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THE EFFECT OF NOISE BANDWIDTH ON THE CONTRALATERAL
SUPPRESSION OF TRANSIENT EVOKED OTOACOUSTIC
EMISSIONS

by

David Scott Velenovsky

A Dissertation Submitted to the Faculty of the
DEPARTMENT OF SPEECH AND HEARING SCIENCES

In partial fulfillment of the Requirements
For the Degree of

DOCTOR OF PHILOSOPHY

In the Graduate College
THE UNIVERSITY OF ARIZONA

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As members of the Final Examination Committee, we certify that we have read the dissertation prepared by David Scott Velenovsky

entitled The Effect of Noise Bandwidth on the Contralateral
Suppression of Transient Evoked Otoacoustic Emissions

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ABSTRACT

The acoustic reflex system (AR) and the influence of the olivocochlear bundle (OCB) affect the ear's response to sound. Both systems are controlled by efferent neurons that originate in the superior olivary complex (SOC). The AR system affects middle ear function through its action on the stapedius muscle, and the OCB affects cochlear function through its action on outer hair cells (OHC). The AR is sensitive to the loudness of a stimulus; that is to say, changes in sound pressure level (SPL) and bandwidth of a stimulus affect the threshold and magnitude of stapedius muscle contraction. Investigators have shown that the magnitude of the OCB effect is influenced by changes in SPL.

Transient evoked otoacoustic emissions (TEOAEs) were collected from 10 women in quiet and in the presence of three different contralateral noise bands centered at 2000 Hz. The NB (100 Hz bandwidth) and WB (2200 Hz bandwidth) noises were at 60 dB SPL. The SPL of the EQ (100 Hz bandwidth) noise was adjusted such that it was equal in loudness to the WB noise.

The results of the present study indicate that an increase in loudness as associated by an increase in noise bandwidth affects the contralateral suppression of TEOAEs. Only the WB noise was associated with a reduction of TEOAE amplitudes. It is believed that this effect results because the WB noise has greater effective energy representation across frequency on the basilar membrane. Although the spectrum level of the WB noise is lower than that of the NB noise at the transducer, it receives more gain from the action of the cochlear amplifier than the NB noise and thus has greater energy representation on the cochlear partition. While the effective energy level on the basilar membrane may have been essentially the same for the WB and EQ noises as a result of the action of the cochlear amplifier, the WB noise was the more effective suppressor because its energy was summed across multiple critical bands. The energy in the EQ noise was confined to a single critical band.

INTRODUCTION

At least two feedback systems influence activity of the afferent auditory system: the acoustic reflex mechanism (AR) and the olivocochlear bundle (OCB) (Borg, 1973; Galambos, 1956). The AR exerts its control on the middle ear system by altering the impedance of the middle ear as a result of contraction of the stapedius muscle (Møller, 1962; von Békésy, 1960). The action of the OCB is not as clearly understood. Medial olivocochlear (MOC) fibers of this system are known to terminate on outer hair cells (OHC) and lateral olivocochlear fibers (LOC) terminate on auditory afferent fibers of inner hair cells (IHC) (Gacek, 1961; Ishii, & Balogh, 1968; Nakai, & Igarashi, 1974; Rasmussen, 1953; Smith, 1961; Smith & Rasmussen, 1963; Spoendlin, 1963; Warr, Guinan, & White, 1986). Early investigations using Guinea pig and cat demonstrated that electrical or acoustical stimulation of the OCB results in attenuation of the amplitude of the whole nerve action potential of the auditory nerve, but the mechanism of this attenuation was unknown (Fex, 1967; Galambos, 1956; Geisler, 1974; Wiederhold, & Kiang, 1970). Recently, the influence of the OCB on otoacoustic emissions (OAEs) has been investigated extensively. This is

due in large part to the belief that OAEs are influenced by OCB neurons (Berlin, Hood, Hurley, & Wen, 1994; Collet, 1993; Collet et al., 1990).

The ARS and OCB efferent systems are similar in several respects: a) the effects of both systems are mediated by neurons that originate in the superior olivary complex (SOC), specifically the medial superior olive (MSO) (Borg, 1973; Rasmussen, 1946; Spoendlin, 1963); b) fibers from MSO neurons of both the ARS and OCB project bilaterally (Borg, 1973; Rasmussen, 1946; Spoendlin, 1963); c) both systems can be inhibitory (Borg, 1973; Galambos, 1956); d) both systems can be activated with contralateral stimulation (Collet et al., 1990; Desmedt, 1962; Møller, 1962).

There are also similarities between the two systems in their responses to pure tone and noise stimuli of varying bandwidths and intensities. The magnitude of the impedance change due to the acoustic reflex varies directly with the SPL of the stimulus in normal human ears (Møller, 1962). It has also been revealed that, for a given intensity, broadband noise will elicit a larger change in impedance than that associated with a pure tone (Block & Wightman, 1977; Flottorp, Djupesland, & Winther, 1971;

Margolis & Popelka, 1975). This implies that the AR is sensitive to a change in loudness that results from increasing the bandwidth of a stimulus maintained at a constant SPL. Others have demonstrated that when stimuli such as pure tones and noise bands are matched for loudness, the magnitudes of the elicited acoustic reflexes are similar (Block et al., 1977; Gorga, Lilly, & Lenth, 1980). Some investigators have hypothesized that a critical band exists for the acoustic reflex (Flottorp et al., 1971).

Several investigators have studied the influence of contralateral stimulation on TEOAEs (Berlin et al., 1993b; Collet, 1993; Collet et al., 1990; Collet, Veuillet, Bene, & Morgon, 1992; Hood, Berlin, Hurley, Cecola, & Bell, 1996; Ryan, & Kemp, 1996). Investigators have determined that as the SPL of a contralateral stimulus is increased, the amplitude of TEOAEs is reduced (Collet et al., 1990; Hood et al., 1996). One study has revealed that TEOAE amplitude decreases as the bandwidth of a contralateral stimulus is increased (Norman & Thornton, 1993).

Given the similarities between the acoustic reflex system and the mechanism of contralateral suppression of emissions, it is reasonable to ask

if TEOAE contralateral suppression effects are similarly responsive to loudness.

