

INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps. Each original is also photographed in one exposure and is included in reduced form at the back of the book.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

U·M·I

University Microfilms International
A Bell & Howell Information Company
300 North Zeeb Road, Ann Arbor, MI 48106-1346 USA
313/761-4700 800/521-0600



Order Number 1353121

**Contralateral stimulation does not influence the suppression
tuning curves of spontaneous otoacoustic emissions**

Tooley, Carolyn Jean, M.S.

The University of Arizona, 1993

U·M·I
300 N. Zeeb Rd.
Ann Arbor, MI 48106



**CONTRALATERAL STIMULATION DOES NOT INFLUENCE THE SUPPRESSION
TUNING CURVES OF SPONTANEOUS OTOACOUSTIC EMISSIONS**

by

Carolyn Jean Tooley

**A Thesis Submitted to the Faculty of the
DEPARTMENT OF SPEECH AND HEARING SCIENCE
In Partial Fulfillment of the Requirements
For the Degree of
MASTER OF SCIENCE
In the Graduate College
THE UNIVERSITY OF ARIZONA**

1 9 9 3

STATEMENT BY AUTHOR

This thesis has been submitted in partial fulfillment of requirements for an advanced degree at The University of Arizona and is deposited in the University Library to be made available to borrowers under rules of the Library.

Brief quotations from this thesis are allowable without special permission, provided that accurate acknowledgment of source is made. Requests for permission for extended quotation from or reproduction of this manuscript in whole or in part may be granted by the head of the major department or the Dean of the Graduate College when in his or her judgment the proposed use of the material is in the interests of scholarship. In all other instances, however, permission must be obtained from the author.

SIGNED: Carolyn Jean Tooley

APPROVAL BY THESIS DIRECTOR

This thesis has been approved on the date shown below:

Theodore J. Glattke
Theodore J. Glattke

29 April 93
Date

ACKNOWLEDGMENTS

I wish to thank the four subjects who participated in this study, as well as those who helped in preliminary data-collecting and took part in discussions concerning this area of research. I would also like to thank my friends and family who have encouraged me throughout the length of this project, especially David Young. A special thank you is extended to Dr. Ted Glattke for his helpful discussions, suggestions, and extensive hours spent editing this final edition.

TABLE OF CONTENTS

LIST OF FIGURES.....	5
LIST OF TABLES.....	6
ABSTRACT.....	7
INTRODUCTION.....	8
Suppression Tuning Curve.....	8
Cochlear Mechanics.....	10
Contralateral Acoustic Reflex Threshold.....	11
Contralateral Stimulation.....	12
METHODS.....	15
RESULTS.....	19
DISCUSSION AND CONCLUSION.....	21
APPENDIX A.....	33
REFERENCES.....	34

LIST OF FIGURES

FIGURE 1, Idealized illustration of a STC.....	9
FIGURE 2, Averaged STCs for EA with and without contralateral noise present.....	29
FIGURE 3, Averaged STCs for JH with and without contralateral noise present.....	30
FIGURE 4, Averaged STCs for DS with and without contralateral noise present.....	31
FIGURE 5, Averaged STCs for MW with and without contralateral noise present....	32

LIST OF TABLES

TABLE 1, SOAE frequencies (Hz) and standard deviations with and without contralateral noise present.....	24
TABLE 2, SOAE amplitudes (dB SPL) and standard deviations with and without contralateral noise.....	25
TABLE 3, Two-tailed paired T values (df=8) between intensity levels of suppression tones for trials 1 and 2.....	26
TABLE 4, Q ₁₀ values and slopes (dB/octave) of STCs for trials 1 and 2.....	27
TABLE 5, Q ₁₀ values and slopes (dB/octave) of averaged STCs.....	28

ABSTRACT

It is well established that the suppression of otoacoustic emissions can be characterized by a tuning curve. The tip of a suppression tuning curve (STC) is normally found to be slightly above the emission frequency, and STCs are similar in shape to tuning curves for mechanical activity of the basilar membrane.

Little is known about the effects of contralateral acoustic stimulation on the STC. Studies have examined the effect, but may have been confounded by activation of the acoustic reflex and the possibility of transcranial crossover of the contralateral signal. This study compared the sharpness (Q_{10}) of STCs of SOAEs with and without contralateral stimulation that employed wideband noise. The intensity of the contralateral noise was well below the acoustic reflex threshold.

The STCs of four female subjects were examined. The results revealed no change in the sharpness (Q_{10}) of the tips of the STCs with and without contralateral stimulation. The lack of change in the STCs is inconsistent with prior findings based on recording obtained from single neurons in the auditory nerve in anesthetized animals and may reflect an insensitivity of the STC measures to dynamics of the efferent auditory system.

INTRODUCTION

Spontaneous otoacoustic emissions (SOAEs) are narrowband signals produced by the cochlea in the absence of any deliberate acoustic stimulation. These signals can be detected with a sensitive microphone placed in the ear canal (Kemp, 1979). The site of origin of SOAEs is unknown; however, it is speculated that SOAEs are associated with action of the "cochlear amplifier" (Davis, 1983), especially the outer hair cells (OHCs) (Glatcke & Kujawa, 1991). Measures of the suppression of SOAEs have been used to confirm the cochlear origin(s) of SOAEs in humans and experimental animals and to demonstrate frequency specificity of SOAEs. Studying the influence of contralateral stimulation on the suppression characteristics of SOAEs provides a noninvasive method to gain information about the possible influence of the efferent neural supply to the OHCs.

Every SOAE can be altered by the ipsilateral presentation of a tone that is close to the SOAE frequency. Introduction of the tone causes the SOAE to decrease in amplitude. A graph that illustrates the frequency and minimum SPL of signals that are effective in suppressing an emission is called the suppression tuning curve (STC) of that emission. STCs for SOAEs reflect the frequency specificity of the cochlea for the SOAE frequency (Kemp, 1979). An STC is illustrated in Figure 1.

Suppression Tuning Curve

The STC represents the various intensities and frequencies of the suppressors that attenuate the SOAE by a pre-selected amount (Harris & Glatcke, 1992). The amount of SOAE attenuation selected as the criterion for SOAE suppression has been inconsistent among studies. Rabinowitz and Widin (1984) used 6 and 10 dB, and Schloth and

Zwicker (1983) used 3, 6, and 10 dB of SOAE intensity reduction. For a fixed suppression criterion, such as -3 dB, SOAE STCs have been demonstrated to be highly replicable (Zizz & Glattke, 1988).

