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From otoacoustic emission to late auditory potentials P300: the inhibitory effect

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This study verifies the effects of contralateral noise on otoacoustic emissions and auditory evoked potentials. Short, middle and late auditory evoked potentials as well as otoacoustic emissions with and without white noise were assessed. Twenty-five subjects, normal-hearing, both genders, aged 18 to 30 years, were tested . In general, latencies of the various auditory potentials were increased at noise conditions, whereas amplitudes were diminished at noise conditions for short, middle and late latency responses combined in the same subject. The amplitude of otoacoustic emission decreased significantly in the condition with contralateral noise in comparison to the condition without noise. Our results indicate that most subjects presented different responses between conditions (with and without noise) in all tests, thereby suggesting that the efferent system was acting at both caudal and rostral portions of the auditory system.

Key words: efferent pathways, contralateral suppression, otoacoustic emissions, auditory evoked potentials

Abbreviations

(EOAE) Evoked Otoacoustic Emission;
(MOC) medial olivocochlear;
(AC) auditory cortex;
(MGB) medial geniculate body;
(IC) inferior colliculus;
(CAPD) Central Auditory Processing Disorder;
(ABR) Auditory Brainstem Response;
(MLR) -Middle Latency Response;
(LLR) Late Latency Response;
(TEOAE) Transient Evoked Otoacoustic Emission;
(CAS) Contralateral Acoustic Stimulation;
(AEP) Auditory Evoked Potentials;
(BIC) Binaural Interaction Components;
(CAEP) - Cortical Auditory Evoked Potential

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INTRODUCTION

The auditory system consists of ascending and descending (corticofugal) systems. The functional role of the efferent auditory system in humans has not yet been completely established and is difficult to demonstrate. Some methods employed in animals to study descending auditory pathways are often invasive and damaging to the auditory pathways, and consequently, they are not applicable in humans (Khalfa et al. 2001, Di Girolamo et al. 2007).

The rostral part of the efferent auditory pathway has received little attention and the anatomy and physiology of this region are not totally known in humans. In animals, this portion of the efferent system has been better studied (Khalfa et al. 2001, Perrot et al. 2006). Neurons in the deep layers of the auditory cortex (AC) project back to the medial geniculate body (MGB), inferior colliculus (IC) and subcollicular auditory nuclei. Corticothalamic fibers project only to the ipsi-

lateral MGB and thalamic reticular nucleus. However, corticocollicular fibers project bilaterally to the IC. The corticofugal projections are bilateral to the subcollicular nuclei. The central nucleus of the IC projects not only to the MGB and the superior colliculus, but also to medial olivocochlear (MOC) neurons, which mostly project to contralateral cochlear outer hair cells (Rasmussen 1946, Andersen et al. 1980, Huffman and Henson 1990, Warr et al. 1997, Suga et al. 2002, Suga and Ma 2003).

Studying the caudal portion of the efferent system in humans, many authors showed that there is a reduction of the cochlear response due to an inhibitory effect induced by contralateral noise activation of the MOC using a non-invasive procedure by recording of evoked otoacoustic emissions (EOAE) suppression (Collet et al. 1990, Veuillet et al. 1991, Berlin et al. 1993, De Ceulaer et al. 2001, James et al. 2002, Guinan 2006).

This mechanism provides an anti-masking effect that increases the discrimination of signal variation by reducing the cochlear amplification of the response to the noise. In addition, it supplies a feedback gain-control system for moderate sound levels that mediates selective attention and focuses attention during learning (Galambos 1956, Harkrider and Smith 2005, Lilaonitkul and Guinan Jr 2009).

On the other hand, in humans, the rostral part of the efferent auditory pathway is less understood, because direct clinical evaluation is difficult. However, there is some evidence for cortical influence on cochlear micromechanical verified through EOAE. This effect has been demonstrated in patients having undergone surgical removal of Heschl's gyrus (Khalfa et al. 2001), by electrical cortical stimulation in epileptic patients (Perrot et al. 2006) and by abnormal cortical functioning resulting in impaired cortical feedback to the brainstem and to the MOC system in dyslexic children (Veuillet et al. 2007).

