Medial Olivocochlear Efferent System in Humans Studied With Amplitude-Modulated Tones

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Maison, Stéphane, Christophe Micheyl, and Lionel Collet. Medial olivocochlear efferent system in humans studied with amplitude-modulated tones. J. Neurophysiol. 77: 1759–1768, 1997. Evoked otoacoustic emissions (EOAEs) are assumed to be generated by outer hair cells (OHCs). It is now generally accepted that EOAEs represent a means of functional exploration of the active micromechanical properties of OHCs. Efferent fibers of the medial olivocochlear system (MOCS) are connected along the sides and the bases of OHCs. Some studies have shown that a suppression effect on EOAE amplitude is induced by the MOCS neurons during contralateral stimulation, presumably by modification of OHC motility. The contralateral acoustic stimuli used in experiments on the MOCS suppression effect have consisted mainly of sounds without a slow temporal fluctuation in their envelopes (band-pass noise, high-pass noise, pure tones, or clicks). To elucidate further the parameters of MOCS activation, in the present study we looked at the contralateral suppression effect of amplitude-modulated (AM) tones. The results showed that EOAE amplitude was reduced with AM tones compared with no contralateral acoustic stimulation. The suppression effect mainly depended on three parameters. 1) Contralateral stimulation intensity: EOAE suppression occurred only with intensities ≥40 dB SL. 2) The greater the modulation depth, the greater the suppression effect: statistical analysis showed a significant effect for 75 and 100% modulation depth. 3) The 100- and 140-Hz modulation frequencies gave the greatest suppression effect for 100 and 75% modulation depths. The suppression effect was frequency specific. The greatest decreases were observed when the carrier frequency of the contralateral AM tone was close to the frequency of the EOAE under study, i.e., 1 and 2 kHz. Acoustic cross talk and middle ear effects, which cannot be completely excluded, are discussed. However, the demonstrated frequency specificity of the EOAE suppression effect, together with observed presence of contralateral EOAE suppression in patients without stapedial reflex and the very weak intensities used (i.e., below acoustic reflex threshold), suggested that it was unlikely that the observed effects were due merely to middle ear reflexes. Our results confirm further the contralateral suppression effect on human cochlea mechanisms and show that the suppression effect can be influenced by amplitude modulations of the suppressor, characteristic of sounds in the environment.

INTRODUCTION

It is well established that the organ of Corti receives efferent innervation from olivocochlear neurons. The discovery of the olivocochlear bundle by Rasmussen (1946) allowed two types of efferent fibers to be distinguished according to cell body location (Warr 1975; Warr and Guinan 1979; Warr et al. 1986). The first system comprises lateral efferent fibers, the cell bodies of which are situated around the lateral superior olivary nucleus. These fibers, which are unmyelinated, make synapses with the radial afferent fiber dendrites (Liberman 1980; Smith and Rasmussen 1963), mainly on the ipsilateral side. A second system is composed of medial efferent fibers, the cell bodies of which are situated in the medial superior olivary nuclei (Spangler and Warr 1991). The projections of these fibers, which are myelinated, are mainly contralateral (Wilson et al. 1991), and terminate on the cell bodies of the outer hair cells (OHCs) (Liberman and Brown 1986).

