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The locus of deficits in dysphonemic dyslexia: an ERP analysis

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Brain stem, middle auditory and auditory event-related potentials (ERPs) were recorded from a group of 13 dysphonemic dyslexic children and 10 matched normal controls. Auditory stimuli were used to elicit ERPs. The focus of our study was an ERP investigation of a slower auditory categorization, rather than simple detection, of pure tones. Brain stem and middle auditory evoked responses did not show any retardation in dyslexics compared with non-dyslexic children. When subjects performed a tone categorization task in which they were required to detect a rare tone interspersed in a series of frequent tones, dysphonemic dyslexics showed longer N2 latency. Compared with non-dyslexics, N2 delays were maximal over the left temporo-parietal region and bilaterally over the occipital sites. These areas have been postulated to be crucial in oral language and reading comprehension.

Déficit en dislexia disfónica: un estudio con potenciales evocados. En el presente estudio se registraron potenciales evocados de tronco, medios y auditivos en una muestra de 13 niños disléxicos y 10 niños normales. El objetivo del mismo fue llevar a cabo un estudio respecto a la mayor lentitud de categorización auditiva, más que la simple detección, de tonos puros, en sujetos disléxicos. Las respuestas de potenciales de tronco y potenciales medios no mostraron diferencias entre las dos muestras de niños. Cuando los sujetos realizaron una tarea de categorización de tonos en la que se pedía que discriminaran un determinado tono interpuesto en una serie de tonos frecuentes, el grupo disléxicos mostró una latencia N200 más larga. Comparados con la muestra de niños normales, el retardo del componente N200 fue máxima en la región temporo-parietal izquierda y bilateral en la región occipital. Estas áreas han sido reconocidas como cruciales en el lenguaje y la comprensión lectora.

Developmental dyslexia is normally characterized by unexpected problems in learning to read for children of average or above average intelligence. It has been suggested that dyslexia appears as a consequence of a language based disorder and that deficits in visual processing are seldom the cause (Darlington, Barceló, Fernández y Rubia, 1999; Mann & Brady, 1988; Vellutino, 1987). Reading problems may be caused by a failure in the development of phonological awareness (Bradley & Bryant, 1983; Goswami, 1993; Pratt & Brady, 1988; Pennington, Van Orden, Smith, Green & Haith, 1990), hampering, mainly, the proper establishment of spelling-to-sound correspondences, and consistently, phonological decoding skill, namely the ability for appropriate analysis and retrieving the sound structure of orthographic units (Backman, Bruck, Hebert & Seidenberg, 1984; Bouma & Legein, 1980; Katz, 1986; Lovett, 1987; Snowling, 1981; Taylor, Lean & Schwartz, 1989;Vigil-Colet, Pérez-Ollé y García-Albea,1998; Waterman & Lewandowski, 1993). In addition, dyslexic readers are worse than normal readers in tasks engaging continuous phoneme perception (Brady, Shankweiler & Mann, 1983; Liberman, Meskill, Chatillon & Shupack, 1985; Tallal, 1980), and verbal working memory (Mann & Liberman, 1984; Mann, Cowin & Schoenheimer, 1989; Rugel, 1974; Vellutino, Pruzek, Steger & Mesulam, 1973). This suggests that dyslexic children could have a more generalized auditory-phonetic perceptual deficit leading to the development of degraded representations of verbal information in memory and problems in segmenting words in phonemes, hampering posteriorly the establishment of spelling-to-sound correspondences and decoding skills.

Behavioral evidence has shown that dyslexic children are slower than normal children only when an auditory selection or classification is required by the auditory task (Nicolson & Fawcett, 1993, 1994).

