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Research report

## Evidence of a medial olivocochlear involvement in contralateral suppression of otoacoustic emissions in humans

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### Abstract

Otoacoustic emissions (OAEs) evoked by click stimuli were recorded in both ears of 20 normal human subjects, in the presence and absence of a contralateral masking broad band noise. No difference in the amplitude of OAE suppression was noted between the first tested ear and the second one. In addition, 20 pathological subjects were tested according to the same protocol. Ten of them belonged to a group of patients whose vestibular nerve was sectioned on one side to relieve incapacitating vertigo and thus represented a group in whom olivocochlear efferents were severed. A great reduction of suppression observed in the operated ear suggested that olivocochlear efferent fibers are necessary to obtain a full suppressive effect. Three of the pathological subjects were patients who had undergone a decompression of the facial nerve which necessitated the same surgical approach as vestibular neurotomy, but without any section of vestibular fibers. This surgical control group demonstrated that the surgical act by itself cannot explain the difference observed in the neurotomized group. Finally, seven of the pathological subjects were patients with Bell's palsy, which paralyzes the facial nerve and abolishes the stapedial reflex. No suppression difference was observed between healthy ears and ears without stapedial reflex. Therefore, it appeared that the stapedial reflex was not involved in the contralateral suppression of EOAEs. However, as the tensor tympani muscle remained functional in these patients, its involvement in the suppressive effect cannot be excluded.

*Keywords:* Evoked otoacoustic emission; Acoustic reflex; Medial olivocochlear efferent; Vestibular neurotomy

### 1. Introduction

Evoked otoacoustic emissions (EOAEs) are sounds that can be recorded in the external ear canal in response to acoustic stimuli [12], and whose origin is thought to be the motile activity of outer hair cells (OHC) [2]. EOAEs are complex signals of short duration that are theoretically composed of all the frequencies contained in the stimulus. When a sound is applied contralaterally to the ear in which EOAEs are recorded, their amplitude decreases [6,7,21] and their phase is shifted [9,19]. Although these effects are now well known and characterized, the mechanisms underlying these modifications are still controversial. Since no decrease in EOAE amplitude was observed when recording subjects with unilateral deafness, the suppressive effect was thought to be elicited for contralateral levels too weak to involve a bone conduction mechanism [7]. This suggests

an interaction between both ears which could originate either in a middle ear or an inner ear mechanism.

The first hypothesis of a middle ear mechanism involves the acoustic reflex pathway. The acoustic reflex leads to the contraction of the contralateral stapedius muscle that would decrease the amplitude of the EOAE signal by decreasing the amplitude of tympanum vibrations. However, the findings of Veuillet et al. [22] demonstrated that variations of the middle ear pressure could not explain all the variations of EOAE amplitude in the presence of a contralateral sound. Moreover, the suppressive effect was found to be larger for very weak intensities [21] which would normally not elicit the acoustic reflex.

The second hypothesis, which involves a direct cochleo-cochlear interaction, is based on anatomical data showing that OHCs are innervated by efferents coming from the contralateral superior olivary complex [18]. Electrophysiological experiments have shown that electrical stimulation of the crossed part of the olivocochlear bundle (OCB), in animals in which middle ear muscles were severed, was able to decrease the responses in the auditory

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nerve [10,13,23,24] as well as otoacoustic emissions [16]. This cochleo-cochlear interaction is also the most probable assumption in humans because the suppressive effect was found to be frequency specific in EOAEs [21] and distortion product otoacoustic emissions [4]. In fact, a contralateral narrow band noise induces a specific decrease in the EOAEs, affecting the very frequencies included in the contralateral sound. This suggests a mechanism that would respect a tonotopical distribution, such as the medial olivocochlear efferent innervation along the cochlea. Moreover, the suppressive effect is very reduced on the 4 kHz component of OAEs [17,21], a result which was interpreted as a functional expression, in humans, of the discontinuity of efferent innervation observed in cats by Guinan et al. [11], around this frequency.

Although much physiological evidence supports the hypothesis of an inner ear mechanism, the physiological mechanism underlying the suppressive effect is still a matter of discussion. The current study was aimed at providing consistent experimental data in order to determine the respective involvements of the crossed OCB (COCB) and the stapedial reflex in the suppressive effect in humans.

