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Introduction

Several reports in the last decade have detailed the incidence and activity of spontaneous otoacoustic emissions (SOAEs) as measured in the sealed ear canal. These emissions have been generally described as narrow-band or tonal, occurring mainly in the 1-2 kHz region, up to 20 dB SPL, continuous, and "finger-printlike" in terms of their stability and uniqueness for each ear (38, 13, 32, 2, 28, 29, 23, 3). SOAEs are seldom perceived by the subjects and have not been shown to be correlated to tinnitus (31, 1). Experimental evidence of spontaneous, as well as of the related evoked and synchronized, otoacoustic emissions indicates that they result from an auditory transducer in the cochlea that functions as a feedback mechanism (18, 38). These findings have helped spur along a major change in the thought regarding the cochlea's action in processing sound (see 11 and 22 for background).

While studies have shown that SOAEs are common in humans, the same is not true for all animal species (36, 38, 25, 21). In their work with chinchillas, Zurek and Clark (1981) found two SOAEs in animals only after exposure to high-intensity noise. From this, they hypothesized that SOAEs may be related to cochlear damage. Thus, spontaneous otoacoustic emissions could prove to be useful in providing a noninvasive means of learning more about cochlear function.

Another phenomenon possibly related to cochlear irregularities is a rippling seen in audiogram microstructure, i.e. thresholds obtained at small frequency intervals. These bidirectional changes in sensitivity as a function of small changes in frequency were first noted by Elliot (1958), who described them as peaks and valleys (sensitivity minima and maxima, respectively) that occurred mainly under 2 kHz and that were of a unique and consistent pattern for a given ear. This has since been correlated by several workers (30, 19, 33, 35, 10, 26, 20). Kemp (1979) and others have commented on the correlation between patterns in audiogram microstructure and the presence of SOAEs in humans. It may be that both phenomena arise from a disruption in the same active cochlear mechanism.

Our aim in this study is to evaluate the audiogram microstructure of a chinchilla with a documented spontaneous otoacoustic emission. This chinchilla presents a unique opportunity to examine relationships between acoustical and audiometric data in an organism whose environment and exposure to noise are well controlled, and, at some point in the future, to complete this with histological findings. To our knowledge no other fine-structure audiometric data have been obtained from a chinchilla.

METHODS

Subject

The subject for this experiment was chinchilla No. 184, who was born and raised in a sound-shielded laboratory colony (5). The animal was rendered functionally monaural by removal of the malleus and incus from its left ear (9). Prior to noise exposure, no otoacoustic emission was detected in the test (right) ear. The chinchilla was subsequently exposed to two 9-day durations of 0.5 kHz octave band noise at 95 dB SPL.

SOAE Recording

On January 25, 1985, approximately one month following the second noise exposure, the animal was prepared for acoustic search (8). Anesthesia (45 mg/kg sodium pentobarbitol injected intraperitoneally) was administered and a Knowles EA 1842 microphone ~~was~~ attached to a #7 Grason-Stadler ear probe at the distal end of one of three tubes was sealed in the chinchilla's right ear canal. This probe assembly was connected to a Hewlett-Packard 358 A sweep frequency Wave Analyzer. Following insertion, a 4 Hz band filter was swept across the frequency spectrum.

An SOAE was detected at 2150 Hz at a level of approximately 10 dB SPL. In a quiet environment, a normal-hearing listener could hear

this sound by listening at the animal's ear canal. Explorations over the next several months showed that the SOAE had increased somewhat in frequency, staying around 2200 Hz. At the present time the emission is still audible in a quiet environment.

Behavior Training and Testing

After ossicular removal, the animal was trained to report the presence of pure tones in a food-reward operant conditioning procedure (4, 6). Thresholds were obtained through a computerized adaptive tracking method. The animal signalled readiness for a test retrial by pressing and holding down a lever. Within a variable time frame of 1-7 seconds, either a tone of 2 seconds duration would be presented or a blank trial would occur. The response latency window for reporting a signal by releasing the lever was 2 seconds. Hits and correct rejections, i.e., holding down the lever throughout the trial, were rewarded with food pellets (Noyes 45 mg.)

