

***THE EFFECT OF AUDITORY FRTIGUE
ON
MASING LEVEL DIFFERENCE (MLD)***

Register No 8503

**An Independent project submitted as part fufilment for
First year M.Sc. (Speech and Hearing)
to the University of Mysore.**

All India Institute of Speech & Hearing
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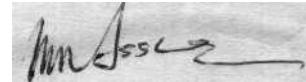
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CERTIFICATE

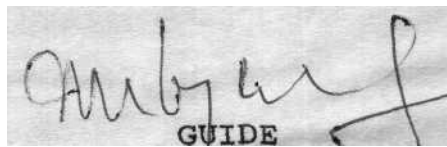
This is to certify that the Independent Project entitled "THE EFFECT OF AUDITORY FATIGUE ON MASKING LEVEL DIFFERENCE (MLD)" is the bonafide work on part fulfilment for the Degree of Master of Science (Speech and Hearing) of the student with Register No. 5803



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CERTIFICATE

This is to certify that the Independent Project entitled "THE EFFECT OF AUDITORY FATIGUE ON MASKING LEVEL DIFFERENCE (MLD)" has been prepared under my supervision and guidance.



A handwritten signature in black ink, appearing to read 'M.N. Vyasamurthy', is written over a rectangular stamp. The stamp contains the word 'GUIDE' in a bold, sans-serif font.

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DECLARATION

I hereby declare that this Independent Project entitled " THE EFFECT OF AUDITORY FATIGUE ON MASKING LEVEL DIFFERENCE (MLD) " is the result of my own study under the guidance of Dr.M.N.VYASAMURTHY, Department of Audiology, All India institute of Speech and Hearing, Mysore, and has not been submitted earlier at any University for any other diploma or degree.

Mysore

Date: April 1986

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

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INTRODUCTION

Auditory fatigue is one of the terms used to describe a temporary change in threshold sensitivity following exposure to another auditory stimulus (Ward, 1963).

Empirical investigation of the locus of auditory fatigue have often been concerned with the question of whether fatigue producing mechanism lies within the cochlear or central to it.

In order to answer the question of origin of auditory fatigue Van Békésy (1929) in his experiment found that fatiguing one ear has caused no decrease in the response of the contralateral ear and hence concluded that auditory fatigue is a peripheral phenomenon (cited by Wernick and Tobias, 1963).

Rawden-Smith (1936) conducted a study on similar lines of Van Békésy (1929) but found that responses of the contralateral ear was reduced when one ear was stimulated.

Other information on the locus of auditory fatigue can be drawn from studies of the electrophysiological activity within the cochlea.

The amplitude of cochlear microphonics was found to be reduced in noise exposed subjects (Legoux and Pierson, 1981; Elderedge et al. 1973; Elderedge and Covell, 1958; Benitez et al., 1972).

Legouix and Pierson (1981) have also observed the alteration in input - output function in the cochlear microphonics after the exposure to the noise.

"Johnstone and Sellick (1972) reported that Endocochlear Potential (EP) could be potentiated following exposure to an intense pure tone"(cited by Legouix and Pierson, 1981).

These studies indicate that the action of the auditory fatigue must take place prior to the initiation of neural activity, most probably the structures directly responsible for the production of these potentials.

Davis et al., (1953) suggest that Compound Action Potential (CAP) too provides a good index of cochlear changes.

smoorenburg and Van Hensden (1979) found that the latency of CAP is reduced at suprathreshold level but not at threshold level after noise exposure.

In a similar study sohmer and Pratt (1975) and salvi et al., (1983) have observed more of peripheral auditory mechanism involvement in the process of auditory fatigue.

The other studies indicating cochlear origin (the presence of recruitment) following the fatiguing stimulus are: Davis et al., (1950); Rawnsley (1953); Reudi (1959)(cited by Wernick and Tobias, 1963).

Recent neuro-physiological findings have generated a renewed interest in possible central factors operating in the process of auditory fatigue. In particular, the demonstration of a subcortical inhibiting system exerting its action in the cochlear via the efferent auditory pathways has provided a new basis for looking at the fatiguing mechanism.

Guiot (1969) showed that stimulation of left ear had a definite influence upon the TTS measured on the right ear. He explains this by disinhibition of central inhibitory process during auditory fatigue.

Nazneen (1984) has observed that TTS produced in right ear is significantly greater than in left ear and she further reports that this ear difference in TTS indicates that the action of the efferent auditory system during binaural stimulation is more intense in right ear than in the left ear.

Hernandez-Peon (1956) while recording in the cochlear nuclei, noticed a decrease in the evoked potentials when the Cat was presented with stimulation to an alternate modality, such as exposure to the smell of fish. Thus he indicates that "attentiveness" was a factor that called into play a selective inhibitory mechanism operating at a locus peripheral to the higher centers and possibly within the cochlea.

Wernick and Tobias (1963) in their study exposed subjects to 4000Hz pure tone at 40 dB SL or 90 dB SL for 3 minutes under conditions of (a) Mental task and (b) Revire. Subjects consistently showed greater TTS and longer recovery time when the fatiguing stimulus was present in the mental task condition.

These results indicate the involvement of the central factors in auditory fatigue.

Similar findings were also reported by Capps and Collins (1965).

On the other hand, Ward and sweet (1963) exposed their subjects to a 4KHz tone at 100 dB SPL for 3 minutes and under two conditions (a) Sitting quietly (b) adding columns of the figures. They observed no significant difference between the TTS measured at any time after exposure. They concluded that the efferent system need not be invoked to account for any aspect of auditory fatigue.

Bell and Stern (1964) also found no significant difference between TTS measured in subjects exposed to 4KHz tones at 50 dB SPL and 100 dB SPL for 3 minutes under the condition of (a) long division (b) light tracking (c) reverie.

Riach and Sheosh (1964) conducted their study on similar lines of Wernick and Tobias (1963) and reported that the TTS

difference found in subjects under mental task and recovery during fatigue is just a chance factor.

Fialkowska et al., (1983); Kylen et al., (1977) report that the noise induced auditory fatigue possibly alters the synaptic mechanism within the cochlea and is then transferred to the auditory pathways.

Babigian et al., (1975); Rosenblith et al., (1950) based on their studies on evoked potentials during fatigue and during recovery from fatigue conclude that there are central effects produced by exposure to intense sounds and that the observed results are not simply a reflection of a fatigue occurring at the cochlea.

Morest et al., (1979) suggests a selective susceptibility of different auditory pathways in the CNS to acoustic over stimulation.

Morest and Bhone (1983) in their study on noise exposed Chinchillas observed that both peripheral and central degeneration, the latter included mainly degeneration of cochlear nucleus and transneural degeneration of axonal endings in Superior Olivary Complex and Inferior Colliculus.

Again these studies indicate the involvement of structures of the auditory system beyond cochlea in the process of auditory fatigue.

So the question of involvement of retrocochlear structures (central factors) in the auditory fatigue mechanism is not completely evidenced.

On the other hand, Masking Level Difference (MLD), the one of the psychoacoustic phenomena is proved to have its origin in Superior Olivary Complex (SOC), a retrocochlear structure (Lynn et al., 1985; Hanely, et al., 1983; Vyasamurthy et al., 1985).

MLD is the difference between the binaural masked thresholds obtained under two conditions viz, homophasic (NoSo-the Noise and signal are in phase at the two ears) and the antiphasic condition (NoSII or NII So - the phase of either signal or noise is reversed at two ears). MLD is usually expressed in dB.

Need for the present study:

Several studies mentioned earlier clearly indicate that the question of whether the structures beyond the cochlea are involved or not in the process of auditory fatigue is completely not yet answered.

So the present study is designed to findout whether auditory fatigue has any effect on the MLD values measured for pure tones of 500Hz is both NoSII and NIISo conditions.

The findings of this study hence provides some evidence to support whether structure beyond the cochlea i.e. SOC is involved in the process of auditory fatigue or not.

HYPOTHESIS:

1. There is no significant difference between pulsed thresholds at 1KHz (test frequency) before and after fatigue.
2. There is no significant difference between MLD values (NoSII) obtained before and after fatigue.
3. There is no significant difference between MLD values (NIISo) obtained before and after fatigue.

Limitations:

1. The MLD values are obtained using pure tone stimulus of 500 Hz frequency only.
2. The sample size was limited to 10 normal hearing adults subjects.
3. The age range was limited.

