

Improvements in Spelling after QEEG-based Neurofeedback in Dyslexia: A Randomized Controlled Treatment Study

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Abstract Phonological theories of dyslexia assume a specific deficit in representation, storage and recall of phonemes. Various brain imaging techniques, including qEEG, point to the importance of a range of areas, predominantly the left hemispheric temporal areas. This study attempted to reduce reading and spelling deficits in children who are dyslexic by means of neurofeedback training based on neurophysiological differences between the participants and gender and age matched controls. Nineteen children were randomized into an experimental group receiving qEEG based neurofeedback ($n = 10$) and a control group ($n = 9$). Both groups also received remedial teaching. The experimental group improved considerably in spelling (Cohen's $d = 3$). No improvement was found in reading. An indepth study of the changes in the qEEG power and coherence protocols evidenced no fronto-central changes, which is in line with the absence of reading improvements. A significant increase of alpha coherence was found, which may be an indication that attentional processes account for the improvement in spelling. Consideration of subtypes of dyslexia may refine the results of future studies.

Keywords Spelling · QEEG-based neurofeedback · Dyslexia · Treatment · Reading problems

Introduction

About 9% of children leaving primary education in the Netherlands are considered to have severe reading and spelling problems (Blomert 2005). An estimated 40% of this occurrence is due to dyslexia. This group functions comparable to other children on ability tests, with a specific exception: word reading and spelling skills. International studies suggest that 5–10% of children in western societies suffer from dyslexia (Walker and Norman 2006). Various authors (Goswami 2003; Habib 2000; Ramus et al. 2003) have postulated three neurocognitive deficits that are involved in dyslexia: the phonological theory, referring to a specific deficit in representation, storage and recall of phonemes; the magnocellular theory, suggesting a deficit in the magnocells of the primary visual area, and the cerebellar theory, based on the idea of a lesion in the cerebellum, leading to automatization deficits. Many imaging techniques (fMRI, PET, Stimulation techniques, MEG) have pointed to differences in functioning between dyslectics and normals in support of these theories. For a selective review see Walker and Norman (2006). This study limits itself to the first theory that has most evidence in support of it and is more applicable to neurofeedback as well.

Various areas that have been found to be involved in dyslexia will be considered below, along with the locations according to the 10–20 system. Angelakis et al. (1999) point to the association of various areas and specific linguistic abilities. F7 activity shows primary activation of a phonological task; P3 and P4 are involved in semantic and mathematical tasks, and T5 and T6 in semantic tasks.

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Klimesch et al. (2001) found children with dyslexia not to desynchronize their Beta-1 activity during a reading task in areas related to Broca's Area (FC5; speech production, articulation) and the Angular Gyrus (CP5, P3; understanding semantic and mathematical). This is in accordance with Rippon and Brunswick (2000), who found the smallest task-no task difference in beta activity in the left parieto-occipital area. Simos et al. (2002) made use of fMRI in order to test the processes involved in a successful intervention in dyslexia. In line with the phonological theory, they found regions of the left superior temporal gyrus (T3) to be recruited more often after treatment than before. Thornton and Carmody (2005) concluded likewise after a review that the left temporal region is disrupted. They suggest that the disruption is in place before children learn to read, and that it is related to underdevelopment of white matter in the area. In a clinical vignette they show the great variety in patterns of deviations from normal functioning, including coherence deviations. Walker and Norman (2006) once more point to the importance of the left temporal-parietal cortex, involving among others the angular gyrus (between T5 and P3).

However, other areas have also been reported to be dysfunctional in dyslexia. Ackerman and Dykman (1995) and Flynn et al. (1992) found beta in the right parietal and occipital areas to be decreased during reading in individuals who were dyslectic. Arns et al. (2007) found that children with dyslexia exhibited increased slow EEG (Delta and theta) activity in the frontal and right temporal regions of the brain, increased beta-1 at F7 and increased EEG coherence in frontal, central and temporal regions. Coherence for the lower frequency bands (Delta and Theta) was symmetrically increased and coherence for the higher frequencies (Alpha and Beta) showed a specific right-temporocentral distribution.

