Changes in SOAE and Puretone Threshold After Exposure to Narrow Band Noise

Register No.M9911

This Independent Project submitted as part fulfilment for the
First Year M.Sc., (Speech and Hearing),
submitted to the University of Mysore, Mysore.

ALL INDIA INSTITUTE OF SPEECH AND HEARING
MYSORE 570006

MAY 2000

Dedicated to

Mataji, Pitaji,
Mummi and Papa.

CERTIFICATE

This is to certify that this Independent Project entitled : Changes in SOAE and Puretone Threshold After Exposure to Narrow Band Noise of Master of science (Speech and Hearing) of the student with Register No.M9911.

Mysore
May, 2000

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CERTIFICATE

This is to certify that this Independent Project entitled : Changes in SOAE and Puretone Threshold After Exposure to Narrow Band Noise prepared under my supervision and guidance.

Mysore
May, 2000

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DECLARATION

This Independent Project entitled : Changes in SOAE and Puretone Threshold After Exposure to Narrow Band Noise is the result of my own study under the guidance of Mr.Animesh Barman, Lecturer in Audiology, 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

May, 2000 Reg. NO.M9911

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हे शिक्षक तुम वृक्ष की भाँति हो
जो स्वयं धूप में रहकर सबको देता है छाया
आपकी अमूल्य सहायता के बल पर
यह संपूर्ण कार्य है हो पाया
धन्यवाद शब्दों में दे दूँ
ऐसा संभव नहीं किसी क्षण
आदर और सत्कार की माला
कृतज्ञ भाव से करूं मैं अर्पण

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INTRODUCTION

Noise is defined as "unwanted sound with more or less random disturbance" (Robert and Ycung, 1957).

Noise is a random frequency current or voltage signal extending over a considerable frequency, spectrum and no useful purposes, unless it is intentionally generated for test purposes (The illustrated dictionary of Electronics, 1980; Ruter and Turner).

The fact that noise can be detrimental to auditory sensory system has been known for centuries. The effects of noise on hearing may be temporary or permanent

The most found anatomical changes in cochlea are as distortion of outer hair cells and direct mechanical distiuctions and metabolic exhaustion at high intensities (>125 dB). White noise exposures causes damage in upper basal and lower second turn. With increase exposures the damage zones spread equally basal ward and apical ward. Narrow band exposures with centre frequencies (250 to 8 kHz) lead to tonotopical localization of damage area.

The susceptibility to acoustic traumatic inner ear damage probably varies among different species. Upto 90 dB, the critical intensity level, no damage is produced practically, from 90 to 130 dB, permanent acoustic traumatic damage of metabolic type and above 130 dB, severe irreversible structural damage is unavoidable (Spoendlin, 1971).

The basic physiological characteristics of peripheral auditory system are as follows:

a) *Threshold and Tuning* - OHC loss elevates the thresholds of neurons associated with the region of damage. Damage to stereocilia on OHCs or the loss of OHCs typically results in elevation of threshold in the tip of tuning curve and sometimes improvement in the threshold in tail of tuning curves. By contrast damage to stereocilia on OHCs results in an increase in threshold in both tip and tail of tuning curve resulting in little loss of tuning.

b) *Intensity Coding* - When threshold is elevated by a noise exposure the dynamic range between threshold and uncomfortable sound levels is reduced. This is associated with loudness recruitment.

c) *Temporal Coding* - Acoustic over stimulation leads to breakdown in temporal integration so that hearing-impaired listeners show relatively little improvement in threshold.

Theoretical and practical interest to audiologist in study of TTS:

1) The similarities between TTS, auditory adaptation, Noise induced permanent threshold shift (NIPTS) indicate that the anatomical and physiological processes which underlie may be differentiated qualitatively.

2) TTS may be used effectively to study the auditory fatigue and related phenomenon 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 SN loss.

4) A series of clinical studies on TTS have attempted to evaluate the predictability of NIHL a ad to state some damage risk criteria (Ward, 1970).

There are various tests to assess the effect of noise on cochlea. These include tonal audiometry, SDS, reflexometry, threshold octave masking, aural harmonics etc. Though these tests show significant different results between normals and subjects with noise exposure, these tests have their own limitations.

It is intuitively obbious as various investigators point out that a person with a high pre-exposure hearing level has less hearing to lose and will not show much TTS as a person with good hearing.

The primary factors influencing the development of TTS are spectrum, intensity, duration and temporal patterns.

There are many factors of variability in tonal audiometric procedure eg. physical variables (calibration, ambient noise, positioning of headset etc), physiologica1 variables (pathological conditions, tinnitus, TTS etc.), psychological variables like motivation, attitude, personality etc. The responsibility to demonstrate the real extent of hearing-

impairment may become a very difficult task if patient is uncooperative. Practical precautions are necessary in performance of basic audiological tests in order to avoid loudness perceptive references.

As far as noise being a hazard to the organ of hearing, studies of TTS are considered by some to be of academic interest because (1) no significant direct life long tests have been conducted with the same individuals (2) susceptibility to TTS and NIPTS in some animals was not significantly correlated (Ward and Nelson, 1970).

Thus the use of procedures is required which ensure the detection, identification and quantification of any simulation with a reasonable degree of certainity One such procedure is use of OAE measurments.

