EFFECT OF ROAD TRAFFIC NOISE EXPOSURE ON BRAINSTEM AUDITORY EVOKED POTENTIALS IN TRAFFIC POLICEMEN

By

Dr. KAVANA G. VENKATAPPA, MBBS

Dissertation submitted to the

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DOCTOR OF MEDICINE
IN
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Under the guidance of
Dr. VINUTHA SHANKAR.M.S. MD
PROFESSOR

DEPARTMENT OF PHYSIOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE, KOLAR

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Dr. Vinutha Shankar.M.S. **MD**
Professor
Department of Physiology

Date :

Place : Kolar

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This is to certify that, the Ethical committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has unanimously approved **Dr. Kavana G. Venkatappa**, Post-Graduate student in the Department of PHYSIOLOGY of Sri Devaraj Urs Medical College to take up the dissertation work entitled "**Effect of road traffic noise exposure on brainstem auditory evoked potentials in traffic policemen"** to be submitted to the Sri Devaraj Urs Academy of Higher Education and Research, Tamaka, Kolar.

Member Secretary *Principal*

Date :
Place : Kolar

A WORD OF GRATITUDE

To my respected Teacher and Guide

Dr. Vinutha Shankar M.S. M.D
Professor
Department of Physiology
Sri Devaraj Urs Medical College,
Kolar.

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Dr. Kavana G. Venkatappa

Date :
Place : Kolar

LIST OF ABBREVIATIONS

BAEP	-	BrainStem Auditory Evoked Potentials
BAER	-	Brainstem Auditory Evoked Response
ABR	-	Auditory Brain Stem Response
AVCN	-	Anterior Ventral Cochlear Nucleus
PVCN	-	Posterior ventral cochlear nucleus
DCN	-	Dorsal cochlear nucleus
IPL	-	Inter Peak Latency
HC	-	Head Circumference
PTA	-	Pure Tone Audiometer
NIHL	-	Noise Induced Hearing Loss
AC & BC	-	Air Conduction & Bone Conduction
kHz	-	Kilo Hertz
dBA	-	Decibel, SPL measured with “A” weighting network
SPL	-	Sound Pressure Level
SEL	-	Sound Exposure Level
Leq	-	Equivalent continuous noise level
Max P	-	Maximum Peak

ABSTRACT

Background and objectives

Noise pollution in mega cities is considered to be one of the most important and pressing problems. A major contribution to the noise is vehicular noise. Traffic policemen bear the brunt of prolonged and high intensity exposure to this environmental pollutant. Both the intensity of noise and the length of exposure determine its ability to damage hearing and continued exposure to greater than 85dB of noise may cause gradual but permanent damage to hearing.

Brainstem auditory evoked potentials (BAEP) have been used as a diagnostic technique in audiology in investigating hearing loss in addition to audiometer. Brainstem auditory evoked response potentials are the potentials recorded from ear and vertex in response to brief auditory stimulation to assess conduction through auditory pathways up to midbrain. Studies carried out for evaluating the effects of noise on human BAEP are minimal. With this background, the present study of effect of noise on BAEP in traffic policemen vis- a vis of the levels and duration of exposure to vehicle noise is carried out and compared it with age matched control population.

Materials & Methods

Thirty traffic policemen manning traffic and 30 aged matched controls who were involved in administrative work were selected considering inclusion & exclusion criteria. These two groups were asked to fill questionnaire to assess the auditory effects of noise, then they were subjected to pure tone audiometric assessment and BAEP recordings. The resulting data was statistically analysed.

Results:

Noise levels recorded were significantly high in study group (traffic policemen) than controls. Three of traffic policemen felt that their hearing ability was below average by self assessed questionnaire. There was increase in hearing thresholds at frequencies of 4kHz (AC and BC), 6kHz (AC) and 8kHz (AC) in traffic policemen compared to that in controls. Wave latencies were significantly prolonged in traffic policemen than controls. Within the study group, the wave latencies were prolonged in traffic policemen with hearing loss than traffic policemen without hearing loss. There was a positive correlation between exposure index (noise level in dBA x duration of exposure to noise in years). Wave latencies (wave I, II and III) were significantly prolonged in traffic policemen without NIHL as compared to controls.

Conclusion

There is increase in hearing thresholds and wave latencies of BAEP in traffic policemen who were exposed to continuous and loud noise. With this background, some preventive modalities for hearing conservation in the form of safety equipment (ear plugs, ear muffs etc), periodic checkups (audiometer, BAEP etc) and duty scheduling for exposure limitation can be suggested and awareness should be created among traffic policemen about the harmful effects of noise on hearing by implementing education and training programmes.

Key words : Noise levels, traffic policemen, pure tone audiometer, brainstem auditory evoked potential.

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF DIAGRAMS

INTRODUCTION

Noise is defined as sound wave that is aperiodic with irregular vibrations with no definite pitch that affects the physiological / psychological well being of individuals.

Noise pollution in mega cities is considered to be one of the most important and pressing problems. Increasing urbanization has led to mounting volumes of noise. Noise has been a bane and seems to have altered the ecological balance. A major contribution to the noise is vehicular noise. Both the intensity of noise and the length of exposure determine its ability to damage hearing. Louder the noise one is exposed to, over a continued period, the faster it leads to hearing loss. The traffic policemen engaged in controlling traffic noise, particularly at heavy traffic junctions, belong to the high risk group to be affected by health hazards of noise and air pollution. Most of the traffic policemen use a mask to prevent the ill effects of air pollution. However, a majority of them remain unaware about the health effects of noise on their hearing ability as this is an insidious process and takes long time to become overt.[1,2,3,4] Noise louder than 80 dB(A) is considered to be potentially hazardous and continued exposure to >85 dB(A) of noise may cause gradual but permanent damage to hearing. In India, occupational permissible exposure limit for 8 hour time weighted average is 90 dBA. Health effects of noise include both the auditory as well as non-auditory effects. Many studies have been carried out to study these effects in different categories of population exposed to high intensity and frequencies of sound in their workplaces. There are only minimal studies carried out regarding the estimation of noise levels and auditory effects of noise generated by automobiles among traffic

policemen particularly in India. This may be one of the reasons for not providing hearing protection devices to this group of work force.[5, 6, 7, 8]

An evoked potential is an electrical manifestation of the brain's reception of and response to an external stimulus. Evoked potentials have been studied in patients with neurological diseases since the early 1950s but it was only in the early 1970s that evoked potentials began to have definite clinical utility. Brainstem auditory evoked potentials, visual evoked potentials and short-latency somatosensory evoked potentials are reliable diagnostic tests that yield reproducible results in routine clinical practice. They provide an objective measure of function in their related sensory systems and tracts; they have been studied in large groups of normal subjects and in patients with a wide variety of neurologic diseases.[9]

Brainstem Auditory evoked potentials (BAEP) are the potentials recorded from ear and vertex in response to brief auditory stimulation to assess conduction through auditory pathways up to the midbrain. BAEP comprises of five or more waves within ten milliseconds of the stimulus and three interpeak latencies. Each individual wave and interpeak latencies providing information about an area of auditory pathway starting with cochlear nerve to the level of inferior colliculi. These were first described by Jewett and Williston in 1971.[9, 10]

Recently the brain stem auditory potentials have been widely studied in audiology, neurology, neonatology and anaesthesiology and have been used as a diagnostic technique in audiology in investigating hearing loss in addition to audiometer. Among all objective methods of hearing evaluation, brainstem auditory evoked potential is considered the most used precocious potential on clinical practice.

Recent technological advances have led to more widespread use of BAEP for audiological differential diagnosis and for estimation of hearing sensitivity in those persons who are difficult to test behaviorally.

Few studies are carried out for evaluating the effect of noise on auditory function of traffic personnel unlike organized industrial sector where periodic checks by audiometer are carried out as per legal requirements and also studies carried out for evaluating the effects of noise on human BAEP are minimal. Therefore, we have proposed a study of effect of exposure to noise on BAEP in traffic policemen vis- a vis of the levels and duration of exposure to vehicle noise and compare it with age matched control population.

AIMS AND OBJECTIVES OF THE STUDY

1. To assess noise induced hearing loss (NIHL) in traffic policemen by self assessed questionnaire and pure tone audiometer.

2. To record BAEP in traffic policemen and compare it with age matched controls.

3. To correlate exposure index (duration of exposure to noise X sound pressure level) with BAEP changes in traffic policemen.

4. To compare BAEP in study group with & without hearing loss with that of age matched controls.

REVIEW OF LITERATURE

A. Historical Review

Noise had a glorious birth. While there were rumblings before 1905, it was Einstein's explanation of Brownian motion that started the field. His motivation was not the mere explanation of the erratic movement of pollen, but much bigger: that noise could establish the existence of atoms. Immediately after Einstein there was an incredible flurry of ideas of the most profound kind that continues to this day. But noise, considered by many as unwanted sound.

Noise induced hearing loss has been recognized since early 19th century as a result of chronic exposure in certain occupations (eg., blacksmiths). By the late 19th century occupational hearing loss was recognized in a broader group of workers (boiler workers and rail road workers, industrial workers). Knowledge of the effects of impulse noise on hearing increased markedly during and after World War II. The United States military established noise exposure regulations in 1956, but civilian occupation standards were not promulgated until 1969 and not adopted widely and enforced until establishment of the Occupational Safety and Health Administration in 1970. The Occupational Safety and Health Administration originally adopted a permissible exposure limit of 85dB(A) for an 8 hr, time weighted, average noise exposure, with an exchange rate designed to allow somewhat higher exposure levels(115 dB) for progressively shorter durations. The National Institute for Occupational Safety and Health estimates that approximately 10% of workers are exposed to unsafe noise levels on the job.[15] First case of noise induced hearing loss was regulated and compensated in India in the year 1996. Occupational

permissible exposure limit for 8 hour time weighted average is 90 dBA.[77] The development in the field of evoked potentials recording is closely linked to the discovery of electricity.

1752- Benjamin Franklin with his kite experiment charged his leyden jar by using kite during electrical storms and postulated the presence of two opposing forces of electricity that is positive and negative.

1791- Luigi Galvani discovered that the nerves were a good conductors of electricity.

1850- Helmholtz was able to measure conduction velocity of nerve in frog.

1861- The method of electro diagnosis based on faradic and galvanic current was introduced by Erb.

1875- The distinction of making the first observation of electrical activity of brain goes to Richard Canton, who reported that he had detected currents from electrodes placed on the skull of exposed brain in rabbits and monkeys. Volta made the first electric battery.

1890- Waller was the first to demonstrate electrocardiogram.

1903- Einthoven invented string galvanometer with which he first recorded electrical currents generated by human heart.

1929- Hans Berger recorded the first human electro-encephalogram from electrodes placed on the scalp.

1939- Davis was the first to record electrical potentials on the human skull in response to auditory stimuli. The potentials generated in the cortex with latencies that ranged from 50 to 500ms. Some years

later thanks to computers, faster and shorter amplitude responses were recorded, known as middle latency potentials, between 10 to 80ms.

1967 - Sohmer and Feinmesser were the first to record BAEP initially.

1971- Jewett and Williston were the first to describe BAEP waveforms.

B. Anatomy of 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[84]

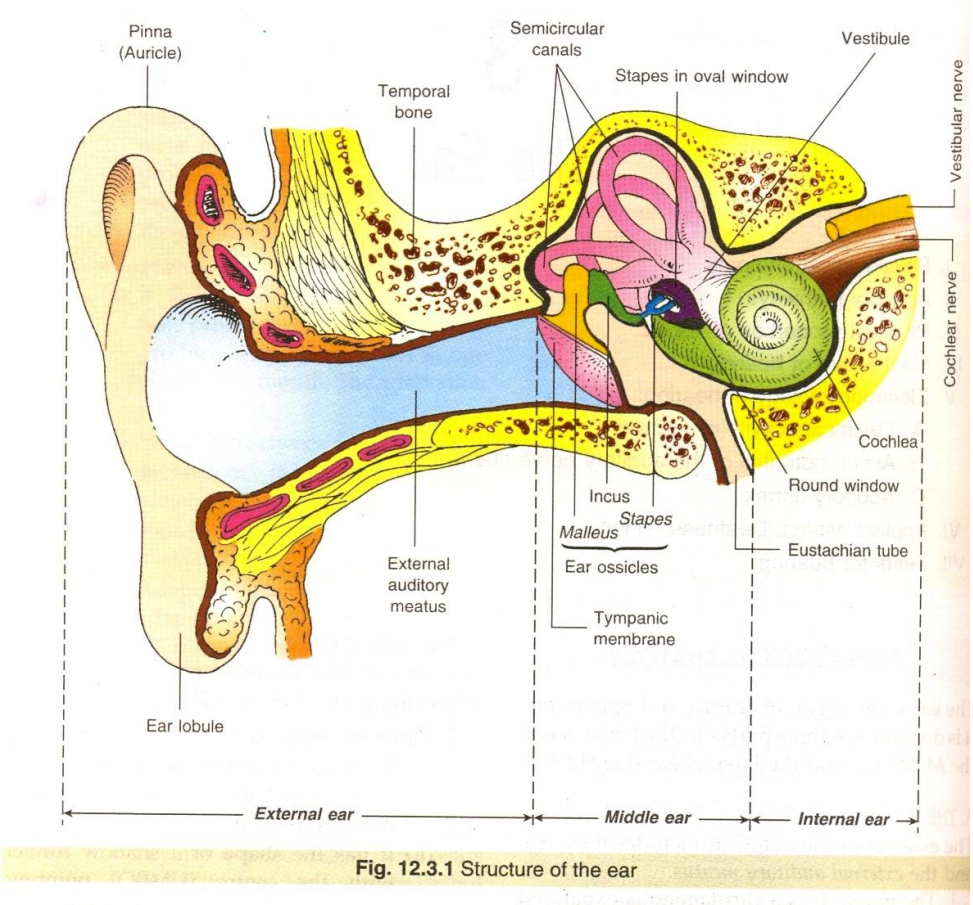


Fig. 12.3.1 Structure of the ear

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. [11]

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

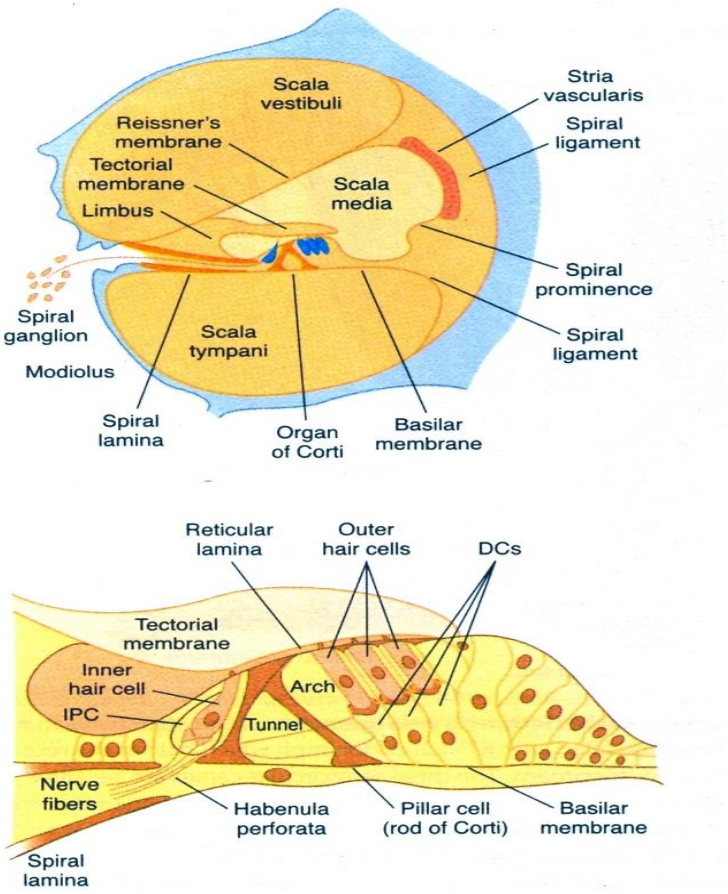


FIGURE 13–4 **Top: Cross-section of the cochlea, showing the organ of Corti and the three scalae of the cochlea. Bottom: Structure of the organ of Corti, as it appears in the basal turn of the cochlea.** DC, outer phalangeal cells (Deiters' cells) supporting outer hair cells; IPC, inner phalangeal cell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

B. Physiology of Hearing

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.[13]

2) Development of action potential in hair cells:

The stimulus to depolarize the sensory cell is the deflection of the stereocilia. The 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

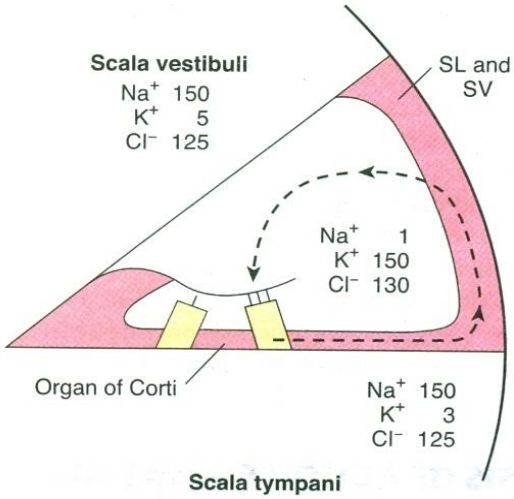


FIGURE 13–7 Ionic composition of perilymph in the scala vestibuli, endolymph in the scala media, and perilymph in the scala tympani. SL, spiral ligament. SV, stria vascularis. The dashed arrow indicates the path by which K^+ recycles from the hair cells to the supporting cells to the spiral ligament 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.[14]

