Contralateral Suppression of TEOAEs: A tool to identify the distribution of Efferent auditory nerve fibers to the cochlea

Reg. No. M0119

An Independent project submitted in part fulfillment for the first year M.Sc (Speech and Hearing) to University of Mysore

All India Institute of Speech and Hearing
Mysore-570006

May- 2002

DEDICATED TO

The Pioneers in the field of

Efferent Auditory system

&

Mukesh and Sharad

CERTIFICATE

This is to certify that this independent project entitled **"Contralateral Suppression of TEOAEs: A tool to identify distribution of efferent auditory nerve fibers to the cochlea"** is a bonafide work in part of fulfillment for the degree of Master of Science (Speech and Hearing) of the student (Register No.MOl 19)

DIRECTOR

All India Institute of Speech and Hearing.
Mysore-570006

Mysore,
May, 2002

CERTIFICATE

This is to certify that this independent project entitled **"Contralateral Suppression of TEOAEs: A tool to identify distribution of efferent auditory nerve fibers to the cochlea"** has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any other University for the award of any degree or diploma.

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DECLARATION

This Independent project entitled **"Contralateral Suppression of TEOAEs: A tool to identify distribution of efferent auditory nerve fibers to the cochlea"** is the result of my own study under the guidance of, **Mr. Animesh Barman,** Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore and not been submitted in any other University for the award of any degree or diploma.

Mysore,

May, 2002 **Reg.** No. **M01**19

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INTRODUCTION

In the family of sciences, Audiology is a young but lusty infant. Though, born less than 6 decades ago, this field has already generated an extensive international literature. During its development, each decade seemed to be dominated by a new advance in auditory physiology, made by various pioneers in the field of Audiology with their important contributions. One among them is, "Rasmussen", who described the existence of an efferent innervation of the mammalian cochlea way back in 1946.

An efferent auditory system can be found in, literally all classes of vertebrates and in some invertebrates (Roberts & Meredith, 1992). The descending (efferent) olivo cochlear (OC) system is known to contain cell bodies and axons originating from specialized nuclei within and surrounding the brainstem superior olivary region. These descending centrifugal fiber bundles provide direct, bilateral input to the cochlea via the anatomically segregated medial and lateral efferent deviation (Brown, 1987; Warr, 1992). There is an important difference in projection between the two subtypes of OC neurons. Medial olivary cochlear (MOC) neurons projected mainly to the outer hair cells (OHCs) whereas, lateral olivary cochlear (LOC) neurons projected mainly to the inner hair cells (IHC) region. Medial efferent innervation is largest near the center of the cochlea with crossed innervation biased towards the base compared to the uncrossed innervation. In contrast,

lateral innervation is relatively constant in the center and base of the cochlea (Guinan, Warr & Norris, 1984; Liberman, Dodds & Pierce, 1990).

Clinical interest in the medial efferent system has been awakened by recent advances in the prepackaged computer technology designed for the clinical study of 'otoacoustic emissions' (OAEs) (Kemp, Bray, Alexander & Brown, 1986; Kemp, Ryan & Bray, 1990; Robinette, 1992).

OAEs are defined as sub-audible sounds generated at the level of the normal cochlea. These sub-audible emissions are preneural (Anderson & Kemp, 1979).

OAEs can be mainly classified as -

(1) Spontaneous OAEs (SOAEs)

(2) Evoked OAEs (EOAEs)

SOAEs are low-level, tonal signals measured in the external ear canal in the absence of any known stimulus (Bright, 1997). In general SOAEs are relatively less prevalent than evoked emissions and there is some evidence that, the occurrence of spontaneous emissions may be gender and ear dependent (Penner & Zhang, 1997).

EOAEs are believed to be generated by electromotile activity of the outer hair cells of the cochlea in response to an evoking stimulus (Robinette & Durrant, 1997). EOAEs are mainly classified as transient evoked OAEs (TEOAEs), distortion-product OAEs (DPOAEs) and stimulus frequency OAEs (SFOAEs) depending on the type of stimuli and process of generation of OAEs.

In general OAEs are thought to be the product of intrinsic, nonlinear mechanical activity within the cochlea (Probst, 1990). They are believed to provide a direct functional view of OHCs (Probst, 1990). Since OHCs are innervated predominantly by neurons of the medial efferent system which arise in the ipsilateral or contralateral superior olivary complex of the brainstem, the stimulation of medial efferent system would affect the function of OHCs and thus leading to difference in the generation of OAEs.

The efferent fibers can be activated by the electrical stimulation at the floor of the fourth ventricle (Galambos, 1956) or by contralateral acoustic stimulation (Buno, 1978). The stimulation of efferent system by these method reduces the compound action potentials of the auditory nerve (Ni) or reduces the amplitude of both spontaneous (Mott, Norton, Neely & Warr, 1989) and evoked otoacoustic emissions (Collet, Kemp, Veuillet, Duclaux, Moulin & Morgon, 1990; Veuillet, Bazin & Collet, 1991). This is due to reduction in gain of the cochlear amplifier.

Most of the physiological studies on the OC systems have investigated the action of the MOC neurons (reviewed by Guinan, 1996). MOC neurons are excited by sound (Fex, 1962) and thus are part of MOC reflex. MOC responses are tuned and have a well defined characteristic frequency (CF). Single unit labeling studies indicate that, an individual MOC neurons projects to the regions of the cochlea innervated by type I fibers of about same characteristic frequency at least in the basal turn of the cochlea (Warr & Guinan, 1979; Robertson & Gummer, 1985; Brown, 1989; Liberman, 1988b). Thus, the MOC neurons are expected to be frequency specific.

Need for the study:

It is clear from the above discussion that the uncrossed and crossed. MOC are mostly distributed, either in the center or base of the basilar membrane. Also, acoustic stimulation to the contralateral ear is expected to show some amount of frequency specific suppression of TEOAEs. Thus, the frequency at which the suppression of TEOAEs is more, might help us to determine whether the uncrossed or crossed MOC plays a major role on the physiology of OHC due to contralateral acoustic stimulation.

Hence, the present study was aimed -

1) To evaluate the frequency specificity of MOC neurons based on contralateral suppression of TEOAEs.

2) To examine whether the effect on TEOAEs is due to ipsilateral or contralateral MOC fibers.

3) To see the differential effects of narrow band noise (NBN) and broad band noise (BBN) on suppression of TEOAEs.

REVIEW OF LITERATURE

OAEs are the acoustic energy produced as a result of the micro-mechanical activity of OHCs on the organ of corti. This is based on the evidence from animal experiments that mammalian OHCs are motile in response to changes in cellular potentials (Brownell, Bader, Bertrand & Ribaupierre, 1985). They appear to be responsible for sharp tuning of the basilar membrane. They are the active mechanisms vulnerable to cochlear pathology (Johnstone, Patuzzi & Yates, 1986) due to intensive noise exposure, ototoxic drugs, etc. These OHCs are innervated predominantly by efferent auditory nerve fibers.

The efferent auditory pathway is formed by the descending motor nerve fibers which take their origin at the neuronal somata in the superior olivary complex (reviewed by Brown, 2001). These descending fiber tracts are known as olivary cochlear bundle (OCB). The pathway was first described by Rasmussen (1946). The total number of OC neurons ranges from 500 to 2500 per cochlea depending on the species (Warr, 1992).

The OCB is composed of mainly 2 separate systems -

(1) The lateral olivary cochlear (LOC) projections

(2) The medial olivary cochlear (MOC) projections

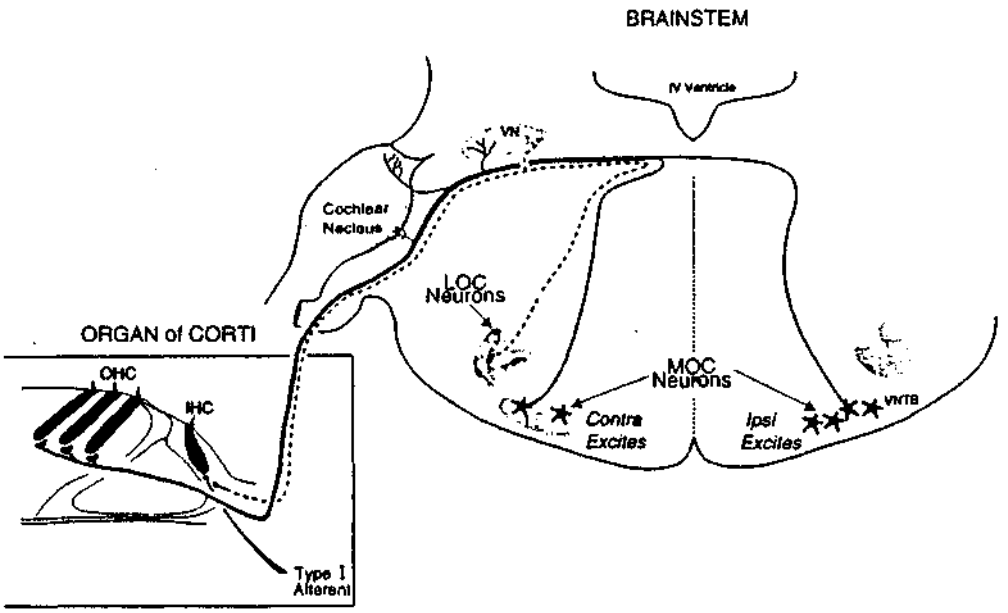


Fig 1. Depicts the pathway and distribution of MOC and LOC neurons.

