EFFECT OF CONTRALATERAL ACOUSTIC STIMULATION ON SOAEs

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DECLARATION

I hereby declare that the Dissertation entitled **"Effect of Contralateral Acoustic Stimulation on SOAEs"** *is the result of my own study under the guidance of Mrs.* C.S.Vanaja, *Lecturer, Department of Audiology, All India Institute of Speech and Hearing, Mysore and has not been submitted earlier at any University for any other Diploma or degree.*

Mysore
May 1999

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CERTIFICA TE

This is to certify that this dissertation entitled **"Effect of Contralateral Acoustic Stimulation on SOAEs"** *is the bonafide work in part fulfilment for the degree of "MASTER OF SCIENCE (SPEECH AND HEARING) " of the student with Register Number M 9701.*

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This is to certify that this dissertation entitled **"Effect of** Contralateral Acoustic Stimulation on SOAEs" *has been prepared under my supervision and guidance.*

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CONTENTS

INTRODUCTION

In humans, the role of ear is extremely important. It is one of the most important links in the speech chain, which enables proper communication. Such enhanced communication skills, as seen in humans, have a momentous role in the existent structure of human society, as society is dependent upon verbal communication.

Since time immemorial, the ear has been recognized as the organ of hearing. However, only twenty years ago has it been demonstrated that the ear besides receiving sounds, also produces sounds [Kemp, 1978]. Although these sounds are of very low intensity, they are loud enough to be reliably measured by instruments specifically designed for the purpose. These sounds emitted by the ear are called Otoacoustic emissions (OAEs).

From the outset OAEs have caused excitement and elicited skepticism. Their paradoxical relation to hearing process and the sheer novelty of cell based sound generation has stimulated research. For those who had studied and admired the workings of human cochlea from a far through various audiological test and psychoacoustics, OAEs provide a direct means of communication with the sensory cells and hence the metabolic processes previously available only to the laboratory physiologist. For those scientists engaged with the physics and engineering of man made signal detecting and processing systems, OAEs provide an opportunity to contribute to hearing research and clinical audiology in a novel way that the first auditory biophysicists would have found hard to believe. Therefore, the discovery of OAE has led to a deluge of studies. Probst et al (1991) in their extensive review on otoacoustic emissions have classified OAEs as follows:

1) Spontaneous otoacoustic emissions (SOAEs)

Those which are emitted spontaneously from the ear, in the absence of any external stimuli. These are seen in about 50% of the ears with normal hearing [Martin et al 1987].

2) Evoked otoacoustic emissions (EOAEs)

These are elicited in response to certain external stimuli. These may be of the following three types -

a) Transient evoked otoacoustic emissions (TEOAE): Those which are emitted in response to a brief acoustic external stimuli. This is seen in about 98% of the ears with normal hearing [Dijk & Wit, 1987].

b) Distortion product otoacoustic emissions (DPOAEs) : These are emitted in response to two simultaneously presented pure tones. These are characteristically found to occur at specific frequencies. These are found to occur in all ears with hearing acquity levels within 25 - 30 dBHL [Lonsbury-Martin, & Martin, 1990].

c) Stimulus frequency otoacoustic emissions (SFOAEs) : These are emitted in response to a continuous tone of a specific frequency. The emission resembles the stimulus in terms of the frequency.

The OAE as a clinical tool, provides several advantages. First, the test is objective in nature, and does not require patient co-operation for it to be administered. Thus it may be conveniently used for measuring the hearing acquity of young children including neonates. Unlike BSERA it does not require cumbersome procedure such as electrode placement and measurement of impedance at the electrodes. Nor does it require an air tight seal as in the case with tympanometry. While the time taken to test a patient using OAE varies with the exact procedure used for measurements, it is shorter than that required for BSERA measurements but longer than that required for tympanometric measurements.

The first major class of OAEs the spontaneous emissions represent narrow band acoustic signals generated naturally within the cochlea, by definition, are measured in the ear canal in the absence of external stimulation. The presence of SOAEs was theoretically predicted by Gold (1948). Case reports of "objective tinnitus", probably a special form of SOAEs, have been mentioned sporadically in the clinical literature since the first published report appeared in 1962 [Loebell, 1962]. The first spectral analysis of an SOAE was probably described in 1970 by Kumpf and Hoke. However, Kemp (1978) can be credited with the initial discovery of SOAEs in clinically normal ears and Zurek (1981) performed the first systematic study that examined a large series of ears with SOAEs. Bialek and Wit (1984) provided evidence that, at least, some extremely narrow band SOAEs are generated by synchronized driving forces, and not thermal noise. As a rule, such emissions can be recorded repeatedly with relatively minor frequency changes, over long periods of time involving months or years. However, it is quite common for the amplitudes of SOAEs to vary substantially both within and between recording sessions The origin of all types of OAEs is belived to be the hair cells, specifically the outer hair cells (OHCs) [Probst et al, 1991]. Many pathologies causing hearing loss, such as noise induced hearing loss (NIHL), ototoxicity etc. are known to selectively damage the OHCs. In these cases OAEs have shown to indicate the severity of damage of OHCs directly (Wier Pasanen & McFadden, 1988).

With the discovery of OAE and the involvement of OHCs in their generation interest has shifted to the examination of emissions with electrical and contralateral acoustic stimulation. It is assumed that the efferent fibres from the medial part of the superior olivary complex terminate as large vesicle filled endings on the bases of OHCs [Brown, 1988]. Many researchers opine that electrical stimulation of the contralateral olivocochlear bundle (COCB) raises

the acoustic thresholds of auditory nerve fibres [Galambos, 1956; Widcrhold & Kiang, 1970]. The mechanism by which the COCB controls the IHCs and hence afferent fibre reponse is thought to rely on biochemical response of the OHCs which are known to be motile and may actively modulate the local mechanical response of the basilar membrane. The OAE represent an objective measurement of the active micromechanical function of the OHCs. Several investigators have demonstrated suppressive effect of contralateral acoustic stimulation (CAS) on spontaneous, transient and distortion product otoacoustic emmission [Collet et al, 1990; Puel & Rebillard, 1990; Veuillet et al, 1991; Berlin et al, 1993a; Chery Croze et al, 1993; Norman & Thornton, 1993).

Need for the study :

Many investigators have studied the effect of contralateral acoustic stimulation on otoacoustic emission. However, the data available in relation with SOAE is limited and very few studies have been documented in the literature demonstrating the effect of contralateral acoustic stimulation on the frequency and amplitude characteristics of SOAE. Also not much is known about the frequency selectivity of the medial efferent system and its contralateral suppressive effect on SOAE, till-date.

Purpose of the study :

The study was taken up with the aim of achieving the following purposes.

a) To examine the effect of contralateral acoustic stimulation on the amplitude and frequency characteristics of SOAEs.

b) To determine if the influence of CAS vary with increase in intensity of wide band noise.

c) To investigate the frequency selectivity of the medial efferent system :

(i) To analyze if the narrow band noise (centred at standard audiometric frequencies across the frequency range of 1 - 8 KHz) has differential effect on the SOAEs.

(ii) To study the effect of increase in intensity of contralatcral NBN presentation across various frequencies on SOAEs.

REVIEW OF LITERATURE

OAEs are defined as "sounds generated within the cochlea, by the outer hair cells, which can be detected at the tympanic membrane" [Norton & Stoves, 1994]

While the OAEs are a recent discovery, it was as early as 1948, when Gold proposed that a purely passive basilar membrane filtering action was not sharp enough for frequency selectivity. He further suggested that an active biomechanical cochlear feedback was responsible for sharp frequency selectivity. This was the first suggestion towards explaining that the ear was not merely a passive organ.

However, the discovery of OAE is attributed to Kemp, at the Institute of Laryngology & Otology, London in 1978. He reported that on presenting brief broad spectrum sound stimuli to the ear, the ear emitted another sound of similar spectra but of very small intensity. Initially these were thought to be the echos of the stimulus which were labelled as "Kemp's Echos". However, over the years, it has been confirmed that these sounds are not echos but sounds emitted from the ear. These confirmations have come after studying the latency of the reflected sound and the emissions.

The source of the OAE is believed to be the OHC, which were demonstrated to be electromotile [Brownell, 1985]. Later, OHC were demonstrated to be motile to pharamacologic stimulation (Slepecky et al, 1980) and also to acoustic stimulation [Drown, 1988].

The cause of such motility of the OHC was earlier believed to be due to the presence of actin and myosin in the stercocilia [Tilney et al, 1980]. However, it has now been established that the motility of the OHC is due to volume changes in the OHC by the movement of ions and not due to actin and myosin fibres [Wilson et al, 1980].

OHCs contract and elongate at rates which no muscle fibre is capable of. This has been confirmed by studying the changes in OHC following electrical stimulation [Ashmore, 1986]. Furthermore, OHC movement occur as a direct conversion of electrical potential energy to mechanical energy. This was demonstrated by the presence of OHC movements even after the depletion of cellular stores of "Adenosine Triphosphate" (ATP) [Brown, 1988]. Thus, it has been suggested that if OHC length changes are rapid enough, they may be responsible for the OAEs [Davis, 1983; Schloth & Zwicker, 1983].

The mammalian cochlea receives efferent innervation from both the ipsilateral and contralateral superior olivary complex (SOC). These descending fiber tracts, known as the olivocochlear bundle (OCB), represents the final link of a chain of neurons from the Cortex to the cochlea [Desmedt, 1975]. The OCB is composed of two separate systems. The medial olivocochlear (MOC) projections primarily to the outer hair cells (OHCs) and the lateral olivocochlear (LOC) projections primarly to the inner hair cells (IHCs) [Warr & Guinan, 1979].

In contrast to the well known function of the afferent system (viz., conveying auditory information to the central nervous sytem), the function of the efferent system is not well defined. One hypothesis of the function of the OCB is that it controls the mechanical state of the cochlea and resulting synaptic transmission to afferent terminals [Klinke & Galley, 1974]]. Since the studies by Buno (1978) and Murata et al (1980), it has been agreed that acoustic stimulation of one cochlea can alter afferent nerve fibre response in contralateral cochlea. [Folsom & Owsley, 1987].

It has been demonstrated in several studies that electrical stimulation of the OCB in animals also affects cochlear mechanics, specifically the otoacoustic emissions [Guinan, 1986; Mountain, 1980; Siegel & Kim, 1982a].

