PREVALENCE OF HEARING IMPAIRMENT AND EAR DISORDERS AMONG SCHOOL CHILDREN IN KIGALI, RWANDA

A CROSS SECTIONAL SURVEY

MUGABO RAJAB MUSTAFA 2009

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DECLARATION

This dissertation is my original work and has not been presented for a degree in any other university.

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This dissertation has been submitted with my approval as the university supervisor.

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DEDICATION

This work is dedicated to:

Almighty Allah for His mercy and blessings.

My sister, Ms Bora Mustafa and my mother, Mrs Saidat Mustafa for all their efforts to lift me to the world of academia.

My wife, Ms Birungi Amida and my daughters, Fath, Thana and Leila for their love and inspiration throughout the course of this study.

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1.0 LIST OF ABBREVIATIONS

ABR	-	Auditory brainstem response
ASR	-	Acoustic stapedial reflex
AOM	-	Acute otitis media
ASOM	-	Acute suppurative otitis media
BAHAs	-	Bone anchored hearing aids
BOR	-	Brancial-oto-renal syndrome
BTE	-	Behind-the -ear
CHL	-	Conductive hearing loss
CMV	-	Cytomegalovirus
CSOM	-	Chronic suppurative otitis media
CT Scan	-	Computerised tomography scan
dB	-	Decibels
DPOAE	-	Distortion product otoacoustic emissions
EcoG	-	Electrocochleography
HI	-	Hearing impairment
HL	-	Hearing level
Hz	-	Hertz
ITE	-	In-the-ear
ITC	-	In-the-canal
kHz	-	Kilo Hertz
MEE	-	Middle ear effusion
NSHI	-	Non-syndromic hearing impairment
OAEs	-	Otoacoustic emissions
OM	-	Otitis media
OME	-	Otitis media with effusion
PTA	-	Pure tone audiometry
PHCI	-	Permanent childhood hearing impairment
SHI	-	Syndromic hearing impairment
SL	-	Sensation level
SOC	-	Superior olivary complex
SPL	-	Sound pressure level
SRT	-	Speech reception threshold
SNHL	-	Sensorineural hearing loss
TEOAEs	-	Transient evoked otoacoustic emissions
WHO	-	World health organistion

2.0 ABSTRACT

Objective: To determine the prevalence of hearing impairment and ear disorders among school children in Kigali, Rwanda.

Design: Descriptive cross-sectional survey.

Participants: School children 6-13 years in the selected schools.

Main outcome measure: Hearing impairment, defined as audiometric threshold values of more than 25 dB HL at any of the testing frequencies (1, 2 and 4 kHz).

Methods and materials: A total of 1073 children from eleven public primary schools in the city were randomly selected and examined. Otoscopy, tympanometry and audiometry screening was carried for all the study participants. Children who failed audiometry screening had pure tone audiometry done to determine their hearing thresholds.

Results: The prevalence of hearing impairment was 13.3% with 11.4% due to conductive hearing impairment, 1.2% sensorineural hearing impairment and 0.9% as mixed hearing impairment. The prevalence of disabling hearing impairment was 1.4%.

The commonest ear disorder was impacted wax found in 18% of the children followed by otitis media with effusion accounting for 6.7%.

Conclusion: The prevalence of hearing impairment in Kigali is high. Conductive hearing impairment is the commonest and most of the causes are preventable and/or treatable.

There is an urgent need to create awareness among the general population and health care providers about the dangers of ear disease and its consequences on hearing and education of the children.

3.0 INTRODUCTION

Hearing impairment among children is a relatively serious problem that can have a significant impact on their development. Consequences of hearing impairment include inability to interpret speech sounds, often producing a reduced ability to communicate, delay in language acquisition, economic and educational disadvantage, social isolation and stigmatization.

Hearing impairment is the most frequent sensory deficit in human populations. According to estimates of the World Health Organization (WHO) in the year 2005, 278 million people (4.6%) worldwide have disabling hearing impairment, with nearly two thirds of these living in poorly developed countries, accounting for approximately 185 million people. [1, 2] The reasons include absence of regular screening programmes for ear disease, poverty, malnutrition, ignorance and paucity of accessible healthcare in poorly developed countries. A further 364 million people are also estimated to have a mild hearing impairment. [1]

It has been estimated that over 1.2 million of children in Sub-Saharan Africa in the 5-14 years age range have a moderate or more severe bilateral hearing loss. [3]

According to the 2002 Rwanda National census, just less than 5 per cent of the population is disabled, but this is likely to be an underestimation. There is no accurate data on prevalence of different types of disabilities but, according to the census, physical disabilities are the most common, followed by deafness, mental deficiencies, blindness and trauma. [4]

Prevalence studies on hearing impairment done in Sub-Saharan Africa have found variable results ranging from 2%-15%. [5] The variability in prevalence depends to some extent on the definition of hearing impairment used, the methodology and/or the population studied. These differences make it difficult to draw valid comparisons from these studies.

In an effort to harmonise and standardise surveys on hearing impairment, WHO (1999) introduced an ear and hearing disorders survey protocol which was followed for this study.

Ear disease and resultant hearing impairment are significant public health issues in developing countries and among many Indigenous populations in developed countries. Ninety-five per cent of school children suffer from middle-ear disease, sometime in the first 10 years of their life. [6] In the auditory-verbal environment of mainstream schools, the resulting hearing-loss can endanger education of the affected children.

Hearing impairment should be identified as early in life as possible, if its long-term consequences are to be prevented. School-age children are in many ways a captive group and it is through the school that they are most accessible. In school-age children even mild and fluctuating losses may impede learning and deny the children the chance to reach their full academic potential. Unfortunately, the instruments required for testing hearing abilities in the young children are not widely available in developing countries. In developed countries, children are screened for hearing-loss routinely at periodic intervals. Implementation of such screening procedures is not feasible in the developing countries at the present moment. Screening at school entry is perhaps the most practical way of ensuring that children are evaluated for hearing capabilities, at least once. However, this is still not feasible in most developing countries including Rwanda.

Before embarking on public health programs to prevent and treat hearing impairment in children, the prevalence of hearing impairment should be determined. There is still a paucity of prevalence information for the majority of countries in sub-Saharan Africa. Without such data, future hearing health service planning and provision will, in many countries, be difficult to implement. Epidemiological data are crucial to the planning and implementation of all audiological and education services for hearing-impaired children. No prevalence studies on hearing impairment have been done in Rwanda and the aim of this study was to determine the prevalence of hearing impairment and ear disorders among school children in Kigali, Rwanda.

4.0 BACKGROUND

4.1 Definitions

There is a diversity of definitions of hearing impairment, thus, comparison among studies is difficult. In previous epidemiologic studies of the prevalence of hearing impairment, investigators have used varying definitions of hearing impairment, making it difficult to compare the results. In this study, the WHO definitions will be used:

Hearing impairment is a broad term used to describe the loss of hearing in one or both ears. It refers to complete or partial loss of the ability to hear from one or both ears.

Hearing loss means any reduction of or difficulties with hearing

Deafness refers to the complete loss of ability to hear from one or both ears/profound hearing impairment.

Disabling hearing impairment in a child less than 15 years of age is defined to be a permanent unaided hearing threshold in the better hearing ear of greater than 30 dB HL average at the frequencies of 0.5, 1, 2, and 4 kHz. [7] However, the concept of permanent childhood hearing impairment (PCHI) is more relevant in a clinical or service development context in developed countries, but less so in a public health context for developing countries.

4.2 Anatomy and physiology of the auditory system

The auditory system is composed of three anatomical compartments: the outer, middle, and inner ear. Sound waves impinging on the head are captured by the pinna and conveyed through the external auditory canal to the tympanic membrane. The vibrations of the tympanic membrane, caused by the airborne sound waves, are transmitted through the middle ear to the inner ear by ossicles. These auditory ossicles consist of the malleus, which is connected to the tympanic membrane; the stapes, which is attached at its base to the oval window of the vestibule; and the incus, which is situated between the malleus and stapes and articulates with both. The sound vibrations of the tympanic membrane are propagated through a piston-like mechanical motion of these ossicles toward the base of the stapes, which moves in and out of the oval window at the entrance to the cochlea. This process amplifies the environmental sound by approximately 20-fold.

The cochlea is the end organ of hearing and is shaped like a snail shell with 2¾ turns. Inside, two membranes longitudinally divide the cochlea into 3 sections: the scala tympani, the scala media, and the scala vestibuli. The cochlear duct is embedded in the perilymph. It is filled with endolymph and contains the organ of Corti between the tectorial and the basilar membranes. The organ of Corti contains two types of sensory cells: a row of inner hair cells and three rows of outer hair cells. The inner hair cells are pure receptor cells that transmit signals to the acoustic nerve and the auditory cortex. The outer hair cells have both sensory and motor elements that contribute to hearing sensitivity and frequency selectivity by amplifying sound reception.

As mechanical sound energy waves transmit through the tympanic membrane, ossicular chain, and oval window, a traveling wave is created through the scala vestibula with a resultant displacement of the basilar membrane and firing of cochlear hair cells. High frequency waves stimulate more proximal portions of the cochlea, while low frequency waves stimulate more distal regions of the cochlea. This neural discharge is tonotopically preserved as it travels through the eighth nerve. With low frequency sounds located ventrolaterally and high frequency sounds more dorsally.

The transfer of neural impulses from the peripheral nervous system to the central nervous system occurs at the cochlear nuclei, located in the lower brainstem. The cochlear nuclei are thought to recode the auditory signal and transfer the data predominantly to the contralateral superior olivary complex. In addition, the cochlear nuclei pass information ipsilaterally to the lateral lemniscus. On entering the thalamus, the auditory information takes two pathways to the temporal lobe. Primary auditory reception occurs in Heschl's gyri in either hemisphere of the brain.

4.3 Classification of hearing impairment

Hearing impairment can be classified as shown in the table below.

Table 1: Classification of hearing loss.

CRITERIA	SUBCATEGORY
Cause	Genetic (monogenic or Multifactorial)
	Nongenetic
Association	Syndromic
	Nonsyndromic
Onset	Prelingual
	Postlingual
Type	Sensorineural
	Conductive
	Mixed
Severity	
Mild	Loss of 21-40 dB
Moderate	Loss of 41-60 dB
Moderately severe	Loss of 61-80 dB
Severe	Loss of 81-100 dB
Profound	Loss of > 100 dB
Frequencies	
Low	< 500 Hz
Middle	5001-2000 Hz
High	> 2000 Hz

*This classification is taken from Parving and Newton [8]

4.4 Types of hearing loss

Conductive hearing loss results from failure of the conducting mechanism to transmit the sound impulses from the external ear to the cochlea. It may be due to blockage or diseases of the external auditory canal, the tympanic membrane, the ossicles and the middle ear cavity. Audiometrically there are normal bone conduction thresholds (less than 20 dB) and an air-bone gap greater than 15dB.

Sensorineural hearing loss can result from failure of the normal transduction of sound waves into electrical impulses within the cochlea or failure of normal transmission of the electrical impulses within the auditory nerve and its central pathways with an air-bone gap of less than 15dB.

Mixed hearing loss results from a combination of both conductive and sensorineural hearing factors with bone conduction thresholds of greater than 20 dB and air-bone gap greater than or equal to 15dB.

4.5 Etiology of hearing impairment

Hearing loss is the most common sensory deficit in humans. It is estimated that between 1 and 3 children in a thousand are born with hearing impairment significant enough to compromise the development of normal language skills and social development. [9]

Hearing loss can be caused by environmental factors as well as genetic factors. It is estimated that 50% of all childhood deafness is due to genetic causes. [10, 11]

Cases in which the etiology of hearing impairment cannot be identified are classified as unknown causes.