This hypothesis can be assessed using Event-Related Potentials (ERPs), a noninvasive technique for measuring neural activity associated with sensory and cognitive information processing. Within specific experimental paradigms, some segments of the ERPs seem to reflect endogenous cognitive activity, such as selective attention and discrimination decisions. An auditory selective-attention task (an auditory oddball paradigm) can be used to see ERP modulations during stimulus selection processes. In this task, a series of tones are presented, with differing tone frequencies. Subjects are instructed to listen to the tones and press a button key whenever the target tone stimulus is presented, while ignoring the rest of the tones (non-target stimulus). This paradigm typically results in a negative peak to all tones (N2 component), and a broad late positive peak to the target tones (P3 component).

When dyslexic and normal children are compared in this type of task, research has emphasized the P300 component (Taylor & Keenan, 1990; Holcomb, Ackerman & Dykman, 1986; Ollo &

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Squires, 1986). Usually, the P3 amplitude is lower in dyslexic than in normal children (Paz y Muñiz, 1989; Lovrich & Stamm, 1983; Holcomb, Ackerman & Dykman, 1986). Less frequently noted is a delayed P300 latency in dyslexics (Fawcett, Chattopadhyay, Kandler, Jarratt, Nicolson & Proctor, 1993). Fawcett et al. (1993) reported significant longer N200 latencies to the rare tones for dyslexic readers in comparison with normal readers. On the other hand, Ollo & Squires (1986) did not find N200 or P300 amplitude or latency differences between normal and reading-disabled children during a similar auditory selective discrimination task.

Inconsistent findings may arise because of the characteristics of the samples studied. In fact, an important limitation in the ERP literature is a tendency to consider children with reading disabilities, or even those with learning disabilities, as a homogeneous group, despite widespread evidence for distinct subtypes (Dool, Stelmack & Rourke, 1993). Taking into account the existence of different dyslexic subtypes, Mazzotta & Gallai (1992) found that phonological dyslexics differed significantly from normals in presenting a longer auditory P3 latency and smaller amplitude on the N2-P3 wave, being consistent with previous studies with developmental dyslexics.

In the present study, we have focused our attention on the N2 component latency, because longer N2 latencies have been reported with developmental dyslexics (Fawcett et al., 1993), but not with dysphonemic dyslexics (Mazzotta & Gallai, 1992). We recorded the N2 latency for rare tones in two samples of children, one with a reading level according to their school grade and the other with severe reading problems. These problems were mainly characterized by a difficulty in the graphemes-to-phonemes conversion skill, and by visual mistakes in identifying words. The N2 component is a negative wave, peaking between 150 and 250 msec in the auditory modality, elicited during auditory stimulus selection tasks. This component has been dissociated into a N2a component («mismatch negativity») and a later frontally distributed N2b component, representing a sign of effortful «controlled» stimulus processing, with a peak latency that correlates with the time taken to categorize the eliciting stimulus (Näätänen & Picton, 1986). Taking into account that ERP measures appear to be often more sensitive to perceptual and cognitive processing than behavioral measures (Rugg, 1987), we expected to find differences in the N2 latency obtained for the rare tones, corroborating in dysphonemic dyslexics previous findings in developmental dyslexics (Fawcett et al., 1993).

If dysphonemic dyslexia were associated with specific alterations in the discrimination and integration of relevant auditory features preventing further proper development of phonological awareness, then the N2 latency, which has been postulated to reflect the taken to stimulus evaluation and categorization according to task relevance (Courchesne, Elsmasian & Yeung-Courchesne, 1987; Näätänen & Picton, 1986; Simson, Vaughan & Ritter, 1977), should have longer latency in dysphonemic dyslexics than matched control children.

Auditory brainstem responses (ABRs) were recorded in order to assess the integrity of the auditory periphery and auditory brainstem pathway to determine whether deficits in sensory processes contribute to the cognitive deficits in these subjects. Auditory middle evoked potential components were also examined in order to assess the cortical registration of the auditory stimulation.

