry). The impact of auditory damage on the hearing of such children is apparent as observed by Sharma, et al, (2005) that cross modal re-organization of the brain (re-assignment of auditory brain cells to other functions) was an outcome in children with long term hearing loss.

Sininger (1999) established that paediatric hearing loss adversely affects development of auditory skills and speech and language (Geers and Moog, 1994). In particular, bilateral severe, unilateral, mild or moderate, and profound loss can cause significant effects on speech and language development, if early detection is ignored (Erenberg et al, 1999; Geers and Moog, 1989; Thompson et al, 2001). Comparing the results of observed this study with the literature, it could be anticipated that the children diagnosed with hearing impairment may present with speech and language developmental challenges. The negative impact on families of children with hearing impairment has also been mentioned and typically bears on the parent's response to the diagnosis and adaptation to the children's needs (Feher-Prout, 1996, Young and Tattersall, 2007).

Family history of hearing loss was reported by 6.3% of mothers. This observation could arise from congenital infections resulting in auditory damage. The findings in this study are supported

by Willems (2000) who supposed the existence of genetic components and gene linkage to with susceptibility to hearing loss, and further confirmed by Canalis (2000), that hearing loss arising from genetic factors are deemed to cause more than 50% of all incidents of congenital hearing loss in children. These genetic factors, as suggested by Canalis (2000) may be autosomal dominant, autosomal recessive, or X-linked (related to the sex chromosome) and hence the associated hearing loss could be hereditary or due to other factors present either in utero (prenatal) or at the time of birth.

The present study however showed that most of the 72 children suspected of hearing loss did not show any family histories of hearing loss. This observation is in agreement with Olusanya et al., (2007) that 798,000 out of the 133 million (0.6%) annual live births in developing countries are likely to present with permanent congenital and early-onset hearing loss (Olusanya, 2007), and also with White (2004) that the incidence of congenital hearing loss in developed countries was estimated at 2 to 4 per 1000 live births (0.2% to 0.4%). On the contrary, Amedofu et al., (1997) reported in a study that 51.5% of 128 children aged between 1-5 years and attending the Ear, Nose and Throat clinic at the Komfo Anokye Teaching Hospital (KATH) presented with congenital sensorineural hearing loss (CSHL).

Hicks and Bacon (1999) reported that several medications or drugs such as salicylates, aminoglycoside antibiotics and loop diuretics showed potential harmful effects and adversely affected hearing. Whereas no indications of use of such drugs were reported by the mothers, alcohol (considered a drug) intake or consumption was nonetheless reported among pregnant mothers surveyed in this study. The prevalence was however was very low (2%). Majority of the mothers (85.4%) had full term delivery and more than 74.0% had spontaneous vaginal delivery

while 18.8% were in labour for 24 hours. Other experiences (premature rupture of membrane, bleeding during labour forceps delivery, labour induced during delivery and labour induced) were reported by less than 10% of the respondents.

### **5.3.2 Risk factors of Infant/newborn**

The various factors which pre-dispose infants and newborns to developing hearing loss were investigated in this study. About 23% of mothers in the study confirmed that their newborns had breathing problems at birth and had been admitted to neonatal intensive care units (NICUs), while 24% of them had anoxia. These findings are in agreement with Marlow et al., (2000), and Billings and Kenna (1999) who reported that studies on premature babies, especially those who spent time in NICU, showed an increased risk of SNHL compared to full-term infants. More than 10% of children had experienced neonatal jaundice or seizures during their infancy. Low birth weight (8.9%) and congenital heart disease (1.0%) was also present in these children. These results are consistent with the findings of Northern and Downs (1991) that low birth weight is associated with hearing loss in children. The results are further confirmed by Lewis (2000), who revealed that other factors influencing birth weight may also directly contribute to hearing loss.

Cerebral palsy was reported among 11.5% of the children in this present study. However seizure disorders and head trauma was low in these children. Meningitis, measles and cerebral malaria accounted for hospitalization of some of the children. These infections could contribute to the observations of hearing loss in some of the children. As reported in the literature, childhood infections such as meningitis, encephalitis, and head injury can also cause hearing loss (Eavey et al., 1995).None of the children in this study was hospitalized for encephalitis.

The most prevalent conditions diagnosed at birth in these children were chicken pox (4.2%), sickle cell disease (2.1%) while none was diagnosed with rubella. Most importantly, 21.9% of the children in this study were diagnosed with ear infection approximately 2 years after birth. These findings suggest that ear infections may have some level of influence on the development of hearing loss among children. Also, early detection of these risk factors can lead to prevention of severe and permanent hearing loss among these infants. In all protocols developed by ASHA, AAA and BSA, early detection of warning signs as well as the disease itself among both the parent and infant is emphasized to ensure that severe and permanent hearing losses are prevented.

## **5.4 AUDIOLOGICAL TESTING**

### **5.4.1 Otoacoustic Emissions**

Otoacoustic emissions, VRA and play audiometry tests were performed on the children. According to Stach (2003), OAEs specifically provide information related to the function of the outer hair cells and has been significantly used in routine audiological assessments in newborn hearing screening programmes. They are also used to verify behavioral responses and obtain additional frequency-specific information in pediatric populations. In-line with ASHA (2004) recommendations which require that audiology assessment of infants and young children should include thorough case histories, otoscopy, and behavioral and physiologic measures, OAE tests were done.

The OAE results showed less passes (40.1%) than refers (59.9%) in both ears. This is suggestive that about 40.0% of the children had normal outer hair cell function. The passes in the right ear (40.6%) were marginally higher compared with the left ear (39.6%), while the converse was true

for refers (right ear: 59.4%; left ear: 60.4%). According to the literature (Kemp *et al.*, (1990; Gorga, *et al.*, 1993), the pediatric audiology test battery is expanded by OAEs via provision of a physiologic means of assessing' preneural auditory functions. Although relations exist between OAEs and behavioral thresholds (Martin *et al.*, 1990; Gorga, *et al.*,2002) as well as improvement in strategies for predicting thresholds using OAEs (Boege and Janssen, 2002), variability among individuals suggests the exercise of caution in attempting to predict behavioral thresholds from OAEs(Gorga *et al.*, 2003). Hence, on the bases of the OAE pass and refer rates, it was needful to avoid assigning behavioural assessments on the children in this study in accordance with the literature.

