"CAUSES OF ACQUIRED HEARING LOSS IN CHILDREN, ADULTS AND GERIATRICS"

Reg No.M9607

AN INDEPENDENT PROJECT SUBMITTED AS PART FULFILLMENT Of FIRST YEAR M.Sc (SPEECH AND HEARING) TO THE UNIVERSITY OF MYSORE. MYSORE.

ALL INDIA INSTITUTE OF SPEECH AND HEARING
MANASAGANGOTHRI, MYSORE - 570 006
INDIA

MAY 1997

Dedicated
With love to
Akka -Appaji
&
Impu, Chinni

'"You mean everything to me "

CERTIFICATE

This is to certify that this independent project entitled "CAUSES OF ACQUIRED HEARING LOSS IN CHILDREN, ADULTS AND GERIATRICS" is the bonafide work in partfulfillment for the First year Master of Science (Speech and Hearing) of the student with register number M9607.

Mysore
May,1997

Dr.S.NIKAM
Director,
All India Institute of Speech and Hearing,
Manasagangothri,
MYSORE - 570 006.
INDIA

CERTIFICATE

This is to certify that this independent project entitled "CAUSES OF ACQUIRED HEARING LOSS IN CHILDREN, ADULTS AND GERIATRICS" Ahas been prepared under my supervision and guidance.

Mysore
May, 1997

Dr.(Miss) S. NIKAM.
Guide

DECLARATION

This independent project entitled *"CAUSES OF ACQUIRED HEARING LOSS IN CHILDREN, ADULTS AND GERIATRICS"* is the result of my own study under the guidance of *Dr. (Miss) S. NIKAM,* Professor and Head, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier at any University for any other Diploma or Degree.

Mysore
May, 1997

M9607

ACKNOWLEDGEMENTS

I would like to express my deep sense of gratitude to my guide **Dr. (Miss) S.NIKAM,** Professor and Head, Department of Audiology, All India Institute of Speech and Hearing, Mysore, for her valuable help, suggestions and guidance at every phase of this project.

I am thankf'ul to Dr. **(Miss) S. NIKAM,** Director, All India Institute of Speech and Hearing, Mysore, for allowing me to take up this project.

It will be very difficult for me to forget the kind help and suggestions extended to me by my respected teacher **Mrs. VANAJA,** Lecturer, Department of Audiology, All India Institute of Speech and Hearing, Mysore, during my route to success. **Mr. ANIMESH BURMAN,** Lecturer in Audiology, All India Institute of Speech and Hearing, Mysore, was another source of help to me. I will be grateful to him for the same.

Dear **Akka** and **Appaji,** you have given me every thing I have aspired for and blessed me over the years, no words can express how great I feel about you both.

I am also thankful to **Annaiah** and my **Sisters Rudri, Indra** for their inspiration and encouragement that has brought me a long way.

Thanks a lot to my **Bhavaji Mr. Mahadev,** Lecturer in Economics, Sagar, for his encouragement in my studios.

Seena and Govindanna your friendship has been among those things whose value could never be explained by words. Thanks for being there with a generous heart to help wherever I needed.

To **SHIBU** and **PREETHI,** who shared my ups and downs in project work, my frenzy and fervous and laziness and laughter and who has been a major influence on my life at AIISH. THANK YOU.

Thanks a lot to all my friends **Visa, Veena, Archana, Shami, Rajesh, Kanaka, Muthu, Savitha Kamat,** and **Suresh** to be for being constant companion throughout my AIISH life, and for being a support all the ways.

I thank all the staffs of Records Section who helped me during my data collection (file study). I also thank all the subjects who participated in spot study without whom this project would have been impossible.

Spaceage Electronic Typing, thanks for their neat computer processing of this work.

Lastly, I thank God for all this blessings

Reg. No. M9607

CONTENTS

INTRODUCTION

Speech is the most effective mode of communication among human beings. Communicationthrough speech is dependent upon various factors and the most important factor among these is "the sense of hearing". Hearing or audition may be thought as general understanding or awareness of an individual about his surrounding environment.

The sense of hearing is possible because of the ear. The ear can be broadly divided into outer, middle and inner ear. The ear of a young adult (18 to 22 years of age) which, has had no known pathology, no H/o infection nor any kind of dis order is called as "normal ear" - (Newby, 1985). It is also defined as, the sense by which sounds are appreciated. [Medical Dectionary Dorland 1961].

Martin (1975) defined hearing loss as 'any loss of sound sensitivity, partial or complete, produced by abnormality anywhere in the auditory system". The severity of hearing loss may vary from mild to prfoound. Hearing loss can be caused by several factors which may operate before, during or anytime after birth. It could also be heredietary. Such loss could be apparent at the time of birth or may manifest itself in late childhood or early adulthood. Congenital hearing loss may be defined as more or less pronounced loss of hearing, present at birth.

Acquired hearing loss on the other hand may be acquired in postnatal period or early childhood or adulthood or in later life. Hearing loss may be acquired as a result of general complications of the external ear and external

auditory canal, such as injuries, foreign bodies, cerumen, extosis, collapsible ear canal, otitis externa. Problems of the middle ear such as perforated drum, eustachian tube malfunction, cleft palate, otitis media, otoslerosis can also cause hearing loss. Hearing loss may also occur due to problems in the inner ear, auditory nerve or central auditory pathway.

The causes of hearing loss in different age group, sex, seasons may not be same. It has been reported that adhesive otitis media is most common among children [Prescode 1976]. Ramalingam (1990) observed that serous otitis media occur more frequently in children below the age of 10 years. It is more common in winter season and males were more commonly affected. Incidence of ear discharge is higher in developing then developed countries. He further reported that it is a major cause for deafness in India.. Results of an investigation by Wrellind (1991). Showed that otitis media plays a major hearing problem in children..

There is a greater prevalence of otoslerosis among women than men. It has been noted that during such periods as puberty, pregnency, child birth and menopause, there is a marked increase in the occurrence of the disease among females. It generally occurs between age of 20 - 30 years. It appears to affect those of fair complecxion 8 times as often as those of dark complexion. In the united state, the racial factor shows that it is about seven times more prevelent among cancasians the negroes [-Prescod (1976)].

A review of to literature shows that "Acoustic neuroma" occurs twice as frequently in women as men, (Shambaugh (1967)]. It is mainly an adult

disease since it frequently occurs in adolesence and increases in frequency around the third and fourth decade of life [Neilson (1942)].

"Presbycusis" usually becomes quite evident aroundthe age 50 years, in some instances before and at times later. Men seem to have a proctivity toward it at an earlier age than women [Prescod (1976)].

Bunch (1929) observed that presbycusis is not only more prevalent among white males than white females, but it is found more often among white males or than black males or females.

Only a few studies have been carried out on causes of hearing loss in Indian population. Review of literature revealed a few studies on CSOM (Arora (1976)]and a few other conductive causes such as otoslerosis, ear discharge. Misra et al (1961), Sachdev and Bhatia (1965), Karpur (1965) of acquired hearing loss]. A majority of these studies have focussed on single causes of acquired hearing loss. Also most of these studies have been done single population such as children, or adults or geriatric population

The present study was undertaken to investigate the possible cause of acquired hearing loss in patients registered at speech and hearing centre. To compare the causes of acquired hearing loss among children, adults, and geriatric population and to compare the causes of acquired hearing loss during different seasons.

REVIEW OF LITERATURE

Among the various sense organs, the ear and its functions has a crucial role in the human civilization. The auditory system plays a very important role in verbal communication which is the most acceptable form of communication, among the human beings. Damage to this system or hearing impairment will therefore have severe implications on an individual.

Hearing impairment may be defined as any change for the worse in the structure and function of auditory system, usually outside (Davis and Silverman 1978). This impairment could be caused due to various factors the causes can therefore be broadly classified in to two types.

Acquired

Congenital

In 1966, Committee on Nomenclature of the Conference of Executive of American schools for the deaf, defined the deaf as "those in whom the sense of hearing is non-functional for the ordinary purpose of life" They classified the deaf into two groups on the basis of the age at which deafness occured.

Congenital deaf: Those who are born deaf and

Acquired deaf:Those who are born with normal hearing but in whom the sense of hearing becomes non-functional later through illness.

Prescord (1978) listed some acquired causes which had to hearing loss, they are:

I. General complications of the external car and external auditory canal like:

Injuries * Frostbite

* Accidents

Foreign bodies

Cerminoma

Exostris

Collapsible ear canal

Otitis Extrena:

Diffuse otities extrena

Swimmer's ear

Chronic otitis extrenia

Otomycosis

Hemorrahagia (O.E)

Furuncolosis

External cholesteatoma

Herps Zoster oticus.

II. Special problems of the middle ear cleft.

Perforated drum

Enstusium tube malfunction

Barotitis

Cleft Palate

Otitis media (SOM, AOM, COM)

Otoslerosis (stepedial, Cochlear).

III. Inner ear disorders:

Drug induced loss

Noise induced loss

Lesions of viral and bacterial origin

Rubella
Mumps
Syphilis
Tubercolosis
Searlet fever

Miniere's disease

Acoustic neuroma

Sudden hearing loss

Presbycusis

Central auditory disorders.

In an another classification sataloff (1966) listed acquired hearing loss on the basis of type of hearing loss, thus according to Sataloff (1966).

I. Conductive hearing loss:

(A) Causes with visible obstruction in External auditory canal.

Extosis of ear canal

Impacted cerumen

Fluid in external auditory canal

Collapse of ear canal during Audiometry

External otitis

Foriegn body in the ear canal

Carcinoma of external canal

Granuloma, cysts of external canal.

B. Causes with abnormalities visible in the Bar drum:

Myringitis

Ruptured drum

Perforated ear drum

Retracted ear drum

Flaccid ear drum

Senile ear drum

(c) Causes within middle ear communicating structures:

Catarrhal deafness and adhesions

Aerotitis media

Hemotympanum

Secretory otitis media

Serious otitis media and adenoids

Acute otitis media, COM

Tympanoslerosis,

Carcinoma, Allergy

Nasopharyngeal tumours.

X-ray treatment

Systamatic diseases

Glomus jungulare and Glomus tympanicum.

