

Sensitization and Desensitization Effects in TEOAEs

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Jinsook Kim · Junghak Lee. Sensitization and Desensitization Effects in TEOAEs. *Korean Journal of Communication Disorders*, 3, 84-104. Sensitization and desensitization effects in human transient evoked otoacoustic emissions (TEOAEs) were explored following exposure to brief pure tones. Their differential effects of frequencies, low, mid and high, and intensities, moderate and intense, were also carefully examined. Sixteen normal hearing young females, eight of them with one or more spontaneous otoacoustic emissions (SOAEs) and the other eight without SOAEs served as subjects. A repeated multivariate analysis of variance (MANOVA) was performed for statistical analysis. The general shape of the recovery pattern as a function of post-exposure time in TEOAEs demonstrated a sensitization effect between 20 seconds and 1 minute 40 seconds and a desensitization effect between 2 and 4 minutes. Both frequency and intensity of the exposure signal significantly differed in the magnitude and configuration of sensitization and desensitization. Depending on the exposure to pure tones, the time course was divided into the three types - the combination of sensitization and desensitization effects, only significant desensitization effect, and the initial sensitization effect. The characteristics and modalities of recovery pattern following the sound exposure are remarkably similar to the findings of behavioral sensitization and desensitization effects. This similarity suggests that in the nature of the motile activity of the cochlear partition, principally the outer hair cells are responsible for changes in OAEs and behavioral thresholds following the acoustic overstimulation. Additionally, the sensitivity of TEOAEs to intense sound exposure indicates that TEOAEs can be used for monitoring hearing changes following the sound exposure.

I. Introduction

When a sensory system is exposed to a prolonged, intense stimulation, it is generally expected that an adaptation, fatigue, or habituation of the system's processing abilities will eventually occur. Under certain conditions an exposure stimulus

can increase and decrease the responsiveness of a particular mechanism. These so-called sensitization and desensitization phenomena can be observed in the auditory system by means of hearing threshold tests, electrophysiological procedures, acoustic reflex measurements and otoacoustic emissions (OAEs).

In 1952 Hirsh and Ward reported the recovery of the hearing threshold following intense acoustic stimulation to be a complex and multiphasic process. They reported an initial rapid rate of recovery which continued up to about one minute post exposure suggesting "it may demonstrate facilitation, i.e., a temporary reduction in the absolute threshold below the normal value." The second decrease in threshold sensitivity was immediately followed in hearing sensitivity referred as the "bounce phenomenon" reaching a maximum by about two minutes after which a slower rate of recovery predominated. Subsequently, many investigators (i.e., Hughes, 1954; Noffsinger and Tillman, 1970; Noffsinger and Olsen, 1970) demonstrated better or worse hearing thresholds following a short term tonal stimulation by tones of appropriate intensity and frequency. Also, the pattern of recovery from such exposure can result either in sensitization, desensitization, or a complex multiphasic interaction of both.

The characteristics of sensitization and desensitization were dependent on the duration, intensity and frequency of the exposure signal. The greatest sensitization effects were observed when the exposure tone frequency was below the test tone frequency. The maximum sensitization often occurred at about 30 seconds when the test tone was lower in frequency than the exposure tone but the timing of maximum sensitization was shifted from 60 to 70 seconds when the test tone was equal to or higher in frequency than the exposure tone. After returning to baseline sensitivity, a small amount of desensitization is observed, especially for the higher exposure intensities (Hughes, 1954; Noffsinger and Tillman, 1970).

The increased responsiveness following the stimulation was also detected in the acoustic reflex in cats by Simmons (1960) and in humans by Deutsch (1972, 1973) and Ruth (1977). In contrast to the work of Deutsch, Ruth found that the post-exposure behavior was dependent primarily upon the intensity of the exposure sound. Additionally, a significant sensitization effect was noted following presentation of contralateral sound exposure. This led him to infer that central nervous system pathways common to both ears and within the realm of the acoustic reflex arc must

be responsible for sensitization.