In spite of the many associations established, now it is not possible to diagnose dyslexia based on neurophysiological assessment. Yet intervention techniques like neurofeedback may profit from the knowledge collected thus far, by addressing specific frequencies at specific locations or by attempting to normalize deviant coherence. Thornton and Carmody (2005) report the lack of any neurofeedback efficacy study directed at reading disability. They report on four cases, suggesting effects far beyond those of classical remediation and rehabilitation programs. Walker and Norman (2006) illustrated a task-related approach, making use of what they call "reading difference topography." While reading, brain dynamics change in people who experience dyslexia and those who do not. Subtraction of the QEEG while reading from qEEG at rest shows the difference in the two states, which then can be compared to the differences in normals. Based on this approach Walker and Norman reported an average increase of at least two grade levels of reading speed

and comprehension in 12 children with dyslexia. In the present study, we report on a randomized controlled trial in which the effects of QEEG neurofeedback training as an additional intervention to linguistic education were determined in 19 children from the Netherlands who had dyslexia. The following research questions are addressed:

1. Does neurofeedback training improve reading and spelling abilities of dyslexic clients?
2. Does neurofeedback training lead to changes in the QEEG?
3. Are the QEEG changes related to changes in reading and spelling?

Methods

Participants

Nineteen children (11 males and 8 females; average age = 10.33; range 8.0–15.98), who were diagnosed with dyslexia by their remedial teachers, participated in this study. Diagnosis was based on a structured protocol assessing the reading and spelling development of the children from grade 1 (Wentink and Verhoeven 2003). The remedial teachers selected these children, who all were attending regular schools.

Exclusion criteria included a personal or family history of mental illness, brain injury, neurological disorder, serious medical condition, drug/alcohol addiction; and a family history of genetic disorder. All subjects voluntarily gave written informed consent or assent.

Procedure

All participants filled out questionnaires, were tested before and after neurofeedback training on their reading and spelling ability, and received neuropsychological tests and a QEEG assessment.

Neurofeedback protocols were based on the outcome of the qEEG assessment. In addition, coherences were checked and compared to the outcome of Arns et al. (2007). Treatment protocols were directed at abnormal functioning according to the following decision rules: (1) increased slow activity (delta) differing more than 1.5 Z-scores from the norm at T6; (2) increased coherence in the alpha- or betaband at F7–FC3 or F7–C3 with $Z > 1.5$; (3) increased coherence at T3–T4 with $Z > 1.5$; (4) in case of clear indications for different training protocols these were incorporated into the protocol.

All participants were given 20 sessions of neurofeedback training during 10 weeks, irrespective of the phenomenology, severity or subtype of the dyslexia. During the neurofeedback

training both the experimental and the control group received additional counselling regarding reading and spelling, apart from the regular language curriculum.

Language Tests

The children were administered a range of tests to further investigate correlations between EEG and neuropsychological findings to sub-tests of dyslexia. The included tests were measures of tasks related to reading—rapid naming of letters, articulation, phoneme deletion (Instituut voor Orthopedagogiek 2004) and spelling (Geelhoed and Reitsma 1999).

Electroencephalographic Data Acquisition

Participants were seated in a sound and light attenuated room, controlled at an ambient temperature of 22°C. Participants were required to refrain from caffeine, alcohol and smoking for at least 2 h prior to testing. Electroencephalographic and neuropsychological assessments were completed in order. EEG data were acquired from 28 channels: Fp1, Fp2, F7, F3, Fz, F4, F8, FC3, FCz, FC4, T3, C3, Cz, C4, T4, CP3, CPz, CP4, T5, P3, Pz, P4, T6, O1, Oz and O2 (Quikcap; NuAmps; 10–20 electrode international system). Data were referenced to linked mastoids with a ground at AFz. Horizontal eye-movements were recorded with electrodes placed 1.5 cm lateral to the outer canthus of each eye. Vertical eye movements were recorded with electrodes placed 3 mm above the middle of the left eyebrow and 1.5 cm below the middle of the left bottom eyelid. Skin resistance was <5 K Ohms and above 1 K Ohm for all electrodes. A continuous acquisition system was employed and EEG data were EOG corrected offline (Gratton et al. 1983). The sampling rate of all channels was 500 Hz. A low pass filter with attenuation of 40 dB per decade above 100 Hz was employed prior to digitization.

The EEG data were recorded for 2 min during eyes open (EO). Subjects were asked to sit quietly. During EO subjects were asked to fix their eyes on a red dot presented on a computer screen.