The association between the OAE tests and demographics (age and gender) were investigated. The youngest group (0.5– 2.5 years) of children constituted the largest population and also presented with most passes ( $n=41/77$ , 53.2%) as well as refers ( $n=67/115$ , 58.3%). In general, the comparisons of the OAE test results with age and gender revealed non-significant associations for right and left ears.

#### **5.4.2 Visual Reinforcement Audiometry and Play Audiometry**

Visual reinforcement audiometry was used to assess 44 out of the cohort of 96 children aged 6months to 30 months and presenting with developmental delay. This VRA procedure allowed for assessment of hearing thresholds with the presentation of a stimulus at the selected test frequencies.

Normal hearing was least prevalent across all test frequencies. The most prevalent degrees of hearing loss at low frequency (500 Hz) were severe hearing loss (20.5% for both ears), profound hearing loss (25.0% for both ears) and mild hearing loss (right ear: 29.5%; left ear: 20.5%). At

higher frequencies (1 kHz – 2 kHz), mild (29.5% for both ears) and profound (right ear: 29.5%; left ear: 27.3%) hearing losses were most prevalent.

Investigation of associations between hearing loss as measured by VRA and age at the test frequencies were performed. The inferential statistical (chi-square tests and *p*-value calculations) established non-significant association between hearing loss via VRA audiometry and age at the test frequencies. Hearing loss was most prevalent among the youngest (0.5years to 2.5 years) children across all the test frequencies. In particular, more children presented with severe (20.5%), mild (22.7%) and profound (25%) hearing losses for the 0.5 – 2.5 year olds in both ears. Mild and profound hearing losses were most prevalent at 1000 Hz. For both degrees of hearing loss, the differences in the number of children presenting with hearing loss in the right ear (27.3%) were marginally lower compared to the left ear (29.6%). The prevalence of profound hearing loss was highest for both ears (right ear: 32.8%; left ear: 27.3%). However, mild hearing loss was more prevalent in the left ear (29.6%). The results of this study are consistent with the findings of Joanne *et al.*, (2000) which established a significant positive correlation between age and hearing loss among a pediatric population during the first 5 years of life. Similarly, Rose *et al.*, (2003) suggested that the prevalence of hearing loss is less for younger children for both gender.

With respect to gender, the statistical analysis established no significant association between hearing loss via VRA tests and gender among the 44 children in both ears. More males (59.1%) than females (40.9%) underwent VRA audiometric tests. Mild and profound hearing losses were the most prevalent degrees of hearing loss in both ears across the range of test frequencies. Although mild hearing loss was higher in males compared to females in the right ear, the reverse

trend was true for profound hearing loss where the prevalence in males was lower than in females. In particular, hearing loss was generally more prevalent in males in all the degrees of hearing loss in both right and left ears with the exception profound hearing loss in the right ear and severe hearing loss in the left ear at 2000Hz.

The findings of this study agree with the literature (Gunasekera, *et al.*, 2008) which reported a non-significant association between gender and hearing loss. Furthermore, the findings reported by Lu *et al* (2011) which established no significant correlation between hearing loss and gender among pre-school children confirm the present study. The observed higher prevalence of hearing loss in the male children could therefore be attributable to the higher male population. On the contrary, a study conducted in Iran showed hearing loss was significantly more common in boys (Sarafraz and Ahmadi, 2009).

Thirty six children were tested with play audiometry. Severe (right ear: 22.2%; left ear: 25.0%) and profound (right ear: 27.8% - 33.3%); left ear: 27.8%) hearing losses were most prevalent across the test frequencies. A non-significant association between hearing loss as measured by play audiometry and age was established across the range of test frequencies. The degree of hearing loss varied with age. In particular, the youngest group of children presented with most degrees of hearing loss in both ears of which severe and profound hearing losses were the most prevalent. The variation in the older 6.6 – 10.0 year old was least. Similarly, the inferential statistics established no significant associations between hearing loss (via play audiometry) and gender across the range of frequencies for both ears.

### 5.4.3 Tympanograms

Tympanometry was performed to assess the middle ear function of the 96 children. Significant associations between tympanograms and OAE test results were established for right and left ears. Thirty seven ears had Type A tympanograms, and 11 had Type B tympanograms. All right ears with Type C tympanograms (11 ears) referred in the OAE test. For the left ear, the number of passes ( $n=37$ , 48.7%) was marginally lower compared to the referrals ( $n=39$ , 51.4%) with Type A tympanograms ( $n=76$ ). All left ears with Type B tympanograms (12) referred in the OAE tests, while only 12.5% passed with Type C tympanograms ( $n=8$ ).

The pass and refer rates with Type A tympanograms suggest normal middle ear function in the children. The 100% referral rates with Type B tympanograms with normal ear canal volume in both ears are suggestive of the presence of fluid in the ears and possible otitis media in the children (Katz, 1994). Since otitis media is a medical condition, further medical assessments would be required for confirmation. The fact that the subjects with Type C tympanograms passed OAE suggests that the negative middle ear pressure has had very little adverse effect (if any) on their hearing. The contrary is true for subjects with Type C tympanograms who referred OAE and signify the presence of adverse effects of negative middle ear pressure on their hearing.

Equal numbers (31 at 500 Hz, 32 at 1000 Hz and 31 at 2000 Hz) of the right ears with Type A tympanograms presented with a hearing loss ranging from mild to profound. There was a significant association between VRA test results and tympanograms for left ears at the high frequencies (1000 Hz – 2000 Hz) except at 500 Hz. Similarly, majority of the ears at 500 Hz and 1000 Hz and 2000 Hz with Type A tympanograms presented with hearing loss ranging from mild to profound in the play audiometry tests for right and left ears. However, no significant

association between play audiometry test results and tympanograms at all tested frequencies for right and left ears were found.