Method

Subjects

Twenty-three Spanish children between age 8 years 6 months and 9 years 6 months participated in all conditions of this study. Ten (7 males and 3 females) were classified as normal readers, and 13 (7 males and 6 females) were classified as disabled readers. All subjects had normal hearing (audiometric tested), normal or corrected to normal vision, and no history of seizures or psychotic disturbances. No abnormality was found in conventional EEG examination. All subjects attended school regularly. Reading disabled subjects were referred to our laboratory, where they were tested by a psychological evaluation team, in order to identify reading problems. They were selected from a larger sample of disabled readers previously evaluated, because their reading errors were consistent with problems in reading phonology.

Control subjects were matched to the dyslexic subjects on the variables of sex, age, socioeconomic status, grade placement and Performance IQ. Control subjects were recruited from the same local elementary schools than disabled readers, and selected from a larger sample. They were required to have no current or past significant deficits in reading or spelling.

All subjects were administered the Spanish versions of the Quick Neurological Screening Test (Vila, 1981), the Conners Rating Scale for Parents and Teachers, the WISC-R IQ test, and academic achievement tests of oral and silent reading and spelling (MAE-ADL, TALE), which are specific Spanish tests for the assessment of dyslexia. In addition, a non-standardized clinical battery was used to assess several different phonological subskills of these children. This battery assessed phoneme (vocal and consonant) and syllable repetition and segmentation, rhyme ability, and word and pseudoword repetition subskills. Also a command comprehension (based on Token Test) and lexical generation tests were applied. The same test battery was administered to the control group.

Criteria for inclusion in the reading disabled group were: (a) no signs of neuro-developmental disturbances on Quick Neurological Screening Test; (b) a rating < 15 on the teacher and parent-rated Conners index; (c) a WISC-R full scale IQ > 90; (d) a report by at least two teachers stating that a subject was significantly retarded in reading given, his/her age, IQ, and previous schooling; (e) a number of errors on reading and spelling tests that were compatible with a discrepancy in the actual level of reading of at least 18 months below the corresponding grade of schooling; and (f) a pattern of reading errors that were consistent with problems in the phonological processing of written language. Thus, the disabled readers fulfilled the diagnostic criteria of developmental reading disorders following the DSM-III-R (APA, 1987). Normal readers were required to have: (a) negative findings on QNST score; (b) Conners index < 15; (c) a WISC-R full scale IQ > 90; (d) reading and spelling error scores according to their grade of schooling; and (e) non significant classroom behavioral or learning problems.

Table I shows the main characteristics for both groups of subjects. Both groups were matched as possible in age, grade of schooling, and WISC-R Performance IQ. However, the groups differed significantly on Verbal IQ and on subtests typically shown to differentiate disabled and normal readers: Information, Arithmetic, Vocabulary, and Digit Span (not used to compute VIQ). Greatest differences were found in reading and spelling tests, in which the

number of errors of the reading disabled children were, in most of the subtests, within the performance level of subjects with an age range between 6 and half and 7 years. Thus, the general error pattern during letter and word identification, pseudoword reading, and text reading and comprehension, was characterized by repetitions, substitutions and omissions and, in spelling, by substitutions, omissions, and additions. However, the dyslexic readers were selected because they consistently showed substitution errors in word and pseudoword reading characterized as visual similarity errors ('adjetivo' → 'objetivo'), and, in spite of low performance on word reading in comparison with normal readers, pseudoword reading was significantly more severely impaired. The stimulus was unsuccessfully repeated several times, omitted, or more frequently changed by a visually similar real word (v.g. 'tapic'->'tapia'). We think that letter errors are consistent with a poor knowledge or manipulation of the correspondences between basic orthographic units and their phonological counterparts, and that word and pseudoword reading errors are compatible with a dysfunction in the phonological route to read (Marshall, 1985). Furthermore, the disabled readers also had poor scores, in comparison with normal readers, on all phonological awareness subtests, including phonemic and syllable discrimination and segmentation, rhyme generation, and dictation, and problems with syntactic structures were also evident. Taken together, we consider this pattern very similar to dyslexic subtypes defined by several authors as dysphonemic (Boder, 1973), dysphonemic-sequential (Denckla, 1979), or auditory-linguistic (Pirozzolo, 1981). For all these reasons this group of reading disabled children was called as dysphonemic readers.