Neuropsychology

Neuropsychological assessment was completed using a touch screen monitor. Measures included: memory recall and memory recognition (number of correctly reproduced words on trial 1, 5, 6, 7; number of correctly recognized words), word Interference test—equivalent to the Stroop test (Number correct text and color condition), tapping test (Number of taps with the dominant and non dominant hand), timing test (proportional bias) and Switching of

Attention test part A and B (equivalent to the WMS Trails A and B; time to complete the A and B form) (See Gordon et al. 2005, for details of these tests). All tests were fully computerized and the participants' responses were recorded via touch-screen presses. Reliability and validity data on these tasks are reported elsewhere (e.g. Clark et al. 2006; Gordon et al. 2005).

Statistical Analysis and Design

Missing Values

If missing values were present for a given statistical test, those cases were excluded for that analysis. The number of missing values per group is reported in the results sections.

Analysis

Electroencephalographic Variables

Average power spectra were computed for EO condition. Each 2-min epoch was divided into adjacent intervals of 4-s. Power spectral analysis was performed on each 4-s interval by first applying a Welch window to the data, and then performing a Fast Fourier Transform (FFT).

The electrical power was calculated in the following frequency bands delta (1.5–3.5 Hz), theta (4–7.5 Hz), alpha (8–13 Hz), alpha1 (8–11 Hz), alpha2 (11–13 Hz), SMR (12–15 Hz), beta (14.5–30 Hz), beta1 (14.5–20 Hz), beta2 (20–25 Hz) and beta3 (25–30 Hz). These data were then square-root transformed to approximate the normal distributional assumptions required by parametric statistical methods. Changes within and between groups were investigated.

This study used a randomized controlled pretest–posttest design. This allows for a multivariate repeated measures analysis of variance that was applied for a comparison of both groups with regard to reading and spelling. Due to the large number of tests, a Bonferroni correction was applied for achieving an overall alpha of .05. Paired *t*-tests were applied for exploring the differences between pre- and posttest scores. Lastly, associations between changes in reading tests, neuropsychological tests and qEEG variables of interest were explored using correlation analysis.

Results

Reading and Spelling Tests

Analysis of variance indicated main effects of the factor “time” for the variables CVC-words [$F = 11.1$ (1, 17),

$p = .004$], CC-words [$F = 8.5 (1, 17), p = .01$], double syllables [$F = 21.4 (1, 17), p = .000$]; triple syllables [$F = 23.7 (1, 17), p = .000$]; and CC-pseudo words [$F = 9.7 (1, 17), p = .006$]. No interaction effects were found, suggesting comparable changes in both the neurofeedback and control group (Table 1).

The spelling test also showed a main affect of time [$F = 10.2 (1, 17) p = .001$]. Additionally a significant interaction effect was found [$F = 4.5 (1, 17), p = .045$] suggesting a treatment effect of neurofeedback. The neurofeedback group progressed from $m = 69.1$ ($SD = 32.0$) to 80.6 ($SD = 32.2$) a 16.6% improvement, whereas the control group progressed from $m = 66.9$ ($SD = 20.9$) to $m = 70.9$ ($SD = 24.4$) a 6% improvement (Fig. 1). Cohen's d was 3.02, suggesting a large progress in spelling of the neurofeedback group (Becker 1998).

Neuropsychological Tests

The changes in scores on the neuropsychological tests of the experimental group were tested using the t -test for paired observations. Although at first glance a switching of attention subtest and a verbal interference subtest appeared to have improved, this change turned out to be due to outliers. Within-group correlations were non-significant, both for the switching of attention subtest ($r = .53, p = .18$) and the verbal interference subtest ($r = .45, p = .26$).

