1992

Spontaneous otoacoustic emissions

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SPONTANEOUS OTOACOUSTIC EMISSIONS

Independent Study

by

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ACKNOWLEDGMENT

I express my deep gratitude to Dr. W. Clark, Chairman Committee, in Communication Sciences, Central Institute for the Deaf, Washington University, for his guidance, help and encouragement, Dr. S. Highstein, Professor of Neuroscience Department, Washington University, School of Medicine, for his permission to use his laboratory, his great support, help and advise, Dr. J Goldstein, Senior Research Scientist, Central Institute for the Deaf, Washington University, for his advice and support, A. Heidbreder, Senior Design Engineer, Central Institute for the Deaf, Washington University, for his help in the technical and engineering part, and Cindy Wells, Head of animal care, Central Institute for the Deaf, Washington University, whose assistance in animals training made the work possible.

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I. INTRODUCTION

INTRODUCTION
Review of literature

"Otoacoustic emissions are low-level sounds produced by vibratory motion in the cochlea. When caused by external stimuli, they can be detected in the external auditory canal (evoked emissions). However, they can also occur as spontaneous oscillations (spontaneous otoacoustic emissions--SOAEs) within the inner ear" (Kemp, 1978; Zurek, 1981).

"Otoacoustic emissions constitute a release of audiofrequency energy into the ear canal from the cochlea. They are transmitted through the middle ear to the cochlea. Emissions must be considered as an energy leak from the cochlear travelling wave" (Kemp, 1986).

"There are four different kinds of acoustic emissions. Those which can be picked up in the closed ear canal by a sensitive microphone without external stimulation, i.e. in quiet, are called spontaneous emissions" (Zwicker and Schloth, 1984). "These emissions occur continuously. Delayed evoked emissions are seen as responses to short sound impulses and show up after a delay of some 10 ms. Synchronous evoked emissions, appear as a response to a continuous tone; they have the same frequency as the stimulating tone and are synchronous with it so the emission has to be separated through special methods" (Zwicker and Schloth, 1984). "Finally, evoked distortion products are continuous and essentially tonal signals that appear at the difference frequency (f2-f1), at the cubic

difference frequency (2f1-f2), or at some other combination (3f1-2f2, 2f2-f1 etc.) of continuous primary tones introduced by the experimenter" (Kemp (1979); Kemp (1980); Rosowski et al (1984)).

"Spontaneous otoacoustic emissions consist of a narrow band signal that can be measured in approximately 40 % of normal ears in the absence of deliberate stimulation" (Schloth (1982); Kemp (1979); Zurek (1981); Wier, et al (1984); Probst, et al (1986), Norton et al 1989)). The other types of emissions are present in almost all normal ears. (Schloth (1982); Kemp (1979); Zurek (1981); Wier, et al (1984); Probst, et al (1986); Grandori, (1983)).

Zwicker and Schloth (1984) concluded that: "delayed evoked, synchronous evoked, and spontaneous otoacoustic emissions are closely related to each other. Certain rules which can be valid for all four types of emissions can be observed. This implies that they originate at the same source in the cochlea, enabling them to have parallel hydromechanical characteristics. From this point of view, the correlation between the emissions and threshold can be understood as internal slightly damped or even undamped resonances; objectively measured emissions are reflected in hearing sensitivity".

"Spontaneous otoacoustic emissions are low-level acoustic signals measured in the mammalian ear canal in the absence of external stimulation" (Kemp (1978); Kemp (1979); Zurek(1981); Schloth (1983); Wier et al(1984)). "Otoacoustic emissions have aroused a great deal of interest because they are believed to

originate from within the cochlea" (Wilson, 1980; Zurek, 1981). They may provide the basis of a sensitive and non-invasive method for examining cochlear function in humans.

"Spontaneous otoacoustic emissions consist of one or several sinusoid-like signals" (Kohler& Fritze (1986)). Other studies describe the emissions as "narrow band acoustic signals" (Kemp (1979); Wilson (1980)). "Spontaneous otoacoustic emissions comparable to pure sinusoidal tones. This is surprising because pure tones occur rarely in nature; most authors have described the SOAEs as narrow-band acoustic signals" (Wit, et al (1981)).

"A healthy ear is necessary for the expression of SOAEs". Bonfils (1989), Norton et al (1989) suggested that "about one-third of normal human ears exhibit spontaneous otoacoustic emissions (SOAEs)". Other studies suggested that "the existence of spontaneous otoacoustic emissions has been well documented and that they now appear to be common in seemingly normal-hearing humans" (Kemp (1979); Wilson (1980); Wilson (1981); Wit et al (1981); Zurek (1981)). However, Zurek, and Clark (1981) found that "narrow band otoacoustic emissions can apparently be induced by traumatic noise exposure in chinchillas. This suggests that these signals may be associated with microlesions in the organ of Corti and are not a result of normal cochlear function. Accordingly, the high incidence of otoacoustic emissions in humans and zero incidence in their sample of unexposed chinchillas may reflect the vastly different histories of noise exposures of humans and these chinchillas".

"The existence of SOAEs has provided support for models of cochlear mechanics which assume that active, nonlinear biomechanical elements are responsible for the characteristic high sensitivity and sharp tuning of the cochlea" (Kemp (1986); Neely and Kim (1986)). "These active elements are presumed to function collectively as a cochlear amplifier. Under certain conditions, either arising spontaneously or evoked by external stimuli, excessive gain is introduced in the amplifier resulting in oscillations which propagate to the ear canal. For several reasons, these active biomechanical elements are thought to be the outer hair cells (OHCs)" (Mott, et al(1989)). "Isolated mammalian OHCs exhibit a motile response to electrical and chemical stimulation" (Brownel, et al (1985); Zenner (1985); Ashmore and Brownel (1986)). In addition, because of their firm attachments to the tectorial and basilar membranes (Kimura, (1966); Takasaka, et al (1983); Slepecky and Chamberlain (1983)), "outer hair cells are considered to be coupled bi-directionally to the cochlear partition. Therefore, they are capable of the mechanical-to-electrical transduction required for hearing, as well as the electrical-to-mechanical transduction required for the generation of otoacoustic emissions" (Mott, et al (1989)).

"It is generally accepted that the auditory sensory cells are mechanoreceptors that transduce the mechanical stimulus (sound) into electrical impulses. Sound causes bending of the steriociliary bundles of hair cells alternately in the excitatory and inhibitory directions. Steriociliary bending in the excitatory direction is

believed to trigger the release of afferent neurotransmitters at the synaptic sites of the hair cells from which neural impulse emanate" (Dallos (1985)). Because both IHCs and OHCs possess the fundamental morphological characteristics of mechanoreceptors, namely the steriociliary bundles and nerve synapses, it has been generally assumed that both types of hair cells are passive mechanoelectrical transducers.

However, the recent observation by Kemp (1978) that "sound can also be emitted from the cochlea (evoked or spontaneous otoacoustic emission) suggests that the inner ear is also an electromechanical transducer". "For otoacoustic emission to take place, there must be a mechanically active component coupled to the basilar membrane. So reverse processing of sound transduction can occur, causing a vibration of the basilar membrane that is transmitted to the tympanic membrane" (Lim (1986)) . The cellular-biology background helps explain both motile activity of the sensory system and how the motile activity is generated and transmitted, but it does not explain otoacoustic emission itself. Based on data from laboratory animals that produced spontaneous otoacoustic emission., Clark, et al (1984) demonstrated that "the emissions require functional distribution of a biomechanical control mechanism normally present in a region of the organ of Corti and the presence of a functionally active organ of Corti adjacent to the region". Kim (1984) postulated that "the steriocillia of the outer hair cells may be involved in this bidirectional (mechano-electrical and electro-mechanical) transduction process". Ruggero, et al (1984) suggested

that "both spontaneous and evoked otoacoustic emissions, are caused by the disruption of the active feedback mechanism of the OHCs on the basilar membrane. Otoacoustic emissions most likely result from the activity of the outer hair cells, that is either hyperactive or poorly inhibited by the impaired regulatory system (particularly the lateral efferent)".

Other studies have shown that some human ears spontaneously and continuously emit narrow-band acoustic signals that are measurable with a probe microphone in the ear canal (Wilson (1980); Zurek (1981)). "All experimental evidence provided by these studies indicated that the observed otoacoustic emissions (OAEs) are produced by spontaneous mechanical oscillation within the cochlea and, perhaps, of the cochlear partition. So otoacoustic emissions constitute a release of audiofrequency energy into the ear canal from the cochlea, transmitted through the ossicular chain and tympanum. The transmission of emissions to the ear canal is highly unlikely to be a functional part of the hearing mechanism. Rather emissions must be considered an energy leak from the cochlear travelling wave" (Kemp (1979)). There is a great deal of evidence which suggests that "this leak is an inevitable biomechanical property of the healthily functioning cochlea" (Johnsen & Elberling (1982); Wit (1979) & Ritsma (1979); Ruggero (1983)) "that gives rise to measurable psychophysical effects such as fine structure on the audiogram" (Kemp (1979)). "This evidence has led to renewed interest in Gold's idea (Gold (1948)) that active biomechanical

elements control the physical response of the cochlea to sound, amplifying vibration and improving performance" (Davis (1983)).

Ruggero, et al (1983) summarized the occurrence of both spontaneous otoacoustic emissions and evoked otoacoustic emissions as follows:

"1. Hair cells actively influence basilar membrane mechanics.

2. Positive feedback of the hair cells upon the basilar membrane results in amplification of the input mechanical signal at each basilar membrane site.

3. Each cochlear site exerts negative feedback upon itself and upon adjacent cochlear segments.

4. In an intact region of the cochlea, the positive and negative feedback cancel each other.

5. If an area of outer hair cell loss is adjacent to an area of relative normality, the loss of damping ordinarily produced by the area with outer hair loss will permit the normal area to induce its segment of basilar membrane to vibrate with larger amplitude than normal, at its own resonant frequency, impulsive-evoked OAEs will appear upon stimulation with clicks or short tone pips.

6. If the spatial transition between normality and abnormality is particularly sharp, basilar membrane oscillation will be generated in the absence of an external acoustic stimulus and SOAEs will appear in the external ear canal.

7. In a cochlear region where hair cell loss or dysfunction is extensive and continuous, no OAEs (including distortion products emissions and SFEs) can be generated".

In most of the studies, the levels of the SOAEs in human never exceed 20 dB SPL and their frequencies most commonly occur between 1 and 2 KHz (Zurek (1981)), Schloth (1983), Rabinowitz and Widin (1984)). The highest frequencies observed are 7.7 kHz (at 5.5 dB SPL, Zurek), 4.0 KHz (at -4 dB SPL, Schloth), and 5.9 kHz (at 3 dB SPL, Rabinowitz). SOAEs of similar frequency--7.5 Khz, but greater intensity--16 dB SPL have been reported by Rugero, et al (1983). Wilson (1986) reported that "most SOAEs occur in the range of -10 to +20 dB SPL. However in a few cases much higher levels have been reported. In one case investigated by Glanville, et al (1971). Glanville, Coles & Sullivan, (1971), a 4-year old boy had a measured level of 60 dB SPL at one ear canal entrance". Several authors (Kemp, 1980, 1981; Wilson, 1980) suggested that "the reverse transfer function of the middle ear (i.e., from a sound source within the cochlea to the resulting occluded ear-canal sound pressure) is maximum between 1 and 2 kHz and, therefore, acoustic emissions will be strongest and observed most often in this frequency range".

Although present in 34% of seemingly normal human ears, (Zurek 1981), SOAEs are infrequent in animals (Decker, and Fritsch, (1982); Evans, et al (1981); Ruggero, et al(1983); Ruggero, et al (1984)) and were not found at all in a sample of 21 ears of 17 chinchillas raised in a sound- shielded animal colony (Zurek & Clark (1981)). A recent survey of Old-and New-World monkeys showed that spontaneous otoacoustic emissions were detected in approximately 5 % of the monkeys (Martin, et al (1985)). While in humans the levels of SOAEs do not exceed 20 dBSPL, in non-humans,

SOAEs of greater intensity have been observed: a) Up to 40 dB SPL (near 4.7 KHz) in a noise-exposed chinchilla, (Zurek and Clark (1981)), b)About 50 dB SPL (near 9.0 KHz) in a dog (Rugero, et al (1982)), c) About 50 dB SPL (near 5.0 KHz) in another dog, (Rabinowitz and Lage (1983)).

Investigation of frequency and amplitude drift of SOAEs revealed that fluctuations of up to 10 Hz and 5 dB were common over a 60-minute time period. In addition, more amplitude drift was found for weak SOAEs than for SOAEs of greater amplitude. The frequency drift of SOAEs was not obviously related to the amplitude (Frick & Matthies (1988)). These results were consistent with those of Rugero, et al (1983) who reported frequency drifts of 10 Hz over a 30-minute time period. Similar fluctuation in SOAEs was noted in other studies (ANSI. 1-1977; Hammel, (1983); Wier, et al (1984)).

It has been shown that an SOAE can be suppressed by an external tone (Rabinowitz (1981); Wilson, J.P. (1980); Zurek (1981); Zurek and Clark (1981)). It has also been noted that the shape of an isosuppression contour resembles physiological and psychological tuning curves, (Clark et al (1984)); they summarized the effect of the external tone on the emission as follows:

1. Level effect

As the level of the external tone was raised, the SOAE was first reduced slightly (1-4dB) with increase in external tone level of 5-10 dB and then reduced abruptly to the noise floor of the system. The

slope of this abrupt suppression was about -6.5 (i.e., 6.5 dB reduction in SOAE level for a 1 dB increase in the level of the external tone). The slope of the suppression contour did not vary significantly with frequency of external tone. Contrary to observations on spontaneous otoacoustic emissions in humans (Kim, 1984) which showed "significant reductions of slope for external tones above the SOAE frequency, there does not seem to be any significant reduction in the slope of suppression with external tones of frequencies higher than the SOAE for the chinchilla".

2.Frequency effect

The frequency of the SOAE would often shift slightly. The change in SOAE frequency observed for 5 dB of the suppression as a function of frequency of external stimuli, the shift in SOAE frequency varied from +1 Hz to -8 Hz. A recent study plotted the suppression contours and illustrated the suppressive effects of external stimuli on SOAE amplitude. Completed suppression contours closely resembled the well documented psychophysical tuning curves of the ear, Frick & Matthies (1988). Norton, et al (1989) illustrated that "spontaneous otoacoustic emissions were reduced in frequency and/ or amplitude following presentation of brief, intense pure tones. The effects were highly tuned with exposures between 1/8 and 5/8 of an octave below the SOAE, thus producing the maximum changes. Exposure frequencies above the SOAE had no effect. The degree of tuning observed depending upon the post-exposure time sampled, with sharpness maximal between 3 and 120 s post exposure. The recovery functions were bi-phasic, the first phase being rapid and non-

monotonic over about 2 minutes, while the second phase was monotonic and slow, sometimes taking several hours".

Recent experiments have shown that electrical stimulation of the crossed olivocochlear bundle (COCB) alters the amplitudes of distortion-product emissions in the guinea pig (Mountain, 1980) and chinchilla (Siegel and Kim, 1982) and stimulus-frequency emissions in the cat (Guinan,1986). Anatomical and physiological evidence obtained in animals suggests that "part of the olivocochlear bundle may serve as a link between the two cochleas" (Fex,1962,1965), such that "acoustic stimulation of one cochlea may alter the sound-induced activity in the opposite cochlea. For example, auditory nerve fiber discharge rates and temporary threshold shifts of the whole nerve compound action potential are reduced in nonhuman animals by acoustic stimulation" (Mott et al,1989). Several investigators have attempted to demonstrate that acoustic stimulation of the contralateral ear induces changes in SOAEs in human subjects (Grose, (1983); Rabinowitz and Widden (1984)). Other studies reported that "without exception, the effect of the contralateral stimulus, whether noise or tone, was to decrease the amplitude and increase the frequency of the SOAEs", Mott et al (1989). Norton, et al (1989) illustrated that "frequency and amplitude of the SOAEs can be altered by acoustic stimulation of the contralateral ear".

The mechanical and acoustical influences on spontaneous otoacoustic emissions were discussed by Schloth and Zwicker (1983). They concluded that "changes of the impedance of the middle ear,

evoked by the acoustic reflex, lead to a significant increase in the frequency of the spontaneous emission as well as a significant decrease in its level. Air pressure introduced in the closed outer ear canal changes the stiffness of the eardrum. Consequently the impedance of the middle ear and its acoustical transmission factor change; this affects the level of the spontaneous emission".

In a study of spontaneous emissions, Fritze (1983) was not able to detect SOAEs if the hearing loss was equal to or greater than 30 dB, although he observed more SOAEs in "slightly damaged cochleas" than were routinely noted in normal ears. In an earlier study, Zurek (1981), observed four subjects with marked hearing impairment, none of whom showed SAOEs. An additional subject with a severe high frequency hearing loss, displayed an SOAE at a mid frequency where the threshold was normal. Probst, et al (1987) reported that "ears with a noise-induced hearing loss showed a significant reduction in the incidence of both spontaneous emissions and spectral peaks in evoked emissions that was not evident in ears with similar patterns of hearing loss caused by other factors. It is important to emphasize that although transient evoked and spontaneous emissions appear to have limited applicability as objective measures of hearing dysfunction, other classes of evoked emissions (e.g. distortion products) that make use of constant pure tone may be more suitable for predicting hearing losses at specific audiometric frequencies".

In summary, SOAE is an intersting phenomenon. Some studies stated that it is produced by healthy normal hearing ears, while others indicated that there must be a minute pathological lesion in the cochlea to cause the emmission. SOAEs are more common in monkeys than chinchillas. They are suppressed by external tones, and there is still a controversy about the role of the efferent nerve on the SOAE.

Further investigation of this phenomenon is the goal of the present study, and the main objectives are:

1. To determine the characteristics of the spontaneous otoacoustic emissions in chinchillas and Squirrel Monkeys.

2. To screen chinchillas that are born and raised in a low-noise colony, for the presence of the SOAEs.

3. To study the effect of ipsilateral and contralateral suppressor tones, and the interaural attenuation on SOAEs.

4. To determine the incidence and characteristics of SOAEs in Squirrel Monkeys.

5. To study the effect of different types of noise (continuous and interrupted) in developing SOAEs in chinchillas.

6. To study the role of Central Nervous System (efferent nerve and cerebral cortex) on SOAE production.

II. EXPERIMENT ONE

SPONTANEOUS OTOACOUSTIC EMISSIONS IN CHINCHILLAS

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 described as narrow-band or tonal (occuring 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. (Zurek, 1981; Fritze, 1983; Clark, et al, 1984; Weir, et al 1984; Strickland, et al 1984; Probst, et al, 1986, Norton et al, 1989).

While studies have shown that SOAEs are common in humans, the same is not true for all animal species (Zurek and Clark, 1981, Zurek, 1981). In their work with chinchillas, Zurek and Clark (1981) found two SOAEs in animals only after exposure to high-intensity noise. They concluded that "narrow-band otoacoustic emission can apparently be induced by traumatic noise exposure. SOAEs may be associated with microlesions in the organ of Corti rather than resulting from normal cochlear function. Accordingly, the high incidence of otoacoustic emissions in humans and the zero incidence in the sample of unexposed chichillas may reflect the vastly different histories of noise exposure in humans and these chinchillas". Clark, et al (1984), concluded that "a punctate loss of the organ of Corti (OC) may facilitate the occurrence of an SOAE". They further hypothesized that the following conditions are necessary and sufficient for an SOAE to occur: (1) functional disruption of a biomechanical control mechanism normally present in a region of the OC. (2) presence of

functionaly-active OC (especially outer hair cells) adjacent to the region.

Cianfrone and Mattia (1986) reported that "the physiological meaning and nature of SOAEs remain the most puzzling points". Although little doubt remains about their origin, whether SOAEs are a reflection of some normal auditory mechanism (Guinan, 1986) or an expression of some preclinical biomechanical neuromechanical or sensorineural disease is still an open question. In the former case, it is expected that further improvement in recording techniques and data processing will reveal an increase in the rate of occurrence of SOAEs in normal subjects. If the latter assumption is correct, the concept of normal hearing should be revised. Measuring of SOAEs could be looked at as to a possible and important clinical test for early diagnosis or hearing damage prediction".

The present study screens chinchillas, that have a hearing system similar to that of humans, for the presence of SOAEs. The objectives of this experiment is to study the incidence and characteristics of SOAEs among chinchillas that were raised in a low-noise colony, and compare that to SOAEs among chinchillas that were later exposed to two different kinds of noise (continuous or interrupted).

EXPERIMENT ONE

MATERIAL AND METHODS

METHODS

Subjects

Our subjects for these experiments were 131 chinchillas, 262 ears. Figure (1) depicts the distribution of ages and sex of animals that had been screened for the presence of SOAEs. These animals were born and raised in a sound-shielded laboratory colony (Clark & Bohne, 1978). This laboratory colony was adjusted to suit the style of living of these animals (cold weather and dim light as in mountain caves), and was regularly checked by a veteranian at Washington University School of Medicine's Animal Studies Committee .

Measurements

Equipment

The probe tube microphone system used to detect acoustic signals in the ear canal has been described previously (Zurek & Clark,1981). The system consists of an ear piece from an otadmittance meter (Grason-Stradler 1720) with a miniature microphone (Knowles Electronics, E A 1842) coupled by plastic tubing to the end of one of three tubes. The probe tube microphone was calibrated with a standard laboratory microphone (Bruel and Kjaer, 4131). The microphone was connected to a preamplifier and then to a (Bruel & Kjaer, type 2033) high resolution signal analyzer. SOAEs were identified by analyzing the microphone output of the probe through the wave analyzer and displaying it on an x-y recorder

(Bruel and Kjaer, type 2308). The other two tubes were connected to receivers that were driven by Wavetek oscillators (model 148) and attenuators (Hewlett & Packard). The wave analyzer was set outside the sound proof booth while the probe assembly was set inside the booth. The microphone had been calibrated and the results were plotted on a graph-showing the reference level in dBV, for each frequency (fig 2). SOAE had been calibrated on the wave analyzer in relation to the reference level (-20dBV). Subtracting the level of SOAE in dBV from the reference level in the graph (shown in fig 2), at the proper frequency gave us an estimation of the level of SOAE in dB SPL.