(d) Effects of ear Surgery:

Myringotomy

Hearing loss with ear surgery

Simple mastiodectomy

Ossiciloplasty

Tymponoplasty

Fenestration

Middle Ear Prostheses

II. Sensory neural Hearing Loss:

Gradual onset: Presbyensis

Occupational deafness

Sensoryneural aspects of otoslerosis

Sensory neural aspects of paget's and Hoeve's disease

-Effects of hearing aid amplification

- Neuritis of the auditory nerve and systamatic disease

- Unknown causes.

Sudden Bilateral:

Meringitis

Infections

Functional Hearing loss

Ototoxic drugs

Multiple slerosis

Unknown causes.

Sudden unilateral Sensory neural causes of hearing loss:

Mumps

Head trauma and acoustic trauma

Miniere's disease

Virus infections

Rupture of round window membrane or inner ear, membrane

Vascular disorders

Following ear surgery

Fistula of oval window

Following general surgery and anesthesia

Unknown causes.

Ballantyne (1977):

1) Conductive deafness:

Affections of the external auditory canal

Affection of middle ear cleft

Otoslerosis

2) Sensory neural deafenss:

Trauma

Noise

Infections

Cochlear Otoslerosis

Cogan's disease

Drugs

Acoustic tumours

Miniere's disease

Senile - deafness (Presbycusis)

Sudden hearing loss

3) Psychologenic deafness:

Hysterical deafness

Feigured Deafness - Malinguring.

Ballantyne (1977) maintained that psychological disturbance are the mains cause for acquired hearing loss.

Davis H (1960): He described causes of hearing loss in terms of

I Peripheral hearing loss:

(a) Conductive hearing loss:

Wax

External otitis media

Non-supporative otities media

Cholesteoltoma

Otoslerosis

b) Sensory neural hearing loss:

Presbycusis

Drugs

Allergens

Noise

Acoustic trauma

Meningities

Drysacusis

c) Psychogenic hearing loss (Functional deafness):

Hysterical deafness

Trauma or injuries have been found to be very important in causing hearing loss. The trauma can be of the various types. They can be broadly classified as follows:

I. Mechanical injury:

A) External:

Auto accidents

Complications of ear surgery

Maringoctomy

Mastoidectomy

Stepedectomy

II. Burns:

(a) Thermal burns

(b) Chemical burns

III. Irradiation injury:

Barber et al (1973): Conducted a study on 64 patients with head injury. It was found that lateral head blows caused homolateral or bilateral cochlear loss and vestibular lesions that might be bilateral, homolateral or contralateral alone. A lateral oblique head blow gave bilateral cochlear loss with the contralateral cochlear loss with the contralateral loss considerably greater than the homolateral significant cochlear and vestibular losses occurred regardless of severity of head injury. In study also found that psychiatric and intellectual defects were much more frequent in severe than in minor injuries and were much more important than cochlear or vertibular disorders in delaying recovery.

Walsted (1996): In a prospective study, 60 patients who underwent surgery for unilateral acoustic neuroma had the hearing on the contralateral ear tested before and several times after surgery. In 40 patients, a Thehold

increase was found during the following 9 days . The changes were greatest in the low frequencies immediately after surgery but after one week the high frequencies became involved. After three months, the hearing was normalized. The elder patients more than 50 years of age were more often affected whereas sex, tumour size, surgical approach or duration of surgery had no influences. Though the study shows the effect of surgery on hearing loss, this cannot be fully accounted for, since the patient was already suffering from acoustic neuroma.

White et al (1988): They conducted a study in which corerelations were made between pure tone thresholds and computer aided cross sectional measurements of the stria varcularies on histological sections of post mortom cochlear from 24 subjects, losses in the summed cross sectional areas of stria vascularies should a direct correlation with hearing loss.

Histelberger et al (1988): Jannetta has proposed vascular compression of the 8^{th} nerve as the etiology of hearing loss, tinnitus and vertigo in some patients. It has been found that staggers and sensory neural auditory lesions associated with deep diving appear to have several possible etiologies and sites.

King (1976): Studied 4 cases seen with hearing loss in the left ear for 5 years, following barotrauma. Tympanometry showed a mobile stapes but a fixed incus. The incus was mobilised and good mobility achieved. But no improvement in hearing resulted.

Coles (1964): Says incidious development of sensory neural hearing loss may be associated with diving. Inner ear barotrauma can cause an acute or relatively or acute on set of hearing loss and/or vertigo.

Armstrong (1972): They conducted study on perforation of tympanic membrane. Traumatic perforation of the tymapnic membrane deserve the same careful attention as any other injury and hearing should not be invariably left to chance. A foriegn object rupturing the posterio superior quadraint may cause obscured damage to the ossicular chain, a perilymptatic leak or leave an unsuspected foreign body in the middle ear.

Rudge (1996): He did a study on 4 cases of central pontine haemorrange and described audiotry dysfunction that was documented. He found central pontine haemorrahage as an important cause for hearing loss.

In today's mechanised society, damage to the auditory mechanism due to excessive noise exposure is a common occurrence. The adverse effects of noise, or unwanted sound has been the subject of extensive research for many years. In the fields of psychoacoustic and physiological acoustics.

This research grows out of the desire for scientific understanding of this effects on people, especially because of social problems created by the study increased in the intensity and prevelance of noise found in living and work environment, since start of the industrial revolution.

"Noise" may be defined as an audible acoustic energy that adversly effects the physiological or psychological well being of the people.

Auditory effects:

The auditory effects can be classified into two types:

Acoustic trauma

Noise induced hearing loss (nihl)

Lawrence (1948): Described acoustic trama as damage to the ear resulting from a sharply rising wave form, such as an intense blast. He further reported that acoustic trauma visually results in rupture of the eardrums with resultant bleeding and a condudtive loss of hearing. It can also produce sensory neural hearing loss.

NIHL, on the other hand constitutes the vast majority of hearing losses observed in industrial and unilatery situations. NIHL is caused by daily exposure to intense sound over a long period of time, the patho-physiology of this process is not definitely known, but it appears to be related to metabolic, vascular and structural changes in the cochlea during excessive noise exposure.

Structural changes in conductive mechanism:

Eames et al (1975): Impulse noise or any other explosive noises can have a drastic effect on the tympanic membrane. These produce a wave of comprehension which travel down the ear canal and later causes an extreme vibration of tympanic membrane and ossicles, tympanic membrane may rupture or ossicles may damaged them full force of explosion may be transmitted to the inner ear causing severe damage.

Singh and Ahluwali (1968): Obsreved central perforation, reddened and oedematous membrane with bleeding in the external auditory meatus offer exposure to noise.

Structural changes in the cochlea: Cochlea is more valuerable to noise exposure. Hawkins, Bars Garan, Johnson (1976): Found that the sensory neural degeneration due to noise focussed on 1st quadrant of basal turn for intermittent noise, continuous noise damage to this quadrant i.e., region between 9 and 13 mm is characterised by dip of 4 kHz.

Structural changes in Organ of Corti: Damage to the corti depends upon the frequency of the stimuli with high frequency sounds damaging the base and low frequency the apex.

Bohnell (1976) : Damage can be more wide spread than one would expect, based on the travelling wave theory.

Damage to the Reisnneer's membrane: The Reisnner's membrane can rupture along the edges due to exposure to continuous or loud noises. Newly formed rears may than closes the scala vestibuli and scala tympani.

Lipcomb etal (1976) : found that Reisnner's membrane may be bulging into scala vestibuli through out the cochlea or it may collalpse in some parts of the cochlea.

Hair cell damage : Damage to hair cells duee to noise, exposure may range from partial to complete. In general hair cell damage is greatest in the apical region than the basal region. The outer hair cells are more volnerable than

Inner hair cells. Lipscomb et al (1976) : frequently damage is seen in the third row of the outer hair cells.

Earliest sign of damage are swelling and pyknosis of hair cells. With increased exposure to noise proliferation, vasculation of ends plasmic reticulum of hair cell are observed. Not only this, with increased stimulation outer hair cells have a distorted appearance.

Damage to stereocilia : With prolonged exposure to noise, stereocilia of hair cells tend to fuse suggesting that the electrostatic properties of membrane permeability is changed. Loosing of stereocsilia membrane and desintegration of root lets stereocilia enlarged.

Damaged of supporting cells: Engstrous etal (1966) : reported that due to exposure to noise vasiculation m ay appear in supporting cells of hair cells including cells of Henses, Dietey and Claudius.

Structural damage of tectorial membrane: Word - Duall (1971) : observed occasional valied up tectorial membrane surrounded by thin layer of cells.

Damage of Basllar membrane : The Basilar membrane may be dislodged (dislocated) completely in some areas.

Vaso constriction due to noise : Hawkins (1976): afound marked constriction of human often blocking the passage of R.B.C. due to exposure tto noise. This constriction was thought to be due to swelling of endothelial cells.

Steria Vascularies Damage: Histo Pathological findings shows that the surface cells appear uneven, swollen and shronken with inter - cellular gaps.

Lips comb, Alekson, Vesten (1976) : observeed vacules in stria vascularies. This condition is most commonly seen in 3rd turn and in apical parts. Some times the epithelium of stria vascularies is seperated from spiral ligament.

The other important effect of noise exposure is the temporary threshod shift and permanent threshold shift.

Temporary Threshold Shift (T.T.S) :

This is one of the common N.I.H.L. (Noise Induced Hearing Loss) seen. This is caused due to exposure to intense sound (70 to 80 dB spl).

Dangerink et al (1987) : The temporary threshold shift (T.T.S) of 9 habitual smokers and non - smokers were determined twice (via Bekasy Sweep Procedures) after each of three conditions: 10 min of physical excresices, 10 minutes of 105dB SPL exposure to a third octave band noise centered around 2 K and 10 min of both excresices and noise. Smokers evidenced less T.T.S than did non - smokers, particularly when exposed to both noise and excresises and especially in the range of 2.5 to 5 K Hz.

Axelsson etal (1992) : In four separate experiments, they examined the effects of cigarate smoking on temporary threshold shift (T.T.S) following noise exposure.

(1) One experiment compared smokekrs and non-smokers after the subjects had obstained from smoking for atleast 6 hours.

(2) A second experiment tested only smokers who smoked a cigaratte just before noise exposure, during the noise exposure and in a controlled conditions during which they did not smoke.