OAEs are believed to be generated by "active" mechanisms in cochlea which involve OHCs. Since OHCs receive direct efferent innervation they may be affected by contralateral acoustic stimulation of the olivocochlear system [Kim, 1986].

Contralateral suppression of OAEs:

There are several ways of measuring contralateral stimulus effects. One can simply use a contralateral auditory stimulus to examine the various OAE amplitudes. Several investigators have studied the effect of contralateral acoustic stimulation using, spontaneous otoacoustic emissions (SOAE) [Mott et al 1989; Moulin, 1993, Harrison & Burns, 1993], transient evoked otoacoustic emissions (TEOAE) for linear clicks [Collet et al 1990, 1992a; Veuillet et al, 1991; Berlin et al, 1993a,b], TEOAE for tone pips [Berlin et al, 1993b] and acoustic distortion products (DP) {Moulin et al, 1992, 1993, Cherry Croze et al, 1993]. All these studies have recorded OAEs in one ear in the presence and in the absence of contralateral acoustic stimulation (CAS).

Either a pure tone {Mott et al, 1989, Berlin et al 1993a; Harrison & burns, 1993], clicks [Veuillet et al, 1991]; narrow band noise [Veiullet et al, 1991; Cherry Croze et al, 1993] or broad band noise [Collet et al, 1990; 1992a; Veuillet et al, 1991, 1992; Berlin et al., 1993a; Moulin et al, 1993; Harrison & Burns, 1993] has been used for contralateral acoustic stimulation.

All these studies have recorded OAEs in one ear in the presence and absence of contralateral acoustic stimulation. This however, would fail to take inter-subject variation into account: a 1 dB decrease in a subject with 5 dB OAEs probably does not have the same significance as in the case of a subject with 15 dBOAE. Collet et al, (1992a) suggested of measuring equivalent attenuation to escape baseline OAE values. It involves tracing an input/output curve with and without 30 dBSL while noise contralateral stimulation at each

input intensity level of primaries and the equivalent attenuation is calculated. Here ngnin, there is considerable variation but about 80% of subjects have equivalent attenuation greater than 1 dB in absolute value terms.

Influence of efferent stimulation on DPOAE :

The relative simplicity of DPOAE measurement in many laboratory animals and their noninvasive characteristics has led to the use of DPOAEs in studies investigating the influences of a number of phenomena on cochlear function including stimulation of the efferent system. The influence of electrical stimulation of the crossed cochlear efferent fibres on DPOAEs was reported first by Mountain (1980) and Siegel & Kim (1982a, b). Mountain (1980) detected a reduction in DPOAE amplitude at F_2 - F_1 in the guinea pig that reached values of about 70% and was most pronounced in response to low level stimuli. In Chinchillas, Siegel & Kim (1982b) uncovered more complex changes in their study of the distortion products at F_2 - F_1 and $2F_1$ - F_2. Specifically acoustic distortion products were reduced, enhanced or unchanged during electrical stimulation of the crossed efferent fibres. No systematic relationship of these changes to stimulus level, stimulus frequency, or to distortion products at F_2 - F_1 or $2F_1$ - F_2 could be distinguished by these workers.

Recent evidence suggests that the earlier findings were obtained under quite complex conditions. For example, anesthesia was demonstrated to have major effect by itself on the DPOAE at F_2 - F_1 [Brown, 1988], an effect that may be mediated by efferent innervation. Additionally the levels of electrical stimulation used in the early experiments were probably unphysiologic[Kemp & Souter, 1988]. Nevertheless, the initial findings of the effect of efferent stimulation on DPOAE amplitude provided early evidence that the OHCs, as the primary sensory cells innervated by the cochlear efferent system, were linked to the generation of OAEs. More recent evidence

by Puel & Rebillard (1990) demonstrated reductions in DPOAE magnitudes produced by contralateral white noise stimulation in guinea pigs, with surgically sectioned middle ear muscles. That the contralateral stimulation induced decrements in DPOAE amplitude disappeared after the crossed medial efferent system was sectioned at the midline of the brainstem supported the notion that the physiological action of the medial olivocochlear efferents is to modulate cochlear nonlinearities [Puel & Rebillard, 1990]. Other detailed results about the action of efferent innervation on OAE generation were also reported by Guinan (1986) and Kemp & Souter (1988). However, these investigators used SFOAEs and not DPOAEs to monitor the contribution made by the cochlear efferent system to emission generation.

The results of studies performed by different investigators indicate that the CAS suppresses the amplitude of DPOAE. The high frequency (as in comparision with the DP frequency demonstrated maximum suppressive effect.

Influence of efferent stimulation on TEOAE:

The effect of contralateral acoustic stimulation on TEOAE amplitudes have been evaluated as a means to assess the influence of the cochlear medial efferent system on OAE generation. An earlier study by Puel et al (1988) reported that TEOAEs were reduced reliably by about ldB when human subjects attended selectively to a visual task. Puel et al (1988) interpreted these findings to indicate that cochlear microphonics could be modulated by the medial olivocochlear efferent pathway which appeared to be involved in selective attention. In a follow up study, Froehlich et al (1990) comparing the influence of contralateral white noise on TEOAEs in the presence and absence of the same visual task documented the reliability of the reduced TEOAEs, but only in a subset of subjects (-20%) that was much smaller than the number (81%) originally reported by Puel et al

(1988). Froehlich et al (1990) concluded that the task related decline in TEOAE amplitudes observed in both studies was due simply to nonspecific alterations in the general arousal level of the subjects rather than to a modality selective effect on TEOAE generation. Thus the role of selective attention on the generation of OAEs through neural activity mediated by the medial efferent system remains unresolved.

The result of related work, however, indicate that contralateral broad band white noise significantly reduced the amplitudes of ipsilaterally tested TEOAEs by about 1 - 4dB (Collet et al, 1990; Froehlich et al, 1990; Ryan et al, 1991; Morlet et al, 1993; Berlin et al, 1993b).

Norman & Thornton (1993) attempted to study the frequency analysis of the contralateral suppression of EOAE by narrow band noise. Analysis of the emissions showed that the 40 dBSL contralateral noise did not produce a significant amount of suppression.. The suppression produced by 60 dBSL noise was spread throughout the frequency range of emissions with limited frequency specificity. But there was some evidence that the amount of suppression increases with the bandwidth of the noise, particularly for noises centred on 1 and 2 Khz; the wide band noise produced much greater suppression than any of the narrow bands.

Thus by careful control of the potentially contaminating influence of the acoustic reflex and cross over of the contralateral stimulus to the test ear by either bone or air conduction, these investigators concluded that the observed decrement in otoacoustic emissions was due to the actions of the cochlear medial efferent system. The contralateral stimulation experiments establish an important model that can be used to explore the function(s) of the cochlear efferent system in humans.

Suppression & SOAEs :

Spontaneous emissions represent an ideal means for the study of response - suppression contours and many investigators have performed suppression experiments using SOAEs. Whereas the typical SOAE - suppression tuning curves are similar to the well known psychophysical tuning curves, the following characteristics can be summarized from the relevant experimental findings. First, the most effective suppression of an SOAE is obtained at a frequency slightly higher than the emission frequency [Wilson, 1980; Wilson and Sutton 1981; Zurek, 1981; Schloth, 1982; Ruggero et al, 1983; Dallmayr, 1985; Rebillard et al, 1987; Bargones and Burns, 1988; Zizz & Glattke, 1988]. Second, the growth of suppression increases more rapidly in response to suppression tones with frequencies lower than the SOAE compared to those with higher frequencies [Zurek, 1981; Schloth, 1982; Rainbowitz & Widin, 1984]. Finally the high frequency component of the suppression contour is often non monotonic in that additional minima are present [Zurek, 1981; Dall mayr, 1985; Bargones & Burns, 1988; Ziza and Glattke, 1988].

Similarly, the influence of contralateral acoustic stimulation on SOAE properties is equivocal whereas most investigators have detected effects that can be explained by the stapedial reflex or acoustic crossover from the opposite ear [Schloth & Zwicker, 1983; Rabinowitz and Widin, 1984]. Mott et al (1989) concluded that they induced alteration in SOAE frequency with contralateral stimulation that was independent of these contaminating factors. They also reported tuning in three of four subjects, such that contralateral tones 3/8 to ½ octave below the SOAE frequency induced greater changes than tones higher or lower in frequency, they also found small, but variable, changes in SOAE amplitude. Long & Tubis (1988) reported similar results in a single subject with an elevated acoustic reflex threshold. They proposed that alterations n OAEs are mediated by

synapses between olivocochlear fibres and OHCs. Moulin et al (1993) investigated the influence of SOAE on acoustic distortion product input/output functions in the presence of contralateral auditory stimulation. They found that as the primary levels for Dps increased, SOAE amplitude and frequency decreased and quickly disappeared into the noise floor. A similar result has been described for a single subject [Ciafrone & Mattia, 1986]. The contralateral broad band noise decreased SOAE amplitude and increased its frequency by 5-20Hz which is in agreement with findings. Harrison & Burns (1993) measured SOAEs prior to, during and following presentation of tonal & broadband stimuli to the contralateral ear. Frequency shifts were positive in all the subjects. But systematic changes in amplitude shifts were observed only for contralateral broad band noise stimulation whereas the effect for tonal stimuli was variable among the subjects.

From the above discussion it can be suggested that the primary effect of CAS on SOAE is interms of increase in SOAE frequency which is incontrast to the effect of CAS on the amplitude of evoked otoacoustic emission.

Several different mechanisms have been suggested for this contralateral suppression effect :

1) The middle ear stapedial reflex :

Tough & Kunov (1989) used stimuli of constant intensity in subject with varying degrees of sensorineural hearing loss in contralateral ear. They found suppression in distortion product emissions which showed a strong correlation with acoustic reflex thresholds and a lack of correlation with sensational level, suggesting that the stapadial reflex may be responsible for suppresion. Also early investigators attributed the effects obtained in these experiments to middle ear reflex [Schloth & Zwicker, 1983; Rabinowitz & Windin, 1984]. Burns et al (1993) have shown the same effects of middle ear

muscles on SOAE as the effects of contralateral acoustic stimulation. However, the discovery by Mott et al (1989) that the latency of frequency shifts in spontaneous emissions is less for intensities of contralateral tones just below the reflex threshold suggests that different mechanisms may be involved above & below the threshold.

