REPEATED REFLEX DECAY TESTING

Register No. 8408

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A dissertation submitted as part fulfilment for
Final year M.Sc. (Speech and Hearing)
to the University of Mysore.

All India Institute of Speech & Hearing
MYSORE. 570 006.

MAY—1986

परम श्रद्धेय माता-पिता,
भाई-बहन, बान्धव,
एवं शुभेच्छुओं को -
जिन्हों ने मेरे सुन्दर भविष्य
का सपना देखा,
यह कृति समर्पित है -

CERTIFICATE

This is to certify that the Dissertation entitled " REPEATED REFLEX DECAY TESTING " is the bonafide work done in part fulfillment for final year M.sc., (Speech and Hearing) of the student with Register Number. 8408

Dr.M.Nithya Seelan
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CERTIFICATE

This is to certify that the Dissertation Entitled "REPEATED REFLEX DECAY TESTING" has been prepared under my supervision and guidance.

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DECLARATION

this Dissertation is my own work done under the guidance of Dr.M.N.Vyasamurthy, Deportment of Audiology, All India Institute of Speech and Hearing, Mysore-6, and has not been submitted earlier at any University for any other Diploma or Degree.

Mysore
Dated May 1936. Register No. 8408

ACKNOWLEDGEMENT

I am truly indebted to may teacher and guide Dr.M.N.Vyasamurthy for his invaluable guidance, suggestions and inspiring encouragement throughout this investigation. I owe him all that, I know in Audiology.

My sincere thanks to:

Dr.M.Nithya Seelan, Director, All India institute of speech and Hearing. Mysore for giving me this opportunity to work.

Dr.(Miss) S.Nikam, Head of the Department of Audiology for permitting me to go ahead with the Dissertation.

To my friends Usha D, Sourav and Sridhar who extended their cooperation during the study.

To my classmates for their constant reinforcement and encouragement.

To Ms Rajalakshmi R Gopal who helped me in finishing the job at a short notice.

To all my subjects for lending their invaluable time in the completion of my work.

To all those
To whom I owe once than what I know

* * *

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INTRODUCTION

The effects of noise exposures on the auditory system and the factors that influence the development of temporary and personant losses. Of hearing sensitivity have been studied extensively. Research indicates that the middle ear muscles reduce the amount of temporary hearing loss that develops during exposures to low frequency tones and noises (Hilding, 1960; Ward, 1973; Karlovich and Wiley, 1974; Zakrisson, 1975). Intense auditory stimulation elicits contraction of the middle ear muscles in the form of aa acoustic reflex (AR). This action affects the transmission of sound through the middle ear and reduces the sound energy entering the cochlea, particularly for low frequencis The physical consequences of this activity are thought to protect the organ of corti from intense sounds.

The acoustic reflex (AR) does not exert a static, unchanging effect on the auditory system, As with all reflex systems, its response is dynamic. The excitability of the middle ear muscle changes with the frequency spectrum, intensity, tenporal patterns, and duration of the acoustic stimulus. If the acoustic reflex (AR) actually serves to protect the ear from over stimulation of Intense sounds, then the magnitude of reflex activity during constant sound stimulation is of paramount importance in evaluating the efficiency of its protective function (Gerhardt, et al 1980).

Acoustic Reflex Decay is a relatively new area of investigation. Decay of the AR refers to a decrease in the magnitude of the reflex response during sustained acoustic stimulation. Anderson, Barr and Wadenberg (1969,70) have suggested that the measurements of acoustic reflex adaptation ia an indication of VIII cranial nerve integrity, Additionally, we have Borg and Odman's (1979) confirmation that decay and recovery originate in the afferent auditory pathway and not in the acoustic reflex

The role of the stapedius reflex in protecting the ear from noise induced hearing loss has been much debated (e.g. Toundorf, 1976). Whatever role it has it is usually considered to be relatively small, largely due to limitations imposed by the reflex decay. However, present literature supports the observation that Changes in the sound parameters lead to a reactivation of the reflex responses. Recently it has bean found (Borg et al 1979) that the human atapedius reflex is in fact active throughout long-term exposure to an industrial noise which ia variable with respect to frequency and amplitude.

Johansson et al (1967) Miyakita et al (1978) have found relationship between the acoustic reflex threshold and susceptibility to noise induced hearing loss or permanent threshold shift. Anae Zachariah (1990) found that the subjects who show greater TTS exhibit low acoustic reflex threshold tad greater magnitude of contraction of the atapedius Muscle, whereas subjects who exhibit high acoustic reflex thresholds show less TTS and less magnitude of contraction of the stapedius muscle. Further Jagadish (1982) round that subjects with lower TOM (Threshold of Octave Masking) have lower ART (Acoustic Reflex Thresholds) and higher magnitude of reflex. Lower TOM, lower ART and higher Magnitude of reflex may be suggestive of susceptibility to noise induced hearing loss.

The present study was designed to find the susceptibility of normal hearing listeners to noise induced hearing loss. (Permanent threshold shift). It has been suggested that several types of auditory pathology, including noise induced damage are gradual processes which eventually culminate in a pure tone hearing loss (Hawkins, 1973). If auditory pathology is to be identified before irrepairable damage results in a pure tone hearing loss, than new tests which are sensitive to subtle dysfunctions must be used. This "Repeated reflex decay testing" is among the objective indices of susceptibility to noise induced hearing loss and can be considered as a more reliable measure than psycho-accustic tests for susceptibility to noise induced hearing loss.

Statement of the problem:

The present study was designed to investigate whether there is any relationship between the reflex decay for repeated stimulus presentations and magnitude of acoustic reflex.

Also the present study was designed to have information regarding the frequency of the stimulus eliciting the reflex and the decaying process.

The present study aims to find answers to following questions:

1) Do the subjects showing repeated reflex decay exhibit greater magnitude of the Acoustic reflex.

3) Is there any relationship between frequency of the stimulus and the repeated reflex decay.

3) Is there any relationship between sensation level of the stimulus and the repeated reflex decay.

REVIEW OF LITERATURE

The Review of Literature concerning the present study deals with the three important research areas:

1) Acoustic Reflex vs Reflex Decay.
2) Reflex decay and its relationship to noise induced hearing loss or permanent threshold shift.
3) Tests of susceptibility to noise induced hearing loss using Acoustic Reflex Characteristics.

1. Acoustic Reflex vs Reflex Decay: "When an acoustic signal is presented to the ear at a high intensity level, the stapedius mascle contracts producing a change in the acoustic immittanee characteristics of the middle ear transmission system. If the reflex activating signal is sustained, then the stapedius muscle gradually relaxes and the acoustic immittance of the middle ear mechanism returns to its prestimulus state. The relaxation of the stapedius muscle and the consequent restoration of the acoustic immittance is called acoustic reflex adaptation or decay". (Flwler and Wilson, 1984).

The neural network involved in the reflex activation of the stapedius muscle during acoustic stimulation is not known ia detail. Experimental studies in animals have shown that the pathways of the acoustic stapedial reflex are located in the lower part of the brain stem (Borg, 1973). The course of the acoustic stapedius muscle reflex are in man is not known but the pathway of the acoustic reflex are in rabbits, as determined by Borg (1973) is depleted in Pig.l.

Pathways of the ipsilateral stapedius Reflex Acoustic input via the ossicles stimulates cochlear hair cells resulting in afferent impulses via the primary acoustic neurons. The primary acoustic neurons constitute the acoustic nerve. The graphic centre for the second acoustic neuron is in the ventral cochlear nucleus. The majority of axons from the ventral cochlear nucleus pass through the trapezoid body to the medial part of the facial motor nucleus, and from this nucleus the electric impulses are transmitted through the facial nerve to the ipsilateral stapedius muscle. In addition, some nerve fibres pass from the ventral cochlear nucleus through the trapezoid body to the ipsilateralmedial superior olive. From this nucleus the electrical impulses are transmitted via a third neuron to the medial part of the ipsilateral facial meter nucleus. The ipsilateral stapedius reflex are thus consists of mainly three but to some extent four neurons (Borg, 1973).

Pathways of the contralateral stapedius reflex - The electrical impulses from the sensory cells in the cochlea pass via the primary acoustic neuron to the ventral cochlear nucleus as in the ipsilateral stapedius reflex. From the ventral cochlear nucleus the electric impulses are transmitted through a second neuron to the region of the medial superior olive. A third neuron connects the medial superior olive to the medial part of the contralateral facial motor nucleus, A fourth neutron transmits the electric impulses from the facial motor nucleus to the contralateral stapedius muscle. Thus the contralateral stapedius reflex are contains four neurons (Djupesland, 1979).

