EFFECT OF CIGARETTE SMOKING ON AUDITORY THRESHOLDS

BY

DR. ASHWINI K. SHETTY, MBBS

DISSERTATION SUBMITTED TO THE

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH,

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IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF MEDICINE
IN
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Under the guidance of

DR. KARTHIYANEE KUTTY, MD.

DEPARTMENT OF PHYSIOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE, KOLAR

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DR.KARTHIYANEE KUTTY, MD.

PROFESSOR & HOD, DEPARTMENT OF PHYSIOLOGY,

SRI DEVARAJ URS MEDICAL COLLEGE, KOLAR.

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DEPARTMENT OF PHYSIOLOGY

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SEAL & SIGNATURE OF THE HOD

DR.KARTHIYANEE KUTTY

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DR. ASHWINI K. SHETTY,

POST GRADUATE STUDENT IN THE DEPARTMENT OF PHYSIOLOGY OF SRI DEVARAJ URS MEDICAL COLLEGE TO TAKE UP THE DISSERTATION WORK ENTITLED

"EFFECT OF CIGARETTE SMOKING ON AUDITORY THRESHOLDS"

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LIST OF ABBREVIATION

PTA	-	Pure Tone Audiometer
AC	-	Air Conduction
BC	-	Bone conduction
kHz	-	Kilo Hertz
dB	-	Decibel
SPL	-	Sound Pressure Level
ABR	-	Auditory Brain Stem Response
OAE	-	Otoacoustic emissions
ERP	-	Event related potentials

ABSTRACT

Background and objectives:

Smoking is widespread addiction among youth. There are approximately 120 million smokers in India, about 37% of all men and 55 of all women. Smoking effects all the systems in the body but a little is known about the effects of smoking on hearing thresholds, although link was established 40 years ago, information on the effects of smoking at the cochlear and auditory central nervous system levels has become available recently.

Hearing thresholds can be assessed using several methods like pure tone audiometry , speech audiometry, auditory evoked potentials & otoacoustic emissions. Pure tone audiometry is a simple, inexpensive, qualitative and quantitative procedure, the only disadvantage being subjective.

The present study aims at studying whether cigarette smoking causes any changes in auditory thresholds in smokers as compared to age matched non smoker.

MATERIALS AND METHODS:

The study included 50 male smokers of age group between 20-40 years and 50 age matched male non smokers, who were selected based on inclusion and exclusion criteria. These subjects were recruited from teaching, non teaching staff of Sri Devraj Urs academy of higher education and research and also attenders of the patients coming to R.L.Jalappa hospital. After taking informed consent and data regarding their smoking history (expressed in pack -years), they were subjected to pure tone audiometric evaluation and test results were entered in audiogram. The data thus

obtained was treated with appropriate statistical test like student t test, Pearson correlation.

RESULTS: There was statistically significant difference in auditory air and bone conduction thresholds at all frequencies for both right and left ear. Study also indicates that if a subject has smoked regularly for a period of 2 or more years it induces changes in both auditory air and bone conduction thresholds. Further, it showed that there is positive correlation of pack years of smoking with air conduction thresholds at all frequencies and bone conduction thresholds at higher frequencies (1kHz, 2kHz, 4kHz) in both right and left ears.

CONCLUSIONS: Smoking causes increase in both air conduction and bone conduction thresholds at all frequencies and if a subject has smoked regularly for a period of 2 or more years it induces changes in both auditory air and bone conduction thresholds.

KEY WORDS: Cigarette smoking, auditory thresholds, Pure tone audiometry.

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF DIAGRAMS

INTRODUCTION

Smoking is widespread addiction among youth and is extensively practiced despite of its harmful effects. Smoking is a greater cause of death and disability than any single disease, says the World Health Organisation.

According to WHO figures, smoking is responsible for approximately five million deaths worldwide every year. Tobacco smoking is a known or probable cause of approximately 25 diseases, and even the WHO says that its impact on world health is not fully assessed. By 2020, Tobacco-attributable mortality is projected to increase to 8.4 million approximately 9% of the worldwide mortality burden.[1] There is believed to be 1.1 billion smokers in the world, 800 million of them in developing countries.[2] There are approximately 120 million smokers in India, about 37% of all men and 5% of all women.[3]

Pandemic of smoking-related death and disease is poised to claim a million lives each year in India. Smoking among persons between the ages of 30 and 69 years is responsible for about 1 in 20 deaths of women and 1 in 5 deaths of men in India and that by 2010, smoking will cause about 930,000 adult deaths (annually) in India.[3]In Karnataka too, tobacco use has reached a prevalence of 41% among men &14.9% among women.

In a long-term prospective cohort study done at Zutphen, The Netherlands showed that average cigarette smoking reduced the total life expectancy by 6.8 years, whereas heavy cigarette smoking reduced the total life expectancy by 8.8 years.[4]

Tobacco contains about 4000 chemicals. Many more toxic chemicals are formed when it is burning, including at least 250 chemicals known to be toxic or capable of causing cancer. It is the major cause in diseases like lung cancer, oral cancer, bronchitis and emphysema. Tobacco-related cancers account for about half of all

cancers among men and one-fourth among women. Tobacco consumption has been explicitly linked to high incidence of heart diseases. A little has been known about the effect of smoking on auditory thresholds although link was established over 40 years ago; information on the effects of smoking at the cochlear and auditory central nervous system levels has become available only recently.

A few population based studies have shown relation between smoking and hearing loss.[5,6] An experimental study have concluded that cigarette smoking results in structural modifications of the cochlea and tuba acoustica.[7]

The present study aims at observing the association between smoking and changes in hearing thresholds in a sample population of Kolar. The hypothesis being tested is "Smoking induces changes in auditory thresholds".

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES:

1. To record auditory thresholds in smokers of age group 20-40 years using pure tone audiometer.
2. To compare the auditory thresholds for various frequencies of smokers and age matched non-smokers.
3. To study association of pack years of smoking with changes in auditory thresholds for both AC and BC at different frequencies.

REVIEW OF LITERATURE

A. HISTORICAL REVIEW OF AUDIOLOGY

6th century BC	Pythagoras, a philosopher and a mathematician, reasoned that sound was a vibration in the air.
175 AD	Galen,a Greek physician recognized that nerves transmitted the sensation of sound to the brain.
1200 AD	Recognition of the fact that sounds entered the interior of the ear via the eardrum & excited on its journey to the brain via auditory nerve.
1600 AD	Felix Platter, a physician from Switzerland studied about bones of the ear and commented on the phenomenon of bone conduction; He also concluded that deafness was some times in the brain (sensorineural) & sometimes in cavity of ear(conductive).
1640 AD	Cranial nerves were discovered by Willis of England.
1825 AD	Weber worked with tuning forks for testing hearing and was called "WEBERS TEST".
1855 AD	Rinne tests hearing using tuning fork & calls it "RINNES TEST".
1863 AD	Helmholtz formulated his "Resonance theory of hearing".
1880 AD	Rutherford put forward his "Telephone theory of hearing".

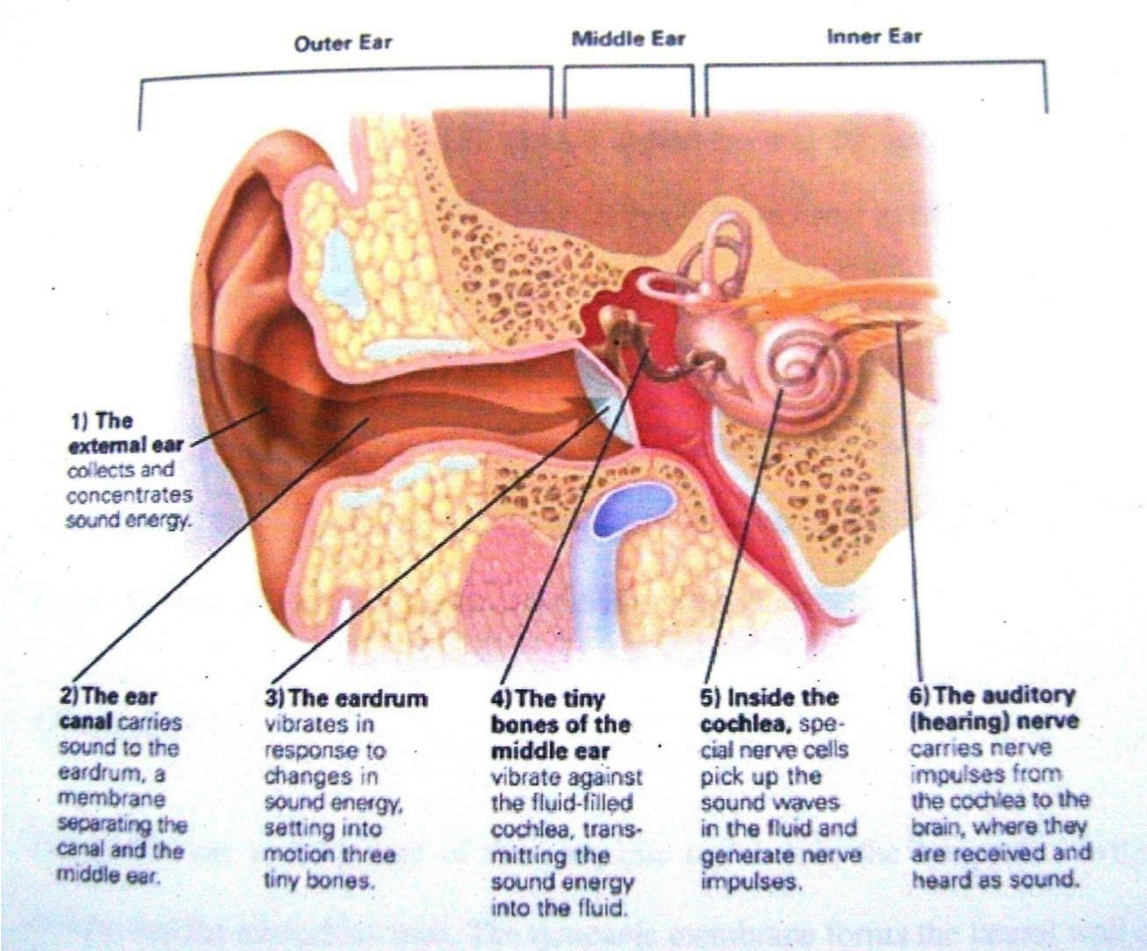
1897 AD	Ist basic model for a pitch range audiometer was developed by Seashore in Iowa.
1928 AD	Georg Von Bekesy, an Engineer for the Hungarian telephone exchange system put forward the "travelling wave theory" .He begins to use large models of cochlea to determine precisely how sounds of different frequencies stimulate the basilar membrane for which he won the "NOBEL PRIZE"
1947 AD	Bekesy introduced a technique for semi automatic audiometry which found application in both standard threshold determination & site of lesion.
1949 AD	Wever put forward "Resonance volley theory of hearing".
1960 AD	Ruben et al recorded cochlear potentials and compound action potentials of vestibulocochlear nerve
1965 AD	Nelson Kiang understands sound encoding in the auditory nerve fibers.
1974 AD	Hecox and Galambos showed the importance of auditory brain stem response (ABR) for screening auditory sensitivity in new borns.
1977 AD	David Kemp discovers that cochlea produces "otoacoustic emissions" & James Hudspeth et al begin detailed experiments to show how hair cells convert sound into electrical impulses.
1980 AD	GSI 66, an automatic screening audiometer was discovered and developed in Australia.

1986 AD	Advancement occurred with the application of digital control circuits to analogue audiometers and hearing aids.
1999 AD	Development of automated pure tone audiometers and hearing aids.
2003 AD	Tympany Inc.designed the otogram ™, an automated pure tone audiometer and speech audiometer.

B. **ANATOMY OF THE EAR:**

The ear is the sensory organ responsible for hearing. It is composed of three parts termed the external ear, the middle ear and the inner ear.

DIAGRAM 1- ANATOMY OF EAR



External ear:

The external ear includes the auricle (pinna) and external auditory canal. The auricle is composed of elastic fibrocartilage covered by perichondrium and skin. The skin

over the lateral aspect of the ear is tightly adherent to the perichondrium whereas on the medial surface, it is more loosely attached. The auricle is attached to the tympanic portion of the temporal bone on the lateral aspect of the skull by extension of the auricular cartilage into the cartilaginous external canal, by three ligaments (anterior, superior, and posterior), by six poorly developed muscles and by its skin and subcutaneous tissue.

The auricle receives sensory innervation from branches of cranial nerves V (auriculotemporal nerve), VII (auricular branch), X (auricular branch) and by the greater auricular nerve from the cervical plexus. Blood supply to the auricle is from the external carotid system mainly by way of the posterior auricular artery and superficial temporal artery. The external auditory canal extends from the concha I cartilage of the auricle to the tympanic membrane. It is approximately 25 mm long in the adult. It courses slightly anteriorly and inferiorly in the adult. The outer l/3rd of the canal is cartilaginous, has thicker skin with subcutaneous tissue and ceruminous glands. The inner 2/3rd is osseous with only epidermis lying on the periosteum of the bony external canal.

Middle ear:

The middle ear is composed of the tympanic membrane, the tympanic cavity, the ossicles and the eustachian tube. The tympanic membrane forms the lateral wall of the middle ear. It is oval in shape, approximately 8 mm wide and 10mm high. The tympanic membrane is about 0.1 mm thick and lies at an angle of 40 degrees in the sagittal plane with the lower aspect displaced medially. It is not flat, rather it is concave medially. The umbo marks the middle of the tympanic membrane and corresponds to the attachment of the tip of the malleus to the tympanic membrane.

Superiorly, the short process of the malleus extends laterally and forms a prominence on the tympanic membrane. From this prominence extend the anterior and posterior malleolar folds. Superior to the folds, lies the pars flaccida (or Shrapnell's membrane), below is the pars tensa. The pars tensa inserts into a bony groove in the tympanic bone termed the tympanic sulcus. The tympanic membrane is composed of three layers, an outer layer of epidermis continuous with the epidermis of the external auditory canal, a middle layer of fibrous tissue (lamina propia) and a medial layer of mucosa. Sensory nerves to the tympanic membrane include the auricular branch of cranial nerve X and the auriculotemporal branch of the mandibular nerve. The blood supply to the tympanic membrane arises from vessels from the external maxillary artery and the stylomastoid artery. The tympanic cavity is a cleft or space within the temporal bone located between the tympanic membrane laterally and the inner ear medially. Posteriorly it communicates with the mastoid air cells and anteriorinferiorly with the eustachian tube orifice. Within the cavity are present the middle ear ossicles, the chorda tympani and a segment of the facial nerve (cranial nerve VII).The middle ear contains three bones or ossicles which transmit sound vibrations to the inner ear. They are from lateral to medial, the malleus, the incus and the stapes. The malleus is firmly attached to the tympanic membrane and the stapes sits within the oval window of the cochlea. Between them lies the incus. The ossicles are held in place by their attachments mentioned above, by their joints with each other, by ligaments and two muscles; the tensor tympani to the malleus and the stapedius muscle to the stapes.

