"A CLINICAL STUDY OF AGE RELATED HEARING LOSS AMONG DIABETES PATIENTS"

By

Dr. SHEETAL.K

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IN

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UNDER THE GUIDANCE OF

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LIST OF ABBREVATIONS

SNHL	Sensorineural Hearing loss
DM	diabetes mellitus
HTN	hypertension
dB	Decible
M	male
F	female
Hz	Hertz
HbA1c	glycated heamoglobin
FBS	fasting blood sugar
PPBS	post prandial blood sugar
PTA	pure tone audiometery
TFT	tuning fork test
Y	yes
N	no

ABSTRACT

Background and objectives

Age related hearing loss is one of the most common health conditions affecting the elderly individuals. With increase in age, risk of presbycusis as well as diabetes increases. An exact cause effect relationship between the two has not yet been established. The present study aims at evaluating the auditory dysfunction in patients with type 2 Diabetes mellitus aged above 50yrs as compared to that of non diabetic individuals, and also tries to find the relation between the duration of diabetes with severity of hearing loss alongwith the type and severity of hearing loss in controlled and uncontrolled diabetes mellitus.

Methodology

A cross sectional study on 106 patients with type 2 Diabetes mellitus and 90 patients with age and sex matched controls was carried out during the period from November 2011 to October 2013. All these patients were evaluated for hearing loss by subjecting them to Pure tone audiometry and other necessary blood investigations like glycated heamoglobin, fasting and post prandial blood sugars alongwith serum creatinine levels.

Results

Hearing loss was evaluated in all these patients undergoing the study. It was observed that the degree of hearing loss increased as the age progressed in both these groups (diabetics and pure presbycusis). There was a bilateral progressive sensorineural hearing loss with a right sloping curve in both diabetics as well as in controls with pure presbycusis, but with a slightly higher range in diabetics

(marked increase at 4KHz and 8KHz). Most of these cases had mild or moderate to moderately severe hearing loss. A prevalence of 73% SNHL was observed in diabetic cases. A significant relation between the duration of the diabetes and severity of hearing loss was seen. We also observed a strong association between the HbA1c and severity of hearing loss.

Conclusion

With ageing the hearing loss increases proportionately in both diabetes mellitus as well as in presbycusis, but a slightly higher range in diabetes. The hearing threshold was seen to affect all the frequencies but mainly the high frequencies (4KHz and 8KHz) in diabetics. As the duration of diabetes increases the severity also increased. Poorer the glycaemic control (HbA1c) more severe was the degree of hearing loss.

Key words – Type 2 diabetes mellitus, presbycusis, SNHL, Pure tone audiometry.

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INTRODUCTION

Age-Related Hearing Loss (ARHL) is one of the three most common chronic health conditions affecting individuals aged 65 years and older.[1] The high prevalence of age-related hearing loss compels otolaryngologists and the audiologist to understand the pathogenesis, neural, genetic, and molecular mechanisms underlying this disorder.

In 1857, Jardao for the first time showed that diabetes mellitus causes hearing loss.[2] Diabetes mellitus is a metabolic disorder, known to cause a progressive damage to the inner ear mainly organ of corti and spiral ganglion neurons causing hearing impairement.[1] Prevalence of diabetes mellitus shows an increasing trend worldwide approximately around 2.8% and it is estimated to increase to 4.4% by 2030.[3]

Eventhough in the past various studies have shown Diabetes mellitus to cause hearing loss, a confirmed cause-effect relationship between the two has not yet been described clearly. It is now thought that there are a series of variables that may be associated with both the conditions, but more studies are required to establish the true role of these factors.

With ageing both hearing loss as well as the risk of diabetes increases.[4] As a result it is difficult to distinguish whether the hearing loss in diabetes is due to normal process of ageing or due to biochemical and the vascular abnormalities that causes hearing loss.

Diabetes mellitus is known to cause bilateral progressive sensorineural hearing loss.[2,4,5]. But the hearing loss seen in these patients would be similar to that of presbycusis but with more severe losses and early onset than expected by ageing alone.[4] Angiopathy and neuropathy have been considered as the two main factors for the vestibular-cochlear dysfunctions in diabetes.

- Angiopathy occurs by endothelial proliferation with increase in the thickness of basement membrane of capillaries (tunica intima) and small vessels thereby reducing the blood supply to cochlea affecting the inner and the outer hair cells.[6,7]
- Neuropathy is directly dependent on microangiopathy. Due to vascular abnormality there is decreased blood and nutrient flow to neurons as a result of which there is myelin and axonal degeneration.[6,7]

As a result of these above factors, diabetic patients are more likely to develop hearing loss. Unfortunately, hearing impairement often receives minimal attention.

Hearing loss affects the most common and simple tasks of daily life thereby affecting the quality of life. [8] Due to the ageing as well as the other comorbidities (DM and HTN) elderly people experience difficulty in understanding the rapid speech, heavily accented language, and speech with few contextual cues and added memory thereby affecting the quality of life.[8] Hence early identification and rehabilitation helps to restore better quality of life.

Most of the reported studies have shown diabetes to affect the hearing threshold in both young and elderly diabetics (type 1 and type 2 diabetes), and in that some studies showing higher frequencies being affected more than lower or mid frequencies [9,10,11,12]. Whereas other studies have reported to affect all the frequencies [4] and some others showing only in the lower frequencies.[13] Although many of these studies have shown contradicting results regarding hearing impairement in diabetic patients, the number of these studies done in India is small.

The present study is undertaken to determine the occurrence of auditory dysfunction in type 2 diabetes patients and whether or not such auditory dysfunction could be correlated with the severity of hyperglycemia.

AIMS AND OBJECTIVES

- To evaluate the occurrence of auditory dysfunction in patients with type 2 diabetes patients aged above 50yrs and to compare the same with age matched control groups.

- To study the relation between degree of hearing loss, the duration and severity of type 2 diabetes mellitus.

- To evaluate hearing loss and pattern in controlled and uncontrolled diabetes mellitus.

REVIEW OF LITERATURE

PRESBYCUSIS AND PATHOPHYSIOLOGY IN HEARING LOSS

Hearing loss is one of the most prevalent chronic health conditions affecting the older individuals. Age related hearing loss is defined as mid to late adult age onset, bilateral, progressive sensorineural hearing loss where all the other underlying causes have been excluded.[1] The term presbycusis (Greek word, Presbys—old man and Akousis—hearing) was first described in 1874.[14]

Hearing has a great impact on the quality of life related to social, functional, and psychological wellbeing of the person.[8] Hearing impairment is a common problem in older age group and the degree of impairment and its prevalence increase with age. [14] Ries in 1994, in one of the health interview survey done in 1990-91, reported that only 9.7% of the older individuals aged older than 65yrs had normal hearing whereas around 50% of the presbycusis patients had hearing impairement or difficulty in understanding the speech, had restricted limitation in daily activity.[15]

Hearing is very important for communication, identifying the potential warning signals to the injurious events and appreciation of music or natural sounds.

Age Related Hearing Loss is said to occur when all the other primary factors such as prolonged noise exposure, medical condition like Atherosclerosis, Diabetes mellitus, Hypertension, Myxedema and intrinsic otological diseases (eg Otosclerosis, Chronic otitis media, Meniere‘s disease), Head injury & Ototoxic drug therapies have all been excluded [1]. It is difficult to assess what component in an individual leads to hearing loss as a consequence of physical stress.[1,16]

Presbycusis, presents with loss of sensitivity for the high-frequency sounds, resulting in difficulties in speech perception, hearing in noisy backgrounds, and distorted loudness perception.[17] As a result this poses a great challenge to the otolaryngologist and audiologist to understand the importance of the ongoing mechanism involved with it. Early identification and rehabilitative measures will improve quality of life in these individuals.

There are multiple variables that have been evaluated which contribute for hearing loss with aging.[16,18] These include:

1. Micro vascular pathology resulting in diminished perfusion and hypoxia to the labyrinthine hair cells and neurons.
2. Formation of free radicals
3. Noise exposure
4. Drug induced effects
5. Effects of cigarette smoking.
6. Genetic cause - Mitochondrial DNA is implicated for hearing loss as well as increased susecptibilty to environmental damage .

Ototoxicity is considered to be an important cause of hearing loss in ageing which often goes unrecognized. It is estimated that approximately 30% of elderly patients presenting with hearing impairment are taking ototoxic drugs[14.] Aminoglycosides are known to cause death of outer hair cells due to free radicals. Cochleotoxic drugs like neomycin lead to progressive high frequency sensorineural hearing loss associated with tinnitus.

The histological features of the cochlear damage caused from the cumulative effect of noise exposure, ototoxic drugs, or process of ageing is usually indistinguishable.

CAUSE FATORS FOR PRESBYCUSIS [14, 16, 18]

A) Ototoxic drugs implicated in hearing loss in elderly:

- Aminoglycosides
- Quinine
- Beta blockers
- Diuretics
- Non-steroidal anti-inflammatory agents
- Salicylates
- Tricyclic antidepressants

B) Systemic disorders which predispose to the hearing loss in presbycusis patients are:

- Hypertension
- Plasma hyperviscosity
- Atherosclerosis
- Hyperlipidaemia
- Metabolic bone disease
- Diabetes mellitus
- Hypothyroidism

PATHOPHYSIOLOGY IN AGEING [1,14,16]

Ageing can affect any stage from the site of the stimulus to the site of perception, depending on the site of degeneration. The various stages at which ageing is affected are:

Factors affecting the external and middle ear in ageing [19]

According to Toynbee, all these factors are seen in ageing:

- Collapse of the cartilaginous component of external auditory canal
- Disturbances in epithelial migration
- Enlargement of EAM cartilage
- Stiffening of the tympanic membrane
- Degeneration of middle ear muscles
- Arthritic changes & ossification of inter ossicular joints
- Calcification of cartilaginous part of Eustachian tube.

Factors affecting the transduction mechanism of the ear [1,14, 18]

The most important part that is affected in presbycusis is inner ear. Each component affected in the inner ear results in different clinical and audiometeric manifestations. Ageing results in sensorineural hearing loss that is mild to moderate affecting either the mid or high frequencies bilaterally.

In the transduction mechanism conversion of mechanical energy to electrophysiological signal occurs in the cochlea. The vibrations of the basilar membrane of the cochlea are set up in response to vibrations transmitted through oval window. This causes movement of the stereocilia of the hair cells on the basilar

membrane resulting in an increased permeability of the hair cell to potassium and depolarisation of the hair cell resulting in generation of action potential. [18]

Within the cochlea all the hair cells are developed within the first trimester of pregnancy. Once formed during development, they are expected to survive for the entire lifetime of the person. Regeneration does not occur after loss of hair cells. Each region in the cochlea transduces a particular frequency of sound, as a result loss of any of this small population of cells will have a noticeable effect on the person.[18]

Presbycusis changes occurring in the cochlea [(20,21,22)] (both sensory and neural component)

- Sensory – loss of hair cells (Inner and Outer hair cells), sustensacular cells at basal end of organ of corti
- Neural – degeneration of neurons of cochlea, resulting in cochlear ganglionic cell loss
- Vascular – atrophy of stria vascularis in the apical & middle turns of cochlea
- Conductive causes – stiffness of cochlear basilar membrane
- Mixed – combination of the above types

Presbycusis changes in the central auditory system [14]

From the cochlea afferent fibres pass to the cochlear nuclei and from there to the higher auditory centres via the lateral lemniscus and midbrain.

The neuronal signal is converted to sound, which is then analysed by the cognitive centres of the brain to permit understanding of content and association with memory. In humans there is strong bilateral representation of auditory information at all levels above the cochlea.

- Decrease in the number of neurons in the cochlea nuclei and auditory centres of the brain.
- Reduction in the size of cells and changes in the neurochemical make up of the cells.[14]

As a result of all the above mentioned factors there is decline in the ability of the central auditory system to process the sound. Further with ageing there are difficulties in understanding the speech and difficulty is noticed when there is background noise requiring more complex auditory processing.

DIABETES MELLITUS

Diabetes mellitus is defined as a metabolic disorder of multiple aetiology characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism resulting from the defects in the insulin secretion, insulin action, or both.[3]

Diagnostic criteria for classification of diabetes mellitus (WHO)

- Fasting plasma glucose concentration of 7.0 mmol /lt (126 mgdl) and above
- Whole blood glucose level of 6.1 mmol/ lt (110 mg dl) and above
- 2hr plasma glucose concentration more than or equal to 200mg/dl during oral glucose tolerance test.

Studies have shown that there is an increased risk of microvascular disease in those person with fasting plasma glucose concentration of 126 mg/dl and above, with the risk of macrovascular complication also being increased with such fasting concentrations eventhough the 2hr post prandial sugar levels values are <140 mg dl/lt .[3]

Diabetes have been classified into various types. The most widely accepted classification internationally is given by WHO in1985 classification[3]

1) Type 1 Diabetes mellitus (DM)

- Seen in younger age / childhood group with age <30yrs.
- Destruction of Beta cells in pancreas
- Total or partial absence of insulin
- Immune mediated

2) Type 2 Diabetes mellitus (DM)

- Associated with insulin resistance in peripheral cells with or without deficient insulin production from the pancreas
- occurs in later adulthood >30yr
- family history of diabetes, obesity, physical inactivity, hypertension, and hyperlipidaemia are seen.
- one of its variant, if occurs at young age it is called as MODYS(maturity onset diabetes in the young).

3) Gestational diabetes mellitus

- Occurs in the later weeks of pregnancy associated with increased insulin requirements which lead to impaired glucose tolerance.
- revert to normal after delivery

4) OTHER SPECIFIC CAUSES [3]

A. Genetic defects of beta-cell function Chromosome 20, HNF4 (MODY1) Chromosome 7, glucokinase (MODY2) Chromosome 12, HNF1 (MODY3) Chromosome 13, IPF-1 (MODY4)	**B. Genetic defects in insulin action** Type A insulin resistance Leprechaunism Rabson-Mendenhall syndrome
C. Diseases of the exocrine pancreas Fibrocalculous pancreatopathy Pancreatitis Trauma / pancreatectomy Neoplasia Cystic fibrosis Haemochromatosis	**D. Endocrinopathies** Cushing's syndrome Acromegaly Phaeochromocytoma Glucagonoma Somatostatinoma
F. Drug or Chemical-induced Diabetes Glucocorticoids Alpha- & Beta adrenergic agonists Thiazides Dilantin Pentamidine	**G. Genetic Syndromes associated with Diabetes** Down's syndrome Klinefelter's syndrome Myotonic dystrophy Porphyria Turner's syndrome

CHRONIC COMPLICATIONS OF THE DIABETES MELLITUS ARE:

I) MACROVASCULAR COMPLICATIONS

- Coronary artery disease
- Peripheral vascular disease
- Cerebro-vascular disease.

II) MICRO-VASCULAR COMPLICATIONS

- Retinopathy
- Neuropathy
- Nephropathy

Causes for hearing loss in diabetes mellitus [5, 23]

Diabetes Mellitus is known to cause an increased rate of triglyceride production due to insulin resistance and hyperinsulinemia. As a result disorder altering the metabolism of glycerides and lipids are known to accelerate the rate of atherosclerosis.

Imbalance in the glucose and insulin metabolism leads to atherosclerotic vasculopathy of the anterior vestibular artery**.** Impaired nutrient transportation through these thickened capillary walls results in decreased blood flow through the narrowed vessels leading to secondary degeneration of the vestibulocochlear nerve causing neuropathy.

Much of the changes in diabetes are seen in the cochlea mainly the stria vascularis and in the spiral ligament with the reduction in the number of spiral ganglion cells and demyelination of the vestibulocochlear nerve.

HISTOPATHOLOGICAL CHANGES IN DIABETES MELLITUS

Various studies have done to study the histopatholgic changes encountered within the cochlea of the diabetic patients.[6,7,24]

- *Demyelination is the early changes to occur in the peripheral nerves.*
- *The thickening of the capillary basement membrane of the stria vascularis.*
- *Atrophy of the spiral ganglion with cell loss in the basal and middle turn of the cochlea.*
- *Loss of outer hair cells*
- *Loss of inner hair cells*
- *Reduction in the number of nerve fibers of the spiral lamina*
- *Reduction in the number of ganglion cells in the ventral and dorsal cochlear nuclei.*
- *Minor loss of ganglion cells in the superior olivary nucleus, inferior colliculus, and medial geniculate body.*

EFFECTS OF GLUCOSE AND INSULIN METABOLISM IN INNER EAR [5, 23]

- Glucose metabolism plays an important role in maintaining endolymphatic potential. Glucose is an most important factor for maintaining the endolymphatic potential other than glutamate and pyruvate.

- Endolymph is similar to that of intracellular medium in its chemical content, as it is rich in potassium and poor in sodium, whereas perilymph is rich in sodium and poor in potassium, and is similar to the extracellular medium.

- Hypoglycemia or Hyperglyceamia affects the active transportation of sodium and potassium thereby producing an imbalance in the endolymph potential.

- As a result potassium tends to shift from the endolymph to the perilymph, while sodium tends to go the opposite way. This passive spontaneous mechanism would lead to high sodium levels in the endolymph and to more water shifting into this compartment, causing the onset of endolymphatic hydrops - vertigo, tinnitus, hypacusis, and aural fullness in diabetic patients.[12, 16]

DIABETIC NEUROPATHY

It is found that approximately around 50% diabetics are affected by neuropathy during their entire lifetime either in the form of polyneuropathy, mononeuropathy or autonomic neuropathy.[3] The development of this mainly depends on the duration and metabolic control of diabetes.

Diabetes is known to affect both myleinated as well as unmyleinated fibres. It is studied that diabetes is associated with loss of myelin corresponding to the individual schwan cells. Apart from this even the cell body and axon are known to be affected. The various risk factors that might affect the progression of neuropathy in diabetic individuals are age, duration of diabetes, glycaemic control, associated co morbidities like hypertension or hypercholestremia or hyperlipidemia.

Apart from these factors recently it is found that Apo E genotype (E3/4 & E4/4) is known to be associated with accelerated neuropathy which are associated with accelerated atherosclerosis,cell adhesion,impairment in cytoskeltal stabilization [(25,26)].Aldose reductase gene is also associated with accelerated neuropathy in type 2 diabetes mellitus.

The D allele of angiotensin I converting enzyme is also associted with increased risk of peripheral neuropathy in type 2 DM.[25]

There are varioue theories that are implicated in the diabetic neuropathy of which the two most commonly accepted ones are :

A) Vascular Etiology

B) Metabloic abnormality

A) VASCULAR ETIOLOGY [25,26]

- Basement membrane thickening
- Endothelial cell swelling and pericyte degeneartion.
- Increased free radicals
- Nerve hypoxia
- Advanced glycation of vessel wall.
- Occlusive platelet thrombi
- Epineural vessel atherosclerosis
- Reduced endothelial nitrate activity

B) METABOLIC ABNORMALITY [27,28]

- Polyol pathway
- Activation of protein kinase C
- Formation of glycation endproducts and increased oxygen stress

A) Diabetic angiopathy

Much of the changes in cochlea occurs because of the microangiopathy. There is endothelial proliferation & accumulation of glycoproteins, with thickening of the capillary and small vessel of the basement membranes. PAS-positive substance are

seen deposited in the internal auditory artery walls, modiolus vessels, and stria vascularis capillaries[6, 7, 24].

- The thickening of the capillary basement membrane of the stria vascularis occurs as result of proliferative response of the capillary cells to injury.[7,14]
- Thickening occurs due to repeated episodes of endothelial cell death and regeneration. The necrotic endothelial cells are trapped in the basement membrane as new layers of tissue are formed by regeneration. As a result basement membrane and tissue regeneration are increased in diabetes while basement membrane degradation is reduced or impaired. [14]
- Lamellar deposition of PAS on the capillary walls of the stria vascularis are seen to occur ten to twenty times thicker than usual which were consistent with atherosclerosis.[14, 7]

METABOLIC PATHWAY IN DIABETES

a) The polyol pathway

Through the polyol pathway, glucose is converted to sorbitol by aldose reductase, which is the rate-limiting enzyme (Greene et al. 1987).[27] Aldose reductase is found in the nervous system, retina, glomerulus and the blood vessels, i.e. in tissues, which do not require insulin for glucose uptake. Sorbitols and other polyols accumulate intracellularily, leading to osmotic damage and swelling.[5] Aldose reductase inhibitors have improved neuropathy to some extent in diabetic patients, including improvement in nerve fiber density and nerve conduction velocity (Greene et al. 1999).[28]

b) Protein kinase C (PKC) [5, 29]

Hyperglycaemia induces synthesis of diacylglycerol, which can activates PKC, (Lee at al. 1989)[29] An activation of PKC has been implicated in many processes relevant to diabetic complications, including regulation of vascular permeability and flow, increased production of cytokines and increased synthesis of basement membranes.[30]

c) Advanced glycation endproducts (AGE) [5,31]

Hyperglycaemia leads to the formation of glycation products through a non-enzymatic process in which glucose is attached to amino groups of proteins. The early glycation products can by rearrangement form more stable products. Those formed on collagen, DNA and other long lived macromolecules slowly undergo further chemical rearrangements to form AGEs (Brownlee et al.) [31]. The formation of AGEs can contribute to tissue damage in several ways, including release of growth factors, stimulation of synthesis of extracellular matrix and cellular hypertrophy and hyperplasia.

d) Increased oxidative stress Hyperglycaemia also leads to increased oxidative stress leading to peroxidation of lipid membranes, proteins and DNA.[32]

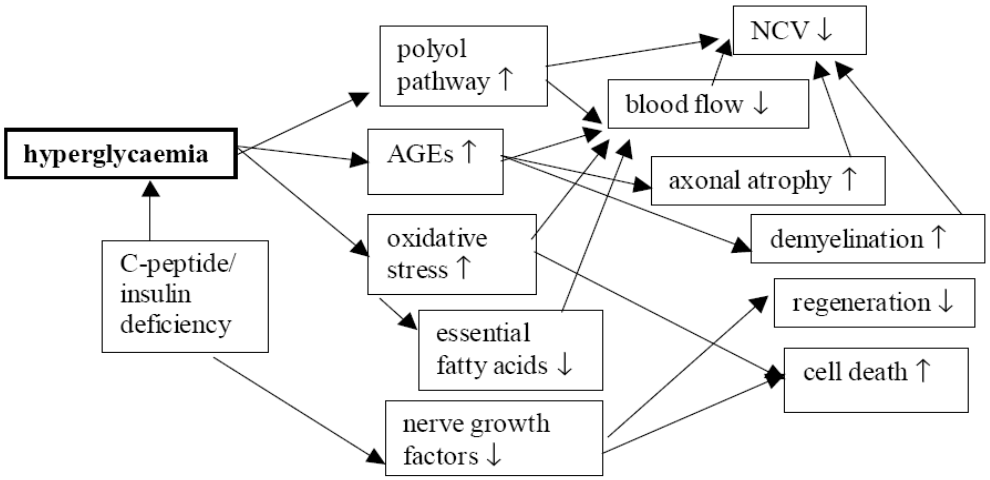


Fig 1:Schematic flow chart showing how hyperglycaemia causes demyleination, regeneration and cell death by generation of various biochemical factors (in courtesy from medicine clinics of North America)

A Microscopic study of temporal bone by Fukushima and Masuda- studied the histopathological changes in the cochlea of type 2 DM patients concluded that there was significant increase in the thickness of the basilar membrane in all turns of cochlea. There was also associated loss of outer hair cells in upper, lower and middle basal turns of cochlea. But no significant changes were seen in the spiral ganglion.[6]

Nageris et al reported in his study that there was no loss of inner or outer hair cells among the diabetic cases. According to him the pathogenesis behind the decrease in the number of OHC are due to microangiopathic changes, there is activation of polyol pathway due to prolonged hyperglyceamia with increased hypoxic changes.[33]

One of the meta-analysis done with 19 case studies concluded that hearing impairment in diabetic patients was 2.1 fold higher than in those without diabetes. Much of these stratified analyses showed a stronger association was observed in younger participants <60 yr compared with studies of older participants. There results had showed an independent association between the hearing impairment and diabetes, although other possible confounding factors like age could not be eliminated. Out of 19 studies it was found that a significant strength of the association was found in all the studies except a study by Aladag who showed to have negative association between diabetes and hearing impairment risk.[4]

A cross-sectional analysis of 5140 non-institutionalized adults aged 20 to 69 years in the nutrition examination survey conducted from 1999 to 2004 by Kathleene. Bainbridge revealed that hearing impairment was more prevalent among adults with diabetes when compared with adults without diabetes. They also concluded that the association between diabetes and hearing impairment was independent of known risk factors for hearing impairment, such as noise exposure, ototoxic medication use, and smoking.[11]

Age is an important variable that plays a role in hearing loss. Diabetes acts an an independent confounding factor and acts synergistically with presbycusis to cause increased hearing loss. Much of the literature in the past have reported that with advancing age the hearing threshold also increases. Axelsson reported in his study that the incidence of pure tone hearing loss increased with age in patients with diabetes, even after correction for presbycusis [10].

Studies have been done to understand the association between duration of diabetes and the hearing loss, but none of the studies have given clear conclusion. Celik et al in his study had observed that as the duration of diabetes increased to 15 years, the incidence of hearing loss also increased. But after 15 years the influence of diabetes on hearing loss was not significant.[34]

Axelson also observed that age-matched patients with diabetes treated with insulin had better hearing that those patients treated with oral medications[10].Wackym and Linthicum had also observed similar results that diabetic patients treated with diet alone had more severe hearing loss than those taking oral hypoglycemic agents, who had worse hearing than those who were on insulin.[7]

A study reported by Asma and Asmi, stated that glycaemic control does not have significant impact on hearing. The hearing threshold was neither affected by insulin treatment nor by the glycaemic control.[35]

Ahmad HA Salahaluddin et al in a cross-sectional study of 836 patients found the prevalence of hearing impairment was higher among men than in women and the associated risk factors for hearing loss were higher in men such as Diabetes Mellitus, Hypertension, retinopathy, nephropathy, neuropathy than in women. There was a statistically significant relationship between both the genders in all the risk factors which is suggestive of Diabetes Mellitus & Hypertension as a contributory factor for hearing loss. [36]

Clícia adriana S, Maia et al in a literature review of 16 various studies concluded that in view of current knowledge, there is evidence that Diabetes Mellitus may cause hearing loss, but without a clear cause-effect correlation. It was also found that a series of variants may favour the association between both diseases, Diabetes Mellitus and hearing loss can be dependent components, or even components of a genetic syndrome and not dependent one on the other.[37]

Kakarlapudi V, et al in a retrospective database review of electronic medical record from 1989 to 2003 in a tertiary referral center reviewed data of 53461 non diabetic age-matched patients and 12575 Diabetic patients and found that sensorineural hearing loss was more common in the Diabetic patients than in age matched non diabetic patients from the same institutions. Poor control of Diabetes, correlated with worsening hearing in patients with diabetes who had sensorineural hearing loss, the studies made them conclude sensorineural hearing loss was more common in patients with diabetes than in the control nondiabetic patients, and severity of hearing loss seemed to correlate with progression of disease as reflected in serum creatinine and it was postulated sensorineural hearing loss may have been due to microangiopathic disease in the inner ear.[12]

Dayna S Dalton et al, in a data from population-based longitudinal study of aging of 344 diabetics of a total of 3571 participants found out that Diabetics were more likely to have a hearing loss than were subjects without diabetes however there was no association between duration of Diabetes or glycaemic control and hearing loss. Individuals with NIDDM and Nephropathy were more likely to have a hearing loss than were those with NIDDM but no nephropathy. [38]

Helzner EP et al in Cross-sectional analysis of a longitudinal cohort study of 2,052 older adults enrolled in the Health, Aging and Body Composition found hearing loss was most common in men, followed women, Diabetes Mellitus were associated with hearing loss after multivariable adjustment and concluded Hearing loss was extremely common in elderly. Because many of the identified hearing loss risk factors are modifiable, some of the burden associated with hearing loss in older people should be preventable [39].

