NOISE - INDUCED VESTIBULAR DYSFUNCTION

NITHYA .B

Register No. M2K15

A Master's Dissertation submitted as part fulfillment for the
II year M.Sc. (Speech and Hearing) to the University of Mysore

All INDIA INSTITUTE OF SPEECH AND HEARING, MYSORE ~ 570 006
MAY 2002

CERTIFICATE

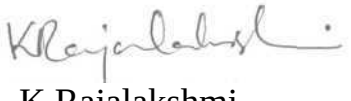
This is to certify that the dissertation entitled "NOISE - INDUCED VESTIBULAR DYSFUNCTION" is a bonafide work done in part fulfillment for the degree of Master of Science (Speech and Hearing) of the student with Register No. M 2KI5

Mysore
May 2002

Director
All India Institute of
Speech & Hearing
Mysore - 570 006

CERTIFICATE

This is to certify that the dissertation entitled "NOISE - INDUCED VESTIBULAR DYSFUNCTION" has been prepared under my supervision and guidance and has not been submitted earlier in any other University for any other Diploma or Degree.



Mysore
May 2002

Dr. K.Rajalakshmi
Lecturer in Audiology
Department of Audiology
A1ISH, Mysore - 570 006

Declaration

I hereby declare that this dissertation entitled "NOISE - INDUCED VESTIBULAR DYSFUNCTION" is the result of my own study under the guidance of Dr. K. Rajalakshmi, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier in any other University for any other Diploma or Degree.

Mysore
May 2002

Register No.M 2K15

TO

SRI
GURU V A YU RAPP AN
&
SRI GAYATHRI
ACCHAN & AMMA

To

My Beloved

Rajalaksmi Madam

Faith is to believe what we do not see;

the reward of this faith is to

see what we believe

Saint Augustine

ACKNOWLEDGEMENT

I extend my immense a gratitude to Dr.K.Rajalakshmi, Lecturer in Audiology, All India Institute of Speech and Hearing Mysore, for her valuable guidance and constant encouragement throughout the completion of this project-Ma'am you are truly the a guiding light and source of wisdom to the uncertain steps of a student, making it firmer by teaching everlasting confidence in Almighty.

I thank Dr. M. Jayaram, Director, All India Institute of Speech and Hearing, Mysore, for allowing me to undertake (his project.

Acchan and Amma the silent love you shower upon me enfolds my soul in bliss and inspires its everlasting sojourn.

Lekhachechi, you are casketted in my heart closer than ever before, distances could now never separate you from me. You are my guiding angel at all walks of my life.

I extend my heart felt thanks to Dr. Rajeshwari lecturer in ENT All India Institute of Speech and Hearing, who patiently guided me throughout this endveour and help me complete it.

Dr. Venkateshan, Dr. Acharya, Dr. Prema, Dr. Basanthi Devi - words would not be enough to thank you all for the confidence you had built in me

about myself about life. Any time I approach,you fill my depleted self morale with zest and vigour to aspire further with realistic goals.

My classmates and my friends at AIISH - a mere mention of your names followed by thanks would not do justice to all the help and affection you have showered on my stay at AIISH. You taught me more about life and how to face it. Your prayers and faith will truly lead me ahead on life's path.

Special thanks to the wonderful library at AIISH and its efficient and helpful librarians.

I thank all Arvind Enterprises team for their fast and efficient typing.

This indiscernible venture was accomplished with many a helping hand. I thank everyone who had been instrumental in completing this study.

Above all I thank Almighty God for the faith and strength he is constantly providing me with every now and then.

TABLE OF CONTENTS

LIST OF TABLES AND FIGURES

INTRODUCTION

Noise is a curse to the modern industrial area. It not only disturbs the persons exposed to it but also pollutes the environment around. Noise is thus hazardous to public health. As urbanisation takes place industries are mushrooming around our country. But most of the industrial development in urban area is not taking adequate noise control measurements in the industries existing, even though there are stringent existing rules and regulations. The result is that our population is at risk of developing noise- induced dysfunction or diseases.

Noise is any undesired sound or any unwanted disturbances within a useful frequency band. Noise is an erratic intermittent or statistically random oscillations. Since these definitions of noise are not mutually exclusive it is usually necessary to depend upon context for distinction.

Psychologically noise is any sound irrespective of its wave form which is unpleasant or unwanted. Noise, like any sound is defined in terms of its amplitude, duration, frequency and spectrum. The intensity of noise is measured in SPL and expressed in dB and its frequency in Hertz.

Noise may be continuos, intermittent, impulsive or explosive. It may be steady- state or fluctuant.

Noise pollution is a public health hazard. The lack of appropriate measures to check and control noise pollution by the authorities leaves an average citizen at risk of developing noise- induced health disorders. These

include physiological disorders ranging from varying degrees of stress to other major physiological manifestations. Noise can also cause various physiological disorders mainly hearing loss, vertigo nausea, headache and cardiovascular diseases.

Increased intensity of noise is one of the fundamental causes of inner-ear damage. Noise destroys the delicate hair cells of the cochlea, affecting the normal auditory function. Such a hearing loss which is due to exposure to hazardous noise is called noise- induced hearing loss or NIHL. Research studies on noise and NIHL revealed that as continuous exposure to noise above 85dBA for 8hrs or more would result in hearing loss if appropriate ear protective devices are not used. When intensity of noise increase the exposure time shortens for the damage effect of cochlea. Exposure to very high intensity sounds of 139 dBA for 0.6 sec would cause profound damage to inner ear.

Thus it is known that loud tones can damage cochlea but vestibular involvement has been given less attention. The vestibular system is phylogenetically a much older system in comparison with the more refined and sophisticated mechanisms of the auditory system whose function is converting physical sound impulses into neuro- physiological sensation. The vestibular system is closely associated with the cochlear system. The endolymph is an example of this association since it is present in both the cochlear duct and in the semi- circular canals.

Connected to the cochlea of the inner ear are the so called sacculus, utricle and semicircular canals. These structures, called the vestibular organs,

share certain fluids with the cochlea and their its innervation are closely connected. These vestibular organs are involved in maintaining body balance and orientation in space. When stimulated in certain ways, a person may loose his sense of balance, become dizzy, his eyes may show nystagmus movements (a fast movement back an forth of the eyeballs) and under extreme conditions he may become nauseated.

Because of their close proximity and fluid connections, it is not surprising to find that intense sounds affect the cochlea and vestibular system. Dickson and Chadwick (1951) reported that, in jet air craft noise over 140 dB or so, a person may feel a sense of disturbances in his equilibrium. Roggevsen and Van Dischoeck (1950) note that in persons who experience vestibular reaction to relatively weak sounds usually presents lesions in the bony walls of the vestibular system . In 1966, Chadwick reported that of 1800 patients with NIHL, 8 had fluctuating hearing loss, tinnitus and attacks of vestibular dysfunction.

Vestibular stimulation by noise has been recognised since it was first described by Tullio (1929) in pigeons. In humans, Man et. al., (1980) and Kilburn et. al., (1992) found an association between noise exposure and vestibular damage.

