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# Changes in EEG Current Sources Induced by Neurofeedback in Learning Disabled Children. An Exploratory Study

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**Abstract** The objective of this work was to explore Neurofeedback (NFB) effects on EEG current sources in Learning Disabled (LD) children, and to corroborate its beneficial consequences on behavioral and cognitive performance. NFB was given in twenty 30-min sessions to 11 LD children to reduce their abnormally high theta/alpha ratios (Experimental Group). Another five LD children with the same characteristics received a placebo treatment (Control Group). In the Control Group no changes in behavior or EEG current source were observed. In the Experimental Group, immediately after treatment children showed behavioral and cognitive improvements, but current source analysis showed few modifications; however, 2 months after treatment many changes occurred: a decrease in current of frequencies within the theta band, mainly in left frontal and cingulate regions, and enhancement in current of frequencies within the alpha band, principally in the right temporal lobe and right frontal regions, and of frequencies within the beta band, mainly in left temporal, right frontal and cingulate cortex regions. In

conclusion, NFB is a possibly efficacious treatment for LD children with an abnormally high theta/alpha ratio in any lead. The changes observed in EEG current sources may reflect the neurophysiological bases of the improvement that children experienced in their behavioral and cognitive activities.

**Keywords** Neurofeedback · Learning Disabled children · EEG current sources · EEG normative database · Control and Experimental Groups

## Introduction

Learning disabilities (LD) are diagnosed when an individual's achievement on individually administered, standardized tests in reading, mathematics, or written expression is substantially below that expected for a particular age, schooling, and level of intelligence. LD are classified as “specific” (in reading, mathematics, or written expression) or “learning disorder not otherwise specified”, which can include problems in all three areas (American Psychiatric Association 1994).

In previous work, it was shown that Neurofeedback (NFB), which decreases the theta/alpha ratio was a useful treatment in LD children. Behavioral improvements were observed immediately after the end of the treatment, but EEG changes were observed only until 2 months later (Fernández et al. 2003).

In the present paper, we evaluate the EEG changes using a current source frequency analysis since this procedure reveals topographic aspects that are not evident in the conventional EEG frequency analysis.

The rationale for the treatment applied in the previous report was based on the following: (a) the most frequent

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EEG abnormality observed in LD children is an excess of theta activity when compared with the EEG of normal children of the same age (Alvarez et al. 1992; Chabot et al. 2001; Fernández et al. 2002; Gasser et al. 2003; Harmony et al. 1990a, b; John et al. 1983); and (b) a minimum quantity of alpha activity at rest is necessary for the correct performance of mental tasks in those regions involved in the tasks both in normal children (Fernández et al. 1998) and normal adults (Fernández et al. 2000).

These facts strongly suggest that for LD children with these EEG abnormalities, reinforcing the reduction of the theta/alpha quotient may produce a trend toward EEG normalization and, as a consequence, an improvement in behavioral and cognitive abilities (Sterman and Egner 2006).

## Methods

The Ethics Committee of Instituto de Neurobiología, Universidad Nacional Autónoma de México approved the experimental protocol.

### Participants

One hundred and nine children between 7 and 11 years of age (24 female) were referred by a social worker from several elementary schools in Querétaro. Sixteen children fulfilled the following inclusion criteria: LD children with normal neurological exam, Intelligence Quotient (IQ) greater than 70 (IQ was assessed by the revised version of the Wechsler Intelligence Scale for Children, WISC-R, Wechsler 1981), mother with at least a third-grade elementary school education, *per capita* income greater than 50% of the minimum wage, and an EEG recorded at rest with eyes closed in which at least one lead showed an abnormally high value of the theta/alpha ratio compared to a normative database (Valdés et al. 1990, see below). Children with paroxysmal activity in the alpha frequency range were excluded. Children with ADHD diagnosis or another psychiatric alteration were also excluded.

A team composed by a neurologist, a neuropediatrician, and a clinical psychologist evaluated the children to establish the “LD not otherwise specified” diagnosis according to the DSM IV criteria (American Psychiatric Association 1994): Learning disabilities (LD) are diagnosed when an individual’s achievement on individually administered, standardized tests in reading, mathematics, or written expression is substantially below that expected for a particular age, schooling, and level of intelligence. The tests used were the arithmetic subscale of the WISC-R, and a writing-reading test standardized by grade for

Mexican children (Iglesias and Derman 1985). In addition, clinical characteristics of the child and his/her academic achievement were taken into account. Children included in this study were classified as suffering from “learning disorder not otherwise specified”; several of them presented problems in attentional processes, as is common in this population (Bernal et al. 2000; Holcomb et al. 1986; Iragui et al. 1993; Silva-Pereyra et al. 2003), but they did not meet the DSM-IV criteria for ADHD (American Psychiatric Association 1994). In addition, cranial computed tomography was performed on each child in order to exclude those with major brain abnormalities. Children excluded from the sample were sent to a specialized service or included in other research project.

Eleven children (six females) received the NFB treatment (Experimental Group), and the other five (one female), received a placebo treatment (Control Group). Five out of the eleven LD children who received the NFB treatment and the five children who received the placebo treatment had been included in an earlier study (Fernández et al. 2003). These two groups were not different in the mean values of age, sex, IQ, ADHD score from TOVA, *per capita* income in the family, and mother’s schooling. The remaining six children of the 11 who received the NFB treatment were recruited in the same way, but no criteria were imposed in order to assign them to the treatment group (paired versus randomized criteria). Just as in the case of the Fernández et al. study (2003), no significant differences were observed between the Control and the Experimental Groups regarding age, IQ, sociocultural level (*per capita* income and mother education), and ADHD score from TOVA.

All children were volunteers; parents’ informed consent was obtained in all cases.

The age of the children in the Experimental Group ranged between 7.16 and 10.68 years (8.94, 1.30; mean  $\pm$  sd), and in the Control Group between 7.83 and 11.36 years (9.7, 1.40; mean  $\pm$  sd).

### Procedure

Before treatment, two or three EEG recordings were taken from each child in order to select the lead where the most abnormal Z value of the theta/alpha ratio was found in two recordings. NFB was applied on the basis of the EEG activity at this lead. The last EEG recording before treatment was used as “before” in the statistical analysis because the first EEG recording could have been affected by the novel environment for the child.

WISC-R and the parent interview (see below) were carried out before treatment and used as inclusion criteria. The Test of Variables of Attention (TOVA; Lark et al.

1999) in its visual mode was also applied, but it was not used as an inclusion criterion.

Immediately after the 20 sessions (NFB or placebo), TOVA and EEG were repeated following the same procedures as before treatment. Two months later, another EEG was recorded. A second application of WISC-R was administered 6 months after the first one, in accordance with WISC-R recommendations. A final interview to the parents was conducted after the treatments in order to obtain a qualitative evaluation of behavioral changes. Academic achievement was communicated by the parents.

### *Behavioral Assessment*

**WISC-R** It is one of the most-used instruments in assessing children's intelligence and general cognitive functions. It is a collection of 13 distinct subtests divided into two scales (a Verbal scale and a Performance scale). The six Verbal scale tests use language-based items, whereas the seven Performance scales use visual-motor items that are less dependent on language. Five of the subtests in each scale produce scale-specific IQs, and the 10 subtest scores produce a Full Scale IQ. The WISC-R was standardized on a sample of 2,200 American children selected as representative of the population on the basis of the 1970 U.S. Census. It shows strong correlations with comparable metrics from WPPSI (0.82) and the Stanford-Binet-IV (0.73). Each of the three IQ scales has an internal consistency reliability coefficient of 0.90 or above in the standardization group over the entire age range covered by the scale. Average internal consistency reliability coefficients, based on the 11 age groups, are 0.96 for the Full Scale IQ, 0.94 for the Verbal Scale IQ, and 0.90 for the Performance Scale IQ. Test-retest stability coefficients are 0.95 for the Full scale IQ, 0.93 for the Verbal scale IQ, and 0.90 for the Performance scale IQ (Wechsler 1981). The average standard errors of the normalized measurements, based on the 5 age groups between 6.5 and 10.5 years, are 3.28 for the Full Scale IQ, 3.86 for the Verbal Scale IQ, and 4.63 for the Performance Scale IQ.

In this study WISC-R was used to exclude mental retardation and to assess the arithmetic performance (only in this sense it contributed to the LD diagnosis), and to determine, together with the data derived from TOVA and parent interviews, if children treated with NFB showed a cognitive improvement.