Table 2: Causes of permanent childhood hearing impairment.*

	Disorders	Early intervention options
Prenatal		
Genetic syndromic	Treacher Collins syndrome Pendred's syndrome Usher's syndrome Waardenburg's syndrome Jervell and Lange-Nielsen syndrome Others (eg, Hunter syndrome)	Genetic assessment and counselling Discourage consanguineous marriages
Genetic non-syndromic	Connexin 26 (35del G mutation) Connexin 31 Others (eg, MYO7A)	Genetic assessment and counselling Discourage consanguineous marriages
Chromosomal	Down's syndrome Edward's syndrome Patau's syndrome Turner's syndrome	Genetic assessment Prenatal diagnosis & termination of pregnancy
Acquired congenital infections	Rubella Cytomegalovirus Herpes simplex Toxoplasmosis Syphilis	Rubella vaccination for girls and infants Health education on secondary prevention Health education/caesarean section Health education/treat mother Health education/treat mother
Natal		
Birth trauma	Birth asphyxia* Prematurity or low birthweight*	Improved obstetric, neonatal and childcare practices
Postnatal		
Neonatal jaundice	Neonatal sepsis Ototoxicity (eg, aminoglycosides) Meningitis* Mumps Measles* Cerebral malaria* Noise (including incubator noise) Otitis media* Impacted cerumen	Early detection and prompt treatment Improved obstetric and neonatal care Education about avoidance/rational use of ototoxic drugs Vaccination (early childhood and at-risk population) Vaccination Vaccination Vector reduction through treated bed nets and prophylaxis Education Improved personal hygiene Improved living conditions, health education for improved aural hygiene, and avoidance of ear buds

*This table is adopted from Olusanya and Newton [12]

4.5.1 Genetic hearing impairment

There are two main forms of genetic hearing loss, syndromic and nonsyndromic. Children with syndromic hearing loss have other clinical features in addition to the hearing loss. The incidence of congenital bilateral hearing impairment (>40dB) is 2–4 per 1000 live births [13] of which 50% have congenital severe-to-profound hereditary hearing impairment. About 30% of the hereditary hearing loss is syndromic, while the vast majority is non-syndromic (70%). [14] Genetic lesions may cause both conductive and sensorineural hearing loss. Genetic anomalies are much more commonly expressed as sensorineural hearing loss than as conductive hearing loss.

Hereditary hearing loss can be transmitted in several inheritance patterns, including autosomal dominant, autosomal recessive, X-linked inheritance and mitochondrial inheritance. The pattern of inheritance is autosomal recessive in 60 to 70 percent of cases, autosomal dominant in 20 to 30 percent, and X-linked in 2 %. [15]

4.5.1.2 Syndromic hearing loss (SHI)

There are over four hundred syndromes with hearing loss that have been described. Some of the syndromes are mentioned below. At least 50 hereditary syndromes produce conductive hearing loss, usually mediated by dysplasia or fixation of the ossicles of the middle ear or by malformation of the external auditory canal. The most common genetically transmitted disorder producing conductive hearing loss is otosclerosis.

Down's syndrome (Trisomy 21)

Down's syndrome is by far the most common and best-known chromosomal disorder in humans. Down's syndrome occurs in 1 out of 700 live births. [16] The cause of Down syndrome is full trisomy 21 in 94% of patients. Mosaicism (2.4%) and translocations (3.3%) account for the rest. Clinical features of Down's syndrome include mental retardation, characteristic facial features, hand anomalies, and congenital heart defects.

External ear canal stenosis and anatomic anomalies of the eustachian tube predispose these children to cerumen impaction, chronic ear infections and hearing loss. Anomalies of the inner ear may also be present causing sensorineural hearing loss. A longitudinal

study conducted by Shott et al [17] found 81% of children with Down's syndrome to have abnormal hearing levels before their treatment.

Pierre Robin syndrome

It is mainly of autosomal dominant inheritance. Phenotypically, it presents with a cleft palate, hypoplasia of the mandible, glossoptosis, cup-shaped pinnae, cochlear abnormalities and narrow internal auditory canals, congenital dislocation of the hip, club foot and mental retardation. The middle ear cleft may be absent or there may be thickening of the stapes footplate. Inner ear deformities include abnormal communication between the cochlea, a poorly developed modiolus or a narrow internal auditory canal.

Treacher Collins syndrome

Treacher Collins syndrome is a disorder of craniofacial development. It is autosomal dominant inheritance with variable expression. The features include slanting of the eyes with inferior displacement of the lateral canthi with respect to the medial canthi, coloboma of the lower lids, micrognathia, microtia and other deformity of the ears, hypoplastic zygomatic arches, and macrostomia. Conductive hearing loss and cleft palate are often present.

Usher syndrome

It is an autosomal recessive disorder characterized by severe-to-profound congenital hearing loss and retinitis pigmentosa.

Waardenburg Syndrome

Waardenburg syndrome is the most common cause of autosomal dominant syndromic hearing loss. The clinical features usually include dystopia canthorum (lateral displacement of the inner canthus of the eyes to give an appearance of a widened nasal bridge), pigmentory abnormalities of the skin, iris (heterochromia), and hair (white forelock), and sensorineural hearing loss.

4.5.1.3 Non-syndromic hearing impairment (NSHI)

About 80 loci for non-syndromic hearing loss have been mapped to the human genome. Although the phenotype is quite similar, NSHI is extremely heterogeneous, with over 40 genetic loci currently known. Based on the type of gene product, these genes can be categorized into several groups- channel and gap junction components, myosin and other cytoskeletal proteins, transcription factors, extracellular matrix proteins and unknown function genes.

There are autosomal dominant, autosomal recessive and X-linked forms of non-syndromic hearing loss. In general, recessive inheritance shows prelingual onset of hearing loss and the severity is severe to profound with all frequencies affected. In autosomal dominant forms, the phenotype is less severe and the onset is usually postlingual.

4.5.2 Acquired/Environmental causes of hearing impairment

Environmental or acquired causes of hearing impairment can be divided into prenatal, perinatal and postnatal factors. Most of these causes of hearing loss are preventable.

4.5.2.1 Prenatal/intrauterine causes

Congenital infections and fetal exposure to teratogens can result in SNHI. The severity with which infections or other toxic agents may damage the fetus in utero is related to the date at which fetal insult occurs but hearing impairment may result at any time of fetal life up to parturition. All of these prenatal insults result in physical abnormalities, which should prompt the clinician to recognize the diagnosis and perform a confirmatory evaluation. Even if these children pass the neonatal screen, careful follow-up of their hearing is necessary.

The infections primarily responsible for congenital and early onset hearing impairment are rubella and CMV. Others include toxoplasmosis, congenital measles, congenital syphilis, herpes and other viral infections.

4.5.2.2 Perinatal causes

The joint committee on infant hearing, US, issued a position statement in 1994, which gave the following perinatal indicators for sensorineural hearing loss in neonates: birth weight <1.5 grams, hyperbilirubinemia requiring exchange transfusion, Apgar scores of 0-4 at one minute or 0-6 at five minutes and mechanical ventilation lasting five days or more, ototoxic drugs, including aminoglycosides but not limited to these given in multiple doses or in conjunction with loop diuretics. Other factors include perinatal sepsis, rhesus incompatibility, congenital heart failure and birth trauma with intracerebral hemorrhage.

The accumulation of perinatal etiologies is the reason for the remarkable majority of acquired hearing losses. One reason might be the general improvement in neonatology. On the one hand this results in reduced mortality, but on the other hand it increases perinatally caused complications like hearing impairment. History of any of the above factors should prompt an evaluation of hearing because these conditions may also result in SNHI.

4.5.2.3 Postnatal causes

Approximately 10-20% of hearing loses are due to postnatal causes. The table below shows etiological classification of postnatal causes of hearing impairment.

Table 4: Causes of postnatal hearing impairment in children.*

1.	Conductive hearing impairment Secretory otitis media Acute otitis media Chronic suppurative otitis media Wax Foreign bodies	
2.	Sensorineural	
	Infection	Viral Bacterial meningitis Chronic suppurative otitis media Syphilis
	Trauma	Temporal bone fractures Perilymph leak Noise
	Metabolic Ototoxicity Meniere's disease	
	Tumors	Meningioma Acoustic neuroma
3.	Functional	Central Flat 40 dB loss and non-organic overlay Reye's syndrome, epileptic disorders, progressive neurological disorders, Central auditory processing disorders.

*Table adopted from Mawson's diseases of the ear 5th edition. [18]

Conductive hearing impairment

Conductive hearing loss results from failure of the conducting mechanism to transmit the sound impulses from the external ear to the cochlea. It may be due to blockage or diseases of the external auditory canal, the tympanic membrane, the ossicles and the middle ear cavity.

Chronic middle ear infection is the main cause of mild to moderate hearing impairment in children in developing countries.

The majority of conductive hearing loss affects low frequencies, while the majority of sensorineural hearing loss affects the high frequencies

Otitis media with effusion (OME)

OME is defined as the presence of serous fluid in the middle ear behind an intact tympanic membrane in the absence of signs or symptoms of acute infection. Malfunction of the eustachian tube, owing to mucosal disease or structural defects such as cranial-base anomalies, is a common substrate for this disorder.

OME is the most common cause of conductive hearing loss in childhood in developed countries. Children with conductive hearing impairment due to OME suffer more subtle effects upon language, attention and behavior. Peak age-specific incidence in the United States occurs in the first 2 years of life, typically between 6 and 18 months. [19]Some studies have shown a bimodal distribution, with peak incidence at 1 to 2 years of age and again at 5 years, the latter age corresponding to school entry. Olusanya et al [20] found a high prevalence of OME of 18.7% of school entrants in Nigeria. However, in other studies, OME is noted to be relatively uncommon in non-white populations living in developing countries. [21, 22]

Acute suppurative otitis media (ASOM)

ASOM is defined as the presence of fluid in the middle ear in association with signs or symptoms of acute local or systemic illness. ASOM is the most commonly diagnosed pediatric disease, with its peak incidence in the first two years of life. Fortunately, acute otitis media rarely results in more than a temporary conductive hearing loss.

The young infants' developing immune system coupled with age-related differences in Eustachian tube anatomy and physiology make them particularly vulnerable to respiratory tract infection and OM. Early exposures to environmental factors (e.g. child care attendance, respiratory viruses, parental smoking) also contribute to early and more frequent OM.

Chronic suppurative otitis media (ASOM)

WHO defines CSOM as "otorrhea through a perforated tympanic membrane present for at least two weeks." CSOM remains one of the most common childhood chronic infectious diseases worldwide, affecting diverse racial and cultural groups both in developing and industrialized countries. Risk factors for the development of COM include young age, overcrowding, inadequate housing, poor hygiene, lack of breastfeeding, poor nutrition, exposure to cigarette or wood-burning smoke, high rates of nasopharyngeal colonization with potentially pathogenic bacteria, eustachian tube dysfunction, and inadequate or unavailable health care. Poverty is a major risk factor in developing countries and in certain disadvantaged ethnic groups in developed countries.

CSOM most often occurs in the first 5 years of life, and it is most common in developing countries, in special populations such as children with craniofacial anomalies, and in certain racial groups. Highest prevalences of CSOM in children are reported among the Inuits of Alaska, Canada and Greenland, American Indians, and Australian Aborigines, and range from 7% to 46%. Intermediate prevalences are reported in the South Pacific Islands, Africa, Korea, India, and Saudi Arabia, ranging from 1% to 6%. The lowest prevalences are found in highly developed industrial countries such as the UK and the US. [23]

The most common sequel of CSOM is chronic hearing loss, either conductive or sensorineural. Chronic infection of the middle ear, causing edema of the middle-ear lining and discharge, tympanic membrane perforation, and possibly ossicular chain disruption, results in a conductive hearing loss ranging from 20 to 60 dB. Available data from Sub-Saharan Africa report COM and meningitis to be the largest cause of infectious hearing loss.

Barotrauma

Barotrauma usually occurs during descent from high altitude in which there is a rapid increase in aircraft pressure in the presence of compromised function of the eustachian tubes owing to upper respiratory tract infection or allergy. The inability to equalize air pressure on the external-canal and middle-ear sides of the tympanic membrane leads to substantial negative pressure within the middle ear, resulting in serous or bloody effusion

and then pain and temporary conductive hearing loss but rarely permanent damage to the inner ear.

Sensorineural hearing impairment

Acquired disorders causing sensorineural hearing impairment include infections (such as meningitis, syphilis, measles, and tuberculosis), ototoxic drugs, trauma, systemic diseases e.g. diabetes mellitus and neoplastic diseases e.g. acoustic neuroma.

Infectious causes:

Bacterial meningitis

Bacterial meningitis from any cause results in sensorineural loss in 10 to 38 percent of patients who survive the disease [24, 25, 26, 27, and 28] and is the main cause of acquired SNHI. [29, 30] The mechanism of hearing loss appears to be suppurative labyrinthitis or neuritis (or both) resulting in the loss of hair cells or damage to the auditory nerve.