Definition of the terms used:

Temporary Threshold Shift (TTS): Refers to an elevation in the threshold of hearing which recovers gradually following the noise exposure.

Masking Level Difference (MLD): Difference between binaural masked thresholds obtained under two conditions viz. homophasic (NoSo – The noise and the signal are in phase at the two ears)

and antiphasic (NoSII and NIISo - the phase of either the signal or noise is reversed (180°) at the two ears).

Fatiguing Stimulus: The acoustic stimulus used to produce auditory shift in the threshold.

Test Frequency: The frequency at which the thresholds were determined after the ear was exposed to fatiguing stimulus.

TTS₂: Temporary threshold shift after the recovery time of 2 minutes.

REVIEW OF LITERATURE

Auditory fatigue is one of the number of terms used to describe a temporary change (usually but not always, a decrease) in threshold sensitivity following exposure to another auditory stimulus. It may be called a "line dead" situation.

Despite, the TTS or post stimulatory fatigue being most studied after effect of auditory stimulation, in auditory fatigue the unsolved problems greatly out number the established facts.

Empirical investigations of the locus of auditory fatigue have often been concerned with the question of whether the fatigue producing mechanism lies within the cochlea or central to it.

The present Review of Literature, related directly to the study, is discussed under the following headings:

- I Auditory fatigue and recruitment.
- II Effects of auditory fatigue on temporal summation.
- III Effects of Noise on cochlear potentials.
- IV Effects of auditory fatigue on 8th nerve activity.
- V Central Factors and auditory fatigue.
- VI structural changes in the auditory system as a result of acoustic over stimulation.
- VII MLD as a soc phenomena.

I. Auditory Fatigue and Recruitment:

One of the studies indicating cochlear origin of auditory fatigue/the presence of recruitment following the presentation of fatiguing stimulus.

Davis, et al., (1950) found a clear indication of recruitment following a high level of stimulation.

Harris and Rawnsley (1953) using the technique of binaural loudness balance, were able to show recruitment for a 1KHz tone at SLs of 10, 20 and 30 dB.

"Epstein and Schubert (1957) using a fatiguing stimulus at 4KHz for duration of 3 minutes, report the presence of recruitment with stimulating intensities of 70 to 100 dB SL. They also found that the degree of recruitment, increased as the intensity of fatiguing tone increased". (Wernick and Tobias, 1963).

II. Effects of auditory fatigue on temporal summation:

It is well known that acoustic trauma modifies the process of auditory temporal summation, especially the relationship between the threshold of a tone and its duration.

Handerson (1969) measured the auditory sensitivity of 2 Chinchillas by using signals of 2000Hz pure tones of 25, 50, 200, 400 and 750 m.secs. Subsequently the Chinchillas were exposed for 3 hours to a 105 dB SPL Octave band of noise centred at 2 KHz.

When the Chinchillas were in the state of TTS, the 12 dB threshold difference between 25 and 750 msec. signals decreased markedly. During the process of recovery, the difference gradually returned to the preexposure magnitude.

He also suggests that when the auditory system is in a state of noise induced TTS, it appears that the time constant of integration is reduced.

Mills et al., (1970) also reported reduction in the time constant for temporal integration at the fatiguing frequency, after fatigue in a subject who was exposed to thermal noise centered at 500Hz for 48 hours at 81.5 dB SPL and second time for 29.5 hours at 92.5 dB SPL.

Jerger (1955) exposed 12 human observers for a thermal noise at 110 dB SPL and observed that pre exposure difference in the threshold of 14.1 dB between 5 and 500 msec. signals at 4 KHz pure tones being decreased to 6.9 dB 4 mins. after the exposure.

Wright (1968) explains the decrease in temporal summation during TTS as hearing loss of cochlear origin causes an abnormally rapid decay in the output of the cochlea at intensities near the threshold.

"Such findings are paradoxical because acoustic overstimulation damages the cochlea and decreases temporal summation.

yet it does not seem to significantly alter the temporal firing patterns of peripheral neurons during TTS or PTS (Handerson(1976); Salvi (1976)(Quoted by Salvi et al., 1983).

Salvi et al., (1983) also observed that a comparison of PST histograms to tone bursts of normal and noise treated units has failed to provide support for an abnormally rapid neural decay in noise treated animals.

Hence, they suggest that perhaps the decrease in temporal summation is the result of some other changes in the neural code, such as change in the bandwidth of spatial summation at the periphery or at higher auditory centers.

III. Effects of noise on cochlear potentials:

Other information on the locus of auditory fatigue can be drawn from studies of electrical activity within the cochlea.

a) Changes in cochlear microphonics (CM):

i) Different types of CM reduction:

Legoux and Pierson (1981) report that the depression of CM after noise exposure is a clear sign of traumatic effect on the sensory cells and that the deterioration of these cells may result from various mechanisms that follow different time courses and some are reversible and others are not.

Benitez et al., (1972) have measured CM in the three cochlear turns of Chinchilla after fatiguing by 500Hz tone. They found that third turn was more affected rather than the other turns.

ii) Modification of CM intensity function and produced by intense noise exposure:

Tondorf and Brogan (1952) reported a greater reduction of the response at high levels so that the curve was truncated. In some particular cases Lawrence (1958) observed a nonmonotonic input-output function following exposure. In plotting the characteristic of transfer they noted that the curve was rapidly modified and displayed a greater symmetry. (Legoux and Pierson, 1978; Legoux et al., 1980).

Legoux and Pierson (1981) also suggests that the changes in the input-output function may lead to some interpretation about the nature of the lesion in the cochlea. This function shifted toward a lower sensitivity, could reflect some impairment in the conduction of the acoustic energy within the cochlea.

iii) CM as an Index for localization of cochlear damage:

Legoux and Pierson (1981) write that there are some investigations which M&B have shown a clear correlation between the decrease in the magnitude of CM response and the number of hair cells that are damaged (eg. Eldredge, et al., 1973).

Benitez et al., (1972) exposed Chinchillas to an octave band of noise centered at 500Hz and at 95 dB SPL for 48 hours and observed a decrease of the intensity function that was maximum for the third turn, the reduction in CM amplitude was graded and less severe from apex to base.

Eldredge and Covell (1958) found that narrow band noises were more effective in producing CM reduction than pure tones presented at an SPL equal to the overall SPL of the noise.

iv) Effect of the time characteristics of the exposure:

Legoux and Pierson (1981) reported that the CM depression produced by an intense sound seems to be directly related to SPL and to the logarithm of the duration of the exposure.

Eldredge et al., (1959, 1961) have suggested that the reduction in CM was the same as long as the sound contains the same energy.

Legoux and Pierson (1981) reveal that it is clear that the trade between intensity and duration can be justified only if the same mechanism alters the hair cells at all intensities, but this does not appear to be the case. The cochlear mechanisms are highly nonlinear in the intensity domain and different processes might occur when the stimulation level is modified. Further they say that a critical intensity must be exceeded to produce a loss at any duration and at very short durations, the

influence of intensity seems to be greater than that of duration. After a certain duration, they state that the CM reduction induced by the noise is maximum and does not grow with time and that this effect seems well correlated with behavioral TTS.

(b) Changes in summing potentials (SP):

Durrant (1976) reported that SP DIF (recorded by differential electrodes) is much more sensitive to effect of noise than the CM and AP, which suggest that marked changes can be produced in the SP even in cases where the CM is hardly affected. He report further that there is a trend towards less depression in the SP from the lower to the upper turns.

Legouix and Pierson (1981) has reported that the cochlea which show the largest negative SP are more susceptible to fatigue.

They further report that when SP and CM are depressed by noise, it is no longer possible to create additional fatigue, at least with the same stimulus as was used for the initial exposure. This stage seems to correspond to the asymptotic threshold shift. When negative SP is depressed or has disappeared after exposure fatiguability is minimal.