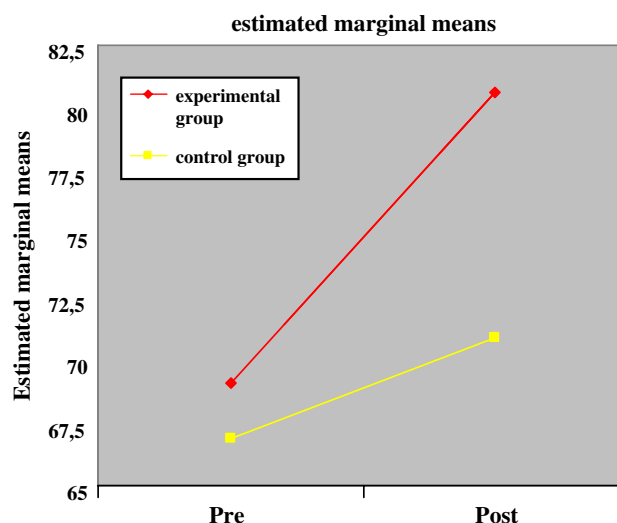


Fig. 1 Pre- and posttest scores on spelling test

EEG Power Spectra

Based on the decision rules mentioned earlier eight personalized power protocols were developed. Coherence training was given using five personalized protocols. The protocols used for each subject and the result in terms of power spectra and coherence are given in Table 2.

No changes in the delta frequency band were noted at any location. At Cz ($t = -2.65, p = .03$) and P3 ($t = -2.51,$

Table 1 Means and standard deviations of experimental and control group on tasks related to reading, rapid naming of letters, articulation, and phoneme deletion

Test	Experimental group				Control group			
	Pretest		Posttest		Pretest		Posttest	
	Score	SD	Score	SD	Score	SD	Score	SD
CVC words ^a	61.80	19.99	68.20	19.32	59.56	18.93	64.67	17.97
VV words ^a	46.80	17.90	50.30	17.84	43.44	20.09	46.78	19.31
2 Syllables ^a	30.00	15.17	33.50	15.44	28.78	12.84	33.78	14.05
3 Syllables ^a	22.70	14.31	25.70	13.58	21.44	10.14	25.67	11.64
RN digits	88.80	11.15	93.60	12.55	91.89	11.85	93.33	11.91
RN letters	89.00	13.33	95.50	9.28	93.67	14.25	93.67	14.00
RN pictures	58.70	8.17	67.00	15.84	57.67	12.16	62.11	14.34
CVC pseudo	81.70	27.85	83.40	26.11	72.33	22.73	78.22	24.22
VV pseudo ^a	50.60	17.96	56.90	18.44	49.22	17.99	50.44	20.24
Two letter pseudo	30.90	14.45	33.80	12.05	29.33	12.67	28.22	13.03
Three letter pseudo	20.60	10.67	22.60	10.82	21.22	11.44	21.67	10.06
Graphemes	24.30	6.99	21.50	3.95	23.78	6.57	23.33	3.64
Articulation	33.80	3.23	35.50	2.88	29.44	7.27	34.00	2.06
Phoneme deletion	17.80	2.86	19.70	0.48	16.89	1.83	17.89	1.62

C consonant, V vowel, RN rapid naming

^a Significant main effect

Table 2 Specification of personalized neurofeedback training protocols and their effect on spectral power and coherence

Gender, age	Power protocol	Coherence protocol	Power pre vs. post (Z-scores)	Coherence pre vs. post (Z-scores)
1 Boy age 8		T3–T4 delta down EO		1.24 vs. 1.01
2 Boy age 11	T6 2–5 Hz down 15–20 up EC	F7–C3 beta down EC	Theta/beta 1.97/–0.54 vs. 0.87/–0.96	1.67 vs. –0.20
3 Girl age 10		T3–T4 delta down EO F7–C3 beta down EO		3 vs. ? 2.59 vs. ? no 2nd measurement
4 Boy age 10	T6 2–12 Hz down EO	F7–FC3 alpha down EO	Delta/theta/alpha 3.93/2.29/2.27 no EO data 2nd measurement	0.92 vs. ? no EO data 2nd measurement
5 Girl age 10	T4 2–8 Hz down EO	T3–T4 delta down EO F7–FC3 alpha down EO	Delta/theta 2.27/1.62 vs. 0.45/0.13	3 vs. –0.02 1.56 vs. –0.40
6 Boy age 9		T3–T4 delta down EO F7–C3 alpha down EO		4.94 vs. 0.61 1.71 vs. –0.58
7 Girl age 8	T6 2–5 Hz down Beta up EO	T3–T4 delta down EO F7–C3 beta down EO	Delta 1.34 vs. –0.04	1.79 vs. –1.03 2.21 vs. –0.37
8 Boy age 12	Fz 18–20 Hz down C3 12–15 Hz up EO		Beta/alpha 1.42/–1.42 vs. 0.32/–1.44	
9 Boy age 9		F7–C3 beta down EO		1.82 vs. 1.07
10 Boy age 8	F3 2–4 Hz down	T3–T4 delta down EO F7–FC3 alpha down EO	1.55 vs. 1.16	2.04 vs. 1.49 3.55 vs. 1.30