Inner ear:

The inner ear consists of two main parts, the cochlea (end organ for hearing) and the vestibule and semicircular canals (end organ for balance). The inner ear can be thought of as a series of tunnels or canals within the temporal bone. Within these

canals are a series of membranous sacs (termed labyrinths) which house the sensory epithelium. The membranous labyrinth is filled with a fluid termed endolymph; it is surrounded within the bony labyrinth by a second fluid termed perilymph. The cochlea can be thought of as a canal that spirals around itself similar to a snail. It makes roughly 2 1/2 to 2 3/4 turns.[12] The bony canal of the cochlea is divided into an upper chamber, the scala vestibuli and a lower chamber, the scala tympani by the membranous (otic) labyrinth also known as the cochlear duct. . The scala vestibuli and scala tympani contain perilymph. The scala media contains endolymph. Endolymph is similar in ionic content to intracellular fluid (high K, low Na) and perilymph resembles extracellular fluid (low K, high Na). The cochlear duct contains several types of specialized cells responsible for auditory perception. The floor of the scala media is formed by the basilar membrane,the roof by Reissner's membrane. Situated on the basilar membrane is a single row of inner hair cells medially and three rows of outer hair cells laterally. The cells have specialized stereocilia and kinocilia on their apical surfaces. Attached to the medial aspect of the scala media is a fibrous structure called the tectorial membrane. It lies above the inner and outer hair cells coming in contact with their stereocilia. Synapsing with the base of the hair cells are dendrites from the auditory nerve. The auditory nerve leaves the cochlear and temporal bone via the internal auditory canal and travels to the brainstem.

DIAGRAM 2- A CROSS SECTION OF THE COCHLEA ILLUSTRATING THE ORGAN OF CORTI

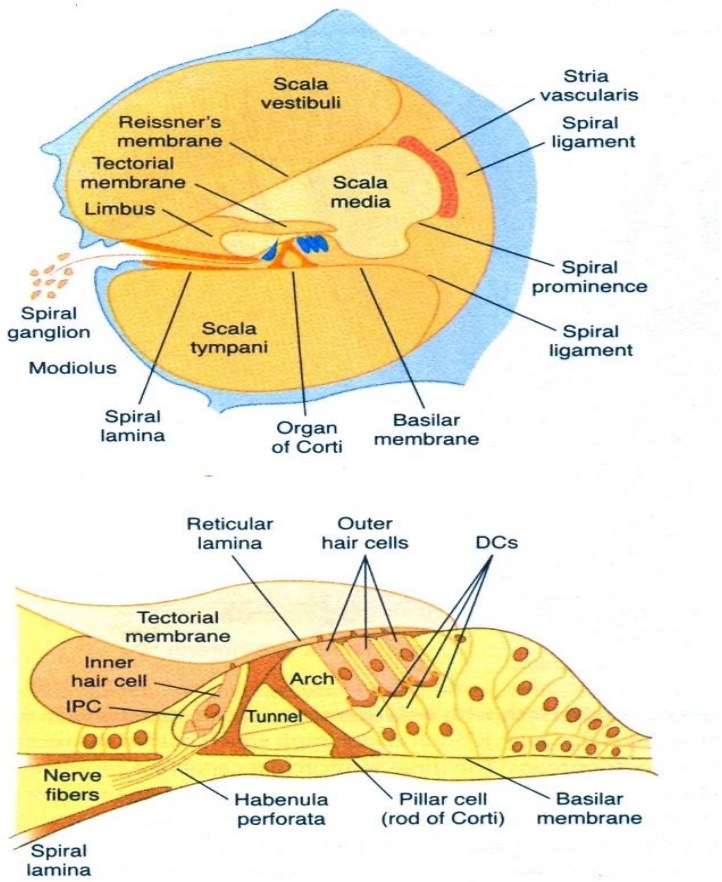


FIGURE 13–4 Top: Cross-section of the cochlea, showing the organ of Corti and the three scalae of the cochlea. Bottom: Structure of the organ of Corti, as it appears in the basal turn of the cochlea. DC, outer phalangeal cells (Deiters' cells) supporting outer hair cells; IPC, inner phalangeal cell supporting inner hair cell. (Reproduced with permission from Pickels JO: *An Introduction to the Physiology of Hearing,* 2nd ed. Academic Press, 1988.)

C. PHYSIOLOGY OF HEARING [8,9,10]

The mechanism of hearing can be broadly divided into:

1. Transmission of the sound from the external ear to the internal ear (conductive apparatus).

2. Development of action potentials in hair cells- Transduction of mechanical energy to electrical impulses (sensory system of cochlea).
3. Conduction of electrical impulses to the brain (Auditory pathway and processing).

1) Transmission of sound from external ear to internal ear:

Sound waves in the external environment that travel through the pinna and the external auditory meatus are transformed by the eardrum and the auditory ossicles into movements of the footplate of the stapes. These movements set up waves in the fluid of the inner ear. The action of the waves on the organ of corti generates action potentials in the nerve fibers. Thus the ear converts sound into action potentials in the auditory nerves. In response to the pressure changes produced by sound waves on its external surface, the tympanic membrane moves in and out. The membrane therefore functions as a resonator that reproduces the vibrations of the sound source. The auditory ossicles thus function as a lever system that converts the resonant vibrations of the tympanic membrane into movements of the stapes against the perilymph filled scala vestibuli of the cochlea. This system increases the sound pressure that arrives at the oval window, because the lever action of the malleus and incus multiplies the force 1.3 times and area of the tympanic membrane is much greater than the area of the footplate of the stapes. Conduction of sound waves to the fluid of the inner ear via the tympanic membrane and ossicles is called ossicular conduction. The movements of the footplate of the stapes set up a series of traveling waves in the perilymph of the scala vestibuli. The distance from the stapes to the point of maximum height varies with the frequency of the vibrations initiating the wave.

High pitched sounds generate waves that reach maximum height near the base of the

cochlea; low-pitched sounds generate waves that peak near the apex.

2) Development of action potential in hair cells:

The stimulus to depolarize the sensory cell is the deflection of the stereocilia. Inside of a hair cell has the normal environment with an intracellular potential of -45mv. The endolymph has an unusually high potassium level, low sodium and a strongly positive potential of +80mv. The endolymph therefore is a special fluid with the composition which is maintained by the stria vascularis. The potential difference of 130 m V across the hair cell is responsible for the influx of potassium towards the negatively charged cell interior when the cell membrane becomes leaky.

DIAGRAM 3- SHOWING THE POTENTIALS ACROSS THE VARIOUS COMPARTMENTS OF THE COCHLEA

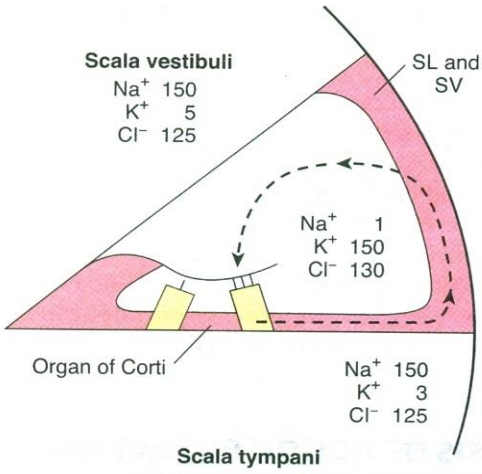
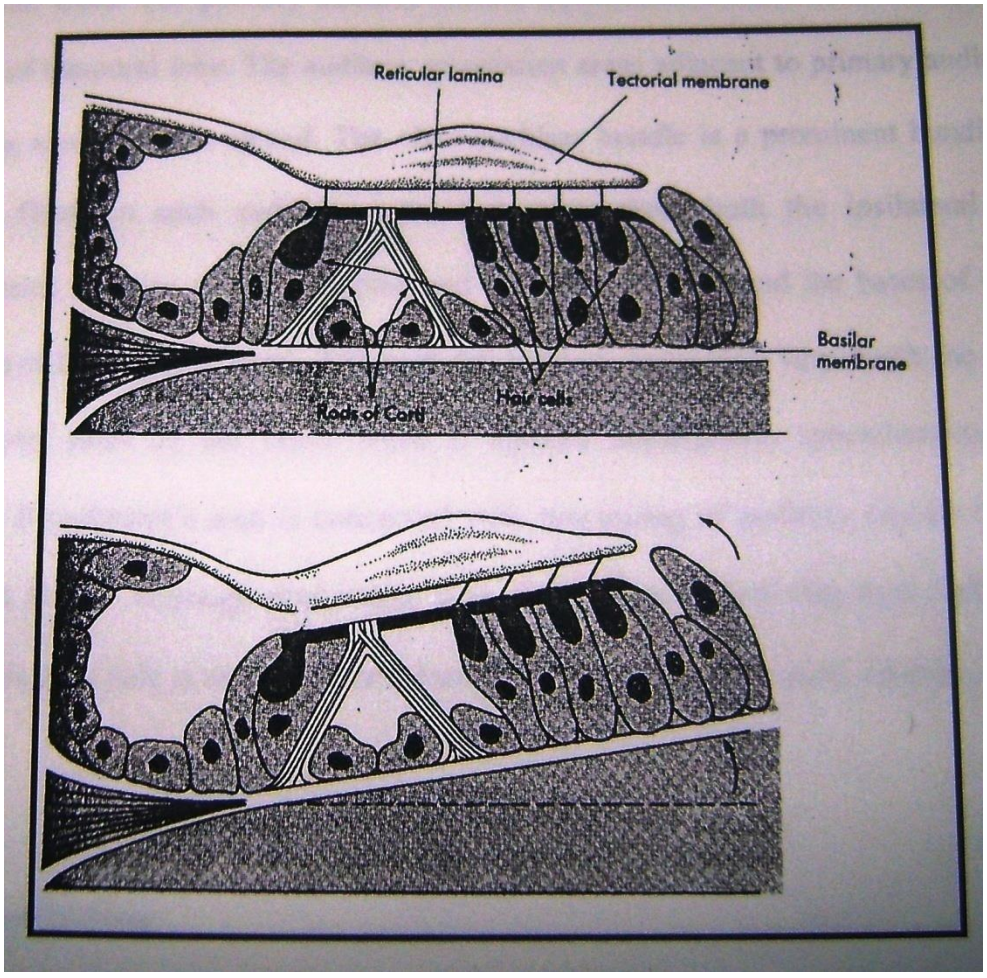


FIGURE 13–7 Ionic composition of perilymph in the scala vestibuli, endolymph in the scala media, and perilymph in the scala tympani. SL, spiral ligament. SV, stria vascularis. The dashed arrow indicates the path by which K^+ recycles from the hair cells to the supporting cells to the spiral ligament and is then secreted back into the endolymph by cells in the stria vascularis.

The stimulus to convert a stable cell membrane at rest to a leaky membrane is the

deflection of stereocilia with its tip links dragging on the membrane of the longer stereocilia to which they are attached thus opening up of ion channels and causing potassium influx. This depolarization releases transmitter substances at the base of the cell which stimulate the afferent nerve endings. There is demarcation of frequencies along each turn of the cochlea with specific stereocilia having specific thresholds for deflection. This results in gradation of the ability to detect specific frequencies in the cochlea. The remarkable sensitivity of the cochlea is brought about by the combination of special characteristics of the endolymph and the mechanical structure of the stereociliary bundle.

DIAGRAM 4- MOVEMENT OF BASILAR MEMBRANE CAUSING STEREOCILIA TO BEND

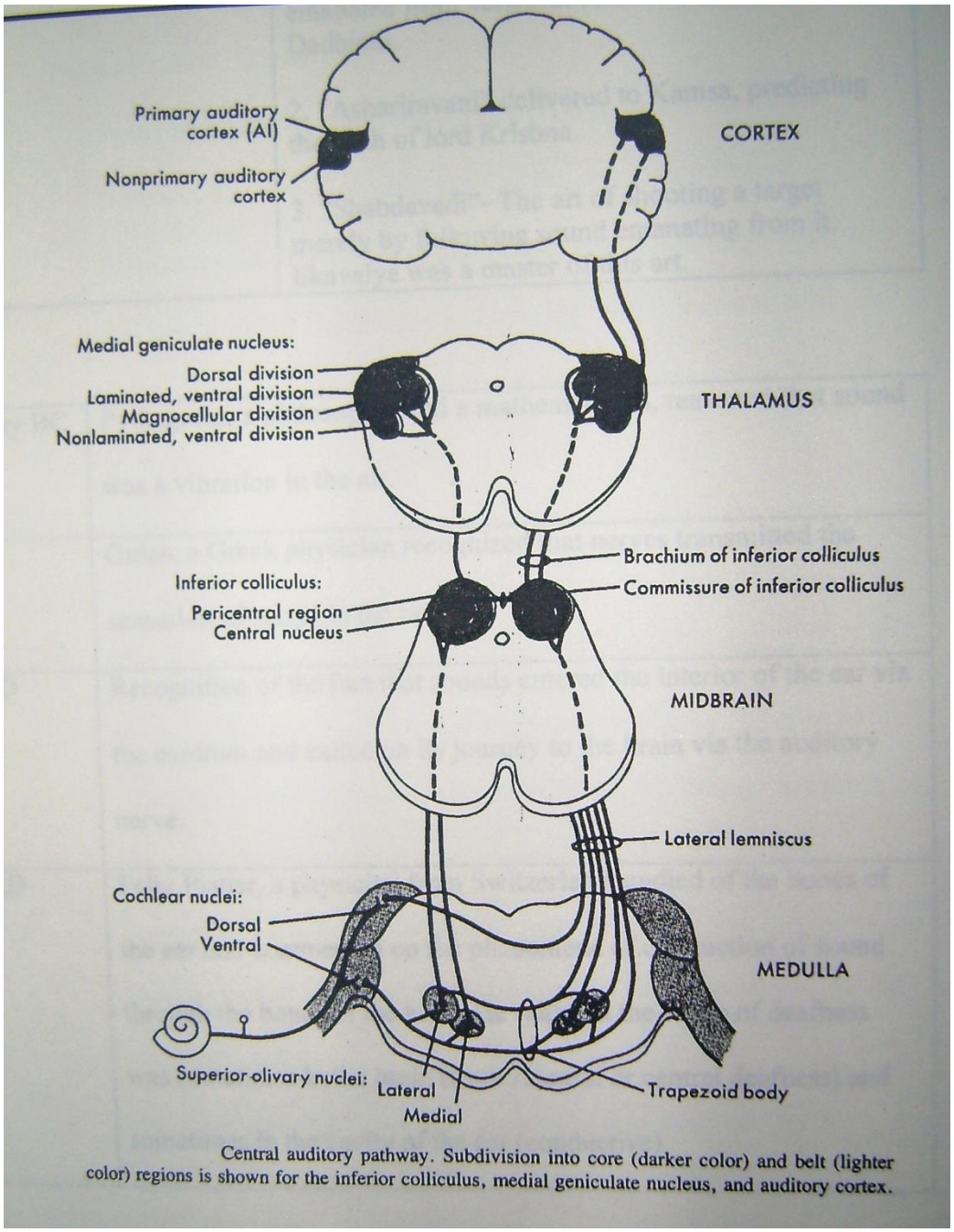


The movement of basilar membrane moves the hair cells which displaces the stereocilia, resulting in generation of an electrical signal in the hair cells. When the stereocilia are pushed toward the kinocillium, the membrane potential is decreased resulting in depolarization and when stereocilia are pushed to opposite direction, the cell is hyperpolarized. When the process is displaced in a direction perpendicular to this axis no change in membrane potential occurs.

3) Auditory pathway and auditory processing:

From the cochlera nuclei,auditory impulses pass via a variety of pathways to the inferior colliculi, the centre for auditory reflexes, and via the medial geniculate body in the thalamus to the auditory cortex. Information from both ears converges on each superior olive, & all higher levels most of the neurons respond to the inputs from both sides. The primary auditory cortex, Broadmann's area 41, is in superior portion of temporal lobe. The auditory association areas adjacent to primary auditory receiving area are wide spread. The olivo cochlear bundle is a prominent bundle of efferent fibres in each auditory nerve that arises from both the ipsilateral & contralateral superior olivary complex and ends primarily around the bases of outer hair cells of the organ of corti. Although the auditory areas look very much the same on both sides of the brain, there is marked hemispheric specialization. For example, Broadmann's area is concerned with processing of auditory signals related to speech, during language processing, it is much more on left side than right side. Area 22 on right side is more concerned with melody, pitch and sound intensity.

