[Frontiers in Bioscience, Elite, 5, 684-696, January 1, 2013]

A potential "tuning" role for the outer hair cells in children with language disorders

Alfredo Durand-Rivera[1], Ethelvina Manzano-Martinez[2], Guillermo Ceballos-Reyes[3]

[1]Laboratorio de Neuroproteccion, Instituto Nacional de Rehabilitacion, INR- SSa. Mexico D.F., [2]Servicio de Terapia de Lenguaje, Instituto Nacional de Rehabilitacion, INR- SSa. Mexico D.F., [3]Seccion de Posgrado e Investigacion de la Escuela Superior de Medicina del Instituto Politecnico Nacional, Mexico D.F.

TABLE OF CONTENTS

1. ABSTRACT

The cochlear outer hair cells serve a tuning function, and any dysfunction of their electromotile response can be reflected in language disorders. Otoacoustic emissions can be used to determine any dysfunction of these cells. A set of clinical records was established to register the neurological and auditory functioning in 42 children, followed by assessment with the Wechsler Intelligence Scale for Children (WISC), the Initial Language Test (ILT), the Auditory and Phonetic Discrimination Evaluation (APDE), tests for measuring Brainstem Auditory Evoked Potential (BAEP) and Transient Otoacoustic Emissions (TOAE). Subjects were classified into 3 groups in this study: Control (C; n = 20), Syntactic Phonological Disorder (SPD; n = 17), and those with Phonological Disability (PD; n = 5). BAEP studies showed a clear response when all children were stimulated to 20 dB. TOAE responses displayed clear and significant differences with half-octave band reproducibility for both ears, the largest effect being observed in the right ear. The results that were compared using ANOVA tests, showed that cochlear processing affects the brain language function, playing a critical role in the language phonetic process.

2. INTRODUCTION

Oral language disorders are highly prevalent among infants. These disorders form a heterogeneous group, ranging from simple phonetic articulation disabilities to severe communication handicaps (1). Alterations in infant speech are associated with defects in various neurophysiological attributes, including memory, attention, executive function, and motor dysfunctions of temporal perception. These deficits are documented at a behavioral level for verbal and nonverbal auditory stimuli, tactile recognition, corporal scheme image, spatial orientation, and visual discrimination, as well as mnesic dysfunction, which is related to the immediate auditory memory and working memory (1, 2). Crespo and Narbona reported that children with a specific language development disorder (dysphasia) had alterations in working memory (phonological and verbal) (1, 3, 4, 5). However, it is difficult to know if this behavioral deficit is due to an alteration in the storage of sensory information or a deficiency at higher levels of cortical processing.

In terms of the dynamics of the hearing process, Ardila (6, 7, 8, 9) postulated the existence of a single sensory pathway controlling auditory perception of

language, suggesting that recognition is independent of production. Auditory processing begins in the ear, where the acoustic signal is first analyzed by the cochlea (Fig 1). In light of our increasing knowledge of cochlear function, a novel approach based on the study by Kemp (10) can be applied to testing otoacoustic emissions (OAEs) by considering their active role in cochlear energy generation. Both spontaneous and evoked OAEs are small sounds caused by the motion of the eardrum in response to external vibrations. These sounds are ultimately processed and amplified by the cochlea and are responsible for frequency selectivity. In addition, the discovery of outer hair cell motility by Brownell in 1983 (11, 12, 13, 14) provided a physical substrate in cochlear processes, because the measurement of the individual strength generated by outer hair cell indicates that activation of a large number of cells could modulate the cochlear basilar membrane's mechanical responses. This property allows the outer hair cells to function as an amplifier (14, 15). Nevertheless, this apparent amplification process is more accurately understood as fine-tuning the auditory response. The inner hair cells are the auditory receptors of the cochlea, and 95% of the auditory nerve fibers project into the encephalon. The outer hair cells (which represent 75% of the total cellular population) are innervated by descending axons that originate from other sites in the encephalon, especially the superior olivary nucleus (12, 16, 17). Activation of this pathway diminishes cochlear sensitivity and frequency discrimination, which are phenomena requiring delicate cochlear tuning (12, 16).

Transient OAEs (TOAEs) are complex acoustic events that occur deep within the human cochlea and are present at early stages in all individuals with normal hearing (11, 12, 13, 14). It is expected that individuals with normal integrity of the middle ear and satisfactory functionality of the cochlear outer hair cells display high reproducibility levels. Previous studies have reported that reproducibility values of 50%–70% are adequate for discriminating between normal hearing and hearing loss (18, 19). On the basis of this understanding of cochlear function, we believe that neuropsychological dysfunction of auditory processing in language disorders could be related to a mild dysfunction of the outer hair cells, which would diminish the delicate cochlear tuning and influence central processing, thereby augmenting language disorders.

The objective of this study was to test our hypothesis that OAEs are altered in language disorders and that certain language disorders are due to cochlear dysfunction, as demonstrated by using OAEs to test cochlear function.

3. SUBJECTS AND METHODS

Inclusion criteria for this study were boys between the ages of 5 years and 7 years 6 months, who were right-handed and had normal intellectual coefficients.

3.1. Subjects

A total of 20 children without language disorders were contacted to form the control group, following the previously indicated inclusion criteria.

We contacted 147 subjects who were diagnosed with a language disorder at the National Institute of Rehabilitation in Mexico City at the Auditory, Phonetic, and Language Pathology Department. Of these subjects, 17 fulfilled the inclusion requirements and were assigned to the pathological group. Furthermore, under the classification of the Specific Disorder of Language Development Subtypes described by Crespo and Narbona, this group was classified as having Syntactic Phonological Disorder (SPD) (3).

We also included a third group of 5 subjects from the 147 children known to have receiving language therapy. Members of this third group showed normal scores on the Initial Language Test (ILT). According to the classification by Crespo and Narbona, this group met the criteria for phonological disability (expressive deficit variant) (3).

After our research project was approved by the Ethics and Research Committee of the National Institute of Rehabilitation in Mexico City and the tutor's or parent's informed consent signature was obtained, the subjects underwent the following tests.

3.2. Clinical history, neurological, audiological, and visual clinical exams

We investigated each patient's general data and cerebral risk antecedents, pathological-neurological dysfunctions (previous or present), psychomotor development, and laterality. None of the children in this study showed signs of cerebral damage or emotional or neurological dysfunction.

3.3. Wechsler intelligence scale for children (WISC)

The Spanish version of this scale was used to assess whether all children in this study had a normal intellectual coefficient (20).

3.4. Initial Language Test (ILT)

This test was designed and standardized for Spanish-speaking children from the ages of 3 years to 7 years 11 months. The ILT incorporates 3 components—semantics, syntax, and morphology—of the 5 language components; phonology and pragmatics are not included in the ILT battery. Average or above-average scores in this test were considered normal, while below-average or deficient scores were regarded as abnormal (21). From these criteria, 3 groups were obtained: control (C), SPD, and Phonological Disorder (PD).