Thus, although noise has long been recognised as a cause of cochlear damage resulting in hearing loss and tinnitus its role in vestibular dysfunction still remains unclear, to many of the audiologists and hearing health care professionals.

Several studies has been done aboard, both experimental and scientific regarding this issues. Indian studies, apart from some studies done by Mr. Praneshwar Rao Retired Audiologist and Vestibular specialist at Aviation Medicine center Department; are negligibly few.

Hence the present study is designed to investigate the vestibular dysfunction due to noise in industrial urban population, in India.

Thus this study aims to scientifically find an association between noise exposure and vestibular dysfunction. If such conditions are experimentally proven, to create awareness in individual of using noise protective devices, avoidance to dangerous levels of noise, treatment and management of noise induced hearing loss and vestibular dysfunction.

REVIEW OF LITERATURE

An association between noise exposure and vestibular function has long been suspected. Noise has long been recognised as a cause of cochlear damage resulting in tinntus and hearing loss. However, its role in vestibular dysfunction remains unclear, and a cause-effect relationship has not been accepted (Nageris, Attias and Feinmesser, 2000).

The first study on the effect of occupational noise hazard was published in 1886. Barr, pointed out that out of 15 of the 1000 individuals studied had "some sensation of giddiness" but little importance was attached to the finding that time. By 1915, vestibular stimulation with noise was recognised by Rodger, who found that 10% of the patients with noise-induced hearing loss (NIHL) complained of giddiness.

Vestibular stimulation by noise has been recognised since it was first described by Tullio (1929) in pigeons. In humans, Man et al (1980) & Kilburn et al (1992) found an association between noise exposure and vestibular damage.

Instances like jet-aircraft noise over 140dB also cause disturbances in equilibrium and lesions in vestibular system according to Dickson and Chadwick (1951). Bekesy in (1960) has provided evidence that the initiation (and cessation) of an intense tone sets up a streaming of fluids not only in the cochlea but also in the canals (semicircular canals).

Roggevsen and Van Dishoeck (1956) note that in persons who experienced vestibular reactions to relatively weak intensity sounds usually present lesions in the bony walls of vestibular system.

In 1966, Chadwick reported that of 1800 patients with NIHL, eight had the fluctuant hearing loss, tinnitus and attacks of vestibular dysfunction characteristic of Meniere's disease. Thereafter both Pulec (1972) and Paparella and Mancini (1983) noted that some patients with Meniere's disease had a history of exposure to hazardous noise and they speculated that the noise exposure may in fact have caused the disease.

Kimura (1982) showed that acoustic trauma produced endolymphatic hydrops in 38% of the exposed animals; these results re-emphasised the fact that histological damage could occur secondary to noise exposure, as was first described by Wittmaack in 1907. This prompted Paperalla (1991) in his text book to list trauma, specifically acoustic trauma, as a cause of Meniere's disease with episodal vertigo attacks.

Both Ylikokski (1988) and Okuno et al (1996) studied military personnel, among whom noise exposure is common. Ylikokski (1988) reported on 10 army offices with NIHL, balance disturbances and a history of long exposure to hazardous noise, Okuno et al (1996) found that of 475 soldiers tested, 1.4% had Meniere's disease and 32.5% had experienced dizzy spells.

Manabe et al (1995) divided 36 patients with NIHL into 2 groups according to the presence or absence of vestibular complaints. Results of electro-cochleograms, performed in all participants and

electronystagrmograms. performed in groups with vestibular complaints showed that episodic vertigo in NIHL may result from a pathophysiological mechanism similar to that of Meniere's disease with highlighting feature of vertiginous attacks.

TuIlioPhenomenon

Intense sound stimuli have been found to induce reflex eye movements in guinea pigs (130-160dBSPL) and monkeys (120-172dBSPL) (Parker et al 1976; 1978) and visual field displacements in humans (125 dBSPL) (Parker et al; 1978). This phenomenon of acoustical activation of vestibular responses has been called the 'Tullio Phenomenon'.

Patients with noise-induced hearing loss (NIHL) show some signs of vestibular pathology in one or more tests (spontaneous, postitional, or cervical nystagmus) (Oostervald et al., 1982), significantly more body sway (Ylikoski et al., 1988) and reduced gain of vestibular-ocular reflex (Shupak et al., 1994). These clinical findings have been taken as evidence that intense sound stimuli can induce vestibular disturbance and as confirming Tullio Phenomenon.

Thus several studies have shown in order for sound to affect the vestibular end organs in the inner ear, very high intensities are required. Furthermore, in patients with noise-induced hearing loss, vestibular signs, if present are subclinical. It seems therefore that even though intense noise clearly affects the cochlea and may have a "masking" effect on the vestibular end organs, the intensities used in this study (113dBSPL) are not able to produce a long term noise induced vestibular disorder in the initially normal ear. These

differences between the response of the cochlear and vestibular end organs to noise may be due to dissimilarities in their acoustic impedences and /or their electrical resting potential (Sohmer et al 1999).

If noise can effect the vestibular system, it is possible that this could affect the ability of a person to maintain balance when standing on a narrow rail. Harris and Von Gierke (1971) exposed subjects wearing earmuffs to white-noise at levels of 120, 130 and 140 dB while standing balanced on a rail above 1 ^{l}A inches wide with eyes open and on a rail about 2 % inches wide with eyes closed. They found that with both ears exposed to same level of noise, only at 140dB there was any impairment to balance performance. At the lower levels there was a reduction in balance performance when the noise was louder in one ear than in the other.

Harris (1972) found that an intermittent lOOOHz tone at a level of 105dB presented monaurally to an unprotected ear impaired the ability to balance on the rails described above compared with performance with the tone at 65dB. However, Vanderhei and Loeb (1976), in a later repetition of the experiment, found no effect of 105dB, 1000Hz, monaurally presented tone on the same balance test. Refer figure 1. These investigations concluded that noise and sound under general field conditions are unlikely to affect equilibrium.

The patient presenting with dizziness tended to be mostly female (average 60 %) to be middle- aged (late 40 to early 60years).

Drachman and Hart 1972; Nezelski, Barber & Mcllmoyl, 1986; Herr, Zun & Mathews 1989; Kroenke et. al., ;(1992)

"In the analyses where the subjects were matched with pairs by age, exposure, blood pressure & serum cholesterol level, elderly subjects was more susceptible to NIHL than were younger subjects Factors independently but casually related to age were important in the development of NIHL among workers exposed to noise levels below 98dB 9A). The inner ear in older subjects seem to me more vulnerable to noise than those in younger ones. (Toppila .E, Pyyko Ilmari, Starck J, (2001))

Noise is undoubtedly one of the world's great health problems and the single most common form of environmental pollution. Today in our industrialized civilization, no one is free from the effects of high level noise. Audioiogy as a profession can trace its origin to the effects of noise of war and its interference with communication. Thus, it is appropriate that it is the audiologist who plays a major role in the diagnosis of the problem, its prevention and its treatment. Occupational noise has been with us since the Industrial Revolution in the 19th century. Historically, this has been the most quantitatively important cause of noise-induced hearing loss and its non-auditory effects over the past one hundred years.