The cortical influence on MOC system was also demonstrated in patients with (central) auditory processing disorder (CAPD), who often have difficulty in understanding speech accompanied by noise (Wiehing and Musiek 2008). Many patients with this kind of disorder present abnormal efferent system functioning that manifests as a reduced suppressive effect of EOAE when compared to controls (Muchnik et al. 2004, Sanches and Carvallo 2006).

Auditory Evoked Potentials (AEP) are used to assess functioning and integrity of the auditory pathway structures. The sound-evoked response of descending pathways has not been studied to any great extent, which makes it difficult to estimate the contribution to the Auditory Brainstem Response (ABR) from the structures of these pathways (Moller 2007). Concerning Middle Latency Response (MLR) and Late Latency Response (LLR), the identification of source contribution becomes more complicated as the chain of activity ascends the auditory pathway, but it is believed that later stages of processing can influence activity at earlier stages from the cochlea to the auditory cortex (Hackett 2007).

Some authors verified the effects of contralateral masking on different sites of auditory pathways, assessed by AEP. For this condition, Salo and coauthors (2003) verified a decrease of N1 amplitude and an increase of P2 amplitude, suggesting that these effects were mediated by the efferent hearing system and that further experiments to clarify the physiology of hearing should be performed. Similarly, Özdamar and Bohórquez (2008) identified a significant alteration in Pb wave of the MLR, suggesting that these results were influenced by central mechanisms.

Based on these assumptions, our hypothesis was that noise stimulates the efferent auditory system and that this stimulation could cause a modification of the responses within the entire auditory system, including the afferent auditory pathways. Thus, the aim of this study was to determine the effects of contralateral white noise on transient evoked otoacoustic emissions and on short, middle and late auditory evoked potentials.

METHODS

This study was carried out in the Auditory Investigation Laboratory with the approval of the Sao Paulo University School of Medicine Ethics Committee (Protocol no. 512/07). All participants provided written informed consent to participate in the study.

Twenty-five subjects, both genders, took part in this study and the ages ranged between 18 and 30 years (mean 25.3 years). The inclusion criteria were the following: no hearing complaint, no middle ear complaint, and normal hearing in both ears.

An audiometric assessment was conducted on all participants. Following otoscopic inspection, tympanometry was carried out using a GSI 38 Auto Tymp (Grason-Stadler Inc., Madison, WI) to document middle ear

integrity and to help rule out conductive hearing loss. An audiologic evaluation for pure-tone thresholds was carried out with a GSI 61 Clinical Audiometer (Grason-Stadler Inc., Madison, WI) using standard audiometric techniques in a sound-attenuated testing room to ensure normal hearing (<20 dB from 250 to 8 000 Hz).

Following the OAE, the electrophysiologic tests (ABR, MLR, LLR including N1, P2 and P300) were carried out in an electric and sound-attenuated testing room.

Transient Evoked Otoacoustic Emissions (TEOAEs) were recorded using an ILO 292 OAE analyzer, version 5.61 (Otodynamics Ltd., Hatfield, UK) using ILO insert phones. The TEOAEs were recorded in a linear mode with and without contralateral acoustic stimulation (CAS). Under all conditions, the mean intensity of the clicks was 60–65 SPL, and 200 sweeps were recorded for each ear (100 with and 100 without noise). This particular intensity was chosen based on previous studies conducted in humans (Hood et al. 1996, Ryan and Kemp 1996, Veuillet et al. 1996, Garinis et al. 2011) and to rule out the involvement of middle ear mechanisms (Giraud et al. 1996). The response level was determined by measuring the signal-to-noise ratio with an analysis time window of 4–20 ms.

