

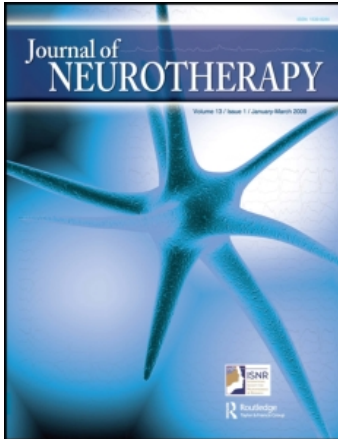
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Quantitative EEG Abnormalities in a Sample of Dyslexic Persons

James R. Evans, Ph.D. and Nan-Sook Park, M.A.

Definitions of terms such as dyslexia and specific reading disability commonly recognize a basis in central nervous system dysfunction. Past research has related this dysfunction to both structural and neural timing abnormalities. The present study used QEEG findings to provide further evidence for neural timing/coherence abnormalities in reading disabled persons. Eight children and two adults were diagnosed with specific reading disability based on standard psychoeducational assessment. QEEGs were obtained from each using Lexicor Neurosearch 24 equipment, and analyzed using the Thatcher Life Span Reference Data Base. Standard print-outs depicting coherence, phase, amplitude asymmetry, and relative power abnormalities of each subject were inspected, and tallies made of the most frequently occurring significant deviations from the norms. The following abnormalities were found in 70% or more of the subjects: (1) abnormal coherence between one or more combination of sites P3, T5, T3, O1; (2) an equal to or greater than 1.4/1 ratio of left to right side coherence abnormalities; (3) coherence abnormalities between posterior sites more often involved decreased rather than increased coherence; (4) at least five abnormalities (or any type) involving site P3; (5) at least three abnormalities (coherence, phase, asymmetry) involving frontal/parietal sites. These data appear to have relevance for neurofeedback. Phase/coherence (neural timing) training and emphasis on site P3 may be especially useful in some cases of reading disability.

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Estimates of the percentage of persons who experience significant reading problems have been as high as 30 percent in this country (Eisenberg, 1978). Many of these are persons who have at least average abilities in other skills areas, and thus can be said to suffer from specific reading disability (dyslexia). Estimates of the percentage of dyslexic persons usually range from 5 to 10 percent (Benton & Pearl, 1978). Most definitions of dyslexia imply that it has a neurophysiological basis. There have been MRI and autopsy studies providing evidence for brain structural differences, e.g., lack of normal left greater than right asymmetry (or reversed asymmetry) in the planum temporale and other posterior cortical regions (Hynd, Marshall, & Gonzalez, 1991), abnormally small and disorganized magnocellular

layers at the thalamic level in the visual system (Galaburda & Livingstone, 1993). However, a popular recent view of the dynamics of dyslexia contends that there is a failure of appropriate neural timing. Specifically, timing problems appear to be involved in a weakness in ability to integrate sensory information which occurs in rapid succession within tens of milliseconds (Tallal, Miller, & Fitch, 1993). In this view, for example, stop consonants such as "ba" and "da" which involve very rapid changes in frequency within themselves may not be discriminated, leading to problems in phoneme awareness, and hence to difficulty "sounding out" words and comprehending phonetic approaches to reading instruction. Similarly, the recent findings of visual system abnormalities in dyslexic persons has

led to speculation that these abnormalities may cause different types of visual information (mediated by magnocellular and parvocellular layers respectively) to be received out of sequence at higher visual processing centers in the brain, thus leading to visual perceptual problems. Again, this is believed to be a failure of neural timing.

Evidence from research such as that cited above has led to the suggestion that dyslexia may be conceptualized accurately as a "dyschronia" (Llinas, 1993). If so, quantitative EEG (QEEG) research may be promising as a means of investigating dyslexia. At this time, only the QEEG offers a relatively inexpensive and non-invasive means of studying neural timing relationships such as those inherent in QEEG measures of phase and coherence. Most QEEG research on dyslexia has involved spectral analyses comparing dyslexics to normally reading persons in terms of amplitude or power in the EEG at various scalp sites. This has led to limited findings supportive of EEG differences in dyslexia. For example, in a frequently cited study involving a small number of subjects, Duffy, Denckla, Bartels, and Sandini (1980) found EEG difference in bilateral medial frontal, left temporal, parietal, and visual association cortical regions. A few older studies have involved measures of neural timing, and have provided some support for abnormal timing in dyslexia. Sklar, Hanley, and Simmons (1972) found reading disabled children to have higher coherence between certain intrahemispheric scalp sites during reading than found in normally reading children, while the latter showed greater coherence between homologous regions across the midline. Evans (1977), using a coherence type measure (cortical coupling), reported a unique pattern of coupling (neural timing) among left posterior scalp sites characterizing average and above average readers. This pattern was observed significantly less often in a sample of children with specific reading disability.