DIAGRAM 5- CENTRAL AUDITORY PATHWAY



Central auditory pathway. Subdivision into core (darker color) and belt (lighter color) regions is shown for the inferior colliculus, medial geniculate nucleus, and auditory cortex.

Sound localization:

Determination of the direction from which a sound emanates in the horizontal plane depends upon detecting the difference in time between the arrival of the stimulus in

the two ears and the consequent difference in phase of the sound waves on the two sides; it also depends upon the fact that the sound is louder on the side closest to the source. Neurons in the auditory cortex that receive input from both ears respond maximally or minimally when the time of arrival of a stimulus at one ear is delayed by a fixed period relative to the time of arrival at the other ear. This fixed period varies from neuron to neuron. Sounds coming from directly in front of the individual differ in quality from those coming from behind because each pinna is turned slightly forward.

Theories of hearing:

The currently accepted theory is that of Bekesy's traveling wave theory which suggests that a wave of displacement progresses in a systematic way along the cochlear partition and produces a local stimulation in its path. Von Bekesy was able to deduce that the form of vibration of the cochlear partition for a given frequency at a given instant in time resembled a wave which traveled along the basilar membrane from the base to apex, reached a peak and then decayed rapidly. The traveling wave experienced a phase delay between the stimulus entering the cochlea and the peak of the basilar membrane displacement. Changing the pitch of the incoming pure tone led to a shift in the displacement of the traveling wave. Higher frequencies were responsible for basilar displacement at the base and the lower frequencies at the apex. Thus the cochlea is tonotopically organized. The outer hair cells despite their paucity of nervous innervation have an important role to play in the sensitivity and frequency discrimination capabilities of the inner ear. The outer hair cells are not surrounded by supporting cells instead it is surrounded by Deiter's cells and fluid called corti co lymph. This arrangement facilitates and modulates inner hair cell function by additional distortion of the basilar membrane-organ of corti-tectorial membrane

complex.

The outer hair cells add 40-50 dB of gain to the hearing mechanism which provides a warning system and survival advantage. It is for this work that Von Bekesy received a Nobel Prize in 1961.

D. AUDIOLOGY AND ACOUSTICS[11]

This section aims to introduce certain terms which are frequently used in audiology and acoustics.

Sound: It is a form of energy produced by a vibrating object. A sound wave consists of compressions and rarefactions of the molecules of the medium (air, liquid or solid) in which it travels. Velocity of the sound is different in different media. In the air, at 20° at sea level, sound travels 344 meters / sec and it is faster in the liquid and still faster in the solid media.

Frequency: It is the number of cycles per second. The unit of frequency is Hertz (Hz), named after German scientist Heinrich Rudolf Hertz

Pure tone: A single frequency sound is called a pure tone.

Pitch: It is the subjective sensation produced by the frequency of the sound. Higher the frequency greater is the pitch.

Complex sound: Sound with more than one frequency is called a complex sound. Human voice is a complex sound.

Intensity: It is the strength of the sound which determines its loudness. It is measured in decibels. At a distance of one meter intensity of

Whisper = 30 dB

Normal conversation = 60dB

Shout = 90 dB

Discomfort of ear = 120dB Pain in ear = 130dB

Decibel: It is I/I0th of a bel, and is named after Alexander Graham Bell, the inventor of telephone.

Formula for decibel is

Sound in decibel = 10 Log power of S l/power of S2

OR

10 Log (SPL OF S 1)2 /SPL OF S2

S1 =Sound being described

S2 =Reference sound

SPL =sound pressure level

Sound can be measured in watts/cm2 or dynes/cm2.

In audiology, sound is measured as sound pressure level (SPL).It is compared with the reference sound which has an SPL of 0.0002dynes/cm^2 ,which roughly corresponds to the threshold of hearing in normal subjects at 1000Hz.If a sound has an SPL of 1000 times the reference sound ,it is expressed as 20*3=60dB. Similarly a sound of 1000000 times the reference sound is expressed as 120dB .[9]

Frequency range in normal hearing: Normal persons can hear frequencies of 20 to 20000Hz but in routine audiometric tests only 125 to 8000Hz are evaluated.

Speech frequencies: Frequencies of 500, 1000, and 2000Hz are called speech frequencies as most of human voice falls within this range. Pure tone average is the average threshold of hearing in these three frequencies. It roughly corresponds to the speech reception threshold.[9]

E. HEARING LOSS:

Hearing loss can be of three types.

(1)**Conductive hearing loss** is caused by any disease process interfering with the conduction of sound from the external ear to stapedio-vestibular joint, Thus the cause

may lie in the external ear (obstructions), tympanic membrane (perforation), middle ear (fluid), ossicles (fixation or disruption) or the Eustachian tube (obstruction).

Characteristics of conductive hearing loss:

1. Negative Rinne test, i.e. BC>AC.
2. Weber lateralized to poorer ear.
3. Normal absolute bone conduction.
4. Low frequencies affected more.
5. Audiometry bone conduction better than air conduction with air bone gap. Greater the air bone gap, more is the conductive loss.
6. Loss is not more than 60 dB.
7. Speech discrimination is good.

(2) **Sensorineural hearing loss** from lesion of the cochlea (sensory type) of VII th nerve and its central connections (neural type). The term retrocochlear is used when hearing loss is due to lesions of VIIth nerve and central deafness, when it is due to lesions of central auditory connections. It may be congenital or acquired. Acquired causes are infections of labyrinth-viral, bacterial or spirocheatal, trauma to the labyrinth or VIIth nerve, e.g. fractures of temporal bone or ear surgery, noise induced hearing loss, ototoxic drugs, presbyacusis, meniere's disease, acoustic neuroma, sudden hearing loss, familial, systemic disorders, e.g. diabetes, hypothyroidism, kidney disease.

Characteristics of sensorineural hearing loss:

1. A positive Rinne's test, i.e. AC>BC.
2. Weber laterlised to better ear.
3. Bone conduction reduced on Schwabach and absolute bone conduction tests.
4. More often involving high frequencies.

5. No gap between air and bone conduction curve on audiometry.

6. Loss may exceed 60 dB.

7. Speech discrimination is poor.

8. There is difficulty in hearing in the presence of noise.

(3) **Mixed hearing loss**: In this type, elements of both conductive and sensorineural deafness are present in the same ear. There is air-bone gap indicating conductive element, and impairment of bone conduction indicating sensorineural loss. Mixed hearing loss is seen in some cases of otosclerosis and chronic suppurative otitis media (CSOM).[9]

DEGREE OF HEARING LOSS [WHO Classification 1980][9]

Normal	0-25 Db
Mild	26-40 dB
Moderate	41-55 dB
Moderately severe	56-70 dB
Severe	71-91 dB
Profound	>91 dB

F.<u>TESTS OF HEARING:</u>

<u>1.Tuning fork tests</u>:

Rinne's test : The Rinne test is probably the most commonly used tuning fork test. The Rinne test is a comparison of the patient's hearing sensitivity by bone conduction versus air conduction. A normal individual will perceive the air conducted sound as louder or the same as bone conducted sound. Proper placement of the tuning fork in each situation is important. When testing by bone conduction, the stem fork should be placed firmly on the mastoid, as near to the posterosuperior edge of the ear canal as

possible. The stem should not touch the auricle of the external canal, which should be held to the side by the examiner's fingers. Touching the external ear itself could give false results due to vibration of the auricle. When testing by air conduction, the fork is held about 2.5 cm lateral to the tragus. In the Rinne test, when the conduction mechanism is normal in an ear (that is, in individuals with normal hearing and in those with sensorineural hearing impairment), air conduction will be heard better than bone conduction as it is a more efficient means of sound transmission. This finding is

termed as positive Rinne.

Bone conduction will be heard better than air conduction when there is a deficit in the conduction mechanism and is referred to as a negative Rinne. A conductive deficit of more than 15 db reverses the tuning fork responses (that is, bone conduction is better than air conduction) at 512 Hz.

Weber’s test: The Weber test is based on the principle that the signal, when transmitted by bone conduction, will be localized to the better hearing ear or the ear with the greatest conductive deficit. The test can determine the type of hearing impairment when the two ears are affected to different degrees. The stem of a vibrating tuning fork is placed on the skull in the midline, and the patient is asked to indicate in which ear the sound is heard.

The usual location described for placement is on the forehead; but better locations are the nasal bones or teeth when a stronger bone conduction stimulus is required. In unilateral hearing losses, lateralization to the poorer-hearing ear indicates an element of conductive impairment in that ear. Lateralization to the better-hearing ear suggests that the problem in the opposite ear is sensorineural

Although tuning fork tests allow the examiner to identify a conductive versus a sensorineural loss, and in some cases lateralize the symptomatic ear, it does not evaluate the degree of impairment or the effects of that impairment on speech understanding.

2**.Pure tone audiometry**

An audiometer is an electronic device which produces pure tones, the intensity of which can be increased or decreased in 5 dB steps. Air conduction Thresholds and measured for tones of 250, 500, 1000, 1500, 2000, 4000 6000 and 8000 Hz. Bone conduction thresholds and measured for 250, 500, 1000, 1500, 2000, 4000 hertz. The amount of intensity that has to be raised about the normal level is a measure of the degree of hearing impairment at that frequency. It is charted in form of a graph called the' audiogram'. The thresholds of bone conduction are a measure of the cochlear function. The difference in the thresholds of air and bone conduction (A-B gap) is a measure of a degree of conductive deafness. The audiometer is so calibrated that hearing of a normal person, both of air and bone conduction is at 0 db and there is no A-B gap.

INTERPRETATION OF AN AUDIOGRAM:

Conductive deafness- is indicated by raised air conduction thresholds (25dB) and a normal bone conduction threshold with a wide air-bone gap of 15 dB or more.

Sensorineural deafness-is indicated by raised air and bone conduction thresholds (both>25dB) and the air bone gap does not exceed 10dB.

Mixed deafness- air and bone conduction thresholds are raised with air bone gap of > 15dB.

3.**Auditory Brainstem Response(ABR):**

Auditory brainstem response test sometimes also referred to as auditory evoked potential gives information about inner ear and brain pathways of hearing. The test can be used with children or others who have a difficult time with conventional behavioral methods of hearing screening .ABR is performed by pasting electrodes on the head and recording brain activity in response to sound.

4.**Otoacoustic Emissions(OAE)**

Otoacoustic emissions are sounds given off by the inner ear when the cochlea is stimulated by sound. When the sound stimulates the cochlea, the outer hair cells vibrate this vibration produces a nearly inaudible sound that echoes back into the middle ear. This sound can be measured with a small probe inserted into the ear canal. People with normal hearing produce emissions, those with hearing loss greater than 25-30dB do not produce these very soft sounds.

4.**Other tests:**

Which include speech audiometry and tests of middle ear like Tympanometry, Acoustic reflex measures, and Static acoustic impedance.

Amongst all these tests pure tone audiometry is a simple, inexpensive, qualitative, quantitative, universal(for comparison) procedure which aims at ascertaining if the subject has an auditory disorder and also degree of hearing loss, only disadvantage being subjective.

G. **SMOKING-THE EPIDEMIC:**

The history of smoking dates back to as early as 5000 BC in Shamanistic rituals, the kind of smoking available was marijuana or cannabis smoking. Cannabis first arrived on the scene or was at least better widely used than tobacco. Tobacco only arrived in

America in the 1600s. Amongst the Indian communities in America tobacco was actually used in treating different ailments including headaches and tooth aches. It was first accepted as a medical aid before becoming a widespread commercial product for temporary pleasure.

Jean Nicot from whose name is derived the word ‘nicotine’, brought tobacco to France in 1560. It then spread from France to England and much of Europe. From Europe, tobacco seeds found their way to Brazil.

In India, tobacco was brought by the Portuguese merchants 400 years ago. Although there were some strains of locally grown tobacco in India, these were outclassed by the new imported varieties from Brazil.

Modern smoking became part of smoking history in the 1900s. It is during the history of smoking in the 1900s that smoking dangers became more and more apparent as people started dying from smoking related lung cancer. In Germany in the 1920s the first ever anti-smoking campaigns began even though they were fiercely opposed in some sections. Second World War saw the intensification of the use of tobacco and cigarettes in cinema. This was so for many years to come as cigarettes dominated Hollywood and were associated with sexism, macho and fashion. Even today there is an entire actor smoking list appearing in movies.

Tobacco companies maximized their endorsement into a generation hungry for a new identity. As more people fell ill from smoking diseases, the health bill continued to balloon. Perhaps the turning point in smoking history was the admitting by tobacco companies of the existence of over 4000 compounds and over 500 additives in

cigarettes. However, overall from 1965 to 2006 smoking declined from about 42% to 20%. The same cannot be said about the cost of smoking healthcare wise.

Smoking is the leading cause of preventable death in the world. The effects of smoking represent a global pandemic of enormous proportions. Tobacco causes around 3.5 million deaths annually, the figure set to rise to around 10 million deaths annually during the 2020s or 2030s. Of the latter figure, 7 million deaths will occur in developing countries.[12]

By recent counts, there are about 94 million smokers in India, Almost 5%of women and a third of all men aged between 30 & 69 smoke and smoking of cigarettes causes death 10 years sooner.[3] Aside from the conventional cigarettes, they also smoke beedis which is a type of Asian cigarette. Prevalence in Karnataka was 41% among men & 14.9% among women.[13]

CIGARETTE AND ITS MAKING:[14,15]

A cigarette is a small roll of finely cut tobacco leaves wrapped in a cylinder of thin paper for smoking with a filter at its mouth end. The tobacco is blended from two main leaf varieties: yellowish "bright", also known as virginia, where it was originally grown, contains 2.5-3% nicotine and "burley" tobacco which has 3.5-4%. In addition to this nicotine blend cigarette also contains sweeteners which are used to affect the flavor, making cigarettes more appealing to consumers, menthol in cigarettes has a numbing effect on sensory nerve endings in the respiratory tract and helps to temporarily soothe sensations of discomfort in areas of inflammation and irritation, ammonia helps in absorption of nicotine in non ionized state.

In addition cigarettes also contain "filters" which are made from stems or other bits of tobacco or made of cellulose acetate. Higher filter contents makes a less dense

cigarette with lower tar delivery. The nicotine & tar delivery to lungs can also be reduced by modifying the type of paper used in the cigarette.

RISKS OF SMOKING:

The dangerous component of a cigarette is "smoke". Smoke contains harmful gases like carbon monoxide & nitrogen oxide. It also contains around 4000 chemicals, out of which 16 priority chemicals include: [14]

1. 1,3 – butadiene
2. Acetaldehyde
3. Acrolein
4. Acrylonitrile
5. Arsenic
6. Benzene
7. Cadmium
8. Carbon monoxide
9. Chlorinated Dioxins and Furans,
10. Chromium (VI)
11. m + p + o Cresol
12. Formaldehyde
13. Hydrogen cyanide
14. N-nitrosonornicotine (NNN)
15. N-nitrosodimethylamine (NDMA)
16. N-nitrosopyrrolidine (NP)

The list of diseases caused by tobacco now includes cancers of the kidneys, stomach, cervix, and pancreas as well as leukemia, cataracts, pneumonia, and gum disease. These illnesses are in addition to diseases previously known to be caused by smoking like bladder, esophageal, laryngeal, lung, oral, and throat cancers, chronic lung diseases, coronary heart and cardiovascular diseases, and sudden infant death syndrome.

Smoking also reduces overall health, contributing to conditions such as hip fractures, complications from diabetes, increased wound infections following surgery, and various reproductive problems. "There is no safe cigarette, whether it is called 'light,' 'ultra–light,' or any other name".