In humans, two pathological models might be very useful to test each physiological hypothesis. One model is the subject in whom efferent fibers have been sectioned. According to the anatomical data of Rasmussen [18], cochlear efferents travel along the vestibular tract. Warren and Liberman [23] demonstrated, in cats, a decrease in the ratio of retrogradely labelled neurons, in the superior oli-

vary complex, following the section of the vestibular nerve at the level of its anastomosis with cochlear fibers. Thus, vestibular neurotomy is thought to result in the sectioning of olivocochlear efferent fibers [25]. In humans, vestibular neurotomy is carried out to relieve incapacitating vestibular disorders. A large group of patients with vestibular neurotomy were tested here in a click-EOAE protocol, designed to quantify the effectiveness of the interaction between both ears. If a reduction of this effectiveness can be demonstrated in these patients, this would support the hypothesis of a COCB involvement in the contralateral suppression. This human model used in psychoacoustic experiments helped to demonstrate the perceptual role of cochlear efferents [20]. A second pathological model is the subject with absence of acoustic reflex. This occurs in the case of Bell's palsy that paralyses the facial nerve unilaterally and consequently the reflex contraction of the stapedial muscle whereas the tensor tympani muscle remains functional. If a quantifiable reduction of suppression can be measured in such patients, the stapedial reflex might also play a role in the suppressive effect. A group of patients with Bell's palsy were tested using the same EOAE protocol as the patients with vestibular neurotomy. In addition to these experimental groups, two control groups were also studied, one consisting of healthy subjects and one of subjects who had undergone a facial nerve decompression without vestibular section in order to rule out a possible effect of the operation itself. Suppression of EOAEs was compared among the groups using a new index of measurement described by Chéry-Croze et al. [5].

Table 1  
Status of the four groups of patients

	Normal subjects	Operated subjects		Bell's palsy patients
		Vestibular neurotomy patients	Patients operated for hemifacial spasm	
Sample size	$n = 20$ (8 F, 12 M)	$n = 10$ (5 F, 5 M)	$n = 3$ (2 F, 1 M)	$n = 7$ (2 F, 5 M)
Age	mean = 23 years, S.D. = 2.54	mean = 44.1 years, S.D. = 13.6	male: 64 years; females: 31 and 57 years	mean = 37 years, S.D. = 20.57
Pathology	None	Meniere's disease $n = 8$ ; acoustic neuroma $n = 1$ ; atypical vertigo associated with Klippel-Trenaunay syndrome $n = 1$	Syndrome attributed to vascular pulsations against the entry zone of the facial nerve (VIIth)	Paralysis of the VIIth nerve
Physiological characteristics	Intact olivocochlear efferent fibers	Olivocochlear efferent fibers cut	Surgical control	Absence of stapedial reflex
Hearing level	Better than 10 dB HL	Better than 10 dB HL on 5 out of the 6 tested frequencies	Better than 10 dB HL	Better than 10 dB HL
Acoustic reflex to contralateral broad band noise	Reflexes bilaterally present	Reflexes bilaterally present	Reflexes bilaterally present	Reflex present on the healthy side, absent on the paralysed side
Tympanometry	Normal	Normal	Normal	Normal
Vestibulometry	Normal	Normal on the intact side; pathologic on the operated side	Normal	Normal

## 2. Material and methods

### 2.1. Subjects

The study, which consisted in the recording of click-evoked OAEs in both ears in the presence or absence of a contralateral broad band noise, was performed on 40 subjects. This population was divided into one reference group of 20 healthy subjects, and three pathological groups comprising 20 patients. All subjects were volunteers and experiments were carried out with the oral consent of healthy subjects and the written agreement of patients. Experiments were performed on four groups of subjects described in Table 1.

Healthy subjects all declared having no auditory congenital disease in their family. A questionnaire ensured that they had no otological disorders following severe infection, use of ototoxic drugs or exposure to long term noise. The normality of their auditory function was tested with audiometry and tympanometry. They all had a hearing level better than 10 dB HL (hearing level in decibels). The presence of acoustic reflexes to contralateral broad band noise and EOAEs in both ears was required to be included in the sample.