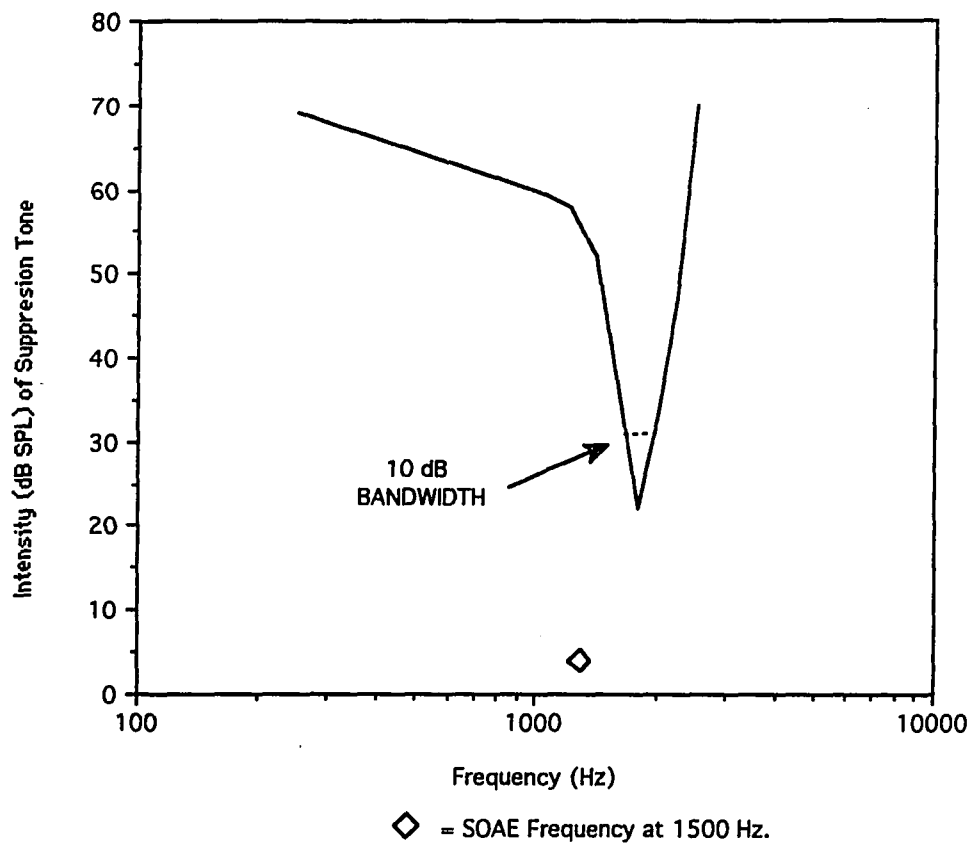


FIGURE 1. IDEALIZED ILLUSTRATION OF A STC.
(RABINOWITZ AND WIDIN, 1984; SCHLOTH AND
ZWICKER, 1983)

STCs are characterized by a "V" shape (Rabinowitz & Widin, 1984; Schloth & Zwicker, 1983). The general shape of STCs is independent of SOAE frequency (Harris & Glattke, 1992). The STC tip, which corresponds to the bottom of the "V", is located at a frequency that is slightly higher than the SOAE frequency and represents an area close to the region associated with the characteristic frequency of the SOAE on the basilar membrane (Rabinowitz & Widin, 1984). Rabinowitz and Widin (1984) demonstrated that the portion of the STC below the emission frequency increases at a rate of 66 to 111 dB/octave, and the portion above the emission is characterized by a steeper slope of 166 to 333 dB/octave. As is the case with other tuning curves obtained in the auditory periphery, the "tail" of the STC extends to lower frequencies, and the suppression of the SOAE gradually becomes less effective (Schloth & Zwicker, 1983).

Cochlear Mechanics

Mountain (1980) found that direct electrical stimulation of the crossed olivocochlear bundle in guinea pigs was associated with a change in the electrical potential of the endolymph. This, in turn, was linked to a decrease in the amplitude of distortion product otoacoustic emissions. Distortion product otoacoustic emissions, unlike spontaneously occurring otoacoustic emissions, are elicited with tonal stimulation of the ear (Kemp, 1979). A reduction of the endolymphatic potential related to stimulation of the efferent fibers innervating the organ of Corti may be a manifestation of hyperpolarization of hair cells which then influences cochlear mechanics (Mountain, 1980). The effects of electric stimulation of the crossed olivocochlear bundle were eliminated when the scala tympani was infiltrated with d-tubocurarine, which blocks the acetylcholine receptors in the post-synaptic membrane

of the OHCs (Siegel & Kim, 1982). Consistent with Mountain, Siegel and Kim attributed changes in hair cell polarization, which influenced cochlear mechanics, to mediation by efferent fibers that synapse with OHCs.

An indirect way to observe changes in cochlear mechanics that may be due to the influence of efferent neurons that synapse with OHCs is to observe SOAEs during simultaneous contralateral acoustic stimulation. SOAE amplitude and frequency changes have been observed with contralateral stimulation employing tonal and wideband noise stimuli, but the changes may have been due to acoustic reflex stimulation and transcranial crossover of the contralateral signal, causing ipsilateral suppression (Grose, 1983; Rabinowitz & Widin, 1984; Schloth & Zwicker, 1983). Changes in SOAEs that are not associated with acoustic reflex stimulation and transcranial crossover of the contralateral signal also have been observed with the introduction of contralateral stimulation (Kujawa & Glattke, 1989; Mott, Norton, Neely & Warr, 1989). These changes in SOAEs support the conclusion that SOAEs are associated with OHCs and that changes in cochlear mechanics result from an efferent effect (Mott, *et al.*, 1989). The use of contralateral stimulation to examine changes of the STC of SOAEs may be a useful measure to aid in the development of models for cochlear function. Changes in the characteristics of STCs of SOAEs during contralateral stimulation may provide information about contribution of efferent fibers to the cochlea during the reverse transmission of SOAEs.

Contralateral Acoustic Reflex Thresholds

Contralateral stimulation using tonal stimuli has been found to cause changes in SOAEs independent of acoustic reflex activity or crossover of the contralateral signal

causing ipsilateral suppression (Mott, et al., 1989). Mott, et al. observed that latencies for SOAE changes (to move from baseline values to mean shifted values) were longer with supra-CART (contralateral acoustic reflex threshold) stimulus levels than for sub-CART stimulus levels. Also, SOAE frequency and amplitude shifts were greater during acoustic reflex stimulation periods than when the reflex was not stimulated. In this study, one subject's reflex decay was 100% at 20 seconds, but the SOAE remained altered during 4 minutes of contralateral stimulus presentation. Another subject who had SOAEs at multiple frequencies in the same ear demonstrated that the various SOAEs reacted differently to contralateral stimulation (Mott, et al., 1989).

Contralateral Stimulation

As a result of contralateral stimulation, the SOAE frequency shifts and the amplitude of the SOAE is decreased (Kujawa & Glatcke, 1989; Mott, et al., 1989). Mott, et al. (1989) found that the greatest shift in SOAE frequency occurs with contralateral tonal stimuli that are 3/8 to 1/2 octave below the SOAE, and amplitude of the SOAE decreased the most with a stimulus frequency similar to the SOAE frequency. It was concluded that frequency and amplitude shifts are "tuned differently" (Mott, et al., 1989). An inverse relationship exists between SOAE amplitude and frequency shifts as a result of an increase in stimulus intensity: amplitude decreases as the stimulus intensity increases, but the frequency shift increases as the stimulus intensity increases. Kujawa and Glatcke (1989) found that the frequency shift due to contralateral wideband noise limited to 50 dB SPL ranged from approximately +2.5 to +8 Hz, and that changes in intensity ranged from approximately -0.6 to -5.5 dB SPL.