Despite much recent evidence that dyslexia is highly related to neural timing

abnormalities, a search of the literature since 1990 revealed no reports of research on quantitative EEG phase or coherence measures in dyslexia. Therefore, it was considered worthwhile to report data from the present pilot study with the hope that it might stimulate further productive research in this area.

Method

Subjects

Subjects for this study were seven children and three adults who had been diagnosed by the author (a licensed clinical and school psychologist) in private practice as having a specific reading disability. In two cases there also was evidence for an attention deficit disorder. Subject characteristics are shown in Table 1. This was not a random sample, but, rather, a sample of convenience composed of former clients who were willing to return for the QEEG evaluation after periods ranging from one week to eighteen years following their original psychoeducational assessment. All subjects were re-evaluated at the time of the QEEG assessment to ensure that they continued to have serious problems with reading (both word recognition and reading comprehension). Discrepancies between Full Scale IQ and reading (word recognition) standard scores ranged from 17 to 53.

Procedure

Lexicor Neurosearch 24 equipment was used in conjunction with Lexicor V151 software, and the appropriate-sized electrode cap from Electro Cap International, Inc. Approximately three minutes of activity was sampled during an eyes closed, resting condition from nineteen scalp electrode sites in the standard International 10-20 montage, referenced to earlobes and grounded just forward of site FZ. Sampling rate was 128 Hz, with 32K gain and high pass filter off. Scalp electrode sites were prepared until impedance for each channel was at or below 5000 ohms. After electrodes were attached, and, prior to gathering data, subjects were

Table 1
Characteristics of Subjects

Gender	Age	Full Scale IQ	Reading ^a Standard Score	Arithmetic ^b Standard Score
Male 8	Mean 15.9	Mean 103.8	Mean 71.4	Mean 93.2
Female 2	SD 8.0	SD 14.9	SD 13.6	SD 12.8

^a Word recognition; based on Wide Range Achievement Test, or Peabody Individual Achievement Test

^b Based on Wide Range Achievement Test

seated in a reclining chair and asked to relax, sit as still as possible and attempt to keep eyes closed and still. The raw EEG data were observed on a computer monitor and instructions given to subjects until eye and other movement artifacts were minimized. Data collection did not begin until it seemed that no further decrease in artifacts was likely to occur.

After the three-minute sample of EEG was collected, wave forms were inspected visually off-line, and artifacts removed prior to data analysis. This was done by the author, a certified QEEG technician. Data analysis was completed using Neurorep Software (Hudspeth, 1994) which involves the Thatcher QEEG Life Span Reference Database (Thatcher, Walker, & Guidice, 1987). Specifically, measures of coherence, phase, and amplitude asymmetry in four different frequency bands are computed among all combinations of eight right and eight left intrahemispheric sites, and between homologous hemispheric sites. Relative power in each of the same four frequency bands at each of sixteen scalp electrode sites (excluding FZ, CZ, PZ) also is calculated. The four frequency bands involved in this analysis were: Delta (.5 to 3.5 Hz), Theta (3.5 to 7.0 Hz), Alpha (7.0 to 13.0 Hz), and Beta (13 to 22 Hz). A total of 832 raw scores are calculated by this program, transformed to Z scores and printed. Z scores differing significantly from the reference data-

base norms for the subjects age, gender, and handedness are indicated (along with level of significance i.e., .025, .005, .001).

Results

After all scores were printed, they were inspected visually to determine which abnormal scores occurred with greatest frequency. It was decided, arbitrarily, that abnormalities found in seventy percent or more of the subjects would be considered worthwhile reporting. It was reasoned that abnormalities occurring with this frequency justifiably could be incorporated into hypotheses for future studies on differences between reading disabled persons and matched controls.