## CHAPTER SIX

### CONCLUSION, RECOMMENDATIONS AND LIMITATIONS

#### 6.1 INTRODUCTION

This research study was conducted to establish the prevalence and identify risk factors of hearing loss among on a cohort of 96 pediatric patients aged 0.5 – 10.0 years at a tertiary teaching hospital in Accra, Ghana. Audiological test batteries including OAE, VRA, play audiometry, distraction assessments and tympanometry were employed and inferential statistics was applied to analyze the tests results with respect to age and gender for purposes of establishing any associations with hearing loss and risk factors.

#### 6.2 CONCLUSION

The findings from the study showed:

- that severe hearing loss, mild hearing loss and profound hearing loss were the most prevalent degrees of hearing loss at all test frequencies were and were higher in the 0.0 – 2.5 year olds.
- non-significant associations between the OAE test results, and age as well as gender, in both ears
- non-significant associations were established between various assessments (VRA, play audiometry, distraction) and age and gender respectively
- significant associations were established between tympanometry, and OAE and VRA results respectively for both ears. On the contrary, no significant association was found between tympanometry and play audiometry

- that meningitis, measles admission into NICUs and anoxia, low birth weight and congenital heart diseases in the pediatric patients, and anaemia in their mothers were the most common risk factors.

### **6.3 RECOMMENDATIONS**

On the bases of the findings of this study, the following recommendations are suggested:

- There is the need to institute a mandatory screening programme for all newborns before discharge from hospitals to ensure early detection and intervention of hearing loss.
- Research and development in hearing impairment among children must be encouraged to realize important findings for policy development and effective implementation of research outcomes
- Information flow between professional staff at hearing assessment centres or clinics and other health personnel at the obstetrics and gynaecological department must be enhanced to detect, minimize and/or prevent identified risk factors at the pre-natal and peri-natal stages of birth
- Strengthening of public awareness programs regarding the need for hearing assessment especially among children must be encouraged
- A prospective longitudinal study over a longer period would allow adequate identification of risk factors and early detection.

### **6.4 LIMITATIONS**

The study was limited by sample size and the use of non-probability sampling methods which may limit the findings to the current population and presentation.

## REFERENCES

- Access Economics (2006). Listen, Hear! The economic impact and cost of hearing loss in Australia. Melbourne: Access Economic. Retrieved from (<http://www.accesseconomics.com.au/publicationsreports/showreport.php?id=71>) [28/7/15]
- Amedofu, G. K., Awuah, P. & Ocansey, G. (2003). Hearing impairment among secondary school students. *Indian Journal of Clinical Practice*, 4(4), 17-20.
- Amedofu, G.K., Opoku- Buabeng, J. & Osei-Bagyina, A. (2003). Hearing loss among school children in Ghana. *Ghana Medical Journal*, 37,148-152.
- Amedofu, G. K., Brobby, G.W., Ocansey, G. (1997). The causes and prevalence of pre-school deafness in Ghana. *Africa Journal Health Science*, 4(1-3), 29-32.
- American Speech-Language-Hearing Association (2014). Causes of hearing loss. Available online at: [www.asha.org/public/hearing/disorders/causes.htm](http://www.asha.org/public/hearing/disorders/causes.htm) [28/7/15]
- American Academy of Audiology (2011). Audiologic Guidelines for Paediatric Assessment retrieved from: [www.cdc.gov/ncbddd/hearingloss/documents/aaa\\_childhood-hearing-guidelines\\_2011.pdf](http://www.cdc.gov/ncbddd/hearingloss/documents/aaa_childhood-hearing-guidelines_2011.pdf) [28/7/15]
- Axelsson, G. L. & Naterstad, K. (2003) Development of an Inducible Gene Expression System for Lactobasillus Sakei. *Letters of Applied Microbiology*, 37, 115-120.
- Berrettini, S., Forli, F., Genovese, E., Santarelli, R., Arslan, E., Chilosi, A. M., & Cipriani, P. (2008). Cochlear implantation in deaf children with associated disabilities: Challenges and outcomes. *International Journal of Audiology*, 47, 199–208.

- Billings, K.R., Kenna, M.A. (1999). Causes of pediatric sensorineural hearing loss: yesterday and today. *Archives Otolaryngol Head Neck Surgery*, 125(5), 517-521.
- Borradori, C., Fawer, C.L. & Buclin, T. (1997). Risk factors of sensorineural hearing loss in preterm infants. *Biologic Neonate*, 7, 1.
- Brobby, G.W. (1988). Causes of congenital and acquired total sensorineural hearing loss in Ghanaian Children. *Tropical Doctrine*, 18(1), 30-32.
- Canalis, R. F. & Lambert, P. R. (2000). *The Laryngoscope*, 110 (9), 158.
- Robertson, C. M. T., Howarth, T.M., Bork, D. L. R. & Dinu, I. A. (2009). Permanent bilateral sensory and neural hearing loss of children after neonatal intensive care because of extreme prematurity. *Paediatrics*, 123 (5), 797-807.
- Centers for Disease Control and Prevention (CDC). (2010). Identifying *infants with hearing loss - United States, 1999-2007*. MMWR Morb Mortal Wkly Rep. 59(8), 220-223.
- Cunningham, M., Cox, E. O. (2003). Hearing assessment in infants and children: Recommendations beyond neonatal screening. *American Academy of Pediatrics*, 111(2), 436-440.
- Dai, C., & Gan, R. Z. (2008). Change of middle ear transfer function in otitis media with effusion model of guinea pigs. *Hearing Research*, 243(1-2), 78-86.
- Dhooge, I.J. (2003). Risk factors for the development of otitis media. *Current Allergy and Asthma Reports*, 3, 321-325.
- Eavey, R. D. di Carmo, C., Bertero, M. & Thornton, A.R. (1995). Failure to clinically predict NICU hearing loss. *Clinical Pediatric*, 34,138.

