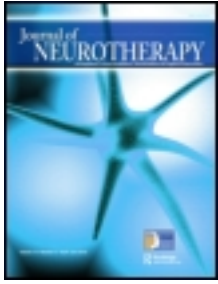


This article was downloaded by: [174.16.85.3]

On: 29 August 2012, At: 15:35

Publisher: Routledge

Informa Ltd Registered in England and Wales Registered Number: 1072954 Registered office: Mortimer House, 37-41 Mortimer Street, London W1T 3JH, UK



## Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience

Publication details, including instructions for authors and subscription information:

<http://www.tandfonline.com/loi/wneu20>

### The Neurophysiology of Dyslexia: A Selective Review with Implications for Neurofeedback Remediation and Results of Treatment in Twelve Consecutive Patients

Jonathan E. Walker MD<sup>a</sup> & Charles A. Norman PhD<sup>b</sup>

<sup>a</sup> Neurotherapy Center of Dallas, TX

<sup>b</sup> Department of Educational Psychology, University of Alberta, Canada

Version of record first published: 08 Sep 2008

To cite this article: Jonathan E. Walker MD & Charles A. Norman PhD (2006): The Neurophysiology of Dyslexia: A Selective Review with Implications for Neurofeedback Remediation and Results of Treatment in Twelve Consecutive Patients, *Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience*, 10:1, 45-55

To link to this article: [http://dx.doi.org/10.1300/J184v10n01\\_04](http://dx.doi.org/10.1300/J184v10n01_04)

PLEASE SCROLL DOWN FOR ARTICLE

Full terms and conditions of use: <http://www.tandfonline.com/page/terms-and-conditions>

This article may be used for research, teaching, and private study purposes. Any substantial or systematic reproduction, redistribution, reselling, loan, sub-licensing, systematic supply, or distribution in any form to anyone is expressly forbidden.

The publisher does not give any warranty express or implied or make any representation that the contents will be complete or accurate or up to date. The accuracy of any instructions, formulae, and drug doses should be independently verified with primary sources. The publisher shall not be liable for any loss, actions, claims, proceedings, demand, or costs or damages whatsoever or howsoever caused arising directly or indirectly in connection with or arising out of the use of this material.

---

## CURRENT CONCEPTS IN NEUROTHERAPY

---

Articles appearing in "Current Concepts" advance hypotheses, descriptions, and reviews of techniques important to clinical neurotherapy. The techniques described are not necessarily supported by clinical research, and opinions expressed regarding the effectiveness or efficacies of these techniques are solely those of the authors.

### The Neurophysiology of Dyslexia: A Selective Review with Implications for Neurofeedback Remediation and Results of Treatment in Twelve Consecutive Patients

Jonathan E. Walker, MD  
Charles A. Norman, PhD

**ABSTRACT.** Dyslexia is a common and important problem in all industrial societies, with a prevalence rate of five to ten percent, for which no consistently effective treatment is available. Recent advances in imaging (morphometric MRI, functional MRI, PET, regional cerebral blood flow), as well as in neurophysiology (evoked potentials, QEEG, event-related desynchronization, coherence studies, magnetic source imaging, reading difference topography) have clarified our understanding of the normal circuitry involved in reading and differences seen in individuals who have trouble learning to read. These studies have important implications for the use of neurofeedback to help dyslexic individuals learn to read more easily. First, we obtain a QEEG and a reading difference topograph. We then train down any abnormalities that are significantly increased and train up any abnormalities that are significantly decreased. Increasing 16-18 Hz activity at T3 (left mid-temporal area) has also proved quite helpful in improving reading speed and comprehension. These combined approaches have been helpful in all cases of dyslexia we have treated, dramatically so in some cases. Each of the 12 individuals treated improved by at least two grade levels after 30 to 35 sessions. doi:10.1300/J184v10n01\_04 [Article copies available for a fee from The Haworth Document Delivery Service: 1-800-HAWORTH. E-mail address: <docdelivery@haworthpress.com> Website: <<http://www.HaworthPress.com>> © 2006 by The Haworth Press, Inc. All rights reserved.]

---

Jonathan E. Walker is affiliated with the Neurotherapy Center of Dallas, TX.

Charles A. Norman is affiliated with the University of Alberta, Canada, Department of Educational Psychology.

Address correspondence to: Jonathan E. Walker, 12870 Hillcrest Road, Suite 201, Dallas, TX 75230 (E-mail: [admin@neurotherapydallas.com](mailto:admin@neurotherapydallas.com)).

Journal of Neurotherapy, Vol. 10(1) 2006  
Available online at <http://jn.haworthpress.com>  
© 2006 by The Haworth Press, Inc. All rights reserved.  
doi:10.1300/J184v10n01\_04

**KEYWORDS.** Dyslexia, QEEG, neurofeedback, coherence training

### **INTRODUCTION**

Dyslexia is a common problem (Watson & Williams, 1995) and recent studies (Bhatnagar, Mandybur, Buckingham, & Andy, 2000; Leisman, 2002; Simos, Breier, et al., 2002) have shown that there is considerable performance variability on various tasks used to refine the diagnosis. Most dyslexics perform poorly on tasks of processing and remembering symbols and some also perform poorly on visual processing tasks, so-called double dyslexia. Rapid naming may also be affected and another classification describes problems in superficial versus deep brain tracts involved in the reading process (Damasio & Geschwind, 1984). Dyslexia is a term primarily used by neurologists; educators describe reading difficulty instead. Traditional tests for dyslexia include psychometric testing or computerized testing (Greene, 1996).