Procedure

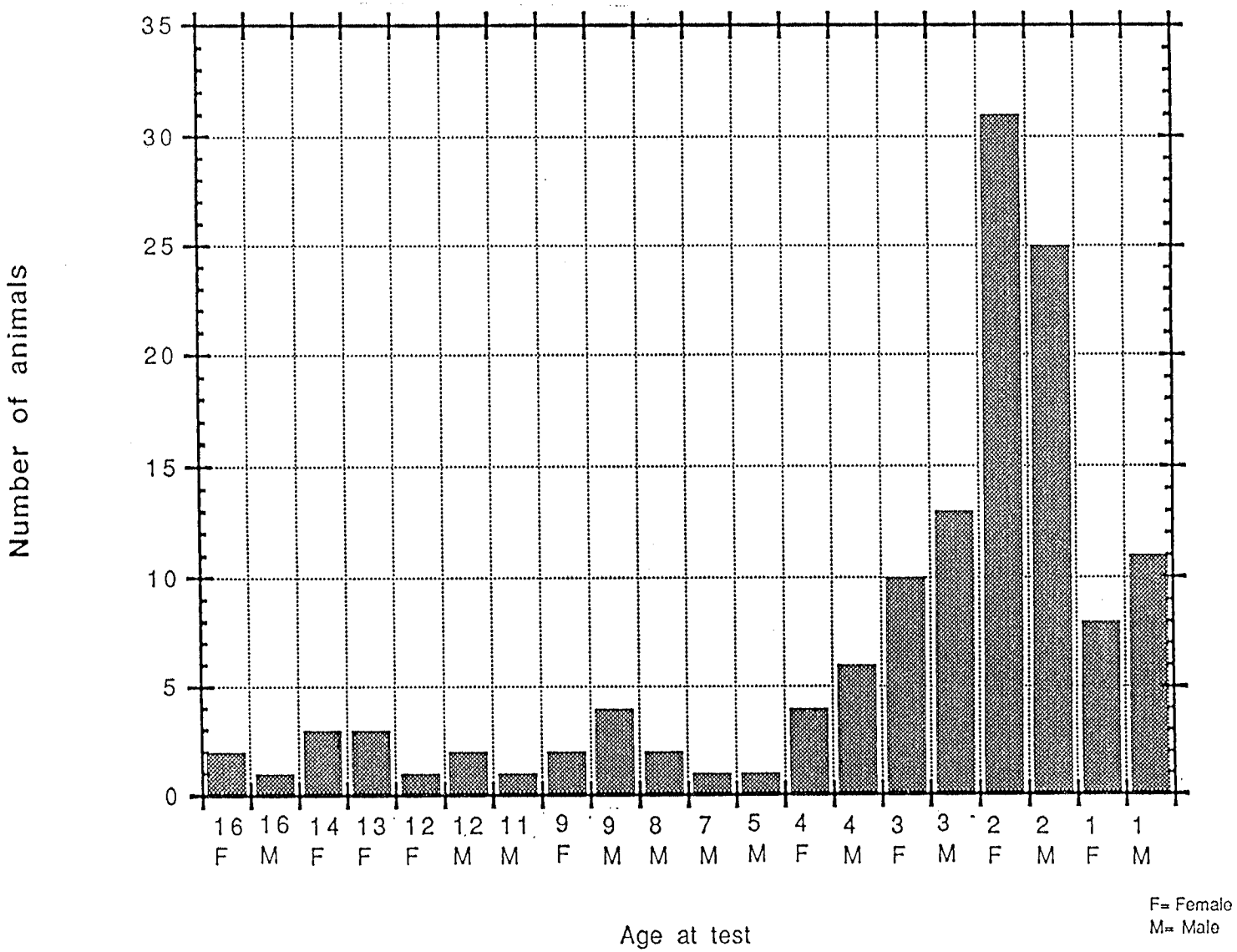
One hundred and thirty one chinchilas were screened for the presence of SOAEs. A special holder was designed to keep the animals steady, because we preferred not to use anaethesia. The animals were examnined in a sound-protected booth lined with foam on all inner surfaces to prevent any external noise from interfering with the experiments. Because the chinchilla is easily adapted for mild restraint, it was no problem to examine the animal in the holder for short periods of time. Screening for emission presence took about two minutes for each animal.

A general examination was given (overall health, weight of the animal, presence of infection, diarrhea....etc.) and the ear piece was placed in a clean container with alcohol after each use. Then the

probe was carefully inserted into the ear canal of the chinchilla, and the body of the probe was taped to the pinna to help holding the probe in its proper place.

After screening all 131 chinchillas, 2 that had been exposed to noise before were exposed to another set of continuous noise (centered at 0.5 KHz at 95 dB SPL) for nine days. Four other chinchillas that had not been exposed to any noise, were exposed to the same kind of noise for nine days. Four chinchillas that had not been exposed to noise were exposed to an interrupted band of noise, centered at 4 kHz at 86 dB SPL for 15 minutes per hour, for 72 days. After the ossicular removal of the left ear, the chinchillas were exposed to an additional 72 days of noise. These 10 chinchillas had been examined for the presence of SOAE during and after exposure to noise. In conclusion, there were 121 chinchilas that were not exposed to noise, and 10 chinchillas that were exposed to noise.

Fig.1:Distribution of ages and sex of chinchillas screened for the presence of SOAEs



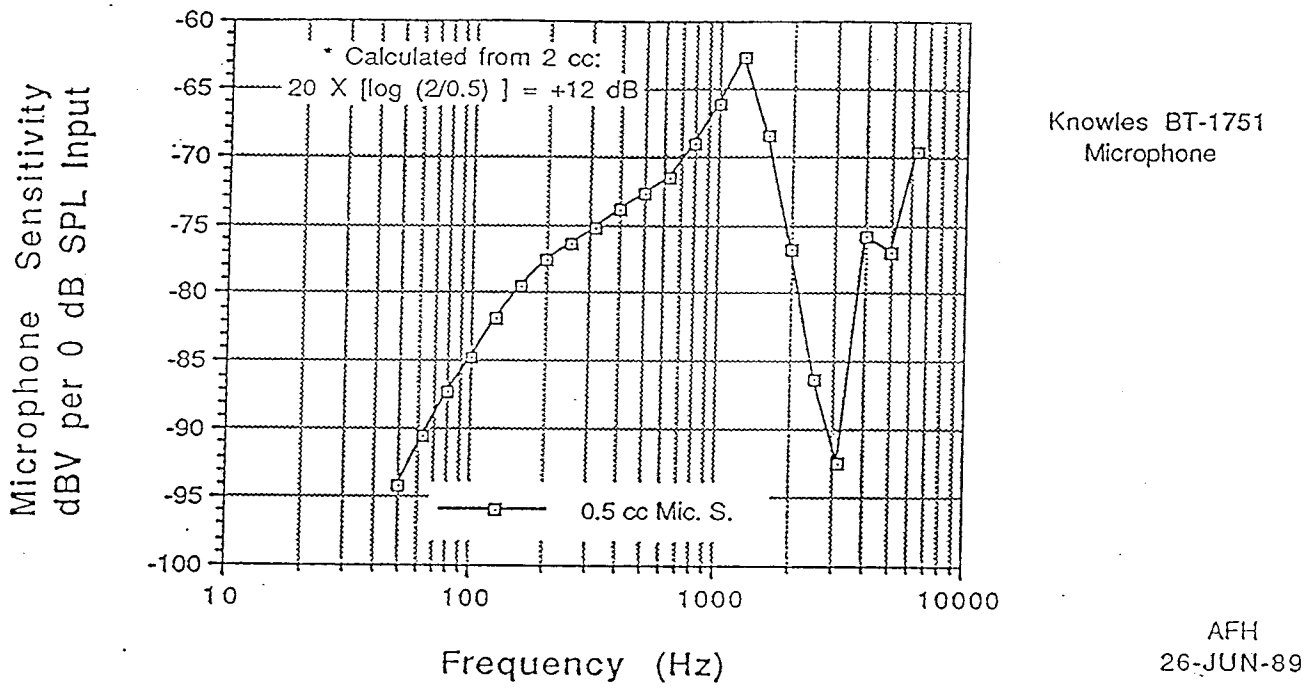


Fig. 2: Microphone sensitivity in 0.5 cc coupler

EXPERIMENT ONE

RESULTS

RESULTS

It was important to notice that no SOAE could be detected from the chinchillas that were not exposed to noise and were raised in a sound-treated colony. SOAEs could be detected in three out of 20 ears, and no emission could be detected from 242 screened ears. Animal no. 184, who was exposed to continuous noise, showed a tonal signal emanating from the right ear around 2262 Hz at approximately 50 dB SPL (fig.3). This animal had SOAE for more than three years. Animal no. 187, who was exposed to interrupted noise, showed an emission from the right ear at 2562 Hz with an amplitude of 27 dB SPL (fig. 4). Two emissions could be detected from the right ear in chinchilla no. 487, the first one at 2500 Hz, 24 dB SPL, the second at 2550 Hz, and 24 dB SPL (fig. 5) .

Table 1. Summary of the SOAE detected from chinchillas before and after exposure to noise.

Animal	before noise	oss. removal	noise	after noise
1. 184	no SOAE	lt ear	continuous	SOAE(2262Hz)
2. 187	no SOAE	lt ear	interrupted	SOAE(2562Hz) (2500Hz)
3. 487	no SOAE	lt ear	interrupted	SOAE (2550Hz)
4. 2087	no SOAE	lt ear	interrupted	No SOAE

Table 1 Continue

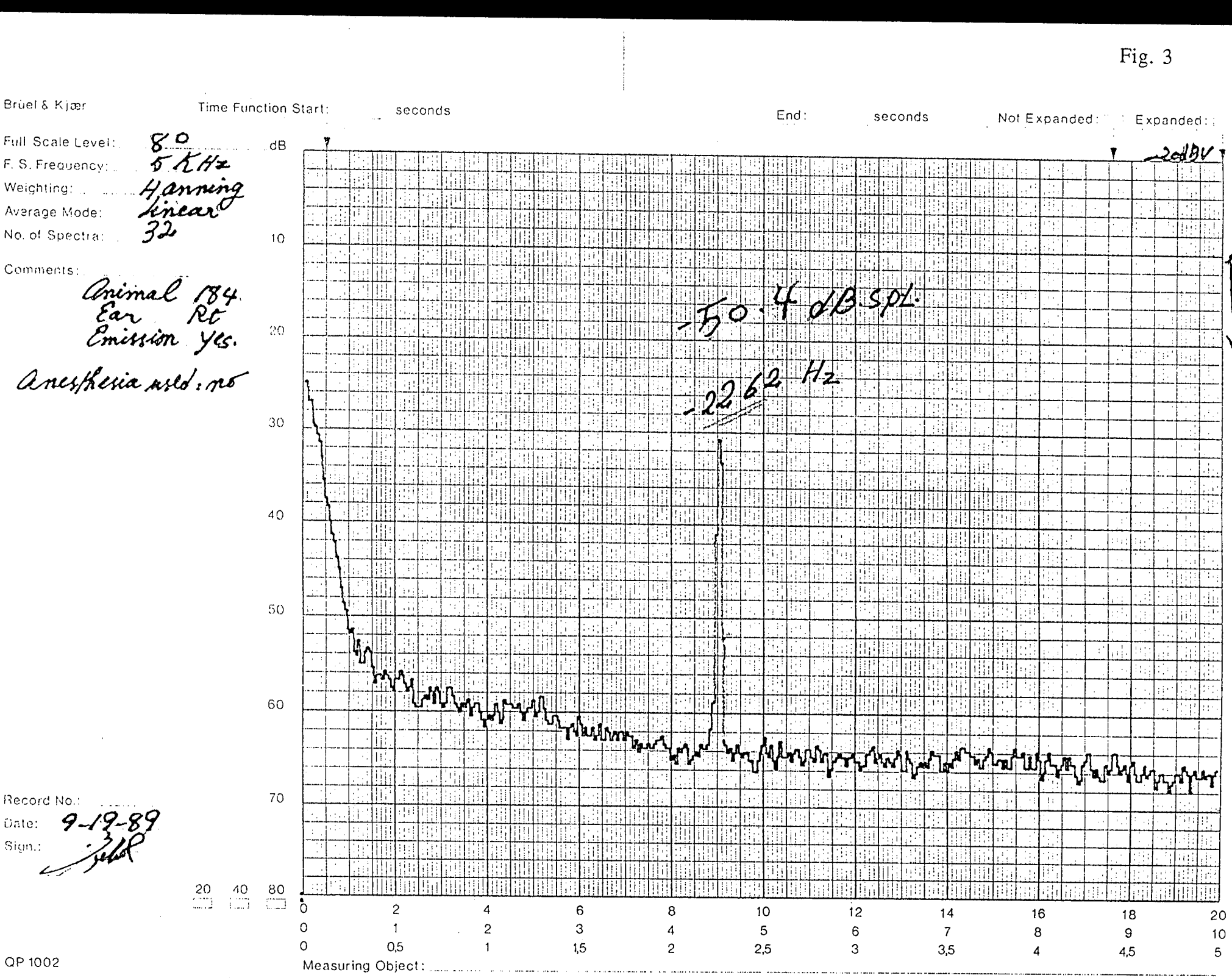
Animal	before noise	ossi. removal.	noise	after nosie
5. 887	no SOAE	no	continuous	No SOAE
6. 1783	no SOAE	no	continuous	No SOAE
7. 2386	no SOAE	no	continuous	No SOAE
8. 1488	no SOAE	no	continuous	No SOAE
9. 4188	no SOAE	no	continuous	No SOAE
10. 5182	no SOAE	no	continuous	No SOAE

Characteristics of emissions:

All recorded emissions from the three animals were narrow-band signals. In 1987, animal no. 184 showed an SOAE 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. In December 1988, the emission was detected at 2137 Hz , at a level of 37.5 dB SPL in the morning; nine hours later, it shifted to 2183 Hz, at a level of 32 dB SPL, and on the following morning the emission returned to approximately 2137 Hz and 37 dB SPL. Emission measurements were repeated for several days. It was found that the average amplitude and frequency shift in the emission on the same day did not exceed 5 dB SPL and 50 Hz respectively. In February 1989, the frequency of the emission was 2287 Hz, at a level of 30 dB SPL; in September 1989 the emission had a frequency of 2262 Hz with amplitude of 50.4 dB SPL. The emission disappeared on 3d March1990. In May 1990, the emission was at 2312 Hz at 30 dB SPL. On May,15,1990, no SOAEs could be detected from animal 184. However, three days later the animal started to have emission again, the emission lasted for one week before disappearing. This happened twice, then the emission disappeared till the present time. Figure 6 summarizes the changes in frequency and level of the SOAEs over the time.

It is interesting to notice that the emanating SOAEs from both animals 187 and 487, were in the same range of frequencies at which hearing loss was detected (figs.7 & 8).

Fig. 3


Brüel & Kjær
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Not Expanded:
Expanded:
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Weighting: Hanning
Average Mode: linear
No. of Spectra: 32
Comments:
Animal 184
Ear RC
Emission yes.
Anesthesia used: no
dB
20dBV
-50.4 dB SPL
-2262 Hz
10
20
30
40
50
60
70
Record No.:
Date: 9-19-89
Sign.:
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QP 1002
Measuring Object:

Fig. 4

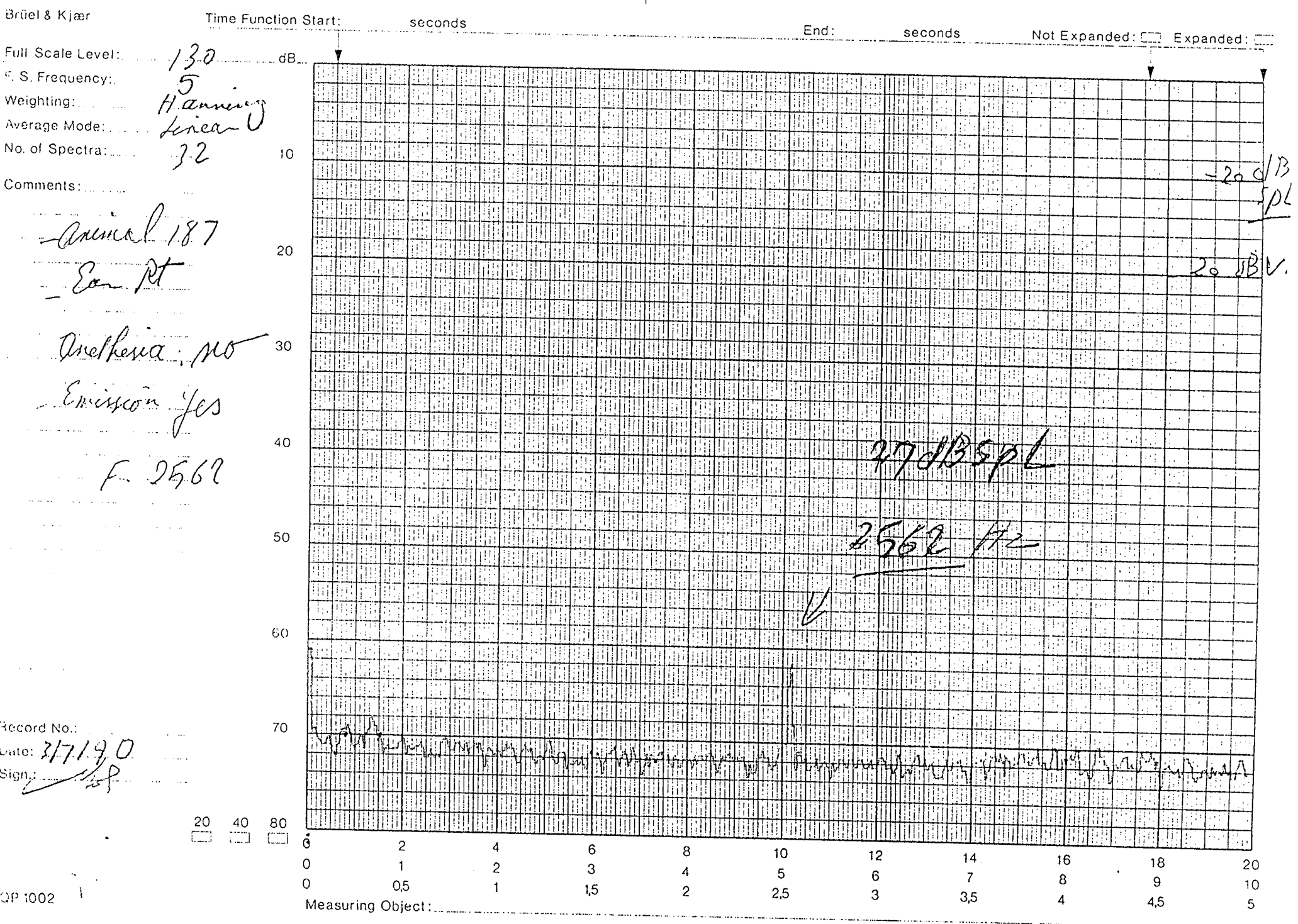

Brüel & Kjær
Time Function Start: seconds
End: seconds
Not Expanded:
Expanded:
Full Scale Level: 130 dB
F. S. Frequency: 5
Weighting: Hanning
Average Mode: Linear
No. of Spectra: 32
Comments:
Animal 187
Ear Rt
Anesthesia: no
Emission: yes
F. 2562
27 dB SPL
2562 Hz
V
20 dB SPL
20 dB V
10
20
30
40
50
60
70
Record No.:
Date: 3/7/90
Sign:
20 40 80
0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0,5 1 1,5 2 2,5 3 3,5 4 4,5 5
Measuring Object:
QP 1002

