*International Journal of Psychophysiology* 1 (1984) 163–177
Elsevier



# EEG SENSORIMOTOR RHYTHM BIOFEEDBACK TRAINING: SOME EFFECTS ON THE NEUROLOGIC PRECURSORS OF LEARNING DISABILITIES

MICHAEL A. TANSEY *

*Private Practice, Union, New Jersey (U.S.A.)*

(Accepted October 12th, 1983)

*Key words:* learning disabilities – electroencephalographic biofeedback training – IQ enhancement – children

This study presents a clinical treatment regime for pathological interhemispheric dysfunction with respect to a population of learning disabled boys. The results obtained replicate and extend earlier findings with respect to operantly conditioned increases in amplitude of sensorimotor transactions and its positive effect on learning disability. Specifically, the biofeedback, and subsequent conditioning, of increased 14 Hz neural discharge patterns (sensorimotor rhythm — SMR) over the central Rolandic cortex, appeared to increase bilateral sensorimotor transactions resulting in substantive reduction/remediation in the learning disabilities of the recipients of such EEG biofeedback training.

## INTRODUCTION

With regard to human brain development and its functional utilization, the brain's synaptic pathways and neural activation matrices are genetically programmed insofar as developmental manifestation, but are also predisposed to functionally respond and further develop with use. This is the basis for all higher learning. It is also an explanation for the effectiveness of electroencephalographic (EEG) biofeedback training. EEG biofeedback training shows tremendous promise as an internal cerebral exerciser. Specifically, via electroencephalographic biofeedback of cerebro-cortical function, the brain is taught to place itself in a self-stimulating posture. Research findings, on EEG Sensorimotor Rhythm biofeedback training indicate that such novel, internal, auto-stimulation of the brain's neural processes results in increased functional efficacy on the part of the cerebro-cortico system undergoing such stimulation (Finley et al., 1975; Seifert and Lubar, 1975; Lubar and Bahler, 1976; Finley, 1977; Sterman, 1977; Shouse and Lubar, 1979; Tansey, 1982; Tansey and Bruner, 1983).

The cerebro-cortico-neural synaptic matrices, which comprise much of our central nervous system, are reactive to the strengthening and modifying action of experiential inputs and functional application. Animal studies have indicated that enriched experiential environments results in increased numbers of dendritic spines within (Parnavelas et al., 1973), as well as increases in the weight of those cortical areas so stimulated (Rosenzweig et al., 1975). Functionally experienced cortical cells were seen to develop synaptic terminals, which functionally inexperienced cortical cells lacked (Pettigrew, 1974). In essence, the animal studies seem to indicate that experiential/functional stimulation may positively alter brain anatomy. The concept that experiential/sensorial and motoric stimulation may positively alter brain anatomy and improve function, in human beings, is the underlying assumption of most of the rehabilitative efforts directed towards the learning impaired. Commonly, the objective of such efforts is the stimulation of the sensorimotor cortex of the

* Address for correspondence: 2810 Morris Avenue, Union, NJ 07083, U.S.A.

brain; by presenting it with an externally produced sensory barrage, whose inputs are expected to have a positive effect on the cerebro-cortico-neural systems processing them. EEG biofeedback training, for the treatment of learning disabilities, also has as its goal the stimulation/exercising of the sensorimotor cortex of the brain, but the cerebro-cortico-neural systems are functionally stimulated from within. Thus, for EEG biofeedback training of the learning impaired, there is the training of increased, discrete, neural discharge over the Rolandic (sensorimotor) cortex of the human brain, with the electrode placement situated so that bilateral sensorimotor transactions may be stimulated from within (Tansey and Bruner, 1983); with resultant increases in potential for higher learning. In function, the sensorimotor cortex produces a 14 Hz neural discharge pattern (brainwave) which is commonly referred to as the sensorimotor rhythm (SMR). As such, the object of EEG sensorimotor rhythm biofeedback training is the production of SMR in increasingly larger amplitude.