Correct detection of an initial, moderately-high level stimulus resulted in its attenuation in 10 dB steps until a miss was recorded. Attenuation was then increased (i.e., decrease in stimulus intensity) in 5 dB increments until a hit was again recorded. Thereafter, the attenuation scheme was down and up in 2.5 dB steps until six alternations from hit to miss have occurred at 2.5 dB step size. Thresholds were determined by taking the average levels of the six reversals. In a typical

session lasting an hour, approximately ten thresholds could be obtained.

Throughout training and testing, the subject was not allowed to go under 80% of its ad libitum weight. To ensure good stimulus control, a certain percentage of blank trials were mixed in with the tone trials. This percentage was adjusted to keep the false alarm rate (number of false alarms divided by the number of silent trials) between 5-15%; for this animal the resulting tone probability was 67-75%. As an additional safeguard, a standard deviation ceiling was of 7 dB was set. The test frequencies were randomized by the computer program.

Apparatus

Audiometric test sessions were conducted with the animal inside a double-walled, sound-shielded booth (IAF Model 1202). The booth was lined with foam on all inner surfaces. A cage built of 1.27 cm. hardwire cloth was centered approximately 0.75 m. above the ground. Immediately outside the cage a BRS/LVE PDC/PPD Series Pellet Dispenser was placed with the pellet chute and lever descending into the cage. In the right corner two speakers, one producing a test signal and one a suppressor signal, were placed approximately 1.5 m from and on a horizontal plane with the cage.

Pure test tone stimuli ~~was~~ were generated by a Hewlett-Packard 3325-A synthesizer and function generator. An interface monitor built at

Central Institute for the Deaf's electroacoustics laboratory was used to visually monitor stimulus frequency, attenuation, and the subject's response and subsequent reinforcement.

For threshold trials incorporating a suppressor tone, a Wavetek Model 148 signal generator was used to produce the suppressor tone. The signal was first amplified by a Crown 060 amplifier, then directed to a Hewlett-Packard 350D attenuator. Signal frequency was monitored by a Bruel and Kjaer 1827 frequency counter and output intensity was measured by a Hewlett-Packard 400F voltmeter.

All test frequencies were calibrated by measuring the dB SPL output of each frequency at 10 dB attenuation at the location of the subject's right ear during testing. For this, a Bruel and Kjaer 2112 audio frequency spectrometer with a 1/2" coupler was used. A similar procedure was used to calibrate the suppressor signal level with the sole variation being attenuation level.

Signal Parameters

The test stimuli used in evaluating audiogram microstructure were pure tones ranging from 1710-2490 Hz at 10 Hz intervals. These tones were 2 seconds in duration, with a rise-fall time of 50 ms. The suppressor tone was a 2200 Hz pure tone attenuated to either 10 or 20 dB SPL. It was left continually on during a given suppressor tone trial.

RESULTS

General Audiogram

Data on the subject's hearing sensitivity were obtained from 0.125-16 kHz at 1/4 octave intervals and are shown in graph 1. As the chinchilla was not trained until after the noise exposure, no baseline audiogram is available. However, comparison of this graph with average hearing thresholds from normal-hearing chinchillas shows a mild threshold shift from 0.5-3 kHz (graph 2). It was noticed that the greatest threshold shift occurred in the same frequency region as the SOAE; this discovery prompted us to evaluate threshold microstructure in that region (shaded area in graph 1).

Audiogram microstructure

Thresholds obtained between 1700-2500 Hz at 10 Hz intervals are shown in graph 3. For the first 400 Hz the microstructure consists of a series of small fluctuations. These range around 24 dB plus or minus 4 dB. At 2090 Hz a well-defined downward slope to 18.6 dB at 2160 Hz is seen. This is followed by a sharp rise to 25 dB at 2190 Hz and, subsequently, by a drop to 14 dB at 2230 Hz. This microstructure takes the form of a prominent "bump" which shows the only plateau throughout the range tested. Thereafter, a general rise in threshold occurs in the higher frequencies tested. It is noteworthy that the frequency range of the strongest fluctuation - the large "bump" described - matches closely the range in which the emission has been recorded.