(3) A third experiment tested only non-smokers who were exposed to noise after chewing nicotine gum after a controlled condition in which they rested chiewing the gum.

(4) The fourth experiment tested smokers and non-smokers in a condition which required than to smoke a cigarratte just prior to noise exposure and in another condition which prevented them from smoking.

Results : Indicated that smokers consistantly ev idance slightly smaller T.T.S than do non-smokers. Non-smokers evidanace significantly greater T.T.S at one frequency after they had non. These results suggest that the smaller T.T.S associated with cigaratte smoking is related to both the chronic and acute effects of smoking and that these effects may be more attributable to corbon monoxide than to nicotine.

Shepperd et al (1983) : The hearing threshold of a total of 58 workers aged 20 - 65 years were measured annually over a period of 4 years using Bekery sweep frequency audiometry. Using these data, accurate values of the yearly rate of change of hearing threshold tend due to presbycusis was obtained. In the laser investigated about 30% of the years showned significant deterioration in hearing ability due to noise in groups of younger and older workers it was found that the younger workers exhibited less threshold shift than expeected where as the older workers showed the greater threshold shift than expected.

Birningham (1972) : The potential danger to hearing produced by exposure to noise has long been recognized. In 1831 Fosbroke included acoustic

trammma among the cause by an explosion like common lire and loss of heariing produced gradually by continuous exposure to noise, an examplifiied by the deafness of a black smith.

Baurer (1976) related the noise produced by airplanes to hearing loss.Bunch (1937) pointed out that loss of activity for high tones was greater than for low tones and emphasized the severe, immediately loss of hearing after exposure to loud noise followed by a period of recovery, known as a temporary threshold shift. Repeated exposure was associated with a gradually increasing hearing loss involving increasing frequencies. He concluded that the medico legal and componsatory implications of industrial noise induced hearing loss necessitated a more scientific evaluation of the problem.

Nimier (1890): Hypothesized that the deafness resulting from an explosion was produced not by trauma to the middle ear, but by the effects on the nervous apparatus of the cochlea.

Bunch (1937): performed audiometry on 9 men who had been exposed to small arms fire or similar explosive noises he observed that the weakest e xplosiv e noise significient to produce trauma caused a decrease in acuity at 4.096 dB; with greater insult the gap broadined in both directions.

Although the delelterious effects of noise on hearing have been recognized for sometime, relatively few studies on acoustic trauma due to noise have been made. The ever increasing exposure of our p opulation to deafening sounds in industry, military service, and even routine civilian life

has made the problem a major concern of contemporary otologists. Indeed, one of the most important obligation of the otologist to his patients is the conservation of hearing.

The presented historical review of acoustic trauma documented proof of resulting hearing loss and established damaged risk criteria for b oth study state and impulse noise. A detailed evaluation of impulse noise produced by the weapons and ammunition most frequently used by the sports man hunter revealed that only a few of the rim fining tests fell short of the damage risk criteria for impulse noise. The muzzle energy exerts the greatest effect on the peak sound pressure level. Research on loss of hearing in general has been relatively scant. Some studies have been conducted on deafness due to noise producing mechanary and deafness sustained as the battle field, but there are few published reports on investigations of hearing loss sustained by the sports man hunter from the firing of hunting guns. Since hunting is a popular sports, the noise caused by firing hunting guns poses a significant danger to the hearing to the hunters firing these guns.

Axelson and Animson (1987) : In a longitudinal study, 2325 children were tested at age 7, 10, and 13 with screening audiometry the screening level was 20 dB HL. Approximately 75% of the children passed the screening levsel at all ages. Hearing loss was more frequent in boys than in girls at age 13 (16% : 19%). The left ear was more commonly affected than the right ear. High frequency dips increased for boys with age, but not for girls. The increasing incidence of hearing loss for boys with age was attributed to noisy leisure time activities.

Jukka Ylikoshi (1987) : They analysed pure tone audiometry curve of 361 Finnish conscripts who suffered acute acoustic tramma during their military service. In more than 75% of other ears the heaaring loss was found in the high frequency region (above 2 Khz). In the remaining 25% the loss was found in the speech frequency range.

Mc Namara etal (1985) : This study used the Franimgham heart study cohort has the refernce population. The majority of Inner displayed sensory neural hearing loss. A multivariable model was constructed to determine which variables had a significant impact upon hearing loss. Under the model, age, sex, illness, family history of hearing loss, miniere's disease and noise exposure were significant population risk factors. Age was by far and most critical risk factor.

There are various acquired causes such as infection / inflammation.

Scott Qiebink (1996) : Studied long term effects of otities media on hearing in both conventional and high frequency region in children. And found that a history of otities media was associated with poorer high frequency hearing, but the presence of subtle residual middle ear dysfunctions was not associated with an additional effect on high frequency hearing. Active middle ear disease significantly effected both conventional and high frequency threshold.

The number of intubulations and frequency of otities media during follow up were significantly and positively associated with poorer thresholds. Several other factors, including middle ear appearance at intabulation,

presence of tympanoslerosis, age, male gender and use of ototopical ear drops, were also associated with poorer high frequency hearing but failed to reach significance after their inter corelation with number of intubutions and frequency of ototies media was considered.

Sanders (1972) : Says idiopathic sudden deafness, generally unilateral and always sensory neural is more common than generally recognized. The site of lesson producing deafness may be either cochlear or neural (retro cochlear). The causes generally are believed to be either vascular or viral. Recently there in evidence to suggest that the two causes need not be separate or mutually exclusive but in fact a viral etiology may cause certain vascular changes that results in a reduced blood flow to the cochlea, and leads to sensory neural hearing loss.

According to Crow Et al (1942) considering the etiology of the sensory neural high tone loss it has been proposed to be caused by a permanantly impaired middle ear ventilation. Recently a tendency to high tone loss has sbeen suggested to be due to sequence of chromic serous otities media (SOM) treated with middle ear ventilation tubes.

Sehilden Et al (1987) : They studied the effects of early assymatric hearing loss in children owing to otities media (O.M.E), on binaural hearing. Five children who had suffered from predominently unilateral O.M.E believed the ages of 2 and 4 years and who were treated for O.M.E at any time participated in lthis study when they were about 12 years of age. The result indicated that the children's ABRs and BICs were comparable to normative data, that there

was evident suppression of transient evoked oto acoustic emmisions in four of the five children and that was also affected. The main threshold shift started at 1 Khz in 8% at 2 Khz in 49% at 4 Khz in 19% and at 6 Khz in 6% of the ears. Impulse noise from large calibre weapon and explosions appeared to cause low-tone hearing loss slightly more often than small arms fire. No etiological differences were apparent between the ears with dip - type hearing loss and ears with more abrupt like hearing loss.

Arora M.N. et al {1976): Survey conducted during the year 1971 through 1972. Among the total population of 3,600 people 1,720 referred to E.N.T. and audio evaluation. In this sample of 1,720 individuals 44 subjects were found to have CSOM i.e., 2.55% in general population, and observed to be major cause of conductive deafness, the results of sex incidence was for Males (14) 31.8% and females (30) 68.2% and age in years showed 45.45% for children with age range 0-14 years, 47.73% for adults with age range of 15 - 45 and 6.82% with the age range of above 46 years.

Misraa et al (1961): Carried out an otolaryngological examination in 120-children and found the incidence of hearing loss to be 34%. In majority of such cases, deafness was found to be of conductive nature.

Sachder and Bhatia(1965): From a study of hospital record found that 30% of the patients attending on E.N.T. outpatient department suffer from chronic supparative otitis media.

Kapur (1965) : In a study of hearing loss in school going children in India, found an incidence of hearing loss between 16.26 and 18.23%

Hacker and **Bali (1972):** Reported that 9.2% of rural population was suffering from discharging ears.

Mitruko - etal (1996) : Conducted a study on 2 patients of age 53/w had noted auditory and vestibular problem since the age of 15 years and another patient of age 68/w had noted problems of the same type since the age of 30 years. They are auditory examinations disclosed mild threshold elevation in pure tone audiometry and markedly pure scores in speech audiometry and good scores in audiometry comprehension test. They were dignored has having auditory nerve disease of unknown cause. Thus showing that certain unknown causes of hearing loss could be unknown.

Ototoxicity has grown out to be another very important acquired cause of hearing loss among adults, children and geriatric population.

Quick (1973) : and some authours have further suggested that alcohol may even be ototoxic causing structural damage to the inner ear which may lead to sensory neural hearing loss and tinnitus.

Robinson Etal (1988) : The toxic effects of cisplatin (cis - diamminedichloro platinum II) on the organ of corti are well established. The present study presents animal experiments on the toxic effects of cisplatin in the stria vascularies and in the organ of corti.

Hoffman Etal (1997) : A new method of local Zentamycin administration was tested in the bullfrog - inner ear to achieve ototoxic induced hair cell destruction. This study demonstrates that thin in a useful and simple method

to investigate the process of vestibular ototoxicity and hair cells regenaration, including aspects of hair cells destruction and repair.

Powers (1972) : Metabolic dysfunction is one of many factors mentioned has a primary or contributing cause of minere's disease (fluctuating hearing loss) which in turn is found to be a very important acquired cause of hearing loss.

After evaluation of 98 patients with minirer's disease, he concluded the following:

1) The five hour glucose tollarance test is helpful in identifing hypoglycemia, which may be a contributing factor in minirer's disease.

2) Non - Myxedematous hypothysoidism may play an important role in some patients with meniere's disease.

3) Further endocrine studies will be necessary to determine the significance of the lowered adreno cortical reserve.

Michael Rolver (**1978)** : Syphilis can cause sensory neural hearing loss that is potentially treatable thus sypilis should be considered in any patient with sensory neural hearing loss of obscure origin, despite a negetive history, for syphilis. The FTA - ABS is the prefer test to exclude hearing loss of syplilitic origin.

Wright (1988) : Delayed endolymphatic hydrops is characterized by a profound sensory neural hearing loss in one ear with the onset of episodic vertigo from that ear after a prolonged period of tone (iprilateral dalayed endolymphatic hydrops) or the development of fluctuating hearing loss or episodic vertigo in the opposite ear after a prolonged period of time.