- Eggermont, J. J., Ponton, C.W. (2003). Auditory-evoked potential studies of cortical maturation in normal hearing and implanted children: Correlations with changes in structure and speech perception. *Acta Oto-Laryngologica*, 123(2), 249–252.
- Eggermont, J.J., Ponton, C. W., Don, M., Waring, M.D., Kwong, B. (1997). Deprivation-induced cortical reorganization in children with cochlear implants. *Acta Oto-Laryngologica*, 117(2), 161–163.
- Eileen, D. (2007). Noise and hearing loss: A review. *Journal of School Health*, 77 (5), 225–231.
- Erenberg, A., Lemons, J., Sia, C., Trunkel, D. Ziring, P. (1999). Newborn and infant hearing loss: Detection and intervention. American Academy of Pediatrics. Task Force on Newborn and Infant Hearing, 103(2), 527-30.
- Feher-Prout, T. (1996). Stress and coping in families with deaf children. *Journal of Deaf Studies and Deaf Education*, 1(3), 155-166.
- Feldman, R. (2006). From Biological Rhythms to Social Rhythms: Physiological Precursors of Mother Infant Synchrony. *Development Psychology* 42, (1), 175-88.
- Fortnum, H. M., Summerfield, A. Q., Marshall, D.H., Davis, A. C. & Bamford, J .M. (2001). Prevalence of permanent childhood hearing impairment in the United Kingdom and implications for universal neonatal hearing screening: questionnaire based ascertainment study. *British Medical Journal*, 3239(7312), 536–40.
- Geers A. (2006). Factors influencing spoken language outcomes in children following early cochlear implantation. *Advance Otorhinolaryngol*, 64, 50-65.
- Gelfand, S. A. (2009). *Essential of Audiology*. (3<sup>rd</sup>. Ed). New York: Thieme Medical Publishers, Inc.

- Greenberg, R.S., Daniels S.R., Flanders, W.D., Eley, J.W. & Boring J.R. (2005). *Medical Epidemiology* McGraw Hill, 4<sup>th</sup> Ed.
- Hellstrom-Lindahl, E., Gerbounova, O., Seiger, A., Mousavi, M., & Nordbieg (1998). Regional distribution of nicotinic receptors during parental development of nicotine –affected development. *Human brain and Spinal Cord Develop Resource*, 108, 147-160.
- Hicks, M. L., & Bacon, S. P. (1999). Effects of aspirin on psychophysical measures of frequency selectivity, two-tone suppression, and growth of masking. *Journal of the Acoustical Society of America*, 106(3), 1436–1451.
- ISO 389 (1991). *Specification for standard reference zero for the calibration of pure tone air conduction audiometers*, International Organization for Standardization, Geneva.
- Katz, J. (2002). *Clinical Audiology*. (5 Ed). Maryland: Library of congress cataloguing-n-publishing data.
- Joanne, E.R., Margaret, R. B., Sandra, C. J., Stephen, R. H., Jackson, R., Martha, M., Eloise, C. N. & Susan, A. Z. (2000). Otitis media in early childhood in relation to preschool language and school readiness skills among black children. *Pediatrics*, 106 (4), 725- 736.
- Joint Committee on Infant Hearing. (2007). Year 2007 Position Statement: principles and guidelines for early hearing detection and intervention programs. *Pediatric*, 120,898.
- Kancherla, V., Naarden, V., Braun, K., Yeargin-Allsopp, M. (2013). Childhood vision impairment, hearing loss and co-occurring autism spectrum disorder *Disability and Health Journal*.
- Karosi, T., Sziklai, I. (2010). Etiopathogenesis of otosclerosis. *European Archives Otorhinolaryngol*, 267, 1337–1349.

- Kemper, A. R., & Downs, S. M. (2000). A cost-effectiveness analysis of newborn hearing screening strategies. *Archives of Pediatric and Adolescent Medicine*, 154 484-488.
- Kennedy, C. R., McCann, D. C., Campbell, M. J., Law, C. M., Mullee, M., Petrou, S. & Stevenson, J. (2006). Language ability after early detection of permanent childhood hearing impairment. *New England Journal of Medicine*, 354(20), 2131– 2141.
- Korver, A. M., Konings, S., & Dekker, F.W. (2010). Newborn hearing screening vs. later hearing screening and developmental outcomes in children with permanent childhood hearing impairment. *Journal of the American Medical Association*, 304, 1701-8.
- Kral, A., O'Donoghue, G. M. (2010). Profound deafness in childhood. *New England Journal of Medicine*, 363, 1438-50.
- Lee, D.S., Lee, J.S., Oh, S.H., Kim, S.K., Kim, J.W., Chung, J. K., Lee, M.C. & Kim, C.S. (2001). Cross-modal plasticity and cochlear implants. *Nature*, 409 (6817), 149–150.
- Lewis, W. H., (2000). Gray's Anatomy of the Human Body, 201b Ed. On-line at <http://www.bartleby.com>
- Lehnhardt, E., Laszig, R., (2009). Praxis der Audiometrie Stuttgart: Georg Thieme Verlag, 47–52
- Lü, J., Huang, Z., Yang, T., Li, Y., Mei, L., Xiang, M., Chai, Y., Li, X., Li, L., Yao, G., Wang, Y., Shen, X. & Wu, H. (2011). Screening for delayed-onset hearing loss in preschool children who previously passed the newborn hearing screening. *International Journal of Pediatric Otorhinolaryngol*, 75 (8), 1045-9.
- Mathers C, Smith A, Concha M. (2000). *Global Burden of Hearing Loss*. [online] retrieved from: [www.who.int/healthinfo/statistics/bod\\_hearinglosspdf](http://www.who.int/healthinfo/statistics/bod_hearinglosspdf). [Accessed 26.08.2014]