Fig. 5

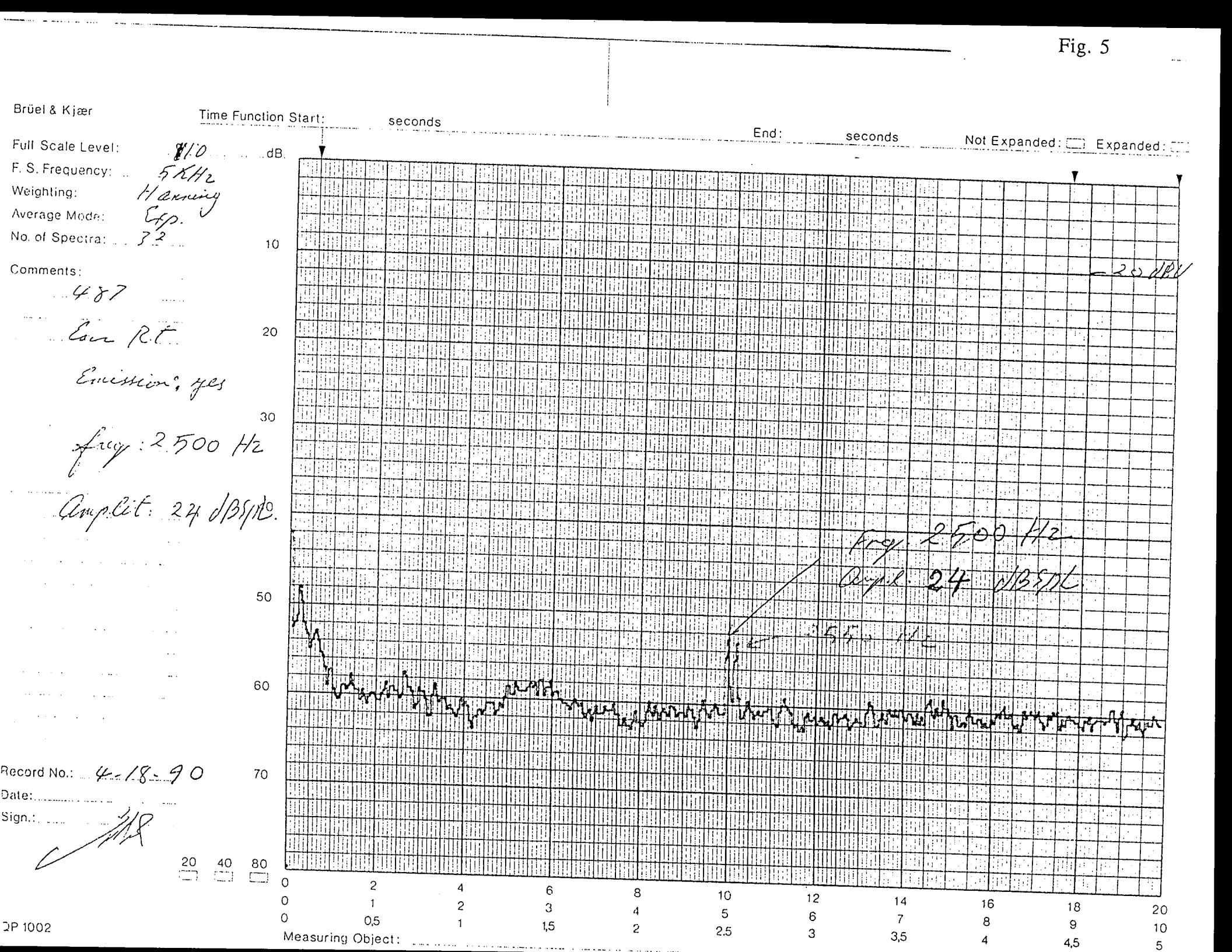

Brüel & Kjær
Time Function Start: seconds
End: seconds
Not Expanded:
Expanded:
Full Scale Level: 110 dB
F. S. Frequency: 5 kHz
Weighting: Hanning
Average Mode: Exp.
No. of Spectra: 32
Comments:
487
Ear R.E.
Emission: yes
freq: 2500 Hz
Amplit: 24 dBSPL
freq. 2500 Hz
ampl. 24 dBSPL
10
20
30
50
60
70
Record No.: 4-18-90
Date:
Sign.:
20 40 80
0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0,5 1 1,5 2 2,5 3 3,5 4 4,5 5
Measuring Object:
QP 1002

Fig. 6: Summerises the changes in frequency
and amplitude of the SOAE of animal 184 over time

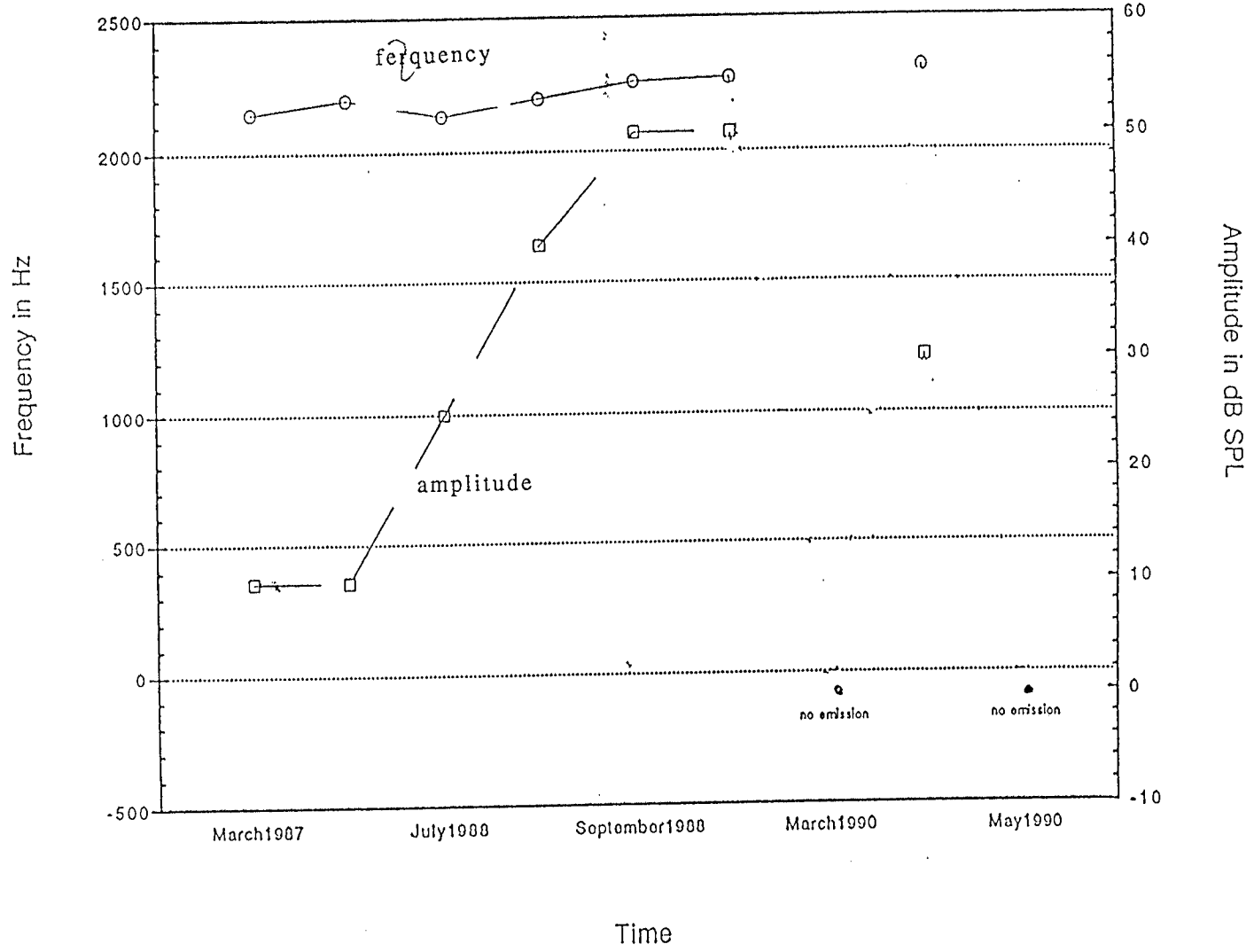


Fig. 7: Hearing thresholds of animal 187 before and after exposure to interrupted noise, frequency of SOAE ~ 2562 Hz

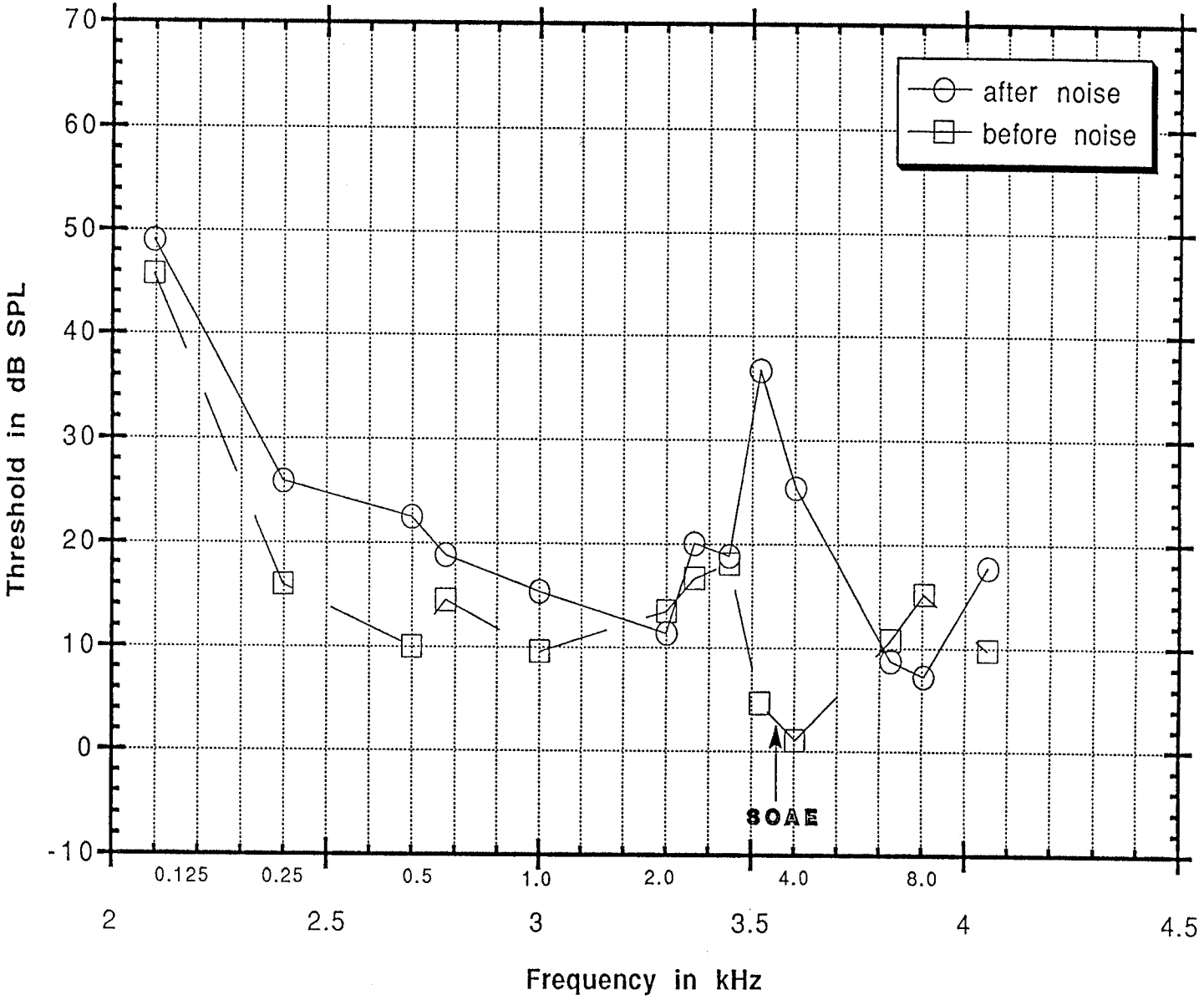
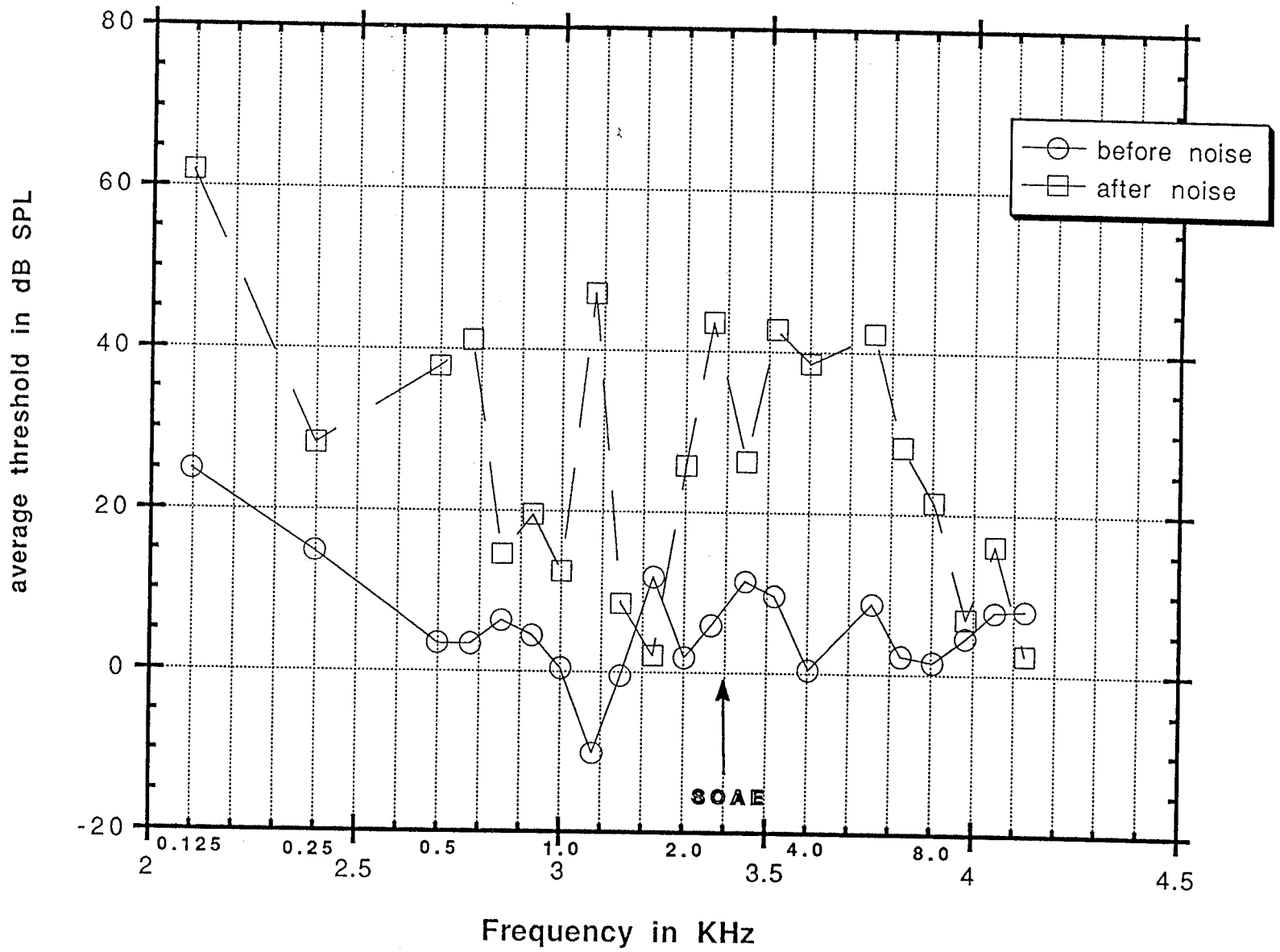


Fig. 8: Hearing thresholds of animal 487 before and after exposure to interrupted noise, frequency of SOAE~ 2.5 kHz



EXPERIMENT ONE

DISCUSSION

DISCUSSION

SOAEs, although present in 34% of seemingly normal human ears (Zureck, 1981), are infrequent in animals (Decker and Fritsch, 1982; Evans et al, 1981, Ruggero, 1984; Zurek and Clark, 1981). In the present study these emissions were detected in 3 out of 262 ears of the 131 chinchillas raised in a sound-shielded animal colony.

SOAEs are strong narrow-band signals emanating from the external canal without any external stimulation. These sinusoid-like signals are tonal, robust, and sometimes audible without the aid of amplification by positioning the listener's ear close to the pinna of the emitting chinchillas (Kemp 1979; Zurek & Clark 1981; Clark, et al 1984; Fritz & Kohler 1986; Mott, et al 1989; Bonfils et al 1990; Martin, Probst & Martin 1990). Figs 3,4,&5 depict the characteristics of the SOAEs seen in the present study. These characteristics correspond to description from previous reports in the form of the sinusoid-like signal, its audibility without amplification, and its stability over time (animal 184 produced SOAEs for 3 years, 1987-1990).

The frequency and amplitude drifts of SOAEs were discussed in several studies. Frick & Matthies (1988) reported that "for four of five SOAEs tested in three of four subjects, the standard deviation for frequency was less than or equal to 1.25 Hz. These findings were consistent with Rugerro, et al (1983), who reported "frequency drifts

of 10 Hz over a 30 minute time period. The amplitude drift of spontaneous emissions also appeared to be small". "The standard deviation for amplitude was less than 1 dB SPL for four of the five emissions observed" (Frick & Matthies1988). "A considerable amount of experimentation over the past 10 years has shown that SOAEs are stationary signals that can be recorded with relatively minor frequency changes either within a test session" (Mot, Norton, Neely & Warr 1989) or "between experiments that are separated by months or years" (Zurek 1981). Martin, Probst and Martin (1990) showed that "values for frequency fluctuations obtained with repeated measurements over time periods of several days or months are usually less than 1%". In the present study frequency and amplitude drifts of SOAEs never exceeded 10 Hz and 5 dB SPL respectively in a single day. The change of the frequency and amplitude of SOAEs in chinchillas was also studied over time as shown in Fig. 6. The value for frequency fluctuations obtained for three years was 7% and for a single day was 0.5%. The difference between the fluctuations in a single day and in three years is not random. Fluctuations seem to be affected by many factors (e.g. the external tone, multiple use of anesthesia and room temperature).

Zurek and Clark (1981) concluded that "the narrow-band otoacoustic emissions can be induced by traumatic noise exposure". Ruggero, Rich and Freyman in another hypothesis postulated that "there is a positive feedback of the hair cells upon the basilar membrane that results in amplification of the input mechanical signal at each basilar membrane site. In addition, each cochlear site

exerts a negative feedback upon itself and upon adjacent cochlear segments. In an intact region of the cochlea the positive and negative feedbacks largely cancel each other, but the net effect at each site is positive and maximal when the input frequency corresponds to its resonant frequency; since the net feedback is positive, and distortion-product acoustic emissions are produced upon continuous stimulation. If an area of outer hair cell loss exists adjacent to an area of relative normality, the removal of damping produced by the former will permit the latter to induce its segment of basilar membrane to vibrate with larger amplitudes than normal, at its own resonant frequency; impulsively evoked OAEs will appear upon stimulation with clicks or short tone pips. If the spatial transition between normality and abnormality is particularly sharp, basilar membrane oscillations will be generated in the absence of an external stimulus and SOAEs will appear in the external ear canal" (Ruggero, Rich and Freyman, 1983).

Histopathological evaluation of the cochlea of emitting ears revealed discrete basal turn lesions near the positions corresponding to the frequencies of the emissions, and in the emitting ear there was an abrupt change in the height of the tissue between the outer hair cells and the squamous epithelium. In the nonemitting ear, the transition between the outer hair cells and the organ of Corti surrounding the outer hair cell loss was much more gradual (Clark, et al1984). In summary the previous studies supported the idea that SOAEs are produced due to pathological lesions. In the present experiment, the three emitting animals were exposed to noise

leading to pathological trauma, one to continuous noise, and two to interrupted noise. It is interesting to notice that the emanating SOAEs detected in the three chinchillas were in the same range of frequencies at which hearing loss was detected in these animals. This observation supports the idea that SOAEs are almost certainly induced by acoustic trauma.

III. Experiment Two

IPSILATERAL SUPPRESSOR TONES

INTRODUCTION

Otoacoustic emissions (OAEs) are low-level sounds produced by vibratory motion in the cochlea. They may be detected in the external auditory canal as a result of external stimuli (evoked emissions) or as spontaneous oscillations within the inner ear (spontaneous otoacoustic emissions--(SOAEs) (Kemp, 1978; Zurek, 1981). "Although their exact origin is unknown, SOAEs appear to be linked closely to active, physiological, vulnerable, biomechanical elements in the cochlear partition. Several models of cochlear mechanics attribute SOAEs to spontaneous oscillation of the cochlear partition, involving the outer hair cell (OHC) subsystem" (Davis, 1983; Neely, 1983; Kim, 1986; Neely and Kim, 1983, 1986). These models assume that OHCs are mechanical force generators, bidirectionally coupled to the cochlear partition, and capable of both mechanical to electrical and electrical to mechanical transduction. Davis (1983); and Neely 1983; Neely and Kim (1983, 1986) and others propose that "OHCs amplify basilar membrane motion in a frequency and place-specific manner, which provides great sensitivity and sharp tuning within the cochlea. Such systems are inherently unstable. While under normal circumstances, both forward and reverse transduction occur only in response to external stimulation, small alterations in such a system can and does lead to spontaneous oscillations" (Norton et al, 1988).

External tones introduced into the ear canal have been observed to suppress the level of spontaneous emissions and also cause some changes in the frequency of SOAEs. Clark, et al (1984) reported that "the shape of an isosuppression contour resembles physiological tuning curves; as the level of the external tone was raised, the SOAE was first reduced slightly (1-4 dB) with increases in external-tone level of 5-10 dB and then was reduced very abruptly to the noise floor of the system". Clark also reported that "although the effect was not straight-forward, it was clear that most of the tested external tone frequencies reduced the SOAE frequency or had no effect, with maximum reduction in the 1 and 8 KHz". Other studies reported that SOAE suppression tuning curve (STC) tips were slightly higher in frequency than the SOAE (Rabinowitz & Widin, 1981). Suppressor signals close to the SOAE exhibit an abrupt increase in suppression. Schloth and Zwicker reported that "the low-frequency slopes of the SOAE suppression tuning curve functions range from 66 to 111 dB/octave, whereas the high frequency slopes range from 168 to 333 dB/octave. As the suppression frequency is shifted below the SOAE, the suppression effect becomes less pronounced. Low-frequency SOAE STC tails have a slope ranging between 0 dB/octave (Zurek) and 15 dB/octave" (Schloth & Zwicker, 1983).

Because suppression is a cochlear phenomenon (Sachs & Kiang, 1968), demonstration of SOAE suppression supports the theory that SOAE originate within the cochlea (Rabinowitz & Widin, 1984; Ruggero, Rich, & Freyman, 1983; Schloth & Zwicker, 1983; Zurek, 1981).

The present work was motivated by our interest in both the nature of SOAEs and their interaction with external single tone stimuli; the suppression of the SOAEs by the external tone ipsilaterally has been investigated.

EXPERIMENT TWO

MATERIAL AND METHODS

METHODS

Subject

The subject for this experiment was chinchilla no.184, who was born and raised in a sound-shielded laboratory colony (Clark & Bohne,1978). The animal was rendered functionally monaural by removal of the malleus and incus from its left ear (Clark & Bohne, 1978). Prior to noise exposure, no otoacoustic emission was detected in the right ear. The chinchilla was subsequently exposed to two 9-day durations of 0.5 KHz octave band noise at 95 dB SPL in December of 1984. After approximately one month following the second noise exposure, an SOAE was detected at 2150 Hz at a level approximately of 10 dB SPL. In a quiet environment, a normal-hearing person could hear this sound by listening at the animal's ear canal. Until April, 1990 the animal was still continuously emitting.