Over the past decade, there has emerged a growing literature indicating a commonality, with respect to the neurophysiological precursors and behavioral sequelae, for populations primarily defined as dyslexic, reading disabled, or more generally, learning disabled. Dyslexia has been attributed to an inherited central nervous system dysfunction (Cole, 1979), to have been associated with anatomic differences in cortical architecture (LeMay, 1981), with deficits in short-term memory (Spring, 1976; Aaron, 1978; Deegener, 1979; Nelson and Warrington, 1980; Byrne, 1981), deficits in information processing capability due to a developmental lag in neurophysiologic functioning (Satz et al., 1971; Thompson, 1973; Mindell, 1978; Bakker et al., 1981), a dysfunction in intersensorial integration (Bravo, 1976), and functional asymmetries in the normally complementary contributions of, and inter-hemispheric interactions between, the right and left cerebral hemispheres (Gardner, 1973; Nielson, 1976; Van der Honert, 1977; Witelson, 1977; Bakker, 1979; Cole, 1979; Hier, 1979; Benton, 1980; Gordon, 1980; Bakker et al., 1981).

Populations primarily defined as reading disabled have also been identified as showing an inherited central nervous system dysfunction (Bakwin, 1973; DeFries et al., 1978; Lewitter et al., 1980), to exhibit deficits in short-term memory (Snef and Freundl, 1971; Rugel, 1974; Cummings and Faw, 1976; Cohen and Netley, 1978; Freeman and Beasley, 1978; Moore et al., 1982), deficits in information processing ability due to a developmental lag in neurophysiologic function (Badian and Wolff, 1977; Lovegrove and Brown, 1978), deficits in intersensorial integration (Levine and Fuller, 1972), and functional asymmetries in the normally complementary contributions of, and inter-hemispheric interactions between, the right and left cerebral hemispheres (Yeni-Komshian et al., 1975; Badian and Wolff, 1977; Kershner, 1977; Wheeler et al., 1977; Gross et al., 1978; Kershner, 1979; Pirozzolo and Rayner, 1979; Garren, 1980; Naylor, 1980; Newell and Rugel, 1981).

Populations primarily defined as learning disabled have also been identified as having an inherited central nervous system dysfunction (Silver, 1971), abnormality in cortical architecture (Geschwind, 1979), deficits in short-term memory (Worden et al., 1982), deficits in long-term memory (Bauer, 1977; Batey and Sonnenschein, 1981), deficits in information processing due to a developmental lag in neurophysiologic function (Grotberg, 1970; Rossi, 1972; Swanson, 1981), deficits in intersensorial integration and sensorimotor development (Van Eyck, 1980), and functional asymmetries in the normally complementary contributions of, and interhemispheric interactions between, the right and left cerebral hemispheres (Gomez, 1972; Rourke et al., 1973; Hartlage, 1975; Rourke, 1975; Rourke and Finlayson, 1975; Dean, 1979).

It has been long understood that the brain responds to a complex of sensorial-perceptual-cognitive inputs, as an integrated entity. Neurophysiologically there is a bilateral cerebro-cortico-neural integration of such inputs, as mediated by the corpus callosum, for the reception, processing, storage, retrieval, and ongoing integration of such experience into ongoing adaptive behavior. Therefore, it is not surprising to find functional asymmetries, in the normally complementary contributions of, and inter-hemispheric interactions between, the right and left cerebral hemispheres as a common denominator of dyslexia, reading disorders, and more generally, learning disabilities. It is reasonable, then, to perceive the overlapping nature of the neuropsychophysiological aspects of these learning impediments as sequelae of a generalized deficiency of the bilateral efficacy of the sensorimotor cortex of the brain of the learning impaired; whose manifestations have been erroneously categorized into separate clinical entities.

In this study, it is hypothesized that increased bilateral sensorimotor transactions can be facilitated via EEG biofeedback training of the 14 Hz neural discharge pattern (sensorimotor rhythm) over the Rolandic cortex of the brain. The effects, of such internal cerebral stimulation, are hypothesized to manifest as an increase in the amplitude of the sensorimotor rhythm, and a reduction in the learning disabilities of the recipients of such EEG biofeedback training.

## METHOD

### *Subjects*

The subjects were 6 Caucasian boys, each with a history of learning disabilities. They ranged in age from 10 years 2 months to 11 years 10 months.

### *Apparatus and Procedure*

EEG biofeedback training of the sensorimotor rhythm was conducted in weekly, 30 min, training sessions. A single channel electroencephalograph was used to assist the subjects in emitting the $14 \pm 0.5$ Hz neural discharge rhythm over the central Rolandic cortex. The monitored 14 Hz neural discharge signal (SMR) was protected via filtering from contamination and low amplitude scalp EMG.