**TOVA** In its visual mode TOVA was administered to all children. TOVA is a computerized, continuous performance test in which the subject has to respond to a target that is presented less frequently (22.5%) than non-target

stimuli in the first half of the test, and more frequently (77.5%) in the second half. The "ADHD score" is the index of the TOVA that tells how similar the performance is to that of the Attention Deficit/Hyperactivity Disorder (ADHD) profile. The "ADHD score" is calculated from the  $z$  score of the response time, response time variability, commission rate, and hit rate. The test-retest reliability of TOVA has shown significant reliability coefficients for response time variability (infrequent target 0.92; frequent target 0.98), omission (infrequent target 0.88; frequent target 0.88), response time (infrequent target 0.89; frequent target 0.99), and commission (infrequent target 0.71; frequent target 0.93) in healthy school-age children (Leark et al. 1999). Consistency studies between the Conners Parent Rating Scale and TOVA have shown similar results in ADHD children in approximately 85% of children (Schatz et al. 2001). An "ADHD score" value below  $-1.8$  is suggestive of ADHD, though it is not conclusive.

### *Interview of Parents*

An experienced clinical psychologist interviewed the mother, father, or tutor of the child on the basis of an ad hoc questionnaire to evaluate socioeconomic status (mother schooling and *per capita* income are inclusion criteria) and family integration, psychomotor and emotional development, social interaction, cognitive abilities, academic achievement, and pathologic antecedents of the child. This interview had two objectives: (a) as an inclusion criterion, and (b) to assess the qualitative changes observed by the parents after treatment regarding attention, memory, learning, behavior, attitude toward school, social interaction, and emotional changes of the child.

**EEG** The EEG was recorded with two purposes: (a) as an inclusion criterion (the subject should have at least one lead with an abnormally high value of the theta/alpha ratio, and he/she should not present paroxysmal activity in the alpha range), (b) to compute the EEG current sources in all frequencies in three conditions: before, immediately after, and 2 months after NFB treatment. MEDICID IV, the equipment used to record the EEG, was also used to analyze the EEG and give the NFB. This equipment was developed by Neuronic A.C., and has been used in numerous studies. The norms (Valdés et al. 1990) and the description of the NFB program (Fernández et al. 2003) will be described below.

**EEG Recording:** Subjects were seated in a comfortable chair in a dimly lit room. Digital EEG was recorded at rest with eyes closed during 20 min from 19 leads (10–20 International System) using linked ear lobes as reference. A1A2 reference was used in order to have the same

conditions as in the normative data. The amplifier bandwidth was set between 0.5 and 30 Hz. The EEG was sampled every 5 ms using the MEDICID IV System and edited off-line. An expert electroencephalographer using visual editing selected 24 artifact-free segments of 2.56 s for quantitative analysis.

*EEG Analyses:* The analyses were done off-line. The fast Fourier transform and cross-spectral matrices were calculated every 0.39 Hz, and the absolute power (AP) in theta (3.6–7.5 Hz) and alpha (7.6–12.5 Hz) bands was computed. The ranges of these bands were selected according to normative data provided by MEDICID IV. Population parameters of the normative data were based on the regression function of age-dependent mean values and the standard deviation obtained from 211 normal subjects between 6 and 90 years old; the ages were distributed in logarithmic form; thus, they are concentrated in the age range considered in the present paper (Valdés et al. 1990, 1992).

*Z Values for the Theta/Alpha Ratio*—Z values for the theta/alpha ratio were calculated in the following manner: AP in each band was computed for the average reference, and the geometric power (Hernández et al. 1994) was subtracted from the cross-spectral matrix. The value “log(theta AP/alpha AP)” was computed, and Z values for this logarithm were calculated by means of the equation:

$$Z = [\log(\text{thetaAP}/\text{alphaAP}) - \mu]/\sigma$$

where  $\mu$  and  $\sigma$  are, respectively, the mean value and the standard deviation of the normative sample of the same age as the subject. Taking into account that the EEG abnormality considered in this population is a high value of the theta/alpha ratio, it was deemed that Z should be greater than 1.645 to be considered as abnormal (this Z value corresponds to a 1-tail distribution,  $p = 0.05$ ). The presence of at least one abnormal value in one lead was used as an inclusion criterion. The lead with the highest abnormal value was selected to give the treatment.

*EEG Current Sources:* Frequency domain variable resolution electromagnetic tomography (FD-VARETA) was used to calculate the distributed sources at 0.78 Hz intervals. This is a technique for estimating the source generators of EEG data (Bosch-Bayard et al. 2001; Valdés et al. 1996). VARETA is a discrete spline distributed solution (Riera et al. 1997) that imposes different amounts of spatial smoothness for different types of generators selected by a data-driven search procedure. In VARETA, current sources are restricted to cortical gray matter by use of a probabilistic mask that prohibits solutions where the mask is zero, i.e., in cerebrospinal fluid, white matter, and subcortical nuclei. As is well known, for neural primary currents to be measurable at the scalp they must not only be produced by neurons with an open field, but also their

activity must be synchronized spatially and temporally (Kutas and Dale 1997). In addition, the deeper the sources, the weaker the signal recorded at the scalp. These are the a priori theoretical considerations that make everyone reluctant to deal with deeper sources.

All solutions had to belong to the cortex. The probabilistic brain atlas used was developed at the Montreal Neurological Institute (Collins et al. 1994; Evans et al. 1993, 1994; Mazziotta et al. 1995). The mean head used in this work was obtained (Evans et al. 1994) by averaging a set of 305 normal MRI scans transformed to Talairach space. The MRIs had been subjected to non-linear warps to match a set of 50 common landmarks. The probabilistic brain atlas is intended to provide approximate solutions that make the use of individual MRI scans unnecessary by sacrificing precise anatomical localization. The three concentric sphere volume conductor model was fitted to the Montreal Neurological Institute mean head by means of the least-squares algorithm. Brain electric source analysis (BESA) (Scherg and Von Cramon 1985) coordinates for each electrode were then used to project each electrode onto the averaged skin. For the head volume conductor model, it was assumed that the conductivity of the three spheres was 1.0 for the skin, 0.0125 for the skull, and 1.0 for the brain. The ratio of the spheres' radii was standardized to 1.0 for the skin, 0.94 for the skull, and 0.86 for the brain.

Special-purpose 3D graphical tools were developed in order to allow interactive evaluation of the large amount of transformed data available. In a generalization of topographic maps, 3D color-coded images or brain electromagnetic tomographies (BETs) are generated, in which the color code reflects the magnitude of the current at each point of the grid or voxel. In this manner, BETs for each frequency were obtained. The complete description of the procedure can be found in Bosch-Bayard et al. (2001).

Frequency Domain VARETA has previously been used by our group to evaluate the topography of brain lesions (Fernández-Bouzas et al. 1999, 2000), as well as to study cognitive processes (Fernández et al. 1998, 2000; Harmony et al. 1999, 2001, 2004).

In the current paper, the EEG current sources were used to evaluate the effects of the treatment.

## Treatment

*NFB Treatment* NFB was conducted using an NFB program adapted to the MEDICID IV recording system and software. The EEG signal was obtained from a lead situated at the site with the most abnormal theta/alpha ratio, referred to linked earlobes. At the first session, a threshold level was selected such that the subject obtained a reward

(a 500 Hz tone) between 60% and 80% of the time; the threshold was updated every three minutes. In the following sessions, the first threshold was calculated as the average threshold of the previous session.

Throughout the continuous recording of the selected lead, the theta/alpha ratio was computed for 1,280 ms every 20 ms and compared with the previous threshold. If the ratio was lower than the threshold, then the reward (tone) was given. Subjects were told that it was important to maintain the duration of the sound as much as possible, and consequently the tone acquired a positive meaning. The first threshold used in a given session is the average between the first and the last thresholds used in the previous session. At the beginning of each session, the child was told that if his/her performance was good, he/she would receive candies at the end of the session.

Each child received 20 sessions of training (each of which lasted 30 min) over a period of 10–12 weeks. The NFB treatment was given by trained postgraduate students of the Master's Degree Program in the Instituto de Neurobiología.

The theta/alpha ratio at the beginning and the end of each session was recorded in the lead selected to give the treatment. It was considered that a child had learned to decrease the theta/alpha ratio when the ratio at the end of the session was less than 75% of the ratio at the beginning of the session. It should have happened in at least 15 sessions of the 20 sessions. In addition, in the EEG recorded at rest before and immediately after treatment, the theta/alpha ratio in the lead selected to give the NFB training was computed, and these two ratios were compared.

**Placebo Treatment** In the placebo treatment all conditions were exactly the same as in NFB, except that in this case the tone onset and its duration were randomly given, not contingent upon EEG activity.