Sandars (1996) : said that some of the acquired hearing loss manifest suddenly. Most cases of sudden sensory neural hearing loss remeain idopathic, and the majority are unilateral. From 1989 - 1993, 823 patients with sudden sensory neural hearing loss were e valuated. Of these, 14 (1.7%) had sudden bilateral sensory neural heaaring loss. He reviewed the charts of these 14 patients to compare sudden bilateral sensory neural hearing loss with sudden unilateral sensory neural hearing loss. Usually bilateral sensory neural hearing loss was asymmetric. Most bilateral cases received combined steriod and vasodialated treatment, while unilateral cases were more likely to receive onlyone of these treatment. Bilateral sensory neural hearing loss patients were older at the onset of hearing loss had a higher incidence of vascular disease, and were more likely to have positive antinuclear, antibody titer. Many of the specific causes of sudden bilateral sensory neural hearing loss that have been described encomposs a bind spectrum of syndromes and diseases such as infections, mononeutiosis, intracranial aneurysm, cerebrovasculaar accidents, maningitis, intravascsular malignant lymphomatosis, meningial carcinomatosis, leukemia, syplitis, sarcoidosis, multiple slerosis, ulecrative colitis, cogan's syndrome, polyneuritis nodosa, human immuno deficiency virus, auto immense inner ear disease, (AIED) and sickle cell disease.

Tumours can also cause significant amount of hearing loss.

According to Pulee (1972) facial nerve neuroma is one of the cause for the conductive and sensory neural deafness, facial paralysis, otorrahea, or serious intra cranial problems by extension into the middle or posterior

cranial possae. He studied 14 patients with facial nerve neuronal and found an above statement.

According to R.S. Mecrea (1972) carcinoma of the middle ear is one of the cause fosr hearing loss. He studied 6 cases of carcinoma of middle ear there of six alive and free from neoplasm 1 for 8 years and 2 for 3 years. He found that it leads to loss of function of the seventh and 8th cranial nerve, deafness *in* that ear complete and permanent vertigo.

Thus, Hearing loss is one of the most challenging problems confronting medicine, because it can effect personality so adversly. A mild hearing loss some times may produced more psychological disturbance than a severe hearing deficit. It is thin effect of hearing loss on the patient's emotions, rather than the actual deafness. The hearing loss may even bother the peoople arround him more than it does the patient. Deafness is the rather strange symptom, for it is not accompanied by pain, discomport, itch or fear, as at true of cancer and other diseases that imple patient to seek medical and hearing loss is really more a symptom than a disease.

METHODOLOGY

The present study was conducted to study the common causes of acquired loss among adults, children and geriatrics population registered at All India Institute of Speech and Hearing, Mysore.

This study was conducted in two parts

(1) Spot study

(2) File study.

The rationale for conducting the study in two parts was in order to see if there is any difference in acquired causes of hearing loss obtaining from these two studies. Among the adults, children and geriatrics.

Spot study:

Subjects: The subjects randomly selected for the study included children (age Range of 0-15 years), adults (age range of 16-50 years) and geriatrics (those above 50 years).

The spot study was carried out for 200 subjects which included 76 children, 80 adults and 44 geriatrics, as shown in Table 3.1.

Children		Adults		Geriatrics	
Male	Female	Male	Female	Male	Female
44	32	42	38	26	18

Table 3.1. Showing number of subjects taken for spot study: .

Only those subjects with aququired hearing loss were considered for Ihe study.

Materials: A case history form which contained questions that extracted information regarding causes of acquired hearing loss was prepared. General case history format was prepared for adults, childdren and geriatrics population. [A sample of case history form is given in Appendix A].

Procedure: Case history was taken through interview method. The patients were seated comfortably on the chair and were instructed as follows:

"I will be asking you some questions regarding your problem, you are requested to think and give the correct answer. You can ask for repetition if the question are not clear". Prior to this general conversation was initiated with the subjects to build rapport. In the case of children the information regarding the child was obtained from the parents. Care was taken to use simple language while asking questions to the subjects. And the subjects were given adequate time to answer. Also, the subjects were not interrupted while answering, care was also taken to repeat the questions, if necessary to make sure that the subjects understood the questions. Based on the information collected from case history, the possible causes of hearing loss were, tabulated, the obtained results were converted to percentage (%).

File Study:

Subjects: A total of 800 files were studied. Among the 800 files studied there were 373 adults, 118 children and 309 geriatrics population as shown in Table 3.2.

Children		Adults		Geriatrics	
Male	Female	Male	Female	Male	Female
67	051	215	158	161	148
118 Children		373 Adults		303 Geriatrics	

Table 3. 2. Showing Number of subjects taken for spot study

Procedure: The file study was carried out by going through the case files of patients registered in a speech and hearing center. The case files from the year April 1996 to February 1997 were studied because this would help to determine the various causes of hearing loss in patients seen during different periods (seasons) of the year. File were selected randomly for each month. Each file was carefully studied to notedown the possible causes of hearing loss.

RESULTS AND DISCUSSIONS

Data regarding causes of hearing loss was collected from 1000 subjects {800 subjects from file study and 200 subjects from spot study}. Results of the collected samples are discussed below:

(I) Causes of Hearing Problem: From the Table 4.1, it is seen that wax (22.(J%) and car discharge (19./%) have highest percentage of occurrence in general. T.B. (1.8%) and otoslerosis (1.1%) causes of hearing problem.

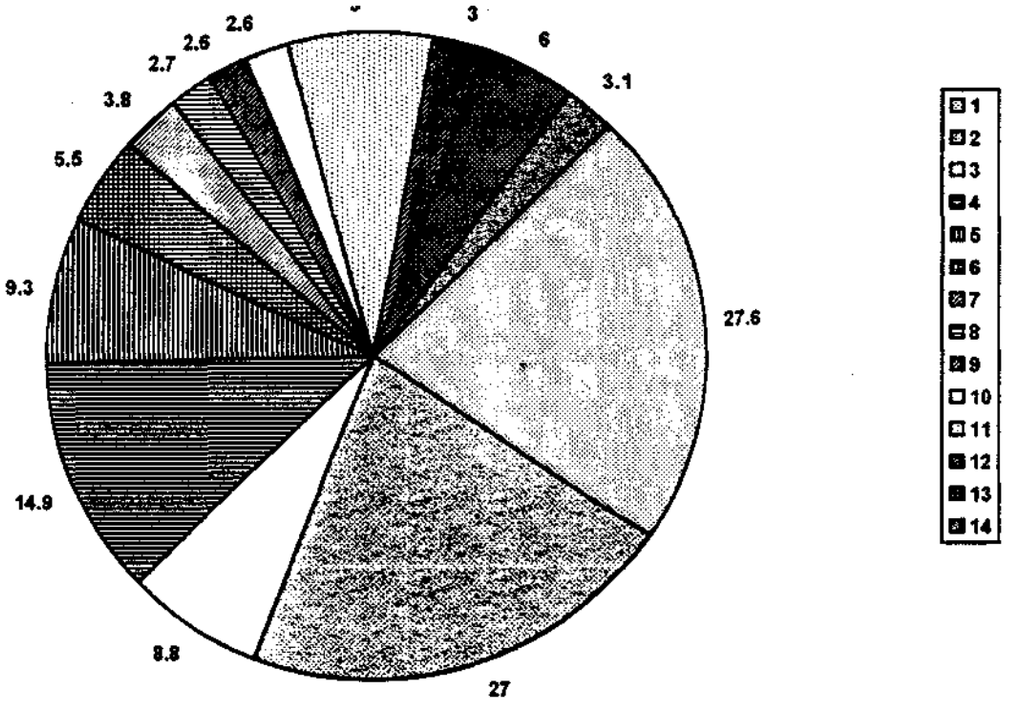
(II) Comparison of Causes of hearing loss in different age groups:

Variability compare with reference to file study and study (Table 4.3)
observed among different age groups for example: for wax percentage of occurrence was 61.3% in children, 10.3% for adults and 8.7% for geriatrics. In children ear discharge with 61.3% and wax with 47.9% have the highest percentage. In adults Otomycosis 16.9% and wax 15% have the highest percentage while in geriatrics wax 19.2% and diabeties 19.8% rated the higest percent of occurrence among other possible causes of hearing
problem.

It can be observed from Table 4.3 and figure 4.3(a) and Figure 4.3(b) that occurrence of ear discharge, Rhinities, foreign body, Tympanic membrane perforation were more in children. These causes generally lead to conductive hearing loss. Similar results have been reported in literature. Results of investigations done by Wrellind (1991) found that otitis media was a major hearing problem in children. Misra et al (1961) reported that in 126 children

	File studv[r]			Spot stud)[r]			TOTAL			
Cause	C	A	G	C	A	G	C	A	G	Total %
Ear Discharge	66.1%	9.6%	7.1%	53.9%"	13.7%	20.4%	61.3%	10.3%	8.7%	19.7%
Rhinuitis	68.6%	8.84%	3.8%	-	.		46.9%	9.4%	6.2%	15.6%
Roreign body	20.3%	3.2%	2.9%	23.6%	16.2%	13.6%	21.6%	5.5%	4.2%	6.8%
Otomaycosis	24.5%	11.7%	8%	35.%	41.2%	18%	28.8%	16.9%	9.3%	16.6%
	50%	11.2%	11.9%	44.7%	32.5%	70.4%	47.9%	15%	19.2%	22.9%
Diabeties	-	6.1%	10.6%	0	26.2%	84%	-	9.7%	19.8%	11.4%
Blood Pressure		4.8%	6.7%	0	36.2%	7.27%	–	10.3%	15.2%	10.1%
Ototoxicity	–	2.1%	6.1%	0	7.5%	25%		3%	8.4%	4.4%
T.M.Perforation	16.1%	3.7%	4.2%	27.6%	22.5%	22.7%	20.6%	7%	6.5%	5.5%
NIHL	-	4.5%	3.5%	11.8%	31.2%	22.7%	4.6%	9.2%	5.9%	7.2%
Trauma.	-	9.1%	-	11.8%	16.2%	0	4.6%	2.8%	0	2.2%
Smoking	-	18%	-	0	23.7%	20.4%	–	**4.1%3**	3.3%	3.1%
Otos-clerosis		9.3%	-	0	11.8%	4.5%		1.9°/o	0.5%	1.1%
Presbycusis	-	-	27%	0	0	72%			9%	3.2%
Typhoid	7.3%		-	-		–	7.2%	2.6%.	0.8%	2.9%
T.B.	-	4.1%	6%	2.6%	8.7%	30.4%	1%	1.5%.	2.5%	1.8%

Table 4.1 Shows % of diffrerent possible causes of hg loss in different groups.