It is now well established that this suppression effect can be obtained in subjects without acoustic reflex [Bells Palsy, Post surgical otosclerosis] [Veuillet et al, 1991; Berlin et al, 1993a; Moulin et al, 1993]. So presence of an acoustic reflex is not required to get this effect. Collet et al (1990) have documented suppression in a subject with no acoustic reflex, showing that this cannot be the only mechanism involved. The suppression effect is also absent in subjects with vestibular neurectomy [Williams et al, 1993]. It is likely that the medial olivochlear bundle is cut during vestibular neurectomy which is required to obtain suppression effect. Also Lind (1994) reported of a reduction of rms amplitude in the last 10.24 ms of the response onset latencies varied from less than 40 ms to 140 ms offset latency from 20 msec to 80 msec. Acoustic reflex latencies have not been reported with such short latencies and this makes it unlikely that this reflex can be responsible for the observed effect.

But one cannot exclude a double pathway in this effect [Harrison & Burns, 1993] including acoustic reflex and medial olivocochlear bundle, as some connections are suggested between the two pathways.

2) Interaural cross talk :

Emissions can also be suppressed by ipsilateral stimulation [Kemp & Chum, 1990]. However, Collet et al (1990) showed that a presentation of a contralateral sound at an intensity which produced a significant amount of suppression in normal subjects has no effect in a subject with a total unilateral hearing loss. Intensity levels of sound equivalent to the highest possible level of crossover sound directly into

the ipsilateral ear has been shown not to produce the same effect as the contralateral sound [Ryan et al, 1991]. Also TEOAEs have been recorded in the healthy ear with contralateral stimulation of the deaf ear. CAS by a white noise of 80 dBSPL also did not have any effect. Cross talk has thus been ruled out [Collet, 1993].

3) An efferent effect

CAS can either have a direct effect via the lateral efferent fibres which innervate cochlear afferent neurons near the inner hair cells, or an indirect effect via medial efferent fibres which innervate the outer hair cells. Puel & Rebillard (1990) conducted experiments on guinea pigs, sectioning the brain to interrrupt all the fibres crossing the brainstem midline. They found that distortion product emissions were unaffected by this but that contralateral suppression was stopped, which suggests that the afferent pathway projecting from the contralateral car to the ipsilateral olivary complex is involved. Berlin et al (1993b) found robust emissions but an absence of contralateral suppression in two patients with absent audiometric brainstem responses and middle ear muscle reflexes despite normal audiograms in the 2 KHz region, and suggested that these apparently paradoxical findings might be due to an auditory nervous sytem dysfunction which disrupts access to the efferent system. Prasher, Ryan & Luxon (1994) examined this effect in patients with extrinsic & intrinsic lesions of the brainstem which may affect the efferent pathway either within the vestibular nerve which carries the efferent bundle to the cochlea or within the brainstem at the level of the SOC. Suppression was reduced or absent in these patients and the site and size of the lesion determined whether the suppression was affected unilaterally or bilaterally. Lesions affecting the auditory afferent pathway without significant alteration in hearing appear to affect the efferent pathway too. It has also been demonstrated that a single injection of gentamycin in the awake guinea-pigs induce a transient blockade of the suppression by contralateral white noise of tone pips evoked

compound action potential [Smith et al, 1994; Aron, Erre, Avan, 1994] likely via the reversible blockade of calcium channels observed in vitro [Dulon et al, 1989].

Thus from the above discussion it is logical to assume that at least a part of the observed effect is due to the effect of medial efferent system on the outer hair cells. Apart from the effect of efferent system on the OHCs there are several other extraneous factors that affects the measurement of suppression.

Influence of maturation and aging :

In premature and full term neonates no inhibition effect of contralateral auditory stimuli on OAEs has been found, [Morlet et al, 1993]. In contrast Ryan (1994) have demonstrated a slight effect in full term babies. The exact age of onset is not yet known. Reduction is found in equivalent attenuation with aging, but the efferent system continues to function even in the aged, with a fall off which might, however, explain difficulties in hearing against spectrum noise [Castor et al, press - cited in Collet, 1993]

Influence of sleep :

Froehlich et al (1993) have shown it to be possible to record the suppressive effect from the human efferent system during sleep. At the onset of sleep, for about 15 minutes, in about 50% of the subjects.

Cochlear action site :

Veuillet (1992) have shown white noise contralateral stimuli to be less effective at EOAE frequencies around 4 Khz suggesting a more fragile cochlear area. At higher and lower frequencies, contralateral auditory stimulation reduces the other components.

Perceptual correlates :

The suppression effect can be with tone decay test results [Collet et al 1992b] and is different in musicians and non musician subjects [Michyel, in press - cited in Collet, 1993]. It is also correlated

with detection thresholds in noise measured with pure tone [Michyel & collet, 1993]. To date, the suppression effect does not appear to be correlated with frequency selectivity although relationships were observed between EOAEs and psychoacoustical tuning curves. [Michyel & Collet, in press - cited in Collet, 1993].

Clinical Applications :

a) Sensorineural hearing loss - Evoked otoacoustic emissions are found when best frequency hearing loss is less than or equal to 40 dBHL. [Collet et al, 1992b, 1993]. The medial efferent system can be explored in endocochlear deafness patients, on condition that they show EOAEs and that the contralateral hearing loss is not top great to rule out an interaural attenuation related to a 30 dBSL white noise. Equivalent attenuations similar to control values have been recorded [Collet et al, 1992a].

b) Noise induced hearing loss - EOAE intensity is greater in work related than in edocochlear deafness. These findings do not however seem to be related to the medial olivocochlear system as no difference is found in EOAE decrease under contralateral stimulation between the two populations [Collet et al, 1991].

c) Hyperacusis-otoacoustic emission decrease is found with contralatral stimulation, but not in certain cases of hyperacusis, where there has ever been noted as increase [Collet et al 1992a].

d) Otoneurological applications - In retro-cochlear hearing loss there seems to be little or no contralateral effect on EOAEs.

A fourth ventricle tumor case showing no contralateral auditory stimulus effect on EOAEs has also been described [Collet et al, 1992c].

e) Tinnitus - Veuillet et al (1992) showed that in cases of unilateral tinnitus, the medial cochlear efferent system is almost or completely non-functional in the affected ear as compared to the other Chery Croze et al (1993) have confirmed and even extended this findings,

further showing that in unilateral cases the efferent system is the least functional in the ear presenting tinnitus.

There would thus seem today to be three main fields of application of medial cochlear efferent system exploration in humans; hyperacusis, tinnitus and otoneurology. There is no contralateral auditory stimulus effect. But it is not known whether it is this due to a disconnection of the medial efferent system or to a technical middle ear effect ? During sleep, OAEs get constantly inhibited by the medial efferent system, irrespective of the sleep stage. It was found that throughout the night OAE amplitude increases and efferent influence decreases, but it is not established if this effect is a result of an absolute decrease, or simply due to the rise in OAEs over time ? The essential point however, is that the medial efferent system can indeed be recorded during sleep.

METHODOLOGY

This study was taken up with an aim of examining the effect of contralateral wide and narrow band noise presentation on SOAEs.

1) Subjects :

Comprised of 30 adult volunteers aged 17 to 28 years, both male and female (mean age = 20.5 years). All subjects had pure tone hearing threshold in the frequency range of 250 Hz to 8000 Hz, less than 20 dBHL. The immittance measurements revealed "A type" tympanograms and normal reflexes on screening. None of the volunteers reported of any history of ototoxic drug usage, noise exposure, tinnitus or giddiness.

2) Equipment used: The following equipment were used in the study.

(a) Pure tone Audiometer :

The Grason Stadler (GSI - 61) clinical audiometer was used with (i) TDH 50P headphones (calibrated as per ANSI - 1989) (ii) Radioear B-71 bone vibrator (calibrated as per ANSI - 1981) (iii) Eartone 3A insert receiver.

(b) Immittance Meter :

The Grason Stadler (GSI - 33) middle ear analyzer version - 2 (calibrated as per ANSI - 1969) was used to assess the middle ear function of the volunteers.

(c) SOAE Measuring System :

The SOAE measuring system was a computer based system. The software being used for the purpose was the Madsen Celesta 503 cochlear emission analyzer (programme version 3.00). Using a computer based instrument gives the OAE measurement immense flexibility.

3) Test Environment :

The tests were carried out in a quiet room. The test room had adequate lighting and comfortable temperature.

Test Procedure :

Initially all the volunteers were screened for their pure tone thresholds (frequency range of 0.25 to 8 kHz) using the GSI - 61 clinical audiometer. Further, using the GSI - 33 middle ear analyzer the immittance measurements were performed for all the subjects.

For the OAE measurement the subjects were made to sit on a chair comfortably and were not required to perform any task. Each ear was tested twice in order to confirm the incidence of SOAE. Only those subjects having one or more measurable SOAEs were included in the study.

An ABA research design was employed for evaluating the effect of contralateral stimulation on SOAEs.

Baseline (A) : This measurement was performed in order to determine baseline frequency and intensity values of SOAE. The SOAEs were analyzed across the frequency range of 0 - 10 kHz. Each recording consisted of 620 sweeps. The recording was acceptable only if the number of artifacts were less than 62 (which is 10% of the total number of averages taken).

Experiment (B) : This was further divided into two sub steps.

1) SOAE were measured in presence of the wide band noise (WBN) in the contralateral car using the cartonc 3A insert receiver of the GSI 61 audiometer. The recordings were performed with each intensity level (30,50 and 70 dBHL) of noise separately.

2) This step included the measurement of SOAE with the presentation of narrow band noise (NBN) in the

contralateral ear that were centred on six standard audiometric frequencies 1 kHz, 2 kHz, 3 kHz, 4 kHz, 6 kHz and 8 kHz. The effect of these noise bands presented through the eartone 3A insert receiver were tested separately at the intensity level of 30, 50 and 70 dBHL.

Intensity of the noise was chosen based on two factors (1) Intensity should be high enough to produce a significant amount of suppression. (2) Intensity should also be below the level at which significant interaural transmission of the sound or activation of the acoustic reflex could be expected.