Effects of suppressor tone on threshold at 2200 Hz

Since the microstructure peak occurring in the SOAE frequency region bears resemblance to a pure tone masking function (e.g., Small, 1959), a separate procedure was devised to test the possibility that the SOAE could be acting as an internal masker. Studies have demonstrated that external tones even at subthreshold levels can partially suppress otoacoustic emissions if they are sufficiently close in frequency to the emission (7, 38, 24, 36, 33).

It was hypothesized that if the SOAE were producing an internal masking effect on thresholds for test stimuli in its frequency range, then its suppression by a subthreshold external tone should result in lowered thresholds. Thresholds at 2200 Hz were obtained as before in the presence of continuous 2200 Hz tones at 10 and 20 dB SPL, respectively, in separate sequences. Tone trials with and without the external tone were alternated in an ABAB sequence.

In the 10 dB suppressor tone paradigm the mean threshold in the external tone condition was 15.5 dB, whereas the mean threshold without the tone was 15.4 dB. There is no significant difference between the two conditions, indicating that the emission did not act as internal masker. It must be noted, however, that these thresholds are over 8 dB lower than the thresholds obtained in the fine-frequency audiogram. Possible explanations include the perception of beats near threshold (Wilson, 1980), especially if

the SOAE had shifted in frequency, and a host of other factors that could occur in the time elapsed between the test procedures. On the other hand, the thresholds obtained in the 20 dB suppressor paradigm, which average to 24.34 dB with the external tone and 26.47 dB without, also indicating no significant effect of the emission on threshold at 2200 Hz, are much closer to that measured in the fine-frequency audiogram.

DISCUSSION

Thirty years before their actual discovery, spontaneous otoacoustic emissions were predicted by Gold (1948) in his model of an active cochlear processing mechanism. As he noted, "the assumption of a 'passive' cochlea, where elements are brought into mechanical oscillation solely by means of the incident sound, is not tenable." Gold attributed the cochlea's selective frequency-tuning ability at low intensities to a feedback channel that served as a resonator, enhancing basilar membrane movement at threshold intensities. He then conjured that "if the feedback ever exceeded the losses" of energy in the transduction process "then a resonant element would become self-oscillatory."

Kemp's (1978) subsequent identification of evoked acoustic emissions in human ears prompted a wave of research on emissions of various types - evoked, spontaneous and synchronized - in both human and animal populations. There is a strong consensus in the literature that otoacoustic emissions do in fact originate from the cochlea and indicate the presence of active cochlear processing. Evidence of this includes the emissions' latency (when a stimulus is involved), susceptibility to ototoxic agents, similarity of suppression curves to frequency tuning curves, non-linearities arising from interaction with external acoustic stimuli, and other phenomena (19, 33, 38, 1, 7, 11, 22).

	time	frequncy in Hz	amplitude in dB SPL
1	March 1987	2150.000	10.000
2	August 1987	2200.000	10.000
3	July 1988	2137.000	25.000
4	August 1988	2200.000	40.500
5	September 1988	2262.000	50.400
6	December 1988	2275.000	50.000
7	March 1990	0.000	0.000
8	April 1990	2312.000	30.000
9	Ma 1990	0.000	0.000

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Changes in spontaneous otoacoustic emissions produced by acoustic stimulation of the contralateral ear *

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pontaneous otoacoustic emissions (SOAEs) were measured in human ear canals before, during and after presentation of tonal li to the contralateral ear. Stimuli were presented in 1/8 octave steps from 2 octaves below to 1 octave above the SOAE ved contralateral acoustic reflex threshold. For certain conditions there was an abrupt For a fixed level the effect was frequency selective; the maximum frequency shift was ave below the SOAE. SOAE amplitude usually decreased but in some cases increased or nges were observed, the maximum shifts were observed for tones at or near the SOAE bserved for stimulus levels below 60 dB SPL. The effect is believed to be mediated by livocochlear bundle which arise in the medial region of the superior olivary complex and ese results support those models which attribute SOAE generation to OHCs, and are ear mechanics. A simple model is presented that proposes that efferent activity alters the g changes in OHC membrane conductance.