Procedure

ERP Recording. Subjects were familiarized with the laboratory previously to ERP testing. The subject was seated in a comfortable armchair at a sound and light-attenuated room in individual sessions of two hours. The following recordings were carried out:

1. Auditory brainstem responses (ABRs) were recorded from subjects placed in supine position. Ag/AgCl electrodes were attached on vertex (Cz) and over both mastoids, with a ground electrode on the forehead. Electrode's impedance was maintained below 5KΩ. They were connected to an averaging computer (Nicolet Pathfinder II) with a band pass of 200-300 Hz. Condensation and rarefaction click stimuli (0.1 msec duration, 80 dBHL intensity) were presented biaurally through headphones. Click stimuli were delivered at a rate of 11.1/sec and 200 responses were averaged. The first 10 msec were analyzed after a stimulus onset. The amplitude was measured from baseline to peak of each component. The peak latency of each component was measured from the beginning of the click stimuli. Wave IV sometimes appeared on the rising phase of wave V, but it was not always evoked. Therefore, wave IV was regarded as the same component as wave V and designated as wave V. The latency of the wave IV was measured to the peak of wave V.

2. Auditory middle evoked potentials were recorded from subjects placed in supine position. Ag/AgCl electrodes were attached on the vertex (Cz) and over both mastoids, with a ground electrode on the forehead. Electrode's impedance was maintained below 5KΩ. They were connected to an averaging computer (Nicolet Pathfinder II) with a band pass of 30-250 Hz. Condensation and rarefaction click stimuli (0.1 msec duration, 60 DBHL intensity) were presented biaurally through headphones. Click stimuli were delivered at rate of 6.1/sec and 100 responses were averaged. The amplitude was measured from baseline to peak of each component. The first 100 msec were analyzed after a stimulus onset. Na latency wave was identified in the maximum negative peak between 10-20 msec, Pa latency wave was identified in the maximum positive peak between 20-30 msec, and Nb latency wave was identified in the maximum negative peak between 30-40 msec.

3. Auditory event-related potentials. The electroencephalogram (EEG) was recorded with Ag/AgCl electrodes from frontal (F3, Fz, F4), temporal (T3, T4, T5, T6), central (C3, Cz, C4), and occipital (O1, Oz, O2) scalp sites according to the International 10-20 system (Jasper, 1958), all referred to linked mastoids. The electrooculagram (EOG) was recorded between supra- and infra-orbital sites around the right eye for vertival movement, and outer canthi of the left and right eyes, for possible eye movement artifact. The EEG was amplified with a bandpass of 0.5-70 Hz. Impedance of the EEG electrodes was maintained below 5KΩ.

Stimulus presentation and data acquisition were controlled by a NIC EEG 1A/97 system (Nicolet Biomedical Instruments). Digitized single-trail data and speed and accuracy of response on every trail were stored on a backup tape for later analysis.

Auditory event-related potentials were collected in a standard oddball paradigm. Subjects were instructed to detect an occasional (20%) high frequency target tone (2000 Hz, 50 msec duration) interspersed in a series of frequent (80%) low frequency (1000 Hz, 50 msec duration) tones. The order of target tones within the series of frequent tones was determined on a pseudorandom basis with the stimulus series. The interstimulus interval between tones was 1.5 sec. Subjects were instructed during a 30-stimulus practice run to familiarize themselves with the task. Tones were presented biaurally through headphones at an intensity of 65 Db SPL. The number of stimuli were 420. Subjects were required to move the index finger of their dominant hand whenever they heard the target tone. The task was considered correct when there were no more than 10% of failures in the responses to target tones.