Diagram 4 - Movement of Basilar Membrane Causing Stereocilia to Bend[84]

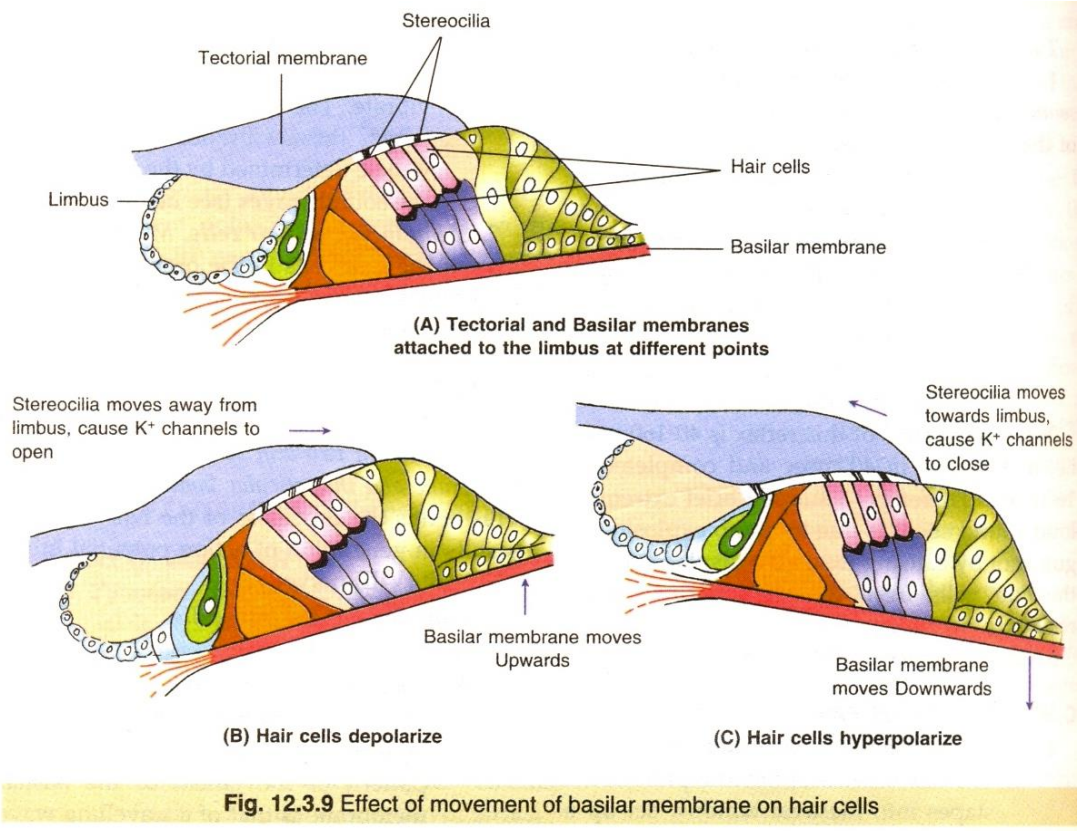


Fig. 12.3.9 Effect of movement of basilar membrane on hair cells

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 concered with melody, pitch and sound intensity.

Diagram 5 - Auditory Pathway[84]

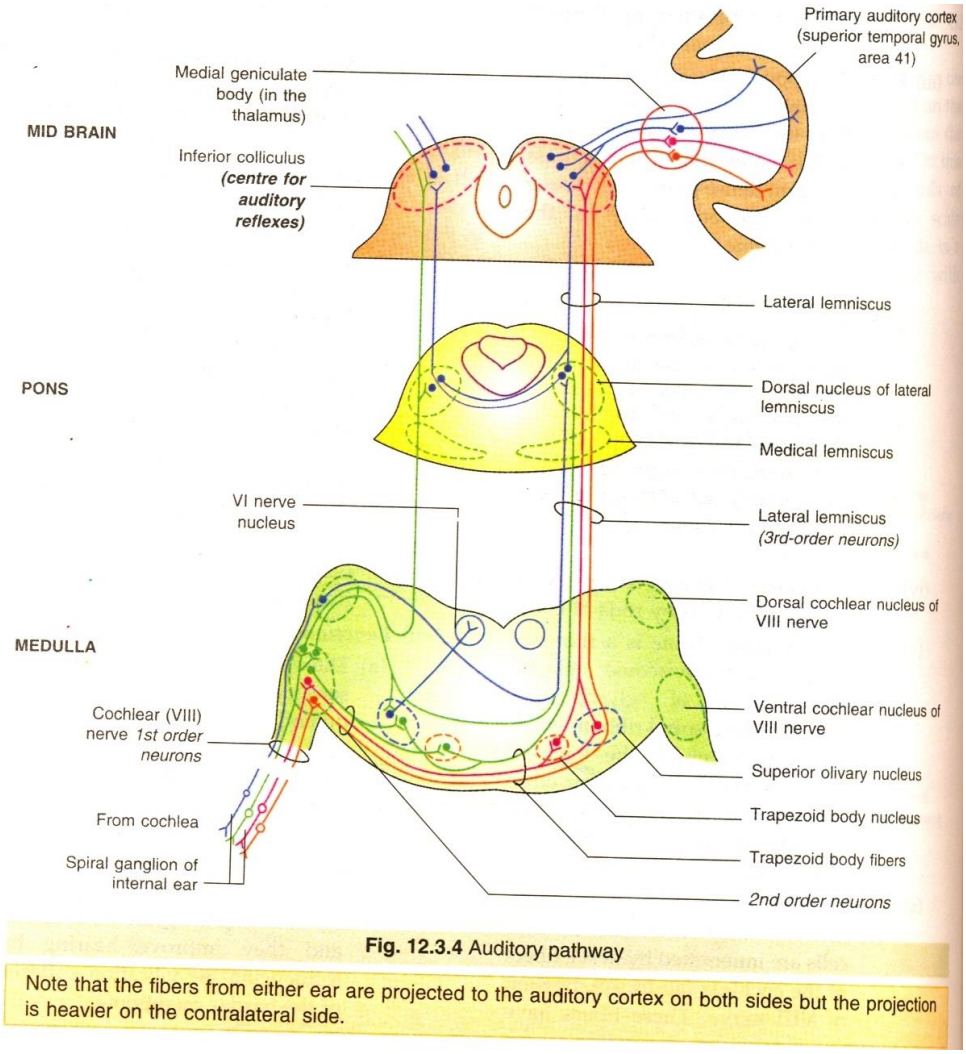


Fig. 12.3.4 Auditory pathway

Note that the fibers from either ear are projected to the auditory cortex on both sides but the projection is heavier on the contralateral side.

Sound localization

Determination of the direction from which a sound emanates in the horizontal plane depaends 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 haring mechanism which provides a warning system and survival advantage. It is for this work that Von Bekesy received a Nobel Prize in 1961.

C. Audiology And Acoustics

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, (iquid, 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).

Noise levels are often expressed as dB(A) which refers to sound pressure level with "A" network where the low and extremely high frequencies are given much less weightage compared to those in middle range which are more important and are responsible for NIHL.

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. [13]

D. 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).[13]

E. Noise Induced Hearing Loss (NIHL):

Occupational noise exposure is the most important preventable cause of hearing loss. NIHL is generally attributable to unprotected exposures above 95dB (A). It often becomes clinically apparent in middle age when age related threshold shifts are added to prior noise induced shifts. There is usually a history of recreational or occupational noise exposure, usually without hearing protection, occurring over many years. With continued noise exposure, hearing loss is progressive.

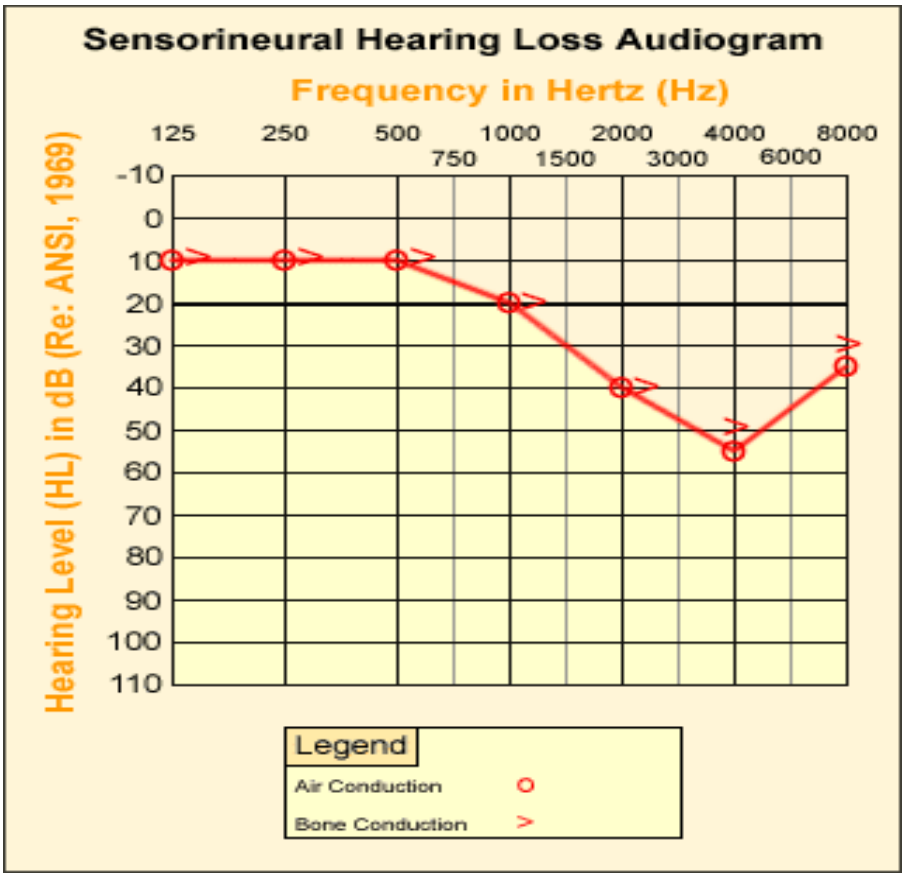
Patients with NIHL typically present with gradual, bilateral, high frequency, sensorineural hearing loss. NIHL adversely affects quality of life.

Noise can be thought of, as any sound which is unpleasant or unwanted or alternatively as a signal that delivers no information, and whose intensity varies randomly with time. For the purpose of the present study, noise is sound of sufficient intensity to damage hearing. Damage to cochlear hair cells from noise depends on the frequency, intensity and duration of exposure to noise as well as individual susceptibility. Noise may be intermittent or continuous. The interval between exposures is important, as recovery may be possible from intermittent noise. Continuous noise exposure is more damaging than interrupted exposure. The classic audiometric evidence of early noise damage is the appearance of a notch on the audiogram between 3 and 6 KHz. The notch usually occurs at 4 KHz and then progresses to include the adjacent frequencies. It is usual for the audiogram to show recovery in both ears for frequencies above 6 kHz. If this recovery is not present, the diagnosis of NIHL should be reconsidered. It is accepted that many severe cases of NIHL do not show such a recovery (sometimes this recovery is absent because of tinnitus. At first this notch is temporary (temporary threshold shift), and after rest away from noise, hearing will usually return to normal. With continuing exposure this loss becomes permanent, i.e. permanent threshold shift. Noise damage is usually similar in both ears unless one ear is more exposed to noise than the other. Where stable noise exposure continues the permanent loss at 4 KHz will reach a maximum level after about 15years. Initial hearing loss is asymptomatic, but problems, particularly in discriminating speech in background noise will begin when loss spreads to the lower frequencies, 2 or 3 KHz. At the same time as being noise

exposed, the person is ageing, and age related loss will be superimposed on the noise damage. This occurs gradually over a period of years. Highest frequencies are affected first, causing the characteristic noise induced notch to disappear from the audiogram.

Tinnitus is a subjective sensation of noise in the ears or head. There are various descriptions of tinnitus, either as a high-pitched ringing, hissing or whistling, or a low-pitched rushing or buzzing. Short periods of high-pitched whistling can be experienced before TTS or PTS is established and can be taken as a warning sign of impending hearing damage. NIHL can occur without the person ever having noticed tinnitus.[15]

Diagram 6 : Audiogram showing dip at 4kHz and recovery at 6kHz (NIHL).



Pathogenesis of NIHL

Sound damages the ear first at a frequency of about 4 kHz and one of the reasons for this is the acoustic resonance characteristics of the external ear. This hard-walled tube, closed at one end, amplifies acoustic energy in the upper frequencies by about 10 decibels. In addition, individual variation in the acoustic transfer characteristics of the tube is a factor in the large variability in people's susceptibility to noise.

Transduction of sound vibration to nerve impulses occurs in the cochlea. The hair cells in the organ of Corti may be damaged directly by noise, or indirectly by very high levels of continuous sound which causes vasoconstriction of the vessels of the stria vascularis in the cochlea blood supply. This renders the hair cells relatively anoxic and thus secondarily damaged. The amount and type of direct hair cell damage depends on the intensity of the sound. Above a certain minimum of frequency and intensity, the outer hair cells show signs of metabolic exhaustion with drooping of the stereocilia. This correlates with the common phenomenon of temporary threshold shift (TTS), which recovers within a few hours. Higher sound levels damage the outer hair cell stereocilia further, including destruction of the inter-cilial bridges, and recovery takes longer. Even higher levels of sound lead to collapse of the stereo cilia, and the hair cell are eventually phagocytosed.

Outer hair cells amplify the movement of the basilar membrane of the cochlea by contracting when stimulated by sound. This increases the stimulus delivered to the inner hair cells which transduce the mechanical movement to trigger a nervous impulse in the afferent nerve endings of the 8th nerve. If the outer hair cells are not

functioning, greater stimulation is required to initiate a nervous impulse; thus the threshold sensitivity of the inner hair cells is raised which is perceived as a hearing loss. Hair cells in the basal coil of the cochlea are the most sensitive to noise damage; they are responsible for transducing higher frequencies and this accounts for the high frequency hearing loss found in noise-damaged ears.[16]

NIHL is preventable. Persons who have to work at places where noise is above 85dB (A) should have pre-employment and then annual audiograms for early detection. Ear protectors (ear plugs or ear muffs) should be used where noise levels exceed 85 dB (A). They provide protection up to 35 dB.[13]

Permissible exposure in cases of continuous noise or a number of short term exposures [Government of India, Ministry of Labour, Model Rules under Factories Act 1948] :

Noise level *	**Permitted daily exposure**
dB(A)	**(hours)**
90	8.0
92	6.0
95	4.0
97	3.0
100	2.0
102	1 ½
105	1.0
110	½
115	¼

*5 dB rule of time-intensity states that "any rise of 5 dB noise level will reduce the permitted noise exposure time to half".[13]

The Workplace Exposure Standard is:[15]

- Leq (8hrs) 85 dB(A)
- Maximum Level 115 dB(A)
- Peak Level 140 dB

F. Evoked Potentials

These are electrical manifestation of the brains reception of and response to an external stimulus. Pattern shift visual, brainstem auditory and short-latency somatosensory evoked potentials are reliable diagnostic tests that yield reproducible results in routine clinical practice. The clinical utility of evoked potentials is based on their ability (1) to demonstrate abnormal sensory system function when the history and/ or neurologic examination are equivocal; (2) to reveal the presence of clinically unsuspected malfunction in a sensory system when demyelinating disease is suspected because of symptoms and signs in another area of the central nervous system; (3) to help define the anatomic distribution of a disease process and (4) to monitor changes objectively over time in a patients status. These tests provide sensitive, quantitative extensions of the clinical neurologic examination. They primarily afford numerical data; sometimes the absence of a wave or an abnormal configuration of its potential field also provides useful information.