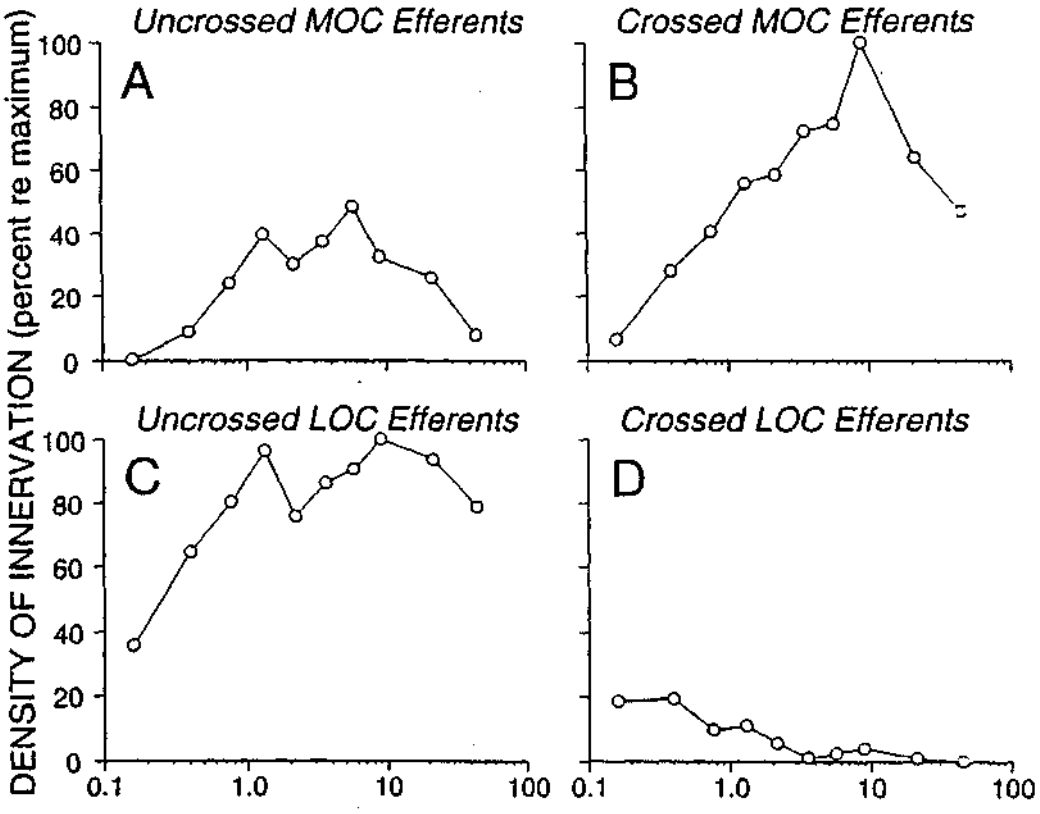
The MOC projections distribute primarily to OHCs (Warr & Guinan, 1979). They contains the myelinated nerve fibers, hence are readily stimulated by extracellular currents (Hallin & Torebjork, 1973; Fitzgerald & Woolf, 1981).

The LOC projections distribute primarily to IHCs (Warr & Guinan, 1979). They contain unmyelinated nerve fibers, hence are not readily stimulated by extracellular currents (Hallin & Torebjork, 1973; Fitzgerald & Woolf, 1981).

The mammalian cochlea receives efferent innervation from both ipsilateral and contralateral superior olivary complex. Approximately 72% to 74% of MOC fibers travel to the contralateral cochlea and supply the OHCs. The remaining 26% to 28% course ipsilaterally whereas, approximately 89% to 91% of LOC fibers destined to terminate in the ipsilateral IHCs and remaining 9% to 11% project to contralateral IHCs (Guinan, Warr & Norris, 1983; Warr, 1992).

Medial and lateral efferents have different patterns of innervation along the length of the cochlea. Medial efferent innervation is largest near the center of the cochlea with crossed innervation biased towards the base compared to the uncrossed innervation. In contrast, lateral innervation is relatively constant in the center and base of the cochlea (Guinan, Warr & Norris, 1984; Liberman, Dodds & Pierce, 1990).

The following graphs (1, 2, 3, 4) depict the density of LOC and MOC efferents for both crossed and uncrossed distributions as a function of cochlear position. The graphs were constructed using the combined anterograde tracing data (Guinan, Warr & Norris, 1984) and immuno-staining data (Liberman, 1996) from the cat.



Graph 1, 2, 3, 4 : Depicts the density of crossed and uncrossed MOC and LOC neurons across the basilar membrane.

Although, the existence of an efferent innervation to the mammalian cochlea was described more than 50 years ago (Rasmussen, 1946), the functional role of auditory efferent fibers in hearing is still a matter of debate. However, continued attempts have been made to understand the functions of efferent system. Conclusions regarding its functions have been drawn from both animal as well as human research. In general the OCB has an inhibitory effect on the auditory periphery. Because of its predominantly inhibitory nature, it has been hypothesized that the efferent system serves a protective

role in the auditory system (Rajan, 1988). It is also hypothesized that the activation of OCB enhances the detection of sound in noise (Micheyl & Collet, 1996) and maintains the cochlea at an optimum mechanical state for efficient function of active processes (Johnstone, et al., 1986).

Most of the physiological studies on the OC systems have investigated the action of the MOC neurons (reviewed by Guinan, 1996). This conclusion stems from the fact that MOC axons are relatively large in diameter between, 0.5 and 2.75 μm in cat and myelinated. Hence, can be easily stimulated (Hallin & Torebjork, 1973; Fitzgerald & Woolf, 1981).

The MOC fibers can be activated by -

(1) The electrical stimulation at the fourth ventricle (Galambos, 1956)or

(2) The contralateral acoustic stimulation (Buno, 1978).

Much of the existing data on efferent effects has been obtained by exciting the olivo cochlear bundle (OCB) with shocks from an electrode at the midline of the floor of the fourth ventricle (Reviewed by Guinan, 1996). This location is used as efferent fibers are close to the surface and easy to access (Galambos, 1956). McCue & Guinan (unpublished) found that both crossed and uncrossed medial efferent fibers can be stimulated by a midline electrode.

The stimulation of efferent system can affect the activity of different physiological processes, which can be discussed under the following headings.

The effect of medial efferent stimulation on -

(1) Action potentials
(2) Cochlear microphonics and MOC potential
(3) IHC potential
(4) Auditory nerve fibers
(5) Stapedial acoustic reflex
(6) Otoacoustic emissions

Action potentials

Perhaps the best known effect of efferent activity is to depress the compound action potential of auditory nerve, N1 (Galambos, 1956; Desmedt, 1962; Wiederhold & Peaks, 1966). This inhibition can be seen with N1 evoked by clicks or by tone pips. Efferent activity evoked by electrical stimulation and contralateral sound produces qualitatively similar effects (Buno, 1978; Murata, Tanohashi, Horidawa & Funai, 1980; Folsom & Owsley, 1987; Liberman, 1989). These inhibition were greatest at low sound levels (Galambos, 1956).

Cochlear microphonics (CM) and MOC potential

In addition to inhibiting N_1 medial efferents also affect non-neural cochlear potentials. Efferent stimulation increases the amplitude of CM and evokes a slow potential, the "MOC potential". The increase in CM is typically larger at high sound levels than at low sound level and can be as large as 4 dB (Fex, 1959; Kittrell & Dalland, 1969; Konishi & Slepian, 1971; Teas, Konishi & Nielsen, 1972; Gifford & Guinan, 1987).

The endocochlear potential bathing the hair cell cilia is around +80 to +90mv. Stimulation of OCB decreases this endocochlear potential by few millivolts. This decrease in large endocochlear potential is termed as MOC potential (Fex, 1959, 1962; Konishi & Slepian, 1971; Brown & Nuttall, 1984; Gifford & Guinan, 1987).

This change in the CM and endocochlear potential can be understood from the electrical properties of the cochlea. Efferent stimulation by increasing OHC basolateral conductance and hyperpolarizing the OHCs, increases the current flow (Davis, 1965; Dallos & Cheatham, 1976) and thereby increases the CM. This increase in OHC conductance also leads to a decrease in basilar membrane motion and to the inhibition of N_1. In addition to the above, the increased conductance and hyperpolarization of the OHCs causes an increased DC current flow through the OHC steriocilia, thereby

decreasing the large, positive endocochlear potential. The MOC potential is the decrease in the endocochlear potential and the potentials causes throughout the cochlea by the increased DC current flow.

IHC potential

An important step in understanding how efferents that synapse on OHCs affect the firing of auditory nerve fibers, that innervate IHCs, was provided by intracellular recordings from IHCs. Brown and co-workers (Brown, Nuttall & Masta, 1983; Brown & Nuttall, 1984) stimulated medial efferents in guinea-pigs while recording intra-cellularly from IHCs. Efferent stimulation reduced both AC and DC receptor potentials in the IHCs without producing a conductance change in the IHCs. The lack of a conductance change indicates that the efferent effect was not produced by efferent synapses, that were directly on the IHCs and therefore the site of medial efferent inhibition is functionally peripheral to the IHCs. Furthermore, for tone burst at the most sensitive frequency of IHCs, efferent induced level shifts in IHC receptor potentials were approximately equal to efferent induced level shifts of N_1. Thus, the efferent inhibition of N_1 could be accounted for by the efferent induced decrease in the IHC receptor potential (reviewed by Guinan, 1996).