Alteration of SOAE amplitude and frequency has been determined to be especially

robust and consistent with wideband noise. Differences in magnitude and direction of SOAE amplitude and frequency shifts vary with different stimuli. Contralateral presentation of wideband noise is consistently associated with amplitude reduction and an upward shift in frequency of the SOAE. The effects during contralateral pure tone stimulation are not as consistent (Kujawa & Glatke, 1990).

It has also been observed that the amplitude of transient evoked otoacoustic emissions decreases during contralateral stimulation using wideband noise (Collet, *et al.*, 1990). Transiently evoked otoacoustic emissions appear after the introduction of brief stimuli to the ear (Kemp, 1978). Comparison of recordings of transient evoked otoacoustic emissions with and without simultaneous contralateral stimulation has been suggested as a means to detect retrocochlear lesions (Ryan, Kemp, and Brass, 1991).

As Kiang, Liberman, Sewell, and Guinan (1986) have reviewed, direct electrical stimulation of the efferent system in cat is associated with an alteration in the frequency threshold curves of single neurons of the auditory nerve. These changes are a decrease in tuning curve tip sensitivity with little or no change in tail sensitivity. Contralateral stimulation using wideband noise is an indirect method of stimulation which should produce similar results.

The purpose of this study was to determine if contralateral stimulation will influence the frequency selectivity of the cochlea as reflected in SOAE STCs. This study examined the characteristics of the STCs of SOAEs during simultaneous contralateral stimulation using wideband noise. More specifically, STCs of SOAEs were compared with and without simultaneous contralateral stimulation to determine the difference(s), if any, of the tuning of the OHCs. Precautions were taken to maintain the contralateral

wideband signal below acoustic reflex threshold and to avoid transcranial crossover of the contralateral stimulus.

METHODS

Approximately 30 women were screened for SOAEs. Women were employed as subjects because they constitute more than 90% of the student population in the University of Arizona Speech and Hearing Department. Also, the prevalence of SOAEs is greater in women than in men (Bilger, Matthies, Hammel & Demorest, 1990). Subjects screened for SOAEs were 18 years or older and, upon otoscopic observation, their tympanic membranes appeared normal. The subjects completed a questionnaire on which they indicated whether they were taking any ototoxic medications. Individuals taking ototoxic medications were excluded from the study.

Sampling of the sounds from the subject's ear was completed using a spectral averaging technique to attenuate unwanted noise. The subject was seated in a comfortable reclining chair in a sound-treated booth. An Etymotic Research ER-10/ER-2 probe system was inserted into the subject's ear using a disposable formfitting foam earplug. The probe microphone output was led to an Etymotic Research ER-10-72 preamplifier and custom amplifier with a high pass filter system that attenuated noise levels below 400 Hz by 30 dB per octave. The output was coupled to a Bruel and Kjaer 2033 real time spectral analyzer. The recorder was monitored through the use of an oscilloscope and loudspeaker. The screening procedure for SOAEs involved examining average ($n=8$) spectral displays that were 500 Hz wide and centered at 750 Hz, 1250 Hz, 1750 Hz, 2500 Hz, 3500 Hz, 4500 Hz, 5500 Hz, 6500 Hz, and 7500 Hz. SOAEs used for this study were 10 dB SPL above the noise floor of the spectrum analyzer. The noise floor generally ranged between -12.0 and -20.0 dB SPL.

When an SOAE was detected, a tone was generated by a computer-controlled

function generator (Quatech) within +10% of the SOAE frequency to suppress the SOAE. This was done to insure that the recorded signal was truly a SOAE and not an artifact. No individual selected for this study had multiple SOAEs in the test ear.

After a SOAE that was 10 dB SPL above the noise floor of the spectrum analyzer was confirmed to exist in one of the subjects' ears, the subjects' hearing was screened. Hearing thresholds of 10 dB HL or less at 250 Hz through 8000 Hz were necessary for participation in this study. An immittance study was performed. All subjects were required to have normal middle ear function with middle ear pressure not less than -50 mm H₂O or greater than 50 mm H₂O. Acoustic reflexes were tested using wideband noise to insure that the intensity of the contralateral signal fell below activation of the subject's reflex. The four individuals who passed all selection criteria returned for the first session of data collection and began being compensated at the rate of \$4.00 per hour.

During the first session, the amplitude (dB SPL) and frequency of the SOAE were recorded from 10 averages of 32 spectral samples, and each session lasted approximately 1/2 hour. To be included in the next phase of the study, the subjects' emissions had to meet amplitude and frequency stability requirements: specifically, an amplitude change of less than 2 dB SPL and frequency shift of less than 5 Hz during the 10 samples. If these criteria were met, the subject returned at another time for further data collection.

During the subject's second visit, a STC was generated without any contralateral stimulation. The intensities and frequencies of 10 averaged spectra based on 8 samples were recorded with no ipsilateral suppression tone alternating with 10 averaged spectra

based on 8 samples with an ipsilateral suppressor that was 5%, 10%, 15%, or 20% above or below the frequency of the SOAE. An ipsilateral suppression tone also was generated at 30% below the frequency of the SOAE. Attenuation of the SOAE by a criterion amount of 3-5 dB SPL was used for designating the "threshold" value of the suppressor tone, that is to say, the value that was represented as a point on the STC. The mean frequency and intensity of the SOAE were computed from the 10 averaged spectra to insure that the suppression was 3 to 5 dB SPL. The SPL of the ipsilateral suppression tone was limited to 70 dB SPL by the equipment to insure that excessive sound intensities were not introduced to the subject's ear.

The recording session lasted approximately 2 1/2 hours. The subject was allowed a short break halfway through the session if it was desired. This was done to avoid fatigue which is associated with low frequency noise that interferes with measurements. A STC similar to the one represented in Figure 1 was the result of the first recording session.

The third session involved the same procedure, but with the introduction of a simultaneous contralateral stimulus. The contralateral stimulus consisted of 800 msec bursts of wideband noise generated by a Grason-Stadler 901B Noise Generator that were routed through a Grason Stadler 829D Electronic Switch and amplified by a Crown amplifier. The electronic switch and sampling of the spectral analyzer were synchronized using a Grason-Stadler 471-1 Interval Timer. After amplification, the noise was attenuated by a Hewlett Packard 350D attenuator and routed to the earphone. The contralateral noise was coupled to the subject's ear using a Grason-Stadler insert earphone to reduce the effects of transcranial crossover.