The linear transient click, a combination of four identical acoustic impulses of 80-μs duration presented at a rate of 50 Hz in alternating blocks (with and without CAS), was registered on channel A delivered by ILO 292 system protocol (Lyon Mode-Otodynamics Ltd.). The data set from the test with CAS was designated memory store 1, and that from the test without CAS was designated memory store 2.

Whereas that middle-ear muscle reflex has been found near 65 dB HL, normally 60 dB HL broadband noise is used as MOC elicitor (Guinan et al. 2003). The CAS consisted of continuous broadband white noise at 60 dB SPL delivered through channel B of the ILO and presented by an ILO General Purpose TE SGS-8 probe (insert phone).

The individuals were tested in a sound booth in a quiet room and all of them were tested bilaterally. Aafter the two probes were in place, TEOAEs were recorded in alternating blocks (with and without CAS) and were always recorded in the same order. TEOAE suppression was calculated by subtracting the with-CAS TEOAE level from the without-CAS TEOAE level. The analysis of TEOAE suppression was conducted using only the response with limited frequency range to only 4–6 kHz, as referenced by Hood and colleagues and (1996).

Electrophysiological evaluation was through investigation of the ABR, MLR, N1-P2 and the P300 with and without contralateral white noise using a two-channel electroneuromyograph (Biologic Traveler Electrodiagnostic Testing System; Biological Systems Corp., Mundelein, IL, USA). The stimulus was 70 dB nHL and the noise was 60 dB nHL (Burkard and Hecox 1983, Burkard and Sims 2002, Özdamar and Bohórquez 2008, Cone 2009) with a +10 dB signal-to-noise ratio for all tests applied. The signal and the noise were delivered through TDH 39 earphones. The individual was asked to remain with eyes closed during the recording of potentials, in order to control eye movement artifacts.

For the ABR, a rate of 19 clicks per second with 0.1 μs duration was used with a filter slope 12 db/octave, with the low filter setup in 30 Hz and the high filter in 3 000 Hz and 2 000 sweeps.

For the MLR, we used the rate of 10 clicks per second with 0.1 μs duration, filter of 20–200 Hz and 1 000 sweeps. Both ABR and MLR measurements were duplicated to ensure fidelity.

The oddball paradigm was used in P300 recordings. This paradigm was based on distinguishing between a target stimulus repeated randomly (20% of the time) and the non-target stimuli with frequent repetition (80% of the time). Subjects were asked to count the stimuli when they discriminate the target stimulus. Monaural auditory stimulus was presented for the without-noise condition; this stimulus was delivered to each ear separately for both conditions. For the with-noise condition, contralateral noise was added. Frequencies were 1 000 Hz for the frequent stimuli (non-target) and 1 500 Hz for the rare (target) stimulus.

The electrode sites used were the following: Cz (coronal midline), A1 (left ear) and A2 (right ear) for the ABR, N1–P2 and P300; C3 (left coronal), C4 (right coronal), and A1 and A2 for the MLR. For all tests FPz (frontal hairline) was used as ground. Electrode impedances were always less than 5 kΩ.

Standard Bio-logic ER-3A insert earphones were used to deliver the sound stimuli for the electrophysiologic tests.

All tests used in the study were performed in dichotic condition, i.e., one ear received clicks stimuli while

Table I

Mean, median, standard deviation (SD) and the *P*-value of transient evoked otoacoustic emissions (TEOAE) (in dB) in conditions without noise (A) and with noise (B) (upper); and mean, median, standard deviation (SD) and *P*-value of waves I, III and V with respect to latency (in ms) and amplitude (in μv) in conditions without noise (A) and with noise (B) (down)

	Otoacoustic emissions	
	A	B
Mean	7.66	6.53
Median	8.25	7.40
SD	5.14	4.80
P-value	<0.00001*	