Past research has shown that the left superior temporal gyrus plays an important role in integrating auditory, visual, perceptual and memory inputs in order to accomplish fluent reading. Adult patients with lesions in this area (e.g., strokes) have deficits in auditory comprehension and a general disruption in language processing (Strub & Black, 1986). Evoked potential studies have also shown abnormalities in this area in dyslexic individuals (Duffy, Denckla, Bartels, Sandini, & Kiessling, 1980). Most studies using quantitative EEG have not found a consistent abnormality in the resting eyes-closed state. In a recent study Walker and Norman (2004) found a linear increase in beta activity at T3 (roughly overlying the superior sylvian gyrus) with progressive increases in reading difficulty. This area may be vitally important in reading comprehension, especially as reading difficulty increases.

#### ***Lesion Deficit Correlation***

Damasio and Geschwind (1984) reviewed studies of lesion deficits in understanding dyslexia. Unfortunately, there are numerous lesion variables (extent, location, and distribution overlap) and detection of deficits varies with

the method and timing of testing. Baseline data is rarely available. Many studies indicate an association of word comprehension (encompassing phonemic, lexical, and semantic representations) with the left superior temporal gyrus. Lesions restricted to this area tend to produce pure word deafness, leaving lexical, semantic, orthographic, phonological, and syntactic knowledge intact (Tanaka, Yomodori, & Mori, 1987). Lesions of the left supramarginal gyrus (anterior to T3) are associated with deficits in spelling (Roeltgen & Heilman, 1984) and verbal short-term memory (Vallar & Shallice, 1990). Lesions affecting the left middle temporal gyrus and the inferior temporal gyrus (near T3) and the inferior temporal gyrus (inferior to T3) frequently disrupt performance on semantic tasks (Alexander, Hiltbrunner, & Fischer, 1989). Lesions of the retrosplenial cortex (anterior to O1) produce memory encoding difficulties accompanying semantic task performance (Rudge & Warrington, 1991).

#### ***Cortical Stimulation and Language***

Bhatnagar et al. (2000) reviewed previous studies involving cortical stimulation and added three cases of their own, using flexible grids of electrodes to stimulate the brain after the patients were out of surgery and in a more natural environment. There was considerable variation between the three patients. Stimulation of multiple sites in the frontal, temporal, and parietal lobes was associated with difficulties in comprehension and expression of language. The Bhatnagar et al. (2004) suggest that there is a multi-layered arrangement. Some language functions are overlaid in a common peri-sylvian region that is involved in to sequencing of motor movements, verbal memory, and phonemic identification. It is theorized that the anterior temporal lobe serves syntactic functions while the posterior temporal lobe is associated with naming functions and the frontal lobe is associated with motor speech functions. Multiple frontal, parietal, and temporal sites were implicated in errors of syntax construction.

### ***Morphometric Studies***

Foster, Hynd, Morgan, and Hugdahl (2002) have reviewed studies showing that 60 to 70 percent of normal individuals have an asymmetry of the planum temporale, the left being larger. Many dyslexic persons have either symmetric plana or the right is larger. The superior surfaces of the temporal lobes are symmetric in dyslexic individuals, but larger on the left in controls (Kushch, Gross-Glenn, Jallad, & Lubs, 1993). Pennington et al. (1999) found people with reading disabilities to have smaller insulae (deep temporal) and anterior superior neocortices (roughly F7/F8) but larger retrocallosal cortex (anterior to O1) when compared to controls. These abnormalities occurred bilaterally. It should be noted that the anterior superior cortex includes Broca's area. Decreased gray matter density was found in the left temporal lobe involving superior (above T3), middle (T3), inferior (below T3), and mesial (no surface representation) structures in dyslexics (Brown et al., 2001). Studies on the corpus callosum have found variable and inconsistent abnormalities. Subjects with reading difficulty exhibited decreased diffusion and anisotropy bilaterally in the temporal/parietal white matter (Klingberg et al., 2000). These abnormalities may correlate with fewer connections between various language specialized areas.

### ***Functional MRI Studies***

Posterior ventral areas of the superior temporal gyrus (above T3) are preferentially activated by speech sounds and speech versus silent presentations, more on the left than on the right in fMRI studies (Binder et al., 1977). These posterior ventral superior temporal areas are probably involved more with phoneme recognition and grapheme-phoneme translation than with lexical-semantic processes. Differences between words and non-words are detected in neighboring regions, including the temporal poles (F7/F8), the middle temporal gyrus (T3), the inferior temporal gyrus (below T3) and the angular gyrus (between T5 and P3; Binder & Price, 2002).