Noise-induced hearing loss and noise induced physiological and psychological disorders continues to be a significant public health problem. In 1987, the National Institute of Occupational Safety and Health rated NIHL as

one of top 10 work-related problems, involving at least 11 million worker (NIOSH, 1987).

Like all biological insults, the effects on any individual organism from exposure to excessive noise levels are extremely variable and relatively unpredictable, especially at moderate exposure levels. Although many theories and suggestions have been made for this variability, with variations in intrinsic protective responses coming in for recent scrutiny, a satisfactory explanation is still lacking (Henderson. Subramaniam, Boettcher, 1993). However exposure to excessive noise levels, the occurrence of noise damage becomes inevitable. It is generally agreed that sound levels below about 80dB are unlikely to cause any damage to the human ear no matter how long one is exposed to them. Sounds of 130dB or greater will damage the auditory mechanism after very short periods of exposure in almost all repeatedly exposed individuals. Between these two extremes, the safe period of exposure decreases as the sound level increases, although the degree of noise damage displayed by one individual is variable and relatively unpredictable as a result of natural biological variability (Alberti, 1987, NIH, 1990, Saunders, Deal and Shneider, 1985). Refer table 1 Hearing losses from a wide range of noise sources have similar audiometric patterns. The low- frequency sensitivity as either normal or near normal, whereas sensitivity in the 3000 to 6000 Hz region is reduced. Yet, the acuity of an individual to an 8000Hz sound is much better and like the lower frequencies can be normal or near normal. This audiometic notch is

characteristic of many forms of damage from noise exposure(Ref.Fig.2)

Audiometric Notch

What are the causes of the non monotonic nature of noise-induced hearing losses that creates an audiometric notch? Several explanations have been proposed for this notch. These include (a) a poor blood supply to the part of the cochlea that corresponds to the 3000 to 6000 Hz region (Crow, Guild & Polvogot, 1934); (b) a greater susceptibility for damage of the supporting structures of the hair cells in this region (Bohne, 1976); (c) the orientations of the stapes foot-plate into the inner ear is such that it primary force vector aims towards those hair cells in this region, with the effect of eventual action Hilding, 1953; Schuknecht & Tonndorf, 1960); and (d) Permanent noise exposure has its greatest effect approximately one-half octave above the peak frequency of the noise spectrum.

Since all spectra are enhanced at 3000Hz by the outer ear canal resonance, the greatest loss will be in the 4000 to 6000Hz region

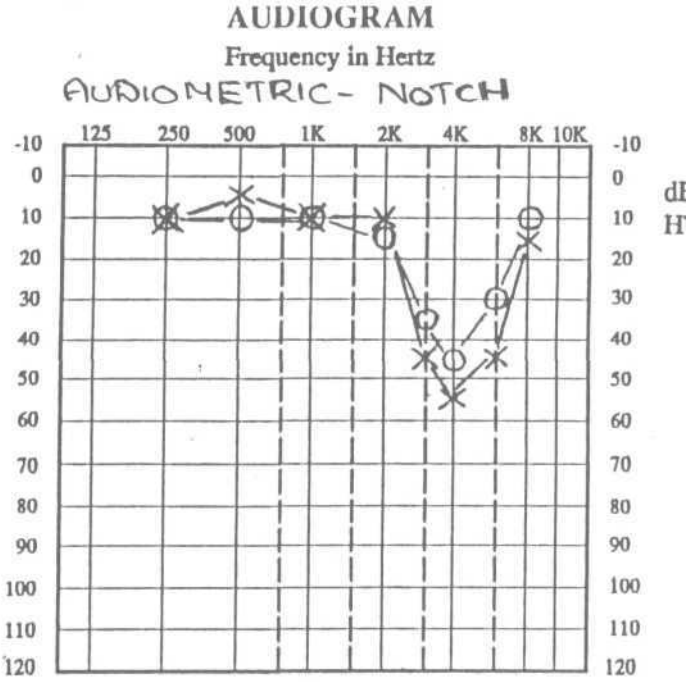


Fig 2. Audiogram Showing A "Notched" Pattern that is Characteristic of NIHL

Table 1 Allowable sound exposure times.

Sound Level Intensity In dBA	Maximum Hours of Exposure Per 8-Hours Workday
80dBA	24 hrs.
82dBA	16hrs.
83dBA	12 hrs.40 mins.
84dBA	10 hrs.04 mins.
85dBA	8 hrs.
86dBA	6 hrs.21 mins.
87dBA	5 hrs.03 mins.
88dBA	4 hrs.
89dBA	3 hrs. 10 mins
90dBA	2 hrs. 31 mins
91dBA	2 hrs.
92dBA	1 hrs. 34 mins
93dBA	1 hrs. 16 mins
94dBA	21 hrs.
95dBA	48 mins
96dBA	38 mins
97dBA	30 mins
98dBA	24 mins
99dBA	19 mins
l00dBA	15 mins
l0ldBA	12 mins
102dBA	9 mins 6 secs.
103dBA	7mins 30 secs.
106dBA	3 mins 45 secs.
108dBA	1 min 52 secs.
112dBA	56secs.
115dBA	28.07 secs.
118dBA	14.03 secs.
121dlBA	7.01 secs.
124dBA	3.31 secs.
121dBA	1.45 secs.
127dBA	1.45 secs.
130dBA	.25 secs.
133dBA	.26 secs.
136dBA	.13 secs.
139dBA	.06 secs.

(From the American Conference of Governmental Industrial Hygienists, 1994-1995)