- Mason, I. A., Herrmann, K. R. (1998). Universal infant hearing screening by automated auditory brainstem response measurement. *Pediatrics*, 101(2), 221-228.
- Marthers, C., Smith, A., Concha, M. (2000). Global Burden of Hearing Loss. [online] Available from: [www.who.int/healthinfo/statistics/bod\\_hearinglosspdf](http://www.who.int/healthinfo/statistics/bod_hearinglosspdf). [Accessed 26.08.2014]
- Moeller, M. (2000). Early intervention and language development in children who are deaf and hard of hearing. *Pediatrics*, 106(3), 1–9.
- Mehra, S., Eavey, R. D., Keamy, D. G. (2008). The Epidemiology of Hearing Impairment in the United States: Newborns, Children and Adolescents. *American Academy of Otolaryngology-Head and Neck Surgery Foundation*, 140(40), 461-472.
- Mytton, J. & Mackenzie, I. (2005) Observed and expected prevalence of permanent childhood hearing impairment in Oldham. *Journal of Public Health*, 27(3), 298-302.
- National Institute of Health (NIH) (2010). Health Statistics.  
E20<http://www.nidcd.nih.gov/health/statistics/Pages/hearing-aids-adults.aspx>
- Neville, H., Bavelier, D. (2002). Human brain plasticity: evidence from sensory deprivation and altered language experience. *Program Brain Resource* 138, 177–188.
- Niedzielska, G., Katska E., Kusa W. (2000). *Hearing loss in chronic alcoholics. Anales Universitatis Marrie Curie - Sklodowska Medical*, 56, 99 – 101.
- Nishimura, H., Doi, K., Iwaki, T., Hashikawa, K., Oku, N., Teratani, T., Hasegawa, T., Watanabe, A., Nishimura, T., Kubo, T. (2000). Neural plasticity detected in short and long-term cochlear implant users using PET. *Neuroreport*, 11, 811–815.
- Nishimura, H., Hashikawa, K., Doi, K., Iwaki, T., Watanabe, Y., Kusuoka, H., Nishimura, T., Kubo, T. (1999). Sign language ‘heard’ in the auditory cortex. *Nature*, 397(6715), 116.

- Northern, J. L., & Downs, M. P. (1991). *Hearing Loss in Children* (4<sup>th</sup>Ed.) U.S.A. William and Wilkins.
- Olusanya, B.O., Okolona, A.A., Ijaluola, G.T, (2000). The hearing profile of Nigerian school children. *International Journal Pediatric Otorhinolaryngol*, 55 (3), 173-9.
- Olusanya, B. & Okolona, A.A. (2006). Early Hearing Detection at Immunization Clinics in Developing Countries. *International Journal of Pediatric Otorhinolaryngology*, 70, 1495-1498.
- Olusanya, B.O., Newton, V.E. (2007). Global burden of childhood *hearing impairment and disease control priorities for developing countries*. *Lancet*, 369, 1314-7.
- Pau, H. W., Sievert, U., Just, T. & Sade, J. (2009). Pressure changes in the human middle ear without opening the Eustachian tube. *Acta Oto-Laryngologica*, 129, 1182–1186.
- Petitto, L.A., Zatorre, R.J., Gauna, K., Nikelski, E. J., Dostie, D, & Evans, A. C. (2000). Speech-like cerebral activity in profoundly deaf people processing signed languages: implications for the neural basis of human language. *Proceedings of National Academy Sciences USA*, 97(25), 13961–13966.
- Paludetti, G, Conti, G., D.I., Nardo, W.D.E., Corso, E., Rolesi, R., Picciotti, P.M., Fetoni, A.R. (2012). Infant hearing loss: from diagnosis to therapy Official Report of XXI Conference of Italian Society of Pediatric Otorhinolaryngology. *Acta Otorhinolaryngol Italian*. 32(6), 347-70.
- Picard, M. (2004). Children with permanent hearing loss and associated disabilities: Revisiting current epidemiological data and causes of deafness. *The Volta Review*, 104, 221–236.

- Ponton, C.W., Vasama, J.P., Tremblay, K., Khosla, D., Kwong, B., & Don, M. (2001). Plasticity in the adult human central auditory system: evidence from late onset profound unilateral deafness. *Hearing Research*, 154, 32-44.
- Ponton, C., Don, M., Eggermont, J.J., Waring, M.D., Kwong, B., & Masuda, A. (1996). Auditory system plasticity in children after long periods of complete deafness. *Neuroreport*, 8, 61-65.
- Probst, F. J., Fridell, R. A., Raphael, Y., Saunders, T. L., Wang, A., Liang, Y., Morell, R. J., Touchman, J. W., Lyons, R. H., Noben-Trauth, K., Friedman, T. B. & Camper, S. A. Genetics of Nonsyndromic Recessively Inherited Moderate to Severe and Progressive Deafness in Humans 271 (1998). Correction of deafness in shaker-2 mice by an unconventional myosin in a BAC transgene. *Science*, 280, 1444-7.
- Prizant, M.B.& Myer, E. C.(1993) Socioemotional Aspect of Language and Social Communication Disorder in Young Children and their families. *American Journal of Speech –Language pathology*, 2, 56-71.
- Ptok, M. and Ptok A. (2001) Etiology Profile of the patients implanted in the cochlear implant program. *The international journal of otolaryngology and head and neck surgery*, 4, (3), 870-876.
- Richard, J. H., Smith, M. D., Bale J.F., White, K.R., (2005). Sensorineural hearing loss in children. *The Lancet*, 365 (9462), 879-890.
- Roder, B., Stock, O., Bien, S., Neville, H., Rosler, F. (2002). Speech processing activates visual cortex in congenitally blind humans. *European Journal Neuroscience*, 16, 930–936.