In a population of 140 patients operated on for vestibular neurotomy, 10 patients only were able to be included in the experiment according to the following post-operative vestibular and acoustic criteria. The required acoustic criteria were normal tympanometry, normal audiometry, the presence of broad band noise acoustic reflexes, and click-evoked otoacoustic emissions in both ears. The section of the vestibular nerve was verified in three ways. The patient was questioned to ensure that no vertigo persisted. The video recording of the operation was re-examined before experiments. The unilateral peripheral vestibular disorder was confirmed by three successive nystagmographic recordings using Ulmer Videonystagmoscope apparatus (Collin ORL) including examination of spontaneous horizontal optokinetic nystagmus and responses to bithermal caloric test: the first Videonystagmographic recording (VNG) was performed between 2 and 8 days after the operation, the second 3 months later, and the last one the day of otoacoustic recordings. In the vestibular neurotomy group, four patients were gathered into a subgroup because of the exact symmetry of their audiograms (hearing level was identical on all the 6 tested frequencies). This subgroup was designed to eliminate the possible involvement of asymmetry of hearing in the asymmetry of otoacoustic responses.

Patients operated on for hemifacial spasm (the surgical control group) underwent the same surgical processes as the vestibular neurotomized patients, using the retrosigmoid approach, but without any section of the vestibular nerve. Patients were chosen using identical acoustic criteria to those required of the normal subjects (normality of tympanometry, audiometry and the presence of bilateral

broad band noise acoustic reflexes). A vestibular examination ensured that they had no vestibular disorder on the operated side.

The selection criteria of patients with a paralysis of the VIIIth nerve (Bell's palsy patients) consisted in having normal hearing and having an absent stapedial reflex to contralateral broad band noise on the paralysed side and a normal reflex in the other ear.

### 2.2. Surgery

All the patients were operated on by the same surgeon under the same conditions. The retrosigmoid approach was chosen because it gives the best and easiest view of the acoustico-facial bundle, with low prevalence of complications. The patient was recumbent and a craniotomy limited to a 1.5 cm trepanation just behind the sigmoid sinus was carried out. The cerebellum fell away spontaneously without the need for retraction.

In the case of vestibular neurotomy, acoustic and vestibular nerves were separated and the vestibular tract was sectioned [14].

In the case of hemifacial spasm, the retrosigmoid surgical approach was coupled with an endoscopic procedure. The endoscope allowed the surgeon to recognize the irritative vascular contact from the normal arterial loop. Using an operating microscope the offending artery was carefully mobilized and held away from the nerve with a small Teflon foam pad [15].

### 2.3. Auditory screening

Tonal audiometry was performed in a soundproof room, using a Madsen DAIII audiometer at 0.25, 0.5, 1, 2, 4 and 8 kHz, according to ISO 6189 standards.

Tympanometry and acoustic reflex recordings were performed in a soundproof room using an Amplaid 702 impedancemeter.

### 2.4. Recording of EOAEs

EOAEs were recorded in a sound-proof room, according to the method of Bray and Kemp [1], using the Otodynamics ILO88 apparatus (Kemp et al., 1990). A miniaturized probe, composed of a loudspeaker (Knowles 1712 transducer) and a microphone (Knowles 1843), placed in the external ear canal, delivered acoustic stimulations and recorded responses. EOAEs were analysed during the 20 ms following the beginning of stimulation by non-filtered clicks of 80  $\mu$ s at a rate of 50/s. The Otodynamics ILO88 system controlled stimulus presentation and EOAE recordings. The recording sampling rate was 25,600 Hz. The amplitude of the response was calculated from 2.5 to 20 ms (stimulation artefact eliminated). In one session of recording 260 responses were averaged within a band-pass of 500 to 6000 Hz. All the recordings were performed

by means of the linear differential cochlear echo method. This stimulation mode uses the combination of four clicks of the same amplitude and polarity allowing one to record the non-linear residual cochlear response specifically. The intensity of the stimulus waveform expressed in decibels peak SPL was measured in the external ear canal and adjusted by ILO88 software. Responses were recorded in two buffers and averaged separately. The reproducibility of OEAEs was tested by the correlation between signals from the two buffers. The ear from which EOAEs were recorded was called ipsilateral.

### 2.5. Contralateral acoustic stimulation

Contralateral acoustic stimulation (CAS) was delivered via a Clinical Audiometer Amplaid 455 through a Telephonics TDH 39P (bandwidth 50 to 8000 Hz) earphone. CAS was a broad band noise (BBN) composed of frequencies comprised between 50 and 8000 Hz. The BBN spectrum was flat at the intensity used in the experiment. The subjective perception threshold of the contralateral white noise was measured for each subject.