Abnormalities were spread quite evenly over the four frequency ranges involved in these analyses. The following coherence abnormalities were found in seventy percent or more of the subjects: (1) abnormal coherence between one or more combinations of sites P3, T3, T5, O1 (70%); (2) more intrahemispheric coherence abnormalities involving sites P3, T3, T5, O1 (coupled with any other left hemisphere site) than abnormalities involving P4, T4, T6, O2 (70%); (3) more coherence abnormalities within the left posterior quadrant (among sites T3, T5, P3, O1) than within the right posterior quadrant (70%); and (4) an equal to or greater than 1.4-1 ratio of left to right (all

sites) intrahemispheric abnormalities (70%). All subjects had at least one coherence abnormality, and for the majority of subjects (60%) the abnormalities involved many more instances of decreased (as opposed to increased) coherence. Another subject had an equal number of abnormalities involving decreased and increased coherence. Other abnormalities found in seventy percent or more of the subjects were as follows: (1) at least five abnormalities (coherence, phase, amplitude asymmetry) involving frontal/parietal coupling (80%); (2) at least five abnormalities (of any type including relative power) involving site P3 (70%).

Discussion

The patterns of cortical area abnormalities observed in this sample are similar to those which have been found in other EEG, MRI, and autopsy studies of dyslexics. That is, abnormalities occurring most frequently in the left posterior hemisphere (especially parietal and planum areas), and in right parietal and bilateral frontal areas (Hynd & Semrud-Clikeman, 1989; Semrud-Clikeman, Hooper, Hynd, Hern, Presley, & Watson, 1996) Thus, present observations provide yet another in the accumulating pieces of evidence supporting a central nervous system dysfunction perspective on dyslexia. Perhaps most importantly, these data suggest that neural timing (phase, coherence) abnormalities may be more discriminative of dyslexia than EEG amplitude and power which most often have been studied in related research on this population. Being a very neurologically complex act, it is not surprising that various specific brain centers should be involved in reading. For example, the area of the left angular gyrus often has been implicated as an especially important area. There, at the interface of visual, auditory, and kinesthetic/tactual sensory association areas (occipital, temporal, and parietal lobes; approximately site P3 in the International 10-20 EEG Electrode System) much of the sensory integration necessary for successful reading

likely takes place (or fails to do so in dyslexia). Perhaps for successful integration to occur there, and among other brain areas involved in reading, these areas must "communicate" and operate as a system involving precise neural timing among them. As Llinas (1993) puts it, "while such systems may be physically separated in space, they are next to each other in time." Timing such as this implies synchronicity of neural firing among spatially disparate cortical areas—a process which might be disrupted by even relatively minor lesions in certain critical areas, e.g., by the focal cellular abnormalities which consistently have been found in autopsy studies of dyslexics (Hynd, Marshall, & Gonzalez, 1991). Relevant to this, it should be noted that in the present study the majority of coherence abnormalities (both right and left sides) involved abnormally *decreased* coherence, implying abnormally decreased communication between sites. If this reasoning is accurate, persons with specific reading disabilities (and perhaps other learning disabilities) might be referred to more basically as "dysynchronous persons" and might most accurately be identified using measures of neural timing such as QEEG phase, coherence, or some combination thereof. Such reasoning also suggests that neurofeedback may prove to be an especially useful therapy in many cases of dyslexia. If cortical communication or coupling between areas is abnormal due to lesions or other factors interrupting synchronicity, appropriate feedback regarding timing (phase/coherence) might enable some persons essentially to "tune" the cortical systems underlying reading readiness. For example, appropriate feedback might enable adjusting of nerve conduction times to enhance synchronicity, or might motivate development of effective alternative neural pathways between sites.

Obviously, the present study has many weaknesses which limit generalization of findings. For example, the number of subjects was small, the age range was very wide, and at least three were not "pure" dyslexics in that attentional problems, visu-

al-motor integration difficulties, and other problems were present. Future researchers, however, may use the present findings profitably to generate specific hypotheses regarding expected QEEG differences between dyslexic groups and control groups of normally reading persons. Clinicians and researchers experimenting with neurofeedback treatment of dyslexia may wish to use these findings as a guide toward which sites to focus on during training of phase/coherence.

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