It is Known that the reticular formation can influence the activity of the middle ear muscles of the cat, particularly the stapedius muscle (Hugelin, 1960) and that higher auditory centers, including the auditory cortex, can also exert control of these muscles (Baust and Berlucchi, 1964).

The superior olivary complex, Which also contains elements of the reflex pathways of the muscles end the efferent innervation of the spiral organ, possibly regulates the conduction of centrifugal impulses along the reflex circuits of the middle ear muscles and these two other regulatory systems. The resultant affects on middle ear muscle activity of interaction between the auditory pathways mod the cortex via the centrifugal extra-reticular auditory control system, the cortex, and the retleular substance, and between the reticular substance and the auditory pathways, are difficult to predict and remains to be resolved. Activity patterns are further complicated by direct control exerted by fibres descending from non-auditory cortical areas.

(Pyramidal Aract, germma system) the possible proprioceptive control mechanisms of the middle ear muscles, and reflex interconnections between afferent fibres from some other motor systems and cutaneous receptors of the external ear canal and the face (Anderson, 1975).

Normal Acoustic Reflex Decay:

The first studies in which an acoustic impedance bridge was used to study reflex decay were performed by Metz (1946, 51). Then two studies by Djupseland and Johansson in the late 1960s demonstrated that acoustic reflex adaptation was frequency dependent with the reflex maintained longer for low-frequency activator signals (500 and l000Hz) than for high frequency activators (3000Hz).

The effects of the intensity level of the activator signal on the characteristics of reflex adaptation also appears to be frequency dependent. Excepting wiley and Karlovich (1975), most studies indicate that reflex adaptation decreases as the intensity level of l000Hz activators increases (Rosenhall et al, 1979; Wilson et al, 1978). For 2000Hz activators, changes is the intensity level of the activator signal do not appear to have systematic effects on the amount of reflex adaptation (Givens and Seidemann 1979; Wiley and Karlovich, 1975).

Fowler mad Wilson (1984) cite a typical examples of reflexes to sustained activator signals (10.2 secs) from a young adult with mammal bearing and are shown in Fig.2. The reflex is maintained at a constant level for the 500 and 1000Hz activators (top panels). With the 2000HZ activator (lower, left panel), the magnitude of the reflex diminishes over time with the onset of adaptation (90%) occurring at 3.3 sec; in this example, the half-life time (50%) for 2000Hz was not attained during the 10.2 sec. activator period. At 4000Hz (lower, right panel), the magnitude of the reflex is diminished and both the onset of adaptation (1.1 sec) and the half-life time (2.2 sec) were measurable; the reflex offset can be observed between 10 and 11 sec. This example clearly demonstrates the frequency-dependent characteristics of acoustic reflex adaptation. The admittance data were recorded with a 226-Hz probe and were corrected for ear canal volume.

Diagnostic use of Acoustic Reflex Decay:

The primary use of acoustic reflex decay is in the differential diagnosis of cochlear and retrocochlear pathology. Of particular interest is the Identification of eighth nerve and lew brain stem lesions, such as acoustic neuromas, brain stem infarct and multiple sclerosis. Specific characteristics of the acoustic reflex, such as threshold and adaptation, may distinguish cochlear from retrocochlear pathology (Fowler and wilson, 1984).

Hirsch end Anderson (1980) have provided useful set of rules for the interpretation of reflex adaptation data. (1) RD^{+++} if the reflex magnitude declines $\geq$ 50% within 5 sec. at 500 and 1000Hz. (2) RD^{++}, if the reflex declines $\geq$ 50% within 5 sec. at 1000Hz but not at 500Hz and (3) RD^{+}, if the reflex declines < 50% within 5 sec. at 500 and l000Hz. RD^{+++} is considered a positive retrocochlear sign, RD^{++} is quesionable and should be followed over time, and RD^{+} is not significant.

2) Reflex Decay and its relationship to noise induced hearing loss or permanent threshold shift:

The acoustic stapedius muscle reflex offers special possibilities for studying decay, since the course of its decay can be observed on a continuous basis in non-anesthetized animals as well as in humans. In animal research, reflex decay is accessible for further neurobiological analysis. In humans, this decay has implications for clinical audiology as well as for evaluation of the protective action of the stapedius reflex against hearing damage by noise (Borg and Odman, 1979).

The role of the stapedius reflex in protecting the ear from noise-induced hearing loss has been much debated (Tonndorf, 1976) Whatever role it has it is usually considered to be relatively small, largely due to the limitations imposed by the reflex decay

Karlovich et al (1977) speculate that acoustic reflex dynamics, particularly reflex relaxation, reflex adaptation and reflex temporal summation, probably are involved in producing the differential effect of contralateral noise on temporary threshold shift

Wiley and Karlovich (1978) concluded that the relations between temporary threshold shift and acoustic reflex dynamics are, however, complex and temporary threshold shift, reductions associated with acoustic reflex activity will vary dependent on the spectral, intensity and temporal characteristics of the ref leam-activating signals.

Borg and Odman (1979) conducted a study on 10 normal hearing subjects, the aim was to study the time course of the decay and recovery of ipsilateral and contralateral stapedius reflex-respons to 2 KHz tone. Reflex responses were found to follow a closely similar time course with respect to both decay and recovery in simultaneous bilateral recordings. The similarity is compatible with the assumption that decay and recovery originate in the afferent auditory system and not in the muscles. Recovery was 50% complex, 250 msec after the end of the stimulus.

The individual correlation between decay and recovery was negative but weak, Which is interpreted as showing that these processes have a tendency to balance each other, but are based partly on different mechanisms.

Berg, Nilsson, Lid' en (1979) investigated the fatiguability of the acoustic stapedius muscle reflex in an actual noisy industrial environment in normal hearing subjects. In a Laboratory situation a small depression was found with a considerable individual variability, the stapedius reflex recovered slowly, approximately as a linear function of time. In a field study on an entire day of exposure in a ship building yard, the reflex depression was on the average 4 dB in response to a stimulation of 2KHz 10 minutes after the end of the work day. This corresponds to less than 8 dB immediately at the end of the exposure.

Rosenhall et al (1980) studied fatigue of the stapedius reflex in 100 normal ears. Stimulus frequencies 500 and l000Hz were used and the decrement of the stapedius reflex at 5, 10, 15 dB above the reflex thresholds was estimated. No decrement at all (less the 10% decay 10 seconds after the onset of a stimulus 10 dB above the reflex threshold) was present in a majority of the ears. Between 27 and 58% of the ears showed reflex decay depending on variations of different factors such as stimulus frequency, intensity of the stimulus and the method to register the reflex. Reflex decay was observed more at l000Hz than at 500Hz stimulus frequency. Reflex decay was also more often seen 5 dB above the reflex threshold the at higher intensity levels.

Gezhardt and Hepler (1983) report that the extent to which the acoustic reflex reduces the temporary hearing loss that would be caused by exposure to noise is dependent upon the magnitude and response duration (decay) of the acoustic reflex. These two factors are influenced by the nature of the stimulus and the effect that the noise exposure has upon the acoustic reflex response. The effect of the acoustic reflex on the auditory system is not static since its magnitude and temporal features change as the amplitude distribution of the stimulus changes and as the duration of the stimulus increases. Tests of susceptibility to noise induced hearing loss using Acoustic Reflex characteristics.

Johansson et al (1967) suggested that the acoustic reflex can be used to evaluate an individuals ear susceptibility. Further more Miyakita et al (1978) measured the magnitude threshold and latency of the aeouatlc reflex and the acoustic impedance of the middle ear.

They found that reflex latency decreases with increasing stimulus level, and a positive correlation was found between the reflex latency and the reflex thresholds. From these results it is conceivable that a person hawing a high reflex thresholds might be susceptible to mound with short rise time and short duration, such as an impulsive noise because of its prolonged latency and small magnitude of the acoustic reflex. However, the results obtained by them shows large individual differences between 190 subjects. The authors concluded that the acoustic reflex threshold can be used as an indicator showing individual susceptibility to impulsive noise.

Anne Zachariah (1980) studied the relationship between "Acoustic reflex threshold and temporary threshold shift" in seventy normal hearing subjects in the age range of 17 to 30 years, on the whole analysis of her data revealed that subjects with "tender" ears who exhibit low acoustic reflex thresholds show greater temporary threshold shift and a greater magnitude of the contraction of the acoustic muscles through the acoustic reflex and that subjects with "tough" ears who exhibit high acoustic reflex thresholds show less temporary threshold shifts and less magnitude of the contraction of the acoustic muscles. Further the author emphasizes that acoustic reflex threshold could be used as a possible predictor for susceptibility to noise induced hearing loss.