- Rose, L. A., Andrew, S., Deborah, E. & Saravana, E., (2003). Pre-school hearing screening. Pass/Refer rates for children enrolled in a head start program in Eastern North Carolina. *American Journal of Audiology*, 16(1), 4-12.
- Rovet, J., Walker, W. & Bliss, B., (1996). Long-term sequelae of hearing impairment in congenital hypothyroidism. *Journal of Pediatrics*, 128,776.
- Ruben R.J. (2000). “Redefining the survival of the fittest: communication disorders in the 21<sup>st</sup> Century” *The Laryngoscope*, 110, 241-245.
- South African Hearing Institute (2000). Hearing loss incidence in South Africa.  
[http://www.sahi.org.za/hearing\\_loss\\_statistics.html](http://www.sahi.org.za/hearing_loss_statistics.html)
- Sarafraz, M. & Ahmadi, K. H. (2009). A practical screening model for hearing loss in Iranian school-aged children. *World Journal of Pediatrics*, 5(1), 64-72.
- Sataloff, R.T. & Sataloff, J. (1993). Occupational Hearing Loss, 2<sup>nd</sup> Ed. Marcel Dekker, Inc., New York, NY.
- Seely, D.R., Gloyd, S.S., & Wright, A. D., Norton, S. J., (1995). Hearing loss prevalence and risk factors and Sierra Leonean children. *Otolaryngol Head and Neck Surgery*, 121 (8), 853-8.
- Sharma, A., Gilley, P. M., Dorman, M. F., Baldwin, R. (2007). Deprivation-induced cortical reorganization in children with cochlear implants. *International Journal of Audiology*, 46(9), 494–499.
- Sharma A., Dorman, M .F. & Kral A. (2005). The influence of a sensitive period on central auditory development in children with unilateral and bilateral cochlear implants. *Hearing Research*, 203, 134–143.
- Sharma, A. (2005). Latency as a biomarker for central auditory development in children with hearing impairment), *Journal of the American Academy of Audiology*, 16, 564–573.

- Smith, A.W. (2001). WHO activities for prevention of deafness and hearing impairment in children. *Scandinavian Audiology Supplementum*, 30, 93-100.
- Smith, T., Groen, A.D., & Wynn, J.W. (2008). Randomised trial of intensive early intervention for children with pervasive developmental disorder. *American Journal of Mental Retardation*, 105(4), 269-85.
- Strickland, D. S., & Shanahan, T. (2004). Laying the groundwork for literacy. *Educational Leadership*, 61(6), 74–77.
- Sue, C. M., Lipsett, L. J., Crimmins, D. S., DipAud, C. S. T., Boyages, S. C., Presgrave, C. M., Gibson, (1998). Cochlear origin of hearing loss in MELAS syndrome. *Annal of Neurology*, 43,350–359.
- Swanepoel, D., Störbeck, C., Friedland, P. (2009). Early hearing detection and intervention services in South Africa. *International Journal of Pediatric Otorhinolaryngol*, 73(6),783-6.
- Taha A. A., Pratt S. R., Farahat, T. M., Abdel-Rasoul, G. M., Albatony ,M. A., Elrashiedy, A. L., Alwakeel, H. R., Zein, A. (2010). Prevalence and risk factors of hearing impairment among primary school children in Shebin El-Kon District, Egypt. *American Journal of Audiology*, 19 (1), 46-60.
- Tharpe, A.M., Ricketts, T., Sladen, D.P. (2003). FM Systems for children with minimal to mild hearing loss. *Access Achieving Clear Communication Employing Sound Solutions*. Proceeding of Chicago Conference, Chapter 20, 191.
- Tucci, D. L., Merson, M. H., Wilson, b. s. (2010). A Summary of literature on global hearing impairment: Current status and priority for action, 31 (1), 31-41.

- Thompson, D. C., McPhillips, H., David, R. L., Lieu, T.L., Homer, C. J., Helfand, M. (2001). Universal newborn hearing screening: Summary of evidence. *Journal of the American Medical Association*, 286, 2000-10.
- Traxler, C. B. (2000). The Stanford Achievement Test, 9th Edition: National norming and performance standards for deaf and hard-of-hearing students. *Journal of Deaf Studies and Deaf Education*, 5(43), 337-348.
- Werker, J., Fennell, C., Corcoran, K., & Stager, C. (2002). Infant's ability to learn phonetically similar words: Effects of age and vocabulary size. *Infancy*, 3, 1-330.
- Werker, J., Polka, L., & Pegg, J. (1997). The conditioned head turn procedure as a method for testing infant speech perception. *Early Development and Parenting*, 6, 171-178.
- Werker, J., & Tees, R. (1984). Cross-language speech perception: Evidence for perceptual reorganization during the first year of life. *Infant Behaviour and Development*, 25(1), 121-133.
- Werker, J. & Tees, R. (1987). Speech perception in severely disabled and average reading children. *Canadian Journal of Psychology*, 41, 48-61.
- White, K. R., Forsman, I., Eichwald, J., Munoz, K. (2014). The evolution of early hearing detection and intervention programs in the United States. *SeminPerinatol*, 2010(34), 170-9.
- World Health Organization, Deafness and hearing loss. [Online]. Available from: [www.who.int](http://www.who.int).
- White, K.R. (2003). The current status of EHDI programs in the United States. *Mental. Retardation and Developmental Disabilities Research Review*, 9(2), 79-88.

- White, K.R., Vohr, B.R., Maxon, A.B., Behreus, T.R., McPherson, M.G., & Mauk, G.W. (1994). Screening all newborns for hearing loss using transient evoked otoacoustic emissions. *International Journal of Pediatric Otorhinolaryngology*, 29(3) 203–217.
- Willems P.J. (2000). Genetic causes of hearing loss. *New England Journal of Medicine*, 2000(342), 1101-1109.
- World Health Organization (2010). Deafness and hearing impairment. *Fact Sheet No 300*. Geneva.
- World Health Organization (2012), Deaf and hearing impairment, Geneva (Retrieved online <http://www.who.int/mediacentre/factsheets/fs300/en/index.html>)
- Young, A., Tattersall, H., Uus, K., Bamford, J., McCracken, W. (2003) Universal Newborn Hearing Screening. *Australian Journal of the Education of the Deaf*, 2003, (9) 5-10.
- Yoshinaga-Itano, C. (2004). Levels of evidence: Universal newborn hearing screening and early hearing detection and intervention systems. *Journal of Communication Disorders*, 37(5), 451-465.
- Yoshinaga-Itano, C. (1999). Benefits of early intervention for children with hearing loss. *Otolaryngologic Clinics of North America*, 32(6), 1089-1103.
- Yoshinaga-Itano, C., Sedey, A.L., Coulter, D.K., & Mehl, A.L. (1998). Language of early & later identified children with hearing loss. *Pediatrics*, 102(5), 1161-1171.
- Zahnert, T. (2010). Schwerhörigkeit – Ätiologie, Diagnostik und auditive Rehabilitation. *Laryngo-Rhino-Otologie*, 89(11), 669–93.

