

TTS AND MENTAL CALCULATION

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CERTIFICATE

This is to certify that the Independent Project entitled TTS AND MENTAL CALCULATION is the bonafide work on part fulfilment for the Degree of Master of Science (Speech and Hearing) of the student with Register No.8703.

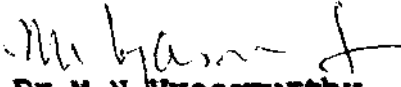


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CERTIFICATE

This is to certify that the Independent Project entitled TTS AND MENTAL CALCULATION has been prepared under ray supervision and guidance.


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DECLARATION

I hereby declare that this Independent Project entitled TTS AND MENTAL CALCULATION is the result of my own study under the guidance of DrM.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

Register No.8703

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To

MIKKY my sweetheart
you will remain fresh in my
memory till the end of my days

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"Thanks a Million Ashok:

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Thou Thought' st to help me
and such thanks I give

As one near death
to those that wish him life

- Shakespeare,

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INRODUCTION

One of the common functional characteristics of all sensory systems is a reduction in sensitivity following exposure to any stimulus of significant duration and intensity. For some systems (Eg. gustatory, olfactory) the sensation may disappear totally while others (eg. auditory) there is a reduction in apparent magnitude or an increase in the threshold. Such changes are temporary so long as the stimulation does not exceed critical limits which is the case in an everyday life for most receptor systems. Ears exposed to stimuli like gunshots or to long periods of high intensity noise results in sensitivity which may be permanently impaired. Often such damage occurs slowly from repeated exposure that individually cause only temporary shifts in sensitivity.

Auditory fatigue is an interesting phenomena, more because of its absence than its presence. It is surprising that the ear, assailed as it is, both day and night, by sounds and noises of all sorts, suffers so little decrement in acuity. No flap or lid enables us to protect our ears from unwanted disturbances and we must even leave them open when we sleep. We learn to disregard the unwanted sounds we hear, at the same time preserving a selective attention for what we consider significant. All said and done excessive unwanted noise has its ill effects on the organisms.

Thus the study of TTS is of theoretical and practical interest to audiologists because:

1. The similarities between TTS, auditory adaptation, and PTS (NIPTS) indicate that the anatomical and physiopathological processes which underlie may be differentiated only quantitatively (Derbyshire and Davis 1935; Davis et al. 1950; Shimizu et al.1967; Gisselson and Sorenson,1959; Tondorf et al.1955).
2. TTS may be used effectively to study the auditory fatigue and related phenomena, because in contrast to adaptation it permits post stimulatory study and in contrast to PTS it does not presuppose permanent damage.
3. TTS measures are among the important auditory tests performed to assess sensori neural hearing loss.
4. A series of clinical studies on TTS have attempted to evaluate the predictability of NIHL and to state some Damage Risk Criteria (Ward, 1970).

Two psychophysical measures commonly employed to assess the reduction of sensitivity of auditory system following noise exposure are.

1. Loudness decrement (obtained during the sensitivity reducing stimulation).

2. Shifts in threshold (obtained after the stimulation) Both being indices of ear's sensitivity. They reflect different types of changes in the auditory system. The former being termed as auditory adaptation and the latter termed as auditory fatigue (post stimulatory auditory fatigue).

Adaptation studies generally use weak to moderate levels of stimulation. Fatigue studies on the other hand are concerned with the effects of moderate to quite intense levels of noise stimulation.

Though there are evidences showing that these two psychophysical measures differ in the types of physiological changes it is also true that these differences are not often complete. This overtly becomes evident when the various physiological changes underlying decrements in ear's sensitivity are considered. They include-neural changes, hair-cell changes, endolymphatic changes etc.

Stimulation results in reversible neural changes that indicate neural adaptation, reduction in hair-cell response, and in all probability a variety of cochlear environmental changes that interfere with both hair-cell and nerve-cell functioning. Auditory sensitivity reflects the level of neural activity so one, must

consider a broad range of partially independent changes in the ear in attempting to understand the source of the decrease in sensitivity.