Measurements

Equipment

As was described in experiment 1, the miniature microphone was coupled by plastic tubing to the end of one of three tubes. Pure-tone stimuli were generated by a Wavetek generator (model148) connected to one of the other two tubes in the probe assembly.

Procedures

An attenuator was used to control the level of the stimulus produced by the calibrated receiver (see fig 9). During the 15-20 min. recording session, the chinchilla was immobilized with a light dose of Ketamine hydrochloride (15 mg/kg, I.M.). The external tone was introduced directly to the chinchilla's ear through the receiver and the resulting (SOAEs) were analyzed and recorded by the wave analyzer through the microphone in the probe assembly. The external tone was applied at different frequencies and the level of the suppressor tone in each frequency was increased until the SOAE disappeared.The suppression contour was plotted graphically. The animal recovered from the anesthesia without any complications.

The frequency and amplitude shift of the emission of chinchilla 184 due to external tone application was measured.

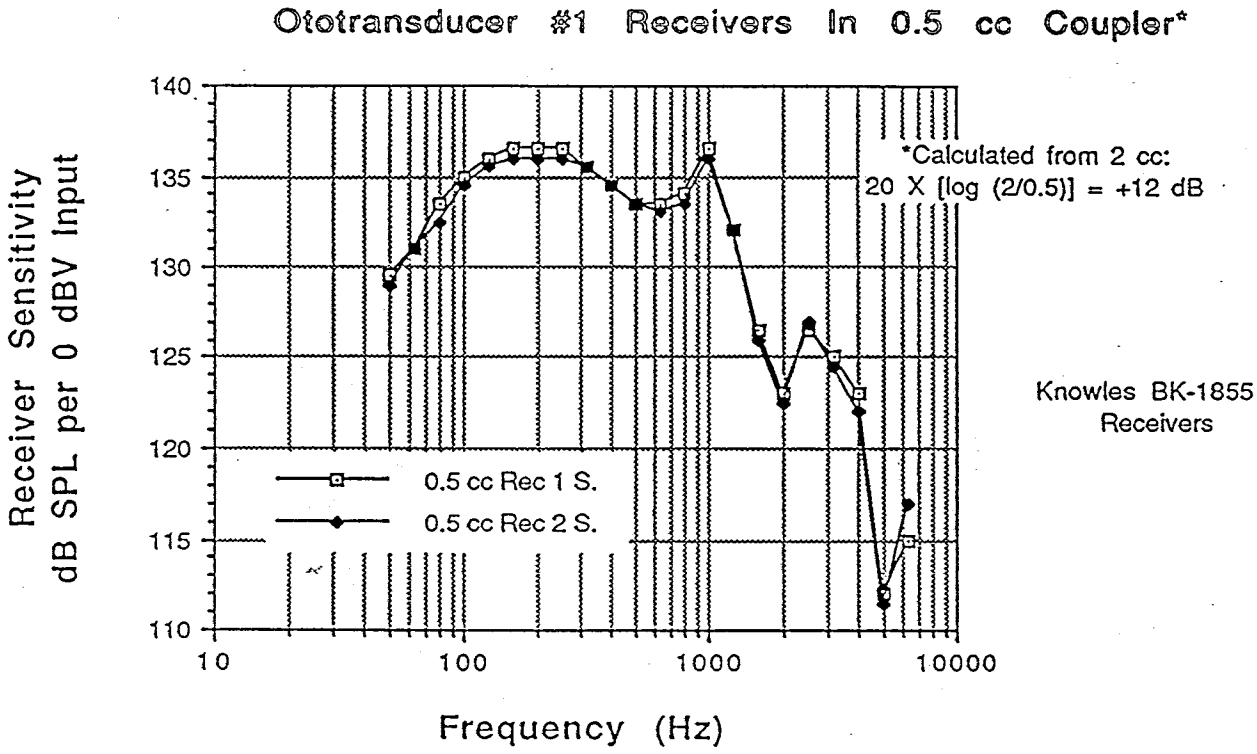


Fig. 9: Receiver sensitivity in 0.5 cc coupler

EXPERIMENT TWO

RESULTS

Results

It has been shown that an SOAE can be suppressed by an external tone, and that the shape of an isosuppression contour resembles physiological and psychological tunning curves (Clark, et al 1984; Frick and Matthies, 1988).

Frequency and Amplitude of the External Tone

Figure (10) shows the suppression of SOAE at 2262 Hz by application of a 2 KHz external tone. It is clear from this graph that as the level of external tone was raised, the SOAE (frequency 2262 Hz) was first reduced slightly--1 dB with an increase of 10 dB in external tone level (L et). Then it was reduced abruptly--more than 28 dB SPL, with an increase of Let 10 dB SPL. This sudden change in the slope of the suppression tone curve occurred at L et of 57 dB SPL. When the frequency of the external tone was 1800 Hz (fig. 11) the sudden slope was at Let of 69 dB SPL (about 28 dB SPL suppression at an increase of L et 10 dB SPL). Figures (12, 13, and 14) depict the effect of the external tone on the SOAE. As we noticed when the amplitude of the external tone was high (fig 13 & 14) the suppression was more than when the intensity of the external tone was less (fig 12).

The amplitude of the SOAE was plotted as a function of the external tone frequency (Fig.15). We noticed that when the frequency of the external tone was near to that of the SOAE, a higher

Let was needed to suppress SOAE than when the frequency of the external tone was far from that of the SOAE. In fig. 15 the frequency of SOAE was 2230 Hz, with F et 2500 Hz; the sudden change in the slope of the suppression curve was at 79 dB Let. With F et 1700 Hz; the change in the slope was at 87 dB L et, and at F et 1500 KHz the sudden change in the slope was at 93 dB Let.

Figure16 confirmed that when the frequency of the external tone was near that of the SOAE, we needed a less intense external tone to suppress SOAE than when the frequency of the external tone was far from that of the SOAE. This figure illustrates that the frequency of SOAE was 2135 Hz, and about 50 dB SPL external tone was needed to suppress the emission when the external tones were with frequencies 1.4, 1.8, 2, 2.35 and 2.8 KHz, while 75 dB SPL was needed to suppress the emission in case of external tone with frequencies 1 KHz and 4 KHz and there was very minimal effect at the frequencies of 5.7 and 8 KHz on SOAE.

Therefore, we noticed that the sudden change in the slope of the suppression contour is affected by the frequency and the amplitude of the external tone.

Amplitude shift of SOAE by the external tone

The effect of the external tone on the amplitude of the SOAE was studied. In figure 17, the amplitude shift of the SOAE in dB SPL was plotted as a function of the frequency of the external tone . The frequency of the SOAE was 2225 Hz. It was clear that the amplitude shift was higher at external tone frequencies above those of SOAE than at external tones with frequencies less than those of the SOAE. The shift in amplitude was 13 dB SPL when the F et was 2400, about 4.5 dB at F et of 2300 Hz, 2200 Hz and 2100 Hz, 3 dB SPL at F et of 2000 Hz and 1900 Hz, and around 1 dB SPL shift in the amplitude of the SOAE at F et of1800 Hz and 1700 Hz.

Frequency shift of the SOAE by the external tone

The frequency shift of the SOAE by the external tone was also studied. The frequency shift of the SOAE in Hz was plotted as a function of the frequencies of the external tone of an SOAE (fig. 18) . The shift in SOAE frequency varied from 10 Hz at F et 1700 Hz, to 75 Hz at F et 2500 Hz. It was noted that the frequency shift of the SOAE was higher at external tones with frequencies above those of the SOAE than by the external tones with frequencies less than those of the SOAE.

From these findings it is clear that the shift of both the amplitude and the frequency of the SOAE was higher when the frequency of the external tone was above that of the SOAE.

Fig. 10 The effect of the ipsilateral tone on SOAE
The frequency of the SOAE 2262 Hz

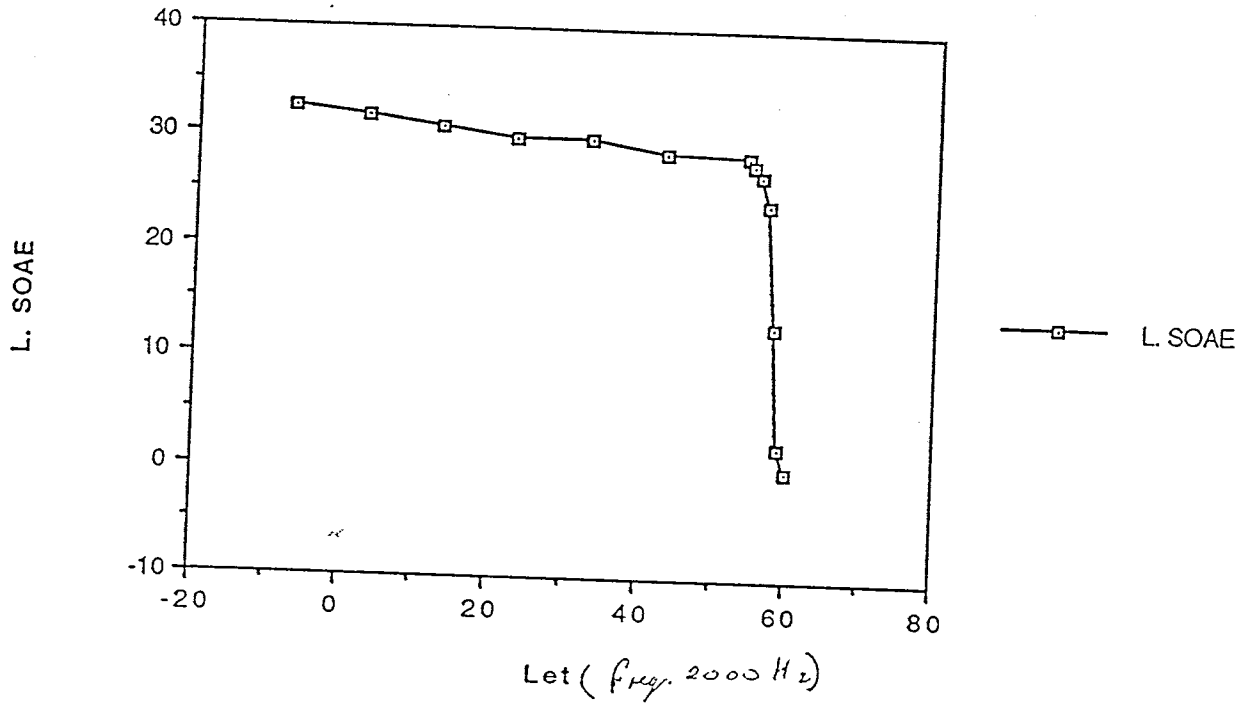


Fig. 11 The effect of Ipsilateral external tone on SOAE.
The frequency of SOAE 2262 Hz

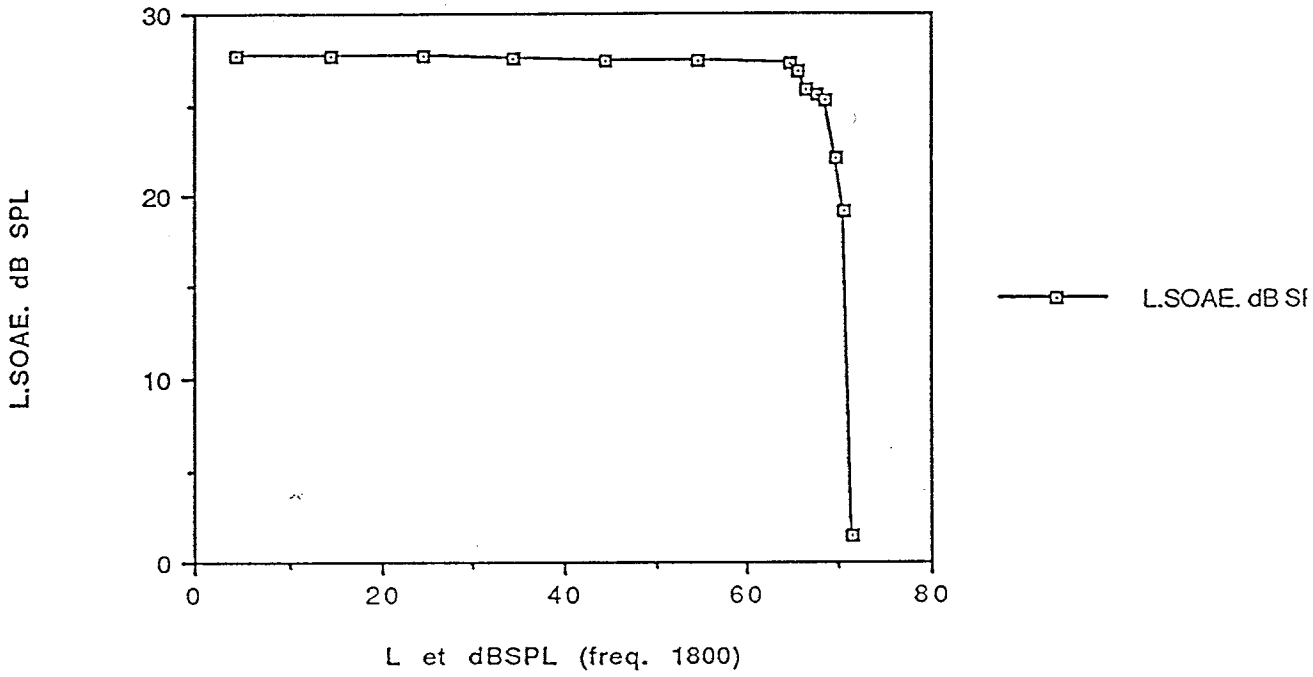


Fig. 12

Brüel & Kjær | Time Function Start: seconds | End: seconds | Not Expanded: | Expanded:

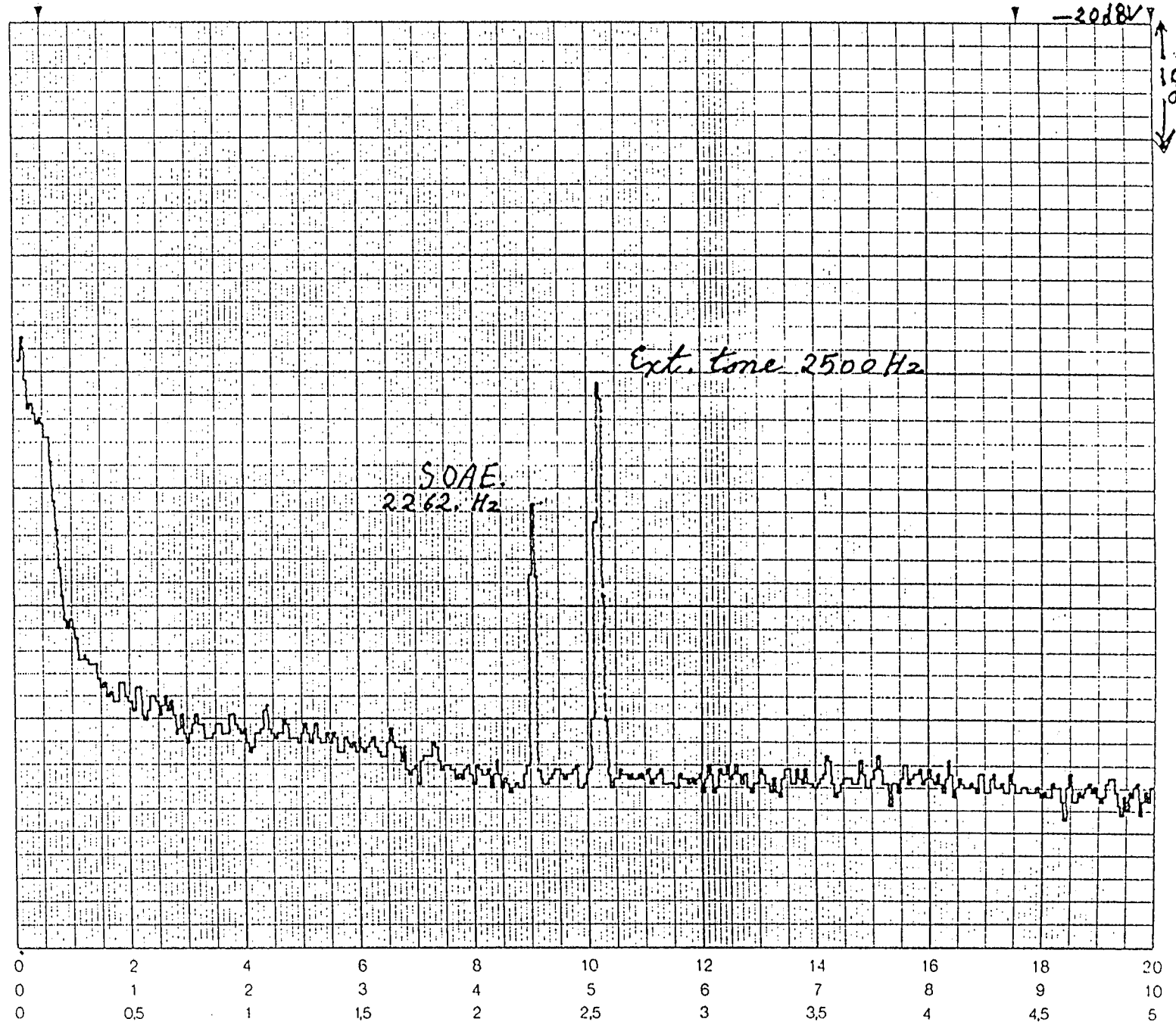
Full Scale Level: 80
F. S. Frequency: 5 KHz
Weighting: Hanning
Average Mode: Linear
No. of Spectra: 32

Comments:
Animal 184
Anesthesia yes
SOAE yes
f: 2262. Hz

ipsilateral supp. tone
at f = 2500 Hz

N.B atten. 47

Record No.:
Date: 8-2-89
Sign.:



DP 1002

Measuring Object:

Fig. 13

Brüel & Kjær

Time Function Start: ... seconds End: ... seconds Not Expanded: ... Expanded: ...

Full Scale Level: 70 dB

F. S. Frequency: 5 K.Hz.

Weighting: Hanning

Average Mode: Linear

No. of Spectra: 32

Comments:

Animal 184
Ear Rt
Anesthesia pbos

SOAE yes
f: 2262 Hz
isolated
fet = 1500 Hz
att. 34

Record No.: ...

Date: 8.2.89

Sign.: ...

20 40 80

10 20 30 40 50 60 70

External tone f: 1500 Hz

SOAE f: 2262 Hz

20dBV

10 dB

0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0,5 1 1,5 2 2,5 3 3,5 4 4,5 5

Measuring Object: ...

DP 1002

Fig 14 | 1262.5 | Fig. 14

Brüel & Kjær

Time Function Start: seconds End: seconds Not Expanded: Expanded:

Full Scale Level: 70
F. S. Frequency: 5K Hz
Weighting: Hanning
Average Mode: Lineak
No. of Spectra: 32

Comments:
Animal 184
Ear Rt
Anesthesia yes
-SOAE: Yes
-f: 2262 Hz

37

ipsilat Ext. tone:
f: 1700 Hz

att. 37

dB
10
20
30
40
50
60
70

20 40 80

-20dBV

10 dBV

Ext. tone f: 1700 Hz

SOAE f: 2262 Hz

0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0,5 1 1,5 2 2,5 3 3,5 4 4,5 5

Measuring Object:

Record No.:
Date: 8-2-89
Sign.:

> 1002

Fig. 15 The effect of ipsilateral external tone on SOAE

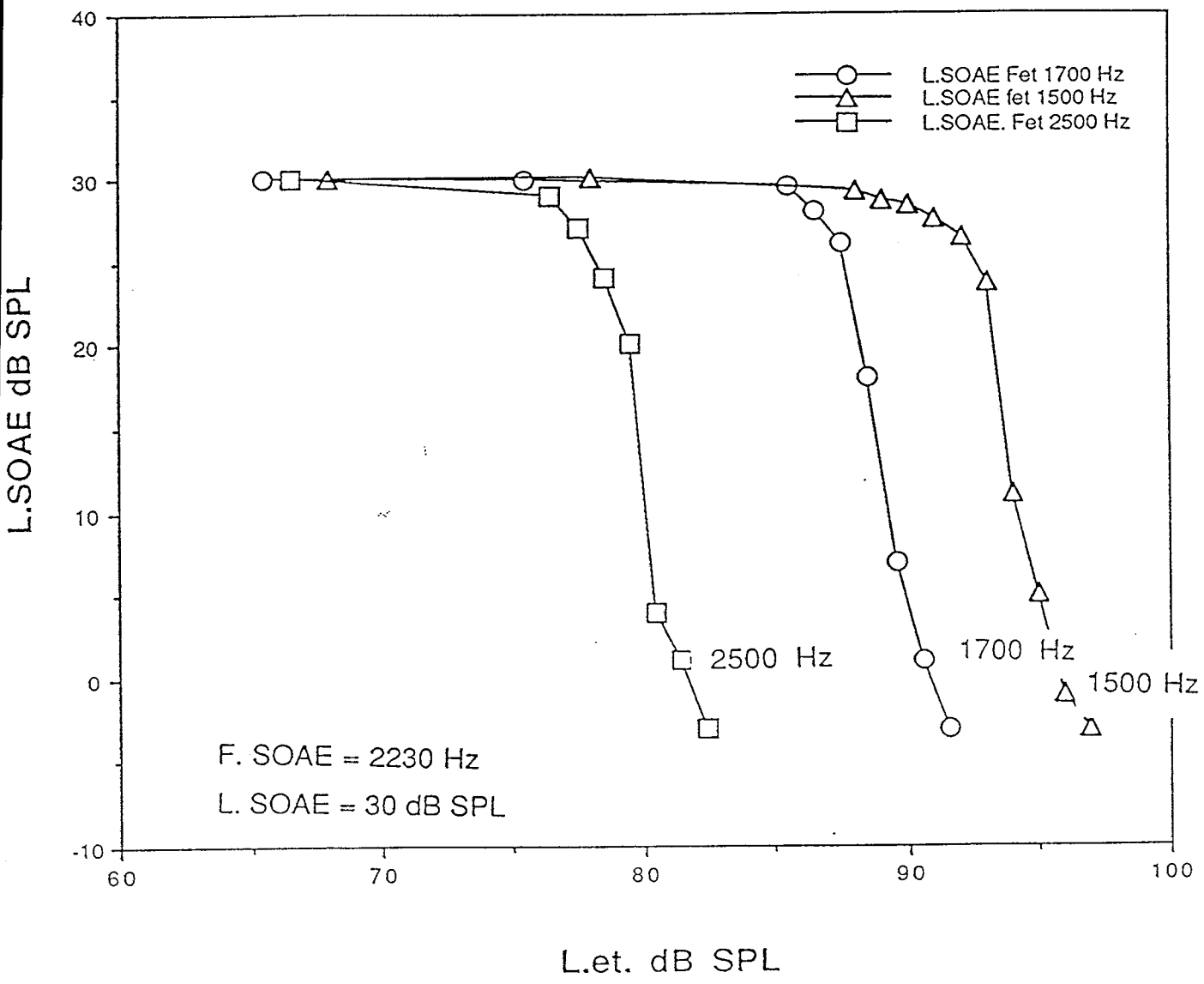


Fig. 16 The effect of the ipsilateral external tone on SOAE

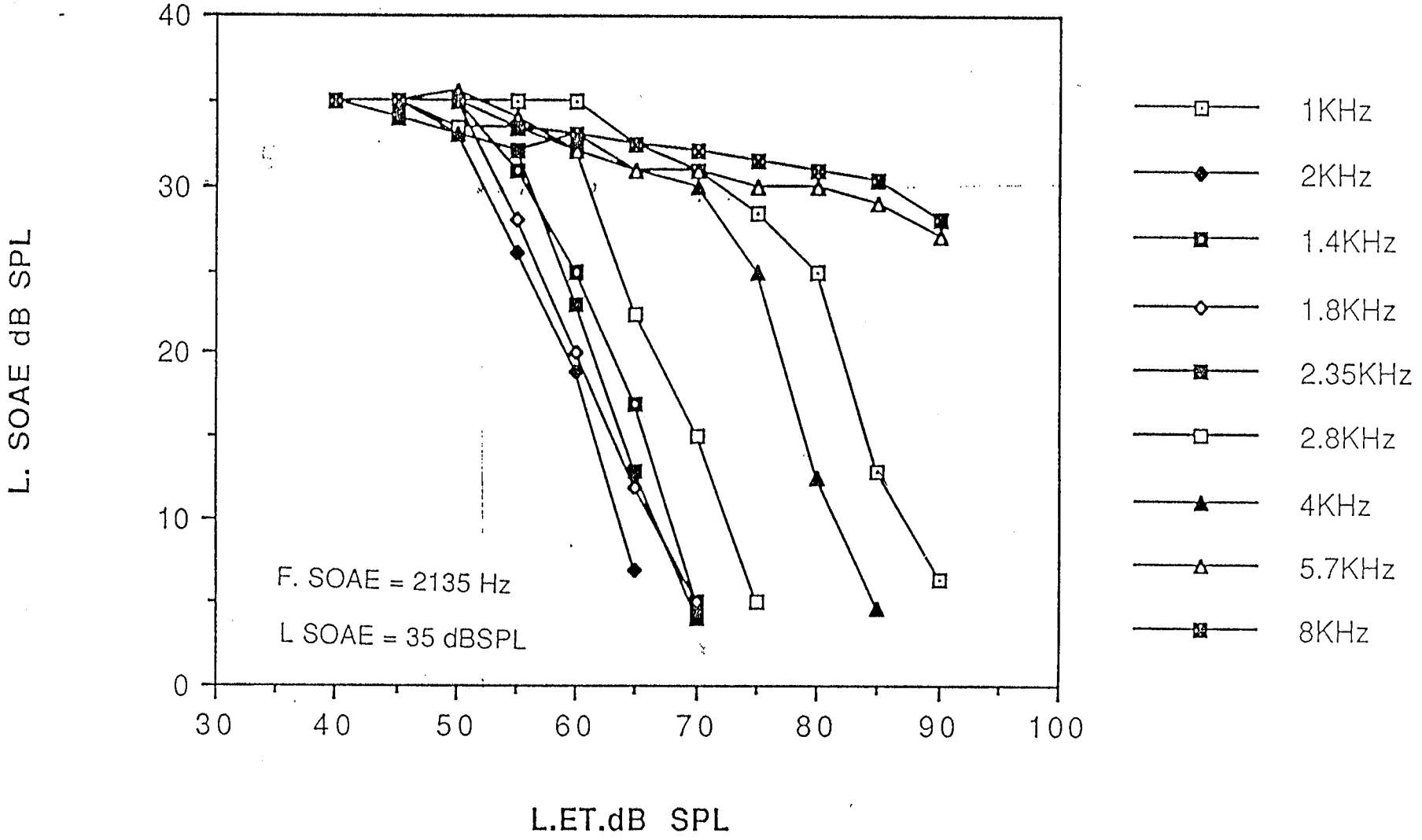


Fig. 17 SOAE amplitude shift as an effect of the ipsilateral external tone.

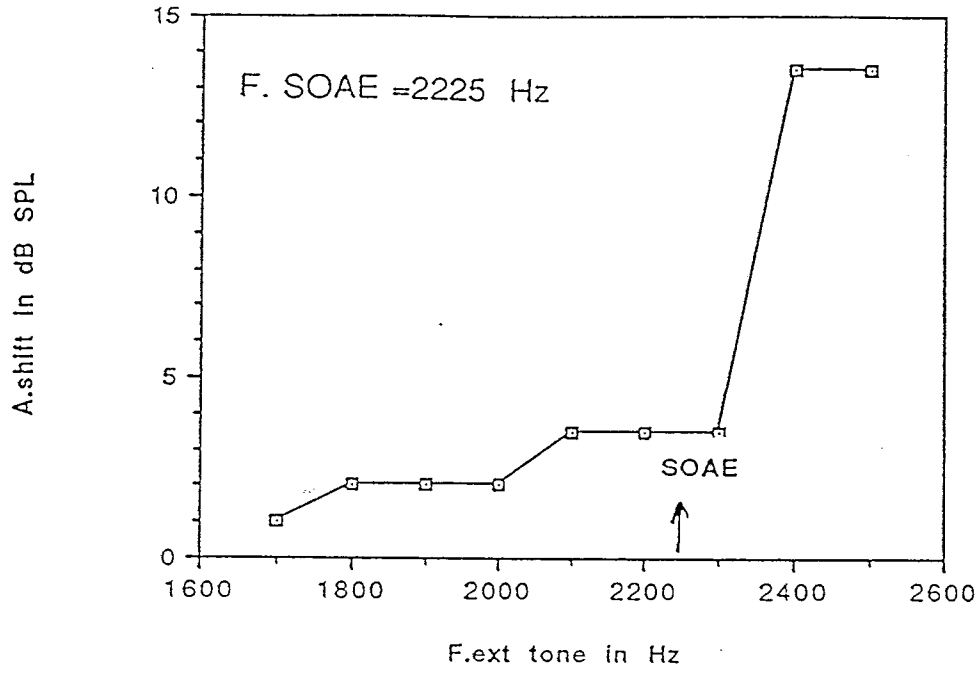
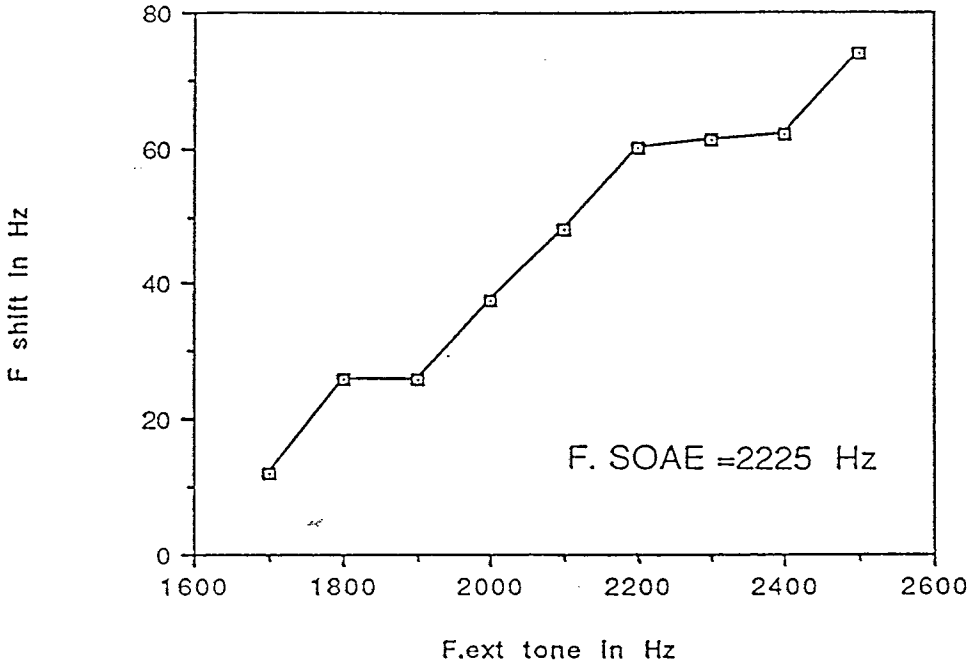


Fig. 18 SOAE freq. shift as an effect of the ipsilateral external tone



DISCUSSION

Discussion

In this experiment the effect of the ipsilateral external tone on SOAEs was studied. It is known that both ipsilateral and contralateral external tones showed suppression effect. The suppressor tuning curve is similar to the physiological and psychological tuning curve, and there is a shift in frequency as well as in amplitude as a result of direct effect of the external tone on the SOAEs (Clark et al,1984; Zizz and Glattke,1988; Frick and Matthies,1988; Mott et al,1989). The results of the present experiments are in agreement with the previous data. Figs 12,13 &14 show that both ipsilateral and contralateral external tones have a suppressor effect on SOAEs.

Rabinowitz and Widin (1984) noted that "the ambient level of an emission was maintained until the intensity of the external tone reached a critical level necessary for suppression of the SOAE. These levels varied with the frequency of the external tone. For tones below the frequency of the emission a steeply sloped masking function was found, with a 5-dB reduction in SOAE level for each 1-dB increase in external tone intensity. Gradual suppression (1 dB/dB) was also noted for tones well above the SOAE frequency; also the function became steeper as the external tone was reduced toward the emission". Norton et al (1989) concluded that "following intense or mechanical stimulation, the OHC stereocilia, particularly on the first row are disarrayed and/or floppy. This behavior is associated with damage to either the stereocilia rootlet or the structures that

couple it to the cuticular plate". Neely and Kim (1983, 1986) have proposed an active bidirectional transduction mechanism in the steriocilia and the cuticular plate region. They propose that "mechanical energy is released from an internal source within the cochlear partition through OHC stereocilia. The amount of energy released is inversely related to the level of an input stimulus. Irregularities in the OHC subsystem result in excess gain in the absence of external stimuli, producing SOAEs. Stereocilia stiffness probably regulates the gain control on the energy source. If the stereocilia and tectorial membrane are decoupled the amplifier output may fall to zero, resulting in a disappearance of an SOAE. Less dramatic changes in stiffness may result in decrease in the gain control of the OHC-BM feedback loop, and thus a decrease in the rate and magnitude of the oscillation of the SOAE generator.

In the present study the suppression due to the ipsilateral tone was minimal at first, then at a critical level the suppression suddenly increased to the limit of the level of the noise floor of the system. This occured wih all ipsilateral external tones at different frequencies. It was noticed that the suppressor effect of the external tone was high when the frequency of that tone was close to the frequency of the SOAEs.

IV. EXPERIMENT THREE

CONTRALATERAL SUPPRESSOR TONES

INTRODUCTION

The influence of contralateral acoustic stimulation on a spontaneous otoacoustic emission (SOAE) was examined to assess possible effects of acoustically-elicited, olivocochlear-bundle (OCB) activity and middle-ear-muscle contractions. An OCB effect, which might be frequency selective (Fex, 1963; Buno, 1978; Cody and Johnstone,1982) was particularly intriguing since the demonstrations by Moutain (1980) and Siegel and Kim (1982) that electrical stimulation of the crossed OCB affects cochlear mechanical processes, as seen via changes in distortion product acoustic emissions. Rabinowitz and Widin (1984) reported that the effect of the contralateral tone appears to have resulted from intra- and /or extra-cranial acoustic conduction to the ipsilateral ear whereupon the SOAE was then suppressed. With contralateral noise, increase in noise level (from 44 to 84 dB in 10-dB steps) caused the level of the SOAE to be reduced and the frequency to be increased. Suppression began at 64 dB SPL, with SOAE changes of -1 dB and 2 Hz; at 84 dB SPL, these changes reached -5 dB, 7 Hz. He concluded that the fact that acoustic reflexes, which modify the state of the middle ear, are able to influence both the level and the frequency of a SOAE indicates that the mechanism of SOAE generation is coupled with the mechanical environment bounding the cochlea. This conclusion is also consistent with other results (Schloth and Zwicker 1983; and Grose, 1983).

Mott et al (1989) concluded that several observations suggest that the effects of the contralateral stimulation were not due to acoustic reflex activation:

1) Longer latencies were observed for supra-contralateral acoustic reflex threshold (CART) levels than the sub-CART levels.
2) SOAE frequency and amplitude shifts observed during reflex activation conditions were larger than those observed during non-reflex conditions; amplitude always decreased during reflex conditions.
3) At the end of the experiment, acoustic reflex decay at 4000 Hz was measured. The reflex decayed 50 % within 7s and 100% within 20 s. However, during the long term contralateral acoustic stimuli (CAS) condition SOAE frequency was elevated for the entire four minute stimulation period.
4) Although the results were not completely unambiguous, SOAEs arising at multiple frequencies in the same ear were affected differently by the same set of the contralateral stimuli.

Neither acoustic reflex activity nor suppression by trans-cranial conduction of sound energy can account for these observations. They can be explained by assuming that efferent activity evoked by the contralateral stimulus alters the intrinsic tuning of the emission generator by causing changes in OHC membrane properties. These results were consistent with those of other investigators who concluded that the efferent system plays a role in cochlear mechanics (Mountain, 1980; Siegel and Kim, 1982; Guinan, 1986). However, Mott et al add the additional speculation

that efferent activity indirectly alters the mechanical response of the cochlea by affecting the phase of the feedback force, and not by direct altering more fundamental mechanical properties, such as stiffness.

In the previous experiment, we investigated the effect of the ipsilateral stimuli on the SOAEs. In this experiment, we measured the effect of the contralateral external tone on the SOAE, studied the difference between the suppressor curve resulting from the contralateral external tone and the suppressor curve resulting from the ipsilateral one, the value of interaural attenuation was measured (IAA), subtract this value from the intensity of the contralateral external tone (L contra), then introduce ipsilateral external tone with intensity equal to (L contra-IAA) which in turn is equal to the intensity of the contralateral external tone when it reaches the ipsilateral ear.

EXPERIMENT THREE

MATERIAL AND METHODS

METHODS

Subject

The subject for this experiment was animal 184, the same animal used in experiment no. 2.

Measurement

Equipments

As was described in experiment 1 & 2, the miniature microphone was coupled by plastic tubing to the end of one of three tubes. Pure-tone stimuli were generated by a Wavetek generator (model148) connected to one of the other two tubes in the probe assembly.

In this experiment, two sets of probe assemblies (discussed before) were used so the microphone could be inserted into the ear canal of the emitting ear, and an external tone could be introduced contralaterally.

Procedures

The chinchilla was anesthetized with the same kind of anesthesia (Ketamine hydrochloride) in a dose of 15 mg/kg body weight. The external tone was applied into the contralateral ear (left side) by an ear piece of one of the probe assemblies.The microphone of the other probe assembly was used to detect the SOAE

from the right side, which was analyzed and recorded on the wave analyzer. The external tone was applied at different frequencies. Its level was raised until the emission disappeared and a suppressor contour was plotted.

Interaural attenuation was measured as follows:

1. The calibrated external tone was delivered through the probe system to one ear.

2. The signal was detected by the microphone inserted in the same ear. On the other hand the same signal was detected by the probe system in the contralateral ear. The result was analyzed and recorded on the wave analyzer.

3. The difference between the level of the sound introduced in one side (LI) and the level of this sound detected from the other side (L contra) was equal to the interaural attenuation (IAA= LI-L contra).

4. The value of the interaural attenuation was subtracted from the level of the contralateral tone (Lipsia = Lcontra- IAA) .

5. An external tone was introduced ipsilaterally with a level equal to the difference between the interuaral attenuation and the contralateral tone.

6. We studied the difference between the effect of the contralateral tone and the effect of a tone with a level equal to the difference between that and the interaural attenuation that was introduced into the emitting side.

EXPERIMENT THREE

RESULTS

RESULTS

Contralateral Suppression Contour Curve

As was mentioned earlier the effects of the contralateral external stimuli on the SOAE were studied using the emitting animal 184 (fig. 19 & 20). The level of the SOAE was plotted as a function of the level and frequency of the contralateral suppressor tone (figs. 21 & 22).

Figure 21 shows the effect of the suppressor tone on the level of the SOAE. The attenuating effects were similar to those of the ipsilateral supressor tone. As the level of external tone was raised, the SOAE was first reduced slightly--1.3 dB with an increase of 5 dB in external tone level (SOAE frequency was 2137 Hz and (Fet) frequency of external tone was 2500 Hz), with a following abrupt reduction--9 dB with an increase of 1 dB in external tone. This change in the suppression contour curve (sudden change in the slope of the curve A) was observed when the external tone had a frequency close to that of the SOAE (Fet 2500 Hz & F SOAE 2287 Hz). On the other hand, when the frequency of the external tone was not close to the frequency of the SOAE, the shape of the slope differed.

Figure 22 shows that the attenuations of the level of SOAE in both curves (B & C) with an external tone of 1500 Hz and 1700 Hz respectively are gradually increased. The level of the SOAE is reduced 10 dB with an increase of 5 dB in external tone. It can be

also noticed that both the level and the frequency of the contralateral external tone have an effect as a suppression tone on the SOAE; as the level of the contralateral suppressor tone was raised the level of the SOAE was reduced. The SOAE was reduced abruptly when the external tone had a frequency near that of the SOAE; the SOAE was reduced slightly and gradually when the frequency of the SOAE was far from that of the SOAE.

Amplitude Shift and Frequency Shift

When a contralateral suppressor tone was applied, a shift in both the frequency and the amplitude of the SOAE was observed. This shift was investigated by measuring the change in level and frequency of the SOAE as a function of the level of the applied contralateral external tones of various frequencies (Fig. 23). At an external tone frequency of 1700 Hz, the frequency shift of the emission was slight (12 Hz). The frequency shift of the emission was more elevated when the frequency of the external tone was higher and closer to the frequency of the SOAE (2225 Hz). A shift of 23 Hz at external tone of 1800-2000 Hz, while a 36 Hz at an external tone of 2100 Hz were recorded. The shift was even greater when the frequency of the external tone was above that of the SOAE--50 Hz at an external tone of 2300 Hz and 2400 Hz; 60 Hz at 2500 Hz.

The amplitude shift as a function of different contralateral external tones frequency applied at a constant level was also studied (fig.24). The effect was similar to that of the ipsilateral external tone.

There was a minimal shift in the amplitude when the frequency of the external tone was less than that of the SOAE (less than 5 Hz) and that the shift was suddenly and abruptly increased (about 13 Hz) when the frequency of the applied external tone was more than 2300 Hz.

It was also observed that both the frequency and the amplitude shift were more if the frequency of the external tone was close to or above that of the SOAE.

Summary of the Ipsilateral and Contralateral External Tone Effect on SOAE

Figures. 25, 26 & 27 depict the difference in effect of the contralateral and the ipsilateral external tone on the SOAE. The frequency of the SOAE was 2262 Hz and 2137 Hz in cases of ipsilateral and contralateral external tone respectively. The suppression was gradual with the contralateral external tone; and with the ipsilateral external tone there was minimal suppression, then sudden suppression in the level of the SOAE (Figures 25 & 26), but with a frequency of 2500 Hz (which is very close to the frequency of SOAE), the suppression of the SOAE was similar in both cases (Figure 28). The difference in the level of the ipsilateral and the contralateral external tone was plotted as a function of the level of the SOAE (ipsi) in dB SPL, the relation was linear, and it was clarified that the contralateral attenuation curve started earlier, and gradually slopes down till the end of the curve. In contrast the

ipsilateral attenuation curve shows that more energy is needed to start the attenuation.

Figures 29 & 30 summarize the frequency and amplitude shift of the SOAE, as an effect of the ipsilateral and the contralateral external tone. It was clear that both (contralateral and ipsilateral) have a similar effect regarding the shift of the frequency in Hz and the amplitude in dB SPL of SOAE.

Fig 19 1150

Fig. 19

Brüel & Kjær

Time Function Start: seconds End: seconds Not Expanded: Expanded:

Full Scale Level: 100 dB

S. Frequency: 5KHz

Weighting: Hanning

Average Mode: Linear

No. of Spectra: 32.