$p = .04$) theta increased, although the aim was to train it down at T6. Paired sample correlations were significant and substantial (both $r = .89$, $p = .00$). This suggests that the increase was found in all participants. Alpha 1 was increased at CP4 ($t = -3.42$, $p = .046$) and at O1 ($t = -2.55$, $p = .04$). Paired sample correlations were .76 ($p = .03$) and $r = .94$ ($p = .00$), again suggesting an increase in all participants. Alpha 1 was intended to go down at locations F3 and T6. Beta 1 decreased at Oz ($t = -2.51$, $p = .04$) with a paired sample correlation of $r = .76$ ($p = .03$). Training aimed at a decrease at T6 and Fz.

Coherence Training

Coherence was trained in the delta, alpha and beta ranges. Whereas for delta coherence differences between pre- and posttreatment were found at FC4–FC3, T4–FC4 and C4–T4, these were caused by some extreme scores in the group. In the alpha band a consistent change was found at CP3–F3 ($t = -6.95$, $p = .00$) with a paired sample correlation of $r = .93$ ($p = .00$). Other differences occurred at P3–F3, O1–F3 and C4–T4, yet they showed insignificant

correlations. In contrast with the training goal of a decrease, coherence went up in the delta and alpha bands.

Whereas a multitude of coherences went down in the beta range, only P4–T4 ($t = 3.62$, $p = .01$) showed a consistent decrease in all subjects ($r = .72$, $p = .04$). The training locations of beta coherence were F7–C3.

Associations

Coherence differences were correlated with the spelling test, switching of attention test and verbal interference test. No significant differences were found. An interesting result, however, was the substantial association of increases in alpha coherence at P3–F3, with spelling ($r = .49$, $p = .18$), switching of attention ($r = -.62$, $p = .10$), and verbal interference ($r = .51$, $p = .20$).

Discussion

This is the first randomized controlled study on neurofeedback treatment for dyslexia. The main result is a large

and clinically relevant improvement in spelling, whereas no improvement in reading abilities was found. This contrasts with the few uncontrolled studies on neurofeedback and dyslexia that report increases in reading grade levels (Thornton and Carmody 2005; Walker and Norman 2006). Several explanations for this anomalous result will be given below. First of all, we found several improvements that were not different from the control group. Many children with dyslexia receive remedial teaching, as in our study, and part of the improvement may be attributed to this. This once more points to the importance of a control group while investigating neurofeedback.

Based on phonological theory (e.g., Shaywitz and Shaywitz 2005) one would expect higher EEG activity in fronto-temporal areas. In this study, protocols involved training in the fronto-temporal (power protocols) and frontal-central and parietal areas (coherence protocols). The absence of any effects in these areas is in line with the absence of effects on reading.

We did, however, find substantial gains in the children's spelling. This effect can tentatively be explained as follows. Given the variety of designs applied, a common factor could be the normalization of the EEG. Here, the previously mentioned effects of neurofeedback on switching of attention and verbal interference tests can play a role: the correlations were not significant, yet this may have been a matter of power, given the magnitude of the correlations and the small number of subjects. It can thus be assumed that attentional processes are involved in the improved spelling.

Of course the present study has several limitations. This study is limited by the small number of participants. Besides, qEEG was post-tested in the neurofeedback group only. Our dependent variables consisted of specific (sub) tests of reading and spelling. Functioning in a classroom environment may be less specific than the demands from a test and more susceptible to general nonspecific factors of treatment. Furthermore, our method of assumption-based neurofeedback may have been less than optimal for each individual child. Training was based on EO/EC qEEG only; no reading task differences were taken into account. Besides, training was not designed to optimize normalization but focused on presumed associations of deviations with dyslexia. Finally, no selection was made with regard to any subtype of dyslexia. Wilmer et al. (2004) suggest two distinct motion processing deficits, associated with different kinds of reading sub skills, that may require different feedback protocols.

Future studies are advised to base their treatment on individual qEEG's compared to databases, because it may well be that there are several subtypes of dyslexia (as has been found in ADHD). The large improvements in spelling in this study suggest that further research in this area is

warranted. Neurofeedback can make an important contribution to the treatment of dyslexia.

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