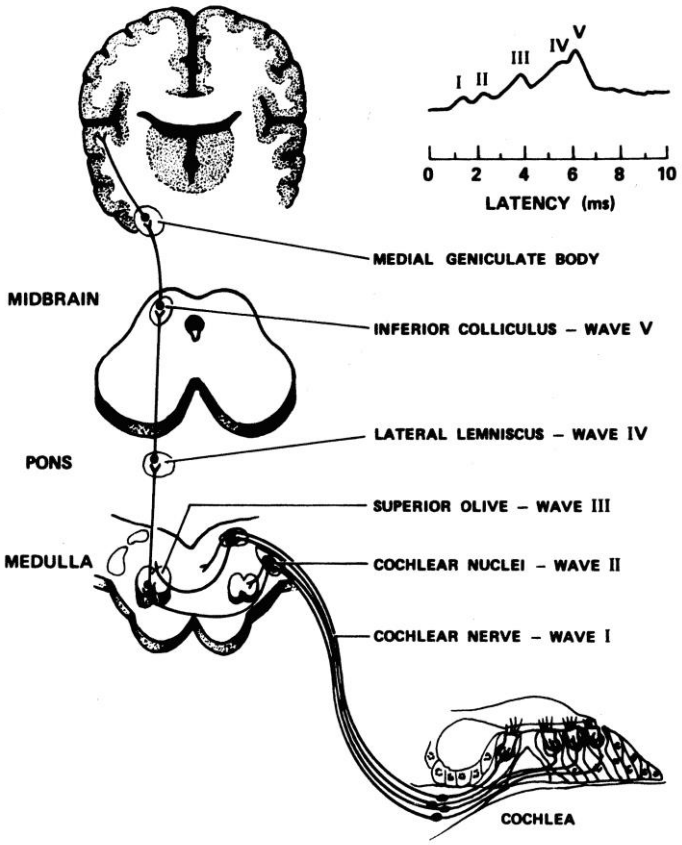
G. Anatomical And Physiological Basis of BAEP:

The external and middle ear transmit the sound waves to the fluid compartment of the inner ear containing cochlea. The cochlea is a coiled

structure having basilar membrane upon which organ of corti rests with its receptor elements and hair cells. Each hair cell contains about sixty very fine hairs or steriocilia, which are embedded in a gelatinous tectorial membrane overlying the organ of corti. The high frequency sounds affect the basal end of cochlea and the low frequencies affect the apical end of cochlea. The amplitude of movement is directly related to the intensity of the acoustic signals. The stimulation of cochlea results in eighth nerve activity of those fibres innervating the portion of the basilar membrane set into vibration. The latency of eighth nerve discharges following an acoustic signal will be shorter from the basal compared to the apical end of cochlea. The eighth nerve neurons are bipolar situated in spiral ganglia, their dendrites go to hair cells and axons to the cochlear nucleus. The cochlear nucleus has three subnuclei: anterior ventral cochlear nucleus [AVCN],posterior ventral cochlear nucleus[PVCN],and dorsal cochlear nucleus[DCN].The output of AVCN is through ventral acoustic striae forming the bulk of trapezoid body to terminate in the superior olivary nuclei and inferior colliculus. The neuons in the AVCN discharge at short latency to acoustic stimuli with a pattern like that of eighth nerve. The output of PVCN mostly goes through ventral and middle acoustic striae to terminate in the superior olivary nuclei and inferior colliculus. DCN terminates in the superior olivary nucleus and contralateral inferior nucleus through dorsal striae. The discharges from these neurons are different from AVCN by having a longer latency. The cochlear nucleus terminates in the superior olivary nuclear complex, which as medial and lateral components at the base of pons.The medial superior olivary nucleus receives the input from both ipsilateral and contralateral AVCN which are excitatory. The Lateral superior olivary nucleus also receives ipsilateral excitatory inputs from AVCN and PVCN and inhibitory inputs from contralateral AVCN and PVCN via trapezoid body. From the olivary nucleus, the impulses travel to ipsilateral and

contralateral lateral lemnisci and to inferior colliculi. The olivary nuclei are the first sight in the auditory pathways where the neurons are affected in a non linear manner and binaural stimulation. Inferior colliculi and lateral lemniscal nuclei converge the input from contralateral cochlear nucleus and superior olivary nucleus. The impulse from inferior colliculi travel to medial geniculate body and then to auditory cortex. The orderly orientation of the neurons in dorsal cochlear,medial superior olivary, and lateral superior olivary nuclei results in summation of synaptic potentials to result in high amplitude electrical fields. The nuclei are connected by large myelinated fiber tracts and their synchronous discharge also generates cohesive voltage fields. There are five or more distinct wave forms recorded within ten milliseconds of the auditory stimulus. [9, 10]

Diagram 7- Central Auditory Pathway With Genesis of Waves



(Available from: http://www.drdavindson.ucsd.edu-fig 01.09.jpg)

H. BAEP – Normal Waveforms and Their Genesis

Wave I: The ABR wave I response is the far-field representation of the compound auditory nerve action potential in the distal portion of cranial nerve (CN) VIII. The response is believed to originate from afferent activity of the CN VIII fibers (first-order neurons) as they leave the cochlea and enter the internal auditory canal.

Wave II: The ABR wave II is generated by the proximal VIII nerve as it enters the brain stem.

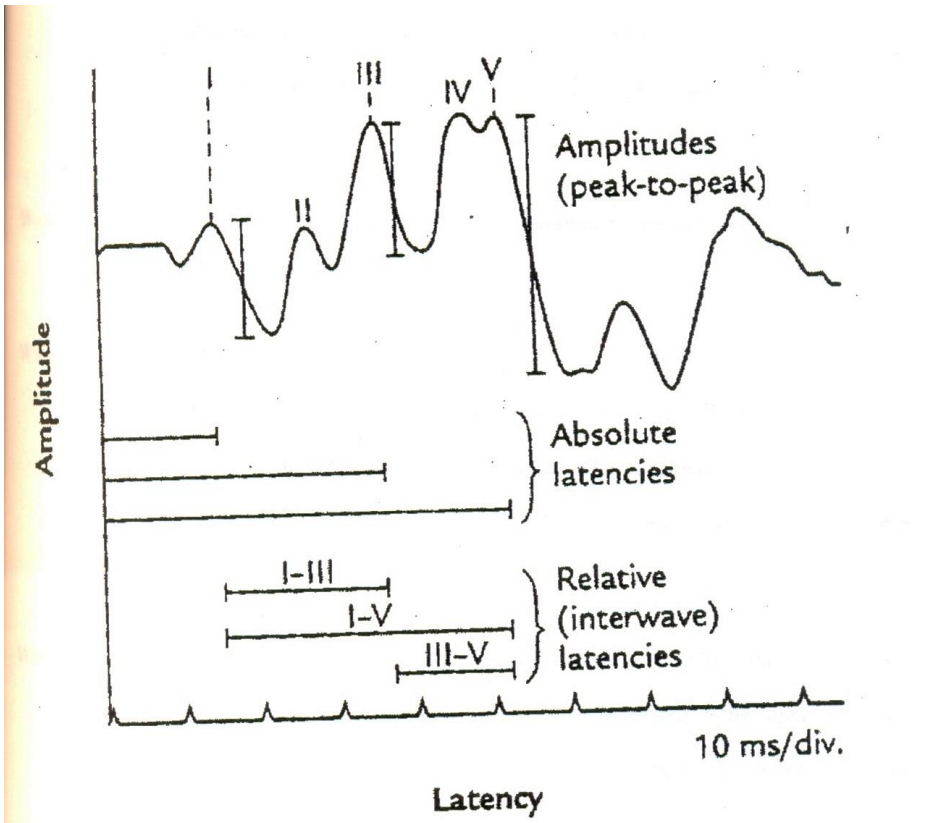
Wave III: The ABR wave III arises from second-order neuron activity (beyond CN VIII) in or near the cochlear nucleus. Literature suggests wave III is generated in the caudal portion of the auditory pons. The cochlear nucleus contains approximately 100,000 neurons, most of which are innervated by eighth nerve fibers.

Wave IV: The ABR wave IV, which often shares the same peak with wave V, is thought to arise from pontine third-order neurons mostly located in the superior olivary complex, but additional contributions may come from the cochlear nucleus and nucleus of lateral lemniscus.

Wave V: Generation of wave V likely reflects activity of multiple anatomic auditory structures. The ABR wave V is the component analyzed most often in clinical applications of the ABR. Although some debate exists regarding the precise generation of wave V, it is believed to originate from the vicinity of the inferior colliculus. The second-order neuron activity may additionally contribute in some way to wave V. The inferior colliculus is a complex structure, with more than 99% of the axons from lower auditory brainstem regions going through the lateral lemniscus to the inferior colliculus.

Wave VI and VII: Thalamic (medial geniculate body) origin is suggested for generation of waves VI and VII, but the actual site of generation is uncertain. I-III interpeak latency is the difference between wave III and I is a measure of conduction from eight nerve across subarachnoid space into core of lower pons. III-V interpeak latency is a measure of conduction from lower pons to midbrain. I-V interpeak latency is a measure of conduction from proximal eighth nerve through pons to midbrain.[9]

Diagram 8 – BAEP : Normal Waveforms and Their Genesis



Typical Auditory brain stem response wave form morphology with display of latency and amplitude measurements (Adapted from Reference 1)

I. Variables Affecting BAEP

i) **Age** - Age has a distinct effect on BAEP wave forms with increasing age after childhood, there are increases in absolute and inter peak latencies of BAEP waveforms. These differences were experimentally noted in 1980 by Jerger and Hall when they examined these latencies in 70 normal subjects.[17] Allison et al. in 1984 constructed a histogram of age changes over the 18-95 years range. Using a simple developmental model Eggermont in 1988 has defined three exponential functions with time constants of 4 weeks , 40 weeks and 4 years which describe maturational latency changes.[18] The waveforms in infants are often are often higher in amplitude than in adults presumably on the basis of smaller head size and greater proximity of the recording electrodes to BAEP generators according to Stockard et al. [19] Till the age of 18 months, BAEP values are age dependent and the effect of age is more pronounced in premature infants. Older adults have a slightly longer I-V interpeak latency compared to younger individuals. With increasing age there is a tendency for all the BAEP waveforms to be delayed significantly. This is most probably the result of the presbycusis seen in most adults. In addition there will be selective prolongation of I-III inter peak latency. Stephen Harkins found that the peak latency of BAEP increased in elderly (mean age 71.2yrs) and was shown to be due to peripheral processes (mild presbyacusis). Presbyacusis, the gradual hearing loss associated with aging, affects more than one third of those over 75 and is probably due to cumulative loss of hair cells and neurons.[14] Arora et al found that absolute latency of wave 1 was significantly delayed in twenty elderly patients with presbyacusis compared with twenty age and sex matched controls with I-III and I-V IPLs normal.[20] Ottaviani et al found that latency increase of BAEP observed in presbyacusis (60-

80)yrs is mainly correlated to the audiometric shape of the hearing loss.[21] It is well known that with increasing age there is a physiological alteration in the hearing acuity which is called presbyacusis. Jorgensen in 1961 studied the histological inner ear changes occurring with age. He noticed collapse of the organ of corti, narrowing of the stria vascularies with increased thickening of its wall, adherence of the Reissnner's membrane to the stria vascularis and loss of ganglion cells in the spiral canal.[82]

ii) **Sex -** The BAEP waveforms show significant differences between males and females. Absolute latency of wave V and I-V inter peak latency are consistently prolonged in males by 0.15 to 0.2 msec for 60 to 70 decibels stimulation intensities. The amplitude of the BAEP s is significantly higher in females. These gender differences are seen in children only after 8 years of age. I-V inter peak latencies decreases by almost 1 msec at the time of the menstrual period. These differences between males and females are most probably related to differences in head size. Allison et al. in 1983 suggested that brainstem auditory pathway length varies as the cube root of brain volume and correlating thus with age and sex related variations in brain weight, calculated that the male:female ratio should be 1.034+ 0.008, comparing this with their normal data they found that latency sex differences were within predicted limits.[22] Females have shorter latency which is attributed to higher core body temperature and shorter length of brain stem auditory pathway which was experimentally proved by Stockard et al. in 1979.[23] Most authors feel that there is no relationship between height and inter peak latencies within gender groups.

iii) **Temperature** - with decreasing central body temperature the latencies of BAEPs are increased. The temperature-latency relationship

is nonlinear. Latencies increase roughly 7% for each 1° centigrade. The amplitude of the BAEPs first increases as the temperature decreases down to 27 ° to 28 ° and then amplitudes decrease linearly with temperature drop and usually disappear at around 20 °. This exponential relationship between body temperature and BAEP latencies was experimentally demonstrated by Markand et al. in 1987. [24]

iv) **Hormonal effects** - Yadav A et al studied these BAEP latencies in 20 women with normal cycles in four different phases and showed that there is a trend of increase in peak latencies of wave III and V in estrogen-peak midcycle while decrease in latencies in progesterone-peak midluteal phase. These findings suggest that normal cyclical variations in the levels of estrogen and progesterone during menstrual cycle do affect the auditory pathways and effects are better seen on the central component.[25]

v) **Drugs** - BAER's are resistant to the effect of drugs but a slight prolongation of wave V latency with barbiturates or alcohol is attributed to lowering of body temperature. Begleiter et al. in 1981 studied 17 alcoholic subjects abstinent for at least 3 weeks and found marked prolongations in I-V IPL. They suggested demyelination or oedema as the mechanism.[26] Effects of aminoglycoside antibiotics on BAEP wave forms was studied with the idea of developing a means of following the ototoxicity of those drugs by Guerit et al in 1981 and they found that the minor latency changes noted disappeared with cessation of medication.[27] Amino glycoside antibiotics such as streptomycin and gentamicin obstruct the mechanosensitive channels in the stereocilia of hair cells and cause the cells to degenerate, producing nerve deafness and abnormal vestibular function. [14]

vi**) Anthropometric variables**- Head size is one of the important parameters affecting BAEP waveforms. There are three important head measures that is (AP) anterior to posterior which is a measurement taken from nasion to inion. Another measurement that is Right to Left taken from mastoid of one ear to the mastoid of the opposite ear. The third measurement that is (HC) head circumference taken across the head through occiput is also considered when taking measures of head size. Masaru Aoyagi and others determined that head size which reflects brain size is one of the important factors for the basis of gender differences.[28] Another study done by James J. Dempsey in 1986 has demonstrated head size as an important source of inter subject variability which should be considered in order to increase the clinical usefulness of BAEP.[29] Study done by Dennis R. Trune showed that head diameter, correlated more highly with BAEP waves than did gender.[30] Wave latencies and interpeak latencies can been normalized using the formula=latency/HC*mean HC to avoid intersubject variability.

J. Interpretation of Abnormal BAEP Waveforms:

BAEP abnormalities may include one or more of the following

1. Absence of waveforms- BAEP waveforms are most of the times absent in brain dead patients. Goldie et al. in 1981 found that 27 out of 35 brain-dead patients had no identifiable BAEP waveforms, including wave I. [31]

2. Abnormal absolute or interpeak latencies - In 80% of the patients with cerebellopontine angle tumors there was prolongation of all BAEP waveforms. This was experimentally demonstrated by study done by Parker et al in 1980 and various other studies suggest the same.[32]

3. Amplitude ratio abnormality- As wave I is generated outside central nervous system and wave V inside and the normal V/ I ratio ranges between 50% to 300%. In assessing hearing impairment if the ratio is lower than 50% it indicates central impairment and if it is more than 300% it may be due to peripheral hearing impairment.[9]

4. Right to left asymmetry-Observing BAEP waveforms and comparing waves in both ears might help in localization of the lesion.[9]

K. Clinical Appilications of BAEP Recording:

Studies done by Avasthi and Subhendu have shown an increase in absolute latencies of all the waves of BAEP in patients with advanced hypertension and following treatment significant decreases were observed in wave latencies demonstrating their role in prognostic follow up of these patients.[33]

Kumar and Tandon have demonstrated clinical utility of BAEP in patients with chronic pain following acupuncture which was indicated by changes in their latencies.[34]

Neil Bhattacharyya demonstrated that these potentials can be used as an effective screening tool in the evaluation of suspected retrocochlear pathology such as an acoustic neuroma or vestibular schwannoma.[35]

In a study done by Pramod Sharma and others they concluded that BAEP recording as a reliable and effective technique for determining auditory functions in the neonates especially changes of early bilirubin toxicity.[36]

J.K. Nousak showed that these BAEP latencies are accurate in estimating hearing threshold.[37]

Young,G Bryan showed that persistent abnormalities of BAEP reliably indicate the likelihood permanent vegetative state or death.[39]

Eswaran, Hari recorded these potentials on fetus during labour and concluded that it can be a important screening tool.[40]

Nora, and Daniel B. showed that BAEP peak latencies correlated positively with electroencephalographic abnormalities in children with liver disease.[41]

Lew, and Henry L. showed BAEP recording can be a objective tool for evaluating hearing dysfunction in traumatic brain injury patients.[42]

Di Zafetriou proved that definite stastically significant association between abnormal BAEP recordings and full term delivery perinatal aetiology of cerebral palsy, spastic tetraplegia, speech, visual and myoskeletal impairments, epilepsy,mental retardation inability to walk independently and cortical atrophy on neuroimaging. They concluded that abnormal recordings in children with spastic CP are indicative of poor prognosis and associated with a multihandicap state. BAEP testing should be incorporated in to the diagnostic plan of all children with spastic CP newly referred to neurodevelopmental centres.[43]

Leocani in a study showed that patients with multiple sclerosis can have an abnormal ABRs. These may be used to detect auditory neuropathy or neural conduction disorders in newborns. Because ABRs are reflective of auditory nerve and brainstem function, these infants can have an abnormal ABR screening result even when peripheral hearing is normal.[44]

Flint Boettcher A showed that latencies were prolonged in people with presbyacusis and can be used as a tool for early detection.[45]

Kurita. A et al compared 20 controls with diabetics and showed that diabetics had significantly longer wave latencies.[46]

A study done by Reyes Contreras et al observed significant differences in 1-5 interpeak latencies in HIV infected groups when compared with controls. They concluded that HIV infections may produce subclinical pathologic changes in the cochlear nerve and brainstem, which can be detected by BAEP recordings.[47]

Kalita J and Mishra UK monitored BAEP in patients with japanese encephalitis and demonstrated significant reduced V/I amplitude ratio which may be due to raised intra cranial tension or brainstem involvement in japanese encephalitis.[48]

Schwarz G et al studied BAEP in subjects with respiratory insufficiency following encephalitis and observed prolongation of all wave and interpeak latencies due to proximity of respiratory control centre in the brainstem.[49]

Romero et al demonstrated that children with perinatal encephalopathy showed larger wave latencies than control groups and these indicated neurological function.[50]

Pelecki et al studied BAEP in patients with acromegaly and observed prolongation of wave latencies and delay in brainstem transmission which shows involvement of central nervous system in acromegaly.[51]

Ferri et al showed that Down syndrome patients with severe retardation showed significantly longer I-V interpeak interval than those with moderate retardation this could be due to the presence of additional central nervous system abnormalities.[52]

Atis et al recorded BAEP in patients with chronic obstructive pulmonary disease and found that there was prolonged latencies due to altered functions of cochlear nerve and brainstem which was attributed to chronic hypoxic-hypercapnic status occurring in brainstem.[53]

Vandana, and Tandon OP, recorded BAEP in children with chronic malnutrition and showed the prolongation of wave latencies I to IV which was due to poor development of peripheral auditory pathways in those children.[54]

Kalita J and Mishra UK studied BAEP in patients with tuberculous meningitis and varying degrees of abnormalities in wave forms which they correlated with radiological findings to improve the diagnostic significance.[55]

Church et al studied BAEP wave forms in children with craniosynostosis which is a devastating disorder characterized by premature closure of the cranial plates before or shortly after birth and found out that there was prolongation of I-III interpeak latency which was due to compression of auditory nerve as it emerged out of internal auditory meatus and posterior fossa. They recommended the recording of BAEP as a standard clinical care for this patient group as the best way to detect auditory nerve compression.[56]

Gamez J, and Minoves T studied BAEP in patients with neurogastrointestinal encephalomyopathy which showed prolongation I-III and I-V interpeak latencies which suggested delay in central conduction time along brainstem. BAEP may be usefull in the neurophysiological evaluation of central white matter lesions in neurogastrointestinal encephalomyopathy.[57]

Kochar et al observed BAEP and Somatosensory evoked potential waveforms in patients with cerebral malaria and concluded that both were abnormal in these patients and were predictive of worst prognosis.[58]

Ramachandran R and Mackenzie I showed BAEP has a role in intraoperative neurophysiological monitoring during surgeries like microvascular decompression for trigeminal neuralgia which aids in predicting post operative hearing loss.[59]

In another study done by Ikuta et al. have also suggested that differences in waveforms are seen in evoked potentials of schizophrenics, manic depressives and epileptics as compared to healthy adults.[60]

MATERIALS AND METHODS

Source of Data

Subjects: Study group consisted of 30 traffic policemen and 30 age matched controls.