Kamakshi (1981) concluded from her study that there is a good correlation between "Fatigability of reflex and temporary threshold shift". The fatigability of reflex test can be used as a predictor of susceptibility to noise induced hearing loss. It can also be seen that subjects who had greater temporary thresh eld shift (TTS) scores had greater "Fatigability of reflex" and they also exhibited reflex decay within 3 minutes and the subjects

Who had low TTS (Temporary Threshold Shift) scores adhibited less "Fatigability of reflex" and did not exhibit reflex decay within 3 minutes.

Jagadish (1982) found a positive correlation between acoustic reflex thresholds and threshold of octave masking values, and from his study it may be stated that magnitude of reflex can also be used as an indicator for detecting the individuals who are susceptible to noise induced hearing loss, It appears that subject who exhibit low acoustic reflex thresholds may also show greater magnitude of reflex and also show lower threshold of octave masking values.

Need and characteristics of a good test of susceptibility for NIH

As the noise and noise induced hearing loss have been increasing in their magnitude day by day, considerable attention is need on the control of noise and prevention of noise induced hearing loss. One of the ways,is to protect the susceptible ears/persons from the damage or exposure to noise and for this an adequate test to identify the susceptibility is highly needed. Different tests have beam proposed so far, and a good test should have the following characteristics (Summerfield et al 1958).

1. The equipment to be employed must be simple and rugged.
2. It must be sufficiently uncomplicated so that subjects and relatively naive testers can perform the teat without difficulty.

3. It must be both valid and reliable.
4. The test results must be immediately classlfiable into degrees of susceptibility that are meaningful to industrial personnel who have to assign employees to specific tasks.
5. The test must not take more time for its administration.

Distribution of Susceptibility:- Ward (1965) reviewed the concept of susceptibility to hearing loss following continuous exposure and concluded that susceptibility was normally distributed in a population. Summerfield et al (1958) suggested that if susceptibility to noise induced hearing loss is distributed normally throughout the population then the number of highly susceptible ears will be only a very small proportion of the population. And if this is in fact that case, then research efforts might better be spent in learning how to achieve better protection of the bearing of the much larger number of workers Who are moderated susceptible to noise induced hearing loss.

Bishnoi (1975) conducted a study aimed to find the distributional pattern of individual susceptibility to noise induced hearing loss in Indian Population. He used Wilson's test to determine the degree of susceptibility. His main findings were that:

1. The temporary threshold shift scores are distributed normally over a range of 0 to 30 dB.

2. TTS of 10 dB is a better index of the degree of susceptibility to TTS.
3. No differences were noticed between (a) males and females (b) normal hearing and impaired hearing subjects. (c) no ear difference; and
4. There exists a negative correlation between TTS and resting thresholds.

The above review of literature clearly indicates that there is a need for the objective and less time consuming tests to find out the susceptibility to noise induced hearing loss. The present study has been undertaken in lieu of the above said facts.

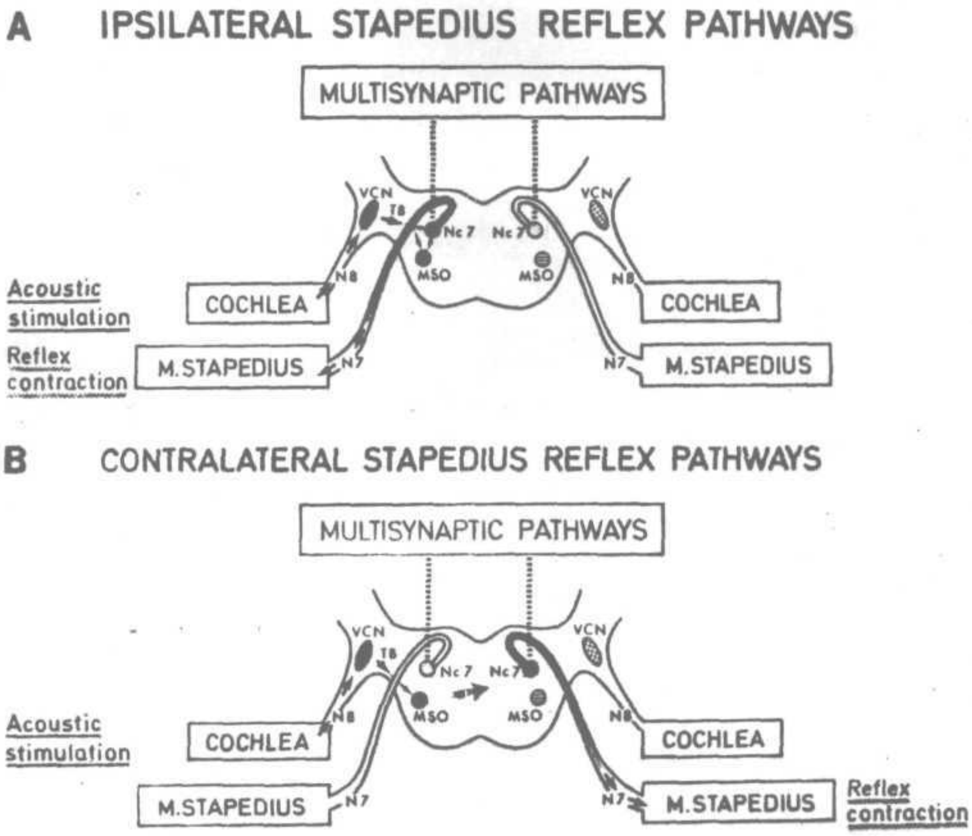


Figure 1. Schematic diagram over ipsi- and contralateral stapedius reflex pathways, based on experimental work in rabbits (Borg, 1973). N8, acoustic nerve; VCN, ventral cochlear nucleus; TB, trapezoid body; MSO, medial superior olive; N7, facial nerve.

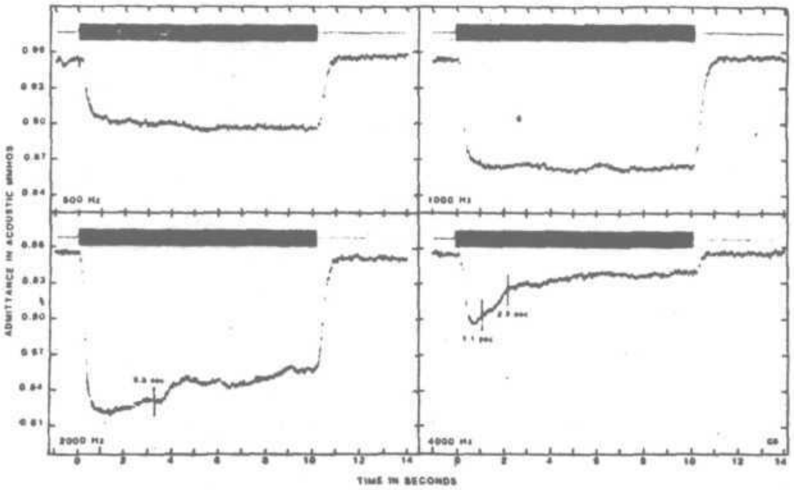


Figure 2. Reflex adaptation responses obtained with four 10.2-sec reflex-activator signals (500, 1000, 2000, and 4000 Hz) presented 10 dB above the reflex threshold of a young adult with normal hearing. The data were corrected for ear canal volume and were measured contralaterally with a 226-Hz probe. The onset of adaptation is noted on the 2000- and 4000-Hz tracings, and the half-life time is noted on the 4000-Hz tracing.

METHODOLOGY

1. SUBJECTS:

Twenty three normal hearing subjects (12 females and 11 males) within the age range of 17 to 23 years were selected for this study.

They were divided into four different groups to measure there "Repeated Reflex Decay" at four different frequencies and four different sensation levels, this constituted experiment No.l. Further 12 out of 23 subjects were selected for experiment No.2 i.e. to determine the magnitude of acoustic reflex in subjects who do not show any noticeable reflex decay and Who Show reflex decay. The probe ear was selected randomly. All the subjects had normal hearing (15 dB HTL ANSI 1969). The subjects were selected on the following criteria:

1) They should not have had any history or chronic ear discharge, tinnitus, giddiness, earache or any other otological complaints.

2) They should have A type tympanogram, static compliance within normal range of 0.30 to 1.60 cm^3 (Jerger, 1970) and middle ear pressure within normal limits of ± 50 mm H_2O (Porter, 1972) in the probe ear.

3) They should be able to tolerate high intensity pure tones without any complaint of headache, giddiness, etc.