Rebaseline (a) : After the experimental procedure was carried out the SOAEs were recorded without any presentation of noise in the contralateral ear. This was performed in order to check the test retest reliability of SOAE in terms of amplitude and frequency. The SOAE amplitude and frequency recorded during the experimental procedure was compared with that of the average of the two baselines in order to evaluate the effect of noise presentation in the contralateral ear.

RESULTS AND DISCUSSION

The examination of data obtained on performing SOAE measurements on thirty subjects resulted in forty three identifiable SOAEs. All the SOAEs obtained in the baseline and experimental conditions were analysed in terms of their amplitude (intensity) and emission frequency. The results of this study were examined with respect to

(a) Effect of contralateral WBN stimuliation on SOAEs.

(b) Effect of contralateral NBN stimulation on SOAEs.

Effect of contralateral WBN stimulation on SOAEs :

The effect of contralateral WBN stimulation on SOAEs was examined for alterations in

(a) SOAE amplitudes

(b) SOAE frequency

for the different intensity level of 30, 50 and 70 dBHL.

Contralateral WBN stimulation and SOAE amplitudes :

The SOAEs were recorded with a decrease in amplitude in all the subjects with the presence of wide band noise in the contralateral ear. Also with the increase in the intensity of contralateral wide band noise presentation there was a consistent decrease in the SOAE amplitudes. For the purpose of statistical analysis the standard 'T-test' was used at 0.05 level of significance. The results obtained after the statistical treatment of data are displayed in Table-1.

Baseline	30 dBHL	50 dBHL	70 dBHL
M = 8.98 SD = 4.89	M = 8.34 SD = 4.79	M = 6.02 SD = 5.01	M = 3.58 SD = 5.08
	T = 4.52 P < 0.001	T = 10.95 P < 0.0001	T = 11.15 P < 0.0001

Table 1: Displays the mean (M) in dBSPL, standard deviation (SD), T and P values obtained on comparision across baseline SOAE and the SOAE amplitude obtained in the presence of contralateral WBN at intensity level of 30, 50 and 70 dBHL.

Baseline M = 8.98 SD = 4.89	30 dBHL M = 8.34 SD = 4.79	T = 4.52 P < 0.0001
	50 dBHL M = 6.02 SD = 5.01	T = 12.52 P < 0.0001
	70 dBHL M = 3.58 SD = 5.08	T = 18.04 P < 0.0001

Table 2: Illustrates the mean (M) in dBSPL, standard deviation (SD), T & P values obtained on comparision between baseline SOAE and SOAE amplitudes measured with contralateral WBN presentation at 30, 50 and 70 dBHL.

The analysis of the data reveals a significant difference (at 0.05 level) between the baseline SOAE and the SOAE amplitudes obtained in the presence of contralateral presentation of WBN at the intensity level of 30, 50 and 70 dBHL. Also the decrease in SOAE amplitude was significant when compared across the three different intensity levels of contralaterally presented WBN. The mean amplitude of SOAEs obtained in the baseline and the experimental condition are expressed with the help of a bar diagram in Fig. 1.

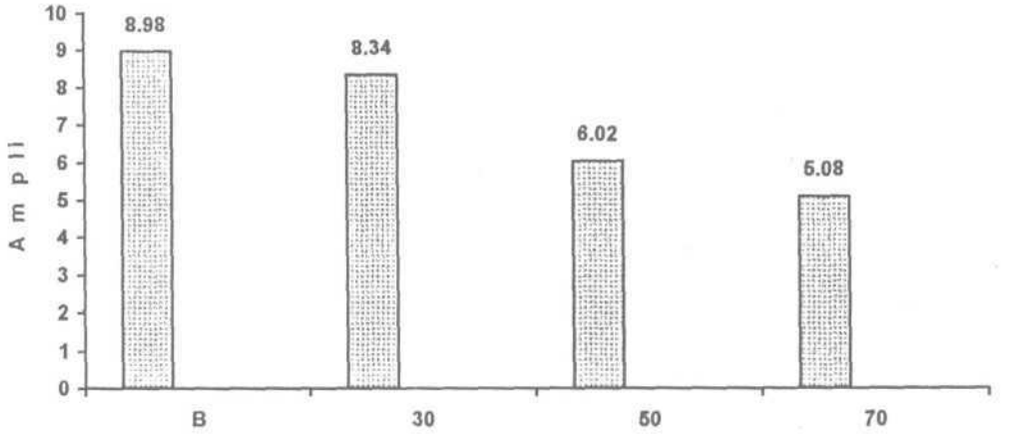


Fig.1: Illustrates the mean amplitude (dBSPL) of SOAE in the baseline condition (B) and with the presentation of contralateral WBN at intensity level of 30, 50 and 70 dBHL.

Contralateral WBN and SOAE frequency :

The presence of contralateral wide band noise induced changes in the SOAE frequency for twenty six subjects (37 - SOAE, 87%). In all the subjects there was an increase in SOAE frequency by 13 - 26 Hz. This may be attributed to the narrow band analysis performed on the spectrum of signal recorded in the ear canal in 13 Hz step size. Of the 37 SOAEs recorded from 26 ears an increase in SOAE frequency was seen for three subjects (3 - SOAE, 8%) for the presentation of contralateral WBN at the intensity level of 30dBHL. Further, when the intensity level of contralateral WBN was increased to 50 dBHL, eighteen subjects (24 - SOAEs, 65%) showed an increase in frequency by 13 Hz. When the intensity level was increased to 70 dBHL, twenty six subjects (37 - SOAE, 100%) demonstrated an increase by 13 Hz in the SOAE frequency. Three subjects (6 - SOAE, 16%) demonstrated an increase of 26 Hz in SOAE frequency. Only four subjects displayed no

changes in SOAE frequency even with the maximum intensity level of 70 dBHL of' WBN in ihe contralateral ear.

Discussion:

The results reported here support the findings of several investigators that stimulating the crossed olivocochlear bundle can affect the characteristics of spontaneous otoacoustic emission. But this suppressive effect could be the result of several factors (1) The middle ear reflex (2) a masking effect due to an interaural "cross talk" (3) an action of olivocochlear efferent system or (4) some systemic effects or unknown mechanisms. The possibility that a middle ear reflex is responsible for the effects at such low intensity level of contralateral acoustic stimulus presentation is doubtful and was ruled out by Puel and Rebillard (1990). A possible bone conduction "cross hearing" can also be excluded as the contralateral WBN was presented through an insert earphone and the maximum intensity level of noise stimulation did not exceed 70 dBHL. Therefore, the observed effect may be attributed to the change in mechanical non linearity of cochlea which induces alteration in SOAE that can be recorded, but other mechanical changes might also be involved. According to Siegel & Kim (1982a) at least part of the cochlear non linearity has a biological origin.

It has been demonstrated that living OHC, can reversibly shorten or lengthen its longitudinal dimension (Flock, 1986). For the better understanding of the mechanism by which the contralateral olivocochlear bundle can affect the contractile properties of OHCs and thereby the SOAE can be divided into two parts, (1) A mechanical model (Geisler, 1974), (2) Pharmacological basis for the observed effect.

Mechanical model : A modification of the familiar variable - resistance electrical circuit was presented by Geisler (1974) as a model of the crossed olivocochlear bundles effect mediated by efferent synapses on the outer hair cells. (Fig. 2)

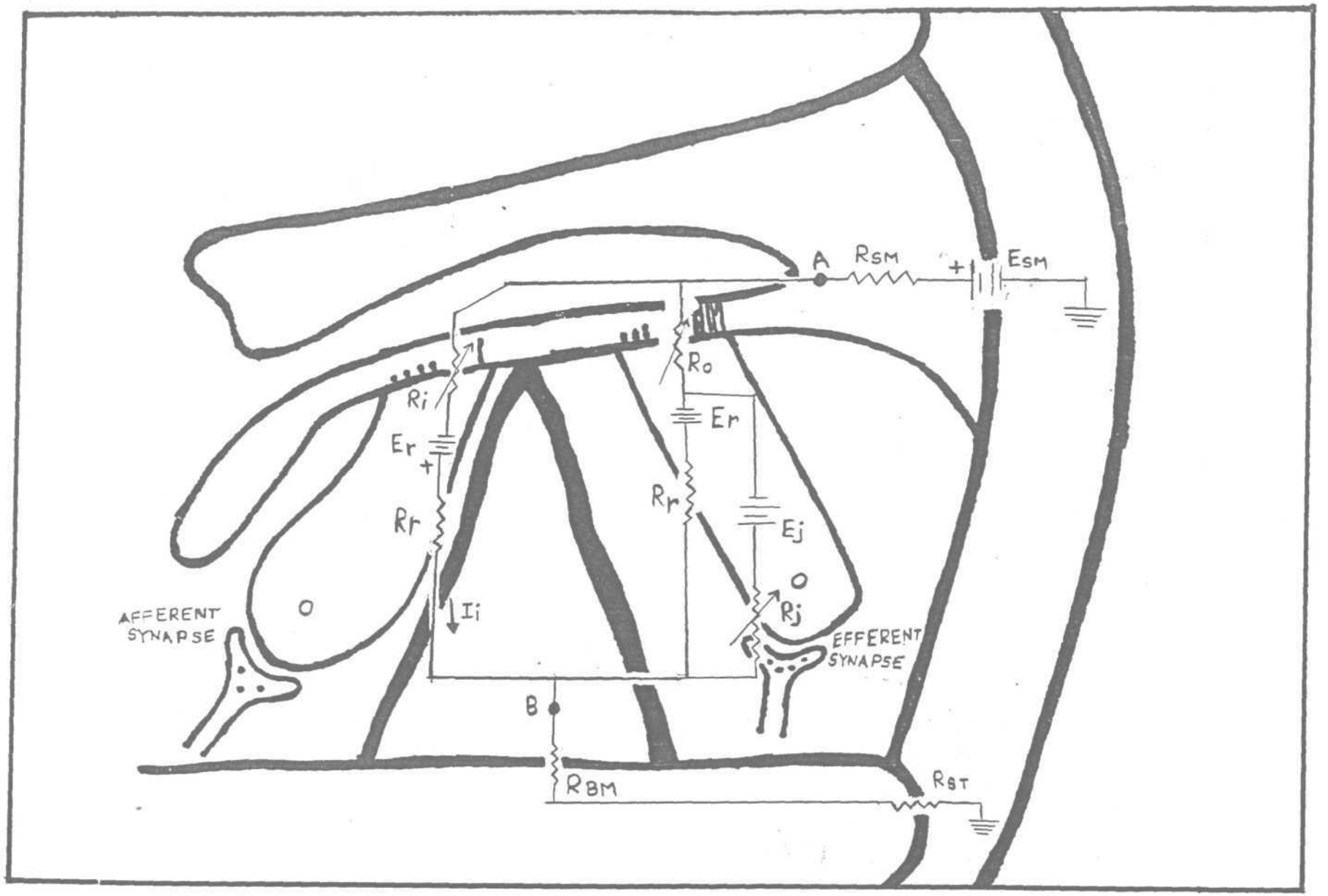


Fig.2: The mechanical model of cochlea

The subcuticular portions of hair cell membranes are assumed identical and are represented by battery Er in series with resistance Rr. Hair cell cuticular plates are assumed exposed to endolymph and are represented by resistances Ri and Ro. The post synaptic membrane at the efferent synapse is represented by resistance Rj in a series with battery Ej where Ej > Er (Eccles, 1964). The essential mechanism of the model is that the efferent synapse branch when activated shunts current away from the afferent synapse branch.