Method of Collection of Data

Data was collected by self assessed questionnaire, by estimating auditory threshold by pure tone audiometer and by recording brainstem auditory evoked potentials from subjects after obtaining informed consent for the study. 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. Subjects of age group between 25 and 60years.
2. Subjects with active traffic management duties.

Control group included

1. Subjects of age group between 25 and 60years.
2. Subjects involved in administrative work.

Exclusion criteria

1. Subjects over 60 years of age.
2. Subjects with history of use of ototoxic drugs like streptomycin, cisplatin, neomycin, gentamycin and ear infections.

3. Subjects with chronic medical illness like diabetes, hypertension etc.

4. Subjects with history of head injury.

Methodology

Noise levels were measured at different traffic junctions in Kolar city and in and around Sri Devaraj Urs Medical College, Kolar using Sound level meter.

Sound Level Meter: is the instrument that displays the amplitude level of sound as its being recorded. In this study, sound level meter 2231 type with the Front Plate BZ 7110 and software Module "M-11" was used.

The amount of Peak, SPL (sound pressure level), SEL (sound exposure level), Leq (Equivalent Continuous Level) and Max Peak (Maximum peak) was measured.

All the measurements were done in the peak of the traffic. The traffic policemen deputed at those junctions were included in the study who were involved in manning the traffic >8hrs per day. The subjects in and around Sri Devaraj Urs Medical College involved in administrative work were taken as the control group. The noise levels were also measured here.

Self assessed questionnaire[61] was given to all the subjects to assess the knowledge, attitude and practices towards noise pollution. The questionnaire was filled by the subjects and it included the questions regarding the self assessment of their hearing ability, past and present exposure to loud sound, duration of exposure to noise and the use of personal protective devices such as earplugs and earmuffs.

Quality of hearing was below average in three of the subjects in study group (traffic policemen) by self assessed questionnaire.

All subjects in both groups were subjected to pure tone audiometry.

Pure Tone Audiometer

An audiometer (Elkon giga 3) 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.

The method is based on American Society for Speech and Hearing Association [ASHA] 1978 guidelines for manual pure tone audiometry (PTA). The procedure is as follows.

1. The subject is made to wear earphones during air conduction testing and a vibrator during bone conduction testing. He is instructed to respond whenever and as soon as the tone comes on, regardless of how faint the tone is and to stop responding as soon as the tone goes off. The subject is also told that one ear is tested first and then the next ear is tested. A pulse of tone is presented at

a set frequency and set dB hearing level using adjustment knobs on the audiometer.

2. Mode of response is by pressing button.

3. The response should not be considered as one, unless the latency of response is consistent and the subject responds appropriately to the termination as well as initiation of the tone.

4. Subjects should be reinstructed if false positive response (in the absence ofa tone) or false negative (in the presence of a tone) are obtained. False positive responses can be minimized by varying the interval between audible stimuli, employing pulsed or warbled tones or by asking the subject to report the number pulsed tones given at a particular level.

5. The subject is familiarized with the tone by one of the two methods

   a) The attenuation is set at low limits and the intensity is slowly and continuously increased until a response occurs.

   b) The tone is presented at 30 dB hearing level and at 50 dB hearing level if no response occurs at 30 dB. If there is no response even at 50dB, tone is increased in 10 dB steps until a response occurs.

6. The duration of tone is 1-2 seconds. The inter-stimulus interval is varied, but is never less than the duration of the stimulus.

7. After the first response, the tone is decreased by 10 dB whenever the subject responds and is increased by 5 dB if the person fails to respond.

   'Threshold' is defined as the lowest intensity at which the subject

responds at least half the time and at least three times on ascending runs.

8. The earphones should be so placed that the grid is directly over the entrance to the ear canal. Hair should be manipulated so that it is not trapped underneath the headphones and other obstacles such as earrings should be removed.

9. Diagnostic testing should be done at the following octave frequencies- 250, 500, 1000, 2000, 4000 and 8000 hertz, following the above steps each time.

The ambient noise at any octave frequency should be less than 25 dB.

The bone vibrator for bone conduction checking must be placed on the mastoid process, no closer than a thumb's width to prevent acoustic radiation and diagnostic testing at 250, 500, 1000, 2000 and 4000 hertz is done as above.

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.

Degree of Hearing Loss [WHO Classification 1980][13]

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

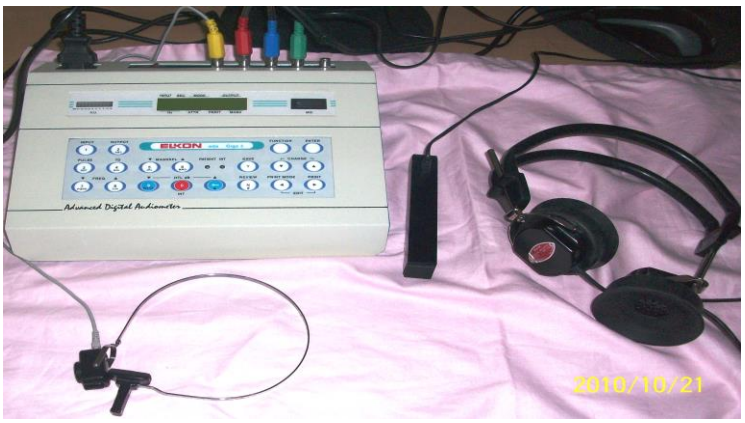
In the present study, eight of them in test group had noise induced hearing loss (sensorineural) with 5 with mild hearing loss (26-40 dB) and 3 with moderate hearing loss (41-55 dB).

Then BAEP was recorded in all the subjects included in the study in whom audiometry was done. BAEP was recorded in an electrically shielded room by using EMG RMS MARK II machine. Head circumference (HC) was measured. Surface electrodes were placed with two active electrodes placed over both the mastoid processes, with a reference electrode placed over vertex, and ground electrode over the forehead. One cycle of 4-kHz sinusoids at an intensity of 90 decibels was delivered through head-phones with alternating phase at interval stimulus of 75 ms. Signals were amplified and band-pass filtered from 3 to 100 khz. Signals were analysed with sampling intervals of 10 micro seconds and for 10.24 mili seconds after stimulus onset. After averaging 2000 sweeps the signals were digitally band-pass filtered. Peak latencies of waves were automatically detected with a time resolution of 0.01 mili seconds to minimize measuring errors. Wave latencies and interpeak latencies of BAEP obtained were normalized using the formula = $\frac{latency}{HC}$ x mean HC.

The wave latencies and interpeak latencies of BAEP in traffic policemen were compared with the age matched controls. Then BAEP in traffic policemen was correlated with the exposure index (duration of exposure to noise x noise levels). The data thus obtained from the study was statistically analysed.

9. Method of Recording BAEP

10. Recording noise level using sound level meter



11. Pure Tone Audiometer

RESULTS & ANALYSIS

In the present study, 30 traffic policemen (test group) manning traffic and 30 aged matched controls who were involved in administrative work were selected considering the inclusion and exclusion criteria and were subjected to Pure tone audiometer (PTA) and BAEP recordings. The data was analysed using appropriate

statistical methods and discussed hereinafter.

Presentation of Data

Master chart showing wave latencies and inter peak latencies with age and sex of the subjects and their duration exposure to noise.

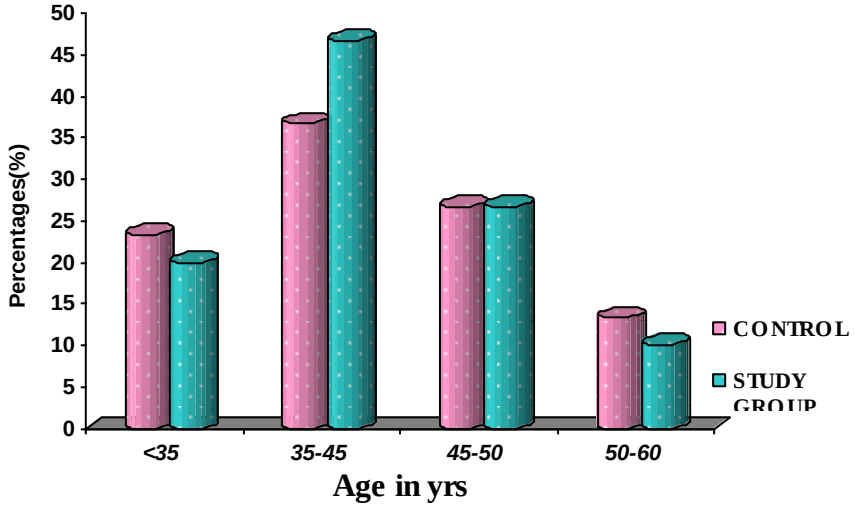
Statistical Treatment of the Data

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. BAEP recording was compared between traffic policemen and age matched controls. The Pearson correlation between noise level, duration of exposure to noise and wave latency and inter peak latencies was also done with significance test by student't' test and ANOVA test. Conclusions are drawn based on the outcome of this statistical treatment.[38, 76]

Table 1 : Comparison of Age (years) in control and study group

Variable	Control Group		Study Group		P Value
	NO.	%	NO.	%	
Age in years					
• <35	7	23.3	5	16.6	0.898
• 35-45	11	36.7	14	46.6	
• 45-50	8	26.7	8	26.7	

• 50-60	4	13.3	3	10.0	
Total	**30**	**100.0**	**30**	**100.0**	**-**



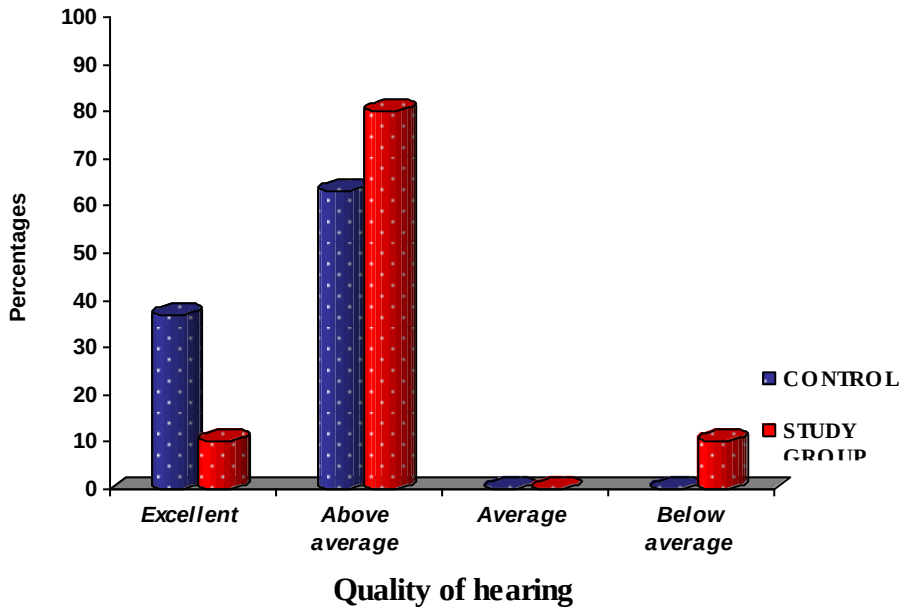
Graph – 1 : **Comparison of Age (years) in control and study group**

Age distribution: as shown in table 1, 60 subjects were selected. Study group comprised of 30 traffic policemen of which 6 were aged <35yrs constituting 20% as compared to 7(23.3%) in control group and the subjects aged between 35-45yrs were 14 and 11 in study and control group making up for 46.7 and 36.7% respectively. There were 8 (26.7%) each in study & control group aged between 45-50yrs and in the age group between 50-60yrs, there were 4 (13.3%)& 3 (10%) in study and control group respectively. The average age was 42.46±6.78yrs & 41.63±7.66yrs in study & control group and there was no statistical difference between the two groups with p value of 0.898.

Table 2 : Comparison of clinical assessment of hearing loss by self assessed questionnaire[61]:

Self Assessment of hearing loss	Control (n=30)		Study Group (n=30)		P Value
	Number of subjects	%	Number of subjects	%	
Quality of hearing					

• Excellent	11	36.7	3	10.0	0.011*
• Above average	19	63.3	24	80.0	
• Average	0	0.0	0	0	
• Below average	0	0.0	3	10.0	
Hearing over phone					
• Without difficulty	30	100.0	28	93.3	0.492
• Do miss Some conversation	0	0.0	2	6.7	
Hearing in crowd					
• Without difficulty	30	100.0	28	93.3	0.492
• Do miss Some conversation	0	0.0	2	6.7	
Sound of TV/ radio					
• Usually louder	0	0.0	1	3.3	1.000
• Usually same loudness	30	100.0	29	96.7	
Do people often indicate that you are talking too loudly?					
• Yes	0	0.0	1	3.3	1.000
• No	30	100.0	29	96.7	
Tinnitus					
• More than once a day / Work related	0	0.0	2	10.0	0.237
• No tinnitus	30	100.0	28	90.0	



Graph 2 : Comparison of clinical assessment of quality of hearing by self assessed questionnaire

Table 2: shows the clinical assessment of hearing by questionnaire method. In the present study, Graph 2 shows only three (10%) of the subjects in study group felt that their hearing ability was below average compared to none in control group (p Value 0.01). 6.7% of the subjects reported that they usually missed some conversation while hearing over phone in the study group and while talking to someone in crowd. 3.3% of the traffic policemen reported that while watching television they usually kept the sound louder to hear properly and mentioned that others often indicated to them that they were talking louder. 10% of the traffic policemen complained of tinnitus more than once a day and is work related.

Table 3 : Comparison of assessment of use of earplugs / earmuffs.

Use of earplugs/earmuffs	Control (n=30)		Study Group (n=30)		P Value
	Number of subjects	%	Number of subjects	%	
Ever used earplugs or earmuffs					
• **No**	30	100.0	30	0.0	1.000
• **Yes**	0	0.0	0	0.0	
Reason for non-usage					
• Uncomfortable	2	6.7	4	13.3	0.001**
• Not available	0	0.0	20	66.7	
• Dislike	28	93.3	6	20.0	
Other PPEs					
• Hands	0	0.0	2	6.7	0.024*
• Cotton	0	0.0	4	13.3	
• Don't use anything	30	100.0	24	80.0	
Do these PPEs effective if used?					
• **Average**	0	0.0	8	26.7	0.005**
• **Good**	30	100.0	22	73.3	

Table 3 : Depicts the distribution of subjects according to the usage of ear plugs / earmuffs which showed none of the subjects used them. The reason for their non-usage in study group was non availability in twenty (66.7%) traffic policemen. 20% of the subjects felt that they don't like to use earplugs and it was uncomfortable in 13.3% of the subjects and in the control group most of them reported that earplugs are of no use as they were not exposed to loud noise. 80% of them in test group did not use any method to reduce exposure to noise, while 6.7% of them used hands and 13.3% of the subjects used cotton to avoid noise exposure. Most of the traffic policemen (73.33%) said that these personal protective equipments will be effective if used in a situation where one is exposed to chronic and loud noise.

Table 4 : Comparison of duration of exposure to noise (years)

Duration of exposure to noise	Control (n=30)		Study Group (n=30)	
	Number of subjects	%	Number of subjects	%
1-5 years	10	33.3	7	23.3
5-10 years	8	26.7	10	33.3
11-15 years	11	36.7	9	30.0
>15 years	1	3.3	4	13.3
Mean ± SD	8.77±4.27		10.03±4.74	

Table 4 : shows the comparison of duration of exposure to noise (years). The mean (SD) was 8.77±4.25years in controls and 10.03±4.74years in study group which showed no statistical difference with p value of 0.281.