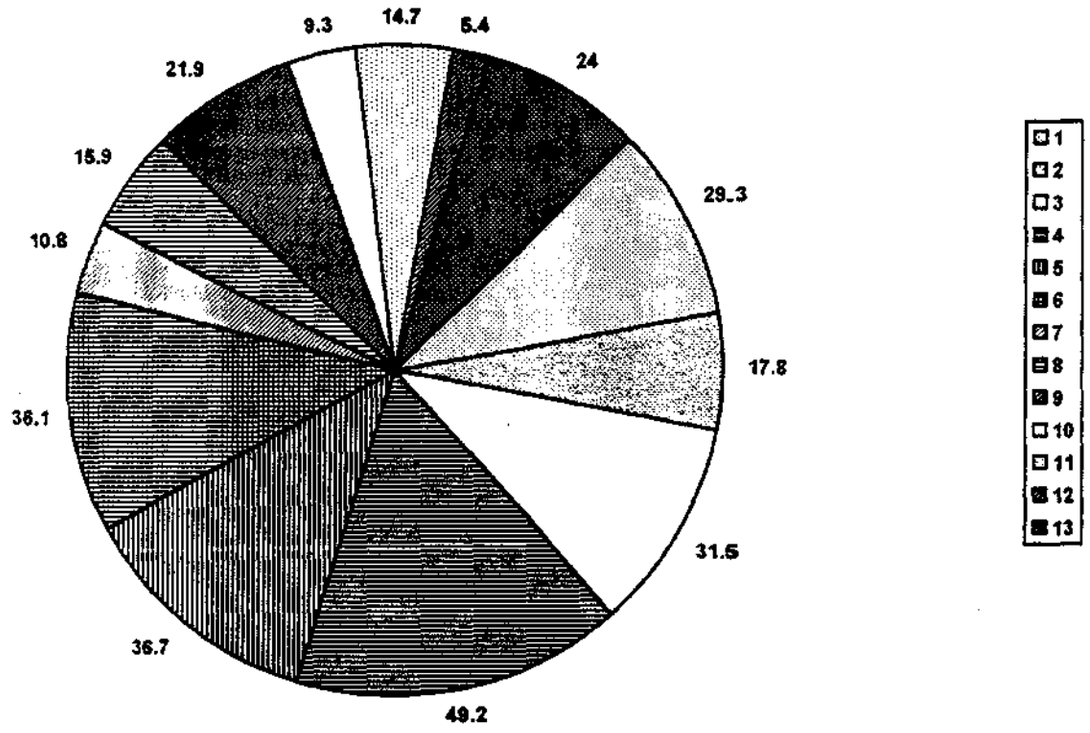


1. Ear discharge 2. Rhinities 3. Foreign body 4. Otomycosis 5. Wax 6. Diabetes

13. Smoking 8. Ototoxicity 9. TM perforation 10. NIHL 11. Presbycusis 12. Trauma

7. B.P. 14. Otosclerosis

Figure 4.1 (a). Graphical representation of possible causes of acquired hearing probolems obtained throgugh file study.



1. Ear discharge 2. Foreign body 3. Otomycosis 4. Wax 5. Diabetes 6. B.P.

7. Ototoxicity 8. TM perforation 9. NIHL 10. Trauma 11. Smoking 12. Otosclerosis

13. Presbycusis

Figure4.1(b). Graphical representation of possible causes of acquired hearing probolems obtained through spot study.

		C		A		G		TOTAL
		M	F	M	F	M	F	
File study	No.of subject	67	51	215	158	161	148	800
	%	8.3%	6.3%	6.8%	19.7%	20.1%	18.5%	
Spot study	Nos. of subject	44	32	42	38	**26**	18	200
	%	22%	16%	**21%**	19%	13%	9%	
					GRAND TOTAL			1000

Table 4.2 Showing the Number and percentage of children, adults and geriatrics included the file study and spot study.

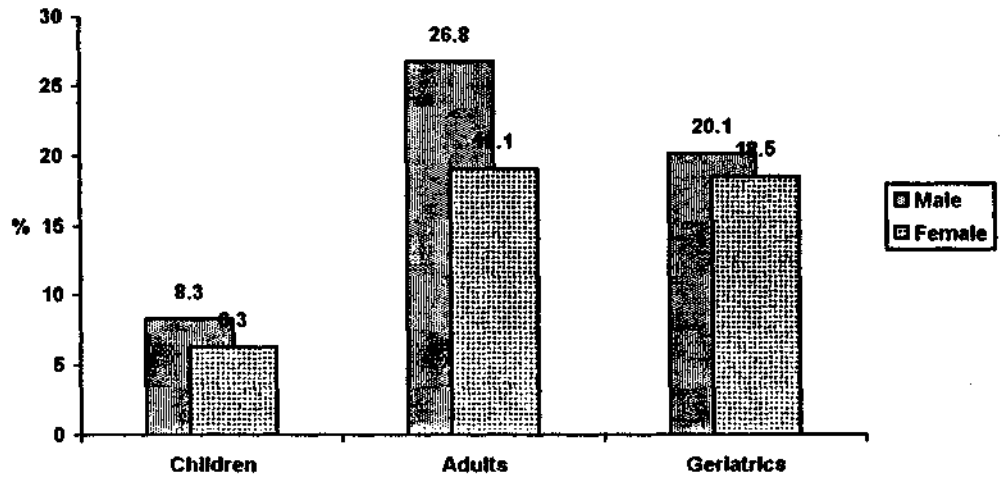


Figure 4.2 (a). Showing the percentage of children, adults and Geriatrics included in the file study.

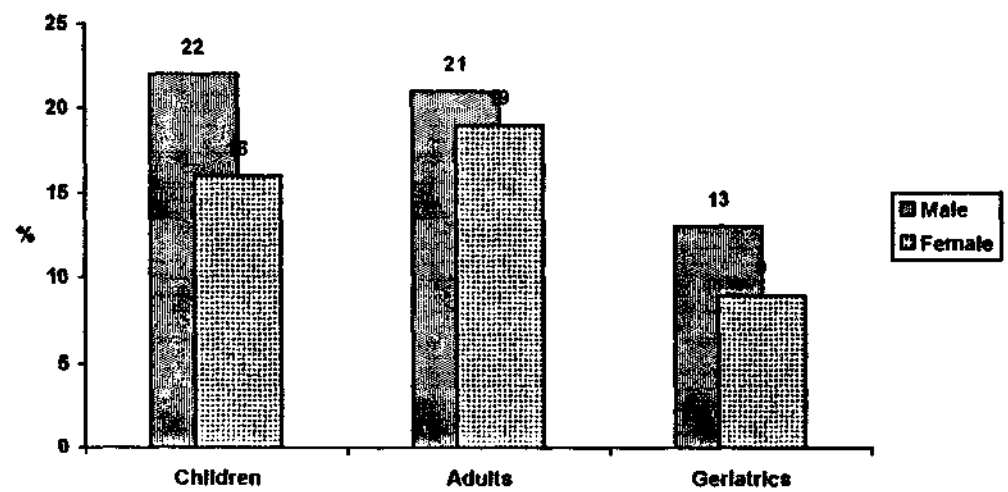
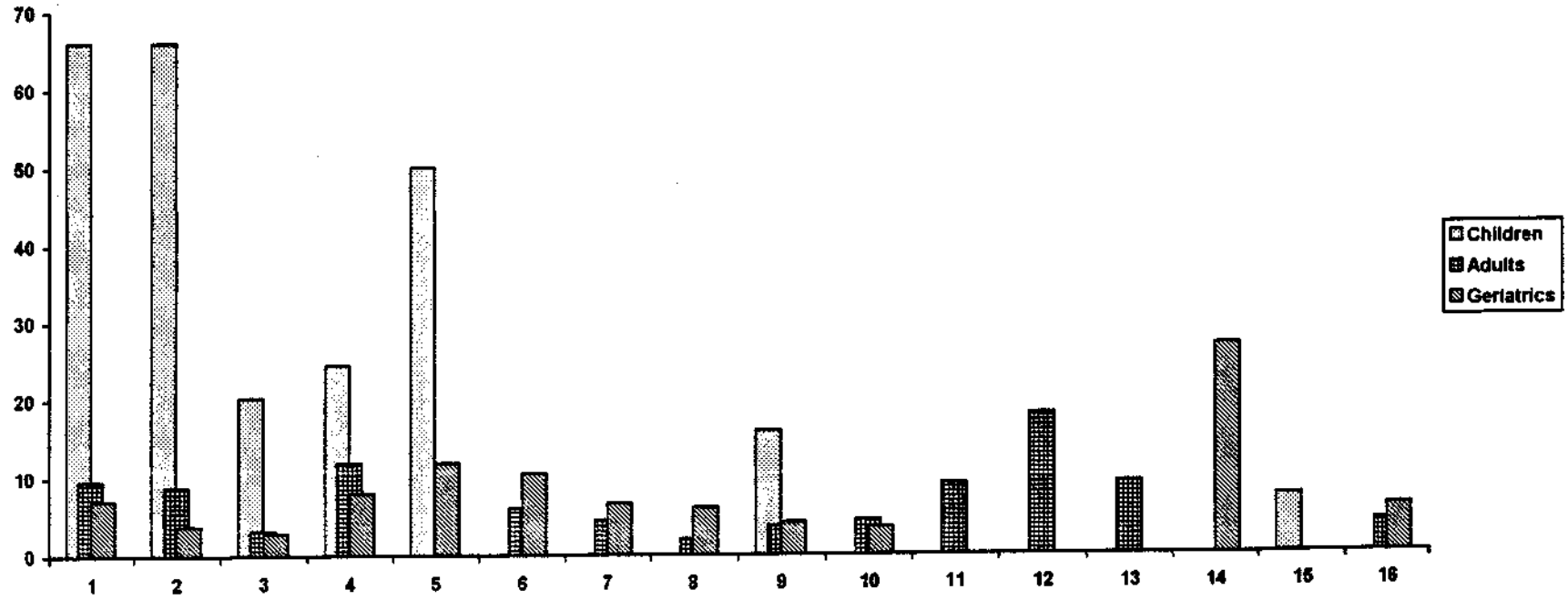


Figure 4.2 (b). Showing the percentage of children, adults and Geriatrics included in the spot study.