Otoacoustic emission (OAEs) are sounds generated within the normal cochlea, either spontaneously or in response to acoustic stimulation. Absence of outer hair cells (OHCs) is associated with a lack of OAEs, supporting the hypothesis that OHCs are responsible for OAE generation. Numerous observations support the cochlear origin of OAEs.

1) OAEs are independent of syr aptic transmission and are preneural.
2) OAEs are unaffected by stimulus rate, unlike neural responses.
3) Evoked OAEs are frequency dispersive (i.e. the higher the emission frequency, the shorter its latency) and their amplitudes grow nonlinearly with stimulus level.

4) OAE tuning or suppression curves are very similar to psychophysical and 8th nerve tuning curves.
5) OAEs are vulnerable to noxious agents such as ototoxic drugs, intense noise and hypoxia which are known to affect the cochlea.
6) Finally they are absent high frequency regions with cochlear hearing loss greater than 40 - 50 dB and present where hearing sensitivity is normal.

There are 2 basic OAE phenomena spontaneous OAEs and evoked OAEs. Spontaneous OAEs occur in the absence of external stimulation and evoked OAEs occur during or after the external stimulation.

SOAE consist of narrow band signals that can be measured in the absence of deliberate external stimulation.

Characteristic frequency range of SOAE is 0.5 to 6 kHz, highest incidence is between 1 and 2 kHz however these emissions upto 9 kHz have been reported in human subjects. SOAEs consist of one or several sinusoid like signals,small broadening of spectra of SOAEs are likely due to small oscillations and shfits of their centre frequency.

SOAE amplitude ranges from -16 to 20 dB SPL. In case of more than one emissions or multiple SOAEs the difference between two emissions is no less than 50 Hz.

SOAE is associated with frequency region where there is preservation of hearing sensitivity (>15 dB HL). They are absent if

hearing loss is more than 25-30 dB HL however it may be detected in some ears with mild sensorineural loss when the case has hearing loss more than 25 dB HL.

Infants may have a somewhat lower prevalence of SOAEs however in older subjects SOAEs may be eliminated due to elevated hearing thresholds. It has beet found that women exhibit SOAEs more than men. SOAEs are vulnerable to cochlear insults known to effect OHCs namely hypoxia, ototoxic drugs and noise.

All these factors effect the microstructure of cochlea. As mentioned above, there are several methods to find changes occurring in cochlea SOAE is one among them. Hence this study has been taken up with the following aims :

1. To find out the amount of SOAE amplitude and Puretone threshold (PTT) shift after exposure to narrow band noise.

2. Comparision of SOAE amp itude suppression vs. PTT shift.

3. To compare the sensitivity of SOAE to that of puretone audiometry in monitoring cochlear changes.

REVIEW

A number of stud es of acoustic trauma have been published which attempt to make a distinction between direct mechanical damage to the cochlea and pattern of damage.

Cochlea is more vulnerable to noise exposure Hawkins et al. (1976) found the sensorineural degeneration due to noise focussed on first quadrant of basal turn for intermittent noise. Continuous noise damages two quadrants i.e. region between 9 and 13 mm characterised by dip at 4 kHz. Minute black droplets were found in cochlea in scala vestibuli and scala tympani indicating presence of lipid, osmoiphi substances by Lipscomb et al. (1977).

Organ of corti is most vulnerable to higher intensity bombardment. Damage depends on frequency of stimuli, with high frequency damaging base and low frequency sounds damaging the apex. However Bohre (1976) found that damage can be more wide spread than one would expect baa d on travelling wave theory.

The Reissner's Membrane may distend or bulged into scala vestibuli, throughout the cochlea or may collapse in some parts of cochlea (Lipscomb et al. 1977). They also reported that more frequently damage is seen in 3rd row of outer I hair cells decreasing towards inner hair cells. According to Liberman and Beil (1978) most of the threshold shift in the noise exposed ears said to be accounted for by loss or damage to sensory cells.

Vasculation in supporting cells of hair cells including cells of Henson was reported by Engstrom et al. (1976) Tectorial membrane is lifted up from organ of corti in its damaged area. Ward-Duvall in 1971 observed occasional rolled up tectorial membrane surrounded by thin layer of cells.

Hawkins et al. (1971) found marked constriction of lumen often blocking the passage of red blood cells. Constriction is due to swelling of endothelial cells. Lipscomb et al. (1977) observed common vacuoles in strivascularis, condition present in all turns in localized but most common in 3rd turn and in apira1 parts. The epithelium of striavascularis is separated from spiral ligament.

Selters (1964) differentiated long term temporary threshold shift to intermediate one. The later is produced by sound intensities up to 60 to 70 dB SPL, lasts longer than a second and disappears within a couple of minutes. Furthermore intermediate TTS when induced by tones should be most prominent when F=Fe (Fe=frequency of exposure) whereas long term TTS is produced mainly at frequencies which are 1/ 2 an octave to an octave higher than the exposure frequency.

The conventional puretone audiometry is the most straight forward, rapid and scientifically tried and tested quantitative method to obtain information on an individuals hearing function. Thus any changes in the hearing susceptibility can be identified using this.