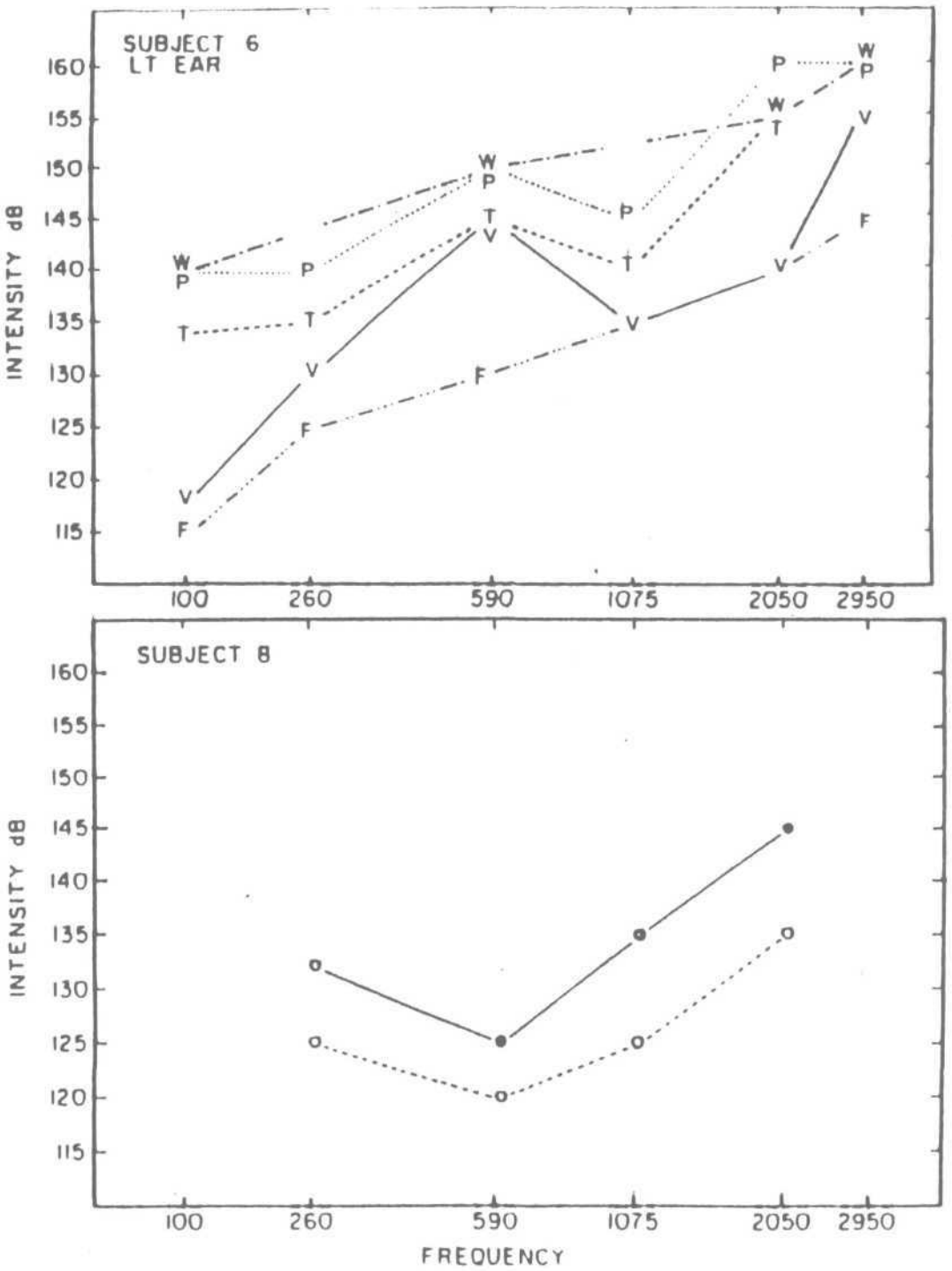


Fig. 1 Threshold curves for small eye movements (Brokenline) & marked nystagmus (solid line) & threshold curves for vibration (V) Tickle (T), Pain (P), Warmth (W) & Feelings (F) of Subjects.

(Tonndorf, 1976; Caiazzo & Tonndorf, 1977). Because of these phenomenon hearing losses due to noise (Including music) exposure are relatively easy to spot.

However, many clinical cases of music or noise exposures do not possess an audiometric notch. Indeed, Barrs,Althoff, Krueger, and Olsson (1994) found that only 37% of workers suffering from noise exposure possessed an audiometric notch. It is quite- possible that in advanced cases of exposure or advanced age where there as significant age related hearing loss ("Presbycusis"), the hearing sensitivity at 8000Hz may have also deteriorated, leaving a flat audiometric configuration. In addition, depending on the noise spectrum, the frequency region of greatest damage may be above the audiometric test frequencies. For example, using data derived from violin players , the frequency of greatest damage can be at 8000Hz, and unless a higher frequency pure tone were to be assessed (e.g. 10,000Hz), a notch would not be apparent.

Alberti (1982) argued that industrial noise exposure tends to be symmetrical . This rationale is based on the highly reverberant environment many workers find themselves in. A damaging sound from a worker's left side may be just as intense as the workers right side, given a sufficiently reverberant environment. However, in less reverberant environments (or ones with sufficient high frequency energy.), the exposure to one ear may be significantly different than that to the contralateral ear('head shadow') thus resulting in an asymmetrical audiometric pattern.

The difference in proximity of the ear to noise source accounted for the differing exposures between the two ears.

The Vestibular System

In humans, there is a complex system for maintaining gaze and balance. The system is dependant upon visual, vestibular, proprioceptive, and superficial sensory- inputs, which are integrated in the central nervous system. At every level from the vestibular receptors to the central cortex, inputs are modulated by afferent & efferent pathways. The particular three- dimensional orientation of the vestibular system offers exquisite directional sensitivity.

Although the vestibular system is important for balance, many patients cope well with no vestibular function. Similarly vision is important for balance, but eye closure does not cause balance problems in the otherwise unimpaired & the blind have good balance. By contrast, a loss of proprioception can cause a severe loss of balance.

The structures subserving compensation for vestibular dysfunction are unknown, but it has been shown that brainstem, cerebellar & cortical in addition to the requirement for all sensory inputs, including vision, somatosensory afferents & remaining labyrinthine input, which are involved in the perception of space, body posture and body locomotion (Lacour& Xerri, 1984)

Functions of the vestibular system

The vestibular system allows detection of body motion in all three planes, consequent upon linear and angular accelerations stimuli applied to the

head. In addition, the vestibular system detects the gravitational vector, necessary for head & body orientations.

Vestibular information is integrated with other inputs to contribute to:

* The maintenance of the fovea on the object of visual fixations.

The vestibular system stabilizes the fovea for high- frequency stimuli, such as occurring during walking and running (>2Hz) (Gross man et. al., 1988) whereas the visual system stabilizes fovea for low-frequency stimuli. Without these stabilizing system, there would be a drop in visual acuity with head movements.

* Maintenance of balance.

* Activity of the autonomous nervous system.

* Level of arousal & mood.

The integrity of vestibular system is assessed , in the most simplest way , using Caloric test.

The Caloric Test

In order to provide more diagnostic information concerning the vestibular organs, it as necessary to stimulate the semi circular canals and record the resultant eye movements. The only procedure available to most clinics to achieve this is the caloric test.

Caloric test is the only routine diagnostic test in which right & left semicircular canal function can be examined separately. This is the major advantage. However, it also has the advantage of being relatively cheap, and therefore widely available.

The patient is positioned on a couch, with the head at as angle of 30° to the horizontal, to bring the horizontal semicircular canal to vertical alignment. A thermal stimulus is applied to the endolympth, by irrigating the outer ear canal with cool & warm water. This is though to induce convection currents and hence a flow of endolympth. This will result in cupular deflection and a neural activity within the vestibular nerve (Fitzgerald & Hallpike, 1942)

As only one ear is irrigated at a time, this will create an asymmetry in vestibular activity & hence stimulate the vestibular ocular reflex (VOR). Nystagmus will result, beating towards the 'warmer' ear. The maximum intensity of this nystagmus occurs approximately 10-12 sec after usually persists for 90-120sec with fixation. The eye movement response is observed directly with and without fixation using Frenzel glasses or an infra-red viewer in the dark. Alternatively, the test can be performed with ENG recording.