IV. Effect of auditory fatigue on 8th nerve activity:

Salvi et al., (1983) studied TTS produced in Chinchillas exposed to octave band noise centered at 500Hz at 95 dB SPL for

5 days and they also measured the auditory nerve activity of the Chinchillas. The threshold shifts of the fibers were approximately 35 to 65 dB: these values were equal to or slightly greater than those measured behaviorally. Most units had broad V-shaped turning curves due to a greater loss in sensitivity near the (CF) than in the low frequency tail. In 17% of the units, the thresholds were actually lower in the tail than at the CF, so that the tuning curves were 'W' shaped. The latencies of the fibers were within normal limits in terms of absolute intensity, but shorter than normal in terms of intensity relative to threshold. Other measures such as spontaneous discharge rate, the discharge rate - intensity function and the firing pattern to tone bursts at CF appeared to be normal.

Fialkowska et al., (1983) in their study on the effect of TTS on ECoG and ABR wave forms observed the increase in the latency of III and V peaks of ABR, determined by the increase in the latency of Peak-1 and Auditory Compound Action Potentials (ACAP) and unaltered intervals between successive peaks. They also reported an elevation of thresholds of the wave forms.

They conclude that the noise induced auditory fatigue possibly alters the synaptic mechanism within the cochlea and is then transferred to the auditory pathways.

Similar effects on ECoG and ABR by TTS has been reported by Sohmer and Pratt (1975), Kylea et al., (1977): Pratt and Sohmer (1978).

Salvi et al., (1978) in their study on Chinchillas during Asymptotic threshold shift (ATS) indicated that intense sound reduced the sensitivity, frequency selectivity and spontaneous activity of the units in the cochlear nucleus and that the damage to the cochlea modified the activity of neurons throughout the auditory pathways. (cited by Fialkowska et al., 1983).

Benitez et al., (1972) in their study on electrophysiological correlates of TTS, exposed 15 Chinchillas to 95 dB SPL of octave band noise centered at 500 Hz to 48 hours observed that endocochlear potentials were unchanged, and shifts of sensitivity for CM in the 2nd and 3rd turns showed closest numerical correspondings to behavioral TTS. Loss of sensitivity of action potentials was greater. Changes in visual detection level for the Average Evoked Response were consistent with behavioral TTS. They concluded that pathologic-physiology associated with TTS is most probably peripheral and possibly because of the disorder in the mechano-electric modulation by hair cells, plus failure to synchronize primary neural responses.

But Babigian et al., (1975) in their study on "Central auditory fatigue" measured the CM, neural (N1-N2) potentials and IC (Inferior colliculus) potentials from the Kangaroo rats who were exposed to two different fatiguing stimuli.

They observed a 10-20 dB depression in IC response and CM amplitude being unaltered from Pre-exposure level when the exposure stimulus was puretones of 1.6 KHz at 95 dB SPL for 3 minutes.

After exposure to 1.6 KHz at 85 dB SPL for 2 minutes. They observed the IC response decrease by 6 to 8 dB, while N1-N2 response of the cochlea changed only slightly.

They further report that whenever the amplitude shift in both the IC and cochlear response occurred, they exhibited different recovery rates.

For the same exposure, the neural discharge frequency was reduced by 60-90%, while the cochlear potentials for the same exposure were only slightly affected, if at all.

They also observed significant difference in the response of excitatory-Inhibitory(E.I) collicular neurons before and after fatigue.

The reduction in the amplitude of IC response continued for 30 minutes after the cessation of the sound exposure. Paralleling these changes there was a post-exposure amplitude decrease of the cochlear potentials.

They conclude that these results show a central affect of auditory fatigue which is not entirely a reflection of peripheral sensitivity changes and support the general conclusion that central neurons are affected by excessive sound stimulation.

V. Central Factors and Auditory Fatigue:

The consensus of authors have been almost unanimous that auditory fatigue is of peripheral (cochlear) origin and is based on repeatedly reported fact that very loud sounds produce histologically verifiable cochlear damage.

But Elloit (1961) observed that changes in auditory sensitivity unaccompanied by hair cells loss in cats after acoustic overstimulation. Similar observations were made by Hunter-Duvar (1971).

Ward et al., (1972) found that animals with only a small area of normal hair cells in the apex can respond normally to very weak high frequency tones.

A similar lack of correspondence between audiogram and pathology has been reported by Benitez et al., (1962) and Bredberg (1968). Bredberg (1968) described normal appearing hair cells associated with PTS, as well as a normal threshold despite a 35% loss of Inner Hair Cells (IHC) and 45% loss of Outer Hair Cells (OHC) in an 81 year old man.

These disparate findings suggest that central factors, in addition to peripheral ones may play an important role in auditory fatigue.

Recent neurophysiological findings have generated a reviewed interest in possible central factors operating in auditory fatigue.

In particular, the demonstration of a subcortical inhibiting system exerting its action in the cochlea via the efferent auditory pathways has provided a new basis for looking at the fatiguing mechanisms. "In 1956, Galanbas electrically stimulated the roof of the medulla of a cat and observed a simultaneous reduction in the potentials evoked by clicks at the cochlear nuclei". (cited by Wernick and Tobias, 1963).

Hernandez-Peon (1956) recording from the cochlear nuclei, noticed a decrease in the evoked potentials when the cat was presented with stimulation to an alternate modality, such as exposure to the smell of fish. Thus he indicated that attentive-
neas was a factor that called into play in selective inhibitory nachanism operating at a locus peripheral to the higher centers and possibly within the cochlea itself.

Wernick and Tobias (1963) in their study on central factors in pure tone auditory fatigue exposed 20 subject to 4KHz pure tone at 40 or 90 dB SL for 3 minutes under conditions of (a) Mental task or (b) Revire. They also used an experimental conditions viz. Experimental condition-1: Arithmetic task during the present of fatiguing stimulus.
Experimental condition-ii: Revire with fatiguing stimulus.
Control condition-1: -Arithmetic task with no fatiguing stimulus.
Control condition-II:-Revire with no fatiguing stimulus.

Subjects showed consistently greater TTS and larger recovery time when the fatiguing stimulus was present in the mental task condition i.e.

(i) At low level fatigue mean TTS_{10 sec} was 6.4 and 3.5 dB respectively in mental task and recovery condition. Mean recovery time was 90 seconds and 36 seconds respectively in mental task and recovery.

(ii) At 90 dB fatiguing stimulus mean TTS_{10 sees.} was 43.3 dB and 34.1 dB with mean recovering time of 44 minutes and 34 minutes mental task and recovery conditions respectively.

These results indicate the involvement of central factor in auditory fatigue. Wernick and Tobias also suggest that the effect of central factors on auditory fatigue may be due to modification of inhibitory impulses in the efferent pathways caused by an accumulation of waste materials brought about by chemical decomposition.

Ward and Sweet (1963) conducted a similar study in which they exposed 12 normal subjects to a 4KHz tone at 100 dB SPL for 3 minutes under 2 conditions viz.

- (a) sitting quietly
- (b) adding columns the figures.

Pre and post exposure thresholds were measured at 5.6KHz. They observed no significant difference between TTS at any time after exposure, in fact, the greatest mean difference at any time

was in opposite direction i.e. TTS at 2 minutes 15 seconds was 14 dB for revire and 12 dB for mental task condition. They concluded that the efferent system need not be invoked to account for any aspects of auditory fatigue.

Similar findings were reported by Bell and Stern (1964); Riach and Sheposh (1964); Price and Oatman (1967). They further write that the difference in results observed between mental task and revire conditions is just a chance factor.

But Capps and Collins (1965) replicated the lower level fatigue condition of the Wernick and Tobias study in order to explore the possibility that difference in task or procedures might account for the failure of the subsequent experiments to detect the difference in fatigue as a function of mental activity during fatiguing period.

They exposed 10 normal hearing adults to either mental task or revire condition, with no control group. They observed both, larger mean TTS and a longer recovery time for mental task condition.

They also offer some explanation to account for the failure of other investigators to obtain these results.

Ward and Sweet used a mental task of adding columns of figures which may be possibly not as effective as the mental arithmetic calculations used by Wernick and Tobias.

Bell and Stern used doing long division as mental task which may not be as effective as mental arithmetic tasks of Wernick and Tobias and also in their study (Bell and Stern) the effectiveness i.e. arousal value of mental task might be decreased with repeated use.

Capps and Collins also indicate that Riach and Sheposh did not have appropriate REV condition.

Hence the possibility of central influence on auditory fatigue cannot be ruled out on the grounds that the data results from chance variation. The choice of mental task and instructions for revire appears to be of primary considerations in experimental design to study TTS as a function of mental activity.