The auditory function of these projections are still largely unknown. Nevertheless, electrophysiological studies of the olivocochlear efferent system show an inhibitory action of electrical stimulation of the medial olivocochlear bundle on auditory afferent responses. Electrical stimulation of the medial olivocochlear system (MOCS) at the level of the fourth ventricle (where MOCS fibers decussate) suppresses the compound action potential (N1 wave) in response to acoustic stimulation by clicks (Fex 1962) and enhances the cochlear microphonic potential (Fex 1959; Gifford and Guinan 1987). Electrical stimulation also decreases stimulated discharge in the auditory afferent fibers (Guinan and Gifford 1988a; Winslow and Sachs 1987). Studies in which researchers used acoustic stimulation of the contralateral cochlea, a condition closer to natural MOCS stimulation conditions, have shown modification of afferent fiber responses in the contralateral cochlear nerve (Buño 1978; Murata et al. 1980). This has been confirmed in cats (Liberman 1989; Warren and Liberman 1989a,b) and in humans (Folson and Owsley 1985). In addition, Rajan and Johnstone (1988) have shown that contralateral acoustic stimulation (CAS) has effects similar to those of electrical stimulation of the MOCS at the floor of the fourth ventricle. Moreover, CAS has the same effect as electrical stimulation of the MOCS on temporary threshold shifts (Rajan 1988a,b). The disappearance of the latter effect after section of the olivocochlear efferent bundle is an argument in favor of a functional role of these efferents (Rajan 1988a).

These studies have been accompanied by others that have revealed how the MOCS acts on the auditory periphery, namely those involving recordings of evoked otoacoustic emissions (EOAEs), which reflect mechanical properties of the OHCs. Mountain (1980) and Siegel and Kim (1982) demonstrated a change in acoustic distortion product amplitude in animals under electrical stimulation of the MOCS. Brown et al. (1983) have shown that electrical stimulation of the MOCS modifies the receptor potential of the inner hair cells, supposing that this effect is mediated through

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The present study involved 89 subjects, of whom 86 were healthy (ages 24.8 ± 6.4 yr, mean ± SD; 38 male, 48 female) with no history of auditory pathology and normal audiometric functions (thresholds of 10 dB hearing level or better between 250 and 8,000 Hz at octave intervals on pure tone audiogram). Middle ear reflexes were present. The other three subjects (ages 21, 38, and 51 yr, 3 females) had total unilateral hearing loss secondary to the mumps, but normal audiometric functions in the contralateral ear. On the affected side, the patients had no subjective auditory response at any audiometric frequencies between 250 and 8,000 Hz. The audiogram of the unaffected ear was normal (thresholds of 10 dB hearing level or better at octave frequencies between 250 and 8,000 Hz).

Audiometry and tympanometry

Tonal audiometry was conducted in a soundproof room with the use of a Madsen OB 828 audiometer. Hearing thresholds were measured at 250, 500, 1,000, 2,000, 4,000 and 8,000 Hz (according to International Standard Organization standards). Tympanometry was conducted in a soundproof room with the use of an Amplaid 702 impedance meter.

Recording and analysis of EOAEs

EOAEs were recorded and analyzed according to the methodology proposed by Bray and Kemp (1987). The probe comprised a Knowles 1843 microphone and a BP 1712 earphone transducer, both embedded in a plastic earplug. Stimulus presentation, data recording, and averaging were carried out with the use of Otodynamics ILO88 software and hardware (Kemp et al. 1990). The stimulus was a tone pip at a frequency of 1 or 2 kHz (1 cycle rise and fall, 2 cycles plateau). The presentation rate of the stimuli was 50 Hz. The linear cochlear echo method was used. This technique uses a combination of four identical acoustic impulses (size and polarity). In this condition, the meatal and middle ear echoes are not self canceling and their durations increase as the stimulus level increases. These ringing components have been eliminated by applying low intrameatal components. To minimize stimulus artifact of the input sound, the EOAEs are analyzed with the use of an eight-channel analyzer. The analysis window was 20 ms; 512 responses were averaged. A pass band of 500–6,000 Hz was employed. In all subjects with normal hearing, EOAEs were recorded in the right ear. In patients with total unilateral hearing loss, EOAEs were recorded in the normal ear. EOAE amplitude was computed from the whole response in a time window of 6–20 ms.