Diniz and Guida showed a significantly higher audiometric thresholds among patients with diabetes mellitus when compared to non diabetics. A prevalence of 62% of hearing loss was seen compared to 30% hearing loss between controls [40].

In a study done by Subesi and Haziran which compared the relation between hearing loss and the retinopathy complication associated with DM This study reported that there was a significant hearing loss involving the low, mid and high frequencies in both the groups (only DM & DM with retinopathy). But significant difference between both the groups were seen mainly at low frequency [41].

Yiling J. Cheng, Catherine C. Cowie, examined potential mediators of the reported association between Diabetes and hearing impairment amongst 1,508 participants, aged 40–69 years, who completed audiometric testing during 1999–2004 in the National Health and Nutrition Examination Survey. Using logistic regression, Diabetes was associated with a 100% increased hearing impairment and a increased odds of high-frequency hearing impairment in preliminary models after controlling for age, sex, race/ethnicity, education, smoking, and occupational noise exposure, they also postulated that there is no evidence suggesting that their observed relationship between diabetes and hearing impairment is due to hypertension or dyslipidemia.[42]

ANATOMY AND PHYSIOLOGY OF HEARING

EXTERNAL EAR

The external ear consists of the pinna and the external auditory canal from the meatus to the tympanic membrane. Pinna is composed of a single layer of yellow elastic cartilage and has no useful muscles. External auditory meatus, measures about 2.5 cm long. The medial 2/3rd is bony and lateral third of the canal is the cartilaginous portion. Cartilaginous portion consists of cerumen-producing glands and hair follicle. The concha has a resonance of about 5 kHz, and the irregular surface of the pinna introduces other resonances and anti-resonances. The external auditory canal (EAC) is essentially a tube that is open at one end and closed at the other and behaves like a quarter-wave resonator. The resonant frequency is determined by the length of the tube. For a tube of 2.5 cm, the resonant frequency is approximately 3.5 kHz

The acoustic properties of the external ear is most prominent at 4-kHz. In localization of sound sources, the head acts as an attenuator at frequencies at which the width of the head is greater than the wavelength of the sound. Thus at frequencies greater than 2 kHz, a head shadow effect occurs, in which inter-aural intensity differences of 5 to 15 dB are used to localize sound sources. At lower frequencies, where the wavelength of the sound is larger than the width of the head, little attenuation is provided by the head. Inter-aural time differences (~0.6 ms for sound to travel across the head) are the salient cues for localization. The resonating frequency in infants is approximately 8 kHz in infants and decreases to adult values after approximately 2.5 years of age.

MIDDLE EAR

The middle ear functions as an impedance-matching device as it couples the low impedance of air to the high impedance of the fluid-filled cochlea.

Middle ear transformer mechanism:

The transformer system of the middle ear, although working as a complex whole, may be divided into 3 stages; that provided by the ear drum (catenary lever), that provided by the ossicles (ossicular lever), and that provided by the difference in area between the tympanic membrane and the stapes foot-plate (hydraulic lever)

a) Catenary lever:

Helmholtz, in 1863, was first to propose the concept of a catenary lever to the action of the tympanic membrane. The familiar example of this type of lever is the tennis net. Studies by Tonndorf and Khanna using sensitive method of time average holography were able to determine that the vibratory patterns of Tympanic membrane were similar to that hypothesized by Helmholtz.

It is estimated that even though the curvature of the TM is variable, the catenary lever provides atleast a two times (2x) gain in sound pressure at the malleus.

a) Ossicular lever:

Dahmann, proposed that the malleus and incus acts as a unit, rotating around an axis running between the anterior malleolar ligament and the incudal ligament. This concept generally accepted today, measures the lever arms from the rotational axis to the tip of the malleus and to the tip of long process of the incus. This lever ratio is 1.15 **:** 1

b) Hydraulic lever:

Helmholtz's third concept of impedance matching involved what is today commonly referred to as the areal ratio. Briefly stated, sound pressure collected over the large area of the tympanic membrane and is transmitted to a smaller foot plate area, results in an increase in force proportional to the ratio of the areas.

This idea is valid as long as both areas are vibrating in a piston like manner. This ratio average is 20.8 **:** 1 .

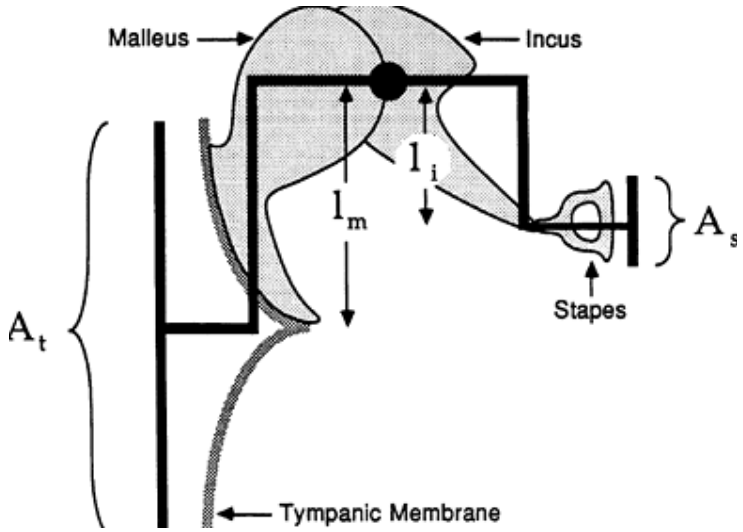


Fig 2 - Schematic diagram of ossicular chain and tympanic membrane showing the differences in area and vibratory pattern of the ossicles

SOUND PRESSURE TRANSFORMER :

Catenary lever: force acting on TM/force acting on malleus	2.0
Ossicular lever: force acting on malleus/force acting on stapes	1.15
Areal ratio: Area of tympanic membrane/area of footplate	21.0
Total lever advantage	48.3 (34 dB)
External ear contribution	15 dB
Total system gain	49 dB

Distortion of sound signals does not occur in the middle ear, for the sound levels greater than 130 dB sound pressure level (SPL).The middle ear, including the tympanic membrane, ossicular chain with supporting ligaments, and middle ear space, can be viewed as a passive mechanical system with both mass and compliant elements and therefore resonant properties. The ratio of the volume velocity of the stapes to sound pressure at the tympanic membrane increases in humans to approximately 900 to 1000 Hz, which is the resonant frequency of the middle ear, and decreases at higher frequencies. Phase shift or time lag between movement of the tympanic membrane and the stapes generally increases with frequency.

Less than half of the power entering the middle ear actually reaches the cochlea as shown in fig 3, because of the absorption of energy by the ligaments and middle ear.

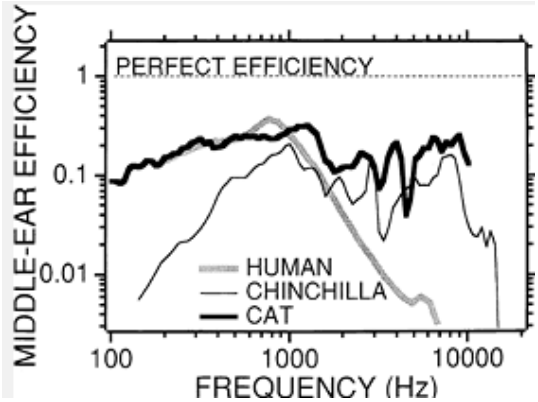


Fig 3- Efficiency of the transfer of power through the middle ear. For all species shown, less than half the power that enters the middle ear actually reaches the cochlea. Energy loss is caused by absorption by the tympanic membrane, ossicular ligaments, and middle ear

Auditory function is profoundly affected by cochlear impedance as well as the combined acoustic effects of the head, external ear, and middle ear. Humans cannot detect and recognize sounds greater than approximately 20 kHz because such high-frequency sounds are not transmitted efficiently through the middle ear to the cochlea. Whereas low-frequency energy is not transmitted to the cochlea, and the frequency region of greatest energy concentration is 3 to 4 kHz. Thus, these acoustic properties are primarily responsible for the ability of intense low-frequency sounds to produce high-frequency hearing losses and injuries in the basal region of the cochlea.

Two striated muscles, the tensor tympani and the stapedius, are located in the middle ear. The former attaches to the malleus and is innervated by the trigeminal nerve. The stapedius muscle attaches to the stapes and is innervated by the stapedial branch of the facial nerve. One function of the middle ear muscles is to protect the cochlea from loud sounds. When sounds louder than approximately 80 dB SPL are presented monaurally or binaurally, consensual reflex contraction of the stapedius muscle occurs. This contraction increases the stiffness of the ossicular chain and

tympanic membrane, attenuating sounds less than approximately 2 kHz. Although the tensor tympani contracts as part of a startle response, acoustic reflex data from human subjects with neurologic involvement of cranial nerves V and VII suggest that the tensor tympani does not normally respond to intense acoustic stimulation. Studies have shown that the stapedial reflex protects the cochlea, particularly from low-frequency (<2 kHz) sounds in excess of 90 dB. In any latency of the acoustic reflex greater than 10 ms, the cochlea may be unprotected from short-duration, unanticipated impulsive sounds.

COCHLEA

The inner ear is an intricately shaped membranous tube suspended within a bony tube – the labyrinth. The inner ear has two functions.

1. The transduction of sound pressure into neurochemical impulses in the auditory (eighth cranial) nerve takes place in the cochlea.
2. To maintain optic fixation in the presence of movement and to help to maintain balance occurs in the vestibular system.

Cochlea comprises of 2 3/4 turns of coil and is snail shaped .It measures about 35mm in length. The base of it protrudes into the middle ear as the promontory of the medial wall. The bony wall of the cochlea has two defects, each covered by a thin membrane. These are the round window and the oval window. The footplate of the stapes, is placed over the oval window and is held in place by the annular ligament.

A cross-section of one turn of the cochlea is divided into three segments as shown in fig 4, which include scala vestibuli, the scala media and the scala tympani. The

scala media contains endolymph and the other two contain perilymph. There is communication between the perilymph of the scala vestibuli and the scala tympani at the apex of the cochlea known as the helicotrema.

The scala media is a closed, triangular cavity bounded above by Reissner's membrane and below by the basilar membrane as seen in fig 4. The stria vascularis forms the base of the triangle lying against the bony wall of the cochlea. The organ of Corti sits on the basilar membrane and it is here that the transduction of sound happens. Scala Media contains endolymph, similar to intracellular fluid with a potassium concentration of 144 mEq/L and a sodium concentration of 13 mEq/L. The scala media has a positive direct current (DC) resting potential of approximately 80 mV that decreases slightly from base to apex. This endocochlear potential is produced by the heavily vascularized stria vascularis of the lateral wall of the cochlea. The scalae vestibuli and tympani contain perilymph, an extracellular fluid-like material with a potassium concentration of 4 mEq/L and a sodium concentration of 139 mEq/L. The sodium-potassium-adenosine tri phosphatase (Na^+-K^+-ATPase) .The organ of Corti sits on the basilar membrane and it is here that the transduction of sound happens .The basilar membrane is approximately 0.12 mm wide at the base and increases to approximately 0.5 mm at the apex.

THE ORGAN OF CORTI

The basilar membrane runs between the inner and outer bony spiral laminae. It is narrower and more taut at the base of the cochlea and wider and floppier at its apical end. At its inner end sits the organ of Corti. This comprises the limbus, the tectorial membrane, the inner and outer rods (or pillars) of Corti with the tunnel of Corti between them, one row of inner hair cells, three rows of outer hair cells and supporting cells of Claudius, Deiter and Hensen as seen in figure 4. Supporting cells provide structural and metabolic support for the organ of Corti. The phalangeal processes of the Deiters cells form tight cell junctions of the reticular lamina.

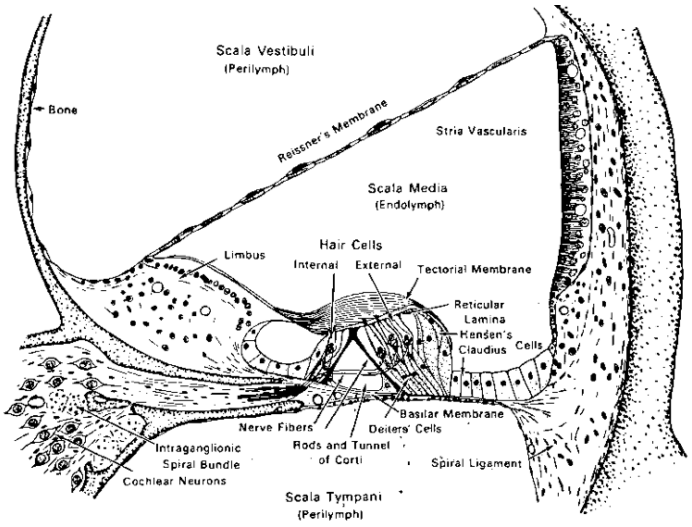


Fig 4 – schematic diagram of organ of corti with basilar membrane (courtesy scott brown)

There are approximately 12 000 outer hair cells and 3500 inner hair cells in humans. The auditory branch of the eighth cranial nerve (the cochlear-vestibular nerve) contains fibres that run from the cochlea to the brain stem – afferent fibres – and efferent fibres that run in the opposite direction. whereas the dendrite projects through the osseous spiral lamina.

Around 90% of the afferent fibres come from the inner hair cells. Each fibre comes from only one cell but each cell may have up to 10 fibres(type 1 neuron). The remaining 10% of the afferent fibres and all of the efferent ones are associated with the outer hair cells. Each of the nerves (type 2neuron) associated with the outer hair cells they branch and is connected with many cells. In addition to the afferent innervation pattern of the cochlea, approximately 1,800 efferent fibers, originating from the ipsilateral and contralateral superior olivary complex, project to the cochlea.

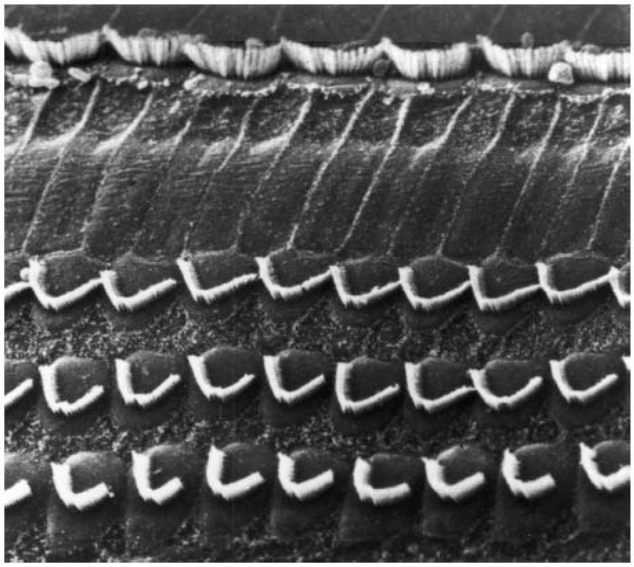


Fig 5 – Schematic diagram showing arrangement of inner and outer hair cells (courtesy from scott brown)

Each hair cell has many hairs (cilia). The cilia of each outer hair cell are arranged in a ‘W’ shape (with a very shallow central notch) and those of the inner hair cells in a gentle curve. The hairs of the outer hair cells are embedded into the tectorial membrane whereas at rest the hairs of the inner hair cells are not. The cilia of each hair cell are connected by tip links. The outer hair cells contain contractile actin and myosin fibres which allow for the cells to alter their length.

THE STRIA VASCULARIS

This is metabolically very active structure with a rich blood supply. It is responsible for the maintenance of the chemical and electrical composition of the endolymphatic space. It is a convoluted structure with many folds and indentations to increase the surface area. There are three types of cells arranged in three layers. The inner basal cells are arranged in one continuous layer. These cells may be of neural crest or possibly mesodermal in origin. The outer layer lines the lumen of the cochlear duct and consists of epithelial cells. Between these two layers are intermediate cells. These are a type of migratory melanocyte. It is likely that these cells are responsible for the endocochlear potential and high potassium content of endolymph that are necessary for transduction of sound.The intermediate and basal cells are joined by gap junctions. Gap junction proteins such as Connexin probably also have a role in maintaining the high potassium concentration within the endolymph.

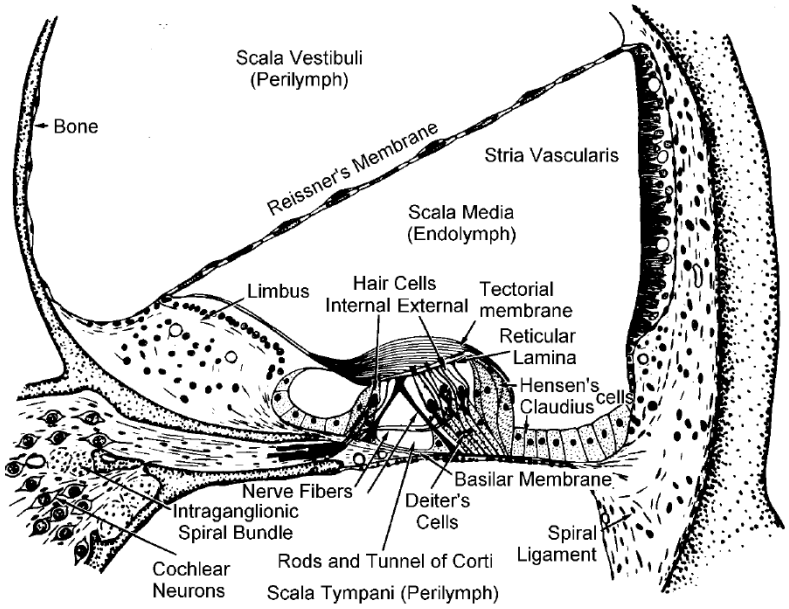


Fig 6 : Schematic diagram showing mid modiolar view of cochlear duct

TRANSDUCTION OF SOUND -

When the stapes footplate moves in response to incoming sound pressure waves the vibrations are transmitted into the scala vestibuli. This is possible because of the helicotrema and the round window. As the stapes footplate moves in, the round window membrane moves out the pressure waves make the organ of Corti move. Because of the hinge action of the limbus, the basilar membrane and the tectorial membrane do not move exactly in unison. This means that the cilia of the outer hair cells that are embedded in the tectorial membrane are displaced laterally. The tip links mean that more of the cilia are deflected. As the cilia move, potassium and calcium channels on the upper surface of the hair cells are exposed. This allows for equalisation of the chemical and electrical differences between the endolymph and the hair cell. It is this depolarisation that triggers the action potential in the cochlear-vestibular nerve. The cells have other ion exchange pumps in their walls that restore the original balance by removing the potassium (repolarisation).

The stereocilia - hair cell complex is critical to transduction. Stereocilia are bundles of actin filaments that form tubes and are inserted into the cuticular plate. They also are cross-linked between themselves. Stereocilia of inner hair cells probably do not contact the tectorial membrane, but those of outer hair cells are in direct contact. Deflection of the stereocilia by the traveling wave opens and closes nonspecific ion channels at the tips of the stereocilia, resulting in current flow (potassium) into the sensory cell. The flow of potassium ions into the sensory cell is modulated by the opening and closing of ion channels of the stereocilia. The potassium flux is caused by the endocochlear potential of +80 mV added to the negative intracellular potentials of hair cells. The resulting intracellular depolarization causes an enzyme cascade involving calcium.

This ultimately leads to the release of chemical transmitters, and the subsequent activation of the afferent nerve fibers.

The displacement pattern of the basilar membrane is a traveling wave. The basilar membrane is stiffer at the base than in the apex. The stiffness component is distributed continuously. Therefore, the traveling wave always progresses from base to apex. The maximal amplitude of basilar membrane displacement varies as a function of stimulus frequency. Traveling waves produced by high-frequency sounds (10 kHz) have maximal displacement near the base of the cochlea, whereas the waves to low-frequency sounds (125 Hz) have the maximum toward the apical region. Traveling waves generated by high-frequency sounds do not reach the apical region of the cochlea, whereas waves to low-frequency sounds can travel the entire length of the basilar membrane.

The mechanism by which the sharply tuned peak is generated within the mechanical traveling wave involves an enhancement known as the cochlear modifier. This is an activity of the outer hair cells that enhances the motion of the basilar membrane at frequencies near the best frequency of the particular cochlear location. This enhancement contributes to the fine frequency-selective abilities of the ear and to the sensitivity of the ear and ability to detect extremely faint sounds. The notion of an active process in the cochlea, the cochlear amplifier, is supported by the phenomenon of otoacoustic emissions. That is, when a short-duration signal is presented to the ear, an echo emanating from the cochlea can be recorded in the external auditory meatus. Because the energy of the echo can be greater than the energy of the short-duration signal, an active process, the cochlear amplifier, is assumed. Factors that may contribute to the cochlear amplifier include motility of outer hair cells and the mechanical properties of the stereocilia and tectorial membrane.

INTENSITY RESOLUTION

It is the afferent nerve fibres from the inner hair cells that carry the information about hearing to the brain.The outer hair cells act as controlling and finetuning mechanisms.

When the organ of Corti moves in response to sounds of low intensity the cilia of the inner hair cells do not contact the tectorial membrane. The brain receives impulses from the afferent fibres of the outer hair cells and is, therefore, aware that there is a sound. However, there is no perception of sound as there are no impulses from the inner hair cells. Signals are sent along the efferent nerve fibres to the outer hair cells, causing them to shorten in length.This pulls the tectorial membrane closer to the basilar membrane, allowing the cilia of the inner hair cells to impact the tectorial membrane. Deflection of the cilia leads to depolarisation and action potentials from the inner hair cells and perception of sound.

High-intensity sounds lead to more movement of the organ of Corti. In order to protect the cilia, efferent impulses cause the outer hair cells to lengthen and push the tectorial membrane away from the basilar membrane.

A protein named prestin has been identified in outer hair cells and is considered to be the motor protein of outer hair cells and the driving force of electromotility of hair cells. Another point of view focuses on rapidly acting potassium and calcium ion channels presumed to be the basis of the cochlear amplifier and its regulation. A third approach suggests that a collection of motor proteins within a hair cell can generate oscillations that depend on the elastic properties of the cell.

The principal neurotransmitter substance of cochlear efferent fibers is acetylcholine. Acetylcholine acts on receptors to produce hyperpolarization of the cell membrane and doubling of the input conductance of the cell. The acetylcholine receptor

has both muscarinic and nicotinic features. In addition to acetylcholine, I-aminobutyric acid and several neuroactive peptides are neurotransmitters for the efferent system.

Four gross (extracellular) potentials can be recorded in the cochlea, endolymphatic (endocochlear) potential, cochlear microphonic, summating potential and whole-nerve action potential. Unlike the other cochlear potentials, the endolymphatic potential is not generated in response to acoustic stimulation. The stria vascularis is considered to be the energy source, of the cochlea, crucial for transduction. The nature of the energy source is related to the heavy vasculature of the stria vascularis and to the Na^+-K^+-adenosine triphosphatase. This pump has been localized to several types of cochlear cells, including marginal cells of the stria vascularis, outer sulcus cells, and fibrocytes near the attachment of the Reissner membrane and in the spiral ligament. Whereas Na^+-K^+-ATPase must play a significant role in ion transport in the cochlea.

Applied anatomy - Malfunctioning of the mechanisms involved in production of endolymph and the endolymphatic potential can produce hearing loss, sometimes called metabolic presbycusis. When the flow of endolymph through the ductus reuniens is blocked, endolymphatic pressure increases, and hydrops occurs.

COCHLEAR MICROPHONICS

The cochlear microphonic is an alternating current (AC) voltage usually recorded within the cochlea or near the round window. It represents the potassium ion current flow through mainly the outer hair cells; that is, the electrical resistance of outer hair cells is altered by the motion of the basilar membrane. When stereocilia are bent away from the modiolus, the resistance of the hair cells decreases. The result is an increase in current flow and a small decrease in endolymphatic potential. When stereocilia are bent toward

the modiolus, resistance increases and current flow decreases with an accompanying increase in the endolymphatic potential.

The summating potential is a DC potential recorded in the cochlea in response to sound. It follows the envelope of the stimulating sound. Recordings of this DC potential can be made in the scala tympani, media, or vestibuli and in some circumstances from a gross electrode in the human ear canal. The potential can be positive or negative, and it can reverse polarity, depending on electrode location or stimulus frequency and level. The summating potential probably has several origins, but it largely reflects the DC shifts caused by stimulus-driven intracellular potentials of outer hair cells. Inner hair cells contribute to these to a lesser extent.

Eighth Nerve Physiology

The auditory nerve has approximately 30,000 fibers in humans. Approximately 90% to 95% of neurons (type I, radial fibers) innervate inner hair cells, whereas 5% to 10% (type II, outer spiral fibers) innervate to the outer hair cells. The type 1 neurons have radial fibers with bipolar cell bodies in the spiral ganglion. Outer spiral fibers are monopolar and unmyelinated. Most auditory nerve fibers in mammals discharge in the absence of acoustic stimulation.

The nerve fibers have been classified into three categories on the basis of rate of spontaneous discharge high (18 to 120 spikes per second), medium (0.5 to 18 spikes per second), and low (0 to 0.5 spikes per second). Fibers with high rates of spontaneous activity respond to auditory signals at lower levels than do fibers with medium or low rates of spontaneous activity. In other words, the most-sensitive fibers have the most-spontaneous activity. Fibers with high spontaneous rates have thick dendrites that tend to terminate on the side of inner hair cells facing outer hair cells. Fibers with low and medium spontaneous rates have thin dendrites that terminate on the side of the inner hair

cell facing the modiolus. Spontaneous activity of nerve fibers is not random but is proving to be anatomically and functionally significant. The tuning curve of a single auditory nerve fiber is perhaps the most basic measure of auditory nerve function Tone bursts covering a wide range of frequencies are used, and the lowest level of signal is recorded for a given frequency that produces a specific rate of discharge. The resulting isoresponse curve is called a tuning curve.

Although thresholds of auditory nerve fibers are related to the rate of spontaneous discharge, most afferent nerve fibers (60%) have high spontaneous rates and thresholds within 20 dB greater than the thresholds for the animal. The remaining low-spontaneous fibers have thresholds that cover approximately 60 dB. The dynamic range of most auditory nerve fibers is approximately 30 dB from threshold to saturation, although some low-spontaneous fibers have a much wider dynamic range.

One of the most common features of sensorineural hearing loss is recruitment of loudness. It is assumed that loudness depends on the total activity of the auditory nerve. The number of fibers activated increases slowly as intensity is increased, and only the tips of tuning curves are activated. As the intensity increases further, the tails of the tuning curves are encountered, and the number of fibers activated increases rapidly. In the case of sensorineural hearing loss, the tips of the tuning curves are missing, and the fibers are not activated until the level of the signal is sufficient to reach the tails of the tuning curves. Abruptly, many fibers then are abruptly activated simultaneously.