	Wave I		Wave III		Wave V	
Latencies	A	B	A	B	A	B
Mean	1.61	1.64	3.74	3.79	5.60	5.65
Median	1.60	1.64	3.72	3.76	5.56	5.64
SD	0.15	0.13	0.17	0.21	0.17	0.18
P-value	0.072#		0.003*		0.009*	
Amplitudes	A	B	A	B	A	B
Mean	0.26	0.21	0.28	0.26	0.45	0.44
Median	0.25	0.21	0.25	0.26	0.45	0.43
SD	0.13	0.10	0.12	0.11	0.15	0.15
P-value	0.010*		0.384		0.917	

* *P*-value statistically significant; # marginal statistical significance

Table II

Mean, median, standard deviation (SD) and p-value of waves Na (C3 and C4 electrode site positions) and Pa (C3 and C4 electrode site positions) latencies (in ms) and of Na–Pa (C3 and C4 electrode site positions) amplitudes (in μv) in conditions without noise (A) and with noise (B)

	Na (C3)		Pa (C3)		Na (C4)		Pa (C4)	
Latencies	A	B	A	B	A	B	A	B
Mean	16.69	18.77	33.43	31.14	18.90	19.12	33.45	33.83
Median	18.72	18.33	33.35	33.35	18.72	18.72	33.35	33.93
SD	1.19	1.74	2.49	2.78	1.68	2.31	2.19	2.58
P-value	0.661		0.068#		0.369		0.238	

	C3		C4	
Amplitudes	A	B	A	B
Mean	2.39	2.11	2.21	1.89
Median	1.89	1.73	1.88	1.31
SD	2.03	1.76	2.05	2.09
P-value	0.128		0.097#	

marginal statistical significance

Table III

Mean, median, standard deviation (SD) and *P*-value of waves N1, P2 and P300 latencies (in ms) and amplitudes (in μv) in conditions without noise (A) and with noise (B)

	N1		P2		P300	
Latencies	A	B	A	B	A	B
Mean	88.86	89.84	156.06	158.40	319.06	316.66
Median	89.00	89.50	151.00	154.00	326.00	316.00
SD	8.79	7.35	19.77	17.52	41.58	42.78
P-value	0.339		0.189		0.604	
Amplitudes	A	B	A	B	A	B
Mean	3.88	3.23	3.50	2.92	10.19	9.19
Median	3.58	2.97	3.43	2.70	9.59	7.79
SD	1.67	1.31	1.82	1.45	4.42	5.64
P-value	<0.0001*		<0.0001*		0.090#	

**P*-value statistically significant; # marginal statistical significance

the other received white noise, the only difference being the OAE using blocks of interrupted noise and the other potentials using continuous noise.

Latency and amplitude of the waves were analyzed and for the P300 we subtracted the target minus the no target stimuli and the latency was measured at the most positive point (amplitude) from 250 to 650 ms.

Any change in the relationship between with and without noise conditions in either latency or amplitude observed was considered to be indicative of an inhibition effect regardless of the size of the effect.

For the statistical analyses, Kolmogorov-Smirnov test was applied to determine normality of variables distribution. For comparison between with and without noise conditions we used ANOVA (one factor), and for others analyses we used ANOVA with repeated measures. The level of significance was set at 0.05. Significant values were designated with an asterisk (*). The power analyses was calculated based on 1.4 times the value of the confidence interval plus the variability of the data, resulting in 0.78 power.

Because the tests and variables studied have different units and magnitudes, it was necessary to transform the data so as to put all variables on the same scale and then compare the conditions with and without noise. The variable of interest was the condition "with noise". as we were concerned with the changes that might occur after the insertion of noise; for that reason, all variables were considered based on the "with noise condition". Thus, all the variables have the same percentage scale of "without noise condition" in relation to the "with noise". For this transformation, the formula $\frac{(A-B)}{B}\times 100$ was applied, where A was the condition "without noise" and B was the condition "with noise". Negative values of this measure indicated that values were larger in the condition without noise, while positive values indicated that they were smaller.

For the last analysis a multinomial model (with 16 response categories) was done *via* weighted least squares (Agresti 2002). Estimates and standard errors of the inhibition probabilities were done.