Smith et al., (1968) reported no significant difference between mental task and revire effects on TTS when the mental task was similar to that of Wernick and Tobias and also using paper-pencil tasks as mental task when subjects were exposed to 1KHz tone at 100 dB SPL for 10 minutes.

But they reported consistently greater TTS when the subjects were asked to track their thresholds for 1KHz tone presented in one ear and fatiguing stimulus was present in the contralateral ear, than when the subjects were in the condition of revire during the presentation of fatiguing stimulus.

They further report that tracking thesholds was more attention demanding or arousing than the mental arithmetic. But they

found no significant difference between TTS values obtained as a function of drugs administered to either increase or decrease the arousal.

They conclude that the question of whether TTS may be influenced by concomittent activity in which the observer is engaged is yet to be answered.

Dunn and Grauer (1981) in their study on attention factors in centralateral threshold shift measured the thresholds of 3 normal subjects in the left ear at 3 KHz before and after receiving in the right ear either a 13 minutes fatiguing stimulus i.e. 500Hz pure tone presented at 75 dB SPL or 13 minutes silence. They observed that only when subjects attention was directed towards the fatiguing tone, the threshold increased. In other cases threshold dropped after exposure. They suggested that this clearly indicates the involvement of central attentional variables in the process of auditoryfatigue.

Cody and Johnstone (1982) observed the action of efferent auditory pathway in the process of auditory fatigue in the guineapigs who were exposed to 10 KHz pure tone 107 dB SPL for 1 minute. They showed that monaural losses in hearing sensitivity could be reduced if an acoustic stimulation of the same frequency was simultaneously delivered to the other ear. The decrease in the threshold was eliminated when the contralateral stimulus was set at a frequency other than the ipsilateral trauma frequency and also after the administration of strychnine, a known blocker of auditory efferent activity.

Rajan et al., (1983) observed that TTS of the cochlear action potential in one (ipsilateral) ear caused by brief intense pure tones, were decreased either by (i) contralateral acoustic stimulation at the same frequency (ii) destruction of the contralateral cochlea. The effect of contralateral cochlea persisted, though slightly decreased in effect, after a delay of one hour between the destruction and ipsilateral exposure. However, contralateral cochlea destruction resulted in none of the effects upon normal ipsilateral thresholds or input-output curves for action potential classically seen when the efferent pathways are stimulated. The results suggest that the crossed effects reported here are due to a complex interaction between the activity of both the cochleas, possibly resulting in a reduction in a central inhibitory influence on an efferent feedback pathway that is then expressed during the ipsilateral exposure.

Guiot (1969) showed that stimulation of left ear had a definite influence upon the TTS measured on the right ear.

Further he says that if any summation effects were to occur, a reduction of sensitivity should have resulted rather than an increase as was actually recorded. A reasonable interpretation of this outcome can be formulated if one admits that a central inhibitory process, in conjunction with fatigue to be intervened in the production of TTS.

Thus, TTS can be shown to demonstrate peripheral and neural effects. Ramdolph and Gardner (1973) in their study of an inter-aural phase effect in binaural TTS. showed that in homophasic condition particular neural units in an afferent pathway are constantly stimulated and ultimately fatigued, the post exposure threshold resulting from restimulation of the same neurons will be shifted. In antiphasic condition (i.e. fatiguing and test stimuli are out of phase) conversely, the exposure and test tones could inturn activate different neural units and produce less TTS.

But Melnick (1967) observed that when listeners were exposed to 500 and 750 Hz/^{tones}at 120 and 110 dB respectively, TTS at test signals of 750 and 1000Hz was greater for monaural thanfor binaural exposure and no phase effects was noticed. For an exposure signal combination of 4000 and 6000Hz, the binaural-monaural difference was eliminated, but there was a significant phase effect for the two binaural exposure condition i.e. More TTS occurred when the exposure signal was 180* out of phase. They consider these results as representing another enigma.

Nazneen (1984) has reported that TTS observed in the right ear is significantly greater than the TTS observed in the left ear. So she suggests that the ear difference does exist in TTS for binaural stimulation using 2 KHztone. Further she reports that the faes thasw right ear shows more TTS than the left ear is an indication

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of the action of the efferent auditory system during binaural stimulation which more intense in the right ear than in the left ear.

VI. Structural changes in the auditory system as a result of acoustic overstimulation

Most of the studies regarding structural changes in the auditory system after exposure to loud sounds are concerned with permanent threshold shifts (PTS) and several of them reveal mainly structural changes in the cochlea (for eg. Liberman and Kiang 1978; Engstrom and Engstrom 1979; Rubel et al., 1982 etc).

Duvall et al., (1974) exposed Chinchillas to a 700-2800Hz noise at 123 dB for 15 minutes and noted a significant structural changes occurring in the stria which included stria widening, temporary disappearance of intermediate cells, alteration of internal membrane system and abnormal vessel transport.

Morest et al., (1979) report that in the cochlear nucleus the small cochlear nerve endings are especially susceptible to acoustic overstimulation. More over, transneural degeneration of synaptic endings occurred in certain regions of SOC without the loss of nerve cell bodies in the cochlear nucleus that normally project there. This suggests for a selective susceptibility of different components of auditory pathways to acoustic overstimulation (cited from Morest and Bhone 1983).

Morest and Bhone (1983) also reported transneural degeneration of axonal endings in SOC and IC. They also reported degeneration of both coarse and fine fibers in the cochlear

nerve root where it enters the cochlear nucleus. These changes were observed in Chinchillas exposed to an octave band noise centered at 4 KHz with 108 dB SPL for 1% hours.

MLD as a SOC Phenomenon;

Masking level difference (MLD) is one of the important psycho-acoustic phenomenon* MLD is the difference between binaural masked thresholds obtained under two conditions viz. homophasic (NoSo-the noise and signal are in phase at the two ears) and antiphasic (NoSII or Nil So - the phase of either the signal or noise is reversed (180*) at the two ears).

The MLD values in normal hearing subjects range from about 3 dB at high frequencies to as much as 15 dB at low frequencies, especially at 500 Hz (Green and Yost, 1975; Jeffress 1972; MdFadden, 1975; Moore, 1977). Several theories and models have been put forward to explain MLD (eg: Durlach, 1972; Jeffress. 1972; Hafter et al., 1969). However, Green and Yost (1975) point out that none of the existent models fits well into the neurophysiological mechanisms.

It has been reported (Harrison and Howe, 1974; Gibson, 1978) that the olivary nuclei represent the most caudal brain stem structure receiving auditory afferent information from both ipsilateral and contralateral ears. Further, Moushegion et al..

1964 have shown that single unit discharge patterns in the medial superior olive (MSO) are differently affected by ipsilateral and contralateral stimulations.

Of recent, Hannley et al (1983) report the results in which they compared auditory brain stem responses (ABR), the MLD for 500 Hz pure tones and acoustic reflexes in 20 patients with confirmed multiple sclerosis. Their rationale for their study was based on the conjecture that the measures -- MLD? wave III of ABR, and the acoustic reflex -- have in common a demonstrated (Borg, 1973? Jewett, 1970: Lev and Sohmer, 1972? Buchwald and Huang, 1975) mediation by lower brain stem structures in the region of the superior olivary complex (SOC). They observed that the size of MLD varied with the integrity of wave-III of the ABR and that absence of Wave-III of the ABR was associated with the absence of MLD and abnormal contralateral reflex.

They Hannley et al., (1983) have concluded that both the wave-III of ABR and the MLD have a common neuromechanism in the region of SOC.

In a similar study Lynn et al., (1981) measured binaural speech detection MLD in 26 patients with confirmed brain stem and cerebral level lesions and 10 normal hearing control subjects. They observed no significant difference between MLD values of normal subjects and patients with cerebral level or rostral patbiney

mid brain and thalamic levels of lesions. For patients with pontomedullary level lesions? MLDs were significantly smaller than for other groups. Hence they concluded the MLD originates from pontomedullary region of the brain stem.

Vyasamurthy et al., (198[^]) measured the ABR tracings in 3 conditions (i) homophasic (NoSo) (2) antiphasic (NoSII) and (3) antiphasic (NTTSo) in normal hearing subjects. They observed significant increase in the latency of III and V peak of ABR in antiphasic condition in comparison to homophasic condition.