Because similar but slightly less sensitization was produced with the contralateral stimulation when they compared the ipsilateral and contralateral exposure effects, the loci of this response was speculated to be partially central by many investigators in behavioral, acoustic reflex, and otoacoustic emission studies. For example, both the frequency and amplitude of the SOAEs changed following contralateral stimulation (Mott et al., 1989). The influence of a contralateral auditory stimulation on OAEs was also studied by many other investigators (Collet et al., 1990a, 1990b, 1992; Veuillet et al., 1991; Moulin et al., 1993). Although the researchers examined with different types of OAEs, such as SOAEs, TEOAEs, and DPOAEs, they commonly found a contralateral auditory stimulus effect on active cochlear micromechanics, probably OHCs, on which medial efferent neurons of the olivocochlear bundle terminate. Therefore, the medial efferent system, excited at brainstem level via the afferent auditory pathways, was noticed as the essential property for contralateral effects on SOAEs.

It is widely accepted that OAEs are produced by the movement of outer hair cells (OHCs) in the cochlea. The primary effect of exposure to loud sound was examined as one piece of evidence supporting the cochlear origin of OAEs because it is well-established that the outer hair cells (OHCs) represent the cochlear component that is primarily affected by excessive sound (e.g. Clark and Bohne, 1978). Several investigations revealed that all four classes of OAEs were influenced by excessive acoustic stimulation. In human studies, temporary threshold shifts (TTS) after relatively short exposures indicated reductions in the amplitudes of SOAEs and TEOAEs (Anderson, 1980; Kemp, 1981, 1982, 1986; Norton et al., 1989).

Otoacoustic emissions from human ears do exhibit a "bounce" phenomenon as seen in the threshold of hearing after brief moderate noise exposure (Kemp, 1981). This "bounce" phenomenon was mentioned by Hirsh and Ward (1952) in the recovery pattern for pure tone thresholds. Kemp (1982) also reported hearing sensitivity enhancement in the cochlear echo response about 1 minute after a brief loud sound which was followed by a trough and a second rise in acoustic output. This recovery pattern is remarkably similar to the psychophysical threshold recovery pattern reported earlier. He finally hypothesized that cochlear mechanical activity recovers somewhat

during stimulation and overshoots within a minute of cessation of stimulation after initial depression by strong stimulation. And the recovery contour of this mechanical activity demonstrates a clear link to the auditory function because the behavioral study shows the similarity of the recovery pattern in the threshold and loudness function.

Furthermore, Kemp (1986) attempted to describe a possible control system for cochlear mechanical activity using a model. In this experiment, he argued that the cochlea acts as a feedback system in order to hold the active elements (OHCs) in a constant condition. That is, the inflow of cellular energy is greater than normal during sustained strong stimulation, which allows the system to recover to near normal and enter a hyperactive state on termination of the stimulation. This notion was supported by reproducing "bounce" behavior utilizing his model. However, he added that this model did not give a complete picture of cochlear mechanical control dynamics. In conclusion, the author hypothesized (Kemp, 1988) that these otoacoustic changes mirrored functional cochlear mechanical disturbances. The fine setting and/or metabolic status of the outer hair cells had presumably been disturbed and traveling wave improvement has been reduced.

The systematic behavior of spontaneous otoacoustic emissions in humans was examined following presentation of brief, intense pure tones as a function of exposure frequency relative to SOAE frequency, exposure intensity, and duration (Norton et al., 1989). The authors found the time course of recovery for both frequency and amplitude was biphasic and remarkably similar to psychophysical threshold recovery patterns. Regarding an increase in SOAE frequency and/or amplitude relative to a baseline during the early rapid recovery phase, Norton et al. stated that it was similar to what earlier TTS investigators called sensitization which is an improvement in behavioral threshold 15 to 45 sec following exposure to brief pure tones or narrow-band noise. The authors also indicated that SOAE sensitization tended to be greatest following less intense exposure and occur for a wide range of SOAE and exposure frequency.

In the same experiment, they also found that the effects of SOAEs following exposure tones were highly tuned with exposures between $1/8$ and $5/8$ of an octave below the SOAE producing the maximum changes for both frequency and amplitude.

Exposure frequencies above the SOAE had little effect, and frequencies below 5/8 octave showed less effect than for frequencies closer to the emission. These findings are comparable to the so-called 'half-octave shift' in TTS behavior. TTS tends to have the most effect not at the exposure frequency but 1/2 octave or more above the exposure frequency (McFadden, 1986). The degree of tuning was dependent upon the post exposure time sampled. Additionally, the general SOAE effects increased nonlinearly with the exposure level and duration.