3.5. Auditory and phonetic discrimination evaluation (APDE)

This test was designed for Spanish-speaking subjects and is useful for detecting dysfunctions derived from an auditory discrimination deficit at an early age, starting at 3 years of age. The analysis consisted of 5 subtests: Environmental Sound Discrimination (ESD), Auditory Figure-Ground Discrimination (AFGD), Phonological Word Discrimination (PWD), Logatome Phonological Discrimination (LPD), and Auditory Sequential Memory (ASM). This test offers 2 possible quantitative scores: a direct assessment score or a

Table 1. Intellectual quotient (Wisc) frequency analysis scores

	Controls			Syntactic Phonological Disorder			Phonological Disorder		
	VIQ	EIQ	TIQ	VIQ	EIQ	TIQ	VIQ	EIQ	TIQ
Mean	122	119	123	98	105	102	106	112	110
Standard Deviation	13.57	16.29	13.32	12.90	11.12	9.29	8.63	8.97	7.79
Lowest Peak Value	92	91	96	80	96	91	96	104	101
Highest Peak Value	153	142	153	132	132	120	119	124	121

N = 20 Controls; N = 17 Syntactic Phonological Disorder; N = 5 Phonological Disorder, VIQ = Verbal Intellectual Quotient, EIQ = Executive Intellectual Quotient, AIQ = Total Intellectual Quotient

dominance level score (22); for our study, we used the direct assessment score.

3.6. Brain stem auditory evoked potential (BAEP)

BAEP recording was conducted by stimulating the ears with clicks on rarefaction polarity at 20 dB or less, and also at 70 dB for a duration of 0.1 ms. These clicks were delivered through headphones. The contralateral ear was stimulated by the use of a masking white noise of 0 dB when the intensity was 20 dB and 50 dB when the intensity was 70 dB. Brain electrical activity was measured and recorded using silver-chloride disposable disk electrodes placed in derivations Cz, A1, A2, and Fpz, according to the 10/20 International System: vertex (Cz, reference), Fpz (ground), and mastoid processes (A1 and A2, active).

Incoming signals from electrodes were maintained under 5 kOhms and were redirected to a Viasis Healthcare Niccolet computer, where band-pass filters were programmed to allow the passage of frequencies ranging from 100 to 3000 Hz at a sensitivity of 10 μV and an examination time of 15 ms. Participants were presented with a total of 2,000 stimuli and used the cursor from the computer as part of the system to measure latencies of wave V, which was most prominent at the threshold level. The response was duplicated at least once in order to ensure reproducibility. All studies were performed individually without any medication. All subjects showed a response at 20 dB, a level that under our conditions, was considered normal audition (23, 24, 25, 26).

3.7. Transitory otoacoustic emission (TOAE)

Results were obtained using a Madsen Capella Cochlear Emissions Analizer device within a soundproof chamber (anechoic chamber). Sensors were placed at the external auditory meatus. The stimulus was nonlinear and applied in sets of 4 clicks. The first 3 clicks for each group were administered with the same polarity, and the fourth click was presented with the opposite polarity and at a 3-fold greater amplitude than each of the previous stimuli. The sum of the stimuli within each group and every single auditory response following the specific stimulus was considered as zero. Any difference due to nonlinear conduction in the ear was preserved (13, 14, 15).

Whole-wave reproducibility is the value of the cross-correlation among forms for A and B waves, which are expressed as percentages. This correlation was re-recorded and computed after each set of 20 stimuli (13, 14, 15). To assess the reproducibility of the frequency waves, forms A and B were filtered within a bandwidth of approximately 1000 Hz, focused around the indicated frequency (13, 14).

3.8. Statistical analysis

The statistical analysis of the APDE was performed using a one-way analysis of variance (ANOVA) among the 3 groups. We employed a Student's *t*-test for independent samples from the BAEPs and TOAEs within each group (right ear versus left ear), and the final analysis among all groups was conducted with a one-way ANOVA.

4. RESULTS

The WISC confirmed that all children included in the study had a normal intellectual coefficient, as shown in Table 1. Based on the results of the ILT, the subjects were classified into 3 groups: a control group (C) displaying average or above average results; a group positively diagnosed with SPD, whose results fell below average or were clearly deficient; and a group of 5 children with scores in the normal range as determined by the ILT but who had a previous diagnosis of PD.

In the APDE, we observed no statistically significant differences for the ESD, AFGD, or LPD groups. In contrast, we found significant differences in the PWD and ASM between the C and SPD groups (Table 2). Regarding neurophysiological BAEP recordings, all children responded similarly at 20 dB for both ears; further exploration at 70 dB revealed that I, III, and V waves and their intervals were within normal limits, confirming the integrity of the auditory system. No statistically significant differences were found for either the right or left ear within the 3 groups for all the considered parameters (Table 3). Moreover, there were no statistically significant differences among all the previously measured parameters between the groups C, SPD, and PD for both the right and left ears (Tables 4 and 5). For OAEs, we selectively studied whole-wave reproducibility and half-octave band reproducibility. The analysis between the right and left ears in the whole-wave reproducibility and the half-octave band reproducibility within each group showed no statistically significant differences (Table 6). Additionally, for the analysis of the total reproducibility (both right and left ears) among groups C, SPD, and PD, no statistically significant differences were found (Tables 7 and 8).

In the half-octave band reproducibility, significant differences were noted for the left ear at a frequency of 3 kHz between the C and PD groups (Table 7). Similarly, the right ear showed significant differences between groups C and SPD at 4 kHz, between groups C

Table 2. Auditory and phonetic discrimination evaluation (APDE)

	ANOVA (DIRECT SCORES)						Post hoc ANOVA Analysis (Direct Scores)		
Subtest		**Sum of squares**	**df**	**Mean square**	**F**	**Sig**	**Group**	**Group**	**Sig**
Media Sound Discrimination (MSD)	Between-groups	6.928	2	3.464	0.687	0.509	Control (M =11.75) (б = 2.197)	Syntactic Phonological Disorder (M = 10.88) (б = 2.205)	0.477
	Within-groups	196.715	39	5.044			Control (M =11.75) (б = 2.197)	Phonological Disorder (M = 11.40) (б = 2.608)	0.948
	Total	203.643	41				Syntactic Phonological Disorder (M = 10.88) (б = 2.205)	Phonological Disorder (M = 11.40) (б = 2.608)	0.893
Auditory Figure-Ground Discrimination (AFGD)	Between-groups	1.541	2	0.770	0.725	0.491	Control (M = 4.80) (б = 0.768)	Syntactic Phonological Disorder (M = 4.47) (б =1.231)	0.601
	Within-groups	41.435	39	1.062					
	Total	41	41				Control (M = 4.80) (б = 0.768)	Phonological Disorder (M = 5.00) (б = 1.225)	0.921
							Syntactic Phonological Disorder (M = 4.47) (б =1.231)	Phonological Disorder (M = 5.00) (б = 1.225)	0.575
Phonological Word Discrimination (PWD)	Between-groups	55.484	2	27.742	3.883	0.029	Control (M =37.80) (б = 1.609)	Syntactic Phonological Disorder (M = 35.47) (б = 3.085)	0.031
	Within-groups	278.635	39	7.144					
	Total	33.119	41				Control (M = 7.80) (б = 1.609)	Phonological Disorder (M = 35.60) (б = 4.393)	0.239
							Syntactic Phonological Disorder (M = 35.47) (б = 3.085)	Phonological Disorder (M = 35.60) (б = 4.393)	0.995
Logatome Phonological Discrimination (LPD)	Between-groups	213.769	2	106.885	2.692	0.080	Control (M =17.80) (б = 4.786)	Syntactic Phonological Disorder (M = 13.53) (б = 7.690)	0.113
	Within-groups	1548.635	39	39.709					
	Total	1762.405	41				Control (M =17.80) (б = 4.786)	Phonological Disorder (M = 12.60) (б = 2.891)	0.237
							Syntactic Phonological Disorder (M = 13.53) (б = 7.690)	Phonological Disorder (M = 12.60) (б = 2.891)	0.955
Auditory Sequential Memory (ASM)	Between-groups	61.908	2	30.954	5.313	0.009	Control (M = 8.18) (б = 2.198)	Syntactic Phonological Disorder (M = 5.53) (б = 2.831)	0.007
	Within-groups	227.235	39	5.827					
	Total	289.143	41				Control (M = 8.18) (б = 2.198	Phonological Disorder (M = 6.40) (б = 1.342)	0.346
							Syntactic Phonological Disorder (M = 5.53) (б = 2.831)	Phonological Disorder (M = 6.40) (б = 1.342)	0.760