Hair cells, outer; Spontaneous otoacoustic emissions; Efferent activity

Phys 8-78-3077 McClaine EL-Fouly

ns (SOAEs) are d in the mam- external stimu- e of SOAEs has vided support for models of cochlear mecha- which assume that active, nonlinear biomech- al elements are responsible for the characteris- high sensitivity and sharp tuning of the cochlea Kim, 1986; Neely and Kim, 1986). These active elements are presumed to function collectively as a cochlear amplifier. Under certain conditions, arising either spontaneously or evoked by external stimuli, excessive gain is introduced in the amplifier resulting in oscillations which propagate out of the cochlea to the ear canal.

For several reasons, these active biomechanical elements are thought to be the outer hair cells (OHCs). Isolated mammalian OHCs exhibit a motile response to electrical and chemical stimulation (Brownell et al., 1985; Zenner, 1985; Ashmore and Brownell, 1986). In addition, because of their firm attachments to the tectorial (Kimura, 1966; Takasaka et al., 1983) and basilar (Slepecky and Chamberlain, 1983) membranes, OHCs are considered to be bidirectionally coupled to the cochlear partition. Therefore, they are capable of the mechanical-to-electrical transduction required for hearing, as well as the electrical-to-mechanical

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rtions of this work were presented at the 10th Midwinter Meeting of the A.R.O. (Mott, J.B., Norton, S.J., Neely, S.T. and Warr, W.B. (1987) Abstr. Assoc. Res. Otolaryngol. 10, 71).

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The question still remains, however, as to whether SOAEs are a manifestation of normal cochlear processing or a result of some cochlear pathology. Surveys of the incidence of SOAEs in humans have thus far shown that whereas 1/3 to 1/2 of normal human ears possess at least one emission (38, 32, 2, 28, 3), only two cases have been documented in which SOAEs occurred in regions with hearing losses greater than 30 dB HL (15, 34). Fritze (1983) notes that twelve subjects with "minor inner ear deafness" showed an elevated incidence of SOAEs relative to subjects with normal audiograms but that no emissions were found in twelve subjects with "severe inner ear deafness" (losses of 30 dB or more). In a study of SOAEs in children, Strickland et al (1985) hypothesized that if SOAEs are associated with outer hair cell damage, the incidence should increase with length of exposure to damaging sounds. They found the incidence of emissions in both the infant and child populations not to be significantly different from that of adults.

From the results of these studies it appears that a correlation between spontaneous otoacoustic emissions and cochlear pathology is tenuous. But it must be pointed out that the studies above defined their subjects as normal hearing based on clinical audiometric results at octave intervals. It is possible that with the interval size utilized in conventional audiometry subtle cochlear lesions could easily be missed (7).

The idea that spontaneous otoacoustic emissions may result from cochlear trauma arose from the discovery that SOAEs in chinchillas were only found in noise-exposed animals who were also shown, upon post-mortem histological analysis, to have punctate lesions in the organ of Corti at regions corresponding to the frequencies of the emissions (38, 7). On the basis of these data, Clark et al (1984) hypothesized that two conditions, a localized area of damage surrounded by normal regions in the organ of Corti, were "necessary and sufficient for an SOAE to occur." If this is a disruption of the feedback mechanism hypothesized by Gold, then an SOAE could be the predicted "self-oscillatory element."

When more detailed audiometric data of human and animal subjects have been obtained, the results have been interesting. Bright et al (1985) reported that "it has been discouraging to find that spontaneous emissions do not appear to occur in ears that have hearing loss above 30 dB." Immediately thereafter, however, they note that SOAEs "do co-exist with reported irregularities in the audiometric threshold pattern when the thresholds are measured with continuous sweep-frequency techniques...." They do not elaborate on the nature of these irregularities except to say that their basis "remains to be seen."

Both Ruggero et al (1983) and Clark et al (1984) have demonstrated that emissions appear to occur in regions showing sharp changes in hearing sensitivity when thresholds are measured at 1/4 octave or

smaller intervals. The Ruggero et al study's human emission occurred at 7.5 kHz, a frequency showing a sudden increase in sensitivity after a region of hearing loss. Even more noteworthy are Clark et al's findings on chinchilla No. 3476-R. In this animal the SOAE occurred at 6470 Hz, the only frequency from 125-16000 Hz showing no permanent threshold shift following noise exposure. These results indicate that the emissions are occurring in regions of the cochlea that, when assessed through detailed audiometric techniques, are unique.