CAS

CAS was either a pure tone or an AM tone. It was generated with the use of a 16-bit digital-to-analog (D/A) converter at a sampling rate of 44.1 kHz and delivered through a TDH 39 earphone (bandwidth 50–8,000 Hz). The D/A card was installed on an IBM-compatible computer. The contralateral stimulus carrier was generated with the use of the following equation

\[
S = A \cdot \cos (2\pi Fc \cdot t)
\]

To maintain the energy of the modulated stimuli equal to that of the unmodulated stimuli, a correction factor was applied

\[
c = 1/\sqrt{[1 + (1/m^2)]}
\]

where \(m\) is the modulation depth (MD) (from 0 to 1). This correction factor was multiplied by the signal. The corrected modulated stimulus was

\[
S_m = S \cdot c \cdot [1 + m \cdot \cos (2\pi Fm \cdot t)]
\]

The coding of AM by the auditory system has been investigated extensively at different stages of the afferent pathways: at the level of the auditory nerve (AN) (Javel 1980; Palmer 1982; Smith and Brachman 1977), the cochlear nucleus (Evans and Nelson 1966; Frisina 1983; Möller 1976), the inferior colliculus (Nelson et al. 1966; Rees and Möller 1983), and the auditory cortex (Schreiner et al. 1983). On the other hand, little is known concerning AM coding by the efferent pathways. To date, only one study has focused on the dynamic discharge properties of efferent fibers in guinea pigs (Gummer et al. 1988). The results obtained by Gummer et al. indicate that the MOCS is particularly sensitive to AM. Modulation transfer functions in the MOCS neurons present a large peak centered around 100 Hz. Several of the above studies have shown MOCS to be activated by BBN, NBN, and clicks but not by pure tones (Berlin et al. 1993; Collet et al. 1990, 1992; Ryan et al. 1991; Veuillet et al. 1991, 1992, 1996).

To test the involvement of the MOCS in the coding of AM tones, we studied the effect of contralateral AM tones on EOAE amplitude.
Effects of AM tones

Table 1 indicates the stimulation parameters used in the different experiments. To clarify description, the contralateral stimulation parameters have been divided into carrier and modulator parameters.

**Effect of AM tone stimulation.** Forty-eight subjects (20 male, 28 female; ages 21.5 ± 5.7 yr) participated in this experiment. Ipsilateral stimulation was an 1-kHz tone pip set at 60 ± 3 dB SPL. Contralateral stimulations were either a 1-kHz pure tone (unmodulated) or a 1-kHz AM tone [MD 100%, modulation frequency (MF) 100 Hz]. The order of presentation of these frequencies was randomized.

In a second step, four separate series of experiments were performed to assess the effect of contralateral stimulation parameters. In all these experiments, EOAES were evoked by a 1-kHz tone pip, the SPL of which was set at 60 ± 3 dB. EOAES were successively recorded with and without contralateral stimulation consisting either of a 1-kHz tone or an AM tone.

**Effect of MD.** Twelve subjects (5 male, 7 female; ages 24.7 ± 6.3 yr) received a contralateral AM tone (1-kHz carrier frequency, 100-Hz MF) modulated with a depth varying between 0 and 100% by 12.5 point steps. EOAES were recorded with the use of a 1-kHz tone pip. Ipsilateral and contralateral intensity levels were 60 dB SPL and 40 dB SL (i.e., 46.6 ± 4.0 dB SPL), respectively.

**Combined effect of MF and MD.** Twenty-one subjects (10 male, 11 female; ages 25.7 ± 4.4 yr) participated in this experiment. The MFs varied from 50 to 800 Hz in half-octave steps, MDs were 25, 50, 75, and 100%. Contralateral stimulus intensity was set at 40 dB SL (44.4 ± 2.6 dB SPL). The order of presentation of MFs and MDs was randomized.

**Effect of carrier frequency.** Twenty-six subjects (12 male, 14 female; ages 27.3 ± 6.4 yr) participated in this experiment. Ipsilateral stimuli used were successive 1- and 2-kHz tone pips. Contralateral stimulus intensity was set at 40 dB SL (44.5 ± 2.6 dB SPL). MF was set at 100 Hz and MD at 100%. The carrier frequencies were 500, 750, 1,000, 1,500, 2,000, 3,000, and 4,000 Hz, in random order.