But no significant difference in amplitude of the wave forms and interpeak (V-III) latencies between homophasic and antiphasic condition was reported.

They further suggest that the prolongation of III and V waves latencies during antiphasic condition suggests that some changes in neural mechanisms in SOC may be taking place. Since the interpeak (V-I) latency also shows prolongation effect during antiphasic condition, the prolongation effect is not likely to the changes in the peripheral level. Obviously SOC is involved in the MLD phenomenon.

Hence it has been concluded that MLD is a SOC phenomena.

From the review of literature on TTS, one can see that no pertinent literature is available regarding the locus of auditory

fatigue. And therefore, this study has been taken up so that some additional information can be provided to answer the question whether the mechanism producing the auditory fatigue lies only in the cochlea or structures beyond it.

METHODOLOGY

SUBJECTS:

5 male and 5 female subjects having normal hearing in the age range of 18 to 23 years were selected from the student population of All India Institute of Speech and Hearing, Mysore. The selection of subjects was done mainly on random basis.

The subjects selected for this study, had no history of any ear discharge, earache, tinnitus, giddiness, headache, brain damage or exposure to loud sounds.

All the subjects had hearing sensitivity within 20 dB HL (ANSI, 1969) in the frequencies 250 Hz, 500 Hz, 1KHz, 2KHz, 4KHz, 8 KHz.

INSTRUMENT USED:

Grason-stadler Audiometer (GSI-10) with TDH-39 earphones mounted in Mx41/AR supra-aural cushions. The audiometer was calibrated according to the specifications given by ANSI 1969.

Detail description of the different control of the instrument and also setting for MLD measurement is given in Appendix 'A'.

TEST ENVIRONMENT:

The study was carried out in an acoustically sound treated room at All India Institute of Speech and Hearing, Mysore. The

ambient noise levels present in the test room were below the proposed maximum allowable noise levels.

PROCEDURE:

All the subjects were screened at 20 dB HL (ANSI, 1969) for the frequencies 250 Hz, 500 Hz, 1KHz, 2KHz, 4KHz, 8KHz to find the presence or absence of a hearing loss in both the ears.

The study was conducted in two conditions: (1) Threshold measurements using 1KHz pulsed tones before and after fatigue. (2) MLD measurements in both NoSII and NIISo before and after fatigue.

Condition-I:

Thresholds were estimated at 1KHz for both the ears using pulsed tones which had on and off time of 200 msec.

The subjects were then exposed to 500Hz tone at 95 dB HL in both the ears simultaneously for ten minutes.

TTS was then determined in the right ear:

- (i) Immediately after the termination of the fatiguing stimulus (TTS_0) i.e. TTS_0 = Threshold at 1KHz pulsed tone with on and off time of 200 msec, immediately after the termination of the fatiguing stimulus - Threshold at 1KHz pulsed tone before the ear was fatigued.

(ii) After two minutes of recovery time (TTS2)

TTS2= Threshold at 1KHz (pulsed tone) obtained 2 minutes after the termination of the fatiguing stimulus - threshold at 1KHz (pulsed tone) obtained before the ear was fatigued.

A minimum of 24 hours rest period was given to each subject before testing them in the condition-II of the study.

Condition-II:

For obtaining Masking Level Difference (MLD) values each subject was presented binaurally with a narrow band noise of 50 dB SPL, centered around 500 Hz and 500 Hz pulsed tone which had on and off time of 200 msec under following conditions.

(i) homophasic (NoSo) i.e. when both the noise and signal are in phase at the two ears.

(ii) antiphasic (NoSII) i.e. when the phase of the signal is reversed (180°) at the two ears.

(iii) antiphasic (NIISo) i.e. when the phase of the noise is reversed (180°) at the two ears.

Then each subjects was presented with the fatiguing stimulus of 500Hz tone at 95 dB HL binaurally for 10 minutes.

Immediately after the termination of the fatiguing stimulus MLD values were obtained for each of the subjects by using the

The data was then analysed statistically using the nonparametric Wilcoxon Matched Pairs, Signed-ranks test.

RESULTS AND DISCUSSION

Table-i reveals the temporary threshold shift (TTS₂) at 1KHz in right ear after a binaural exposure to a 500Hz pure tone at 95 dB HL for 10 minutes. The result show that all the subjects except one had higher thresholds 2 minutes after the exposure (i.e. exhibited TTS₂). The mean TTS- value is also indicated in the same table.

Table-2 shows the MLD values obtained for 500 Hz pure tone in NoSII condition (i.e. signal in the both the ears are out of phase by 180*), before and after fatigue. The results show that only 2 subjects did not show any difference in the MLD values obtained before and after fatigue. All other subjects showed decrease in MLD values obtained after fatigue.

Table-3 shows the MLD values for 500 Hz pure tones in NIISo condition (i.e. the noise in both the ears are out of phase by 180°) before and after fatigue (fatiguing stimulus 500 Hz pure tone, at 95 dB HL for 10 minutes). The results show that in 6 subjects MLD values were decreased after fatigue 4 subjects showed no variation inMLD values obtained in post fatigue condition in comparison to MLD values obtained before fatigue in the same (NTISo) condition.

"The Wilcoxon's-Matched Pairs Singed Ranks Test" was used to find out whether there is significant different between the

thresholds obtained before and 2 minutes after fatigue (TTS_2) and also between MLD values obtained in NoSII and NIISo conditions before and after fatigue.

The analysis of the data reveals that there is a significant difference between – The thresholds obtained before and 2 minutes after the fatigue (at 0.001 level of significance);

The MLD values obtained in NoSII conditions before and after fatigue (at 0.01 level significance):

The MLD values obtained in NIISo condition before and after fatigue (at 0.05 level of significance).

According to the results obtained from the present study, the null hypothesis that –

There is no difference between thresholds at 1KHz before and after fatigue:

There is no difference between the MLD values obtained in both NoSII and NIIESo conditions before and after fatigue are rejected.

DISCUSSION:

Empirical investigations of the locus of auditory fatigue have often been concerned with the question of whether fatigue producing mechanisms lies within the cochlea or structures beyond it.

Several investigators (eg. Legouix and Pierson 1981: Johnstone and Sellick. 1972? Davis et al., 1950; Sohmer and Pratt, 1975; Jerger, 1955; Wright, 1968: Durrant, 1936) report that the auditory fatigue involves only cochlea.

On the other hand the existence of central influence on auditory fatigue has been observed by many authors (eg. Rawden-Smith 1936; Wernick and Tobias, 1965; Capps and Collins 1965; Salvi et al., 1983; Benitez, et al., 1972; Babigian et al., 1975; Cody and Johnstone 1982; Dunn and Grauer 1981; Rajan et al., 1983).

Review of literature concerning the locus of auditory fatigue do not provide definite answer wot the question of whether the auditory fatigue is mediated by cochlea or structures beyond it.

Recently Haaniley et al., 1983; Lynn et al., 1981 and Vyasamurthy et al., 1985 have shown that Masking Level Difference (MLD) one of the psycho-acoustic phenomena to be mediated by Superior Olivary Complex (SOC) a structure beyond the cochlea.

The present study was undertaken to test whether auditory fatigue has any effect on the MLD values for pure tone of 500Hz.

The results of the present investigation show that the MLD values obtained in both NoSII and NIISo conditions are significantly decreased after fatigue.

Thus the present study suggest the involvement of SOC a structure beyond cochlea in the auditory fatigue. In other words, the present study reveals that the central factors are involved in the auditory fatigue. This finding is the agreement with the previous studies such as-Rawden-Smith 1936: Wernick and Tobias, 1965; Cupps and Collins 1965; Salvi et al., 1983; Benitez, et al., 1972; Babigian et al., 1975; Cody and Johnstone 1982; Dunn and Grauer 1981; Rajan et al., 1983.

Table-1: Temporary Threshold Shifts (TTS2) at 1KHz pulsed tone

Subjects	Threshold at 1KHz before fatigue in dB	Threshold at 1KHz 2 minutes after fatigue in dB.	TTS2 in dB	Mean TTS2 in dB	Level of Significance
1	15	20	5		
2	15	25	10		
3	10	20	10		
4	15	20	5		
5	10	15	5	6.0	0.01
6	10	15	5		
7	10	10	0		
8	15	20	5		
9	10	20	10		
10	15	20	5		

Table-2: MLD values for 500 Hz pure tones (No SII condition) before and after fatigue.