### 2.6. Experiment

Click EOAEs were recorded in both ears according to the efferent system investigational protocol proposed by Collet et al. [7]. In a range comprised between 55 and 75 dB peak SPL, five ipsilateral stimulus intensity levels were tested in 3 dB steps, in a randomized presentation order with and without the contralateral sound. Firstly, the weakest stimulus level eliciting a significant response (response > noise) was searched for in both ears. Then, the five stimulus levels were taken behind the highest threshold in order to be equal in the two ears of each subject.

In patients, the pathological ear was systematically tested first. In healthy subjects, 10 subjects were tested first in the right ear and 10 subjects were tested first in the left ear.

### 2.7. Measurement of CAS effectiveness

Input/output functions were represented and MOC effectiveness was quantified by the Euclidean distance calculation method [5]. Usually, Equivalent attenuation (EA) was computed according to the definition of Veuillet et al. [21] and Collet et al. [8]. EA is defined as the increase in the stimulus level (dB) which would be necessary to obtain the same response, in the presence of CAS, as it would be in its absence. In our case, EA was not suitable because too many subjects presented input/output functions that were not exactly parallel in both the presence and absence of CAS. We preferred to compute Euclidean distance (ED) which is independent of the slope of regression lines (Fig. 1).

Its calculation required five intensity levels (I1 to I5)

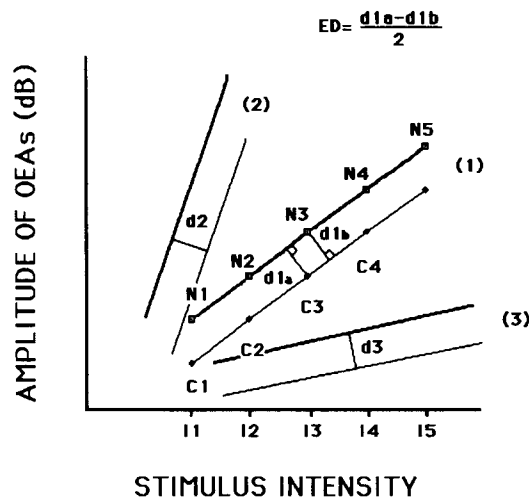


Fig. 1. Euclidean distance (ED) calculation method (from Chéry-Croze et al. [5]): five intensity levels (I1 to I5) correspond to five pairs of EOAE amplitudes, one measured in the presence of CAS (C1 to C5) and the other in its absence (N1 to N5). The ED is the mean between the distances (d) separating each experimental point N1 to N5 from the regression line of C1 to C5 points by an orthogonal projection, and the distances separating C1 to C5 points from the regression line of N1 to N5 points.

corresponding to five pairs of EOAE amplitudes, one in the presence of CAS (C1 to C5) and the other in its absence (N1 to N5). Fig. 1 shows the calculation of ED which is the mean between the distances (d) separating each experimental point N1 to N5 from the regression line of C1 to C5 points by an orthogonal projection, and the distances separating C1 to C5 points from the regression line of N1 to N5 points [5].

### 2.8. Statistics

Both ears were compared in each group using a paired Student's *t* test. Probabilities below 0.02 were considered significant. In each condition medians and semi interquartiles were computed. To estimate differences between groups, Student's *t* test (unpaired samples) was applied.

In the normally hearing group, in order to avoid effects due to a possible lateralization of the suppression, ears were not separated into right ear and left ear groups but into a 'first tested ear group' and a 'second tested ear group', each one comprising the same number of right and left ears.

## 3. Results

### 3.1. Normal subjects

The effectiveness of the suppressive effect was quantified by the calculation of Euclidean Distance. Fig. 2 shows the mean results for normally hearing subjects. No statisti-

Table 2  
Experimental results

Sides:	Normal subjects		Vestibular neurotomy (VNT) patients; n = 10		VNT patients: subgroup with symmetrical hearing; n = 5		Patients operated for hemifacial spasm		Bell's palsy patients	
	first	second	intact	operated	intact	operated	intact	operated	healthy	paralysed
Mean contralateral suppression of EOAEs (dB)	mean = 0.81 S.D. = 0.43	mean = 0.78 S.D. = 0.43	mean = 0.98 S.D. = 0.12	mean = 0.39 S.D. = 0.08 **	mean = 0.98 S.D. = 0.29	mean = 0.29 S.D. = 0.2 ***	mean = 0.80 S.D. = 0.33	mean = 0.60 S.D. = 0.2	mean = 1.11 S.D. = 0.4	mean = 1.05 S.D. = 0.23

Difference between intact and operated sides: \*\*  $P < 0.02$ , \*\*\*  $P < 0.01$ .