## APPENDIX I

### INFORMED CONSENT

Dear Respondent:

This survey is designed to **study “the prevalence and some causes of hearing loss among children”**.

The research is purely for academic purpose and your responses to these questions will be kept under strictest confidentiality.

Thank you in advance for your cooperation.

### INFORMED CONSENT FORM

PARTICIPANT HOSPITAL ID NUMBER: \_\_\_\_\_

PARTICIPANT NAME: \_\_\_\_\_

### **STUDY TITLE: The prevalence and some causes of hearing loss among children.**

Hearing loss is a major health condition among children in Ghana. The prevalence and the risk factors of this condition are currently unknown. This is the first time a study of this nature is being conducted in Ghana. Prof. Geoffrey Amedofu, Dr. S. Anim-Sampong and Rejoice Afi Acquah from Hearing Assessment Centre and School of Biomedical and Allied Health Sciences, University of Ghana are conducting a research to determine the prevalence and some causes of hearing loss among children in Ghana.

You are to understand that taking part in the research is entirely voluntary. You are further to note that you may refuse to take part or withdraw from the research at any time without anyone objecting.

We are going to ask you to provide information about yourself, your health and that of your child. You may feel uncomfortable providing such information. You are likely to spend the best part of the morning at the survey site. We will ask you to provide information about yourself, your health and that of your child. There will be no extra charges to you during this study.

The study will enable us to determine the prevalence and some causes of hearing loss among children in Ghana and this will help in improving their care and management.

Do you have any questions about what I have told you or what we are going to do for you? Should you later have any question relating to this study clarified, please do not hesitate to contact us at the address and telephone numbers below.

## CONSENT

I have fully explained to ..... the nature and purpose of the above described study and the inconveniences that are involved in its performance. I have answered and will answer to the best of my ability, all questions relating to the study.

_____	_____	_____
SIGNATURE	FULL NAME OF RESEARCHER	DATE
Prof. G. K. Amedofu (Hearing Assessment Centre, Department of Ear, Nose and Throat, P. O. Box 77, Korle Bu Teaching Hospital, Accra. (Tel: 0244377392) and Dr. Anim-Sampong (School of Biomedical and Allied Health Sciences, (Tel: 0207774000) will be available to answer any questions you may have.		

I ..... have read (or have read to me in a language that I fully understand) the proposed study and that I have understood what is going to be done. Also, all questions have been fully answered to my satisfaction. My signature or thumbprint below indicates that I have understood what is going to be done and that I agree to take part in the study.

_____	Date: _____
(Signature/thumbprint of Subject)	

**APPENDIX II****QUESTIONNAIRE****QUESTIONNAIRE ON THE PREVALENCE AND CAUSES OF HEARING LOSS  
AMONG CHILDREN AT THE HEARING ASSESSMENT CENTER**

1. Coded name of child \_\_\_\_\_
2. Age of Child \_\_\_\_\_
3. Parents/Mothers age \_\_\_\_\_
4. Gender            Male       Female
5. Age suspected of hearing loss \_\_\_\_\_

**FAMILY HISTORY**

6. Are parents' blood relations?            Yes       No
7. Family history of hearing loss            Yes       No
8. Another affected child with hearing loss            Yes       No
9. Any affected siblings with hearing loss?            Yes       No

**MATERNAL FACTORS**

10. Apart from the routine drugs, were there any drugs given during pregnancy? Yes  No   
If yes, specify \_\_\_\_\_

11. Was mother hospitalized during pregnancy? If yes why?  
\_\_\_\_\_  
\_\_\_\_\_

12. Maternal illness during pregnancy

- |              |                              |                             |
|--------------|------------------------------|-----------------------------|
| Bleeding     | Yes <input type="checkbox"/> | No <input type="checkbox"/> |
| Anaemia      | Yes <input type="checkbox"/> | No <input type="checkbox"/> |
| Diabetes     | Yes <input type="checkbox"/> | No <input type="checkbox"/> |
| Preeclampsia | Yes <input type="checkbox"/> | No <input type="checkbox"/> |

13. Rh Immunoglobulin given?            Yes       No

14. During pregnancy, was mother exposed to:

Measles	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Mumps	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Chicken Pox	Yes <input type="checkbox"/>	No <input type="checkbox"/>
German measles	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Excessive Noise	Yes <input type="checkbox"/>	No <input type="checkbox"/>

If other; specify

---

15. During pregnancy was mother diagnosed with:

Syphilis	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Herpes Virus	Yes <input type="checkbox"/>	No <input type="checkbox"/>
If other; specify	<input type="checkbox"/>	<input type="checkbox"/>

---

16. During pregnancy was the mother taking alcohol?      Yes       No

### **DELIVERY/LABOUR HISTORY**

17      Full term pregnancy      Yes       No   
 If No, Specify

---

Spontaneous vagina delivery	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Labour induced	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Labour long than 24 hours	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Premature membrane ruptures	<input type="checkbox"/> Yes	<input type="checkbox"/> No
Bleeding	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Forceps/assisted delivery	Yes <input type="checkbox"/>	No <input type="checkbox"/>
Cesarean section	Yes <input type="checkbox"/>	No <input type="checkbox"/>
If yes, give reasons		

---

If other; specify

---

**INFANT NEWBORN FACTORS**

- 18 Small birth weight less than 2kg Yes  No   
 If yes, specify \_\_\_\_\_
- Baby in an intensive care unit Yes  No   
 If yes, specify how long \_\_\_\_\_
- Problems with breathing Yes  No   
 Anoxia Yes  No   
 Congenital Heart Diseases Yes  No   
 Seizures Yes  No   
 Septicaemia Yes  No   
 Neonatal Jaundice Yes  No   
 Paralysis Yes  No   
 Defects of the ear, nose, and throat Yes  No   
 If other; specify \_\_\_\_\_