Hearing loss and inner ear damage after exposure to tones of high intensity was assessed by Dolan, et al. (1975) with tones of 125 Hz,

1 kHz, 2 kHz, 4 kHz at SPLs in range 120-157.5 dB for duration of one hour at 1, 2 and 4 kHz and for 4 hours at 125 Hz. exposure to 4 kHz produced damage in a restricted region of cochlea and hearing loss for a relatively narrow range of requencies. Exposure to 125 Hz produced widespread inner ear damage and hearing loss throughout the frequency range of 125-6000 Hz. The discrepancy between the damage to cochlea and hearing loss at 125 Hz and at 4 kHz may be due to the difference between the exposure duration.

While deriving the audiological profile of NIHL Cooper and Owen (1971) found that the tonal thresholds increased systematically from approximately 7 dB at 250 Hz to 22 dB at 8 kHz.

Test of basic audiometric battery may not indicate the full extent of auditory dysfunction related to noise induced hearing loss as suggested by Robert (1976). Tests of adaptation and discrimination of speech under adverse listening conditions may reflect the auditory changes more accurately.

James et al. (1977) tested the thresholds at 5,2,5.7 and 8 kHz after an exposure of an octave band noise centred at 4 kHz and observed that the threshold shift reached asymptomatic level after the Ist or 2nd exposure having a greatest shift at 5.7 kHz.

According to Charles et al. (1978) high frequency hearing loss produced by low frequency noise and that noise bands matched within 1 dB A were not equally hazardous as indicated by damage risk criteria.

Lessened ability of cochlear system to integrate acoustic energy over longer durations in cochlea r impaired auditory system was observed by Chung et al. (1980). In 4 kHz region the difference between the amounts of temporal integration of 2 groups (NIHL and normals) is less in the masked conditions. Subjects with NIHL showed less temporal integration than normals. Frequency effects were present in both groups.

Botte et al. (1994) described short term effects of tone exposure (1000 Hz, 90 dB for 15 minutes) and ITS was measured for 60 phone test tone. The TTS was found greatest above the test frequency. Maximum TTS (17.8 dB) occurred at 150 sec. post exposure at 0.6 octave above the exposure frequency.

An experimental data introduced by LePage (1987, 1990) shows that the CF of each portion of cochlear partition can vary as a function of cochlear condition, especially on account of OHC loss. It is most likely that the cochlear partition stiffness depends on OHC tonus, which reflects motility of OHC especially because the OHC turgor depends on cochlear condition (Brownwell et al. 1985).

A model proposed by Goldstein (1990) is able to predict most of the cochlear phenomena that are related to suppression. The model is described as nonlinear mixing between a sensitive compressive band pass filter and an intensive linear like low pass filter. The suppression phenomena are predictable because of nonlinear mixing.

Fatigue frequency related changes of SOAE (i.e. no or slight modifications with fatiguing; tones higher or close in frequency to SOAE and strong modifications with much lower fatiguers) are the main results, obtained from SOAEs fatiguing experiments.

The SOAE suppression tuning curves and fatiguing frequency dependent effects may involve different peripheral mechanisms and structures.

Ruggero et al. (1982) found the suppression of SOAE at 7529 Hz and 16 dB SPL by an external continuous tone. A 3 dB isosuppression curve was broadly displaced, relative to SOAE. towards higher frequencies. An audiogram notch exists at frequencies just below that of the SOAE.

Firtz (1983) found that following experimental sound exposure, SOAEs disappear with fatiguing stimuli of sufficient energy and duration, then emission return gradually to their original amplitude. During the development of this process the frequency of emission usually shifts downwards and then slowly moves back to original value. The extent of this frequency shift is quantitatively similar to shift determined psychophysically (in subjec tive binaural frequency comparison), under identical exposure conditions. The disappearance of the original emission may coincide with the appearance of SOAEs in an adjacent frequency range in which emissions are rarely seen. Like in subjective tests, the main effect of sound exposure occurs 1/2 to one octave above the exposure frequency.

It was suggested that following a brief noise exposure, changes occur in the cochlear partition especially in the outer hair cells, which cause both ITS and temporary alternation in SOAE (Norton et al. 1989).

In the early stages, the hearing loss is reflected by SOAEs is less severe than the cochlear pathologies identifiable with traditional subjective and objective methods (Ruggero et al. 1983).

Effects of noise exposure on the threshold microstructure near an SOAE and on the amplitude and frequency of SOAE as reported by Furst et al. (1992) are as follows :

a) The exposure to wide band noise for a short time causes a temporary reduction in SOAK frequency and amplitude and alters reversibly, the threshold microstructure invicinity of SOAE.

b) The threshold at SOAE frequency is most sensitive to noise exposure.

c) Intensive stimulation causes a relatively small increase, or even a decrease in threshold at frequencies near SOAE.

It was demonstrated by Kemp (1982), Norton et al. (1989), that following exposure to intense acoustic stimuli SOAE amplitude and frequencies are reduced, and they return to their original values in a time course similar to that for behaviour threshold recovery.

Auditory fatigue can be considered a suitable test to assess some cochlear mechanisms and diseases otherwise not easily detectable since spontaneous OAEs originate from active sources within cochlea. Cinanfrone, et al. (1993) show sensitive and early vulnerability to noise, displaying informative time courses after over stimulation in the short (0-6 sec) and in the long term (1-10 min) depending on the frequency of fatiguing tone.