Post stimulatory auditory fatigue:

The most common index of auditory fatigue is the TTS (Temporary threshold shift). Usually it is measured by first determining the normal threshold, then exposing the ear to fatiguing stimulation and finally finding the post exposure threshold. Difference between pre and post exposure threshold defines the severity of the fatigue.

Thus precisely TTS can be defined as any post-stimulatory shift in threshold. It is a time linked process unlike masking and adaptation where recovery time is less than a minute and they are not time linked.

Primary factors influencing TTS are:

1. Recovery interval (RI)
2. Intensity of fatiguing exposure (I)
3. Test frequency (F_t)
4. Exposure frequency (F_e)
5. Duration of exposure (D)
6. Intermittent exposure.

Miscellaneous factors affecting TTS:

1. Interactive effects
2. Resting thresholds.
3. Latent and residual effects.
4. Vitamin A deficiency.
5. oxygen deficiency.
6. Salt (excess of it).
7. Vibration.
8. Age, Sex and Experience.
9. Impulse signals.
10. Drugs and levels of consciousness.

TTS and Methodological considerations:

Reliable threshold determination is time consuming and reflects not only the sensitivity of the receptor system, but the non-sensory factors as well. They are:

1. Practice
2. Motivation
3. The psychophysical procedure used
4. Chance fluctuations

These variables have to be reduced to minimum. For instance the practice effect may lead to change in susceptibility (Merish, and Ward, 1952; Loeb and Fletcher, 1963; Riach et al. 1964).

This may be due to conditioning affect of the acoustic reflex rather than changes in ear's resistance to fatigue.

Thus the divergences among some of the studies may be due to differences in the testing procedures.

If, TTS is primarily a peripheral physiological process, except in so far as higher centers are involved in the behaviour from which we infer just what the threshold itself is, one would think that the changes in TTS associated with changes in levels of consciousness would be relatively minor. That is, it should make no difference in the TTS produced by a given noise whether or not the listener is concentrating on the noise or reading a book, awake or asleep, hypnotized or alert – unless, of course, any of these affect the transmission of sound to the cochlear by reducing or enhancing the action of the middle ear muscles.

There is no concrete evidence about effect of drugs on TTS.

When the factors causing a change in an organisms normal response to stimulus are conditioned upon the state of the central nervous system it is not surprising

when we find increased variability. Central inhibition is a labile phenomena. Consequently, both the variability and binaural nature of auditory fatigue can be accounted for, if we, assume that the loss in sensitivity is due to the intervention of central factors. The phenomenon would partake, then, less of the nature of sensory fatigue than of the nature of inhibition, and phenomena of disinhibition (Pavlov's inhibition of inhibition) would be likely to appear.

The phenomena of auditory fatigue appears, then to be complicated by some type of central inhibition, which makes it hard to discover, by psychophysical experiment the actual loss of sensitivity within the sense organs due to previous stimulation.

A series of studies done by Bronstein shows that after exposure to a loud tone the auditory threshold not only returns to normal, but also, often it falls below normal for a period of time. The increased sensitivity, may amount to 10-15dB and extends to frequencies other than that of stimulating tone. As with fatigue, there is some sensitization in the opposite ear, when only one ear has been stimulated. This and other features of the phenomena, suggest sensitization and fatigue are both due, in part, to cortical factors.

Need for such a study:

To know if the higher centers play a role in influencing the amount of post exposure threshold shift (Central Inhibition).

Null Hypothesis:

There is no difference between the TTS_0 produced under 'Mental reverie' condition and the TTS_0 produced under some 'mental activity' condition.

There is no difference between the $TTS-$ produced in the first and second conditions mentioned above.

REVIEW OF LITERATURE

Central component in contralateral threshold shift.

Most of the studies that have tried to find the central component of TTS have employed post stimulatory paradigm.