The Fitzgerald- Hallpike test technique involves irrigating both ears in turn with water 7° below & 7° above body temperature (30° c & 44°c respectively). The optimal order of testing is a controversial issue. However left cool- right cool- left warm- right warm is the order of choice; because the cool. Irrigation tends to have greater acceptability for most patients. The four irrigations will provide information concerning right & left beating nystagmus, from both ears. The strength of the response can be ascertained in each case, either by measuring the duration of nystagmus (i.e when direct observation is employed) or maximum slow phase velocity(with ENG recording).

Evoked Otocoustic Emissions

The functioning of the normal cochlea is strongly dependent on an active mechanism that is physiologically vulnerable. The mechanism depends upon the integrity of OHCS, (outer hair-cells), and particularly their stereocilia. The OHCS are easily damaged by noise exposure, ototoxic chemicals, infection and metabolic disturbances. When they are damaged, the active mechanism is reduced in effectiveness or destroyed completely. This has several important consequences. 1. Sensitivity is reduced, so that the tips of tuning curves are elevated by up to 40-50dB.

2. The sharpness of tuning on the Basilar Membrane (BM) is greatly reduced. The tip of the tuning curve may be elevated or may disappear altogether, leaving only the broad tuning of the passive BM. 3. Non-linear effects such as compressive input-output functions on the BM, two-tone suppression and combination tone generation are reduced or disappear altogether. 4. Evoked and spontaneous otoacoustic emissions are reduced or disappear at least in the frequency range corresponding to the damaged place. The IHCS (inner hair cells) are the transducers of the cochlea converting the mechanical vibrations on the BM into neural activity. They are less susceptible to damage than the OHCS. When they are damaged, sensitivity is reduced.

Evoked Otocoustic emissions are reduced in magnitude by cocblear hearing loss. Human ears with hearing losses exceeding 40 to 60dB usually show no detectable emissions.(Corge, Neely, Ohlrich et.al., 1997). The emissions appear to be particularly associated with OHC (Outer Hair Cell)

functions. The emissions are abolished in ears that have been exposed to intense sounds or to drugs that adversely affect the operation of the cochlea. In the former case, the emissions may return after a period of recovery. This suggests that the emissions are linked to the active mechanism. The measurement of cochlear emissions provides a sensitive way of monitoring the physiological state of cochlea & is now being commonly applied in clinical situations

METHODOLOGY

The present study was designed to explore the vestibular dysfunction in patients with noise- induced hearing loss (NIHL).

A. The cases for this study were selected from the group who reported at AIISH for hearing evaluation.

The selected persons were interviewed & detailed case history was taken. Those who satisfied the following criteria were selected. For the study.

a) No history of otological infections.

b) No associated physiological problems with medical history of hypo/ hypertension, diabetes, systemic diseases, etc.,

c) The age range was between 20-30 yrs (to rule out presbycusis).

The subjects were divided into 2 groups 1) control group 2) Experimental group.

Both the groups were medically investigated for ruling out the conditions mentioned in a & b.

1) **Control group:-** For the control group 10 normal hearing subjects with history of no noise- exposure were taken. The evaluations at AIISH confirmed normal hearing thresholds.

2) **Experimental group:** For the experimental group the following criteria were used

   a) History of exposure to noise.

b) A 4 KHz dip in audiometric pattern as an indication of NIHL.

Those cases for this study were selected from the group who have reported at A.I.I.S.H for their routine hearing evaluations from AT& S industries Nanjangud. (The audiology department at A.I.I.S.H has a hearing conservation program for the industries of Mysore.)

These cases were interviewed & a detailed case history regarding their kind of work, duration of level of noise exposure , discomfort, usage of Ear-protective devices (EPD's) etc., were recorded.

B. Instrumentation

The following instruments were used for the study.

* A calibrated G SI-61 two -channel diagnostic audiometer (Grason- Stadler Inc.) with TDH- 50p earphones housed in MX-41/AR ear cushions, and a B- 71 bone vibrator was used to evaluate the hearing status.

*A Calibrated GSI-33 middle ear analyzer (Grason- Stadler Inc.) was used for examine the middle- ear status.

* A Calibrated DP Echoport plus-ILO292 TEOAE Screener was used for recording TEOAE'S.

C. Test Batteries

1. Hearing test

a) Pure-tone audiometry

b) Speech audiometry

c) Impedance audiometry

d) OAE test

2) Vestibular function test

Bi-thermal calorie test

D. Test procedure

The routine pure-tone audiometry was carried out using modified Hughson & Westlake procedure. Puretone average (PTA) obtained. Speech audiometry was carried out assessing SRT, PBMAX. Then the UCL was assessed using speech as stimuli.

Impedance audiometry was carried out with tympanometry, acoustic reflex testing, (ART) & reflex decay testing (RDT).

Cochlear mechanics were evaluated by recording TEOAES in ILO-292 oto acoustic emission system. TEOAES were recorded with click stimulation. Clicks were generated using short electrical pulses (<80 μs) resulting in a wide band signal. TEOAES were collected using the nonlinear mode. In the averaged spectra (0.012-6.25 KHz) TEOAES were considered present when the signal to noise ratio of at least one frequency component was l0dB or better. The Otoacoustic Emissions were recorded Transient Otoacoustic Emissions (TOAE) test & its presence/ absence was noted.

The vestibular function was tested using Fitzgerald Hallpike Bi- thermal caloric test with cold & warm water irrigation of the ear canal, patient lying in supine with head at 30° angle & the temperature of cold water being 33°c & warm water being 40° c. nystagmus was elicited & type of nystagmus, its latency & duration noted.

RESULTS & DISCUSSIONS

The data collected from 10 normal & 10 experimental group were analyzed in terms of their PTA (Pure Tone Average), SRT (Speech Reception Threshold), PBMAX (Phonetically Balanced Maximum Words), UCL (Uncomfortable Level), Tympanogram, . Acoustic reflex threshold (ART), Reflex decay (+ve /-ve), TEOAE (Transient Evoked Oto- Acoustic Emissions) (present/ absent), caloric responses in terms of nystagmus, type, latency of onset & duration.

The data hence obtained were statistically analyzed Mean & standard deviation were calculated for control group & experiment group. Statistically significant difference between mean & variance of normal & experimental groups was inferred using 't' test.

Sampling method - Purposive fixed sampling.

Independent variable - Noise exposure

Statistical measures obtained from experimental and control group for PTA, SRT, PBMAX & UCL have been shown in table A & table B.