The activation of the inhibitory synapse by stimulation of the olivo cochlear bundle (OCB) would cause a reduction of the resistance Rj. This decrease in resistance would depend on the intensity of the contralaterally presented stimulus (noise) which would activate the efferent synapse at the base of the outer hair cells. This resistance change would hyperpolarize the membrane of the outer hair cells (Flock & Russell, 1973). The activation of the circuit through the efferent loop would increase the steady current flowing through resistance RSM, & R_{BM} & R_{ST}- As per the Thevenins theorm applied between points A and B reducing Rj would cause a decrease in Thevenins resistance. As Ej > Er it would increase the magnitude of the Thevenins voltage source which depends on the magnitude of stimulation through the efferent circuit. The thevenins voltage would cause increased counter clockwise current flow in the resulting one loop circuit following efferent stimulation. This would cause the endocochlear potential to drop and the potential in scala tympani to rise. Therefore, the ac current flowing through resistance R_S and R_{ST} would increase when resistance R_O is varied periodically. The R_O would theoretically depend on the direction of deflection of stereocilia during acoustic stimulation. Now the increased ac current would cause larger ac potentials at the points corresponding to scala media and scala typani. Simultaneously but not necessarily in phase, variation of resistance Ri would not change this later effect appreciably, provided that the ac and dc components of the current flowing through the outer hair cells were much larger than those flowing through the inner hair cells. The restriction on ac current distribution

seems reasonable as the stereocilia of the outer hair cells are in direct contact with lectorial membrane. In contrast the stereocilia of inner hair cells are not in direct contact with the tectorial membrane but are stimulated by the tectorial membrane at high intensity levels of signal presentation (Davis et al, 1958; Wang & Dallos, 1972).

Proceeding still further, it is clear that the decrease of dc voltage at point A caused by activation of the efferent synapse, coupled with the corresponding increase of the dc voltage at point B, forces the dc voltage between points A and B to drop. This decrease would in turn cause a reduced current flow Ii and a subsequent increase in the magnitude of the polarization of the sub cuticular membrane of the inner hair cells. Thus, activating the efferent synapse on the models outer hair cell would hyperpolarize the subcuticular portions of both inner and outer hair cells. It would appear that communication between the inner and outer hair cells is possible via field potentials, without the necessity for postulating synaptic, electronic (ephaptic), or transmitter interactions between inner and outer hair cells (Klinke and Galley, 1974). This hyperpolarization of the outer hair cells would reduce the excitatory activity of outer hair cells and consequently the amplitude of otoacoustic emission. The reduction in OAE amplitude would be in direct relation with the efferent excitation. Secondly the hyperpolarization of the outer hair cells will affect the active mechanical processes of the cochlea and thus the micromechanical characteristics of the basilar membrane. This in turn may cause alteration on the damping mechanisms or the reverse transduction and thus cause a change in the emission frequency of SOAEs. However the rationale underlying the positive shift or increase in SOAE frequency needs further exploration.

This electrical model of cochlea can be further supported with the information available regarding the neurotransmitters in the efferent system and its mode of action on the outer hair cells.

Pharmacological evidence : Efferent fibres, originating bilaterally from neurons located in the medial superior olivary nucleus of the brainstem, provide extensive innervation of the basal region of the OHCs (Warr & Guinan 1979). This efferent innervation is believed to be cholinergic (Norris & Guth, 1974) and has a number of unusual pharmacological properties, for example strychnine block of the inhibition of the auditory nerve compound action potential produced by efferent (olivocochlear bundle) stimulation. (Gifford & Guinan, 1987). Now there is little doubt about the major neurotransmitter role of acetylcholine (Ach) at medial efferent synapes with OHCs. Among the many biochemical and or neurochemical publications (Eybalin, 1993) for this assumption is the immuno-localization of choline acetyl transferase in the medial efferent cochlear innervation Altschuler et al (1985) and in the presynaptic endings below outer hair cells (Eybalin & Pujol, 1987). A mixed nicotinic-muscarinic receptor pharmacology has been reported for the efferent system of the inner ear, consistent with the existence of both ionotropic and metabotropic mechanisms. (Bobbin, 1996). Evidence for muscarinic acetylcholine receptor action in the cochlea has included both agonist binding studies and muscarinic acetylcholine receptor against-induced changes in inosital phosphate production (Niedzielski & Schocht, 1992). Specific evidence for muscarinic acetylcholine receptor action on cochlear OHCs is limited to a suggested inhibition of the response to acetylcholine when either pertusis toxin or heparin is dialysed into isolated OHCs (Evans, 1996). However, the most recent electrophysiological and Ca^{2+} imaging studies examine this possibility and find only support for an OHC nicotinic acetylcholine receptor (Blanchet et al, 1996; Evans, 1996).

Whole-cell voltage clamp experiments have demonstrated that acetylcholine acts with millisecond latency to activate the Ca^{2+} permeable mcotinic acetylcholine receptor localized to the basal pole of the guinea pig cochlear OHCs (Housley & Ashmore, 19901). The hyperpolarization of the OHCs has been shown to arise from the secondary activation of a Ca^{2} -

dependent k^+ (Evans, 1996). The type of receptors involved at these synapses is still a matter of controversy, although both nicotinic & muscarinic receptors have been demonstrated by various methodological approahces. Nicotinic receptors have been physiologically determined (Housley & Ashmore, 1991) and immunochemically detected at OHC basal poles (Plinkert et al, 1991). On the other hand, muscarinic receptors to be localized both on hair cells (Kakehata et al, 1993). and presynaptically (Bartolami et al, 1993a, b). It may be that the OHC cholinergic receptors are of peculiar subtype, somewhere in between nicotinic and muscarinic (Housely and Ashmore, 1991) which could explain the unusual cholinergic pharmacology of these receptors, especially their strychnine sensitivity. Thus the Acetylchoine of the medial efferent may mimic the effect of the electrical stimulation of the olivocochlear bundle (Kujawa et al 1993). It may be hypothesized that the observed phenomenon then may be a direct effect due to increase in acetylcholine transmission through the medial efferent which are in direct contact with the outer hair cells.

The electronic model of cochlea and its analogus pharmacological evidence reviewed here can account for the changes in the amplitude and emission frequency of SOAEs in the presence of contralateral acoustic stimulation.

Effect of contralateral NBN stimulation on SOAE :

The effects of contralateral NBN stimulation (centred at 1 kHz, 2 kHz, 3 kHz, 4 kHz, 6 kHz, 8 kHz) were examined for alterations in

(a) SOAE amplitudes

(b) SOAE frequency

at different intensity level of 30,50 and 70 dBHL.

Contralateral NBN stimulation and SOAE emission frequency :

In contrast to the results obtained for contralateral WI3N stimulation no change in the emission frequency was noticed for any band of contralateral NBN stimulation in the thirty subjects. It may be inferred that the effect of contralateral WBN was greater than the NBN centred at different frequencies at equal intensity levels.

Contralateral NBN stimulation and SOAE amplitudes :

In contrast to the effect of contralateral WBN presentation on SOAEs, the effect of contralateral NBN presentation was variable with respect to the SOAE amplitude and the centre frequency of narrow band noise. Therefore, for the purpose of statistical analysis the SOAE amplitudes were grouped together on the following criteria.

(1) The SOAEs on the basis of their emission frequencies were grouped into eight categories with the frequency range of 1 kHz in each category (Table -3),

Frequency range	No. of SOAEs
1000 - 2000 Hz	8
2001 - 3000 Hz	10
3001-4000 Hz	11
4001-5000 Hz	3
5001-6000 Hz	6
6001 - 7000 Hz	3
7001-8000 Hz	1
>8001 Hz	1

Table 3: The number of SOAE in different categories.

(2) The amplitude (intensity) of SOAE obtained on the contralateral stimulation of narrow band noise (x) centred at frequency immediately lower to that of the emission frequency were grouped together.

e.g for an SOAE at 3608 Hz, x = 3 KHz.

(3) The amplitude of SOAE obtained on contralateral NBN stimulation of centre frequency (y) immediately higher to that of the emission frequency were grouped together.

e.g. for an SOAE at 3608 Hz, y = 4 KHz.

(4) Similarly the SOAE amplitude obtained for contralateral stimulation of (x - 1 kHz) narrow band noise formed another group and also the amplitude obtained for (Y + 1 kHz) band of stimulation were accumulated together.

(5) Two more separate groups were obtained for (x - 2 kHz) and (y + 2 kHz) bands of noise.

As further no changes in the SOAE amplitude was noticed for other narrow bands of stimulation in relation with the emission frequency all the other values were assigned to form a(z) group.

All the forty three values could not be obtained in all the above mentioned groups due to methodological limitations.

e.g. No value of x could be obtained for emission frequency between 5 - 6 kHz. Such emission amplitudes could not be obtained for the data anlysis. The standard 't' test was used at 0.05 level of significance for the purpose of statistical analysis. The results can be discussed as follows:

SOAEs Vs X - narrow band noise :

Here the baseline amplitude (dB) values of SOAE were compared with the amplitude of SOAE obtained in the presence contralateral stimulation with X- NBN at the intensity level of 30, 50 and 70 dBHL respectively. The X - NB noise was centred at a frequency immediately lower to the emission frequency.