Subject	MLD value before fatigue in dB.	MLD value after fatigue in dB.	Difference in MLD value obtained before & after fatigue in dB	Mean MLD difference in dB	Level of significance
1	12.5	10.0	2.5		
2	12.5	10.0	2.5		
3	12.5	10.0	2.5		
4	15.0	10.0	5.0		
5	10.0	10.0	0.0	2.25	0.01
6	10.0	7.5	2.5		
7	10.0	7.5	2.5		
8	12.5	12.5	0.0		
9	12.5	10.0	2.5		
10	10.0	7.5	2.5		

Table-3: MLD values for 500Hz pure tones (N--S condition) before and after fatigue.

Subject	MLD value before fatigue in dB.	MLD value after fatigue in dB	Difference in MLD value obtained before & after fatigue in dB	Mean MLD difference in dB	Level of significance
1	10.0	10.0	0.0		
2	10.0	10.0	0.0		
3	10.0	7.5	2.5		
4	12.5	10.0	2.5		
5	7.5	7.5	0.0		
6	10.0	7.5	2.5	1.50	0.05
7	7.5	5.0	2.5		
8	10.0	10.0	0.0		
9	10.0	7.5	2.5		
10	10.0	7.5	2.5		

SUMMARY AND CONCLUSIONS

Auditory fatigue is one of the terms used to describe a temporary change in threshold sensitivity following exposure to another auditory stimulus (Ward, 1963).

Despite, the extensive research carried out in the field of auditory fatigue; it is not completely understood that whether the mechanism of the auditory fatigue involves cochlea only or structures beyond the cochlea also.

The present study was aimed to study the effect of auditory fatigue on MLD values measured for 500 Hz pure tone in both NoSII and NIISo conditions.

10 normal hearing subjects (5 males and 5 females) in the age range of 18 to 23 years were selected. Each subject was tested under two conditions:

(1) Measurement of thresholds using 1KHz pulsed tones in the right ear before and 2 minutes after the binaural exposure to a fatiguing stimulus of 500Hz pure tone presented at 95 dB HL for 10 minutes.

TTS_2 was calculated by subtracting the original thresholds measured using 1KHz pulsed tones from the thresholds obtained using 1KHz pulsed tones 2 minutes after fatigue.

(2) Measurement of MLD values for 500 Hz pure tones in both NosII and NIISo conditions before and after binaural exposure to a fatiguing stimulus of 500 Hz pure tone presented at 95 dB HL for 10 minutes.

Between the two measurement conditions a minimum of 24 hours rest was given to each subject. All the subjects were tested in sound treated room of All India Institute of Speech and Hearing, Mysore using a calibrated Grason-Stadler Audiometer (GSI-10) audiometer with TDH-39 earphones mounted in MX41/AR supraaural cushions.

The data obtained were subjected to statistical analysis (Wilcoxon's method) and the following are the results obtained from the study.

1. The test ear showed significant difference (0.01 level) between the thresholds obtained for pulsed tones at 1KHz before and 2 minutes after fatigue i.e. significant TTS_2 was observed.

2. There was a significant difference (0.01 level) between the MLD values obtained for 500 Hz pure tone before and after fatigue in NoSII condition i.e. the MLD values decreased after fatigue.

3. There was a significant difference (0.05 level) between the MLD values obtained for 500 Hz pure tone in NITTSO condition before and after fatigue i.e. the MLD values decreased after the fatigue.

Hannley et al., (1983): Vyasamurthy, et al., (1985) have shown that Masking Level Difference (MLD) is mediated by superior olivary complex (SOC) a structure beyond the cochlea in the auditory pathway.

Since the present study shows that the MLD value is reduced after the ear is fatigued and since MLD is mediated mainly by SOC, it is reasonable to conclude that SOC is involved in the auditory fatigue. Following this reasoning, it is likely that the central factors are involved in the auditory fatigue. The present finding is in support of the many studies (Rawden-Smith 1938; Wernick and Tobas, 1965; Capps and Collins 1965; Salvi, et al., 1983; Benitez, et al., 1972; Babigian et al., 1975; Cody and Johnstone 1982; Dunn and Grauer 1981; Rajan et al., 1983; Fialkowska et al. 1983; Guiot, 1969; Morest and Bhone, 1983) which show that the central factors are involved in the auditory fatigue.

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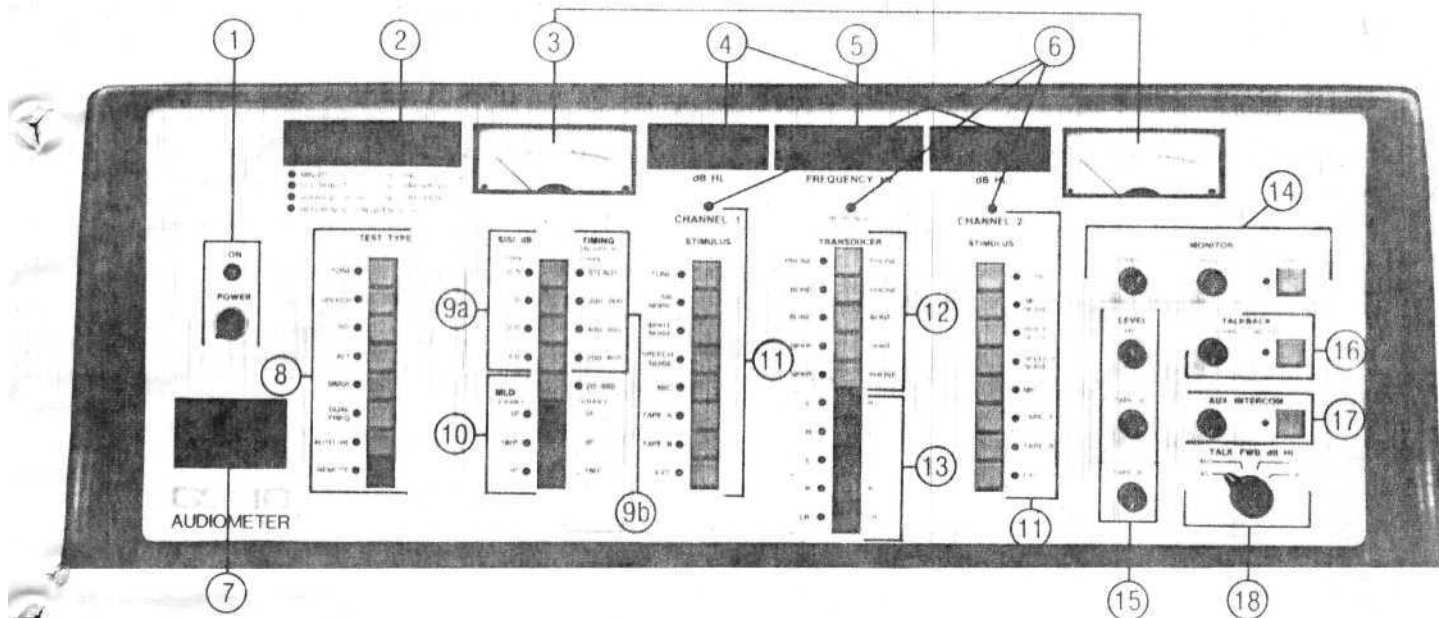


Figure 2-2. Top Front Panel

2.4.2 Front Panel Controls and Indicators

The controls and indicators on the front panel of the GS10 are shown in Figure 2-2 and Figure 2-3.

(1) **POWER ON/OFF Switch and Indicator Lamp** - turning the switch to ON illuminates the lamp above the switch and initializes the unit to a TONE test type.

(2) **Left-hand Display** — shows time, scoring (% correct, number presented), AUTO HL threshold levels along with the number of ascending trials required to reach threshold or the Channel 2 frequency when DUAL FREQ is selected.

NOTE

One of four LEDs will indicate which operation is displayed for a particular test mode.

(3) **dB (VU) Meters, Channel 1 and Channel 2** — monitor the levels of the signals on Channel 1 and Channel 2.

NOTE

The Channel 2 dB (VU) Meter may be used to monitor talk-back levels from the patient by activating the TALKBACK CHANNEL 2 METER pushbutton.

(4) **dB HL Displays, Channel 1 and Channel 2** — show the hearing level of the signal in their respective channels.