RESULTS

Comparison between conditions without and with noise

There was no statistically significant difference between right and left ears *(P*>0.05; ANOVA) so both

ears were grouped and 25 subject responses (50 ears) were registered in terms of mean, median and SDs for peak measures established in conditions without and with noise.

A statistical analysis performed separately for otoacoustic emissions revealed that these values did significantly differ between the two conditions (Table I). The condition without noise exhibited significantly higher mean amplitude than the condition with noise.

Upon evaluation of the ABR latency measures, waves III and V exhibited a statistically significant difference when comparing with- and without-noise conditions, while wave I showed a marginal statistical significance. On the other hand, for amplitude, only wave I exhibited a statistically significant difference (Table I).

Upon analysis of Na and Pa waves of the MLR, there was only a marginal difference in Pa latency at the C3 electrode site position and in the Na–Pa amplitude at the C4 site position (Table II).

For latencies of waves N1, P2 and P300, there was no statistically significant difference, but for N1 and P2 amplitudes there was a statistically significant difference when comparing with and without noise and there was a marginal statistical significance in P300 amplitude (Table III).

In general, the mean latencies of the waves were increased in the noise conditions, whereas the mean amplitudes were diminished with noise conditions for short, middle and late latency responses (Tables I–III). However, not all analyses showed statistically significant differences.

Table IV

Mean, median and standard deviation variation (in percentage) of all variables

Variables	Mean	Median	Standard deviation
Otoacoustic emission	8.48%	12.0%	76.9%
Wave I latency	−1.44%	0.0%	5.8%
Wave III latency	−1.35%	−1.0%	3.0%
Wave V latency	−0.96%	−0.7%	2.5%
Na (C3) latency	−0.05%	0.0%	6.7%
Pa (C3) latency	−1.72%	−1.7%	7.8%
Na (C4) latency	−0.49%	0.0%	8.2%
Pa (C4) latency	−0.83%	0.0%	6.8%
N1 latency	−0.92%	−2.2%	8.2%
P2 latency	−1.36%	−0.3%	8.1%
P300 latency	1.32%	−0.4%	10.7%
Wave I amplitude	52.41%	27.3%	132.0%
Wave III amplitude	27.60%	10.6%	88.0%
Wave V amplitude	4.08%	3.6%	29.0%
C3 amplitude	43.20%	2.3%	134.0%
C4 amplitude	41.71%	32.5%	63.2%
N1 amplitude	27.58%	21.1%	49.1%
P2 amplitude	26.17%	19.0%	55.8%
P300 amplitude	32.46%	17.6%	58.9%

P-value statistically significant for this comparison ($P<0.001$)

Comparison between tests

In general, for otoacoustic emissions, the condition with noise caused a decrease in the amplitude. However, for the AEP, we observed increased latencies and decreased amplitudes. Table IV also shows that the percentage difference for "wave I amplitude" had a high standard deviation. Repeated measures ANOVA for comparisons between all variables showed a statistically significant difference. Thus, the effects of noise on response measures were different at the levels of the auditory pathway compared in this study.

As the data correspond to repeated measures on the inhibition caused by noise performed in five tests, and all 25 subjects were inhibited when submitted to OEA, there is no variability for a statistical analysis and we must assume that the inhibition probability for OEA is 100%.

The remaining data is summarized in Figure 1 and Table V. As can be seen, the estimate of having inhibition on the ABR if the subject has inhibition on the OEA is 96%; the lowest probability is P300 at 80%.

DISCUSSION

The goal of the present study was to determine the effects of contralateral white noise on transient evoked otoacoustic emissions and on short, middle and late

auditory evoked potentials, by comparison of conditions without and with noise.

We tried to evaluate in the same subject the inhibitory effect for different levels of the auditory system from the peripheral level (cochlear) and ascending to more central stations of the system. We wanted to know if the inhibitory effect could be seen in the same subject for all levels of the system considering that most of the studies evaluate just one portion of the system. In the literature we could see many studies in the first attempt to search evidence of descending cortical control at the cochlear level as well as at the auditory brainstem function that is also thought to be modulated by higher level processes *via* top-down processing.