AUDITORY CENTRAL NERVOUS SYSTEM

The ascending and descending auditory pathways are described in the schematics representation.

Eighth-nerve afferent fibers carried till it reaches at the level of the cochlear nucleus. Five major cell types are found within the cochlear nucleus, each with distinct cell morphologic and physiologic features, such as response to stimulus onset, and frequency modulation. From the cochlear nucleus, most fibers cross the brainstem to the contralateral superior olivary complex; a much smaller number of fibers run to the ipsilateral superior olivary complex.

The superior olivary complex

This is considered the first center in the ascending auditory system, where inputs from both ears converge. Auditory nuclei above the superior olivary complex can be excitatory or inhibitory with inputs from each ear. Stimulation of the contralateral ear typically is excitatory to cell bodies of the auditory CNS, whereas stimulation of the ipsilateral ear is inhibitory.

The medial and lateral superior olivary complex

The medial SOC is the origin of the crossed efferent fibers that terminate on outer hair cells, whereas the lateral superior olivary complex is the origin for the uncrossed efferent fibers that terminate on inner hair cells. Although many functions have been attributed to the efferent auditory system, especially protecting the cochlea from loud sounds.

The inferior colliculus nucleus

It is a complex nucleus with at least 18 major cell types and at least five areas of specialization. It is involved in probably all forms of auditory behavior, including differential sensitivity for frequency and intensity, loudness, and binaural hearing. The inferior colliculus is clearly more than a relay center.

The medial geniculate body

This sends projections to the auditory cortex, but its specific functions are unknown. The auditory cortex is located in the sylvian fissure of the temporal lobe; many secondary auditory areas are clustered around the primary area.

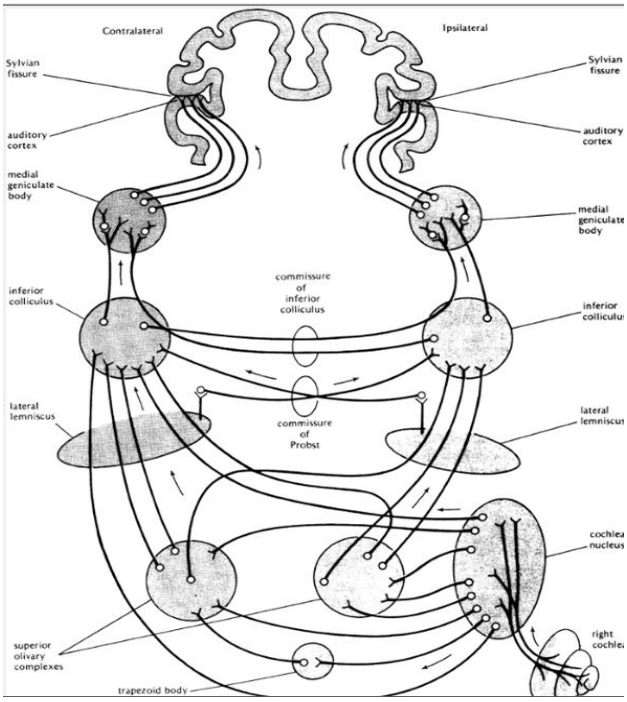


Fig 7 : Schematic diagram of the ascending (afferent) pathways of the central auditory system from the right cochlea to the auditory cortex

In each area, the cells are tonotopically organized in a columnar manner, each column having a special attribute. The cells in one column can have different tuning at a similar characteristic frequency, whereas another column can be associated with intensity encoding, another with providing inhibitory responses to stimulation of one ear and excitatory responses of the other ear, and so on.

Bilateral lesions of the temporal lobe have been shown to produce wide-ranging effects (cortical deafness, in which several auditory behaviors are severely affected, including speech discrimination, localization of sound, temporal processing of information, and the detection of faint, short-duration signals). Another important feature of the auditory system is its tonotopic nature. From the basilar membrane to the auditory cortex, the system is organized spatially with respect to frequency. Each place on the basilar membrane responds best to a specific frequency i.e., high-frequency sounds are localized to the base, and low-frequency sounds, to the apex. The tonotopic organization of the cochlea is preserved at the cochlear nucleus.

APPRECIATION OF PITCH OF THE SOUND THEORIES OF HEARING

The two qualities of sound are: (1) The pitch and (2) The loudness or intensity. The pitch of the sound depends upon the frequency of sound waves, and the loudness depends upon the amplitude of sound waves.

The frequency of sound audible to human ear lies between 20 & 20000 Hertz or cycles/second. The range of greatest sensitivity lies between 2000 and 3000 Hz.

Theories of Hearing are generally classified into two groups. According to the first group, the analysis of sound frequency is the function of cerebral cortex and the cochlea merely transmits the sound.

According to the second group of theories, the frequency analysis is done by cochlea, which later sends the information to cerebral cortex.

THEORIES OF FIRST GROUP

(1) Telephone theory of Rutherford

This was postulated by Rutherford in 1880. This is also called frequency theory. According to this theory, the cochlea plays a simple role of a telephone transmitter like converting the sound waves into electrical impulses of same frequency. The impulses are sent to cortex, where perception and analysis of sound occur. It is believed that, the nerve fibers can transmit only to the maximum of 1000 impulses per second. Thus, the telephonic theory fails to explain the transmission of sound with frequency above 1000/sec.

(2) Volley theory

In 1949, Wever postulated this theory. According to this theory, the impulses of sound waves with frequency above 1000 cycles per second were transmitted by different groups of nerve fibers. Volley means groups, however, this theory has no evidence to prove it.

THEORIES OF SECOND GROUP

(1) The Resonance theory of Helmholtz.

This was the first theory of hearing to emerge in 1863. According to Helmholtz, analysis of sound frequency is the function of cochlea. The basilar membrane contains many basilar fibers, which he called the resonators and compared them with the resonator piano. When the sound with a particular frequency is applied, the basilar fibers in a particular portion of basilar membrane are stimulated.

The resonance theory was not accepted because the individual resonators could not be identified in cochlea.

(2) Place theory

According to this theory, the nerve fibres from different portions of basilar membrane of organ of corti, give response to sounds of different frequency.

Accordingly ,the corresponding nerve fibres from organ of corti give information to the brain regarding the portion of organ of corti that is stimulated. It is more widely accepted theory.

(3) Travelling wave theory:

From place theory, emerged yet another theory. This is called ‘travelling wave theory this theory explains how the travelling wave is generated in the basilar membrane.

METHODOLOGY

STUDY DESIGN

Cross sectional study

STUDY POPULATION

Study population includes patients aged 50 years and above with Age related hearing Loss and patients with diabetes mellitus attending Endocrinology department/ diabetology and Otorhinolaryngology outpatient department at R.L.Jalappa Hospital Kolar, Tamaka.

SAMPLE PERIOD

December 2011 to October 2013

CRITERIA FOR SAMPLE SELECTION

A. Inclusion Criteria:

1. Patients aged 50 yrs and above presenting to diabetology clinic and ENT outpatient department with age related hearing Loss among Diabetic patients with/without complications with no previous h/o ear diseases
2. Non diabetic control group age and sex matched healthy subjects with no previous h/o ear disease.

B. Exclusion Criteria:

1. Patients less than 50 years old.
2. Patients with any middle ear pathology

3. Patient with sensorineural hearing loss with a known cause like congenital hearing loss, Meniere‘s disease, Labyrinthitis, Temporal bone fracture, syphilis, meningitis.
4. History of known ototoxic drug intake.
5. History of systemic diseases like hypertension, cardiac diseases and renal failure.

METHODS OF COLLECTION*:*

The study group were classified into 2 groups.

Group A - Patients with Type2 DM aged above 50years.

Group B – control group who were age and sex matched Non diabetic cases without any co morbidity.

Among Diabetes patients, were further classified based on treatment with insulin(I) / oral hypoglycemic drugs (O) /no treatment and associated complications like neuropathy(C), retinopathy(A) and nephropathy(B).All the patients were subjected to questionnaires regarding- medical history, smoking and alcohol history and other associated co morbid disease, past h/o ototoxic drug intake, previous ear pathology, previously undergoing surgeries, and family h/o hearing loss. Patients were evaluated with the following modalities.

1. General physical examination.
2. Complete examination of the ear, nose, and throat
3. Otoscopic examination including siegalisation.
4. Assessment of hearing with tuning forks.

PURE TONE AUDIOMETRY TEST

Pure tone audiometry is the most routine audiometric evaluation to measure the degree of hearing loss. It was performed using a pure tone audiometer in a sound proof room in ENT department. Ear phones were used to test hearing by air conduction and a small vibrator placed over the mastoid to test hearing by bone conduction. The signals presented to subject by an audiometer were characterized by its frequency, sound pressure level and wave form which are controlled. Patient was clearly explained about the procedure and the purpose of the test and consent was taken.

Pure Tone Audiometry: Air conduction threshold

The test was based on measurement of hearing threshold for a range of pure tones presented through earphones according to the ascending method – HUGHSON WESTLAKE METHOD, Up 5, Down 10 method.

Air conduction threshold were measured for tones of 250Hz, 500 Hz, 1000 Hz, 2000 Hz, 4000 Hz and 8000Hz. Patient were instructed to hear tones of short duration either in right / left ear. To start with tones might be very faint. He/she were instructed to raise his/her arm corresponding to side of ear and keep it raise as long as the sound was heard.

THRESHOLD DETERMINATION A clearly audible 40dB was presented to the subject at 1000Hz, if there was difficulty in hearing then it was again raised to 60dB. The level of the tone was then reduced until tone became inaudible by every 10dB. The tone was then raised by every 5dB till the response was obtained. The level at which the subjects gave the response after raising 5dB was the considered as

THRESHOLD for that particular frequency. The test was continued till the next higher frequency 8000Hz. Again returned to 1000Hz and then tested at 500Hz and 250Hz

Pure Tone Audiometry: Bone Conduction Threshold

This was calculated by placing a vibrator on the mastoid process. The measurement was performed similar to that of air conduction method but till 4000Hz. THRESHOLD DETERMINATION- The bone vibrator was placed over the mastoid process to which side Webers had laterlized at 1000hz.

Continuously producing clear tones was given until loudest sound was heard. If the test tone or the response was not obtained then tones were increased every 10dB till sound was heard. Then the level was reduced in steps of 20dB until tone became inaudible. The level was then increased in steps of 5dB. Masking was given wherever necessary.

The severity of Hearing loss was graded (as per WHO classification)

A	NORMAL	0-25dB
B	MILD	26-40dB
C	MODERATE	41-55dB
D	MODERATELY SEVERE	56-70dB
E	SEVERE	71-90dB

OTHER INVESTIGATIONS DONE :

- Fasting blood sugar level and Post prandial blood sugar. The patients were analysed according to FBS and PPBS values.

  FBS <110mg/dl and FBS > 110mg/dl

  PPBS - <140mg/dl and PPBS >140mg/dl

- HbA1c

  HbA1c - <7% - good glyceamic control

  HbA1c - 7%-8%- Fair glycaemic control

  HbA1c - >8% poor glycaemic control

- Serum creatinine levels

  <1.5mg/dl and > 1.5mg/dl

Fundus examination was done in diabetic patients.

OBSERVATIONS

A total of 196 patients aged more than 50yrs with and without diabetes mellitus attending the Otorhinolaryngology and Diabetology clinic in R.L.Jalappa Hospital were studied for the sensory neural hearing loss during the period of November 2011 to September 2013 as a cross sectional study.

There study groups were divided into:

- Group A – Cases (Diabetes patients aged > 50yrs)
- Group B – Controls (Non Diabetic patients aged >50yrs without any other co-morbidities)

The total number of the patients studied were196. Among them, Group A were 106 patients and Group B were 90 patients.

The various parameters that were studied in the present study are:

1. The mean value of pure tone averages was taken for each group as mentioned below:

a) Group 1- 50-60yrs

b) Group 2- 61-70yrs

a) Group 3- 71-80yrs

b) Group 4- 81-90yrs

c) Group 5- >90yrs

This mean was compared between the Right and Left ear of Group A (Diabetic) with corresponding right and left ear of Group B (Non Diabetic)

Firstly the mean was compared between males of both groups, secondly among the females of both groups, and lastly the comparison was done between males and females within the same groups.

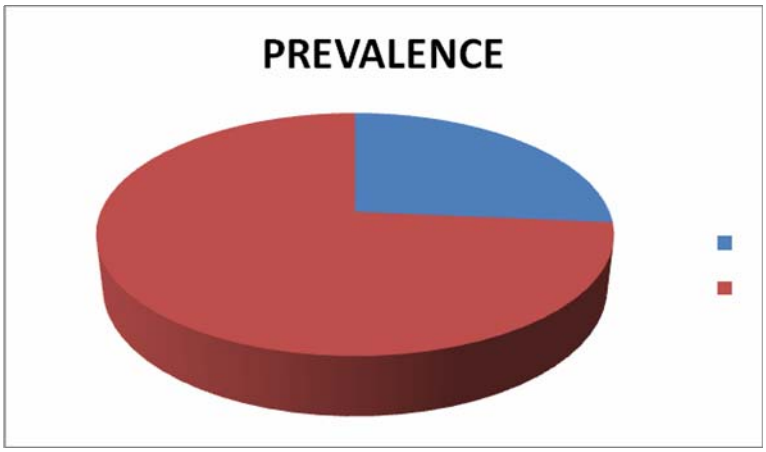
2) The mean pure tone average was compared for different frequencies (250, 500, 1000, 2000, 4000, 8000Hz) among the males of different age groups between cases and controls. This was done similarly among females.
3) The FBS value was compared with the severity of hearing loss.
4) The PPBS value was compared with the severity of hearing loss.
5) The HbA1c value was compared with the severity of hearing loss.
6) The Creatinine values were compared with the severity of hearing loss.
7) The prevalence of SNHL among the Group A and Group B was studied.
8) Age and degree of SNHL was compared between the groups.
9) Duration of diabetes in the Group A was compared with degree of SNHL.

RESULTS

Table 1 - PREVALENCE OF SNHL IN DIABETIC GROUP

DIABETIC CASES	TOTAL NO OF PATIENTS	INCIDENCE
NORMAL HEARING	28	26.4%
SNHL LOSS	78	73.58%

Graph 1 - PREVALENCE OF SNHL IN DIABETIC GROUP

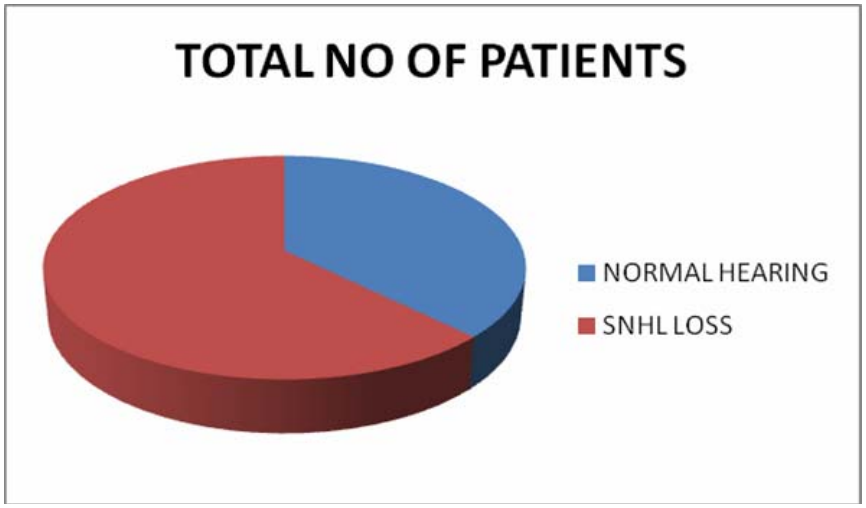


INFERENCE- In our study it was observed that out of 106 diabetic patients, 78 cases were found to have SNHL, therefore incidence of sensorineural hearing loss among the patients suffering from diabetes mellitus was 73.58%.

Table 2 - PREVALENCE OF SNHL IN NON DIABETIC GROUP

Non Diabetic cases	TOTAL NO OF PATIENTS	PREVALENCE
NORMAL HEARING	33	36.6%
SNHL LOSS	55	61.1%

Graph 2 - PREVALENCE OF SNHL IN NON DIABETIC GROUP



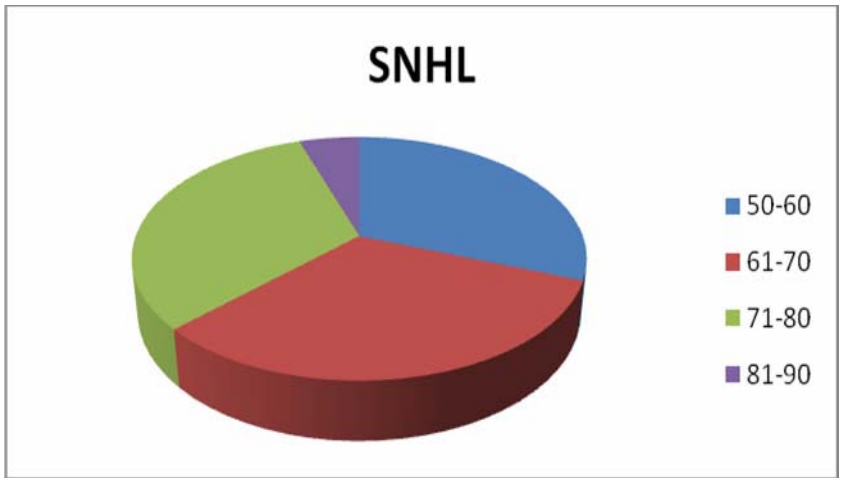
INFERENCE-

In our study it was observed that out of 106 diabetic patients, 55 controls were found to have SNHL, therefore incidence of sensorineural hearing loss among the patients suffering from diabetes mellitus was 61.1%.

Table 3 : AGE WISE DISTRIBUTION OF HEARING LOSS AMONG DIABETICS

GROUP A	Age in years	No of cases	SNHL	Prevalence
Group 1	50-60	36	24	66.6%
Group 2	61-70	35	25	71.42%
Group 3	71-80	29	25	86.2%
Group 4	81-90	6	4	66.6%

Graph 3- AGE WISE DISTRIBUTION OF HEARING LOSS AMONG DIABETICS



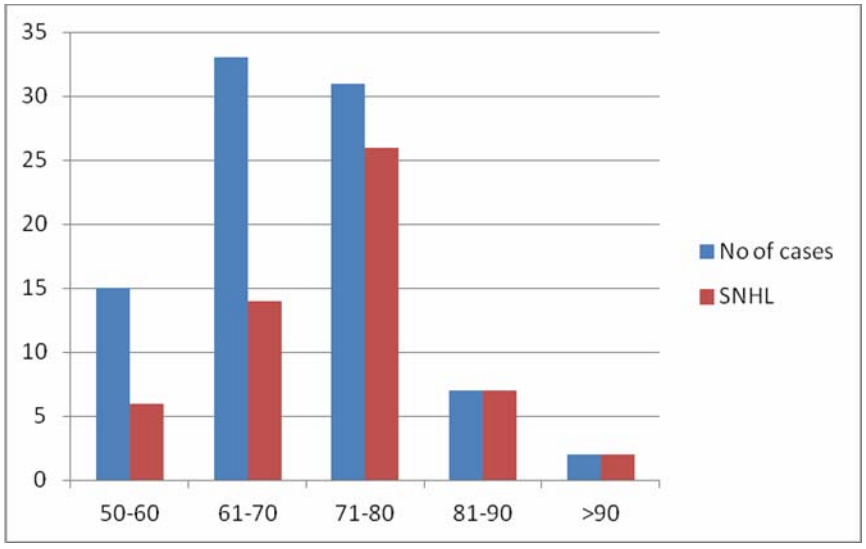
INFERENCE –

Of 24/36 cases in 50-60yrs of age, 25 /35 cases in 61-70yrs of age, 25/ 29 cases in 71-80yrs of age, and 6/ 8 cases in 81-90yrs of age, had SNHL. The incidence of SNHL in these four groups was 66.6%, 71.42%, 86.2% and 66.6% respectively.

Table 4 :AGE WISE DISTRIBUTION OF HEARING LOSS AMONG NON DIABETICS

GROUP B	Age in years	No of cases	SNHL	Incidence
Group 1	50-60	15	6	40%
Group 2	61-70	33	14	42.42%
Group 3	71-80	31	26	83.87%
Group 4	81-90	7	7	100%
Group 5	>90	2	2	100%

Graph 4 : AGE WISE DISTRIBUTION OF HEARING LOSS AMONG NON DIABETICS



INFERENCE –

6 of 15 cases in 50-60yrs of age, 14 of 33 cases in 61-70yrs of age, 26 of 31 cases in 71-80yrs of age, and all 7 in 81-90yrs of age had SNHL. Therefore the incidence in these four groups was 40%, 42.42%, 83.86% and 100% respectively.

Table 5- COMPARISON OF DURATION OF DIABETES MELLITUS WITH SNHL

DURATION	Inference						Total
	A	B	C	D	E	F	
<10	23	16	12	10	7	3	71
10-15	3	6	8	1	2	O	20
15>	2	5	4	3	1	O	15
Total	28	27	24	14	10	3	106

Table 6: PERCENTAGE OF SNHL WITH DURATION OF DIABETES

Degree of hearing loss	Incidence (%) in >10yrs	Incidence (%) in < 10yrs
Mild	30%	22.5%
Moderate	40%	17%
Moderately severe	<5%	9%
Severe	10%	10%

Table 7 : PREVALENCE OF SNHL

Duration in years	Prevalence of SNHL
<10yrs	63%
>10yrs	85%

INFERENCE-

On comparing the duration of diabetes with the severity of hearing loss it was observed that the duration of diabetes mellitus was grouped into 3 divisions <10yrs , 10-15yrs and >15yrs.

The prevalence of SNHL, in group where duration was less than 10yrs was 63% when compared to >10yrs where incidence was 85% which was significantly high.

Among the first group (<10yrs) of the 71 cases majority of the cases were of normal hearing (23) or mild 22.8% (16 cases), moderate 17% (12 cases) or moderately severe 9% (10 cases) with few severe 10% (7 cases) and profound (3 cases) hearing loss. Thereby inferring that patients with duration less <10yrs had **normal to moderate** hearing loss.

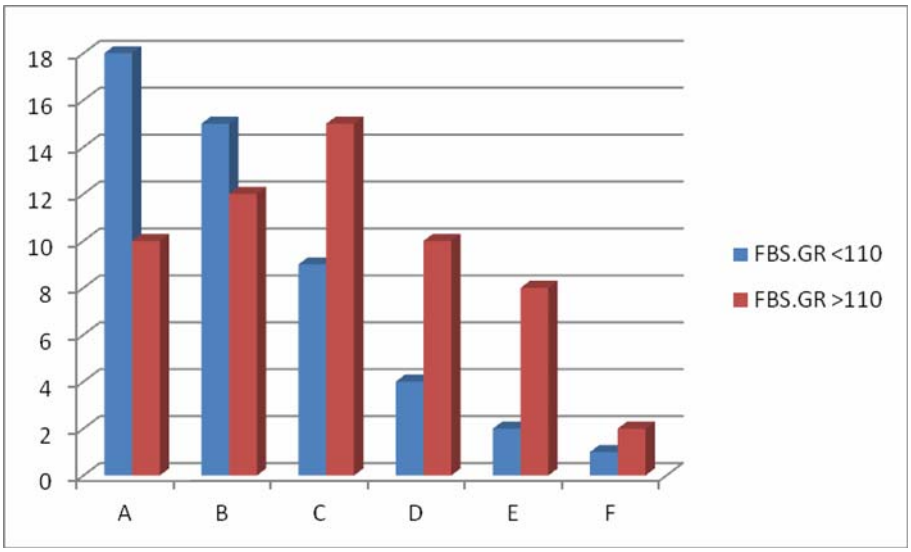
While in group 2 (10-15yrs duration), of the 20 cases most of them were mild 30% (6 cases) or moderate 40% (8 cases), with only few normal hearing (3 cases), moderately severe (<5%) one and severe hearing loss one. Thereby inferring that patients with >10yrs and <15yrs duration had **mild to moderate or moderately severe** hearing loss.

In the group 3 (>15yrs duration), of the 15 cases the number of patients with normal hearing (2 cases), mild hearing loss (5 cases), moderate hearing (4 cases), moderately severe (3cases) and severe (1 case). Here patients with >15yrs duration had either mild or moderate hearing loss.

Table 8 : FBS AND SEVERITY OF HEARING LOSS

Inference	Total subjects	FBS	
		<110	>110
A	28	18	10
B	27	15	12
C	24	9	15
D	14	4	10
E	10	02	8
F	03	01	02
Total	**106**	**49**	**57**

Graph 5 : FBS AND SEVERITY OF HEARING LOSS



INFERENCE :

On comparing the results of FBS values with severity of hearing loss it was seen that out of 106 cases 49 cases had FBS levels <110mg/dl and 57 cases >110mg/dl.

The incidence of SNHL in patients less than 110mg/dl was 60.2% compared to patients with more than 110mg/dl where the incidence was 83%.

Among these cases with <110mg/dl (49 cases) majority of them were normal (18 cases) or had mild hearing loss (15 cases). Only a few had moderate (9 cases) to moderately severe (4 cases), severe (2 cases) and profound (1 case) sensorineural hearing loss.

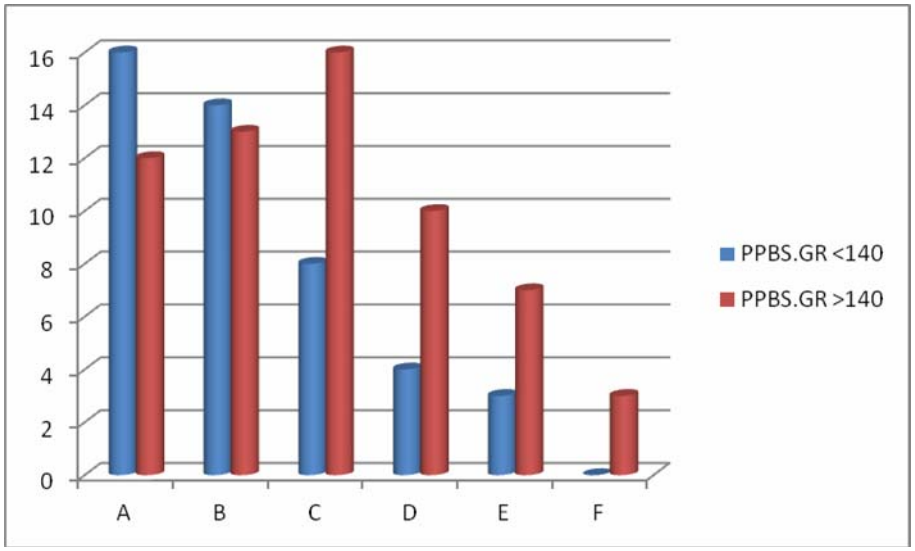
Among the 57 cases with >110mg/dl it was seen that (10 cases) had normal hearing, (12cases) had mild, (15 cases) had moderate, (10cases) had moderately severe, (8 cases) severe and (2 cases) had profound hearing loss. Patients with moderate, moderately severe, severe to profound hearing loss most of them had PPBS values >110mg/dl. However there were few cases with normal hearing (10 cases) who had PPBS values >110mg/dl.

The observation yields that the clustering of patients with FBS >110mg/dl is towards moderate to profound hearing loss therefore inferring that cases with high FBS (sugar levels) had more severe hearing loss in comparison to controls.