Primary visual areas are not differentially activated by words or pseudo-words. The left lateral extrastriate (lateral to O1) region is pref-

erentially activated by letter strings versus faces or visual textures (Puce, Allison, Asgari, Gore, & McCarthy, 1996). Some studies suggest this may be involved in differentiating familiar letters from non-sense (unfamiliar) characters (Pugh et al., 1996). Stimulation by orthographic versus non-orthographic activation also occurs in the left inferior frontal area (F7; Fiez & Petersen, 1998) and the left anterior insula (deep temporal, no surface representation; Price, Wise, & Frackowiak, 1996).

A hypothesis suggesting that dyslexics have a specific impairment within the visual magnocellular pathway (Stein & Walsh, 1997) has been largely discredited (Amitay, Ben-Yehudah, Banai, & Ahissar, 2002). Over-activation of Broca's area in dyslexics may represent increased effort in performing language related tasks (Shaywitz et al., 1998). Pugh et al. (2000) summarized the neuroimaging literature on normal reading in dyslexia. They propose that in normally developing readers, a dorsal left posterior temporo-parietal system predominates at first and is associated with the analytical processing necessary for learning to integrate the orthographic features with phonological and lexical-semantic features of printed words. Later, a posterior ventral occipital/temporal circuit comes to constitute a fast word-identification system underlying fluent word recognition in skilled readers. These two posterior systems are functionally disrupted in developmental dyslexia. Reading disabled persons demonstrate higher reliance than normals on bilateral frontal inferior (F7, F8) and right posterior hemisphere (P4) regions to compensate for left posterior deficits.

A remediation program focused on auditory processing and oral language training improved oral language and reading performance and also increased activation toward normal in the left temporo-parietal cortex and left inferior frontal gyrus of dyslexics (Temple et al., 2003). This study suggests that hypoactivation of these areas is a crucial determinant of dyslexia. However, several other areas showed an increase in activity after remediation (bilateral cingulate gyrus, left hippocampal gyrus, left lingual gyrus, right pre-cuneus/posterior cingulate, right parietal-occipital sulcus, and bilateral hypothalamus). Furthermore, no correlation was found (these areas are not near the



surface areas of the 10/20 system) between improved reading scores and activation of the left temporo-parietal region or the inferior frontal region.

### ***Positron Emission Tomography Studies (PET)***

Gross-Glenn, Duara, Barker, and Loewenstein (1991) have made a case for involvement of the cerebellum in developmental dyslexia, the so-called "dyslexia automatization hypothesis." They found classical signs of cerebellar dysfunction in dyslexic children, including dystonia, discoordination, dysequilibrium, and decreased muscle tone. PET studies indicated decreased activation in the right cerebellar hemisphere and vermis in 80 percent of the subjects when learning a button-press sequence compared to controls. They proposed that the reduced quality of an articulatory representation might lead to impaired sensitivity to onset, rhyming, and the phonemic structure of language, leading to deficits in phonological awareness and in naming speed (the "double deficit" hypothesis). Other PET studies in dyslexics have shown bilateral hyperactivation of the lingual gyrus (medial occipital) with relative right frontal (F4) hypo-activation (Hagman et al., 1992). Another study found a bilateral increase in metabolism in the medial temporal regions (not projecting to the cortical surface; Rumsey et al., 1992). Other studies showed failure of activation in left posterior temporal (near T3) and inferior parietal (between P3 and T5) regions in dyslexic adults (Paulesu et al., 1996). Other studies supported a disconnection between posterior and anterior language areas (Horwitz, Rumsey, & Donohue 1998; Rumsey et al., 1999).

### ***Regional Cerebral Flow***

An important study points to the left angular gyrus (between T5 and P3) as the most likely site of functional impairment in dyslexic adults (Ramsey et al., 1999). Higher blood flow is associated with better reading skill in controls and lower blood flow is associated with poor reading in dyslexics.

### ***Electrophysiological Imaging of Brain Function in Dyslexia***

Nobre and McCarthy (1995) reviewed evoked potential studies in normal word recognition. Word recognition is often impaired in dyslexia, and evoked potential studies are only used for studying discrete events. Quantitative EEG, on the other hand, may be used in combination with recall of read materials to determine which areas of the brain are involved in both word recognition and comprehension. Most QEEG studies have shown no consistent difference between dyslexics and controls in the eyes-closed state as expected, but some changes have been seen with activation (i.e., reading during acquisition of the QEEG). Flynn and Deering (1989) found no difference between dysphonetic dyslexics (phonologically impaired) and control subjects, but dyseidetic (visually impaired) children showed a marked increase in left temporal and parietal theta activity during reading and spelling tasks. Flynn, Deering, Goldstein, and Rahbar (1992) failed to confirm the theta activation but found reduced beta activity bilaterally in the dyseidetic children and decreased right parietal/occipital beta activity in the dysphonetic children. Walker and Norman (2004) hypothesized that easy reading would not require activation of "reading areas," but progressively difficult reading would. This proved true at T3 (roughly overlying the superior temporal gyrus), but no other areas were activated by difficult reading. This suggests that training the T3 area to activate during difficult reading tasks might be useful in making children with dyslexia easier to teach and help them to catch up with their classmates, using intensive remedial instruction.