H.EFFECT OF SMOKING ON AUDITORY SYSTEM:

There is no direct evidence for the mechanisms of damage to the auditory system associated with cigarette smoke exposure. However, at least three different putative mechanisms may play a role in the manifestations of peripheral and central auditory problems associated with nicotine exposure. [16]

The first mechanism may be related to hypoxia; both nicotine and carbon monoxide in cigarette smoke have been shown to reduce the oxygen supply to fetal tissue by restricting utero-placental blood flow.[17, 18] Moreover, there may be direct intake of nicotine by the fetus, since nicotine can easily cross the placenta, potentially increasing fetal nicotine plasma levels by up to 15% and amniotic fluid levels by up to 54% in the mid trimester .[19] Thus, nicotine-induced vasospasms and carbon monoxide may deplete oxygen levels to the cochlea .[17,18] In older individuals nicotine induced vasospasms as well as atherosclerotic damage may play a role in perpetrating (hypoxic) damage to the cochlea and even spiral ganglion cells . [20]

The second putative mechanism may pertain to the interaction between

nicotine and nicotinic acetylcholine receptors (nAChRs) within the auditory system. Nicotine binds to nAChRs that normally modulate the effects of a neurotransmitter called acetylcholine. Since neurotransmitters function as chemical message carriers facilitating communication between cells by binding to the receptors on the cell surface, loss or damage of the receptors would eliminate the modulatory influences of the receptors. There is now evidence that nAChRs are critical components of the auditory pathway, from the cochlea to the temporal lobe, and the descending auditory pathway.[21,22] Moreover, emerging data indicates that prenatal exposure to nicotine or chronic nicotine use during adolescence damages the nAChR binding sites, producing cognitive impairments in the auditory and visual modalities .[23,24,25]

Finally, the neurophysiological mechanism that may potentially explain the association between adolescent smoking and neurocognitive deficits is protracted development of the auditory central nervous system pathways. There is incontrovertible evidence that many components of auditory central nervous system development, including the auditory thalamocortical and cortigofugal pathways, continue into late adolescence .[24,25,26] Moreover, these pathways are particularly susceptible to damage, if environmental toxins like nicotine are introduced during their developmental emergence.[23,27]

I. REVIEW OF OTHER WORK DONE:

Cigarette smokers are exposed to nicotine directly, as well as a number of additional chemicals including formaldehyde, benzene, arsenic, vinyl chloride, ammonia and hydrogen cyanide via second hand smoke inhalation.

Over a century ago, the association between excessive smoking and deafness was noted by de LaCharrière in 1875.[28] Information on the effects of smoking at the cochlear and auditory pathway has become available only recently. Various studies

have shown cigarette smoking has got deleterious effects on peripheral and central auditory nervous system.

A study done to evaluate the association between cigarette smoking and hearing loss in a large population based cohort of adults aged 48 to 92 years showed that the prevalence of hearing loss in current smokers increased from 26% in the 48-59 years age range to 56% and 71% in the sixth and seventh decades of life, respectively. Furthermore, as pack years of cigarette smoking increased from 0 to > 40, the prevalence of hearing loss increased from 17% to 29% in the 48-59 years age range, from 36% to 59% in the 60-69 year old smokers, and from 60% to 74% in the 70-79 year old smokers. This study concluded that environmental exposures may play a role in age related hearing loss [5] .A retrospective cross sectional study in 13,308 men aged 20-68 years showed that a significantly higher incidence of any type of hearing loss was found in current (11.8%) and past smokers (11.7%) than in non smokers (8.1%).The risk of increment of the smoking status for developing hearing loss among subjects under the age 35 was 43% and 17% among those above 35 years. Sensorineural impairment and conductive impairment were found to be associated particularly with smoking.[29]

The association of cigarette smoking with development of hearing impairment over a 5 year follow up was studied in 1554 non hearing impaired Japanese male office workers of age group between 30 to 59 years and it showed that workers who smoked were at a greater risk for hearing loss at 4 kHz than at 1 kHz compared to nonsmokers and ex-smokers. After controlling the confounding factors like age , body mass index, alcohol consumption, triglyceride levels, etc., this study showed that as numbers of cigarettes smoked per day and pack years of smoking increased, the risk for high-

frequency hearing loss increased in a dose dependent manner, whereas low-frequency hearing loss remained unchanged.[6]

From the above studies it is shown that not only does cigarette smoking place an individual at risk for hearing loss, but as pack years of smoking increase accompanying hearing loss also increases and the effects of smoking may be exacerbated in younger individuals.

A number of studies have shown that noise induced hearing loss is exacerbated by cigarette smoking and that the effects of age may further compound the hearing problem. Most recently prospective observational cohort study done on a group of long-term smokers and a control group of non-smokers taken from a population of noise-exposed employees in the brick manufacturing industry showed that the median age-corrected hearing thresholds at 3 and 4 kHz in the smokers group were significantly higher than those in the non-smokers group. No statistical difference in the hearing thresholds between both groups was found in any other tested frequency (0.5, 1, 2, 6 and 8 kHz).[30] Likewise, study done on 4624 steel company workers in Japan to examine synergistic effect of smoking and occupational exposure to noise on hearing loss showed that smoking was associated with increased odds of having high frequency hearing loss in a dose dependent manner. The synergistic index was 1.16 and smoking was not associated with low frequency hearing loss.[31]

A study done on 263 residents of a rural village to determine the combined effect of smoking and age on hearing impairment who were not exposed to noise by the department of community health, Malaysia demonstrated that there was a statistically significant trend in the number of pack-years of smoking and age as risk factors for hearing impairment .[32]

Smoking, age and noise exposure together pose a greater risk for hearing loss

than each factor alone was concluded by a cross sectional study done on 535 male adult workers of metal factory workers.[33]

All of the above studies indicate that although age in itself may produce a decline in hearing over time, the contributions of a variety of risk factors including smoking further perpetuates the hearing loss. Thus, if individuals monitor risk factors like blood pressure, diabetes, cholesterol, body mass index and choose a healthy lifestyle that promotes good cardiovascular health, age related hearing loss may be abated considerably.

There are various studies in literature which shows auditory processing difficulties associated with prenatal exposure to cigarette smoke. A recent study that measured high-density event related potentials (ERPs) within 48 hours of birth in healthy babies of smoking and nonsmoking mothers showed significant differences in auditory neurophysiology of exposed and non-exposed babies.[34] Yet another study noted that maternal use of more than 10 cigarettes per day during pregnancy produced significant reduction in babbling in babies and that the risk of not babbling doubled by 8 months of age. This risk further increased in children who were breast fed for < 4 months .[35]

These findings indicate that prenatal cigarette smoke exposure alters speech, sound or auditory discrimination ability and that such changes may ultimately be responsible for not only developmental speech-language problems, but also neurocognitive impairments documented at later ages.

METHODOLOGY

MATERIALS AND METHODS:

The study group consisted of 50 cigarette smokers and 50 age matched non smokers.

SELECTION OF SUBJECTS:

The subjects were recruited based on various inclusion and exclusion criteria from teaching and non teaching staff of Sri Devraj Urs Academy of Higher Education and Research and also attenders of patients coming to R.L.Jalappa Hospital, Kolar after taking informed consent. Ethical clearance was also obtained from Institutional Ethical Clearance Committee for the study.

CRITERIA FOR SELECTION OF STUDY GROUP

Inclusion criteria:

Study group included :

1. Male smokers of age group between 20 and 40years .
2. Subjects who are not exposed to occupational noise.

Control group included :

1. Non smoking males of age group between 20 and 40years .
2. Subjects who are not exposed to occupational noise

Exclusion criteria:

Study group:

1. Subjects over 40 years of age.
2. Subjects with history of use of ototoxic drugs like streptomycin, cisplatin, neomycin, gentamycin .
3. Subjects with chronic medical illness like diabetes, hypertension etc.
4. Subjects with history of head injury and history of ENT infections in past 3 months.

Control Group: Same as above subjects being non smokers.

METHODOLOGY:

Based on above predetermined inclusion and exclusion criteria, subjects were divided into study (smokers) and control (non smokers) groups.

Study subjects thus selected were given a questionnaire to collect information regarding their smoking history expressed in pack years. Pack years of smoking will be defined as the number of packs (one pack=20 cigarettes) smoked per day multiplied by the duration of smoking in years.A detailed general physical and systemic examination was conducted in all subjects.Also a detailed ear, nose and throat examination was carried out to rule out any unidentified pathology.

An assessment of auditory thresholds was done for different frequencies by using pure tone audiometer (ELKON-GIGA3) for both study and control groups in a sound proof room.

Pure tone audiometer contains sound thresholds in decibels and frequencies in Hertz. Auditory threshold is the lowest level of sound threshold in decibels, at a particular frequency of sound at which the human ear can perceive it as a sound. The parameters studied in pure tone audiogram are air conduction and bone conduction hearing thresholds of both the ears at various frequencies of sound.

They are recorded on audiogram chart which depicts the auditory thresholds of the particular ear. The audiogram was recorded for both the ears separately.

The data collected was entered in master chart and statistically analysed using appropriate statistical test like student t test.

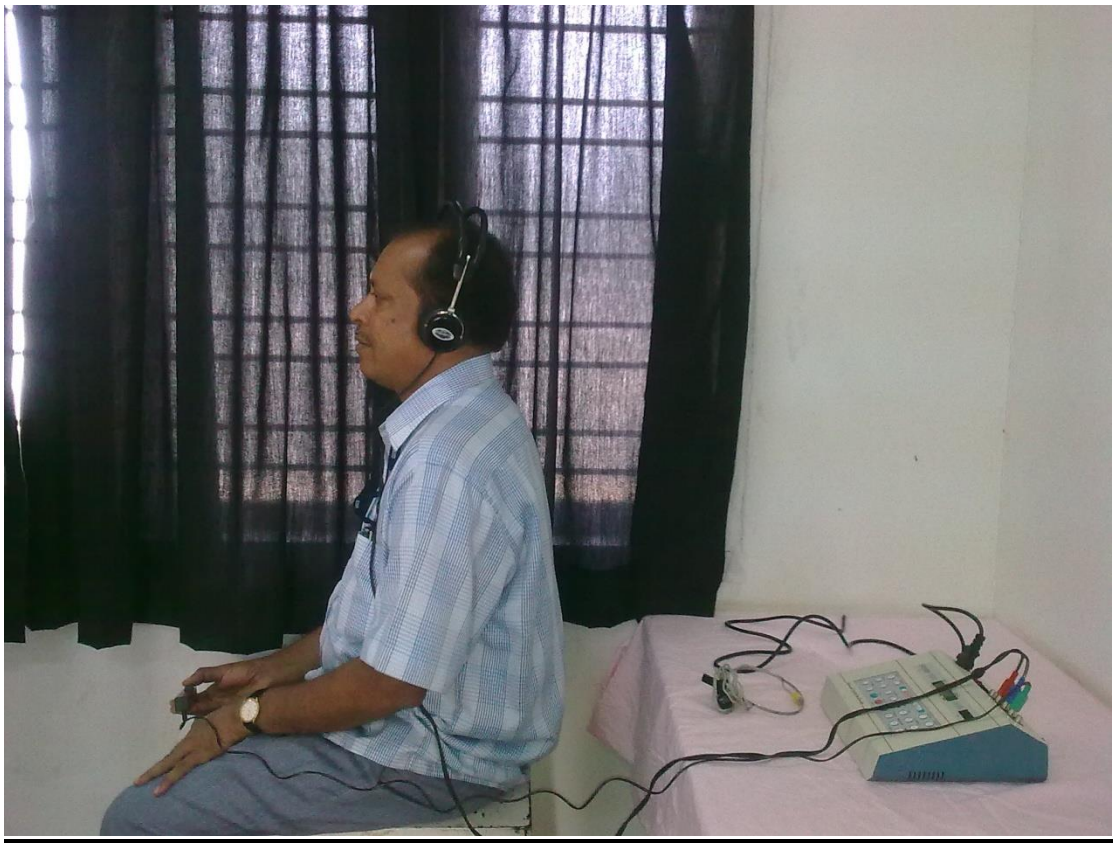
Statistical Treatment of the data:[37,38,39,40]

The data was suitably arranged into tables for discussion under different headings. Descriptive statistical analysis was carried out on this data. Results on continuous measurements are presented as mean $\pm$ standard deviation and results on categorical

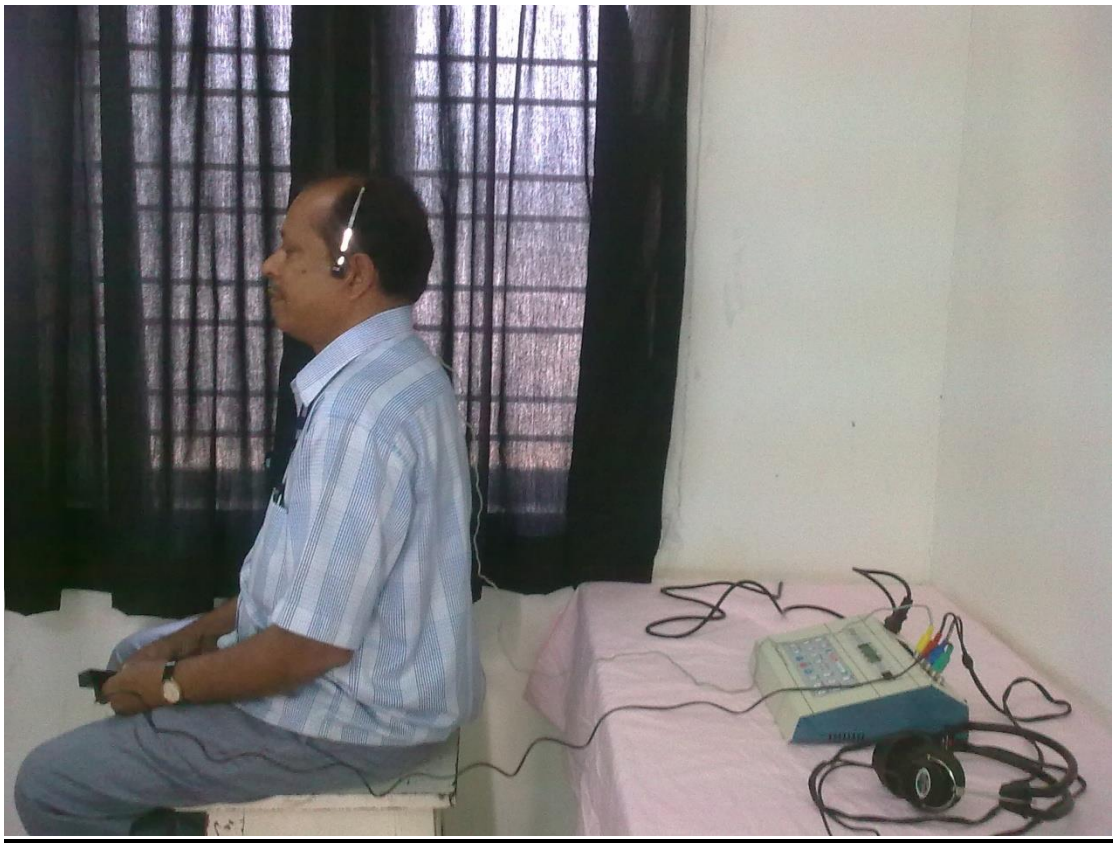
measurements are presented in number%. Significance was assessed at 5% level of significance. AC, BC hearing thresholds recording was compared between cigarette smokers and age matched non smokers. The Pearson correlation between pack years of smoking and AC ,BC conduction hearing thresholds for both ears was also done with significance test by student‘t’ test and ANOVA test.