In monitoring the sensorimotor rhythm (SMR), 3 saline electrodes are used (impedance levels in saline of 1 k$\Omega$). Electrode contact with the skin/scalp surface is checked with, built-in electrode check, LCD indicators. To monitor, and subsequently train, bilateral sensorimotor discharge patterns, the active electrode is placed so that its $6.5 \times 1.3$ cm contact surface lays lengthwise along the midline of the top of the skull (overlaying the cerebral longitudinal fissure), centering about 2.6 cm behind Cz (10/20 system). In this position, the active electrode centers over the Rolandic cortex (pre- and postcentral gyri) of both the right and left cerebral hemispheres. The reference and ground electrodes are placed on opposite ears via earclips (see Fig. 1).

The monitored SMR was then transmitted to the electroencephalograph for signal processing [1] and subsequent auditory feedback. The single channel EEG unit provided both amplitude and frequency modulated audio feedback. The feedback tone is modulated so that the greater the amplitude of the brainwave the louder the tone. In addition, the repetition rate of the tone (the number of beats per second) covaried with the rate of occurrence of the monitored brainwave as it exceeded threshold. The phase shift of the unit is set for 225°C. This phase shift setting seems to time the occurrence of the audio feedback signal so that it is perceived as being more in synchrony with the trainee's sense of brainwave production. For all subjects, the monitored SMR was submitted to on-line computer analysis. Evaluations of the changes in the mean value of SMR amplitude, across each training session, was recorded as a performance measure of the acquisition of the operant task — autostimulation of the sensorimotor cortex resulting in increases in the amplitude levels of its neural discharge pattern. The EEG sensorimotor rhythm training sessions were scheduled weekly. Each training session commenced with a 10–15 min review of each boy's status for the previous week. EEG biofeedback training was performed for 30 min. Instructions during the EEG biofeedback training sessions were presented to the boys while they were in a reclining position with their eyes closed. The instructions were: 'Now, let yourself become hollow and heavy. Just let yourself be a heavy, hollow rock; quiet, hollow, and heavy — and let the beeps come out'. Intermittent, positive reinforcement (verbal praise for 'beep' production) was provided every few minutes. Every 10 min, the initial-orientation instructions were repeated. A contract was made to

[1] Integrated Biofeedback Systems, 100 Mercer Street, P.O. Box 1148, Hightstown, NJ 08520, U.S.A.

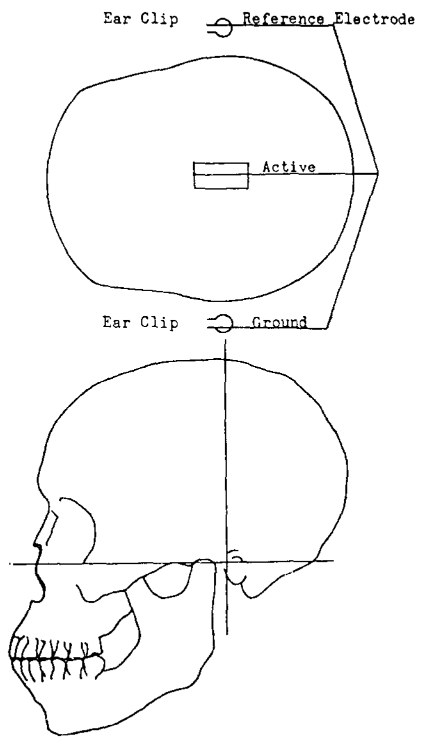


Fig. 1. Diagram of EEG electrode placement.

provide a tangible reward, in the form of a 'Matchbox' car, at the end of each EEG sensorimotor rhythm training session, contingent upon the boy being trained having 'let a lot of beeps come out'.

A multi-subject, A-B-A, experimental design was utilized to assess the impact of operantly conditioned increases of the 14 Hz neural discharge rhythms, as monitored over the sensorimotor (Rolandic) cortex of the brain, where the active electrode was situated so as to maximally facilitate bilateral hemispheric transactions and conditioning. In search of relations between monitored cerebro-cortico-neural events and ongoing behavior, the pretreatment baselines, against which to evaluate the effects of hypothesized altered cerebral function, were selected as follows: (1) an assessment of each boy's learning disability; and (2) the administration of the Wechsler Intelligence Scale for Children — Revised (WISC-R).