Table 5 : Comparison of Sound pressure Level (dB) (A)

SPL (dBA)	CONTROL (n=30)		STUDY GROUP (n=30)	
	Number of subjects	%	Number of subjects	%
64-70	30	100.0	0	0.0
71-80	0	0.0	9	30.0
>80	0	0.0	21	70.0
Mean ± SD	67.13±1.77		84.49±3.44	

Graph 3 : Comparison of Sound pressure Level (dB) (A)

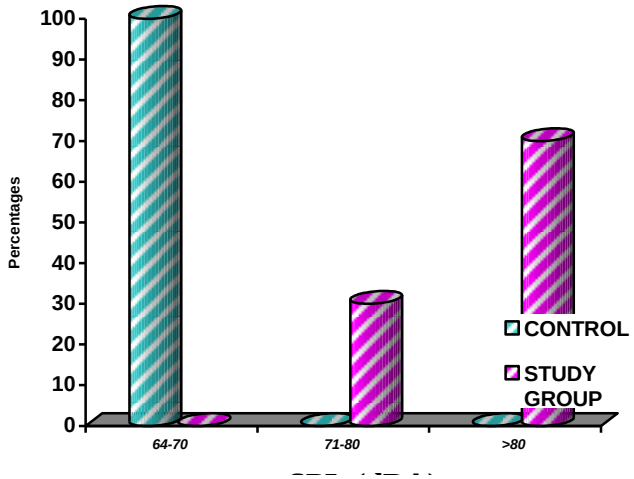


Table 5 : shows the comparision of sound pressure level. Sound pressure level (dBA) in study group was (mean±SD) 84.49±3.44(dBA) and 67.13±1.77(dBA) in controls. SPL was significantly high in study group who were continuously exposed to noise for >8hrs per day as compared to controls with p value <0.001**. (Graph 3)

Table 6 : Comparison of duration of exposure to noise (years) and sound pressure level (dBA) within the study group.

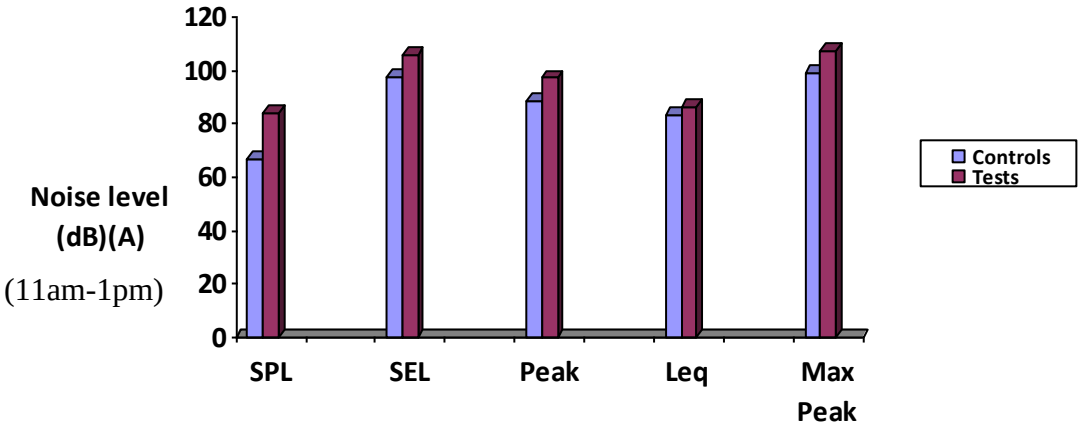
VARIABLES	**Study group with hearing loss (n=08)**	**Study group without hearing loss (n=22)**
Duration of exposure to noise (Years) (Mean±SD)	12.25±3.69	8.95±3.37
SPL (dBA)	85.80±3.16	81.85±3.37

Table 6 : Shows mean duration of exposure to noise is significantly high in traffic policemen with hearing loss (12.25±3.69 years) compared to traffic policemen without hearing loss (8.95±3.37 years). Sound pressure level (dBA) is also significantly increased in study group with hearing loss (85.80±3.16 dBA) than without hearing loss (82.85±3.37).

Table 7 : Comparison of noise levels in Controls and Study group.

NOISE LEVELS	Timings	SDUMC, Kolar (Controls) dB(A)	Traffic Junctions Kolar (Study group) dB(A)
SPL (dBA)	11am-1.30pm	66.99±2.08	83.83±3.84
	4.30-6pm	67.27±2.01	84.48±4.30
SEL (dBA)	11am-1.30pm	97.57±3.27	106.00±2.75
	4.30-6pm	99.08±2.74	105.75±2.31
PEAK (dBA)	11am-1.30pm	88.64±1.92	97.12±6.36
	4.30-6pm	89.08±1.74	98.42±8.58
Leq (dBA)	11am-1.30pm	83.13±1.03	86.41±1.56
	4.30-6pm	82.13±1.03	86.41±2.56
MAX P (dBA)	11am-1.30pm	98.97±1.74	106.91±4.43
	4.30-6pm	99.57±1.61	106.37±4.05

Graph 4 : Comparison of noise levels in Controls and Study group



Graph 5: Comparison of noise levels in Controls and Study group (tests)

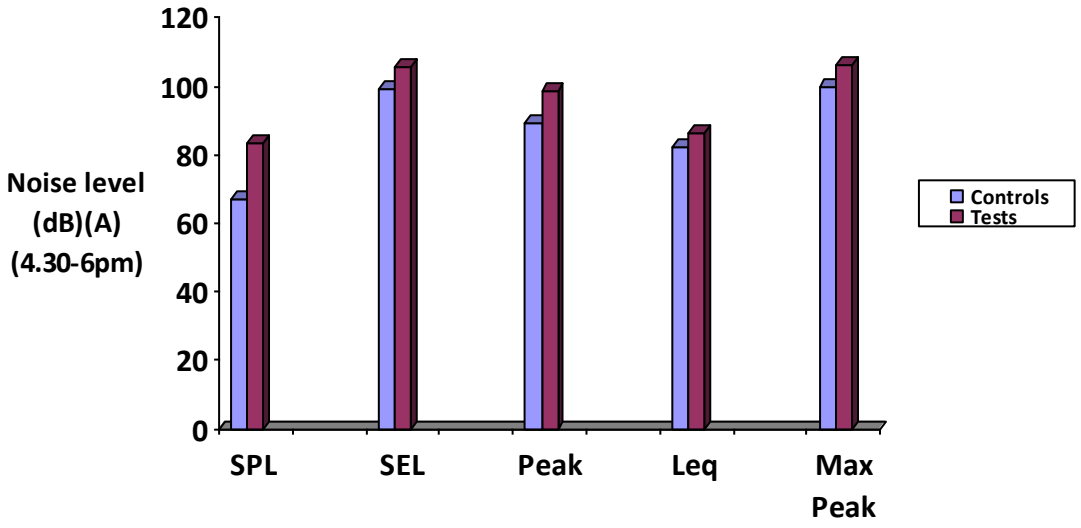


Table 7 : Shows the recording of noise levels at various traffic junctions in Kolar & in around Sri Devaraj Urs Medical College, Kolar where in all the parameters measured were high in the study group compared to controls. (Graph 4 & 5)

Table 8 : Comparison of **Exposure Index** [sound pressure level in dB(A) X duration of exposure to noise in years].

Exposure Index	CONTROL (n=30)	STUDY GROUP (n=30)
Mean ± SD	585.56±280.66	855.36±377.59

Graph 6:

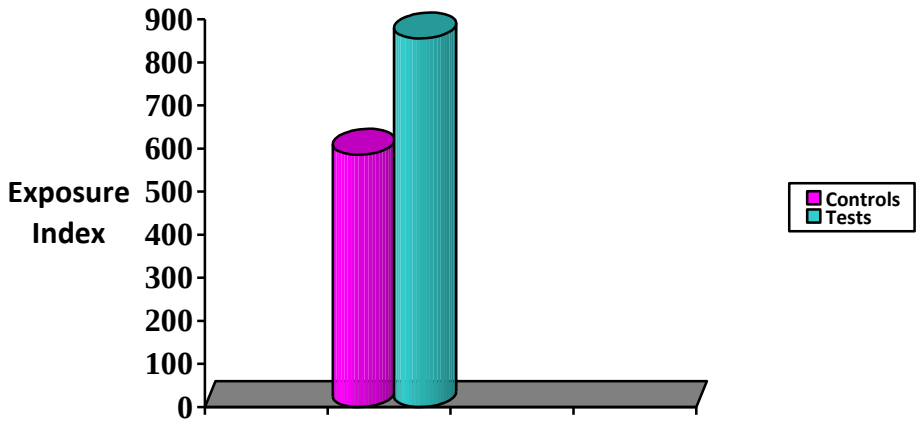


Table 8 : shows the comparison of exposure index which is calculated by multiplying SPL(dBA) and duration of exposure to noise(years) wherein the mean±SD was 585.56±280.66 in controls & 855.36±377.59 in test group and the exposure index was statistically high in test group with p<0.001**. (Graph 6)

Table 9 : Sensorineural hearing loss in study and control group.

GROUPS	SENSORINEURAL HEARING LOSS				TOTAL
	YES		NO		
	Number of subjects	%	Number of subjects	%	
Study Group	8	26.66	22	73.33	30
Controls	0	0	30	100	30

Table 10 : Degree of sensorineural hearing loss in study group (traffic policemen).

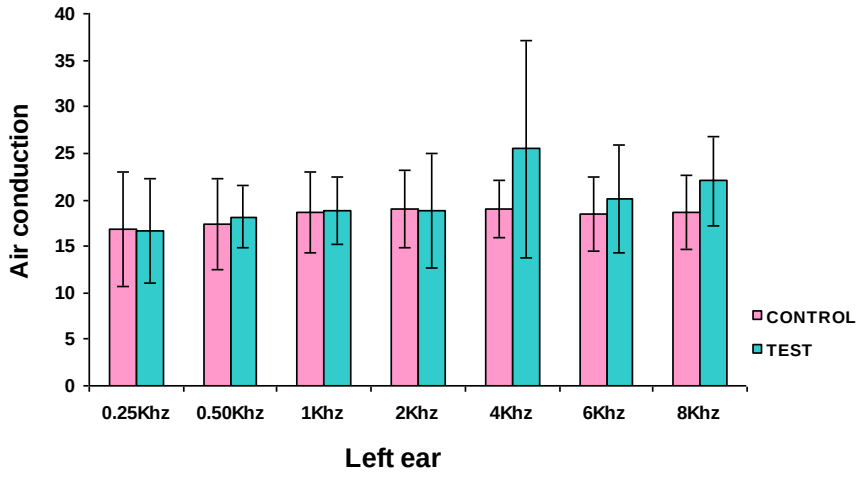
Degree of Sensorineural Hearing Loss	STUDY GROUP (30)	
	Number	**Percentage (%)**
Normal (0-25 dB)	22	73.33
Mild (26-40 dB)	05	16.66
Moderate (41-55dB)	03	0.1
Moderately severe (56-70Db)	00	0
Severe (71-91 dB)	00	0
Profound (>91 dB)	00	0
TOTAL	**30**	**100**

Table 9 and table 10 shows sensorineural hearing loss in study & control group. In the study group, eight (26.66%) had sensorineural hearing loss and there were none in the control group. Among 8, five (16.66%) had mild and 3 (0.1%) had moderate degree hearing loss.

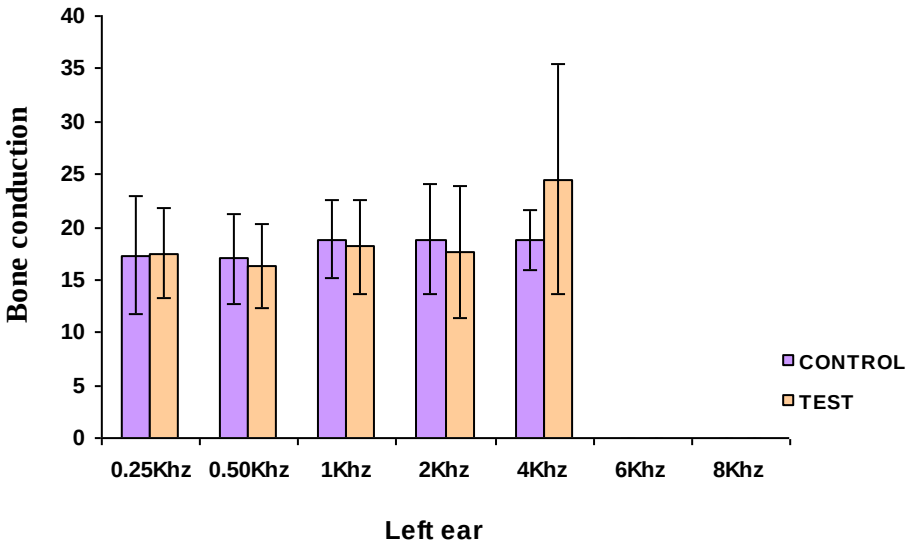
Table 11: Comparison of auditory acuity between control and study group

Frequency	Air conduction			Bone conduction		
	Control	Tests	p value	Control	Tests	p value
Left ear	**(dBA)**	**(dBA)**		**(dBA)**	**(dBA)**	
• 0.25Khz	16.83±6.23	16.67±5.62	0.914	17.33±5.53	17.50±4.31	0.897
• 0.50Khz	17.33±4.87	18.17±3.34	0.443	17.00±4.28	16.33±3.92	0.532
• 1Khz	18.67±4.34	18.83±3.64	0.873	18.83±3.64	18.17±4.45	0.528
• 2Khz	19.00±4.23	18.83±6.11	0.903	18.83±5.2	17.67±6.26	0.436
• 4Khz	19.00±3.05	25.50±11.69	<0.001**	18.83±2.84	24.50±10.6	0.001**
• 6Khz	18.50±3.97	20.17±5.79	0.040*	-	-	-
• 8Khz	18.67±3.92	22.00±4.84	0.001**	-	-	-
Right ear						
• 0.25Khz	15.83±5.88	16.50±5.11	0.641	18.17±5.33	17.33±4.87	0.530
• 0.50Khz	17.17±4.86	18.17±3.59	0.368	18.17±4.64	17.50±3.66	0.539
• 1Khz	18.83±4.29	19.00±3.32	0.867	19.17±3.49	18.83±4.09	0.735
• 2Khz	19.17±4.37	18.83±5.68	0.800	19.00±5.48	18.83±5.52	0.907
• 4Khz	18.50±2.67	25.16±11.78	<0.001**	18.67±2.92	23.16±12.2	0.002**
• 6Khz	18.00±3.62	20.00±6.02	0.015*	-	-	-
• 8Khz	18.00±3.85	22.16±4.49	<0.001**	-	-	-

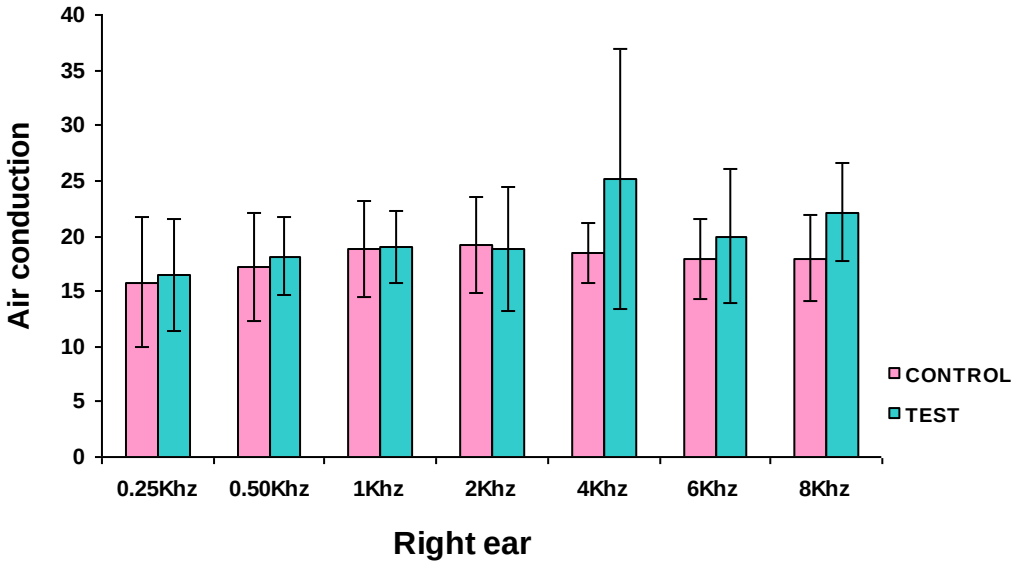
Graph 7: Comparison of Air Conduction between control and study group in left ear.



Graph 8: Comparison of Bone Conduction between control and study group in left ear



Graph 9: Comparison of Air Conduction between control and study group in right ear.



Graph 10 : Comparison of Bone Conduction between control and study group in right ear

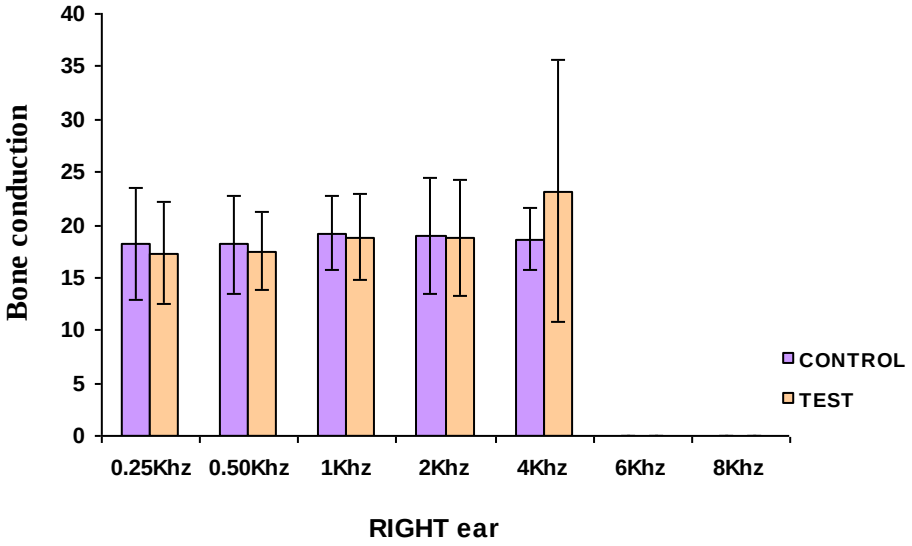


Table 11 : Shows the comparison of hearing thresholds between control and test group in both the ears. It is evident that in the test group, the mean thresholds at frequencies 4Khz (AC and BC), 6Khz(AC) and 8Khz(AC) are increased compared to that in the controls with the significant p value of <0.001 at frequency of 4Khz (AC &BC), 0.04 at 6Khz(AC) and 0.001 at 8Khz(AC) in the left ear and also in the right ear the auditory acuity was statistically significant with a p value of <0.001 at 4Khz(AC &BC) , 0.015 at 6Khz(AC) and <0.001 at 8Khz(AC) respectively. (Graph 7, 8, 9 and 10).