### Statistical Analysis

**Behavioral Analysis** Given the small size of the samples, a normal distribution was not warranted, so parametric analyses were not appropriate. The statistical significance of the differences of behavioral and EEG data-between before and after treatment for each group was assessed by a multivariate non-parametric permutational test (Galán et al. 1997) for dependent variables. For TOVA data two different analyses were performed: one for the ADHD score and another one for percentage of omissions (%O), percentage of false alarms (%FA), and average response time (RT). We performed two analyses of the WISC-R data: one for the total IQ, and the other one for verbal and

performance scales. In all behavioral analyses the global null hypothesis tested the equality of all variables included in each analysis, and the marginal null hypotheses tested the equality of particular variables.

The Kolmogorov–Smirnov test was performed to test if the assumption of normality of data distribution was met. The comparison between groups regarding the changes produced by NFB were computed by an ANOVA-RM; the factors considered were condition, as the within factor, and group, as the between factor.

**EEG Current Source Data** The values resulting from the source analysis at each point of the grid, for all the frequencies studied, were used to calculate differences in the BET images before and after NFB treatment independently for each group. For each frequency, multivariate nonparametric permutational tests (Galán et al. 1997) were computed to compare the following conditions: before versus immediately after, and before versus 2-months after NFB.

Permutational tests have the following advantages: the tests are distribution free, no assumptions of an underlying correlation structure are required, and they provide exact *p*-values for any number of subjects, time points, and recording sites (Galán et al. 1997). Multivariate statistics can be used to summarize and test differences between two conditions obtained from the maximum value of all the univariate statistics. This may also be the maximum of the *t* distributions calculated between the two sets of data, *t*<sub>max</sub>, for all frequencies and across all voxels. The distribution estimated by permutation techniques for *t*<sub>max</sub> can then be used to set significance levels controlling the experiment-wise error for simultaneous univariate comparisons, thus avoiding a type I error (Blair and Karninski 1993, 1994).

To analyze the differences between groups with respect to the changes between before, immediately after, and 2 months after NFB treatment, we performed an ANOVA-RM test, as previously described.

## Results

### Behavioral Results

#### WISC-R

Tables 1 and 2 show the age, IQ, and ADHD score from TOVA before and after treatment, as well as the sex and the lead with the most abnormal theta/alpha ratio for each subject in the Experimental and the Control Groups, respectively. In the Control Group no significant differences were observed in WISC-R when before and after

**Table 1** Subject characteristics—Experimental Group

Subject	Age	IQ before	IQ after	ADHD score before	ADHD score after	Sex	Lead with the most abnormal theta/alpha ratio
W003	10.56	70	73	−1.44	0.12	M	T3
W008	10.30	78	85	−4.72	−3.71	F	P4
W024	8.44	102	118	−0.98	−1.69	M	T3
W035	10.01	84	90	1.32	1.25	M	F4
W048	7.26	72	76	−0.41	−	M	T3
NRA05	10.68	72	69	−6.56	−5.33	F	C3
NRA09	8.41	70	70	−2.56	−0.25	M	T3
NRA15	7.16	80	79	−4.21	−1.21	F	O2
NRA17	9.09	75	82	−0.55	−0.47	F	O1
NRA18	8.72	71	82	−0.77	0.22	F	T5
NRA19	7.65	72	69	−6.78	−0.64	F	P4
<b>Mean</b>	<b>8.94</b>	<b>76.91</b>	<b>81.18</b>	<b>−2.51</b>	<b>−1.17</b>		
<b>SD</b>	<b>1.30</b>	<b>9.47</b>	<b>14.03</b>	<b>2.68</b>	<b>1.97</b>		

**Table 2** Subject characteristics—Control Group

Subject	Age	IQ before	IQ after	ADHD score before	ADHD score after	Sex	Lead with the most abnormal theta/alpha ratio
W005	10.81	75	76	0.60	−1.04	M	F4
W009	8.80	70	73	−2.59	−4.75	M	T4
W031	7.83	92	92	−0.95	−4.13	M	P4
W041	11.36	79	82	0.22	0.77	M	F4
W052	9.77	80	85	−2.15	−4.22	F	Pz
<b>Mean</b>	<b>9.71</b>	<b>79.20</b>	<b>81.60</b>	<b>−0.97</b>	<b>−2.67</b>		
<b>SD</b>	<b>1.44</b>	<b>8.16</b>	<b>7.50</b>	<b>1.40</b>	<b>2.41</b>		

treatment scores were compared. In the Experimental Group, IQ scores before and after NFB treatment were compared; results are shown in Fig. 1. The IQ and verbal scores from WISC-R increased significantly ( $p = 0.02$  and  $p = 0.001$ , respectively), but the performance score did not. ANOVA-RM analyses revealed no significant differences between groups for IQ, verbal, or performance score.

In the total IQ scale, a change of more than three points exceeds the standard error of the normalized measurement. In the Experimental Group, 6 out of 11 children (54.5%) improved their total IQ by four points or more, while for five of them it remained approximately the same (3 or below). In the Control Group, only one girl (w052) increased her total IQ by more than three points.

### TOVA

The ADHD score from TOVA was invalidated for one child in the Experimental Group, so the statistical analysis was performed with 10 subjects. Significant increases ( $p = 0.01$ ) in the ADHD score from TOVA were observed

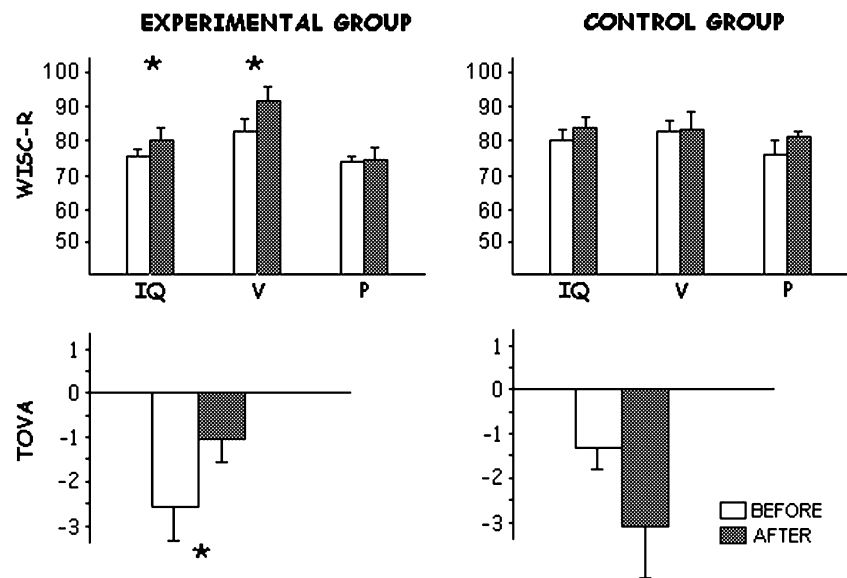
(Fig. 1). When reaction time, false alarm percent, and omission percent were compared before and after NFB treatment, a highly significant ( $p = 0.001$ ) decrease of omission percent was observed in the Experimental Group. The samples exhibited a normal distribution; thus, this assumption for ANOVA tests was fulfilled. The ANOVA-RM results were highly significant only for the interaction group  $\times$  condition ( $F(1,13) = 10.937$ ,  $p = 0.006$ ).

In the Experimental Group, 6 out of 10 children (60%) improved their ADHD score by more than one point. In two of the remaining four children, the ADHD score improved by less than one point and the other two decreased by less than one point. In the Control Group, the ADHD score of four out of five children (80%) showed deterioration by more than one point; the remaining child (W041) improved by less than one point.

### Parent Interview

All parents of children in the Experimental Group reported behavioral improvement in memory, attention, and attitude

**Fig. 1** WISC-R mean values and standard errors before and after NFB treatment in Experimental and Control Groups. Significant differences (\*) were only observed in the Experimental Group. V = verbal scale, P = performance scale



toward school. According to the parents, these changes were manifested as betterment in their children's academic performance except in child W035, in whom no changes in academic performance were observed. All the parents also reported highly significant positive changes in their children's socialization, self-confidence, and independence behaviors, with the exception of one mother who said that her daughter had become disrespectful and defiant.

According to the parents, two children in the Control Group (w009, w031) presented no improvement. The parents of the remaining three children of the Control Group reported behavioral improvements in memory, attention, and attitude toward school. Only girl w052 showed an improvement in academic performance.