Above review indicates that SOAEs could be an important tool to monitor auditory fatigue. It is imporant to do an extensive study to see how the short-term or long-term exposure to either wide band noise (WBN) or narrow band noise (NBN) can affect the SOAEs. However, effect of NBN on SOAEs has been carried out. The present study is designed to see the affect of NBN on SOAE and also to see whether SOAE could be a sensitive tool to predict individuals who are more susceptible to loss due to noise exposure.

METHODOLOGY

The aim of this study is to compare the pre and post noise exposure SOAE measurements a ad to find its sensitivity against puretone threshold measurments.

A. Subjects

Comprised of 33 adult volunteers (45 ears) age 17 to 22 years (average age 20.5 years). Subjects were selected based on following criterions:

(i) History : The selected subjects had no history of otological problems such as hearing loss, ear pain, ear discharge, exposure to noise etc.

(ii) *FTA* : All the subjects had puretone thresholds within 15 dB HL in the frequency range from 250 to 8000 Hz. The testing was done using Madsen OB 822 audiometer and TDH 39 earphones housed in MX 41 AR. The bone vibrator used was B-71.

(iii) *Middle ear pathology* was ruled out by performing immittance audiometry using GSI-33 (version-2). All the subjects had 'A' type tympanogram with reilexes at normal level.

(iv) Individuals with presence of SOAE either in one ear or both the ears were taken for study. The SOAE measurement was carried out using Madsen Celesta 503. Difference between amplitude of SOAE

and noise floor being more than 3 dB was taken as criterion for SOAE to be present (Lonsbury et al. 1990).

B. SOAE measurement

The system parameters considered were as follows:

1) *Maximum frequency :* frequency up to 10000 Hz. was selected.
2) *Prg sweep :* The number of repetitive samples were 500.
3) *Rej sweep* : The number of samples rejected on the basis of S/N level criterion was 10% of prg. sweep.

C. *Procedure*

i) *Test Environment*

Testing was done in a well illuminated sound treated room with permissible noise levels as per the reference given by ANSI-S3-1-1977. The subjects were provided with comfortable chair to sit during the test.

ii) *Instructions*

The subjects were given standard instructions before performing PTA, immittance or SOAE measurements. During SOAE measurments subjects were told not to move head. Being an objective test the subjects were not required to perform any task.

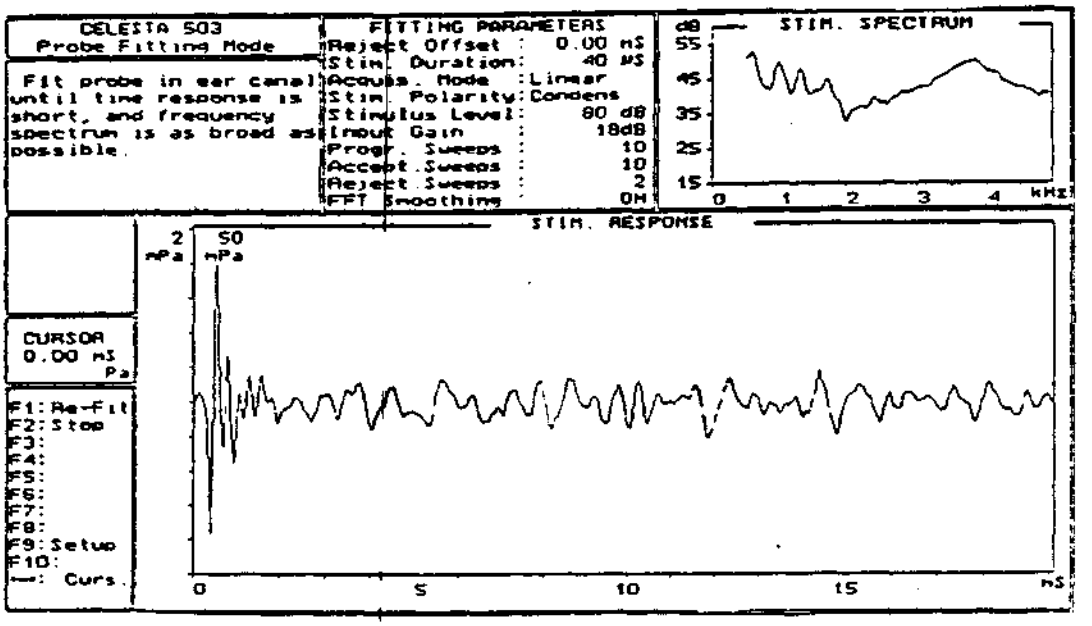
iii) *Probe Insertion*

Ear canal examination is done prior to insertion of probe during immittance or SOAE measurement. Ear tip of suitable size for the ear being tested was located, placed *i* nd then inserted into patient's ear canal, to obtain an air tight seal. A proper tip and good probe fit was obtained prior to the study by introducing short transient click stimuli to the ear canal of the subject. Its power spectrum is level from 1 to 5 kHz which is the limit of probe design. Figure (a) shows an acceptable profit.

(iv) *Measurement of SOAE*

Following the proper probe fit baseline SOAEs were measured. Subsequently, all the subjects were exposed to narrow band noise for 5 minutes at the specific frequency which was around one octave below the frequency at which SOAEs were present for a particular subject. Noise introduced was one octave below the SOAE frequency since it has maximum effect on SOAE (Selters, 1964; Lenoir et al. 1985).