Comments:

Animal: 184
Anaesthesia: Yes
dose 0.06 ml.
Ketamine I.M

SOAE Yes
Freq 2137 Hz

Contralateral tone
Yes. Freq 2.5 KHz
at: 39

Record No.:

Date: 7/21/89

Sign.:

22 dB SPL

10 dB SPL

SOAE
f: 2137 Hz

Contralat Ext. tone
Freq 2500 Hz

10 20 30 40 50 60 70

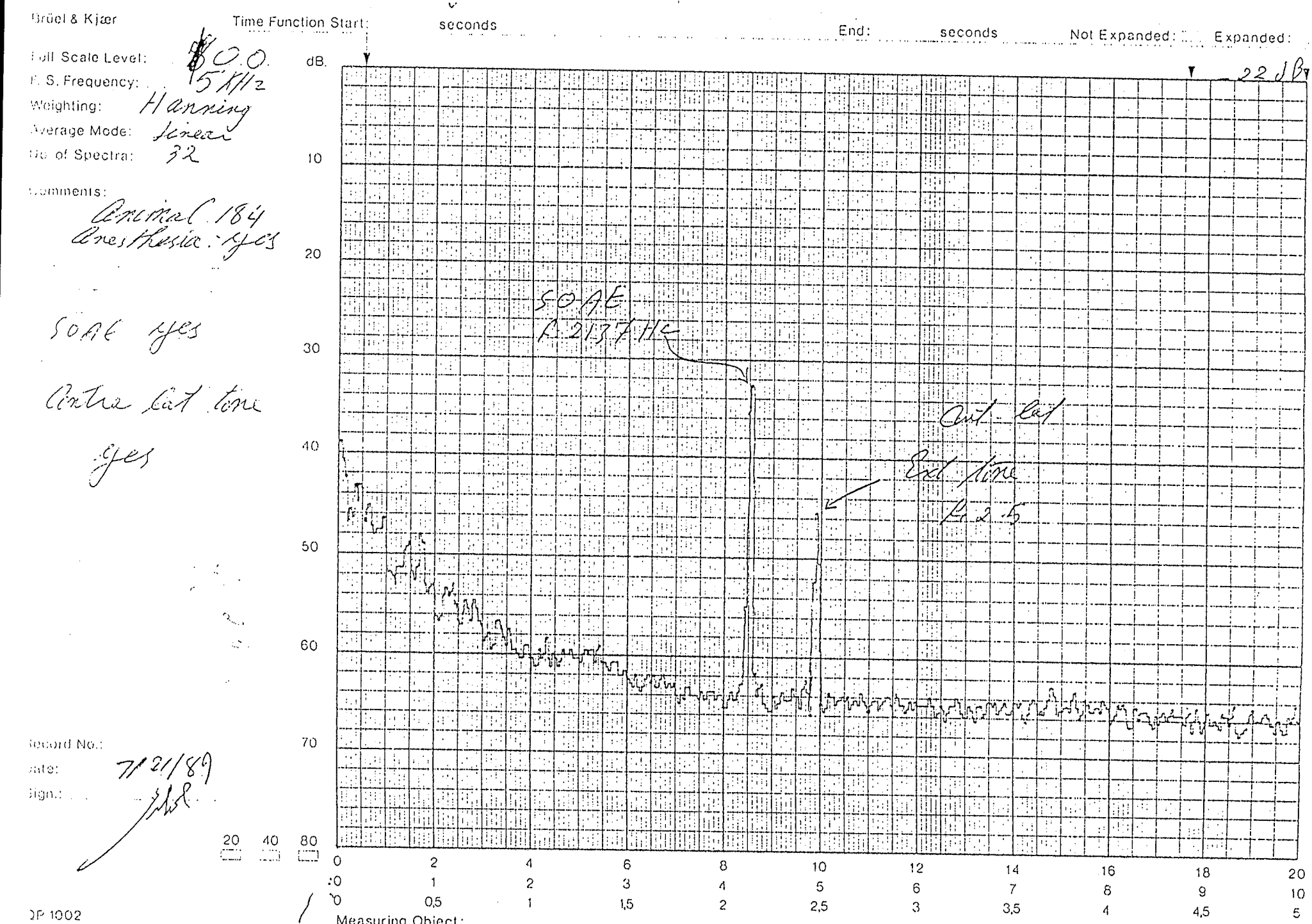
20 40 80

0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0,5 1 1,5 2 2,5 3 3,5 4 4,5 5

DP 1002

Measuring Object:

Fig. 20


Brüel & Kjær
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End: seconds
Not Expanded:
Expanded:
Full Scale Level: 80.0
F. S. Frequency: 5 kHz
Weighting: Hanning
Average Mode: linear
No. of Spectra: 32
Comments:
Animal C 184
Anesthesia - yes
SOAE yes
Extra Ext tone
yes
dB.
22 dBV
SOAE
f 2137 Hz
Ext. Ext
Ext tone
L 2.5
Record No.:
Date: 7/21/89
Sign.:
10
20
30
40
50
60
70
20 40 80
0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0,5 1 1,5 2 2,5 3 3,5 4 4,5 5
Measuring Object:
QP 1002

Fig 21: The effect of the contralateral external tone (freq. 2500 Hz) on SOAE (Freq. 2137)

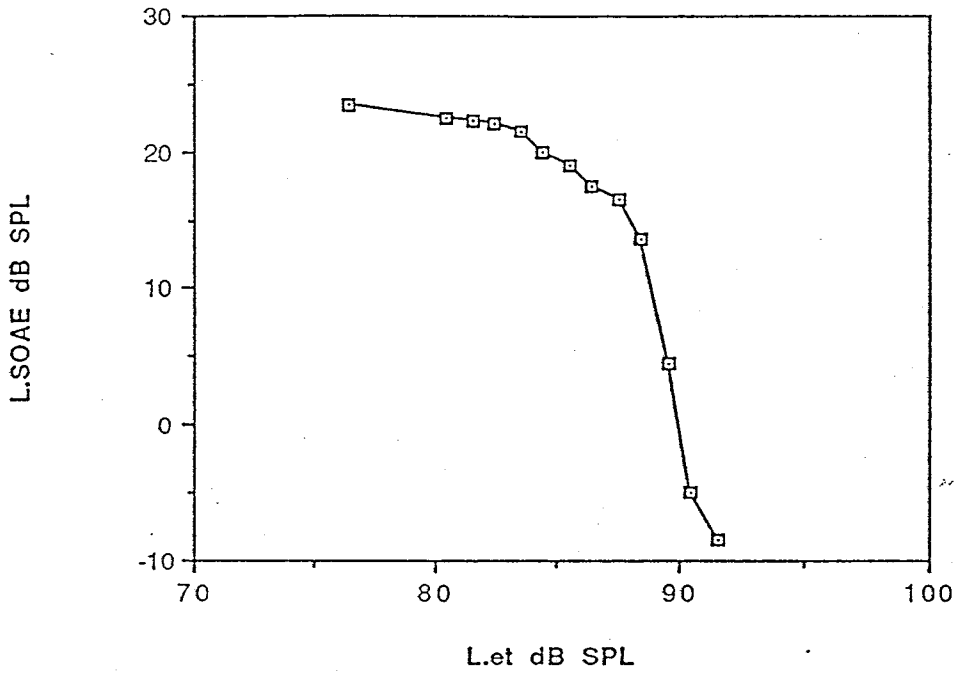
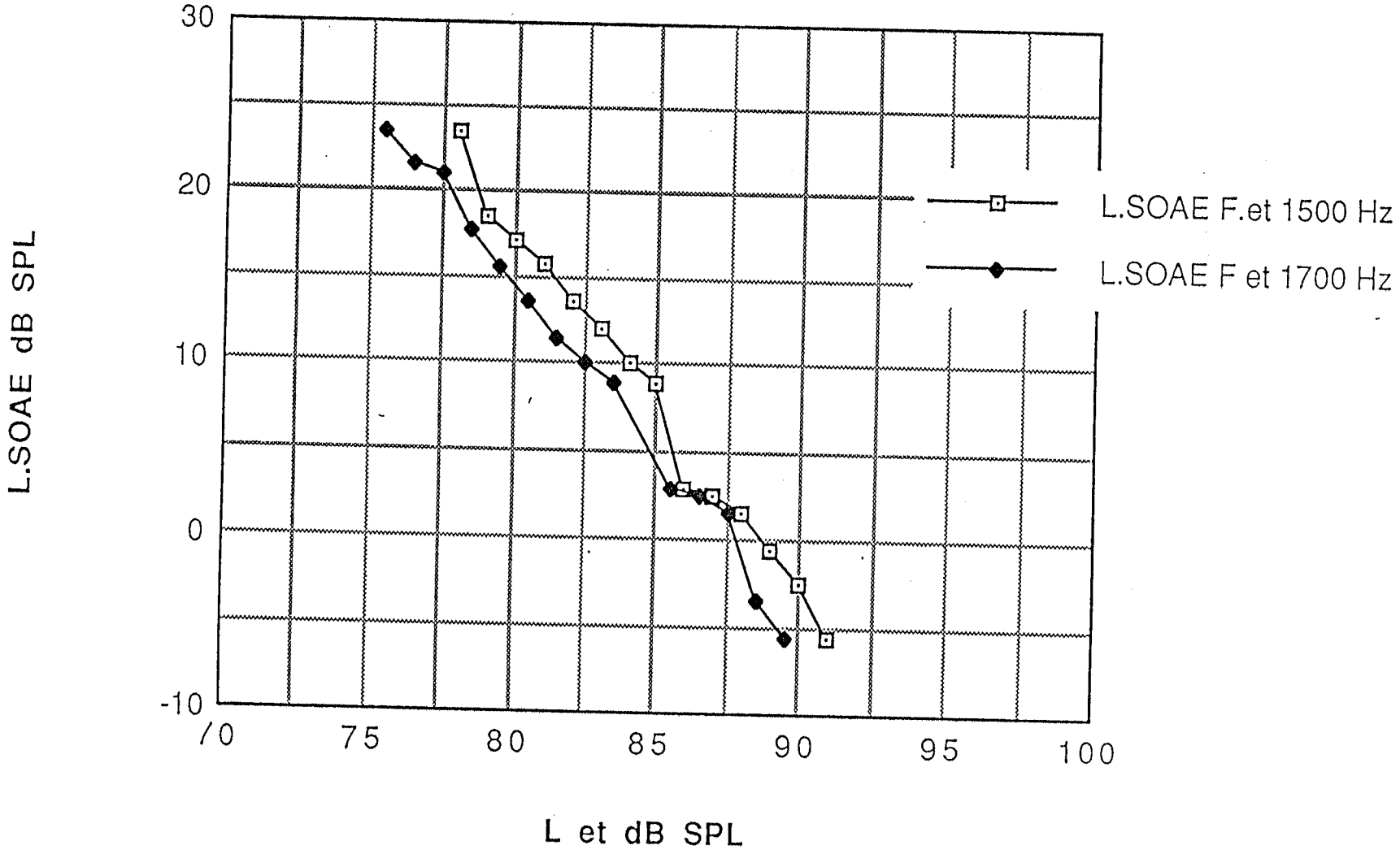


Fig 22: The effect of the contralateral external tone (freq. 1500 & 1700 Hz) on SOAE (freq. 2137 Hz)



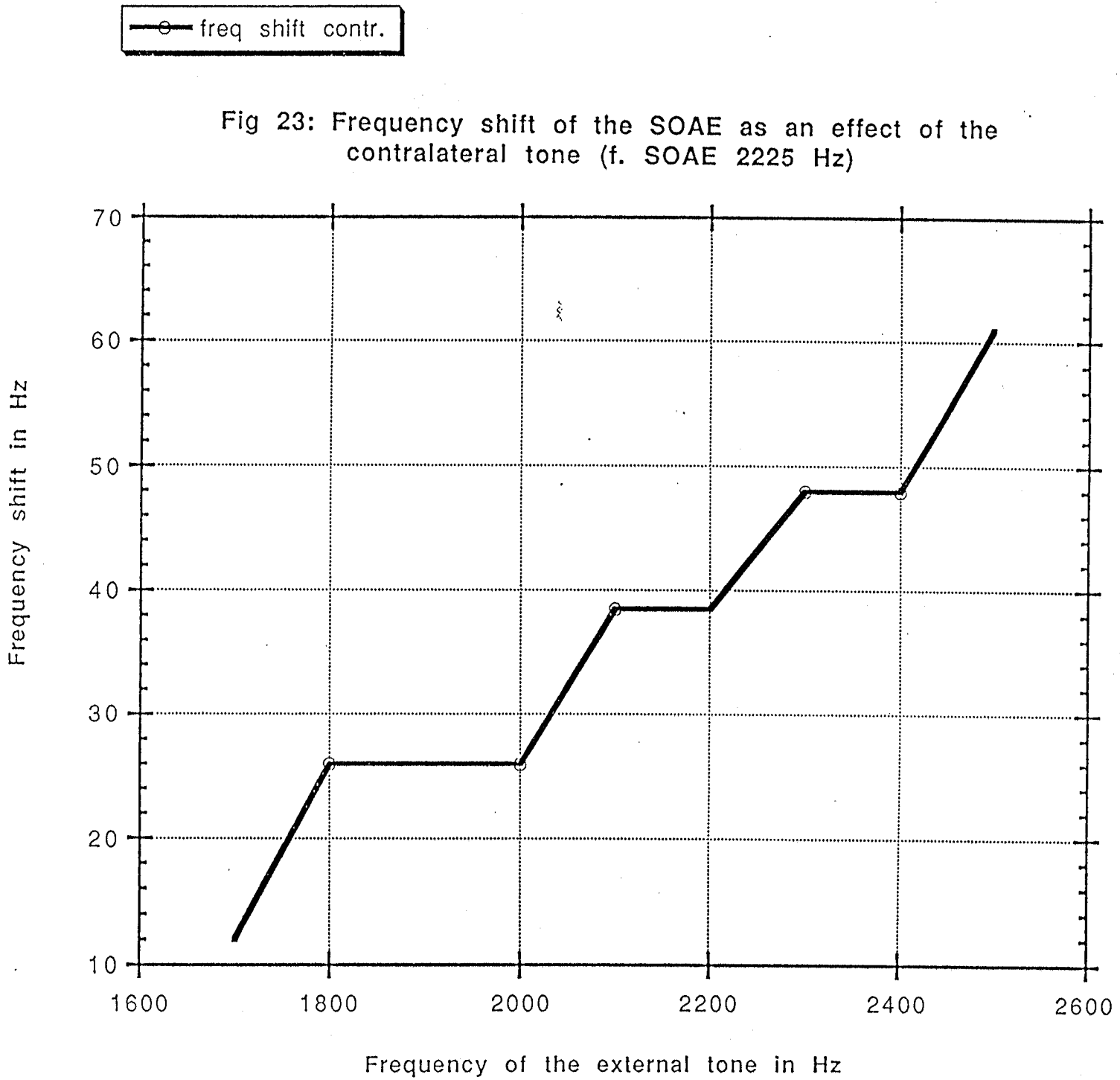


Fig 23: Frequency shift of the SOAE as an effect of the contralateral tone (f. SOAE 2225 Hz)

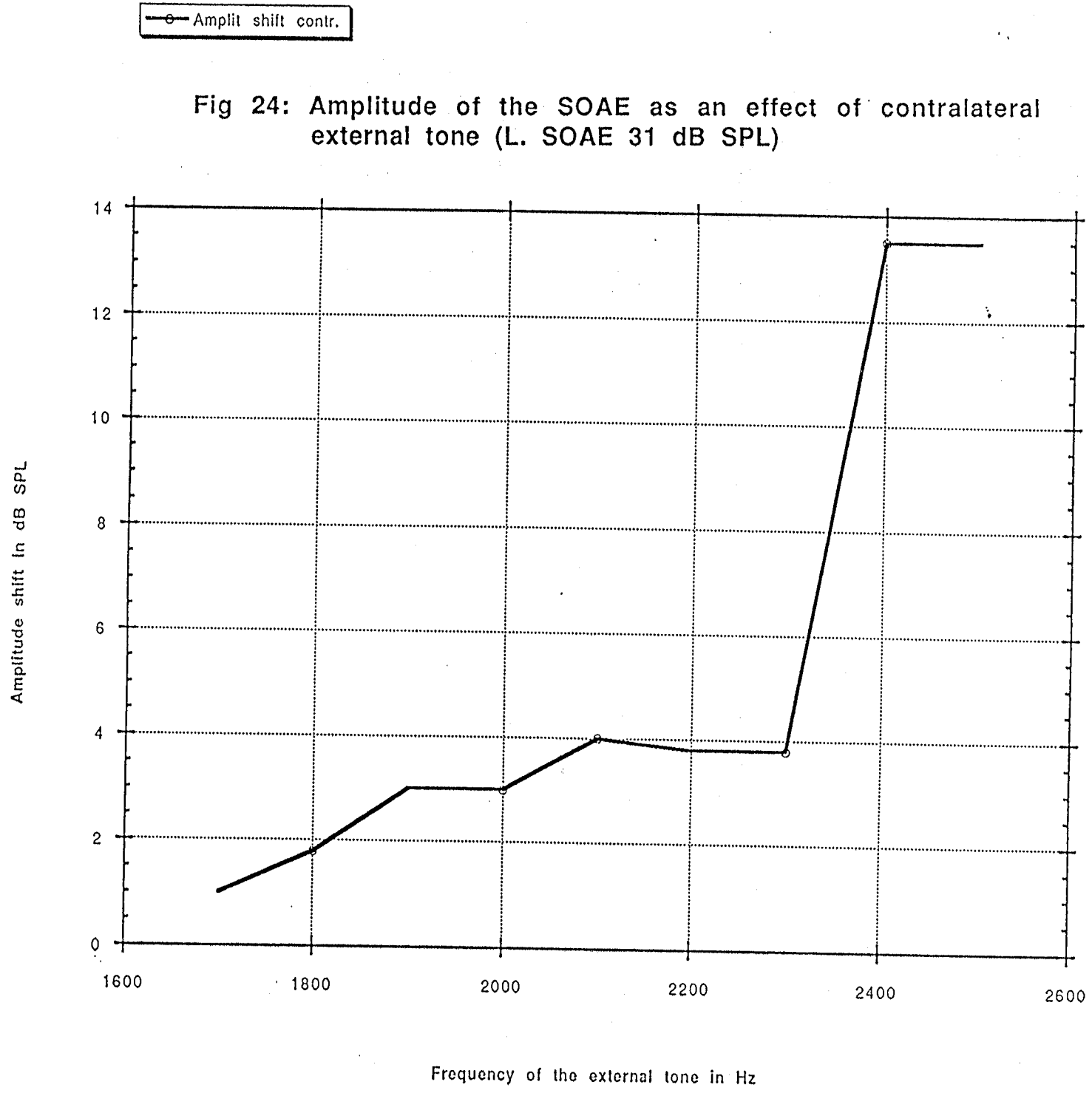
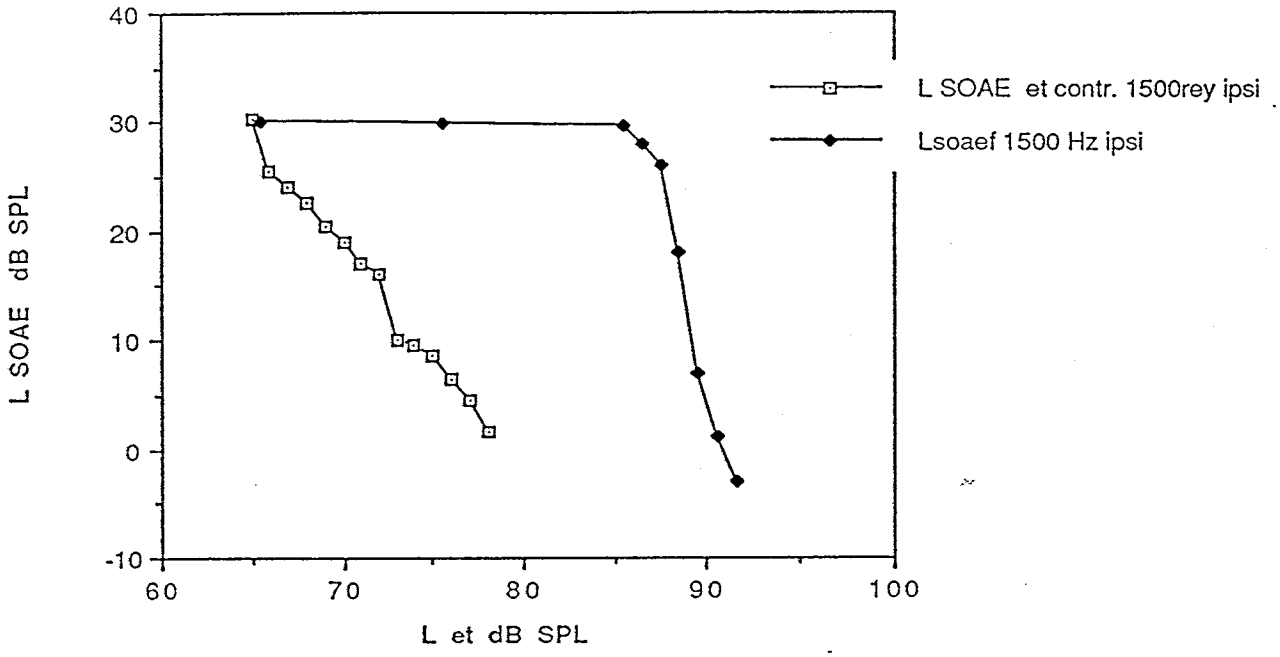


Fig 24: Amplitude of the SOAE as an effect of contralateral external tone (L. SOAE 31 dB SPL)

Fig. 25: the effect of the ipsi and contrlatera tone (both have freq. 1500 Hz) on SOAE



. 26: The effect of the Ipsi and contralateral external tone (both have freq. 1700 Hz) on SOAE