Control N = 20, Syntactic Phonological Disorder N = 17, Phonological Disorder N = 5 M = mean, б = Standard deviation, sig = significance < 0.05, df = Degrees of freedom

Table 3. Brain stem auditory evoked potential student´s t

	Levene's Test for Equality of Variances		t-test for Equality of Means			Right Ear		Left Ear	
Controls	**F**	**Sig.**	**t**	**df**	**Sig. (2-tailed)**	**Mean**	**SD**	**Mean**	**SD**
Threshold	3.112	0.086	0.634	38	0.530	7.30	0.34	7.24	0.21
I	0.020	0.889	-0.282	38	0.780	1.80	0.18	1.81	0.15
III	1.922	0.174	-0.273	38	0.787	3.97	0.19	3.99	0.27
V	0.118	0.734	-0.409	38	0.685	5.87	0.21	5.89	0.23
I – III	0.397	0.532	-0.450	38	0.655	2.14	0.19	2.17	0.24
III – V	1.572	0.218	0.169	38	0.867	1.91	0.18	1.90	0.25
I – V	0.019	0.892	-0.233	38	0.817	4.06	0.26	4.08	0.26
Syntactic Phonolo-gical Disorder									
Threshold	0.081	0.777	-0.393	32	0.697	7.21	0.44	7.32	0.41
I	0.245	0.624	-1.099	32	0.280	1.79	0.11	1.83	0.14
III	1.996	0.167	-0.886	32	0.382	3.92	0.14	3.98	0.25
V	2.386	0.132	-0.745	32	0.462	5.87	0.15	5.92	0.28
I – III	1.668	0.206	-0.011	32	0.992	2.13	0.13	2.13	0.18
III – V	0.013	0.909	-0.081	32	0.936	1.93	0.14	1.93	0.15
I – V	0.544	0.466	-0.204	32	0.840	4.06	0.17	4.07	0.19
Phonological Disorder									
Threshold	1.406	0.270	-1.016	8	0.340	7.08	0.19	7.22	0.25
I	11.680	0.009	-0.255	4.554	0.810	1.75	0.02	1.77	0.10
III	5.688	0.044	0.224	4.928	0.832	3.99	0.22	3.96	0.07
V	0.059	0.814	-0.307	8	0.767	5.86	0.19	5.90	0.23
I – III	4.240	0.073	0.346	8	0.738	2.23	0.20	2.19	0.10
III – V	0.771	0.405	-0.470	8	0.651	1.88	0.12	1.93	0.19
I – V	0.034	0.859	-0.111	8	0.915	4.11	0.18	4.12	0.16

N = 20 Controls; N = 17 Syntactic Phonological Disorder; N = 5 Phonological Disorder; F = F value; Sig = significance = $p < 0.05$; t = t values; df = degrees of freedom; SD = Standard deviation I, III, V = Observed Waves at 70 dB I - III, III - V, I - V = Intervals obtained at 70 dB Student´s t – Distribution results for each cohort (right versus left ear side), without statistically significant results were encountered among the three groups.

and PD at frequencies of 5 kHz, and between groups SPD and PD at 5 kHz (Table 8).

5. DISCUSSION

Electrophysiological studies of related evoked potentials have been used to evaluate children's auditory perception of language (2). It has also been assumed that if a child does not present with an auditory deficit or dysfunction upon audiometric or BAEP testing, then cochlear processing is adequate. In this work, we have tried to elucidate the potential role of cochlear processing in language function using TOAEs that ultimately measure the functioning of the outer hair cells. These cells function as a delicate cochlear amplifying and tuning element (10, 11, 12, 14, 15, 16, 17). Consequently, our first goal was to prove that all the children in our sample had normal hearing. We found a BAEP response (wave V) at 20 dB for both pathways in each of the 3 groups, indicating that all subjects presented with normal bilateral hearing responses (23, 24, 25, 26). Similarly, no significant differences existed for wave V at the threshold level (20 dB) compared with the contralateral pathway (right versus left ear), or among groups (C, SPD, and PD). This finding indicates that a homogeneous auditory threshold existed in all patients (Table 3).

The latency behavior for waves I, III, and V and interwave intervals between I–III, III–V, and I–V at 70 dB showed no significant differences for any wave or interval within a group (right versus left ear), nor among groups (C, SPD, and PD), indicating that in addition to having normal hearing, the subjects exhibited complete integrity and adequate functionality of the auditory pathway (23, 24, 25, 26). We observed no differences among the subjects, as has been reported for children with specific language disorders (2). We conclude that the children did not have any auditory perception impairment (hypoacousia) and enjoyed full integrity of the auditory pathway (Tables 4 and 5) (2, 16, 23, 24, 25, 26).

TOAEs constitute a series of complex acoustic events that are associated with normal hearing in early life (11, 12, 13, 14). Recent studies suggest that reproducibility values of 50%–70% are adequate to discriminate between normal hearing and hearing loss (18, 19). Thus, one would expect high reproducibility values from people with normal integrity of the inner ear and full functionality of the outer hair cells. It is important to note that the function of outer hair cells cannot be inferred from auditory evoked potentials because the outer hair cells are predominantly innervated by descending axons (12, 16, 17). We also know that because of their neural characteristics, outer hair cells are capable of modifying the cochlear mechanical response, a phenomenon known as cochlear amplification (12, 14, 17), and cochlear tuning.

In our work, a whole-wave reproducibility higher than 70% was observed in all 3 groups without significant differences among the cohorts (right versus left ears), which indicated that the overall cochlear functioning was adequate (Tables 6, 7, and 8) (10, 13, 18, 19, 27). Nevertheless, when we performed the same analysis by half-octave band, we observed that children with SPD had deficits on the right side at 4 kHz as compared to the C group (Table 8). The PD group exhibited the greatest alterations, as deficits in group C were observed in the left ear at 3 kHz (Table 7), and in the right ear at 4 and 5 kHz.