Auditory nerve fibers

The discharge of neural impulses by auditory nerve in the absence of acoustic stimulation is called spontaneous firings. The stimulation of medial efferents reduces the spontaneous activity of auditory nerve fibers even in very insensitive fibers (Wiederhold & Kiang, 1970; Guinan & Gifford, 1988; Kawase, & Liberman, 1993). This observation is not due to an efferent induced reduction of cochlear amplifier gain. Guinan & Gifford (1988) hypothesized that this is due to the MOC potential.

Medial efferent stimulation shifts the thresholds of auditory nerve fibers to higher sound levels throughout the tuning curves (Guinan & Gifford, 1988). The largest threshold shifts are in the tip region of the tuning curve, which is the most sensitive part. For auditory nerve fibers with high (>3 kHz) characteristic frequencies (CFs) the threshold shift is usually greatest at the fiber's CF and decreases for higher and lower frequencies. The result is that efferent stimulation makes tuning curve wider (Wiederhold, et nl. 1970; Guinan & Gifford, 1988c). For some fibers, particularly low spontaneous rate (SR) and medium SR fibers with CFs of 1-2 kHz, the largest increase in threshold was at the low-frequency edge of the tuning curve tip (Guinan & Gifford, 1988c). In such cases efferent stimulation made the tuning curve more narrow, opposite to the typical efferent effect on high CF auditory nerve fiber.

Medial efferent stimulation has little effect on tuning curve tails. For high CF fibers in which 1 kHz was in the tail, threshold shifts at 1 kHz averaged less than 1 dB (Guinan & Gifford, 1988c).

Stapedial Acoustic reflex

Borg (1971) measured the acoustic reflex threshold in the awake rabbit, before and after COCB sectioning. He found that ART were decreased by 12 dB after the sectioning. These results indicate that the efferent system has some influence over the acoustic reflex.

Higson, Stephenson & Haggard (1996) reported the binaural summation of acoustic reflex. They found out ipsilateral acoustic reflex threshold (ART) with and without contra-lateral acoustic stimulation. Contralateral stimuli was of the same frequency as that of reflex activating stimulus and intensity was ipsilateral ART + (contralateral ART - ipsilateral ART). They noted an improvement in the ART by about 4.4 dB. But, 3 subjects out of 34 showed negative summation i.e., elevation in threshold and some showed no difference.

Ajith Kumar & Animesh Barman (2002) studied the effect of efferent induced changes on acoustic reflex threshold and amplitude. AR threshold and acoustic reflex amplitude (at ART+10 dB) were obtained at

three frequencies (500 kHz, 1 kHz, and 2 kHz) in the presence and absence of Contralateral NBN and WBN (centered around reflex activating stimuli) at 30dBSL. Results showed a consistent reduction in amplitude and increase in threshold for 1 kHz and 2 kHz tones in the presence of contralateral WBN but not NBN. The observed effect was attributed to the change in the electrical or mechanical properties of cochlea brought about by the efferent auditory system. Study concluded that the efferent system inhibits the cochlear responses for high stimulus intensity levels and may play an active role in the protection of the cochlea from acoustic injury.

Otoacoustic emissions

A variety of experiments indicate that medial efferent activity influences OAEs. Such effects are of interest because OAEs are thought to reflect aspects of basilar membrane motion. OHCs over the basilar membrane are known to be cochlear amplifier. OAEs are believed to be generated by active mechanisms in the cochlea which involves OHCs. Since OHCs receive direct efferent innervation. They may be affected by contralateral acoustic stimulation (CAS) of olivocochlear bundle (Kim, 1986).

There are wide variety of mechanisms by which medial efferents might affect OAEs.

At low to moderate sound levels, medial efferent induces depression of basilar membrane (Dolan & Nuttall, 1994).

It affect the operation of OHCs, i.e., it may reduce the OHC receptor potential, which would reduce OHC motion (Santo-Sachi & Dilger, 1988).

It hyperpolarizes the cell, which moves the membrane potential away from the optimum voltage for voltage to length transduction (Roddy, Hubbard, Mountain & Xue, 1994).

Efferent induced contractions of OHCs distort the organ of corti, thereby lowering the gain of the cochlear amplifier (Rajan, 1990).

Finally, medial efferents reduce the endocochlear potential which reduces the gain of the cochlear amplifier (Sewell, 1984).

Efferent stimulation is shown to effect all types of OAEs. Medial efferents produce small changes in SOAEs. SOAE frequency shifts to higher frequencies and amplitude can change in either direction (Mott, et al., 1989; Harrison & Burns, 1993).

Efferent stimulation usually decreases DPOAEs, but sometimes it increases them (Mountain, 1980; Siegel & Kim, 1982). Efferent inhibition of

DPOAE is greatest for low-level primaries and decreases as primary tone level is increased (Mountain, 1980; Moulin, Collect & Duclaux, 1993).

Similarly, activity in medial efferents affects Click evoked OAEs Tone-burst OAEs and stimulus frequency OAEs. The usual effect is to inhibit with the greatest inhibition for responses to low level sounds (Guinan, 1986, 1991; Collet, et al., 1990; Ryan, Kemp & Hinchcliffe, 1991; Norman & Thornton, 1993).

Although early literature emphasizes efferent effects at low sound levels (Galambos, 1956; Mountain, 1980), recent work suggests that the most significant effect of medial efferents may be at moderate and high sound levels (Guinan & Stonkovic, 1995).

The efferent stimulation through contralateral acoustic stimulation is dependent upon the type of contralateral stimulus. The contralateral acoustic stimulus to stimulate efferent system can be a pure tone (Mott, Norton, Neely & Warr, 1989; Berlin, Hood, Wen, Szabo, Cecola & Rigby, 1993a; Harrison & Burns, 1993), Clicks (Veuillet, Bazin & Collet, 1991), Narrow band noise (Veuillet, Bazin & Collet, 1991; Chery-croze, 1993) or by Broad band noise (Veuillet, 1991, 1992; Berlin, et al., 1993a). Among all BBN seems to be most effective stimulus since, the OCB activation increases with increase in bandwidth of CAS (Norman & Thorton, 1993).

Among the TEOAE, DPOAE and SOAE with the contralateral BBN, TEOAE achieves the maximum suppression (reviewed by Hall, 2000).

Experiments conducted on subjects with stable SOAEs, where there are clear amplitude peaks at particular frequency have shown changes in both intensity and frequency of these peaks with contralateral pure tones (Mott, et al., 1989).

Veuillet, et al., (1991) studied the suppression of emissions evoked using 1kHz and 2 kHz tone pips by contralateral NBN at intensity of 50 dB SPL and found that the amount of suppression was greatest when the noise band was centered on the central emission frequency.

Moryl (1992) studied the suppression of click evoked emission by contralateral pure tone and found suppression in some frequency bands of the emission from 250 Hz and 500 Hz tone but, no significant effect from higher frequency tone at the same intensity.

Norman & Thorton (1993) found that 0.5 kHz NBN produced most suppression at low frequencies, the 1 kHz band at mid-frequencies and 2 kHz band at high frequencies, but a significant result was obtained only at 1 kHz band within the emission where the amount of suppression was itself significant for almost all the noise bands.

Thus, from the above studies, it can be inferred that the change in the response of OAEs may show some degree of frequency specificity if the contralateral stimulus is frequency specific.

METHOD

The following method was adopted to evaluate the frequency specificity of medial efferent auditory nerve fibers through contralateral suppression of TEOAEs and to investigate whether crossed or uncrossed MOCB plays a role in TEOAE suppression

SUBJECTS

32 normal hearing adults in the age range of 15-25 years served as subjects for the study. 32 subjects involved 21 females and 11 males, who passed the following criteria:

1. Subjects did not report of having any past or present history of otological or neurological problems.
2. All of them had auditory thresholds within 15 dB HL over the frequency range of 250 Hz - 4000 Hz to say that person's hearing was within normal limits (ANSI, 1969).
3. They had 'A' type tympanogram with reflexes present.
4. Did not report of any difficulty in understanding speech in the noisy situation, and
5. Had a signal-to-noise ratio of >6 dB SPL in the baseline averaged response of TEOAEs.

INSTRUMENTATION

1. A calibrated two channel OB-922 diagnostic audiometer was used for pure tone audiometry.
2. A calibrated GSI-33 (version-2) middle ear analyzer was used to assess the middle ear status.
3. A calibrated two channel GSI-16 diagnostic audiometer was used to obtain behavioral thresholds for noise [NBN with center frequency of 1 kHz, 2 kHz, 3 kHz, 4 kHz and BBN] and to present noise through the contralateral ear.
4. TEOAEs were measured using ILO-292 echoport plus.

STIMULUS PARAMETERS

TEOAEs were recorded by presenting clicks at intensity around 75 dB SPL. The response of 260 sweeps of clicks were averaged to obtain the standard non-linear click evoked emission and amplitude of emission were measured. The stimulus stability was to be above 90% to consider the response for the study.

NBN with center frequency at 1 kHz, 2 kHz, 3 kHz, 4 kHz at 50 dB SL (ref. Noise threshold) and BBN at 50 dB SL (ref. Noise threshold) were

fed through the insert receiver to the contralateral ear to activate the medial efferent fibers.