Event-related desynchronization has also been used to study dyslexia. Rippon and Brunswick (2000) found a global absence of task-related alpha amplitude reduction for both a reading task and a picture completion task. There was also a marked asymmetry (right greater than left) in beta activity in the parietal/occipital regions, again in both tasks, in the dyslexic group. Dyslexics had an increase in frontal theta with the phonological tasks, whereas control subjects exhibited a decrease. Both dyslexic and control subjects showed a task-related

reduction in frontal theta with the visual task. Improvements in cognitive skill have been associated with reduction in theta, and Orekhova, Stroganova, and Posikera (1999) have suggested that higher levels of theta may be evidence of less active task engagement (i.e., more “idling”).

Another approach has been to look at coherence between brain areas in dyslexia. Leisman (2002) found that controls have significantly greater coherence in the 1 to 30 Hz range between the hemispheres at several homologous sites compared to dyslexics. Dyslexics demonstrated significantly greater coherence at 1 to 30 Hz within hemispheres compared to normal. He suggested that developmental dyslexia may represent a functional hemispheric disconnection syndrome. Pugh et al. (2000) found a disruption in functional connectivity between the left angular gyrus and related occipital and temporal sites, but only on tasks requiring phonologic assembly and not on print tasks. Evans (1996) also found decreased coherence between multiple left posterior hemisphere sites ( $P3 > T5 > T3 > O1$ ) in dyslexic subjects, with several different combinations being noted in 70 percent of the dyslexic subjects.

Duffy et al. (1980) and Duffy, Denckla, McAnulty, and Holmes (1988) are the only group that have reported quantitative evoked potentials (QEPs) in dyslexic subjects. They found decreased responsiveness to auditory stimuli (non-verbal) in the middle posterior portions of the left (between T3 and T5) temporal lobes. When a verbal task was used as a stimulus (count “TYKE” versus “TIGHT” presentations), more posterior regions were affected including a prominent right posterior occipital/temporal region. They found that they could discriminate between anomia, dysphonemic, and global types using several tasks and found differences in both QEEG and QEPs using a discriminant involving several such tasks. No significant differences were found in the left parietal/occipital areas, suggesting that all dyslexics are impaired in this area. Differences found in the subtypes may reflect compensatory activation of areas not normally required for reading rather than a pathological change.

### ***Magnetic Source Imaging (MEG)***

Simos, Fletcher, et al. (2002) found decreased activation of the left temporo-parietal region in dyslexics during a visual pseudo-word rhyme-matching task. After an intensive 80-hour intervention that produced significant improvement in reading skills their scores on basic word reading tests increased into the normal range. They observed increased activation in the left posterior superior temporal gyrus of the dyslexic children. Salmelin, Service, Kiesila, Uutela, and Salonen (1996) described impaired processing of word forms in adult dyslexic subjects. A left inferior temporal occipital area (below T3) was differentially engaged in the normal subjects as early as 180 milliseconds after word presentation. Dyslexic subjects either failed to activate this area or showed a slowly increasing late response. Letter-string specific responses peaking around 150 milliseconds after presentation in the left inferior occipital temporal cortex in fluent readers were undetectable (no surface representation) in dyslexic subjects. Children with dyslexia exhibited little or no activation of the left posterior superior temporal gyrus (close to T5) compared to controls. Activation was increased in the right posterior superior temporal gyrus compared to control subjects. After a two-month intervention that produced significant improvement in reading skills, activity in the posterior left superior temporal gyrus increased by several orders of magnitude, of magnetic flux, in each participant (Simos, Fletcher, et al., 2002).

### ***Suggestions for Neurofeedback Training Based upon Cortical Stimulation and Imaging Studies***

Most studies indicate a central role for the superior temporal gyrus and certain adjacent areas (in and around T3) in normal reading comprehension with impairment in one or more of these areas in persons with dyslexia. Beta 2 activity (15-18 Hz) is an indicator that the area is engaged in the reading process (Walker & Norman, 2004). With an easy learning task, this area will only generate slightly more beta 2 activity. With progressively more difficult reading tasks more beta 2 activity will be generated by that area, but if the task becomes too diffi-

cult, the area may disengage and stop generating beta2 activity (Walker & Norman, 2004).

By training such a critical brain area to function normally (i.e., train that area to be quiet at rest, become active with an easy learning task, and to become increasingly more active with more difficult learning tasks) reading skills should improve. Higher frequency beta (21-30 Hz) may well be dysfunctional and an indica-

tion of anxiety and would need to be down-trained. Then coherence abnormalities are trained to normalization. The first 12 patients in our clinic reported an improvement of at least two grade levels, as judged by their reading teachers, in reading speed and comprehension using this protocol (30 to 35 ten-minute sessions each). Results are presented in Case I, Case II and Table 1.