Table 9 : PPBS AND SEVERITY OF HEARING LOSS

Inference	Total subjects	PPBS group	
		<140	**>140**
A	28	16	12
B	27	14	13
C	24	08	16
D	14	04	10
E	10	3	7
F	03	00	03
Total	**106**	**45**	**61**

Graph 6 : PPBS AND SEVERITY OF HEARING LOSS



INFERENCE:

On comparing the PPBS values with severity of hearing loss it was seen that out of 106 cases 45 cases had PPBS levels <140mg/dl and 61 cases >140mg/dl. Among these cases (45 cases) majority of them had normal (16 cases) or mild hearing loss (14 cases). Only a few had moderate (8 cases) to moderately severe (4 cases) & severe (3 cases) hearing loss.

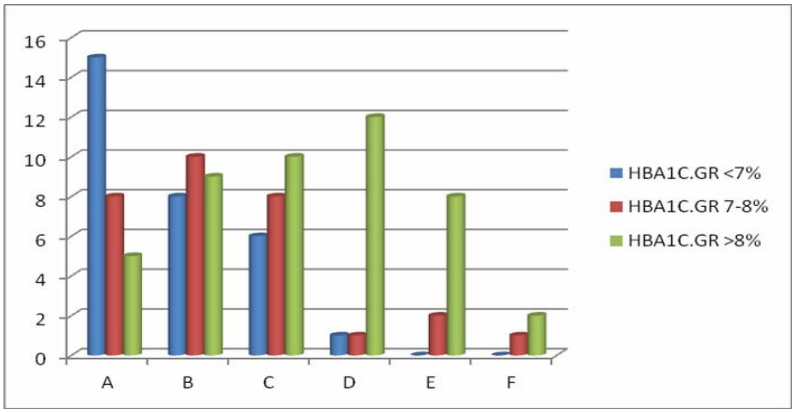
Among the 61 cases with >140mg/dl it was seen that (12 cases) had normal hearing, (13cases) had mild, (16 cases) had moderate, (10cases) had moderately severe, (7 cases) severe and (3 cases) had profound hearing loss. Patients with moderate, moderately severe, severe to profound hearing loss most of them had PPBS values >140mg/dl. However there were few cases with normal hearing (12 cases) who had PPBS values >140mg/dl.

The observation yields that the clustering of patients with PPBS levels >140mg/dl is towards moderate to profound hearing loss therefore inferring that cases with high PPBS (sugar levels) had more severe hearing loss in comparison to controls.

Table 10 : HbA1c VALUE COMPARED WITH THE SEVERITY OF HEARING LOSS

Inference	Total Subjects	HbA1c Grading		
		<7%	7-8%	>8%
A	28	15	8	5
B	27	8	10	9
C	24	6	8	10
D	14	1	1	12
E	10	0	2	8
F	3	0	1	2
Total	**106**	**30**	**30**	**46**

Graph 7 : HbA1c WITH SEVERITY OF HEARING LOSS



INFERENCE- On comparing the relation between the HbA1c and severity of hearing loss it was seen that out of 106 patients in Diabetic group, 62% (<8% HbA1c) had SNHL and 89% (>8%HbA1c) had SNHL which was highly significant.

Further patients with good glycaemic control (<7%) were around 30 and most of them had normal hearing sensitivity 40% (15cases) with few cases had mild 30% (8 cases) and moderate hearing loss 20% (6cases).

In cases with glycaemic control around 7-8% (30 cases) most of the cases had either normal (8cases) or mild (10cases) to moderate hearing loss (8cases).

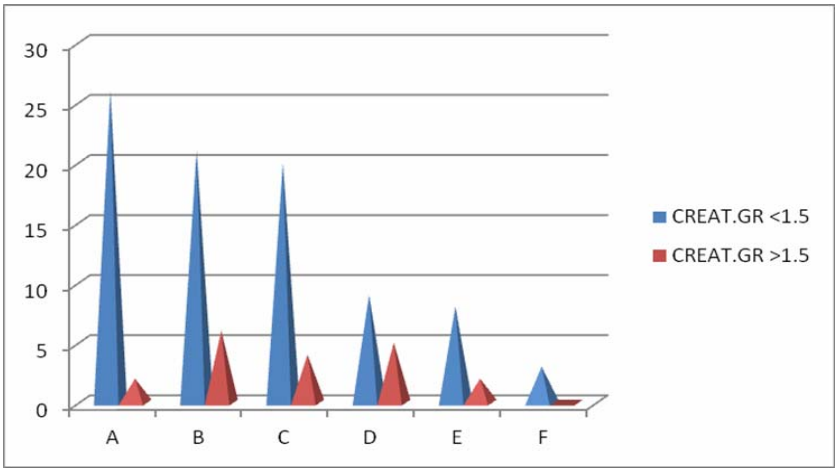
However of 46cases with >8% of glycaemic control, majority of the cases had moderate (21%) to moderately severe (26%) hearing loss and few cases had severe to profound HL (19% each).

Thereby, inferring that poorer the glycaemic control, severe is the hearing loss.

Table 11 – SERUM CREATININE LEVELS AND SEVERITY OF HEARING LOSS.

Inference	Total subjects	Creatinine group	
		1.00 <1.5	2.00 >1.5
A	28	26	2
B	27	21	6
C	24	20	4
D	14	9	5
E	10	8	2
F	3	3	0
Total	**106**	**87**	**19**

GRAPH 8 – CREATININE LEVELS COMPARED WITH SEVERITY OF HEARING LOSS



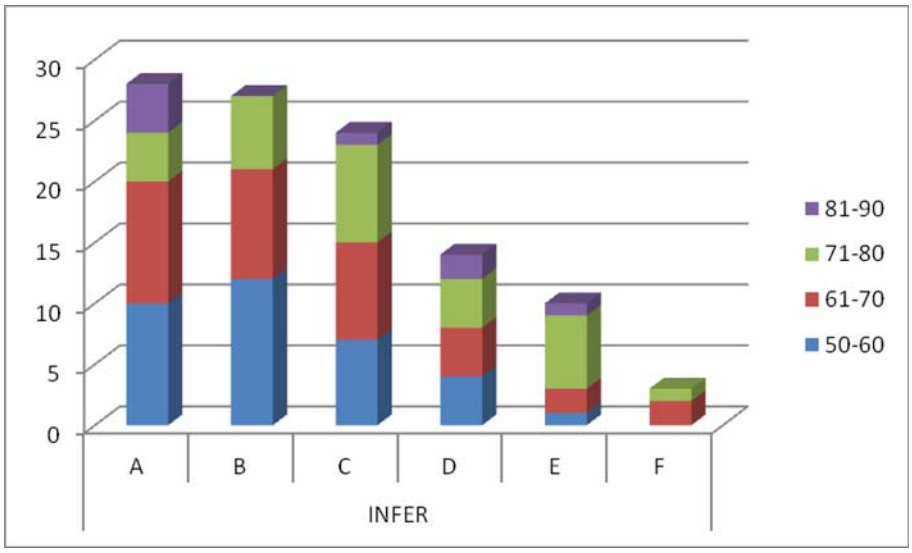
INFERENCE-

On comparing the levels of serum creatinine with severity of hearing loss it was seen that of the 106 patients in diabetic group, 87 patients had serum creatinine levels <1.5mg/dl. Among them most of the patients had normal or mild to moderate hearing loss with only a few patients having moderately severe to severe hearing loss (26- normal , 21- mild, 20- moderate, 9- moderately severe 8- severe, 3- profound). In 19 cases in whom the serum creatinine levels >1.5mg/dl, 6 had mild, 4 moderate, 5 moderately severe, 2 severe and none with profound hearing loss. It can be inferred that the serum creatinine levels does not affect the severity of hearing loss.

Table 12 : AGE AND SEVERITY OF SNHL IN DIABETIC GROUP.

Age group	Total no	Inference					
		A	B	C	D	E	F
1.00	34	10	12	7	4	1	0
2.00	35	10	9	8	4	2	02
3.00	29	4	6	8	4	6	01
4.00	8	4	0	1	2	1	0
Total	**106**	**28**	**27**	**24**	**14**	**10**	**3**

Graph 9 : AGE AND SEVERITY OF SNHL IN DIABETIC GROUP.



INFERENCE

Among the diabetic group out of 106 cases, 28 cases had normal hearing, (27 cases) had mild (B), (24cases) moderate (C), (14 cases) moderately severe (D), (10 cases) severe (E) and 3 cases had profound (F) hearing loss.

Among the different age groups, in Group 1 (50-60yrs), of the 34 cases most of them were normal to moderate hearing loss [10 normal (A), 12 mild (B), 7 moderate (C)]. Only 4 cases had moderately severe and 1 case had severe hearing loss. Thereby inferring that in this age group most of the cases had either normal hearing or up to moderate hearing loss.

Group II (61-70yrs), of the 35 cases most of them were normal to moderate hearing loss [10 normal (A), 9 mild (B), 8 moderate (C)]. Only 4 cases had moderately severe (D), 2 cases with severe (E) & 2 cases with profound (F) hearing loss. Thereby inferring that in this age group most of the cases had either normal hearing or up to moderate hearing loss.

Group III (71-80yrs), of the 29 cases most of them were mild to moderate or moderately severe hearing loss [4 normal (A), 6 mild (B), 8 moderate (C), 4 moderately severe (D), 6 severe (E) and 1 profound (F)]. Thereby inferring that this age group most of the cases had either moderate to severe hearing loss.

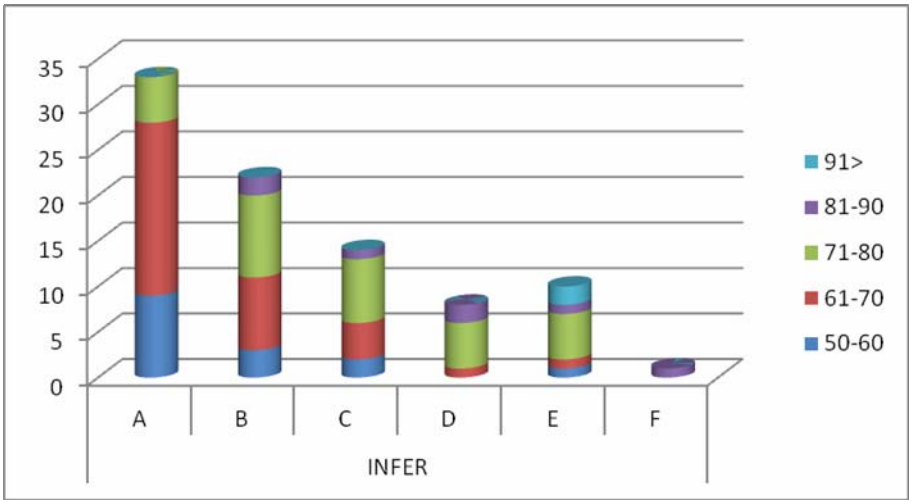
Group IV (81-90yrs), of the 8 cases, 4 had normal hearing. In the remaining 4cases, one had moderate, two moderately severe and one severe hearing loss.

Most of the cases in the diabetic group belonged to the category of mild, moderate or moderately severe hearing loss.

Table 13- AGE AND SEVERITY OF SNHL IN CONTROLS

AGE.GR	Total	INFER					
		A	B	C	D	E	F
1.00	15	9	3	2	0	1	0
2.00	34	19	9	4	1	1	0
3.00	32	5	10	7	5	5	0
4.00	7	0	2	1	2	1	1
5.00	2	0	0	0	0	2	0
TOTAL	**90**	**33**	**24**	**14**	**8**	**10**	**1**

Graph 10: AGE AND SEVERITY OF SNHL IN CONTROLS



Among the Non diabetic group out of 90 cases, 33 cases had normal hearing, 24 had mild hearing loss (B), 14 had moderate hearing loss(C), 8 had moderately severe (D), 10 severe and 1 had profound hearing loss.

Among the different age groups, in Group 1 (50-60yrs), of the 15 cases most of them were normal hearing loss [9 normal (A), 3 mild (B), 2moderate (C)]. Only 1 case had severe hearing loss. Thereby inferring that this age group most of the cases had normal hearing with only few mild hearing loss.

Group II (61-70yrs), of the 34 cases most of them were normal to mild hearing loss with only few moderate hearing loss [19 normal (A), 9 mild (B), 4 moderate (C)]. Only 1 case had moderately severe (D) & 1 case with severe hearing loss. Thereby inferring that this age group most of the cases had either normal hearing to mild hearing loss.

Group III (71-80yrs), of the 32 cases most of them were mild to moderate or moderately severe & severe hearing loss [5 normal (A), 10 mild (B), 7 moderate (C), 5 moderately severe (D), 5 severe (E)]. Thereby inferring that this age group most of the cases had either mild to severe hearing loss.

Group IV (81-90yrs), of the 7 cases, none had normal hearing, 2 mild, one moderate, two moderately severe and one severe hearing loss.

Most of the cases in the Non diabetic group belonged to the category of mild or up to moderately severe hearing loss.

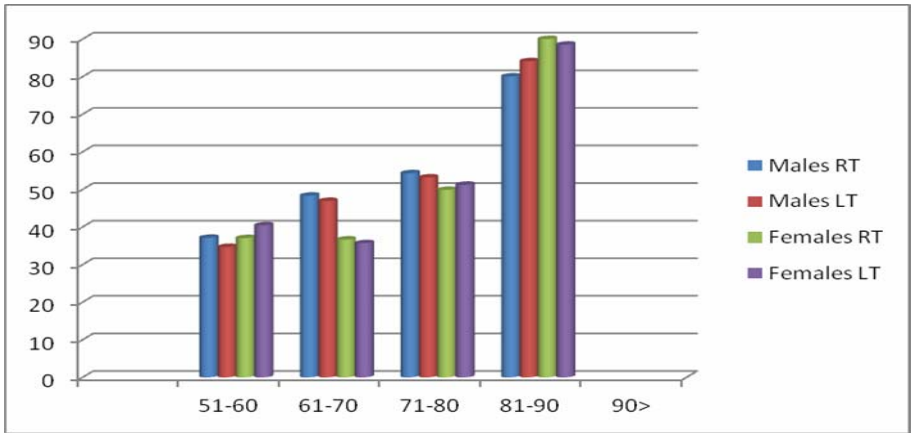
A) Comparison of the mean Pure Tone Threshold average between diabetic and non diabetic males and females.

Table 14 – AVERAGE PTA COMPARISON BETWEEN MALES AND FEMALES OF DIABETIC GROUP

	Males					Females				
AGE	**TOTAL**	**RT**	**SD**	**LT**	**SD**	**TOTAL**	**RT**	**SD**	**LT**	**SD**
51-60	14	33.17	17.57	32.89	16.20	22	36.46	21.16	36.35	20.83
61-70	16	48.33	21.87	46.93	20.91	19	36.66	14.60	35.68	15.78
71-80	15	54.283	21.87	53.17	20.33	14	49.87	23.81	51.23	21.55
81-90	4	86.04	11.65	84.10	14.35	2	90	15.34	88.5	17.68
90>	0	0	0	0	0	0	0	0	0	0

Age in years (diabetic group)	**PTA average in males(D)**	**PTA average in females(D)**	**Difference in dB Loss between the two**
50-60yrs	33.03dB	36.405 dB	2-3dB
61-70yrs	47.63 dB	36.17 dB	10 dB
71-80yrs	53.72 dB	50.55 dB	4-6 dB
81-90yrs	85.073 dB	89.25 dB	4-6 dB

Graph 11 – AVERAGE PTA COMPARISON BETWEEN MALES AND FEMALES OF DIABETIC GROUP



INFERENCE –

On comparing the results between the males and females of diabetic group, the average hearing threshold on both the sides were plotted on a bar chart. It was seen that the hearing threshold increased linearly with the increase in the age and that the threshold in both the ears was almost symmetrical in both the sex groups. However it was seen that on comparing the average threshold, males had slightly higher hearing loss than the females. But when the difference in the dB loss was compared between the it was almost same with 3-4dB variation only with no much significant difference in hearing threshold between the two.

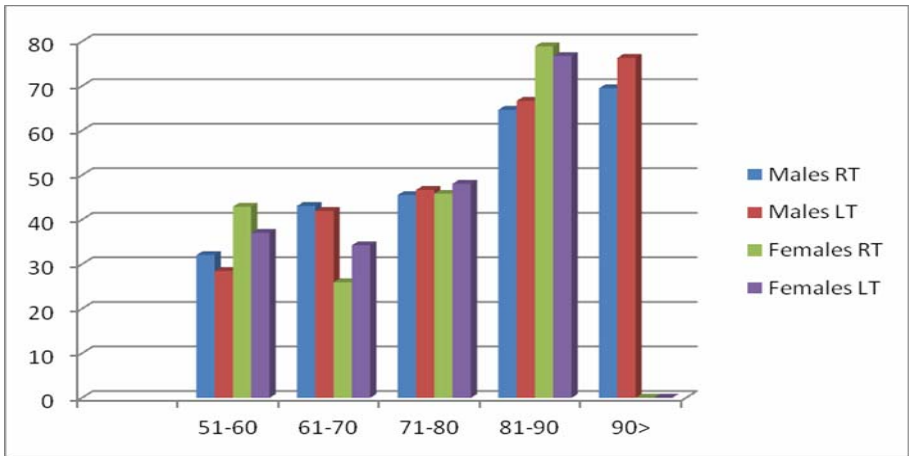
Table 15 : PTA COMPARISON BETWEEN MALES AND FEMALES OF NON DIABETIC GROUP

Males						Females				
AGE	**TOTAL**	**RT**	**SD**	**LT**	**SD**	**TOTAL**	**RT**	**SD**	**LT**	**SD**
51-60	11	32.05	12.65	28.49	10.22	4	42.916	35.04	37.05	28.715
61-70	24	43.08	19.19	41.953	18.25	10	35.92	8.89	34.25	9.703
71-80	23	45.53	19.97	46.66	18.03	9	45.81	27.49	48.06	26.82
81-90	6	64.66	26.99	68.67	19.25	1	73.92	13.64	76.74	12.04
90>	2	78.85	3.53	76.33	4.73	0		0		0

Average PTA in males and females among Non diabetic group

Age in years (Non diabetic group)	PTA average in Males (ND)	PTA average in females(ND)	Difference in dB Loss between non diabetics males and females
50-60yrs	30.27dB	36.983 dB	2-3 dB
61-70yrs	42.516 dB	35.085 dB	10 dB
71-80yrs	46.09 dB	42.93 dB	4-6 dB
81-90yrs	66.66 dB	69.33 dB	2-4 dB

Graph 12 – PTA COMPARISON BETWEEN MALES AND FEMALES OF NON DIABETIC GROUP



INFERENCE

On comparing the results between the males and females in the control group the average hearing threshold on both the sides were plotted on a bar chart. It was seen that the hearing threshold increased linearly with the increase in the age and that the threshold in both the ear was almost symmetrical in both the sex group. However with increasing age it was observed that males had slightly higher hearing loss than the females except at 51-60yrs and 81-90yrs where females had slightly higher loss than males.

C) **Comparison of the mean values of the Pure Tone threshold average for different frequencies at different age groups among the Diabetic and the Non Diabetic Females in the right and the left ear**

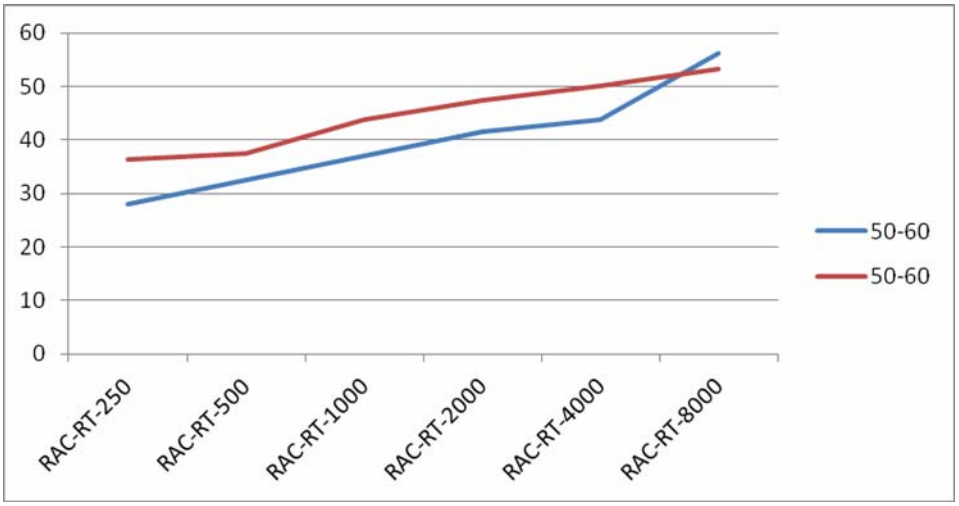
1) **50-60yrs**

Right ear

Table 16 – PTA AT 50-60YRS AGE IN FEMALES RE, (CASES VS CONTROLS)

AGE	RAC-RT-250	RAC-RT-500	RAC-RT-1000	RAC-RT-2000	RAC-RT-4000	RAC-RT-8000
Cases(F) 50-60	28.04	32.6	36.95	41.52	43.86	56.23
Control(F)50-60	36.25	37.5	43.75	47.5	50	53.25
p value	0.03	0.02	0.004	0.03	0.009	0.06

Graph 13- PTA at 50-60yrs age in females RE, (cases vs controls)



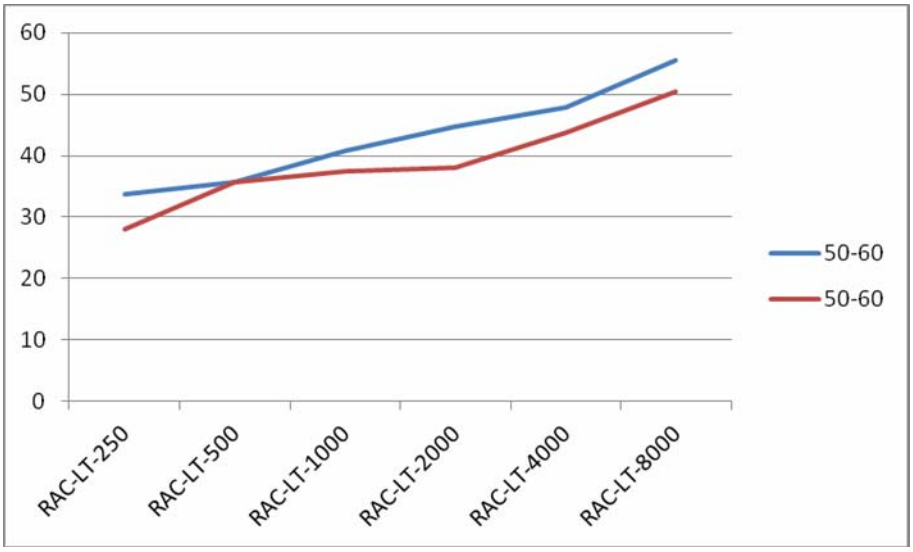
INFERENCE : On comparing the mean pure tone average in right ear between the diabetic and the non diabetic females in the age group of 50-60yrs it was seen that firstly, both the groups had symmetrical hearing loss with an increase at higher frequencies and secondly, pure tone average of the control group was showing a slight increase (5-7dB) in hearing loss at all the frequencies when compared to the cases except at 8000hz

Left ear

Table 17 : PTA AT 50-60YRS AGE IN FEMALES LE, (CASES VS CONTROLS)

AGE	RAC-LT-250	RAC-LT-500	RAC-LT-1000	RAC-LT-2000	RAC-LT-4000	RAC-LT-8000
Cases(F)50-60	30.69	33.36	34.68	44.78	45.95	55.45
Control(F)50-60	34.84	35.67	40.87	45.89	50.75	51.5
p value	0.047	0.12	0.01	0.31	0.03	0.03

Graph 14– PTA AT 50-60YRS AGE IN FEMALES LE, (CASES VS CONTROLS)



INFERENCE : On comparing the mean pure tone average in the left ear between the diabetic and the non diabetic females in the age group of 50-60yrs it was seen that firstly, both the groups had symmetrical hearing loss with an increase at higher frequencies and secondly, pure tone average of the control group was showing a slight increase in hearing loss at all the frequencies when compared to the case except at 8000hz.

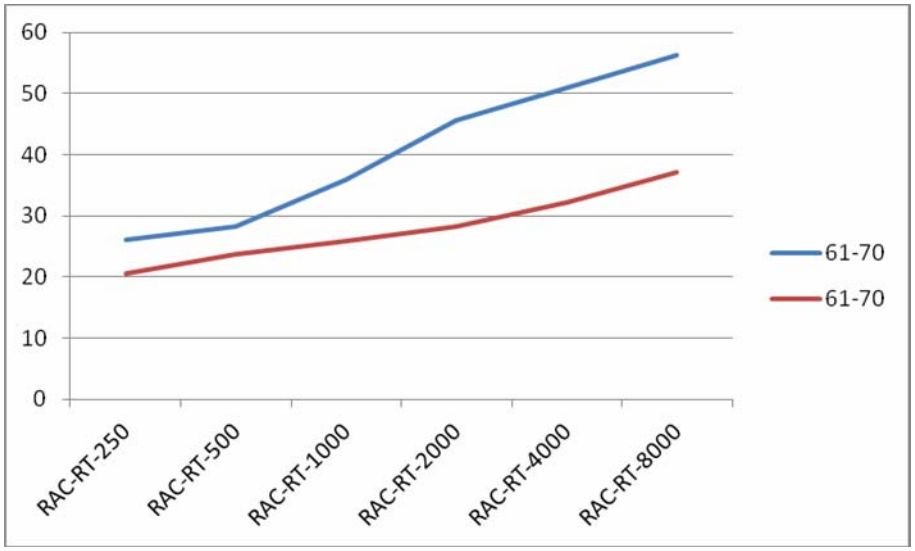
2)61-70yrs

RIGHT EAR

Table 18 – PTA AT 61-70YRS AGE IN FEMALES RE, (CASES VS CONTROLS)

AGE	RAC-RT-250	RAC-RT-500	RAC-RT-1000	RAC-RT-2000	RAC-RT-4000	RAC-RT-8000
61-70	26	28.33	36	45.67	51	56.33
61-70	20.55	23.66	25.88	28.22	32.22	37.22
p value	0.02	0.008	0.001	0.0001	0.0001	0.0001

Graph 15– PTA AT 61-70YRS AGE IN FEMALES RE, (CASES VS CONTROLS)



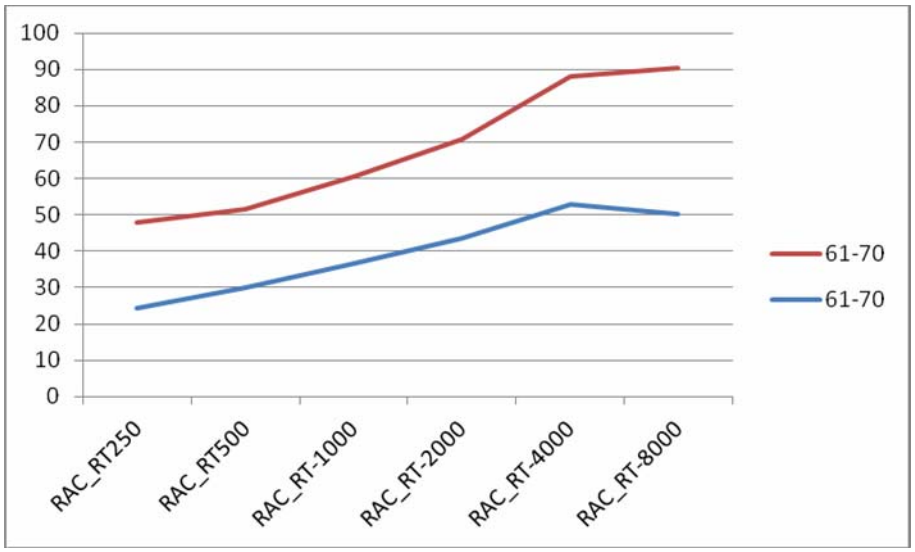
INFERENCE – On comparing the mean pure tone average between the diabetic and the non diabetic females in the age group of 61-70yrs in the right ear it was seen that, both the groups had hearing loss which increased progressively with increase in the frequencies. The difference between both groups was about 5 -10 dB at 250, 500, 1000 Hz and 17- 20 dB at 2000, 4000, 8000 Hz. The degree of hearing loss was statistically significant ($p<0.05$) at all the frequencies.