PTA (Pure Tone Average)

The mean PTA for Right ear (PTAR) experimental group was found to 8.48 dB, where as for control group it was 9.15dB. The independent sample

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
PTAR	Exptl	10	8.4800	1.4620	.4623
	Ctrl	10	9.1500	7.5923	2.4009
PTAL	Exptl	10	8.6400	2.5907	.8192
	Ctrl	10	8.1300	2.5325	.8008
SRTR	Exptl	10	10.0000	3.3333	1.0541
	Ctrl	10	9.5000	3.6893	1.1667
SRTL	Exptl	10	10.5000	3.6893	1.1667
	Ctrl	10	10.5000	3.6893	1.1667
PBMAXR	Exptl	10	99.5000	1.5811	.5000
	Ctrl	10	99.5000	1.5811	.5000
PBMAXL	Exptl	10	100.0000	.0000	.0000
	Ctrl	10	100.0000	.0000	.0000
UCLR	Exptl	10	101.0000	2.1082	.6667
	Ctrl	10	101.0000	2.1082	.6667
UCLL	Exptl	10	105.0000	.0000	.0000
	Ctrl	10	103.0000	2.5820	.8165

Table A Mean, standard deviation and standard error for experimental and control group for the values of PTA, SRT, PBMAX & UCL for Right and Left Ears.

	t-test for Equality of Means				
	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
PTAR	-.274	18	.787	-.6700	2.4450
PTAL	.445	18	.662	.5100	1.1456
SRTR	.318	18	.754	.5000	1.5723
SRTL	.000	18	1.000	.0000	1.6499
PBMAXR & L	.000	18	1.000	.0000	.7071
UCLR	.000	18	1.000	.0000	.9428
UCLL	2.449	18	.025	2.0000	.8165

Table B:- T test for equality of means for the experimental and control group for the values of PTA, SRT, PBMAX & UCL for Right and Left Ears.

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
LATRC	exptl	10	40.0000	3.3333	1.0541
	Ctrl	10	36.5000	5.7975	1.8333
LATRW	exptl	10	42.0000	2.5820	.8165
	Ctrl	10	37.5000	5.4006	1.7078
LATLC	exptl	10	42.0000	2.5820	.8165
	Ctrl	10	36.0000	5.6765	1.7951
LATLW	exptl	10	40.0000	3.3333	1.0541
	Ctrl	10	37.0000	5.8689	1.8559
DURRC	exptl	10	222.0000	22.5093	7.1181
	Ctrl	10	215.5000	26.5047	8.3815
DURRW	exptl	10	222.0000	22.5093	7.1181
	Ctrl	10	220.5000	24.9944	7.9039
DURLC	exptl	10	227.0000	17.0294	5.3852
	Ctrl	10	214.0000	29.5146	9.3333
DURLW	exptl	10	222.0000	22.5093	7.1181
	Ctrl	10	215.0000	28.3823	8.9753

Table C Mean, standard deviation and standard error for experimental and control group for the values of Latency and Duration of Nystagmus for Right and Left Ears.

	t-test for Equality of Means				
	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference
LATRC	1.655	1	.115	3.5000	2.1148
LATRW	2.377	1	.029	4.5000	1.8930
LATLC	3.043	1	.007	6.0000	1.9720
LATLW	1.406	1	.177	3.0000	2.1344
DURRC	.591	1	.562	6.5000	10.9962
DURRW	.141	1	.889	1.5000	10.6367
DURLC	1.206	1	.243	13.0000	10.7755
DURLW	.611	1	.549	7.0000	11.4552

Table D:- T test for equality of means for the experimental and control group for the values of Duration & Latency of Nystagmus for Right and Left Ears.

test revealed a non significant difference between these two groups (mean difference = 0.67 t value = .274; p < 0.787) from mean values it is clear that both groups have statistically equal values.

The mean PTA for Left ear (PTAL) for experimental & control group are found to be 8.64dB & 8.13dB. The independent sample T test revealed as non significant difference between these two groups. (Mean difference = 0.5100 t values = .445 p < 0.662) from these mean values it is clear that both groups have statically equal values.

SRT (Speech Reception Threshold)

The mean SRT for Right ear (SRTR) for experimental group were found to be 10.00 where as for control group it was 8.13. The Independent Sample T test revealed a nonsignificant differences between these two group (Mean difference = 0.5000 and t value = 0.318; p<0.754. From the mean value it is clear that both groups are equal value.

The means SRT for Left ear (SRTL) for experimental group was found to be 10.5 and control group also 10.5. The Independent Sample T test revealed a nonsignificant difference between these two groups Mean difference - 0.0000 t = 0.0000 p< 1.000). From the above value it is clear that both groups have statically equal value.

PBMax of Right ear (PBMAX R) for both experimental and control group were 99.5% and hence statically equal values.

Similarly PBMax of Left ear(PBMAX L) were of equal values i.e., 100% and hence statistically non significant.

UCL (Uncomfortable Level)

UCL of Right ear (UCLR) for experimental group were 105dB where as for control group it was of 103dB. The Independent T test revealed a non significant difference between these two groups. (Mean difference 0.000 t=.000 p<1.00.

Impedance Audiometry

1) All tympanograms of both experimental and control group were of 'A' type.
2) Acoustic reflexes were present in all cases (except in one case of experimental groupi where 4K reflexes were absent both ipsitaterally & constralaterally but rest of the frequency relaxes were present in this cases).

Bi-thermal Caloric-test

Statistical measures obtained from experimental and control group for Duration and Latency of Nystagmus is shown in Table C & D.

Latency of Nystagmus

The mean latency for Right ear cold (LATRC) & for experimental & control group was 3.5 & 4.5 . the Independent Sample T test revealed a non-significant difference between there two groups.

(Mean diff = 3.5 t= 1.655 p< 0.115)

The Mean latency for Right ear warm & of the experimental & control group were of significant difference.

(Mean = 4.5 t = 2.377 p< 0.29)

But as the normative value ranges from 0-60sec if can be considered non- significant. Similarly latency of Left ear cold (LATLC) of experimental & control group were of significant differences statistically (M.D = 6.00 t= 3.043 p< 0.007) But since these normative values also range from 0-60 secs the values can be considered non-significant.

Latency of Left Ear warm (LATLW) were nonsignificant statistically (MD=3:t=1.406p<.177)

Duration of Nystagmus

Duration of Right Ear cold (DURRC) was statistically nonsignificant (M.D=6.5 t=0.591 p<0.562). duration of Right Ear warm (DURRW) was statistically nonsignificant (M.D=1.5 t=0.141 p<0.889). Duration of Left Ear cold (DURLC) was statistically nonsignificant (M.D.= 13 t=1.206 p<0.243). Duration of Left Ear warm was statistically nonsignificant (M.D.=7 t=0.611 p<0.549).

The type of Nystagmus - Horizontal for both control and experimental group were observed.

TEOAE (Transient Evoked Oto - Acoustic Emissions)

TPOAE'S were present in all controlled group where as in experimental group six of them had significantly reduced (TEOAE'S) and in rest of the four

(TEOAE'S) were present In cases were (TEOAE'S) were absent outer hair cell damaged has been suspected.

All subjects were using EPD'S for 4-5 hours.

Intensity level of exposure - 95 - 102dB.

Noise- exposure & its related vestibular dysfunctions has long been suspected. But its causes & effect relationship would not be confirmed due to a number of variabilities present in each study.

This study can be discussed in three steps.