REVIEW OF THE LITERATURE

Otoacoustic Emissions

Predicted over 50 years ago by Gold (Gold, 1948), otoacoustic emissions (OAEs) were first demonstrated by Kemp (1978). Kemp reported that upon acoustic stimulation, a very small amount of energy was emitted from the cochlea by way of the middle ear system (Kemp, 1978). Kemp's discovery had a profound effect on auditory theory. Historically, the cochlea was viewed as a passive organ that converted mechanical vibrations into neural energy (Brownell, 1990; Johnstone, Patuzzi, & Yates, 1986; von Békésy, 1960). Von Békésy's pioneering work led to an elegant explanation of cochlear function via the traveling wave (von Békésy, 1960), which became the foundation of modern auditory theory (Kim, 1986). However, a clear understanding of cochlear function eluded researchers for many years after von Békésy's discoveries. This was due to the surgical and technical difficulties involved in measuring the function of a living cochlea (Kemp, 1979). Kemp's (1978) discovery of the existence of an active source of energy in the cochlea, and Brownell's (1985) discovery of outer hair cell (OHC) motility gave hearing scientists a clearer picture of cochlear function.

Stimulated otoacoustic emissions are believed to result from a motile response of the outer hair cell (OHC) (Brownell, 1985; 1990). Kim (1986) hypothesized that there are two types of motile mechanisms to control cochlear biomechanics: a) a fast motile mechanism located in the cilia bundle that follows the cycle-by-cycle events at the characteristic frequency of the hair cell; and b) a slow motile mechanism, located in the specialized elongated cylindrical portion of the hair cell, which is thought to control the operating point of the mechanical amplifying action of the fast motile mechanism. Simply put, the slow mechanism brings the mechanical amplifying action of the fast process to bear on the inner hair cells (IHC), thus activating them (Kim, 1986). If this process is eliminated, then IHCs are not stimulated until the motion of the cochlear partition is sufficient to activate them directly (Kiang, Liberman, Sewell, & Guinan, 1986).

Suppression Effects

In 1946, Rasmussen described the “efferent cochlear bundle” in cats and later determined that efferent fibers terminated in the cochlea (Rasmussen, 1946, 1953). Other investigators revealed that efferent fibers terminated directly on outer hair cells in the cat, Guinea pig and squirrel monkey (

Ishii et al., 1968; Kimura & Wersall, 1962; Nakai et al., 1974; Smith, 1961; Smith et al., 1963; Spoendlin, 1963). Structural similarities between the auditory efferent system in humans and other mammals have been reported (Gacek, 1961). Prior to the discovery of OAEs, contralateral suppression effects were studied in laboratory animals by measuring changes in auditory nerve responses that resulted from electrical or contralateral acoustic stimulation of the OCB. The experimental procedures for the aforementioned methods were invasive and complicated (Gifford & Guinan, 1987; Guinan & Gifford, 1988a, 1988b, 1988c; Wiederhold, 1970; Wiederhold & Peake, 1966). Galambos was the first to demonstrate that electrical stimulation of the contralateral OCB resulted in attenuation of auditory nerve responses to acoustic stimuli in the cat (Galambos, 1956).

The discovery of OAEs made the non-invasive study of auditory efferent effects at the cochlear level possible (Berlin et al., 1993b; Collet et al., 1990). Investigators have turned their attention to the study of the effects of contralateral stimuli on various parameters of evoked otoacoustic emissions in humans and in animals and have demonstrated that stimulation of the ear opposite the one receiving the evoking stimulus reduces the

amplitude of the otoacoustic emission (Berlin, Hood, Cecola, Jackson, & Szabo, 1993a; Berlin et al., 1994; Berlin et al., 1993b; Collet et al., 1990; Collet et al., 1992; Harrison & Burns, 1993; Kujawa, Glatcke, Fallon, & Bobbin, 1992; Puel & Rebillard, 1990; Ryan, Kemp, & Hinchcliffe, 1991; Veuillet, Collet, & Duclaux, 1991). This effect is believed to be mediated by the MOC system (Berlin et al., 1994; Collet, 1993; Collet et al., 1990; Giraud, Collet, Chery-Croze, Magnan, & Chays, 1995) , and suggests that the contralateral suppression of auditory nerve responses described previously may result from activity at the cochlear level.

The suppression effect on evoked otoacoustic emissions in humans is a small but consistent decrease of 1-4 dB in overall emission amplitude in the presence of a contralateral acoustic stimulus (Berlin et al., 1993b, 1994; Collet et al., 1990, 1992; Ryan et al., 1991, 1996; Veuillet et al., 1991; Veuillet, Duverdy-Bertholon, & Collet, 1996). Collet is credited as the first person to describe contralateral suppression of evoked otoacoustic emissions in humans (Berlin et al., 1993b; Collet et al., 1990). Collet's group determined that white noise used as a contralateral stimulus at 30 dB SPL and greater resulted in suppression of TEOAE amplitudes and as the level of the contralateral stimulus was increased, the amplitude of the

TEOAE further decreased (Collet et al., 1990). The most significant suppression effects occurred between 1000 and 4000 Hz in the TEOAE spectra (Collet et al., 1990). Berlin et al. (1993) examined the suppression effects of various contralateral stimuli by measuring the reduction of TEOAE amplitudes at discrete time intervals from 2 to 20 milliseconds after stimulus onset. Narrow bands of noise were the most effective suppressors, pure-tones the least effective (Berlin et al., 1993b). All noise bands had similar suppression effects across frequency. Berlin et al.'s results imply that the contralateral effect is not tuned (Berlin et al., 1993b). Norman and Thornton (1993) similarly found a lack of tuning of the contralateral effect on TEOAEs (Norman & Thornton, 1993). In Berlin et al.'s study the greatest TEOAE amplitude reduction resulting from contralateral stimulation with narrowband noise, clicks or puretones appeared 8 ms after stimulation and occurred between 8 and 16 ms depending on the type of contralateral stimulus (Berlin et al., 1993b). Transient evoked otoacoustic emissions are most susceptible to contralateral suppression effects when the intensity of the click stimulus is below 65 dB SPL (Hood et al., 1996; Veuillet et al., 1996).

Animal studies have provided evidence regarding the source of OAE contralateral suppression effects. Puel and Rebillard (1990) determined that no distortion product otoacoustic emission (DPOAE) contralateral suppression effects were measurable in Guinea pigs after a midline sagittal section of the brainstem, cutting all crossed fibers, was performed. Kujawa et al. (1992) reported that after applying antagonists to acetylcholine, a suspected neurotransmitter in the MOC system, contralateral suppression of DPOAEs was blocked. When the antagonists were removed, the effect was reversed (Kujawa et al., 1992). While the aforementioned studies involved DPOAEs, the findings support the hypothesis that contralateral suppression of OAEs is mediated by the MOC.