Calibration of the contralateral wideband noise was performed by attaching the insert earphone to a Bruel and Kjaer 2204 sound level meter using an HA-2A coupler. The wideband noise was approximately three octaves wide (500 to 3500 Hz). The spectrum of the wideband noise after being coupled to the insert earphone and sound level meter coupler indicated a reduction in high frequency energy indicating a lower overall intensity level than anticipated. Using the C weighting scale, the wideband noise was measured at 62 dB SPL. The contralateral stimulus was used to elicit a CART as measured on a Teledyne TA-4D acoustic impedance meter in all subjects. Subject EA's CART was at 97 dB SPL, with JH's being 92 dB SPL, DS's was 97 dB SPL, and MW's was present at 82 dB SPL. The contralateral stimulus level employed in this study was found to be between 20 to 35 dB below CART.

STCs were derived from each session to illustrate the frequency and intensity combinations necessary to attenuate the SOAE 3 to 5 dB SPL with and without contralateral stimulation. High and low frequency slopes and Q_{10} values were calculated for each STC. Q_{10} was calculated by dividing the SOAE frequency by the bandwidth (Hz) 10 dB above the tip of the tuning curve. The Q_{10} values were used to compare the STCs with and without contralateral stimulation.

RESULTS

Four subjects who had SOAEs that met the previously stated selection criteria (amplitude change of less than 2 dB SPL and frequency shift of less than 5 Hz) were employed in this study. Mean frequencies and standard deviations of SOAEs for each subject were calculated for both trials with and without contralateral noise present. See Table 1. Target frequencies noted in Table 1 were computed as mean frequencies from 10 averages of 32 spectral samples with no contralateral noise present. Subject MW had a target frequency of 4555 Hz, with EA's being 2740 Hz, DS's 1751 Hz, and JH's 1662 Hz. The high frequency of MW's SOAE is important to note as it may be related to results that differ from those of the other three subjects.

Mean amplitudes and standard deviations of SOAEs for each subject were also calculated for both sessions with and without contralateral noise present. See Table 2. SOAE amplitude ranged from -6.9 to -1.2 dB SPL without contralateral noise present. With contralateral noise present, SOAE amplitude ranged from -6.3 to 1.0 dB SPL.

A two-tailed paired t-test was performed between suppression tone intensity levels for trials one and two. See Table 3. No significant difference exists between suppression tone intensity levels ($p < .01$) of trials one and two for all subjects except MW.

STCs with and without contralateral noise present were generated for all four subjects for each trial. Q_{10} values were then calculated for each STC with the tip of the tuning curve representing the characteristic frequency, and a two-tailed paired t-test was performed between all trial one Q_{10} scores and all trial two Q_{10} scores. No

significant difference ($p < .01$) exists between Q_{10} scores from trials one and two for all subjects. High and low frequency slopes were also calculated for STCs generated for trials one and two. The mean low frequency slopes for Trials 1 and 2 were -80.75 dB/octave and -88.4 dB/octave respectively. The mean high frequency slopes for Trials 1 and 2 were 107.0 dB/octave and 148.25 dB/octave respectively. See Table 4.

STCs with and without contralateral noise present were generated for all subjects using averaged levels of suppression tone intensities for trials one and two. Figures 2, 3, 4, and 5 illustrate overlaid "averaged" STCs with and without contralateral noise present.

High and low frequency slopes and Q_{10} values were computed for the averaged STCs of all subjects. See Table 5. A two-tailed paired t-test indicated that no significant ($p < .01$) difference exists between the Q_{10} values of the averaged STCs with and without contralateral noise present. MW's Q_{10} value without contralateral noise present is 5.6 and with contralateral noise present is 8.4 indicating a difference in tuning sharpness. The other three subjects' Q_{10} scores did not reflect similar changes.

DISCUSSION AND CONCLUSION

The data revealed STCs similar to those obtained by Harris and Glatcke (1992). The STC tip, which corresponds to the minimum of the STC function was located at a frequency slightly higher than the SOAE frequency, a finding that is similar to Rabinowitz & Widin's observations (1984). As the tail of the STC generated extended to the lower frequencies, suppression of the SOAE gradually becomes less effective as Schloth and Zwicker also observed in their study (1983).

Low frequency slopes ranged from -52.5 to -114 dB/octave for STC without contralateral noise present. The high frequency slopes without contralateral noise present ranged from 43.0 to 206 dB/octave. Both high and low frequency slopes included values outside the 66 to 111 dB/octave range for low frequency slopes and 166 to 333 dB/octave range for high frequency slopes that Rabinowitz and Widin (1984) described.

With contralateral noise present, low frequency slopes ranged from -78.0 dB/octave to 98.5 dB/octave and high frequency slopes ranged from 67.0 dB/octave to 316 dB/octave.

STCs were generated twice with and without contralateral noise present. Suppression using a fixed suppression criterion has been demonstrated to be highly replicable (Zizz & Glatcke, 1988). The criterion for suppression of the emission in this study was a reduction of 3 to 5 dB SPL. Similar to Zizz and Glatcke's findings, no statistically significant difference between the STCs for Trial 1 and Trial 2 was found to exist.

Because no significant difference existed between Trials 1 and 2, the suppression

tone intensity levels were averaged to produce a single STC with no contralateral noise present and a single STC with contralateral noise present for each subject.

Changes in the STCs as reflected in the Q_{10} scores with and without contralateral noise present did not exist at a statistically significant level. The difference between subject MW's Q_{10} scores (5.6 without contralateral noise present and 8.4 with contralateral noise present) was greater than the difference for the other three subjects. MW's SOAE frequency differs greatly from the other three subjects. Her target SOAE frequency is 4555 Hz, while the other three subjects' SOAE frequencies occur in the mid-frequency range.

High characteristic frequency tuning curves have sharply tuned tips with steep slopes. Progressively lower characteristic frequency tuning curves have more sensitive tails and broader, less finely tuned tips (Kiang, *et al.*, 1986). Perhaps the effects of contralateral stimulation using wideband noise are more easily observed when stimulating the basal end of the basilar membrane where MW's SOAE is presumed to be generated because of the characteristics of the micromechanical responses of the organ of Corti elements.

Although changes in SOAEs have been observed with the introduction of contralateral stimulation (Kujawa & Glatcke, 1989; Mott, *et al.*, 1989), the results from this study do not reveal a difference in the STC Q_{10} values when contralateral stimulation was present. Changes in the Q_{10} values were anticipated on the basis of previous studies that revealed slight alteration in the frequency tuning curve Q_{10} values (Kiang, *et al.*, 1986). Kiang, *et al.* observed the sensitivity of tuning curves to decrease

with little or no change in tail sensitivity during stimulation of the olivocochlear bundle. The entire tip of the tuning curve disappeared when OHCs were missing due to damage from kanamycin. Both missing OHCs and stimulation of the olivocochlear bundle affect tip sensitivity similarly. Some difference exists between changes in the tail of a tuning curve with missing OHCs and one with stimulation of the olivocochlear bundle. These differences are noted to exist as "kanamycin removes OHCs and their stereocilia, but OCB [olivocochlear bundle] stimulation only changes some OHC property" (p. 177).