Ultimately, the similarity of the SOAE data and the effects of comparable pure tone exposures on behavioral thresholds led the authors to suggest a common underlying mechanism, outer hair cell, as the responsible region for both. This notion was supported by the data about the effects of pure tone exposure on click evoked otoacoustic emissions (Hayes and Norton, 1991) and SOAEs (Furst, 1992). They conjectured metabolic changes within the OHC as a factor for post exposure changes in SOAE characteristics and their subsequent recovery contour.

In noise exposure study of TEOAEs, Hotz et al. (1993) reported significant changes after exposure by firearms in military service. One hundred forty-seven normal hearing young men who were in obligatory military service in Switzerland were tested. The subjects were required to wear hearing protectors during exposure to firearms. The authors embarked on this study emphasizing the feasibility of using TEOAEs as an objective field procedure and sensitivity comparison TEOAEs with pure tone thresholds. While pure tone audiometry revealed no significant mean changes during the military service, all mean changes of TEOAEs were in the direction expected from cochlear damage. The results indicated that TEOAE testing may be more sensitive than pure tone audiometry in detecting early cochlear damage from noise.

The purpose of the present investigation was to systematically evaluate sensitization and desensitization effects in the human TEOAEs and characterize the frequency and intensity of exposure tones and the interaction of both. Although several investigators, as previously mentioned, reported sensitization or desensitization effects using TEOAEs, those were described as by-products of studies of cochlear mechanism and temporary threshold shifts in TEOAEs. Therefore, it is necessary that more data on sensitization and desensitization effects be collected on TEOAEs. In

addition, the analysis of the differential effects of exposure frequencies, including low, mid, and high, and intensities, including moderate and intense, will provide valuable information for the use of TEOAEs in scrutinizing the micromechanical activity of the cochlea and examining the noise exposed cochlea.

II. Methods

1. Subjects

Sixteen normal hearing young females with ages ranging from 21 to 29 years (mean age of 24.6 years) served as subjects. The subject selection was undertaken considering age and gender factors evidenced in the several investigations (Bilger et al, 1990; Probst et al, 1991; O-Uchi et al, 1994). Only one ear per subject was tested in the experiment. Eight of sixteen selected ears had one or more SOAEs and the other eight did not have SOAEs. All subjects had thresholds better than 10 dB HL at the standard audiometric frequencies between 0.25 and 8.0 kHz in the experimental ear. Middle ear function as assessed by clinical immittance procedures was also within normal limits. Also, the subjects had a negative history for long-term noise exposure, acute acoustic trauma, ear infections, ototoxic drug medication, sudden hearing loss, Meniere's disease, and hereditary deafness. Informed consent was obtained from all subjects prior to testing.

2. Instrumentation

TEOAEs were measured using the ILO88 hardware and software (Otodynamic OAE systems) controlled by a HAI, DATA STAR 486 computer. The level and timing of analog sinusoidal stimuli were computer controlled. The ILO B - type probe designed for adults was used.

The exposure was pure tone generated by a Grason-Stadler model 1705 audiometer and a Hewlett Packard model 3311A function generator. An insert earphone (Nicolet Model TIP-50) was used for the presentation of acoustic stimulation. Both

the probe and the tube of the insert earphone were housed in the disposable and pliable foam eartip and fitted in the individual ear. The duration of the stimulation was monitored visually with a stop watch.

The sound pressure levels and attenuator linearity were monitored prior to, periodically during, and after completion of the experiment by means of a Bruel and Kjaer type 2231 digital sound level meter.

3. Exposure Condition

The exposure intensity was 70 and 100 dB SPL representing moderate and intense levels. The frequency was 500 Hz for low, 4 kHz for high, and $\frac{1}{2}$ octave below the strongest frequency measured in the screening process for those who had pre-exposure SOAEs for the middle frequency. For those who did not have pre-exposure SOAEs, arbitrary 1500 Hz pure tone was presented for the middle frequency. As a result of these combinations, the following six exposure conditions were used in this study: (1) 70 dB SPL at 500 Hz, (2) 70 dB SPL at 4 kHz, (3) 70 dB SPL at $\frac{1}{2}$ octave below the strongest frequency or 1500 Hz, (4) 100 dB SPL at 500 Hz, (5) 100 dB SPL at 4 kHz, (6) 100 dB SPL at $\frac{1}{2}$ octave below the strongest frequency or 1500 Hz. The duration of the exposure to all these conditions was 90 seconds. Each of the sixteen subjects experienced the same conditions involving six different test properties.