(5) **FREQUENCY Hz Display** — shows the selected frequency for all tests. It is blank in the SPEECH mode.

(3) **CHANNEL 1, CHANNEL 2 and RESPONSE Indicator Lamps** — illuminated for the duration of each test stimulus presentation. The RESPONSE indicator lamp is lit when the patient presses down on the hand switch button.

(7) **Monitor Loudspeaker** — allows the operator to listen to stimuli as they are being presented via Channel 1 and/or Channel 2 or to listen to patient comments via the TALKBACK system.

(8) **TEST TYPES**

- a. TONE — pushbutton initializes the test protocol for air/bone conduction testing, masked, or unmasked
- b. SPEECH - pushbutton sets up the GSI 10 for speech audiometric tests (i.e., speech reception thresholds, speech discrimination) via a test microphone for live-voice testing or through an optional tape cassette for recorded speech.
- c. SiSi — pushbutton initializes test procedure.
- d. ALternate — pushbutton initializes the tone for the alternate presentation of Channel 1 and Channel 2 stimuli to the selected transducers.

NOTE

The timing is set internally and does not appear on the front panel.

- e. SIMULtaneous - pushbutton initializes the test protocol for the simultaneous presentation of 'one-type stimuli from Channel 1 and Channel 2.

NOTE

The timing is set internally and does not appear on the front panel.

- f. **DUAL FREQUENCY** — pushbutton activates the second test frequency capability for Channel 2. This frequency is shown on the Left-hand Display. The REFERENCE FREQUENCY Hz LED will light under this display when this test mode is chosen.
- g. **AUTO HL** — pushbutton initializes an automatic Hughson-Westlake procedure. The Left-hand Display reads —P . and the AVERAGE dB HL/NO. PRESENTED LED under this display will light.
- h. **REMOTE (option)** — pushbutton will allow the operator to communicate with an external computer via an RS232C interface board.

(9) **CHAN 1 TIMING**

- a. **SISI dB** — pushbuttons select a SISI pulsed increment of 0.5, 1.0, 2.0 or 4.0 dB.
- b. **TIMING ON/OFF ms CHAN 1** — pushbuttons select the timing on Channel 1. The timings available are STEADY, 200 ms ON/200 ms OFF, 400 ms ON/400 ms OFF, 200 ms ON/800 ms OFF and 20 ms ON/980 ms OFF.

(10) **MLD (Masking Level Difference)** — pushbuttons

select the phase of the signal routed through Channel 1 and Channel 2.

In 0°/0°, both the Channel 1 and Channel 2 signals are in phase to both transducers. This is considered to be the baseline or reference condition as it provides fundamentally no MLD.

In 180°/0° the Channel 1 signal is out of phase between the transducers and the Channel 2 signal is in phase.

In 0°/180° the Channel 1 signal is in phase and the Channel 2 signal is out of phase between the transducers.

For more information on MLD see *Appendix*.

(11) **STIMULUS Channel 1 and Channel 2**

- a. **TONE** — pure tones available are 125, 250, 500, 750 and 1000, 1500, 2000, 3000, 4000, 6000, 8000 and 12,000 Hz ± 1 % accuracy. With the Bekesy option, the sweep frequency range is 125 to 12,000 Hz with ± 2 % accuracy for digital readout and ± 3 % accuracy for chart readout. The total harmonic distortion is less than 2%.
- b. **NB NOISE** — centered at each test frequency and available for all test frequencies with a 3 dB down bandwidth of less than 1/3 octave. Each narrow band is calibrated in effective masking
- c. **WHITE NOISE** — calibrated for pure tone effective masking if a tone type signal is selected and for speech effective masking if a speech type signal is selected. Electrically, there is equal energy per Hz with 3 dB down points at 60 Hz and 15,000 Hz with a 12 dB/octave roll off thereafter
- d. **SPEECH NOISE** calibrated in octave masking level and consists of equal energy per Hz from 250 to 1000 Hz with a 12 dB/octave roll-off from 1000 to 6000 Hz.

- e. **MICROPHONE** — input capability for live-voice speech testing when the SPEECH test type is activated. The microphone also allows for communication with the patient when the TALKFORWARD pushbutton is depressed and/or with a test assistant when the AUX INTERCOM pushbutton is depressed.
- t. **TAPE A and TAPE B** — accepts recorded speech material from an optional two-channel tape cassette or reel-to-reel tape recorder.
- g. **EXTERNAL** — pushbuttons allow for the acceptance of external signals. It is calibrated for speech in a speech test mode or to the selected frequency in a tone test mode as long as the dB (VU) meter reads 0 dB.

NOTE

An additional pushbutton may be selected along with EXT on Channel 1 because the signal represented by that additional pushbutton may be routed to an external device for modification and then rerouted back to the GSI TO. Only one pushbutton at a time may be selected from the Channel 2 STIMULUS group.

To exit EXT push a TEST TYPE pushbutton.

(12) **TRANSDUCER Output Selector** - permits the

easy and simultaneous selection of the transducer for each stimulus available through Channel 1 and Channel 2. The labels to the left of this row of pushbuttons describe the transducer receiving the test signal from Channel 1. The labels to the right of this row of pushbuttons describe the transducer receiving the test signal from Channel 2. The following combination of transducers is available:)

CHANNEL 1	CHANNEL 2
PHONE	PHONE
BONE	PHONE
BONE	BONE
SPKR	SPKR
SPKR	PHONE

(13) **Routing Output Selector** — permits the easy and

simultaneous selection of the route for each stimulus available through Channel 1 and Channel 2. The labels to the left of this row of pushbuttons describe the route receiving the test signal from Channel 1. The labels to the right of this row of pushbuttons describe the route receiving the test signal from Channel 2. The following combination of routing is available:

CHANNEL 1	CHANNEL 2
left	Right
R	L
L	L
R	R
LR	LR

LR-LR mixes stimuli from both channels to each transducer and drive; both the left and right transducers with this combined signal.

NOTE

(1) All routing pushbuttons are invalid for BONE BONE- The routing is set internally. When the transducer selection of BONE

BONE is exited, the routing selection will default to R-L.

(2) *BONE-PHONE* and *SPKR-PHONE* routings can be utilized only in L-R and R-L. If either *PHONE-PHONE* or *SPKR-SPKR* is selected, then all routing pushbuttons are valid.

(14) MONITOR Controls (CHAN 1, CHAN 2, SPKR)

— pushbutton is used to turn the monitor speaker on and off. The monitor phone is activated by plugging this phone into the MONITOR PHONE-OPERATOR jack on the rear panel of the GS! 10. The monitor speaker or the monitor phone allows the operator to listen to stimuli as they are being presented via Channel 1 and/or Channel 2 or to listen to patient comments via the TALKBACK system. The CHAN 1 and CHAN 2 controls adjust the gain of each channel to the monitor speaker, monitor phone and auxiliary insert phone simultaneously,

NOTE

(1) When *MIC* is selected on either channel, that channel's input to the monitor speaker and monitor phone is deactivated to reduce acoustic feedback. The input to the auxiliary insert phone remains active if selected.

(2) When *TALK FORWARD* is selected, both Channel 1 and Channel 2's input to the monitor speaker, monitor earphone and auxiliary insert phone are deactivated.

(15) MIC, TAPE A and TAPE B— controls are used to

adjust the signal from these external sources so that their level is accurately reflected by the dB HL displays on the front panel. These inputs are adjusted by turning the appropriate control until an indication of 0 dB on the average is obtained on the selected channel dB (VU) meter.

NOTE

When the *EXTERNAL* input jacks for Channel 1 and Channel 2 are used, the gain control on the external device must be adjusted to obtain 0 dB HL indication on the appropriate channel dB (VU) meter.

(16) TALKBACK CHAN 2 METER Control — knob

controls the talkback gain through the monitor speaker, monitor phone and auxiliary insert phone. Activating the pushbutton allows the talkback signal to be monitored on the Channel 2 VU meter. When the TALKBACK CHAN 2 METER is active, the same knob sets the level of the Channel 2 dB (VU) meter.

NOTE

CAUTION should be taken when setting the TALKBACK CHAN 2 METER to read 0 dB when utilizing the monitor speaker, monitor earphone and auxiliary insert phone. The hearing level in these monitors may become uncomfortably loud.