The noise was presented through TDH 39 earphone in cases having SOAE present in one ear and through insert receiver for those who had SOAEs present in both the ears as to minimize the effect of noise due to cross over. Intensity and duration parameters were kept constant for all the subjects. SOAE measurement was repeated immediately after the noise expos ore to NBN which were later compared with baseline SOAEs.



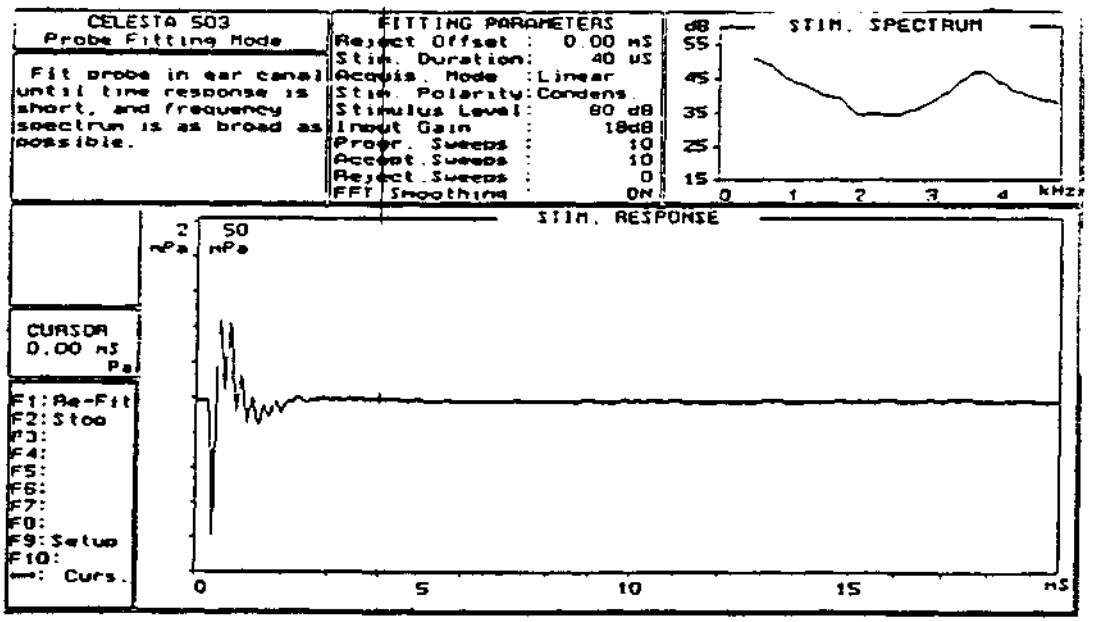


Fig.a(ii) Shows an acceptable probe fit

SOAE test results were displayed on an amplitude spectrum. The peaks identified and marked as spontaneous emissions were recorded in tabular format at the top of the screen.

In cases where multiple SOAEs were present at pre exposure state, the noise presentation criterion was similar to that in cases with single SOAEs.

(v) *Puretone Threshold Measurement*

Baseline hearing thresholds around the frequencies at which SOAEs were present, were obtained after one month for the same ears using puretones. The tracking method used was 2 steps down and one up i.e. decrease by 2 dB and increase by 1 dB.

Narrow band noise around one octave below the frequency at which SOAEs were present vas introduced to subjects ears. Pre and post exposure hearing thresho ds were determined around the frequency where SOAEs were present for a particular subject.

(vi) *Analysis*

Pre and post exposure SOAE responses as well as puretone thresholds were tabulated and T-test was used to find any significant difference between the mean SOAEs and puretone thresholds obtained during pre and post exposure condition. Spearman's rank correlation method was used to find the correlation between amount of suppression in SOAE and in puretone threshold shift after the exposure of noise.

RESULTS

In the present study SOAEs were studied as baseline and post exposure to noise. The collected samples were analysed and descriptive statistics and results of 't' test *were* obtained.

1) *Relationship between baseline SOAEs* ***and the*** *post exposure SOAEs*

Out of 62 baseline SCOAE recordings 67% SOAEs were found to be absent after exposure to noise. Amplitude was reduced in 17% of SOAEs whereas the amplitude remained same in 8% and increased in 8% after noise exposure.

The frequency ranges of SOAEs obtained pre- and post-exposure state are 941-8455.9 Hz and 941-8418 Hz respectively with maximum number of SOAEs (64.5%) falling between 3-4 kHz. SOAEs were found to be more in female subjects with male to female ratio of around 1:6.

One of the recording samples of baseline SOAEs and post exposure SOAEs are illustrated in figure b and c.

The amplitudes (signal-to-noise ratio) of SOAEs obtained at pre and post exposure range from 3-14 dB SPL (S/N ratio below 3 dB was considered as absence of SOAE) with an average of 5.5 and 5.2 respectively. The enhancement of amplitude ranged from 1-2 dB SPL (average 1.6 dB SPL) reduction ranged between 1 to 10 dB SPL (average 5.4 dB SPL).