PURETONE AUDIOMETER:

RECORDING OF AC THRESHOLDS:



RECORDING OF BC THRESHOLDS:



PURE TONE AUDIOMETRY:

INSTRUMENT :ELKON GIGA3

PRINCIPLE:

An audiometer is an electronic device which produces pure tones, the intensity of which can be increased or decreased in 5 dB steps. Air conduction Thresholds and measured for tones of 250, 500, 1000, 1500, 2000, 4000 6000 and 8000 Hz. Bone conduction thresholds and measured for 250, 500, 1000, 1500, 2000, 4000 hertz. The amount of intensity that has to be raised about the normal level is a measure of the degree of hearing impairment at that frequency. It is charted in form of a graph called the' audiogram'. The thresholds of bone conduction are a measure of the cochlear function. The difference in the thresholds of air and bone conduction (A-B gap) is a measure of a degree of conductive deafness. The audiometer is so calibrated that hearing of a normal person, both of air and bone conduction is at 0 db and there is no A-B gap.

METHOD:[36]

The method is based on American Society for Speech and Hearing Association (ASHA) 2005 guidelines for manual pure tone threshold audiometry(PTA) and is as follows.

Determination of Manual Thresholds

Before conducting threshold testing, a complete case history should be obtained and otoscopy completed. The audiologist should be able to monitor the listener's alertness and physical condition at all times.

Ear examination:Visual inspection of the pinna and ear canal, including otoscopy, should precede audiometric testing to rule out active pathological conditions and the potential for ear canal collapse caused by audiometric earphones. The ear canal

should be free of excessive cerumen before testing. Testing should begin with the better ear when identifiable, otherwise it is arbitrary.

Participant seating: The participant should be seated in a manner to promote safety and comfort as well as valid testing. Such seating considerations may include the following:

• Avoid giving inadvertent visual cues to the participant.

• Enable easy observation of participant responses to stimuli.

• Allow for the monitoring and reinforcement of responses.

• Permit observation of participant comfort, safety, and health.

Some of the factors that influence the manual assessment of pure-tone thresholds are (a) the instructions to the participant, (b) the response task, and (c) the audiologist's interpretation of the participant's response behavior during the test.

Instructions:

The test instructions should be presented in a language or manner appropriate for the participant. Interpreters (oral or manual) should be used when necessary. Supplemental instructions may be provided to enhance understanding, such as written directives, gestures, and demonstrations. Test instructions shall accomplish the following:

• Indicate the purpose of the test, that is, to find the faintest tone that can be heard.

• Emphasize that it is necessary to sit quietly, without talking, during the test.

• Indicate that the participant is to respond whenever the tone is heard, no matter how faint it may be.

• Describe the need to respond overtly as soon as the tone comes on and to respond overtly immediately when the tone goes off.

• Indicate that each ear is to be tested separately with tones of different pitches.

• Describe inappropriate behaviors such as drinking, eating, smoking, chewing, or any additional behavior that may interfere with the test.

• Provide an opportunity for any questions the listener may have.

Response task: Overt responses are required from the participant to indicate when he or she hears the tone going on and off. The response task used here is pressing and releasing the signal switch.

Interpretation of response behavior: The primary parameters used by the audiologist in determining threshold are the presence of "on" and "off" responses, latency of responses, and number of false responses:

• Each suprathreshold presentation should elicit two responses: an "on" response at the start of the test tone and an "off" response at the end of the tone.Participants who are unable to correctly signal the termination of the tone, after proper instruction and reinstruction, may be demonstrating auditory problems and may need more detailed testing.

• The latency of the "on" responses varies usually with the level of presentation.If the first response to a tone in an ascending series is slow, present a 5-dB higher tone until the response is without hesitation.

• False responses may be of two types:

(a) False positive, a response when no tone is present

(b) False negative, no response to a tone that the audiologist believes to be audible to the participant. Either type complicates the measurement procedure. Reinstruction may reduce the occurrence rate of either type.

Threshold Measurement Procedure

The basic procedure for threshold determination consists of

(a) Familiarization with the test signal

(b) Threshold measurement.

The procedure is the same regardless of frequency, output transducer, or ear under test.

a.)Familiarization:.

The purpose of familiarization is to assure the audiologist that the participant understands and can perform the response task. Familiarization is a recommended practice for general populations and should be used whenever warranted by the mental or physical status of the patient. The participant should be familiarized with the task before threshold determination by presenting a signalof sufficient intensity to evoke a clear response.

The following two methods of familiarization are commonly used:

1. Beginning with a 1000-Hz tone, continuously on but completely attenuated, Gradually increase the sound-pressure level of the tone until a response occurs.

2. Present a 1000-Hz tone at a 30 dB hearing level (HL). If a clear response occurs, begin threshold measurement. If no response occurs, present the tone at 50 dB HL and at successive additional increments of 10 dB until a response is obtained.

b)Threshold determination: The method described, an ascending technique beginning with an inaudible signal, is recommended as a standard procedure for manual pure-tone threshold audiometry.

1. Tone duration: Pure-tone stimuli of 1 to 2 seconds' duration.
2. Interval between tones: The interval between successive tone presentation shall be varied but not shorter than the test tone.

3. Level of first presentation: The level of the first presentation of the test tone shall be well below the expected threshold.
4. Levels of succeeding presentations: The level of each succeeding presentation is determined by the preceding response. After each failure to respond to a signal, the level is increased in 5-dB steps until the first response occurs. After the response, the intensity is decreased 10 dB, and another ascending series is begun. (An exception is as explained previously under Interpretation of response behavior—Latency).
5. Threshold of hearing: Threshold is defined as the lowest decibel hearing level at which responses occur in at least one half of a series of ascending trials. The minimum number of responses needed to determine the threshold of hearing is two responses out of three presentations at a single level (American National Standards Institute, 2004a)

Variability of threshold measures: The audiologist should establish limits on acceptable test-retest variability for a given participant.

Standard Procedures for Air-Conduction Measures

Supra-aural or circumaural earphones shall be held in place by a headband with the earphone grid directly over the entrance to the ear canal.

Earphone placement:

The audiologist should instruct participants to remove hats, headbands, eyeglasses, earrings, or anything that may interfere with proper positioning of the earphone cushions on the ears. After visual inspection of the outer ear , the audiologist should place the earphones on the participant and adjust them to fit her or his head properly. Insert earphones shall be placed comfortably deep in the ear canal and in accordance with manufacturer recommendations.

Stimuli:

Continuous or pulsed pure-tone signals should be used. Pulsed tones have been shown to increase a test participant's awareness of the stimuli (Burk & Wiley,2004).

Frequency:

The frequencies tested differ, depending on the technique used.

1. Monitoring technique. Threshold assessment should be made at 500, 1000, 2000, 3000, 4000, 6000, and 8000 Hz when monitoring as part of hearing loss prevention programs. When monitoring for other purposes (e.g., ototoxicity, medical management), thresholds may be measured at other test frequencies as appropriate.

2. Diagnostic technique. Threshold assessment should be made at 250, 500, 1000, 2000, 3000, 4000, 6000, and 8000 Hz, except when a low-frequency hearing loss exists, in which case the hearing threshold at 125 Hz should also be measured. When a difference of 20 dB or more exists between the threshold values at any two adjacent octave frequencies from 500 to 2000 Hz, interoctave measurements should be made.

Order:

When appropriate information is available, the better ear should be tested first. The initial test frequency should be 1000 Hz. Following the initial test frequency, the audiologist should test, in order, 2000, 3000, 4000, 6000, and 8000Hz, followed by a retest of 1000 Hz before testing 500, 250, and 125 Hz. A retest at 1000 Hz is not necessary when testing the second ear. Although the order of frequencies is not likely to significantly influence test results, presentation frequencies in the order described may help ensure consistency of approach to each test participant and minimize the risk of omissions (American National Standards Institute, 2004b).

Masking for diagnostic audiometry:

Appropriate masking should be applied to the nontest ear when the air-conduction threshold obtained in the test ear exceeds the interaural attenuation to the nontest ear. Because the procedures for masking are not confined to pure-tone measures, these procedures are not discussed in this set of guidelines.

Standard Procedures for Bone-ConductionMeasures

Standard bone-conduction vibrator placement should allow mastoid or forehead placement with proper force applied (American National Standards Institute, 2004b; Dirks, 1964). The test ear should never be covered for standard boneconduction measurements. The contralateral ear will be covered when masking is being used. The audiologist shall place the transducer(s), not the participant. It may be necessary to clip the transducer wire to the participant to avoid unintentional movement.

It may be necessary to include the following instructions during bone-conduction testing:

• Advise the participant to sit quietly and avoid movement that will dislodge the bone vibrator from the proper position.

• Request that the participant notify the audiologist when the bone vibrator slips or moves in any way from the original placement.

Frequency: Thresholds should be obtained at octave intervals from 250 to 4000Hz and at 3000 Hz. Testing at frequencies below 500 Hz demands excellent sound isolation for cases with normal or near normal sensitivity but may be accomplished when such an environment is available. Higher frequencies may be tested if the transducer has sufficient frequency-response characteristics.

Order: The initial frequency tested should be 1000 Hz. After the initial test frequency, the audiologist should test 2000, 3000, and 4000 Hz followed by a retest of 1000 Hz before testing 500 and 250 Hz.

Masking: If the unmasked bone-conduction threshold is 10 dB better than the airconduction threshold at that frequency in either ear, masking must be used. Because the threshold values on which the calibration of bone vibrators is based were measured with masking noise in the contralateral ear, the audiologist may prefer always use masking in the testing procedure.

Responses:Vibrotactile responses to bone-conducted signals are possible,especially at low frequencies (Boothroyd & Cawkwell, 1970). Suspected vibrotactile responses should be noted on the audiogram form.

Record Keeping

Recording of results:

Results may be recorded in graphic or tabular form or both.

Separate forms to represent each ear may be used. Results must be legible and should be of sufficient quality to allow copying and electronic storage and communication. The privacy and confidentiality of audiometric records must be maintained and protected in accordance with all applicable state and federal regulations, such as the Health Insurance Portability and Accountability Act of 1996 (Final Regulations for Health Coverage Portability for Group and Medicaid Services, 2004).

Audiogram form: When the graphic form is used, the test frequencies shall be recorded on the abscissa, indicating frequency on a logarithmic scale, and hearing levels shall be recorded on the ordinate, using a linear scale to include the units of decibels. The aspect ratio of the audiogram is important for standardization. The correct aspect ratio is realized when a square is formed between any given octave pair

on the abscissa and any 20 dB increment on the ordinate. For conventional audiometry, the vertical scale is to be designated hearing level in decibels; the horizontal scale is to be labeled frequency in hertz. By convention, frequency is recorded in ascending order from left to right, and hearing level is recorded in ascending order from top to bottom, ranging from a minimum value of −10 dB to the maximum output limits of the audiometer (usually 110 or 120 dB HL). It is advisable when reporting extended high-frequency audiometric results to use a separate graph that incorporates the appropriate decibel scale (HL vs. SPL) and frequency range measured.

Audiogram symbols: When the graphic form is used, the symbols presented in the Guidelines for Audiometric Symbols (American Speech-Language-Hearing Association, 1990a) should be used.

RESULTS AND ANALYSIS

RESULTS & ANALYSIS

The study included 50 male cigarette smokers of age group between 20-40 years and 50 age matched male non smokers, who were selected based on inclusion and exclusion criteria and subjects were recruited from teaching ,non teaching staff of Sri Devraj Urs academy of higher education and research and also attenders of the patients coming to R.L.Jalappa hospital. After taking informed consent and data regarding their smoking history (expressed in pack -years), they were subjected to pure tone audiometric evaluation. The data was analysed using appropriate statistical methods and discussed hereinafter.

Table 1: Age distribution of subjects studied:

Age in years	Smokers		Non-Smokers	
	No	%	No	%
20-25	15	30.0	16	32.0
26-30	17	34.0	19	38.0
31-35	11	22.0	7	14.0
36-40	7	14.0	8	16.0
Total	50	100.0	50	100.0
Mean ± SD	29.14±5.28		28.16±5.73	

Table 1 shows age distribution of subjects included in the study .The study groups are age matched with p=0.376.

Graph1: Age distribution of subjects studied

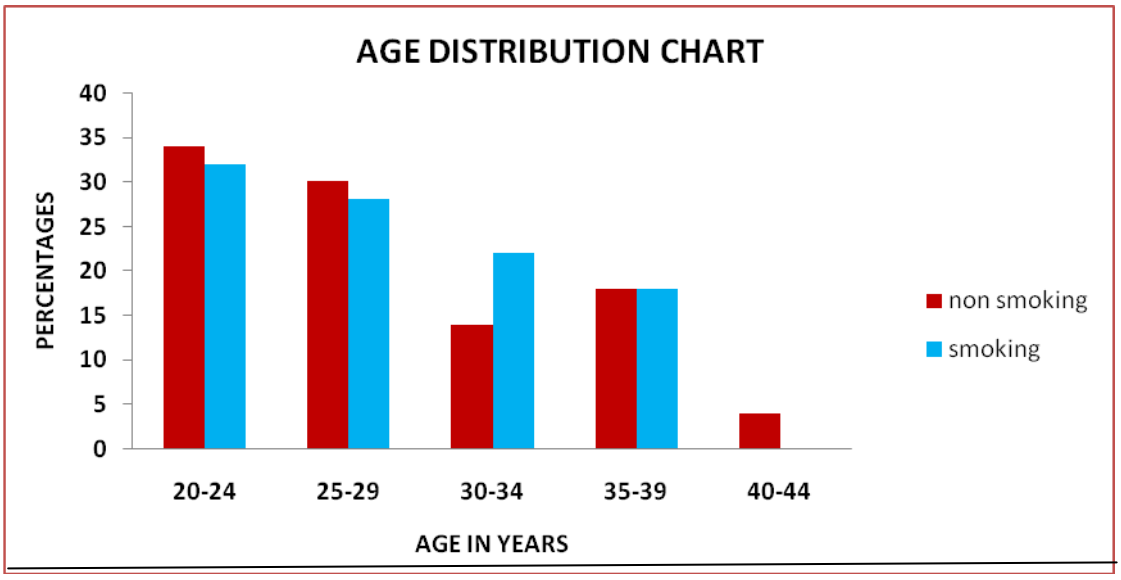


Table2: Comparison of AC thresholds in decibels between smokers & non smokers in right ear.

Frequency (right ear)	AC		
	Smokers	Non-Smokers	p value
0.25khz	18.50±6.94	13.70±4.61	<0.001**
0.5khz	20.00±6.85	13.80±3.72	<0.001**
1khz	18.30±8.84	11.60±3.42	<0.001**
2khz	19.20±9.66	11.80±5.42	<0.001**
4khz	22.50±10.11	12.90±5.98	<0.001**
6khz	23.40±10.76	12.10±5.06	<0.001**
8khz	18.50±11.17	11.90±5.14	<0.001**

Results are presented in mean ± SD, Student t test has been used to compute p value

Table 2 depicts that there significant difference in AC thresholds between smoker and non smoker groups at all frequencies in right ear.