Syphilis

Both congenital and acquired syphilis can produce unilateral or bilateral sensorineural hearing loss. Syphilis can mimic a number of other disorders of hearing and balance, including Meniere's disease and immune-mediated sensorineural hearing loss.

Tuberculosis

Tuberculosis of the temporal bone can cause multiple perforations of the tympanic membrane, chronic granulomatous otitis media, Bezold's abscess, and both conductive and sensorineural hearing loss. It is rare in the absence of primary pulmonary tuberculosis. Tuberculous otitis media may mimic chronic otitis media and, hence, may be seen clinically as persistent disease after mastoid surgery.

Viruses

The role of viruses in the production of sensorineural loss is controversial. The evidence of viral-induced sensorineural loss is strongest for maternal rubella, cytomegalovirus, and

herpes zoster. Although measles, mumps, and other common viruses are often cited as causative agents, their role in postnatal hearing loss remains unproved. Sensorineural hearing loss may also occur in patients with HIV infection or as a result of opportunistic infections of the temporal bone or cerebellopontile angle.

Ototoxicity

A variety of commonly used drugs have ototoxic properties. The best known are the aminoglycoside antibiotics, loop diuretics, salicylates, and antineoplastic agents such as cisplatin and antimalarial drugs. Most ototoxic substances cause hearing loss by damaging the cochlea, particularly the auditory hair cells and stria vascularis. Careful monitoring of patients, particularly those with compromised renal function or those receiving more than one ototoxic drug can prevent ototoxicity.

Trauma

Closed or penetrating injuries of the skull and temporal bone can result in conductive or sensorineural hearing loss or both. Concussive injuries of the skull without fracture may result in high-frequency sensorineural hearing loss similar to that seen in acoustic trauma. Perilymph fistula may occur following head trauma, straining or occasionally during barotraumas. Leakage of fluid from the inner ear may cause progressive or fluctuating sensorineural loss and vestibular symptoms.

There is individual susceptibility to trauma from noise. Clinically important sensorineural hearing loss may occur in some people exposed to high-intensity noise. Serial audiometry is the only way to be certain of the possible deleterious effects of noise in a given patient.

Irradiation of the head and neck region directed at lesions within the temporal bone or adjacent areas such as the nasopharynx may result in a conductive hearing loss due to osteoradionecrosis or a sensorineural hearing loss presumably due to radiation labyrinthitis.

Tumors

Primary or metastatic tumors can produce either a conductive hearing loss, by interfering with the motion mechanics of the middle ear and ossicles, or a sensorineural hearing loss, by invading the inner ear or auditory nerve. The most common primary tumors arising within the temporal bone are acoustic neuroma, chemodectoma, squamous-cell carcinoma, adenocarcinoma, and basal-cell carcinoma. The most common metastatic lesions to the temporal bone are adenocarcinoma of the breast in women and prostatic and renal-cell carcinoma in men.

Idiopathic and Degenerative Disorders

Degenerative disorders of unknown cause can produce sensorineural hearing loss. Meniere's syndrome is characterized by fluctuating sensorineural hearing loss, episodic vertigo, and tinnitus. Sudden idiopathic sensorineural hearing loss, causing moderate-to-severe sensorineural deafness, may be due to viral labyrinthitis.

Presbycusis, the hearing loss associated with aging affects more than one third of persons over the age of 75 years. The most common histopathological correlate of presbycusis is the loss of neuroepithelial (hair) cells, neurons, and the stria vascularis of the peripheral auditory system.

4.6 Severity of hearing impairment

Regardless of the type, hearing loss can be defined in terms of Decibels lost. The table below shows the grades of hearing impairment currently used by WHO (1991) from none, slight, moderate, severe to profound. Moderate, severe and profound hearing impairment in the better ear define the group of people having disabling hearing impairment. WHO uses disabling hearing impairment in the estimation of global deafness and hearing impairment for the global burden of disease rankings.

Table 5: Grades of hearing impairment

Grade 0 None		25 dB or less	No/slight problems Hears whispers
Grade 1 Slight		26 - 40 dB	Hears/repeats words in normal voice at lm
Grade 2 Moderate	Child 31 - 60 dB Adult 41 - 60 dB		Hears/repeats words in raised voice at 1m
Grade 3 Severe		61 - 80 dB	Hears words shout-ed into better ear
Grade 4 Profound		81 dB or more	Cannot hear/under-stand shouted voice

[Average 0.5, 1, 2, 4 kHz in better ear]

4.7 Clinical presentation

Hearing impairment in infants and very young children may be suspected by parents and any parent voicing a suspicion of hearing impairment in their child has considered it carefully first. Others in contact with hearing impaired children may be able to compare their responses to sound with those of other children they are in contact with and mention their suspicion. Clinicians may also suspect hearing impairment in children with craniofacial anomalies and other clinical syndromes known to be associated with hearing impairment. All such suspicions should be taken seriously and the infant/child's hearing tested.

Children with hearing impairment may also present with poor speech and language, behavioral problems, inattention and poor school performance. Children with conductive hearing impairment may have difficulty hearing low frequencies such as human speech, while children with sensorineural hearing loss may have difficulty hearing high frequencies such as doorbells, telephones or high-pitched voices.

4.8 Assessment of hearing impairment

Hearing impairment should be identified as early in life as possible, if its long-term consequences are to be prevented. The technology used for screening of hearing, should be age-appropriate and the child also should be comfortable with the testing situation.

The mainstay of ancillary testing remains behavioral (pure-tone and speech) audiometry. Measurement of the auditory evoked brain-stem response is useful in very young or uncooperative patients.

Decibel scale:

Clinically, loudness is expressed in decibels HL; the threshold for the perception of a sound at a given frequency by normal persons is 0 dB HL. Normal conversational levels are 45 to 60 dB, and the loudness of a jet engine at 31 m [100 ft] is 140 to 150 dB. The threshold for a handicapping hearing loss that is, one severe enough to interfere with speech acquisition in a child or effective conversation in an adult is approximately 25 to 30 dB.

4.8.1 Behavioural tests of hearing:

The startle response

The startle response can be elicited by a sound such as a shout and is either a whole body startle (Moro response) or little more than a blink (auropalpebral reflex). The startle response does not fulfill the criteria of a screening test but is nevertheless a useful part of the neonatal examination and should alert the physician to a possible hearing problem.

Auditory response cradle

The auditory response cradle measures changes in head turn, body movement, and respiration in babies less than 5 kg weight in response to bursts of high pass noise at 90 dB HL. This level is required as neonates do not produce repeatable behavioral responses to sound at less than 70-90 dB HL.

Distraction test

It involves attracting and releasing the child's attention with a play activity, the presentation of a series of frequency-specific auditory stimuli outside the child's visual field, and observing the child's response of turning towards and localizing the sound source. The testing is performed in a quiet, sound treated room. This form of testing may be used in a screening program or as part of a hearing assessment to quantify hearing impairment in children from the age of six to seven months. Children usually begin to inhibit their responses to this technique quickly from the age of 12 months.

The co-operation test

For most children between the ages of 18-30 months, there is a stage when the responses to the distraction test are inhibited and conditioning is not possible. At this stage a test of language comprehension can be done in which the child is requested to hand toy items to the mother, another adult or place items in a box. The level of voice required is measured. This is a suprathreshold test; levels required being $\geq$ 10 dBA above the threshold. Children with normal hearing will respond at levels of 40 dBA.

Performance test/Play Audiometry

The assessment of hearing of children of a developmental age of two and a half years upwards or until co-operation with pure tone audiometry is achieved. The child is conditioned to perform a simple action in response to a sound stimulus.

In the initial conditioning process, the child is given both visual and acoustic clues to the presentation of the sound stimulus in order to facilitate compliance without dependence on hearing sensitivity.

Pure tone Audiometry

Pure tone audiometry is the most common measurement of hearing sensitivity. The American academy of Pediatrics recommends pure-tone audiometry to be done at 4, 5, 12 and 18 years.

Signals are delivered through air and bone. The pure tone screening is considered important, because even slight abnormalities in hearing acuity can reduce the

intelligibility of speech message and cause learning problems in children.

Air conduction assesses the function of the entire auditory system from the most peripheral aspect to the central portion. Air conduction testing alone provides little information regarding the etiology of hearing loss and specific auditory pathology. When used in conjunction with bone conduction testing, they help determine both the type and severity of the hearing loss. Bone conducted sound is transmitted directly to the cochlea and is thought to be a better reflection of sensory hearing.

The clinically normal region on an audiogram is 0 to 20dB HL. Conversational speech is in the 40-50db HL region; with the most significant frequencies for understanding speech being 500 through 4000Hz. Hearing sensitivity within the speech frequency region is summarized by means of calculating the pure-tone average.

Audiometric results are valid only when the patient's responses are caused by stimulation of the test ear. Crossover occurs when the acoustic energy presented to one ear can stimulate the non-test ear, resulting in obtained responses, which represent the performance of the non-test rather than the test ear. The main mechanism of crossover is presumed to be bone conduction stimulation caused by vibration of the earphone cushion against the skull at high stimulus intensity levels. The amount that crosses over is a reflection of attenuation. The interaural attenuation of air-conducted tones varies from 40 to 80dB depending on whether ear inserts or headphones are being used. Interaural attenuation values are also frequency dependent, being smaller for low frequencies and higher for high frequencies. Interaural attenuation values for bone conduction can occur even at about 0dB for bone conduction signals.

Masking is the audiometric technique used to eliminate responses by the non-test ear whenever air and bone conduction stimulation exceeds interaural attenuation. An appropriate noise is presented to the ear not being tested when the stimulus is presented to the test ear. The level of masking noise must exceed the threshold for that ear. Excess levels of masking noise must be avoided in order to prevent crossover from the masking noise.

Speech Audiometry

Speech audiometry helps determine how well a person hears and understands speech. Spondee or spondaic words are the speech stimuli used to obtain the speech reception

threshold (SRT). A spondee is defined as a two-syllable word spoken with equal stress on both syllables and is presented in similar fashion as pure tone audiometry. The SRT is the softest intensity level at which a patient can correctly repeat 50% of the words. SRT is measured with speech signal, and the PTA is measured within the conversational range frequency, therefore these two values should be in close agreement. An unusually good SRT relative to the PTA should alert the physician to the possibility of nonorganic hearing loss, such as malingering.

Word recognition scoring is a common clinical approach to evaluate a person's ability to hear and understand speech. Lists of 20 to 50 words are presented to the patient at supra-threshold levels, usually 30dB above threshold. Out of this list, a percentage correct is calculated. Word recognition scores of 90% or higher is considered normal while scores below this level indicate a problem with word recognition. Patients with conductive hearing loss usually show excellent word recognition. Patients with cochlear lesions have poorer discrimination. Patients with retrocochlear lesions usually have even poorer discrimination scores.

4.8.2 Impedance Audiomety

Acoustic immittance is a measurement of energy or air pressure flow, which involves the ear canal, eardrum, ossicular chain, tensor tympani, stapedius muscle, cochlea, cranial nerves VII and VIII, and the brainstem. Impedance is the resistance to the flow of acoustic energy. Admittance is the ease of which acoustic energy can flow. Immittance is usually evaluated by tympanometry and the acoustic reflex. It is a useful diagnostic tool to identify the presence of fluid in the middle ear, evaluate Eustachian tube function, evaluate the facial nerve, and help predict audiometry.

Tympanometry

Tympanometry reflects the mobility/compliance of the tympanic membrane when air pressure is varied from +200 to -200 dPa within the ear canal. There are three types of tympanograms: A, B, and C.

A normal, or type A tympanogram has peak compliance between -100 and +100dPa, and within a normal range of compliance. Peaks that are located within this pressure range may be overly compliant, 'Ad,' as with an atrophic tympanic membrane or under

compliant, 'As,' such as with ossicular chain fixation or tympanosclerosis.

A type B tympanogram has no peak compliance and very little change in compliance with varying pressures. This pattern is most often associated with a middle ear effusion. Type B tympanogram is 93.4% sensitive in detecting those children aged 3-5 years with a hearing loss greater than 25dB hearing level. [31]

'Type C' tympanograms have a peak compliance that is located in the negative pressure ranges beyond -100dPa. This is usually seen in patients with Eustachian tube dysfunction and inadequate ventilation of the middle ear. 'Type C' may be a precursor to a 'Type B' tympanogram as the development of negative pressure precedes the presence of the effusion. The volume of air medial to the probe is also obtained with tympanometry. In general, ear canal volumes range from 0.5 to 1.0ml for children and 0.6 to 2.0ml in adults. Volume measurements greater than these may suggest tympanic membrane perforation or the presence of a patent ventilation tube.