LEFT EAR

Table 19– PTA AT 61-70YRS AGE IN FEMALES LE, (CASES VS CONTROLS)

AGE	RAC-LT-250	RAC-LT-500	RAC LT-1000	RAC-LT-2000	RAC-LT-4000	RAC-LT-8000
61-70	24.3	29.83	36.57	43.67	53	50.33
61-70 p value	23.69 0.23	21.66 0.04	23.88 0.02	27.22 0.001	35.22 0.0001	40..22 0.02

Graph 16– PTA AT 61-70YRS AGE IN FEMALES LE, (CASES VS CONTROLS)



INFERENCE – On comparing the mean pure tone average between the diabetic and the non diabetic females in the age group of 61-70yrs in the left ear it was seen that, both the groups had hearing loss which increased progressively with increase in the frequencies. The difference between both groups was about 8 and 13 dB at 500, 1000Hz and approximately 15- 18 dB at 2000, 4000, 8000 Hz. The degree of hearing loss was statistically significant ($p<0.05$) at all frequencies except at 250Hz..

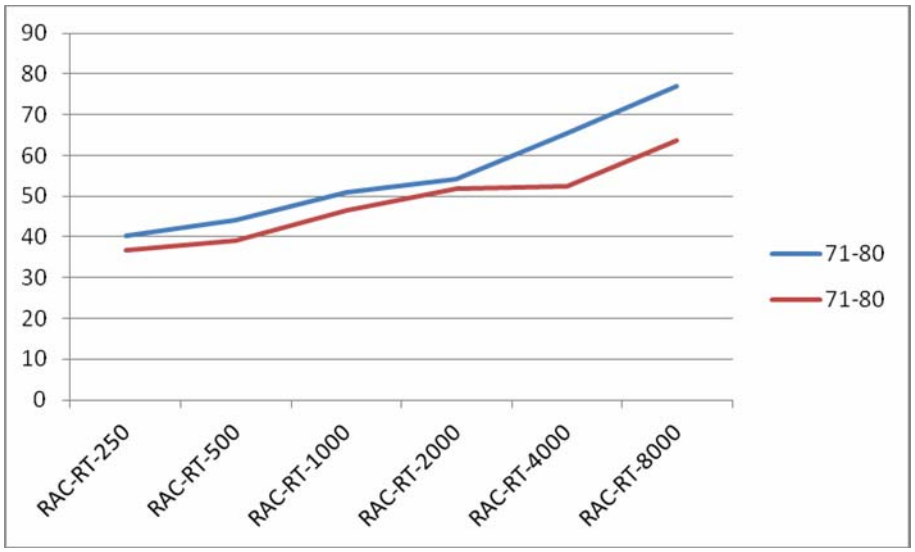
2) 71-80yrs

RIGHT EAR

Table 20– PTA AT 71-80YRS AGE IN FEMALES RE, (CASES VS CONTROLS)

AGE	RAC-RT-250	RAC-RT-500	RAC-RT-1000	RAC-RT-2000	RAC-RT-4000	RAC-RT-8000
71-80	40.35	44.28	51.07	54.28	65.35	77.14
71-80	36.87	39.12	46.45	51.87	52.5	63.75
p value	0.04	0.03	0.04	0.08	0.01	0.009

Graph 17 – PTA AT 71-80YRS AGE IN FEMALES RE, (CASES VS CONTROLS)



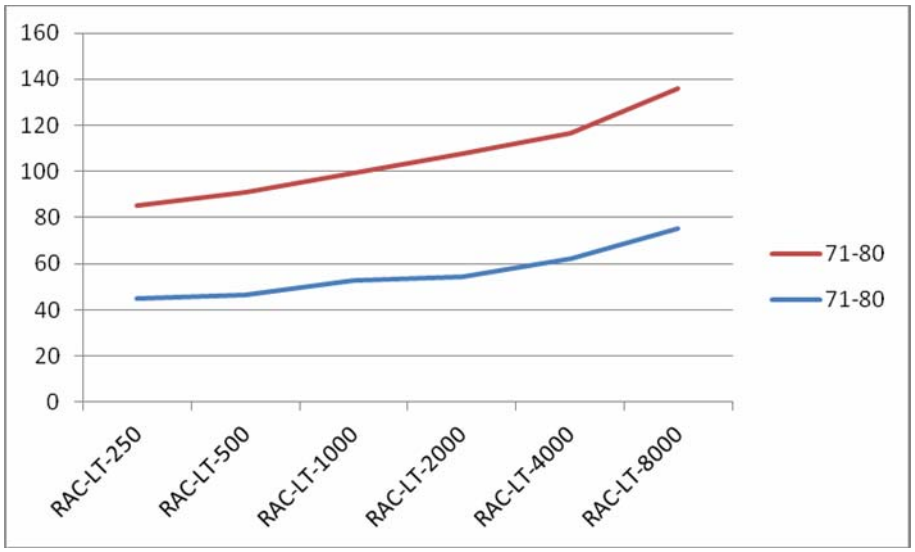
INFERENCE - On comparing the mean pure tone averages between the diabetic and the non diabetic females in the age group of 71-80yrs in the right ear it was seen that, both the groups had hearing loss which increased progressively with increase in the frequencies. The difference between both groups was approximately 4 -5 dB at 250, 500, 1000 & 2000 Hz and 13- 15 dB at 4000 & 8000 Hz. The degree of hearing loss was statistically significant ($p<0.05$) at all the frequencies.

3) LEFT EAR

Table 21– PTA AT 71-80YRS AGE IN FEMALES LE, (CASES VS CONTROLS)

AGE	RAC-LT-250	RAC-LT-500	RAC-LT-1000	RAC-LT 2000	RAC-LT-4000	RAC-LT -8000
71-80	44.8	46.25	53	54.44	62.32	75.35
71-80	40.5	44.54	46.23	53.42	54.34	60.5
p value	0.04	0.32	0.009	3.45	0.01	0.001

Graph 18– PTA AT 71-80YRS AGE IN FEMALES LE, (CASES VS CONTROLS)



INFERENCE - On comparing the mean pure tone averages between the diabetic and the non diabetic females in the age group of 71-80yrs in the left ear it was seen that, both the groups had hearing loss which increased progressively with increase in the frequencies. The difference between both groups was about 2 -6 dB at 250, 500, &1000, 2000 Hz and 8–15dB at 4000, & 8000 Hz. The degree of hearing loss was statistically significant except at 500 and 200Hz.

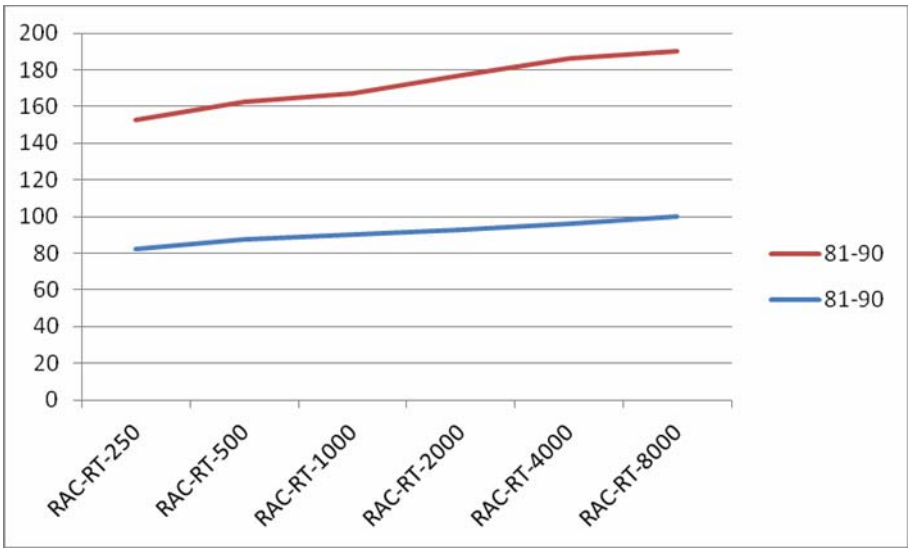
4) **81-90 yrs**

RIGHT EAR

Table 22- PTA AT 81-90YRS AGE IN FEMALES RE, (CASES VS CONTROLS)

AGE	RAC-RT-250	RAC-RT-500	RAC-RT-1000	RAC-RT-2000	RAC-RT-4000	RAC-RT-8000
81-90	74.67	84.5	90	92.5	96	100
81-90	70	75	79.59	84.86	87.38	90
p value	0.03	0.001	0.004	0.01	0.03	0.01

Graph 19- PTA AT 81-90YRS AGE IN FEMALES RE, (CASES VS CONTROLS)



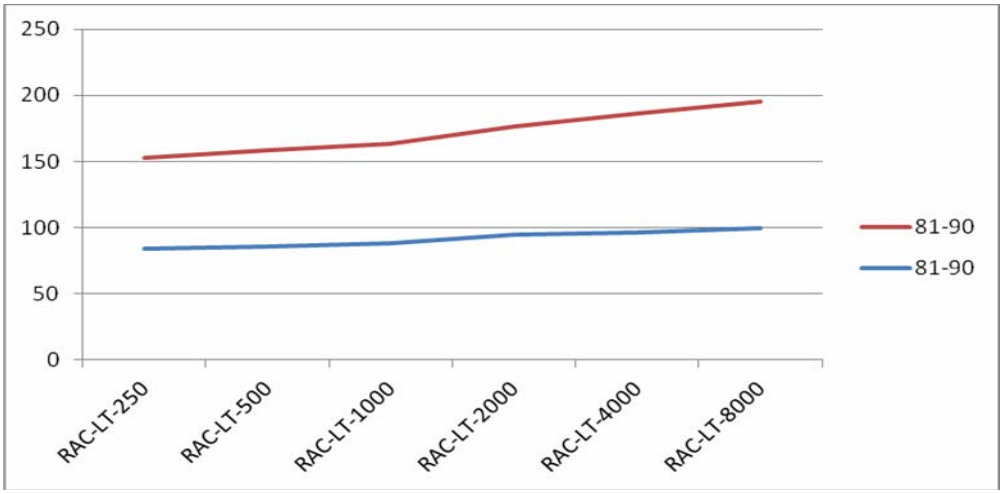
INFERENCE - On comparing the pure tone average between the diabetic and the non diabetic females in the age group of 81-90yrs in the right ear it was seen that, both the groups had hearing loss which increased progressively with increase in the frequencies. The difference between both groups was about 5 dB at 250Hz, whereas in all other frequencies the difference in hearing loss was 8-10dB. The degree of hearing loss was statistically significant at all frequencies <0.05.

LEFT EAR

Table 23- PTA AT 81-90YRS AGE IN FEMALES LE, (CASES VS CONTROLS)

AGE	RAC-LT-250	RAC-LT-500	RAC-LT-1000	RAC-LT-2000	RAC-LT-4000	RAC-LT-8000
81-90	74.3	86.33	88.57	94.50	96.52	100
81-90	68.45	72.64	75.24	82.35	90	95
p value	0.02	0.001	0.006	0.009	0.03	0.04

Graph 20- PTA AT 81-90YRS AGE IN FEMALES LE, (CASES VS CONTROLS)



INFERENCE -

On comparing the pure tone average between the diabetic and the non diabetic females in the age group of 81-90yrs in the left ear it was observed that, both the groups had hearing loss which increased progressively with increase in the frequencies. The difference between both groups was about 6dB at 250Hz, 4000Hz & 8,000Hz. Whereas in other frequencies the difference in hearing loss ranged from 12-14dB. The degree of hearing loss was statistically significant at all the frequencies <0.05

Average difference in decibel loss at low and high frequencies in different age groups in diabetic and non diabetic females

Table 24 - DIFFERENCE IN DB LOSS IN DIFFERENT AGE GROUPS AT LOW AND HIGH FREQUENCY IN DIABETIC AND NON DIABETIC FEMALES (RE &LE)

Age group	Low frequency (RE)	High frequency (RE)	Low frequency (LE)	High frequency (LE)
50-60yr	5-7DB	5-7dB	5-7dB	5-7Db
61-70yr	5-10 dB	17-20 dB*	8-13 dB	15-18 dB*
71-80yr	4-5 dB	13-15 dB*	4-5 dB	13-15dB*
81-90yr	8-10dB	8-10DB	12-14 dB (Only 6dB at 250Hz)	6-8dB

Inference : It was observed in our study diabetic females had more hearing loss compared to non diabetics in all the age groups and all frequencies. However the difference in the average decibel loss when taken at low (250, 500 and 1000 Hz) and high frequencies (4KHz and 8KHz) between both the groups at different age groups showed that the maximum difference was seen at higher frequencies compared to lower frequencies except at 81-90 yrs where the difference at frequencies was constant 8-12dB.

D) Comparison of the mean values of the Pure Tone threshold average for different frequencies at different age groups among the Diabetic and the Non Diabetic Males in the right and the left ear.

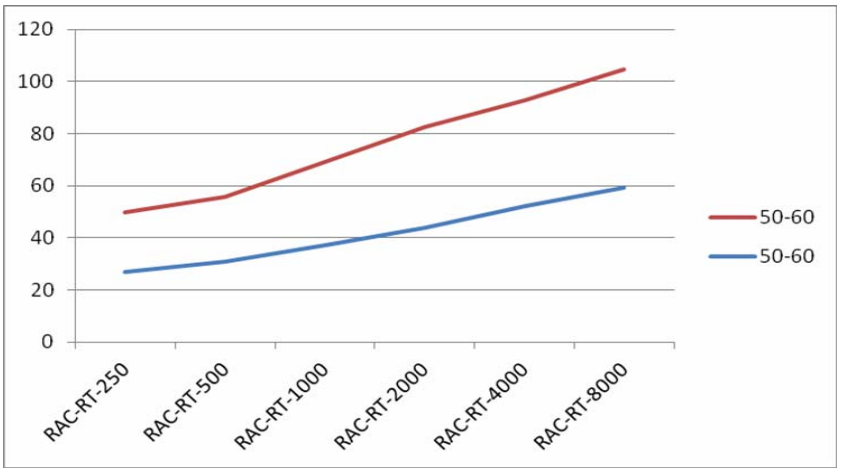
1) 50-60yrs

RIGHT EAR

Table 25 – PTA AT 50-60YRS AGE IN MALES RE, (CASES VS CONTROLS)

AGE	RAC-RT-250	RAC-RT-500	RAC-RT-1000	RAC-RT-2000	RAC-RT-4000	RAC-RT-8000
50-60	27	30.67	37	43.67	52.33	59.33
50-60	22.63	25.18	32.18	38.81	40.45	45.36
p value	0.04	0.03	0.03	0.02	0.001	0.001

Graph 21 – PTA AT 50-60YRS AGE IN MALES RE, (CASES VS CONTROLS)



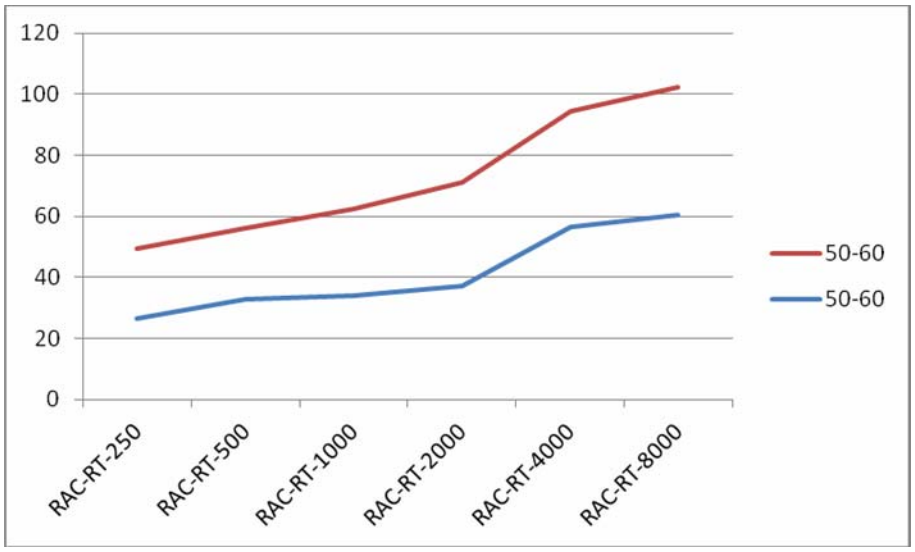
INFERENCE – On comparing the results between the diabetic and the non diabetic males in the age group of 50-60yrs in the right ear it was seen that, both the groups had hearing loss which increased progressively with the increase in the frequencies. A difference in the hearing loss of 5-6dB was noted in the lower frequencies and approximately12-14 dB loss in higher frequencies. A statistically significant p values was found at 4 and 8KHz.

LEFT EAR

Table 26 : PTA AT 50-60YRS AGE IN MALES LE, (CASES VS CONTROLS)

AGE	RAC-LT-250	RAC-LT-500	RAC-LT-1000	RAC-LT-2000	RAC-LT-4000	RAC-LT-8000
50-60	26.4	32.86	34	37.20	56.33	60.47
50-60	23	23.22	28.27	34	37.89	42
p value	0.07	0.02	0.01	0.09	0.0001	0.0001

Graph 22– PTA at 50-60yrs age in males LE, (cases vs controls)



INFERENCE – On comparing the mean pure tone averages between the diabetic and the non diabetic males in the age group of 50-60yrs in the left ear it was seen that, both the groups had hearing loss which increased progressively with the increase in the frequencies. A difference in the hearing loss of 3-8dB was noted in the lower frequencies and approximately15-18 dB loss in higher frequencies. A statistically significant values was found in all frequencies but more at higher frequencies.

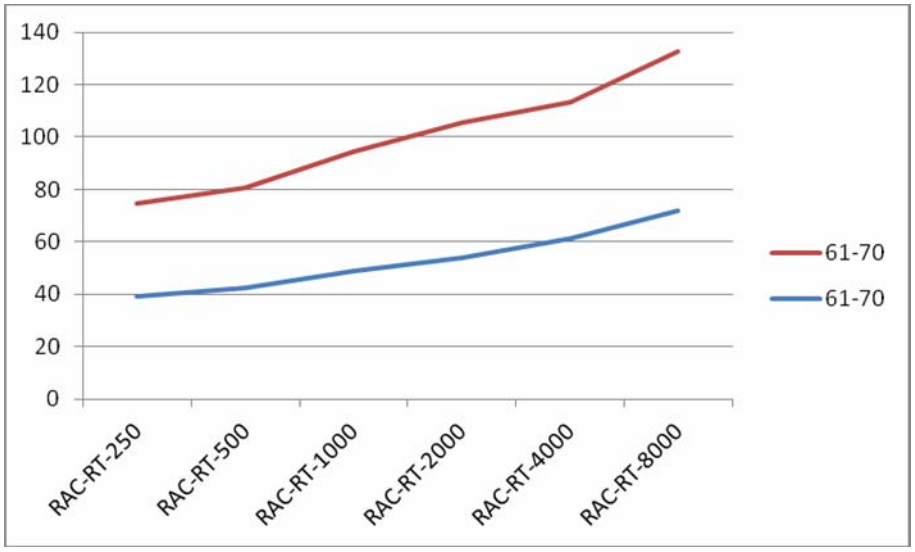
2) 61-70yrs

RIGHT EAR

Table 27 – PTA AT 61-70YRS AGE IN MALES RE, (CASES VS CONTROLS)

AGE	RAC-RT-250	RAC-RT-500	RAC-RT-1000	RAC-RT-2000	RAC-RT-4000	RAC-RT-8000
61-70	39.4	42.25	49	53.75	61.25	72
61-70	35.25	38.25	45.25	51.75	52	60.75
p value	0.07	0.08	0.05	0.23	0.008	0.001

Graph 23– PTA AT 61-70YRS AGE IN MALES RE, (CASES VS CONTROLS)



INFERENCE – On comparing the pure tone averages between the diabetic and the non diabetic males in the age group of 61-70yrs in the right ear it was seen that, both the groups had hearing loss which increased progressively with the increase in the frequencies but the difference in the hearing loss was approximately 10-12dB at 4000 and 8000 Hz. However the hearing loss was almost symmetrical at low frequencies with only about 3-4dB difference in the hearing loss.

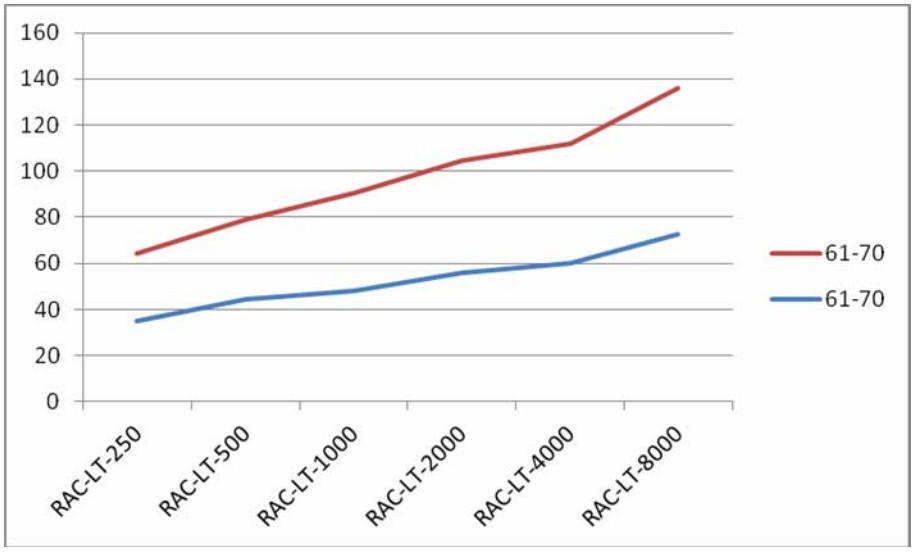
A statistically significant p values was found at most of the frequencies.

LEFT EAR

Table 28– PTA AT 61-70YRS AGE IN MALES LE, (CASES VS CONTROLS)

AGE	RAC-LT-250	RAC-LT-500	RAC-LT-1000	RAC-LT-2000	RAC-LT-4000	RAC-LT-8000
61-70	35.25	44.25	48.25	55.75	60	72.75
61-70	28.91	34.79	42.45	48.62	51.91	63.5
p value	0.03	0.009	0.01	0.01	0.007	0.002

Graph 24– PTA AT 61-70YRS AGE IN MALES LE, (CASES VS CONTROLS)



INFERENCE –

On comparing the pure tone averages between the diabetic and the non diabetic males in the age group of 61-70yrs in the left ear it was seen that, both the groups had hearing loss which increased progressively with the increase in the frequencies but the difference in the hearing loss at 4KHz & 8KHz was approximately 10dB. However the hearing loss in low frequencies was almost symmetrical with about 5-7dB difference only. A statistically significant p values was seen in all the frequencies but significant mainly in higher frequencies.

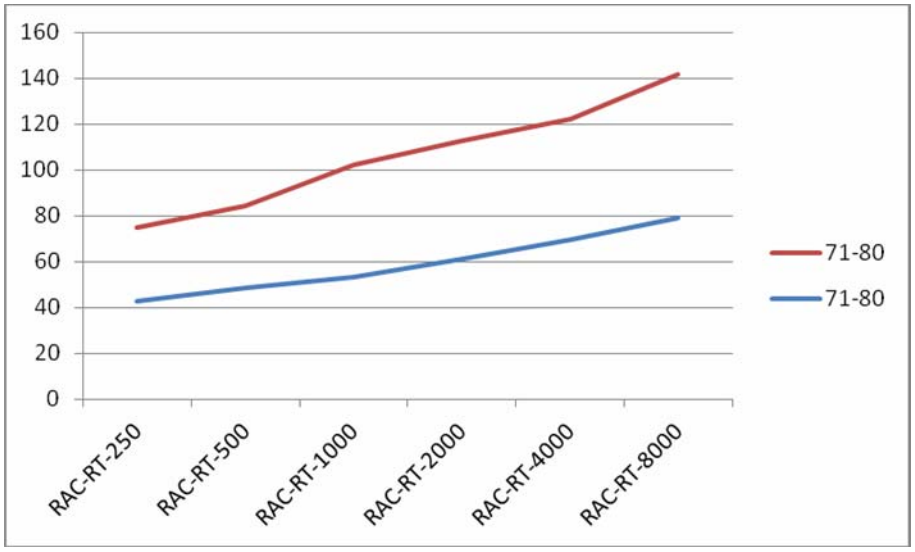
2) 71-80yrs

RIGHT EAR

Table 29– PTA AT 71-80YRS AGE IN MALES RE, (CASES VS CONTROLS)

AGE	RAC-RT-250	RAC-RT-500	RAC-RT-1000	RAC-RT-2000	RAC-RT-4000	RAC-RT-8000
71-80	42.86	48.57	53.21	61.07	69.28	78.92
71-80	31.74	35.89	49.09	51.58	53.04	62.6
p value	0.01	0.003	0.08	0.01	0.006	0.001

Graph 25– PTA AT 71-80YRS AGE IN MALES RE, (CASES VS CONTROLS)



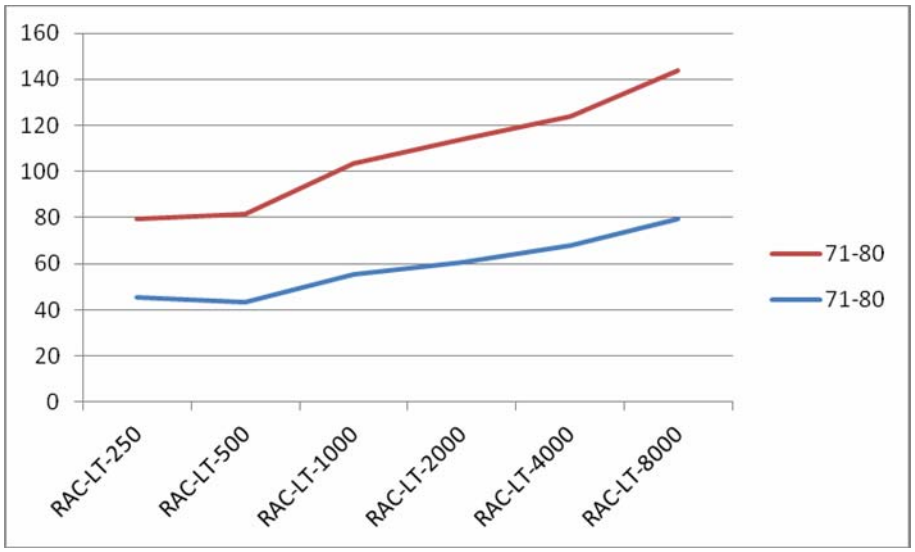
INFERENCE – On comparing the pure tone averages between the diabetic and the non diabetic males in the age group of 71-80yrs in the right ear it was seen that, both the groups had hearing loss which increased progressively with the increase in the frequencies. The difference in the hearing loss observed in all the frequencies ranged from 10 -16dB loss except at 1000Hz which was around 4dB loss. Similarly this group also showed a statistically significant p values in most of the frequencies.