Auditory Efferents

Efferent innervation of the cochlea in mammals is provided by the olivocochlear bundle (OCB), which comprises two basic groups of neurons, the lateral olivocochlear bundle (LOC) and the medial olivocochlear bundle (MOC) (Guinan, Warr, & Norris, 1983). Both the LOC and the MOC arise from the superior olivary complex (SOC). Axons from the LOC project to the ipsilateral cochlea and terminate on the afferent fibers of IHCs. Axons of MOC neurons project predominantly to

the contralateral cochlea, terminating on the cell bodies of OHCs. Lateral olivocochlear fibers are not likely to affect OHC function or contralateral suppression effects. Therefore, they will not be discussed further.

The pathway of the MOC system has been characterized mainly from animal studies. However, results demonstrated in human temporal bone studies imply that the MOC pathway is similar in all mammals (Gacek, 1961). Medial olivocochlear axons extend dorsomedially from the superior olivary complex, eventually combining into a bundle close to the floor of the fourth ventricle (Gacek, 1961; Rasmussen, 1946; Warr et al., 1986). Medial olivocochlear fibers that project to the opposite cochlea cross the midline at this point and those MOC fibers that project ipsilaterally join with the crossed MOC fibers from the opposite side. The OCB leaves the brain as a component of the vestibular nerve. At the base of the modiolus, the OCB separates from the vestibular nerve and as Oort's vestibulocochlear anastomosis, joins the cochlear portion of the vestibulocochlear nerve (Gacek, 1961; Webster, 1992). After passing through Rosenthal's canal, the olivocochlear axons are known as the intraganglionic spiral bundle, and they leave the spiral ganglion and travel throughout the osseous spiral lamina and then enter the organ of Corti

(Ishii et al., 1968; Kimura et al., 1962; Nakai et al., 1974; Rasmussen, 1953; Smith, 1961; Smith et al., 1963). The myelinated axons of the MOC pass through the osseous spiral lamina and enter the organ of Corti through the habenula perforata, at which point they lose their myelin sheaths. These demyelinated axons, now known as outer radial fibers, cross the tunnel of Corti and terminate at the base of the cell bodies of outer hair cells (Ishii et al., 1968; Kimura et al., 1962; Smith et al., 1963; Spoendlin, 1963).

Inputs to MOC neurons arise from the ventral cochlear nucleus from both ears, but mainly come from the contralateral side. Included in the synapse of the MOC fiber to the OHC is the subsynaptic cistern. The MOC system is thought to control the action of the slow motile mechanism of the outer hair cell by way of this organelle (Kim, 1986). Thus it is believed that the mechanism of the MOC efferents is to interrupt the transfer of the mechanical amplification of the fast motile response to the inner hair cells.

Most of what is known of the AR pathway has been learned from animal studies. The stapedial reflex pathway is believed to involve 3 to 4 synapses (Borg, 1973). From afferent fibers of the auditory nerve, the first synapse

occurs at the anterior ventral cochlear nucleus (AVCN) (Borg, 1973). From the AVCN, fibers pass through the trapezoid body to terminate in the superior olivary complex (SOC). Some fibers project directly to the ipsilateral facial motor nucleus (Nc7) from the trapezoid body. The remaining fibers project to the lateral (LSO) and medial (MSO) superior olivary complex as well as the lateral preolivary nucleus (LPO) (Borg, 1973). According to Borg, only lesions to the MSO affected acoustic reflex activity. Therefore the MSO is the likely site of the second synapse. Fibers from the MSO project to stapedius muscle motoneurons ipsilaterally and contralaterally (Borg, 1973). The third synapse is thought to occur at the stapedius motoneurons in the medial area of the facial motor nucleus (Borg, 1973; Kamerer & Rood, 1978). The stapes motoneurons project to the stapedius muscle in the middle ear (Borg, 1973). Contraction of the stapedius muscle alters the impedance of the middle ear by increasing the stiffness of the ossicular chain (Møller, 1962; von Békésy, 1960). As a result, the transmission properties of the middle ear will be altered: low frequency energy may encounter increased reactance while the transmission of higher frequencies may be enhanced (Møller, 1962; von Békésy, 1960).

Loudness and Critical Bands

Fletcher and Munson (1933) defined loudness as a “psychological term used to describe the magnitude of an auditory sensation” (Fletcher & Munson, 1933). When the SPL of a sound is increased within a certain range (approximately 20 dB SPL and above) it is perceived as being louder by human subjects (Fletcher & Munson, 1933). Fletcher also acknowledged that the complexity of a sound influences the perception of loudness in humans (Fletcher & Munson, 1933). In fact, the perception of loudness can be altered without changing the overall intensity of the signal. This is accomplished by distributing the energy of a stimulus across the boundaries of critical bands (Zwicker, Flottorp, & Stevens, 1957). The term “critical band” was coined by Fletcher in 1940. He hypothesized that when a white noise just masks a tone, only a relatively narrow band of frequencies surrounding the tone and equal to it in power actually does the masking (Fletcher, 1940). Sound energy outside the band contributes little or nothing to the masking of the tone (Scharf, 1970). The critical band is described by Scharf as “that bandwidth at which subjective responses rather abruptly change”. Crossing the boundaries by widening the bandwidth results in an increase in the loudness of a noise (Scharf, 1961, 1970; Zwicker et al., 1957). Several researchers have confirmed the existence

and boundaries of the critical band (Boer & Bouwmeester, 1974; Bonding, 1978; Green, Mason, & Kidd, 1984; Hubner & Ellermeier, 1993; Saunders, Rintelmann, & Bock, 1979; Scharf, 1961, 1970; Schneider, 1988; Ward, 1990; Weber, 1978; Zwicker & Feldtkeller, 1955; Zwicker et al., 1957) .

The critical band is approximately 15-20% of the center frequency of the band (Scharf, 1970). This value is slightly larger for very high and low frequencies. Critical bands are thought to correspond to a constant distance of approximately 1.3 mm on the basilar membrane (Scharf, 1961).

Loudness and the Acoustic Reflex

Flottorp et al. (1971) examined the relationship between critical bandwidth and the acoustic reflex. They found that for a given center frequency, the acoustic reflex threshold was constant until the bandwidth of the stimulus became wider than a value they designated as the critical reflex band (Flottorp et al., 1971). At this point, the reflex threshold decreased at a rate of approximately 3-6 dB per octave for center frequencies of 250, 500, 1000, 2000, and 4000 Hz (Flottorp et al., 1971). Further, Flottorp et al. found that critical reflex bands were wider than critical bands determined by psychophysical methods. Additionally they reported that a

stimulus with a bandwidth broader than the critical reflex band elicited larger acoustic reflexes than stimuli with a bandwidth narrower than the critical reflex band when both stimuli were at equal SPLs (Flottorp et al., 1971). Møller reported that a broadband noise elicited an acoustic reflex of greater magnitude than a sinusoid when they were matched for intensity (Møller, 1962). Similar results were reported by Deutsch (1972) and Margolis and Popelka (1975). Others have reported that when stimuli are matched for loudness, acoustic reflex magnitudes are similar, which suggests that the acoustic reflex responds to loudness (Block et al., 1977; Gorga et al., 1980; Margolis & Popelka, 1975; Ross, 1968). The effect is seen regardless of whether reflexes were elicited contralaterally or ipsilaterally (Ross, 1968).