4. Test Conditions

For OAE measurements, thirty sample number were averaged per spectrum which took about 8 seconds to complete one spectrum. The sample number thirty was chosen because that showed stable frequency and amplitude over days in the pilot data. All the other conditions except sample numbers used the default conditions of the Otodynamics OAE systems.

The TEOAEs were defined when they were at least 5 dB above the noise floor of the system and stable in both amplitude and frequency from the test ear. Also, the reproducibility of 50 % or above was included for the criteria. The minimum of three pre-exposure baseline spectra were obtained at the beginning of each experimental

condition. Post-exposure measurement commenced immediately upon exposure offset every 20 sec for the first 5 min, and then every 30 sec for the second 5 min. This resulted in tracking the recovery pattern for a total time of 10 min and twenty-six points during that time. As TEOAE measurements took about 8 sec (it was varied slightly according to the calmness of the subject), a period of about 12 sec for the first 5 min and 22 sec for the next 5 min, during which time no stimuli were present, followed and preceded every measurement.

The presentation order of the exposure conditions was counterbalanced for each subject. Only one high-level exposure (100 dB SPL) was done per session, with sessions separated by a minimum of one hour and a maximum of one week.

5. Procedure

Prior to the session, subjects were asked questions regarding their general hearing histories. The consent form was presented for their signature and an extra copy of the consent form was given to the subjects to keep.

All testing was accomplished in a commercial double-wall sound isolation room (Acoustic Systems). The electronic component was set a warm-up period of at least 15 minutes prior to initiation of the test.

Subjects were seated in the sound isolation room and examined bilaterally with an otoscope for determination of the presence of cerumen or tympanic membrane perforation. Pure tone audiometry and immittance screening was conducted consecutively. For determination of the existence of SOAEs, two recordings for each SOAE were performed. Subjects were instructed to remain as relaxed and quiet as possible during the test. The foam eartip was placed in the ear canal of the subject for the experimental measurements.

6. Repetitive Measurements

The repeatability of this study was examined with the selected exposure intensity and frequency. The exposure intensity was 100 dB SPL, the frequency was $\frac{1}{2}$ below the strongest frequency for those who had SOAEs and was 1500 Hz for

those who did not have SOAEs. Out of sixteen subjects, only eight subjects participated in the repetitive measurements. The eight subjects consisted of four randomly selected from the group who had SOAEs and four also randomly selected from those who did not have SOAEs.

III. Results

1. Characteristics of Pre-exposure TEOAEs

In the screening process, a sample number of 260 (default) and 30 were tested for all subjects. The mean TEOAEs obtained from all 16 subjects for sample number 260 and 30 were 13.4 (SD: 3.4) and 13.9 (SD: 3.3) dB SPL, respectively. The *t*-test between the sample number 260 and 30 failed to reach significance indicating that the sample numbers 260 and 30 were not different.

The pre-exposure TEOAEs were obtained at the beginning of each experiment with a minimum of three pre-exposure baseline spectra. Table 1 presents the data for the pre-exposure TEOAEs according to the 6 exposure conditions. As pre-exposure measurement was not affected by the exposure condition, the different exposure condition here means a different time set. Although the inter-subject variability was relatively high as evidenced by the standard deviation and range values recorded, the different time set seemed to have a minimal influence on the measurement. The group difference between the groups with and without SOAEs was examined with one-way analysis of variance (ANOVA) for all exposure conditions. All failed to reach significance for both measurements, indicating that two groups, with and without SOAEs did not differ in pre-exposure TEOAEs.

Table 1. Descriptive statistics for pre-exposure TEOAE responses of the groups with and

without SOAEs in dB SPL

		Exposure conditions					
		1	2	3	4	5	6
TEOAE responses Group with SOAEs	Mean	13.4	13.9	14.2	14.3	14.0	13.6
	SD	2.9	2.9	3.3	2.9	3.3	3.3
	Range	7.4	7.5	8.3	7.6	10.0	9.1
TEOAE responses Group without SOAEs	Mean	13.9	14.3	14.1	14.2	14.1	14.3
	SD	3.9	3.5	3.4	3.7	3.6	3.9
	Range	9.7	9.3	8.6	9.3	9.6	9.5

2. General Effects of Sound Exposure

The increase and decrease of responses were observed following tonal stimulation over time (Figure 1). The amplitude response of TEOAE was analyzed for the statistical analysis. A MANOVA was also conducted to determine whether measured post-exposure TEOAEs differed systematically in levels of exposure intensities, frequencies, and post-exposure time. All three main effects reached significance, indicating that the post-exposure TEOAEs were different between two exposure intensities, among three exposure frequencies, and among twenty-six points of the post-exposure time. All the interaction effects including intensity by frequency, frequency by post-exposure time, intensity by post-exposure time, intensity by frequency by post-exposure time reached significance in MANOVA, indicating all combinations of interaction made a difference in post-exposure TEOAEs. The effects of the group with SOAEs and without SOAEs failed to reach significance in one-way ANOVA, indicating that those groups were not different in TEOAE responses.