In general our results showed for the noise condition a decrease in the amplitude of otoacoustic emissions, and an increase in latencies with a decrease in amplitudes of the waves of most AEP, compared with the condition without noise.

Comparison between conditions without and with noise

There was a suppressive effect of contralateral noise on TEOAE. It is well established that the phenomenon of contralateral suppression of TEOAE provides an objective and noninvasive clinical tool for exploration of the function of the medial olivocochlear efferent system. This can be done through contralateral auditory stimulation, which may alter active cochlear micromechanics and hence affect evoked otoacoustic emissions and diminish TEOAE amplitudes that are attributed to the efferent system (Collet et al. 1990, Khalfa et al. 2001, Kumar and Vanaja 2004, Muchnik et al. 2004, Perrot et al. 2006, Lilaonitkul and Guinan Jr 2009). Such contralateral effects indicate that the central binaural processor modifies its inputs from each cochlea by affecting cochlear mechanics.

Table V

Estimates and standard errors of the inhibition probabilities

Test	Estimate	Standard error
ABR	96%	4%
MLR	92%	5%
LEP	88%	7%
P300	80%	8%

(ABR) auditory brainstem response; (MLR) middle latency response; (LEP) late evoked potential; (P300) cognitive evoked potential

The use of global response to estimate TEOAE suppression (Hood et al. 1996) allows analysis of the cochlear regions between 4–6 kHz. While it provides some information, the contribution of the high frequency part of the cochlea is missed.

Additionally, the introduction of ipsilateral or contralateral noise during auditory evoked potentials has been shown to negatively affect the amplitudes and latencies of recorded waves (Weihing and Musiek 2008). For ABR, some authors have found an increased latency and diminished amplitude of wave V (Burkard and Hecox 1983, Hecox et al. 1989, Burkard and Sims 2002). Polyakov and coworkers (1998) also found that early ABR peaks, generated peripheral to binaural convergence, may be affected by contralateral stimulation and that these contralateral effects were in a pattern compatible with suppression, most probably by efferents of the olivo-cochlear bundle. The efferent system is the most "likely" reason for the changes in amplitude and latencies seen in our study because it cannot be explained by the binaural interaction com-

ABR	MLR	LEP	P300	Frequency
Y	Y	Y	Y	16
Y	Y	Y	N	4
Y	Y	N	Y	2
Y	Y	N	N	1
Y	N	Y	Y	1
Y	N	Y	N	0
Y	N	N	Y	0
Y	N	N	N	0
N	Y	Y	Y	0
N	Y	Y	N	0
N	Y	N	Y	0
N	Y	N	N	0
N	N	Y	Y	1
N	N	Y	N	0
N	N	N	Y	0
N	N	N	N	0
Y = yes		N = no		Total = 25

Fig. 1. Frequency of inhibited subjects. (ABR) auditory brainstem response; (MLR) middle latency response; (LEP) late evoked potential; (P300) cognitive evoked potential.

ponents (BIC) as these effects are supposed to increase the amplitude and decrease the latency value (Dobie and Berlin 1979, Hosford et al. 1979, Hall and Harvey 1985, Kral and Eggermont 2007). Rosenhamer and Holmkvist (1983) found a significant increase in the latencies of waves III and V for conditions with 90 dB HL contralateral white noise and clicks at 70 dB HL. For white noise at 80 dB HL, there was an increase in wave V latency, while there was no modification of latency or amplitude below this threshold. Similar findings were observed in the present investigation; there were statistically significant increases in latencies of waves III and V with white noise conditions. This reported effect on latencies was more evident than the effect observed on the amplitudes.

One of the reasons that could explain these effects is because the auditory brainstem function is modulated by higher-level processes *via* top-down processing. This cognitive-sensory interaction is made possible by a multitude of afferent fibers carrying sensory information to the midbrain (inferior colliculus) and auditory cortex in concert with the corticofugal pathway, an extensive system of descending efferent fibers that synapse all along the auditory pathway, extending even to the outer hair cells of the basilar membrane (Gao and Suga 2000).