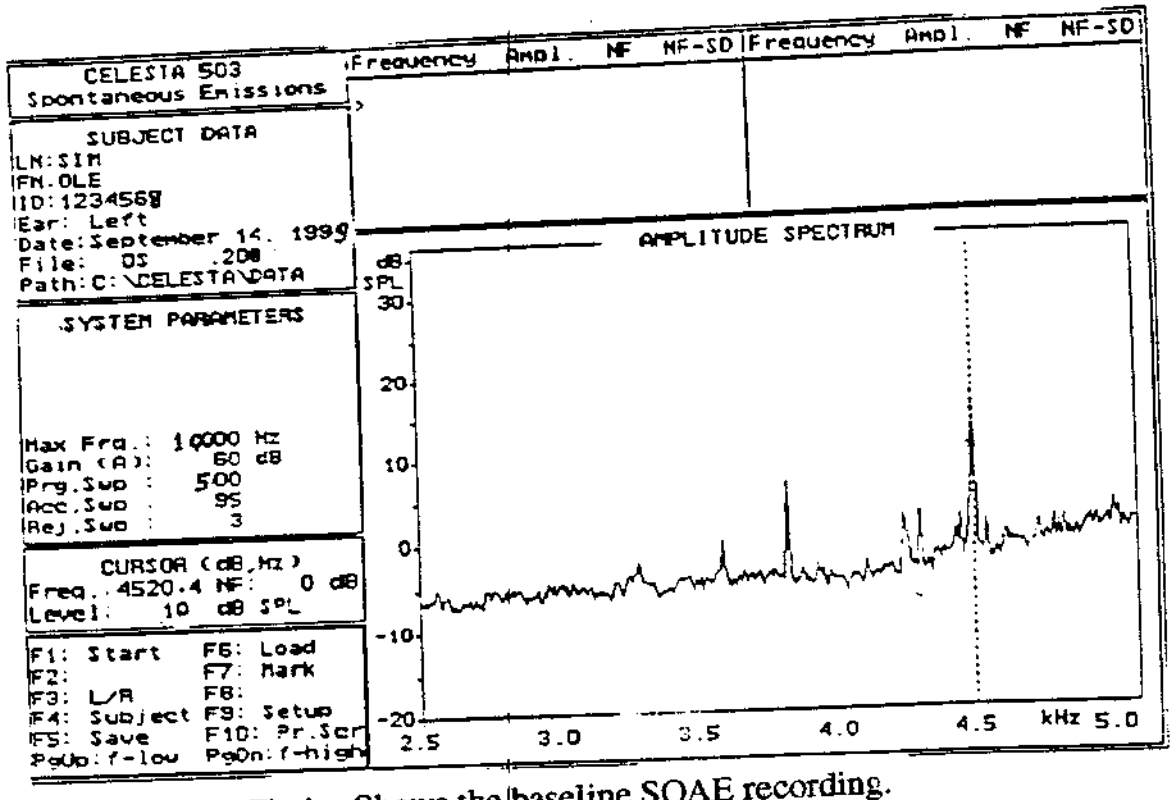


Fig.b : Shows the baseline SOAE recording.

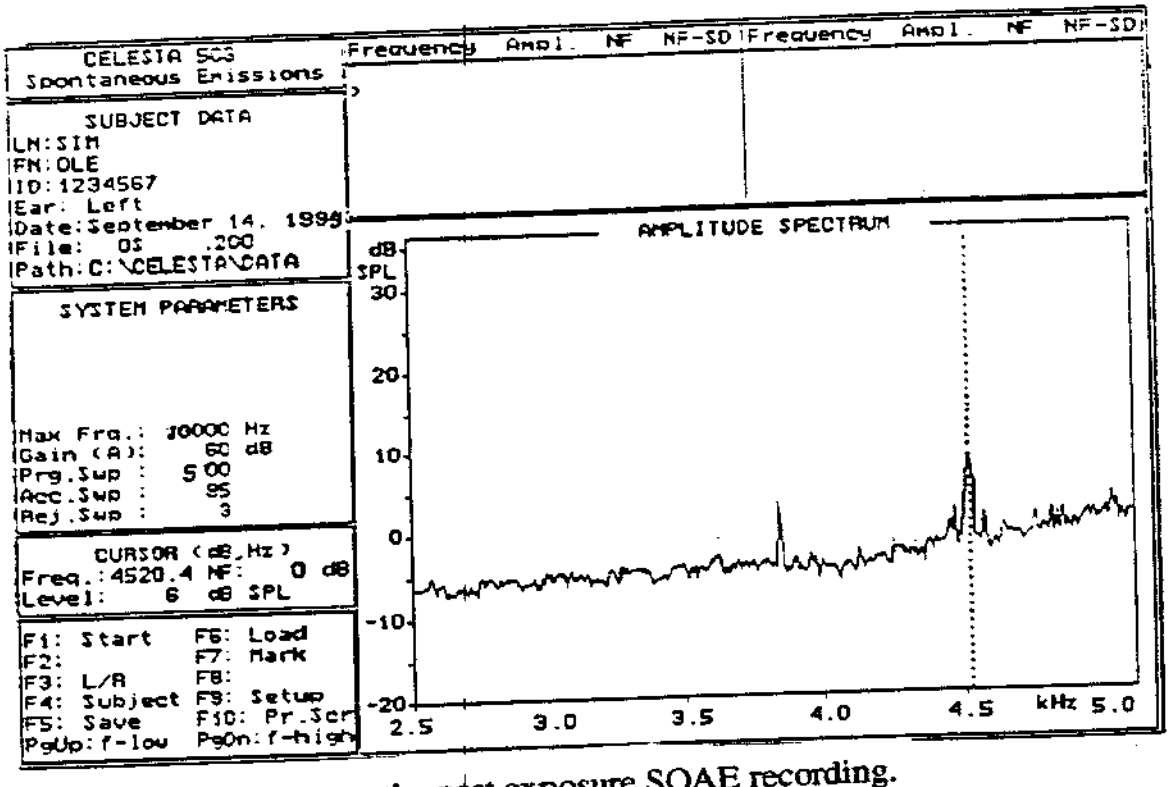


Fig.c : Shows the post exposure SOAE recording.