Type A tympanogram suggesting normal Function
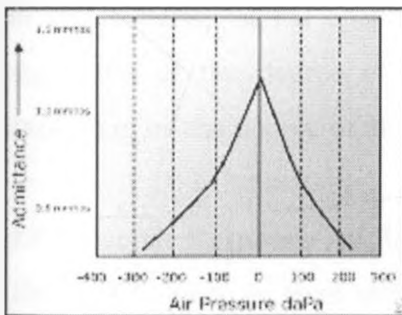
Type B tympanogram with no peak pressure
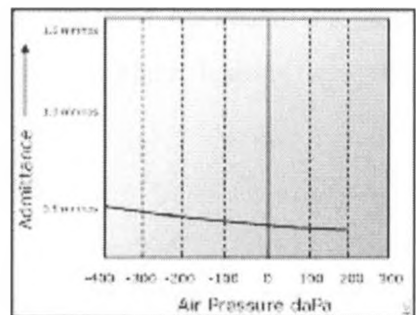
Type C tympanogram depicting normal compliance but significant negative pressure.

4.8.3 The acoustic stapedial reflex (ASR)

ASR is defined as the lowest intensity required to elicit a stapedial muscle contraction. The afferent portion of the reflex is the ipsilateral eighth nerve to cochlear nuclei. There is a c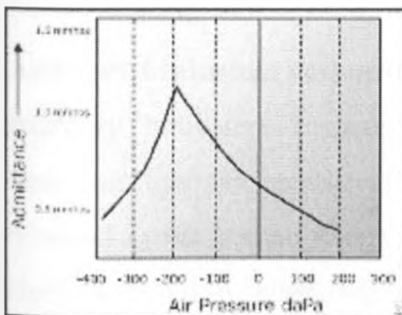omplex interaction between the ipsilateral cochlear nuclei and the bilateral motor nuclei of the seventh nerve. The efferent limb of the reflex is the seventh nerve, which innervates the stapedial muscle. Contraction of the stapedial muscle tilts the anterior stapes away from the oval window and stiffens the ossicular chain and results in increased impedance, which is measured as a small decrease in compliance by an ear canal probe. Measurement of various parameters of the acoustic reflex, including threshold, latency, decay and amplitude, provides variable information in the differentiation of cochlear, retrocochlear and brainstem pathology.

Acoustic reflex thresholds for tones in patients with normal hearing are usually 70-80 dB above their tone thresholds, and about 5dB greater for the contra lateral threshold.

The efferent limb of the reflex is explored in the diagnosis of ossicular chain disorders, such as otosclerosis and discontinuity, and facial nerve pathology.

Eighth nerve lesions will demonstrate an absent acoustic reflex when stimuli are presented to the affected ear. An important differentiation between the acoustic reflexes in eighth nerve versus cochlear lesions is that the reflex will be absent or abnormal regardless of the degree of hearing loss, whereas in cochlear lesions it is usually dependent on the degree of hearing loss.

4.84 Electric Response Audiometry:

These are objective tests useful in assessing hearing in newborns. These tests do not depend upon a behavioral change in response to sound and so all degrees of hearing loss can be determined.

Auditory brainstem responses (ABRs)

Auditory brainstem responses are impulses generated by the auditory nerve and brainstem that can be recorded on the scalp after a transient stimulus. ABRs have been detected in the human neonate as early as 25 weeks of gestation. It is not affected by sleep, sedation, or attention, and is therefore very appropriate for estimation of auditory

sensitivities in infants and children who have failed conventional testing secondary to behavioral techniques.

Otoacoustic emissions (OAEs)

Otoacoustic emissions are low energy sounds produced by the cochlea. They are thought to be acoustic byproducts of the outer hair cells, which are thought to underlie the amplification of the basilar membrane. Clinically, they are most often evoked using transient and distorted product stimulation. The evoking response causes outer hair cell motility which results in a mechanical wave that travels from the cochlea through the middle ear and tympanic membrane to the external ear canal where it is recorded. There are two types of OAEs- transient evoked otoacoustic emissions (TEOAEs) and distorted product otoacoustic emissions (DPOAEs).

Electrocochleograghy (EcoG).

EcoG can be defined as a method of measuring stimulus-related potentials of the most peripheral portions of the auditory system. The three major components of the EcoG are the cochlear microphonic, summating potential (SP), and the action potential (AP). The cochlear microphonic and summating potential reflect cochlear bioelectric activity.

This examination is performed most often for intraoperative monitoring of cochlear and eighth nerve status and in the diagnosis of Meniere's disease. Transtympanic EcoG has proven itself a reliable test to detect the presence of endolymphatic hydrops.

Other investigations that can be done for assessment of hearing in children include:

I. Children with congenital hearing impairment should undergo other tests- Electrocardiography, ophthalmic, thyroid and renal tests to rule out associated syndromes. Chromosomal studies may be of benefit in seeking particular genetic syndromes.

II. Serological tests for cytomegalovirus, toxoplasmosis and syphilis are performed routinely in some centers in children below 6 months. If maternal rubella is suspected, specific IgM or IgG antibodies may be detected.

III. Radiological investigations including skull X-rays, CT scan or MRI can be performed to localize malformations, position of the cochlear or other lesions of the middle ear and inner ear.

4.9 Screening for hearing impairment

Mass screening of hearing in children is based on the concept of secondary prevention, irrespective of the age at which the child is screened. A prerequisite for implementation of all screening programs is that the condition screened for should represent an important health problem, not only in terms of prevalence, but also in terms of devastating consequences – provided the condition is undetected, and thus untreated.

Delay in identification of hearing impairment in children not only has severe effects on their speech and language development, social skills, academic progress, psychological condition, and future job-opportunities but also has serious consequences for family members. Early diagnosis of hearing loss and adequate aural rehabilitation services can considerably minimize the damage.

According to a new guideline by the Joint Committee on Infant Hearing (JCIH) in 2000 [32] on the early identification of hearing impairment and intervention programs, all infants with hearing impairment should be identified before 3 months of age, and infants with confirmed hearing impairment should receive intervention before 6 months of age. Implementation of such screening procedures is not feasible in developing countries at the present moment. Screening at school entry or of school children is perhaps the most practical way of ensuring that children are evaluated for hearing capabilities, at least once.

Neonatal period

The goal of neonatal screening is to detect all hearing losses present at birth so that training can be instituted early enough to allow the deaf child to achieve speech and language that is normal as possible. It has been shown that early training produces better speech and language skills in the long term. Neonatal hearing screening can be universal or targeted. Universal neonatal hearing screening is where all newborns are screened for hearing whereas targeted neonatal hearing screening is where newborns at risk are

screened. High-risk criteria for neonates (birth to 28 d): Family history of congenital or early SNHL, Congenital infection known to be associated with SNHL, Craniofacial anomalies, Birth weight of less than 1500 g, Hyperbilirubinemia, Exposure to ototoxic medications, Bacterial meningitis, Low Apgar scores at birth, Prolonged mechanical ventilation and Findings of a syndrome associated with SNHL.

Tests used in neonatal screening are OAEs and ABR. Universal neonatal hearing screening programs have provided the opportunity to detect neonates with permanent congenital or early-acquired hearing impairment, and thus initiate auditory rehabilitation before the age of 3 months. This has been implemented in United Kingdom and a number of states in the United States.

Screening at 8 months

The goal is to detect congenital hearing impairment and early onset sensorineural and conductive hearing impairment, particularly that due to secretory otitis media, so that management may be implemented during the critical phase of language acquisition.

The distraction test is used at this stage.

Screening at 3 years

The goal is to detect middle ear dysfunction, early onset and mild frequency cochlear hearing impairment and to assess language acquisition, so that any hearing impairment may be promptly investigated and any effect upon language assessed. Play audiometry can be used for screening.

School entry screening

The goal is to detect middle ear dysfunction, unilateral hearing impairment, acquired and progressive hearing impairment so that appropriate educational and medical action can be instituted. In school-age children even mild and fluctuating losses may impede learning and deny the children the chance to reach their full academic potential. PTA is used for screening at this age.

4.10 Management of hearing impairment

Solutions to hearing impairment focus on prevention, early detection and management, and rehabilitation. 50% of deafness and hearing impairment in developing countries is avoidable through prevention, early diagnosis, and management.

The ultimate goal of management of a child with hearing impairment is for the child to grow up into a socially well-adjusted, integrated, competent adult, with good communication and good employment prospects.

Management of a child with hearing impairment involves the active participation from the outset of a number of different disciplines. Communication between them is best if they come together in multidisciplinary units to coordinate the assessment and management of these children. There is need for visual and developmental assessment so that other handicaps may be identified and corrected.

Speech therapy, psychological assessment, bereavement counseling and genetic counseling will be required in addition to auditory training.

The involvement of a teacher for hearing impaired children is mandatory for every deaf child from the time of diagnosis. Every child with unilateral or bilateral, mild, moderate or severe hearing impairment should be notified to the education service whether or not hearing aids are prescribed.

Prevention

The prevalence of congenital and acquired hearing impairment may be minimized through prevention. Many cases of sensorineural hearing impairment can be prevented through:

- Immunizing children against childhood diseases, including measles, meningitis, rubella and mumps, according to national recommendations.
- Immunizing women of child-bearing age against rubella before pregnancy.
- Testing for and treating syphilis and certain other infections in pregnant women.
- Improving antenatal and perinatal care.
- Neonatal care to avoid factors associated with subsequent hearing impairment
- Avoiding the use of ototoxic drugs unless prescribed by a qualified health care worker and properly monitored for correct dosage.

- Control of environmental noise through reducing exposure (both occupational and recreational), using personal hearing protection and engineering noise control.
- Genetic counseling to all parents of children with congenital hearing impairment and to all school leavers whose hearing impairment is congenital or early onset.

Many cases of conductive hearing impairment can be prevented from becoming chronic through appropriate detection, followed by appropriate medical and/or surgical interventions.

Medical therapy

Medical management is aimed at alleviating Eustachian tube dysfunction, allergic rhinitis and treating middle ear disease. Treatments include antibiotics (parenteral or topical), topical steroids, antihistamines and decongestants. Wax may be treated with syringing, aural toilet or ceruminolytics.

Surgery management

Some causes of CHL may be managed or aided surgically. Children with persistent chronic or recurrent otitis media with resultant effusions may benefit from the placement of myringotomy tubes to ventilate the middle-ear space to prevent negative pressure in this area.

If otitis media results in the destruction or fixation of the ossicles, surgery may improve ossicular function. Chronic perforation of the tympanic membrane is closed by my myringoplasty to reduce CHL and to prevent re-infection of the middle ear. Cholesteatoma is a surgical disease.

Bone-anchored hearing aids (BAHAs) may be useful in some patients. Examples are patients with microtia.

Amplification

SNHL cannot be treated medically. Amplification with hearing aids is used to give the child as much auditory input as possible. Some children with conductive hearing impairment that is not amenable to surgical treatment can also benefit from hearing aids.

The main purpose of a hearing aid is to amplify speech so that it is audible and comfortable to listen to.

Cochlear implants

A cochlear implant is a device to restore some hearing in severely-profoundly deaf people when their organ of Corti has not developed, or is destroyed by disease or injury, to such an extent that no comparable hearing can be obtained with a hearing aid.

The cochlear implant takes the place of the damaged organ of corti and delivers to the inner ear a processed signal that stimulates more central neural structures, probably the spiral ganglion.

4.11 Sequel of hearing impairment

Hearing impairment and deafness are serious disabilities that can impose a heavy social and economic burden on individuals, families, communities and countries. Children with hearing impairment often experience delayed development of speech, language and cognitive skills, which may result in slow learning and difficulty progressing in school.

In adults, hearing impairment and deafness often make it difficult to obtain, perform, and keep employment. Both children and adults may suffer from social stigmatization and isolation as a result of hearing impairment.

5.0 LITERATURE REVIEW

Hearing impairment is the most frequent sensory impairment in humans, with significant social and psychological implications. In spite of the number of published studies about hearing impairment (HI), the currently available data use different criteria, which cause difficulties in comparison and estimation of the problem.