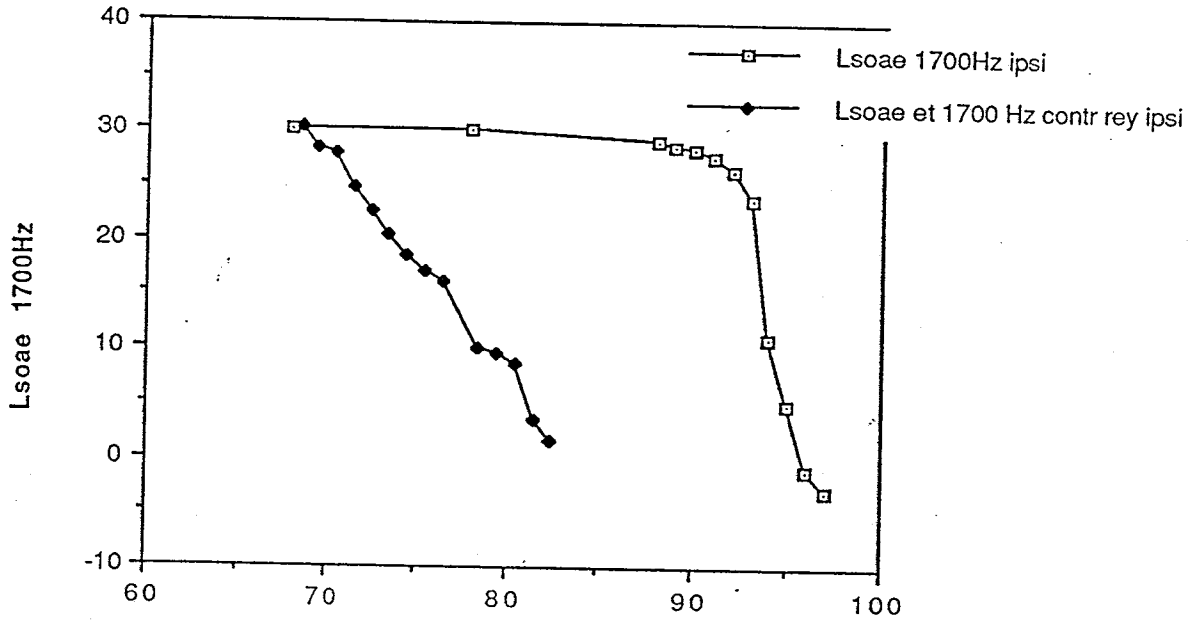
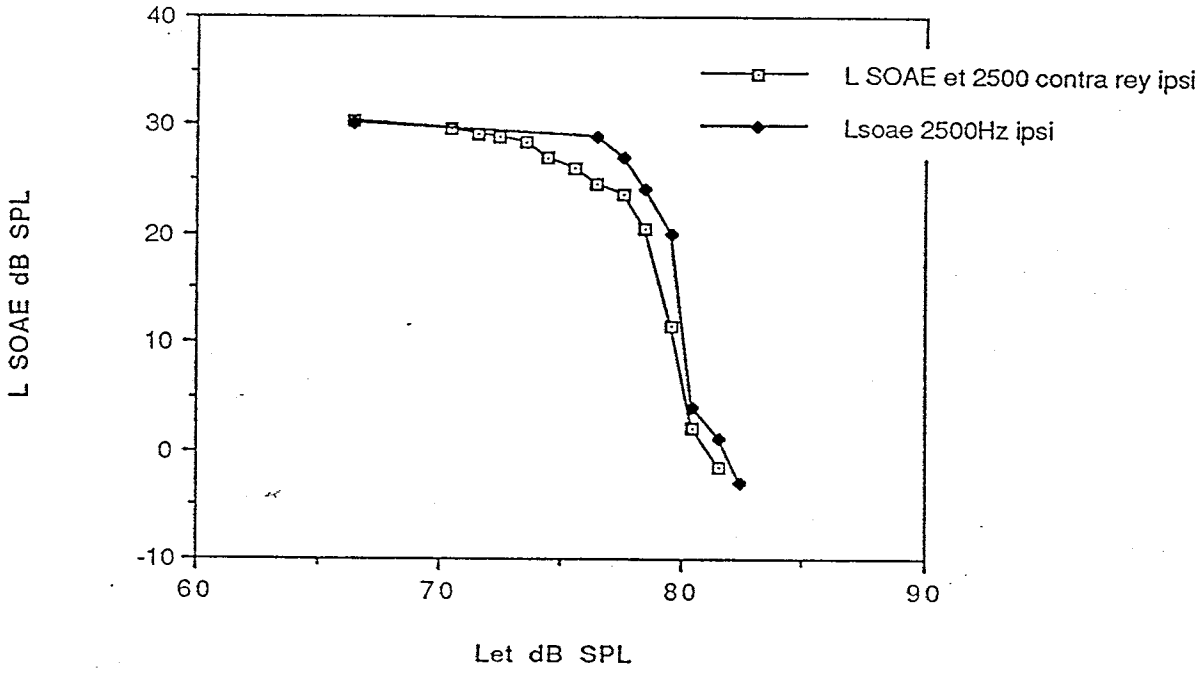


Fig. 27: The effect of ipsi and contralateral external tone (both have freq. 2500 Hz) on SOAE



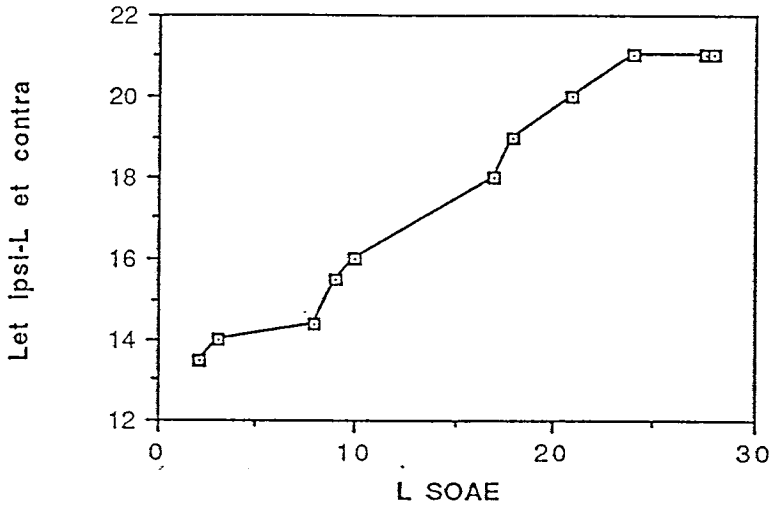


Fig. 28: The difference in the level of the ipsilateal and the contralateral external tones was plotted as a function of the level of SOAE

Fig. 29: Freq. shift of SOAE as an effect of the ipsi and contralateral tone

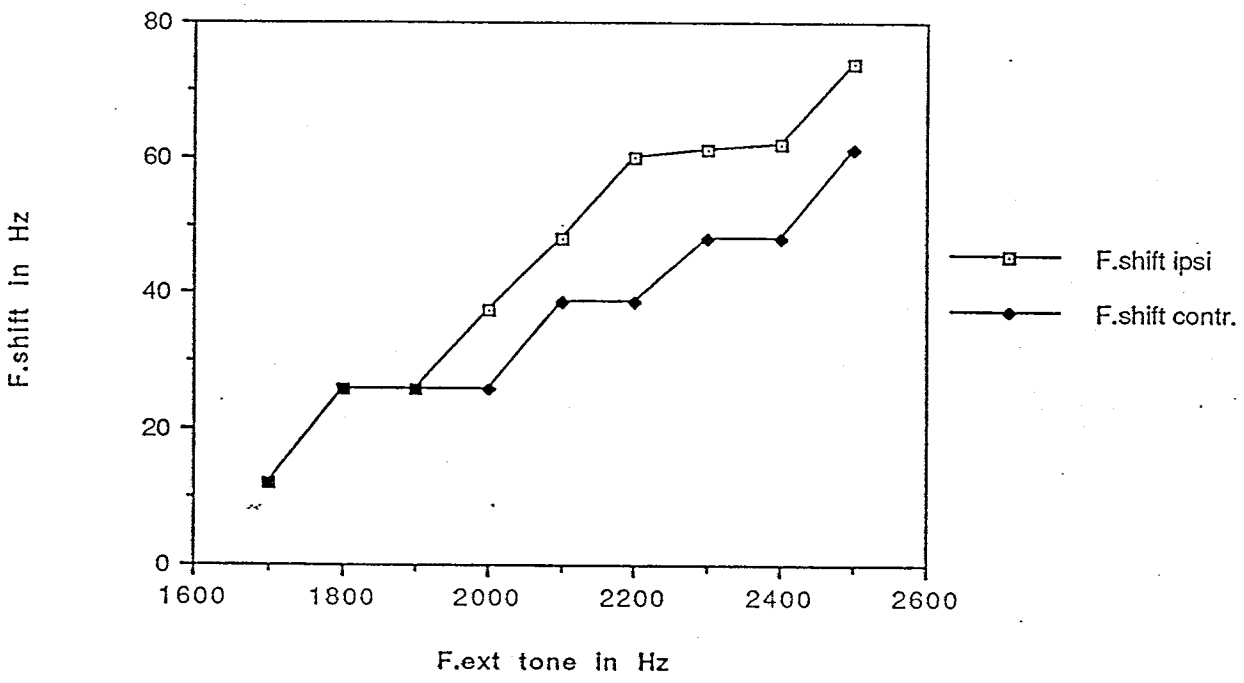
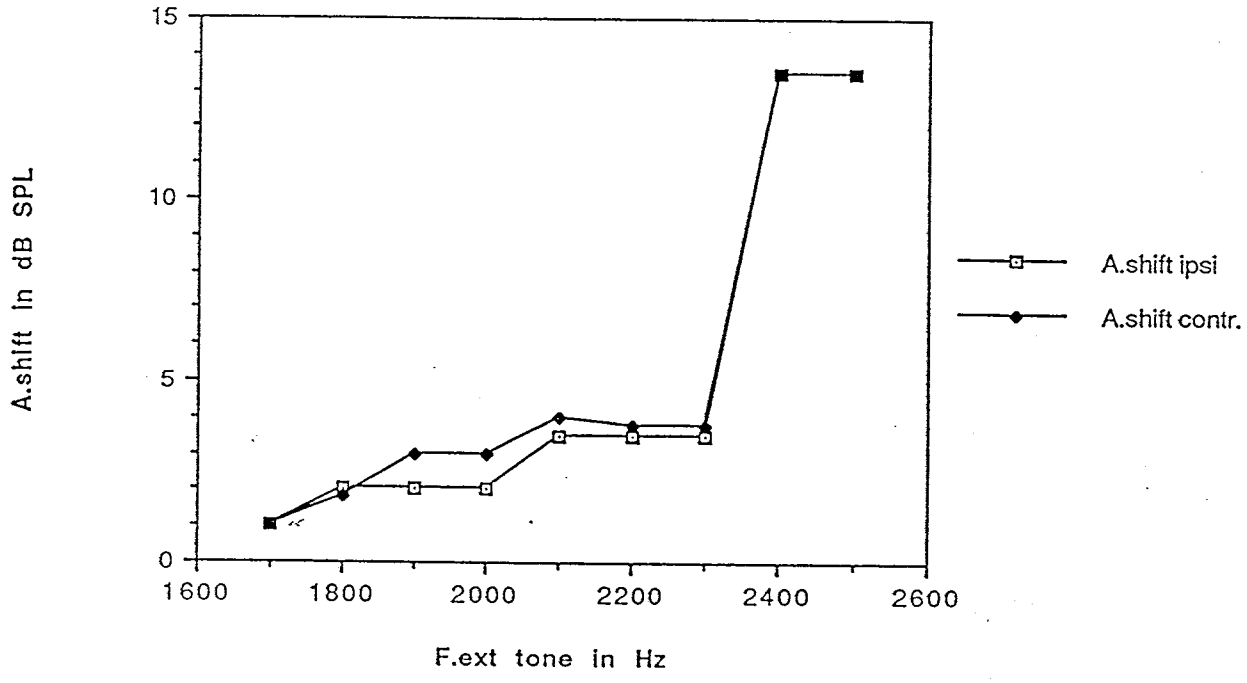


Fig. 30 Amplitude shift of SOAE as an effect of ipsi. and contralateral tone



DISCUSSION

DISCUSSION

Dealing with contralateral stimulation, anatomical and physiological evidence obtained in animals suggests that part of the olivocochlear bundle may serve as a link between the two cochleas (Fex, 1962 and 1965; Robertson and Gummer, 1985; Liberman and Brown, 1986) such that acoustic stimulation of one cochlea may alter sound-induced activity in the opposite cochlea. For example auditory nerve fiber discharge rates (Buno, 1987; Murata et al,1980) and temporary threshold shifts of the whole nerve compound action potential (Cody and Johnstone, 1982; Rajan and Johnstone, 1983) are reduced in nonhuman animals by contralateral acoustic stimulation. Mott et al (1989) summarized that contralateral acoustic stimulation can produce alterations in the frequency and amplitude of spontaneous otoacoustic emissions. Neither acoustic reflex activity nor suppression by transcranial conduction of sound energy can account for these observations. They can be explained by assuming that the efferent activity evoked by contralateral stimulus alters the intrinsic tuning of the emission generator by causing changes in OHC membrane properties. Efferent activity indirectly alters the mechanical response of the cochlea by affecting the phase of the feedback force, and not by directly altering more fundamental mechanical properties such as stiffness (Mott et al,1989).

In the present study the suppression due to contralateral external tone applications was studied, SOAEs decreased gradually without any sudden change except for those with frequencies very

close to that of the SOAE; the suppression curve was similar to that of the ipsilateral external tone. It was also noticed that the contralateral external tones started to suppress SOAE earleir and gradually, than the ipsilateral exteranl tones which needed more intensity till it reached the point at which the level of the SOAE was suddenly dropped. This observation was clarified in Fig. 28 which showed that the difference between the level of the ipsilateral external tone and that of the contralateral external tone as a function of the level of the SOAE (ipsi) in dB SPL was linear. This difference in the effect of the ipsilateral and contralateral acoustic stimulation helped us to realize that there is an efferent effect on SOAEs. This is consistent with other investigators who concluded that the efferent system plays a role in cochlear mechanics (Mountain, 1980; Siegel and Kim, 1982; Guinan,1986; Norton et al,1989), and raises a question about the source of the mechanical energy producing SOAEs. There is nearly no doubt now that the central nervous system has a role in controlling the SOAÉ emission, but is the source of the SOAE in the cochlea (peripheral) ? or in the cerebral cortex (central)?

Temporary threshold shift following exposure to intense acoustic stimuli (both contralateral and ipsilateral) is a well-documented phenomenon in both animals and humans (Ward. W.D, 1973). Clark et al (1984) submitted that the shift in SOAE frequency caused by the external suppressor tone is not straightforward, and that most of the tested external tone frequencies reduced the SOAE frequency or had no effect with maximum reduction in the 1 and 8 KHZ. Temporary threshold shift tends to be greatest at frequencies

1/2 to 1 octave above the frequency of the exposure stimulus. This so-called half-octave shift has also been demonstrated in whole-nerve responses (Yates, Cody and Johnstone, 1983) as well as single 8th-nerve fibers (Lonsbury-Martin and Meikel, 1978). In a study by Norton et al (1989), both behavioral temporary threshold shift (TTS) and SOAE data showed a half-octave shift, which means that this effect originates within the cochlea.

In the present study of the frequency shift as a function of the external tone (both contralatral and ipsilateral), the results agree with previous studies that there was a frequency shift as a result of external tone application, and there was a relation between the amount of this shift and the frequency of the applied external tone. In Fig 29, it was clear that as the frequency of the external tone increased, the shift of the SOAE frequency increased (for example, at 1800 Hz external tone the shift was 26 Hz, and at 2400 Hz external tone the shift was more than 60 Hz).

V. EXPERIMENT FOUR

Spontanoeus Otoacoustic Emissions in Squirrel Monkeys

INTRODUCTION

A recent survey of Old and New World monkeys showed that spontaneous otoacoustic emissions (SOEs) were detected in approximately 5% of the monkeys (Martin et al, 1985). The investigative effort aimed at corroborating the incidence of SOEs in macaques alone seemed worthwhile since previous detailed observations on an emitting monkey indicated that the level, frequency, bandwidth properties for nonhuman-primates SOEs (Martin, et al1988) were very similar to those recorded in humans.

Martin, et al (1988) reported that "both cochleas of Rhesus monkeys were exhibiting stable spontaneous and stimulus-frequency emissions over 1-year period". "The amplitude and frequency of both SOAEs and stimulus frequency emissions (SFEs) were routinely recorded, while transionally evoked (EOE) and dist-product emissions (DPEs) at the frequency 2f1-f2 were examined occasionally . Between evaluation sessions, both the frequencies and amplitudes of SFEs remained relatively stable in both ears, while the frequencies and amplitude of SOAEs were less constant. Isossupression contours of SOAEs as a function of frequencies and level of tonal maskers, revealed sharp tuning consistent with normal frequency selectivity" (Martin, et al 1988).

In another study by the same authors, both cochleas of a Rhesus monkey exhibiting stable spontaneous and stimulus-

frequency emissions were evaluated histologically; in the emitting-monkey's cochlea, "there was a mild loss of sensory cells scattered throughout the organ of Corti, which was generally greater for OHCs. The striking observation which varied in degree across the emitting monkeys was a generalized irregularity in the cellular organization of the OHC region; it was most pronounced in the low- and mid-frequency regions of the organ of Corti" (Martin, et al, 1990).

Although the incidence of SOAEs in humans is considerably greater than in nonhuman primates, the incidence of these emissions in monkeys is still much higher than that reported for other common laboratory animals (Evans, et al, 1981; Zurek and Clark, 1981; Ruggero, et al, 1984; Clark, et al,1984). "Based on the incidence findings of investigations that had been done, and the high degree of similarity in the parametric features of SOAEs in humans and monkeys, monkeys appear to be uniquely qualified to serve as an animal model for human SOAEs" (Martin and Martin, 1988).

Based on the forementioned studies where the Rhesus monkey is considered one of the best laboratory animals for the study of SOAEs, the main aim of this experiment is to evaluate the incidence and charecteristics of emissions in a different species of monkeys (Squirrel monkey).

EXPERIMENT FOUR

MATERIAL AND METHODS

METHODS

Subjects

The subjects were chosen from the colony in Prof. Stephen Highstein Laboratory, in the Neuroscience Department at Washington University. Fourty one squirrel monkeys, 12 females and 29 males, were used. Ninteen monkeys were excluded, because they were either carrying a baby or pregnant. Five animals were born in the laboratory; their ages ranged between 3 and 5 yearrs. In spite of the lack of knowledge of the exact ages of the rest of the monkeys as they were caught in wild, they were considered mature (more than 6 years of age).

Measurements

Equipment

The same instruments that were used in detecting and measuring the SOAEs in chinchillas in the previous experiments were used in this experiment. The probe -tube microphone system was used to detect acoustic signals in the ear canal; the microphone was previously described in a study of human ears (Zurek, 1981). It consists of an ear -piece from an otoadmittance meter (Grason-Stradler 1720) with a miniature microphone (Knowles Electronics, E A 1842) coupled by plastic tubing to the distal end of one of three

tubes. The probe-tube microphone was calibrated in a sound field with a standard laboratory microphone (Bruel and Kjaer, 4131). This probe- tube microphone was connected to a sensitive wave analyzer (Hewlett-Packard 3581A) to scan the amplitude spectrum of the microphone signals, that was displayed on an *x-y* recorder (Bruel and Kjaer, 2308).

Anesthesia

We used light anesthesia to prevent any side effects or permanent effects on the animals. We first used Ketamine Hydrochloride 0.25 ml intramusculary and Atropine 0.25 ml intramusculary. Then we used Azopromazine in addition to the previous two drugs because ketamine was light and had a short duration. We tried Rompun 0.2 ml intramuscularly (20mg/ml) and Nembitol (Sodium pentobarbitol) 15 mg /kg body weight intraperitoneally.

Procedure

Masks and gloves were used to prevent the possibility of cross infection. Each animal received its anesthesia in the colony and was then transferred to the sound-isolation chamber. The monkey was placed on a table in the chamber with a light directed to the ear, and the outer ear canal was checked to exclude the presence of any possible infection. The probe was then carefully inserted into the ear canal. SOAEs were recorded on the wave analyzer and plotted.

Each monkey was carefully checked several times in a set for test-retest reliability, the background noise was measured, any artefact was carefully excluded, After screening all the monkeys, the emitting monkeys were remeasured by the same procedure.

EXPERIMENT FOUR

RESULTS

Results

The survey of the 41 squirrel monkeys at Washington University's Department of Neurobiology laboratory showed that spontaneous otoacoustic emissions were detected in 9 monkeys (12.95%) and 10 ears (21.20%). One month later, the monkeys were retested. However two out of the 9 emitting monkeys had been sacrificed in another study (Neuroscience experiments) and so were lost in this study. Spontaneous otacoustic emissions could not be detected in one of the previously emitting ears. This brought the total of emitting ears to 7 out of 78 ears (8.97%). No changes were found on the third trial.

The emissions were continuous narrow-band acoustic signals emanating from the ear (fig. 32 & 33). Table 2 summarizes the emmitting monkeys, the side (left or right) and the frequency and amplitude of emissions. There was considerable variability in the amplitude and frequency of the emissions. These signals were found in the range of 1-5 KHz aproximately, with the intensities ranging from 31 to 14 dB SPL above the noise floor level. There were also slight frequency and amplitude shifts on the day of examination--5 Hz and 2 dB SPL respectively.

Table 2: this table summarises the monkeys that had SOAE and the side, frequency and the amplitude of these emissions.

Animal No	Ear	Frequency	Amplitude
1. 253	Rt	2050 Hz	31 dB SPL
2. M 90	1-Rt	2200 Hz	24 dB SPL
	2-Rt	2225 Hz	24 dB SPL
3. 401	Lt	2575 Hz	14 dB SPL
4. 255	Lt	1125 Hz	21 dB SPL
5. 1137	Rt	2625 Hz	21 dB SPL
6. 556	1.Rt	975 Hz	27 dB SPL
	2.Rt	2562 Hz	22 dB SPL
7. 0.16	Lt	5135 Hz	19 dB SPL

A light anesthetic (Ketamine Sulphate) was used. One of the animals showed a sensitivity reaction in the form of red rashes on his face, but no other complications were observed. One of the drawbacks of ketamine sulphate is its short duration of action, so it was not easy to record the emissions from the adult monkeys with kitamine alone, especially when they were injected in the colony (one floor away from the test room), and carried to the booth. Azopromazine and sometimes Rompon were used instead of Ketamine, because of its longer duration of action. No sensitivity reactions were detected.