TABLE 1. Effect of Neurofeedback in Improving Reading Level in 10 Additional Cases

Case	Age	Grade	Pre-Neurofeedback Reading Grade Level	Neurofeedback Protocols (5 sessions each)	Post-Neurofeedback Reading Grade Level
3	16	10	9	↓ 2-7 Hz/↑ 12-15 Hz at FP2 ↓ 1-8 Hz plus ↓ 18-30 Hz at OZ ↓ coherence of beta at P3/O1 ↓ coherence of beta at FP2/O2 ↑ coherence of delta at F3/O1 ↑ coherence of theta at C4/P4 ↑ coherence of delta at F4/O2	12
4	10	4	1	↓ 2-7 Hz/↑ 15-18 Hz at FP1 ↓ 1-5 Hz at O2 ↓ 1-8 Hz at PZ while reading ↑ coherence of beta at F4/O2 ↓ coherence of theta at F3/P3	4
5	11	5	2	↓ 1-4 Hz at O2 ↓ 1-5 Hz at F8 ↑ 15-18 Hz at T3 while reading ↓ 10-12 Hz at O1	5
6	7	1	Pre-K	↓ 4 Hz at F7 ↓ 5-6 Hz at F8 ↓ 29-30 Hz at FZ ↓ coherence of beta at O1/T3 ↓ 1-10 Hz at F3 while reading ↓ 2-7 Hz/↑ 15-18 Hz at C3	2
7	11	5	4	↓ 2-7 Hz at F3 plus F4 ↓ 2-7 Hz at O1 plus O2	10

			Pre-Neurofeedback		Post-Neurofeedback
Chart	Age	Grade	Reading Grade Level	Neurofeedback Protocols (5 sessions each)	Reading Grade Level
				↑ coherence of beta at F7/F4	
				↑ coherence of theta at C4/T4	
				↓ coherence of delta at O1/F3	
				↓ 2-7 Hz/↑ 15-18 Hz at PZ	
				↑ coherence of theta at PZ/O2	
				↓ coherence of theta at T3/C3	
8	12	6	5	↓ 2-7 Hz/↑ 15-18 Hz at T5	7
				↓ 2-7 Hz/↑ 15-18 Hz at O2	
				↓ 2-7 Hz/↑ 15-18 Hz at O1	
9	9	3	2	↓ 2-7 Hz/↑ 12-15 Hz at C4	4
				↓ 2-7 Hz/↑ 15-18 Hz at O2	
				↓ 2-7 Hz/↑ 15-18 Hz at O1	
				↓ 2-7 Hz/↑ 15-18 Hz at P4	
10	10	5	4	↓ 1-4 Hz at CZ	7
				↓ 1-2 Hz/↑ 9-11 Hz at O1	
				↓ 1-11 Hz at FP1 while reading	
				↑ coherence of beta at T3/T4	
				↓ coherence of alpha at F1/T3	
				↓ coherence of delta at T5/P3	
				↑ coherence of theta at C3/T3	
11	10	3	2	↓ 2-7 Hz/↑ 15-18 Hz at C3	4
				↓ 1 Hz/↑ 15-18 Hz at T4	(gifted and talented)
				↓ 1-10 Hz at F3 while reading	
				↓ 30 Hz at F8	
				↓ coherence of delta at F7/T5	
				↓ coherence of alpha at O1/T3	
				↓ coherence of theta at F3/T5	
12	7	2	1	↓ 1-8 Hz/↑ 15-18 Hz at CZ	3
				↓ coherence of alpha at F7/F3	
				↑ coherence of delta at CZ/PZ	
				↑ coherence of alpha at CZ/C4	
				↑ 11-12 Hz at O2	



Future efforts to improve our treatments will include enhancing our understanding and use of reading difference topographies. These are obtained by mapping baseline eyes open data and subtracting that data from maps obtained while reading moderately difficult material. We can then train the person while reading to decrease slow or “idling” rhythms (4-10Hz) and increase faster rhythms (15-18Hz) which are more appropriate for paying attention and learning.

During analysis of the QEEG, we use raw single Hz difference maps between reading and eyes open conditions. These maps show how the EEG changes when the person reads. We compute these difference maps with NeuroRep (Hudspeth, 2004) by subtracting the eyes open raw numbers from the task raw numbers. If the task voltage is higher than the eyes open baseline, the remainder will be positive and it will indicate how much the EEG voltage increased when person read. If the remainder is negative, the maps will show how much the voltage decreased while reading. We use NeuroRep because all the single Hz topographs are created with the same color scale so it is easy to look at a page of topographs and see where the largest changes happen. The results from NeuroRep give only absolute differences. In order to get an idea of percent change, we compare the change in voltage in the difference topographs to the eyes open baseline. For example, a 2 micro-volt change in the single Hz differences is important if the baseline EEG voltage is 2.7 uV. However, a 2 uV difference is not very important if the baseline voltage is 7 uV. NeuroGuide (Thatcher, 1998) can make single Hz difference maps as well. It has the advantage of calculating the maps as percent change maps. However, NeuroGuide scales each map with a different color scale so we cannot look at a page and see where the biggest changes occur. SKIL (Serman & Kaiser, 2005) may also be able to calculate and create different topographs as well.