II. EQUIPMENT:

The equipmeat used was Madsen z0174 Immittance Audiometer.

Brief Description of the Instrument:

The Madsen Z0174 Immittance Audiometer is one of a series of Madsen instruments designed to measure middle ear function. It is a "computerized" instrument Which uses microprocessor technology to provide a very versatile measurement system. It is designed to conduct manual, semi automatic and automatic measurements of tympanometry and acoustic reflexes. The results can be viewed as they are obtained on the MONITOR. They can also be printed quickly or transmitted via the DATA XMII option to another device much as a computer. But since this option warn not available to the experimenter, tracing paper was used, to trace the visual display.

The main functions of the ZO174 Include:

Screening:- Automatic tympanogram and acoustic reflexes.

Tympanogram and Reflexes: Automatic tympanogram and reflex thresholds.

Expanded Reflexes: Automatic recording at two sequential freguencies

Averaged Reflexes: At any selected stimulus.

Reflex Decay: At any two selected stimuli.

SPECIFICATIONS:

COMPLIANCE:

Probe tones: 226 and 1000 and either 660 or 800 Hz.

Total Range = 0.1 ml to 7.0 ml.

Reflex: 0 to 0.15 ml
0 to 0.3 ml

Tympanometry: 0 to 1.5 ml
0 to 3.0 ml
0 to 4.5 ml

AIR PRESSURE SYSTEM:

Range: Normal + 200 to - 400 daPa
Extended + 400 to -800 dAPa
Rote 1
Extended range must be selected manually.

Air Pamp: Plunger type
Stopper motor - digitally controlled
speeds - selectable 400 daPa/sec
200 daPa/sec
100 daPa/sec
50 daPa/sec

Manual operation to max. 400 daPa/sec. Bar graph pump position indicator.

Air Release: Mechanical safety release
- valve set to + 800 and -1200 daPa

Manual air release: Solenoid activated by push button. Automatic air release, solenoid - activated by computer.

ACOUSTIC REFLES : 1. "Real time" for decay measurements
Selectable modes
(Ipsilaterally) 2. Time division multiplexed.

Contralateral stimulation: 1. Pure tones:
500, 1000, 2000, 4000Hz, 0-110 dBHL
with range extender to 125 dBHL
6000Hz to 110 dBHL
8000Hz to 100 dBHL
2. Pulsed tones: All frequencies
3. Click Train: 100 sex click
4. White noise - 0-110 dBSPL with
range extender to 125 dBSPL.
5. External input.

Ipsilateral stimulation : 1. Pure tones:
(226Hz only) 500, 1000, 2000Hz 115 dBSPL
4000Hz, 0-90 dBSPL
2. Click train: 100 sec clicks.

Bilateral stimulation : Any two tones may be selected for simultaneous bilateral presentation

Dual contralateral stimulation : The tones from the ipsilateral ranges may be presented to the contra earphone, permitting simultaneous presentation of any two tones to the contralateral ear.

External Input : An external signal source may be connected.

: Input impedance 47K
Input voltage 5VRMS max.
Frequency range lOOHz - lOKHz

OPERATION MODES

The ZO174 will operate in three modes for tympanometry and acoustic reflex measurements. These modes are 1) automatic, 2) semi-automatic and 3) manual.

CALIBRATION:

The 20174 requires periodic checking of its acoustical calibration, for the probe tones and the various stimuli used for eliciting the acoustic reflex. As transducers change their characteristics over time, or as a result of rough handling of them, the instrument may need re-calibration. Re-calibration will always be necessary if any one of the transducers is changed. All calibration cheeks and calibration changes may be performed by the user. In addition to the supplied calibration check cavity, the equipment required is a precision sound level meter with octave filter and with 6cc and 2cc couplers. A philips head screwdriver will also be needed. The Z0174 has three elements that require separate calibration procedures:

Probe tones
Ipsilateral stimuli
Contralateral stimuli

The calibration data for these are stored in a user-programmable memory Which does not require battery or other power in order to retain the data. Inside the Z0174 is a callbration/Run switch which, in conjunction with certain of the front panel push-buttons, allows for re-programming.

At any time during the calibration procedure, pushing the stop/Reset button will cause the 20174 to exit from the calibration mode. When the calibration has been completed, pushing Autostart will transfer the new calibration data to permit memory. Resetting the calibrate/Run switch to Run and closing the cover Makes Z0174 ready for use again.

For research purposes, Z0174 calibration on be changed for a short period (in practice for as long as the power is kept on) by returning to HSU without pushing autostart, while retaining the original calibration. The original calibration can be retrieved by Simply turning the power off and on again.

PROBE TONE - Compliance Calibration.

To recalibrate, proceed as follows:

a) Push probe into calibration check cavity, using 10 mm probe tip
b) Push air release
c) See that compliance now reads 2.0 cc ± 0.1
d) If it does not, push and hold probe tone and then push autostar
e) Compliance will now read 2.0cc.

Ensure that probe and tip are properly seated, or calibration will not be correct.

TEST ENVIRONMENT:

The experiment was carried out is sound treated room at the Audiology Department, All India Institute of Speech and Hearing, Mysore.

Location of the Instrument: The instrument was placed inside a large sound treated room.

1) Humidity was neither too high or low to the point where either the subject or clinician were uncomfortable.
2) It was away from noisy drafty or excessive vibration area.

PROCEDURE:

The test procedure consisted of dividing the subjects into four distinct groups. This grouping was based on the sensation level at which "Repeated Reflex Decay" (contralateral stimulation) was measured. Four sensation levels (5, 10, 15 and 20dBSL reference acoustic reflex threshold of the individual subject) were chosen. Repeated decay for each subject was measured at 500Hz, 1KHz and 2KHz and 4KHz. Five subjects were taken to measure repeated decay at 5 dBSL and 15 dBSL. Four and nine subjects were taken respectively to measure repeated decay at 10 and 20 dBSL.

The experimental tasting was carried out in two stages:

a) Stage-l: The acoustic reflex thresholds (contralateral stimulation using T & R setting of the instrument. Further "Repeated Reflex Decay" was observed at single frequencies by presenting the acoustic stimulus five times (Five presentations) and by setting the instrument in the DECAY mode. The time internal between the stimuli was less than 3 second 500Hz and 2KHz were selected for one experimental session and lKHz and 4KHz were selected for another experimental session. The selection of the frequencies in any experimental session or across the two experimental sessions was random. the second experimental session was carried after a gap of a minimum of twentyfour hours). A rest period of one minute was given in individual experimental session in between the two frequencies tested for Repeated Decay.

The selection of frequencies inany particular experimental session, the time gap between the two experimental sessions end the rest period ell account to control interactive effect of different frequencies so that decaying process at any one frequency does not affect the decay at the another frequency. (Fig.3)

b) stage-2: Procedure to determine the magnitude of the acoustic reflex in(contralateral stimulation)at 2KHz in subjects selected from 15 dBSL and 20 dBSL group The instrument was kept in the "AVERAGING" mode. First the averanged magnitude

(10 averagings) at the acoustic reflex threshold was obtained and this was subtracted from averaged magnitude (10 averagings) obtained at 20 dBSL, (ref ART of individual subjects) to find the magnitude of acoustic reflex. The selection of the frequency 2KHz was done on the basis that at this frequency, some subjects showed decay and some did not, whereas at 4KHz, all the subjects showed decay and at 1KHz, none of them showed from the above said two groups.(Fig 3)