Schloth (1983) and Zwicker and Schloth (1984) examined threshold microstructure and SOAE distribution in humans. They concluded that strong otoacoustic emissions of any sort "are directly related to high sensitivity of hearing at low levels." Similar relations have been found between evoked emissions and minima (areas of maximum sensitivity) in audiogram microstructure (17, 35, 19, 33).

The fine-structure audiometric results with chinchilla No. 184 also show that the animal's SOAE is correlated to sharper changes in hearing sensitivity than seen in other frequency regions. While the emission has fluctuated within the range stated, it appears to occur most often at 2200 Hz, on a downward slope towards a threshold minimum in the microstructure. This is consistent with findings in humans. The fact that the presence of a suppressor tone at the frequency of the emission did not alter

thresholds significantly leads us to believe that the sharp fluctuations in hearing sensitivity ~~is~~ are not due to internal masking, as some in the literature have hypothesized (34).

The results of this study must be interpreted with caution. More single-study subjects of this sort need to be carried out before we can draw general conclusions about any significance spontaneous otoacoustic emissions may have in helping us better understand the effects of noise on the cochlea. This is especially true in attempting to make inferences about the significance of SOAEs and cochlear function in humans from animal data. McFadden et al (1983) noted that it is not clear if human and animal auditory systems are the same. With regard to this important point, however, we feel that, with auditory sensitivity and frequency response being the most similar to the human auditory systems of all animals, the chinchilla is the most appropriate animal model for studying the ear. In addition, further studies of chinchillas with emissions would be strengthened by the following methodological variations: 1) obtaining a baseline audiogram prior to noise exposure to ensure that the threshold shift is valid, 2) measuring the SOAE more frequently, 3) measuring any effects that suppressor tone may have on the SOAE--threshold changes may also result if the test tone, suppressor tone and/or the emission combined to form beating.

Despite its preliminary nature, we feel that the results of our audiometric study on this chinchilla with a spontaneous otoacoustic emission are significant and warrant further studies of hearing sensitivity in subjects with emissions.