Table 1
Group means in criterion variables. Two tailed t-test. Standard error in parentheses

Variable	NR	DR	t	p
N	10	13		
Age	106.4 (3.1)	108.9 (7.2)	1.10	n.s.
Grade	4.4.(0.5)	4.3(0.5)	0.41	n.s.
WISC-R IQ	111.4(6.1)	101.9(6.7)	3.47	0.002
Performance IQ	113.3(5.1)	107.2(12.4)	1.59	n.s.
Verbal IQ	105.6(6.9)	92.1(10.4)	3.50	0.002
Information	12.6(2.0)	10.4 (2.4)	2.61	0.016
Vocabulary	11.1(1.4)	8.3(3.6)	2.45	0.025
Arithmetic	12.8(1.8)	10.4(2.1)	2.77	0.011
Digit Span	10.4(2.1)	6.9(3.4)	2.77	0.011
Reading				
% Letter ID	93.0(7.4)	86.2(6.2)	2.36	0.028
% Syllable ID	97.5(3.5)	96.1(4.6)	0.76	n.s
% Word ID	93.4(4.4)	82.5(4.3)	5.90	0.000
Passage Reading	5.3(3.4)	9.8(3.5)	-3.11	0.005
Passage Comprehension	7.0(0.9)	6.2(2.8)	0.92	n.s.
% Word Reading	87.0(3.7)	70.5(3.9)	10.13	0.000
% Non-word Reading	80.7(8.9)	45.7(15.3)	6.40	0.000

Data Quantification

Brainstem evoked potentials have been established carrying out the classical method of considering positive peak latency values. The middle evoked potentials have been determined throughout the Na-Pa-Nb latency complex in Cz electrodes. Averages of auditory ERPs were constructed offline for each subject at each electrode but only for artifact-free trials. Separate averages were computed for target and non-target stimuli. Peak latency for the N2 component associated with target stimulus, was determined in each electrode as the point of maximum voltage on the greater negative peak between 150 and 350 msec from stimulus onset (Figure 1). In this figure, we present individual wave form recording for one subject. Amplitude was also calculated from baseline, being defined as the averaged potential of the 50 msec period before stimulus onset, to the maximum negative peak and expressed in µv. Trails with eye movement contamination were discarded before averaging.

Discussion

Recent behavioral evidence suggests that developmental dyslexics are slower than normal children during auditory selective discrimination tasks, but not during simple sensory detection tasks (Nicolson & Fawcett, 1993, 1994). These results seem to show that dyslexic children could have a deficit in stimulus categorization not restricted to linguistic stimuli, even when they are confronted with simple auditory stimulation, that could hamper the development of basic reading skills. However, negative results have been obtained studying N2 latency component in dysphonemic dyslexia (Mazzotta & Gallai, 1992), in which phonological processing is the underlying primary deficit.

The hypothesis of slow speed in stimulus categorization in dysphonemic dyslexia implies that these subjects could have a deficit in perceptual or central decision processes, but not in those processes associated with sensory recording of the stimulus. This is the reason we applied an audiometric test and an evoked response battery to evaluate a broad range of auditory functions in