Bekesy in 1929 made the first attempt, in a recorded version, to localize the place of origin of the fatigue phenomena. He fatigued one ear for 2 minutes with an 800Hz sinusoid of 108dB intensity and used a loudness doubling method to ascertain any fatigue effects in the contralateral ear. He found none and concluded fatigue to be a peripheral phenomena exclusively.

Rawdon and Smith in 1936 reported a similar experiment and concluded differently. Using methods of limits, he determined the intensity of the threshold before and after the introduction of monaural fatiguing tone in the opposite ear. On examination of the two, he found there was a considerable decrease in hearing sensitivity following the introduction of the fatiguing tone. They concluded central factors were involved in auditory fatigue. Further experimentation revealed that this post stimulatory contralateral threshold shift was influenced by the introduction of various non-auditory stimulation to the subject. For instance the threshold returned to normal and then

demonstrated an oscillating fluctuation when the lights in the sound proof room were turned off. A control experiment revealed that the normal threshold was not affected by this procedure. The phenomena found by Rawdon-Smith thus appeared to be more of the nature of inhibition than of fatigue. They speculated that the threshold fluctuation observed during and after the introduction of unexpected stimuli corresponded to disinhibition or inhibition. They thought that cortical intervention played a partial role in the fatigue phenomena in combination with a peripheral effect.

Causse and Chavasse in 1942 pointed out that the sound intensity used in the Rawdon and Smith experiment were high enough to affect the contralateral ear via air-bone conducted sound. Thus the premise that only central intervention of some kind could affect the threshold of the unfatigued ear was not entirely correct. In their replication of the original experiment they could not find a fatigue effect in the unstimulated ear. A clear effect was noticed in the stimulated ear, however they concluded that the effects reported by Rawdon-Smith are not present when extra-cranial sound leakage is controlled.

Rosenblith et al in 1950 have stated that cortical and cochlear responses grow in parallel as a function of intensity and that during recovery from sound exposure the N_1 response behaves similarly to the cortical one. Thus central effects produced by intense sounds are present and not a reflection of fatigue occurring in cochlea.

Ingham in 1957 experimented on subjects who monitored their thresholds at 1K before, during and after stimulation of the non test ear by a low intensity 400Hz tone. The results suggested an increased sensitivity during the first 10 minutes after stimulation. A close examination of threshold over time during contralateral stimulation reveals a gradual decrease in sensitivity during that period. All these findings have to be interpreted with some skepticism because Blegvad in 1968 has reported that there is a considerable individual variation in the contralateral fatigue. Threshold shifts in the same individuals were not always in the same direction.

Later Grauer and Dunn in 1978 performed two experiments to study perstimulatory auditory "Central fatigue" In the first experiment the subjects tracked thresholds for 13 minutes at 0.3, 0.6, 1, 2, 3 or 4K while being stimulated in the contralateral ear by a 0.5K tone at

75dB SPL. Bekesy tracings showed a significantly rising threshold at 2, 3 and 4KHz. In experiment-II subjects tracked threshold for 13 minutes of a 3KHz pulsed tone while a 0.5K tone pulsed simultaneously at 75dB SPL in the contralateral ear. Results from previous central masking research would predict enhanced effect, while results from ipsilateral fatigue studies would predict a much reduced effect. The data showed virtually no effect at all, thus supporting a fatigue hypothesis.

Again Dunn and Grauer in 1981 studied this on young adults. They tracked fixed frequency Bekesy thresholds in left ear to a 3KHz tone for 4 minutes before and for 4 minutes after receiving in the right ear either a 13 minute fatiguing tone at 75dB SPL or 13 minute silence. Half the time subjects, when receiving the fatiguing tone, read a magazine and the other half of the time, their attention was directed to the right ear. In the control condition with no fatiguing tone, subjects read half the time and the other half of the time they counted soft tone pips in right ear. Only when subjects attention was drawn towards the fatiguing tone did the thresholds rise. Other cases thresholds dropped. This lowering of thresholds may be due to response bias. This cannot explain why response bias did not occur when attention was directed towards stimulus during the 13 minutes

interval. This may have been due to contralateral fatigue which was great enough to effect the response bias. The other explanation may be that subjects might have been exhibiting the effects of auditory fatigue from ambient noise in a normal environment in the initial recording and some recovery had occurred during the time these subjects were in the quiet room. There is one more evidence of extracranial leakage which means that thresholds would be decreased even slightly after 13 minute stimulation, during which subjects were directing their attention away from sound (because a small amount of peripheral fatigue would be registered in the test ear). But this speculation was also not found to be true.