Loudness and Contralateral Suppression of TEOAEs

Norman and Thornton (1993) examined contralateral suppression of TEOAEs in humans related to critical bandwidth. They used four noise bands of varying widths centered at five different frequencies as contralateral stimuli. The bandwidths were defined as multiples of the critical band for each frequency: .25, .5, 1, and 2 times the critical bandwidth. They found increased suppression with an increase in the

bandwidth of noises centered at 1 and 2 kHz. They also observed that suppression resulting from a wide band noise at 50 dB SL was more than two times greater than any resulting from the 60 dB SL narrow band noises (Norman & Thornton, 1993). The 1 kHz centered noise band suppressed a wide range of the TEOAE spectra (.5 to 3.5 kHz). The 2 kHz centered band affected the TEOAE spectra in the 1 to 2.5 kHz range. The greatest amount of suppression associated with narrow-band noise was seen when the noise bandwidth was twice the critical band of the center frequency (Norman & Thornton, 1993).

In general, Norman and Thornton's data are similar to those found by investigators examining the relationship between loudness and the acoustic reflex, that is to say a wider bandwidth elicits a greater response magnitude. In the case of the acoustic reflex it is an impedance change of greater magnitude, and for contralateral effects, it is reflected in greater reduction of TEOAE amplitudes. No attempt has been made to determine the contralateral suppression effects on TEOAE amplitudes for noise bands that are matched for loudness, or to relate suppression effects to any perceived differences in loudness of the noise bands.

PURPOSE

The AR mechanism and the MOC are efferent feedback systems that affect the response of the auditory periphery to sound. The similarities between these two systems have been described. The AR is responsive to changes in loudness (Block et al., 1977; Gorga et al., 1980; Margolis & Popelka, 1975; Ross, 1968). It is not known whether the same is true for MOC effects. To determine this, the following questions were addressed in this study: a) Will the 60 dB SPL wideband noise spanning several critical bands for a given center frequency used in this study be judged as louder than the 60 dB SPL narrowband noise centered at the same frequency whose bandwidth does not exceed the critical band ? b) Will the wide band noise be a more effective contralateral suppressor of TEOAEs than the narrowband noise? c) If the dB SPL level of the narrowband noise is increased such that it is judged to be equally as loud as the wideband noise at 60 dB SPL, will this stimulus have the same suppressive effects on TEOAE amplitude as the wideband noise ?

METHOD

Subject Criteria

Subjects were women 20 to 37 years old in good general health. It was determined by questionnaire that they had no history of middle ear surgery, neural disease, or were not taking any medication currently known to affect cochlear, auditory afferent or efferent function. They had no family history of hearing loss other than presbycusis and no history of long-term unprotected exposure to toxic noise levels (industrial noise levels, shooting, military exposure, etc.).

Subject Selection

To determine a subject's eligibility, the following screening procedures were performed:

- A case history (see above)
- Otoscopic evaluation of ear canals and tympanic membranes
- Tympanometry; middle ear pressures less than or equal to ± 50 deca Pascals were accepted; compensated acoustic admittance was within .3 to 1.5 milliliters

- Hearing was screened at 15 dB HL, bilaterally, for 500 through 8000 Hz at octave intervals.
- Subjects were screened for the presence of TEOAEs using a non-linear click at a level of approximately 75 dB peak SPL. Reproducibility by band scores were 80% or greater for frequency bands of 1000, 2000, and 3000 Hz (Norman & Thornton, 1993)
- It was determined if potential subjects had robust emissions (defined as a response value of 5 dB or greater) for a linear click stimulus at 60 dB peak SPL
- Subjects were screened for spontaneous otoacoustic emissions although the results were not used to determine a subject's suitability to participate in the study.
- Subjects were screened to determine if they exhibited suppression (≥ 0.8 dB overall reduction of TEOAE level) of TEOAEs in both ears using the above described click as the ipsilateral stimulus and a 2200 Hz wide noise band centered at 2000 Hz as a contralateral stimulus at a level of 60 dB SPL (Subjects with a reduction of less than 0.8 dB overall did not consistently show a reduction of TEOAEs on repeated trials)

Instrumentation

Hearing was screened in an Industrial Acoustics Corporation (IAC) sound booth using a Beltone 10D portable audiometer calibrated to ANSI S3.6-1989 standards. Tympanometry and acoustic reflexes were assessed using a Teledyne Avionics Model TA-4D acoustic impedance meter.

Clicks were generated by Otodynamics ILO 88 XP software using a portable IBM compatible PC, an ILO sound card, and an ILO 88 amplifier. The click stimulus was delivered to the ear via a small probe coupled to the ear by a soft plastic tip. The probe was also supplied by Otodynamics. It contains a miniature transducer for stimulus delivery and a miniature microphone to measure the resulting emissions. The emissions were analyzed using Otodynamics software. Further analysis was performed using Echomaster 312 software developed by Wen at the Kresge Hearing Research Laboratory in New Orleans, Louisiana (Wen, Berlin, Hood, Jackson, & Hurley, 1993).

The contralateral noise stimuli were produced by Tucker-Davis Technologies (TDT) software (SigGen v 1.3 and BioSig v 2.1) and hardware (TDT System II) controlled by an ACT IBM compatible PC.

There were two noise bands, both centered at 2000 Hz. The first was 100 Hz wide (NB), well within the 320 Hz critical band for 2000 Hz (Scharf, 1970; Zwicker et al., 1957). The second band was 2200 Hz wide (WB). This bandwidth is approximately seven times the critical bandwidth for 2000 Hz (Scharf, 1970; Zwicker et al., 1957). At this bandwidth, 10 dB SPL must be added (on average) to a comparison tone to match the 2200 Hz noise band in loudness (Scharf, 1970). Therefore, at SPLs of 40 dB or more, it is as if a 10 dB increase in intensity has occurred when the bandwidth is increased from 100 Hz to 2200 Hz. An actual 10 dB SPL increase in a contralateral stimulus is associated with a measurable change in contralateral suppression effects (Hood et al., 1996). The 2200 Hz bandwidth also exceeds Flottorp's critical reflex band for 2000 Hz (Flottorp et al., 1971). The noise stimuli were delivered through Etymotic Research ER-2 transducers and monitored using an Etymotic Research ER-10A probe microphone system. The noise level and bandwidths were monitored using a Stanford Research Systems SR 760 FFT Spectrum Analyzer. The probe was coupled to the ear using a foam insert plug. The levels of the noise stimuli were controlled using Hewlett Packard 350D attenuators. The ILO probe was calibrated in a 1 cc test cavity supplied by ILO using the probe cavity test option from the ILO 88 test menu. The

ER-10A microphone system is known for having a flat frequency response up to approximately 9000 Hz. To assure consistent performance the SPL and noise shape of the WB noise was checked daily in a 2 cc coupler and then compared to a standard saved to disc. The psychoacoustic portion of the study was conducted using TDT's Psychosig software (v 1.3) and TDT System II hardware.