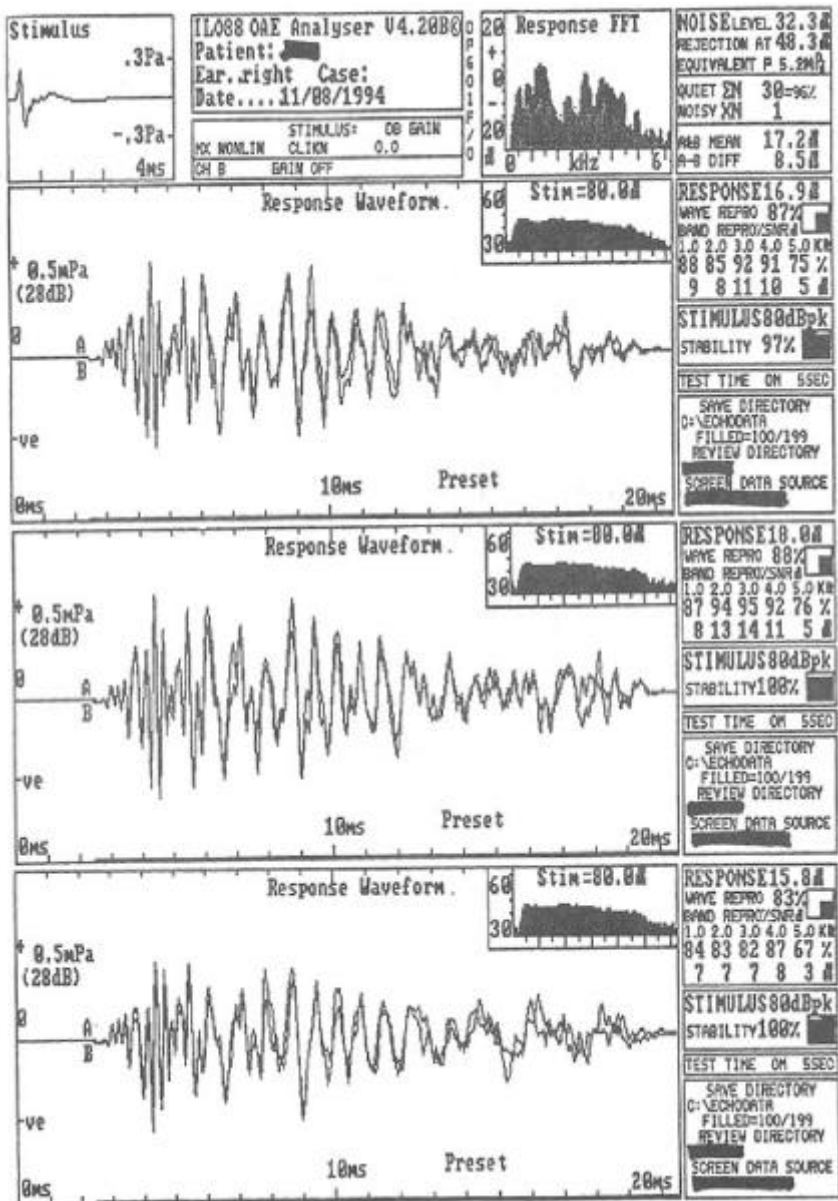


Figure 1. The effects of sound exposure. The first panel is before the exposure, the second panel is 1 minute post-exposure representing sensitization, and the third panel is 2 minute post-exposure representing desensitization. Note the slight changes in reproducibility and 1 to 2 dB SPL changes in response level.

3. Post-Exposure Behavior

A. Recovery Patterns and Time Course

In Figure 2, the mean amplitude shift is plotted as a function of post-exposure time. Between 20 seconds and 1 minute 40 seconds post-exposure, OAE amplitude increased showing sensitization. Then between 2 and 4 minutes post-exposure, OAE amplitude decreased showing desensitization. Subsequent recovery to 10 minute post-exposure was gradual and essentially monotonic.