#### EEG Theta/Alpha Ratio

All children of the Experimental Group learned to decrease the theta/alpha ratio during the course of the NFB sessions. However, this learning was not so evident when the comparison between EEG at rest recorded before and immediately after NFB treatment was made, because only in 4 of the 11 children the theta/alpha ratio immediately after NFB was less than 75% of the ratio obtained before NFB; however, 2 months later, 7 of the 11 children showed a decrement of this ratio.

#### EEG Current Sources

In the Control Group no significant differences were observed in EEG current sources. The results for the Experimental Group are shown in Table 3. The bands considered were delta (1.5–3.5 Hz), theta (3.6–7.5 Hz),

alpha (7.6–12.5 Hz), and beta (12.6–19 Hz). All current decreases occurred in the delta and theta bands, and all current increases occurred in the alpha and beta bands. In the comparison between before and immediately after NFB, few significant differences were observed: the current showed a significant reduction in the 3.51 Hz frequency in the right occipitotemporal region and increases in the 7.8 Hz frequency in the cingulate region and in the 9.36 Hz frequency in the left inferior occipital gyrus. Two months later, many changes were found, namely, a current reduction, mainly in the frequencies within the theta band, in the cingulate region, temporal lobe, frontorbital gyrus of the left hemisphere, and in the right parahippocampal area; also current increases in frequencies were observed within the alpha band in temporal and frontal regions of the right hemisphere, and in frequencies within the beta band in frontal, temporal, and cingulate areas. Figures 2 and 3 show examples of the significant differences between 2 months after NFB and before NFB in 6.24 and 8.16 Hz frequencies, respectively. At 6.24 Hz frequency the current decreased after NFB and at 8.16 Hz frequency current increased after NFB.

With the objective of formalizing the comparison between groups across time, an ANOVA-RM test was performed, considering condition as the within factor, and group as the between factor. No significant differences were observed in any factor or interaction. These results should be taken with caution, since the assumption about the normal distribution of the data was not fulfilled.

#### Discussion

Although the exact physiological bases underlying NFB are not well understood, NFB has proven useful in the



**Table 3** Significant EEG current sources changes in experimental group

Band	Freq (HZ)	Decreases		Increases	
		B > iA	B > 2mA	B < iA	B < 2mA
Delta	0.78		R middle occipital gyrus.		
	3.51	R anterior occipito-temporal region, and R uncus.			
Theta	4.29		L posterior cingulate region, and bilateral anterior cingulate area.		
	6.24		L superior temporal gyrus, L insula, inferior part of L precentral and postcentral gyri, R parahippocampal gyrus, and R lateral occipitotemporal gyrus.		
	6.63		L lateral frontorbital gyrus.		
	7.02		Bilateral lateral frontorbital gyrus, and L middle frontal gyrus		
	7.80			L anterior cingulate region.	
Alpha	8.19				R uncus, R inferior temporal gyrus, R hippocampal and parahippocampal gyrus, R superior frontal gyrus, and R medial frontal gyrus.
	9.36			L inferior occipital gyrus	
	10.53				L superior postcentral region, and superior region of the R middle frontal gyrus.
	10.92				Medial region of the R middle temporal gyrus, and posterior part of the superior frontal gyrus
	17.16				Superior part of the R precentral gyrus, R medial frontal gyrus, R cingulate region, and L superior temporal gyrus.
Beta	17.55				Posterior region of the bilateral cingulate gyri.
	18.72				Medial region of the L lateral occipitotemporal gyrus.

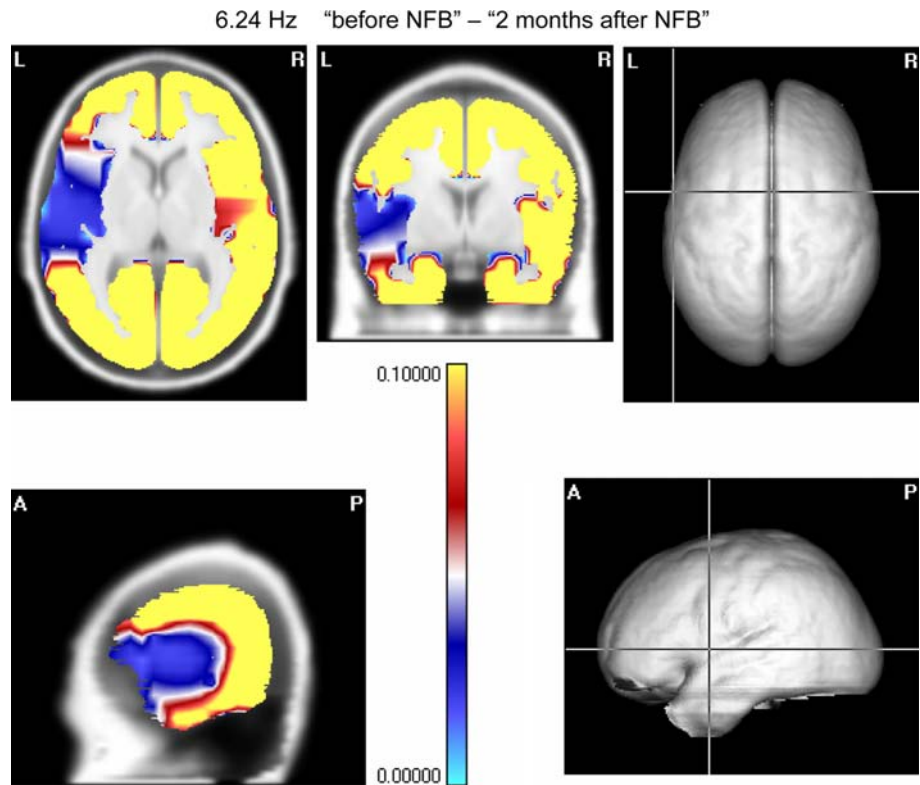
B = before NFB, iA = immediately after NFB, 2mA = 2 months after NFB

treatment of many psychiatric conditions, such as in ADHD (Beauregard and Levesque 2006; Butnik 2005; Fox et al. 2005; Fuchs et al. 2003; Leins et al. 2007; Levesque et al. 2006; Linden et al. 1996; Lubar and Lubar 1999; Lubar et al. 1985; Lubar et al. 1995a, b; Monastra et al. 2002, 2005; Nash 2000; Penberthy et al. 2005; Pop-Jordanova et al. 2005; Rossiter 2004a, b; Thompson and Thompson 1998), anxiety (Abarbanel 1999; Hammond 2005; Moore 2000), and affective disorders (Baehr et al. 1999; Hammond 2005; Rosenfeld 2000), as well as in the treatment of addictions (Trudeau 2000), mainly alcoholism (Kelley 1997; Peniston and Kulkosky 1999), and

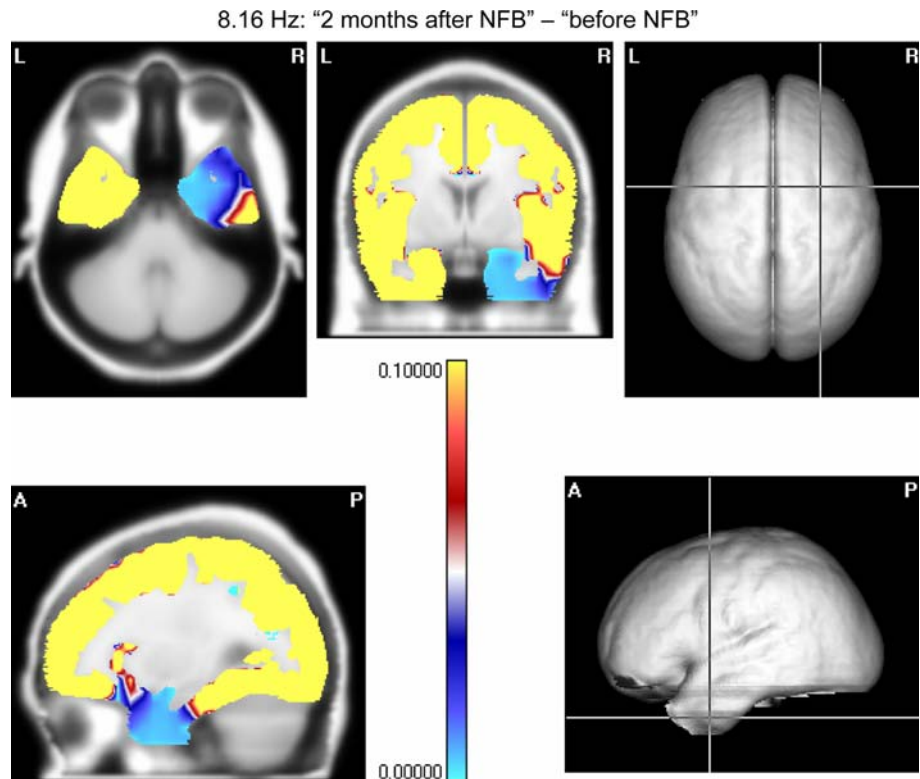
neurological disorders, such as epilepsy (Egner and Sterman 2006; Goldstein 1997; Lubar and Bahler 1976; Seifert and Lubar 1975; Sterman 2000; Sterman and Egner 2006; Sterman and Friar 1972; Walker and Kozlowski 2005) and traumatic brain injury (Ponsford and Kinsella 1998; Stamatina and Lubar 2004; Tinius and Tinius 2000). However, to the best of our knowledge, only one study considering exclusively LD children has been previously reported (Fernández et al. 2003).