General Procedures

Subjects were seated in an IAC single wall sound booth. Otoscopy was performed. Hearing was screened bilaterally at the aforementioned frequencies. Immittance testing was performed bilaterally. The ER-10A probe was arbitrarily placed in either the right or left ear canal. The subjects were screened for the presence of spontaneous otoacoustic emissions (SOAEs). The frequency and level of any SOAE seen was recorded. The noise bands were generated by the TDT equipment and evaluated in situ using the SRS 760 spectrum analyzer. The noise bands were modified if necessary by adjusting the SPL of the high and low frequency boundaries of the noise bands to attain a flat frequency response across the specified bandwidths. Any modifications were done within SigGen and then reloaded into BioSig. Next, the noise bands were used as

contralateral stimuli in the same ear to determine if, at experimental levels (60 dB SPL), they would elicit an acoustic reflex. After assessment of acoustic reflexes, a psychoacoustic procedure using a simple adaptive up-down procedure (Leek, Hanna, & Marshall, 1992; Levitt, 1971) was performed to determine the SPL difference between the NB and WB noises when they were perceived as equally loud by the subject. The subject listened to the two noise bands (NB and WB) presented sequentially. The NB noise was the standard, the WB noise was the variable. Each noise had a duration of 1 second and was separated in time by a 0.5 second interstimulus interval (Zwicker et al., 1957). After both noisebands were presented, the subject judged whether the WB noise was louder than the NB noise. This was indicated by pressing one of two buttons on a hand held box connected by a cable to the TDT hardware. One button was labeled “yes”, the other “no”. When the “yes” button was pressed the WB noise level was reduced by 2 dB SPL. When the “no” button was pressed, then the WB noise level was increased by 2 dB SPL. The trial was terminated after 10 direction reversals (i.e., software instructed to change from increasing SPL to decreasing SPL and vice versa by the subject’s button pressing), indicating that the subject could not perceive a difference in loudness between the two stimuli. Point of subjective equality was

determined by averaging the values of the maximum and minimum excursions determined by the reversals. There were two trials types; one began with the WB and NB noises at 60 dB SPL. The other began with the WB noise at 40 dB SPL and the NB noise at 60 dB SPL. Each trial type was performed twice for a total of four trials per ear.

Next, the ILO probe was placed in the ear opposite the one with the Etymotic probe. Transient evoked otoacoustic emissions were recorded for a linear click level of 60 ± 1 dB peak SPL (Hood et al., 1996; Veuillet et al., 1996). First, reliability trials were performed to determine the stability of the TEOAE. These consisted of a quiet run (no contralateral stimulus) followed by another quiet run. The two runs were then compared to each other to determine if there was a difference in emission levels. This was done twice. Next, TEOAEs were measured in the presence of a noise band. The levels of the noisebands were $60 \text{ dB SPL} \pm 1$ dB as measured in the ear canal. This level was chosen because it was intense enough to elicit a contralateral suppression effect, it could be produced by the TDT system without significant harmonic distortion and was not likely to elicit an acoustic reflex. Prior to each run in the presence of noise, emissions were gathered without a contralateral stimulus present.

Later the emission levels obtained in the presence of the noise were compared to the levels obtained in quiet that preceded them and a dB difference was calculated. For the noise trials the contralateral stimulus was present in the ear at least 30 seconds before the ipsilateral stimulus was presented. Finally, the 100 Hz band noise was presented at an SPL at which it was perceived as equal in loudness to the 2200 Hz noiseband at 60 dB SPL. This was the EQ noise. This level was determined by adding the dB difference between the NB and WB noise at equal loudness for each subject (determined by the psychoacoustic procedure) to the NB noise level (60 dB SPL). There was a total of 16 trials per ear. The quiet condition was always presented first. The order of presentation of the noise conditions was varied.

RESULTS

Thirty eight women meeting the subject criteria were screened to determine if they demonstrated contralateral suppression of TEOAEs resulting from the WB noise centered at 2000 Hz. The noise was presented at 60 dB SPL. Of the 38 women, 10 had suppression effects, defined as a reduction of amplitude of the overall TEOAE level by 0.8 dB or greater in both ears. These ten were chosen as subjects for this study. All had hearing thresholds ≤ 15 dB HL at 500, 1000, 2000, 4000, and 8000 Hz bilaterally. All had symmetric tympanograms with middle ear pressure of ± 50 deca Pascals in both ears. Contralateral acoustic reflex thresholds were ≤ 95 dB HL bilaterally using pure tones at 1000 and 2000 Hz as stimuli. None of the noise stimuli elicited a contralateral acoustic reflex in any of the 20 ears. All had TEOAE response levels of at least 5 dB in both ears when a linear click level of 60 dB ± 1 dB peak SPL was used as a stimulus. All participants in this study were found to have spontaneous otoacoustic emissions (SOAEs) in at least one ear. Seven had SOAEs in both ears. Six had a greater number of SOAEs in the left ear than the right, one had an equal number of SOAEs in both ears, and three had more SOAEs in the right ear than in the left.

Loudness Study

The investigation of the loudness of the bands of noise involved four trials per ear for each subject. Each trial began with NB and WB noises at 60 dB SPL or with the WB noise at 40 dB SPL and the NB noise at 60 dB SPL. Two trials were conducted with the noise bands at equal SPLs and the noise bands at unequal SPLs. There was no specific order of trial presentation. The mean SPL difference between the NB and WB noises when they were perceived as equally loud by the subjects in this study was 13.6 dB. The values ranged from 11 to 16 dB SPL. The SPL required for equal loudness of the NB and WB noise bands differed between right and left ears of individual subjects by an average of 1.6 dB SPL and ranged from 0 to 6 dB SPL. For three subjects, the WB noise was perceived as louder in the right ear. The WB noise was perceived as louder in the left ear by 5 subjects, and the WB noise was perceived as equally loud in each ear by 2 subjects. For all subjects in all ears, the SPL difference between the NB and WB noises required for a loudness match was slightly higher for trials that began with the WB noise at 40 dB SPL. The average difference was 2.2 dB SPL and ranged from 0 to 6 dB SPL. The dB SPL difference between the WB and NB noises is represented in Figure 1.