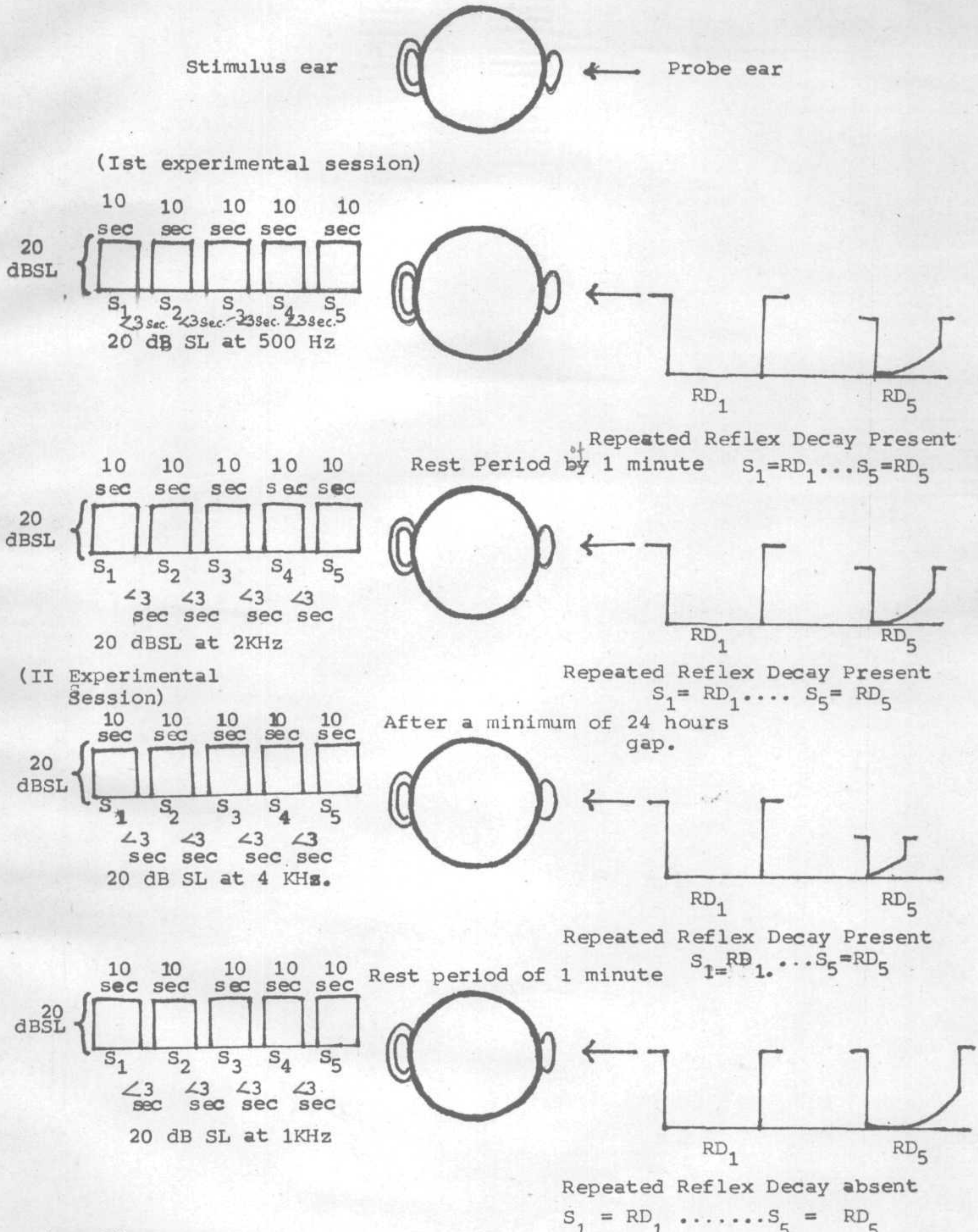
To find the magnitude of reflex decay and the averaged magnitude of acoustic reflex, thin tracing paper and felt/sketch pan were used. Visual display of the initial magnitudes of the first and the fifth presentations were traced down with the help of trace paper. Two readings of the averaged magnitude were similarly obtained. Magnitude of the Acoustic Reflex and the Acoustic Reflex Decay was measured in centimeters.

Amount of reflex decay was calculated using the formula:

$$\text{Amount of reflex decay} \quad \frac{(\text{Intial magnitude of Ist presentation} \quad \text{Initial magnitude of 5th presentation})}{\text{Total number of presentations(5)}}$$

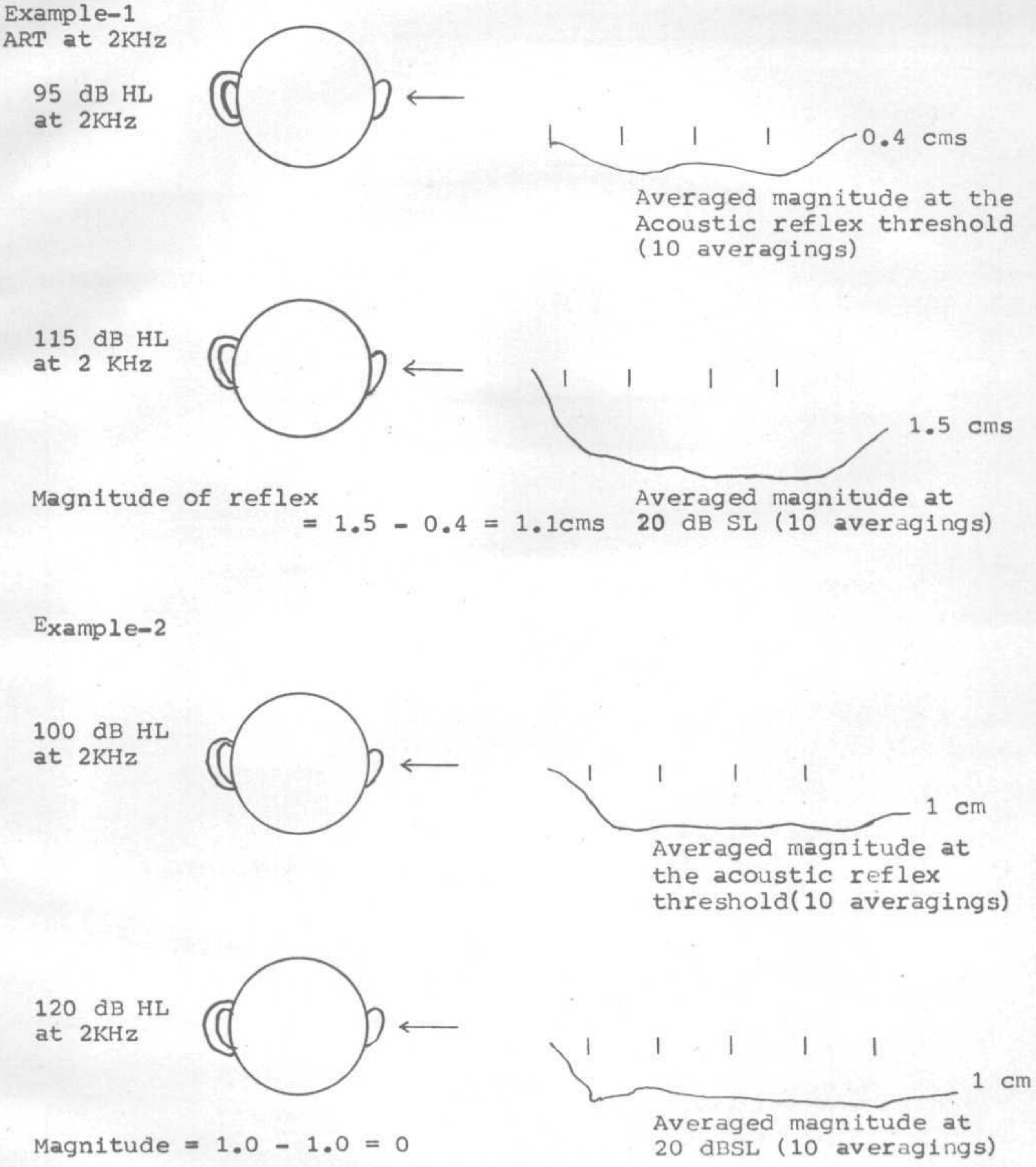
in centimeters.

Example to show the procedure of Repeated Reflex Decay Testing



(Fig.3)

Examples to show the computation of magnitude of reflex



Note: Example-1: Magnitude of Acoustic reflex in the subject who showed repeated reflex decay.

Example-2: Magnitude of acoustic reflex in the subject who did not show repeated reflex decay.

Fig.(4).

RESULTS AND DISCUSSION

The data obtained were subjected to statistical analysis. The results obtained are displayed an the tables and can be discussed under following headings:

1. Amount of reflex decay on presentation of tone repeatedly (5 times) at 5 dBSL, 10 dBSL, 15 dBSL and 20 dBSL for frequencies 5OOHz, 1KHz, 2KHz aad 4KHz.

3. Magnitude of Acoustic reflex ia subjects for 2 KHz at 20 dBSL who do mot show any noticeable reflex decay.

3. Magnitude of Acoustic reflex ia subjects for 2KHz at 30 dBSL who show reflex decay.

1. amount of reflex decay on presentation of tone repeatedly (5 times) at 5 dBSL, 10 dBSL, 15 dBSL, and 20 dBSL for frequencis 5OOHz, 1KHz, 2KHz and 4 KHz.

Tables 1(a), 1(b), 1(c), l(d) show the amount of mean reflex decay at 5 dBSL.

There is no reflex decay at 500 Hz aad 1KHz, whereas the decay is present at 2KHz aad 4KHz. The amount of decay ia more at 4KHz, than at 2KHz.