Hatcher et al [33] carried out a school-based prevalence survey of 5368 children from elementary schools in Kenya. They found 5.6% of children screened with hearing impairment of greater than 30 dB in at least one ear and 2.2% with bilateral hearing loss. A prevalence rate of 2.4 per 1000 was noted for profound bilateral hearing loss (failure to respond to a test tone at one or both frequencies at an 80 dB HL level). One-point-one percent had CSOM while 8.6% of the children had wax obstructing the tympanic membrane. Workers in this study were not able to measure the ambient noise before the screening process at all the sites and this made the interpretation of their results difficult. However, the sample size was large and representative of the study population.

Bastos et al [34] carried out a prevalence survey among 854 urban and rural schoolchildren in Tanzania. A screening threshold level of 25 dB HL was used. They found hearing loss within the speech frequency range was 37% in urban children and 18% in rural children. They found prevalence of hearing loss above 30 dB HL of 3% and a high frequency loss was more common in urban than rural children The prevalence of chronic otitis media was found to be 1.6% with no difference among urban and rural children.

A prevalence study done by Westerberg et al [35] among school children in Zimbabwe found 2.4% of children had hearing impairment (threshold of greater than 30 dB in at least one ear and for at least one of the test frequencies). Sensorineural healing loss was found in 1% of children and conductive hearing loss was found in 1.4% of children screened. Disabling hearing impairment was found in 0.9% of the children. The most common causes of conductive hearing loss were cerumen impaction and serous otitis

media with 25% and 32% respectively. The main objective of this study was to determine the prevalence of significant hearing impairment defined as greater than 30 dB HL at 1, 2 and 4 kHz in a quiet classroom. However, the grading of hearing impairment used in this study is not commonly used and makes it difficult to compare results of this study with other prevalence studies.

A community-screening program of 2015 children aged between 5-15 years was conducted by Seely et al in Sierra Leone [36] and found 9.1% of children screened to have mild or greater hearing loss. The prevalence of profound hearing loss was 4.0 per 1000 children. The risk factor most strongly associated with hearing loss was a history of otorrhea persisting longer than 1 month.

In the study by Olusanya et al [20] in Nigeria, Parental interviews, otoscopy, pure-tone audiometric screening (frequency 0.5–4 kHz) and tympanometric examinations were conducted for a representative sample of 359 school entrants in an inner city area of Lagos. The prevalence of hearing loss was 13.9%. Middle ear abnormalities were noted in 20.9% of the study population, of which 18.7% were reported with otitis media with effusion. The most common disorder was impacted cerumen, documented in 52.6% children. Hearing loss in this study was defined as pure tone average greater than 15dB HL in the worse ear. This could explain the high prevalence of hearing loss in this study.

S. Elango [37] conducted audiometric screening to find out the prevalence of hearing loss and ear disorders in 1307 primary school children in Malaysia. Hearing loss was found in 5.81% of the screened children and 7.26% had middle ear disorders. Screening audiometric test failed to detect 46% of cases with middle ear disorders.

Rao et al [38] conducted a study to determine hearing impairment and ear diseases among 855 children of school-entry age (first standard) in rural South India. Prevalence of hearing impairment was 11.9% and impacted wax was found to be the commonest cause

of hearing impairment (86.3%). Hearing impairment was predominantly conductive type (81.6%).

In the third national health and nutrition examination survey, Niskar et al [39] found the prevalence of hearing loss among children 6 to 19 years of age in the US to be 14.9%. Most of the hearing loss was unilateral and slight in severity (16- to 25- dB HL). The definition of hearing impairment of audiometric threshold level of 16dB HL in this survey was lower than that used in most prevalence studies conducted in developing countries. This could explain the high prevalence of hearing loss obtained in this study that is comparable to rates in developing countries.

6.0 STUDY JUSTIFICATION

Hearing impairment in children affects the development of speech and language, social and emotional development, and influence behavior and academic achievement. The world health organization is currently trying to implement primary ear and hearing care (PEHC) as part of primary health care in developing countries. This public health approach is seen to be the best way to prevent deafness and hearing impairment on a large scale and help people hard of hearing especially in developing countries where other programmes are almost non-existent.

Before embarking on public health programs to prevent and treat hearing impairment in children, the prevalence of hearing impairment should be determined.

No prevalence studies on hearing impairment have been done in Rwanda and the aim of this study was to provide prevalence data on hearing impairment among school children in Kigali, Rwanda.

7.0 OBJECTIVES

7.1 Global objective

To determine the prevalence of hearing impairment among school children in Kigali, Rwanda.

7.2 Specific objectives

1. To determine the prevalence of hearing impairment among school children and to relate this to age and gender
2. To determine the prevalence of ear disorders among the school children
3. To demonstrate the relationships between the various ear conditions and hearing impairment
4. To determine the proportions of children with the various grades of hearing impairment

8.0 METHODOLOGY

8.1 Study area

The study was conducted in the three districts (Nyarugenge, Gasabo and Kicukiro) of Kigali, Rwanda.

8.2 Study population

The study was conducted among school children in primary schools in Kigali. According to information obtained from the Ministry of Education Kigali, there are 139 primary schools in Kigali. The total number of school children in the city is 141145; 71851 (51%) are girls and 69294 (49%) are boys.

8.3 Study design

This was a school-based descriptive cross-sectional survey.

8.4 Sample size

Determination of the Sample Size

To calculate the required sample size for this study, sampling fraction (n/N), and the design effect (W) are taken into consideration.

The sample size is therefore, calculated from the following Fisher formula:

$n = A / \{(E^2 + (A/N)\}$

Where;

n = minimum sample size required (approximately)

P = assumed population prevalence of 5.6%, as that found in the Kiambu study (Kenya)

$Q = 100 -$ Prevalence $= 94.4\%$

$A = 3.8416PQW$

E = maximum acceptable random sampling error, (5%)

W = the likely design effect (3.0)

N = population size. (141,145)

Substituting the following in the formulae, we get, $n \approx 866$ children.

Thus the required minimum sample size was 866 children.

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8.5 Sampling method

Sampling was done using Computer generated random numbers. A random sample of primary schools, stratified by districts was chosen from all public primary schools in Kigali. The number of schools chosen in each district was proportional to the total number of school children in that district. The number of children screened in each district was also proportional to the number of children in each district. Two classes were then randomly selected in each of the eleven primary schools. The stream examined in each class was also randomly selected in each of the eleven schools.

8.6 Inclusion criteria

School children between 6-13 years in the selected schools

8.7 Exclusion criteria

Children whose parents did not consent to this study

Children who did not sign an informed assent

School children who did not cooperate during the examination process

8.8 Study period
This study was conducted in two-month period of September and October 2008.

8.9 Training
All research participants had a two-day training to get acquainted on all steps involved in this study- registration/interview of subjects, measurement procedures, quality control and recording of data.

8.10 Materials and equipment
1. Diagnostic audiometer (AD 27)
2. MTP10 hand held ympanometer and plugs
3. Sound level meter (CEL-269)
4. Head light (Battery operated)
5. Otoscope (Heine)
5. Aural speculums

7. Jobson Horne probes
8. Foreign body/wax hooks
9. Crocodile forceps
10. Metallic syringes
11. Warm water and cold water containers
12. Kidney dishes
13. Towels and Macintosh
14. Instrument container
15. Wax drops
16. Consumables- Cotton, Disposable gloves, Surgical spirit, H2O2, ear drops.

8.11 Measurement procedures

An introductory letter was obtained from the permanent secretary in the Ministry Education to the directors of the schools where the study was conducted. The letters were sent in advance explaining the purpose of the study and the date the research team would be visiting the school.

On the first visit to the school, the purpose of the study was explained to the headmaster of each of the schools. Community consent was then obtained from the headmaster and a class of each grade to be screened randomly selected. Parental consents were then left for dispatch to each of the parents of children in the selected classes.

On the day of the examination, the purpose of the study was again explained to the director and the teachers and help sought from teachers to assist in the screening process. The classes to be examined were then informed. A quiet classroom was then chosen at the school where the examination was conducted and the ambient noise measured by a sound level meter. Doors and windows of the classroom were also closed to reduce on background noise. The examination procedure was then explained and demonstrated to the children in the native Kinyarwanda language.

The examination was done as a one-stage approach and begun with recording of demographic data of the children with the help of their class teachers. All children from 6-13 years in the selected classes were included in the study. Children outside this age bracket were examined but not included in this study.

Each subject was also asked whether he/she has a parent or sibling with difficulty in hearing and the answer recorded on the proforma. An alternative question was asking whether the subject has a sibling of school going age who does not attend school and why.

Children who had their parental consent and signed an informed assent in the selected classes were then examined by otoscopy, tympanometry and pure tone audiometry.

Otoscopy

The procedure was carried out using a battery-powered otoscope (Heine). The procedure was explained to each subject before it was done. The examination was done on the right ear and then the left ear. Examination began with the inspection of the pinna, pre-auricular and post-auricular areas for any abnormalities. The pinna was then gently pulled upwards and outwards and the speculum of the otoscope gently inserted into the canal with the instrument held between the thumb and index finger with the ulnar aspect of the hand resting gently against the subject's cheek or neck. The external auditory canal and tympanic membrane were inspected and findings recorded on the proforma. Subjects who were found to have copious otorrhea had their ear canals mopped to allow visibility of the tympanic membrane.

Tympanometry

Tympanometry was performed with MTP10 handheld tympanometer on both ears after otoscopic examination starting with the right ear and then the left ear. Subjects found to have impacted wax on otoscopy had audiometric screening first and wax removal before doing tympanometry. Tympanometry was not done in children who had dry tympanic membrane perforation and CSOM. The subjects were first informed about what was to done in the procedure and asked to remain still and avoid swallowing or coughing but breathe normally. The pinna was drawn backwards and upwards and the probe plug inserted with a slight twist motion. Automatic tympanometry was then performed and the type of curve obtained recorded on the proforma. For children we got abnormal tympanogram (type B and C), tympanometry was repeated twice to ascertain the finding.

Audiometry screening

Audiometry screening was performed at a tone of 25 dB HL at 1, 2 and 4 kHz frequencies with testing repeated at 1 kHz. If the response obtained on retesting at 1 kHz was different from the first one, audiometry screening was repeated for that subject. Subjects who failed to hear the screening tone at any of the frequencies had further audiometric testing for thresholds to determine the severity of hearing impairment. A pass was defined as correct responses to signals at all frequencies in both ears whereas a fail was recorded if no response to one or more frequencies in at least one ear was obtained.

Subjects who failed the audiometry screening and were found to have wax or foreign bodies obstructing the external auditory canal had them removed and testing with audiometry repeated. Children who passed the audiometry screening but who were found to have cerumen or foreign bodies in the external auditory canal during otoscopy also had them removed.

Children who were found to have survey-related or other diseases were given appropriate medications and those who required surgery were referred to the main public referral hospital.

All findings for each subject were recorded in a modified WHO EARFORM/proforma and then entered in a computer data base.

Pure-tone audiometry

All subjects who failed the audiometry screening had pure tone audiometry done to determine their hearing threshold levels. This procedure was explained to each subject before testing was done. The subject being tested was seated facing the examiner but shielded from the control panel. The subject was then fitted with headsets and testing done for the right ear and then the left. Each time the subject heard the sound, he/she responded to the tester by raising a hand.

The presentation of sound at the start of the test was at 60 dB HL at 1 kHz. If no response was obtained at this threshold, it was increased in 10 dB steps until the subject responded to the sound. Once the subject heard a sound the threshold of hearing would then be established by decreasing thresholds by 10 dB steps and increasing by 5 dB steps until

the threshold was established by the subject confirming threshold on 3 successive occasions. If a response was obtained at 60 dB, it was then decreased by 10 dB until the subject could not hear the sound. The threshold was then established by increasing threshold by 5 dB and decreasing by 10 dB until the threshold was determined by the subject confirming threshold on 3 consecutive occasions.