The absence of statistically significant changes in Q_{10} does not necessarily indicate the absence of influence by the efferent fibers. The changes observed during OCB stimulation are very small. A change in threshold of the tuning curve tip is the most easily observable response to OCB stimulation which was noted in Kiang, *et al.*'s study (1986). Q_{10} values reflect the sharpness of the tuning curve which changed very little, if at all, in Kiang, *et al.*'s study. It is possible that a larger response may be seen using a higher intensity level of contralateral stimulation than employed in this study. Mott, *et al.* (1989) observed that changes in SOAEs became much larger with increasingly higher levels of contralateral stimuli which suggests that a greater change in Q_{10} values may be observed with contralateral stimuli closer to CART.

Contralateral stimuli in this study were between 20 and 35 dB below CART. A higher intensity level of contralateral stimuli could be obtained without reaching CART, and it may elicit a more observable response due to efferent stimulation upon the Q_{10} values.

TABLE 1. SOAE FREQUENCIES (Hz) AND STANDARD DEVIATIONS
WITH AND WITHOUT CONTRALATERAL NOISE PRESENT

<u>SUBJECT</u>	<u>TARGET SOAE FREQUENCY</u>	<u>SOAE FREQUENCY WITHOUT CONTRALATERAL NOISE</u>		<u>SOAE FREQUENCY WITH CONTRALATERAL NOISE</u>	
		<u>TRIAL #1</u>	<u>TRIAL #2</u>	<u>TRIAL #1</u>	<u>TRIAL #2</u>
EA	2740 Hz	M=2739.1 SD=-1.5	M=2743.9 SD=-1.3	M=2742.4 SD=1.0	M=2743.3 SD=-2.0
JH	1662 Hz	M=1659.6 SD=-1.8	M=1654.1 SD=2.1	M=1656.3 SD=-2.2	M=1658.0 SD=-1.1
DS	1750 Hz	M=1756.0 SD=-3.7	M=1758.2 SD=-2.7	M=1757.3 SD=-3.9	M=1760.1 SD=-4.1
MW	4555 Hz	M=4557.5 SD=-2.8	M=4564.0 SD=-2.6	M=4552.8 SD=-1.9	M=4571.3 SD=4.4

TABLE 2. SOAE AMPLITUDES (dB SPL) AND STANDARD DEVIATIONS WITH AND WITHOUT CONTRALATERAL NOISE

<u>SUBJECT</u>	<u>TARGET SOAE FREQUENCY</u>	<u>SOAE AMPLITUDE WITHOUT CONTRALATERAL NOISE</u>		<u>SOAE AMPLITUDE WITH CONTRALATERAL NOISE</u>	
		<u>TRIAL #1</u>	<u>TRIAL #2</u>	<u>TRIAL #1</u>	<u>TRIAL #2</u>
EA	2740 Hz	<i>M</i> =-2.5 <i>SD</i> =0.5	<i>M</i> =-2.2 <i>SD</i> =0.6	<i>M</i> =-6.0 <i>SD</i> =0.7	<i>M</i> =-6.2 <i>SD</i> =0.7
JH	1662 Hz	<i>M</i> =4.6 <i>SD</i> =1.7	<i>M</i> =1.2 <i>SD</i> =2.7	<i>M</i> =1.0 <i>SD</i> =1.6	<i>M</i> =-0.2 <i>SD</i> =1.6
DS	1750 Hz	<i>M</i> =-4.8 <i>SD</i> =1.3	<i>M</i> =-6.0 <i>SD</i> =0.7	<i>M</i> =-4.8 <i>SD</i> =1.4	<i>M</i> =-4.2 <i>SD</i> =1.0
MW	4555 Hz	<i>M</i> =-5.3 <i>SD</i> =0.6	<i>M</i> =-7.0 <i>SD</i> =0.2	<i>M</i> =-5.4 <i>SD</i> =0.8	<i>M</i> =-6.3 <i>SD</i> =0.5

TABLE 3. TWO-TAILED PAIRED T VALUES (DF=8)
BETWEEN INTENSITY LEVELS OF SUPPRESSION TONES FOR TRIALS 1 AND 2

<u>SUBJECT</u>	<u>T VALUES BETWEEN TRIALS 1 & 2 WITH CONTRALATERAL NOISE</u>	<u>T VALUES BETWEEN TRIALS 1 & 2 WITHOUT CONTRALATERAL NOISE</u>
EA	1.5X10 ⁻¹⁸	-2.9
JH	-3.1	1.1
DS	-0.5	-0.7
MW	4.3	-3.6

TABLE 4. Q₁₀ VALUES AND SLOPES (dB/OCTAVE) OF STCs FOR TRIALS 1 AND 2

SUBJECT	<u>WITHOUT CONTRALATERAL NOISE</u>			<u>WITH CONTRALATERAL NOISE</u>		
	<u>Q₁₀</u>	<u>LOW FREQ. SLOPE</u>	<u>HIGH FREQ. SLOPE</u>	<u>Q₁₀</u>	<u>LOW FREQ. SLOPE</u>	<u>HIGH FREQ. SLOPE</u>
EA						
TRIAL #1	15.4	-88.0	99.0	15.4	-98.5	89.0
TRIAL #2	20.2	-92.0	51.0	17.1	-88.0	81.0
JH						
TRIAL #1	14.7	-75.0	43.0	12.0	-78.0	67.0
TRIAL #2	13.1	-55.0	78.0	10.6	-79.0	85.0
DS						
TRIAL #1	6.4	-80.0	206	5.6	-87.5	242
TRIAL #2	7.4	-89.5	158	4.6	-88.0	316
MW						
TRIAL #1	30.4	-52.5	105	19.4	-93.5	153
TRIAL #2	20.8	-114	116	10.3	-94.5	153

TABLE 5. Q₁₀ VALUES AND SLOPES (dB/OCTAVE) OF AVERAGED STCs

<u>SUBJECT</u>	<u>WITHOUT CONTRALATERAL NOISE</u>			<u>WITH CONTRALATERAL NOISE</u>		
	<u>Q₁₀</u>	<u>LOW FREQ. SLOPE</u>	<u>HIGH FREQ. SLOPE</u>	<u>Q₁₀</u>	<u>LOW FREQ. SLOPE</u>	<u>HIGH FREQ. SLOPE</u>
EA	5.1	86.0	73.0	5.8	93.0	85.0
JH	4.4	81.3	60.5	5.1	78.5	76.0
DS	11.0	85.0	311.0	11.0	88.0	279.0
MW	5.6	57.0	111.0	8.4	94.0	153.0