Graph2: Comparison of AC thresholds in decibels between smokers & non smokers in right ear

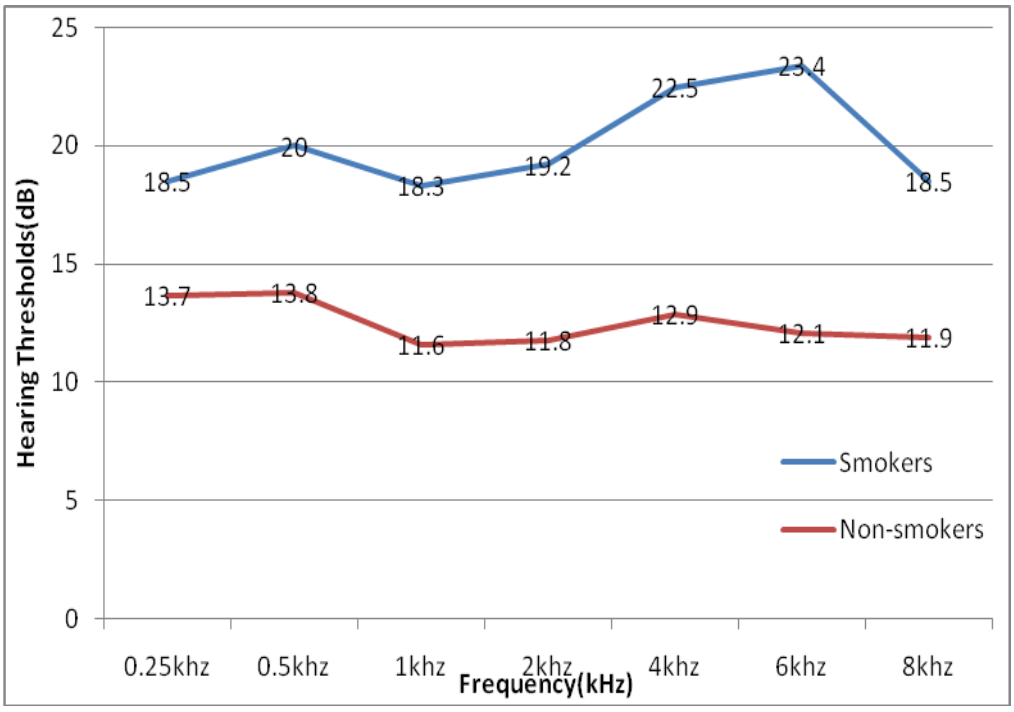


Table 3: Comparison of AC thresholds in decibels between smokers & non smokers in left ear.

Frequency (left ear)	AC		
	Smokers	**Non-Smokers**	**p value**
0.25khz	19.60±6.21	13.50±4.87	<0.001**
0.5khz	20.10±6.66	13.90±4.77	<0.001**
1khz	19.80±9.53	12.10±4.05	<0.001**
2khz	20.00±9.58	11.40±5.05	<0.001**
4khz	23.20±11.19	12.70±5.64	<0.001**
6khz	24.60±11.9	12.50±5.37	<0.001**
8khz	19.10±10.38	12.80±5.90	<0.001**

Results are presented in mean ± SD, Student t test has been used to compute p value

Table 3 depicts that there significant difference in AC thresholds between smokers and non smokers group at all frequencies in left ear.

Graph3: Comparison of AC thresholds in decibels between smokers & non smokers in left ear.

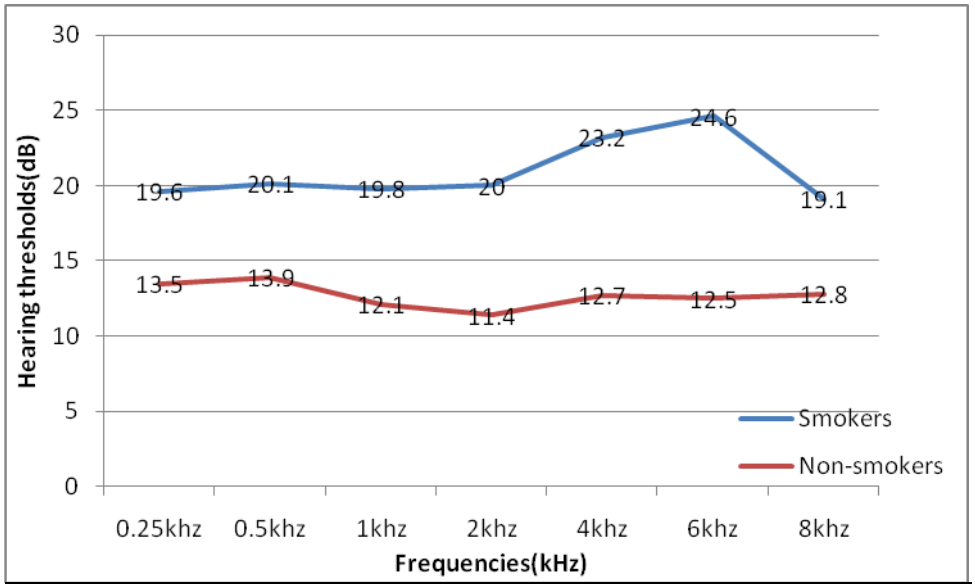


Table4: Comparison of BC thresholds in decibels between smokers & non smokers in right ear.

Frequency (right ear)	BC		
	Smokers	**Non-Smokers**	**p value**
0.25khz	10.70±5.72	6.20±2.96	<0.001**
0.5khz	11.30±5.70	6.50±2.90	<0.001**
1khz	11.40±7.22	5.50±3.07	<0.001**
2khz	11.60±8.66	5.10±3.98	<0.001**
4khz	13.20±8.68	5.50±3.68	<0.001**

Results are presented in mean ± SD, Student t test has been used to compute p value

Table 4 depicts that there significant difference in BC thresholds between smoker and non smoker groups at all frequencies in right ear.

Graph 4: Comparison of BC thresholds in decibels between smokers & non smokers in right ear

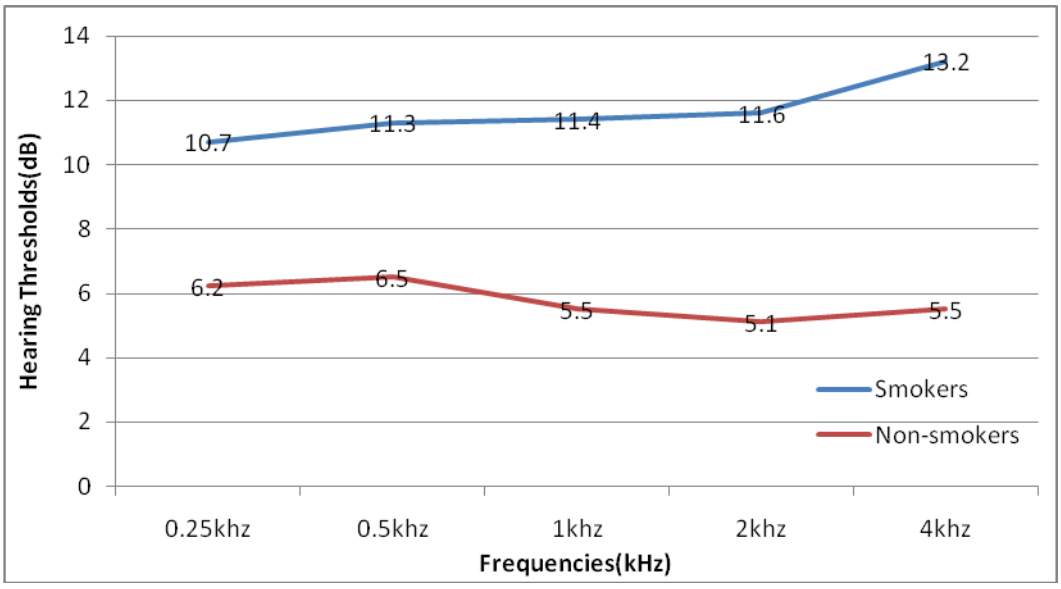


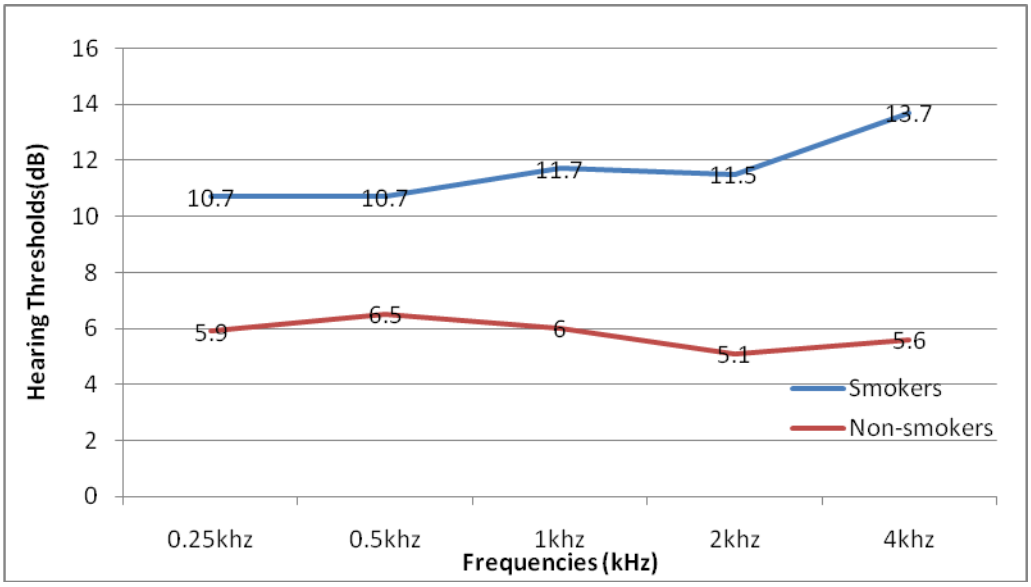
Table 5: Comparison of BC thresholds in decibels between smokers & non smokers in left ear

Frequency (left ear)	BC		
	Smokers	Non-Smokers	p value
0.25khz	10.70±5.63	5.90±3.30	<0.001**
0.5khz	10.70±5.63	6.50±3.07	<0.001**
1khz	11.70±7.26	6.00±3.19	<0.001**
2khz	11.50±8.7	5.10±3.71	<0.001**
4khz	13.70±8.85	5.60±3.87	<0.001**

Results are presented in mean ± SD, Student t test has been used to compute p value

Table 5 depicts that there significant difference in BC thresholds between smoker and non smokers groups at all frequencies in right ear.

Graph5: Comparison of BC thresholds in decibels between smokers & non smokers in left ear



For further analysis of data the smokers group were divided based on number of pack years(PY) as <2 PY, 2-5 PY, >5 PY and for nonsmokers, the number of pack years was set to zero(0 PY).[51]

Table 6: Pack years of smoking among study group:

Pack years	**Number of subjects**	**%**
0 pack years	50	50
<2.0 pack years	24	24.0
2-5 pack years	13	13.0
>5 years	13	13.0
Total	100	100.0

After which the hearing thresholds for both AC &BC of either ears of 0PY, <2PY, 2-5PY & >5PYwere compared and results have been tabulated below

Table 7: Comparison of AC and BC thresholds of right ear of study subjects (n=100) - ANOVA

Study group	AC						
	0.25 kHz	0.5 kHz	1 kHz	2 kHz	4 kHz	6 kHz	8 kHz
0PY	13.7±4.6	13.8±3.7	11.6±3.41	11.8±5.41	12.9±5.98	12.1±5.05	11.9±5.13
<2 PY	14.58±5.50	16.04±6.08	13.96±6.25	14.38±6.31	18.96±9.78	17.50±7.66	14.58±8.33
2-5 PY	21.15±5.46	23.08±5.22	18.85±6.50	21.54±7.18	22.31±9.71	24.23±9.32	16.54±8.99
>5 PY	23.08±6.93	24.23±5.72	25.77±10.17	25.77±12.39	29.23±8.13	33.46±9.66	27.69±13.01
p value	<0.001**	<0.001**	<0.001**	0.001**	0.001**	<0.001**	0.001**
	BC						
	0.25 kHz	0.5 kHz	1 kHz	2 kHz	4 kHz	6 kHz	8 kHz
0PY	6.2±2.95	6.5±2.90	5.5±3.07	5.1±3.97	5.5±3.67	-	-
<2 PY	9.58±5.50	11.04±5.51	8.75±6.12	8.33±6.37	10.00±7.94	-	-
2-5 PY	10.77±5.72	10.38±5.94	9.62±5.58	11.54±5.91	14.62±9.00	-	-
>5 PY	12.69±5.99	12.69±5.99	18.08±6.63	17.69±11.48	17.69±7.80	-	-
p value	<0.001**	<0.001**	<0.001**	<0.001**	<0.001**	-	-

Results are presented in mean ± SD, ANOVA test has been used to compute p value

In table 7 comparison between AC and BC thresholds were done between 0 pack years <2 pack years, 2-5 pack years and >5 pack years groups by using ANOVA and it depicts that there is significant difference in AC and BC thresholds between the groups at all frequencies in the right ear.

A Post-hoc Tukey test was carried out for table 7 to determine which groups differ from each other and p values were obtained

Table 7a: Comparison of auditory threshold in AC and BC for Right ear in study subjects-Post hoc analysis

	Frequency(Right ear)						
	0.25khz	**0.5khz**	**1khz**	**2khz**	**4khz**	**6khz**	**8khz**
Post –hoc analysis	**AC**						
0PY **Vs** <2 PY	0.926	0.26	0.6	0.6	0.14	0.04*	0.8
0PY **Vs** 2-5PY	<0.0001*	<0.0001*	<0.0001**	<0.0001**	<0.0001*	<0.0001**	0.012*
0PY **Vs** > 5PY	<0.0001*	<0.0001*	<0.0001**	<0.0001**	<0.0001*	<0.0001**	<0.0001**
<2 PY **Vs** 2-5PY	0.006**	0.003**	0.153	0.046*	0.557	0.071	0.835
<2PY **Vs** > 5PY	<0.001*	<0.001**	<0.001**	0.418	0.007**	<0.001**	0.001**
2-5 PY **Vs** >5PY	0.685	0.867	0.059	0.418	0.154	0.024*	0.016*
Post –hoc analysis	**BC**						
0PY **Vs** <2PY	0.06	0.03	0.13	0.34	0.18	-	-
0 PY **Vs** 2-5PY	0.01*	0.06	0.05*	0.01*	<0.0001**	-	-
0 PY **Vs** >5PY	<0.0001*	<0.0001*	<0.0001*	<0.0001**	<0.0001**	-	-
<2 PY **Vs** 2-5PY	0.818	0.941	0.913	0.472	0.241	-	-
<2PY **Vs** > 5PY	0.261	0.684	<0.001**	0.003**	0.024*	-	-
2-5 PY **Vs** >5PY	0.666	0.566	0.003**	0.127	0.607	-	-

p values are obtained based on Post –hoc Tukey test

In table 7a it is depicted that there is no significant difference between AC and BC thresholds 0 pack years and < 2 pack years of smoking at all frequencies except 6 kHz (AC) in right ear but significant differences of AC and BC thresholds were found between 0 pack years and smokers with 2-5 pack years, >5 pack years at all frequencies.

Table 8: Comparison of AC and BC thresholds of left ear of study subjects (n=100) –ANOVA

Study group	AC						
	0.25 kHz	0.5 kHz	1 kHz	2 kHz	4 kHz	6 kHz	8 kHz
0PY	13.5±4.82	13.9±4.76	12.1`±4.05	11.4±5.05	12.7±5.62	12.5±5.36	12.8±5.90
<2 PY	16.67±4.82	16.67±6.37	16.04±6.75	15.21±6.51	18.33±9.52	17.92±8.71	15.21±8.01
2-5 PY	22.31±4.84	23.46±4.27	20.38±5.19	23.08±6.93	26.15±9.82	26.54±9.87	19.23±8.62
>5 PY	22.31±7.53	23.08±6.30	26.15±13.56	25.77±12.39	29.23±12.05	35.00±11.18	26.15±12.61
p value	<0.001**	<0.001**	<0.001**	<0.001**	<0.001**	<0.001**	0.001**
	BC						
	0.25 kHz	0.5 kHz	1 kHz	2 kHz	4 kHz	6 kHz	8 kHz
0PY	5.9±3.30	6.5±3.07	6.0±3.19	5.1±3.70	5.6±3.86	-	-
<2 PY	9.79±5.00	9.79±4.77	8.96±6.59	8.13±5.86	10.21±8.91	-	-
2-5 PY	11.92±6.30	10.38±5.94	10.77±5.34	11.92±7.23	14.62±6.60	-	-
>5 PY	11.15±6.18	12.69±6.65	17.69±6.96	17.31±11.48	19.23±8.13	-	-
p value	<0.001**	<0.001**	<0.001**	<0.001**	<0.001**	-	-

Results are presented in mean ± SD, ANOVA test has been used to compute p value

In table 8 comparison between AC and BC thresholds were done between 0 pack years , <2 pack years, 2-5 pack years and >5 pack years groups by using ANOVA and it depicts that there is significant difference in AC and BC thresholds between the groups at all frequencies in the left ear.