This difference in the BIC study and ours makes us think about the difference in dichotic and diotic stimulation. While BIC uses diotic stimulation, i.e., the same click in both ears, our study used dichotic stimulation, i.e., one ear received click and the other received white noise stimuli.

On the other hand, Özdamar and Bohórquez (2008) did not observe significant modification of ABR waves, but they identified a significant alteration in Pb, which is a middle latency auditory evoked potential component. It was inferred that these observed effects could not have been due to cochlear or brainstem events but were influenced by central mechanisms. However, the underlying physiological mechanisms remain unknown.

Another study also showed effects of noise on MLR waves. Gott and Hughes (1989) found increased Pa latency with increasing ipsilateral broadband noise, which further supports a central mechanism, although in general, amplitude for the MLR Pa component is smaller for true binaural recordings than for the sum of monoaural responses (Dobie and Norton 1980, Özdamar et al. 1986).

Investigating MLR in normal-hearing adults when stimulated by clicks and music in the contralateral ear, Eisencraft and colleagues (2006) found a reduction of Pa amplitude at all electrode sites in the contralateral ear with a music stimulus; however, this reduction was not statistically significant.

Weihing and Musiek (2008) suggest that all of these findings may differ as a result of the magnitude of the noise that was applied. Other parameters, including the stimulated ear and noise type, may also contribute to the "discrepancies" among these results. For MLR, the present study found only marginal statistical significance in Pa latency at the C3 electrode site and in Na–Pa amplitude at the C4 electrode site. However, most responses exhibited a slight increase in wave latency or decrease in amplitude, which agreed with some previous studies.

Related to the late potentials, Salo and coauthors (2003) examined the effect of contralateral masking on cortical auditory evoked potentials N1 and P2 at different masking intensities. These results showed that the N1 amplitude was significantly decreased with contralateral white noise. In contrast, the P2 amplitude was significantly increased with contralateral white noise. In addition, peak latencies were not affected by masking. These effects were suggested to be mediated by the efferent hearing system and were similar to situations occurring during activation of the medial olivocochlear efferent system by contralateral noise that causes a decrease of otoacoustic emissions. Our study also found a decrease in N1 amplitude and also in P2 amplitudes (statistically significant) which was not found in Salo .and others (2003) reports. The latencies of N1 and P2 also were unaffected in our study.

Salisbury and coworkers (2002) examined the effect of background noise on P300 amplitude and latency when stimuli were presented at supra-threshold levels. It was found that performance accuracy was unaffected by background noise and that P300 latency increased when noise was present, yet amplitude remained unaffected. The present investigation differed from that of Salisbury and others (2002) regarding P300 because it found a decrease in P300 amplitude (marginal statistical significance), but latencies were unaffected. Before Salisbury and colleagues (2002), Polich and others (1985) also found an increase of about 10 ms with the presence of a white noise masking the stimulus in P300.

Tomchik and Lu (2006) studied the auditory system of animals and suggested that primary afferent neurons adapt to noise, reducing their evoked firing rates in response to an additional stimulus (e.g., clicks), which may increase the latency of responses. In addition, it was also suggested that broadband noise disrupts the phase-locking of primary afferents to an added stimulus. This may reduce the amplitude of the response to a tone because the tone-evoked potentials measure synchronized activity in the auditory pathway. Furthermore, efferent feedback increases the signal power by activation of efferent neurons that suppress some of the afferent responses to noise. This could be a possible explanation for findings of the present study; efferent system participation may have generated increased latencies and decreased amplitudes for most of the auditory potentials in conditions of white noise; this would have been beyond the range of suppression of otoacoustic emissions.