FIGURE 2. AVERAGED STCs FOR EA WITH AND WITHOUT CONTRALATERAL NOISE PRESENT

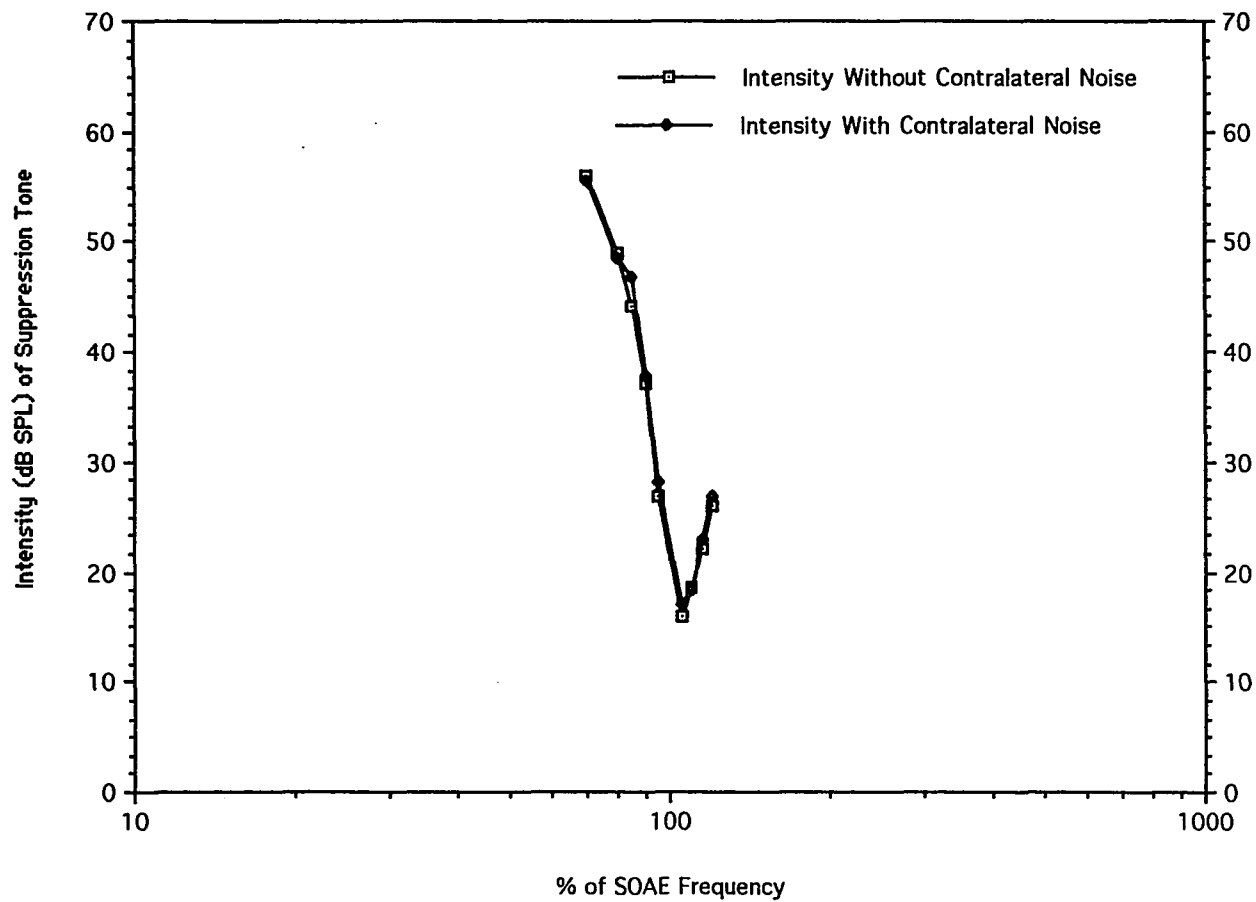


FIGURE 3. AVERAGED STCs FOR JH WITH AND WITHOUT CONTRALATERAL NOISE PRESENT