### ***Two Cases Reports Illustrating the Application of These Findings to Persons with Dyslexia***

*Case I.* A 15-year-old boy presented with a chief complaint of dyslexia. He also complained of short-term memory difficulty and in-

termittent depression. He was in a special school, in the ninth grade, but reading at a fifth grade level. His initial QEEG revealed an increase in the relative power of theta at FP1, F3, F4, FZ, and CZ. There was an elevated theta/beta ratio at FZ and CZ. Reading difference topography revealed a diffuse increase in frequencies 1 to 10 while reading, especially marked in the occipital areas in the 1-2 Hz bins (normal readers usually decrease the amount of 1-10 Hz diffusely while reading). Coherence analysis revealed increases in the coherence of delta at F8/T6 and at F4/O2.

He completed 30 sessions of neurofeedback with 5 sessions of each of the following protocols:

- |   | <u>Based on</u>               |
|---|-------------------------------|
| 1. Decrease 1-7 Hz at CZ.                       | Eyes open QEEG                |
| 2. Decrease 2-7 Hz and increase 15-18 Hz at CZ. | Eyes open QEEG                |
| 3. Decrease 1-10 Hz at O1 while reading.        | Reading difference topography |
| 4. Increase 11-12 Hz at O1.                     | Eyes open QEEG                |
| 5. Decrease coherence of delta at F8/T6.        | Eyes open QEEG                |
| 6. Decrease coherence of delta at T4/O2.        | Eyes open QEEG                |

Following the 30 neurofeedback sessions, he was reading at grade level (Grade 10). He was no longer having memory problems and was not depressed, by self-report. A repeat QEEG showed normalization of the reading difference topograph with a decrease in frequencies 1 to 10 in the reading condition. The coherence abnormalities were also normalized. There was still mild slowing (in the theta band) at F4, C3, C4, P4, O1, O2, and PZ. A new finding was an increase in beta (15-18 Hz) fronto-polar, frontal and central regions bilaterally. No new symptoms were noted.

*Case II.* This 9-year-old girl had the chief complaints of dyslexia, spelling difficulty and bad handwriting. She was in the fourth grade, but was reading at first grade level and hated reading. The initial QEEG showed an elevated

theta/beta ratio, maximal at C3/P3. Reading difference topography showed a diffuse increase in the 1 to 10 Hz frequencies in the reading condition. There was an increase in the intrahemispheric coherence of alpha at T4/T6, a decrease of beta coherence at CZ/PZ and an increase in delta coherence at F1/F7. She completed 53 neurofeedback sessions as follows:

- |   | <u>Based on</u>                 |
|---|---------------------------------|
| 1. Decrease 2-8 Hz and increase 15-18 Hz at CZ (10 sessions). | Eyes open QEEG                  |
| 2. Decrease 2-8 Hz and increase 15-18 Hz at P3 (10 sessions). | Eyes open QEEG                  |
| 3. Decrease 1-10 Hz at O1 while reading (10 sessions).        | Reading difference topographies |
| 4. Decrease coherence of alpha at T4/T6 (5 sessions).         | Eyes open QEEG                  |
| 5. Decrease coherence of beta at CZ/PZ (5 sessions).          | Eyes open QEEG                  |
| 6. Decrease coherence of delta at FP1/F7 (5 sessions).        | Eyes open QEEG                  |
| 7. Increase 11-12 Hz at O2 (8 sessions).                      | Eyes open QEEG                  |

Following the training she was reading about grade level (Grade 6), as judged by her reading teacher. She was reading for pleasure for the first time. Her handwriting and spelling were much improved.

The follow up QEEG revealed normalization of all of the abnormalities trained. Reading difference topographies revealed the normal decrease in 9 to 10 Hz activity in the reading condition, though the occipital 4 to 7 Hz frequencies and F7 and F8 1 to 2 Hz frequencies were still increased during reading (better, but not normal). No new abnormalities were seen. Our experience with 10 additional patients is summarized in Table 1.

Recent advances in neuroimaging have led to an improved understanding of the brain areas

involved in reading and of the abnormalities leading to dyslexia. QEEG is the only modality that indicates the state of connectivity between these critical areas. We describe an approach to correcting the abnormalities of magnitude and coherence which characterize dyslexic states. Our preliminary results suggest that this approach is likely to result in improved reading skills over relatively short time periods (a few weeks to a few months).