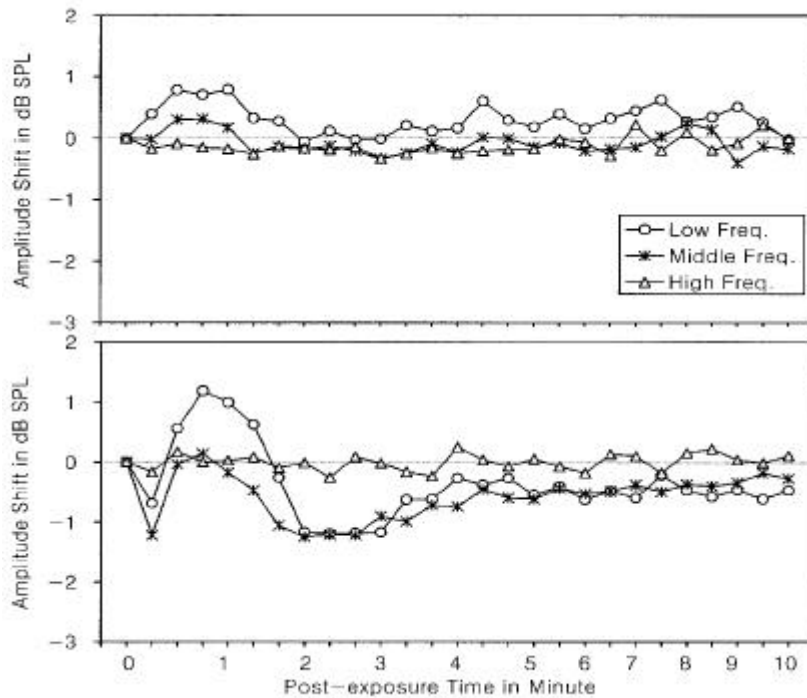


Figure 2. Mean post-exposure TEOAE amplitude shifts as a function of time following 90 second pure tone exposures at 70 dB SPL (upper panel) and 100 dB SPL (lower panel). Each line represents different exposure frequency. Note the larger increase and decrease at 100 dB SPL exposure (lower panel) and the largest effect with the low frequency exposure.

In general, there appear to be three different post-exposure recovery patterns manifested. As the result of the low frequency and intense sound exposure (500 Hz - 100 dB SPL), the first type is characterized by a combination of sensitization and desensitization followed by nearly full recovery by at least 10 minutes post-exposure. The second type of recovery followed the middle frequency and intense sound (- ½ octave or 1500 Hz - 100 dB SPL) exposure, and could be characterized by only a desensitization effect. These curves initially drop very sharply, exhibiting significantly decreased amplitude accompanied by the first rapid recovery, then decreasing again followed by the second slow recovery toward baseline. The 70 dB SPL exposures for both low and middle frequency resulted in the third type of recovery pattern. These contours can be characterized by an initial sensitization followed by mild to no desensitization. Full recovery to the baseline responses was achieved at least 4 minutes post-exposure.

B. Frequency and Intensity Effects

It appears that both frequency and intensity of the exposure signal influence the magnitude and general shape of the recovery pattern as a function of post-exposure time. The effects were more pronounced with the exposure intensity of 100 dB SPL. When the exposure intensity was 100 dB SPL, the amplitude of OAEs were significantly reduced immediately following exposure, showing a rapid recovery, then decreasing again. Following the exposure condition of 70 dB SPL, all effects were reduced compared to 100 dB SPL exposure except the sensitization effect of the middle frequency at the first one minute post-exposure period. The amplitude shift increased following the 70 dB SPL exposure, showing sensitization effect, then decreased showing desensitization. Subsequent recovery for both intensities was gradual and monotonic. The low frequency exposure showed the largest effect of all, the high frequency exposure the least effects, and the middle frequency the middle effect. The greatest amplitude of increase and decrease occurred with 500 Hz - 100 dB SPL exposure condition (Figure 2).

C. Sensitization and Desensitization Effects

The magnitude of sensitization was approximately 1-1.5 dB SPL. The post-exposure time course for sensitization was most prominent between 20 and 40 seconds with residual effects out to 1 minute 40 seconds. Sensitization effect was greatest for the low frequency intense sound exposure condition. For both low and middle frequency exposure signals, the moderate intensity sound exposure produced sensitization approximately at the same time course. For the high frequency exposure conditions, nearly no sensitization effect was observed.