Tables 2(a), 2(b), 2(c), 2(d) show the mean values of reflex decay at 10 dBSL.

The reflex decay is present at all frequencies and increases from 500Hz to 4 KHz. That is to say, there is more decay at higher frequencies than at lower frequencies. However, in this group, the amount of decay at lKHz is more than the amount of decay at 2KHz.

Tables 3(a), 3(b), 3(c) and 3(d) show the mean values of reflex decay at 15dBSL.

From the above tables, it is clear that, there is more decay at higher frequencies than at lower frequencies, the mean decay increases with frequencies higher the frequency, greater the decay.

Tables 4(a), 4(b), 4(c) and 4(d) show the mean values of reflex decay at 20dBSL.

Here again the amount of reflex decay present at higher frequencies is more than that of lower frequencies.

On comparing the mean amount of repeated reflex decay obtained at different sensation levels, the trend is that there is greater amount of reflex decay obtained at higher sensation level (for e.g. 20dBSL) and very little reflex decay is obtained at lower sensation levels (5 dBSL). This holds good for all the groups excepting the 10 dBSL group. In this group, the mean amount of reflex decay obtained at 1KHz and 4KHz is more than the wean amount of reflex decay obtained for 1KHz and 4KHz for 15 dBSL and 20 dBSL groups. Even at 2KHz, the amount of decay obtained in 10 dBSL group is more than that obtained in 15 dBSL group for the same frequency.

The data in the tables indicate that there is least variability at 500Hz, across all the groups and the variability increases at higher frequencies. This can be seen clearly from the tables 4(a), 4(b), 4(c) and 4(d) of the 20 dBSL group,

2. Magnitude of Acoustic reflex in subjects at 2KHz, 20 dBSL who do set show any noticeable reflex decay.

Table 5 clearly shows that the magnitude of Acoustic reflex at 20 dBSL is very little.

3. Magnitude of Acoustic raflex in subjects at 2KHz, 20 dBSL who show reflex decay.

From table 6, it is clearly evident that there is greater magnitude of acoustic reflex at 20 dBSL.

On comparing tables 5 and 6, the mean scores of .18 and 0.7 are obtained respectively. Hence, we canaay that the subjects who showed reflex decay, also showed greater magnitude of acoustic reflex and the subjects who did not show reflex decay showed lesser magnitude of acoustic reflex.

The variability data indicate that the subjects who show reflex decay are more variable than the subjects who do not show reflex decay.

Discussion:

The value of the teat battery approach in assessing human auditory function has been recognised for many years and may prove a more reliable approach to susceptibility testing. If several correlates are identified, they could be used as a test battery for susceptibility to noise induced hearing loss (Eldrege and Miller 1969; Ward and Durall 1971; Borg, 1968; Brasher et al 1969; Coles 1969; Zakrisson and Borg 1974; Zakrisson et al 1975; reported the influence of the acoustic refles to temporary threshold shift (TTS).

The relation between TTS and ART exists because when reflex occurs there will be attenuation of low frequency sounds reaching the cochlea. As TTS is related to the intensity of the signal reaching the cochlea, the reduction in the intensity of the signal brought about by reflex action can be expected to result in less TTS. On the basis of TTS many tests have been developed to identy subjects Who are susceptible to noise induced hearing loss or PTS

Anne zachariah (1980) has found that the subjects with low acoustic reflex thresholds show greater TTS and greater magnitude of contraction of the stapedius muscle through the acoustic reflex and the subjects who exhibit high reflex thresholds show less TTS and less magnitude of contraction of stapedius muscles. Jagadish (1933) has found that subjects with lower threshold of octave masking (TOM) values have lower ART and higher magnitude of reflex. Further he reports that lover TOM, lower ART and higher magnitude of reflex may be suggestive of susceptibility to noise induced hearing loss.

Based on the above research a study was carried out to investigate the relationship between 'Repeated Reflex Decay Testing" and susceptibility to noise induced hearing loss.

The subjects who showed "Repeated Reflex Decay" also showed greater magnitude of acoustic reflex and the subjects who did not show noticeable reflex decay, showed very little magnitude of acoustic reflex.

This study can be correlated with Anne Zachariah's (1980) study, the subjects who have high TTS, show repeated reflex decay and greater magnitude of acoustic reflex and subjects who have low TTS, do not show repeated reflex decay and show less magnitude of acoustic reflex. Jagadish (1982) also supports the above contention.

This study also gives information about the repeated reflex decay at various frequencies and at different sensation levels.

Djupseland tad Johansson (1967) demonstrated that acoustic reflex adaptation was frequency dependent with the reflex maintained longer for low frequency activator signals than for high frequency activators. The present study supports this here repeated reflex decay is greater at higher frequencies than at lower frequencies indicating that reflex decay is a frequency dependent phenomenon.

Wiley and Karlovich (1975) report adaptation data across subjects for the 500Hz activating signals at levels of 5, 10 and 15 dB above individual acoustic reflex thresholds, they found on comparison of acoustic impedance change over the entire signal interval that reflex adaptation increases with increase in in activating signal level. The present study supports Wiley and Karlovich (1975) data and draws support to infer that greater amount of repeated reflex decay is seen at higher sensation levels

However, earlier investigations by Wilson et al (1978) and Resenhali et al (1978) report that reflex adaptation decrease, as the intensity level of lOOOHz activator increases for 2000Hz activators, changes in the intensity level of the activator signal do not appear to have systematic effects on the amount of reflex adaptation.

It is difficult to make specific comparisons between the acoustic reflex data here and those of earlier investigations.

since most other studies employed only a single activator level and involved observation of acoustic reflex activity over substantially shorter durations.

The effects of the intensity level of the activator signal on the characteristics of reflex adaptation also appear to be frequency dependent.

Finally a major portion of the confusion regarding acoustic reflex adaptation can be due to differences. in experimental methodology, spectral, temporal or intensity characteristics of the reflex-activating signals used. A more complete description of the adaptation properties of the acoustic reflex for comparable signal parameters appears warranted to evaluate adequate inferences drawn regarding the role of the middle ear musculature in psycho-acoustic phenomena (such as TTS). The purpose of this study, was to examine systematically the response of the acoustic reflex to sustained signals at different frequencies, at difference levels above acoustic reflex thresholds.

Table-1(a): Amount of reflex decay on presentation of tone repeatedly at 5 dBSL, 500Hz measured in cms.

Subjects	Probe ear	ART at 500Hz	Amount of decay
1.	Left	90 dB	- 0.04
2.	Left	100 dB	- 0.12
3.	Right	100 dB	+ 0.02
4.	Right	95 dB	- 0.07
5.	Right	90 dB	+ 0.04

M = -0.034

n-l - .0645

+ Sign indicates Reflex decay

- sign indicates No reflex decay

Table-1(b): Amount of reflex decay on presentation of tone repeatedly at 5 dBSL, 1KHz measured in cms.

Subjects	Probe ear	ART at 1KHz	Amount of decay
1	Left	100 dB	+ 0.01
2	Left	100 dB	- 0.04
3	Right	90 dB	0
4	Right	90 dB	- 0.04
5	Right	85 dB	- 0.03
			M = - 0.02
			σ n-1 - .25495

+ Sign indicates reflex decay

\- Sign indicates no reflex decay

Table-1(c): Amount of reflex decay on presentation of tone repeatedly at 5 dBSL, 2KHz Measured in cms.

Subject	Probe war	ART at ZKHs	Aaouat of decay
1	Left	90 dB	+ 0.3
2	Left	90 dB	- 0.15
3	Right	90 dB	+ 0.47
4	Right	110 dB	+ 0.12
5	Right	90 dB	+ 0.14

M = .123

σ n-l - .2467

+ Sign indicates reflex decay

- sign indicates ao reflex decay

Table-1(d) Amount of reflex decay on presentation of tone repeatedly at S dBSL, 4KHz measured in cms

Subjects	Probe ear	ART at 4 KHz	Amount of decay
I	Left	110 dB	+ 0.09
2	Left	160 dB	+ 0.11
3	Right	120 dB	+ 0.14
4	Right	120 dB	+ 0.16
5	Right	115 dB	+ 0.22

M = .144

σ n-1 = .0878

+ Sign indicates reflex decay

- Sign indicates no reflex decay

Table-2(a): Amount of reflex decay on presentation of tone repeatedly at 10 dBSL, 500HZ measured in cms.