The present paper is focused on the changes produced by NFB treatment on the EEG current sources as well as on behavior of LD children with abnormal theta/alpha ratios in

**Fig. 2** Probability maps for the 6.24 Hz frequency. Longitudinal, coronal, and sagittal slices of regions where the maximum difference was observed between 2 months after NFB and before NFB. At the right, the levels of the slices on the average brain are shown. The color scale shows the probability of the null hypothesis (current 2 months after NFB equal to current before NFB). Significant decreases (blue color) at 6.24 Hz were observed in the left superior temporal gyrus, left insula, and inferior part of left precentral and postcentral gyri



**Fig. 3** Probability maps for the 8.16 Hz frequency. Significant differences (blue color) in current between 2 months after NFB and before NFB. Current 2 months after NFB was higher than before NFB in the right uncus, right inferior temporal gyrus, right hippocampal, and parahippocampal gyri



their EEGs. Two groups were studied: an Experimental Group, which received NFB treatment, and a Control Group, which received a sham treatment.

According to the Guidelines for Evaluation of Clinical Efficacy of Psychological Interventions (La Vaque et al. 2002), our study could be classified as *possibly efficacious*

for LD children with a higher abnormal EEG theta/alpha ratio in some lead. It is because of the limited number of published studies with this population and the lack of a randomized assignment to a control condition internal to the study. We studied a placebo Control Group; however, the assignment of the children to any group was not randomized. In the previous study, 10 children were assigned to the Control or Experimental Groups in a way that, on average, no significant differences existed in age, sex, IQ, ADHD score, *per capita* income in the family, or mother schooling (Fernández et al. 2003). In the present study, six new children were added to the Experimental Group. Thus, it is neither a randomized nor a paired study. For this reason, the present paper constitutes an exploratory study; this clearly limits any conclusions based on the study.

In the present study, sample sizes were relatively small. We selected children by imposing an important number of restrictions. In the first place, children were LD with no other comorbidity such as ADHD. Therefore, the results of the study cannot be generalized to the whole LD population; however, these restrictions allowed a more profound study of Learning Disability *per se*. Another advantage of this strict selection of subjects was that samples were homogeneous, thus rendering a small variability in the data.

Although it is often suggested that the best evidence in clinical assessment arises from randomized double blind studies that includes a control group of patients with the same symptoms (Vernon et al. 2004), in NFB studies this type of experimental design is not readily found, probably because for most of the pathologies some effective standard treatment exists. Since no effective treatment exists for LD children, and according to the ethical principles guiding human research expressed in The World Medical Association Declaration of Helsinki (2004), it is possible to consider a group of placebo-treated LD children as control, in order to demonstrate that if changes in EEG, behavior, and cognition occur in children who received NFB, they were produced by the treatment rather than by non-controlled variables, such as increased attention or care given by parents, teachers, and experimenters.

In the Control Group one girl increased her IQ, ADHD score, and academic performance; we do not have any explanation for this improvement. In two other children of this group parents reported an improvement in behavior that was not evident in TOVA and WISC-R; in these cases we suppose it was a placebo effect.

Although no statistically significant differences between before and after treatment were found, in this control group a clear deterioration in TOVA (80%) was observed. This fact is difficult to explain; one possible reason is that the deterioration was induced by the sham treatment: the

randomization of the tone, implies that children cannot detect any coincidence between the tone and their physiological state; therefore, the tone is not a meaningful signal; it becomes noise for the system, producing a deterioration in cognitive performance; this explanation has been offered for autistic children (Rubenstein and Merzenich 2003).

In contrast, in the Experimental Group there were significant positive statistical differences between before and after treatment in WISC-R and in TOVA when the multivariate non-parametric permutational test was performed. In TOVA, the ANOVA-RM analysis for group and condition showed a significant result for the interaction, which means that the differences in ADHD score before versus after NFB were due to the different treatments. Improvements higher than the standard error in IQ were observed in 54.5% of the children, and by more than one point in TOVA were observed in 60% of the children. One-hundred percent of the children improved in one or both tests. The other evaluation used was the parent interview; in the Experimental Group all parents reported an improvement after treatment; however, as in the Control Group, it seemed that the placebo effect was present; therefore, we consider this interview of limited value, except when an objective change in academic performance was observed (10 out of 11 children in the Experimental Group, and 1 out of 5 children in the Control Group showed academic improvement). The duration of the treatment was around 10–12 weeks. In this period it is not possible to evaluate changes in the LD symptoms. A subgroup of children, the 10 children studied earlier (Fernández et al. 2003) were re-evaluated 2 years after treatment; in this follow-up study, all children of the Control Group continued presenting LD, but 4 out of 5 children in the Experimental Group had a normal diagnosis (Becerra et al. 2006). In other pathologies, such as ADHD (Lubar 1991) or epilepsy (Sterman 2000), around 80% of the subjects had significant positive changes with the NFB treatment. Due to the limitations in the present study we cannot reach firm conclusions about the percentage of children in whom NFB produces positive changes.

Other studies that included subjects with ADHD and LD have also shown positive findings (Linden et al. 1996; Lubar et al. 1995a, b; Tansey 1991, 1993). In these studies the NFB protocol was different from the theta/alpha ratio used in the present work, since EEG abnormalities of the ADHD children are different from those found in LD children, both in frequency and in topography.

The theta/alpha ratio has demonstrated to be a useful measure for characterizing EEG abnormalities in children (Matousek and Petersén 1973). It has several advantages: first, this quotient eliminates the major artifact sources in EEG recordings (eye movements and muscular activity);

second, the use of this quotient (and any other AP quotient) eliminates approximately 40% of EEG AP interindividual variability (Fernández et al. 1995; Valdés et al. 1992).

In clinical practice, most NFB treatments include 40–60 sessions. Rossiter and La Vaque (1995) demonstrated that 20 sessions of an NFB program significantly reduce the cognitive and behavioral symptoms of ADHD; we have demonstrated that the same occurs in LD children with abnormal values of the theta/alpha ratio (Fernández et al. 2003). These results may be explained by the theory of operant conditioning in relation to the characteristics of the reinforcer given: learning is more efficient if both the stimulus used for reinforcement and the instructions given to the subjects are simple (Stevenson and Wright 1966). On the other hand, it should be not forgotten that LD children present perceptual deficits (Neville et al. 1993), and that a complex stimulus may prolong the time required for its analysis, thus reducing the stimulus efficiency to induce NFB.

The present work follows almost all functional characteristics required for effective operant conditioning recommended by Sterman and Egner (2006). An important feature of the present experiment is that reinforcement was not contingent upon every response (reduction of the theta/alpha ratio); rather, the reinforcer was given intermittently (only between 60% and 80% of the time). It is well established that intermittent reinforcement produces greater resistance to extinction than continuous reinforcement (Hilgard and Marquis 1940). Moreover, it may have been an important factor in maintaining positive behavioral and cognitive changes associated with NFB, although further research is needed to validate this proposition.

In the present work, the placebo treatment had no effect on the current sources when comparing between before and immediately after treatment or before and 2 months after treatment conditions. In contrast, although few changes were observed immediately after NFB in the Experimental Group, 2 months later numerous EEG current source changes were found in this group. These changes consisted principally of a current decrease in frequencies within the theta band, mainly in left frontal regions and cingulate region, and a current increase in frequencies within the alpha band, principally in the right temporal lobe and right frontal regions, and in frequencies within the beta band, mainly in left temporal regions, right frontal regions, and cingulate cortex.