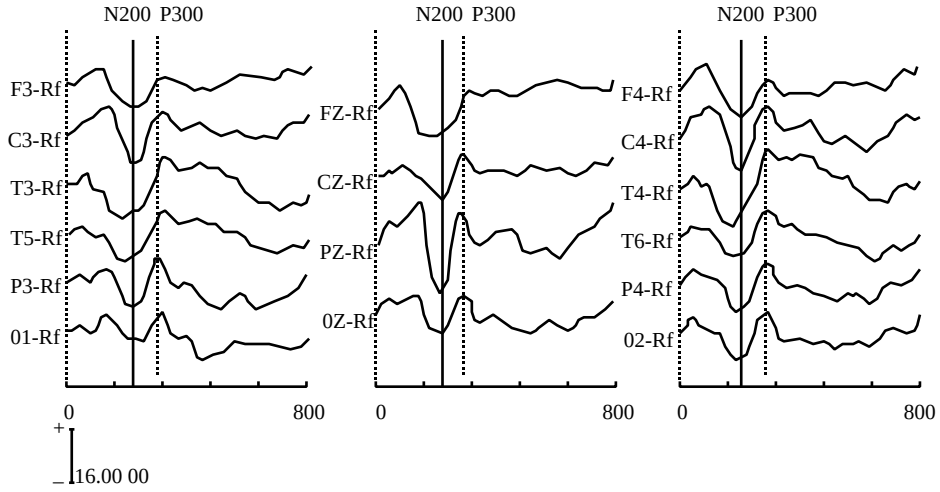

Figure 1. Individual wave form recording for one subject

The ABRs and Na, Pa and Nb auditory middle evoked potential latencies were compared between both groups using Student's «t-test» procedure (two-tailed). Auditory N2 latency and amplitudes comparisons between both groups were performed in each electrode site using Student's t-test (two-tailed).

Results

We did not find significant differences between groups in the ABRs or auditory middle evoked potential latencies, Na (t= -0.95, p<0.34), Pa (t= -0.98, p<0.33), and Nb (t= 1.04, p<0.30). These results involve the absence of pathologies in this area. Amplitudes in auditory ERPs did not reflect differences between groups. The analyses of N2 target tone latency (Table II) yield significant differences between both groups, O1 (t=-2.99, p< 0.008), Oz (t=-3.07, p< 0.005), O2 (t= -3.00, p<0.006), P3 (t= -2.89, p <0.011), and T5 (t= -3.12, p < 0.005). The dyslexic group presented longer N200 latencies in comparison with the normal group (Figure 2).

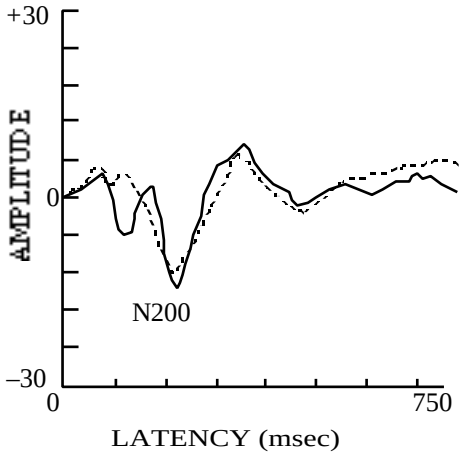

Figure 2. Individual wave forms recorded from Cz placement in a normal (solid line) and a dysphonemic dyslexic subject (dashed line), inwhich N200 component is observed

Table 2
Two-tailed t-test comparisons in N2 peak latencies recorded in each electrode site between normal and dysphonemic dyslexic children

	Normal	Dysphonemic		
Electrode	**Mean (Sd)**	**Mean (Sd)**	**t-value**	**p**
F3	226.2 (33)	255.7 (54)	-1.49	n.s
F4	231.4 (29)	258.2 (58)	-1.43	n.s
Fz	227.9 (34)	269.0 (57)	-2.00	n.s
T3	227.8 (28)	253.8 (54)	-1.49	n.s
T4	221.5 (44)	260.4 (56)	-1.80	n.s
C3	219.8 (24)	251.2 (66)	-1.59	n.s.
C4	225.3 (29)	250.5 (57)	-1.39	n.s.
Cz	226.2 (24)	247.5 (43)	-1.47	n.s.
T5	210.8 (28)	265.1 (53)	-3.12	0.005
T6	212.4 (39)	249.6 (56)	-1.77	n.s.
P3	212.8 (18)	260.6 (57)	-2.89	0.011
P4	220.8 (34)	265.4 (66)	-2.07	n.s.
Pz	220.7 (26)	255.7 (61)	-1.86	n.s.
O1	207.1 (19)	248.4 (44)	-2.99	0.008
O2	213.6 (27)	262.6 (46)	-3.00	0.006
Oz	209.6 (17)	254.4 (48)	-3.07	0.005