Suppression Studies

The amplitude differences between a TEOAE recording made in the Quiet, NB, WB or EQ conditions and the recording in quiet that preceded it were examined over time and across frequency. The Echomaster program (Wen et al., 1993) was used to calculate the difference in dB between two TEOAE traces. This was done by subtracting the TEOAE waveform obtained in the experimental condition from the TEOAE waveform collected in the control condition (Wen et al., 1993). The control is always a quiet trace. Therefore, suppression is indicated by positive values. The dB difference between two traces was calculated at 2 millisecond (ms) intervals from 2 to 20 ms. Thus, difference values were determined for periods of 2-4, 4-6, 6-8 ms...18-20 ms. This program was also used to determine the dB difference between two TEOAE traces by frequency from 195 to 5275 Hz at discrete frequency intervals.

The mean dB difference for the WB noise across all subjects/all ears was 1.12 dB. The values for the Quiet, NB, and EQ conditions were -0.08 dB, 0.05 dB, and 0.16 dB, respectively. The mean dB differences in right ears for the Quiet, WB, NB, and EQ conditions were -0.09 dB, 1.01 dB, 0.02 dB and 0.23 dB, respectively. The respective values for left ears for the

Quiet, WB, NB and EQ noises were -0.07 dB, 1.23 dB, 0.07 dB, and 0.09 dB. Suppression effects were evaluated across frequency and time in right and left ears. The WB noise centered at 2000 Hz was associated with a reduction of TEOAE spectrum amplitudes from 400 to 5000 Hz and from 4 to 18 ms post stimulus. The dB difference for the Quiet, NB, WB and EQ conditions averaged across subjects and ears for the 2 ms time periods is shown in Figure 2. Suppression resulting from the WB noise was also calculated for three time periods: 4 to 10 ms (“early”), 10 to 16 ms (“mid”), and 16 to 20 ms (“late”) (Collet et al., 1990). Values for these time periods for right ears were 0.75 dB, 1.45 dB, and 1.11 dB, respectively. Results for left ears were: 0.96 dB, 1.42 dB, and 1.66 dB for the early, mid, and late time periods. The amount of suppression increased after 10 milliseconds. In some individuals, the EQ noise was associated with TEOAE amplitude reduction, but at discrete “islands” across frequency rather than the general overall effect across frequency seen with the WB noise. The difference in WB and EQ effects by frequency in subject DGT’s right ear is illustrated in Figure 3.

Statistical Analysis

Results of the loudness study were subjected to a two factor repeated measures analysis of variance (ANOVA). The two factors were ear (right or left) and WB starting SPL (60 dB or 40 dB). Results indicate no ear effect and no interaction between ear and starting WB SPL. Starting WB SPL did have a significant effect ($F=11.77$, $p \leq .01$) but the effect was not strong (ETA squared = .567).

Transient evoked otoacoustic emissions were evaluated in all subjects under all conditions (Quiet, NB, WB, and EQ), although the order of condition presentation was varied systematically. Thus, a two factor repeated measures ANOVA was used to analyze the overall dB difference data. The two factors were ear and condition (Quiet, NB, WB, and EQ). There was no significant effect for ear ($F=.74$ at .411 level). There was a significant effect for condition ($F=42.70$ at .0001 level). An ETA squared value of .827 indicates a strong effect. A power of 1 was attained. There was no interaction between ear and condition ($F=2.85$ at the .056 level).

Post hoc analyses using the Tukey HSD test were applied to the aforementioned results to determine where the significant difference between conditions (Quiet, NB, WB, and EQ) were. Only the WB results

were significantly different (at .05 level) from all other conditions. Because there was no ear effect, data for the right and left ears was combined.

Repeated measure ANOVAs and Tukey posthoc tests were also performed on combined data for 2 millisecond time intervals. When mean suppression values for all subjects for both ears at 2 ms time periods were analyzed, significant effects (at the .0001 level) were noted at all time intervals except for 2-4 ms ($F=2.82$ at .058, not significant) and 18-20 ms ($F=8.02$ at .001). All other time periods had F values that ranged from 15 to 44 at the .0001 level. For each 2 millisecond time interval, the WB noise effect was significantly different at the .05 level from all other conditions except at the 2 to 4 millisecond time period.

DISCUSSION

The WB noise was perceived as louder than the NB noise by all subjects. The loudness of the WB noise was judged similar for both the right and left ears by all subjects. In all subjects, the WB noise was associated with the greatest amount of suppression. When the SPL of the NB noise was increased so that it was equal in loudness to the WB noise, there was no change in its suppression effects.

Loudness Studies

The method used to determine the difference in SPL between the NB and WB noise at equal loudness was a simple adaptive “up-down” procedure (Leek et al., 1992; Levitt, 1971). Levitt stated that the advantages of this method is its simplicity, efficiency, robustness, and small sample reliability. The SPL difference results obtained with this method are similar to those found by earlier investigators. Results of studies by Feldtkeller and Zwicker (1956), Zwicker, Flottorp and Stevens (1957) and Scharf (1970) predict that a noise with a bandwidth approximately five times its center frequency, or two tones that are separated by approximately five times the center frequency will be perceived as being louder than a similar stimulus of equal intensity with energy within the critical band of that center

frequency. A difference of approximately 10 dB SPL is seen between a stimulus with energy inside the critical band and a stimulus with energy distributed across a frequency range five times the same critical band when their levels are adjusted so that they are judged equally loud (Feldtkeller & Zwicker, 1956; Scharf, 1970; Zwicker et al., 1957). The critical band for 2000 Hz is approximately 320 Hz (Feldtkeller & Zwicker, 1956; Scharf, 1961, 1970; Zwicker et al., 1957). Thus a noise with a 100 Hz bandwidth would need to be at least 70 dB SPL to be judged equally loud as a noise with a 1600 Hz bandwidth at 60 dB SPL when both are centered at 2000 Hz. The WB noise used in this study was 2200 Hz wide, approximately seven times the critical bandwidth for 2000 Hz. Based on Feldtkeller and Zwicker's data, one would expect approximately a 12-13 dB SPL difference between the NB and WB noises used in this study at equal loudness (Feldtkeller & Zwicker, 1956). In the present study the mean dB SPL added to the NB noise so that it was perceived as equally loud to the WB noise was 13.6 dB SPL. Individual values ranged from 11 to 16 dB SPL. The results from this study closely agree with the results of Feldtkeller and Zwicker (1956) and Scharf (1970).