It was obvious that the spectral analysis of the recording was affected by the type of the anesthetic used. With kitamine, the spectral analysis of the recording depicted higher background noise and the regular oscillation in the inner ear. When Rompun or Nambitol was used, the spectral analysis depicted lower background noise and no recording of any oscillations (figs.34 & 35). This difference was probably due to the effect of generation or propagation.

fig. 32

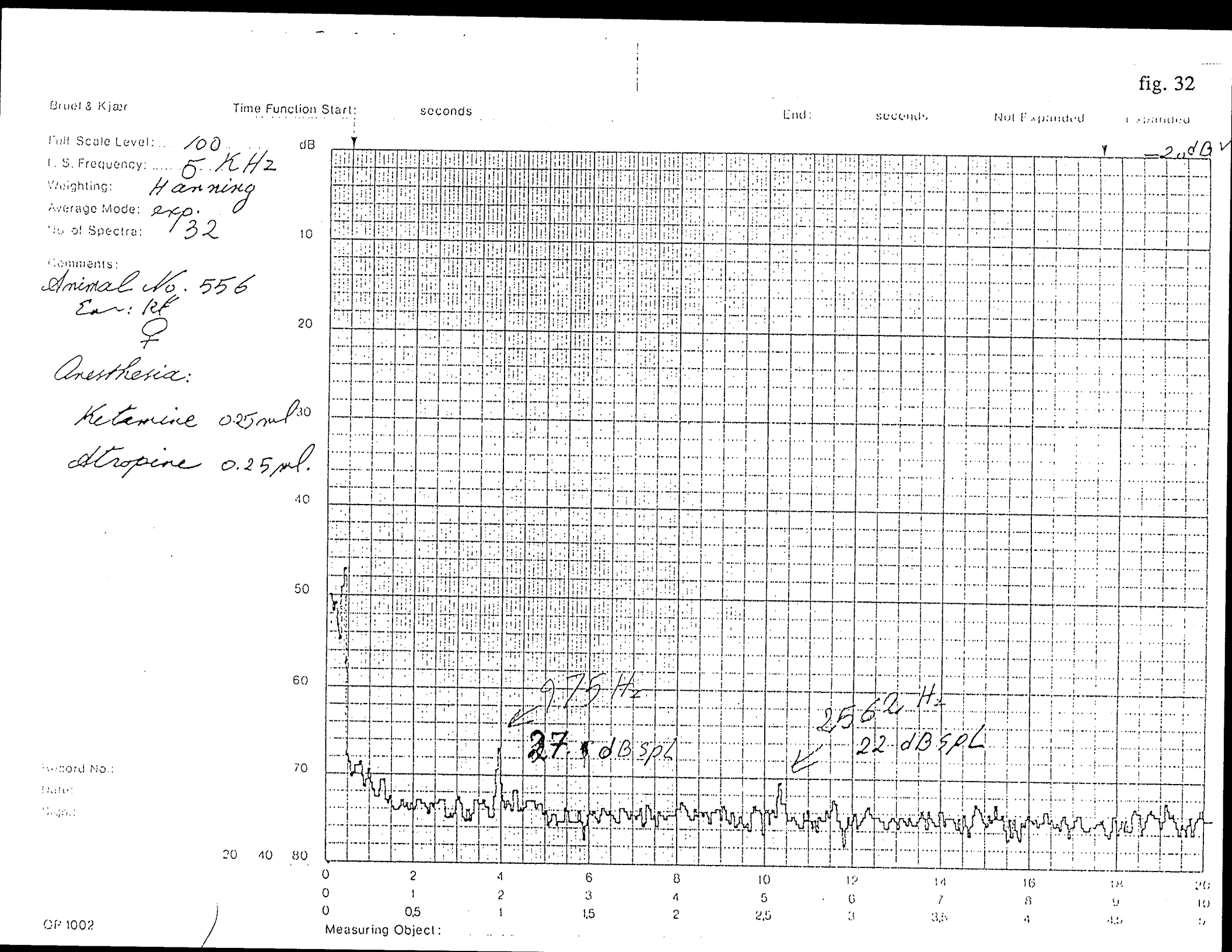
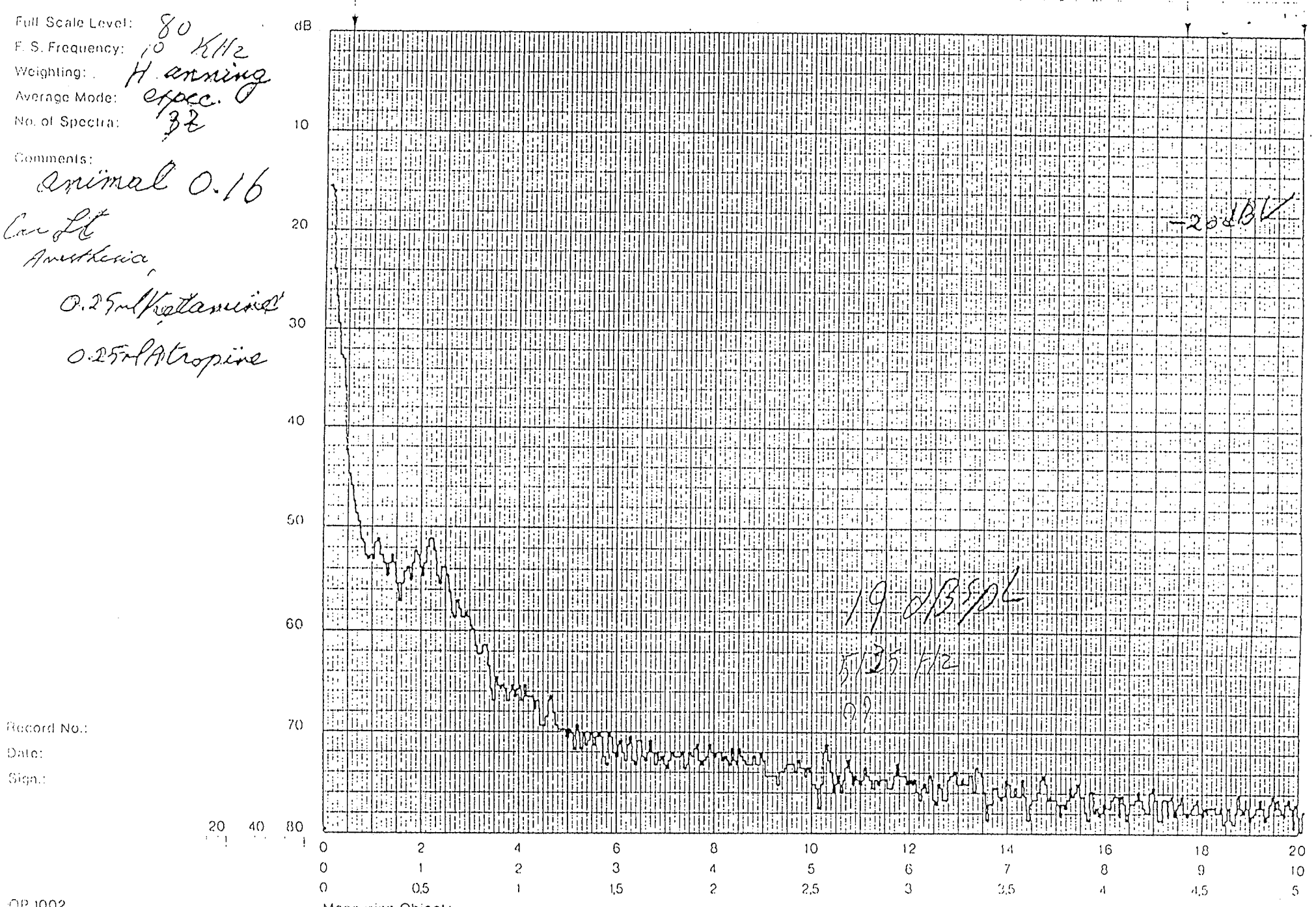

Brüel & Kjær
Time Function Start: seconds
End: seconds
Not Expanded
Expanded
Full Scale Level: 100
F. S. Frequency: 5 KHz
Weighting: Hanning
Average Mode: exp.
No. of Spectra: 32
Comments:
Animal No. 556
Ear: Rt
♀
Anesthesia:
Ketamine 0.25 ml
Atropine 0.25 ml.
dB
10
20
30
40
50
60
70
80
-2 dB
2.75 Hz
27.5 dB SPL
2562 Hz
22 dB SPL
Record No.:
Date:
Sign.:
20 40 80
0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0,5 1 1,5 2 2,5 3 3,5 4 4,5 5
Measuring Object:
QP 1002

fig. 33


Brüel & Kjær
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End: seconds
Not Expanded:
Expanded:
Full Scale Level: 80 dB
F. S. Frequency: 10 kHz
Weighting: Hanning
Average Mode: exp.c. 0
No. of Spectra: 32
Comments:
animal 0.16
Cannula
Anesthesia,
0.25ml Ketamine
0.25ml Atropine
-20dB
19dB
5.35 kHz
Record No.:
Date:
Sign.:
dB
10
20
30
40
50
60
70
20 40 80
0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0.5 1 1.5 2 2.5 3 3.5 4 4.5 5
QP 1002
Measuring Object:

fig. 34

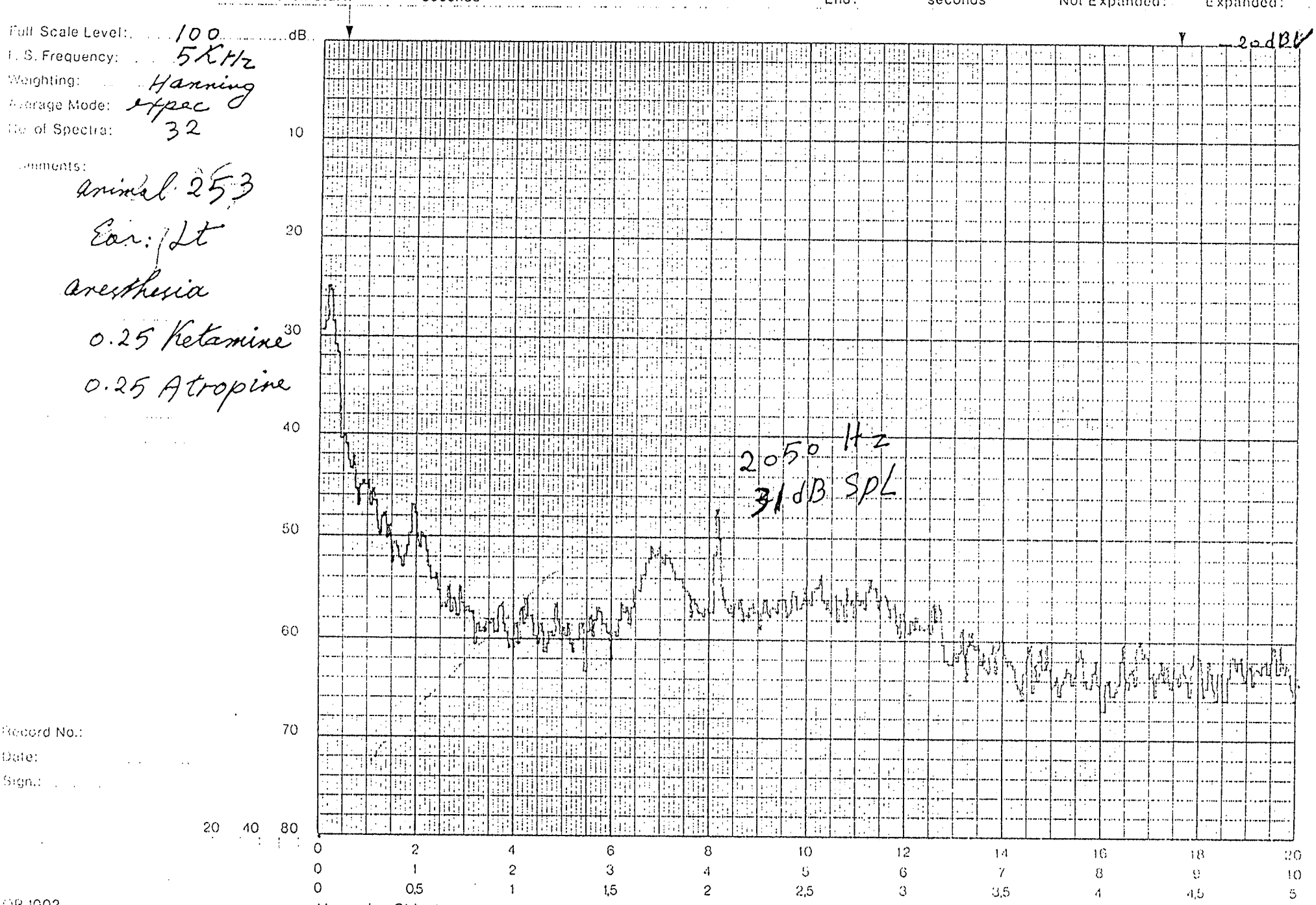
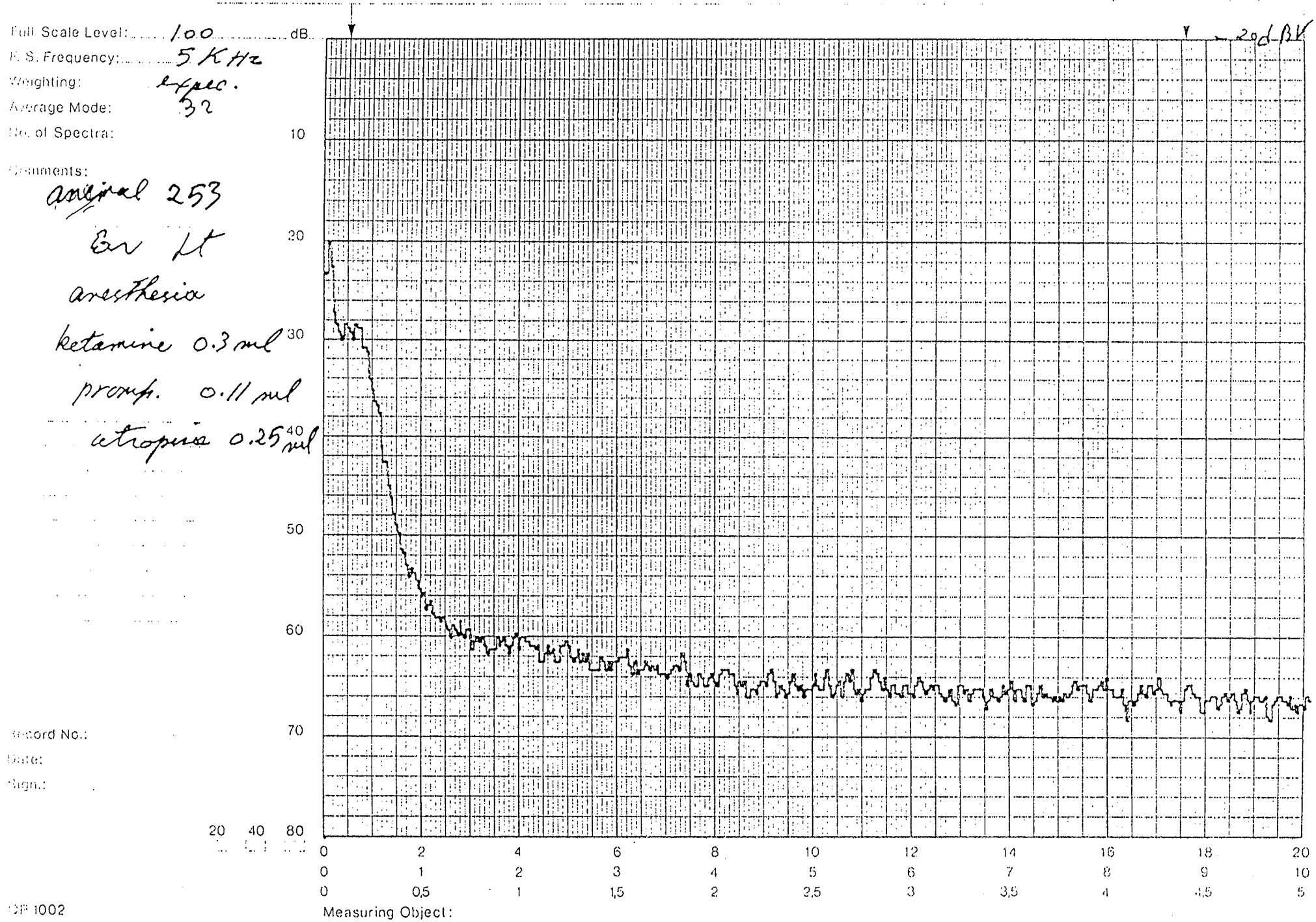

Brüel & Kjær
Time Function Start: seconds
End: seconds
Not Expanded:
Expanded:
Full Scale Level: 100 dB
F. S. Frequency: 5KHz
Weighting: Hanning
Average Mode: spec
No. of Spectra: 32
Comments:
animal 253
Ear: Lt
anesthesia
0.25 Ketamine
0.25 Atropine
20dBV
2050 Hz
31 dB SPL
10
20
30
40
50
60
70
80
Record No.:
Date:
Sign.:
20 40 80
0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0,5 1 1,5 2 2,5 3 3,5 4 4,5 5
SP 1002
Measuring Object:

fig. 35


Brüel & Kjær
Time Function Start: seconds
End: seconds
Not Expanded:
Expanded:
Full Scale Level: 100 dB
F. S. Frequency: 5 KHz
Weighting: l-spec.
Average Mode: 32
No. of Spectra:
Comments:
original 253
Ear Lt
anesthesia
ketamine 0.3 ml
promp. 0.11 ml
atropine 0.25 ml
Record No.:
Date:
Sign.:
20 dB
10
20
30
40
50
60
70
80
20 40
0 2 4 6 8 10 12 14 16 18 20
0 1 2 3 4 5 6 7 8 9 10
0 0,5 1 1,5 2 2,5 3 3,5 4 4,5 5
Measuring Object:
DP 1002

EXPERIMENT FOUR

DISCUSSION

DISCUSSION

The purpose of the fourth experiment was to determine the incidence of SOAEs in a population of nonhuman primate ears representing a variety of species closely related to man, other than chinchillas. The survey of the 41 squirrel monkeys at Washington University's Department of Neurobiology laboratory showed that spontaneous otoacoustic emissions were detected in 9 monkeys (21.95%) and 10 ears (12.20%). SOAE signals were found in the range of 1-6 KHz, with intensities ranging from 5 to 30 dB SPL above the noise floor level. In studying 122 nonhuman primate ears (Martin; Lonsbury-Martin & Probst 1985), multiple SOAEs were recorded from three ears of different monkeys. Thus SOAEs were observed in 2.5% of the ears and 5 % of the monkeys (the subjects were macaques, baboons, squirrel and Rhesus monkeys). The emissions recorded from the monkeys were associated with lower frequencies (1.4-4.9 KHz) and higher amplitude (2-15 dB SPL). In a further survey among 102 pigtail monkeys, twenty-six SOAEs were detected in 12 ears of nine monkeys which represented 9% of the subjects and 6% of the ears tested. Spontaneous emissions were recorded bilaterally from three primates and unilaterally from either the right or the left ear. SOAEs measured in that study tended to extend both in the upper frequency range by an octave to 8 KHz and the lower-amplitude range by about 13 dB to -11 dB SPL (Martin, Lonsbury-Martin & Probst,1988).

In the present study the higher incidence of SOAEs in squirrel monkeys than in chinchillas supports the idea that several of the characteristics of SOAEs emitting from monkeys resemble human emissions more closely than those of other species such as chinchilla (Martin, Lonsbury-Martin & Probst,1985). The incidence in monkeys was higher also than the results in the other two studies (Martin, Lonsbury-Martin & Probst,1985 & 1988), and the amplitude of the SOAE reached 30 dB in one case in our study, while it did not reach more than 11 dB in the other studies. In general the present study is in agreement with the previous two studies which concluded that the incidence of SOAE in monkeys is greater than in chinchillas and close to the incidence in humans.

In the fourth experiment, SOAEs could not be detected from the emitting monkeys when we used Nambitol or Rompun for anesthesia, and we could detect SOAEs when we used Ketamine sulphate. It was known that Ketamine sulphate causes what has been described as dissociative anesthesia, because during induction the recipient feels dissociated from his environment. Analgesia and amnesia are conspicuous but muscular relaxation is poor. On the other hand Nambitol and Rompun have different pharmacological effects. Barbiturates (Nambitol) reversibly depress the activity of all excitable tissues. Xylazine works as sedative analgesic as well as a muscle relaxant. Its sedative and analgesic activity is related to the central nervous system, and its muscle relaxant effect is based on inhibition of the intraneural transmission of impulses of the central nervous system (Goodman and Gilman, 1975). There is a

correspondence between the hypothesis that SOAE is caused by contraction of the outer hair cell muscles and the observation in the present study that when the anesthetic affected the central nervous system, we could not detect SOAE.

VII. SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

In the present study, SOAEs were detected in chinchillas that had been exposed to noise but not in those that were living in a quiet environment. The frequencies of these emissions were in the same range of frequencies at which hearing loss was detected in the subsequent chinchillas.

External tones had a suppressor effect on the SOAEs. While the suppression due to the ipsilateral tone was minimal at the start, followed by a sudden increase at a critical level, the contralateral external tone suppressed the SOAEs gradually except for those with frequencies very close to that of the emissions where they still showed this sudden pattern of suppression.

A frequency and amplitude shift was noted due to external tones (ipsi & contralateral) application. The increase in shift of the SOAE frequency was directly proportional to the increase in the frequency of the external tone.

It was also found in this study that the incidence of SOAEs is higher in monkeys than in chinchillas. Regarding the effect of anesthesia on SOAEs, anesthetic drugs that had a temporary effect on the central nervous system abolished the SOAEs, while weak muscle relaxants had no effect on the emissions. This observation supports the idea that there is a central nervous system effect on the SOAEs.

The question now is: " Are SOAEs central (cerebral cortex) or peripheral (cochlea) in origin?

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