The thresholds were then established in the same manner at 2, 4 and 0.5 kHz and then finally, the threshold was established again at 1 kHz

Study flow chart:

Identification of Quiet classroom

Ambient noise measurement

Registration/interview of subjects

Informed assent

Otoscopy

Tympanometry   Wax
                                    Foreign body

Audiometry screening

Fail   Pass

Pure tone thresholds   Discharge from study

Wax removal
Foreign body removal
Consultation and information
Referral
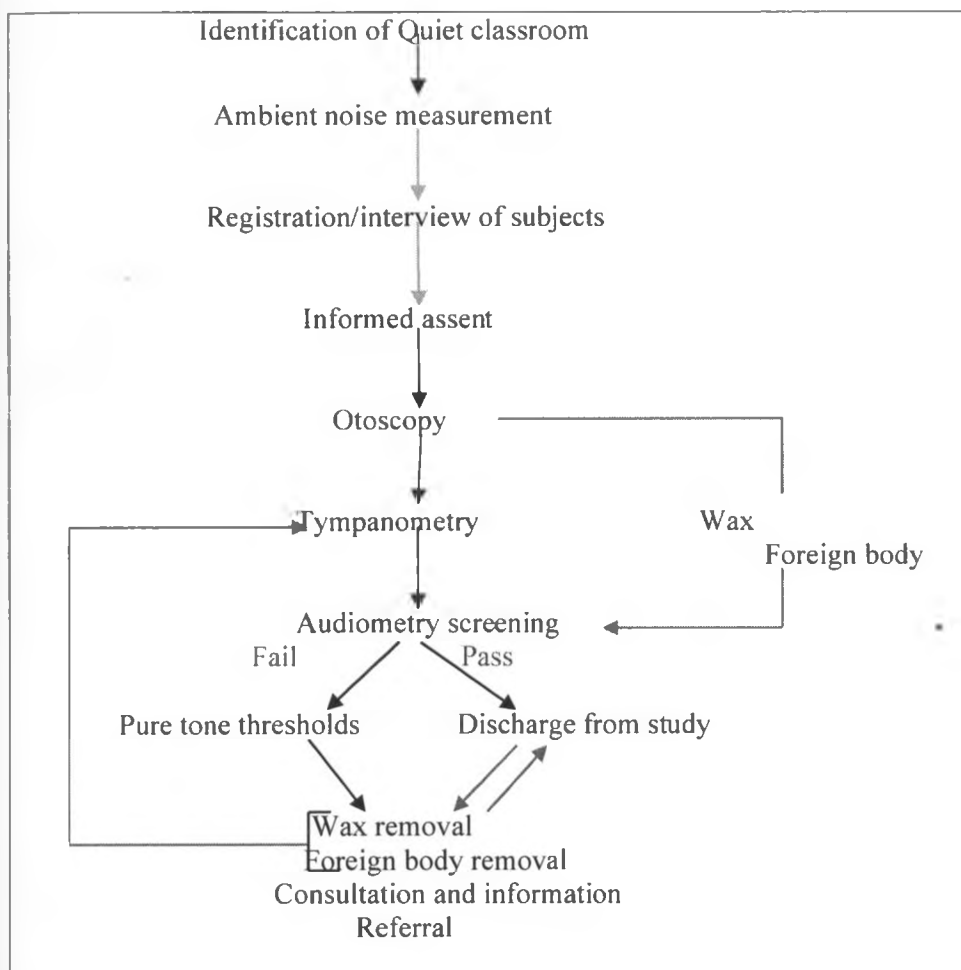
8.12 Data analysis

Data was checked for completeness, consistency and accuracy. Data base was then created by coding using statistical package for social sciences (SPSS version 11.0) with the help of a statistician. Data was then analyzed for ear disorders and hearing impairment and results presented in text, tables, graphs and charts. Chi square was used to measure statistical significance. Conclusions and recommendations based on the results were made.

8.13 Quality control

The questionnaire was pre-tested prior to commencement of the study and appropriate changes made.

Regular calibration of machines was done at the beginning, during and at the end of the study. An investigator (Audiologist) with confirmed normal hearing thresholds in a sound-proof booth acted as a control for the audiometric testing at each site each day.

The principal investigator carried out all otoscopic and tympanometry screening and audiometry screening and pure-tone audiometry were performed by an audiologist.

8.14 Ethical considerations

The study was approved by Rwanda national ethics committee

Approval was also obtained from the Ministry of Education and Ministry of Health.

Community consent was obtained from the Director of each school and parental consent from each parent. All children who agreed to participate in the study signed an informed assent.

Subjects found to have survey-related or other diseases were treated at the screening site or referred to the main referral Hospital.

The results and recommendations of this study will be available for use to Rwanda government, Rwanda bureau of statistics, WHO and the entire medical fraternity.

All information from all subjects will be kept confidential at all times.

There was no cost for participating in the study.

9.0 RESULTS

A total of 1073 school children from eleven public primary schools in Kigali city were examined for hearing impairment and ear disorders in September and October 2008. This represented 89% of the children enrolled in the 22 selected streams in the eleven schools. This representative sample of children in the city was randomly selected from three administrative districts in the city (Gasabo, Kicukiro and Nyarugenge). Majority of the children 483 (45%) screened were from Gasabo because it has more schools and a higher number of children (Figure 1).

Figure 1: Distribution of the Study Participants by District (n = 1,073)
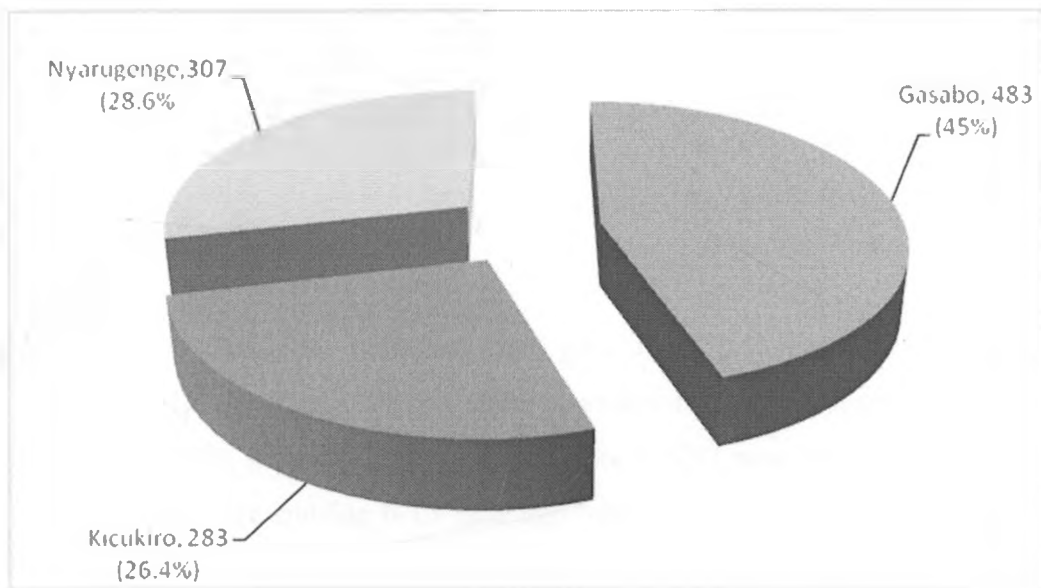
A total of 22 streams, 2 from each school were examined with all classes 1-6 represented (Figure 2). Majority of children examined were in class three 315 (29.4%) followed by 252 (23.5%) in class two. Class six had the least number of children with 65 (6.0%) examined.

Figure 2: Grades and number of children examined
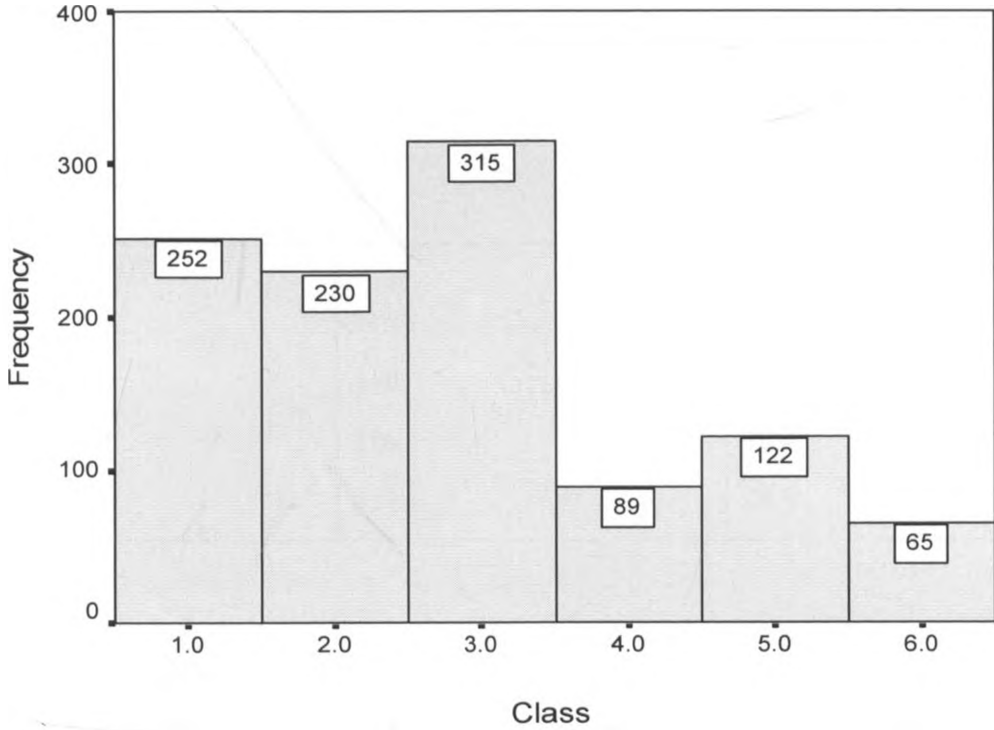
Majority of the children examined were girls 545 (50.8%) and 528(49.2%) were boys (Table 6). The age range for the children varied from 6-13 years with mean of 10.11 years (IQR 9-12). Majority of the children 326(30.4%) were in the age range 10-11years. Children who were outside 6-13 year age range were examined but excluded from the study.

Table 6: Age and sex distribution of children examined (n = 1,073)

Factor	Frequency	Percentage
Sex		
• Male	528	49.2
• Female	545	50.8
Age (in years)		
• 6-7	116	10.8
• 8-9	310	28.9
• 10-11	326	30.4
• 12-13	321	29.9

EAR DISORDERS

Overall prevalence of ear disorders was found to be 34%. The most common ear disorder was impacted wax found in 193 (18%) of the children (Table 7). All children had their wax removed by instruments or syringing after audiometry screening. We observed that it was easier to remove impacted wax with instruments than syringing. We also observed that some parents had removed wax from their children's ears prior to the set date for the examination. Those who failed audiometry screening before removal of wax were done pure tone audiometry after removal of wax.

Otitis media with effusion (type B tympanogram) was found in 72 (6.7%) children. Majority of children 40 (55.6%) with OME were below the age of 10 years (Figure 3).

Eustachian tube dysfunction (type C tympanogram) was found in 43 (4%) of the children and of these 14 (32.6%) had hearing impairment. Tympanometry was not performed on 46 ears which were found to have ear discharge and/or tympanic membrane perforation on otoscopy.

Prevalence of CSOM was found to be 2.1%. Only one child with CSOM was also found to have a definite cholesteatoma in the middle ear.

Other disorders included foreign bodies found in 15 (1.5%) children and dry tympanic membrane perforation found in 11(1%) of children.

Table 7: Prevalence of Ear Disorders (n = 1,073)

Disorder	Frequency	Per cent
Wax	193	18.0
OME	72	6.7
ET dysfunction	43	4.0
CSOM	22	2.1
Foreign body	15	1.5
Dry perforation	11	1.0
Otitis Externa	1	0.1
Other:		
Cholesteotoma	1	0.1
Tympanosclerosis	5	0.5
Fungal Infection	2	0.2
AOM	0	0

Table 8: Tympanometry findings (n = 2146 Ears)

Tympanometry	RE	LE	Total
Type A	964 (89.8%)	965 (89.9%)	1929
Type B	45 (4.2%)	55 (5.1%)	100
Type C	40 (3.7%)	31 (2.9%)	71
Not Done	24 (2.2%)	22 (2.1%)	46

RE – Right ear, LE-Left ear.

Table 8 shows the tympanometry findings. Type A tympanogram was found in 1929 ears (90%). Type B tympanogram was found in 100 ears (4.7%) and type C in 71 ears (3.3%).

Tympanometry was not done in 46 ears (2.1%) due to presence of tympanic membrane perforation and /or otorrhea.