In contrast to sensitization effect, desensitization effect was strongest for middle frequency intense sound exposure condition. For this condition, in fact, only desensitization effect were observed. The mean magnitude of desensitization was approximately 1-3 dB SPL. The post-exposure time course was between 2 and 4 minutes, reaching the peak at 2 to 3 minutes. Slightly less but still good desensitization was noticed following low frequency intense sound exposure. The high frequency moderate intensity sound exposure produced little or no desensitization.

D. Repeatability

The nearly duplicated TEOAEs were observed when the post-exposure TEOAEs were measured repeatedly at the same exposure condition. Statistically, the dependent *t*-test failed to reach significance, evidencing that the resulted TEOAEs were not different under the corresponding experimental conditions.

IV. Discussion

The systematic and physiological analysis of sensitization and desensitization effects following sound exposure in human TEOAE is of considerable practical and theoretical interest. Being a sensitive, rapid, place specific and non-invasive measurement of the cochlear effects of injurious acoustic agents, OAEs are able to observe very rapidly changing and subtle effects without sophisticated experimental apparatus. Therefore, OAEs offer valuable data concerning the characteristics of sensitization and

desensitization phenomena.

Importantly, the twenty-six post exposure TEOAEs obtained in the present study permitted the generation of recovery contours. The recovery pattern of TEOAE is remarkably similar not only to those of previous SOAE and TEOAE noise studies (Kemp, 1986; Norton et al., 1989.) but also to those of behavioral and acoustic reflex investigations (Hirsh and Ward, 1952; Hughes, 1954; Hirsh and Bilger, 1955; Ruth, 1977). Recall Hirsh and Ward (1952) reported initial rapid recovery over 1 minute post-exposure followed by a decrease in hearing sensitivity which was referred to as the "bounce phenomenon", reaching a maximum by about 2 minutes, then accompanied by a slow recovery. These two distinctive phases of threshold recovery were labeled later by Hirsh and Bilger (1955) as R-1 and R-2 and subsequently verified by several investigators (Lightfoot, 1955; Jerger, 1955, 1956). Indeed, this time course is analogous to the post exposure time course in the present study. When the exposure frequency was low and middle, on cessation of the acoustic stimulation a rapid recovery was observed out to 1 minute 40 seconds, reaching a peak at between 20 and 40 seconds, but then decreasing (bounce) between 2 and 4 minutes, reaching maximum between 2 and 3 minutes. Subsequent recovery was essentially monotonic and gradual.

With the intense sound exposure, the significantly reduced amplitude for TEOAEs was observed immediately following the exposure. This initial decrease following a moderately intense sound exposure was observed by several previous investigators (i.e., Ruth, 1977; Norton et al., 1989). In the study of acoustic reflex threshold, Ruth reported an initial large amount of the acoustic reflex threshold shift accompanied by a rapid recovery. Although the means of measurement were different, this finding is remarkably similar to the present study. Norton et al., in their SOAE study, also reported this amplitude decrease as ". . .the SOAE was absent or obscured by the noise floor." Since this initial decrease was followed by a second decrease (bounce), the authors described the recovery pattern as a typical biphasic post-exposure recovery contour which is observed in the present study. Behavioral studies also defined the same time courses.

The similarity of the present study and the effects of comparable exposures on behavioral thresholds imply common underlying mechanisms that are responsible for both. This notion is in accordance with the literature (Norton et al., 1989; Rossi et al.,

1991). Although their exact origin is unknown, OAEs appear to be closely linked to active and biomechanical elements in the cochlear partition, involving the outer hair cell (OHC) subsystem (Davis, 1983; Neely, 1983; Kim, 1986; Neely and Kim, 1983, 1986). Also, the behavioral threshold changes after noise exposure is well known to be due to damage to the cochlear partition. Therefore, the common fundamental mechanisms involved in post-exposure effects of behavioral threshold as well as TEOAEs seem to be alterations to OHCs. This is not an unproven premise but the results in the present study support the hypothesis.