TEST PROCEDURE

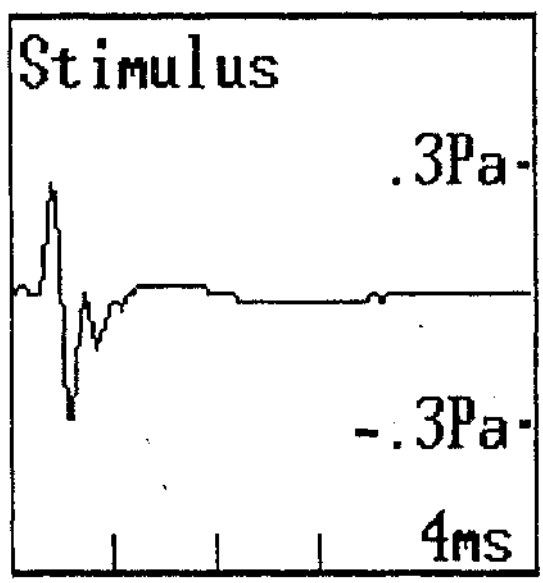
The test procedure started with the case history of the individual. In the case history, information was collected about the individual's hearing condition in quite and noisy situation, presence or absence of otological and neurological problems etc. Individuals who did not report of having any such problems were considered for the behavioural and physiological tests.

Individual's behavioral thresholds for pure tone were evaluated for frequencies between 250 Hz to 4000 Hz. Subjects who had auditory thresholds within 15 dB HL for these frequencies were considered for immittance audiometry.

Immittance audiometry was carried out with a probe tone frequency of 226 Hz. Acoustic reflex thresholds were evaluated with 500 Hz, 1 kHz, 2 kHz, and 4 kHz stimulation tones. Subjects who had 'A' type tympanogram with reflexes present were taken for the study.

MEASUREMENT OF TEOAEs

For the measurement of TEOAEs, the patients were made to sit comfortably on a chair inside a sound treated room. The probe with a tip was positioned in the external ear canal and was adjusted to give flat frequency spectrum across frequency range, as shown in figure 2.



After the establishment of good probe fit, data were obtained in three phases:

1. Using ILO-292 Echoport plus OAE analyzer, TEOAE response for 260 sweeps of clicks were averaged at intensity around 75 dB SPL. This was considered as the baseline TEOAE response and was used as the reference, to assess the suppression of TEOAE response seen in the presence of contralateral acoustic stimulus.
2. Insert receiver of the audiometer was placed in the external ear canal opposite to that of the probe ear. TEOAEs were measured by presenting 50 dBSL NBN with a center frequency of 1 kHz, 2 kHz, 3 kHz, 4 kHz respectively through the insert receiver.
3. TEOAEs were once again measured by presenting BBN in the contralateral ear at 50 dB SL.

A minimum of 1 minute gap was given between any two recordings, to reduce the influence of one recording over other recording.

The overall TEOAE amplitudes and amplitudes across frequencies were recorded after each recording. The data obtained was tabulated and statistically analyzed using paired t-test, to see the significance of difference between the means across frequencies.

RESULTS AND DISCUSSION

RESULTS

The mean (M) and Standard Deviation (SD) values of TEOAE response without and with contralateral NBN and BBN were calculated. The data was statistically analyzed to find the significance of difference between the means of two experimental conditions.

Results obtained with NBN of center frequency at 1 kHz, 2kHz, 3 kHz and 4 kHz and BBN were as follows:

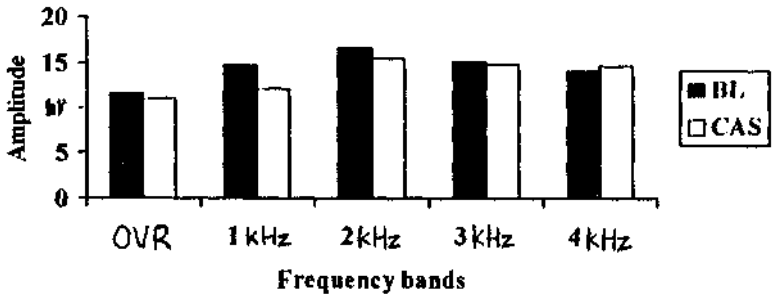
TEOAE amplitude without and with 1 kHz contralateral NBN

The overall TEOAE response and TEOAE response across frequency bands without and with contralateral 1 kHz NBN were obtained. The following table-1 and graph-5 shows the mean values of TEOAE amplitude in two conditions. The table also depicts the SD and t-values of TEOAE response.

Table: 1: Depicts the Mean, SD and t-values of TEOAE response without and with 1 kHz NBN

	OVR		1 kHz		2 kHz		3 kHz		4 kHz	
	BL	CAS	BL	CAS	BL	CAS	BL	CAS	BL	CAS
M	11.60	11.07	14.78	12.12	16.68	15.5	15.06	14.87	14.15	14.56
SD	4.61	4.57	5.75	5.09	7.38	6.95	6.35	6.28	5.41	6.42
't'	5.273		5.60		1.92		0.46		-0.96	

Graph-5: Mean values of TEOAE response without & with 1 kHz NBN



BL-Baseline; CA5 — Contralateral Acoustic Stimulation

As shown in the table and graph, there was difference in mean amplitude at all the frequency bands and overall (OVR) amplitude between the two experimental conditions. The mean amplitude was higher without the contralateral noise compared to with contralateral noise, except for 4 kHz frequency band as can be seen in fig.3. The difference was statistically significant in OVR and at 1 kHz frequency band (at 0.01 level).

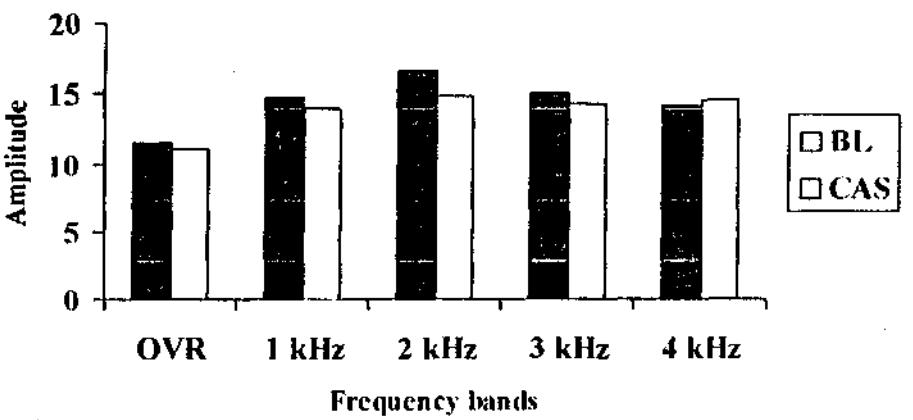
Among the 32 subjects, around 92% of the subjects showed a decrease in the amplitude at 1 kHz frequency band when compared to baseline TEOAE response. The difference in amplitude ranged from **1** dB SPL to 4 dB SPL.

TEOAE amplitude without and with 2 kHz contralateral NBN

Table-2 : Depicts the Mean, SD and t-values of TEOAE response without and with 2 kHz NBN

	OVR		1kHz		2 kHz		3 kHz		4 kHz	
	BL	CAS	BL	CAS	BL	CAS	BL	CAS	BL	CAS
M	11.60	11.13	14.78	14.03	16.68	14.90	15.06	14.34	14.15	14.59
SD	4.61	4.6	5.75	5.7	7.38	7.3	6.35	6.7	5.41	5.6
't'	4.39		1.22		3.73		1.15		-0.97	

Graph-6: Mean values of TEOAE response without & with 2 kHz NBN



The above mentioned table-2 and graph-6 shows, a decrease in the overall amplitude and amplitude across frequency bands with the CAS except at 4 kHz. The decrease in amplitude was maximum at 2 kHz compared to

other frequency bands as can be seen in fig.4. However, the difference was statistically significant at OVR and only 2 kHz frequency band. At 4 kHz there was a slight increase in the amplitude with contralateral NBN.

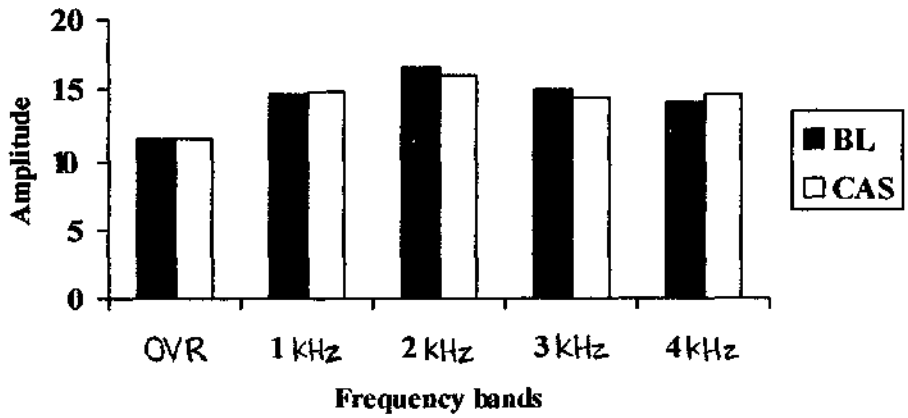
Around 71% of the subjects showed a decrease in the TEOAE amplitude at 2 kHz frequency band. The difference in amplitude ranged between 1 dB to 6 dB SPL.