In perstimulation subjects must attend to stated auditory dimension of the primary stimulus during the presentation of the fatiguing stimulus. While in post stimulatory paradigm there is no such attention demand, here the subject is making an attempt to ignore the fatiguing stimulus. Thus there is an important potential difference between the two types of fatigue, implying that a mechanism which is capable of producing a contralateral fatigue effect is also affected by manipulations of attentional variables.

Central factors and post-stimulatory thresholds in contralateral or ipsilateral ear.

In 1963 Wernick and Tobias reported a central factor of auditory fatigue in humans. Their subjects in the experiment were asked either to solve an arithmetic problem or to maintain a state of reverie during the time of exposure to puretones of various intensities and frequencies. Post stimulation intensive thresholds of the same ear showed a greater elevation and took a longer time to recover when the fatiguing tone was presented in the mental task vs. reverie condition. The authors concluded that there is a central factor in pure tone auditory fatigue and that the degree of effect is a function of subject's mental activity during stimulation.

Reports by Ward and Sweet (1963); Bell and stern in 1964; Riach and sheposh in 1964; failed to confirm this study. Capps and Collins in 1965 suggested that this failure to the type of mental task used.

Price and Oatman in 1967 conducted 3 experiments. They isolated the procedural artifacts that could explain the results of the data of wernicke and Cobias. The first experiment they replicated the Wernicke and Tobias (1963) study and produced same data. Here mental activity in form of mental arithmetic during a pure tone exposure resulted in more auditory fatigue than the same exposure

during Reverie. In experiments II and III they were asked to prepare themselves for the threshold measurements and there was no increase in thresholds in mental task condition. The results indicated that if subjects could resume post exposure threshold tracking without being required to do something else simultaneously (such as write an answer to a problem), the differences between the experimental groups disappeared. Thus central auditory fatigue seems to be an artifact.

Fricke (1966) exposed subjects to 1000B or 120dB white noise for 15 minutes after which they listened either for interruptions in the noise or to a story to which they were supposed to attend. While there was some tendency for the noise and story TTS to be greater at 100dB differences were generally not significant as a function of the attention demanding story.

Smith and Loeb in 1968 conducted four experiments concerned with TTS obtained from longer exposure under different activating or attention demanding condition and under the influences of drugs. Two arithmetic tasks failed to produce significant differences in TTS. TTS was consistently greater when subjects were exposed while tracking a 1KHz tone than while exposed during reverie. D-amphetamine and secobarbital did not differ from a placebo in their effects on TTS. An activation hypothesis

relating changes in the magnitude of TTS to reticular activation was not confirmed, rather the significant effects obtained seem related to the nature of the distracting stimuli employed.

One possible explanation why the mental arithmetic task showed negative findings is that the task was too difficult for the population used. It seemed possible that longer exposure time would produce greater shifts and allow more opportunity for differentiated effects to become apparent. But this hypothesis was also refuted in their second experiment where the exposure time was greater and mental mathematics was a paper and pencil task. Again here there was no statistical significance. Third experiment was a tracking task where they were asked to track their thresholds for a 1K tone presented via an earphone in the opposite ear. This tracking test was carried out under two different conditions of stimulus intensity and compared it with their corresponding reverie state. There was a statistically significant difference in the tracking condition that is TTs was greater ($P < .05$) There was a significant difference attributable to the intensity of the fatiguing stimuli ($P < .01$) and time ($P < .01$) of the measurement. There was a significant interaction of the fatiguing stimuli intensity* mental

arithmetic and reverie conditions and time ($P < 0.05$). Though the difference between conditions appeared much greater at 100dB than that at 90dB the interaction of conditions and intensity was significant beyond the 0.1 level.