**Effect of carrier intensity.** Twenty-four subjects with normal hearing (11 male, 13 female; ages 27.5 ± 6.5 yr) participated in this experiment. Contralateral stimuli were set at −10, 0, 10, 20, 30, 40, 50, and 60 dB SL (i.e., −5.1 ± 2.5, 4.9 ± 2.5, 14.9 ± 2.5, 24.9 ± 2.5, 34.9 ± 2.5, 44.9 ± 2.5, 54.9 ± 2.5, and 64.9 ± 2.5 dB SPL), presented in random order. For each intensity, MF and MD were 100 Hz and 100%, respectively.

In the three subjects with hearing loss, EOAES were recorded without and with a contralateral 1-kHz AM tone (MD 100%, MF 100 Hz) with intensity varying from −10 to 60 dB over the mean threshold of the normal subject sample.

**Measurements and statistics.**

In all cases, the amplitude of the EOAES was measured in decibels SPL. Statistical analysis was performed with the use of SigmaStat software (version 1.02) and included paired t-test and analysis of variance (ANOVA) for repeated measures.

**Results.**

**Effect of contralateral AM tone.**

Figure 1 shows an EOA trace recorded from one subject with normal hearing without (A) and with (B) a 1-kHz AM tone modulated at 100 Hz at a depth of 100% and presented contralaterally at 40 dB SL (i.e., 46.2 ± 5.5 dB SPL).

In Fig. 2 are presented results demonstrating contralateral EOA suppression as a function of CAS. Each CAS was set at 40 dB SL. Figure 2 shows that EOA amplitude did not decrease significantly under contralateral 1-kHz pure tone stimulation (amplitude attenuation 0.08 ± 0.10 dB, mean ± SE). However, the 1-kHz AM tone did significantly decrease EOA amplitude (P < 0.001) with amplitude attenuation equal to 0.40 ± 0.12 dB.

**Effect of contralateral MD.**

Figure 3 represents contralateral attenuation of EOAES as a function of the MD of a 1-kHz pure tone modulated at a frequency of 100 Hz and a depth varying between 0 (pure tone) and 100% by steps of 12.5 points. The EOA amplitude attenuation induced by the contralateral tone increased with contralateral MD, becoming statistically significant at a depth of 37%.

**Combined effect of contralateral MD and MF.**

The data shown in Fig. 4 provide insight into the pattern of variation of the attenuation effect as a function of contralateral MD and MF. EOA amplitude was reduced significantly for MFs ranging in half-octave steps from 50 to 570 Hz for an MD of 100% and for MFs going from 50 to 400 Hz.
Cochlear Response

FIG. 1. Comparison of evoked otoacoustic emissions (EOAEs) without (A) and with (B) a contralateral amplitude-modulated (AM) tone in 1 subject. EOAEs were induced by a 1-kHz tone pip. The contralateral stimulus was a 1-kHz AM tone [modulation depth (MD) 100%, modulation frequency (MF) 100 Hz]. Ipsi- and contralateral stimulus levels were 60 dB SPL and 40 dB sensation level (SL) (i.e., 46 dB SPL), respectively. Left: EOAE recordings. Echo: EOAE amplitude (dB SPL). Analysis time was 6–20 ms after stimulus onset (stimulus ringing is rejected). Right: amplitude spectra of EOAE waveforms and of the stimuli (i.e., 1- and 2-kHz tone pips) employed for generating EOAEs. Frequency range: 0–6 kHz. Ordinates are in dB SPL.

Effects of intensity of contralateral carrier

Figure 5 shows mean EOAE attenuation as a function of the SL of the contralateral 1-kHz tone for an MF of 100 Hz and an MD of 100% for subjects with normal hearing and for patients with a total unilateral hearing loss.