Table 4. ANOVA brain stem auditory evoked potential left ear scores

							Controls		Syntactic Phonological Disorder		Phonological Disorder	
		Sum of squares	**df**	**Mean square**	**F**	**Sig**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Threshold	Between-groups	0.082	2	0.041	0.418	0.661	7.24	0.21	7.32	0.41	7.22	0.25
	Within-groups	3.850	39	0.099								
	Total	3.933	41									
I	Between-groups	0.019	2	0.009	0.435	0.651	1.81	0.15	1.83	0.14	1.77	0.10
	Within-groups	0.836	39	0.021								
	Total	0.855	41									
III	Between-groups	.003	2	0.002	0.024	0.977	3.99	0.27	3.98	0.25	3.96	0.07
	Within-groups	2.541	39	0.065								
	Total	2.544	41									
V	Between-groups	.008	2	0.004	0.060	0.942	5.89	0.23	5.92	0.28	5.90	0.23
	Within-groups	2.576	39	0.066								
	Total	2.584	41									
I – III	Between-groups	0.020	2	0.010	0.226	0.799	2.17	0.24	2.13	0.18	2.19	0.10
	Within-groups	1.767	39	0.045								
	Total	1.787	41									
III – V	Between-groups	.009	2	0.005	0.104	0.901	1.90	0.25	1.93	0.15	1.93	0.19
	Within-groups	1.772	39	0.045								
	Total	1.782	41									
I - V	Between-groups	0.010	2	0.005	0.098	0.907	4.08	0.26	4.07	0.19	4.12	0.16
	Within-groups	2.074	39	0.053								
	Total	2.084	41									
	Total	1.932	41									

N = 20 Controls; N = 17 Syntactic Phonological Disorder; N = 5 Phonological Disorder; df = Degrees of freedom; sig = significance < 0.05, I, III, V = Observed Waves at 70 dB I - III, III - V, I - V = Intervals obtained at 70 dB ANOVA analysis was used for the left and right side ear encompassing Controls; Syntactic Phonological Disorder and Phonological Disorder groups. No statistically significant results were encountered in any case.

The PD group was more severely affected than not only the C group, but also the SPD group (Table 8).

From a clinical point of view, the SPD group showed poor performance on the APDE test, specifically regarding PWD and ASM. This finding is consistent with reports that language disorders in children are commonly associated with a variety of neuropsychological events, including a lack of perception for verbal and nonverbal auditory stimuli, and auditory memory dysfunction, especially in children with some form of phonologic failure (Table 2) (1, 2). Indeed, children with SPD have difficulty comprehending language that is presented as noncontextualized phraseology (3). The children with PD showed no alterations compared to the C group in language competences (Table 2), as tested by the ILT and APDE, both of which explore semantics, syntax, morphology, and phonology. In phonological disorders, the main problem is imprecise articulation. Indeed, isolated phonemes can be produced, but they tend to lose word structure or are omitted in diverse ways when used within a certain word context (3). For this reason, the PD group was considered to fair normally in the ILT, and no statistically significant differences were observed with group C on the APDE.

However, both groups (SPD and PD) share failures in comprehension and integration of words and in phonological performance. Therefore, these children probably need alterations for detection of certain critical features in the acoustic signal (phoneme recognition (H), Figure 1), because the first frequency analysis performed in the cochlea (Figure 1 (B)) (6, 7, 8, 9) was not adequate as the outer hair cells were not modulating the mechanical response of the basilar membrane (14, 15). As described by Ardila (6, 7, 8, 9), there are neurons in the primary auditory cortex that are highly tuned to specific frequencies

Table 5. ANOVA brain stem auditory evoked potential right ear scores

							Controls		Syntactic Phonological Disorder		Phonological Disorder	
		Sum of squares	**df**	**Mean square**	**F**	**Sig**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Threshold	Between-groups	0.198	2	0.099	0.699	0.503	7.30	0.34	7.27	0.44	7.08	0.19
	Within-groups	5.532	39	0.142								
	Total	5.731	41									
I	Between-groups	0.008	2	0.004	0.484	0.620	1.80	0.18	1.79	0.11	1.75	0.02
	Within-groups	0.881	39	0.032								
	Total	0.889	41									
III	Between-groups	0.031	2	0.015	0.484	0.620	3.97	0.19	3.92	0.14	3.99	0.22
	Within-groups	1.237	39	0.032								
	Total	1.268	41									
V	Between-groups	0.000	2	0.000	0.004	0.996	5.87	0.21	5.87	0.15	5.86	0.19
	Within-groups	1.442	39	0.037								
	Total	1.442	41									
I - III	Between-groups	0.039	2	0.019	0.656	0.525	2.14	0.19	2.13	0.13	2.23	0.20
	Within-groups	1.156	39	0.030								
	Total	1.195	41									
III - V	Between-groups	0.010	2	0.005	0.178	0.838	1.91	0.18	1.93	0.14	1.88	0.12
	Within-groups	1.055	39	0.027								
	Total	1.064	41									
I - V	Between-groups	0.013	2	0.006	0.129	0.880	4.06	0.26	4.06	0.17	4.11	0.18
	Within-groups	1.919	39	0.049								
	Total	1.932	41									

N = 20 Controls; N = 17 Syntactic Phonological Disorder; N = 5 Phonological Disorder; df = Degrees of freedom; sig = significance < 0.05, I, III, V = Observed Waves at 70 dB I - III, III - V, I - V = Intervals obtained at 70 dB ANOVA analysis, was used for the right side ear encompassing Controls; Syntactic Phonological Disorder and Phonological Disorder groups. No statistically significant results were encountered in any case.

Table 6. Transitory otoacoustic emissions student´s t

	Levene's Test for Equality of Variances		t-test for Equality of Means			Right Ear		Left Ear	
Controls	**F**	**Sig.**	**t**	**df**	**Sig. (2-tailed)**	**Mean**	**SD**	**Mean**	**SD**
Whole-wave-reproducibility	5.381	.026	1.532	38	.134	94.5	5.2	90.05	11.98
1 Khz	6.110	.018	1.302	25.271	.205	95.25	6.08	90.60	14.76
2 Khz	.738	.396	.734	38	.467	94.20	8.78	91.80	11.67
3 Khz	1.972	.168	1.148	38	.258	90.60	10.25	85.95	14.92
4 Khz	7.076	.011	1.384	28.592	.177	81.40	13.94	72.05	26.79
5 Khz	1.042	.314	.685	38	0.5	49.00	28.89	42.05	34.99
Syntactic Phonological Disorder									
Whole-wave-reproducibility	.084	.774	.124	32	.902	88.00	18.54	87.29	14.37
1 Khz	.016	.899	.276	32	.784	91.29	16.59	88.70	16.91
2 Khz	.027	.871	-.033	32	.974	87.64	20.91	87.88	20.10
3 Khz	.154	.697	.309	32	.760	83.11	22.96	80.88	19.10
4 Khz	.800	.378	.109	32	.914	62.76	28.59	61.82	21.26
5 Khz	1.077	.307	.099	32	.922	43.47	32.03	42.35	33.88
Phonological Disorder									
Whole-wave-reproducibility	1.673	.232	1.014	8	.340	88.00	14.81	73.40	28.57
1 Khz	.743	.414	.361	8	.728	87.40	14.32	82.60	26.08
2 Khz	4.742	.061	1.281	8	.236	93.80	5.16	68.60	43.67
3 Khz	3.376	.103	.964	8	.363	75.20	20.21	56.88	37.58
4 Khz	.022	.887	.359	8	.729	56.40	22.27	50.80	26.81
5 Khz	.744	.414	-2.162	8	.063	3.80	14.28	28.20	20.80