The results obtained after the statistical analysis of the data are displayed in Table 3 and 4.

	NBN		
Baseline	30 dBHL	50 dBHL	70 dBHL
M = 8.38 SD = 4.57	M=8.17 SD = 4.66	M = 7.00 SD = 4.57	M = 5.67 SD = 4.82
T= 1.87 P = 0.07	T = 8.22 P<0.0001	T = 9.15 P<0.0001	

Table 3: Depicts the mean (M) in dBSPL, standard deviation (SD), T & P values obtained on comparision across the SOAE amplitudes for baseline and contralateral X - NBN noise presentation at 30 dB, 50 dB, 70 dB

Baseline M = 8.38 SD = 4.57	30dBHL M = 8.17 SD = 4.66	T= 1.87 P = 0.07
	50 dBHL M = 7.00 SD = 4.57	T = 7.14 P< 0.0001
	70 dBHL M = 5.67 SD = 4.82	T= 10.90 P<0.0001

Table 4: Illustrates the mean (M) in dBSPL, standard deviation (SD), T & P values obtained on comparision between SOAE baseline amplitudes and SOAE amplitudes obtained with contralateral X -NBN presentation at 30, 50 and 70 dBHL.

The analysis of suppression reveals that the 30 dBHL of contralateral X - NBN presentation did not affect the amplitude of SOAE in the test ear. Thus 30 dBHL contralateral X - NBN did not produce a significant amount of

suppression. The suppression produced by 50 and 70 dBHL of x-NBN presentation was significant throughout the frequency range of emissions.

SOAEs Vs V - NBN presentation.

The t-test results obtained on comparision between the baseline SOAE amplitude and the amplitudes obtained with Y - NBN presentation are summarized in Table 5 & 6. The Y - NB noise was centred at a frequency immediately higher to the emission frequency.

	NBN		
Baseline	30 dBHL	50 dBHL	70 dBHL
M - 9.52 SD = 5.24	M = 9.04 SD = 5.60	M = 8.02 SD = 5.61	M = 6.58 SD = 5.64
	T = 2.72 P = 0.0101	T = 6.28 P < .0001	T = 8.48 P<0.0001

Table 5: depicts the mean (M) in dBSPL, standard deviation (SD), T & P values obtained on comparision across baseline SOAE amplitudes and amplitudes for contralateral Y - NBN presentation at 30, 50 and 70 dBHL.

Baseline M = 9.52 SD=5.24	30 dBHL M = 9.04 SD = 5.60	T = 2.72 P = 0.0101
	50 dBHL M = 8.02 SD = 5.61	T = 7.07 P< 0.0001
	70 dBHL M = 6.58 SD = 5.64	T = 9.31 P<0.0001

Table 6: Illustrates the mean (M) in dBSPL, standard deviation (SD), T&P values obtained on comparision between baseline SOAE amplitudes and SOAE amplitudes with contralateral Y - NBN presentation at 30, 50 and 70 dBHL.

Examination of the data reveals a significant suppression in the presence of 30 dBHL of contralateral Y - NBN which is in contrast with the results obtained for X-NBN presentation. Also the magnitude of suppression increased with the increase in the intensity level of contralateral Y - NBN presentation.

SOAEs Vs (X - 1 kHz) NBN presentation -

The baseline SOAE amplitudes were compared with amplitude of SOAE obtained for contralateral NBN which was approximately 1 KHz lower than the SOAE emission frequency. The results are documented in Table 7 8.

	NBN		
Baseline	30 dBHL	50 dBHL	70 dBHL
M = 9.36 SD = 5.33	M = 9.13 SD = 5.23	M = 9.06 SD = 5.26	M = 8.46 SD = 5.12
	T = 2.53 P = 0.08	T = 0.57 P<0.57	T = 3.84 P < 0.0006

Table 7 : Documents the mean (M)in dBSPL standard deviation (SD), T & P values obtained on comparision across the SOAE amplitudes for baseline and contralateral (X -1 kHz) NBN noise presentation at 30, 50 and 70 dBHL.

Baseline M = 9.36 SD = 5.33	30 dBHL M = 9.13 SD=5.23	T = 2.53 P = 0.08
	50 dBHL M = 9.06 SD = 5.26	T = 2.75 P < 0.09
	70 dBHL M = 8.46 SD = 5.12	T = 4.95 P<0.0001

Table 8: Displays the mean (M) in dBSPL, standard deviation (SD) T & P values obtained on comparision between SOAE baseline amplitudes and SOAE amplitudes obtained with contralateral (X - 1 kHz) NBN presentation at 30, 50 and 70 dBHL.

The results presented here show that the (X-l kHz) NBN contralateral presentation resulted in significant suppression of SOAE amplitudes at intensity level of 70 dBHL only. No significant amount of suppression was obtained for contralateral noise presentation at intensity level of 30 and 50 dBHL.

SOAEs Vs (Y + 1 kHz) NBN presentation :

The data was obtained by making a comparision of baseline SOAE amplitude with the SOAE amplitude in the presence of contralateral (Y + 1 kHz) NBN approximately 1 - 2 kHz above the SOAE frequency.

	NBN		
Baseline	30 dBHL	50 dBHL	70 dBHL
M = 7.31 SD = 3.30	M = 7.22 SD = 3.25	M = 7.18 SD = 3.1	M = 6.54 SD = 3.36
P = 1.44 T = 0.16	T = 0.37 P = 0.71	T = 4.10 P = 0.0005	

Table 9: Illustrates the mean (M) in dBSPL, standard deviation (SD) T & P values obtained on comparision across the SOAE amplitudes for baseline and contralateral (Y + 1 kHz) NBN presentation at 30, 50 and 70 dBHL.

Baseline M = 7.31 SD = 3.30	30 dBHL M = 7.22 SD = 3.25	T = 1.44 P = 0.16
	50 dBHL M = 7.18 SD = 3.12	T = 1.14 P < 0.26
	70 dBHL M = 6.54 SD = 3.36	T = 5.92 P<0.0001

Table 10: Demonstrates the mean (M) in dBSPL, standard deviation (SD), T & P values obtained on comparision between SOAE baseline amplitudes and SOAE amplitudes obtained with contralateral (Y + 1 kHz) NBN presentation at 30, 50 and 70 dBHL.

The results depicted in Table 9 and 10 indicate a significant suppression only for the intensity level of 70 dBHL for the contralateral (Y + 1 kHz) NBN presentation. Other intensity levels of noise did not produce significant suppression.

SOAEs Vs (X - 2 kHz) and (Y + 2 kHz) NBN presentation.

The results obtained on comparision of baseline SOAE amplitudes with amplitudes of SOAE determined for contralateral (X -2 kHz) NBN and (Y +2 kHz) NBN presentation are represented in Table 11,12 and Table 13 and 14 respectively.

	NBN		
Baseline	30 dBHL	50 dBHL	70 dBHL
M = 9.83 SD = 3.61	M = 9.83 SD = 3.17	M = 9.62 SD = 3.32	M = 9.45 SD = 3.84
	P = -0.61 T=1.00	T = - 1.95 P = 0.65	T = 0.33 P = 0.74

Table 11: Depicts mean (m) in dBSPL, standard deviation (SD), T & P values obtained with comparision across the SOAE amplitudes for baseline and contralateral (X - 2 kHz) NBN presentation at 30, 50 and 70 dBHL.

Baseline M = 9.83 SD = 3.61	30 dBHL M = 9.83 SD = 3.17	T = 0.61 P = 1.00
	50 dBHL M = 9.62 SD = 3.32	T = 1.95 P =0.057
	70 dBHL M = 9.45 SD = 3.84	T=- 0.09 P = 0.09

Table 12 : Represents the mean (M) in dBSPL, standard deviation (SD), T & P values obtained on comparision between SOAE baseline amplitude and amplitudes obtained with contralateral (X - 2 kHz) NBN presentation at 30, 50 and 70-dBHL.

	NBN		
Baseline	30 dBHL	50 dBHL	70 dBHL
M = 8.98 SD = 3.42	M = 8.71 SD = 2.96	M = 8.46 SD = 3.1	M = 8.18 SD = 3.64
	T= - 0.64 P=0.81	T = - 1.64 P = 0.09	T = - 1.18 P=0.16

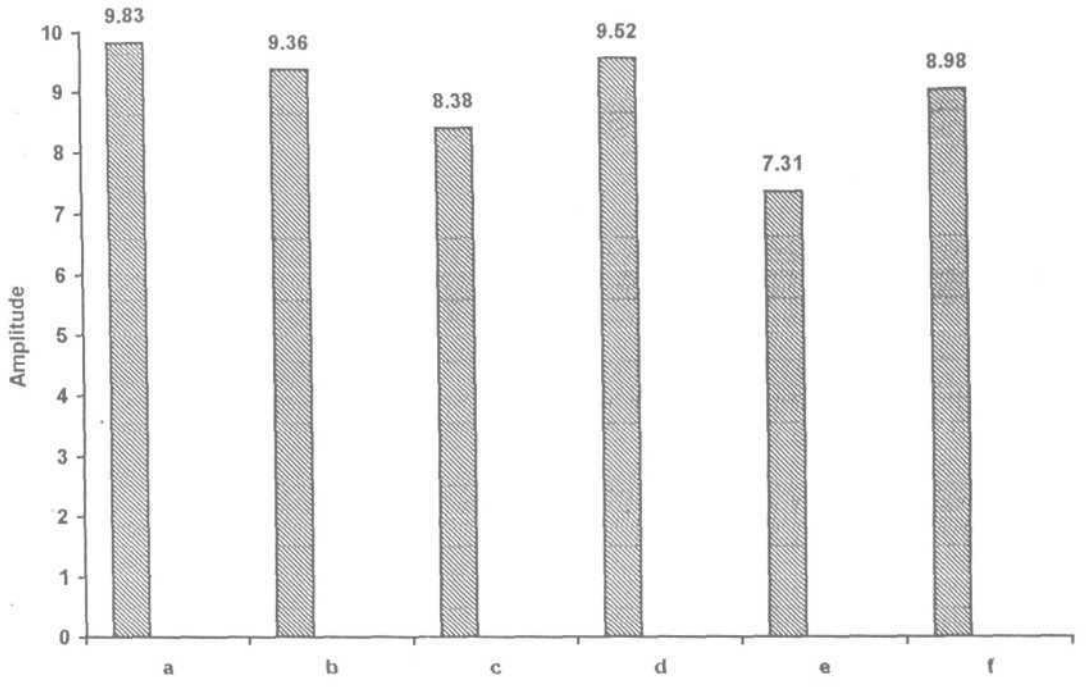
Table 13 Illustrates the mean (M) in dBSPL, standard deviation (SD), T & P values obtained on comparision across the SOAE amplitudes for baseline and contralateral (Y +2 kHz) NBN presentation at 30, 50 and 70 dBHL.