Figure 3: OME and age distribution
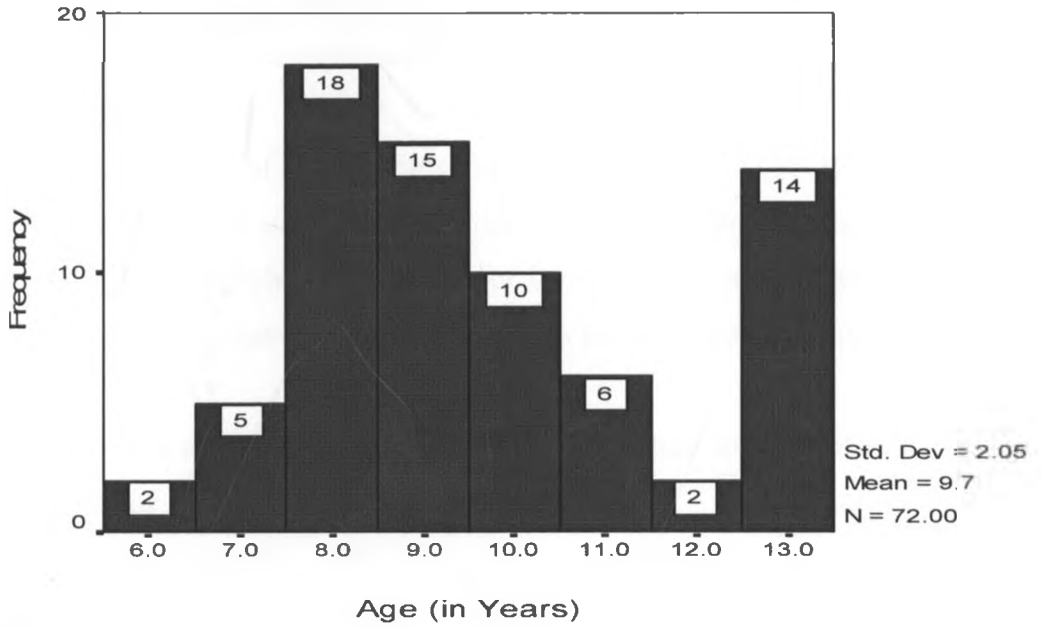
Table 9: Association between Age and Disorder

Disorder	Age				P-Value
	6-7, n (%)	8-9, n (%)	10-11, n (%)	12-13, n (%)	
Wax	25 (13.0)	57 (29.5)	60 (31.1)	51 (26.4)	0.566
Foreign body	2 (13.3)	2 (13.3)	7 (46.7)	4 (26.7)	0.433
CSOM	2 (9.1)	4 (18.2)	5 (22.7)	11 (50.0)	0.220
OME	7 (9.7)	33 (45.8)	16 (22.2)	16 (22.2)	*0.012*
Dry perforation	2 (18.2)	1 (9.1)	3 (27.3)	5 (45.5)	0.392
ET dysfunction	12 (27.9)	15 (34.9)	8 (18.6)	8 (18.6)	*0.001*

Age was a statistically significant factor for the presence of OME and ET dysfunction (P 0.012 and P 0.001 respectively.

HEARING IMPAIRMENT

On audiometry screening, 189 (18%) children failed the screening (Figure 2). Forty six (24%) children who failed audiometry screening were found to have normal hearing on pure tone audiometry.

The overall prevalence of hearing impairment was found to be 13.3%. Bilateral hearing impairment was present in 49 (4.5%) children while unilateral hearing impairment was found in 94 (8.8%) children. Prevalence of hearing impairment slightly higher among boys (7.2%) when compared with 6.2% for girls but this was not statistically significant (P 0.233).

The prevalence of disabling hearing impairment (pure tone average of 1, 2 and 4 kHz in the better hearing ear greater than 30dB) was 1.4%

Figure 4: Audiometry Screening
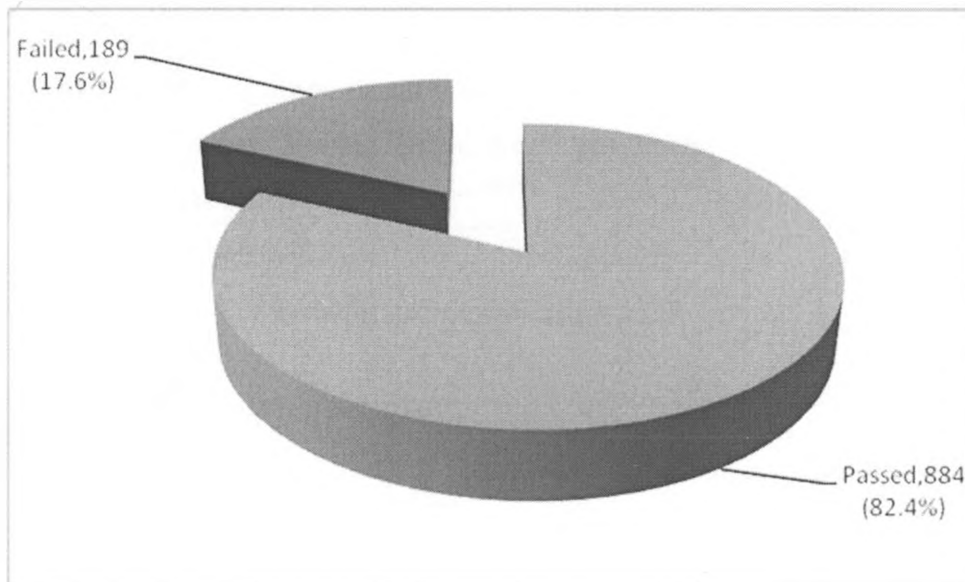
Table 10: Association between sex, age and hearing impairment

Factor	Presence of HI		P-Value
	Yes, n (%)	No, n (%)	
Sex			
Male	77 (7.2)	451 (42.0)	0.233
Female	66 (6.2)	479 (44.6)	
Age			
6 -7	18 (1.7)	98 (9.1)	
8-9	38 (3.5)	272 (25.3)	0.454
10-11	38 (3.5)	288 (26.8)	
12-13	49 (4.6)	272 (25.8)	

Age and sex of the patient were not found to be associated with presence of hearing impairment.

Majority of hearing impairment was due to conductive type accounting for 11.4% (Table 11). Sensorineural hearing impairment was found in 1.2% while mixed hearing impairment was found in 0.9%.

Table 11: Types of hearing impairment (n = 143)

Type of HI	Frequency	Per cent
CHL	122	11.4
SNHL	13	1.2
Mixed	10	0.9

Fifty children (26%) with impacted wax failed audiometry screening. Thirty eight children (19.7%) who had impacted wax were found to have hearing impairment. Of these, 21 (55.3%) had their hearing improve or return to normal after removal of wax. Twenty six (36.1%) of children with OME were found to have hearing impairment. Of those children with CSOM, 95.5% were found to have hearing impairment.

OME, ET dysfunction, CSOM, foreign body and dry tympanic membrane perforation were all strongly associated with hearing impairment ($P<0.001$) as shown in table 13.

Table 12: Association between ear disorder and hearing impairment.

Disorder	Presence of HI		OR (95% CI)	P-Value
	Yes, n (%)	No, n (%)		
Wax	38 (19.7)	155 (80.3)	1.8 (1.2 – 2.7)	0.004
OME	26 (36.1)	46 (63.9)	4.3 (2.5 - 7.2)	**<0.001**
ET dysfunction	14 (32.6)	29 (67.4)	3.4 (1.7 – 6.6)	**<0.001**
CSOM	21 (95.5)	1 (4.5)	159.9 (21.3 – 1199.4)	**<0.001**
Dry perforation	8 (72.7)	3 (27.3)	18.3 (4.8 – 69.9)	**<0.001**
Foreign body	7 (46.7)	8 (53.3)	5.9 (2.1 – 10.6)	**<0.001**
tympanosclerosis	3 (60.0)	2 (40.0)	9.9 (1.6 – 60.0)	0.002
Otitis Externa	0	1 (100.0)	-	-
AOM	0	0	-	-
Cholesteotoma	1(100.0)	-	-	-
Fungal Infection	0	2 (100.0)	-	-

Table 13: Prevalence and pattern of hearing impairment (n = 143)

Grade	Frequency	Percent
Mild	103	72.0
Moderate	24	16.8
Moderately Severe	4	2.8
Severe	3	2.1
Profound	9	6.3
Total	143	100.0

Table 13 shows prevalence and pattern of hearing impairment based on the worse ear.

Table 14: Prevalence and pattern of bilateral hearing impairment (n = 49)

Grade	Frequency	Percent
Mild	42	85.7
Moderate	5	10.3
Moderately Severe	1	2.0
Severe	0	0
Profound	1	2.0
Total	49	100.0

Table 14 shows prevalence and pattern of hearing impairment based on the better ear.

Table 15: Family History of hearing impairment (n = 1,073)

	Frequency	Percentage
Yes	83	7.7
• Brother/Sister	69	83.2
• Parents	14	16.8
No	986	91.9
Uncertain	4	0.4

Eighty three (7.7%) children reported having a parent/sibling with difficulty in hearing and 0.4% were uncertain (Table 15). A few children reported having their siblings drop out of school or being at home and not attending school due to difficulty in hearing.

Table 16: Relationship of family history of hearing impairment and hearing impairment in children examined.

Family History	Presence of HI		OR (95% CI)	P-Value
	Yes, n (%)	No, n (%)		
Yes	10 (7.1)	73 (7.9)	0.9 (0.5 – 1.8)	0.749
No	131 (92.9)	855 (92.1)		

Family history of hearing impairment was not associated with hearing impairment in the children examined (P 0.749).

10.0 DISCUSSION

Hearing impairment in children results in significant consequences on their development and education. This usually results in poor performance in school and dropping out with eventual poor social economic status and stigmatization.

The burden of hearing impairment and ear disorders has not been described in Rwanda. This study is therefore the first to assess the prevalence of hearing impairment and ear disorders among school children in Rwanda.

Screening of school children is perhaps the most practical way of ensuring that children are evaluated for hearing capabilities, at least once.

With free-mandatory universal primary education in place in Rwanda and with enrollment of about 95%, it is imperative that children with hearing impairment and ear disorders are identified and treated to avoid them being denied their chances to education. Although the prevalence of hearing impairment found in this may not reflect the exact prevalence in the whole country, it gives an estimate of what may be expected.

A total of 1073 children from 22 streams in eleven schools were recruited and examined during the course of this study. This represents enrollment of 89% of the total children in the selected classes. This enrollment is comparable to the Zimbabwean study. [35] A slightly high number of children examined were girls 545 (50.8%) and 528(49.2%) were boys. This is probably a reflection of the Rwandan general population with more women than men (male: female of 12:10). Majority of the children 326 (30.4%) were in the age range 10-11years.

Sample size for this study was drawn from three administrative zones of Kigali city (Gasabo, Kicukiro and Nyarugenge). Majority of children examined 45% were from Gasabo district because it has more schools and hence a large number of school children.

Children examined were in the age range 6-13 years since most of the primary school children in Rwanda fall in this age bracket. Primary school education in Rwanda has six classes (class 1 – class 6) followed by three years of post-primary education. Each class has several streams which go up to ten in some schools.

The Overall prevalence of ear disorders in this study is 34% (365). The commonest ear disorder was impacted wax with a prevalence of 18%. This is followed by otitis media with effusion, eustachian tube dysfunction, chronic suppurative otitis media, foreign body and dry perforation of the tympanic membrane at 6.7%, 4%, 2.1%, 1.5% and 1% respectively.

The prevalence of impacted wax in the present study is higher than that reported in the Kenyan, [33] Swaziland, [38] and the Tanzanian, [39] studies but much lower than that reported in Nigeria. [20] Only 19.7% of children with impacted wax were found to have hearing impairment and 55.3% had their hearing improve or return to normal after removal of wax. The high prevalence of impacted wax may be explained by the fact that majority of the children are asymptomatic or have no significant hearing impairment and that would not alert their parents to seek medical intervention. This also may be explained by the fact that impacted wax is not taken as a disease process but a natural phenomenon of the ear which does not require any treatment.

OME was found in 6.7% of the children with those below 10 years more affected. Prevalence reported in Zimbabwe, [35] Swaziland, [40] South Africa, [42, 43] and Nigeria (20) were 0.5%, 5%, 3.8% and 18.7% respectively. This shows a highly variable prevalence OME in Africans studies. Most other surveys done in Africa have not employed tympanometry testing. We found a high number of children with OME at 13 years than expected since OME is uncommon after 12 years of age.[45] Prevalences of OME in western countries are higher than those in African studies. Point prevalence estimates of 3% to 25% are reported among United States and Scandinavian children ages 6 months to 11 years. [46]

Eustachian tube dysfunction diagnosed by presence of a type C tympanogram was found in 4% of the children. No comparative figures for this disorder were found in literature.