LEFT EAR

Table 30– PTA AT 71-80YRS AGE IN MALES LE, (CASES VS CONTROLS)

AGE	RAC-LT-250	RAC-LT-500	RAC-LT-1000	RAC-LT-2000	RAC-LT-4000	RAC-LT-8000
71-80	45.35	43.57	55.36	60.5	68.21	79.64
71-80	34.35	38.26	48.35	53.39	55.65	64.32
p value	0.007	0.04	0.03	0.01	0.01	0.001

Graph 26– PTA AT 71-80YRS AGE IN MALES LE, (CASES VS CONTROLS)



INFERENCE –

On comparing the pure tone averages between the diabetic and the non diabetic males in the age group of 71-80yrs in the left ear it was seen that, both the groups had hearing loss which increased progressively with the increase in the frequencies. The difference in the hearing loss observed in 4KHz & 8KHz frequencies ranged from 13 -15dB, whereas in the low frequencies the hearing loss ranged from 5-7dB.A statistical significant p values was seen in most of the frequencies. (mainly at 4KHz and 8KHz)

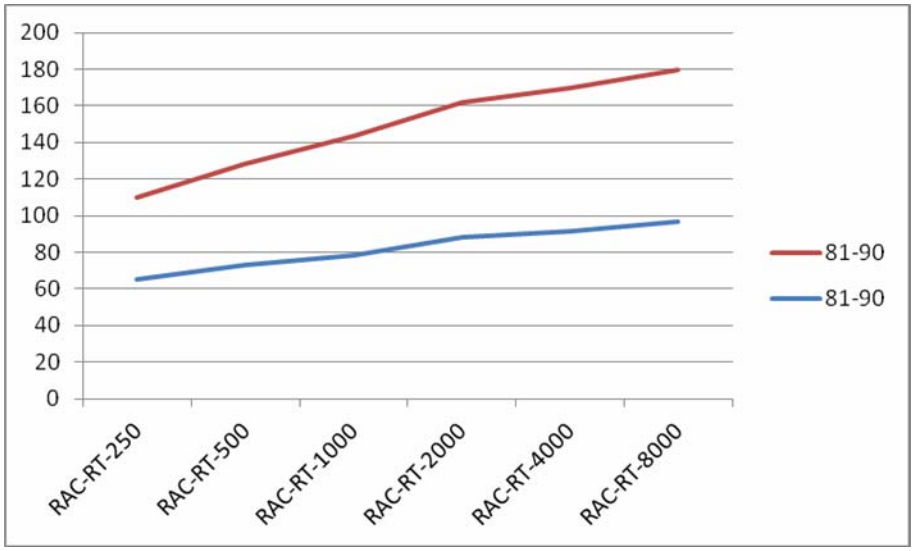
3) 81-90yrs

RIGHT EAR

Table 31– PTA AT 81-90YRS AGE IN MALES RE,(CASES VS CONTROLS)

AGE	RAC-RT-250	RAC-RT-500	RAC-RT-1000	RAC-RT-2000	RAC-RT-4000	RAC-RT-8000
81-90	65.53	73.33	78.56	88.23	91.67	96.67
81-90	52.46	55.35	65.08	74.54	78	83
p value	0.003	0.001	0.01	0.008	0.007	0.002

Graph 27– PTA AT 81-90YRS AGE IN MALES RE, (CASES VS CONTROLS)



INFERENCE -

On comparing the pure tone averages between the diabetic and the non diabetic males in the age group of 81-90yrs in the right ear it was seen that, both the groups had hearing loss which increased progressively with the increase in the frequencies. The difference in the hearing loss observed at all the frequencies ranged from 13 -15dB. A significant p values was seen in almost all the frequencies <0.05.

LEFT EAR

Table 32– PTA AT 81-90YRS AGE IN MALES LE,(CASES VS CONTROLS)

AGE	RAC-LT-250	RAC-LT-500	RAC-LT-1000	RAC-LT-2000	RAC-LT-4000	RAC-LT-8000
81-90	68.33	73.33	81.66	88.33	98.33	96.66
81-90	42.46	52.49	64.65	76.88	80.64	84.5
p value	0.001	0.001	0.001	0.03	0.001	0.007

Graph 28– PTA AT 81-90YRS AGE IN MALES LE,(CASES VS CONTROLS)

INFERENCE - On comparing the results between the diabetic and the non diabetic males in the age group of 81-90yrs in the left ear it was seen that, both the groups had a significant hearing loss which increased progressively with the increase in the frequencies. The difference in the hearing loss noted at 250 and 500 Hz ranged from 20- 25dB, whereas in high frequencies it was 13-15dB. Similar to the right ear significant p values was observed in all the frequencies.

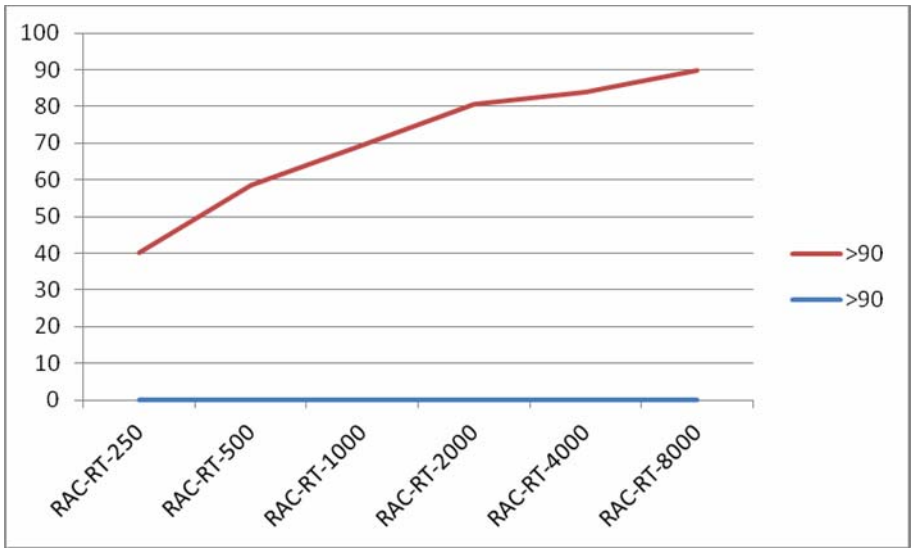
4) >90yrs

RIGHT EAR

Table 33– PTA AT >90YRS AGE IN MALES RE,(CASES VS CONTROLS)

AGE	RAC-RT-250	RAC-RT-500	RAC-RT-1000	RAC-RT-2000	RAC-RT4000	RAC-RT-8000
>90	0	0	0	0	0	0
>90	40	58.5	69.5	80.5	84	90

Graph 29– PTA AT >90YRS AGE IN MALES RE,(CASES VS CONTROLS)



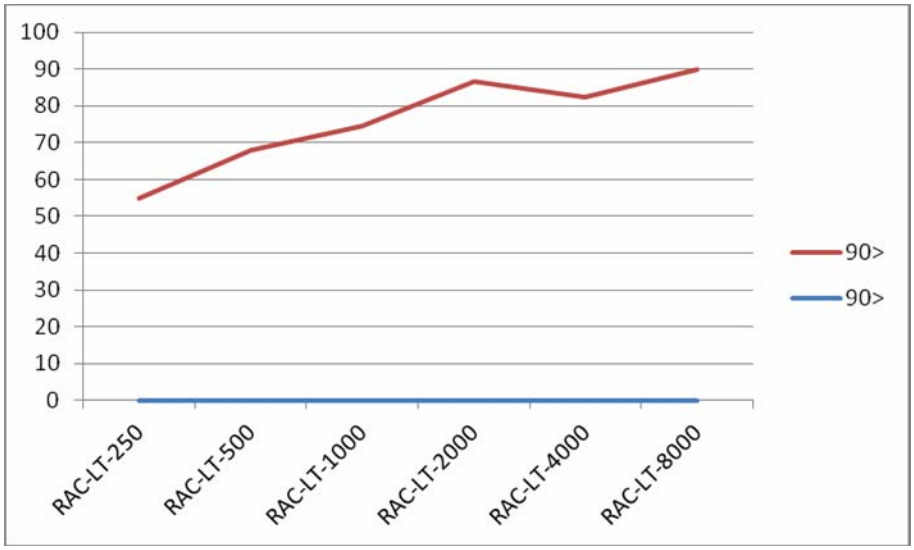
INFERENCE – it was observed that in the non diabetic age group the hearing loss seem to increase progressively with the increase in the age group. However the results could not be compared as there were no cases in the diabetic group studied.

LEFT EAR

Table 34– PTA AT >90YRS AGE IN MALES LE,(CASES VS CONTROLS)

AGE	RAC-LT-250	RAC-LT-500	RAC-LT-1000	RAC-LT-2000	RAC-LT-4000	RAC-LT-8000
90>	0	0	0	0	0	0
90>	55	68	74.5	86.5	82.5	90

Graph 30– PTA AT >90YRS AGE IN MALES LE,(CASES VS CONTROLS)



INFERENCE – it was observed that in the non diabetic age group the hearing loss seem to increase progressively with the increase in the age group. However the results could not be compared as there were no cases in the diabetic group studied.

DISCUSSION

Age-Related Hearing Loss (ARHL) is one of the most common health conditions affecting the elderly individuals.[1] With the ageing both hearing as well as the risk of diabetes mellitus increases. Diabetes mellitus being one of the most common metabolic disorder affects the both the older as well the younger age group to a lesser extent, and is associated with the hearing impairement due to the complications of neuropathy. But the relation between diabetes and hearing is still a controversy.

Diabetes mellitus is known to cause progressive damage to the inner ear and spiral ganglion neurons, thereby reducing the input from the central auditory pathway leading to the hearing impairement.[2] The hearing loss caused by diabetes mellitus would be similar to that of presbycusis but with a more severe loss than expected due to ageing alone [4,37]. Both these condition are known to act synergistically leading to pronounced hearing impairement in elderly.[4] But it is not clear whether the hearing loss among the diabetes is due to ageing effect or due to the accelerated angiopathic and neuropathic effects of diabetes alone.

The present study done was a cross sectional study where an age and sex matched cases (diabetics) were compared with controls (non diabetics) to study the prevalence of hearing loss, the hearing threshold in age wise subgroups and find an association between the duration and severity of hearing loss as well as whether the blood sugar levels and HbA1c have any relationship with the severity of hearing loss.

There is a wide variation in the prevalence rates in the literature ranging from 0-93%[13].This study showed a prevalence rate of 73.58% among the type II diabetes mellitus group and 60% in presbycusis group. This study rates were similar to that of the study done by Malucelli et al where a prevalence rate was found to be 76.6%.[23]

Aonther study done by Rajendran et al, also showed a similar rates (73.3%) to that of our study.[43] Dalton et al in his study also showed a prevalence rates of 74.7%[38]. Another study by Minami et al [44] observed 78.2% of SNHL among diabetic patients. But there are studies which have shown a low prevalence rates too.[12,45, 40] This variation in the results regarding the relation between diabetes and hearing loss could be attributed to the non randomization, poor study design, sample size, methodology, varying standardizing technique and also because of the other confounding factors.

TYPE OF HEARING LOSS

Diabetes in our study was shown to have a bilateral progressive sensorineural hearing loss affecting all the frequencies with most of them having mild to moderate hearing loss. However it was found that a significant difference was noted at higher frequencies mainly 4KHz and 8KHz between diabetics and pure presbycusis. This was consistent with the study results as that found in Mitchell et al.[46]

Malucelli Augusto et al [23] found a similar result to that of ours where he found diabetes mellitus to affect more of higher frequencies. Other various studies which have observed a similar pattern of SNHL among diabetics are Bainbridge and Hoffman et al,[47] Rajendran et al,[43] Cullen and Cinnamond.[9] But in contrast to our study Tay and Irwin reported that diabetes affected low and mid frequencies than higher frequencies.[13] Whereas Maia and Campos[49], showed no significant relation was found between the two. This was also supported by another study done by Axelson and Fagerberg,[10] who found no correlation between diabetes mellitus and hearing loss was seen.

Also in our study there were few cases which had shown asymmetry in the hearing loss between both the ears and 2 cases had shown mixed hearing loss even

after ruling out all the middle ear pathologies. This could be attributed that in early stages atherosclerotic changes affected more in one particular ear than the other but as age progresses both the ears were affected symmetrically. Also the genetic and environmental changes would predispose hearing loss early in some individuals than those without it.

RELATION BETWEEN AGE AND HEARING LOSS

A) Among Diabetics

Age and its relation to the hearing loss has been studied in the past and has varied results. In our study we found that there was a significant association between the increase in the age and hearing loss in diabetes. We found both the groups had progressive increase in the incidence of SNHL as the age increased but was significantly higher in diabetes group. The difference in the pure tone average seen among the different age groups in diabetics varied from 5 to 30dB in both the sexes. Also, another finding which we observed in our study was lesser age individuals had lesser severity of hearing loss compared to older age groups where comparatively more severe hearing loss was seen. The results were similar to that of various other studies done in the past[45,48,51,52,56].

However, Mitchill et al [46] showed that the difference in the hearing loss among the different age groups remained consistent and not much significant changes were seen as the age progressed. Similarly a meta-analysis study done by Horikawa et al also showed no significant association between age and hearing loss [4].

2) ***Among Non Diabetics***

In non diabetics also, we observed a similar pattern as that seen in diabetics where increased ageing increased hearing loss. However in 80-90yrs the prevalence rate seems to be higher in control group compared to the cases, this could be attributed to a lesser sample size. The difference in mean pure tone average varied from 3-20dB among the different age groups in males and 5-30dB among females.

Overall diabetes mellitus was found to be associated with increased hearing loss, with an earlier onset of SNHL similar to that of age related hearing loss. However it was seen that presbycusis patients develop hearing loss which was slow and increased progressively with the age. Both these conditions are known to have a similar pattern of hearing loss with diabetes having significantly higher loss. These changes associated with the hearing loss in presbycusis could be due to the various confounding factors like genetic, environmental and degenerative changes that mainly affect the cochlea and the neural components with ageing. Whereas in diabetes the probable etiology could be because of the angiopathic and neuropathic effects apart from the ageing process itself.

GENDER DIFFERENCE

Our study showed a prevalence rate of SNHL between the males and female diabetics, of 46% and 53% respectively. But on quantitatively measuring the mean pure tone average the difference between both the genders no much significant difference was seen in the hearing.

There was only 3-4dB difference between the both the groups (except at 60-70yrs where difference was 10dB). This was similar to that of the studies done by, Axelson and Fagerberg,[10] Colonel and Chamyal,[51] where no gender difference was

seen. An Indian study done by Sharma et al, also showed that there was no much sex difference in hearing loss among the diabetes mellitus patients.[52] However there were few studies which showed male preponderance.[36,54, 55,48] Irwin &Taylor[13] and Rajendran et al [43] in their studies observed that females had higher affiliction for hearing loss compared to males.

DURATION OF DIABETES

A lot of conflicting results have been observed between the duration of diabetes and hearing loss. Our study showed that patients with <10yrs of duration of diabetes had 63% incidence of hearing loss whereas in cases with >10yr of duration the incidence rates was 85%. However in patients with >15yrs of duration no much significant results was seen probably due to a relatively less sample size.

We also observed that as the duration progressed the severity of the hearing loss also increased in most of the cases. In patients with <10yrs of duration, they had either normal (32.2%) or mild (22.5%) with few moderately severe (16%) or severe (9%) hearing loss.

This study showed a consistent results to that of a study done by Mitchell et al [46]. Studies done by Pemmaiaha et al[53] and Mohammad et al [45] also observed a significant relation between duration of diabetes and the hearing loss. However a contradicting results was seen in a number of studies where no association between the both was observed. [9,10,12,55]

FBS and PPBS values

Various parameters have been taken into the consideration to know whether alteration in the blood sugar levels affected the degree of hearing loss. Much of the

results in literature are still debated in this regards. We compared both FBS and PPBS levels in our cases and observed that there was a strong association between the severity of hearing loss and blood sugar levels.

We observed an incidence of SNHL of 60.2% among the patients with <110mg/dl as compared to 83% in patients with >110mg/dl. We was also observed that most of the patients with high FBS and PPBS levels the hearing loss was also comparitively severe (either moderate, moderately severe to severe hearing loss). In contrast patients within the normal range had either normal to mild or moderate hearing loss.

Our results observed, was similar to that of Pallavi Panchu[56] and Sharma et al,[52] where higher the blood sugar levels, more was the severity of the hearing loss. This association could be attributed to severe hyperglycaemia which results in biochemical abnormality that activates polyol pathway and accumulation of sorbital within the neurons thereby reducing the myoinositol content and Na+/ K+ ATP ase activity, along with activation of protein kinase C and advanced glycation products all of which would ultimately leads to the atrophy of the spiral ganglion.[5,27,29,31] Alternatively this could also be due to the acceleration of atherosclerosis within the vessel walls of stria vascularis which in turn may alter the blood supply to cochlea affecting the OHC's and IHC's leading to its death.[5,6,23]

HbA1c and SNHL

HbA1c is generally considered as a more reliable indicator of the glycaemic status of the patient for the preceeding 3 months. Understanding the relation between the HbA1c and its severity has been reported in a number of studies but most of them have contradictory results. In our study there was a positive relation seen between the

HbA1c and the severity of the hearing loss. We found that patients with a good glycaemic control (<8%) the incidence of hearing loss was 62%, when compared to patients with poor glycaemic control (>8%) where it was 89%.

Also with poorer glyceamic control most of the cases had either moderately severe (26%) or moderate (21%) hearing loss with few cases having mild and severe (each being19%) hearing loss . In contrast most of the cases with good glycemic control had either normal (40%) or mild (30%) with few moderate (20%) hearing loss.

Studies done by Pallavi Panchu[56] and Pemmaiah et al,[53] showed a similar relation between glycemic control and SNHL where they concluded in their study that poorer the glycemic status more severe was the hearing loss. Sumathi et al, in her study also showed a strong relationship between HbA1c and hearing loss.[57] However some studies have shown no correlation between the glycaemic control and hearing loss [35,12].

Creatinine values and severity of hearing loss.

Serum Creatinine is one of the strong parameters used to detect the severity of diabetes in a long standing disease as this measures the nephropathic changes which is one of a microvascular complication. In our study patients with serum creatinine levels <1.5mg/dl, had hearing loss which were distributed over all the groups of hearing loss. Also in cases with serum creatinine levels >1.5mg/dl there were approximately similar number of cases which were seen in all the groups of hearing loss. As a result not much could be inferred regarding the serum creatinine and severity of hearing loss. However, a study done by Kakarlapudi et al reported a strong association between serum creatinine levels and hearing loss.[12]

Pure tone threshold averages

Most of the studies done in the past have all been non randomized with very few studies showing age and sex matched comparison. As a result not much data is available regarding the pure tone threshold at various frequencies in different age groups. A comparison of PTA was done between,

a) Diabetic males with Diabetic females
b) Non Diabetic males with Non diabetic females
c) Diabetic males with Non Diabetic males
d) Diabetic females with Non diabetic females

Diabetic Males Compared With Non Diabetic Males

On comparing the mean pure tone threshold average between the diabetic and the non diabetic males, our study showed that males in the diabetic group had significantly higher hearing loss compared to the non diabetics and was bilaterally symmetrical. When the mean pure tone average threshold (averages at 500, 1000 and 2000Hz) was taken in both the groups and compared, the difference between the two groups was high at two age groups (71-80yrs and 81-90yrs) around 8-10dB and 15dB respectively. However in other groups the difference was only 3-5dB.

The study also compared the hearing threshold at various frequencies in both the groups which showed a significant hearing loss noted at all frequencies in diabetic males compared to non diabetic males. We also observed that the difference was more marked in the higher frequencies (4KHz & 8 KHz) in all the age groups except at 71-80 and 81-90yrs where the difference was almost consistent in all frequencies. In 50-60yrs and 61-70yrs the maximum difference seen was approximately 15-18dB at 4KHz & 8KHz, whereas in 71-80yrs the was difference 10-16dB in right and 13-

15dB in left ear, and in 81-90yrs the maximum difference seen was 13-15dB (except 20-25dB at 250HZ.)

Diabetic Females Compared With Non Diabetic Females

When diabetic and non diabetic females were compared at different age groups, we observed that the hearing threshold increased progressively with the increase in the age and was bilaterally symmetrical in both the ears & in both the groups.

When the mean of the pure tone average was taken at 500, 1000 and 2000Hz and compared, the difference between the two groups (61-70yrs and 81-90yrs) was 8-10dB and 15dB whereas in other groups it was only 5dB.

On comparing the two groups at various frequencies we also observed that the hearing threshold increased at all frequencies in diabetic females compared to non diabetic females (except at 50-60yrs) and that the difference in hearing loss was more marked in the high frequencies (at 60-70yrs and 70-80yrs), however in other age groups the difference was almost constant.

At low frequencies the difference in mean PTA varied from 5-11dB, and at high frequencies the difference was 13-19dB in the younger age groups. However, in higher age groups the difference was almost constant at all frequencies. (7-12dB)

Rajendran et al[43] in his study also showed that the maximum difference in the hearing threshold between the age and sex matched diabetics and non diabetics was found to be more in higher frequencies than in the low and mid frequencies .However, in his study the hearing threshold was comparatively low at all frequencies as compared to ours as it had included younger age diabetes patients.

Cullen and Cinnamond also reported that a difference of 5-30dB in the hearing threshold was seen between the diabetics and non diabetics but more so prominent at

4KHz and 8KHz. [9] Similarly Huang et al, in his study also concluded that diabetes is more likely to affect the higher frequencies with an approximately 5-30dB loss.[54]

The possible reason why high frequency sounds were affected more in diabetes than the pure presbycusis could be due to the accelerated atherosclerosis and neuropathic effects of hyperglyceamia. Further with atherosclerosis there is increased thickness of capillary basement membrane in stria vascularis as well as the other arteries supplying the cochlea, thereby decreasing the blood flow to the cochlea, mainly to the basal and middle turn leading to the cell degeneration and loss of high frequency hearing sounds. Alternatively this could also occur because of the various biochemical abnormalities as described above apart from the presbycusis changes alone.

On comparing the results between males and females in diabetic group

In this study when the age matched diabetic males were compared with the diabetic females, no significant hearing loss was seen between. There was only 3-4dB difference between the two groups in all the age distribution, expect at 61-70yrs where it was 10dB. However it was also seen that at higher age groups, females had slightly higher but a non significant hearing loss compared to males. This was similar to that of the results seen in some of the studies where no significant sex difference was seen. [(10,52,57)]

In control group, males had slightly higher hearing loss than the females at different age groups except at 51-60yrs & 81-90yrs. In 61-70yrs and 71-80yrs difference in hearing loss was 8dB and 2dB respectively more for males. When the age and other confounding factors are matched it is shown that there is no such significant difference between the sexes.

CONCLUSION

1. The prevalence of hearing loss in Type 2 diabetic patients was high (73%).

2. There was bilaterally symmetrical progressive hearing loss with right sloping curve seen in both diabetic patients as well as non diabetics, with hearing loss noted at all frequencies, more so in higher frequencies as compared to non diabetic group.

3. The hearing threshold increased linearly with the increase in the age in both the cases and the controls but higher in diabetes group. However no significant difference was seen in the hearing threshold between the diabetic males and females.

4. A significant relationship was observed between HbA1c, blood sugar levels and severity of the hearing loss. Poorer the diabetic control, more severe was the hearing loss.

5. The severity of the hearing loss increased as the duration of the diabetes also increased.

SUMMARY

Diabetes mellitus is an endocrine metabolic disorder affecting the multi organ system. Since the relation between the diabetes and the hearing loss is controversial our study aimed at identifying and determining the prevalence and hearing threshold along with the effects of diabetes on hearing in older individuals. The hearing threshold was evaluated in both the groups using a pure tone audiometer. Study also aimed in identifying if short term blood sugar levels affected the severity of hearing loss.

In our study we found a high prevalence rate of hearing loss among type II diabetes patients, 73.2%. The hearing loss was bilaterally progressive symmetrical, affecting all the frequencies but significant loss mainly at higher frequencies. The severity of hearing loss observed was mainly mild, moderate or moderately severe hearing loss.

Our study also showed no much difference in the hearing threshold between the both the sexes.

The glycaemic control among the diabetics had shown a significant relationship between the severity of hearing loss. Poor glycaemic control was associated with higher degree of hearing loss. Our study also showed higher blood sugar levels (FBS >110mg/dl and PPBS >140mg/dl) had a stronger relationship with hearing loss. In contrast serum creatinine levels in our study had showed no relation with the severity of hearing loss.

Duration of diabetes was seen to affect the severity of hearing loss. Lesser the duration of diabetes, less severe was the hearing loss compared to patients with longer

duration of diabetes (>10yrs) which was associated with higher degree of hearing loss.

Our study evaluated the hearing threshold between the diabetic and the non diabetic males, where patients with diabetes had higher degree of hearing loss compared to non diabetics. The pure tone averages at different frequencies had shown to affect in all the frequencies but a maximum difference was seen at higher frequencies mainly, 4KHz and 8KHz. However at higher age groups the hearing loss was noted at all frequencies and the difference in the hearing threshold was almost constant.

Whereas diabetic females was compared with non diabetics, the difference was significant in higher frequencies only at 61-70yrs and 71-80yrs of age, but in other age groups the hearing loss was seen to affect all the frequencies with a consistent loss.

However certain conclusions with respect to attributability and confounding factors, is beyond the scope of this study as we could not truly rule out the effects of presbycusis in older diabetics. It is felt strongly that a larger studies with lesser variables and more randomized studies must be done to evaluate the relation between diabetes and hearing loss.