TEOAE amplitude without and with 3 kHz contralateral NBN

Table-3 : Depicts the Mean, SD and t-values of TEOAE response without and with 3 kHz NBN

	OVR		1kHz		2 kHz		3 kHz		4 kHz	
	BL	CAS	BL	CAS	BL	CAS	BL	CAS	BL	CAS
M	11.60	11.58	14.78	14.87	16.68	16.06	15.06	14.5	14.15	14.78
SD	4.61	4.72	5.75	4.91	7.38	6.76	6.35	6.34	5.41	5.99
't'	0.14		-0.14		1.26		1.33		-1.32	

Graph-7: Mean values of TEOAE response without & *wi*th 3kHz NBN



The inspection of table-3 and graph-7 reveals a differential effect of 3 kHz NBN among the components of TEOAEs. There was a slight change in the overall amplitude of TEOAEs with the introduction of noise. There was almost equal decrease in the amplitude among 2 kHz and 3 kHz. But, 1 kHz and 4 kHz frequency band showed a slight increase in the amplitude as can be seen in fig.5. However, there was no statistically significant difference at any of the frequency bands.

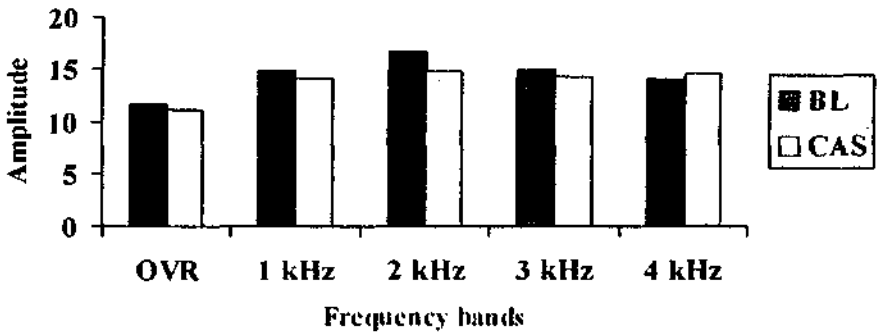
Only 47% of subjects showed a decrease in the amplitude at 3 kHz frequency band ranging from 1 dB to 3 dB SPL

TEOAE amplitude without and with 4 kHz contralateral narrow band noise

Table-4 : Depicts the Mean, SD and t-values of TEOAE response without and with 4 kHz NBN

	OVR		1kHz		2 kHz		3 kHz		4 kHz	
	BL	CAS	BL	CAS	BL	CAS	BL	CAS	BL	CAS
M	11.60	11.43	14.78	14.2	16.68	15.28	15.06	14.5	14.15	14.81
SD	4.61	4.63	5.75	6.23	7.38	6.66	6.35	6.27	5.41	5.73
't'	1.87		0.77		2.74		1.4		-1.6	

Graph-8: Mean values of TEOAE response without & with 4 kHzNBN



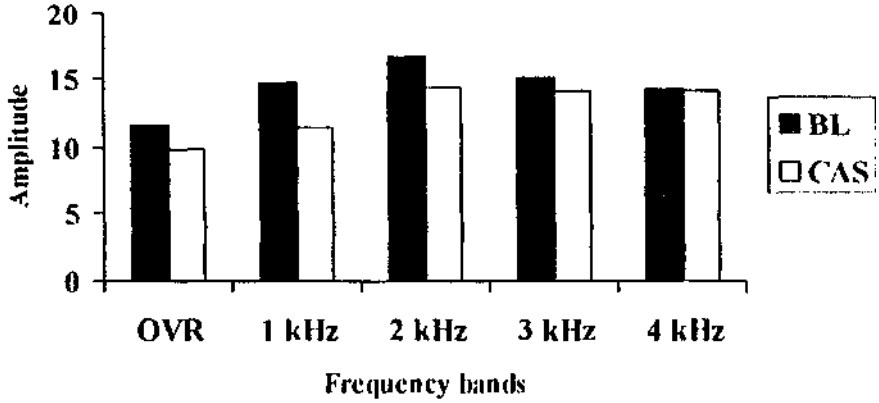
As it is evident in the table-4 and graph-8, the 4 kHz contralateral NBN induced a decrease in the overall amplitude at all the frequency bands except at 4 kHz. The equal amount of decrease was seen at 1 kHz and 3 kHz whereas, reduction was maximum and statistically significant (0.01 level) at 2 kHz frequency band. There was an increase in the amplitude at 4 kHz frequency band with the CAS as shown in fig.6.

TEOAE amplitude without and with BBN

Table-5 : Depicts the Mean, SD and t-values of TEOAE response without and with BBN

	OVR		1kHz		2 kHz		3 kHz		4 kHz	
	BL	CAS	BL	CAS	BL	CAS	BL	CAS	BL	CAS
M	11.60	9.85	14.78	11.40	16.68	14.34	15.06	14.06	14.15	14.0
SD	4.61	4.58	5.75	5.07	7.38	7.09	6.35	5.88	5.41	5.42
't'	8.34		4. 58		3.84		1.75		0.39	

Graph-9: Mean values of TEOAE response without & with BBN



Results indicate a decrease in amplitude at all the frequency bands and overall amplitude with the Contralateral BBN. The difference seen was maximum at 1 kHz followed by 2 KHz, 3 kHz and 4 kHz respectively as can be seen in fig.7. A statistically significance for the decrease was obtained at 1 kHz and 2 kHz and overall amplitude at 0.01 level.

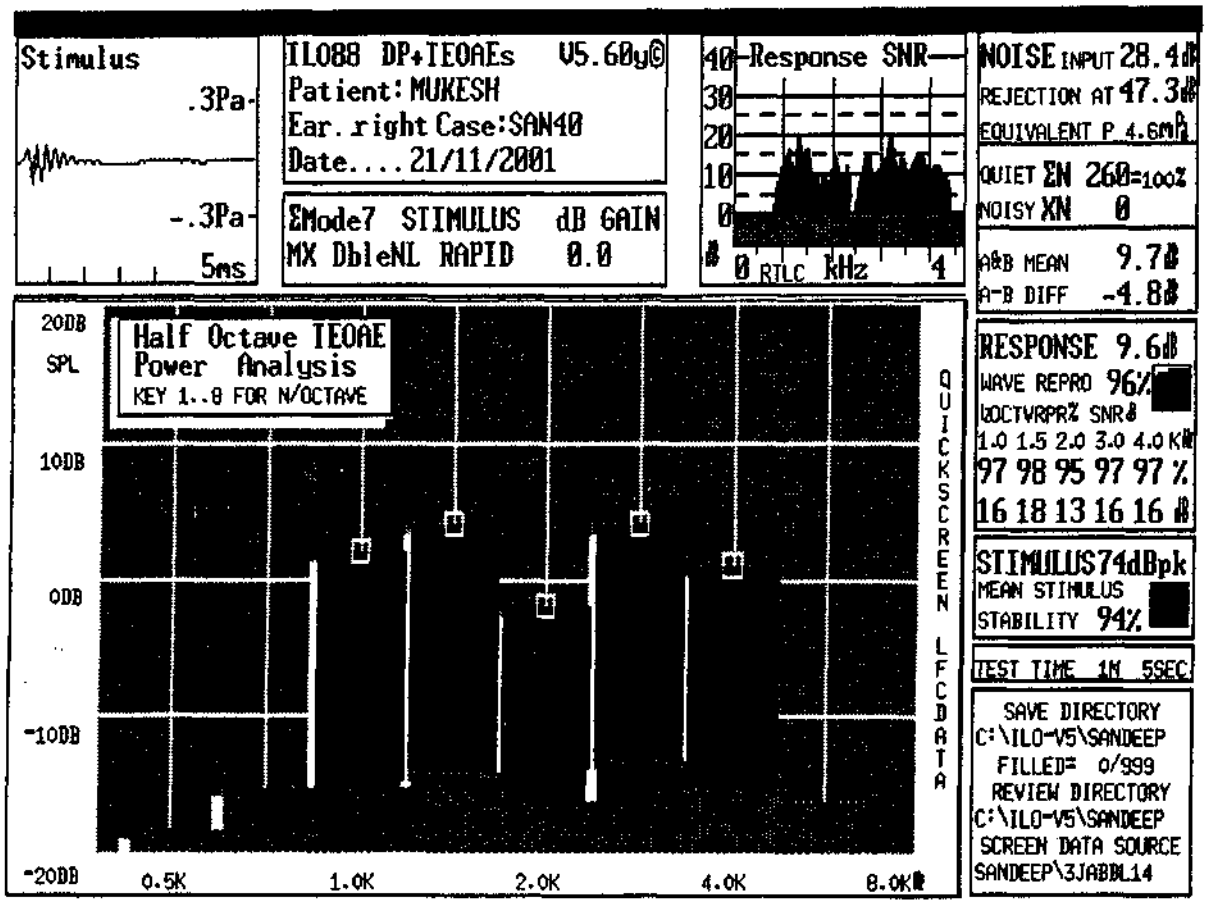


Fig.A: TEOAEs at different frequency bands without contralateral noise.