Comparison between tests

Because tests have different units and magnitudes, for a comparison between all tests, it was necessary to place all variables on the same scale by utilizing data transformation. This comparison revealed high variation between conditions without and with noise. The difference in effectiveness for all tests in the condition with noise could be due to a physiological division of the efferent system into two distinct systems, i.e., caudal (including connections between the superior olivary complex and cochlea) and rostral (including connections between auditory cortex and superior olivary complex). Given that the efferent system was stimulated by noise and that neural generators for all tests may have been located in different regions of this system, differences observed between these tests may be related to differences in efferent activity (Weihing and Musiek 2008).

Complementing previous analyses, the multinomial analysis indicated that most subjects presented with modifications of responses between conditions (without and with noise) in all tests, thereby suggesting that their efferent systems were acting in both caudal and rostral portions of the auditory system.

Considering that all subjects had inhibition verified by OAE, the calculated estimate of the inhibition probabilities was higher in caudal portion of auditory system (AB R – 96%) and lower in rostral portions (P300 – 80%). Thus, the absence of modifications of responses without and with noise was most often noted for components of potentials that were generated in the rostral part of the system and this finding may indicate a participation of attention in these effects, as these structures are more susceptible to the influence of attention.

Such hypotheses are plausible because some investigations have shown that the auditory cortex participates actively in the modulation of the peripheral auditory efferent system. Khalfa and coauthors (2001) studied three patients with intractable temporal lobe epilepsy whose Heschl's gyrus was surgically removed on the side of the lesion. In all three cases, several weeks after the operation, the medial olivocochlear system was clearly less functional on both sides, but especially on the side contralateral to the resection. Perrot and colleagues (2006) found that in 10 epileptic patients, electrical stimulation of the contralateral auditory cortex led to a significant decrease in the evoked otoacoustic emission amplitude, whereas no change occurred under stimulation of non-auditory contralateral areas. Both studies revealed that in humans, the auditory cortex plays a role in the modulation of auditory peripheral activity through direct or indirect efferent fibers and that this descending influence may improve the auditory afferent message by modulating hearing function according to the cortical analysis of the ascending input.

Cone (2009) also suggested that efferent activation by noise and active attention has significant and differing modulatory effects on electroacoustic and electrophysiologic responses along the auditory pathway. In that study, TEOAE showed more suppression in a situation with active attention than in passive listening conditions. Moreover, ABR amplitudes were reduced in all noise conditions; Cortical Auditory Evoked Potentials (CAEPs) also showed the effects of contralateral noise and attention, but were similar to those seen for TOEAEs. These findings could therefore explain the differing effects observed for all tests in the present investigation where attention was not controlled for. These findings also agree with our hypotheses regarding modulation of responses to transient evoked otoacoustic emissions and of short, middle and late auditory evoked potentials in the presence of contralateral white noise.

Researchers have developed and used behavioral measures to assess the complex auditory process of hearing, but only recently have audiologists begun to use them as clinical measures. Although these behav-

ioral tests provide important information regarding an individual's ability to hear in noise, for example, pairing behavioral and physiological tests would most completely and accurately evaluate the auditory system, but to date a paucity of cortical electrophysiological data exists in order to effectively evaluate hearing in noise abilities.

We must also take into consideration the fact that all tests used in the study were performed in the condition with noise after the assessment without noise, which may also have influenced the decreased response due to decreased attention. We suggest in future studies that this order (without and with noise assessment) is randomized.

According to results found in this study and some others mentioned in the literature, in a healthy auditory system an inhibitory effect in the presence of a competitive stimulus is expected. Therefore, future studies should be directed towards verification and quantification this inhibitory effect in normal subjects such that measures may serve as parameters for diagnostic procedures and / or monitoring of auditory function in cases of injury and / or central auditory nervous system dysfunction.

CONCLUSION

These results indicate that most subjects presented different responses between conditions (with and without noise) in all tests, thereby suggesting that the efferent system was acting at both caudal and rostral portions of the auditory system. In future studies, the combined use of both otoacoustic emissions and auditory evoked potentials may be used to elucidate the mechanisms underlying the inhibitory effect of the auditory system.

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