normal and dysphonemic dyslexic children. Auditory brainstem responses give information about the integrity of the auditory periphery and auditory brainstem pathway. On the other hand, auditory middle evoked potential components may assess the cortical registration of the auditory stimulation. Audiometric screening showed these subjects had normal auditive levels. Brain-stem potential values of latency showed that there were no differences between both groups, indicating the absence of functional alterations in brain stem, structural lesions, or deficits in speed of transmission of brain stem, which is consistent with previous research (Legatt,Arezzo & Vaughan, 1988; Grntved, Walter & Grnborg, 1988, Ollo & Squires, 1986). The results of auditory middle evoked potentials suggest the absence of functional alterations in both groups in the auditive pathway, respect to mesencephalic and diencephalic levels, as well as in the primary auditive cortex. These structures, as Deiber, Ibañez, Fischer, Perrin & Manguiere (1988) have stated, can be considered as centers which generate these kinds of evoked potentials. As a whole, these results indicate that dysphonemic dyslexics did not reflect dysfunctional sensory abnormalities during auditory stimulation that could explain their possible deficits with auditory stimulus recognition.

On the other hand, well defined differences between both groups of subjects were detected in target N200 latency. The left temporo-parietal and bilateral occipital electrodes showed significant differences between groups. Dysphonemic dyslexics had longer N200 latencies in comparison with normal readers. This result correlates with the study of Fawcett et al. (1993), that reported longer rare N2 latencies for developmental dyslexics than for normal subjects; although they reported differences in Cz site, the only electrode recorded. Taking into account that N2 latency has been associated with stimulus evaluation and classification according to task relevance, data suggests that occipital and left temporo-parietal regions have a significant temporal delay to be engaged in the auditory stimulus recognition. This may reflect a difficulty of these cortical areas to be activated in a normal fashion.

Precisely, it has been postulated that these cortical areas play a key role in oral language and reading comprehension abilities (Damasio, 1989; Geschwind,1985). Thus, the present pattern may imply an inefficient functional pattern for acoustic processing in dyslexic children, even when non-linguistic information is involved, leading to a greater cognitive effort to cope with the task (Rugg et al., 1988). In summary, the longer N2 latency in the dysphonemic dyslexic group might suggest greater levels of effort required by this group to perform the task adequately, as would be predicted by the «copying-hypothesis» of Van Zomeren, Brouwer & Deelman (1984) as a result of a deficient information processing.

The increased latency in the left hemisphere of dysphonemic dyslexics, is consistent with the proposal of Jorm (1979) that dyslexia is associated with a deficit of the left parietal lobe, implying important neurophysiological problems when subjects are processing visual-linguistic stimuli that are integrated in occipital areas of the left hemisphere. Also, the left posterior quadrant of the scalp has been the most frequently noted as dysfunctional or presenting an unusual functional asymmetry pattern in ERP studies searching for specific electrophysiological signs of developmental phonological dyslexia (Duffy & McNulty, 1985; Fried, Tanguay, Boder, Doubleday & Greensite, 1981). Neuropsychological evidence shows that a brain lesion in the depicted areas may provoke a reading impairment characterized by a severe difficulty to read by phonology (Baxter & Warrington, 1985).

On the other hand, (the pattern of) results suggest another possible deficit in the temporal region, due to a great increase in T5 latency. If we consider the results of Hari et al.(1984), and of Sams et al.(1985), who found that N2 component could be primarily generated in the supratentorial and primary auditory cortex, the delay of N2 latency in electrode T5 would reflect a neurophysiological deficit that would justify a difficulty in the differential evaluation of auditory stimulation.

We think that the involved topography is consistent with a functional deficit in the management of auditory-phonetic information, hampering the development of all those basic skills related with phonological awareness, on which reading progress seems to depend (Goswami, 1993). Consistent with our hypothesis, N2 latency was delayed in phonemic dyslexics only during auditory categorization, and in those cortical areas postulated to be important in phonological route for reading, corroborating previous studies with developmental dyslexics.

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