The above experiment was repeated because in all these results significance was found beyond the .05 level. This time with more number of subjects, they found TTs was greater when subjects were exposed during reverie state. There was a high significance in 0.01 level. There was a significant difference between exposure intensity ($P < .01$) and time of testing ($P < .01$). There was no statistical difference attributable to the interaction of stimulus intensity and mental arithmetic and reverie condition. The remaining interaction were not significant either.

The experimental artifact as suggested by Price and Catman in 1967 cannot really explain the significant differences in the tracking condition.

If there is an effect of the intensity and duration employed in these experiments, it would appear that the effect may be attributable to type of exposure, type of task, and the characteristics of the subjects employed.

The drug experiment suggests the differences previously obtained under certain experimental condition are not due to changes in the general level of arousal but to something more specific. The tracking experiments strongly imply that there may be some sort of attentional effect. Wernicke and Tobias (1963) attributed their findings to efferent gunk i.e. waste products resulting in sensory stimulation which add to fatigue products.

Another possibility is the inhibition of the acoustic reflex when observers are trying to detect a threshold level stimulation as suggested by Loeb and Riopelle (1960) Wernicke and Tobias ruled this out in their experiment because they felt that it would be ineffective at the frequencies and exposure intensities which they employed. But this is again contraindicated by the frequency and intensity used by Smith and Loeb.

The experiment by Guann 1967 does not support the inhibition hypothesis.

Collins and Lapp in 1965 found that tracking of contralateral tone does not influence TTs.

McInick in 1968 found less TTS at 1400Hz following a 2 minute exposure to a 1000Hz tone at 110dB SPL under the same tracking at 250Hz tone in nontest ear condition.

Speculated site of pathology:

Babighian, G. et al in 1975 studied TTs which was related to central auditory fatigue. Based on evoked responses and single-neuronal responses he revealed that there is a central involvement in auditory fatigue. In these experiments cochlear potentials (microphonic and whole nerve action potential) and inferior colliculus electrical responses were simultaneously obtained before and after excessive sound exposure. In general sound exposure produced a greater reduction of the collicular evoked responses than of the cochlear microphonics and action potentials. Recordings from single neurons support the evoked responses findings. Some authors have pointed out that degree and time course of TTS may be affected by the cochlear-cochlear or olivocochlear auditory mechanisms.

Elliot (1961) and Hunter - Duran (1971) say that changes in auditory sensitivity unaccompanied by hair cell loss have been noticed in Monkeys and cats.

Eldredge and Miller (1969), Ward and Durall (1971) - showed that considerable hair cell destruction may be correlated only with a slight TTS.

Ward et al 1972 - found in animals that only a small area of normal hair cells in the apex can respond to very

weak high frequency tones.

Similar lack of correspondence between the audiograms and pathology, have been reported by Benitez et al (1962) and by Bredberg (1968) . Bredberg described normal appearing hair cells associated with PTS, as well as a normal threshold despite a 35% loss of IHC and 45% loss of OHC in 81 year old man..

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

It has been seen that during recovery from TTS a reduction in amplitude of the collicular response continued for 30 minutes after the cessation of sound exposure.

Paralleling these changes there was a post exposure reduction of cochlear potentials. The CM and $N_1 - N_2$ potentials displayed a diphasic recovery.

In 1978 a study by Pratt et al was conducted regarding surface-recorded cochlear microphonics potentials during TTS in man.

CM was recorded by means of surface electrodes, before during and after white noise induced TTS in human volunteers.

The behaviour TTS was not accompanied by a Change in amplitude of CM. These findings indicate that in humans, the site affected by the noise exposure and which probably gives rise to TTS is central to the site of generation of CM.

In a previous study the compound action potential generated in the auditory nerve was found to be of a lower amplitude and longer latency during TTs, and it is thus proposed that the site affected is peripheral to the generation of conducted action potentials.