## REFERENCES

- Alexander, M. P., Hiltbrunner, B., & Fischer, R. S. (1989). Distributed anatomy of transcortical sensory aphasia. *Archives of Neurology*, *46*, 885-892.
- Amitay, S., Ben-Yehudah, G., Banai, K., & Ahissar, M. (2002). Disabled readers suffer from visual and auditory impairments but not from a specific magnocellular deficit. *Brain*, *125*, 2272-2285.
- Bhatnagar, S. C., Mandybur, G. T., Buckingham, H. W., & Andy, O. J. (2000). Language representation in the human brain: Evidence for cortical mapping. *Brain & Language*, *74*, 238-259.
- Binder, J. R., Frost, J. A., Hammeke, T. A., Cox, R. W., Rao, S. M., & Prieto, T. (1997). Human brain language areas identified by functional magnetic resonance imaging. *Journal of Neuroscience*, *17*, 353-362.
- Binder, J. R., & Price, C. J. (2002). Functional neuroimaging of language. In R. Cabeza & A. Kingstone (Eds.), *Handbook of functional neuroimaging of cognition* (pp. 202 - 224). Boston MA: MIT Press.
- Brown, W. E., Eliez, S., Menon, V., Rumsey, J. M., White, C. D., & Reiss, A. L. (2001). Preliminary evidence of widespread morphological variations of the brain in dyslexia. *Neurology*, *56*, 781-783.
- Damasio, A., & Geschwind, N. (1984). The neurological basis of language. *Annual Review of Neuroscience*, *7*, 127-147.
- Duffy, F. H., Denckla, M. B., Bartels, P. H., Sandini, G., & Kiessling, L. S. (1980). Dyslexia: Automated diagnosis by computerized classification of brain electrical activity. *Annals of Neurology*, *7*, 421-428.
- Duffy, F. H., Denckla, B. D., McAnulty, G. B., & Holmes, J. A. (1988). Neurophysiological studies in dyslexia. In F. Plum (Ed.), *Language, communication, and the brain* (pp. 60, 149-170). New York: Raven Press.
- Evans, J. R. (1996). Quantitative EEG abnormalities in a sample of dyslexic persons. *Journal of Neurotherapy*, *2* (1), 1-5.
- Fiez, J. A., & Petersen, S. E. (1998). Neuroimaging studies of word reading. *Proceedings of the National Academy of Sciences of the United States of America*, *95*, 914-921.
- Flynn, J. M., & Deering, W. M. (1989). Subtypes of dyslexia: Investigation of Boder's system using quanti-

- tative neurophysiology. *Developmental Medicine and Child Neurology*, 31, 215-223.
- Flynn, J. M., Deering, W. M., Goldstein, M., & Rahbar, M. H. (1992). Electrophysiological correlates of dyslexic subtypes. *Journal of Learning Disabilities*, 25, 133-141.
- Foster, L. M., Hynd, G. W., Morgan, A. E., & Hugdahl, K. (2002). Planum temporale asymmetry and year advantage in dichotic listening in developmental dyslexia and Attention-Deficit/Hyperactivity Disorder (ADHD). *Journal of the International Neuropsychological Society*, 8, 22-36.
- Greene, J. F. (1996). Psycholinguistic assessment: The clinical base for identification of Dyslexia. *Topics in Language Disorders*, 16, 45-72.
- Gross-Glenn, K., Duara, R., Barker, W., & Loewenstein, D. (1991). Positron emission tomographic studies during serial word-reading by normal and dyslexic adults. *Journal of Clinical and Experimental Neuropsychology*, 13, 531-544.
- Hagman, J. O., Wood, F., Buchsbaum, M. S., Tallal, P., Flowers, L., & Katz, W. (1992). Cerebral brain metabolism in adult dyslexic subjects assessed with positron emission tomography during performance of an auditory task. *Archives of Neurology*, 49, 734-739.
- Horwitz, B., Rumsey, J. M., & Donohue, B. C. (1998). Functional connectivity of the angular gyrus in normal reading and dyslexia. *Proceedings of the National Academy of Sciences of the United States of America*, 95, 8939-8944.
- Hudspeth, W. J. (2004). NeuroRep EEG Report Software Manual. Reno, NV: Grey Matter, Inc.
- Klingberg, T., Hedehus, M., Temple, E., Salz, T., Gabrieli, J. D., Moseley, M. E., et al. (2000). Microstructure of temporo-parietal white matter as a basis for reading ability: Evidence from diffusion tensor magnetic resonance imaging. *Neuron*, 25, 493-500.
- Kushch, A., Gross-Glenn, K., Jallad, B., & Lubs, H. (1993). Temporal lobe surface area measurements on MRI in normal and dyslexic readers. *Neuropsychologia*, 31, 811-821.
- Leisman, G. (2002). Coherence of hemispheric function in developmental dyslexia. *Brain and Cognition*, 48, 425-431.
- Nobre, A. C., & McCarthy, G. (1995). Language-related field potentials in the anterior-mediated temporal lobe: Effects of word type and semantic priming. *Journal of Neurosciences*, 15, 1090-1098.
- Orekhova, E. V., Stroganova, T. A., & Posikera, I. N. (1999). Theta synchronization during sustained anticipatory attention in infants over the second half of the first year of life. *International Journal of Psychophysiology*, 32, 151-172.
- Paulesu, E., Frith, U., Snowling, M., Gallagher, A., Morton, J., Frackowiak, R. S., et al. (1996). Is developmental dyslexia a disconnection syndrome? Evidence from PET scanning. *Brain*, 119, 143-157.
- Pennington B. F., Filipek, P. A., Lefly, D., Churchwell, J., Kennedy, D. N., Simon, J. H., et al. (1999). Brain morphometry in reading-disabled twins. *Neurology*, 5, 723-729.
- Price, C. J., Wise, R. J., & Frackowiak, R. S. (1996). Demonstrating the implicit processing of visually presented words and pseudowords. *Cerebral Cortex*, 6, 662-670.
- Puce, A., Allison, T., Asgari, M., Gore, J. C., & McCarthy, G. (1996). Differential sensitivity of human visual cortex to faces, letter strings, and textures: A functional magnetic resonance imaging study. *Journal of Neuroscience*, 16, 5205-5215.
- Pugh, K. R., Mencl, W., Einar, J., Annette, R., Katz, L., Frost, S. J., et al. (2000). Functional neuroimaging studies of reading and reading disability (developmental dyslexia). *Mental Retardation & Developmental Disabilities Research Reviews*, 6, 207-213.
- Pugh, K. R., Shaywitz, B. A., Shaywitz, S. E., Constable, R. T., Skudlarski, P., Fulbright, R. K., et al. (1996). Cerebral organization of component processes in reading. *Brain*, 119, 1221-1238.
- Rippon, G., & Brunswick, N. (2000). Trait and state EEG indices of information processing in developmental dyslexia. *International Journal of Psychophysiology*, 36, 251-265.
- Roeltgen, D. P., & Heilman, K. M. (1984). Lexical agraphia: Further support for the two-system hypothesis of linguistic aphasia. *Brain*, 107, 811-827.
- Rudge, P., & Warrington, E. K. (1991). Selective impairment of memory and visual perception in splenial tumours. *Brain*, 114, 349-360.
- Rumsey, J. M., Andreason, P., Zametkin, A. J., Aquino, T., King, A. C., Hamburger, S. D., et al. (1992). Failure to activate the left temporoparietal cortex in dyslexia. An oxygen 15 positron emission tomographic study. *Archives of Neurology*, 49, 527-534.
- Rumsey, J. M., Horwitz, B., Donohue, B. C., Nace, K. L., Maisog, J. M., & Andreason, P. (1999). A functional lesion in developmental dyslexia: Left angular gyral blood flow predicts severity. *Brain & Language*, 70, 187-204.
- Salmelin, R., Service, E., Kiesila, P., Uutela, K., & Salonen, O. (1996). Impaired visual word processing in dyslexia revealed with magneto-encephalography. *Annals of Neurology*, 40, 157-162.
- Shaywitz, S. E., Shaywitz, S. E., Pugh, K. R., Fulbright, R. K., Constable, R. T., Mencl, W. E., et al. (1998). Functional disruption in the organization of the brain for reading in dyslexia. *Proceedings of the National Academy of Sciences of the United States of America*, 95, 2636-2641.
- Simos, P. G., Breier, J. I., Fletcher, J. M., Foorman, B. R., Bergman, E., Fishbeck, K., et al. (2002). Brain activation profiles in dyslexic children during non-word reading: A magnetic source imaging study. *Neuroscience Letters*, 290, 61-65.
- Simos, P. G., Fletcher, J. M., Bergman, E., Breier, J. I., Foorman, B. R., Castillo, E. M., et al. (2002). Dys-