In their systematic study of the effects of exposure frequency on behavioral thresholds, Noffsinger and Olsen (1970) noted that the exposure frequency lower than the observed frequency resulted in better sensitization. Although the means of the measurement were different, comparable data were elicited in the present study. For example, the low exposure frequency of 500 Hz evoked the strongest sensitization effect, the middle exposure frequency showed the mild effect, and the high exposure frequency produced little effect. Given the fact that most of TEOAEs response observed were above 500 Hz, the sensitization effect was elicited when the observed frequencies were above the exposure frequency. The 500 Hz exposure frequency revealed not only the great sensitization but also the great desensitization regardless of the observed frequency. Therefore, it can be stated that the nonspecific exposure frequency effect of a pure tone on post-exposure effect was observed in the present study. This is consistent with the findings of Hughes in 1954. He demonstrated that an exposure tone of 500 Hz sensitized the auditory system over a wide range of frequencies.

Moreover, in the same study, Noffsinger and Olsen found that the desensitization appeared more clearly and frequently in higher frequency measurement for instance 4 kHz versus 0.25 and 1 kHz. The greater desensitization effect at higher frequency was also supported by the noise exposure study in TEOAEs by Hotz et al. (1993). They observed 3 mean changes between pre- and post-exposure including overall TEOAE response, lower frequency region TEOAEs (0.5 to 2 kHz), and higher frequency region TEOAEs (2 to 4 kHz), and found significance in overall and higher frequency region TEOAEs for the desensitization effect. Contrastingly, the 4 kHz exposure did not produce notable effects on either sensitization or desensitization in

the present study. The desensitization effect was more pronounced in the middle frequency and sensitization in the lower frequency. However, Noffsinger and Olsen (1970) also noted that the exposure frequency nearest or equal to the test frequency showed the most dramatic post-exposure effects. This is in partial agreement with the present study because desensitization was greatest with the - ½ octave exposure which was the nearest frequency to the observed frequency out of three frequency measurements examined in the present study .

The strongest response levels occurred most frequently in 1000 Hz region and in 2000 Hz region the second most frequently. Eighty-three percent of the strongest response levels of TEOAEs were placed in 1000 and 2000 Hz frequency regions. The location of these frequency regions confirms agrees with previous research (Zurek, 1981; Wier et al., 1984; Martin et al., 1990; Kulawiec and Orlando, 1994). However, it is possible that the distribution of TEOAEs across frequency is influenced by the transfer function of the middle ear, which optimizes transmission of sounds in this frequency region. That is, the transfer function of the middle ear will impede the transmission of sounds below 1000 Hz and above 2000 Hz from the inner ear to the ear canal to the same extent that their transmission is impeded from the ear canal to the inner ear. For this reason, it is important to check the middle ear status before TEOAE measurements. The paucity of TEOAEs below 1000 Hz seems to be attributed to biologic noise.

Good reliability was demonstrated in the present study by revealing highly replicated measurements (1 dB or less variability). It was possible to compare the results of the present study to the general repeated measurements TEOAEs in the absence of sound exposure, while no comparison could be made to the repeated measurement following the sound exposure. TEOAE findings demonstrated excellent agreement with the results of Harris et al. (1991). The authors found that the variability of TEOAE measurements in individual spectral bands was within 1 dB range with the repeated measurements which were observed in three sessions separated by 3-day intervals.

Our findings document the presence of sensitization and desensitization effects in TEOAEs. Although the present study deals with limited subject population and apparatus, the results provided additional information toward our understanding of

OAE phenomena and physiology of the auditory system in general. Furthermore, the sensitivity of TEOAEs to moderate to intense sound exposure revealed in the present study indicates that TEOAEs can be used for monitoring hearing changes following exposure. For instance, clinical monitoring would be possible with patients having noise exposure. The measurements of TEOAEs would offer an effective means of monitoring subtle changes in cochlear status.

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(Transient Evoked Otoacoustic Emissions, TEOAEs) (sensitization) (desensitization)

16 (21-29, 24.6), 16 8

(Spontaneous Otoacoustic Emission, SOAE)가

8 SOAE가 70 100 dB

SPL, 500, 1500 (SOAE가 가 OAE가

1/2), 4000 Hz,

90 3 TEOAE

10 TEOAE 5 20, 5

30 26 TEOAE TEOAE 5 dB

50% 가

26

Repeated Multivariate Analysis of Variance (MANOVA)

가 TEOAE

가 가

가 , SOAE가

TEOAE, 20

1 40 TEOAE 가 가 , 2 4 TEOAE

가 10

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