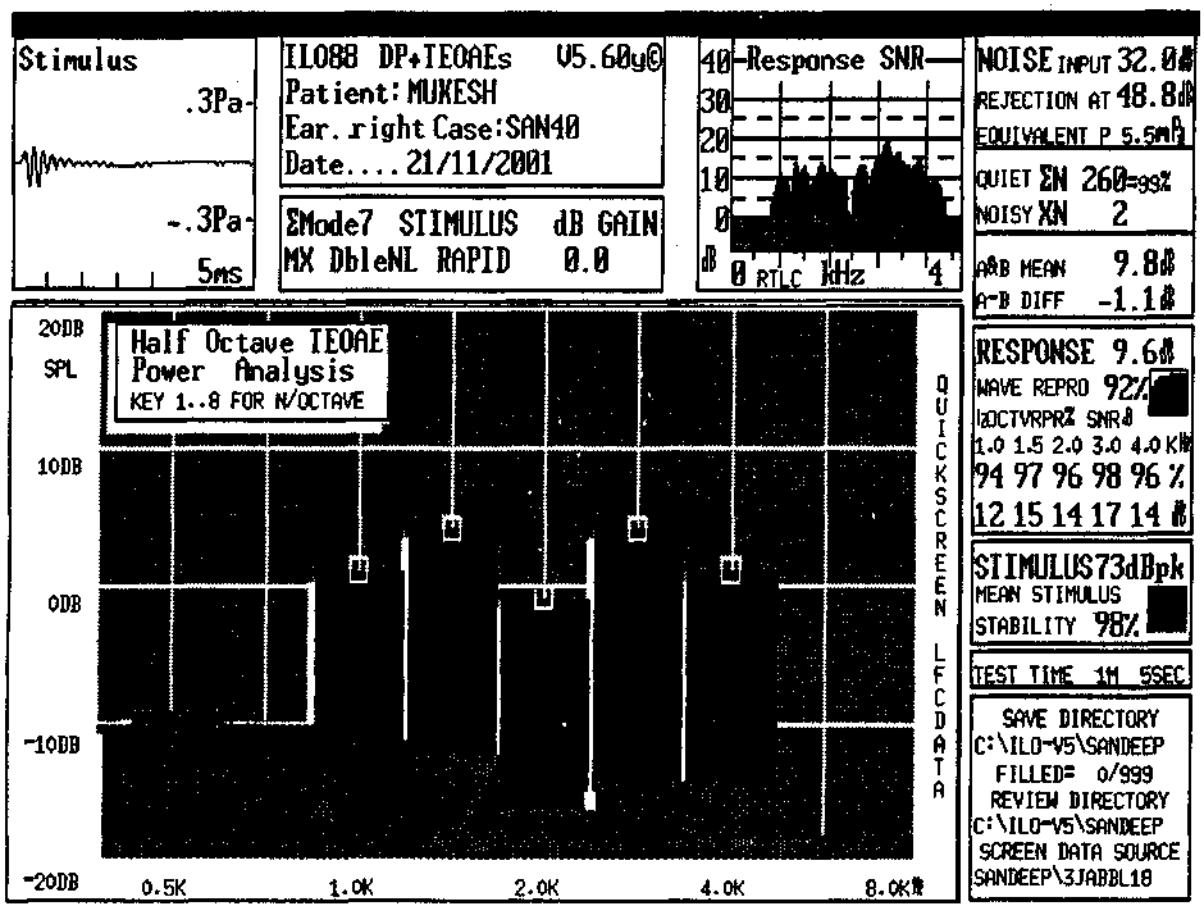


Fig.3: TEOAEs at different frequency bands with 1kHz NBN in the contralateral ear.

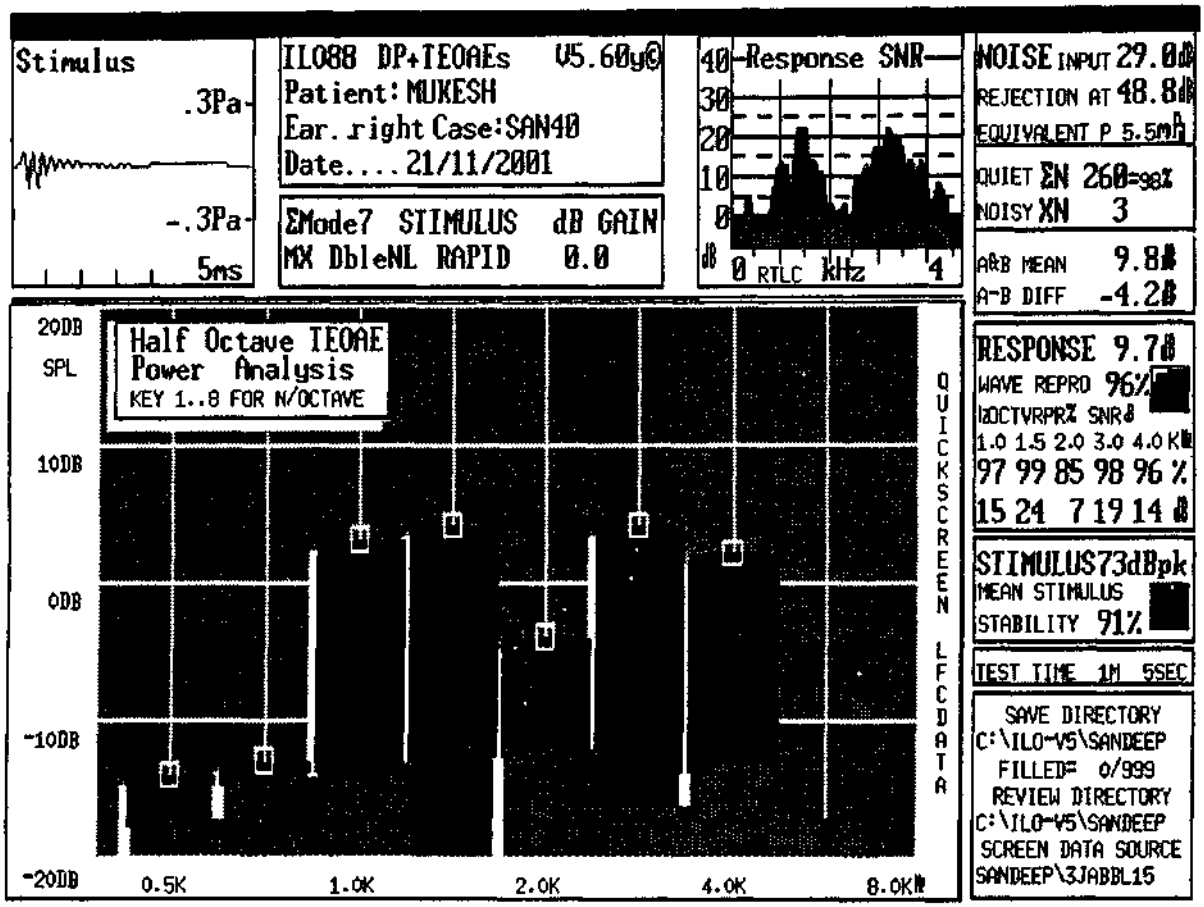


Fig.4: TEOAEs at different frequency bands with contralateral 2kHz NBN.

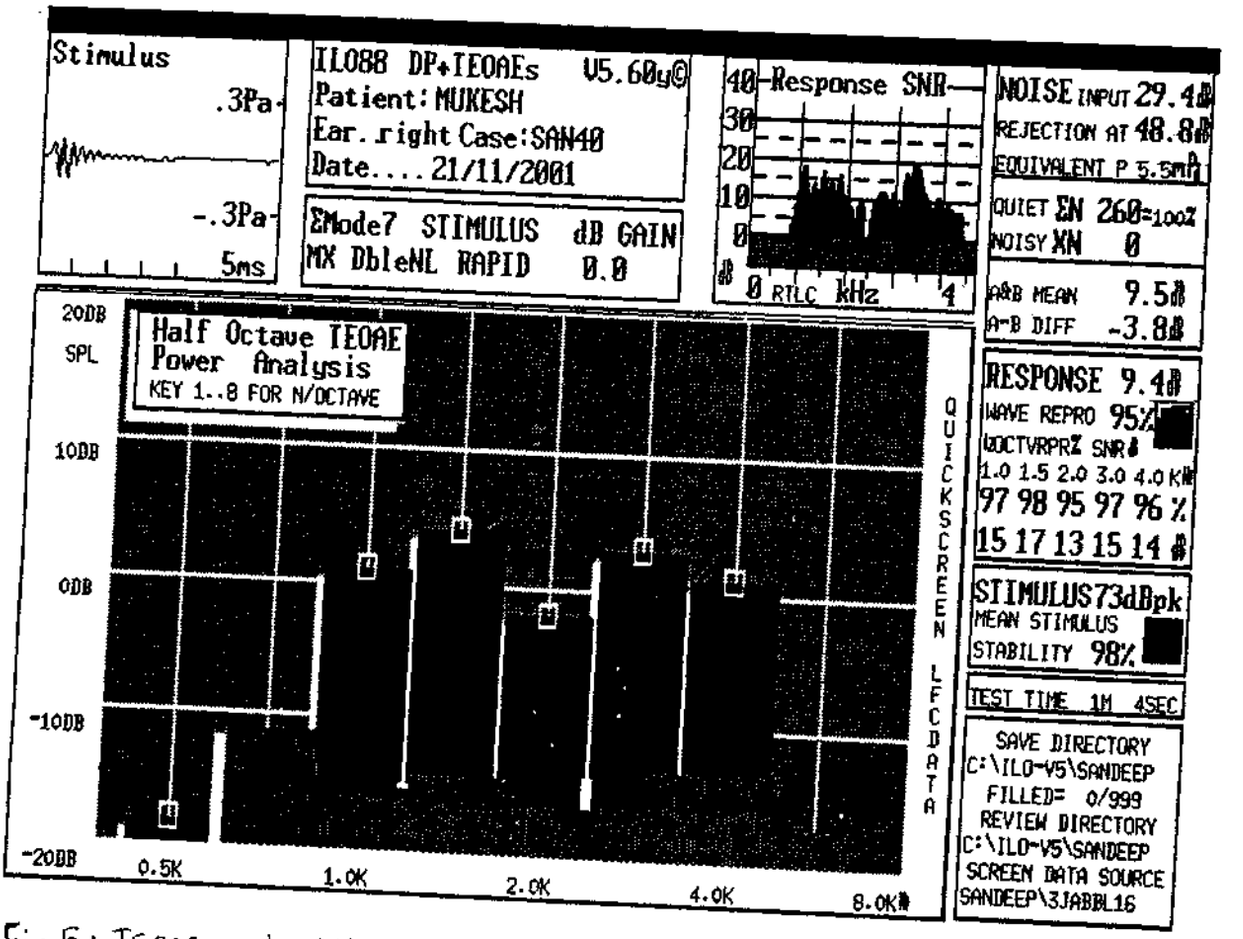


Fig.5: TEOAEs at different frequency bands with contralateral 3KHz NBN.