	File study			Spot study		
Causes	C	A	G	C	A	G
Ear discharge	66.1%	9.6%	7.1%	53%	13.7%	20.4%
Rhinitis	68.6%	8.84%	3.8%	-	-	-
Foreign body	20.3%	3.2%	2.9%	23.6%	16.2%	13.6%
Otomycosis	24.5%	11.7%	8%	35.5%	41.2%	18%
Wax	50%	11.2%	11.9%	44.7%	32.5%	70.4%
Diabities	-	6.1%	10.6%	-	26.2%	84%
Blood pressure	-	45.8%	6.7%	-	36.2%	72.7%
Ototoxicity	-	2.1%	6.1%	-	7.5%	25%
TM Perforation	16.1%	3.7%	4.2%	27.6%	22.5%	22.7%
NIHL	-	4.5%	3.5%	11.8%	31.2%	22.7%
Trauma	-	9.1%	-	11.8%	16.2%	-
Smoking	-	18%	-	-	23.7%	20.4%
Otosclorosis		0.3%			1 1.8%	1.5%
Presbycusis	-	-	27%	-	-	72%
Typhoid	7.3%	-	-	-	-	-
T.B.	-	4.1%	6%	2.65	8.7%	30.4%

Table 4.3: Shows variation in different groups for various possible acquired causes of hearing problem in both file and spot: study.

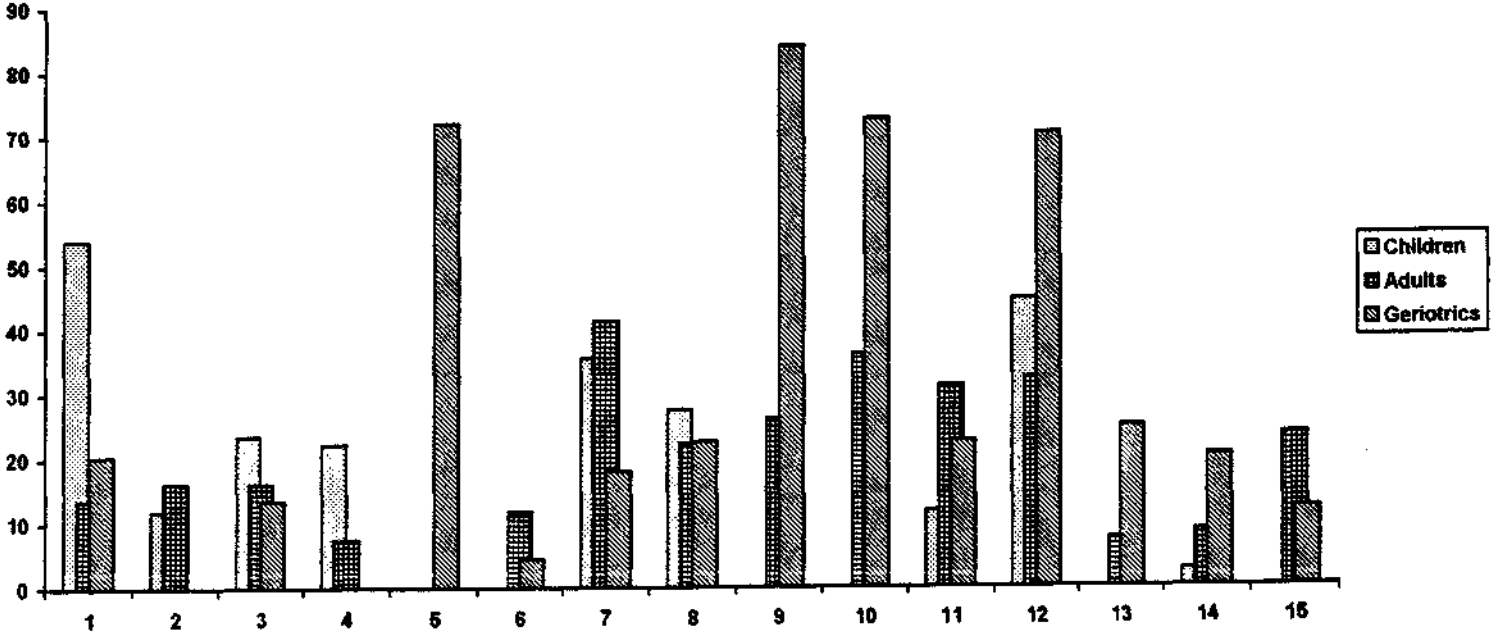


1. Ear discharge 2.. Rhinities 3. Foreign body 4. Otomycosis 5. Wax 6. Diabetes

7. B.P. 8. Ototoxicity 9. TM perforation 10. NIHL 11. Trauma 12. Smoking

13. Otosclerosis 14. Presbycusis 15. Typhoid 16. T. B.

Figure 4.3(a) : Shows variation in various acquired possible causes of hearing loss in children. adults and geriatrics reported in file study



1. Ear Discharge 2. Trauma 3. Foreign body 4. Consagunity 5. Presbycusis

6. Otosclerosis 7. Otomycosis 8. TM perforation 9. Diabeties 10. B.P.

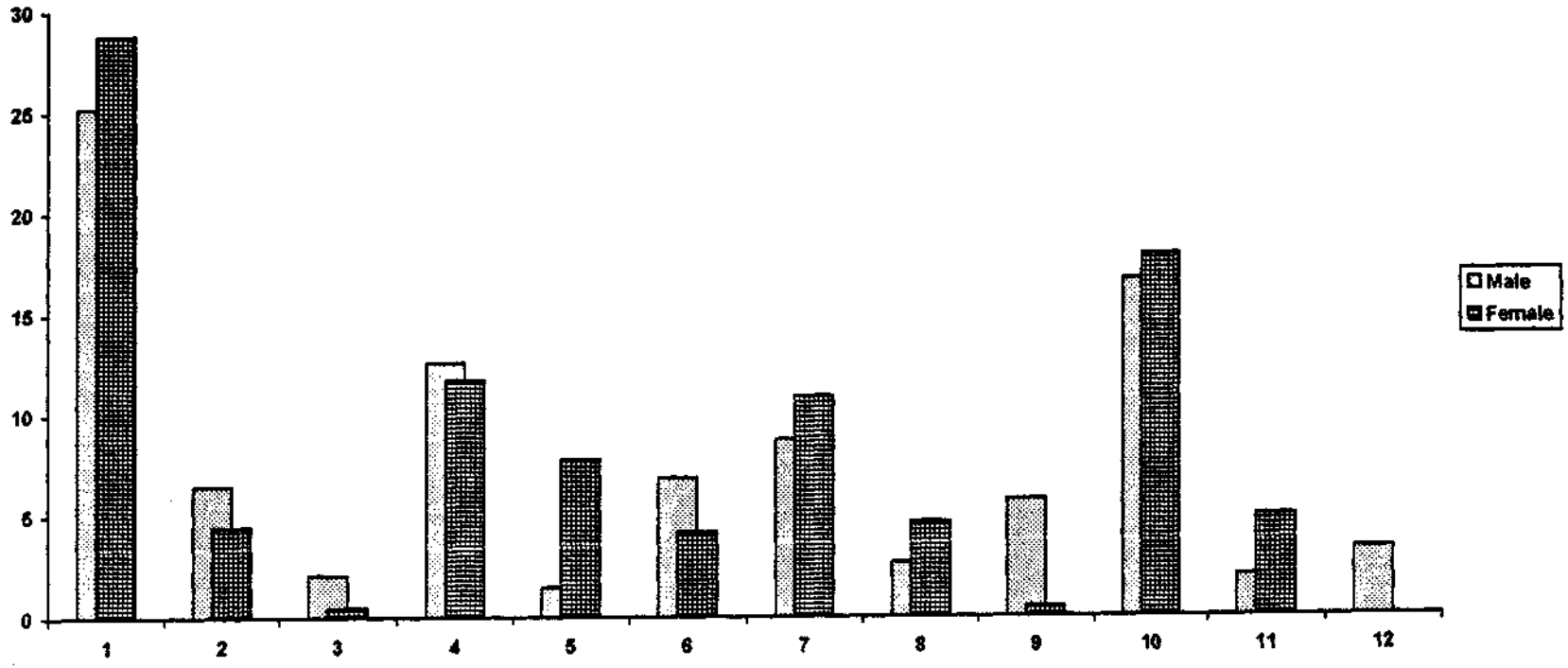
11. NIHL 12. Wax 13. Ototoxicity 14. T.B. 15. Smoking

Figure 4.3(b). Shows variation in various acquired causes of hearing problem in children, adults and geriatrics reported in spot study.

external ear and middle ear. Wax, otomyrosis, NIHL were major causes of acquired hearing loss in adults, study done by Sheppered et al (1983), Birningum (1973), Fosharote (1831), Bundh (1937) have also reported that NIHL was a major cause of hearing loss in adults. Diabeties, blood pressure, ototoxicity, T.B., smoking were the major causes of hearing loss in geriatric population. Cruick (1973), Robinson et al (1988), Hoffman et al (1997) opine that ototoxicity to be a singnificant cause that leads to hearing problem.