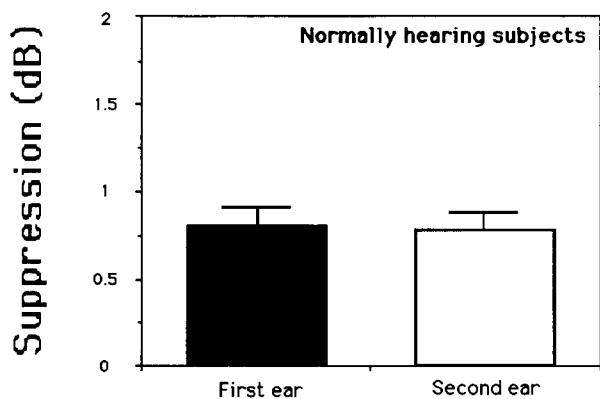


Fig. 2. Mean suppressions in dB are represented with standard error bars, for 20 normally hearing subjects. In black: first tested ear; in white: second tested ear.

cal difference was observed between the two ears (each group comprising 10 left and 10 right ears). The suppression was 0.81 dB in the ear tested first and 0.78 dB in the ear tested second with the same standard deviation of 0.43 (see mean results in Table 2).

On individual values of E.D., depicted in Fig. 3, great interindividual variations can be noted. Every subject had a positive suppression in both ears, although it was sometimes very weak as in the case of subjects BV, FG, DA. In several cases (subjects JD, DS and TV), the suppression was very asymmetric between the two ears. But neither effect of the side (no difference between the right and the left ear) nor of the order of testing (no difference between the first and the second tested ear), was evident.

### 3.2. Patients with vestibular neurotomy

As far as vestibular neurotomy patients are concerned, the mean suppressive effect was found to be very asymmetric: suppression was significantly greater in the healthy ear (0.98 dB) than in the operated one (0.39 dB)

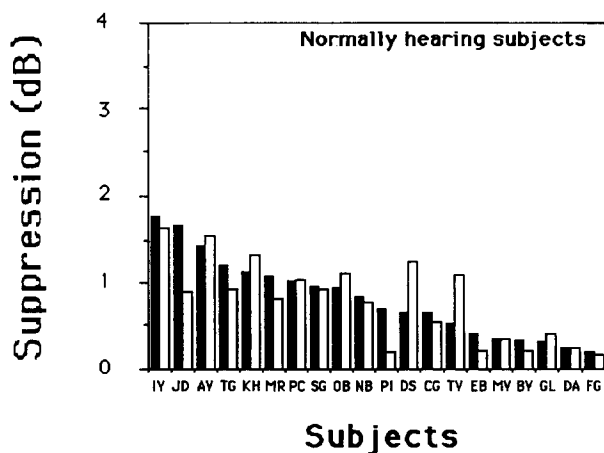


Fig. 3. Individual values of suppression, in dB, are represented in both ears. In black: first tested ear; in white: second tested ear.