A Post-hoc Tukey test was carried out for table 8 to determine which groups differ from each other and p values were obtained

Table 8a: Comparison of auditory threshold in AC and BC for left ear in study subjects-Post –hoc analysis

	Frequency(Right ear)						
	0.25khz	**0.5khz**	**1khz**	**2khz**	**4khz**	**6khz**	**8khz**
Post –hoc analysis	**AC**						
0 PY **Vs** <2 PY	0.07	0.27	0.26	0.34	0.26	0.124	0.87
O PY **Vs** 2-5PY	<0.0001**	<0.0001**	<0.0001**	<0.0001**	<0.0001**	<0.0001**	0.12
O PY **Vs** > 5PY	<0.0001**	<0.0001**	<0.0001**	<0.0001**	<0.0001**	<0.0001**	<0.0001**
<2 PY **Vs** 2-5PY	0.015*	0.004**	0.327	0.026*	0.081	0.034*	0.445
<2PY **Vs** > 5PY	0.015*	0.008**	0.004**	0.002**	0.010*	<0.001**	0.005**
2-5 PY **Vs** >5PY	1.000	0.985	0.221	0.700	0.728	0.077	0.164
Post –hoc analysis	**BC**						
0PY **Vs** <2PY	0.22	0.07	0.23	0.42	0.16	-	-
0PY **Vs** 2-5PY	0.01*	0.005*	0.002*	0.001*	<0.0001**	-	-
0 PY **Vs** >5PY	<0.0001**	<0.0001**	<0.0001**	<0.0001**	<0.0001**	-	-
<2 PY **Vs** 2-5PY	0.523	0.949	0.691	0.359	0.270	-	-
<2PY **Vs** > 5PY	0.766	0.299	0.001**	0.005**	0.007**	-	-
2-5 PY **Vs** >5PY	0.936	0.550	0.022*	0.209	0.329	-	-

p values are obtained based on Post –hoc Tukey test

In table 8a it is depicted that there is no significant difference between AC and BC thresholds between 0 pack years and smokers with < 2 pack years of smoking at all frequencies in left ear but significant differences of AC and BC thresholds were found between 0 pack years and smokers with 2-5 pack years and >5 pack years of smoking at all frequencies.

Above tables 7,7a, 8, 8a indicate that if a subject has smoked regularly for a period of 2 or more years will induce changes in both auditory air and bone conduction thresholds.

Table 9: Pearson's correlation between Pack years of smoking and AC thresholds in right ear of smokers (n=50).

Pack years	**Frequency(Right ear)**						
	0.25khz	**0.5khz**	**1khz**	**2khz**	**4khz**	**6khz**	**8khz**
AC							
r value	0.355	0.334	0.416	0.366	0.398	0.555	0.390
p value	0.011*	0.016*	0.003**	0.009**	0.004**	<0.001**	0.005**

Table 10: Pearson's correlation between Pack years of smoking and AC thresholds in left ear of smokers (n=50).

Pack years	**Frequency(left ear)**						
	0.25khz	**0.5khz**	**1khz**	**2khz**	**4khz**	**6khz**	**8khz**
AC							
r value	0.337	0.366	0.326	0.382	0.379	0.475	0.408
p value	0.017*	0.009**	0.021*	0.006**	0.007**	<0.001**	0.003**

Table 9&10 shows Pearsons' correlation of pack years with AC thresholds of smokers in both right and left ear. These tables show that there is positive correlation at all frequencies with highly significant p values.

Table 11: Pearson's correlation between Pack years of smoking and BC thresholds in right ear of smokers (n=50)

Pack years	**Frequency(Right ear)**				
	0.25khz	**0.5khz**	**1khz**	**2khz**	**4khz**
BC					
r value	0.239	0.151	0.497	0.393	0.355
p value	0.095+	0.297	<0.001**	0.005**	0.011*

Table12: Pearson's correlation between Pack years of smoking and BC thresholds in left ear of smokers (n=50)

Pack years	**Frequency(left ear)**				
	0.25khz	**0.5khz**	**1khz**	**2khz**	**4khz**
BC					
r value	0.173	0.217	0.481	0.408	0.407
p value	0.230	0.130	<0.001**	0.003**	0.003**

Table 11&12 shows Pearson correlation of pack years with BC thresholds of smokers in both right and left ear. These tables show that there is small positive correlation at all frequencies and there is a highly significant p value at frequencies of 1, 2 &4 kHz.

DISCUSSION

DISCUSSION

Cigarette smoking is an important risk factor for chronic lung diseases and cardiovascular diseases. Relatively little data exists on the exposure of cigarette smoking as a risk factor for hearing loss, although link was established over 40 years ago. Information on the effects of smoking at the cochlear and auditory central nervous system levels has become available only recently.[5,6,7] Hence this study was an attempt to observe the association between smoking and changes in hearing thresholds in a sample population of Kolar.

Pure tone audiometry was used to record auditory thresholds in our study, which is a simple, inexpensive, qualitative and quantitative procedure. Study included age matched samples between 20-40 years, as hearing loss is an inevitable consequence of aging. With increasing age there is physiological alteration in hearing acuity called "presbyacusis".

Present study showed that there was statistically significant difference in auditory air and bone conduction thresholds at all frequencies for both right and left ear between smokers and non smokers. The smoker group had higher threshold of hearing at all frequencies as compared to age matched non smokers indicating smoking affects both air and bone conduction auditory thresholds. Similar results were shown in a study which compared the auditory thresholds, within a group of male smokers and non-smokers aged between 18 and 40 years.[52]

Studies have also shown that the percentage of hearing loss was greater for smokers at all measured frequencies .[41] A longitudinal study done on non hearing impaired Japanese male office workers over a period of 5 year showed that the relative risk of low and high frequency hearing loss was higher among smokers as compared to never smokers.[6] Also, men who smoked more than one pack per day had

worse hearing thresholds at 250 to 1000 Hz than non smokers or light smokers but no difference at higher frequencies was documented in another study.[44] Association of smoking with higher prevalence and incidence of hearing loss was shown by various cross sectional, prospective and case control studies in literature.[29, 42, 43]

The possible explanation for conductive hearing loss among smokers may be a higher prevalence of rhino sinusitis or eustachian tube dysfunction. [47,48,49,50] Sensorineural hearing loss might be due to an oxidative damage caused by toxic substances inhaled with the cigarette smoke.[7]

There is no direct evidence for the mechanisms of damage to the auditory system associated with cigarette smoke exposure. It may be related to hypoxia; both nicotine & carbon monoxide in cigarette smoke may induce vasospasms and deplete oxygen levels to the cochlea as well as spiral ganglion cells.[17,18,19,20] Recent studies have also indicated that the mechanism that may be due to the interaction between nicotine and nicotinic acetylcholine receptors (nAChRs) within the auditory system. Nicotine binds to nAChRs that normally modulate the effects of a neurotransmitter called acetylcholine. Since neurotransmitters function as chemical message carriers facilitating communication between cells by binding to the receptors on the cell surface, loss or damage of the receptors would eliminate the modulatory influences of the receptors. There is now evidence that nAChRs are critical components of the auditory pathway, from the cochlea to the temporal lobe, and the descending auditory pathway.[21,22] Moreover, emerging data indicates that prenatal exposure to nicotine or chronic nicotine use during adolescence damages the nAChR binding sites, producing cognitive impairments in the auditory and visual modalities.[23,24,25]Finally, the neurophysiological mechanism that may potentially explain the association between adolescent smoking and neurocognitive deficits is protracted development of the

auditory central nervous system pathways. There is evidence that many components of auditory central nervous system development, including the auditory thalamocortical and cortigofugal pathways, continue into late adolescence .[24, 25, 26] Moreover, these pathways are particularly susceptible to damage, if environmental toxins like nicotine are introduced during their developmental emergence.[23, 27]

Studies have also found negative association between smoking and hearing loss, The longitudinal study of aging done at Baltimore, found no association between cigarette smoking and development of hearing loss in 531 white men.[45]A study which tested hearing with audiometry done at Framingham showed that there is no association between cigarette smoking and hearing loss .[46] A possible explanation for this discrepancy in results may be the different methodologies used in the studies and the fact that they related to different populations (Spanish and North Americans) at different ages.

On further analysis of our study, The subjects were further divided based on pack years (PY) into 0 PY, <2 PY, 2-5 PY, >5 PY.ANOVA revealed that there was significant difference in hearing thresholds between the groups at all frequencies in the right ear and left ear. Later on Post-hoc Tukey test was carried out to determine which groups differ from each other .It showed that there was no significant difference between hearing thresholds at 0 PY and < 2 PY of smoking at all frequencies in both right and left ear but significant differences of hearing thresholds were found between 0PY and 2-5 PY, 0PY&>5PY,<2PY&2-5PY,<2PY&>5PY indicating that if a subject has smoked regularly for a period of 2 or more years it induces changes in both auditory air and bone conduction thresholds.Although earlier studies have shown changes to set in at as early as one year .[51]

Post hoc analysis showed that there is no significant difference of hearing

thresholds between 2-5PY and >5PY. But it was observed that there was positive correlation of pack years of smoking with air conduction thresholds at all frequencies and bone conduction thresholds at higher frequencies(1,2 &4 kHz) of smokers in both right and left ear .Various studies in the past have also shown positive correlation between pack years and hearing loss.[5, 6, 32] Because of the smaller sample size and unequal distribution of samples i.e 0PY(n=50), <2PY (n=24), 2-5PY (n=13), >5PY(n=13) these results need to be confirmed by population based studies.

Present study included only male subjects; consequently generalization cannot be made across genders. Pure tone audiometry is a subjective test and there might be chances of error while recording auditory thresholds. Results need to be confirmed by objective tests like BERA, OAE's.

Cigarette smoking, a well known risk factor for chronic diseases affects hearing sensitivity. Among the various environmental factors associated with hearing loss, smoking is important preventable risk factor hence cessation of smoking habits at the earliest may prevent decline in hearing sensitivity.

SUMMARY AND CONCLUSION

SUMMARY

The present study was conducted in department of physiology SDUMC, Kolar which aimed at observing the association between smoking and changes in hearing thresholds in a sample population of Kolar. The study included 50 cigarette smokers and 50 age matched non smokers recruited based on various inclusion and exclusion criteria. After taking smoking history subjects underwent detailed general systemic and ENT examinations after which they were subjected to pure tone audiometric evaluation. The data thus obtained were stastically analysed which revealed that there is statistically significant difference in auditory air and bone conduction thresholds at all frequencies for both right and left ear , the smoker group having higher thresholds at all frequencies compared to age matched non smokers indicating smoking affects both air and bone conduction auditory thresholds. Study also indicates that if a subject has smoked regularly for a period of 2 or more years it induces changes in both auditory air and bone conduction thresholds. Further, it showed that there is positive correlation of pack years of smoking with air conduction thresholds at all frequencies and bone conduction thresholds at higher frequencies (1 kHz, 2kHz, 4kHz) in both right and left ears. Hence, Smoking is important preventable risk factor for hearing loss therefore cessation of smoking habits may prevent or delay age related declines in hearing sensitivity.

CONCLUSIONS:

The present study aimed at observing the association between smoking and changes in auditory thresholds in a sample population of Kolar revealed that,

1. Smoking causes increase in both air conduction and bone conduction thresholds at all frequencies.
2. If a subject has smoked regularly for a period of 2 or more years it induces changes in both auditory air and bone conduction thresholds.

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ANNEXURES

QUESTIONNAIRE

1. NAME:
2. AGE:
3. SEX:
4. ADDRESS:
5. OCCUPATION:
6. ARE YOU A DIABETIC: YES/NO
7. ARE YOU HYPERTENSIVE: YES/NO
8. DO YOU SMOKE CIGARETTES:YES/NO

   If yes,

   a) Duration of smoking in years:

   b) Average Number of cigarettes smoked per day:
9. DO YOU CONSUME ANY DRUGS REGULARLY :YES/NO

   If yes, Name of drugs and reason for consumption
10. DO YOU HAVE ANY HISTORY OF ENT INFECTIONS IN PAST 3 MONTHS
11. DO YOU HAVE ANY HISTORY OF INJURY IN THE PAST 3 MONTHS FOR WHICH YOU WERE HOSPITALISED:YES/NO
12. DO YOU HAVE HISTORY OF SURGERY IN PAST:YES/NO

    If yes, Reason:
13. DO YOU HAVE HISTORY OF EXPOSURE TO NOISE:YES/NO

MASTER CHART FOR SMOKERS IN RIGHT EAR

Sl.no	Age	No of cigars/ day	No of years	Pack years	0.25khz		0.5khz		1khz		2khz		4khz		6khz		8khz	
					AC	BC	AC	BC	AC	BC	AC	BC	AC	BC	AC	BC	AC	BC
1	24	4	3	0.6	20	15	20	20	15	10	10	5	10	5	15	-	15	-
2	22	2	2	0.2	5	0	5	5	10	0	10	0	10	0	10	-	10	-
3	24	5	5	1.25	15	10	10	10	10	5	10	5	10	5	10	-	5	-
4	29	5	10	2.5	20	0	25	0	20	0	15	0	10	5	15	-	5	-
5	35	20	15	17	20	15	20	15	25	20	25	20	30	20	35	-	25	-
6	36	10	12	6	30	20	30	20	40	25	45	35	40	30	40	-	45	-
7	35	8	5	2	15	15	15	15	20	15	20	15	40	25	25	-	25	-
8	30	6	10	3	25	15	25	15	20	15	25	15	25	20	35	-	15	-
9	29	10	15	7.5	15	5	20	5	15	10	15	5	30	10	30	-	15	-
10	30	6	10	3	20	20	20	20	15	15	20	20	25	15	20	-	15	-
11	28	4	5	1	10	5	10	5	10	10	15	5	25	10	25	-	15	-
12	32	9	10	4.5	15	10	20	10	15	10	15	10	15	10	15	-	10	-
13	20	4	6	1.2	25	10	30	10	25	15	25	15	40	20	35	-	35	-
14	29	10	11	5.5	20	15	20	10	15	15	15	15	15	15	15	-	10	-
15	21	4	4	0.8	15	10	15	5	15	10	20	10	20	10	25	-	10	-
16	20	3	1	0.15	20	15	25	15	15	5	10	10	20	5	20	-	15	-
17	28	15	10	7.5	15	5	15	5	15	10	10	5	15	10	15	-	15	-
18	22	1	2	0.1	10	10	15	10	10	0	15	0	20	5	15	-	10	-
19	21	5	5	1.25	10	10	15	5	0	0	5	5	15	5	10	-	10	-
20	22	2	4	0.4	15	10	20	10	10	5	10	5	15	10	15	-	10	-
21	28	5	6	1.5	10	5	20	15	15	15	15	15	20	20	20	-	5	-
22	24	2	10	1	10	5	10	5	5	5	5	0	5	0	5	-	5	-
23	24	4	3	0.6	20	15	20	20	15	10	10	5	10	5	15	-	15	-
24	22	2	2	0.2	5	0	5	5	10	0	10	0	10	0	10	-	10	-
25	24	5	5	1.25	15	10	10	10	10	5	10	5	10	5	10	-	5	-
26	29	5	10	2.5	20	0	25	0	20	0	15	0	10	5	15	-	5	-
27	35	24	15	18	20	15	20	15	25	20	25	20	30	20	35	-	25	-
28	36	10	12	6	30	20	30	20	40	25	45	35	40	30	40	--	45	--
29	35	8	5	2	15	15	15	15	20	15	20	15	40	25	25	-	25	-