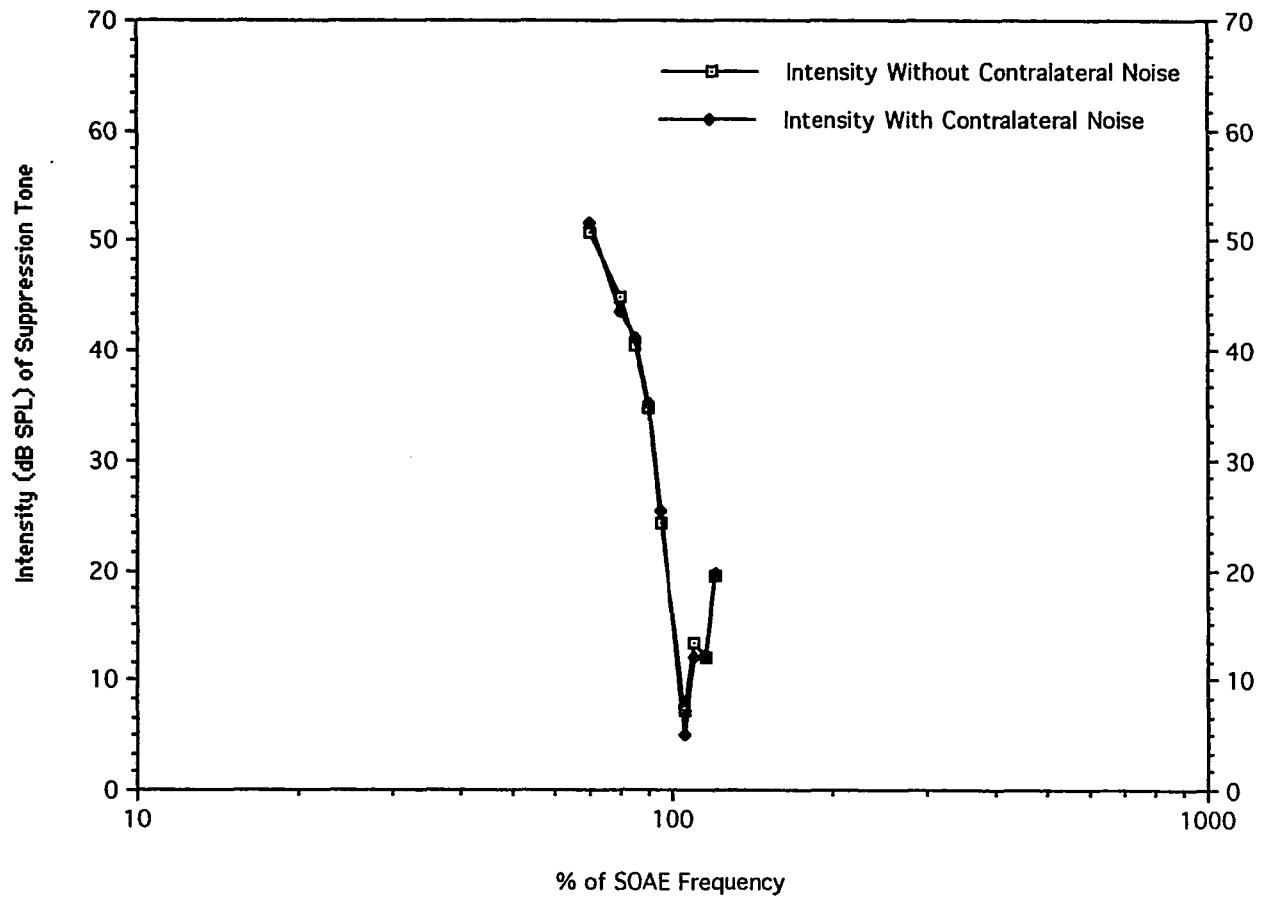


FIGURE 4. AVERAGED STCs FOR DS WITH AND WITHOUT CONTRALATERAL NOISE PRESENT

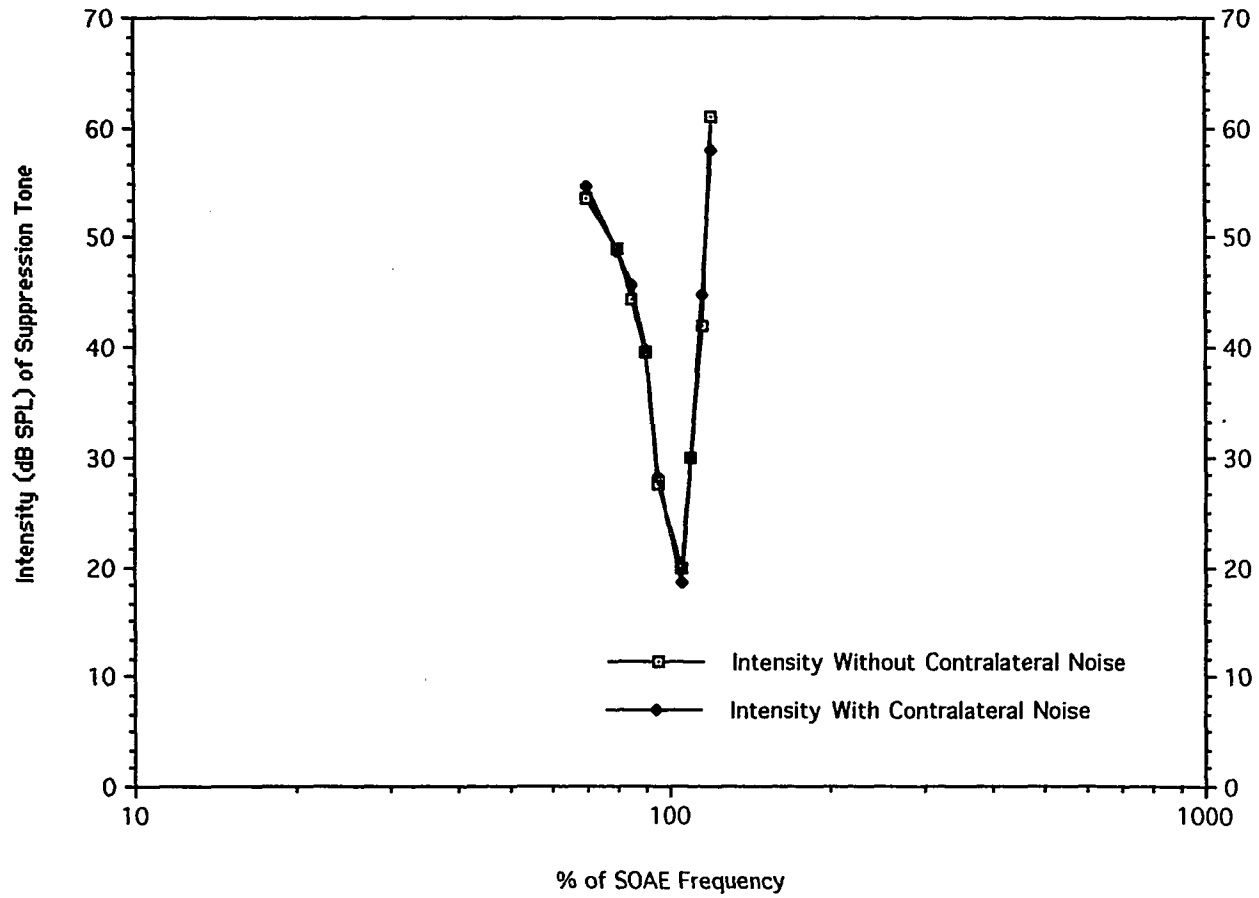
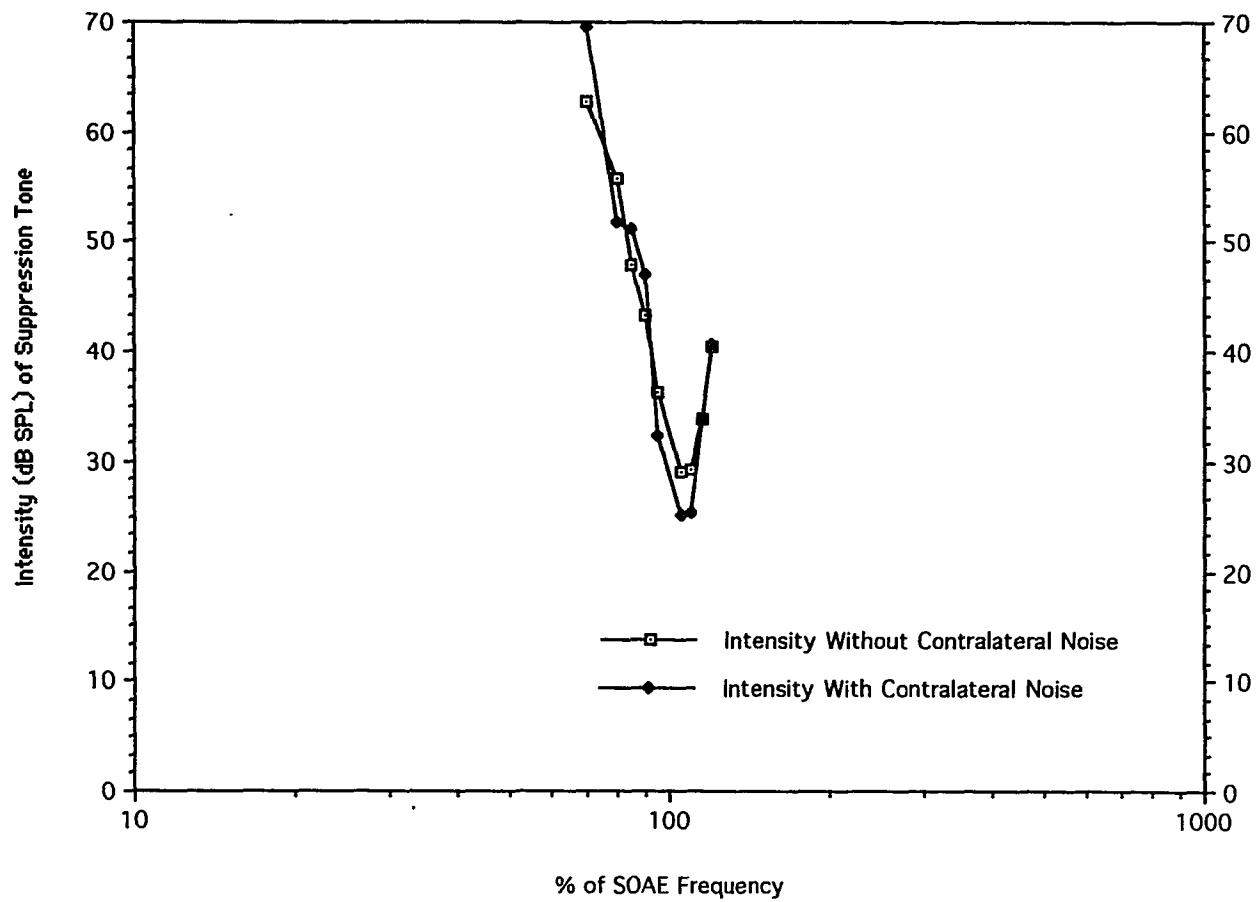