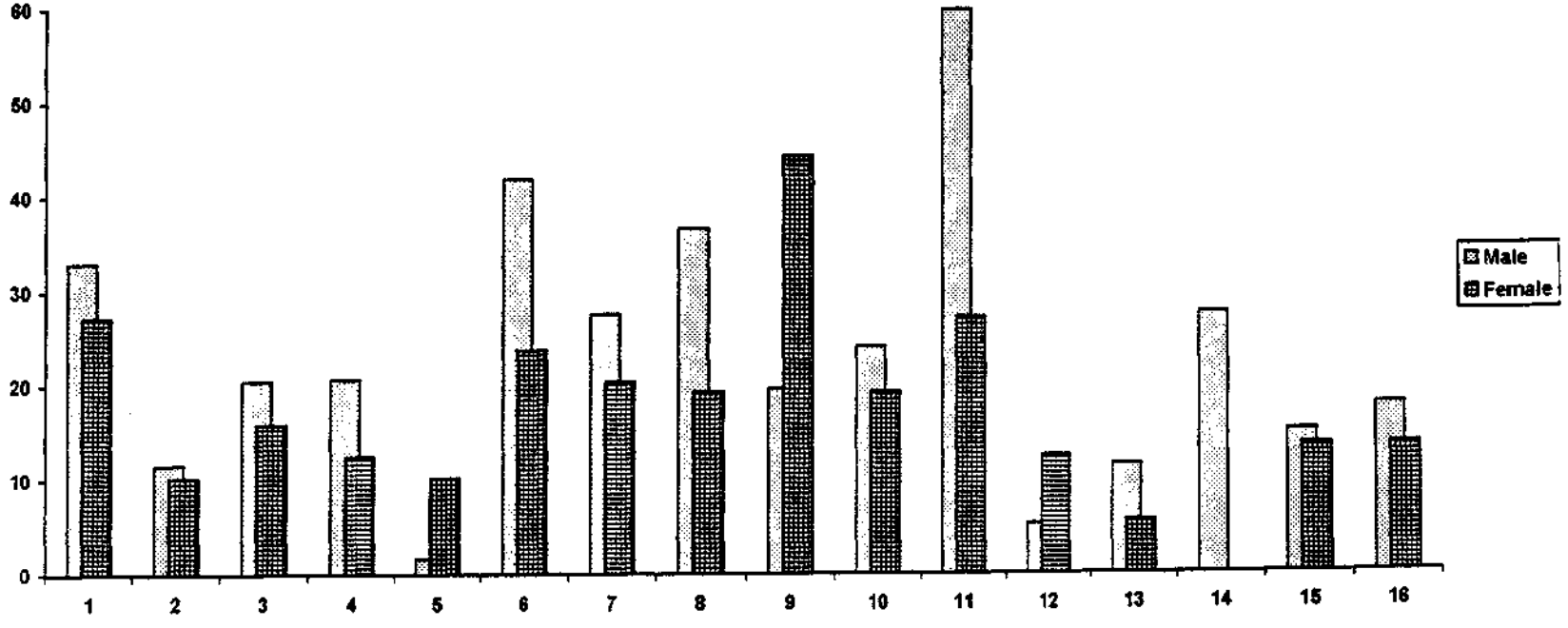
(Ill) Comparision of causes of hearing loss in males and females: Table 4.2 and Figures 4.4(a), 4.4(b) show that the possible causes of hearing problem were more widely distributed. There were more females (7.8%) who had otoslerosis compared to male (1.5%). Diabeties (Male 8.8%, Female 10.92%) NIHL (Male 5.8%, Female .5%) were more in males when compared to females. According to Prescod (1976) there is a greater prevelence of otoslerosis among women then men. In general during periods such as puberty, pregnency, childbirth menopause there is a marked likelyhood for occurrence of earproblems among females. The results of the present study supports this findings. In both the studies i.e., spot study and file study there were more males than females. The reason for males to be more prone to hearing loss than females could be due to the following reasons.

1. More number of males work in industrial area i.e., un healthy condition such as noisy area (Shepperd et al, 1983).
2. More number of males are smokers and alcoholics.



1. Ear Discharge
2. Foreign body
3. Stroke
4. Otomycosis
5. Otosclerosis
6. TM perforation
7. Diabetics
8. B.P.
9. NIHL
10. Wax
11. Otooxicity
12. Smoking

Figure 4.4 (a). Shows causes of hearing loss in both males and females file study.



1. Ear Discharge 2. Trauma 3. Foreign body 4. Consagunity

5. Otosclerosis 6. Otomycosis 7. TM perforation 8. Diabetics

9. B.P. 10. NIHL 11. Wax 12. Otooxicity

13. T.B. 14. Smoking 15. Typhoid 16. Presbycusis

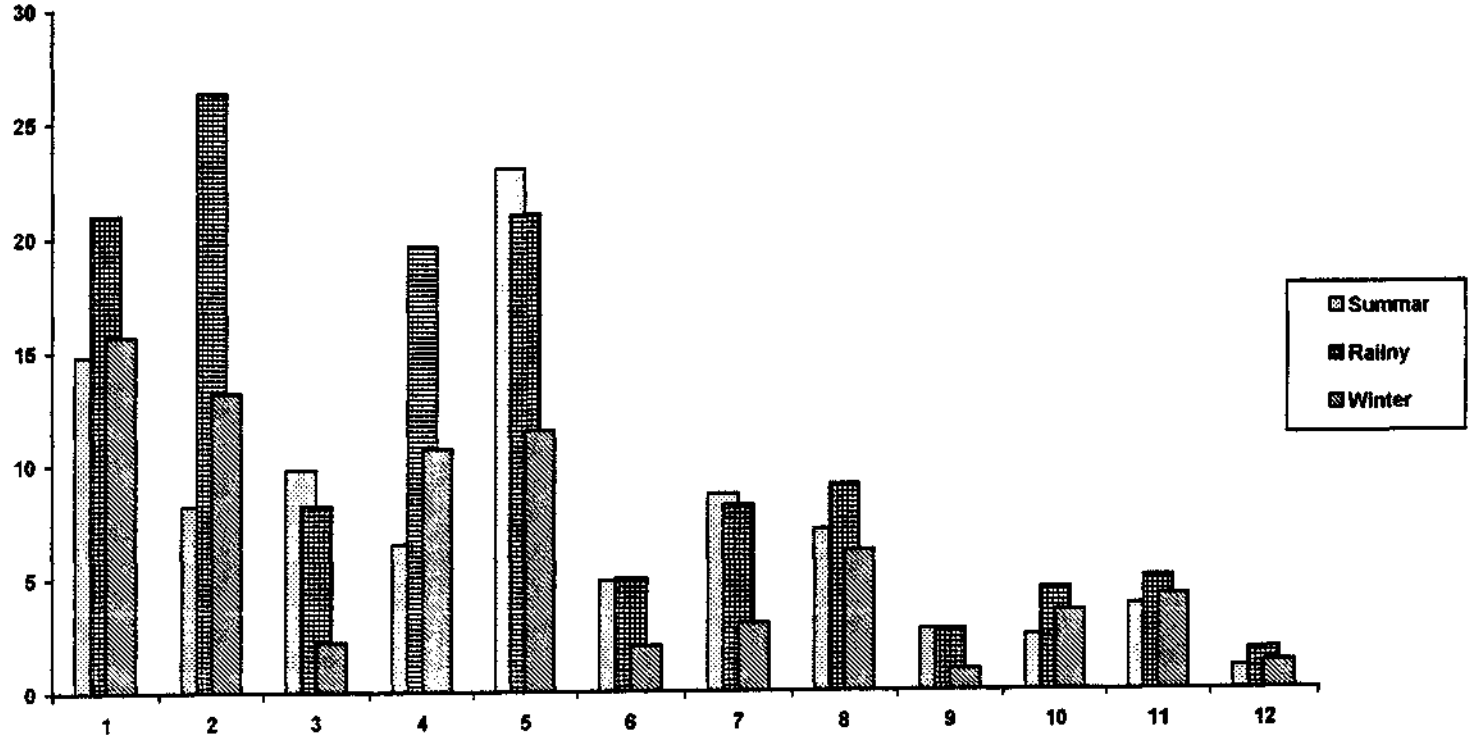
Figure 4.4 (b). Shows causes of hearing loss in both males and females spot study.

Dangerick et al (1987), Axelsson et al (1992) found that smoking could be a significant cause of hearing problems in males. It is also likely that more males than females report to a speech and hearing center seeking redressal.

(IV) Comparison of causes of hearing loss in different seasons: In general Figures 4.5 and 4.5(a) show that, during rainy season, more number of subjects reported with the complaint of ear discharge, Rhinitis and Otornycosis. However the incidence of Tympanic membrane perforation, foreign bodies and wax seem to be occur more in summer season. In winter season percentage of occurrence were less compared to the other season. However patients with CSOM and Otomycosis were found to report more numbers in winter compared to summer season. Ramalingam (1990) found that serous otitis media occurred more frequently in winter season for children below the age of 10 years. This study contradicts the findings of the present study where CSOM was found to be more in rainy season. Patients with diabeties, B.P. Otoslerosis , Cerebro vascular accidents, reporting did not show seasonal variation.

	Summer season	Rainy season	Winter Season
Ear discharge	14.8%	21%	15.7%
Rhinitis	8.2%	26.4%	13.2%
Foreign body	9.8%	8.2%	2.2%
Otomycosis	6.5%	19.6%	10.7%
Wax	23%	21%	15.%
N1HL	4.9%	5%	2%
Tympanic membrane perforation	8.7%	8.2%	3%
Diabeties	7.1%	7.3%	6.2%
Blood pressure	2.1%	4.5%	3.5%
Typhoid	2.7%	2.2%	.9%
Oloslerosis	3.8%	5%	4.2%
Stroke	1%	1.8%	1.2%

Table 4.5 Showing % of possible required causes of hearing loss in different seasons.



I. Ear Discharge 2. Rhinities 3. Foreign body 4. Otomycosis 5. Wax

5. NIHL 7. TM perforation 8. Diabeties 9. Typhoid 10. B.P.

II. Otosclerosis 12. Stroke

Figure 4.5(a). Shows the causes of acquired hearing loss during different seasons.

SUMMARY AND CONCLUSION

Hearing loss in an individual can be caused due to wide variety of factors. It can be hereditary, congenital or acquired. The present study was taken up with an aim to study the common causes of acquired hearing loss among children, adults and geriatrics seeking professional assistance in a speech and hearing centre. The subjects randomly selected and included children (age range 0 - 1 5 years), adults (age range of 16 - 50 years) and geriatrics (above 50 years). The study was conducted in two parts spot study and file study. The results of study can be summerzied as follows:

(1) Among 1000 subjects whose files were scrutinised, the maximum number of subject with acquired hearing loss were adults (393) followed by geriatrics (309) then children (118). Whereas in spot study, the total number of adult subjects were 76, geriatrics 44 and children 76. From both studies it was found that more adults with hearing loss sought professional help.