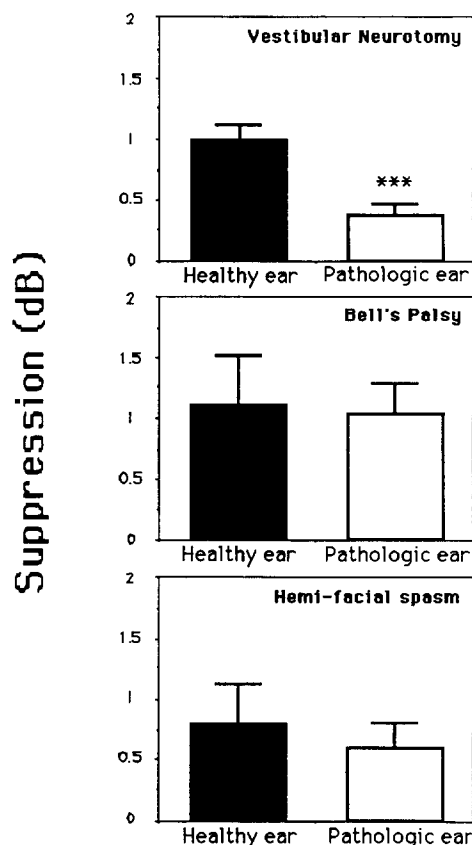


Fig. 4. Mean suppressions, in dB, are represented, with standard error bars, for each group of patients. Upper graph: vestibular neurotomy patients; middle graph: patients without acoustic reflex; lower graph: Patients operated for a hemifacial spasm (surgical control group). Stars indicate a significant paired *t*-test with  $P < 0.01$ . In black: healthy side; in white: pathological side.

with paired *t*-test (see Table 2 and Fig. 4). In the healthy ear, values of suppression were not statistically different from the values obtained in normal subjects whereas in the pathological ear, the suppression was significantly below normality ( $P = 0.02$ ). Although very reduced in the operated ear, the suppression was not totally absent, however (suppression value was statistically superior to 0).

Individual results (Fig. 5) showed that every patient except one had a good suppression in the healthy ear and a very reduced one in the other. The only exception was patient HO who presented a slightly greater suppression in the operated ear (0.37 in the normal ear and 0.41 dB in the operated one), and in whom balance and VNG tests confirmed the presence of a vestibular deficit. This patient had been operated for Menière's disease. The two subjects with other pathologies, either acoustic neuromas (subject RD) or atypical vertigo associated with a Klippel-Trenaunay syndrome (subject OG), both presented a strongly asymmetric suppression.

Patients SL, HO, AM, OG and SB were gathered into a subgroup because of the perfect symmetry of their hearing. Results, reported in Fig. 6, showed a significant difference

between suppression in the normal ear (0.98 dB) and suppression in the other (0.29 dB). The asymmetry was greater in this subgroup, in spite of the presence of subject HO who demonstrated almost equal values in both ears.

3.3. Patients with absent acoustic reflexes

In patients who contracted a Bell's palsy, the mean suppression was strongly symmetric (1.11 dB on the normal side and 1.05 on the paralysed side), and values given by the quantification method showed that there was a tendency toward a slightly higher value than in the group of normal subjects, but significance failed to be reached (Fig. 4). No statistical difference was found between suppression values of either side. These suppression values were not statistically different from those of normal subjects.

Individual values, reported in Fig. 5, showed that in three cases (NT, LR and PB) suppression was higher on the side where middle ear muscles were paralysed than on the normal side. In one case (SY), values were almost identical, and in the three last cases, suppression was higher on the normal side.

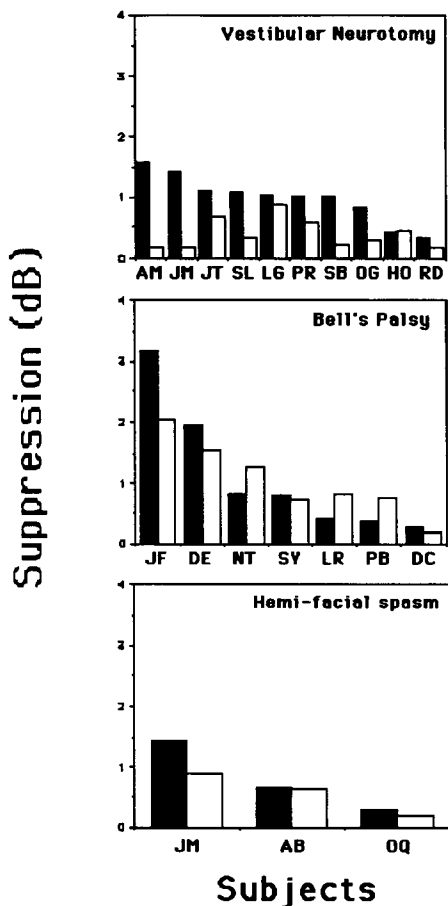


Fig. 5. Individual suppressions are represented, in dB, for each group of patients. In black: healthy side; in white: pathological side.