- lexia-specific brain activation profile becomes normal following successful remedial training. *Neurology*, 58, 1203-1213.
- Stein, J., & Walsh, V. (1997). To see but not to read: The magnocellular theory of dyslexia. *Trends in Neuroscience*, 20, 147-152.
- Serman, M. B., & Kaiser, D. A. (2005). SKIL Topometric Software. Los Angeles, CA: Serman-Kaiser Imaging Laboratory.
- Strub, R., & Black, F. W. (1986). *Neurobehavioral disorders: A clinical approach* (pp. 253-311). Philadelphia, PA: F. A. Davis.
- Tanaka, Y., Yomodori, A., & Mori, E. (1987). Pure word deafness following bilateral lesions. *Brain*, 110, 381-403.
- Temple, E., Deutsch, G. K., Poldrack, R. A., Miller, S. L., Tallal, P., Merzenich, M. M., et al. (2003). Neural deficits in children with dyslexia ameliorated by behavioral remediation: Evidence from functional MRI. *Proceedings of the National Academy of Sciences of the United States of America*, 100, 2860-2865.
- Thatcher, R. W. (1998). EEG normative databases and EEG biofeedback. *Journal of Neurotherapy*, 2 (4), 8-39.
- Vallar, G., & Shallice, T. (1990). *Neuropsychological impairments of short-term memory*. New York: Cambridge University Press.
- Walker, J. E., & Norman, C. A. (2004, August). Normal adult readers recruit increasing beta power at T3 as reading difficulty increases. Presentation at the 12th International Society for Neuronal Regulation Conference, Ft. Lauderdale, FL.
- Watson, C. W., & Williams, D. M. (1995). Information processing patterns in specific reading ability. *Journal of Learning Disabilities*, 28, 216-231.

RECEIVED: 06/30/04

REVISED: 12/01/04

04/22/04

01/25/05

ACCEPTED: 08/23/05

doi:10.1300/J184v10n01\_04