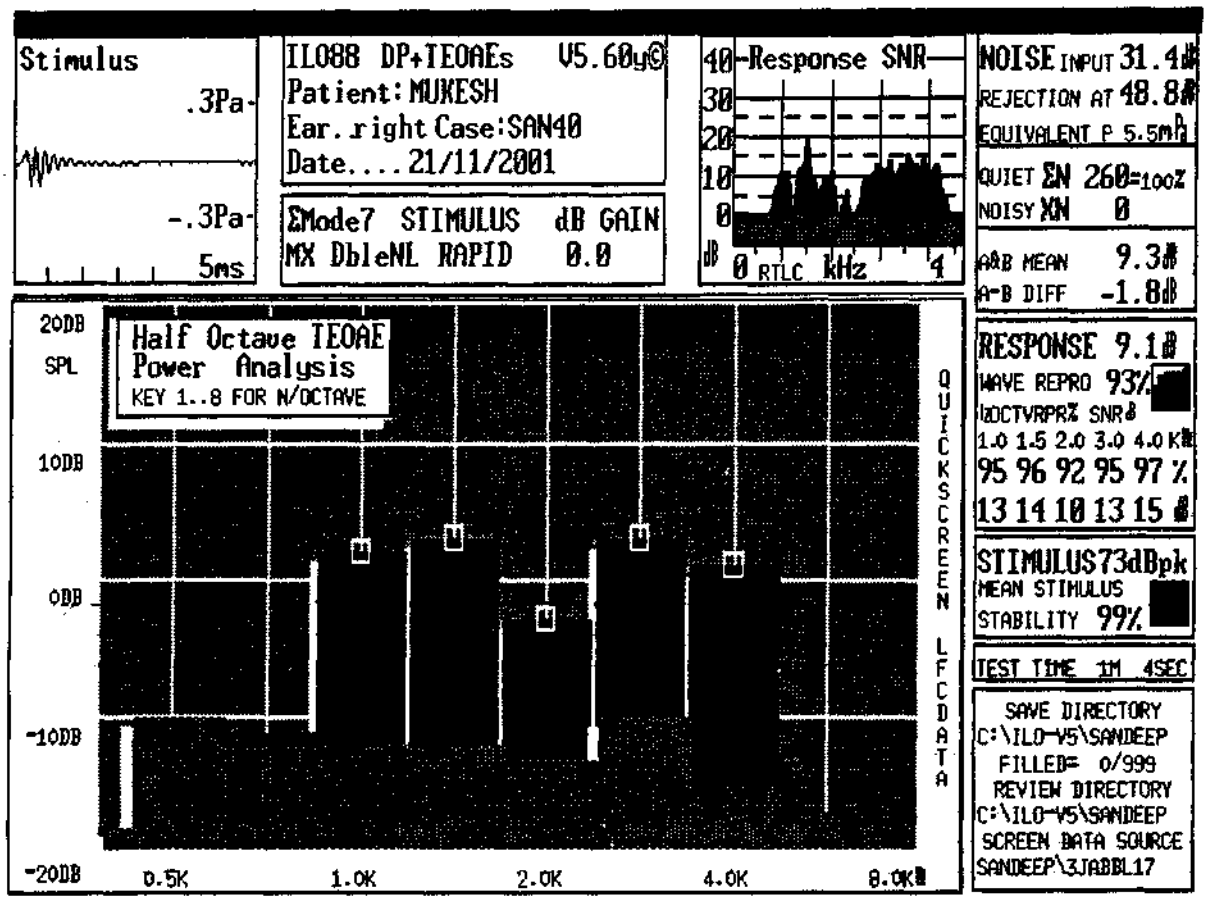


Fig. 6: TEOAEs at different frequency bands with contralateral 4kHz NBN.

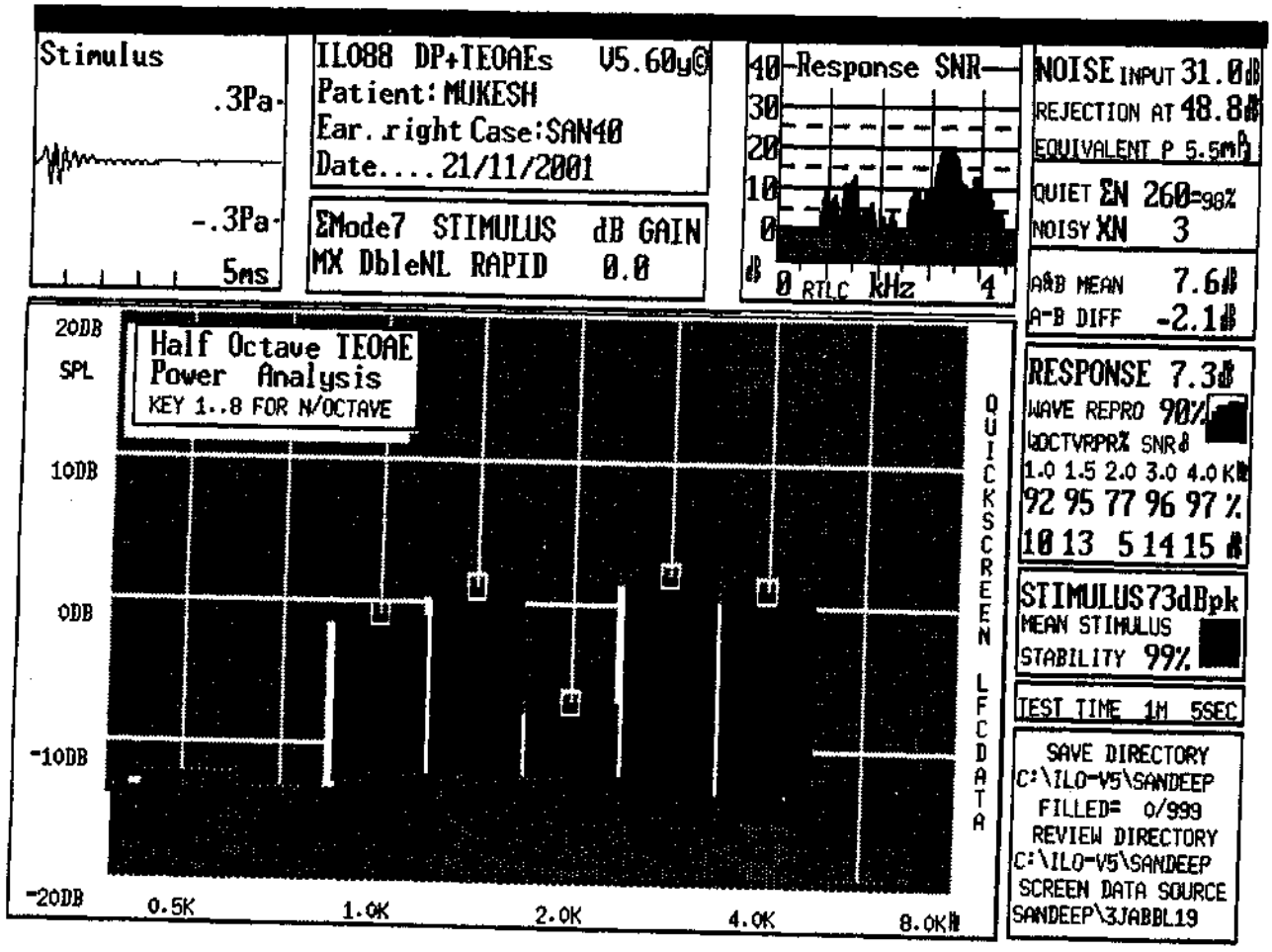


Fig. 7: TEOAEs at different frequency bands with contralateral BBN.

DISCUSSION

The results of the present study showed that -

1) The emissions evoked by non-linear clicks at 75 dB SPL can be suppressed by both contralateral NBN and BBN.
2) Among the NBN, 1 kHz and 2 kHz NBN when presented in contralateral ear had maximum suppression in the respective frequency bands compared to other two frequency bands. However, when 3 kHz and 4 kHz NBN were used, this trend was not seen.
3) With the contralateral BBN, there was a decrease in TEOAE suppression with the increase in the band frequency. But, the suppression seen was more than that seen with Contralateral NBN of any characteristics frequency band.

Contralateral suppression of TEOAEs:

The TEOAE suppression seen in the presence of contralateral NBN and BBN is attributed to the change in the electrical/mechanical properties of the cochlea brought by the medial efferent system. The mechanism by which medial efferent stimulation alters the electrical properties of the cochlea and thereby OAEs, can be understood by the following explanation.