FIGURE 5. AVERAGED STCs FOR MW WITH AND WITHOUT CONTRALATERAL NOISE PRESENT



Human Subject Committee

March 27, 1992

Carolyn J. Tooley, B.A.
c/o Theodore Glattke, Ph.D.
Department of Speech & Hearing Sciences
Main Campus

THE UNIVERSITY OF
ARIZONA
HEALTH SCIENCES CENTER

1690 N. Warren (Bldg. 526B)
Tucson, Arizona 85724
(602) 626-6721 or 626-7575

33

RE: **HSC A92.44 THE EFFECTS OF CONTRALATERAL STIMULATION ON THE SUPPRESSION TUNING CURVES OF SPONTANEOUS OTOACOUSTIC EMISSIONS**

Dear Ms. Tooley:

We received your revised consent form for your above referenced research proposal. The procedures to be followed in this study pose no more than minimal risk to participating subjects. Regulations issued by the U.S. Department of Health and Human Services [45 CFR Part 46.110(b)] authorize approval of this type project through the expedited review procedures, with the condition(s) that subjects' anonymity be maintained. Although full Committee review is not required, a brief summary of the project procedures is submitted to the Committee for their endorsement and/or comment, if any, after administrative approval is granted. This project is approved effective 27 March 1992 for a period of one year.

The Human Subjects Committee (Institutional Review Board) of the University of Arizona has a current assurance of compliance, number M-1233, which is on file with the Department of Health and Human Services and covers this activity.

Approval is granted with the understanding that no further changes or additions will be made either to the procedures followed or to the consent form(s) used (copies of which we have on file) without the knowledge and approval of the Human Subjects Committee and your College or Departmental Review Committee. Any research related physical or psychological harm to any subject must also be reported to each committee.

A university policy requires that all signed subject consent forms be kept in a permanent file in an area designated for that purpose by the Department Head or comparable authority. This will assure their accessibility in the event that university officials require the information and the principal investigator is unavailable for some reason.

Sincerely yours,

William F. Denny

William F. Denny, M.D.
Chairman
Human Subjects Committee

WFD:rs

cc: Departmental/College Review Committee

REFERENCES

- Bilger, R.C., Matthies, M.L., Hammel, D.R., & Demorest, M.E. (1990). Genetic implications of gender differences in the prevalence of spontaneous otoacoustic emissions. Journal of Speech and Hearing Research, 33, 418-432.
- Collet, L., Kemp, D.T., Veillet, E., Duclaux, R., Moulin, A., & Morgon, A. (1990). Effect of contralateral auditory stimuli on active cochlear micro-mechanical properties in human subjects. Hearing Research, 43, 251-262.
- Davis, H. (1983.) An active process in cochlear mechanics. Hearing Research, 9, 79-90.
- Glatcke, T.J., & Kujawa, S.G. (1991). Otoacoustic emissions. American Journal of Audiology: A Journal of Clinical Practice, 1, 29-40.
- Grope, J.H. (1983). The effect of contralateral stimulation on spontaneous otoacoustic emissions. Journal of the Acoustical Society of America, 74, Supplement 1, S38.
- Harris, F.P., & Glatcke, T.J. (1992). The use of suppression to determine the characteristics of otoacoustic emissions. Seminars in Hearing, 13, 67-80.
- Kemp, D.T. (1978). Stimulated acoustic emissions from the human auditory system. Journal of the Acoustical Society of America, 64, 1386-1391.
- Kemp, D.T. (1979). Evidence of mechanical nonlinearity and frequency selective wave amplification in the cochlea. Archives of Otorhinolaryngology, 224, 37-45.
- Kiang, N.Y.S., Liberman, M.C., Sewell, W.F., & Guinan, J.J. (1986). Single unit clues to cochlear mechanisms. Hearing Research, 22, 171-182.
- Kujawa, S.G., & Glatcke, T.J. (1989). Influence of contralateral acoustic stimulation on spontaneous otoacoustic emissions. Asha, October, p. 123.
- Kujawa, S.G., & Glatcke, T.J. (1990). Influence of contralateral stimulus bandwidth on spontaneous otoacoustic emissions. Asha, October, p. 156.
- Mott, J.B., Norton, S.J., Neely, S.T., & Warr, W.B. (1989). Changes in spontaneous otoacoustic emissions produced by acoustic stimulation of the contralateral ear. Hearing Research, 38, 229-242.
- Mountain, D.C. (1980). Changes in endolymphatic potential and crossed olivocochlear bundle stimulation alter cochlear mechanics. Science, 210, 71-72.

- Rabinowitz, W.M., & Widin, G.P. (1984). Interaction of spontaneous oto-acoustic emissions and external sounds. Journal of the Acoustical Society of America, 76, 1713-1720.
- Ryan, S., Kemp, D.T., Hinchcliffe, R. (1991). The influence of contralateral acoustic stimulation on click-evoked otoacoustic emissions in humans. British Journal of Audiology, 25, 391-397.
- Schloth, E., & Zwicker, E. (1983). Mechanical and acoustical influences on spontaneous otoacoustic emissions. Hearing Research, 11, 285-293.
- Siegel, J.H., & Kim, D.O. (1982). Efferent neural control of cochlear mechanics? Olivocochlear bundle stimulation affects cochlear biomechanical nonlinearity. Hearing Research, 6, 171-182.
- Zizz, C.A., & Glatke, T.J. (1988). Reliability of spontaneous otoacoustic emission suppression tuning curve measures. Journal of Speech and Hearing Research, 31, 616-619