NORMAL SUBJECTS. The mean AM tone perception threshold was 4.5 ± 2.6 (SD) dB SPL. No significant shift in EOAE amplitude was observed for contralateral stimulus

Hz for an MD of 75%. For these two MDs, the greatest contralateral EOAE amplitude attenuation effects were obtained for MFs of 100 and 140 Hz. For an MD of 50%, the contralateral attenuation effect was significant only for MFs of 140, 200, and 400 Hz, the greatest and most highly significant effect being obtained for the 400-Hz MF. Last, for an MD of 25%, statistically significant attenuation was observed only at 50 Hz.

FIG. 2. EOAE amplitude reduction as a function of type of contralateral stimulation. EOAEs were elicited by a 1-kHz tone pip. Ipsi- and contralateral stimulus intensities were 60 dB SPL and 40 dB SL (i.e., 42.6 ± 5.1 dB SPL for pure and AM tones), respectively. EOAEs were recorded in 48 subjects without and with 2 types of contralateral stimulation: 1-kHz pure tone, 1-kHz AM tone (MD 100%, MF 100 Hz). Bars: SE. Ordinate: relative EOAE amplitude [EOAE without contralateral acoustic stimulation (CAS) = 0 dB], NONE, without CAS; PT, pure tone; AMT, AM tone. Triple asterisk: $P < 0.001$.

FIG. 3. EOAE amplitude reduction as a function of contralateral MD. EOAEs were elicited by a 1-kHz tone pip. Ipsi- and contralateral stimulation intensities were 60 dB SPL and 40 dB SL (i.e., 46.6 ± 4.0 dB SPL), respectively. EOAEs were recorded in 12 subjects without and with a 1-kHz tone with MF 100 Hz and with MD varying from 0 to 100% by 12.5% steps. Bars: SE of the EOAEs. Ordinate: relative EOAE amplitude. Single asterisk: $P < 0.05$. Double asterisk: $P < 0.01$. Triple asterisk: $P < 0.001$. 

$\text{Triple asterisk: } P < 0.001.
intensities ≤30 dB SL (i.e., 34.5 ± 2.6 dB SPL). However, for contralateral SLs >30 dB SL, EOAE attenuation increased with contralateral stimulus intensity, reaching significance (P < 0.05 at 40 dB SL and P < 0.001 at 50 and 60 dB SL).

Patients with a total unilateral hearing loss. No significant differences were observed in EOAE amplitude for any contralateral stimulation intensity (paired t-test).

Effects of frequency of contralateral carrier

Figures 6 and 7 show mean EOAE amplitude shift as a function of contralateral carrier frequency for two different frequencies of the ipsilateral tone pip used to evoke EOAEs (i.e., 1 and 2 kHz). The modulation parameters, MF and MD, of the contralateral stimulus were held constant at 100 Hz and 100%, respectively. With ipsilateral 1-kHz tone pip stimulation (Fig. 6, left), the greatest and only statistically significant (paired t-test, P < 0.001) mean shift in EOAE amplitude was obtained for a contralateral carrier frequency of 1 kHz. Further insight into the frequency specificity of the suppression effect is provided by measurements over frequency bands near the center frequency of the ipsilateral tone pip as a function of the carrier frequency of the contralateral AM tone. With the ipsilateral tone pip at 1 kHz (Fig. 6, right), a significant reduction in EOAE amplitude was found in the 0.73- to 0.93-kHz band for a contralateral carrier frequency of 1 kHz (P < 0.05) but not in the two other bands. However, even if no statistically significant decreases can be observed for the 0.98- to 1.17-kHz and 1.22- to 1.42-kHz bands, the greatest decreases can be observed for a contralateral carrier frequency of 1 and 1.5 kHz, respectively.