Table 11 : Shows the significant dip (increased hearing threshold) at frequency of 4kHz and recovery at 6Khz and 8Khz in the test group which is typical of noise induced hearing loss (sensorineural).

Table 12 : Comparison of wave latency and Inter peak latency of BAEP.

BAEP	Side involved	CONTROL	TEST	P Value
Wave Latency				
I (ms)	Left	1.74±0.03	1.86±0.22	<0.005*
	Right	1.74±0.04	1.86±0.22	<0.005*
II (ms)	Left	2.81±0.05	2.87±0.09	<0.002*
	Right	2.82±0.07	2.88±0.09	0.018*
III (ms)	Left	3.74±0.02	3.77±0.05	0.002*
	Right	3.74±0.02	3.79±0.05	0.001*
IV(ms)	Left	5.15±0.02	5.18±0.03	0.052*
	Right	5.17±0.02	5.19±0.04	0.051*
V (ms)	Left	5.75±0.02	5.77±0.08	0.05*
	Right	5.75±0.02	5.78±0.08	0.05*
Inter Peak Latencies				
I-III	Left	1.91±0.03	1.92±0.19	0.86
	Right	1.92±0.03	1.93±0.18	0.86
III-V	Left	1.99±0.03	1.99±0.05	0.9
	Right	1.99±0.03	1.99±0.04	0.9
I-V	Left	3.90±0.03	3.91±0.16	0.8
	Right	3.91±0.03	3.92±0.17	0.8

Graph 11: Comparison of wave latency of BAEP

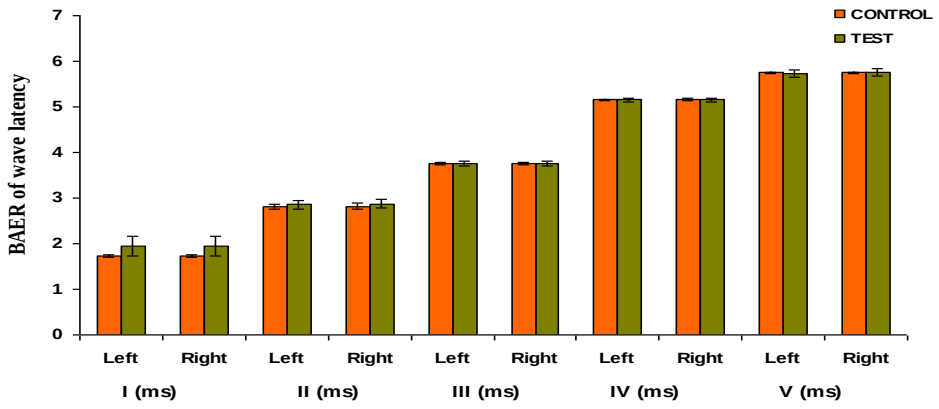


Table 12 : shows the comparison of BAEP in controls and test (study) group. (Graph 11)

Wave I: In the test group, the wave I which is generated in the eighth cranial nerve had mean latency of 1.86±0.22ms in the left ear and 1.86±0.22ms in the right ear which was significantly high with P value of <0.005 for both the ears as compared to the controls where the mean latency of wave I was 1.75±0.03ms in the left ear and 1.74±0.04ms in the right ear

Wave II: In the test group, second wave latency that is wave II which is generated in the cochlear nucleus showed a mean value of 2.87±0.09ms in the left ear and 2.88±0.09ms in the right ear which was significantly high with P value of <0.002 for the left ear and 0.01 for the right ear as compared to the controls where the mean of wave II was 2.82±0.05ms in the left ear and 2.83±0.07ms in the right ear.

Wave III: In the test group, wave III which originates in the superior olivary nucleus had a value of 3.77±0.05ms in the left ear and 3.79±0.05ms in the right ear which was significantly high with P value of <0.002 for the left ear and 0.001 for the right ear as

compared to the controls where the mean of wave III was 3.76±0.02ms in the left ear and 3.76±0.02ms in the right ear.

Wave IV: In the test group, wave IV which originates in the lateral lemniscus had value of 5.18±0.03ms in the left ear and 5.19±0.04ms in the right ear which was statistically significant with p value of 0.05 for the left ear and 0.05 for the right ear as

compared to the controls where the mean of wave IV was 5.16±0.02ms in the left ear and 5.17±0.02ms in the right ear.

Wave V: In the test group, the wave V which originates in inferior colliculi had a mean latency of 5.77±0.08ms in the left ear and 5.78±0.08ms in the right ear which was statistically significant with p value of 0.05 for both

the ears as compared to the controls where the mean of wave V was 5.75±0.02ms in both the ears.

Interpeak latencies:

I-III: In the test group, I-III IPL which is a measure of conduction from proximal eighth nerve across subarachnoid space into the core of lower pons showed a mean value of 1.92±0.19ms in the left ear and 1.93±0.18ms in the right ear compared to the controls where the mean was 1.91±0.03ms in the left ear and 1.92±0.03ms in the right ear which showed no statistical significance.

III-V: In the controls, III-V which indicates conduction from lower pons to midbrain had a mean value of 1.99±0.03 in left ear and 1.99±0.03 in the right ear when compared with test group where in the left ear the mean value was 1.99±0.05 and 1.99±0.04 in the right ear which showed no statistical significance.

V-I: I-V IPL which is a measure of conduction from proximal eighth nerve through pons to midbrain in the control group had mean latency of 3.92±0.03 in left ear and 3.92±0.03 in the right ear when compared to the controls which showed mean of 3.91±0.16 in the left ear and 3.91±0.17 in the right ear males when compared with a value of 3.81±0.04 in female subjects which showed no statistical significance.

Table 13 : Comparison of wave latencies of BAEP in study group with and without hearing loss.

BAEP	Side involved	Study Group		p Value
		No hearing loss	Hearing loss	
Wave Latency				
I (ms)	Left	1.77±0.02	2.05±0.26	0.000**
	Right	1.77±0.02	2.08±0.25	0.000**
II (ms)	Left	2.84±0.05	2.95±0.09	0.000**
	Right	2.85±0.05	2.97±0.10	0.000**
III (ms)	Left	3.77±0.04	3.86±0.02	0.000**
	Right	3.77±0.03	3.87±0.02	0.000**
IV(ms)	Left	5.15±0.06	5.20±0.05	0.000**
	Right	5.17±0.03	5.21±0.04	0.000**
V (ms)	Left	5.75±0.05	5.90±0.04	0.000**
	Right	5.75±0.06	5.91±0.03	0.000**

Table 13 shows comparison of BAEP in study group with and without hearing loss.

The mean of all wave latencies from wave I to wave V is increased in the subjects with hearing loss as compared to the subjects without hearing loss. Application of Student t test revealed a significant difference in latencies with a p value of 0.000**.

Table 14 : Comparison of wave latencies of BAEP.

BAEP	Side involved	CONTROL	STUDY GROUP		P Value
			No hearing loss	Hearing loss	
Wave Latency					
I (ms)	Left	1.74±0.03	1.77±0.02	2.05±0.26	0.000**
	Right	1.74±0.04	1.77±0.02	2.08±0.25	0.000**
II (ms)	Left	2.81±0.05	2.84±0.05	2.95±0.09	0.000**
	Right	2.82±0.07	2.85±0.05	2.97±0.10	0.000**
III (ms)	Left	3.74±0.02	3.77±0.04	3.86±0.02	0.000**
	Right	3.74±0.02	3.77±0.03	3.87±0.02	0.000**
IV(ms)	Left	5.15±0.02	5.15±0.06	5.20±0.05	0.000**
	Right	5.17±0.02	5.17±0.03	5.21±0.04	0.000**
V (ms)	Left	5.75±0.02	5.75±0.05	5.90±0.04	0.000**
	Right	5.75±0.02	5.75±0.06	5.91±0.03	0.000**

Table 14 shows the comparison of BAEP between study group with and without hearing loss and controls. The mean wave latencies from wave I to Wave V is increased in study group with the hearing loss compared to study group without hearing loss and that of controls and application of ANOVA revealed a significant difference in latencies with a p value of 0.000**. (Graph 12)

Graph 12 : Comparison of wave latencies of BAEP

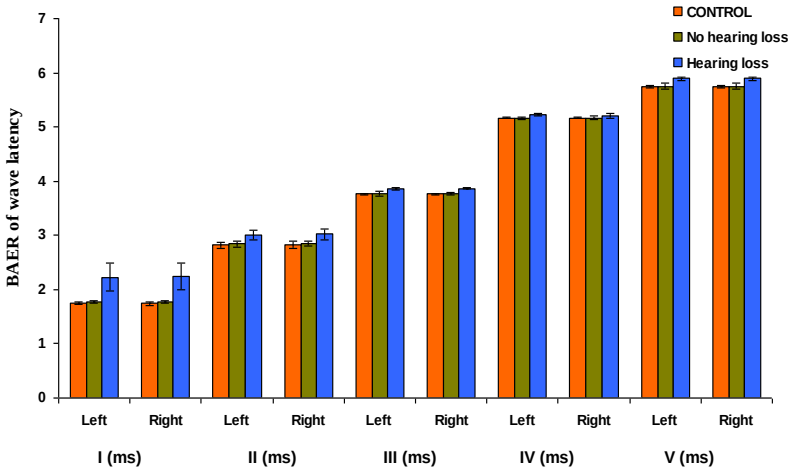


Table 15 : Comparison of wave latencies of BAEP in control group and study group without hearing loss.

BAEP	Side involved	Control	Study group without Hearing loss	P value
Wave Latency				
I (ms)	Left	1.74±0.03	1.77±0.02	0.000**
	Right	1.74±0.04	1.77±0.02	0.000**
II (ms)	Left	2.81±0.05	2.84±0.05	0.000**
	Right	2.82±0.07	2.85±0.05	0.000**
III (ms)	Left	3.74±0.02	3.77±0.04	0.000**
	Right	3.74±0.02	3.77±0.03	0.000**
IV(ms)	Left	5.15±0.02	5.15±0.06	0.9
	Right	5.17±0.02	5.17±0.03	0.9
V (ms)	Left	5.75±0.02	5.75±0.05	0.89
	Right	5.75±0.02	5.75±0.06	0.9

Table 15 - shows wave latencies of BAEP which was statistically prolonged in study group without hearing loss in wave I, wave II and wave III with p value of 0.000. There was no statistical significance in wave IV and wave V between the groups.

Table 16 : Pearson correlation of exposure index with wave latencies of BAEP in study group.

Exposure index vs wave latencies of BAEP	Side involved	TEST	
		r value	p value
Exposure index vs Wave Latency			
I (ms)	Left	0.438	0.01*
	Right	0.444	0.01*
II (ms)	Left	0.421	0.02*
	Right	0.355	0.05*
III (ms)	Left	0.193	0.31
	Right	0.271	0.14
IV(ms)	Left	0.358	0.05*
	Right	0.368	0.05*
V (ms)	Left	0.337	0.06
	Right	0.370	0.370

Table 15 shows positive correlation between exposure index and the wave latencies

For the wave I, there is a moderate correlation(r=0.44) with a p value of 0.01 in the left ear and r=0.421 with a p value of 0.02 in the right ear after application of Pearson correlation. For the wave II, there is a moderate correlation(r=0.42) with a p value of 0.02 in the left ear and r=0.355 with a p value of 0.05 in the right ear. Wave III: there is a small correlation(r=0.19) with a p value of 0.33 in the left ear and r=0.27 with a p value of 0.14 in the right ear. Wave IV: there is a moderate correlation(r=0.35) with a p value of 0.05 in the left ear and r=0.37 with a p value of 0.05 in th e right ear. Wave V: there is a moderate correlation(r=0.337) with a p value of 0.06 in the left ear and r=0.37 with a p value of 0.37 in the right ear.

DISCUSSION

Noise has been a bane and has altered the ecological balance. Traffic policemen engaged in controlling traffic noise, particularly at heavy traffic junctions belong to the high risk group to be affected by hazards of noise.

Recently the brain stem auditory potentials (BAEP) have been widely studied in audiology, neurology, neonatology and anaesthesiology and have been used as a diagnostic technique in audiology in investigating hearing loss in addition to audiometer. Among all objective methods of hearing evaluation, brainstem auditory evoked potential is considered the most used precocious potential in clinical practice. BAEP is one of the audiological tools used when diagnosing individuals exposed to noise, chemical agents which are the common causes of irreversible sensorineural hearing loss in both children and adults.[71] With the above background, the present study has been carried out to study the effect of road traffic noise on BAEP in traffic policemen.

Studies have shown that head size influences BAEP.[28, 29] So, in the present study, wave latencies and interpeak latencies were normalized (latency/HC*mean HC) to avoid intersubject variability.[28, 29, 30] Head circumference (HC) in study group was (mean$\pm$SD) 54.38$\pm$1.09cm and 53.99$\pm$0.87cm in control group which showed no statistical significance.

Age also influences BAEP. So age matched controls were selected with mean$\pm$SD of 42.46$\pm$6.78yrs & 41.63$\pm$7.66yrs in study & control group respectively, the difference in mean being not statistically significant.

First, noise induced hearing loss (NIHL) was assessed by self assessed questionnaire in all the subjects. In the present study, only three (10%) of the traffic policemen felt that their hearing ability was below average while none in the control group had hearing loss by self assessed questionnaire.[61] A similar study among traffic cops in Gujarat showed that 2.3% of the subjects felt that their hearing ability was below average.[61] Impairment of hearing at high frequencies will initially cause a loss of clarity in perceived speech and then interfere with daily activities as hearing loss progresses. Hearing loss-related symptoms, such as trouble in normal and telephone conversation, turning up the radio/television volume and tinnitus, usually occur in the early stages of NIHL.[77, 78]

Most of the traffic policemen (73.33%) said that PPEs (personal protective equipments) like ear plugs, ear muffs will be effective if used in a situation where one is exposed to chronic and loud noise. It was observed that none of the traffic policemen used PPEs and the reason for their non-usage was non-availability in most of the subjects. Similar study done on traffic policemen of Gujarat showed that none of them used PPEs because of non-availability and 67.4% subjects did not use any method to reduce exposure to noise.[61] Ear protectors (ear plugs or ear muffs) should be used where noise levels exceed 85 dB (A). They provide protection up to 35 dB.[13]

Noise levels were measured using sound level meter at various junctions at which subjects were posted. In our study, sound pressure level (dBA) that the study group (mean$\pm$SD) (84.49$\pm$3.44) was exposed to was significantly more than controls (67.13$\pm$1.77). In India, Occupational permissible exposure limit for 8 hour time weighted average is 90 dBA.[77, 79] But the levels in the metros is almost 90 dBA. Prolonged exposure to high noise levels can lead to irreversible nerve

damage and also affect hormones and lead to psychological problems in the long run. A survey in January 2009 listed Bangalore as the noisiest state capital in India.[62]In a study done in Cairo traffic policemen , noise level ranged from 72-110dBA.[64] Among those who had completed 5 years in the traffic wing had hearing loss in various degrees.[77,80] A study done in thirty-two points in Kaula Lumpur recorded a maximum sound of 108.2 dBA and the noise emitted by vehicles were upto 133 dBA.[81]

Leq (Equivalent Continuous Noise Level) can be directly compared with an exposure standard [85dB(A)] to determine whether the noise is likely to be damaging to a person's hearing ability.[15]In the present study, Leq (dBA) measured was significantly more in study group (mean$\pm$SD) 86.41$\pm$2.56 dBA as compared to 82.13$\pm$1.03 dBA in the control group.

Exposure index was calculated by multiplying sound pressure level (dBA) and duration of exposure to noise (years). It was significantly high in study group (mean$\pm$SD) (855.36±377.59) as compared to controls (585.56±280.66). This shows that traffic policemen were more exposed to continuous and loud noise compared to controls. The amount and the type of direct hair cell damage depends on noise intensity and duration of exposure to noise during a typical working day and overall exposure during working life which provides a cumulative effect on hearing.