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ANNEXURES

PROFORMA OF THE CASE SHEET

1. PERSONAL DETAILS

Name Age Sex - M/F

Address DOA DOD

Occupation Hospital no :

2. PRESENTING COMPLAINT

Decreased hearing Y/N

R/L/B

Duration

Tinnitus Y/N

R/L/B

Duration

Giddiness Y/N

Facial weakness Y/N

R/L/B

3. HISTORY OF PRESENT ILLNESS

A) Hard of hearing-

a) Onset : insidious/sudden

b) R/L/B

c) Progressive/ Non progressive

d) Fluctuant/ Non fluctant

B) Tinnitus: Y/N

Subjective/ Objective

Intermittent/ continous

Roaring/ whistling

C) Vertigo: Y/N

Nature : occasional/continous/postural/nausea/vomiting

Precipitating factors

Aggravating factors

Relieving factors

D) Associated symptoms-

Diabetes mellitus- Y/N

Duration

Associated – decreased vision- Y/N Excessive weight gain- Y/N

Neuritis – Y/N Polyuria- Y/N

Medication – OHAs / insulin

4) **PAST HISTORY-**

h/o any exposure to noise trauma

h/o ototoxic drug intake

h/o smoking

h/o Hypertension/ cardiac disease

h/o any previous ear discharge/ surgery

5) **FAMILY HISTORY**

h/o hearing loss in family

6) **PERSONAL HISTORY**

Loss of appetite: Y/N Disturbed sleep : Y/N Alcohol: Y/N

Bowel/bladder disturbance: Y/N Smoking : Y/N

7) **GENERAL PHYSICAL EXAMINATION**

Temperature - pulse BP

Built: poor/ medium/ well built pallor: Y/N Icterus: Y/N Clubbing: Y/N

Koilonychia : Y/N Nutritional status: poor/satisfactory odema: Y/N

Lymphadenopathy: Y/N

8) **ENT EXAMINATION-**

SL.NO	**EAR**	**RIGHT**	**LEFT**
1	PINNA: scardeformity	Y/N Y/N	Y/N Y/N
2	POSTAURICULAR AREA: scar/ Obliterated / accentuated		
3	PRE AURICULAR		
4	External auditory canal		
5	Tympanicmembrane Colour Surface Position mobility		
6	TFT : RINNES TEST WEBERS TEST ABC TEST	+ve/ -ve R/L/C Y/N	+ve/-ve R/L/C Y/N
7	VESTIBULAR FUNCTION Spontaneous nystagmus Fistula test Positional test	 Y/N Positive/negative Positive/negative	 Y/N Positive/negative Positive/negative

9) **NOSE AND PNS-**

A) External Nose and Vestibule

Anterior rhinoscopy

Posterior rhinoscopy

Tenderness PNS : Y/N

10) **EXAMINATION OF THROAT**

Oral cavity and oropharynx

IDL

Neck

11) Systemic Examination

Cardio vascular system

Respiratory system

Abdomen

Central nervous system

12) **DIAGNOSIS-**

13) **INVESTIGATION**

PURE TONE AUDIOMETRY- SNHL/ MHL

FBS-

PBS

Urine sugar levels-

HbA1c

B.urea

S.creat

Ophthalmic findings

PTA (RE) PTA (LE)

Inference – Right ear

Left ear

KEY TO MASTER CHART

R right

L left

B both

C central

RE right ear

LE left ear

FBS fasting blood sugar

PPBS post prandial blood sugar

HbA1c glycosylated Haemoglobin

AC air conduction

BC bone conduction

Hz hertz

NR No response

DB decible

Complications :

A retinopathy

B nephropathy

C neuropathy

D diabetic foot

N normal

Severity of hearing loss:

A- Normal

B- Mild hearing loss

C- Moderate hearing loss

D- Moderately severe hearing loss

E- Severe hearing loss

F- Profound hearing loss

Treatment :

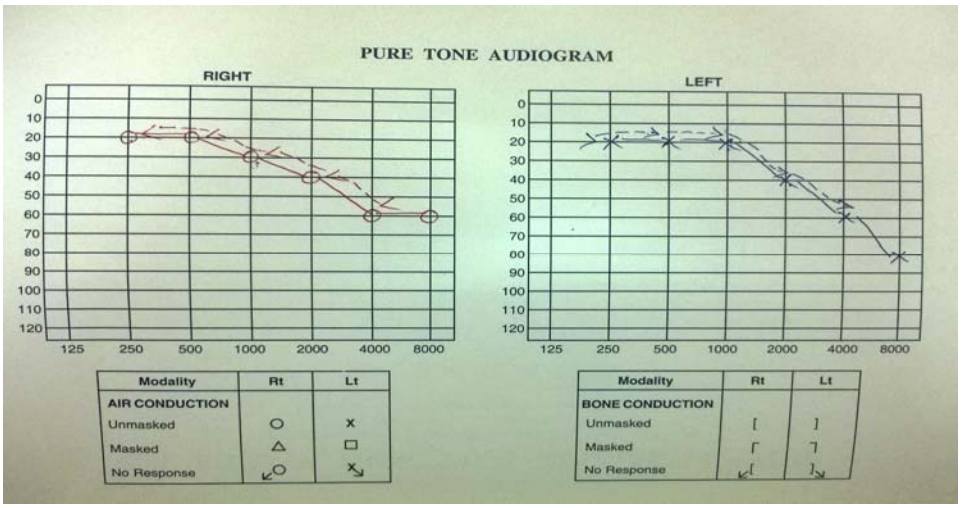
I insulin

O oral hypoglycemic drugs

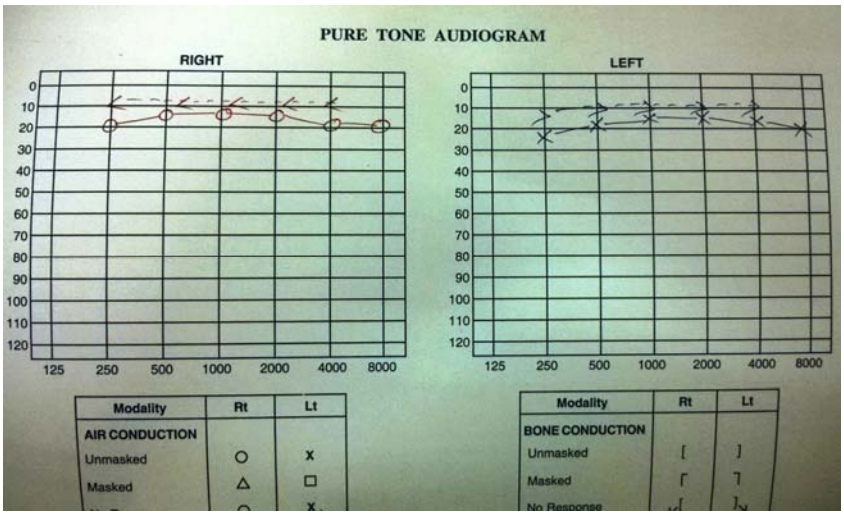
I+O both insulin and oral hypoglycemic drugs

PHOTOGRAPHS

AUDIOMETER



AUDIOGRAM OF DIABETIC PATIENT



AUDIOGRAM OF NORMAL PATEINT

MASTER CHART : CONTROLS

SL NO	NAME	AGE	SEX	IP NO	HL LOSS	R/L/B	TINNITUS	GIDDINESS	RINNES R	RINNES L	WEBERS	ABC R	ABC L	AC IN RIGHT EAR						AC IN LEFT EAR						BC IN RIGHT EAR						BC IN LEFT EAR						PTA RE	PTA LE	INFERENCE RT EAR	INFERENCE LT EAR
														250	500	1000	2000	4000	8000	250	500	1000	2000	4000	8000	250	500	1000	2000	4000		250	500	1000	2000	4000				
1	RAMDAS	67	M	877490	Y	B	N	N	P	P	L	R	R	45	40	40	45	60	65	30	35	30	25	40	50	40	40	40	45	50		30	30	30	20	30		40.6	30	B	B
2	MARI RAO	70	M	872862	Y	B	N	N	P	P	C	R	R	30	40	35	40	60	70	30	40	30	40	55	60	30	30	30	40	50		30	40	30	40	50		38.3	38.3	B	B
3	YAMAKKA	62	F	874925	Y	R>L	Y	N	P	P	C	N	N	15	15	20	25	40	50	10	10	15	10	30	45	10	10	10	5	25		5	5	5	5	15		20	11.6	A	A
4	HUSSAINAMMA	75	F	874915	Y	B	N	N	P	P	L	R	R	25	50	60	40	60	65	20	40	50	50	60	70	20	35	40	40	50		20	35	45	50	55		50	46.6	C	C
5	CHANDAMMA	80	F	880314	Y	B	Y	Y	P	P	R	R	R	80	85	85	90	100 NR	110NR	90	80	85	90	100	110NR	70	75	85	85	100NR		90	80	80	90	90		86.6	85	E	E
6	CHINAPPA	90	M	879588	Y	B	B	Y	P	P	L	R	R	60	65	70	75	80	80	55	65	65	75	80	90	60	60	65	70	75		55	60	60	70	85		70	68.3	D	D
7	VENKATASWAMY	70	M	876801	Y	B	N	Y	P	P	L	R	R	85	90	80	70	60	60	50	65	70	70	80	85	60	70	70	60	50		50	45	60	70	75		80	70	D	D
8	VENKATASWAMY	68	M	876807	Y	B	N	N	P	P	R	R	R	40	55	50	55	60	75	40	50	55	60	65	80	40	55	40	50	70		35	50	50	60	60		53,3	55.6	C	C
9	THIPPAYA	68	M	876797	N	N	N	N	P	P	C	N	N	5	5	10	10	15	30	51	10	10	15	10	15	5	5	10	10	10		5	10	10	10	10		8.3	11.6	A	A
10	NARAPPA	85	M	872208	Y	B	Y	Y	P	P	C	R	R	25	25	30	40	55	30	20	30	40	55	50	30	20	20	40	50	50		20	30	40	50	50		40	40	B	B
11	VENKATAPPA	80	M	880138	Y	B	Y	N	P	P	L	R	R	40	50	55	50	60	60	35	45	45	50	75	90	30	50	55	50	80		30	30	35	50	70		51.6	46.6	C	C
12	NARASIMHAPPA	80	M	880299	Y	B	Y	N	P	P	L	R	R	40	55	55	60	80	90	45	55	55	60	70	80	30	50	60	60	80		35	40	40	40	60		60	56.6	D	D
13	MUNIYAMMA	64	F	880331	N	N	Y	N	P	P	C	N	N	25	25	20	20	25	45	20	25	25	30	45	40	15	20	20	25	25		20	20	25	30	40		25	23.3	A	A
14	THIMAKKA	60	F	872853	N	N	N	N	P	P	C	N	N	20	20	20	30	40	45	20	15	15	20	40	50	10	25	15	25	40		20	20	20	25	50		23.3	16.6	A	A
15	NANJUNDAPPA	75	M	872864	Y	B	Y	N	P	P	L	R	R	70	75	80	90	100	110NR	70	75	80	100	110NR	100NR	55	60	70	80	90		50	65	70	100	100		81.6	85	E	E
16	LAKSHMAMMA	74	F	872854	Y	B	Y	N	P	P	C	R	R	30	30	35	45	65	80	30	30	30	40	60	80	20	25	20	40	40		20	30	25	30	50		36.6	33.3	B	B
17	LAKSHMAMMA	60	M	872852	Y	B	Y	N	P	P	C	N	N	25	20	35	45	65	55	20	15	35	50	65	60	20	20	20	35	50		15	15	30	35	50		33.3	33.3	B	B
18	NARAYANAPPA	75	M	874921	Y	B	N	N	P	P	R	R	R	40	40	45	50	55	70	45	45	50	55	70	70	30	30	40	40	45		45	35	40	50	60		45	50	C	C
19	VENKATARAMAPPA	74	M	874922	Y	B	Y	N	P	P	C	R	R	25	30	35	40	50	55	25	30	35	40	60	60	20	30	35	50	55		20	20	25	50	50		36.6	35	B	B
20	SUBAMMA	62	F	876790	Y	R>L	Y	N	P	P	L	R	N	20	25	20	20	30	30	20	15	20	20	40	50	20	25	20	20	30		10	15	15	15	20		21.6	18.3	A	A
21	PRAMILA	53	F	872866	N	N	N	N	P	P	C	N	N	10	15	20	25	25	30	10	10	15	15	20	25	5	10	10	15	20		5	5	10	10	15		20	13	A	A
22	SHAMALAPPA	96	M	856324	Y	B	Y	N	P	P	L	R	R	75	80	85	85	95	105	65	75	80	85	90	90	70	80	75	75	80		60	70	70	70	80		81.6	78.3	E	E
23	VENKATARMANAPPA	74	M	879590	Y	B	N	N	P	P	R	R	R	30	30	35	40	50	65	30	40	45	45	50	60	30	30	40	45	50		35	40	45	50	55		35	43.3	B	B
24	MUNIYAPPA	65	M	874908	N	N	N	N	P	P	C	N	N	5	10	15	15	20	25	10	10	15	15	15	20	5	5	10	10	10		5	5	10	10	15		13.3	13.3	A	A
25	PEER SAT	82	M	874923	Y	B	Y	N	P	P	L	R	R	60	45	50	65	60	65	40	40	40	45	45	35	45	45	40	55	55		60	40	40	45	45		53.3	41.6	C	C
26	CHINAPPA	87	M	879588	Y	B	Y	N	P	P	L	R	R	85	85	80	85	90	100NR	65	70	75	80	80	85	70	75	70	80	80		55	60	70	70	80		83.3	78.3	E	E
27	SANGAPPA	71	M	879589	N	N	N	N	P	P	C	N	N	20	20	25	30	30	30	15	20	25	30	40	30	10	20	20	25	25		15	20	25	30	40		25	25	A	A
28	HANUMAIAH	80	M	874920	Y	L>R	Y	N	P	P	L	R	R	80	80	80	85	90	90	85	85	85	90	90	80	70	70	80	80NR	90		80	80	75	75	90		81.6	83.3	E	E
29	BABASAB	75	M	882885	Y	B	Y	N	P	P	L	R	R	35	35	40	40	50	60	30	35	35	40	45	50	30	30	40	40	50		30	30	50	60	70		36.6	35.6	B	B
30	VEKATESHAPPA	85	M	889247	Y	B	Y	Y	P	P	L	R	R	100	90	95	110	110	90NR	50	60	80	80	90	95	100	100	110	110NR	110NR		80	90	90	100NR	100NR		106	73.3	F	D
31	CHOWDAPPA	76	M	880308	Y	B	Y	N	P	P	R	R	R	40	40	45	55	60	75	45	40	40	55	60	60	40	45	45	55	55		40	45	45	55	50		46.6	45	C	C
32	MUNIVENKATAPPA	73	M	880309	Y	B	Y	N	P	P	L	R	R	60	80	80	90	90	100	65	75	80	75	80	85	50	55	65	60	60		60	55	65	70	65		83.3	76.6	E	D
33	HANUMANTHAPPA	78	M	880361	Y	B	N	N	P	P	L	R	R	35	40	40	45	60	30	35	30	40	40	45	60	30	35	40	40	60		20	30	30	40	40		43.3	40	C	C
34	ASWATHAPPA	61	M	874918	Y	B	Y	N	P	P	R	R	R	40	40	45	50	60	75	40	40	40	45	50	60	30	40	45	50	50		30	40	45	40	50		45	42	C	C
35	SURAPPA	65	M	882774	N	N	N	N	P	P	C	N	N	15	20	20	15	15	20	25	30	25	20	35	60	0	5	15	15	10		10	20	15	20	25		18.3	25	A	A
36	VENKATARAMAPPA	68	M	880292	Y	B	N	N	P	P	L	R	R	20	30	30	35	40	45	20	20	30	40	50	65	15	20	30	35	40		20	20	20	30	50		31.3	30	B	B
37	MAGILLAPPA	65	M	882623	Y	B	Y	Y	P	P	L	R	R	40	35	35	40	60	60	35	35	35	40	60	65	30	30	35	40	50		35	25	35	40	60		36.6	36.6	B	B
38	SUNDARAJ	85	M	913528	Y	B	Y	N	P	P	L	R	R	30	35	35	40	65	60	20	25	40	45	60	20	30	35	35	40	60		20	30	35	30	50		36	35	B	B
39	BASANNA GOWDA	78	M	922480	Y	B	Y	N	P	P	L	R	R	40	40	30	35	45	55	40	30	30	35	40	50	30	35	40	40	60		40	40	40	40	50		35	31.6	B	B
40	GANGAPPA	68	M	872871	Y	B	Y	N	P	P	R	R	R	55	45	50	55	60	80	50	50	55	60	70	80	50	45	45	50	50		45	40	55	60	70		50	55	C	C
41	KADIRAPPA	57	M	872866	Y	B	Y	N	P	P	R	N	N	35	45	45	55	70	90	30	35	50	55	70	80	30	40	40	50	70		30	30	40	50	60		48.3	46.6	C	C
42	HUAASIANAMMA	79	F	874915	Y	B	Y	N	P	P	L	R	R	65	65	70	80	80	100	50	55	60	70	80	85	50	55	60	70	80		50	50	50	60	75		71.6	61.6	E	D
43	NARAYANAPPA	68	M	861090	Y	B	N	N	P	P	R	R	R	20	15	15	10	15	20	10	10	15	10	10	15	25	30	30	35	35		25	25	30	35	35		15	10	A	A
44	VARADARAJU	78	M	868887	Y	B	Y	Y	P	P	L	R	R	90	95	100	90	85	100NR	50	60	65	70	70	85	90	90	85	80	90		50	55	65	60	80		95	65	F	E
45	VEKATESHAPPA	64	M	886516	Y	B	Y	N	P	P	C	N	N	10	15	20	40	60	50	10	10	20	25	60	70	5	10	10	30	50		5	5	15	20	50		25	18.3	A	A

MASTER CHART : CONTROLS

46	SUBBAMMA	77	F	880306	Y	B	N	N	P	P	R	N	R	30	40	45	55	60	70	40	40	45	45	60	70	20	30	35	50	50	70	70	70	80	80	46.6	43.3	C	C
47	YELLAPPA	65	M	862455	Y	B	N	N	P	P	L	R	R	20	30	35	35	40	60	20	20	30	35	40	50	20	30	25	25	30	20	20	30	40	40	33.3	28.3	B	B
48	VENKATESH	68	M	625834	N	N	Y	N	P	P	C	N	N	40	20	25	25	35	80	25	25	25	10	15	60	30	20	15	15	20	20	10	10	10	10	23.3	13.3	A	A
49	RAJASHEKAR	76	M	895208	Y	B	N	N	P	P	R	R	R	45	55	55	45	50	65	45	55	55	45	50	70	40	40	40	45	40	40	50	50	40	50	51.6	51.6	C	C
50	RANGAMMA	78	F	880324	Y	B	N	N	P	P	R	R	R	85	90	85	85	90	100	95	95	100	100	95	90	80	90	80	75	80	90	85	90	90	90	86.6	90	E	E
51	RAMAIAH	98	M	723502	Y	B	Y	N	P	P	R	R	R	85	90	85	90	100	110	95	95	100	100	95	90	80	80	75	75	80	85	85	90	90	90	86.6	85	E	E
52	MUNIKUDIRAPPA	70	M	880328	Y	B	Y	N	P	P	R	R	R	30	30	40	40	50	65	20	30	30	40	50	75	30	40	45	40	40	40	45	50	50	60	36	36.6	B	B
53	YARAGAPPA	74	M	893845	Y	B	N	N	P	P	L	R	R	60	60	60	65	65	75	60	60	65	65	65	75	50	55	50	60	60	50	55	60	60	55	61.6	63.6	D	D
54	KADIRAPPA	54	M	872861	N	N	N	N	P	P	C	N	N	15	10	20	15	15	30	15	20	10	10	10	15	10	5	5	10	10	10	15	5	5	10	15	13.5	A	A
55	RAMAPPA	80	F	910646	Y	B	Y	N	P	P	R	R	R	60	75	75	65	65	65	60	70	75	70	70	80	50	70	65	65	60	50	70	60	70	60	71.6	71.6	E	E
56	NAGARAJAIH	61	M	874152	N	N	N	N	P	P	C	N	N	10	15	20	20	10	20	10	10	10	15	10	15	10	15	15	20	20	20	20	20	20	25	18.3	11.6	A	A
57	KAMALAMMA	65	F	875135	N	N	N	N	P	P	C	N	N	20	20	25	20	25	25	25	20	20	25	25	20	15	15	10	15	15	5	10	20	15	15	18.3	21.6	A	A
58	SEETHARAMASHETTY	68	M	871062	N	N	N	N	P	P	C	N	N	20	15	20	20	20	50	20	20	30	35	40	55	10	10	10	10	15	15	15	25	30	35	18.3	28.3	A	A
59	AKKAMMA	75	F	875497	N	N	Y	N	P	P	C	N	N	15	10	5	10	20	40	10	15	10	20	20	25	5	5	5	5	15	5	10	10	10	15	8.3	15	A	A
60	SONAPPA	75	M	903661	Y	B	Y	Y	P	P	R	R	R	20	30	35	55	65	70	30	35	40	50	65	75	20	25	30	50	60	25	30	35	45	55	40	41.6	B	B
61	ALLISAB YARAGU	64	M	893845	N	N	Y	Y	P	P	C	N	N	10	10	15	25	35	40	15	15	20	25	30	35	5	5	10	20	30	10	10	15	20	25	16.4	14	A	A
62	NANJAPPA	65	M	901832	Y	B	Y	N	P	P	L	R	R	60	65	75	75	75	100	40	45	60	75	80	90	30NR	55	65	65	70	35	40	55	70	70	70	60	D	D
63	RAMAPPA	76	M	910646	Y	B	N	N	P	P	R	R	R	35	50	60	60	65	75	45	60	70	75	75	75	25	40	50	50	55	35	50	55	60	65	56.6	68.3	D	D
64	DEVRAJAPPA	76	M	905502	Y	L	Y	N	P	P	R	R	R	20	20	25	25	50	10	15	20	20	25	30	40	15	20	20	30	50	20	15	20	30	30	26.6	25	A	A
65	CHINAKAMMA	65	F	915566	N	N	Y	N	P	P	C	N	N	20	15	25	25	30	30	20	25	25	25	30	30	15	15	20	20	20	20	20	20	20	20	21.6	31.6	A	B
66	JEEVAMMA	70	F	910897	N	N	Y	Y	P	P	C	N	N	30	20	25	25	30	60	20	20	25	25	40	40	25	20	20	30	55	25	20	30	30	40	23.3	23.3	A	A
67	SONAMMA	65	F	916508	N	N	Y	N	P	P	C	N	N	20	15	15	10	15	30	25	15	15	15	20	25	15	10	10	10	10	15	10	10	10	15	13.3	15	A	A
68	JAYARAM REDDY	75	M	916800	Y	B	Y	N	P	P	C	N	N	30	35	40	40	55	55	35	40	45	50	55	55	25	30	30	35	40	30	35	40	40	45	38.3	40.6	B	B
69	PRABHU	58	M	917079	Y	B	N	N	P	P	R	R	R	20	25	30	35	40	55	20	25	30	40	55	70	15	20	50	60	60	15	20	40	60	65	30	31.6	B	B
70	CHOWDAMMA	65	F	917223	Y	B	N	N	P	P	R	R	R	25	30	50	60	65	70	30	35	50	55	60	60	20	30	40	50	60	25	30	40	50	50	46.6	46.6	C	C
71	GOVINDA REDDY	55	M	918161	Y	B	N	N	P	P	C	N	N	20	20	25	25	40	60	20	20	15	20	40	30	20	20	25	30	50	20	30	30	30	40	23.3	23	A	A
72	MUNISWAMY	65	M	918044	Y	B	Y	N	P	P	C	N	N	30	40	40	45	45	50	35	35	40	45	50	50	20	30	30	40	40	30	30	30	45	50	41.6	40	C	C
73	RAJU	65	M	918193	Y	B	N	N	P	P	C	N	N	25	20	20	25	40	45	20	25	25	30	35	35	20	25	30	30	30	15	20	20	25	30	21.6	25.6	A	A
74	CHIKAMMA	60	F	917254	Y	B	Y	Y	P	P	C	N	N	30	30	35	40	40	50	35	35	40	40	45	55	30	35	30	40	40	30	40	40	45	45	35	26.6	B	B
75	NARAYANAMMA	88	F	919462	Y	B	Y	N	P	P	R	R	R	90	90	95	110	100NR	10ONR	90	95	100	110NR	11ONR	100NR	80	80	90	100	100NR	80	90	90	80NR	80NR	98.3	101.6	F	F
76	LAKSHMAMMA	65	F	892242	N	N	Y	N	P	P	C	N	N	20	25	25	20	25	20	20	15	25	25	20	30	15	10	10	15	20	15	10	20	20	15	23.3	21.6	A	A
77	KESHAVA REDDY	66	M	893197	Y	B	Y	N	P	P	C	N	N	25	30	40	40	50	70	20	30	35	35	40	70	20	30	40	45	55	25	30	35	40	70	40	35	B	B
78	SUBRAMANYA	77	M	878202	N	N	Y	N	P	P	C	N	N	15	20	20	25	35	70	20	20	20	25	40	40	10	15	15	25	35	15	15	15	20	25	21.6	21.6	A	A
79	NARAYANAPPA	76	M	861090	Y	B	Y	N	P	P	R	R	R	20	25	40	45	60	85	20	20	50	55	85	75	15	25	40	45	55	15	15	45	50	75	36.6	40.6	B	B
80	VANAPPA	53	M	885171	Y	R>L	Y	N	P	P	C	N	N	20	20	25	25	30	35	15	15	20	20	25	25	15	20	20	20	25	10	10	15	15	20	23.3	18.3	A	A
81	VENKATESHAPPA	53	M	886516	N	N	N	N	P	P	C	N	N	15	15	15	20	25	25	20	20	20	30	40	60	10	10	15	20	20	15	20	25	30	40	16.6	25.3	A	A
82	RAJASHEKAR	55	M	895208	N	N	Y	N	P	P	C	N	N	15	20	20	25	30	60	20	25	20	30	40	80	10	20	15	25	30	15	20	30	40	60	21.6	25	A	A
83	SUNDARAJ	55	M	913528	Y	B	N	N	P	P	C	N	N	15	20	20	15	25	30	20	20	20	25	35	40	10	15	15	15	20	15	15	15	20	30	18.3	21.6	A	A
84	SUJATHAMMA	50	F	912875	Y	B	N	N	P	P	L	R	R	85	90	95	100	100	100	65	70	75	80	80	95	70	80	90	90	100	35NR	60	65	70	70	95	75	F	E
85	MALARMANI	73	F	895195	N	N	Y	N	P	P	C	N	N	20	20	25	30	50	70	15	20	20	25	45	65	15	20	25	30	50	10	15	15	20	40	25	21.6	A	A
86	SREERAMAPA	71	M	891620	Y	B	N	N	P	P	R	N	R	25	35	40	50	60	70	20	40	35	40	55	65	20	30	40	45	45	20	30	40	50	55	40.6	38.3	B	B
87	BASANNA GOWDA	62	M	922480	Y	B	N	N	N	N	R	R	R	50	65	65	75	75	85	70	70	70	75	100	100NR	40	50	55	65	75	70	65	65	70	80NR	70	70.5	E	E
88	BHAGAVATHI	67	F	899651	N	N	N	Y	P	P	C	N	N	20	15	20	20	25	25	20	20	25	30	30	35	15	15	15	15	20	15	15	15	20	25	18.3	23.3	A	A
89	MUNIYAPPA	50	M	904945	Y	L	Y	N	P	P	R	N	R	15	20	15	15	20	20	20	15	20	20	25	40	15	20	15	15	10	10	10	15	10	10	16.6	18.3	A	A
90	NARASIMHAPPA	55	M	800738	Y	R	N	N	P	P	L	R	R	35	35	60	60	60	75	30	35	35	55	65	65	30	35	60	60	60	25	30	35	45	45	51.6	41.6	C	C