The synapse between hair cells and auditory nerve fibres is the most likely candidate to be the affected site.

In Chinchillas no change was found in the endocochlear potentials while changes in the CM recorded from the region of the cochlea corresponding to the frequency of the noise showed the best numerical correlation with the behaviour TTS in the same animals. Where as the compound action potentials of the auditory nerves showed an even greater change than other measures (Benitez et al. 1972). They concluded that the noise exposure effects two sites. The transducing mechanism of the hair cells and the synapse between the hair cells and auditory nerve fibres.

Similar experimentation on squirrel Monkeys have not shown clear evidence of hair cell damage, even when the noise exposure caused a PTS of 10-20dB (Hunter-Duvar and Elliot, 1972). Even PTS of 20-50dB resulted in a hair cells loss in a region which did not correspond to the frequency of the hearing loss in the observed animals. But a PTS of 40-60dB caused a hair cells loss corresponding to the noise and hearing loss frequencies in animal tested. Thus the sensitivities of Monkeys and chinchillas to noise exposure are different .

It seems unlikely that the efferent olivocochlear bundle contributes to this effect after noise exposure since no change in TTS was observed in Cats following section of this bundle (Trahiotis and Elliott 1969).

The absence of amplitude changes of the cochlear microphonics implies that there is no impairment of sound conduction to the inner ear, even though this might have been expected from the increase of latency of the auditory nerve compound action potentials (sohmer and Cohen, 1976). The stage affected by the noise exposure must be thus beyond the site of generation of the CM, but before the site of compound action potential. Therefore the synapse between hair cells and nerve fibres, or the auditory nerve dendritic processes in the inner ear may be the site

when the TTS is about 10-20dB.

Similar conclusions have been drawn when other sense organs have been continuously stimulated. The general belief is that the reduction of sensation is due to reduced synaptic efficacy.

Gerken, G.M et al in 1985 reported both hearing loss and continuous tone can decrease the absolute threshold for the detection of electrical stimulation applied to auditory nuclei of the brain. Like wise acme of the evoked responses in the auditory system are considerably larger in the animal with hearing loss, or in the normal hearing animals during presentation of continuous tone. Evoked responses enhancement are seen at the level of midbrain or above and primarily in the unanaesthetized Cats. Continuous tone stimuli have proved to be more effective than continuous noise in producing enhancement. This effect could be a representation of a Change at a higher level of the auditory system.

In 1983 Fialkowska, M.D. et al studied effect of noised induced TTS on the auditory brain stem activity which was associated with fatiguing processes in the cochlea and cochlear nerve as well as with the altered activity of the cochlear nucleus units. Based on these results and on biochemical and structural findings

presented by other authors it was concluded that TTS originates primarily in the cochlea and modifies the activity of neurones through out the auditory pathways.

Thus TTs may be useful as a measure of the individuals susceptibility to industrial noise* but the prognosis on the presumed PTs based on evaluation of the amount of TTS seems to be doubtful.

Results reviewed are confusing. Nevertheless, they are of considerable theoretical interest. Attempts to isolate an attentional component of central auditory fatigue have then been characterized by diverse and often inadequate methods leading to spurious and sometimes contradictory conclusions.

Recently there has been an interest in loudness adaptation with binaural stimulation. Research by Bray et al (1973) and Dirks et al (1974) yielded data supporting the position that all loudness adaptation has a central component. Weiler et al in 1975 reported same findings.

METHODOLOGY

SUBJECTS:

Ten subjects volunteered to undergo testing. Out of them 7 were females and 3 were males. Their age range was from 18 to 23 years with a median age of 20 years. These subjects were chosen at random. All of them had normal hearing, with their hearing sensitivity within 20dB HL (ANSI 1969) at frequencies from 250Hz to 8000Hz, at octave intervals. At the time of testing the subjects complained of no such otologic problems as tinnitus, ear ache, vertigo, headache, ear discharge, etc. None of them reported of having been exposed to loud noise earlier.