Fig. 4. Combined effect of contralateral MF and MD on EOAE amplitude. EOAEs were elicited by a 1-kHz tone pip. Ipsilateral and contralateral stimulus intensities were 60 dB SPL and 40 dB SL (i.e., 44.4 ± 2.6 dB SPL), respectively. EOAEs were recorded in 21 subjects without and with a 1-kHz AM tone (MD 100%) with MFs varying from 50 to 800 Hz in half-octave steps. Contralateral MDs were 100% (A), 75% (B), 50% (C), and 25% (D). Bars: SE of the EOAEs. Ordinate: relative EOAE amplitude. Single asterisk: P < 0.05. Double asterisk: P < 0.01. Triple asterisk: P < 0.001.

Fig. 5. Effect of contralateral stimulus intensity on EOAE amplitude. EOAEs were elicited by a 1-kHz tone pip with intrameatal stimulus level fixed at 60 dB SPL. EOAEs were recorded in 24 subjects (●) without and with a contralateral AM tone (MD 100%, MF 100 Hz) with intensity varying from −10 to 60 dB SL (i.e., from −5.1 to 64.9 ± 2.5 dB SPL). EOAE amplitude attenuation in 3 patients (▲) with total unilateral hearing loss. Contralateral intensities varying from −10 to 60 dB over the threshold of the normal sample. Ordinate: relative EOAE amplitude. Bars: SE of the EOAEs. Single asterisk: P < 0.05. Double asterisk: P < 0.01. Triple asterisk: P < 0.001.
With the 2-kHz ipsilateral probe stimulus (Fig. 7, left), the strongest and only significant decrease in EOAЕ amplitude was observed for a contralateral carrier frequency of 1.5 kHz ($P < 0.05$). The spectral analysis of the 2-kHz tone pip responses shows a significant reduction in EOAЕ amplitude in the 1.95- to 2.15-kHz band ($P < 0.05$). The greatest, albeit nonsignificant, decrease was observed in the 1.71- to 1.90-kHz band with the carrier frequency of the contralateral stimulus at 2 kHz.

For convenient reference, the amplitude spectra of the 1- and 2-kHz tone pips used as ipsilateral stimuli to elicit EOAЕs are illustrated in Fig. 8.

**Discussion**

The main finding of the present study is that AM tones, used as contralateral stimuli, exert a suppression effect on EOAЕ amplitude. In the following discussion, this result is interpreted first in relation to the general mechanisms of contralateral EOAЕ suppression and then in relation specifically to the AM effect.

**General mechanisms of contralateral EOAЕ suppression**

MECHANICAL INTERACTION BETWEEN THE EARS: TRANCRA-NIAL TRANSFER AND ACOUSTIC CROSSOVER. In the present study, with the use of AM tones as contralateral stimuli, a significant attenuation of EOAЕ amplitude was obtained at lower SLs, namely 40 dB SL. With the use of pure tones as the contralateral stimulus, no significant contralateral EOAЕ amplitude suppression effect was observed at even lower SLs. At such a low intensity, transcranial transfer and cross talk are very unlikely to play a role in the EOAЕ attenuation effect. This finding is in agreement with the absence of suppression effect on EOAЕ amplitude found with pure tones from 20 to 60 dB hearing level as contralateral stimulation (Berlin et al. 1993). Besides stimulus intensity, when the contralateral stimulation is presented to the deaf ear of a subject presenting a total...
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FIG. 8. Amplitude spectra of the EOAES (top) and of the stimuli (i.e., 1- and 2-kHz tone pips) (bottom). Spectra were derived from EOA waveforms in 21 subjects, without contralateral stimulation. a: EOAEs elicited by a 1-kHz tone pip. b: EOAEs elicited by a 2-kHz tone pip. Frequency range: 0–6 kHz. Ordinates are in dB SPL.

unilateral hearing loss, no attenuation of EOA amplitude is obtained in the healthy, ipsilateral ear (Collet et al. 1990; Veuillet et al. 1991). This result was confirmed in the present study with the use of AM tones. No effect on EOA amplitude was observed in patients with a total unilateral hearing loss. This observation supports the notion that, whatever the type of contralateral stimulus, the contralateral EOA suppression effect is produced by a sensorineural rather than purely mechanical interaction between the ears.