During the course of the EEG sensorimotor rhythm biofeedback training, the subjects did not engage in any additional therapies for their learning impediments other than detailed in the individual case profiles (assignment to a special class or resource room etc.). In any case, these other therapeutic activities were of documented ineffectiveness insofar as impacting on the recipients learning impediment, and were carried out without the knowledge of the subjects having undergone EEG Sensorimotor Rhythm biofeedback training. As such, all school derived evaluations of learning disability/ability and intellectual functioning were unbiased measures. While much of the WISC-R post-testing was performed by the author, and by definition was not 'blind', the strong construct validity and reliability of the WISC-R, with the standardization of its presentation and objectivity of its scoring, seems to inherently limit the variability of I.Q. scores after retesting. Even if the same person, or someone else, performs the pre- and post-testing, 'all children within the entire range covered by the WISC-R may be expected to gain about 7 points in their full scale I.Q. on a retest after a month has elapsed'. (Kaufman, 1979, p. 49, Wechsler, 1974). These factors will be taken into consideration in evaluating the results.

## RESULTS

### *Acquisition of the operant task: enhancement of sensorimotor rhythm*

The, pre- and post-EEG biofeedback training, mean values of SMR for each subject are presented in Fig. 2. These positive alterations in SMR amplitude serve to illustrate, on the part of the subjects, a learned operant control/conditioning of the neural discharge pattern emanating from the sensorimotor cortex of their brains. The mean amplitude of SMR increased 300% over baseline for Case 1, 50% over baseline for Case 2, 39% over baseline for Case 3, 38% over baseline for Case 4, 279% over baseline for Case 5, and 120% over baseline for Case 6. As a group, the mean amplitude of SMR increased 137.6% over baseline.

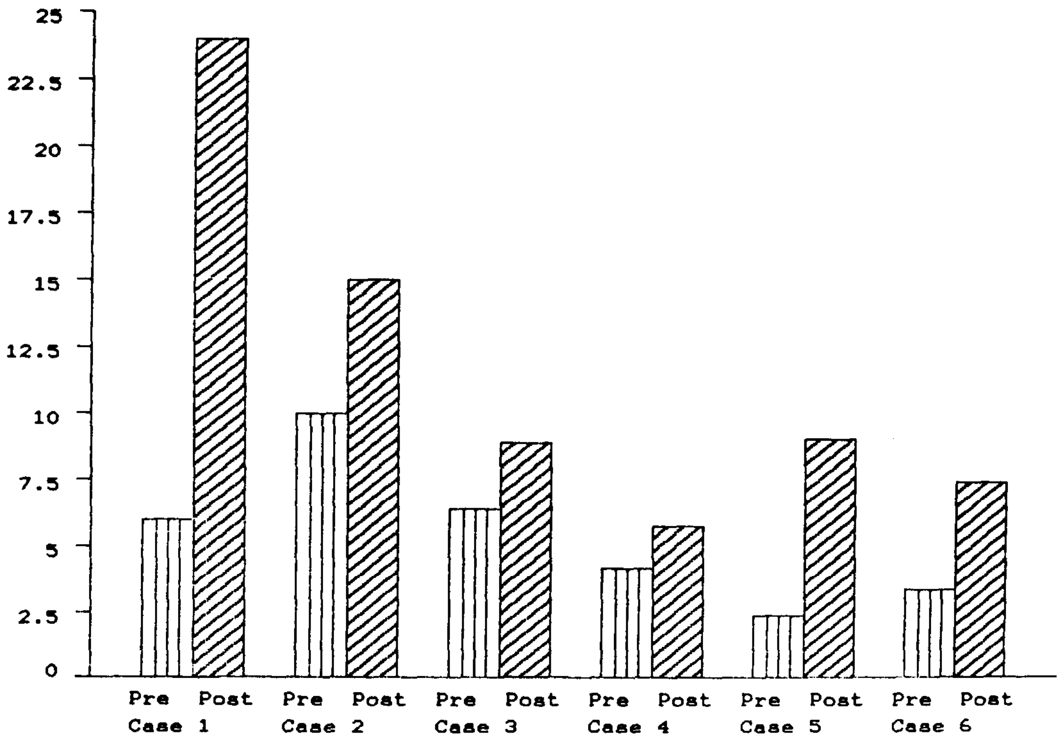


Fig. 2. Pre- and post-EEG biofeedback training amplitude measures of sensorimotor rhythm.