Changes in EEG current sources were more prominent 2 months later than immediately after NFB treatment; however, behavioral improvement was observed immediately after treatment. These results are consistent with NFB producing a primary change in subcortical structures, which is reflected immediately in behavior, but not in postsynaptic cortical activity. It must be kept in mind that

97% of the cerebral activity recorded as EEG originates within the cortex (Nunez 1995; Thatcher et al. 1986). The thalamus is a reasonable candidate to be the locus of the early changes in activity produced by NFB (Sterman 1996). These functional changes may later modify the EEG through modulation of thalamo-cortical circuits (Steriade et al 1990; Sterman 1996). This theory is also supported by the fact that the NFB effect on EEG is generalized: the changes are not limited to the region in which NFB was applied or to the frequencies used in the treatment. Thus, EEG changes seem to be the result of a complex reorganization of EEG activity. Though one possible candidate to produce such complex changes is the thalamus; it is a closed-field structure, and its electrical activity can neither be observed in scalp EEG nor in its current sources.

It is important to point out that all children in the Experimental Group learned to reduce the theta/alpha ratio during the NFB sessions; however, in the EEGs recorded at rest before and immediately after NFB treatment, a reduction of the theta/alpha ratio was observed only in four of them. Two months later the theta/alpha ratio reduction was observed in 7 out of 11 children. One possible explanation is that during NFB training the reinforcement produces activation of thalamo-cortical loops that decrease the theta/alpha ratio; whereas at rest, when there is no reinforcement, the thalamo-cortical loops are not so active and changes in the scalp EEG are not well detected. The enhancement in the theta/alpha ratio that occurred 2 months later could be interpreted as delayed increase in memory; however, we think this effect was due to the EEG reorganization mechanism mentioned above.

A significant increase in alpha and beta activity was found in the right frontal lobe, which has been related to alertness, an essential condition to maintain the tonic state of attention (Posner et al. 2006). In a previous paper, significant differences between normal and LD children were observed in EEG current sources (Fernández et al. 2002). A major dissimilarity between LD and normal children was that LD children had more current in frequencies within the theta band in anterior regions. In the present study, a current decrease in theta frequencies was found in frontal regions as a consequence of the NFB treatment, which can be interpreted as a normalization of EEG current sources in the frontal lobes.

Other relevant changes in EEG current sources were found in theta (reduction) and beta (increase) activity in the cingulate cortex. According to Posner et al. (2006), this structure is associated with the executive attention network, which is activated when stimulus characteristics compete for control of the output in conflict situations, as in TOVA, and it regulates the activity in other brain networks involved in thought and emotion. Using functional magnetic resonance imaging, Levesque et al. (2006)

demonstrated in a controlled study that only ADHD children who received NFB showed (after treatment) activation of the anterior cingulate cortex during the Counting Stroop task. Congedo (2004) reported improvements in the performance on a sustained attention task in subjects who received a NFB treatment consisting of enhancement of the current density power ratio between beta and alpha frequency bands in the cingulate cortex, using low resolution electromagnetic tomography (LORETA).

Changes observed in EEG current sources may explain the improvement children experienced in their behavior, principally in their cognitive activities. In relation to our main goal, the evaluation of NFB effect on EEG current sources, we may conclude that the results suggest that EEG current source analysis is a valuable tool for the understanding of the neurophysiological bases of NFB. Notwithstanding the limitations of our study, mainly the small sample size and the non-random assignment of the children, it was clearly shown that, in LD children with an abnormal theta/alpha ratio, improvement in this ratio normalizes EEG oscillations in cerebral regions that may, in turn, explain the positive behavioral changes produced by NFB.

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## References

- Abarbanel, A. (1999). The neural underpinnings of neurofeedback training. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to Quantitative EEG, Neurofeedback* (pp. 311–340). New York: Academic Press.
- Alvarez, A., Pérez-Avalo, M. C., & Morenza, L. (1992). Neuropsychological assessment of learning-disorder children with paroxysmal EEG activity. *New Issues in Neurosciences*, *IV*, 40–50.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (4th ed.)*. Washington, DC, pp. 179–286.
- Baehr, E., Rosenfeld, J. P., Baehr, R., & Earnest, C. (1999). Clinical use of an alpha asymmetry neurofeedback protocol in the treatment of Mood Disorders. In J.R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG, neurofeedback* (pp. 181–203). New York: Academic Press.
- Beauregard, M., & Levesque, J. (2006). Functional magnetic resonance imaging investigation of the effects of neurofeedback training on the neural bases of selective attention and response inhibition in children with attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback*, *31*, 3–20.
- Becerra, J., Fernández, T., Harmony, T., Caballero, M. I., García, F., Fernández-Bouzas, A., Santiago-Rodríguez, E., & Prado-Alcalá, R. A. (2006). Follow-up study of Learning-Disabled Children treated with Neurofeedback or Placebo. *Clinical EEG and Neuroscience*, *37*, 198–203.
- Bernal, J., Harmony, T., Rodríguez, M., Reyes, A., Yáñez, G., Fernández, T., Galán, L., Silva, J., Fernández-Bouzas, A., Rodríguez, H., Guerrero, V., & Marosi, E. (2000). Auditory event related potentials in poor readers. *International Journal of Psychophysiology*, *36*, 11–23.
- Blair, R. C., & Karninski, W. (1993). An alternative method for significance testing of waveform difference potential. *Psychophysiology*, *30*, 518–524.
- Blair, R. C., & Karninski, W. (1994). Distribution-free statistical analyses of surface and volumetric maps. In R. W. Thatcher, M. Hallett, E. R. John, & M. Huerta (Eds.), *Functional neuroimaging* (pp. 19–28). New York: Academic Press.
- Bosch-Bayard, J., Valdés-Sosa, P., Virués-Alba, T., Aubert-Vázquez, E., John, E. R., Harmony, T., Riera-Díaz, J., & Trujillo-Barreto, N. (2001). 3D statistical parametric mapping of EEG source spectra by means of variable resolution electromagnetic tomography (VARETA). *Clinical Electroencephalography*, *32*, 47–61.
- Butnik, S. M. (2005). Neurofeedback in adolescents and adults with attention deficit hyperactivity disorder. *Journal of Clinical Psychology*, *61*, 621–625.
- Chabot, R. J., di Michele, F., & John, E. R. (2001). The clinical role of computerized EEG in the evaluation and treatment of learning and attention disorders in children and adolescents. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *13*, 171–186.
- Collins, D. L., Neelin, P., Peter, T. M., & Evans, A. C. (1994). Automatic 3D registration of MR volumetric data in standardized Talairach space. *Journal of Computer Assisted Tomography*, *18*, 192–205.
- Congedo, M., Lubar, J. F., & Joffe, D. (2004). Low-resolution electromagnetic tomography Neurofeedback. *IEEE Transactions on Neural Systems and Rehabilitation Engineering*, *12*, 387–397.
- Egner, T., & Serman, M. B. (2006). Neurofeedback treatment of epilepsy: from basic rationale to practical application. *Expert Review of Neurotherapeutics*, *6*, 247–257.
- Evans, A. C., Collins, D. L., Mills, S. R., Brown, E. D., Kelly, R. L., & Peters, T. M. (1993). 3D statistical neuroanatomical models from 305 MRI volumes. *Proceedings of the IEEE-Nuclear Science Symposium and Medical Imaging Conference*, pp. 1813–1817.
- Evans, A. C., Collins, D. L., Neelin, P., MacDonald, D., Kamber, M., & Marret, T. S. (1994). Three-dimensional correlative imaging. Applications in human brain mapping. In R. W. Thatcher, M. Hallett, T. Zeffiro, E. R. John, & M. Huerta (Eds.), *Functional neuroimaging* (pp. 145–162). New York: Academic Press.
- Fernández, T., Harmony, T., Rodríguez, M., Bernal, J., Silva, J., Reyes, A., & Marosi, E. (1995). EEG activation patterns during the performance of tasks involving different components of mental calculation. *Electroencephalography and Clinical Neurophysiology*, *94*, 175–182.
- Fernández, T., Harmony, T., Silva, J., Galán, L., Díaz-Comas, L., Bosch, J., Rodríguez, M., Fernández-Bouzas, A., Yáñez, G., Otero, G., & Marosi, E. (1998). Relationship of specific EEG frequencies at specific brain areas with performance. *NeuroReport*, *9*, 3681–3687.
- Fernández, T., Harmony, T., Silva-Pereyra, J., Fernández-Bouzas, A., Gershenowies, J., Galán, L., Carbonell, F., Marosi, E., Otero, G., & Valdés, S. (2000). Specific EEG frequencies at specific brain areas and performance. *NeuroReport*, *11*, 2663–2668.
- Fernández, T., Harmony, T., Fernández-Bouzas, A., Silva, J., Herrera, W., Santiago-Rodríguez, E., & Sánchez, L. (2002). Sources of EEG activity in learning disabled children. *Clinical Electroencephalography*, *33*, 160–164.