Suppression Studies

The results of this study compare favorably to those of Collet et al. (1990), Berlin et al. (1993) and Veuillet et al. (1991) in that a contralateral noise stimulus is associated with the reduction of the amplitude of TEOAEs evoked by a click. Further, these results are similar to those of Collet et al. and Berlin et al. with respect to the time period after click stimulation within which suppression occurs. Collet et al. found little suppression effect from 5 to 10 ms, but significant effects from 10 to 15 ms (approximately 2 dB) and 15 to 20 ms (approximately 6 dB) using a broadband noise at 50 dB SPL as a contralateral stimulus. Berlin et al. found significant suppression (approximately 2 dB) across time from 2-20 ms with the greatest amount occurring between 12 to 20 ms for $\frac{1}{3}$ octave narrow band noise (approximately 500 Hz wide for a center frequency of 2000 Hz) at a level of 50 dB HL. In this study, there was significant (approximately 1.25 dB) reduction in TEOAE level from 5 to 10, 10 to 15, and 15 to 20 milliseconds. The fact that the WB noise has such a broad effect across frequency is not surprising. Liberman and Brown (1986) reported that a single MOC fiber may project to as many as 10 discrete clusters of outer hair cells, spanning a distance of up to 2.8 millimeters (1 octave) on the basilar membrane in the cat. Brown (1987) reported similar

findings in the Guinea pig. Nakagi and Igarashi (1974) and Liberman and Brown (1986) reported that MOC fibers tuned to a given frequency tend to project to OHCs on the basilar membrane tuned to the same frequency and basalward. It might be said that there is an upward spread of suppression effects. Consider too the propagation of the traveling wave envelope. It is known that the envelope tails towards the base of the basilar membrane, thus exciting auditory nerve fibers at, and basal to, the point of maximum displacement (von Békésy, 1960). When considering the innervation patterns of MOC fibers and the propagation of the traveling wave, it seems reasonable that suppression would be seen at frequencies higher than the high frequency boundary of the WB noise (3100 Hz).

If we accept the possibility that MOC fibers tuned to 3100 Hz may also project to OHCs positioned to respond to frequencies one octave above in humans, then we might expect to see suppression effects up to 6200 Hz. The results of this study support the belief that, although MOC fibers are tuned (Brown, 1989; Liberman, & Brown, 1986), the suppression effect is not (Berlin et al., 1993b).

Suppression resulting from NB and WB noise at equal SPLs

It is interesting to note that the NB noise at 60 dB SPL was not an effective suppressor while the WB noise at the same intensity was associated with a reduction of TEOAE levels. When considering the spectrum levels of the NB and WB noises (40 and 27 dB SPL, respectively) it is tempting to anticipate that the WB noise would have little effect. Even though the energy in the WB noise is spread across a greater frequency range (2200 Hz) than the NB noise (100 Hz), the spectrum level of 27 dB is quite low. However, one must consider the spectrum level of the noise at the transducer and the energy representation of that noise on the basilar membrane as a result of the cochlea's response to that noise stimulus.

Several investigators have described a non-linear response of the basilar membrane to a stimulus that results from an active process in the outer hair cell (Brownell, 1990; Dallos, 1992; Johnstone et al., 1986; Kemp, 1979; Kim, 1986; Veuillet et al., 1991; Zwicker, 1986). The implication of their research is that at certain SPLs (roughly 0 to 60 dB SPL), the active process adds energy to an incoming signal, resulting in greater displacement of the basilar membrane. As the SPL of the stimulus increases from approximately 0 to 60 dB SPL, energy is added to the input

stimulus at a decreasing rate (Johnstone et al., 1986). Ruggero (1997) described non-linear activity at the basilar membrane of chinchillas near the base of the cochlea at stimulus levels up to 100 dB SPL using tone bursts as stimuli. From Ruggero et al. (1997), estimates of gain at the basilar membrane at different input levels can be determined:

<u>Input Level (dB)</u>	<u>Gain (dB)</u>
20	46
30	40
40	33
50	27
60	20

Leek and Summers (1996) used a method developed by Glasberg and Moore (1990) to estimate the excitation patterns of certain vowels and broadband noise stimuli on the basilar membrane. Examination of their data reveals that the energy representation of a stimulus on the basilar membrane is approximately 15 to 30 dB greater than the stimulus SPL at the transducer when the spectrum level of a given frequency in the stimulus was below 50 dB SPL (Leek & Summers, 1996). Data from Johnstone et al. (1986), Leek & Summers, (1996) and Ruggero et al. (1997), imply that

the equivalent SPL of a stimulus will be greater at the level of the basilar membrane than at the level of the transducer coupled to the ear canal.

In this study, the WB input spectrum level was 27 dB, but the energy representation on the basilar membrane in response to this signal may have been much greater when the energy added to the signal by the active process is considered. A signal with a spectrum level of 27 dB may be resolved at the level of the basilar membrane as if 40 dB of gain were provided. For the NB noise, with a spectrum level of 40 dB, the amount of gain may be estimated at 33 dB. Thus the effective spectrum level may be 73 dB. Consider that the bandwidths of the NB and WB noises are 100 and 2200 Hz, respectively. In light of this fact, it seems plausible that a noise with a spectrum level of 67 dB and spread over 2200 Hz would be a more effective suppressor than a noise with a spectrum level of 73 dB spread over 100 Hz. Using the spectrum level to determine an overall dB level on the basilar membrane yields some interesting results:

$$\text{Total dB} = \text{Spectrum Level} + 10 \log (\text{bandwidth})$$

$$\text{For the NB noise, Total dB} = 73 + 10 \log (100)$$

$$\text{Total dB} = 93 \text{ dB SPL}$$

For the WB noise, Total dB = $67 + 10 \log (2200)$

Total dB = 100 dB SPL

Not only would the effective stimulus SPL of the WB noise on the basilar membrane be greater, but the energy would be spread over a wider range of frequencies. That is exactly what was found in this study. The WB noise was a more effective suppressor than the NB noise when both stimuli were presented at 60 dB SPL.