ACOUSTIC REFLEX AND INVOLVEMENT OF THE MIDDLE EAR.

The acoustic reflex results in a reduction in the transmission power of the ossicular chain and could explain the attenuation of EOA amplitude observed on contralateral stimulation, because EOAes travel backward through the middle ear (Harrison and Burns 1993). However, involvement of the middle ear may be questioned, because the suppression effect persists in human subjects with unilateral stapedial reflex loss (Veuillet et al. 1991). Complementarily, it has been shown that the EOA suppression effect is dramatically reduced in patients whose olivocochlear bundle has been cut during surgical neurectomy of the vestibular nerve to release severe vertigo in Ménière’s disease (Giraud et al. 1995; Williams 1993, 1994). Furthermore, contralateral EOA amplitude attenuation is obtained at ipsilateral stimulus frequencies far above the range of frequencies over which the acoustic reflex exerts its effects. Moreover, as pointed out initially by Liberman (1989), frequency specificity of the suppression effect can hardly be explained in terms of middle ear functioning.

MOCS FUNCTIONING.

Some studies have shown that EOA suppression is induced by modification of OHC motility under contralateral stimulation, by medial efferent neurons (Liberman 1988; Liberman and Brown 1986; Mountain 1980; Siegel and Kim 1982). Brown and Nuttal (1984) have suggested that CAS alters OHC membrane potential in the mammalian cochlea, producing a modification of the stereocilia-tectorial complex membrane. Thus the effect observed is an EOA amplitude reduction.

A decisive argument for an involvement of the MOCS in the contralateral suppression effect is the frequency specificity of this effect. In the present study, EOA amplitude was attenuated by contralateral AM tones in a frequency-selective manner. The greatest decreases were observed when the frequency of the contralateral AM tone was close to the frequency of the studied EOA: i.e., 1 and 2 kHz. These results are in agreement with those of Veuillet et al. (1991), who demonstrated frequency specificity of the contralateral NBN stimulation effect on EOA amplitude. Chéry-Croze et al. (1993) showed an equivalent frequency specificity with distortion products under contralateral NBN.

For the 1-kHz EOAes (total EOAes and frequency bands in spectrum analysis), the suppression effect was clearly greater for the AM tone with a carrier frequency of 1 kHz. For the 1.22- to 1.44-kHz band, it was the AM tone with a carrier frequency of 1.5 kHz that showed some frequency specificity. However, the suppression effect induced by a 2-kHz tone pip was less obvious than at 1 kHz. For the 2-kHz EOAes, it was the 1.5-kHz AM tone that produced the greatest suppression effect. The frequency content of the stimulus can partly explain this result (see Fig. 8), all the more in that in the 1.95- to 2.15-kHz band the greatest suppression effect was found for the AM tone with a carrier frequency of 2 kHz. Figure 8 compares the frequency spectra of stimuli used (bottom: 1 and 2 kHz tone pip) and the frequency spectra of responses obtained (top). It appears that, if the spectra of EOA responses elicited by a 2-kHz tone pip were larger, this would be due simply to the stimulus being more intense. This finding of frequency specificity fails to confirm findings of Elberling et al. (1985) but is in agreement with those of Veuillet et al. (1991), Bray (1989), Probst et al. (1986), and Wit and Ritsma (1979). This frequency specificity of the EOA suppression effect may be explained by the existence of a reciprocal tonotopy of the afferent and efferent pathways (Brown 1989; Cody and Johnstone 1982; Liberman and Brown 1986; Robertson 1984; Robertson and Guomer 1985; Robertson et al. 1987; Warren and Liberman 1989b).

Specific mechanisms of AM contralateral suppression

Because a correction factor was applied to the amplitude of the AM tone to hold energy constant between the unmodulated and modulated tones, interpretations based on an en-
ergy factor can be ruled out. In other words, the effect appears to be related to differences in temporal distribution of the whole energy of these two types of CAS.