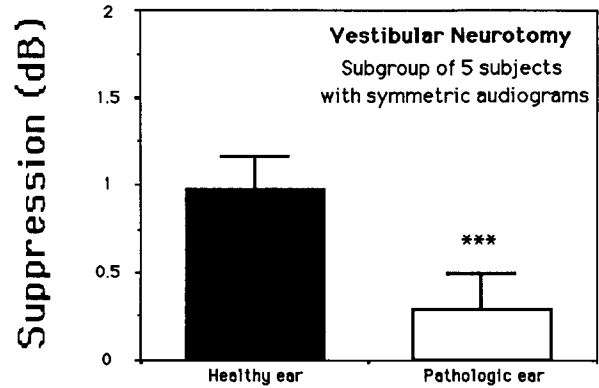


Fig. 6. Mean suppressions, in dB, with standard error bars are represented for the subgroup of neurotomized patients with symmetrical hearing.

3.4. Patients operated for decompression of the VIIth nerve

Means were computed with the three control patients (Fig. 4). Using *t*-test, no significant difference was evidenced between ears although the mean suppression value was slightly lower on the operated side (0.80 in the normal ear and 0.60 dB in the operated one). Suppression values in both ears were not different from those of normal subjects.

Two of the 3 patients (AB and OQ) operated on for a decompression of the VIIth nerve had almost symmetrical suppressions in both ears (Fig. 5). One subject (JM) presented an asymmetry but the operated ear showed a strong suppression (0.8 dB), although it was inferior to that of the healthy ear. Although one subject with vestibular neurotomy (patient LG) reached such a strong value of suppression in both ears (1.1 and 0.8 dB), all the other patients presented much lower suppressive effects in the operated ear.

4. Discussion

Findings in this study demonstrate that suppression of EOAEs by contralateral acoustic stimulation was present in both ears of all the 20 normally hearing subjects, but with large intra-individual variations; suppression varied within subjects between the ears, from exact symmetry to a difference of 0.8 dB. There was no evident reason, such as an effect of the order of testing, that could predict the direction of the variation between ears of a given individual. This study thus confirmed the great interindividual variations obtained by Collet et al. [8] who used the equivalent attenuation method. They found a mean of 2.47 dB of suppression with an S.D. of 1.67 dB. We consistently found a lower suppression (0.73 dB) because we used a different calculation method, but the S.D. was almost half of the mean value (S.D. = 0.43).

#### 4.1. Influence of vestibular neurotomy

In patients who had undergone a vestibular neurotomy, values of suppression as well as interindividual variations were normal on the intact side. But in 9 cases out of 10, the suppressive effect was significantly smaller on the operated side (0.39 dB by mean) than on the other side (0.98 dB) and than that obtained bilaterally in normally hearing subjects (0.81 and 0.78 dB). These results are consistent with the finding of Williams et al. [25,26], who noted no reduction in the amplitude of EOA signals in the operated ear of neurotomized patients. As the asymmetry between ears was greater in our subgroup with exactly symmetrical hearing (0.29 dB in the operated ear and 0.98 dB in the other), we postulate that the slight hearing loss persisting in operated ears does not explain the asymmetric suppression. The pathology of patients before the operation may not be at the origin of the asymmetry of suppression. In fact, 8 patients exhibited Menière's disease but two had other pathologies, i.e. acoustic neuromas and atypical vertigo with tinnitus. Both these latter showed strongly asymmetrical effects. As Williams et al. [26] previously proposed, the operation itself can also be excluded as a possible cause for asymmetric suppression in neurotomized patients. The three control patients operated on under the same conditions as the vestibular neurotomized patients but without vestibular section exhibited a slight but insignificant asymmetry with lower suppressions on the operated side. However, two of the three subjects had almost exactly symmetrical suppression values. Only one subject (JM) showed an asymmetry, but the smallest suppression (1 dB), obtained on the operated side, was within the upper range of normal values ( $0.80 \pm 0.43$ ). Moreover, suppression on the operated side did not differ from that of normal subjects.