N = 20 Controls; N = 17 Syntactic Phonological Disorder; N = 5 Phonological Disorder; F = F value; Sig = significance = $p < 0.05$; t = t values; df = degrees of freedom; SD = Standard deviation 1 kHz, 2 kHz, 3 kHz, 4 kHz, 5 kHz = Describes the half – octave - band reproducibility analyzed. Student´s t analysis of whole – wave - reproducibility and half – octave - band reproducibility for each group (right versus left ear side), without statistically significant results were encountered in any of the three groups.

Table 7. Transitory otoacoustic emissions (left ear scores)

ANOVA (DIRECT SCORES)							**Post hoc ANOVA Analysis (Direct Scores)**		
Left Ear Scores		**Sum of squares**	**df**	**Mean square**	**F**	**Sig**	**Group**	**Group**	**Sig**
Whole-wave-reproducibility	Between Groups	1112.225	2	556.113	2.332	0.11	Control (M =90.05) (б = 11.98)	Syntactic Phonological Disorder (M = 87.29) (б = 14.37)	0.852
	Within Groups	9301.679	39	238.505					
	Total	10413.905	41				Control (M =90.05) (б = 11.98)	Phonological Disorder (M = 73.40) (б = 28.57)	0.092
							Syntactic Phonological Disorder (M = 87.29) (б = 14.37)	Phonological Disorder (M = 73.40) (б = 28.57)	0.194
1 Khz	Between Groups	261.042	2	130.521	0.45	0.644	Control (M =90.60) (б = 14.76)	Syntactic Phonological Disorder (M = 89.70) (б = 16.91)	0.986
	Within Groups	11441.529	39	293.373					
	Total	11702.51	41				Control (M =90.60) (б = 14.76)	Phonological Disorder (M = 82.60) (б = 26.08)	0.622
							Syntactic Phonological Disorder (M = 89.70) (б = 16.91)	Phonological Disorder (M = 82.60) (б = 26.08)	0.696
2 Khz	Between Groups		2	1079.120	2.522	0.093	Control (M =91.80) (б = 11.67)	Syntactic Phonological Disorder (M = 87.88) (б = 20.10)	0.835
	Within Groups		39	427.953					
	Total		41				Control (M =91.80) (б = 11.67)	Phonological Disorder (M =68.60) (б = 43.67)	0.076
							Syntactic Phonological Disorder (M = 87.88) (б = 20.10)	Phonological Disorder (M =68.60) (б = 43.67)	0.173
3 Khz	Between Groups	3404.771	2	1702.386	4.224	0.022	Control (M =85.95) (б = 14.92)	Syntactic Phonological Disorder (M = 80.88) (б = 1910)	0.726
	Within Groups	15719.515	39	403.064					
	Total	19124.286	41				Control (M =85.95) (б = 14.92)	Phonological Disorder (M = 56.88) (б = 37.68)	0.016
							Syntactic Phonological Disorder (M = 80.88) (б = 1910)	Phonological Disorder (M = 56.88) (б = 37.68)	0.060
4 Khz	Between Groups	2167.684	2	1083.842	1.779	0.182	Control (M = 72.05) (б = 26.79)	Syntactic Phonological Disorder (M = 61.82) (б = 21.26)	0.428
	Within Groups	23754.221	39	609.083					
	Total	25921.905	41				Control (M = 72.05) (б = 26.79)	Phonological Disorder (M = 50.80) (б = 26.81)	0.210
							Syntactic Phonological Disorder (M = 61.82) (б = 21.26)	Phonological Disorder (M = 50.80) (б = 26.81)	0.657
5 Khz	Between-groups	862.844	2	431.422	0.388	0.681	Control	Syntactic Phonological	1.000

	Whitin-groups	43367.632	39	1111.991			(M =52.05) (б = 34.99)	Disorder (M = 42.35) (б = 33.88)	
	Total	44230.476	41				Control (M =11.75) (б = 2.197)	Phonological Disorder (M = 28.20) (б = 20.80)	0.686
							Syntactic Phonological Disorder (M = 42.35) (б = 33.88)	Phonological Disorder (M = 28.20) (б = 20.80)	0.684

Control N = 20, Syntactic Phonological Disorder N = 17, Phonological Disorder N = 5 sig = significance < 0.05, df = Degrees of freedom

frequency intensity recognition (C), Figure 1) and respond to intensity changes within particular frequency bands, but in children with SPD and PD, the signal reaching these neurons is not adequate. The primary cortex contains a tonotopic map, in which the relative position of a frequency projection is proportional to the logarithm of the frequency (8). The existence of certain properties in the acoustic signal, such as a frequency change from fo to fk in a given time t (transition), is functional in a specific phonological system (long-term memory for features (D), Figure 1). In our cases, the perception of the frequency change may not be correct because the outer hair cells were not modulating the mechanical response of the basilar membrane (14, 15), which allows feature recognition ((E), Figure 1). Recognition of a phoneme requires the listener to match the features of the signal with phonemic categories (Figure 1). This process represents the first categorical judgment of sound units (phonemes) in language perception. A deficit in this first analysis may lead to the imprecise articulation seen in children with PD (3). Presumably, this type of analysis is accomplished in the first temporal gyrus, around the primary auditory cortex. Phoneme chains are subsequently integrated into more complex units (morpholexical units (K), Figure 1). Morpholexical units are organized into verbal-acoustic memory (memory for words (J)) formed with the repeated presence of identical phonemic sequences ((I), Figure 1). A deficit in the second level of processing could be accounted for in children with SPD, and for this reason, children with SPD have problems in auditory sequential memory and phonological word discrimination. This second categorical judgment may involve the first and second temporal gyri (6, 7, 8, 9).

Conversely, the superior olivary complex plays a fundamental role in binaural auditory development, and this complex is the primary information-receiving center for the cochlear nuclei (16, 28, 29). The superior olivary complex is also the origin of the efferent cochlear-olivary system, which terminates at the outer hair cells (16). This efferent pathway increases the sensitivity and the selectivity to frequencies by providing amplification and tuning. Why do children with SPD and PD have phonological disorders? In children with SPD, phonology is altered in the expression area, with phonological errors (omissions, distortions, and substitutions) affecting the integrity of language (3); therefore, the ILT and APDE (which assess phonological word discrimination and auditory sequential memory) are altered (Table 2). In the TOAEs, the pathological groups exhibit fewer failures (4 kHz, Tables 7 and 8). In the case of the PD group, the subjects' phonology was altered in the receptive area, as they could produce isolated phonemes and syllables (3). These findings are reflected in the TOAE scores: the PD group has the most alterations (3 kHz, 4 kHz, and 5 kHz; Table 7 and 8).