Baseline M = 8.98 SD = 3.42	30 dBHL M = 8.71 SD = 2.96	T = 0.61 P=1.00
	50 dBHL M = 8.46 SD = 3.12	T = - 0.69 P =0.18
	70 dBHL M = 8.18 SD = 3.6	T=- 1.41 P = 0.67

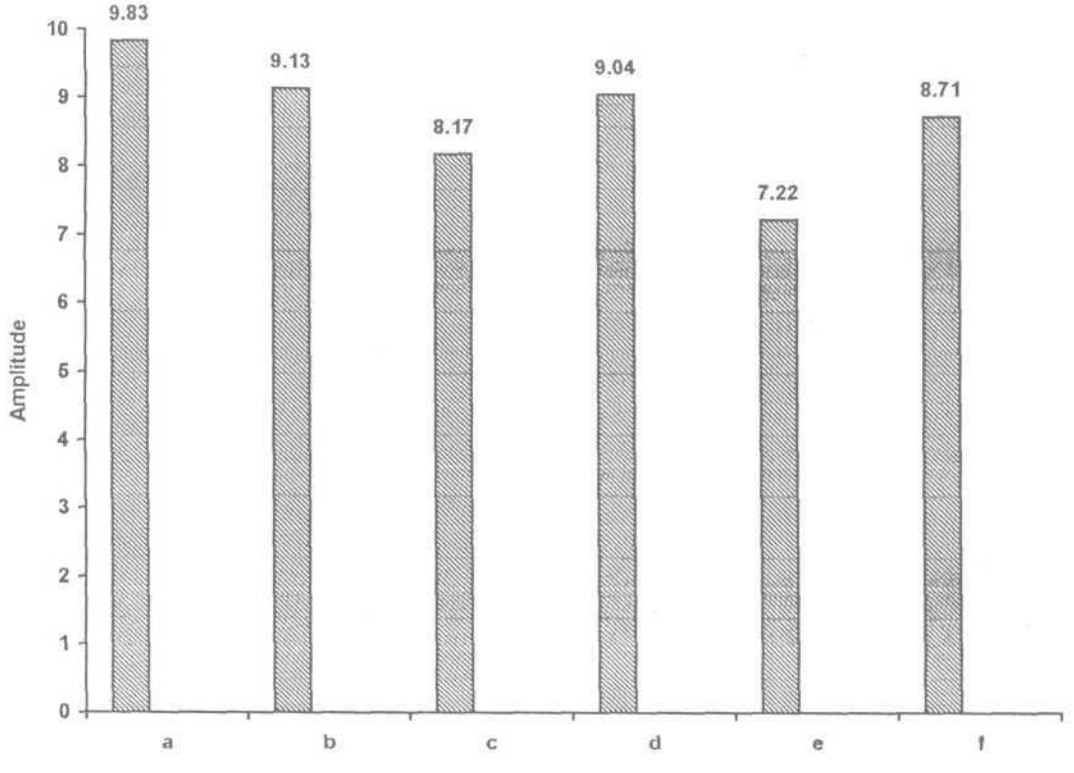
Table 14: Documents the mean (M) in dBSPL, standard deviation(SD), T & P values obtained on comparision between SOAE baseline amplitudes and amplitudes obtained with contralateral (Y + 2 kHz) NBN presentation at 30, 50, and 70 dBHL.

The examination of data reveals that the contralateraly presented NBN approximately 2 - 3kHz higher or lower than the emission was not able to produce significant suppression at the intensity level of 30, 50 and 70 dBHL. The mean amplitude of SOAEs obtained in the baseline and the experimental condition are expressed with the help of a bar diagram in Fig.3.

(A)


9.83
9.36
8.38
9.52
7.31
8.98
Amplitude
10
9
8
7
6
5
4
3
2
1
0
a
b
c
d
e
f

(B)


9.83
9.13
8.17
9.04
7.22
8.71
Amplitude
10
9
8
7
6
5
4
3
2
1
0
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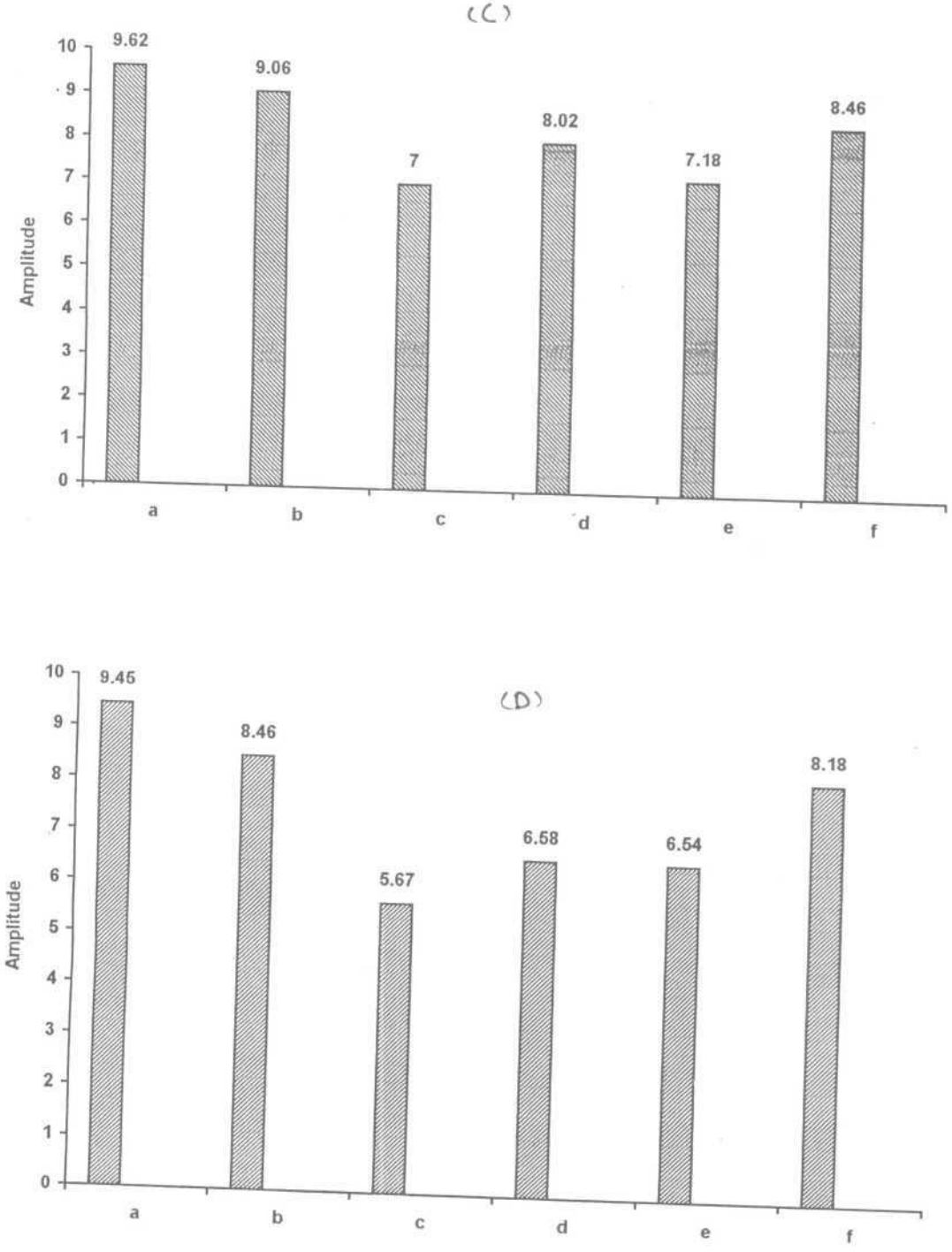
b
c
d
e
f



a = (X - 2 kHz) NBN , b = (X - 1 kHz) NBN , c = X- NBN, d = Y-NBN, e = (Y + 1 kHz) NBN, f = (Y +2 kHz) NBN.

Fig.3: Illustrates the mean amplitude (dBSPL) of SOAE in the baseline condition (A) and with the presentation of contralateral WBN at intensity level of 30 dBHL - (B), 50 dBHL - (C) and 70 dBHL - (D).

SOAEs Vs Z - group :

The Z group consisted of SOAE amplitudes obtained in the presence of contralateral NBN which was approximately higher or lower than the SOAEs frequency by greater than or equal to 3 kHz. The values of SOAE amplitudes obtained in the presence of remaining bands of narrow band noise stimulation were grouped together and compared with the baseline SOAE amplitudes. No significant differences wre obtained ($P > 0.05$) in the SOAEs amplitudes in the presence of other narrow bands of noise at the intensity level of 30, 50 and 70 dBHL.

The data obtained for each subjects was compared between the amount of suppression in terms of emission amplitude and frequency changes obtained with NBN presentation with that of the suppression for WBN presentation in the contralateral ear.

Discussion :

The results obtained hereindicate that the NBN higher to the SOAE emission frequency is more efficient in causing contralateral suppression as compared to the low frequency NBN. Also the NBN which was higher or lower by 1 - 2 kHz of the emission frequency causes contralateral suppression at higher intensity level. The other bands of noise presented to the contralateral ear but approximately greater than or equal to 2 - 3 kHz higher or lower than the emission frequency has no significant effect on emission amplitude.