(2) In all three age groups more male subjects were found to have reported than females in both file and spot studies.

(3) Both studies revealed that the etiological factors in the order of incidence was as follows: Ear discharge, Wax, Rhinitis, Otomycosis followed by Diabeties, Blood Pressure, NIHL.

(4) In general greater incidence of otological problems occured in the rainy season. Incidence of foreign body and wax seemed to occur more in summer. In winter

season frequency of otological problems were less in compated to other seasons.

(5) Almost similar findings were obtained with file and spot studies. Wax, Otomycosis, NIHL were major causes of hearing problem in adults. Whereas, ear discharge/infection, foreign body, Rhinitis were more in children and diabeties, blood pressure, tuberculosis, otomycosis, presbycusis were more in geriatric populations.

BIBLIOGRAPHY

Adelman.S (1983). "Identification of Acoustic neuroma in noise exposed workers" Scandanavian Audiology, 12, 247 - 250.

Arora, M.M.L. et al (1976). "Incidence of chronic suppurative otitis media in general population", (A rural survey), Indian journal of Otolaryngology, 28, 35-39.

Axelson.A.L. (1992).. "The interaction of smoking and noise on temporary threshold shift". Acta otolaryngology 112, 932 - 938.

Ballantyne and Martin JAM (1984). "Conductive deafness" cited in Ballantyne and Martin in "Deafness" (ed 1984 , 91 - 238, Melbourne and New York.

Belfast.C (1972). "Radial surgery for carcinoma of the middle ear" laryngoscope 82 July - December, 1514 - 1523.

Birningham Ala et al (1972). "Acoustic trauma of Sportsman, Hunter due to gun firing, Laryngoscope 82, 1971-89

Coles R.R.A. (1976). "Cochleo - vestibular disturbances in diving" Audiology 15, 273 - 278.

Columbus M.D. (1972). "Symposium on Ear diseases, sudden deafness and its treatment" laryngoscope - 82, 1206 - 1213.

Costa A.G. (1987). "Hearing loss in school children" , Scandinavian Audiology . 16, 137 - 143.

Davis and Siverman "hearing and deafness" cited in Davis and Silverman in Hearing and Hearing loss 9 - 175, Rim hart and Winston Inc.

Dengerink J.F. et al (1987) "The effects of smoking and physical exercise on temproary threshold shifts". Scandinavian Audiology, 16, 131-136.

Fridjhon P.J. etal (1996). " A pilot investigation of high frequency Audiometry in obscure audiometry dysfunction (OHD) patients", British Journal of Audiology 30 (4) , 233 - 237.

Gerlif J.N. et al (1996) "CSF loss and Threshold changes", Audiology and Neurotology . 1 (5) 256 - 264.

Giebrank.S.G. etal (1996) " High frequency hearing loss associated with otitis media. Ear and Hearing 17(1) 1-11.

Girard F.K etal (1978). "Detection of syphilitic hearing loss", Archives otolorayngology, 104, 63 - 67.

Histelberger, E.W. etal (1988). "Vertigo caused by Basilar artery compression of the 8^{lh} nerve", laryngoscope 98, 807 - 809.

Hoffman L.C. et al (1997). "Intraotic Administraties of Gentamycin. A new method to study ototoxicity in the crista ampullaries of the Bullforg". Laryngoscope . 17, 137 - 143.

Izhak L.H. etal (1988). "Cisplatin ototocity in guinea pigs with special reference to toxic effects in the straia vascularies" Laryngoscope .98 865.

Judith white.S.H. etal (1988). "Atrophy of the stria vascularies as a cause of sensory neural hearing loss" Laryngoscope 98, 754.

Jukka Ylikoski (1987) "Audiometric configuration in acute acoustic trauma caused by firemans", Scandinavian Audiology, 16, 115-120.

Krileny. P.R. J. et al (1996). "Effects of intra typically delivered lidocanine on the Auditory system in the Humans". Ear and Hearing 17 (1). 49 - 54.

Landen R.D. et al (1972). "Otological problems of the Alaskannative Population" Laryngoscope . 82, 1793 - 1893,

Lindsay R..J. (1973). "Histopathology of deafness due to postnatal viral disease" Archives otolaryngology ume 98, 258 - 262.

Lingram F.D. (1987). "The effects of smoking and physical exercise on Temporary Threshold shift", Scandinavian Audiology, . 16, 130 - 136.

Martin F.N. (1984). "Introduction to audiology" 1-15, Prentice Hall Englewood, Cliffs, New Jersey.

Mc Namara M.OP. et al (1985). "Hearing loss in the Elderly" Epidermologic study of the Franingham Heart Study". Ear and Hearing, 6 (4) 184-190.

Meyer.E.S. etal (1991). "The Binaural Masking Level differences in children with a history of otitis media" Audiology 30, 91-101.

Mirko T.J. et al (1996) "CSF loss and threshold changes" Audiology and Neurotology 1 (5) 248.

Newby (1964). "Audiology Appleton - Century Crafts, Inc.

Powers H.W. (1972) "Symposium On Mineare's disease" Laryngoscope .82 1716-1725.

Prescod (1978). "Audiological hand book of hearing disorders" 5 - 209, Ed Litton Educational Publishing Inc, Melborne New York.

Ramalingam, K.K. Sriramamurthy "A short practice of otolaryngology" 46 - 57, AITBS publishers Delhi.

Randolph G.G. etal (1969) " Neurofibromatosis as a cause of conductive hearing loss" Archives Otolaryngology 89, 703 - 708.

Reddel C.R. etal (1972) "The presbycusis component in occupational hearing loss" Laryngoscope 82, 1399 - 1409.

Rudge. P. L. et al (1996) "Auditory deficits and hearing loss associated with focal brainsten hoemorrhage" Scandnavian Audiology 25 (2) 133 - 141.

Sanders. E.J. etal (1996) "Sudden bilateral S N hearing loss" Laryngoscope 106, Nov, 1347 - 1350.

Sataloff (1980) "Hearing loss" 2nd edn cited in Sataloff in "classification of hearing losses" Lippen cott company, Philadelphia.

Sehilden S.M. et al (1987) "Measures of binaural hearing in children with a history of assy metric otitis media with effusion", Scandanauvian Audiology 16, 1 15 120.

Sheppard H.N. (1983) "Hearing Deterioration in shipyard workers", Scandinavian Audiology 12, 265 - 271.

Shinedo K.Y. et al (1996). "Auditory nerve disease of both ears revealed by auditory brain responses, electrocochleography and OAE", Scandinavian Audiology . 25, 233.

Shlomo Silverman G.A. (1985) "Functional hearing loss and relationship to resolved hearing levels". Scandinavian audiology" 6 - 183.

William wright J.I. etal (1988) "Delayed Endolymphentic hydrops: A review of 15 cases" Laryngoscope 98 , 840 -.845.

APPENDIX

CASE HISTORY FORM (GENERAL)

Case Name	:		
Case No.	:	Sex	:
Date	:	Nationality	:
Education	:	Income	:
Occupation	:	Mother tongue	:

I. Complaint:

a) HL with no speech defect
b) HL with speech defect
c) HL with no speech
d) Ear infection
e) H/o Ear aches
f) H/o Ear discharge

Duration:
Continuous/ Recurrent
Profuse/ Modest/Scanty and foul
Purulent/ Mucopurulent/ Blood stained

II. Tinnitus High pitched
Low pitched

III. Vertigo Episodes and frequency

sensation of:

(a) Rotation of object

(b) Rotation of head

Occurrence :

(a) In Lying down position
(b) On standing up only

(c) On walking

IV. Discovery of problem:

1) Age when hearing loss was first noticed
2) The condition
   * Remained Unchanged
   * Improved
   * Became worse

3) Who was the first to notice it ?

4) Age at which medical attention was sought ?

V. Past History:

a) Pre-natal history:

i) No illness or complications
ii) Viral infections
iii) Infectious diseases
iv) Tropical diseases
v) Toxaemia
vi) H/o miscarriage
vii) Drug therapy
viii)Deficiency diseases
ix) Excessive vomitting
x) Others
xi) Systemic disorders
* Leukaemia
* Pernicious, Anaemia
* Diabetes
* Nephritis

VI. Birth complications:

- No complications

a) Home delivery
b) Hospital delivery
i) Pre-mature delivery
ii) Post-term delivery
iii) Prolonged labour
iv) Precipitate labour
v) General anaesthesia
vi) Instrumental delivery
vii) Physiological Jaundice in the 1st 3 - 4 days
viii) Rh incompatibility
ix) I lypoxia
x) Breech delivery
xi) Congenital deformity
xii) Birth weight
- under
- over
- normal
xiii) Others:

VII. Post Natal:

1) No head injury
2) Head injury only
3) Head injury with unconsciousness
4) Head injury with bleeding from nose
5) Head injury with bleeding from ears

VIII. Exposure to noise:

1) No exposure to noise
2) Exposure to noise
- Duration
- No. of hours/day

Noise level

* Sevet
* Moderate
* Mild
* Continuous
* Intermittent
* Impact
* Acoustic trauma
* Otitic blast injury

IX Infections:

i) No infections
ii) Mumps
iii) Measles
iv) Chicken pox
v) Small pox
vi) Influenza
vii) Typhoid
viii) Whooping cough
ix) T.B.
x) Syphilis
xi) Meningitis
xii) High fever without convulsions
xiii) High fever with convulsions
xiv) H/o of drug intake:

Streptomycin
Salicylates :
Neomycin:
Others

X. Metabolic disorders:

* Diabities
* Myxaoedema
* Deficiencies

XI Familial background:

H/o defect illness	Relation to patient	Treated /untreated
1. Speech problem		
2. Hearing loss		
3. Mental Retardation		
4. Epilepsy		
5. Mental illness		
6. Physical defects (specify)		

X. Family constellation:

Siblings:

* Number of brothers and sisters well or ill

* Other persons in the house (mention relationship)

XII Consanguinity:

Family history

Yes/No (Specify relation)
Yes/No Higher in family tree

XII. Unusual behaviour observed:

a) None
b) Purposeless hand motions
c) Unusual posturing
d) Hyperactivity
e) Withdrawal
f) Perseveration
g) Echolalia
h) Others:

Provisional diagnosis:

Student Clinician