#### 4.2. Influence of the absence of the stapedial reflex

In subjects with an absent acoustic reflex, symmetry between the ears was almost perfect (1.1 dB in the healthy ear and 1.05 dB in the paralysed ear). These mean values of suppression were greater than those obtained in normal subjects, because one subject (JPF) had very strong suppressions (3.2 and 2 dB). Other individual data showed suppression values comprised between 0.2 and 1.8 dB, exactly in the range of normality. Individual data indicated that, in almost half of the cases, suppression on the pathologic side was greater. Logically, a functional acoustic reflex should not be necessary to induce a suppressive effect. Evidence is provided here that the stapedial reflex is not involved in the suppression of evoked otoacoustic emissions.

#### 4.3. Respective roles of olivocochlear bundle and acoustic reflex in contralateral suppression of EOAEs

According to the most probable hypothesis, the integrity of the vestibular tract was necessary to obtain a normal

contralateral suppressive effect of EOAEs. In the absence of fibers travelling along the vestibular nerve, the suppressive effect was very reduced. The anatomical data of Rasmussen [18] showed that all the medial and lateral olivocochlear fibers travel along the vestibular nerve at the level of the section. Thus, medial olivocochlear efferents seem likely to be the fibers, the section of which inhibits the suppressive effect. Besides, the normality of suppression values in subjects with a non-functional stapedial muscle points to an involvement of the medial olivocochlear efferent system in the contralateral suppression of EOAEs. Two aspects of our results raise questions, however.

Firstly, mean values of suppression obtained on the operated side of neurotomized patients demonstrated that the suppressive effect was not completely absent in de-efferented ears. As a mechanism involving the stapedial muscle cannot explain the smallest part of the suppressive effect, another process may interfere in the contralateral suppression of EOAEs. According to Burns et al. [3], the cochlear efferent system may act synergistically with the middle ear non-acoustic efferent system. This hypothesis could involve a mechanism able to co-activate the tensor tympani muscle and olivocochlear efferents. As the tensor tympani muscle was still functional in patients with VIIIth nerve paralysis, our findings support this hypothesis.

A second problem arises from findings in patient HO who had undergone vestibular neurotomy. He presented a slightly greater suppression on the operated side, despite very weak suppression values in both ears (0.37 and 0.41 dB) – values that were almost equal to mean residual suppressions on the operated side of neurotomized patients. Since this patient exhibited an absence of vestibular reflex on the operated side, no doubt subsisted about the section of the vestibular nerve. No objective auditory criterion enabled us to distinguish this subject from the other neurotomized patients: he was one of those included in the subgroup of subjects with perfectly symmetrical hearing, he had EOAEs of normal amplitude and no spontaneous otoacoustic emissions. This subject demonstrated, however, that suppression can be obtained even when vestibular fibers are non-functional. Therefore, it can be postulated either that all efferent fibers do not travel along the vestibular nerve and, consequently, that not all were cut by the vestibular neurotomy, or that suppression, in this case, may be caused by an additional mechanism that does not involve a cochleo-cochlear interaction, such as an activation of a middle ear muscle, i.e. the tensor tympani muscle.

According to studies of Warren and Liberman [23] in cats, no significant decrease of afferent nerve discharges persisted after the section of the COCB at the level of vestibulo-cochlear anastomosis. These data in cats, coming directly from afferent nerve activity, suggest that all the COCB fibers were damaged during the vestibular neurotomy. Therefore, we can postulate that the residual sup-

pression of EOAEs observed in our study after vestibular neurotomy, involves structures other than COCB. However, the possibility of variations in the course of cochlear efferents should also be considered, in spite of a lack of anatomical data in the literature concerning the exact pathway of cochlear efferent fibers, as well as the category of fibers included in the vestibular tract in humans. In subject HO, the weak and symmetrical values of suppression obtained in the intact as well as in the operated side may be interpreted as a bilateral effect mediated by other structures such as the tensor tympani muscle.

## 5. Conclusion

Our present findings clearly demonstrate the involvement of the medial olivocochlear efferents in the suppression of EOAEs by a contralateral sound. However, a residual suppression that persisted after the section of the vestibular nerve suggested that another mechanism was likely to be involved. As the contribution of the stapedius muscle appears not to be necessary to obtain a full suppressive effect, the participation of a mechanism involving another part of the middle ear, such as the tensor tympani muscle, cannot be excluded. Findings in one neurotomy patient with greater suppression in the de-efferented ear suggest that the efferent fibers may not all travel inside the vestibular tract, at the level of the section. No anatomical data are currently available to confirm this hypothesis in humans.

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