The present work shows that the amplitude of the suppression effect depends on several factors. The first parameter of the suppression effect is the intensity of the contralateral stimulus. Results indicate that the contralateral AM tone induced a significant decrease in EOAE amplitude for contralateral stimulus intensities $\geq 40$ dB SL. Second, the deeper the modulation of the contralateral tone, the larger the suppression effect. Third, for the two MDs (100% and 75%), the greater effect was obtained with the use of an AM tone with MFs of 100 and 140 Hz (Fig. 4). This is why many of the experiments performed in this study specifically involved 100 Hz-MF and 100% MD. However, for an MD of 100%, considerable variations were observed. This result may be related first to the existence of multiple groups of subjects that were run with variations of the parameters and next to the results of previous studies indicating substantial intersubject variability in contralateral suppression effects (Collet et al. 1992; Giraud et al. 1995; Norman and Thornton 1993).

Some data on neuroanatomy may on the one hand indicate that the MOCS is activated by AM tones, and yet be relevant to our MD and MF findings. Modulation gain values recorded from AN fibers indicate the preservation of AM in discharges. AN afferent fibers contact the ventral cochlear nucleus (VCN). Two principal morphologically defined cell types, called bushy (primary-like) and stellate (chopper) cells, have been described in the VCN. Various studies by Moller (1976, 1977), Frisina (1983), and Frisina et al. (1985, 1990a,b) have shown that primary-like units as well as chopper-type cells of the VCN preserve or enhance AM compared with AN fibers. The enhancement takes place in the temporal domain; the response of these cells is strongly synchronized on the stimulus modulation period. These results suggest the hypothesis that certain types of VCN cells are involved in sharpening the representation of the temporal features of the stimulus. Inputs to MOCS neurons are derived, to some extent, bilaterally from the VCN, but mainly contralaterally (Thompson and Thompson 1991). Thus, if the encoding properties of MOCS neurons derive from those of VCN cells, we can suppose that the coding of AM is conserved through the MOCS.

Gummer et al. (1988) studied discharges from olivocochlear efferent neurons in the guinea pig cochlea with the use of AM tones. Modulation transfer functions, in most cases, showed a peak in modulation response at an MF of 100 Hz. It appears that efferent modulation responses have the same behavior as higher auditory neurons in that they have band-pass functions with a maximum reproduction of AM at an MF of $\sim 100$ Hz (see discussion in Gummer et al. 1988). These facts argue for MOCS sensitivity to AM tones. Several neurophysiological studies concerning the cochlear nucleus tend to confirm the predominance of the 100-Hz MF on the suppression effect observed. In the gerbil VCN, the modulation frequencies at the peak of the modulation transfer functions of primary-like units are within the 180- to 240-Hz range, whereas those of chopper units cover a larger range, $\sim 80$–520 Hz (Frisina 1983: Frisina et al. 1990a). The range covered by chopper units corresponds to MFs producing a contralateral suppression effect on EOAE amplitude. The anatomic link between the VCN and MOCS, as well as the previous data, is thus consistent with the results obtained in our study.

The exact mechanism underlying the stronger effect of AM tone CASs on EOAE amplitude via MOCS activity, however, remains speculative. Nevertheless, the demonstrated ability of MOCS fibers to encode AM very efficiently provides a basis for a possible mechanism. Indeed, it might be that CAS amplitude fluctuations reduce the neural adaptation of MOCS fibers compared with that found with continuous stimuli.

In conclusion, the results of the present study, indicating a particular sensitivity of the MOCS to AM stimuli, suggest that this system could play a specific role in the encoding of environmental auditory stimuli, that is, with envelopes showing temporal fluctuation, as is the case in most animal and human communication signals.

The authors are grateful to Professor M. Charles Liberman and Professor John D. Durrant for helpful comments on an earlier version of the manuscript. We gratefully acknowledge the anonymous reviewers.

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Received 16 April 1996; accepted in final form 22 November 1996.

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