Therefore, if the TOAE tests measure outer hair cell activity (10, 11, 13, 14), we can assume that in the children with phonological deficits, a cochlear dysfunction exists for one or more specific frequencies. In our study, these frequencies were at 3, 4, and 5 kHz (Table 7 and 8), which explains why we did not obtain good selectivity and tuning of the frequencies. As such, under Ardila's model, there will be no accurate recognition of frequencies and therefore a failure in recognizing features and inadequate recognition of phonemes, thereby representing faults in the first and second levels of the categorical perception of language.

Our subjects displayed dysfunction more commonly in the right ear, supporting the findings of other studies that in children with normal hearing, TOAE scores are higher for the right ear (29, 30). This finding is in agreement with another report that reported that for dichotic listening tests (directed attention mode), the right ear is more precise for sound-language recognition, thereby supporting the theory that the left hemisphere, contralateral to the right ear, specializes in recognition and response to language sounds (29, 31, 32, 33, 34, 35, 36, 37).

Additionally, neurophysiological studies show that hemispheric asymmetries in children and young adults are exposed to nonsense syllables in the right ear. Older people lose such asymmetry, along with the ability to discriminate speech sounds (29, 38). We hypothesized that a similar process might occur in the children in our study because subjects with a SPD presented with deficits in the APDE, ILT, and the TOAEs. Remarkably, the deficit in TOAEs was less severe than what we observed with PD. In fact, despite our extensive testing during the APDE and the ILT, we detected no dysfunction. However, TOAEs were more heavily altered, due to the fact that this group (PD) is more affected than the control group in both ears and because the deficit in the right ear is greater than that in the SPD group. From this finding, we can conclude that hemispheric processing is inadequate, because of a dysfunction at the cochlear level, specifically in the outer hair cells. This dysfunction may be, in part, the cause of the phonologic deficit present in the children in our study.

Table 8. Transitory otoacoustic emissions (right ear scores)

ANOVA (DIRECT SCORES)							**Post hoc ANOVA Analysis (Direct Scores)**		
Right Ear Scores		**Sum of squares**	**df**	**Mean square**	**F**	**Sig**	**Group**	**Group**	**Sig**
Whole-wave-reproducibility	Between Groups	442.619	2	221.310	1.259	0.295	Control (M =94.50) (б = 5.02)	Syntactic Phonological Disorder (M = 88.00) (б = 18.54)	0.309
	Within Groups	6857.000	39	175.821					
	Total	7299.619	41				Control (M =94.50) (б = 5.02)	Phonological Disorder (M = 88.00) (б = 14.81)	0.593
							Syntactic Phonological Disorder (M = 88.00) (б = 18.54)	Phonological Disorder (M = 88.00) (б = 14.81)	1.000
1 Khz	Between Groups	304.092	2	152.046	1.000	0.380	Control (M =95.25) (б = 6.00)	Syntactic Phonological Disorder (M = 91.29) (б = 16.59)	0.598
	Within Groups	5930.479	39	152.064					
	Total	6234.571	41				Control (M =95.25) (б = 6.00)	Phonological Disorder (M = 87.40) (б = 14.32)	0.419
							Syntactic Phonological Disorder (M = 91.29) (б = 16.59)	Phonological Disorder (M = 87.40) (б = 14.32)	0.810
2 Khz	Between Groups	424.618	2	212.309	0.966	0.390	Control (M =94.20) (б = 8.78)	Syntactic Phonological Disorder (M = 87.64) (б = 20.90)	0.382
	Within Groups	8575.882	39	219.894					
	Total	9000.500	41				Control (M =94.20) (б = 8.78)	Phonological Disorder (M = 93.80) (б = 5.16)	0.998
							Syntactic Phonological Disorder (M = 87.64) (б = 20.90)	Phonological Disorder (M = 93.80) (б = 5.16)	0.696
3 Khz	Between Groups	1144.754	2	572.377	1.849	0.171	Control (M =90.60) (б = 10.25)	Syntactic Phonological Disorder (M = 83.11) (б = 22.96)	0.410
	Within Groups	12075.365	39	309.625					
	Total	13220.119	41				Control (M =90.60) (б = 10.25)	Phonological Disorder (M = 75.20) (б = 20.21)	0.200
							Syntactic Phonological Disorder (M = 83.11) (б = 22.96)	Phonological Disorder (M = 75.20) (б = 20.21)	0.653
4 Khz	Between Groups	4381.346	2	2190.673	4.552	0.017	Control (M =81.40) (б = 13.94)	Syntactic Phonological Disorder (M = 62.76) (б = 28.59)	0.036
	Within Groups	18769.059	39	481.258					
	Total	23150.405	41				Control (M =81.40) (б = 13.94	Phonological Disorder (M = 56.40) (б = 22.27)	0.071
							Syntactic Phonological Disorder (M = 62.76) (б = 28.59)	Phonological Disorder (M = 56.40) (б = 22.27)	0.837
5 Khz	Between Groups	8296.869	2	4148.435	4.887	0.013	Control (M =49..00) (б = 28.89)	Syntactic Phonological Disorder (M = 43.47) (б = 32.09)	0.834
	Within Groups	33109.035	39	848.950					
	Total	41405.905	41						

							Control (M =49..00) (б = 28.89)	Phonological Disorder (M = 3.80) (б = 14.28)	0.010
							Syntactic Phonological Disorder (M = 43.47) (б = 32.09)	Phonological Disorder (M = 3.80) (б = 14.28)	0.029

Control N = 20, Syntactic Phonological Disorder N = 17, Phonological Disorder N = 5 sig = significance < 0.05, df = Degrees of freedom