Subjects	Probe ear	ART at 500Hz	Amount of decay
1	Left	90 dB	+ 0.26
2	Left	90 dB	- 0.04
3	Right	90 dB	+ 0.02
4	Right	90 dB	- 0.04

M = .05

σ n-1 = .1457

+ Sign indicates reflex decay

- Sign indicates no reflex decay

Table-2(b) Amount of reflex decay on presentation of tone, **repeatedly at 10 dBSL, 1KHz measured in cms.**

Subjects	**Prob ear**	ART at 1KHz	**Amount of decay**
1	Left	90 dB	**+ 0.52**
2	Left	90 dB	**+ 0.52**
3	Right	90 dB	**0**
4	Right	**90 dB**	0
			M - 0.26
			σ n-1 = **0.26**

+ sign indicates reflex decay

Table-2(c) Amount of reflex decay on presentation of tone, repeatedly at 10 dBSL, 2KHz measured in cms.

Subjects	Probe ear	ART at 2KHz	Amount of decay
1	Left	90 dB	+ 0.28
2	Left	90 dB	+ 0.48
3	Right	90 dB	+ 0.07
4	Right	90 dB	+ 0.03
			M = .215
			σ n-l = .2079

+ sign indicates reflex decay

Table-2(d) Amount of reflex decay on presentation of tone, repeated at 10 dBSL, 4 KHz measured in cms.

Subjects	Probe ear	ART at 4 KHz	Amount of decay
1	Left	90 dB	+ 0.52
2	Left	90 dB	+ 0.26
3	Right	90 dB	+ 0.51
4	Right	90 dB	+ 0.16

M = .362

n-l = .1808

+ Sign indicates reflex decay

Table-3(a) Amount of reflex decay on presentation of tone, repeatedly at 15 dBSL, 500Hz measured in cms.

Subjects	Probe ear	ART at 500Hz	Amount of decay
1	Left	95 dB	+ 0.02
2	Left	90 dB	+ 0.05
3	Right	90 dB	+ 0.12
4	Right	100 dB	0
5	Right	100 dB	+ 0.1

M = .058

n-l = .051

+ sign indicates reflex decay

Table-3(b) Amount of reflex decay on presentation of tone, repatedly at 15 dBSL, 1 KHz measured in cms.

Subjects	Probe ear	ART at 1KHz	Amount of decay
1	Left	90 dB	+ 0.09
2	Left	95 dB	- 0.01
3	Right	90 dB	+ 0.13
4	Right	100 dB	- 0.02
5	Right	100 dB	+ 0.15

M = 0.068

n-1 = .858

+ Sign indicates reflex decay

- Sign indicates no reflex decay

Table-3(c) Amount of reflex decay on presentation of tone, repeatedly at 15 dBSL, 2KHz measured in cms.

Subjects	Probe ear	ART at 2 KHz	Amount of decay
1	Left	90 dB	+ 0.54
2	Left	90 dB	0
3	Right	100 dB	+ 0.1
4	Right	100 dB	+ 0.15
5	Right	90 dB	- 0.02

M - 0.154

n-l = .2396

+ **sign** **i**ndicates reflex **decay**

- sign indicates no reflex decay

Table-3(a) Amount of reflex decay on presentation of tone, repeatedly at 15 dBSL, 4KHz measured in cms.

Subjects	Probe ear	ART at 4KHz	Amount of decay
1	Left	105 as	+ 9.44
2	Left	100 dB	+ 0.22
3	Right	95 dB	+ 0.16
4	Right	100 SB	+ 0.2
5	Right	90 dB	+ 0.07

M = .218

σ n-l = .1368

+ sign indicates reflex decay

Table-4(a) Amount of reflex decay on presentation of tone, repeatedly at 20 dBSL, 500Hz measured in cms.

Subjects	Probe ear	ART at 500 Hz	Amount of decay
1	Left	90 dB	+ 0.13
2	Left	90 dB	+ 0.97
3	Left	90 dB	+ 0.02
4	Left	90 dB	+ 0.04
5	Right	90 dB	+ 0.09
6	Right	90 dB	+ 0.1
7	Right	100 dB	+ 0.04
8	Right	90 dB	0
9	Right	90 dB	+ 0.06

$M = 0.061$

$\sigma_{n-1} = .0410$

+ Sign indicates reflex decay.

Table-4(b) Amount of reflex decay on presentation of tone, repeatedly at 20 dBSL, 1KHz measured in cms.

Subjects	Probe ear	AST at 1KHz	Amount of decay
1	Left	90 dB	+ 0.17
2	Left	96 dB	+ 0.26
3	Left	100 dB	0
4	Left	90 dB	0
5	Right	90 dB	+ 0.2
6	Right	90 dB	0
7	Right	90 dB	0
8	Right	100 dB	+ 0.07
9	Right	90 dB	

M = **.084**

σ n-1 = .1005

+ Sign indicated reflex decay

Table-4(c) Amount of reflex decay on presentation of tone, repeatedly at 20 dBSL, 2KHz measured in cms.

Subjects	Probe ear	ART at 2 KHz	Amount of decay
1	Left	100 dB	+ 0.21
2	Left	90 dB	+ 0.16
3	Left	90 dB	+ 0.42
4	Left	90 dB	+ 0.32
5	Right	65 dB	+ 0.04
6	Right	100 dB	+ 0.28
7	Right	100 dB	+ 0.4
8	Right	105 dB	+ 0.14
9	Right	90 dB	+ 0.06

$M = .225$

$\sigma_{n-1} = .1386$

+ Sign indicates reflex decay

Table-4(d) Amount of reflex decay on presentation of tone, repeatedly at 20 dBSL, 4KHz measured in cms.

Subjects	Probe ear	ART at 4 KHz	Amount of decay
1	Left	105 dB	+ 0.18
2	Left	90 dB	+ 0.16
3	Left	95 dB	+ 0.52
4	Left	100 dB	+ 0.5
5	Right	80 dB	+ 0.54
6	Right	105 dB	+ 0.42
7	Right	100 dB	+ 0.3
8	Right	105 dB	+ 0.12
9	Right	105 dB	+ 0.12
			M = .3177
			σ n-1 = .1790

+ Sign indicates reflex decay

Table-5 Reflex magnitude at 2 KHz, 20 dBSL in subjects who do not show any noticeable reflex Decay (Mean reflex decay for those subjects is .036)

Subject	Probe ear	ART at 2KHs	Magnitude of AR at 2 KHz	Magnitude of AR at 20dBSL	Magnitude of AR
1	Right	85 dB	1.2	1.4	.2
2	Right	90 dB	.7	.9	. 2
3	Right	100 dB	1	1	0
4	Right	90 dB	.7	.8	.1
5	Left	90 dB	.3	.7	.4

M = .18

σ n-1 = .1483

Table-6 Reflex magnitude at 2KHz, 20 dBSL in subjects who show reflex decay (Mean reflex decay for these subject .275)

Subjects	Probe ear	ART at 2KHz	Magnitude of AR at 2KHz	Magnitude of AR at 20 dBSL	Magnitude of AR
1	Right	100 dB	.2	1.0	0.7
2	Right	100 dB	.5	1.3	.8
3	Right	105 dB	.4	1.1	.7
4	Left	90 dB	.7	1.1	.4
5	Left	100 dB	.4	1.5	1.1
6	Left	90 dB	.5	1.3	.8
7	Left	90 dB	.3	.7	.4

M = 0.7

σ n-1 = .2449

SUMMARY AND CONSCLUSION

The present study was designed to find the susceptibility of normal hearing listeners to noise induced hearing loss (permanent threshold shift). This "Repeated reflex decay testing" is among the objective indices of susceptibility to auditory fatigue and can be considered as a more reliable measure than psychoacoustic tests for susceptibility to noise induced hearing loss.

This study also gives information about the frequency and intensity level of the stimulus eliciting the reflex.

This study was carried eat in a sound treated room at Audiology Department of All India institute of Speech & Hearing, Mysore. Twenty three (12 females and 11 males) normal hearing subjects were tested. The subjects were divided into four groups to measure this "Repeated Reflex Decay" at four different sensation levels and at four different frequencies. Madsen Z0174 Immittance Audiometer was used. The experimental testing was carried out in two stages.

Stage-1: The acoustic reflex thresholds (contralateral stimulation)using T & R setting of the instrument were measured.

Further "Repeated Reflex Decay" was observed at single frequencies by presenting the acoustic stimulus repeatedly (five presentations). Selection of the frequencies for any experimental session was random. Two frequencies were selected for one experimental session and two for another experimental station. This was done to control the interactive effect of different frequencies so that decaying process at any one frequency does not affect the decay at the another frequency.

Stage-2: It consisted of procedure to determine the averaged magnitude of the acoustic reflex at 2KHz in subjects selected from 15 dBSL and 20 dBSL group.