Examination of the data also reveals that none of the narrow bands of noise produced as much suppression as wide band noise in any of the frequency bands of emission. This may be explained as per the suggestion of Moryl (1992) that there might be a critical bandwidth for the contralateral suppression of emissions. From the data presented in this study it is clear that the critical bandwidth, if there is one, cannot be the same as the critical bandwidth for other psychoacoustic phenomena on which the bandwidths tested were based.

A number of factors exist that may have contributed in the above obtained results.

1) NBN bandwidth.

The bandwidth of the narrow band noise presented in the contralateral ear was 3% of the centre frequency (Table - 13).

NBN	Bandwidth
1 KHz	970 - 1030 Hz
2 KHz	1940 - 2060 Hz
3 KHz	2910 - 3090 Hz
4 KHz	3880-4120 Hz
6 KHz	5820-6180 Hz
8 KHz	7760 - 8240 Hz

Table 13 : Bandwidth for the narrow band noise centred at different frequencies.

Therefore, the emissions occuring within the boundry of the narrow band noise (bandwidth) is likely to be suppressed more.

The evidence obtained from the study of Norman & Thornton (1993) suggests that there is a significant increase in suppression with ease in the bandwidth of narrow band noise. Further evidence of the general trend can be obtained from the results of this study which showed greater suppression with contralateral wide band noise presentation then that observed for NBN.

(2) Emission amplitude:

The emission amplitude for some of the SOAE measurements may vary by 1 - 2 dB. This may not be a significant factor for determining the amount of suppression for contralateral WBN presentation. But, while determing the

suppression for collateral NBN stimulation this may have contributed in obtaining the above results. This may be attributed to the fact that the suppression in terms of amplitude (dB) was much larger in the presence of contralateral WBN as compared to other bands of NBN.

Another factor that requires to be considered is that a ldB decrease in a subject with 3 dBSOAE does not have the same significance as in the case of a subject with a SOAE of 16 dB in amplitude.

(3) Tonotopic organization and SOAE frequency :

At this juncture an intriguing question about the frequency specific effect of the medial efferent system is that of its "causation". The tonotopic organization of the medial efferent system is the central tenet of this discussions. Warr & Guinan (1979) have identified a more basal shift in the crossed olivo cochlear bundles (COCB) projections from the medial olivocochlear neurons to OHCs of the contralateral cochlear (Fig. 4). Further the COCB fibres are peaked around 1 KHz to 2 KHz region of the contralateral cochlear. This anatomical findings suggested that the maximum suppression could be obtained at these frequences than elsewhere.

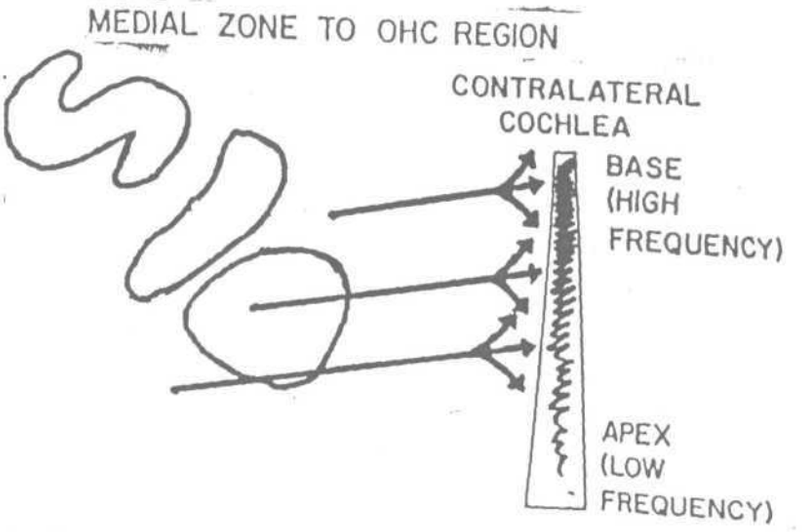


Fig. 4: Depicts the projections of the medial efferent fibres to the contralateral cochlea.

Another contributing factor to this speculation is the higher incidence of SOAE in the 1 - 6 KHz frequency range. This may be attributed to the reverse transfer function of the middle ear has a loss of 12 dB across best frequency range of 1-1.5 KHz. This loss increased at a rate of about 12 dB octave for both lower and higher frequencies (Kemp, 1980).

At the same time the acoustical properties of the earcanal and the frequency response of the recording equipment also determines the emission frequency and amplitude of SOAE.

Thus the results obtained may be largely due to the fact that the limited amount of data in the each frequency range and the scanty number of SOAEs measurements available for analysis above 6 kHz. Also the suppressive effect of noise (especially NBN) was subjected to a high degree of variability.

However, taking all these factors into consideration a comment can be made regarding the frequency specific nature of the medial efferent system in the low frequency region. (1-4 kHz).

(4) Apical end Vs Basal end (of cochlea) :

The site of generation of SOAE is said to have less of damping mechanism which results in spontaneous osillations on the basilar membrane (Probst et al, 1991). Ruggero et al (1983) hypothesized that the hair cells exert an excitatory influence on the basilar membrane at their location and an inhibitory influence on neighbouring locations. In the normal condition inhibition and excitation are in balance and no oscillations takes place. In the case of the damaged cochlea, the inhibitory influence normally exerted by the damaged region is removed, allowing the possibility of oscillation in the nearby undamaged regions. The passive positive damping is greater at the high frequency basal end as compared to the low frequency(Koshigoe & Tubis, 1983) apical end. Thus owing to this factor the contralateral noise at a

particular intensity level will have varied effects for SOAEs generated at different parts on the basilar membrane.

(5) Relations between SOAEs:

In ears with SOAE a common finding is that several emissions are present. Schloth (1983) and Dallmayr (1985) have shown that multiple spontaneous emissions may interact in specific ways including mutual suppression and the generation of acoustic distortion products. Emissions occuring within 50 HZ of each other have been detected when FFT averaging was used (Kohler et al 1986). However, such close emissions were shown to consist of a single SOAE with two different rapidly changing frequency states. The effect of contralateral acoustic stimulus may be variable in ears with multiple SOAEs. Also mutual suppression of SOAEs with larger frequency differences have been convincingly demonstrated by Burns et al (1984). The present study has anlyzed the SOAEs across the frequency range of 0 - 10 kHz in 13 Hz step size. Thus SOAEs occuring within the boundry of 13 Hz may not be identified and can create a bias in the results obtained.

Finally, it would be prentitious to consider any of these factors in isolation, owing to the fact that the amplitude and spectral characteristics of SOAEs measured in the ear canal may be reflective of not only the SOAEs at various sites of vibration across the basilar membrane, but also the anatomical and physiological action of middle and external ear and other technical influences. Thus the confounding influences of the above mentioned factors on each other are inextricably meshed and seem to underscore the perplexing results obtained.

In the light of now known studies on anatomical and physiological pecularities of the medial efferent (COCB) system, the results unequivocally suggest the system of having a high degree of frequency specificity. Although the findings of this study demonstrate some degree of frequency specificity of the COCB, but is quite deficient in resolving unambiguously the issue of

frequency specificity of COCB. The equivocal findings in the prsent study can be attributed to technical limitations, choice of noise bandwidth, high intersubject suppression variability, and most important of all the inherent nature of the mechanism(s) generating the SOAEs. However, carefully planned future experiments by researchers utilizing both physiologic and psychoacoustic measures may prove fruitful in discovering the frequency specific nature of the medial efferent system.

SUMMARY AND CONCLUSION

In the post 'Bekesy era' evidence regarding the contribution of medial olivo cochlear system innervating the outer hair cells to the alterations in the basilar membrane biomechanics has accumulated (Siegel & Kim, 1982). The major land mark in such investigations relating to cochlear biomechanics has been the discovery of otoacoustic emission by Kemp (1978). These emissions provide a non invasive effective method of observing the mechnical non linearities of the basilar membrane. However, the data available in relation with SOAE is limited and very few studies have been documented in the literature demonstrating the effect of contralateral acoustic stimulation on the frequency and amplitude characteristics of SOAEs. Also not much is known about the frequency selectivity of the medial efferent system and its contralateral suppressive effect on SOAEs, till date. The study was taken up with an aim of :

a) To examine the effect of contralateral acoustic stimulation on the amplitude and frequency characteristics of SOAEs.

b) To determine if the influence of CAS vary with increase in intensity of wide band noise.

c) To investigate the frequency selectivity of the medial efferent system :

   (i) To analyze if the narrow band noise (centred at standard audiometric frequencies across the frequency range of (1 - 8 KHz) has differential effect on the SOAEs.

   (ii) To study the effect of increase in intensity of contralateral NBN presentation across various frequencies on SOAEs.

For this purpose, 30 young adults (43 - SOAEs) were studied for their emission frequency and amplitude (dB) with and without the contralateral wide and narrow band noise presentation at the intensity level of 30, 50 and 70 dBHL. The SOAEs were analyzed across the frequency range of 0 - 10 kHz. The results of the study indicate that the contralateral wide band noise caused an increase in SOAE emission frequency by 13 - 26 HZz A consistent decrease in the SOAE amplitude was also noted in the presence of contralateral WBN stimulation. As the intensity of noise was increased the SOAE amplitudes kept on decreasing. In contrast to these results the data obtained for contralateral narrow band noise stimulation was variable. The contralaterally presented NBN (centered at 1 kHz, 2 kHz, 3 kHz, 4 kHz, 6 kHz, 8 kHz) did not cause any alterations in the emission frequency. The narrow band of noise immediately higher to the SOAE frequency caused a significant decrease in the SOAE amplitude No significant suppression could be obtained for 30 dBHL of contralateral NBN centered at a frequency immediately lower to SOAE frequency. Tthe amount of decrease was significant at a higher intensity level of 50 and 70 dBHL. Also the bands of noise 1-2 KHz on either side of the emission frequency could significantly reduce the SOAE amplitude at the intensity level of 70 dBHL only. Narrow bands of noise seperated by more than 2 KHz on the either side of emission frequency did not produce any suppressive effect at the highest intensity level of 70 dBHL.

A number of anatomical, physiological and technical factors and their confounding nfluences on each other which arc inextricably meshed arc also discussed.

Thus the study on the effect of contralateral acoustic stimulation on SOAEs opens the doors to further research on the topic and enhances the use of SOAEs in understanding the complex micromechanical action across the basilar membrane and in resolving the less understood physiological activity of the human cochlea.

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