INSTRUMENT USED:

GSI-16 Audiometer was used for the experiment. TDH-39 earphone coupled with a supra-aural type of a cushion was used. The audiometer was calibrated according to the specification given by ANSI 1969; ISO 1975.

TEST ENVIRONMENT:

The experiment was carried out in a two room situation. Both rooms were sound treated. The ambient noise level was much below the maximum permissible level specified by ISO-1964.

PROCEDURE:

The subjects were made to sit on an armed chair comfortably

facing the tester.

Each subject was tested twice under two different conditions keeping all other variables constant. The first condition was the control where subjects were in a state of mental reverie while the stimulus tone was presented. The second condition being the experimental one where subjects were asked to solve a simple multiplication problem (arithmetic sum) and write down the answer. Each subject was given enough time (more than 2 days) to recover from the threshold shift induced in the control condition, before they underwent the experimental condition.

TEST ADMINISTRATION:

1. The subjects hearing acuity was tested to make sure they all had normal hearing.
2. Their right ear pure tone air conduction thresholds were noted for the frequencies 4KHz and 2KHz .
3. A pure tone of 2KHz was presented continuously for 10 minutes at 100dBHL in the right ear, as the fatiguing stimulus tone.
4. During the presentation of stimulus tone, the subject either sat quietly or solved the problem depending on the test condition (i.e. control or experimental)

5. Immediately after the stimulus tone was withdrawn, their threshold for 4KHz pulsed pure tone, taken as the test tone, was found out. This was repeated after 2 minutes after the withdrawal of stimulus tone, the former being TTS_0 and the latter being TTS_2 .

Here pulsed tone was used because some of the subjects complained of the tone lingering in the ear even after the stimulus tone was withdrawn. Thus pulsed tone made it easy for them to distinguish between the persisting tone in the ear and the tone through the earphone, which avoided confusion.

INSTRUCTIONS:

1. "Raise your finger even if you hear a slight sound in your ear".
2. "Then you will hear a loud sound in your ear continuously for 10 minutes in your right ear".
3. a) For control condition: "Be seated comfortably as long as the tone is heard in your ear".
b) For experimental condition: "As soon as you hear the sound in your ear start solving the problem".
4. "As soon as the tone in your ear stops you are required to again raise your finger even if you hear a slight tone. The tone will be a pulsed one. Repeat this again after 2 minutes when pulsed tone will be presented again".

The pre and post exposure thresholds were noted and the shift in threshold was entered in a tabulated form for further statistical analysis.

RESULTS

The data of the experiment has been epitomized in the four given tables.

Table-1 indicates the subjects shifts in threshold at 4KHz (the test tone) immediately after withdrawal of the fatiguing stimulus (2KHz pure tone). Precisely, this table reveals the TTS_0 of all the 10 subjects. Table-1(I) delineates the subjects TTS_0 measured under the control condition (State of mental reverie) and Table-1(II) denotes their TTS_0 under the experimental condition (exposure to fatiguing stimulus while working out a problem). These tables lucidly signifies that there is some change in TTS_0 under the second condition.

On comparison of the Tables 1(I) and (II) it is evident that subjects 5, 6, 8 and 10 showed an increase in TTS_0 the second time. Subjects 4 and 9 showed no difference. The rest showed a decrease. Table-2 indicates a greater variability of TTS_0 in the second condition. Also the mean of the experimental TTS_0 is clearly greater.

Table-3 elucidates similar data as in Table-1. The only difference being that these are the values for TTS_2 (The shift in threshold 2 minutes after withdrawal of fatiguing stimulus).

Once again these 10 subjects showed a slight modulation of TTS_2 in the experimental condition albeit subjects 1 and 9 showed no difference at all in the two situations. Positively 5 and 10 showed an increase in the experimental condition and the rest showed a decrement while they solved the problem.

Table-4 vividly exemplifies a greater variability in the experimental state for TTS_2 and as before the mean in the second condition is also greater.