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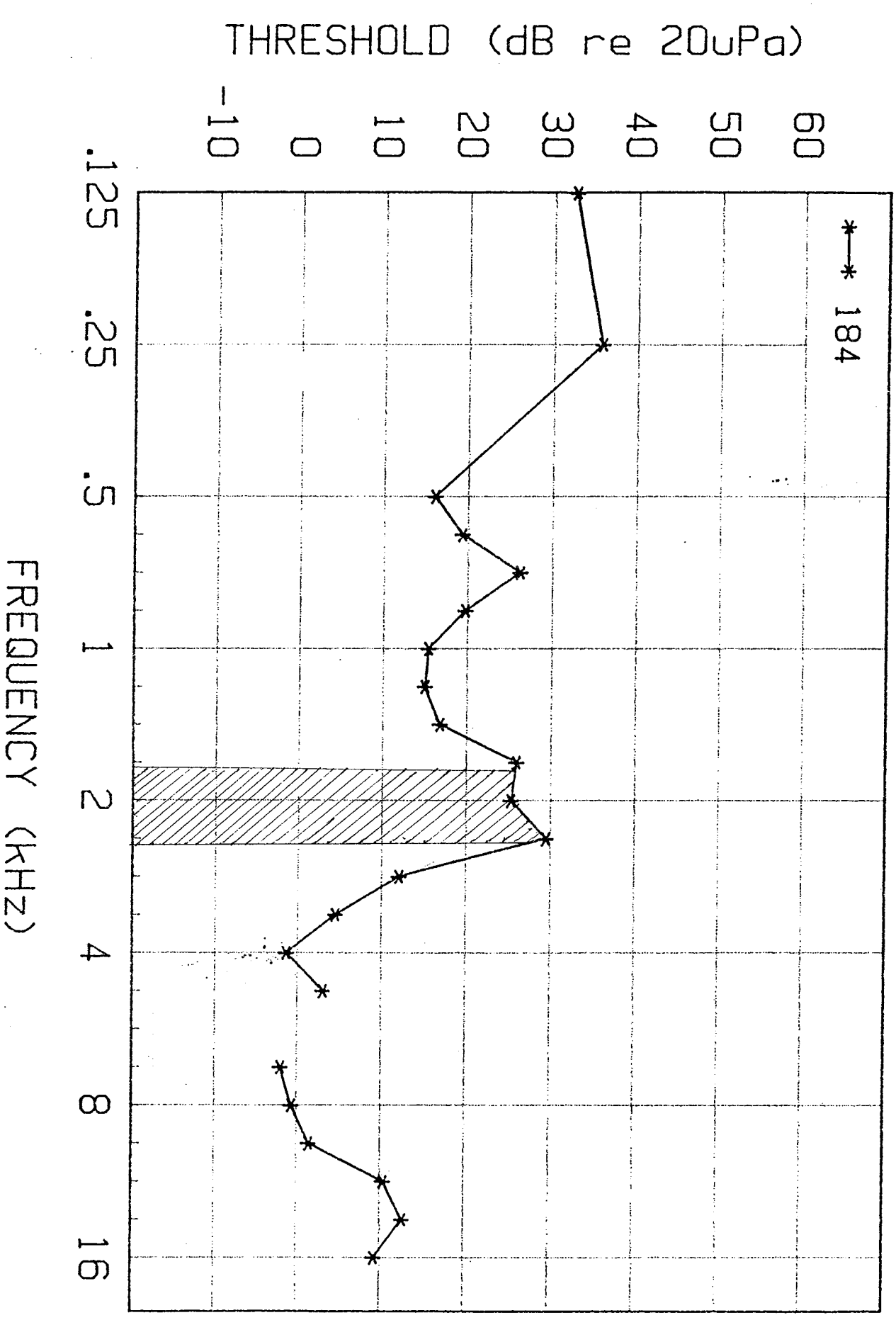
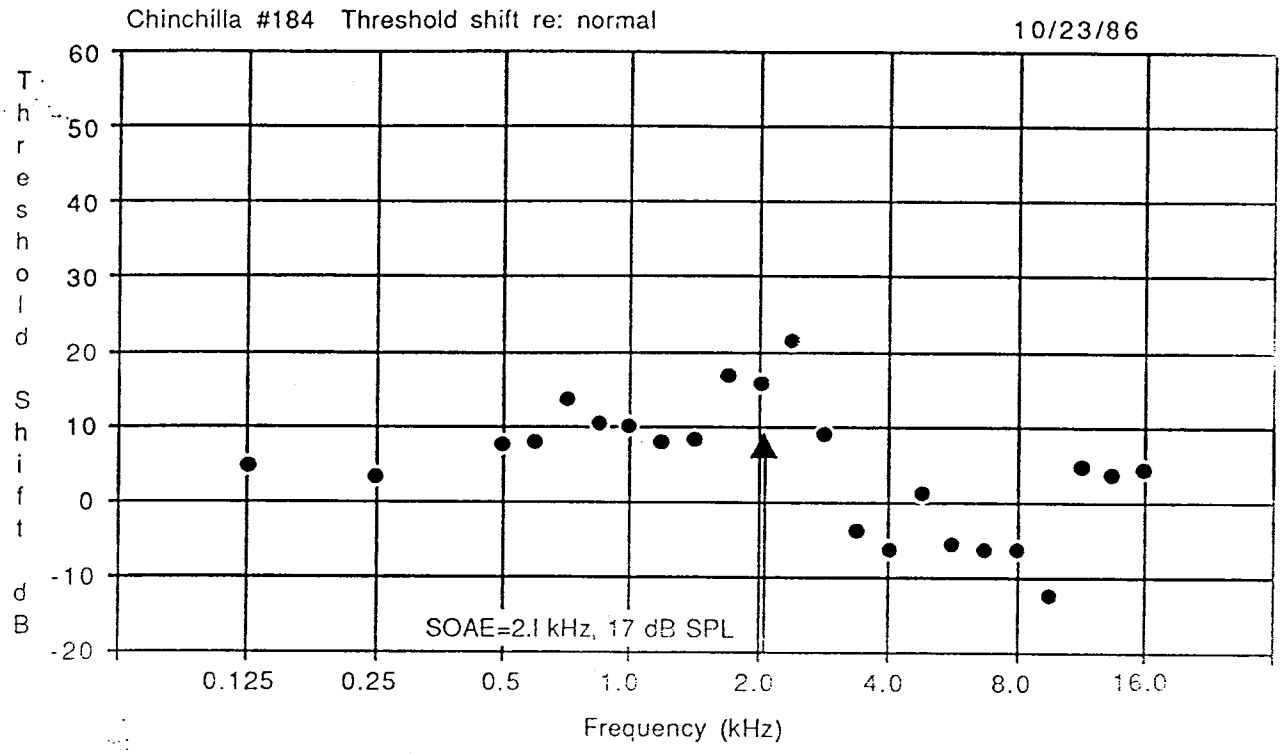
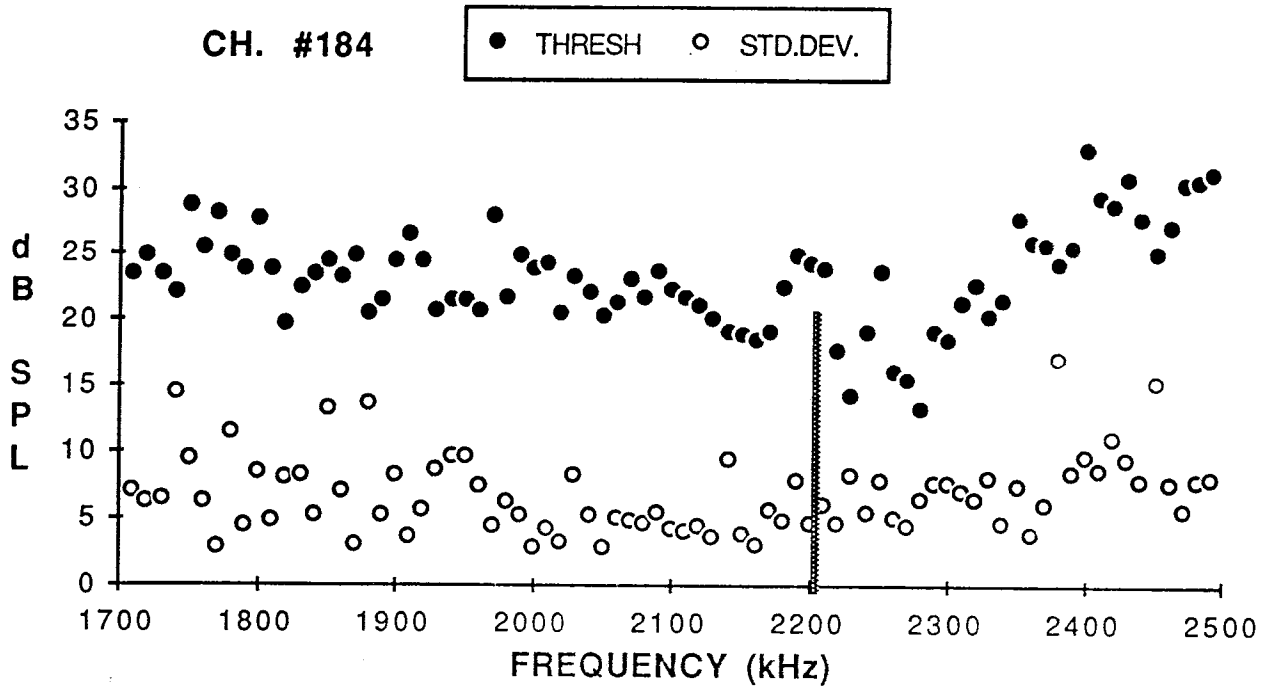

THRESHOLD (dB re 20uPa)
-10
0
10
20
30
40
50
60
.125
.25
.5
1
2
4
8
16
FREQUENCY (KHz)
184

graph 1


Chinchilla #184 Threshold shift re: normal
10/23/86
Threshold Shift dB
60
50
40
30
20
10
0
-10
-20
SOAE=2.1 kHz, 17 dB SPL
0.125
0.25
0.5
1.0
2.0
4.0
8.0
16.0
Frequency (kHz)

graph 2


CH. #184
THRESH
STD.DEV.
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dB SPL
1700
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1900
2000
2100
2200
2300
2400
2500
FREQUENCY (kHz)

graph 3