In our study, pure tone audiometer revealed that, out of 30 traffic policemen, 8 (26.6%) had NIHL and among these, 5 had mild degree of hearing loss and 3 had moderate degree of hearing loss and there were no subjects with NIHL in control group. Duration of exposure to noise (years) and sound pressure level (dBA) in study group with hearing loss (12.25$\pm$3.69 years and 85.80$\pm$3.16 dBA) was significantly prolonged

compared to traffic policemen without hearing loss (8.95±3.37 years & 81.85±3.37 dBA). A study of noise exposure and hearing loss among traffic policemen working at busy streets of Jalgaon Urban centre, Maharashtra showed that 84% of them had hearing loss.[67] A survey on the effects of noise pollution on traffic policemen in the city of Hyderabad, India, carried out by the Society to aid the hearing impaired, revealed that 76% had NIHL.[77]

Higher sound levels damage the outer hair cells, stereocilia, further, including destruction of the interciliary bridges, and recovery takes longer. An even higher level of sound leads to a collapse of the stereocilia and the hair cell is eventually damged permanently. If the outer hair cells are not functioning, a greater stimulation is required to initiate a nervous impulse; thus, the threshold sensitivity of inner hair cells is raised, which is perceived as a hearing loss.[77]

In the present study, the average hearing threshold at frequencies 4kHz (AC and BC), 6kHz(AC) and 8kHz (AC) were significantly increased in the study group as compared to controls. There was a significant dip at frequency of 4kHz and recovery at 6kHz and 8kHz in the study group which is typical of noise induced hearing loss (sensorineural).[15] NIHL observed is bilateral and shows a similar pattern in both the ears which is in accordance with the literatures.[77]A similar study conducted on hearing threshold of Cairo(Egypt) [64] , Dhaka traffic policemen[68] and French traffic officers[69] showed that the mean hearing threshold of traffic policemen was significantly higher than that of controls which affected mainly frequencies of 4-6kHz. In a study 'Audiometric notch as a sign of NIHL', it was found that notch at 4kHz is a well established clinical sign and may be valuable in confirming the diagnosis of NIHL and 6kHz notch is variable and is of little importance.[66]

Sound damages the ear first at a frequency of about 4 kHz and one of the reasons for this is the acoustic resonance characteristics of the external ear. This hard walled tube, closed at one end, amplifies acoustic energy in the upper frequencies by about 10 decibels. In addition, individual variation in the acoustic transfer characteristics of the tube is a factor in the large variability in people's susceptibility to noise. Hair cells in the basal coil of the cochlea are the most sensitive to noise damage; they are responsible for transducing higher frequencies and this accounts for the high frequency hearing loss found in noise-damaged ears.[16]

BAEP was recorded in traffic policemen with and without NIHL and controls. In our study, from the table 12, 13 and 14, it is seen that the mean wave latencies are significantly prolonged in subjects with NIHL as compared with the subjects without NIHL in the study group (traffic policemen) and that of controls. Interpeak latencies showed no statistical significance between study and control groups. Earlier studies too have reported prolongation of wave latencies with normal interpeak latencies.

A study on BAEP in individuals with sensorineural hearing loss showed that 44(40%) had normal results of BAEP & 65(60%) had altered BAEP ie., increase values of absolute latencies of wave I, III and V though with normal values of interpeak latencies I-III, III-IV and I-V. [71] Rosenhamer in 1981 studied 11 ears with rising, 22 ears with flat and 77 ears with sloping audiograms (etiologies consisted mostly of Meniere's disease, noise induced hearing loss and previous sudden hearing loss) and concluded that IPLs were not significantly affected by cochlear hearing loss.[72] Eggermont in 1980 studied 70 normal hearing subjects & 43 subjects with unilateral hearing loss of cochlear origin and found essentially no difference in the I-V IPL between the two groups.[73] Evidence reported in the literature indicates

that wave I of the BAEP is influenced by cochlear contributions from a more basal area of cochlea than is wave V. In high frequency hearing losses, wave I tends to be delayed at all intensities and by a greater amount than wave V.[74]

One probable cause for prolongation of wave latencies of BAEP is that the hair cells in the organ of Corti may be damaged directly by noise, or indirectly by very high levels of continuous sound which causes vasoconstriction of the vessels of the stria vascularis in the cochlear blood supply. This renders the hair cells relatively anoxic and thus secondarily damaged. The amount and type of direct hair cell damage depends on the intensity of the sound. Above a certain minimum of frequency and intensity, the outer hair cells show signs of metabolic exhaustion with drooping of the stereocilia. This correlates with the common phenomenon of temporary threshold shift (TTS), which recovers within a few hours. Higher sound levels damage the outer hair cell stereocilia further, including destruction of the inter-cilial bridges, and recovery takes longer. An even higher level of sound leads to collapse of the stereocilia, and the hair cell is eventually phagocytosed. Outer hair cells amplify the movement of the basilar membrane of the cochlea by contracting when stimulated by sound. This increases the stimulus delivered to the inner hair cells which transduce the mechanical movement to trigger a nervous impulse in the afferent nerve endings of the 8th nerve. If the outer hair cells are not functioning, greater stimulation is required to initiate a nervous impulse; thus the threshold sensitivity of the inner hair cells is raised which is perceived as a hearing loss.[16]

One more explanation is that known that organ of Corti has two functional systems, one is of high intensity composed of internal ciliated cells (inner hair cells) connected to the largest part of afferent neural fibers and the another of low intensity composed by external

ciliated cells (outer hair cells), which constitute the cochlear amplifier and interact with the inner hair cells, soothing it in order to respond to low intensity stimuli. Therefore occurrence of such results on hearing losses can be justified by the fact that the acoustic stimulus reaching cochlear area gets weakened due to peripheral involvement, eliciting the responses of outer hair cells which form synapse with only 10% of the afferent neural fibers and need some time to soothe the inner hair cells, enlarging the latencies of waves of BAEP.[71]

Significant correlation was observed between wave latencies (wave I, II and IV) of BAEP and exposure index highlighting the influence of duration of exposure to noise in addition to noise level on wave latencies.

It is interesting to note that wave latencies (wave I, II and III) were significantly prolonged in traffic policemen without NIHL as compared to controls suggesting that these changes precede the audiometric findings and hence BAEP might serve as a useful tool in detecting early signs and hence necessary precautions can be taken to prevent deleterious effects of noise.

Another study showed that there exist a relationship between severity of noise induced hearing loss indicated by Pure Tone Audiometry and patterns of auditory brainstem response wave abnormalities among workers with noise induced permanent hearing loss. Abnormal wave patterns were detected in 72.7% of ears.[75]

In conclusion, these results suggest that continuous exposure to loud and chronic noise cause sensorineural hearing loss in long run and prolonged wave latencies of BAEP are seen even before it becomes evident in audiometry.

SUMMARY

This study was conducted in the Department of Physiology, Sri Devaraj Urs Medical College, Kolar, to evaluate the effect of road traffic noise exposure on BAEP (Brainstem Auditory Evoked Potential) in traffic policemen. 30 traffic policemen and 30 aged matched controls were subjected to pure tone audiometer and BAEP recordings. Statistical analysis revealed that traffic policemen had significantly higher thresholds of hearing and the wave latencies were significantly prolonged than the controls. Within the study group, the wave latencies were higher in traffic policemen who showed mild to moderate degree of hearing loss than traffic policemen without hearing loss. There was a positive correlation between exposure index (noise level*duration of exposure of noise) and wave latencies (I, III & IV) in the study group. The wave latencies (wave I, II and III) were significantly prolonged in traffic policemen without NIHL as compared to controls suggesting that these changes precede the audiometric findings and hence BAEP might serve as a useful tool in detecting early signs and hence necessary precautions can be taken to prevent deleterious effects of noise.

CONCLUSIONS

1. Only 3 out of 30 traffic policemen studied had self reported hearing loss whereas BAEP showed prolonged wave latencies in all of them (traffic policemen).

2. Sound pressure level (dBA) was significantly high in study group than the controls.

3. The exposure index {noise level (dBA)*duration of exposure to noise in years} was significantly high in study group compared to controls.

4. Traffic policemen who were exposed to loud and continuous noise showed significantly higher thresholds than the controls. Significant differences in thresholds of both the ears are seen particularly in the higher frequencies.

5. Wave latencies were significantly prolonged in study group compared to controls.

6. Within the study group, the wave latencies were significantly prolonged in the subjects who had mild to moderate degree of hearing loss than the subjects without hearing loss.

7. Wave latencies (wave I, II and III) of BAEP were significantly prolonged in traffic policemen without NIHL as compared to controls suggesting that these changes precede the audiometric findings and hence BAEP might serve as a useful tool in detecting early signs and hence necessary precautions can be taken to prevent deleterious effects of noise.

Auditory brainstem response (ABR) audiometry is considered as an effective tool in the evaluation of suspected retrocochlear pathology[35] and BAEP latencies are accurate in estimating hearing threshold.[37]

Traffic constables play a very significant role in controlling the traffic system especially in the metropolitan cities despite several limitations and these policemen engaged in controlling traffic noise, particularly at heavy traffic junctions; belong to the high risk group to be affected by health hazards of noise.

NIHL is major avoidable cause of permanent hearing impairment and the most effective way to prevent NIHL from hazardous noise at the workplace is by using personal protective equipments (PPEs) like ear plugs, ear muffs which was used by none in the present study and non-availability was the reason for their non-usage in most of the subjects. Most of the study subjects are in the economically productive age groups and if they suffer from hearing disability at this age, they would have to live with that disability throughout their life and if effective measures are taken at this stage, health hazards could well be prevented. Thus it is suggested that not only should these PPEs be made available, but also periodic workshops should be carried out to motivate the subjects for their correct and regular usage.

With this background, some preventive modalities for hearing conservation in the form of safety equipment, periodic checkups and duty scheduling for exposure limitation can be suggested and awareness should be created among traffic policemen about the harmful effects of noise on hearing by implementing education and training programmes. **Dealing with Noise & its Effects is a personal responsibility, A Work Place Responsibility And A Community Responsibility**.

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EFFECT OF ROAD TRAFFIC NOISE EXPOSURE ON BRAINSTEM AUDITORY EVOKED POTENTIALS IN TRAFFIC POLICEMEN

Investigator : Dr. Kavana G. Venkatappa

Guide : Dr. Vinutha Shankar M.S

A. SELF ASSESSED QUESTIONNAIRE

1. Demographic Characteristics

NAME	
AGE	
SEX	
EDUCATION STATUS	
DESIGNATION	
DURATION OF EXPOSURE (since joined)	
PLACE OF DUTY, TIMINGS	

2. Clinical Assessement of Quality of Hearing

Quality of hearing Excellent, above average, average, Below average.	
Hearing over phone Without difficulty, do miss some conversation, Miss a lot of what is said.	
Hearing in crowd Without difficulty Do miss some conversation Miss a lot of what is said	

Sound of TV/radio Usually louder Usually same loudness, a little louder	
Do people often indicate that u r talking too loudly Yes No	
Do people often have to talk louder Yes/no	
Tinnitus; almost all the time, >once a day, about a Day, about once a week, >once a year. Is it work related?	

3. According to the use of Ear Plugs / Ear Muffs

Ever used ear plugs Yes /No	
Regularity of usage Seldom /Never	
Reason for non usage Uncomfortable, not available, bad habit, dislike Cause for headache.	
Other usage Hands, cotton, fingers Don't use anything	
How effective are these methods Average, good, better than plugs	

B) Noise levels measured in & around Sri Devaraj Urs Medical College, Kolar (Control Group).

RLJH, KOLAR (Controls)	TIMINGS	SPL (dBA)	SEL (dBA)	PEAK (dBA)	Leq (dBA)	MAX P (dBA)
Administrative block, SDUMC	11am-1.30pm	64	92	86.1	84	96.8
	4.30-6pm	65.2	95.6	87.2	84.3	98.1
Inside hospital	11am-1.30pm	69.9	101.2	92	87.6	101.2
	4.30-6pm	69	101	90	87.6	101.1
Outside hospital	11am-1.30pm	69	101	90	87.6	101.1
	4.30-6pm	70	102.3	92	87.8	101.4
PG block	11am-1.30pm	65.2	95.6	87.2	84.3	98.1
	4.30-6pm	65	95.4	87.2	84	98
Departments, SDUMC	11am-1.30pm	67.4	98.6	88.6	87.2	99.2
	4.30-6pm	68.9	100.1	89	87	99.4
Near canteen	11am-1.30pm	67.4	98.6	88.6	87.2	99.2
	4.30-6pm	69	101	90	87.6	101.1
Police station, Inside campus	11am-1.30pm	66	96	88	87	97.2
	4.30-6pm	67	98.2	88.2	87.1	97.9

B. Noise levels measured at various junctions, kolar (Study Group).

TRAFFIC JUNCTIONS KOLAR (Study Group)	TIMINGS	SPL (dBA)	SEL (dBA)	PEAK (dBA)	Leq (dBA)	MAX P (dBA)
Brindhavan Circle	11am-1.30pm	82.2	104.2	96.3	82	105.8
	4.30-6pm	83.3	104.2	96.3	82	105.8
Mekke Circle	11am-1.30pm	88.7	107	105.6	82.3	109.5
	4.30-6pm	88.9	107.8	106.6	83	109.8
Ammavarpete Circle	11am-1.30pm	79.7	103.3	92.2	78.5	102
	4.30-6pm	80.2	104.5	87	79.8	102.8
KRC Circle	11am-1.30pm	83.3	104.2	96.3	82	105.8
	4.30-6pm	80.4	104.6	87.2	79.8	102.8
Pallavi Circle	11am-1.30pm	84.2	106.2	105.7	91.5	106.9
	4.30-6pm	82	104.2	96	82	105
Clock Tower	11am-1.30pm	89.4	113	92.4	80.3	118.2
	4.30-6pm	89.3	110.3	108.3	93.2	114.2
Doomlight Circle	11am-1.30pm	82	104.4	96	82	105
	4.30-6pm	79.7	103.3	92.2	78.5	102
Bangarpete Circle	11am-1.30pm	82.4	104.4	97.2	83	105.2
	4.30-6pm	79.8	103.8	92..4	78.6	103
M.G Chowk	11am-1.30pm	85.5	107	93	90	105
	4.30-6pm	87.8	107.8	106.6	83	109.8
Yelepete Circle	11am-1.30pm	76.9	104.5	87	79.8	102.8
	4.30-6pm	78.3	103.3	88	80	101.3
Old Bus Stand	11am-1.30pm	87.9	107.8	106.6	83	109.8
	4.30-6pm	88.6	107	105.6	82.2	108.5

(CONTROL GROUP) : HEARING THRESHOLDS OF LEFT EAR.

Sl. No.	0.25Khz	0.25Khz	0.50Khz	0.50Khz	1Khz	1Khz	2Khz	2Khz	4Khz	4Khz	6Khz	6Khz	8Khz	8Khz
	AC (dB)	BC (dB)	AC (dB)	BC (dB)	AC (dB)	BC (dB)	AC (dB)	BC (dB)	AC (dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)
1	15	10	20	15	25	20	20	25	20	20	25		20	
2	20	20	25	20	15	20	25	25	20	20	20		25	
3	15	10	20	15	20	10	20	15	20	20	25		15	
4	20	20	25	20	20	20	25	20	15	20	20		15	
5	15	25	15	20	20	25	15	5	10	20	20		20	
6	20	20	20	15	20	20	15	20	20	20	15		15	
7	20	20	5	10	20	20	20	15	15	20	20		20	
8	20	15	20	20	20	15	15	20	20	20	20		20	
9	20	20	15	15	20	20	20	20	20	15	20		20	
10	25	25	20	20	20	20	20	15	20	20	25		25	
11	15	20	20	20	15	20	20	20	20	20	20		20	
12	15	15	15	20	20	25	25	20	20	15	20		20	
13	5	10	5	15	10	15	20	20	20	20	15		20	
14	25	15	20	20	20	20	20	20	20	20	20		20	
15	20	20	15	20	20	15	20	20	20	20	20		20	
16	5	20	20	15	10	20	15	20	20	15	10		15	
17	15	20	15	20	20	15	20	15	20	15	15		10	
18	25	20	20	25	25	20	25	20	15	15	20		15	
19	15	25	15	20	20	15	20	25	20	20	20		20	
20	10	15	20	15	15	20	15	15	15	20	15		15	
21	5	15	15	5	15	15	5	5	20	20	15		20	
22	10	15	20	25	20	20	25	15	20	15	20		15	
23	20	15	20	20	20	20	15	25	20	20	25		20	
24	5	5	10	10	15	15	15	20	20	20	15		15	
25	20	25	20	15	20	15	20	20	20	20	15		15	
26	25	20	20	25	20	25	20	20	20	20	15		15	
27	15	5	10	15	10	15	20	15	15	20	15		15	
28	20	15	20	20	30	25	20	30	25	20	20		25	
29	25	25	20	20	20	20	20	20	15	10	10		20	
30	20	15	15	20	15	20	15	20	20	20	20		25	

(CONTROL GROUP) : HEARING THRESHOLDS OF RIGHT EAR

Sl. No.	0.25Khz AC(dB)	0.25Khz BC(dB)	0.50Khz AC(dB)	0.50Khz BC(dB)	1Khz AC(dB)	1Khz BC(dB)	2Khz AC(dB)	2Khz BC(dB)	4Khz AC(dB)	4Khz BC(dB)	6Khz AC(dB)	6Khz BC(dB)	8Khz AC(dB)	8Khz BC(dB)
1	15	20	15	20	20	15	20	15	20	15	15		10	
2	25	20	20	25	25	20	25	20	15	15	20		15	
3	15	25	15	20	20	15	20	25	20	20	20		20	
4	10	15	20	15	15	20	15	15	15	20	15		15	
5	5	15	15	5	15	15	5	5	20	20	15		20	
6	10	15	20	25	20	20	25	15	20	15	20		15	
7	20	15	20	20	20	20	15	25	20	20	25		20	
8	5	5	10	10	15	15	15	20	20	20	15		15	
9	20	25	20	15	20	15	20	20	20	20	15		15	
10	25	20	20	25	20	25	20	20	20	20	15		15	
11	15	5	10	15	10	15	20	15	15	20	15		15	
12	20	15	20	15	20	15	20	15	20	20	20		25	
13	25	25	20	20	20	20	20	20	15	10	10		20	
14	20	15	15	20	15	20	15	20	20	20	20		25	
15	20	20	25	20	20	20	25	20	15	20	20		15	
16	15	25	15	20	20	25	15	5	10	20	20		20	
17	20	20	20	15	20	20	15	20	20	20	15		15	
18	20	20	5	10	20	20	20	15	15	20	20		20	
19	20	15	20	20	20	15	15	20	20	20	20		20	
20	20	20	15	15	20	20	25	30	20	15	20		20	
21	25	25	20	20	20	20	20	15	20	20	25		25	
22	15	20	20	20	15	20	20	20	20	20	20		20	
23	15	15	15	20	20	25	25	20	20	15	20		20	
24	5	10	5	15	10	15	20	20	20	20	15		20	
25	25	15	20	20	20	20	20	20	20	20	20		20	
26	20	20	15	20	20	15	20	20	20	20	20		20	
27	5	20	20	15	10	20	15	20	20	15	10		15	
28	15	20	15	20	30	25	20	30	20	25	15		10	
29	25	20	20	25	25	20	25	20	15	15	20		15	
30	15	25	25	20	20	25	20	25	20	20	20		20	