1. Intensity level of exposure
2. Age- related factors
3. Sex-related factors.

1. dB of noise exposure

The cases in this study was exposed to 90 to 102 dB noise. Since the subjects were exposed to low level noise comparatively (102dB) to 140 dB the vestibular functions in this study were normal.

According to studies quoted earlier in review by Dickson & Chadwick (1951) Harris & Von Gierke(1971) a noise of over 140 dB created vestibular dysfunction in cases taken for the study.

2. Age- Factor

The patents taken for the study were of the age range between 20-30 years who did not exhibits noise induced vestibular dysfunction. According to studies by Drachman and Hard (1972) ; Nexelski, Barber & Mcllmoy, 1986; Herr, Zun & Mathews 1989; Kroenke et. al.,; 1992) patients with vestibular

symptoms presenting dizziness tend to be of middle age (late 40's and early 60's). according to Toppila .*E*, Pyyko llmari, Starck J, 2001 elderly subjects were more susceptible to NIHL than younger subjects. Taking into consideration these facts from these studies and the results of the present study we can assume that the noise induced symptoms are not much marked in the cases due to the fact that they were younger in age which reduces the risk of developing noise induced vestibular dysfunction.

3. Sex Factor

The cases taken for this study were all males who presented no noise induced vestibular dysfunction. According to Drachman and Hart 1972; Nezelski, Barber & Mcllmoyl, 1986; Herr, Zun & Mathews 1989; Kroenke et. al.,; 1992. The patients presenting with dizziness tended to be mostly females (average 60%). From this we can assume that noise induced vestibular dysfunction were not being prominent in the present study due to the reason that the cases were all males.

Also it has to be noted that all the cases in the experimental group were using ear protective devices for 4-5 hours out of 8 hours of labor which might be the reason that these cases did not present with prominent noise induced hearing loss symptoms or noise induced vestibular dysfunctions.

SUMMARY AND CONCLUSIONS

20 subjects of 10 normal healthy subjects and 10 exposed to noise were evaluated the 10 subjects related who were exposed to noise had a 4K dip in their audiometric pattern as an indication of NIHL.

These subjects audiometric and vestibular function tests were assessed.

The study was aimed to find out co-relation between noise and vestibular dysfunction i.e., whether noise could produce any vestibular

dysfancation which had damanged cochlear mechanism.

As per the results show in the previous chapter we could not establish a relationship between noise induction and vestibular dysfunction as the vestibular functions in the noise induced population showed no significant difference from the control group. This is mainly due to the fact that the noise required to create vestibular dysfunction should have been of greater intensity (130-140dB) according to various studies quoted earlier in review of literature.

Taking age factor into consideration it gives us a conclusion that in younger individuals since their ears are not susceptible to noise- exposure damage as much as in elderly population their noise- induced hearing loss is much less to be a handicap than in elderly ones. So the effects of noise on vestibular system (as cochlea & vestibular are connected together) will be negligible as evidenced by the present study. Another factor is the intensity, since the intensity of noise in which the experimental group was exposed were 95-102 dB, as the earlier studies quoted supports than a more intense sound (140 and above) in require to produced vestibular dysfunctions. Here also the

age & sex factor have to be considered. The earlier study represented females suffer from dizziness more than males & also the middle- aged. The subjects who were taken for the study were younger in age, where studies quoted had in younger age noise induced hearing loss and its effects are less and the middle aged ones are more prone to vestibular dysfunctions. So we can conclude that younger individuals and males are less prone to vestibular dysfunction. Here since the subjects were of robust health and young age this study could not find a noise- induced vestibular dysfunction in the group studied.

The study had the following limitation:

To assess qualitatively more about vestibular dysfunction ENG (Electro Nystagmo Graph) could not be used.

Hence, further studies could be carried out with ENG measurement for finer vestibular function tracking and also taking into consideration other factors such as 1) Intensity of noise exposure 2) Age 3) Sex.

To conclude:

The topic of effects of nonauditory effects of noise exposure- exposure that manifests itself in other non-hearing-related ways- including vestibular dysfunction is still very much in its infancy. Research that is both valid and reliable is very difficult to perform. Because of the inherent differences in real-world situations and well controlled laboratory situations, data need to be interpreted with caution. Nevertheless, nonauditory factors have been noted and can play an important part in the development and well being of people.

REFERENCES

Alberti, P.W. (1987) Noise and the ear. In d. Stephens (Ed) Scott-Brown's Otolaryngology, 5th ed, vol. 2 Adult Audiology (London: Butter Worths).

Alberti, P.W. (Ed) (1982) cited cited in Behar A., Chasin M. Cheesman, M. (2000) Chapter 2. Hearing & Hearing loss in Noise control A Primer singular publishing group: Canada.

Barr (1886) cited in Nageris, B.I. Attias, J. Feinmesser, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9, 45-48.

Barr T. (1988) cited in Nageris, B.I. Attias, J. Feinmesser, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9, 45-48.

Barr, D., Althoff L., Krueger, W., & Olsson, J. (1994). Work related, noise induced hearing loss: Evaluation including evoked potential audiometry. Otolayrgology - Head & Neck surgery, 110 (2), 117-184.

Bekesy, G., (1960) cited in Sohmer, H., Elidan, J., Plotnik, M, Freeman S, Sockalingam. R., Berkowidz, Z. (1999) Effects of noise on the vestibular system - Vestibular evoked potential studies in rats. Noise & health 5. 41-51.

Bohne, B.A (1976) Safelevel for noise exposure ? Annab of Otology, Rhinology & Laryngology 85, 711-724.

Caiazo, A & **Tonndorf, J.** (1977) Ear canal resonance & temporary threashold shift. Journal of Acoustical Society of America, 61, 578.

Chadwick, D.L. (1966) cited in Nageris, B.I. Attias, J. Feinmesser, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9. 45-48.

Crow, S, **Guild, S. Polrogot, L.** (1934) cited in Behar A., Chasin M. Cheesman, M. (2000) Chapter 2. Hearing & Hearing loss in Noise control A Primer singular publishing group: Canada.

Dickson, E.D.D., and Chadwick W.L., (1951) observation on disturbances of equilibrium and other symptoms induced by jet engine noise. Journal of Laryngology.

Drachman DB, Hart, C.W. (1972) An approach to dizzy patient. Neurology 22, 323-334.

Fitzgerald G, Hallpike C.S. (1942) studies in human vestibular function: 1. Observation as the directional prepondemance of caloric nystagmus resulting from cerebrial lesions Brain 65, 115-137.

Gorga, MP, Neeley S.T., Ohlrich B, Hoover B, Redner J, Peters J (1997) cited in B.C.J. Moore, (1998) Chapter 1, Physiological Aspects of Cochleal Hearing Loss Wluirr publishers: London.

Grossman, G.E. Leigh, R.J. Abel, L.A. Lanska, D.J. Thurston, S.E. (1988) cited in Peter Savundra (1997) Chapter I the Anatomy & physiology of vertigo & balance (pp 1-16) L.M. Luxon & R.A. Davies Handbook of Vestibular Rehabilitation Whurr publishers: San Diego.