Suppression resulting from NB and WB noise at equal loudness

What of the results when the two noises are equal in loudness? In this study the average SPL difference between the NB and WB noise at equal loudness was 13.5 dB. Thus, the average SPL of the EQ noise (NB noise with SPL increased to match WB noise at 60 dB SPL in loudness) was 73.5 dB (60 dB SPL + 13.5 dB SPL). The spectrum level of the noise from the transducer was 53.5 dB SPL. From Ruggero et al. (1997) the gain for a signal at 53.5 dB would be approximately 25 dB, so the effective stimulus spectrum level on the basilar membrane might have been closer to one

presented at a level of 79 dB. The effective level of the noise on the basilar membrane could be estimated using the following equation:

$$\text{Total dB} = 79 + 10 \log (100)$$

$$\text{Total dB} = 99 \text{ dB SPL}$$

This level is greater than the NB noise when it was at 60 dB SPL (“actual” total level 93 dB SPL). In fact, the total dB levels for the WB and EQ noises are within 1 dB of each other. This makes sense as they were judged to be equally loud. However, the energy in the EQ noise is confined to a 100 Hz bandwidth. It is not surprising then, that the WB noise, stimulating a broader area of the basilar membrane, with energy summed across several critical bands, was the most effective suppressor.

A comparison of the calculated effective energy levels on the basilar membrane resulting from the different noise bands is interesting. While the NB and WB noises were at equal SPLs at the transducer, they had unequal effective energy levels on the basilar membrane. The WB noise effective level was greater and it was judged as louder. The EQ and WB noises were at unequal SPLs at the transducer, but had relatively equal effective energy levels on the cochlear partition and were judged equally

loud. Equal loudness may imply equal energy levels on the basilar membrane.

Cochlear amplifier gain in dB and spectrum level formulas were used in the previous sections to determine dB SPL levels for different noises present on the basilar membrane. This calculation was done in an attempt to explain why the WB noise was associated with greater contralateral suppression effects than the NB or EQ noises. Admittedly, there are problems in using this terminology to define actual energy levels present on the cochlear partition. However, it is believed that use of dB levels is a valid way to make a relative comparison of the effective energy levels resulting from each noise on the basilar membrane.

Acoustic Reflexes

Veuille et al. (1991) and Berlin et al. (1993) have commented on the possibility of the influence of the stapedial reflex on the reduction of TEOAE amplitude. Stimulation of the stapedius muscle results in a change in the impedance of the middle ear system by stiffening the ossicular chain (Møller, 1962). This stiffening results in a reduction in the transfer of energy in certain frequencies, mostly 1000 Hz and below, to the cochlea

(Møller, 1962). Transient evoked otoacoustic emissions are very low intensity phenomena (typically 15 dB or less). Therefore, if the acoustic reflex were activated while evoking TEOAEs, it is conceivable that the emission levels would be reduced as a result of the impedance change caused by stapedius muscle contraction. However, it is doubtful that the acoustic reflex was a factor in this study as none of the noise bands used in this study elicited an acoustic reflex. Berlin et al. discussed the possibility of a middle ear muscle response in which a slight change in impedance would result from partial contraction of the stapedius muscle that would not be detected by conventional impedance measurements. Fisch and Schulthess (1963) recorded stapedius muscle EMGs from human subjects when a variety of stimuli were presented in the contralateral ear. The lowest EMG threshold they measured was for a broadband noise stimulus at 70 dB SPL. The lowest EMG threshold for a pure tone was recorded at 90 dB SPL for 2000 Hz (Fisch, & Schulthess, 1963). The noises used in this study were at 60 dB SPL or in the case of the EQ, at a maximum of 76 dB SPL. Although the SPL of the EQ noise may be greater than the lowest EMG threshold found by Fisch and Schulthess (1963), its bandwidth was only 100 Hz. The acoustic reflex threshold for this noise would be expected to be closer to that of a puretone. In fact, for one subject in this

study the acoustic reflex was activated for a NB noise level of 86 dB SPL. Further, Veuillet et al. (1991) measured contralateral suppression in subjects with paralyzed or severed stapedius muscles. Berlin et al. (1993) also demonstrated contralateral suppression in a patient with Bell's Palsy. If the middle ear reflex were the actual cause of suppression effects, then little or no reduction in TEOAE amplitude would be seen in the aforementioned subjects. As mentioned previously, impedance changes mainly affect the transmission of frequencies at or below 1000 Hz. Although suppression was seen at 1000 Hz and below, it was also seen for frequencies above 1000 Hz, and in fact up to 4000 Hz. It is unlikely then, that an acoustic reflex caused the reduction of TEOAE levels seen in this study.

Stability of the TEOAE

One would expect little or no change in emission levels for repeated measures using the same click SPL without a contralateral stimulus. Harris, Probst and Wenger (1991) and Marshall and Heller (1996) found TEOAEs in human subjects to be repeatable and stable in amplitude and spectrum in human subjects over repeated sessions. Most variation was seen for the lower frequencies (Marshall, & Heller, 1996). In this study the Quiet condition represented comparisons of one measurement in quiet

with another. As with the measurements with a contralateral noise present, a quiet result was compared with another preceding it, and then another pair of measurements in quiet were collected and those two were compared. On average across all subjects and ears, the dB difference was less than 0.5 dB at any time period. Subjects who had contralateral suppression associated with the WB noise during the screening procedure continued to show suppression effects upon return for the full evaluation.

Conclusion

Two contralateral stimuli of the same intensity but different bandwidths had different effects on TEOAE amplitudes. The WB noise at 60 dB SPL was associated with suppression, the NB noise at 60 dB SPL was not.

Suppression effects resulting from the WB noise were seen across a broad frequency range. Reduction of TEOAE amplitudes was present from 400 to 5000 Hz. As with the acoustic reflex system, the suppression effect does not appear to be tuned. If only intensity determined the response of the MOC system, then signals of equal intensity would have similar effects regardless of bandwidth; however in this study, they do not. The results of this study imply that the MOC system, like the acoustic reflex system, responds to loudness resulting from variations in bandwidth as well as variations in SPL.

However, unlike the AR system, equal loudness was not associated with equal MOC effects. Kobler, Guinan, Vacher, and Norris (1992) postulated that the very broad tuning of stapedius muscle motoneurons implies that different frequency regions of the cochlea must converge at some point central to the stapedius motoneuron outputs. Animal studies do not support the idea of an auditory afferent convergence point prior to MOC stimulation.

In fact, MOC neurons are sharply tuned, but project to areas of the basilar membrane tuned to respond at and basalward to frequencies to which they are tuned; up to an octave in some cases (Brown, 1989; Liberman & Brown, 1986). This, and the propagation of the traveling wave may explain the broad frequency effects of MOC stimulation. Therefore, one would not predict that MOC effects would be equal when contralateral noise bands that differ by multiples of critical bandwidth are at equal loudness. The results of this study support this conclusion.

Figure 1: dB SPL difference between WB and NB noises at equal loudness across ten subjects

Figure 2: dB Difference for all conditions averaged across all Subjects' right and left ears as a function of time (error bars: standard error of mean)

Figure 3: Comparison of suppression associated with WB and EQ noises across frequency for subject DGT

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