The OHCs are securely attached to the reticular lamina at their apex and to the Dieter's cells (Dallos, 1992; Santi, 1988) at their perinuclear region which are also capable of stretching (LePage, 1989). When there is medial efferent stimulation, these fibres release Ach and open Ca^{++} dependent channels. This action is followed by a conspicuous hyperpolarizing Ca^{++} dependent K^{+} efflux, resulting in an elongation of the OHC. The elongation probably occurs through osmotic factors. A change in OHC length (elongation) or even a reduction in a depolarizing contraction, would conceivably modify the separation between the reticular lamina and basilar membrane (Dallos, 1992; Neely, 1989; Sziklaiv & Dallos, 1993). Results of an investigation demonstrated that the OHCs can alter the shape of the basilar membrane independent of the travelling wave changing its relative position to the rectorial membrane along selected tonotropic regions (LePage, 1989). Therefore Ach-induced alterations in the shape or compliance of the OHCs serve to damp micromechanical activity and reduce the sensitivity of the basilar membrane response (Geisler, 1991; Mountain & Cody, 1989; Neely & Kim, 1986; Rhode, 1984; Zenner, et al., 1989).

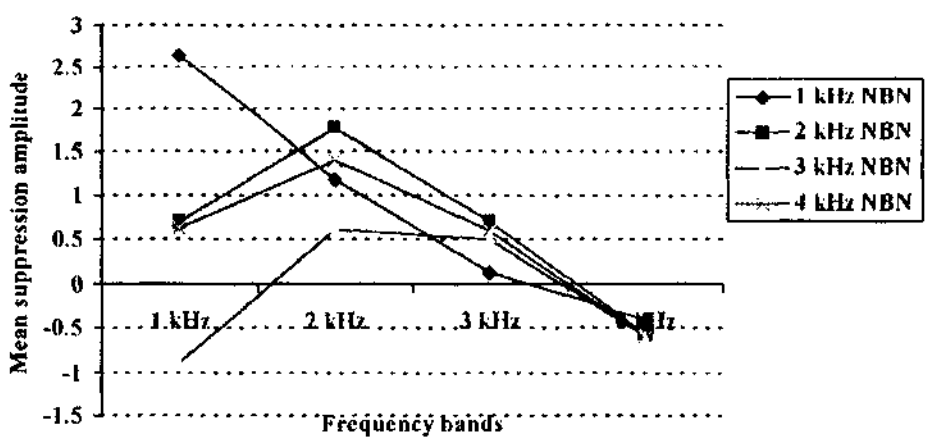
The other mechanisms by which medial efferents might affect OAEs are -

It affects the operation of OHCs i.e., it may reduce the OHC receptor potential, which would reduce OHC morion (Santo-sachi & Dilger, 1988).

Efferent induced contractions of OHCs distort the organ of coiti, thereby lowering the gain of the cochlear amplifier (Rajan, 1990).

Frequency Specificity of TFOAE Suppression

The following graph depicts the suppression of amplitude across four frequency bands (1 kHz, 2 kHz, 3 kHz & 4 kHz) recorded with NBN of CF at 1 kHz, 2 kHz, 3 kHz and 4 kHz.



Graph 10: Depicts the frequency specificity of TEOAE suppression.

Inspection of the graph reveals that, a frequency specific suppression was seen with 1 kHz and 2 kHz contralateral NBN. But this was not seen at

This suggests some degree of frequency specificity in the efferent auditory pathway for the contralateral noise. Results of the present study is in-par with the study by Veuillet, et al., (1992), who found a strong frequency specificity of the suppression produced by 50 dB SPL NBN with CF between 0.9 and 2.9 kHz. It suggests that, for contralateral tones, the greatest inhibition of the response to an ipsilateral tone is produced when the contralateral tone is approximately the same frequency as the ipsilateral tone. Presumably this is because each medial efferent fiber projects to a region of the cochlea tuned to the efferent best frequency (BF).

But the question arises as to why there is significant suppression only at 1kHz and 2 kHz. This could by justified by the following explanation.

Efferent effects evoked by contralateral sounds are largest in the 1 to 2 kHz region of the cochlea which contrast with shock evoked efferent effects which ear greatest from 2 to 10 kHz. This may be because contralateral reflex evoked by moderate level sound is mediated by uncrossed medial efferent fibers and that their distribution is peaked in the center of the cochlea (Guinan, Warr & Norris, 1984; Liberman, Dodds & Pierce, 1990). This may not be only due to the crossed efferents, as they are biased towards the basal region of the cochlea (Guinan, et al., 1984; Liberman, et al., 1990) or it can be due to the combined effect of both crossed and uncrossed MOCB. The maximum rates evoked by contralateral sound are greatest in a broad range

around 2 kHz for sounds up to 90 dB SPL. However 90 dB SPL in much greater than the levels actually used in contralaternl sound experiments. Since the rates of medial efferent fibers with BFs below 2 kHz often saturate at sound levels below 90 dB SPL (Liberman & Brown, 1986; Liberman, 1988a), lowering the contralateral sound will preferentially lower the rates of fibers with BFs above 2 kHz.

The net effect of all of these factors would be to produce large, contralaterally evoked efferent inhibitions primarily in the 1 kHz lo 2 kHz region. This observation is also consistent with the interpretation that suppression of TEOAEs was due to medial efferents, not due to lateral efferents, because lateral efferents innervation is relatively constant throughout the length of the cochlea (Guinan, et al., 1984; Liberman, et al., 1990).

In contrast to the other frequency bands, TEOAE response at 4 kHz showed a consistent increase in amplitude with all the NBN. The underlying physiology for this is yet to be known.

Differential effects of NBN and BBN on suppression of TEOAEs:

A statistically significant TEOAE suppression was seen both with NBN and BBN. However, suppression was more with BBN compared to NBN. This is due to increase in OCB activation with the increase in stimulus bandwidth, when the overall energy is maintained constant. This can be explained by the spatial integration properties of certain neurons in the cochlear nucleus (Evans & Zhoo, 1991; Young, Spirou, Rice & Voigt, 1992). Onset units have large tuning curves with occasionally inhibitory lateral bands in their response maps. These units are able to carry out spatial integration of several auditory nerve fiber responses of different best frequencies due to which OCB activation increases with stimulus bandwidth, whether or not overall energy is kept constant. Similar results were also reported by Norman & Thornton (1993). The significant suppression seen only at 1 kHz & 2 kHz again supports the notion that uncrossed MOC innervation is largest near the center of the cochlea (Guinan, et al., 1984; Liberman, et al., 1990).

SUMMARY AND CONCLUSION

The function of the OC bundle has remained controversial since its discovery as an anatomical pathway 50 years ago (Rasmussen, 1946). However, it is clear that activation of medial olivocochlear fibers has an inhibitory effect on the auditory periphery (Abdal, Ma & Sininger, 1999). The OAE which reflect the OHC integrity, provide an appropriate index of changes in cochlear function as MOC fibers are activated. Since, the anatomical distribution of medial effercnts along the basilar membrane shows some frequency specificity, the contralateral suppression of TEOAEs mediated by medial efferents is also expected to be frequency specific. Also, the frequency at which the TEOAE suppression is more, might help to determine whether the process of suppression is mediated by crossed MOC fibers or uncrossed MOC fibers. Hence the present study was taken up -

1. To evaluate the frequency specificity of MOC neurons based on contralateral suppression of TEOAEs.
2. To examine whether the effect on TEOAEs is due to ipsilateral or contralateral MOC fibers.
3. To see the differential effects of NBN and BBN on suppression of TEOAEs.

Thirty two normal hearing adults were included in the study. The overall amplitude and amplitudes of TEOAEs across frequency bands without

and with contralateral BBN and NBN (1 kHz, 2 kHz, 3 kHz, 4 kHz)were recorded. The presentation of Contralateral noise was at 50 dB SL through the insert receiver to avoid crossover.

Results indicated : (1) frequency specific suppression of TEOAEs at 1 kHz and 2 kHz. (2) frequency specificity decreased within-creasing frequency. (3) BBN produced a greater efferent stimulation than NBN. Among the NBN only 1 kHz NBN & *2* kHz NBN produced statistically significant efferent inhibition.

Conclusion : This suppression effect seen can be attributed to the change in the electrical and mechanical properties of the cochlea brought by the efferent system. Since, MOCB innervation is largest near the center of the cochlea (Guinan, Warr & Norris, 1984). Liberman, Dodds & Pierce (1990) more suppression was seen only in mid frequencies (1 kHz & 2 kHz) but not at higher frequencies. Hence it can be concluded that the uncrossed MOCB plays a major role in Contralateral suppression. 1 lowever BBN showed more suppressive effect because OCB activation increases with stimulus bandwidth, even when overall energy is maintained constant (Norman & Thornton, 1993).

Clinical applications :

To assess the physiology of efferent auditory pathway.

To assess the frequency specificity of efferent pathway.

To assess the speech perception in noise.

Limitation : Study was done using TEOAEs but the literature shows less frequency specificity with TEOAEs compared to other evoked OAEs. Frequencies specificity was assessed only at mid and higher frequencies, low frequencies are not considered.

Further Study : Further study can be taken up

To compare the frequency specificity across different evoked OAEs and

To study the frequency specificity at low frequencies (below 1 kHz), if the facilities are available.

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