CSOM was identified in 22 (2.1%) of the children. Most of the children with discharging ears were found in the rural areas of the city. This may be attributed to paucity of

accessible health care and poor socioeconomic status. This prevalence is similar to that reported in the Swaziland, [44] South African, [42] and the Tanzanian[41] studies. Prevalence of CSOM in Kenya [33] was much lower than in the present study.

Only one child with CSOM had obvious cholesteatoma. This could be an underestimate since we did not suction or examine the ears with a microscope.

Foreign bodies of the ear were found in 15 (1.5%) of children. This is almost twice that reported in the Kenyan study [33] and Swaziland study [40]. One child had bilateral foreign bodies (Beans) that caused her moderate conductive hearing impairment.

We did not find any children with acute otitis externa or acute otitis media. This could be due to the fact that children with acute ear infections are unlikely to be in school.

Tympanosclerosis was identified in 5 (0.5%) of the children and this could have followed previous ear infections.

One hundred eighty nine children (18%) failed audiometry screening at 25 dB (screening level recommended by WHO). This failure rate is higher than that reported in Kenya, [33] Zimbabwe, [35] South Africa, [44] Malaysia, [37] and south India. [38] The variable prevalences however, could be attributed to different methodologies employed and definitions of hearing impairment used in the different studies. Forty six (24%) children who failed audiometry screening were found to have normal hearing on diagnostic audiometry.

Overall prevalence of hearing impairment was found to be 13.3% (143) whilst disabling hearing impairment (defined by WHO as pure tone average of 1, 2 and 4 kHz in the better hearing ear greater than 30dB) was identified in 1.4% (15) of the children examined. The overall prevalence rate is comparable to that reported in South Africa, [44] South India [38] and Nigeria. [20] Rates reported in Kenya, [33] Zimbabwe [35] are lower than in the present study. These differences could similarly be attributed to different methodologies and

definitions of hearing impairment used in the different surveys. Disabling hearing impairment in the Zimbabwean study was reported as 0.9%.

Bilateral hearing impairment was found in 4.5% whereas 8.8% of the children had unilateral hearing impairment.

Conductive hearing impairment accounted for 11.4% of children with hearing impairment. This finding reaffirms the fact that more than 50% of hearing impairment in the third world setting is preventable. Sensorineural and mixed hearing impairment accounted for 1.2% and 0.9% of hearing impairment respectively. The prevalence of SNHI is comparable to that reported in Zimbabwe.[35]

The high prevalence of hearing impairment in this study could be due to lack of awareness of the dangers of ear diseases in the general population and paucity of accessible ENT services in the city.

Some of parents interviewed did not know that their children had hearing impairment. We found parents had different perceptions on the treatment of CSOM with some regarding it as a self limiting disease and others using traditional herbs to treat. Those who had sought medical attention did not know that ear disease can be treated surgically since the option was not given to them at any of the health facilities including the referral hospitals.

Study limitations

Variation of the background noise during the audiometry screening and testing could have affected the results

The fact that some parents had removed wax from their children's ears prior to the examination may have lowered the prevalence of impacted wax in this study

Some parents did not give consent for their children to participate in the study and this may have affected the prevalence rates in this study.

We were not able to measure intra-observer error and sensitivity of the tests used in this study.

11.0 Conclusion

This study demonstrates a high incidence of ear disease and hearing impairment in 1073 school children in Kigali. If we are to extrapolate the findings of this study to the whole city (141145 children), a total 18772 could be having hearing impairment of which more than 50% is preventable and/or treatable. With a free-mandatory primary education in place, it is imperative that measures are taken to identify and treat children with ear disease and hearing impairment if they are to benefit from this program.

12.0 Recommendations

A country-wide survey should be conducted to establish a national prevalence of hearing impairment.

There is an urgent need to improve awareness in the general population and health care providers on the dangers of ear disease and hearing impairment especially in school children.

Efforts should be made to scale up the

A screening program at least at school entry to identify and treat children with ear disease and hearing impairment is desirable.

Primary ear and hearing care should be adopted as part of primary health care in Rwanda.

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APPENDIX I: PROFORMA

WHO Ear and Hearing Disorders Examination Form Version 7.1A

A. CENSUS

1 Country Number			2 Study			3 Admin Number			4 Cluster Division			5 Household			6 Person			7 Optional		

8 Name _____	9 Age in Years			10 Age in Months			11 Male / 12 Female		Exam Status		13 Date					

(d d m m y y)

B. HEARING EXAMINATION

(I) Tympanometry R L
1. Type A Tympanogram .... □ □
2. Type B Tympanogram .... □ □
3. Type C Tympanogram .... □ □
4. Not done .... □ □

(II) Audiometry *(Age 4 years or over)*
1. Ambient noise .... [ ] [ ] dBA
2. Hearing Thresholds   Right (dBHL)   Left (dBHL)

		1 KHz		
		2 KHz		
		4 KHz		
		1 KHz		

SCREENING EXAMINER'S NUMBER .... [ ][ ]   SCREENING EXAMINER'S REMARKS:-

NOT FULLY EXAMINED IN THIS SECTION .... [ ]

C. BASIC EAR ASSESSMENT
Mark all abnormal findings which apply R L
I. Ear Pain .... □ □
II. Auricle
  1. Malformation .... □ □
  2. Normal .... □ □
III. External ear canal
  1. Inflammation .... □ □
  2. Wax .... □ □
    Removed .... □ □
  3. Foreign body .... □ □
    Removed .... □ □
  4. Otorrhoea .... □ □
    Removed .... □ □
  5. Fungi .... □ □
  6. Normal .... □ □
  7. Not seen .... □ □
IV. Ear drum
  1. Perforation .... □ □
  2. Dullness or Retraction .... □ □
  3. Red and Bulging .... □ □
  4. Normal .... □ □
  5. Not seen .... □ □
V. Middle Ear
  1. Otorrhoea .... □ □
  2. Normal .... □ □
  3. Not seen .... □ □
VI. Others _____ □ □
  Specify _____
Normal findings .... □ □
Not examined .... □ □

VII. Additional Information
1. (This question to be answered for subjects reporting deafness or hearing impairment)
  Since infancy/childhood (0-14y) .... □
  Since adulthood (15-59y) .... □
  Since old age (60y +) .... □
  Uncertain .... □
2. (This question to be answered for all subjects)
  Does any brother/sister/offspring/parent of subject have difficulty hearing?
  No .... □   →   Brother or sister ..... □
  Yes .... □   Child of subject ...... □
  Uncertain .... □   Parent of subject .... □

D. CAUSE OF EAR DISEASE AND/OR HEARING IMPAIRMENT
                                   R L

Normal ear **and** normal hearing .... □ □
I. Ear Disease
  1. Wax .... □ □
  2. Foreign body .... □ □
  3. Otitis externa .... □ □
  Otitis media
  4. Acute .... □ □
  5. Chronic suppurative .... □ □
  6. Serous (with effusion) .... □ □
  7. Dry perforation of Tympanic Membrane .... □ □
  8. Other .... □ □
II. Infectious Diseases .... □ □
  Specify _____
III. Genetic Conditions .... □ □
  Specify _____
IV. Non-Infectious Conditions .... □ □
  Specify _____
V. Undetermined Cause .... □ □
  Specify _____

E. ACTION NEEDED

I. No action needed .... □

II. Action needed .... □
  1. Medication .... □
  2. Hearing aid .... □
  3. Language/speech rehabilitation .... □
  4. Special needs education .... □
  5. Vocational Training .... □
  6. Surgery Referral
    Urgent .... □
    Non-urgent .... □
  7. Others .... □
    Specify)

SPECIAL EXAMINER'S NUMBER [ ][ ]

SPECIAL EXAMINER'S REMARKS:-

WHO/PBD/PDH/99.2)
Date of version 30-Aug-99
Date of printing 30-Aug-99

APPENDIX II: AUDIOGRAM

Name.. Age ....

Study Number.................................

Class..

Cell...District....................................

RIGHT EAR	**LEFT EAR**

FREQUENCY IN HERTZ (Hz)   FREQUENCY IN HERTZ (Hz)
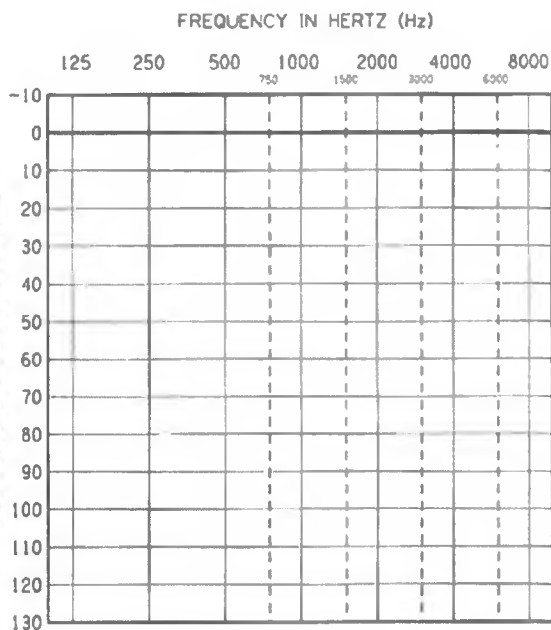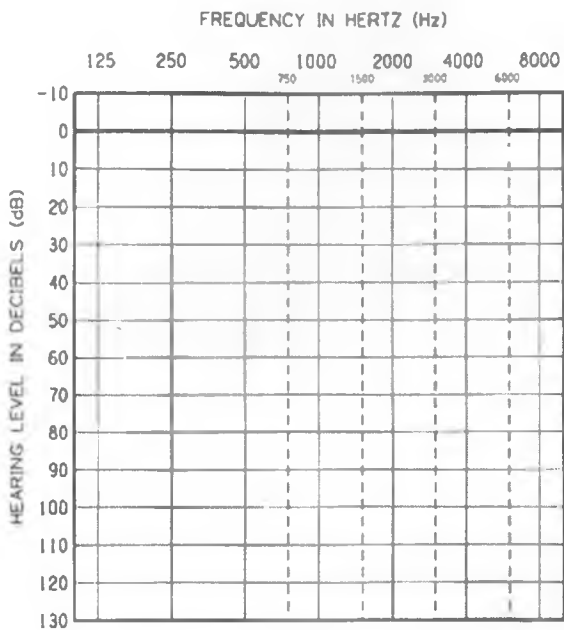
REMARKS:

RIGHT EAR..

LEFT EAR...

PURE TONE AVERAGE:

RIGHT EAR   LEFT EAR

AUDIOMETER--- SERIAL NUMBER-------------------

DATE--- BY: ---------------------------------------

APPENDIX III: ETHICS APPROVAL

REPUBLIQUE DU RWANDA

NATIONAL ETHICS COMMITTEE / COMITE NATIONAL D'ETHIQUE
Telephone: (250) 55 10 78 84   Ministry of Health
E-mail: rnec@moh.gov.rw   P.O. Box 84
                                                 Kigali, Rwanda.

Assurance No. FWA 00001973
IRB 00001497 of IORG0001100
August 19, 2008

Dr.Rajab Mustafa MUGABO,
Department of Surgery
University of Nairobi

RE: Prevalence of hearing impairment and ear disorders among school children in Kigali,

Rwanda.

After reviewing your protocol, during the RNEC meeting of July 19th, 2008, where quorum was me
and revisions made on the advice of the RNEC submitted on 18 August 2008, we hereby provid
approval for the above mentioned protocol.

Please note that approval of the protocol and consent form is valid for 12 months.

You are responsible for fulfilling the following requirements:

1. Changes, amendments, and addenda to the protocol or consent form must be submitted to th
   committee for review and approval, prior to activation of the changes.

2. Only approved consent forms are to be used in the enrollment of participants

3. All consent forms signed by subjects should be retained on file. The RNEC may conduct audi
   of all study records, and consent documentation may be part of such audits.

4. A continuing review application must be submitted to the RNEC in a timely fashion and befo
   expiry of this approval.

5. Failure to submit a continuing review application will result in termination of the study.

Sincerely,

Dr. Kayitesi KAYITENKORE
CHAIR, Rwanda National Ethics Committee.

C.P.I.
- Hon. Minister of Health.
- The Permanent Secretary, Ministry of Health.