The difference in amplitude of pre and post exposure was statistically significant (P:<:.001) (HS) when taken into account those SOAEs which were absent (i.e. difference less than 3 dB) and amplitude was considered as zero. Means of pre and post exposure SOAEs amplitudes were found to be 5.53 and 1.69 respectively. There was no significant difference between the standard deviations of both pre and post exposure recordings in terms of amplitude (Table 1).

Table 1: Depicts the mean. SD and t-value of pre and post exposure SOAE amplitude.

	Mean	SD	t-value
Pre	5.53	2.82	
			10.17
Post	1.69	2.92	

When considering only those SOAEs (20 recordings) which were present post exposure wi*th* reduced amplitude, the 't' test indicated significant difference between the 2 means (pre and post exposure SOAE amplitudes). (P:<.05) (S). Means of the pre and post exposure SOAE amplitudes were 6.9 and 5.29 respectively. However, standard deviation varied i.e. standard deviation of pre exposure SOAE amplitude is more (Table-2).

Table-2 : Depicts the mean, SD and t-value of pre and post exposure SOAE

	Mean	SD	t-value
Pre	6.9	3.52	
			6.60
Post	5.25	2.78	

Along with the reduction in amplitude of SOAEs, downward frequency shifts were seen in 9 post exposure SOAEs which were more often in cases with enhancement of amplitude of SOAE or where SOAE amplitude remained same after exposure to noise.

2) *Relationship between baseline puretone thresholds to post exposure puretone thresholds :*

Puretone thresholds were measured at a particular frequency around which SOAEs were present before and after the exposure to noise. The data was analysed and t-test results were obtained.

Out of 45 ears 40 ears showed elevated puretone thresholds, in 3 ears the threshold remained some and in 2 ears thresholds were found to be improved. The elevation was found to be more in those subjects who had lower thresholds levels at pre exposure level.

The means of pre and post exposure puretone thresholds were found to be 5.45 and 9.70 respectively. There was not much difference between the standard deviations of pre and post exposure SOAEs (Table-3).

Table-3: Depicts the mean, SD and t-value of pre and post exposure puretone thresholds.

	Mean	SD	t-value
Pre	5.45	5.55	
			6.60
Post	9.70	5.38	

The difference in the mean of pre and post exposure thresholds was found to be statistically significant (P:<.001) (HS).

3) ***Correlation between SOAE amplitude suppression and puretone threshold shift after exposure*** *to* ***noise*** *:*

A positive correlation was found between the difference threshold (pre and post exposure puretone threshold difference) and difference amplitude (pre and post exposure SOAE amplitude).

The mean elevation in puretone threshold was found to be 5.1 dB HL whereas mean reduction in SOAE amplitude was 5.4 dB SPL. The ranges of puretone threshold and SOAE amplitude shifts were found to be -4 dB to 12 dB HL an I -2 dB 10 dB SPL respectively.

It was observed that where there was more shift in thresholds, reduction SOAEs was also more. A correlation coefficient of .2134 was found between puretone threshold shifts and difference SOAE amplitude suppression. From the above mentioned findings it is clear that the mean suppressions of SOAE amplitude is more than that of threshold elevation.

DISCUSSION

The modifications of SOAEs after exposure justify that SOAEs are an expression of normal physiological processes and that they are sensitively and strongly vulnerable in conditions where clear psychoacoustic modifications r lay not be detectable. The data obtained in this study agrees with the previous observations on the vulnerability of SOAEs (Kempet al. 1982: Ruggero, et al. 1982; Fritz, 1983; Ruggero, et al. 1983: Norton et al. 1989; -Furst et al. 1992, Cianfrone, et al. 1993).

Several studies indicate cochlear emissions and outer hair cells (OHC) as the main active and vulnerable elements in the cochlea. It is still uncertain what kind of active processes in the cochlea are involved in generation of the various kinds of cochlear emissions. Probably the noise exposure affects the outer hair cells in such a way that their amplification gain is reduced. Reductions in amplifier gain causes a decrease in cochlear partition displacement as a response to certain stimulus level.

In subjects where SC AEs remained same or improved could be attributed to the possibility that SOAEs obtained in such cases did not fall in the band width of frequency which is one octave above the exposure frequency of narrow band noise as all the subjects had downword shift in SOAEs after the exposure. This is against the expected fact that the SOAEs get suppressed at one octave above the frequency of exposure. Downward shift in SOAE frequency in TTS situation has been previously reported by Firtz (1983).