- Fernández, T., Herrera, W., Harmony, T., Díaz-Comas, L., Santiago, E., Sánchez, L., Bosch, J., Fernández-Bouzas, A., Otero, G., Ricardo-Garcell, J., Barraza, C., Aubert, E., Galán, L., & Valdés, P. (2003). EEG and behavioral changes following neurofeedback treatment in Learning Disabled children. *Clinical Electroencephalography*, *43*, 145–152.
- Fernández-Bouzas, A., Harmony, T., Bosch, J., Aubert, E., Fernández, T., Valdés, P., Silva, J., Marosi, E., Martínez-López, M., & Casián, G. (1999). Sources of abnormal EEG activity in the presence of brain lesions. *Clinical Electroencephalography*, *30*, 46–52.
- Fernández-Bouzas, A., Harmony, T., Fernández, T., Silva-Pereyra, J., Valdés, P., Bosch, J., Aubert, E., Casián, G., Otero-Ojeda, G., Ricardo, J., Hernández-Ballesteros, A., & Santiago, E. (2000). Sources of abnormal EEG activity in brain infarctions. *Clinical Electroencephalography*, *31*, 165–169.
- Fox, D. J., Tharp, D. F., & Fox, L. C. (2005). Neurofeedback: An alternative and efficacious treatment for Attention Deficit Hyperactivity Disorder. *Applied Psychophysiology and Biofeedback*, *30*, 365–373.
- Fuchs, T., Birbaumer, N., Lutzenberger, W., Gruzelier, J. H., & Kaiser, J. (2003). Neurofeedback treatment for Attention-Deficit/Hyperactivity Disorder in children: A comparison with methylphenidate. *Applied Psychophysiology and Biofeedback*, *28*, 1–12.
- Galán, L., Biscay, R., Rodríguez, J. L., Pérez-Avalo, M.C., & Rodríguez, R. (1997). Testing topographic differences between event related brain potentials by using non-parametric combinations of permutation tests. *Electroencephalography and Clinical Neurophysiology*, *102*, 240–247.
- Gasser, T., Rousson, V., & Scheiter Gasser, U. (2003). EEG power and coherence in children with educational problems. *Clinical Neurophysiology*, *20*, 273–282.
- Goldstein, L. H. (1997). Effectiveness of psychological interventions for people with poorly controlled epilepsy. *Journal of Neurology, Neurosurgery and Psychiatry*, *63*, 137–142.
- Hammond, D.C. (2005). Neurofeedback with anxiety and affective disorders. *Child and Adolescent psychiatric clinics of North America*, *14*, 105–123.
- Harmony, T., Marosi, E., Díaz de León, A. E., Becker, J., & Fernández, T. (1990a). Effect of sex, psychosocial disadvantages and biological risk factors on EEG maturation. *Electroencephalography and clinical Neurophysiology*, *75*, 482–491.
- Harmony, T., Hinojosa, G., Marosi, E., Becker, J., Fernández, T., Rodríguez, M., Reyes, A., & Rocha, C. (1990b). Correlation between EEG spectral parameters and an educational evaluation. *International Journal of Neuroscience*, *54*, 147–155.
- Harmony, T., Fernández, T., Silva, J., Bosch, J., Valdés, P., Fernández-Bouzas, A., Galán, L., Aubert, E., & Rodríguez, D. (1999). Do specific EEG frequencies indicate different processes during mental calculation? *Neuroscience Letters*, *266*, 25–28.
- Harmony, T., Fernández, T., Fernández-Bouzas, A., Silva-Pereyra, J., Bosch, J., Díaz-Comas, L., & Galán, L. (2001). EEG changes during word and figure categorization. *Clinical Neurophysiology*, *112*, 1486–1498.
- Harmony, T., Fernández, T., Gershenowies, J., Galán, L., Fernández-Bouzas, A., Aubert, E., & Díaz-Comas, L. (2004). Specific EEG frequencies signal general common cognitive processes as well as specific task processes in man. *International Journal of Psychophysiology*, *53*, 207–216.
- Hernández, J. L., Valdés, P., Biscay, R., Virués, T., Szava, S., Bosch, J., Riquenes, A., & Clark, I. (1994). A global scale factor in brain topography. *International Journal of Neuroscience*, *76*, 267–278.
- Hilgard, E. R., & Marquis, D. G. (1940). *Conditioning and learning*. Appleton-Century-Crofts, New York.
- Holcomb, P. J., Ackerman, P. T., & Dyckman, R. A. (1986). Auditory event-related potentials in attention and reading disabled boys. *International Journal of Psychophysiology*, *3*, 263–273.
- Iglesias, A., & Derman, B. (1985). *Prueba de lecto-escritura*. México D.F.: Progreso.
- Iragui, V. J., Kutas, K., Mitchiner, M. R., & Hillyard, S. A. (1993). Effects of aging on event-related brain potentials and reaction times in an auditory oddball task. *Psychophysiology*, *30*, 10–22.
- John, E. R., Prichep, L., Ahn, H., Easton, P., Fridman, J., & Kaye, H. (1983). Neurometric evaluation of cognitive dysfunctions and neurological disorders in children. *Progress in Neurobiology*, *21*, 239–290.
- Kelley, M. (1997). Native americans, neurofeedback, and substance abuse theory. *Journal of Neurotherapy*, *3*, 45–52.
- Kutas, M., & Dale, A. (1997). Electrical and magnetic readings of mental functions. In M. D. Rugg (Ed.), *Cognitive neuroscience* (pp 197–237). London: University College Press.
- La Vaque, T. J., Hammond, D. C., Trudeau, D., Monastra, V. J., Perry, J., & Lehrer, P. (2002). Template for developing guidelines for the evaluation of the clinical efficacy of psychophysiological interventions. *Applied Psychophysiology and Biofeedback*, *27*, 273–281.
- Leark, R. A., Dupuy, T. R., Greenberg, L. M., Corman, C. L., & Kindschi, C. L. (1999). *T.O.V.A. professional guide*. Los Alamitos: Universal Attention Disorders Inc.
- Leins U., Goth G., Hinterberger T., Klinger C., Rumpf N., & Strehl U. (2007). Neurofeedback for children with ADHD a comparison of SCP and theta/beta protocols. *Applied Psychophysiology and Biofeedback*, *32*, 73–88.
- Levesque, J., Beauregard, M., & Mensour, B. (2006). Effect of Neurofeedback training on the neural substrates of selective attention in children with attention-deficit/hyperactivity disorder: A functional magnetic resonance imaging study. *Neuroscience Letters*, *394*, 216–221.
- Linden, M., Habib, T., & Radojevic, V. (1996). A controlled study of the effects of EEG biofeedback on cognition and behavior of children with Attention Deficit Disorder and Learning Disabilities. *Biofeedback and Self-regulation*, *21*, 35–49.
- Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Self-regulation*, *16*, 201–225.
- Lubar, J. F., & Bahler, W.W. (1976). Behavioral management of epileptic seizures following EEG biofeedback training of the sensorimotor rhythm. *Biofeedback and Self-regulation*, *1*, 77–104.
- Lubar, J. F., Bianchini, K. J., Calhoun, W. H., Lambert, E. W., Brody, Z. H., & Shabsin, H. S. (1985). Spectral analysis of EEG differences between children with and without learning disabilities. *Journal of Learning Disabilities*, *18*, 403–408.
- Lubar, J. F., & Lubar, J. O. (1999). Neurofeedback assessment and treatment for Attention Deficit/Hyperactivity Disorders. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp 103–146). New York: Academic Press.
- Lubar, J. F., Startwood, M. O., Startwood, J. N., & O'Donnell, P. H. (1995a). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in TOVA scores, behavioral ratings, and WISC-R performance. *Biofeedback and Self-regulation*, *20*, 83–99.
- Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. H. (1995b). Neurofeedback for the management of Attention Deficit/Hyperactivity Disorders. In M.S. Schwartz (Ed.), *Biofeedback: A practitioner's Guide* (pp. 493–522). New York: Guilford Press.
- Matousek, M., & Petersén, I. (1973). Frequency analysis of the EEG in normal children and adolescents. In P. Kellaway & I. Petersén