(TEST GROUP) : HEARING THRESHOLDS OF LEFT EAR

SL N0.	0.25kHZ	0.25Khz	0.50Khz	0.50Khz	1Khz	1Khz	2Khz	2Khz	4Khz	4Khz	6Khz	6Khz	8Khz	8Khz
	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)
1	15	25	15	20	20	15	20	20	20	20	20		20	
2	10	15	20	15	15	20	15	15	15	20	15		15	
3	5	15	15	5	15	15	5	5	20	20	15		20	
4	25	25	20	20	20	20	20	15	20	20	25		25	
5	20	15	20	20	20	20	20	15	20	20	25		25	
6	25	25	20	20	20	20	20	20	15	10	10		20	
7	20	15	20	15	20	15	20	15	30	30	20		20	
8	20	15	20	15	10	10	20	20	20	20	15		15	
9	20	15	15	15	20	25	25	25	25	25	20		25	
10	15	15	15	15	20	15	20	10	40	40	30		25	
11	20	20	15	15	20	20	15	15	20	20	20		20	
12	25	25	20	20	20	20	20	15	20	20	25		25	
13	20	15	15	15	20	15	20	10	30	30	20		20	
14	20	15	20	15	15	20	15	15	15	20	15		15	
15	20	15	15	5	15	15	5	5	20	20	15		20	
16	25	20	15	15	20	15	20	25	50	50	30		25	
17	20	25	15	15	20	25	25	25	50	45	20		25	
18	20	15	20	20	15	20	15	25	40	40	25		25	
19	15	25	15	20	20	15	20	25	20	20	20		20	
20	10	15	20	15	15	20	15	15	15	20	15		15	
21	15	15	20	20	25	30	20	20	40	40	20		20	
22	20	20	20	20	20	15	20	20	40	40	25		25	
23	15	15	20	15	20	10	15	25	40	40	30		25	
24	20	15	20	15	20	15	20	15	20	20	20		25	
25	25	25	20	20	20	20	20	20	15	10	10		20	
26	20	15	15	20	15	20	15	20	20	20	20		25	
27	25	20	20	20	15	20	15	25	55	50	30		30	
28	20	15	15	15	20	15	20	10	40	40	30		25	
29	20	15	15	15	20	25	25	25	25	25	20		25	
30	25	10	30	15	30	15	40	10	40	10	40		40	

(TEST GROUP) : HEARING THRESHOLDS OF RIGHT EAR

Sl. No.	0.25Khz	0.25Khz	0.50Khz	0.50Khz	1Khz	1Khz	2Khz	2Khz	4Khz	4Khz	6Khz	6Khz	8Khz	8Khz
	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)	AC(dB)	BC(dB)
1	10	15	20	15	15	20	15	15	15	20	15		15	
2	5	15	15	5	15	15	5	5	20	20	15		20	
3	25	25	20	20	20	20	20	15	20	20	25		25	
4	20	15	20	20	20	20	20	15	20	20	25		25	
5	25	25	20	20	20	20	20	20	15	10	10		20	
6	20	15	15	15	20	25	25	25	25	25	20		25	
7	20	15	20	15	20	15	20	15	30	30	20		20	
8	25	25	20	20	20	20	20	20	15	10	10		20	
9	20	15	15	20	15	20	15	20	20	20	20		25	
10	15	15	15	15	20	15	20	10	40	40	30		25	
11	25	25	20	20	20	20	20	20	15	10	10		20	
12	20	15	15	20	15	20	15	20	20	20	20		25	
13	20	15	15	15	20	15	20	10	30	30	20		20	
14	25	25	20	20	20	20	20	20	15	10	10		20	
15	20	15	15	20	15	20	15	20	20	20	20		25	
16	25	20	15	15	20	15	20	25	50	50	30		25	
17	20	25	15	15	20	25	25	25	50	45	20		25	
18	20	15	20	20	15	20	15	25	40	40	25		25	
19	15	20	20	20	15	20	20	20	20	20	20		20	
20	15	15	15	20	20	25	25	20	20	15	20		20	
21	15	15	20	20	25	30	20	20	40	40	20		20	
22	20	20	20	20	20	15	20	20	40	40	25		25	
23	15	15	20	15	20	10	15	25	40	40	30		25	
24	20	15	20	20	20	20	15	25	20	20	25		20	
25	5	5	10	10	15	15	15	20	20	20	15		15	
26	25	20	20	20	20	15	15	20	25	30	25		25	
27	25	20	20	20	15	20	15	25	55	50	30		30	
28	20	15	15	15	20	15	20	10	40	40	30		25	
29	20	15	20	20	20	20	15	25	20	20	25		20	
30	25	10	30	15	30	15	40	10	40	10	40		40	

(TRAFFIC POLICEMEN) : BAEP - WAVE LATENCIES & INTERPEAK LATENCIES

Sl. No.	Duration of Noise exposure (yrs)	HC (cm)	SPL (dB)	Age (yrs)	I(ms)		II(ms)		III(ms)		IV(ms)		V(ms)		I – III (ms)		III – V (ms)		I – V (ms)	
					L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R
1	3	53	80.2	28	1.77	1.76	2.81	2.82	3.75	3.76	5.13	5.14	5.71	5.7	1.98	2	1.96	1.94	3.94	3.94
2	4	54	84.2	29	1.74	1.75	2.72	2.76	3.74	3.76	5.14	5.14	5.71	5.68	2	2.01	1.97	1.92	3.97	3.93
3	3	54	87.9	30	1.75	1.76	2.84	2.85	3.8	3.8	5.15	5.18	5.7	5.72	2.05	2.04	1.9	1.92	3.95	3.96
4	11	56	89.3	47	2.01	2.02	3.11	3.10	3.87	3.85	5.25	5.26	5.89	5.87	2.07	2.07	2.02	2.02	4.09	4.09
5	7	55	89.3	42	1.80	1.78	2.88	2.87	3.77	3.77	5.17	5.15	5.74	5.76	1.97	1.99	1.97	1.99	3.94	3.98
6	6	53.5	82.4	33	1.78	1.79	2.86	2.87	3.82	3.81	5.15	5.17	5.73	5.76	2.04	2.02	1.91	1.95	3.95	3.97
7	11	54	87.9	37	1.77	1.75	2.88	2.89	3.77	3.75	5.15	5.16	5.72	5.75	2	2	1.95	2	3.95	4
8	10	54	85.5	37	1.78	1.76	2.86	2.88	3.75	3.76	5.14	5.15	5.75	5.76	1.97	2	2	2	3.97	4
9	6	55	87.9	42	2.01	2.02	3.11	3.10	3.86	3.85	5.19	5.21	5.91	5.93	2.09	2.07	2.05	2.08	4.14	4.15
10	10	54	84.2	42	1.78	1.76	2.88	2.9	3.76	3.76	5.15	5.17	5.74	5.75	1.98	2	1.98	1.99	3.96	3.99
11	9	54	88.6	38	1.75	1.76	2.86	2.88	3.75	3.76	5.15	5.12	5.73	5.76	2	2	1.98	2	3.98	4
12	8	53	87.9	39	1.77	1.76	2.85	2.8	3.75	3.76	5.14	5.15	5.74	5.75	1.98	2	1.99	1.99	3.97	3.99
13	18	56.5	85.5	44	1.77	1.79	2.85	2.86	3.75	3.76	5.16	5.17	5.75	5.76	1.98	1.97	2	2	3.98	3.97
14	14	54	79.7	39	1.79	1.76	2.84	2.88	3.76	3.77	5.16	5.18	5.73	5.78	1.97	2.01	1.97	2.01	3.94	4.02
15	5	55	79.8	41	1.79	1.78	2.85	2.88	3.75	3.76	5.14	5.18	5.74	5.75	1.96	1.98	1.99	1.99	3.95	3.97
16	10	55.5	87.9	42	2.09	2.31	3.12	3.15	3.85	3.87	5.24	5.25	5.95	5.93	1.51	1.56	2.1	2.06	3.61	3.62
17	17	55.5	87.9	44	2.32	2.35	3.10	3.13	3.88	3.89	5.20	5.21	5.85	5.87	1.56	1.54	1.97	1.98	3.53	3.52
18	18	53	80.4	45	1.80	1.79	2.87	2.87	3.75	3.76	5.17	5.16	5.76	5.77	1.95	1.97	2.01	2.01	3.96	3.98
19	15	54	80.4	45	1.78	1.77	2.87	2.79	3.77	3.77	5.18	5.18	5.75	5.74	1.99	2	1.98	1.97	3.97	3.97
20	6	54.5	84.2	47	1.78	1.80	2.88	2.9	3.82	3.81	5.18	5.17	5.76	5.75	2.04	2.01	1.94	1.94	3.98	3.95
21	10	54	84.2	47	1.79	1.79	2.89	2.88	3.72	3.74	5.19	5.20	5.76	5.78	1.93	1.95	2.04	2.04	3.97	3.99
22	10	56	85.5	48	2.10	2.09	2.95	2.93	3.85	3.85	5.23	5.24	5.84	5.85	2.05	2.03	1.99	2	4.04	4.03
23	13	56.5	82.2	48	2.00	2.04	2.91	2.93	3.86	3.85	5.23	5.13	5.92	5.91	1.86	1.81	2.06	2.06	3.92	3.87
24	10	53	84.3	48	1.78	1.77	2.79	2.76	3.75	3.76	5.16	5.16	5.71	5.69	1.97	1.99	1.96	1.93	3.93	3.92
25	13	53.5	87.9	48	1.76	1.77	2.72	2.76	3.75	3.76	5.14	5.14	5.71	5.68	1.99	1.99	1.96	1.92	3.95	3.91
26	3	54	80.4	49	1.76	1.75	2.82	2.8	3.75	3.73	5.15	5.14	5.72	5.69	1.99	1.98	1.97	1.96	3.96	3.94
27	1 5	54.5	85.5	54	2.45	2.42	3.00	3.04	3.86	3.87	5.20	5.23	5.92	5.91	1.41	1.45	2.06	2.04	3.47	3.49
28	16	56	80.2	53	2.46	2.47	2.93	2.93	3.85	3.86	5.19	5.18	5.91	5.89	1.39	1.39	2.06	2.03	3.45	3.42
29	15	52.5	80.2	51	1.75	1.76	2.84	2.85	3.74	3.76	5.15	5.18	5.7	5.72	1.99	2	1.96	1.96	3.95	3.96
30	8	54	79.7	37	1.76	1.75	2.85	2.83	3.76	3.75	5.14	5.14	5.73	5.74	2	2	1.97	1.99	3.97	3.99

(CONTROL GROUP) : BAEP- WAVE LATENCIES & INTERPEAK LATENCIES

Sl. No.	Duration of Noise exposure (yrs)	HC (cm)	SPL (dB)	Age (yrs)	I(ms)		II(ms)		III(ms)		IV(ms)		V(ms)		I – III (ms)		III – V (ms)		I – V (ms)	
					L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R
1	3	53	69.0	28	1.74	1.75	2.76	2.79	3.75	3.73	5.14	5.14	5.74	5.76	2.01	1.98	1.99	2.03	4	2.01
2	2	54	67.4	29	1.75	1.74	2.75	2.79	3.76	3.77	5.15	5.18	5.73	5.76	2.01	2.03	1.97	1.99	3.98	2.01
3	4	54	65.0	30	1.74	1.75	2.7	2.72	3.75	3.76	5.15	5.16	5.74	5.75	2.01	2.01	1.99	1.99	4	2.01
4	5	53	66.0	30	1.75	1.77	2.84	2.8	3.74	3.76	5.16	5.17	5.75	5.76	1.99	1.99	2.01	2	4	1.99
5	5	55	70.0	33	1.76	1.76	2.83	2.82	3.8	3.8	5.15	5.17	5.78	5.79	2.04	2.04	1.98	1.99	4.02	2.04
6	6	54.5	69.0	43	1.70	1.64	2.88	2.92	3.73	3.74	5.19	5.18	5.77	5.77	2.03	2.1	2.04	2.03	4.07	2.03
7	2	55	69.0	40	1.71	1.76	2.84	2.86	3.73	3.74	5.14	5.15	5.74	5.75	2.02	1.98	2.01	2.01	4.03	2.02
8	6	54	69.9	37	1.72	1.70	2.85	2.87	3.75	3.76	5.18	5.19	5.71	5.7	2.03	2.06	1.96	1.94	3.99	2.03
9	11	53	65.2	45	1.73	1.74	2.84	2.83	3.76	3.75	5.17	5.19	5.71	5.68	2.03	2.01	1.95	1.93	3.98	2.03
10	10	54	67.0	37	1.74	1.70	2.72	2.6	3.75	3.76	5.18	5.18	5.7	5.72	2.01	2.06	1.95	1.96	3.96	2.01
11	9	55	66.0	43	1.73	1.72	2.74	2.78	3.75	3.76	5.17	5.16	5.72	5.75	2.02	2.04	1.97	1.99	3.99	2.02
12	11	54.5	67.0	37	1.74	1.75	2.76	2.79	3.75	3.73	5.14	5.14	5.74	5.76	2.01	1.98	1.99	2.03	4	2.01
13	12	53	67.0	29	1.78	1.75	2.83	2.84	3.73	3.74	5.18	5.17	5.74	5.75	1.95	1.99	2.01	2.01	3.96	1.95
14	14	53.5	67.0	38	1.70	1.68	2.84	2.85	3.75	3.76	5.16	5.15	5.74	5.75	2.05	2.08	1.99	1.99	4.04	2.05
15	5	55	67.0	43	1.75	1.77	2.84	2.8	3.74	3.76	5.16	5.17	5.75	5.76	1.99	1.99	2.01	2	4	1.99
16	5	54.5	66.0	44	1.76	1.76	2.83	2.82	3.8	3.8	5.15	5.17	5.78	5.79	2.04	2.04	1.98	1.99	4.02	2.04
17	10	53	64.0	40	1.76	1.75	2.84	2.83	3.8	3.8	5.14	5.18	5.74	5.75	2.04	2.05	1.94	1.95	3.98	2.04
18	10	54	70.0	48	1.79	1.79	2.85	2.94	3.77	3.75	5.19	5.20	5.73	5.76	1.98	1.96	1.96	2.01	3.94	1.98
19	5	55	69.0	35	1.76	1.74	2.84	2.9	3.77	3.77	5.17	5.19	5.78	5.66	2.01	2.03	2.01	1.89	4.02	2.01
20	6	54	66.0	47	1.77	1.78	2.85	2.94	3.72	3.75	5.18	5.18	5.73	5.76	1.95	1.97	2.01	2.01	3.96	1.95
21	12	53.5	68.9	47	1.78	1.75	2.83	2.84	3.73	3.74	5.18	5.17	5.74	5.75	1.95	1.99	2.01	2.01	3.96	1.95
22	13	52	66.6	48	1.77	1.76	2.82	2.83	3.74	3.76	5.17	5.18	5.73	5.75	1.97	2	1.99	1.99	3.96	1.97
23	13	52.5	69.0	48	1.76	1.76	2.81	2.82	3.8	3.8	5.17	5.18	5.76	5.77	2.04	2.04	1.96	1.97	4	2.04
24	10	53.5	67.0	46	1.70	1.68	2.84	2.85	3.75	3.76	5.18	5.19	5.74	5.75	2.05	2.08	1.99	1.99	4.04	2.05
25	13	54	67.0	48	1.71	1.76	2.84	2.86	3.75	3.76	5.17	5.18	5.77	5.76	2.04	2	2.02	2	4.06	2.04
26	3	55	69.0	49	1.75	1.77	2.83	2.84	3.75	3.73	5.19	5.17	5.77	5.77	2	1.96	2.02	2.04	4.02	2
27	14	55.5	65.0	51	1.82	1.81	2.86	2.87	3.81	3.80	5.19	5.18	5.81	5.80	1.99	1.99	2	2	3.99	1.99
28	16	54	65.0	53	1.80	1.79	2.88	2.92	3.75	3.76	5.21	5.19	5.74	5.74	1.95	1.97	1.99	1.98	3.94	1.95
29	13	53	65.0	51	1.79	1.78	2.84	2.8	3.74	3.76	5.16	5.17	5.75	5.76	1.95	1.98	2.01	2	3.96	1.95
30	15	53.5	65.0	52	1.77	1.76	2.83	2.82	3.8	3.8	5.18	5.20	5.78	5.79	2.03	2.04	1.98	1.99	4.01	2.03

KEY TO MASTER CHART

HC	-	Head Circumference
SPL	-	Sound Pressure level
L	-	Left Ear
R	-	Right Ear
AC	-	Air Conduction
BC	-	Bone Conduction
kHz	-	Kilohertz
dB	-	Decibels
ms	-	Milliseconds
cm	-	Centimeter