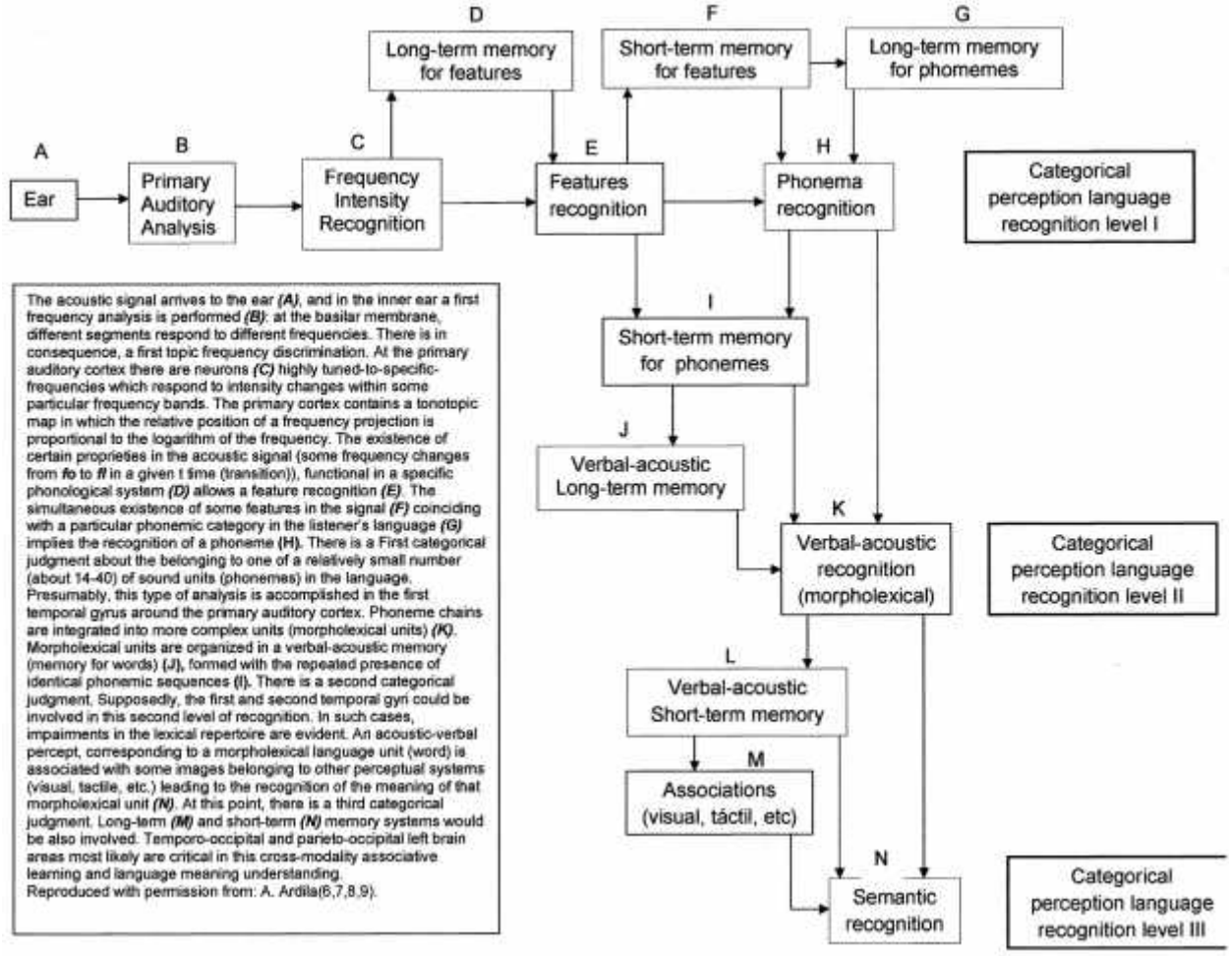

Figure 1. The acoustic signal arrives to the ear (A), and in the inner ear a first frequency analysis is performed (B): at the basilar membrane, different segments respond to different frequencies. There is in consequence, a first topic frequency discrimination. At the primary auditory cortex there are neurons (C) highly tuned-to-specific-frequencies which respond to intensity changes within some particular frequency bands. The primary cortex contains a tonotopic map in which the relative position of a frequency projection is proportional to the logarithm of the frequency. The existence of certain proprieties in the acoustic signal (some frequency changes from fo to fl in a given t time (transition)), functional in a specific phonological system (D) allows a feature recognition (E). The simultaneous existence of some features in the signal (F) coinciding with a particular phonemic category in the listener's language (G) implies the recognition of a phoneme (H). There is a First categorical judgment about the belonging to one of a relatively small number (about 14-40) of sound units (phonemes) in the language. Presumably, this type of analysis is accomplished in the first temporal gyrus around the primary auditory cortex. Phoneme chains are integrated into more complex units (morpholexical units) (K). Morpholexical units are organized in a verbal-acoustic memory (memory for words) (J), formed with the repeated presence of identical phonemic sequences (I). There is a second categorical judgment. Supposedly, the first and second temporal gyri could be involved in this second level of recognition. In such cases, impairments in the lexical repertoire are evident. An acoustic-verbal percept, corresponding to a morpholexical language unit (word) is associated with some images belonging to other perceptual systems (visual, tactile, etc.) leading to the recognition of the meaning of that morpholexical unit (N). At this point, there is a third categorical judgment. Long-term (M) and short-term (N) memory systems would be also involved. Temporo-occipital and parieto-occipital left brain areas most likely are critical in this cross-modality associative learning and language meaning understanding. Reproduced with permission from: A. Ardila(6,7,8,9).

On the basis of our results, we propose that peripheral auditory processing affect the brain language functions, playing a critical role during phonological language processing and in peripheral processing laterality control language acquisition (29, 31, 32, 33, 34, 35, 36, 37, 38). Should this delicate neural mechanism suffer any signal transduction, even a subtle alteration in the cochlea affecting the functionality of the outer hair cells could affect language and speech processes. This knowledge might help clinicians to develop better and more accurate diagnostic strategies, which

may be implemented to shorten rehabilitation and treatment schemes. Finally, considering that language disorders are a public health issue in most countries, further investigation of peripheral processing of language is necessary, and TOAEs may constitute a significant tool in this endeavor.

6. REFERENCES

1. P.A. Conde-Guzón, M.J. Conde-Guzón, M.T. Bartolomé-Albistegui, P. Quirós-Expósito: Perfiles neuropsicológicos asociados a los problemas de lenguaje oral infantil. *Rev Neurol* 48 (1), 32-38 (2009)

2. M.A. Idiazábal-Aletxa, M. Saperas-Rodríguez: Procesamiento auditivo en el trastorno específico del lenguaje. *Rev Neurol* 46 (Supl 1), S91-S95 (2008)

3. N. Crespo-Eguílaz, J. Narbona: Subtipos de trastorno específico del desarrollo del lenguaje: perfiles clínicos en una muestra hispanohablante. *Rev Neurol* 43 (supl 1), S193 – S200 (2006)

4. J. Muñoz, G. Carballo: Alteraciones lingüísticas en el trastorno específico del lenguaje. *Rev de Neurol* 40 (Supl 1), S57-63 (2005)

5. Serra M. Dificultades cognitivas y lingüísticas en los niños con trastorno específico del lenguaje. Rev. Logop. Foniatr. Audiol. 17, 79-93 (1997)

6. A. Ardila, Psicología del lenguaje: Eds. Trillas. México D.F. México (1983)

7. A. Ardila Neurolingüística. Eds. Trillas. México D.F. México (1984)

8. A. Ardila: Toward a model of phoneme perception. Intern. J. Nuerocinece. Vol.70 1-12 (1992)

9. A. Ardila: Orígenes del Lenguaje: un análisis desde la perspectiva de las afasias. Rev. Neurol. 43 (11): 690-98 (2006)

10. DT. Kemp: Otoacoustic emissions, their origin in cochlear function, and use. *Br Med Bull* 63, 223 – 41 (2002)

11. P. Délano, I. Robles, L. Robles: Sistema eferente auditivo. *Rev. otorrinolaringol. cir. cabeza cuello* 65, 55-62 (2005)