- (Eds.), *Automation of clinical electroencephalography* (pp. 75–102). New York: Raven Press.
- Mazziotta, J. C., Toga, A., Evans, A. C., Fox, P., & Lancaster, J. (1995). A probabilistic atlas of the human brain: theory and rationale for its development. *NeuroImage*, 2, 89–101.
- Monastra, V. J., Lynn, S., Linden, M., Lubar, J. F., Gruzelier, J., & La Vaque, T. J. (2005). Electroencephalographic biofeedback in the treatment of Attention-Deficit/Hyperactivity Disorder. *Applied Psychophysiology and Biofeedback*, 30, 95–114.
- Monastra, V. J., Monastra, D. M., & George, S. (2002). The effects of stimulant therapy, EEG biofeedback and parenting style on the primary symptoms of Attention-Deficit/Hyperactivity Disorder. *Applied Psychophysiology and Biofeedback*, 27, 231–249.
- Moore, N. C. (2000). A review of EEG biofeedback for anxiety disorders. *Clinical Electroencephalography*, 31, 1–6.
- Nash, J. K. (2000). Treatment of Attention Deficit Hyperactivity Disorder with neurotherapy. *Clinical Electroencephalography*, 31, 30–37.
- Neville, H., Coffey, S. A., Holcomb, P., Tallal, P. (1993). The neurobiology of sensory and language processing in language-impaired children. *Journal of Cognitive Neuroscience*, 5, 235–253.
- Nunez, P. L. (1995). Toward a physics of neocortex. In P.L. Nunez (Ed.), *Neocortical dynamics and Human EEG rhythms*. New York: Oxford University Press.
- Penberthy, J. K., Cox, D., Breton, M., Robeva, R., Kalbfleisch, M. L., Loboschewski, T., & Kovatchev, B. (2005). Calibration of ADHD assessments across studies: a meta-analysis tool. *Applied Psychophysiology and Biofeedback*, 30, 31–51.
- Peniston, E. G., & Kulkosky, P. J. (1999). Neurofeedback in the treatment of addictive disorders. In J. R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback* (pp. 157–180). New York: Academic Press.
- Ponsford, J. L., & Kinsella, G. (1998). Evaluation of a remedial program for attentional deficits following closed-head injury. *Journal of Clinical Experimental Neuropsychology*, 10, 693–708.
- Pop-Jordanova, N., Markovska-Simoska, S., & Zorcec, T. (2005). Neurofeedback treatment of children with attention deficit hyperactivity disorder. *Prilozi*, 26, 71–80.
- Posner, M. I., Sheese, B. E., Odludas, Y., & Tang, Y. (2006). Analyzing and shaping human attentional networks. *Neural Networks*, 19, 1422–1429.
- Riera, J., Fuentes, M. E., Aubert, E., & Díaz, D. (1997). Solving the forward problem: Spherical vs. realistic electric lead field. *Biomedizinische Technik*, 42, 223–226.
- Rosenfeld, J. P. (2000). An EEG biofeedback protocol for affective disorders. *Clinical Electroencephalography*, 31, 7–12.
- Rossiter, T. R., & La Vaque, T. J. (1995). A comparison of EEG biofeedback and psychostimulants in treating Attention Deficit/Hyperactivity Disorders. *Journal of Neurotherapy*, 3, 48–59.
- Rossiter, T. (2004a). The effectiveness of Neurofeedback and stimulant drugs in treating AD/HD: Part I. Review of methodological issues. *Applied Psychophysiology and Biofeedback*, 29, 95–112.
- Rossiter, T. (2004b). The effectiveness of Neurofeedback and stimulant drugs in treating AD/HD: Part II. Replication. *Applied Psychophysiology and Biofeedback*, 29, 233–243.
- Rubenstein, J. L., & Merzenich, M. M. (2003). Model of autism: Increased ratio of excitation/inhibition in key neural systems. *Genes, Brain and Behavior*, 2, 255–267.
- Schatz, A. M., Ballantyne, A. O., & Trauner, D. A. (2001). Sensitivity and specificity of a computerized test of attention in the diagnosis of Attention-Deficit/Hyperactivity Disorder. *Assessment*, 8, 357–365.
- Scherg, M., & Von Cramon, D. (1985). Two bilateral sources of the late AEP as identified by a spatio-temporal dipole model. *Electroencephalography and Clinical Neurophysiology*, 62, 32–44.
- Seifert, A. R., & Lubar, J. F. (1975). Reduction of epileptic seizures through EEG biofeedback training. *Biological Psychology*, 3, 157–184.
- Silva-Pereyra, J., Rivera-Gaxiola, M., Fernández, T., Díaz-Comas, L., Harmony, T., Fernández-Bouzas, A., Rodríguez, M., Bernal, J., & Marosi, E. (2003). Are poor readers semantically challenged? An event-related brain potential assessment. *International Journal of Psychophysiology*, 49, 187–199.
- Stamatina, S., & Lubar, J. F. (2004). EEG changes in traumatic brain injured patients after cognitive rehabilitation. *Journal of Neurotherapy*, 8, 21–51.
- Steriade, M., Gloor, P., Llinás, R. R., Lopes da Silva, F. H., & Mesulam, M. M. (1990). Basic mechanisms of cerebral rhythmic activities. *Electroencephalography and Clinical Neurophysiology*, 76, 481–508.
- Sterman, M. B. (1996). Physiological origins and functional correlates of EEG rhythmic activities: implications for self-regulation. *Biofeedback and Self-regulation*, 21, 3–33.
- Sterman, M. B. (2000). Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Clinical Electroencephalography*, 31, 45–55.
- Sterman, M. B., & Eger, T. (2006). Foundation and practice of neurofeedback for the treatment of epilepsy. *Applied Psychophysiology and Biofeedback*, 31, 21–35.
- Sterman, M. B., & Friar, L. (1972). Suppression of seizures in an epileptic following sensorimotor EEG feedback training. *Electroencephalography and Clinical Neurophysiology*, 33, 89–95.
- Stevenson, H. W., & Wright, J. C. (1966). Child psychology. In J. B. Sidowski (Ed.), *Experimental methods and instrumentation in Psychology* (pp. 577–605). New York: McGraw Hill.
- Tansey, M. A. (1991). Wechsler (WISC-R) changes following treatment of learning disabilities via EEG biofeedback training in a private practice setting. *Australian Journal of Psychology*, 43, 147–153.
- Tansey, M. A. (1993). Ten-year stability of EEG biofeedback results for a hyperactive boy who failed fourth grade perceptually impaired class. *Biofeedback and Self-regulation*, 18, 33–44.
- Thatcher, R. W., Krause, P. J., & Hrybyk, M. (1986). Cortico-cortical associations and EEG coherence: A two-compartmental model. *Electroencephalography and Clinical Neurophysiology*, 64, 123–143.
- The World Medical Association Declaration of Helsinki. (2004). *Ethical Principles for medical research involving human subjects*. Note or clarification on paragraph 30 added by the WMA General Assembly, Tokio.
- Thompson, L., & Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: effectiveness in students with ADD. *Applied Psychophysiology and Biofeedback*, 23, 243–263.
- Tinius, T. P., & Tinius, K. A. (2000). Changes after EEG biofeedback and cognitive retraining in adults with mild traumatic brain injury and Attention Deficit Hyperactivity Disorder. *Journal of Neurotherapy*, 4, 27–44.
- Trudeau, D. L. (2000). The treatment of addictive disorders by brain wave biofeedback: A review and suggestions for future research. *Clinical Electroencephalography*, 31, 13–22.
- Valdés, P., Biscay, R., Galán, L., Bosch, J., Zsava, S., & Virués, T. (1990). High resolution spectral EEG norms topography. *Brain Topography*, 3, 281–282.
- Valdés, P., Bosch, J., Grave, R., Hernández, J., Riera, J., Pascual, R., & Biscay, R. (1992). Frequency domain models of the EEG. *Brain Topography*, 4, 309–319.
- Valdés, P., Riera, J., & Casanova, R. (1996). Spatiotemporal distributed inverse solutions. In C. Wood (Ed.), *Proceedings of*

*the Tenth International Conference on Biomagnetism BIO-MAG'96*, Santa Fe NM.

Vernon, D., Frick, A., & Gruzelier, J. (2004). [Neurofeedback as a treatment for ADHD: A methodological review with implications for future research.](#) *Journal of Neurotherapy*, 8, 53–82.

Walker, J. E., & Kozlowski, G. P. (2005). Neurofeedback treatment of epilepsy. *Child and Adolescent psychiatric clinics of North America*, 14, 163–176.

Wechsler, D. (1981). *Manual WISC-R Español. Escala de Inteligencia Revisada para el nivel escolar*. México, D.F.: Manual Moderno.