**INFANT/CHILDHOOD HISTORY****19. Child diagnosed of**

- Cerebral palsy Yes  No   
 Seizure Disorder Yes  No   
 Head trauma/ Skull Yes  No   
 If other; specify \_\_\_\_\_

**20. Ever hospitalized for**

- Meningitis Yes  No   
 Cerebral malaria Yes  No   
 Encephalitis Yes  No   
 Measles Yes  No   
 If other; specify \_\_\_\_\_

**21. Child diagnosed of**

- Rubella Yes  No   
 Mumps Yes  No



**AUDIOMETRIC DATA FORM**  
**DEPARTMENT OF AUDIOLOGY, SPEECH AND LANGUAGE THERAPY-SBAHS**  
**COLLEGE OF HEALTH SCIENCES, UG-KORLE BU**

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**PREVALENCE AND RISK FACTORS OF HEARING LOSS AMONG CHILDREN AT**  
**THE HEARING ASSESSMENT CENTER KORLE BU**

**Audiometric Results**

**Table A1: Audiometric results**

Ear	Frequency			Ear	OAE	
	500 Hz	1000 Hz	2000 Hz		Pass	Fail
Right ear				Right ear		
Left ear				Left ear		

**Table A2: Tympanometry results**

Tympanometry	Left	Right
Ear canal volume		
Peak Compliance		
Peak Pressure		



UNIVERSITY OF GHANA  
SCHOOL OF BIOMEDICAL AND ALLIED HEALTH SCIENCES  
DEPARTMENT OF AUDIOLOGY, SPEECH AND LANGUAGE THERAPY

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November 3, 2014

The Head  
Hearing Assessment Centre  
ENT Department  
Korle Bu Teaching Hospital

Dear Sir/Madam,

**PERMISSION TO CARRY MSc AUDIOLOGY RESEARCH PROJECT AT THE  
HEARING ASSESSMENT CENTRE, KBTH**

The Department of Audiology, Speech & Language Therapy (DAS&LT) of the University of Ghana School of Biomedical and Allied Health Sciences (SBAHS) presents its compliments to you and has the pleasure requesting your kind consideration of the above subject.

Mrs. Rejoice Acquah is a 2<sup>nd</sup> year MSc Audiology student of the Department of Audiology, Speech and Language Therapy of SBAHS, University of Ghana. She is conducting a research project in "Prevalence and Risk factors of Hearing Loss among Pediatric Patients at a Tertiary Teaching Hospital in Accra" under the supervision of Prof. G.K. Amedofu and Dr. S. Anim-Sampong. Her research study has been reviewed and passed by the Department's Ethics and Protocols Review Group of the School as meeting all ethical requirements.

The Department would be most grateful if you could kindly grant her permission to carry out this important research project for the common good of the University and your Centre. Thank you.

Yours faithfully,

A handwritten signature in black ink, appearing to read 'S. Anim-Sampong', written over a horizontal line.

**Dr. S. ANIM-SAMPONG**  
For: (Head of Department)

cc: Dean (SBAHS)  
Prof. G.K. Amedofu

## HEARING ASSESSMENT CENTRE

In case of reply the number  
And the date of this  
Letter should be quoted

My Ref. No.....  
Your Ref. No.....



KORLE BU TEACHING HOSP  
P.O. BOX 77  
KORLE BU, ACCRA

Tel: 233-21- 673033-6  
Fax: 233-21- 667759  
Email: korlebu@ghana.com  
Web Site: www.korlebu.com

17<sup>th</sup> November, 2014

The Head  
Dept. of Audiology, Speech and Language Therapy  
School of Biomedical and Allied Health Sciences  
College of Health Sciences  
University of Ghana

Dear Sir,

**RE: PERMISSION TO CARRY MSc AUDIOLOGY RESEARCH PROJECT  
AT THE HEARING ASSESSMENT CENTRE, KORLE BU TEACHING  
HOSPITAL**

Permission has been granted Mrs. Rejoice Acquah to carry out a research project on "Prevalence and Risk Factors of Hearing Loss among Paediatric Patients at a Tertiary Teaching Hospital in Accra".

She is expected to work closely with the Audiologist in charge to ensure safety of subjects and equipment used.

Yours Sincerely,

E N T CLINIC  
KORLE BU TEACHING HOSPITAL

JEMIMA A. FYNN (MRS.) FWACN  
MSc. AUDIOLOGICAL SCIENCE

Cc: Head, ENT UNIT



UNIVERSITY OF GHANA  
SCHOOL OF BIOMEDICAL AND ALLIED HEALTH SCIENCES  
DEPARTMENT OF AUDIOLOGY, SPEECH AND LANGUAGE THERAPY

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October 7, 2014

Mrs. Rejoice A. Acquah  
Dept. of Audiology, Speech and Language Therapy  
SBAHS,  
Korle Bu

Dear Mrs. Rejoice A. Acquah

**ETHICS CLEARANCE**

Following a technical and professional review of your research proposal by the Department Ethics and Protocol Review Committee and by your supervisors, I am pleased to inform you of the Committee's approval your research proposal entitled:

**"Prevalence and Risk Factors of Hearing Loss Among Pediatric Patients a Tertiary Hospital in Accra"**

This is an initial approval. You are therefore required to obtain a final approval from the School's Ethics and Protocol Review Committee per the Schools regulations.

You are required to work closely and in collaboration with your supervisors. Please report all serious adverse events related to this research to the supervisors and this Committee in writing.

Thank you.

Yours sincerely,

A handwritten signature in black ink, appearing to read 'S. Anim-Sampung'.

**DR. S. ANIM-SAMPONG**

For: Chairman DASL&T Ethics and Protocol Review Committee  
DEPARTMENT OF AUDIOLOGY  
**SPEECH & LANGUAGE THERAPY**  
**SCHOOL OF BIOMEDICAL AND ALLIED**  
**HEALTH SCIENCES**