MASTER CHART : CASES

SL NO	NAME	AGE	SEX	IP NO	HL LOSS	R/L/B	TINNITUS	GIDDINESS	DM DURATION	TREATMENT	COMPLICATIONS	AC IN LEFT EAR						AC IN RIGHT EAR						BC IN RIGHT EAR					BC IN LEFT EAR					PTA RE	PTA LE	INFERENCE RE	INFERENCE LE	FBS	PPBS	HbA1c	UREA	CREAT
												250	500	1000	2000	4000	8000	250	500	1000	2000	4000	8000	250	500	1000	2000	4000	250	500	1000	2000	4000									
1	YASMEEN TAJ	60	F	920874	2Y	B	Y	N	15Y	I	A	25	45	55	60	60	85	35	40	50	65	70	80	25	40	50	50	55	30	40	40	50	50	53.3	51.6	C	C	161	266	9.1	36	0.75
2	MUNIYAMMA	80	F	881205	10Y	B	N	N	20Y	I + O	A+B	45	50	55	55	70	75	55	50	50	50	85	100NR	40	50	40	60	70	45	40	40	40	70	53.3	50	C	C	178	288	8.6	30	0.8
3	SONNE GOWDA	74	M	882298	N	N	N	N	5Y	O	N	40	45	60	45	60	90	35	40	50	55	55	90	30	40	55	40	60	30	40	40	50	50	50	48.3	C	C	106	130	7.5	39	1.2
4	RAJAMMA	80	F	910612	8Y	B	Y	Y	15Y	I+O	A	80	80	80	95	110	100NR	75	80	70	70	75	100NR	75	75	80	90	110	75	70	70	65	75	85	73.3	E	E	238	390	8.8	19	0.49
5	GODAVARI BHAI	64	F	872784	N	N	N	N	8Y	I	N	25	25	30	30	30	40	25	30	35	35	40	45	20	25	20	20	25	20	30	30	35	35	28.3	33.3	B	B	142	301	7.4	33	1.1
6	NANJUNDAPPA	64	M	878860	N	N	N	N	10Y	O	N	25	25	25	25	40	55	20	20	20	25	40	75	20	25	20	20	30	20	15	15	20	30	25	21.6	A	A	130	306	6.9	19	1.2
7	NASARULLA KHAN	57	M	864002	N	N	N	N	10Y	O	N	35	35	35	30	45	55	30	35	30	35	45	50	30	25	25	30	40	30	30	25	30	40	33.3	33.3	B	B	220	383	10	29	1.1
8	SRINIVAS	69	M	886783	2M	B	Y	N	8Y	I	N	30	30	40	40	70	80	30	25	25	40	60	75	20	30	30	40	70	25	25	20	30	50	36.6	30	B	B	65	145	7.2	34	0.4
9	MANGAMMA	54	F	926288	N	N	N	N	12Y	O+H	N	30	25	20	20	40	20	30	20	25	15	40	20	25	20	20	35	35	25	20	20	10	35	21.6	20	A	A	150	280	6.3	27	0.6
10	ZAIDA BEE	90	F	880342	15Y	B	Y	Y	35Y	O+H	A	50	60	70	70	90	100	60	60	65	70	80	85	50	55	60	60	90	55	50	60	60	80	66.6	65	D	D	147	292	10.4	26	0.61
11	AMARNARAYANA	53	M	917209	N	N	N	N	2Y	O	N	25	20	25	30	40	50	25	30	30	50	55	55	20	25	20	30	35	20	25	25	30	40	28.3	30	B	B	110	192	6.4	18	0.64
12	HANUMAPPA	70	M	923061	1Y	N	N	N	10Y	O	N	35	40	45	65	65	85	35	40	55	65	70	80	30	30	45	60	60	30	35	50	60	70	50	53.3	C	C	156	243	7.9	25	1.3
13	VWNKATESH	54	M	932815	N	N	N	N	6Y	O	N	25	35	35	40	40	80	20	30	40	40	45	70	15	15	30	30	30	15	20	30	30	30	36.6	36.6	B	B	110	140	7.6	32	0.9
14	MAHASHWAI	52	F	894470	N	N	N	N	6Y	O	N	30	30	30	15	15	25	20	25	25	15	20	30	20	25	20	15	10	20	20	15	15	20	25	21.6	A	A	110	123	6.2	24	0.6
15	VENKATAMMA	72	F	835773	2Y	B	N	N	7Y	O	A	45	45	50	60	65	80	50	55	40	55	65	90	30	30	40	45	45	30	50	30	45	45	51.6	50	C	C	153	163	7.4	30	1.8
16	HANUMANTHAPPA	78	M	865019	5Y	B	Y	N	10Y	O	A+B	80	80	95	90	95	90	85	80	85	90	95	100	80	70	90	90	90	80	70	80	90	90	88.3	85	E	E	185	333	13.6	85	3.3
17	MALASHREE	58	F	886779	3Y	B	Y	Y	10Y	I	C	55	60	65	70	75	90	50	60	65	65	85	90	50	60	60	60	70	50	60	60	55	75	65	63.3	D	D	130	128	8.4	22	0.8
18	SUSHILAMMA	56	F	926596	N	N	N	N	2Y	O	N	35	35	40	60	65	90	40	55	55	60	75	85	30	35	35	50	50	30	55	55	60	75	45	56.6	C	C	112	254	7.7	11	0.5
19	KUPPA SWAMY NAIDU	61	M	848793	1Y	B	Y	N	8Y	I	B	30	45	50	55	70	80	35	40	45	55	70	85	30	40	50	50	60	30	40	40	50	60	50	46.6	C	C	273	328	12	78	1.4
20	RATHNAMMA	75	F	867016	Y	B	Y	N	15Y	I	A	30	45	55	55	70	90	40	40	50	55	70	80	30	40	45	55	65	30	70	80	80	85	51.6	48.3	C	C	136	238	7.9	17	1
21	SEETHAPPA	59	M	919863	3M	B	N	N	6Y	O	B	30	35	45	60	70	75	35	35	40	65	65	70	30	30	40	50	55	30	35	35	50	55	46.6	46.6	C	C	190	240	8	68	1.7
22	PRAKASH GUPTA	61	M	875894	3Y	N	N	N	9Y	O	N	15	20	25	25	40	80	20	15	15	20	45	80	15	15	20	20	35	15	15	10	20	40	23.3	16	A	A	116	193	6.4	21	0.87
23	RAJANNA	55	M	902002	6M	B	N	N	5Y	O	N	25	25	35	75	85	90	25	30	30	70	85	90	25	25	30	70	75	20	25	30	65	80	45	43.3	C	C	79	185	12	27	0.6
24	VASANTHAMMA	60	F	920582	4Y	B	Y	Y	20Y	I	A	30	30	25	25	25	40	35	35	25	20	20	45	25	25	20	20	25	30	30	25	20	20	26.6	26.6	B	B	257	357	11	11	0.5
25	JAYAMMA	57	F	9309059	N	N	N	N	5Y	O	N	25	30	25	35	30	40	25	30	40	40	60	75	20	30	30	30	35	20	30	35	40	55	31.6	36.6	B	B	210	186	8.2	20	0.9
26	VENKATAMMA	52	F	940629	N	N	N	N	12Y	O	B	25	25	30	30	35	35	35	35	30	40	50	80	20	20	25	25	30	10	15	15	20	10	28.3	31.6	B	B	118	179	10	89	3.6
27	MOGILAPPA	64	M	884731	N	N	N	N	10	I	N	10	10	15	15	20	20	10	10	25	25	20	25	10	10	10	5	20	10	10	15	15	20	13.3	20	A	A	82	132	7	22	0.8
28	NARAYANAPPA	71	M	875850	2Y	B	N	N	7Y	I	R	45	45	60	65	70	85	45	40	65	70	75	80	40	40	50	55	60	40	40	60	55	65	56.6	58	D	D	114	189	7	38	0.74
29	AKTHARUNNISSA	60	F	921778	5Y	B	Y	N	10Y	I	A+B	20	25	30	25	30	40	35	40	40	35	55	70	20	20	25	25	30	30	40	40	30	50	26.6	38.3	B	B	292	243	8	51	1.2
30	YASHODAMMA	72	F	907002	N	N	N	N	8Y	O	A	35	40	50	55	55	70	35	45	50	50	55	75	30	30	45	45	60	30	30	45	40	55	48.3	48.3	C	C	140	206	7.4	16	0.4
31	SEETHAPPA	77	M	919863	Y	B	Y	N	5Y	I	B	45	55	65	65	70	90	40	50	65	60	75	90	40	50	55	50	60	40	50	50	50	60	61.6	58.3	D	D	190	246	8.9	68	1.7
32	VIJAYAKUMAR	62	M	916011	Y	B	Y	N	15	I	A	25	30	35	35	40	45	20	35	35	40	45	50	20	30	30	35	40	20	35	35	35	40	33.3	36.3	B	B	202	260	11	15	5
33	RAMAKRISHNAPPA	65	M	927529	Y	B	Y	N	2Y	O	A	75	75	80	85	90	110	60	70	75	80	100	100NR	65	65	70	75	80	60	70	70	75	80	80	75	E	E	156	250	12.2	31	2.7
34	SAMAPANGI RAAIAH	88	M	928602	N	N	N	N	10	O	B	75	75	80	85	90	100NR	60	70	75	75	80	100NR	65	65	70	75	80	60	70	70	75	80	80	73.3	E	E	212	290	11	137	2.3
35	NATARAJ	66	M	918892	Y	B	Y	N	12Y	O+I	D	40	40	50	55	70	90	45	50	55	55	75	90	40	40	50	50	60	40	50	55	60	70	48.3	53.3	C	C	323	250	12	32	0.69

MASTER CHART : CASES

36	ADHI NARAYANA	65	M	945341	Y	B	Y	N	20Y	I	A+B	40	40	55	60	75	90	40	45	65	65	75	85	35	35	65	50	70	35	40	65	65	70	61.6	58.3	D	D	177	247	9.1	65	1.4
37	CHIKKA VENKATESHAP	55	M	945282	N	N	N	N	6Y	I	B	25	25	20	30	30	60	20	20	25	30	35	50	20	25	20	30	30	15	20	20	25	35	25	25	A	A	258	207	7.8	22	0.9
38	RUKKMINIYAMMA	68	F	940986	N	N	N	N	6M	O	N	15	15	20	25	25	40	15	20	20	25	30	40	15	10	20	15	20	15	20	20	20	20	20	21.6	A	A	87	182	6.3	20	0.6
39	RATHNAMMA	80	F	921903	Y	B	Y	N	7Y	O+I	A	80	85	95	90	95	100	85	90	90	95	100	100NR	80	85	85	90	90	80	80	85	90	90	90	91.6	F	F	102	184	7.8	12	0.3
40	GOWRAMMA	57	F	946328	N	N	Y	N	5Y	I	B	20	25	35	40	45	55	20	25	25	35	40	45	15	15	20	40	40	15	20	20	30	40	33.3	28.3	B	B	256	256	7.2	20	0.8
41	RATHNAMMA	58	F	929104	N	N	N	N	12Y	O+I	N	25	30	35	35	45	50	20	20	30	35	45	55	20	20	30	35	45	15	20	30	20	40	33.3	28.3	B	B	145	272	6.8	23	0.62
42	SIDDAMMA	55	F	710644	Y	B	N	N	15Y	O+I	A	35	40	55	55	70	80	35	45	65	60	75	85	30	40	65	65	70	35	45	65	65	70	50	56	C	C	227	262	8.6	18	0.7
43	GOWRAMMA	56	F	946754	N	N	N	N	2Y	O	N	10	10	15	20	25	25	15	10	15	15	20	25	10	10	10	20	25	15	10	15	10	20	15	13.3	A	A	125	284	7.4	18	0.6
44	VIJAYALAKSHMI	58	F	945468	N	N	N	N	3.5Y	O	N	40	45	55	70	70	85	45	40	50	65	70	85	40	40	50	70	65	45	40	45	60	70	56.6	51.6	C	C	215	356	8	10	0.63
45	SRINIVAS	50	M	928183	Y	B	Y	N	15Y	I	A+C	25	25	45	65	70	20	20	20	40	65	75	20	20	25	35	60	70	20	35	60	70	70	45	41.6	C	C	196	333	8.7	46	1.1
46	SHANMUGAM	68	M	934965	Y	L>R	Y	N	7Y	O+I	B+C	45	55	60	75	90	100	40	55	75	80	95	110	45	50	60	70	85	40	55	70	75	85	63.3	70	D	D	318	329	9.6	71	1.1
47	DODDANAYANATH	68	M	918856	N	N	N	N	5Y	O	C+D	15	20	25	25	50	65	10	20	25	30	45	60	15	20	25	40	65	10	20	25	30	40	23.3	25	A	A	95	209	6.9	31	1.2
48	MUNIVEERAPPA	65	M	946710	N	N	N	N	16Y	O+I	A+C	15	20	35	40	40	50	10	20	30	40	40	60	10	20	35	40	40	10	20	30	30	40	31.6	38.3	B	B	278	280	9.1	29	0.8
49	GOVINDAPPA	50	M	945896	N	N	N	N	3M	O+I	N	10	15	10	15	20	25	15	15	10	25	25	40	10	15	10	15	20	15	15	10	20	20	13.3	16.6	A	A	193	213	7	44	0.7
50	SYED ABDUL AZZEZ	75	M	929144	Y	B	Y	Y	15Y	O+I	B+C	30	45	45	70	75	90	35	45	45	65	75	100	30	45	45	60	75	30	40	45	65	70	53.3	51.6	C	C	98	221	9.6	82	2.9
51	KUNDALI VENKTRAPPA	75	M	945948	Y	B	N	N	5Y	O	N	80	80	85	90	95	95	80	85	85	95	100	100NR	80	80	80	85	90	80	80	85	95	100	85	88.33	E	E	162	308	8.6	22	0.9
52	ERGAPPA	65	M	945682	Y	R>L	N	N	25Y	O+I	B	65	60	75	85	90	90	60	65	70	80	90	90	60	60	70	80	90	60	50	70	70	80	73.3	71.6	E	E	261	440	9.24	67	2
53	VENKATAREDDY	55	M	926442	Y	B	Y	N	5Y	O+I	B	70	85	85	90	95	100	65	75	80	95	100	110NR	70	80	80	85	90	60	75	80	90	90	86.6	83.3	E	E	383	337	11.9	127	5.5
54	SEETHAPPA	65	M	932341	N	N	N	N	5Y	O	N	60	65	70	75	85	90	65	65	70	75	90	100	60	60	70	70	80	60	60	70	70	80	70	70	D	D	234	242	12.6	27	0.7
55	NARAYANASWAMY	78	M	922738	Y	B	Y	N	15Y	I	A	30	35	40	40	45	60	35	35	40	45	55	60	30	30	40	40	40	30	30	30	40	50	40	40.6	B	B	153	165	8.8	18	0.6
56	VENKATAREDDY	85	M	926444	Y	B	Y	N	10Y	O	N	75	85	90	100	110NR	100NR	80	85	95	100	110	11ONR	70	80	90	100	100	80	80	80	90	90	91.6	93.3	F	F	202	246	8.3	10.6	0.8
57	MUNISHAMAPPA	65	M	883899	N	N	N	N	5Y	O+I	B	15	20	20	25	30	35	10	15	25	25	30	35	15	20	20	20	30	10	15	20	25	30	21.6	21.6	A	A	74	130	6.9	53	1.7
58	NAGARAJ RAO	71	M	851080	Y	B	Y	N	15Y	O+I	A	50	55	55	60	90	100	45	45	55	70	85	100NR	50	50	50	70	80	40	40	50	70	80	56.6	56.6	D	D	274	269	11.4	24	0.97
59	MUNISHAMI	70	M	922181	N	N	N	N	8Y	O+I	A	8	10	15	15	20	40	55	15	10	15	20	35	10	15	15	15	30	10	10	10	15	30	13.3	16.6	A	A	208	167	8	39	0.6
60	MUNIYAPPA	60	M	918063	Y	R>L	N	N	6Y	O	A	40	50	65	80	90	95	50	55	65	75	85	90	40	45	60	75	90	45	50	60	70	80	65	65	D	D	343	282	12.6	22	0.82
61	POOBHALAN	58	M	917592	N	N	N	N	1Y	O	N	10	15	20	25	30	40	15	15	20	40	45	50	10	15	20	25	30	15	10	15	30	35	20	25	A	A	320	270	10	35	1.1
62	YARAPPA	76	M	936071	N	N	N	N	2M	O	N	15	15	10	25	30	35	10	15	15	20	35	40	15	15	10	20	20	10	10	15	20	30	16.6	16.6	A	A	142	158	6.7	28	0.6
63	SATHYANARAYANA	74	M	938033	N	N	N	N	8Y	O	B	25	30	30	40	45	60	20	20	25	30	40	55	20	25	25	30	35	15	20	20	30	35	33.3	25	B	B	169	283	8	22	0.8
64	MOZAMIL PASHA	60	M	930039	N	N	N	N	4Y	O	B	10	10	15	20	25	25	10	15	15	20	25	25	10	10	10	15	20	10	10	15	15	20	15	16.6	A	A	106	155	8.6	30	0.3
65	ERAPPA	65	M	936686	N	N	N	N	6M	O	N	25	30	40	45	60	65	20	20	25	40	45	50	20	20	30	35	50	20	20	20	30	40	38.3	28.3	B	B	132	180	7.2	27	0.5
66	SHANKAR REDDY	73	M	936509	N	N	N	N	2Y	O	N	25	30	25	35	75	85	20	25	30	30	70	85	20	20	35	70	80	20	20	20	65	80	45	43.3	C	C	140	196	6.3	28	1.1
67	JAI DEV	72	M	924828	Y	B	N	N	16Y	I	A	55	60	70	75	80	90	50	55	55	70	80	90	50	55	70	70	75	45	50	50	70	75	68.3	60	D	D	326	407	11.4	23	1.1
68	KRISHNAREDDY	84	M	902560	Y	B	Y	N	8Y	O	N	50	60	65	80	85	90	50	55	65	80	85	90	40	55	65	80	80	50	55	60	70	80	68.3	65	D	D	106	172	8.8	28	0.4
69	YALASAPPA	68	M	929866	N	N	N	N	6Y	O	N	15	20	20	30	40	50	10	15	15	30	45	60	15	20	20	20	30	10	10	10	25	35	23.3	20	A	A	118	165	8	23	0.9
70	ANNAIAHA	76	M	9282253	Y	B	N	N	20Y	O+I	N	45	45	50	55	75	80	40	40	50	55	60	75	40	50	50	60	60	30	40	40	50	50	50	48.3	C	C	227	276	9	23	0.7
71	NARAYANA SWAMY	75	M	927384	N	N	N	N	12Y	O	D	15	20	25	30	40	55	10	15	30	40	45	60	10	20	20	25	40	10	10	25	30	40	25	28.3	B	B	255	398	10.6	35	0.6
72	SEENAPPA	85	M	927384	Y	B	Y	N	10Y	O	A+B	70	75	80	85	90	100	75	80	85	85	100	110NR	70	70	70	80	80	70	70	80	80	90	80	83.3	E	E	306	358	11	34	0.8
73	NARASIMHAPPA	67	M	927998	Y	B	N	N	10Y	O+I	N	50	60	70	75	75	90	55	60	70	75	75	90	50	60	70	70	70	50	60	65	70	70	68.3	68.3	D	D	240	350	12.8	28	0.6

MASTER CHART : CASES

74	CHOWDAPPA	59	M	927568	N	N	N	N	2Y	O+I	N	25	30	35	30	45	60	15	25	30	35	50	65	20	30	30	30	45	15	15	25	30	45	31.6	30	B	B	118	350	7.6	17	1
75	SHANKAR REDDY	77	M	925880	Y	B	N	Y	2Y	O+I	N	60	65	70	70	85	90	50	55	60	65	65	80	50	60	60	70	65	45	50	60	60	55	70	60	D	D	286	240	10	22	1.4
76	NARAYANAMMA	80	F	797441	Y	B	Y	N	22Y	O	A+B	50	55	55	60	70	85	40	45	55	60	65	70	40	50	50	60	60	40	45	50	55	65	56	53.3	C	C	118	306	12	36	2.3
77	ZAYADA BEE	83	F	880342	Y	B	Y	N	6Y	O	A+C	80	80	90	95	100	100NR	85	85	90	95	100	110NR	70	75	80	90	90	80	80	85	90	85	88.3	90	E	E	286	348	9.6	18	0.4
78	MUNIYAMMA	67	F	912978	Y	B	Y	N	30Y	O+I	A+B+C	40	45	55	55	70	80	40	45	55	55	65	70	40	45	55	60	60	40	50	50	60	60	55	51.6	C	C	306	284	9	22	0.8
79	NANJAMMA	79	F	837148	Y	B	N	N	6Y	O	N	50	50	55	60	75	80	40	45	45	50	55	60	40	50	55	55	70	40	45	45	45	50	55	46.6	C	C	160	204	11.3	48	1.9
80	VASANTHAMMA	60	F	920582	Y	B	Y	Y	6Y	O	A	25	40	45	50	60	90	25	30	45	50	55	80	20	40	40	50	55	20	30	40	50	50	45	41.6	C	C	186	390	8.7	12	0.3
81	MUNIYAMMA	80	F	881295	Y	B	Y	Y	6Y	O	A	15	20	25	30	40	80	10	20	25	40	45	60	10	20	20	25	35	10	20	25	30	40	25	28.3	B	B	274	202	9.6	18	0.4
82	MUNISATHNAMMA	70	F	915212	N	N	Y	Y	15Y	I	N	45	45	50	55	60	80	40	40	45	50	55	65	40	40	50	50	60	40	35	35	40	50	50	45	C	C	190	222	8.4	26	0.8
83	LAKSHMAMMA	56	F	909123	N	N	Y	N	4Y	O	N	15	15	30	45	45	70	25	30	35	55	75	80	15	15	30	40	45	20	25	30	50	65	30	40	B	B	128	196	7.5	14	0.4
84	SUBAKKA	52	F	926983	Y	B	N	N	4Y	O	N	10	15	25	20	20	45	20	15	20	15	15	30	10	10	10	10	10	10	5	10	10	10	20	16.6	A	A	148	190	7.9	8	0.2
85	KAMMAMMA	60	F	909119	Y	B	N	N	8Y	O	N	10	15	10	10	15	15	10	15	10	10	10	15	10	15	15	10	15	5	10	10	15	15	11.6	11.6	A	A	162	208	6.8	11	0.6
86	MANGAMMA	55	F	926288	Y	B	N	N	4Y	O	N	25	20	15	20	40	45	15	25	15	30	30	40	15	20	15	20	40	15	25	15	30	30	18.3	23.3	A	A	180	178	7	14	0.7
87	VENKATAMMA	68	F	940029	N	N	N	N	16Y	O	A	15	20	35	60	50	65	15	25	20	30	50	50	15	20	35	60	50	15	15	20	25	50	38.3	26	B	B	135	143	6.8	20	0.4
88	YASHODAMMA	72	F	907002	N	N	N	N	8Y	O	A	15	15	35	35	50	70	15	25	30	40	40	65	10	15	35	35	50	15	15	20	30	40	28.3	31.6	B	B	118	232	7.4	24	0.6
89	BETAMMA	70	F	882543	Y	B	N	N	15Y	O+I	N	20	20	30	40	60	60	20	20	20	40	60	80	10	20	30	40	60	20	20	20	40	60	30	26.6	B	B	156	250	6.9	10	0.4
90	PAPAKKA	53	F	920582	Y	B	N	N	6Y	O	C	65	80	90	105	11O	100NR	85	90	105	105	105	95	50	80	90	105	11O	85	90	105	105	90	91.6	100	F	F	224	190	12.8	28	0.9
91	PADMAMMA	50	F	926983	N	N	N	N	3Y	O	N	20	15	15	15	20	20	25	20	15	15	20	20	10	10	10	15	20	20	20	15	15	20	15	16.6	A	A	146	407	6.4	12	0.3
92	PADMA	78	F	885284	Y	B	N	N	12Y	I	N	65	65	75	75	80	100NR	60	65	75	75	80	100NR	55	55	65	65	80	50	50	60	65	75	71.6	71.6	E	E	358	116	10.4	18	0.7
93	GADAVARI BAI	70	F	872784	Y	B	Y	N	10Y	O+I	A	15	20	30	45	45	70	25	30	35	50	65	75	15	20	20	30	40	20	20	30	40	60	31.6	38.3	B	B	102	209	8.3	12	0.8
94	PAPILAMMA	73	F	932695	N	N	N	N	15Y	O	N	15	20	25	25	55	55	15	20	20	35	55	55	5	10	15	20	55	10	10	20	30	55	23.3	25	A	A	236	256	7.6	8	0.3
95	MANGAMMA	65	F	896574	N	N	N	N	3Y	O	A	10	10	15	15	10	15	5	10	15	15	10	15	10	10	10	15	10	5	10	15	15	10	13.3	13.3	A	A	130	186	6.3	6	1.6
96	CHOWDAMMA	68	F	815312	Y	B	Y	N	6Y	O+I	A	50	50	60	60	80	90	55	60	70	75	85	100NR	40	40	50	50	70	40	50	60	70	75	56	68.3	D	D	158	140	7.2	12	0.9
97	PADMAVATHI	64	F	903223	N	N	Y	N	19Y	I	A+C	15	20	30	60	60	65	15	20	40	55	60	70	10	10	20	50	60	15	20	30	50	60	36.6	38.33	B	B	96	158	7	7	0.2
98	NANJAMMA	74	F	837148	Y	B	N	N	8Y	I	A	10	15	20	25	30	40	15	15	20	40	45	50	10	15	20	25	30	10	15	20	40	45	20	25	A	A	158	136	7	39	0.8
99	MUNNA BHAI	50	F	885285	Y	B	Y	Y	16Y	I+O	A+C	25	30	40	40	60	90	25	30	45	45	55	80	15	30	30	40	55	20	30	40	40	50	36.6	40	B	B	88	140	6.4	27	0.79
100	VENKATAMMA	70	F	881179	N	N	N	N	4Y	O	A+B	30	30	35	45	65	80	30	30	30	40	60	80	20	20	30	40	60	20	20	30	30	50	36.6	33.3	B	B	177	123	7	89	0.68
101	VENKATALAKSHMAMN	70	F	872563	N	N	N	N	2Y	O	N	30	45	45	70	75	90	35	45	45	65	75	100	25	40	40	55	70	30	30	40	55	70	53.3	51.6	C	C	111	192	7.3	24	0.77
102	MUNIRATHNAMA	70	F	915212	N	N	N	N	2Y	O	N	15	15	20	25	40	50	10	10	15	10	30	45	10	10	15	20	40	10	10	15	10	30	20	11.6	A	A	124	138	8	24	0.7
103	YASHODAMMA	72	F	907002	Y	B	Y	N	15Y	O+I	A	5	10	15	15	20	25	10	10	15	15	15	20	5	10	10	15	20	10	10	15	15	15	13.3	13.3	A	A	140	206	10	16	0.4
104	TAMIL SELVI	50	F	922760	N	N	Y	Y	15Y	O+I	A	45	45	50	55	75	80	40	40	50	55	60	75	35	40	50	60	70	30	35	40	45	50	50	48.3	C	C	250	300	9.1	30	0.6
105	DASAMMA	65	F	928924	Y	B	N	N	8Y	I	A	35	40	60	70	75	80	30	40	60	70	70	80	30	35	50	50	75	30	35	50	65	60	55	55	C	C	258	232	8.7	25	0.55
106	AMARAVATHI	56	F	924137	N	N	N	N	2Y	O	N	20	15	20	20	20	50	20	20	30	30	40	55	10	10	20	20	20	20	20	30	30	45	18.3	25	A	A	137	228	7.2	9	0.6