12. AJ. Hudspeth: Transformación sensitiva en el oído. In Principios de Neurociencia 4ª ed. Eds. Kandel ER., Schwartz JH, Jessell TM. Mc Graw Hill. México D.F. 614 – 22 (2001)

13. Oostenbrink-Meter and Verhaagen-Warnaar: Otoacoustic Esmissions. *Am. J. END Technol* 44, 189 – 198 (2004)

14. MS Robinette, TJ Glattke: Otoacustic emissions clinical applications. Thieme. New York (1997).

15. KH Iwasa, RS Chadwick: Elasticity and force generation of cochlear outer hair cells. *J. Acoust. Soc. Am*, 92 (6), 3169 – 3173 (1992)

16. MS Malmierca: The structure and physiology of the rat auditory system: an overview. In International Review of Neurobiology Eds. Ronald J. Bradley, R. Adron Harris & Peter Jenner. San Diego, California 56, 147-211 (2003)

17. Purves-Dale, G J Augustina, D Fitzpatrick, LC Katz, AS La Mania, JO McNamara: Sistema Auditivo In Invitación a la Neurociencia. Panamericana México D.F. 243-265 (1997)

18. WA Harrison, SJ Norton: Characteristics of transient evoked otoacoustic emissions in normal hearing and hearing impaired children. *Ear & Hearing* 20(1), 75-86 (1999)

19. G Zimatore, A Giuliani, C Parlapiano, G Grisanti, A Colosimo: Revealing deterministic structures in click-evoked otoacoustic emissions. *J Appl Physiol* 88, 1431-37 (2000)

20. D Wechsler: WISC – R – Español: Escala de Inteligencia Revisada para el Nivel Escolar. El Manual Moderno México D.F. (1981)

21. M Ramos, J Ramos: Prueba de Lenguaje Inicial Manual del Examinador. Pro-Ed. N.L. México (1996)

22. Mario F. Brancal, Francisco Alcantud, Antonio M. Ferrer, Marta E. Quiroga: Evalucación de la Discriminación Auditiva y Fonológica (E.D.A.F.) Manual del Test. Ed. Lebon. Barcelona, España (2005)

23. KH. Chappa: Evoked Potentials in Clinical Medicine. (2ª. ed.) Eds. Raven Press New York 123-304 (1983)

24. A. Durand-Rivera: Bases técnicas y fisiológicas de los potenciales evocados auditivos de tallo cerebral. *Para la Salud* 2(19), 20 – 30 (1998)

25. Y. Elias Cuadros: Potenciales provocados auditivos de tallo cerebral en el diagnóstico del daño auditivo y neurológico del adulto. In Registros electrofisiológicos para el diagnóstico de la patología de la comunicación humana. Eds. Flores-Rodriguez T. & Peñaloza-López Y. Instituto Nacional de la Comunicación Humana, México D.F. 157 – 72 (1996)

26. M. Rosete-Díaz: Potenciales provocados auditivos, potenciales provocados de tallo cerebral. Fundamentos y aplicaciones en la hipoacusia infantil. In Registros electrofisiológicos para el diagnóstico de la patología de la comunicación humana. Eds. Flores-Rodriguez T. & Peñaloza-López Y. Instituto Nacional de la Comunicación Humana, México D.F. 143 – 53 (1996)

27. RJ Nozza, DL Sabo, EM Mandel: A role for otoacoustic emissions in screening for hearing impairment and middle ear disorders in school age children. *Ear Hearing* 18(3), 227-39 (1997)

28. JF Brugge: An overview of central auditory processing. In *The mammalian auditory pathway: neurophysiology* Eds. Propper A, Fay R Springer Verlag New York, 1 – 33 (1992)

29. B First-Jill, JI Umler, W Gaggl: Diferential representation of speech sound in the human cerebral hemispheres. *The Anatomical Record* Part A 288A, 345 – 57 (2006)

30. YS Sininger, B Cone-Wesson: Asymmetric cochlear processing mimics' hemispheric specialization. *Science* 305, 1581 (2004)

31. M Benavides, YR Peñaloza-López, S Jiménez-de la Sancha, F García-Pedroza, PK Gudiño: Lateralidad auditiva y corporal, logoaudiometría y ganancia del audífono monoaural. Aplicación en hipoacusia bilateral simétrica. *Acta Otorrinolaringol Esp* 58 (10) 458-63 (2007)

32. A Brancucci, A D'Anselmo, F Martello, L Tommasi: Left hemisphere specialization for duration discrimination of musical and speech sound. *Neuropsychology* 46 (7) 2013 – 19 (2008)

33. K Hugdahl, R Westerhausen, K Alho, S Meduedev, H Hämäläinen: The effect of stimulus intensity on the right ear advantage in dichotic listening. *Neurosci Lett* 24: 41 (1), 90 – 4 (2008)

34. D Kimura: Functional asymmetry of the brain in dichotic listening. *Cortex* 3, 163 – 78 (1967)

35. MR Olivarez-García, YR Peñaloza-López, F García-Pedroza, S Jesús-Pérez, R Uribe-Escamilla, S Jiménez-de la Sancha: Identificación de la lateralidad corporal y orientación espacial en niños con dislexia y en controles. *Rev Neurol* 41(4), 198-205. (2005)

36. B Saetrevik, K Hugdahl: Priming inhibits the right ear advantage in dichotic listening: Implications for auditory laterality. *Neuropsychology* 28, 45 (2), 282 – 87 (2007)

37. M Spajdel, K Jariabková, K Riecanský: The influence of musical experience on lateralization of auditory processing. *Laterality* 12 (6), 487 – 99 (2007)

38. TJ Bellis, T Nicol, N Kraus: Aging affects hemispheric symmetry in the neural representation of speech sounds. *J Neurosci* 20, 191 – 97 (2000)

Abbreviations: OAEs: Otoaocustic emissions; WISC: Wechsler Intelligence Scale for Children; ILT: Initial Language Test; APDE: Auditory and Phonetic Discrimination Evaluation; ESD: Environmental Sound Discrimination; AFGD**:** Auditory Figure-Ground Discrimination; PWD: Phonological Word Discrimination; LPD: Logatome Phonological Discrimination; ASM: Auditory Sequential Memory; BAEP: Brainstem Auditory Evoked Potential; TOAE: Transient Otoacoustic Emission; C: Control; SPD: Syntactic Phonological Disorder; PD: Phonological Disability; OAEs: Otoaocustic Emissions

Key Words: Language; Language disorders; Syntactic phonological disorder; Phonological disability; Outer hair cells; Otoacoustic emissions; Whole-Wave-Reproducibility; Half-Octave-Band reproducibility

Send correspondence to: Alfredo Durand-Rivera, Instituto Nacional de Rehabilitacion, Laboratorio de Neuroproteccion, Clz. Mexico Xochimilco # 289 Col. Arenal de Guadalupe Tlalpan 14370 Mexico D.F., Tel: 38711648, Fax: 30953020 E-mail: alfredo.durand@gmail.com