*The Wechsler Intelligence Scale for Children – Revised pre- and post-EEG biofeedback training profiles*

The pre, post, and difference scores, obtained by each subject are presented in Table I. Statistical criterion for significance are those provided by Wechsler (1974). All 6 subjects manifested increases in their full scale I.Q. scores of at least one standard deviation (15 I.Q. points). Both verbal and performance I.Q. scores increased for all subjects. As a group, their post-EEG biofeedback training scores, substantially exceeded their pre-EEG biofeedback training scores over and above any expected gains due to a retest factor; i.e. more than 3.5 points increase in verbal I.Q., more than 9.5 points increase in performance I.Q., and more than 7.0 points increase in full scale I.Q. (Kaufman, 1979). In addition, subjects with either a significant — greater than 15 points — ($P < 0.01$) verbal > performance I.Q. (Cases 1 and 6) or performance > verbal I.Q. (Cases 2 and 4) discrepancy exhibited a substantially greater increase in the lower of the two I.Q. scores (See Table I). Specifically, the increase in Case 1's performance I.Q. score was 60% greater than that of his verbal I.Q. score, and Case 6's performance I.Q. score showed an 84% greater increase; with the increase in Case 2's verbal I.Q. score being 90% greater than the increase in his performance I.Q. score, and Case 4's showed a 257% greater increase. Further analysis of these WISC-R profiles and data will be included in the reassessment of the subject's behavioral, academic, and intellectual functioning.

*Case profiles*

*Case 1*

Case 1 is a 10 year 7 month old Caucasian male, in the 4th grade, with a history of perceptual deficits since kindergarten; where he had notable

TABLE I

*WISC-R profiles*

| | Scale scores | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | *Case: 1* *(Boy, 10 years 7 months)* | | | *2* *(Boy, 11 years 7 months)* | | | *3* *(Boy, 10 years 8 months)* | | |
| | *Pre* | *Post* | *Difference* | *Pre* | *Post* | *Difference* | *Pre* | *Post* | *Difference* |
| Verbal | | | | | | | | | |
| Information | 12 | 12 | 0 | 5 | 10 | +5 | 4 | 4 | 0 |
| Similarities | 11 | 16 | +5 | 16 | 19 | +3 | 6 | 10 | +4 |
| Arithmetic | 9 | 9 | 0 | 6 | 11 | +5 | 6 | 6 | 0 |
| Vocabulary | 12 | 12 | 0 | 12 | 12 | 0 | 5 | 7 | +2 |
| Comprehension | 12 | 15 | +3 | 9 | 13 | +4 | 9 | 14 | +5 |
| (Digit Span) | 7 | 12 | +5 | 5 | 10 | +5 | 3 | 5 | +2 |
| Performance | | | | | | | | | |
| Picture Completion | 7 | 13 | +6 | 13 | 13 | 0 | 9 | 11 | +2 |
| Picture Arrangement | 11 | 11 | 0 | 12 | 13 | +1 | 11 | 14 | +3 |
| Block Design | 6 | 9 | +3 | 14 | 16 | +2 | 8 | 9 | +1 |
| Object Assembly | 5 | 8 | +3 | 13 | 16 | +3 | 6 | 8 | +2 |
| Coding | 8 | 8 | 0 | 13 | 15 | +2 | 2 | 6 | +4 |
| Verbal I.Q. | 107 | 117 | +10 | 97 | 118 | +21 | 75 | 88 | +13 |
| Performance I.Q. | 82 | 98 | +16 | 121 | 132 | +11 | 81 | 96 | +15 |
| Full scale I.Q. | 94 | 109 | +15 | 109 | 128 | +17 | 76 | 91 | +15 |

difficulty in the copying of letters, in the making of designs, and in the manipulation of scissors. As of Grade 2, he was placed in a class for the perceptually impaired. His classification as educationally handicapped: perceptually impaired was further supported in Grade 4, whereupon testing revealed him to have a 3 year developmental lag in visual-motor integration concurrent with information processing deficits inhibiting academic function. Reading skills were 1 year below expectancy. At the same time, his WISC-R profile showd him to be functioning within the average range of intellectual ability, but there was a 25 point discrepancy between his verbal (107) and performance I.Q. (82) in favor of his verbal I.Q. score. Shortly after this evaluation, this boy undertook EEG sensorimotor rhythm biofeedback training. His WISC-R scores were accepted for utilization in his pretreatment baselines (See Table I).