Brief intense stimilation generally causes a temporary increase in threshold with considerable intrasubject variability. Noise exposure causes a threshold increase to defend the cochlea from exaggerated motion as a result of over stimulation. Elevation in thresholds were reported by various observations previously (Dolan et al. 1975; Cooper etal. 1976; James, et al. 19 77; Charles et al. 1978; Chung, et al. 1980; Botteetal. 1994).

Similar results were obtained in this study. However, the frequency the tested for threshold estimation was approximately the frequency at which SOAEs were present for a particular subject. The SOAE frequency was chosen to test the effect of noise since threshold at SOAE frequency is most sensitive to noise exposure as reported by Furst et al. 1990).

The elevation in thresholds being more in subjects with lower pre exposure thresholds can be attributed to the facts that a person with a high pre exposure hearing level has less hearing to lose and will not show as much TTS as a]person with good hearing and the cochlear impaired auditory system has lessened ability to integrate acoustic stimulation over longer durations (Chung et al. 1980,1982).

A comparision of SOAE with audiometric data particularly with puretone audiametry shows that SOAEs tend to occur in ears with minor hearing loss (Maximally 20 to 25 dB at the corresponding site in the audiograms.

Comparison of SOAE recordings to that of audiometric findings was done and the results indicated that in cases with more suppression of SOAE had higher elevation in thresholds also. Thus there exists a positive correlation between the two. This means that the SOAEs can be used as a substitute for puretone measurement. The mean suppression is found to be more in SOAE compared to puretone threshold shift. Thus SOAE can be regarded as a more sensitive measure to detect minor changes in cochlea. This could be due to the fact that SOAE amplitudes are more prone to changes due to minor changes in the OHC whereas PTT may not show any change.

Clinical applications

At present, the biological and clinical significances of SOAEs are not entirely known. The derection of SOAEs in human ears makes it likely that a true amplification process takes place within the cochlea.

SOAEs appear to be an expression of minor cochlear damage. In the presence of minor hearing loss, synchronization, which is essential in normal hearing, occurs spon :aneously (Lenoir et al. 1985). In early stages, the hearing loss reflected by SOAEs is less severe than the cochlear pathologies identifiable with subjective and objective methods. Consequently SOAEs can be expected to increase the sophistication of audiologic test (Ruggero, et al. 1983). SOAEs may have a dual benefit it can help to confirm known phenomena of inner ear research and are likely to produce new insights to the field.

SUMMARY AND CONCLUSION

Otoacoustic emission recordings show great promise for the objective demonstration of cochlear pathology. The nature of OAEs are unknown. Are they reflective of some normal auditory mechanisms or an expression of some sensorineural disease? It appears that missing OHCs could create conditions that result in SOAEs and thus it is feasible that "pathologic" SOAEs mi ght coexist with "normal" SOAEs. Possibly patterns of SOAEs recorded luring processes that are unknown to damage the cochlea would distinguish between emissions due to irregularities in OHC distribution patterns Various cochlear pathologies which are known to cause micromechanical changes include hypoxia, Mienere's disease, intense noise, ototoxic drugs, etc.

Hence the present study was taken up with the aims :

1) To find the amount of suppression of SOAEs after noise exposure
2) To find the amount of puretone threshold elevation following noise exposure.
3) To compare the sensitivity of both the procedures to monitor the cochlear changes.

45 normal ears from 33 normal subjects were included in the study. SOAE testing using celeesta 503 and behavioural audiometry using Madsen OB822 was carried out for all the subjects. Both the measurements were carried out pre and post exposure to the noise (a narrow band noise of 90 dB SPL with frequency approximately

corresponding to one octave below the frequency of SOAE for a particular subject). Behavioural thresholds were measured at the SOAE frequency.

The following results were obtained.

1) *SOAE suppression* : Reduction in amplitude or complete disappearance of SOAEs was found in 85.4% of the ears and 14.6% of the ears showed same or impro\ed SOAE amplitude. T-test indicated significant difference between the means of pre and post exposure SOAEs amplitude.

2) *Puretone threshold elevation :* Elevation of thresholds around SOAE frequency was found in 88.8% of the ears, whereas 11.2%, ears showed no elevation or inproved thresholds. T-test showed significant difference between the pre and post mean exposure thresholds.

3) *Comparision of sensitivity of SOAE and puretone threshold: A* positive correlation between mean SOAE suppression and mean threshold elevation was found using the spearman's correlation coefficient However, the mean suppression was found to be more in SOAEs compared to behavioural thresholds.

Thus the above mentioned results suggest that post exposure to noise which is known to shift PTT also can affect SOAE amplitude. Hence SOAE can be used as an objective tool to detect an individual who is more susceptible to hearing loss due to noise exposure. The minor cochlear changes due to noise exposure which may not be detected

by the behavioural thresholds measurements in routine examination procedure in routine examination procedure may show some changes in SOAE amplitude. Though SOAEs have certain limitations for eg. these are not present in all the normal ears and in cases with frequency shifts of SOAE in a post exposure condition where the amplitude parameter may not suggest presence or absence of any cochlear involvement, it has many clinical applications. It is an indicator of hearing function. The results of this study suggest that a healthy ear is necessary for the expression of SOAEs. Moreover SOAE recordings could be considered as an objective, easy, non-invasive and very rapid (less than 40 sec) audiological procedure. Therefore SOAEs can be used as reliable technique for studying objectively the normal micromechanical activity within the cochlea.

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