(17) AUXILIARY INTERCOM — provides a means for the

operator to communicate with an assistant located in the test room and, in addition, allows this assistant to monitor the Channel 1 and Channel 2 signal presentations. By pressing the AUX INTERCOM pushbutton, the lamp next to it is illuminated and, the operator may talk to the assistant wearing the AUX INTERCOM insert phone at any time without the patient hearing the conversation.

NOTE

If the operator would like to talk to the patient and the test assistant simultaneously, then the TALK FORWARD pushbutton must be pressed while the AUX INTERCOM is active. Alternatively, Microphone may be selected as an input source on at least one of the channels.

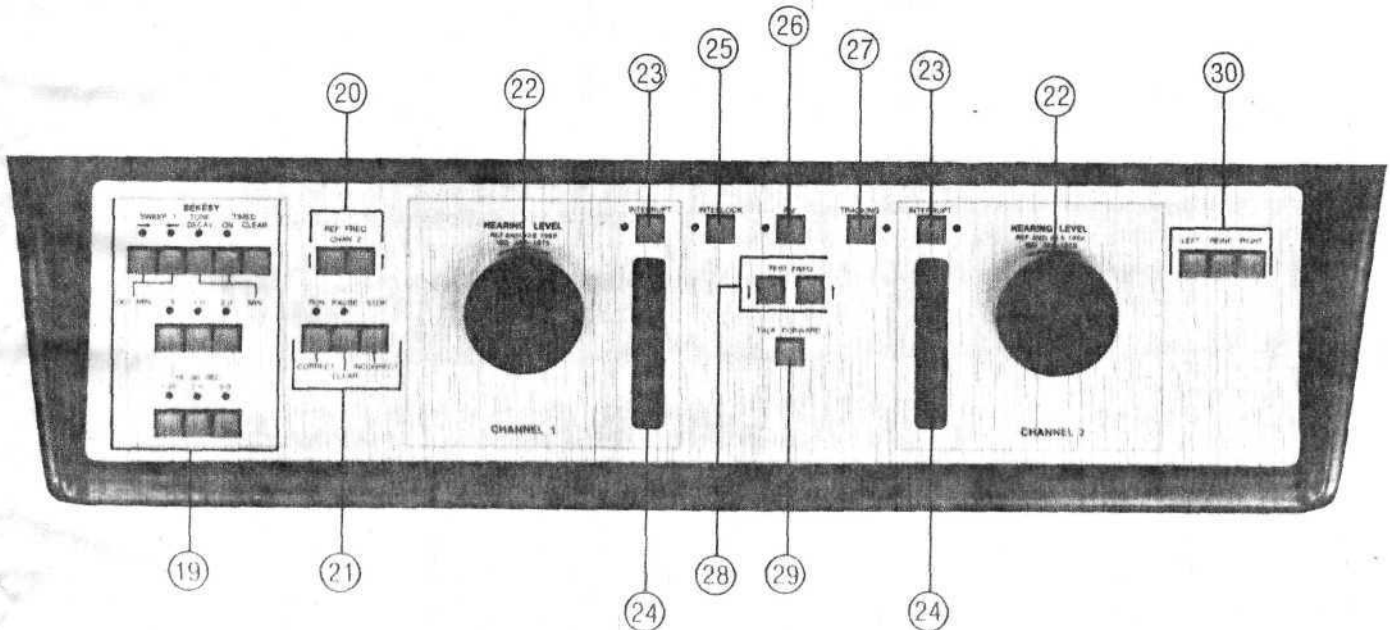


Figure 2-3. Bottom Front Panel

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The AUX INTERCOM knob controls the level of the signal, i.e., the tester's voice supplied to the AUX INTERCOM insert phone. The test assistant will also hear the Channel 1 and Channel 2 monitor signals as well as the patient talkback signal at the levels set by their respective controls on the front panel.

NOTE

Independent control over the monitor and talkback level is not obtainable for the operator and test assistant monitors.

In order to deactivate the Auxiliary INTERCOM, press the AUX INTERCOM pushbutton. The lamp next to it will no longer be illuminated.

- (18) TALK FWD dB HL — variable level control knob, calibrated for use with all of the included transducers at 45, 60, 75 and 90 dB HL when giving patient instructions.
- (19) BEKESY (active only with the Bekesy option)
 - a. SWEEP f- — pushbutton initializes the procedure for a Bekesy forward sweep test, beginning at 125 Hz or at a frequency selected by the operator prior to RUN.
 - b. SWEEP f*- — pushbutton initializes the procedure for a Bekesy reverse sweep test, beginning at 12,000 Hz or at a frequency selected by the operator prior to RUN.
 - c. TONE DECAY — pushbutton initializes the procedure for a Bekesy tone decay test with a hard-copy of the test results being traced out on the X-Y recorder.
 - d. TIMED ON — pushbutton initializes the procedure for a Bekesy timed test with a tone type signal (i.e., fixed frequency). However, a speech type signal may also be selected.

NOTE

TIMED ON can be selected simultaneously with all test types except for SISI and AUTO HL.

TIMED CLEAR- pushbutton allows operator to exit

Timed Bekesy mode.

- f. OCT/MIN, MIN — pushbuttons select an octave rate per minute (0.5, 1.0 or 2.0) for frequency change during a sweep frequency Bekesy test. When in TONE DECAY or TIMED ON, these pushbuttons select a test duration in minutes (0.5, 1.0, or 2.0).
- g. HL dB/SEC (In SWEEP f-> or -, TONE DECAY or TIMED ON) — pushbuttons select a rate of attenuation change of 1.25, 2.5 or 5.0 dB/SEC during the test.

NOTE

These pushbuttons may be used during all non-Bekesy test modes except Auto HL to change dB size to 1.0, 2.5 or 5.0 dB steps.

- (20) REF FREQ CHAN 2 — pushbuttons increase or decrease the Channel 2 frequency when in the DUAL FREQ test type.
- (21) RUN, PAUSE, STOPICORRECT, CLEAR, IN-

CORRECT — pushbuttons control the operations on the Left-hand Display. When TONE, SISI, ALT, SIMUL, AUTO HL or BEKESY is selected, the pushbuttons function as RUN, PAUSE and STOP. CORRECT, CLEAR and INCORRECT are valid for the SPEECH test type.

NOTE

These pushbuttons are invalid for the DUAL FREQ test type since the Left-hand Display is used to display the Channel 2 frequency.

- (22) HEARING LEVEL CHANNEL 1 and CHANNEL 2 Controls — Turning a control clockwise will increase the channel's HL and turning the control counter-clockwise will decrease the channel's HL.

NOTE

The control step size will automatically initialize to 2.5 or 5.0 dB, depending on the test type selected. However, the HL dB/SEC pushbuttons on the BEKESY panel may be used at any time to change step sizes to 1.0, 2.5 or 5.0 dB in all test types except AUTO HL.

The HL value shown on the dB HL display for each channel is the actual HL that will be routed through the selected transducer to the subject, irrespective of input source, frequency and output transducer. These variables are automatically taken into account by the GSI 10 and the necessary adjustments are made internally to maintain the HL. The range of HL on either channel is from - 10 dB HL to the maximum limit of the transducer selected for that channel.

- (23) INTERRUPT — pushbuttons determine the status of their respective tone bars and operate independently. To select the normally ON position, press the INTERRUPT pushbutton, the lamp next to the pushbutton is then illuminated.

NOTE

When the INTERRUPT pushbutton is set to the normally ON position, the corresponding channel is deactivated (interrupted) upon pressing the tone bar and activated (presented) upon releasing the tone bar.

To select the normally OFF position, press the INTERRUPT pushbutton once again. The lamp next to the pushbutton is no longer lit.

NOTE

When the INTERRUPT pushbutton is set to the normally OFF position, the corresponding channel is activated (presented) upon pressing the tone bar and deactivated (interrupted) upon releasing the tone bar.

- (24) Tone bars — each tone bar is independent unless the tone bars INTERLOCK is activated. When in a normally OFF (present) mode with a STEADY tone, activation of the tone bar will result in the immediate presentation of the stimulus. The stimulus will be presented for as long as the tone bar is pressed. When the tone bar is deactivated the channel will turn off immediately.

NOTE

If a pulsed timing is selected and the first pulse has not finished before the tone bar deactiva-