30	30	6	10	3	25	15	25	15	20	15	25	15	25	30	35	-	15	-
31	29	10	15	7.5	15	5	20	5	15	10	15	5	30	10	30	-	15	-
32	28	16	5	4	15	10	20	10	5	5	10	10	15	10	15	-	15	-
33	32	10	10	5	20	10	25	10	20	15	25	15	45	25	35	-	20	-
34	28	4	5	1	15	5	15	5	10	10	10	10	15	15	15	-	20	-
35	30	4	2	0.4	20	15	20	15	15	10	20	15	20	15	20	-	15	-
36	37	10	15	7.5	30	15	30	15	30	25	25	15	25	15	45	-	35	-
37	35	6	6	1.8	15	15	20	20	15	15	15	15	15	10	25	-	15	-
38	32	3	8	1.2	25	20	25	20	30	25	30	25	25	25	30	-	30	-
39	26	1	6	0.3	10	10	15	10	15	10	10	5	15	5	10	-	5	-
40	34	10	10	5	15	10	15	10	10	5	15	10	15	10	15	-	15	-
41	32	6	10	3	20	10	25	10	30	10	35	15	30	10	25	-	25	-
42	36	10	12	6	30	20	30	20	40	25	45	35	40	30	40	-	45	-
43	34	6	10	3	25	15	25	15	20	15	25	15	25	30	35	-	15	-
44	29	10	15	7.5	15	5	20	5	15	10	15	5	30	10	30	-	15	-
45	37	10	15	7.5	30	15	30	15	30	25	25	15	25	15	45	-	35	-
46	28	6	10	3	20	10	15	5	25	10	25	10	20	5	20	-	20	-
47	25	2	2	0.2	10	0	15	5	15	10	20	10	25	5	10	-	15	-
48	37	10	12	6	30	10	30	15	30	15	30	20	30	15	35	-	35	-
49	25	1	5	0.3	20	5	15	10	20	5	20	5	20	10	20	-	25	-
50	39	10	10	5	35	15	35	15	25	10	30	15	30	15	35	-	40	-

MASTER CHART FOR SMOKERS IN LEFT EAR

Sl.no	Age	No of cigars/ day	No of years	Pack years	0.25 khz		0.5 khz		1 khz		2 khz		4 khz		6 khz		8 khz	
					AC	BC	AC	BC	AC	BC	AC	BC	AC	BC	AC	BC	AC	BC
1	24	4	3	0.6	25	15	30	20	20	10	15	5	15	5	15	-	20	-
2	22	2	2	0.2	10	0	5	5	10	0	10	0	5	0	5	-	5	-
3	24	5	5	1.25	15	10	15	10	10	5	15	5	15	5	15	-	15	-
4	29	5	10	2.5	20	0	20	0	20	0	15	0	20	5	15	-	5	-
5	35	20	15	17	25	15	25	15	25	20	25	20	30	20	30	-	30	-
6	36	10	12	6	30	20	30	20	45	25	45	35	45	30	45	-	40	-
7	35	8	5	2	15	15	20	15	25	15	25	15	40	25	30	-	25	-
8	30	6	10	3	25	15	25	15	25	15	25	15	30	20	35	-	20	-
9	29	10	15	7.5	15	5	15	5	10	10	15	5	15	10	30	-	10	-
10	30	6	10	3	20	20	20	20	15	15	20	15	20	15	40	-	30	-
11	28	4	5	1	15	5	15	5	10	10	20	5	15	10	20	-	10	-
12	32	9	10	4.5	15	15	20	15	10	10	20	15	15	10	15	-	10	-
13	20	4	6	1.2	20	10	15	10	25	15	20	15	30	20	30	-	25	-
14	29	10	11	5.5	20	5	20	5	15	15	15	15	15	15	15	-	10	-
15	21	4	4	0.8	20	10	15	5	15	10	15	10	20	10	15	-	5	-
16	20	3	1	0.15	15	15	15	15	10	5	10	10	15	5	15	-	10	-
17	28	15	10	7.5	10	5	15	5	15	10	10	5	20	10	20	-	20	-
18	22	1	2	0.1	10	10	10	10	10	0	10	0	20	5	15	-	15	-
19	21	5	5	1.25	10	10	15	5	25	0	15	5	15	5	15	-	10	-
20	22	2	4	0.4	10	10	10	10	10	5	10	5	20	10	20	-	25	-
21	28	5	6	1.5	15	5	20	15	20	20	20	20	25	25	20	-	15	-
22	24	2	10	1	15	5	10	5	5	5	5	0	5	0	5	-	0	-
23	24	4	3	0.6	25	15	30	20	20	10	15	5	15	5	5	-	20	-
24	22	2	2	0.2	10	0	5	5	10	0	10	0	5	0	15	-	5	-
25	24	5	5	1.25	15	10	15	10	10	5	15	5	15	5	5	-	15	-
26	29	5	10	2.5	20	0	20	0	20	0	15	0	20	5	15	-	5	-
27	35	24	15	18	25	15	25	15	25	20	25	20	30	20	30	-	30	-

28	36	10	12	6	30	20	30	20	45	25	45	35	45	30	45	--	40	--
29	35	8	5	2	15	15	20	15	25	15	25	15	40	25	30	-	25	-
30	30	6	10	3	25	15	25	15	25	15	25	15	30	20	35	-	20	-
31	29	10	15	7.5	15	5	15	5	10	10	15	5	15	10	30	-	10	-
32	28	16	5	4	20	5	20	5	15	10	25	5	30	20	35	-	30	-
33	32	10	10	5	25	10	25	10	25	10	30	15	45	25	30	-	20	-
34	28	4	5	1	20	5	15	5	15	15	10	10	15	10	20	-	15	-
35	30	4	2	0.4	20	15	15	10	20	15	10	10	15	10	25	-	25	-
36	37	10	15	7.5	20	10	25	15	30	25	25	15	35	25	50	-	30	-
37	35	6	6	1.8	20	20	20	10	15	10	15	10	15	15	20	-	15	-
38	32	3	8	1.2	25	10	25	10	30	25	35	20	35	30	40	-	30	-
39	26	1	6	0.3	20	10	20	10	15	10	10	10	15	15	20	-	5	-
40	34	10	10	5	20	15	20	10	15	15	15	15	20	15	20	-	15	-
41	32	6	10	3	20	15	25	10	25	10	25	15	30	15	20	-	25	-
42	36	10	12	6	30	20	30	20	45	25	45	35	45	30	45	-	40	-
43	30	6	10	3	25	15	25	15	25	15	25	15	30	20	35	-	20	-
44	29	10	15	7.5	15	5	15	5	10	10	15	5	15	10	30	-	10	-
45	37	10	15	7.5	20	10	25	15	30	25	25	15	35	25	50	-	30	-
46	28	6	10	3	20	15	25	10	20	15	20	5	10	5	15	-	20	-
47	25	2	2	0.2	15	5	20	5	10	5	15	5	15	0	15	-	15	-
48	37	10	12	6	35	10	30	20	35	10	30	15	35	15	35	-	40	-
49	25	1	5	0.3	20	10	20	5	20	5	15	10	15	5	15	-	15	-
50	39	10	10	5	35	15	35	10	25	10	40	25	40	15	35	-	30	-

MASTER CHART FOR CONTROLS IN RIGHT EAR:

SL.NO	AGE	0.25 khz		0.5 khz		1 khz		2 khz		4 khz		6 khz		8 khz	
		AC	BC	AC	BC	AC	BC	AC	BC	AC	BC	AC	BC	AC	BC
1	27	5	5	10	5	10	5	5	5	5	5	10	-	10	-
2	27	15	0	10	5	10	5	5	5	10	5	10	-	5	-
3	32	15	10	10	5	10	5	15	10	20	5	15	-	15	-
4	36	20	5	15	10	15	10	20	5	15	5	15	-	15	-
5	29	15	10	20	5	15	5	20	10	15	5	15	-	15	-
6	21	15	10	15	5	15	5	20	10	20	10	15	-	15	-
7	30	20	5	20	10	10	5	15	5	20	10	15	-	15	-
8	32	10	5	15	10	15	5	10	5	15	5	10	-	10	-
9	21	10	5	15	5	15	5	15	5	20	5	20	-	20	-
10	26	20	5	20	10	15	5	10	5	20	10	20	-	20	-
11	23	10	5	15	5	10	0	10	0	10	5	10	-	10	-
12	21	10	0	10	5	5	0	5	0	5	0	5	-	5	-
13	22	20	5	15	5	15	5	15	0	5	0	5	-	5	-
14	21	20	5	15	5	10	5	5	0	5	0	5	-	5	-
15	23	15	5	15	5	15	5	5	0	5	0	5	-	5	-
16	27	10	10	15	5	10	10	5	5	5	5	5	-	5	-
17	39	10	5	10	5	10	5	5	5	10	5	5	-	5	-
18	35	15	10	15	10	10	5	5	0	20	10	15	-	10	-
19	24	10	5	20	5	15	10	10	5	10	5	10	-	10	-
20	40	10	10	10	5	10	5	10	5	20	15	15	-	10	-
21	26	10	5	15	10	10	10	5	5	10	10	15	-	20	-
22	27	10	5	10	5	10	0	5	0	10	5	10	-	10	-
23	27	5	5	10	5	10	5	5	5	5	5	10	-	10	-
24	27	15	0	10	5	10	5	5	5	10	5	10	-	5	-
25	32	15	10	10	5	10	5	15	10	20	5	15	-	15	-
26	36	20	5	15	10	15	10	20	5	15	5	15	-	15	-
27	29	15	10	20	5	15	5	20	10	15	5	15	-	15	-
28	21	15	10	15	5	15	5	20	10	20	10	15	--	15	--
29	30	20	5	20	10	10	5	15	5	20	10	15	-	15	-
30	32	10	5	15	10	15	5	10	5	15	5	10	-	10	-
31	21	10	5	15	5	15	5	15	5	20	5	20	-	20	-
32	27	15	5	15	5	10	10	15	5	15	5	5	-	5	-

33	35	20	10	15	10	10	10	15	10	20	15	20	-	20	-
34	21	10	10	10	10	10	10	10	10	5	5	10	-	10	-
35	26	15	10	15	15	15	15	15	15	10	10	15	-	15	-
36	21	15	10	15	5	15	5	20	10	20	10	15	-	15	-
37	30	20	5	20	10	10	5	15	5	20	10	15	-	15	-
38	22	20	5	15	5	15	5	15	0	5	0	5	-	5	-
39	21	20	5	15	5	10	5	5	0	5	0	5	-	5	-
40	32	15	5	15	0	10	0	15	5	15	5	15	-	10	-
41	25	10	5	10	5	5	5	5	0	5	0	5	-	5	-
42	22	5	0	5	5	5	0	5	5	5	5	5	-	10	-
43	26	5	5	5	5	5	5	10	0	5	0	10	-	10	-
44	36	15	10	15	10	15	5	20	15	15	5	15	-	15	-
45	26	10	5	10	5	5	5	10	5	10	5	10	-	10	-
46	30	10	5	10	0	10	5	15	5	10	0	10	-	10	-
47	37	15	5	15	10	10	5	15	0	15	5	15	-	20	-
48	38	15	10	15	10	20	10	15	10	20	5	20	-	20	-
49	39	20	10	15	5	15	5	15	5	20	5	25	-	20	-
50	30	10	5	10	5	10	0	10	0	10	5	10	-	15	-

MASTER CHART FOR CONTROLS LEFT EAR:

SL.NO	AGE	0.25 khz		0.5 khz		1 khz		2 khz		4 khz		6 khz		8 khz	
		AC	BC	AC	BC	AC	BC	AC	BC	AC	BC	AC	BC	AC	BC
1	27	5	5	5	5	5	5	5	5	5	5	5	-	5	-
2	27	15	0	10	5	10	5	10	5	15	5	10	-	10	-
3	32	15	10	10	5	10	5	20	10	15	5	15	-	20	-
4	36	15	5	20	10	20	10	15	5	15	5	15	-	20	-
5	29	20	10	15	5	15	5	15	10	15	5	15	-	20	-
6	21	15	10	20	5	15	5	15	10	20	10	20	-	20	-
7	30	20	5	20	10	15	5	15	5	20	10	15	-	10	-
8	32	20	5	15	10	15	5	20	5	20	5	15	-	20	-
9	21	10	5	10	5	15	5	20	5	20	5	20	-	20	-
10	26	20	5	20	10	15	5	10	5	20	10	15	-	10	-
11	23	15	5	15	5	5	0	5	0	10	5	10	-	10	-
12	21	15	0	15	5	5	0	5	0	5	0	5	-	5	-
13	22	20	5	20	5	15	5	10	0	5	0	5	-	5	-
14	21	5	5	15	5	10	5	5	0	5	0	5	-	5	-
15	23	15	5	15	5	15	5	5	0	5	0	5	-	5	-
16	27	10	10	10	5	10	10	5	5	10	5	5	-	5	-
17	39	5	5	10	5	5	5	5	5	5	5	5	-	5	-
18	35	10	10	20	10	10	5	5	0	10	5	10	-	10	-
19	24	15	5	15	5	10	10	10	5	15	5	10	-	10	-
20	40	15	10	15	5	10	5	10	5	20	15	15	-	10	-
21	26	15	5	10	10	10	10	5	5	10	10	15	-	20	-
22	27	10	5	5	5	10	0	5	0	15	5	10	-	10	-
23	27	5	5	5	5	5	5	5	5	5	5	5	-	5	-
24	27	15	0	10	5	10	5	10	5	15	5	10	-	10	-
25	32	15	10	10	5	10	5	20	10	15	5	15	-	20	-
26	36	15	5	20	10	20	10	15	5	15	5	15	-	20	-
27	29	20	10	15	5	15	5	15	10	15	5	15	-	20	-
28	21	15	10	20	5	15	5	15	10	20	10	20	--	20	--
29	30	20	5	20	10	15	5	15	5	20	10	15	-	10	-
30	32	20	5	15	10	15	5	20	5	20	5	15	-	20	-
31	21	10	5	10	5	15	5	20	5	20	5	20	-	20	-

32	27	15	5	15	15	15	15	10	5	10	10	15	-	10	-
33	35	20	10	20	10	20	10	15	15	15	15	20	-	20	-
34	21	15	15	15	15	15	15	10	10	10	10	20	-	15	-
35	26	10	10	15	10	15	10	10	10	10	10	15	-	10	-
36	21	15	10	20	5	15	5	15	10	20	10	20	-	20	-
37	30	20	5	20	10	15	5	15	5	20	10	15	-	10	-
38	22	20	5	20	5	15	5	10	0	5	0	5	-	5	-
39	21	5	5	15	5	10	5	5	0	5	0	5	-	5	-
40	32	10	5	10	5	15	10	10	5	10	0	10	-	10	-
41	25	5	0	5	0	5	5	10	5	10	0	10	-	15	-
42	22	5	0	5	5	10	5	5	0	10	5	0	-	5	-
43	26	10	5	10	0	5	0	10	0	0	5	15	-	10	-
44	36	10	5	15	5	10	5	15	5	10	5	10	-	15	-
45	26	15	0	10	5	10	5	10	0	10	0	10	-	10	-
46	30	10	5	10	5	10	5	10	5	10	0	10	-	10	-
47	37	15	5	15	5	15	10	10	5	10	5	20	-	15	-
48	38	15	5	15	10	15	10	20	10	20	5	20	-	20	-
49	39	10	10	15	5	10	5	10	5	10	10	15	-	20	-
50	30	10	5	10	5	10	5	15	10	15	5	15	-	15	-