Harris, C.A. (1972) cited in Kryter K.D. (1994) chapter 9. Non auditory system responses to sound & noise & reladious to health. The Handbook of Hearing & the effects of noise. Physiology Psychology & Public health. Academic Press: San Diego.

Harris, C.S. **and Von Gierke, H.E.** (1971) Cited in Kryter K.D. (1994) chapter 9. Non auditory system responses to sound & noise & reladious to health. The Handbook of Hearing & the effects of noise. Physiology Psychology & Public health. Academic Press: San Diego.

Henderson, D., Subramaniam, M. and Boettcher, F.A. (1993). Individual susceptibility to noise induced hearing loss: An old topic revisited. Ear & Hearing 14. 152-158.

Herr RD, Zunh, Matthews J.J., (1989) cited in Tina Laczo-Schroeder (1997) chapter 6. Psychological aspects of vestibular rehabilitation L.M. Luxon & R.A. Davies Handbook of Vestibular Rehabilitation Whurr publishers: San Diego.

Hilding, A.C. (1953) studies on Otic labyrinth : Anatomic explanation of hearing dip at 4096 KHz characteristic of acoustic trauma & preshycusis annals of Otology, Rhindogy & haryngology, 62, 950.

Kilburn K.H., Warshaw R.H., Hanscom B (1992) cited in Manabe, Y., Kurokawa, T. Saito T and Saito H, (1995) Vestibular Dysfanction in Noise induceed hearing loss Acta Otolarygol (Stockli) 1995): Suppl. 519: 262-264.

Kimura R.S. (1982) cited in Nageris, B.I. Attias, J. Feinmessen, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9, 45-48.

Kroneke K, Lucas C.A. Roscnbery M.L., Scherokman, B., Herbes, J.E., Wehrle P.A. Cited in Kryter K.D. (1994) chapter 9. Non auditory system responses to sound & noise & reladious to health. The Handbook of Hearing & the effects of noise. Physiology Psychology & Public health. Academic Press: San Diego.

Boggi, P.O. (1992) rest as in 27. L.M. Luxon & R.A. Davies Handbook of Vestibular Rehabilitation Whurr publishers: San Diego.

Lacour M, Xerri C (1984) cited in Peter Savundra (1997) Chapter I the Anatomy & physiology of vertigo & balance (pp 1-16) L.M. Luxon & r.A. Davies Handbook of Vestibular Rehabilitation Whurr publishers: San Diego.

Man A, **Segal** S, **Naggan** L. Vestibular involvement in acoustic trauma Journal of Laryngol Otol 1980, 94: 1395-400.

Manabe Y, Kurokawa T. Saito T., Saito H. (1995) vestibular dysfunction in noise induced hearing loss Acta Otolaryngology suppl. Stockh. 519: 262-264.

Nageris, B.I. Attias, J. Feinmesser, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9, 45-48.

National Institutes of Health (NIH) 1990 cited in Mencher, G.T., Gerber, S.E., McCombe, A. (1997) Chapter 11 Noise Exposure and Hearing Loss in Audiology & Auditory dysfunction College Hill press: Needam Heights.

Nedzelski JM, Barber HO, Maclllmoy L (1986) Diagnoses in dizziness unit. Journal of Otolarygology 15, 101-104.

Okuno, H., Kumatsuzaki A. Ogawa A. (1996) cited in Nageris, B.I. Attias, J. Feinmesser, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9, 45-48.

Oostervald W,J., Polman, a.R. and Schoonheyt, J. (1982) Vestibular implications of noise induced hearing loss British Journal of Audiology 16: 227-232.

Paparella, M.M., Costa S.S., **Fux, R. Yoon, T.H.** (1991) cited in Nageris, B.I. Attias, J. Feinmesser, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9,45-48.

Paperella M.M., Mancini F (1983) Trauma & Meniere's Syndrome Laryngoscope 93(8) 1004-1012.

Parker D.E., Ritz, L.A. Tubbs, R.L. and Wood D.L. (1976) Sohmer, H., Elidan, J. Plotnik M. Sreeman, S. Sockalingam R., Berkowitz. Z., and Mager, M. (1999) Effects of noise on the vestibular system - vestibular evoked potential studies in rats Noise & Health 5. 41-51.

Parker, D.E., Ritz, L.A., Tubbs R.L. and Wood W.L., (1976) cited in Nageris, B.I. Attias, J. Feinmesser, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9,45-48.

Parker, D.E., Tubbs, R.L. and Littlefield, V.M. (1978) Visual-field displacements in humans beings evoked by acoustical transcients. Journal of Acaustical Society of America 63. 1912-1918.

Pulec J.I. (1972) Menieres' disease: Results of two & one half year study of etiology, natural history & results of treatment laryngoscope 82: 1703-1715.

Rodger T.R. (1915) Non's deafness: a review of recent experimental work and a clinical investigation into the effect of loud noise on the labyrinth in boiler-makers. Journal of Laryngology Otology 30: 91-105.

Roggevsen, L.S., and Van Dishoeck, H.A. (1956) vestibular reactions as a result of acoustic stimulation. Pract. Oto-Rhino-Laryngology 18, 205-213.

Saunders, J.C., Dear, S.P., and Schneider, M.E. (1985) The anatomical consequences of acoutic injury. A review and tutorial. Journal of Acoutic society of America 78: 833-866.

Schuknecht, H. & Tonndorf J. (1960) Acoustic trauma of cochlea from ear surgery laryngoscope, 70, 479.

Shupak A., Bar El, E., Podoshin L. Spitzel, O. Gardon, C.R. & Ben David, J., (1994) Vestibular findings associated with chronic noise induceed hearing impairment Acta Otolarygoloy (stockh) 114: 579-585.

Sohmer, H., Elidan, J., Plotnik, M., Freeman S, Sockalingam. R., Berkowidz, Z. (1999) Effects of noise on the vestibular system - Vestibular evoked potential studies in rats. Noise & health 5. 41-51.

Toppila E, Pyyko Ilmari, Starck. J. (2001) Age & Noise induced hearing loss Scandinavian Audiology 2001. 30: 236-244.

Tullio P. Das 1929 cited in Manabe, Y., Kurokawa, T. Saito T and Saito H, (1995) Vestibular Dysfanction in Noise induceed hearing loss Acta Otolarygol (Stockh) 1995): Suppl. 519:262-264.

Vanderbei, S.L., & Loeb, M. (1976) Effects of bilateral & unilateral continous & impact noise on equilibrium as measured by the rail test. Journal of Applied Psychology 61, 123-126.

Wittmack K. (1907) cited in Nageris, B.I. Attias, J. Feinmesser, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9, 45-48.

Ylikoksi J. 1988 cited in Nageris, B.I. Attias, J. Feinmesser, R., (2000) Noise induced vestibular dysfunction Noise & health 3.9, 45-48.

Ylikoksi Juntunen, J., Matikainen, E. Yilkokski M and Ojala M., (1988) Subclinicai vestibular pathology in patients with noise-induced hearing loss from intense impulse noise. Acta Otolaryngol (Stockh) 105: 558-563.