To verify if the differences in means were statistically significant, the Wilcoxon's matched-pairs, signed-rank test was used. Consequently both the differences in means were confirmed to be non-significant at all three levels of significance (0.025, 0.01 and 0.0005).

Apparently the results connotes that the two Null hypotheses, made prior to the commencement of the experiment, remains unaltered.

Table-I: Indicates values for TTS_0

| subjects | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|--|----|----|----|----|----|----|----|----|----|----|
| I TTS in dB in control condition. | 25 | 30 | 35 | 30 | 35 | 30 | 25 | 20 | 25 | 40 |
| II TTS in dB experimental condition. | 20 | 25 | 30 | 30 | 45 | 35 | 20 | 30 | 25 | 45 |

Table-2: Mean and Standard Deviation of TTS_0

| | Mean (dB) | Standard Deviation |
|---|-----------|--------------------|
| I Control condition TTS_0 | 29.5 | 5.68 |
| II Experimental condition TTS_0 | 30.5 | 8.5 |

Table-3: Indicates values for TTS₂

| Subjects | 1 | 2 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|--|----|----|----|----|----|----|----|----|----|
| I Control TTS ₀ in dB | 15 | 25 | 20 | 20 | 25 | 25 | 20 | 20 | 25 |
| II Experimental TTS ₂ in dB | 15 | 20 | 15 | 15 | 40 | 20 | 15 | 20 | 30 |

Table-4: Mean and standard deviation of TTS₂

| | Mean(dB) | Standard deviation |
|--|----------|--------------------|
| I Control TTS ₂ in dB | 21.5 | 3.2 |
| 11 Experimental TTS ₂ in dB | 23.0 | 7.9 |

DISCUSSION

As per results obtained one is completed to believe the conclusion made by Price and Oatman in 1967 that the involvement of higher centres in determining post exposure TTS is an artifact, Smith and Loeb in 1968 report similarly. In their experiment also the subjects were given a paper and pencil task.

Nonetheless one cannot overlook the other side of the coin. It is an already stated fact that the differences in test results arise due to factors such as:

1. Methodological discrepancies (Hish and Ward, 1952; Loeb and Fletcher, 1963; and Many more).
2. The type of mental problem per se, subjects were exposed, Smith and Loeb in 1968 and Capps and Collins 1965 state that the order of difficulty of the mental task administered is a crucial factor in determining TTS. Taking these two points into consideration one can still justify the results of Rawdon and Smith, 1936, Wernicke and Tobias 1963; and others, who state in favour of such a phenomena.

Nevertheless the issue of central factor's influence on the post exposure threshold shift may continue to intrigue many an experimenter before any inference is made regarding this topic assertively.

SUMMARY AND CONCLUSION

The present study was aimed at finding out if there exists any sort of influence of higher centers on the amount post exposure threshold shift.

The GSI-16 audiometer with TDH-39 earphone coupled to a supra-aural earcushion was used. The audiometer was calibrated as specified by ANSI 1969. 10 normal hearing subjects were chosen. Their pre tone air conduction thresholds at 4KHz were obtained at first. They were then exposed to a fatiguing stimulus of 2KHz pure tone at 100dB HL for 10 minutes in two types of situations. The first time they were exposed to the tone under a state of mental reverie. The second time they were asked to solve a paper and pencil arithmetic problem. There was a minimum gap of 2 days before the subjects were tested for the second condition. In both the conditions TTS_0 and TTS_2 at 4KHz were measured.

Thus, it can be concluded that:

1. There is no statistically significant difference between TTS_0 obtained in the first condition and that obtained in the second condition.

2. There is no statistically significant difference between TTS_2 obtained in the two conditions.

The results show that there is no role of central factors in the amount of post exposure threshold shifts thus TTs can be viewed as a peripheral phenomena primarily.

Suggestion:

It may be worthwhile to think a more foolproof methodology to carryout this experiment which at this stage seems to be the major hurdle in solving this age old enigma.

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