The following conclusions have been drawn:

1) The subjects who showed "Repeated Reflex Decay" also showed greater magnitude of acoustic reflex and the subjects who did not show reflex decay showed less magnitude of acoustic reflex.

2) Anne zachariah (1980) concluded that subjects who show greater magnitude of reflex are susceptible to noise induced hearing loss. Since the subjects who show greater magnitude of reflex also show decay for repeated reflex decay test, it may be concluded that the subjects who show decay for repeated reflex decay test can be considered susceptible to noise induced hearing loss,

3) Repeated reflex decay is greater at high frequencies than at low frequencies, indicating that reflex decay is a frequency dependent phenomenon.

4) Greater amount of "Repeated Reflex Decay" is obtained at higher sensation levels than at lower sensation levels.

Recommendations:

1) It would be worthwhile if the present study is carried out on a large sample of normal subjects.

2) To establish the relationship between "Repeated Reflex Decay" values and TTS on Indian population.

3) A study may be undertaken to verify whether "Repeated Reflex Decay" high TTS, lower ART and "greater magnitude of reflex" go together in a subject or not.

4) It weald be worthwhile to have a longitudinal study on industrial workers, by making use of all the above mentioned suseptibility tests to establish the validity.

Anderson, H., Barr, B., aad Wedeaberg, E., (1969). Intra-aural reflexes in retrocochlear lesions. in Hamburger, C., and wersall, J. (eds), Disorders of the skull Base Region, 10th Nobel symposium, Pp 49-55 (Almquist and Wiksell, Stockholm).

Aaderson, H., B. Barr, and Wedenberg, w., (1970). Early diagnosis of VIIIth nerve tumours by acoustic reflex tests. Acta Oto.Laryngol. 262, 232-237.

Anderson (1976). Intra tympanic muscles. In Scientific foundations of Otolaryngology (Hinchcliffe, R. and Harrison, D eds) Pp.275-280 (Letter Press Great Britian).

Anne Summerfield, Aram Glorig, and D.E.Wheeler. "Is there a suitable Industrial Test of susceptibility to Noise" - induced hearing loss? Noise Control 4 (Ja.1958). 40-46; 54.

Baust, w and Berlucchi, G. (1964). Arch.ital.Biol., 102, 686

Bishnoi, G.D., - The distribution of individual susceptibility for noise induced hearing loss as measured using Wilson's test in Indian population - a dissertation for M.s., submitted to the University of Mysore (1975).

Borg et.al., (1970). Fatigue and recovery of the human stapadius reflex in industrial noise. JASA 64(3), 1979, 846-848.

Borg, E. (1973). On the neuronal organization of the acoustic middle ear reflex. A physiological and anatomical study. Brain Res. 49, 101-123.

Borg, E., and Odman, B., (1979). Decay and recovery of the acoustic stapedius reflex in humans. Acta Oto.Lary. 87: 421-428.

Borg, E., Nilsson, R., and Liden, G., Fatigue and recovery of the human stapedius reflex in industrial noise. JASA 65(3), 1979, 846-848.

Brasher, P.P., et al., - Middle ear muscle activity and TTS — International Audiology, Vol.8, 1969 P.479-584.

Borg: A quantitative study of the effect of the acoustic stapedius reflex on sound transmission through the middle ear of man. Acta-to-Laryngol., Vol.66, 1968, Pp.461-472.

Coles, R.R.A., - Middle ear muscle activity as a possible index of susceptibility to TTS - Sound Vol.3, 1969, P 72-74.

Djupesland, G., and G.Flottrop and F.Winther, (1967). Size and duration of acoustically elicited impedance changes in man. Acta Oto-laryngol. Suppl. 224, 220-228.

Djupseland (1979) - The acoustic reflex. In clinical impedance audiometry (Jerger and Northern ed) Pp.65-82. American Electromedics Corporation.

Eldredge, E., and J.Miller, (1969). Acceptable noise exposure. Damage risk criteria in ward and Fricke, eds. Noise as a public Health Hazard. ASHA Report-4, 110-120.

Fowler, C., wilson, R., (1984). Adaptation of Acoustic reflex. Bar and Hearing 5, 281-288.

Gershardt, et al., (1980). Acoustic Reflex Decay in Chinchillas during a long term exposure to noise. Ear and Hearing 1, 33-37.

Gerhardt, K.J., Hepler, E.L.Jr. Acoustic reflex activity and behavioral thresholds following exposure to noise. J.Acoust,Soc,Am., 1983, Jul., 74(1): 109-14.

Givens, G., and M.Seidemann, (1979). A systematic investigation of measurement parameters of acoustic - reflex adaptation. JSHD. 44, 534-542.

Hawhins, J (1973). Comparative Otopathology aging, noise and ototoxic drugs. Adv. Otol-Rhinol.Laryngol. 20, 125-141.

Hilding, D (1960). The intratympenic muscle reflex as a protective mechanism against loud impulsive noise. Ann. Otol.Rhinol.Laryngol. 69, 51-60.

Hirsch, A., and H.Anderson (1960). Elevated stapedius reflex threshold and pathologic reflex decay. Acta Oto-laryngol. Suppl. 368, 1-28.

Hugelin, A., Dumont, 3., and Paillas, N (1960b), Science, 131, 137.

Jagadiah, A. Study of the relationship between the Acoustic Reflex threshold and threshold of octave masking-Dissertation to Mysore University - 1982.

Jerger, J., (1970). Clinical expariences with impedance audiometry. Archieves of Otolaryngology, 92: 311-314.

Johansson et al., (1967). Acoustic reflex as a test of individual susceptibility to noise. Acta Otolaryngol. 64, 256.

Kamakshi. A study of the relationship between "Fatigability of Acoustic Reflex" and temporary threshold shift.

Karlovich, R., and J.Wiley. (1974). Spectral and temporal parameters of contralateral signals altering temporary threshold shifts, JSHR, 17, 41-50.

Karlovich, R.s. at al - The acouatic reflex and temporary threshold shift: Temporal characteristics, JSHR, 1977, 20, 565-573.

Metz, O. (1946). The acoustic impedance measured on normal and pathological ears. Acta Ota-laryngol. Suppl. 63, 1-254.

Matz, O. (1951). Studies on the contraction of the tympanic muscle as indicated by changes in the impedance of the ear. Acta oto-laryngol. 39, 397-405.

Miyakita et al., - study on the magnitude threshold and latency of aural reflex. DSH Abstract, Vol.19, 1979, p.418.

Porter, T. Normative otoadmittance values for three populations. J.Aud.Res. 12, 1972, p.1 (1976).

Rosenhall, U., O.Liden, and E.Nilsson (1979). Stapedius reflex decay in normal hairing subjects. J.Am.Aud.Soc. 4, 157-163.

Tonndorf, J. (1976). Relationship between the transmission characteristics of the conductive system and noise induced hearing loss. In effects of noise on hearing (ed. D.Handeraon, R.P, Hamernick, D.S., Dosanih and J.H.Milis), P.159, Raven Press, New York.

Ward, W.D. Psychoacoustics in audiometry. Principles and practice Gloring, A (Ed), 1965.

Ward, W., and A.Duvall. (1971). Behavioral and ultrastructural correlates of acoustic trauma. Ann.Otol.Rhin. laryngol.80, 981-896.

Ward, W. (1973). Adaptation and fatigue. Pp.301-314 in J.Jerger, ed. Modern Developments in Audiology (Ed.2). Academic Press, Inc. New York.

Wiley, T.L., and R.S.Karlovich, (1975). Acoustic reflex response to sustained signals. JSHR, 18, 148-157.

Wiley, T.L., and Karlovich - Acoustic reflex dynamics for pulsed signals, JSHR, Vol.21, 1978, 295-308.

Wilson, R.H., J.F., Steckler, H.C. Jones, and R.H., Margolis(1978) Adaptation of the Acoustic riflex. J.Acoust.Soc.Am. 64, 782-791.

Wilson, R.H., and L.M.McBride (1978) Threshold and growth of the acoustic reflex. J.Acoust.Soc.Am., 63, 147-154.

Zachariah Anne - A study of the relationship between Acoustic reflex threshold and TTS. Dissertation to Mysore University - 1980.

Zakrisson and Borg, E. Stapedius reflex and auditory fatigue. Audiology, Vol.13, 1974, P.231-235.

Zakrisson, J.E. The role of the stapedius reflex in post stimulatory auditory fatigue. Acta-Oto-Laryngol. Vol.79, 1975, P 1 to 10.

* Original article not referred.