Case 1's mean amplitude of SMR increased from a baseline of 6 $\mu$V to 24 $\mu$V by his 24th EEG biofeedback training session. Concurrent with this 300% increase in amplitude of sensorimotor rhythm, he exhibited a shift from documented academic underachievement with a developmental reading disorder, to age appropriate academic growth and reading ability. As of his 24th EEG biofeedback training session, he was retested within the school setting (at the third month of Grade 5) and found to have a reading comprehension grade level of 5.8, a reading recognition grade level of 6.4, and a spelling grade level of 5.0. At the same time he was readministered the WISC-R. His verbal I.Q. had increased, 10 points, from average (107) to high average – bright (117). His performance I.Q. had increased, 16 points, from low average – dull (82 — 3 points above borderline) to average (98). His full scale I.Q. had increased, 15 points, from mid (94) to the upper limit of the average range of intellectual functioning (109).

*Case 2*

Case 2 is an 11 year 7 month old Caucasian male who undertook EEG sensorimotor rhythm biofeedback training shortly after a psychoeducational evaluation while in Grade 3. The child study

*Scale scores (continued)*

| *4 (Boy, 11 years 10 months)* | | | *5 (Boy, 11 years 1 month)* | | | *6 (Boy 10 years 2 months)* | | |
|---|---|---|---|---|---|---|---|---|
| *Pre* | *Post* | *Difference* | *Pre* | *Post* | *Difference* | *Pre* | *Post* | *Difference* |
| 12 | 15 | +3 | 8 | 9 | +1 | 13 | 13 | 0 |
| 10 | 13 | +3 | 11 | 18 | +7 | 12 | 16 | +4 |
| 7 | 14 | +7 | 9 | 13 | +4 | 13 | 14 | +1 |
| 8 | 12 | +4 | 11 | 11 | 0 | 10 | 11 | +1 |
| 13 | 17 | +4 | 12 | 16 | +4 | 12 | 17 | +5 |
| 8 | 10 | +2 | 9 | 11 | +2 | 10 | 10 | 0 |
| 13 | 14 | +1 | 11 | 12 | +1 | 6 | 12 | +6 |
| 16 | 16 | 0 | 13 | 14 | +1 | 9 | 14 | +5 |
| 17 | 19 | +2 | 9 | 10 | +1 | 9 | 12 | +3 |
| 15 | 15 | 0 | 7 | 13 | +6 | 11 | 11 | 0 |
| 11 | 13 | +2 | 13 | 14 | +1 | 9 | 12 | +3 |
| 100 | 125 | +25 | 101 | 120 | +19 | 112 | 125 | +13 |
| 131 | 138 | +7 | 104 | 118 | +14 | 91 | 115 | +24 |
| 116 | 136 | +20 | 102 | 122 | +20 | 102 | 123 | +21 |

team deferred classification and/or official action despite findings of this boy's not performing up to his ability, having difficulty focusing on directions, to be deficient in auditory memory and reading comprehension. His reading level was 1 year below expectancy. His WISC-R profile showed him to be functioning within the average range of intellectual ability, but there was a 24 point discrepancy between his verbal (97) and performance I.Q. (121) in favor of his performance I.Q. score. In addition, while engaged in performing mathematics, he was noted to be silent, and, when silently dealing with mathematical concepts and assignments, to maintain a B average on his written tests. Yet, if he was asked to verbally solve a mathematics problem, he appeared unable to comply. Such behavior prompted his teacher to reduce his mathematics grade from his test average of B to a C because of his not being able to verbally demonstrate his knowledge of the concepts used to arrive at his answers. These findings/behavior held true through the first half of his Grade 4 experience; whereupon, he undertook EEG sensorimotor rhythm biofeedback training. His WISC-R scores were accepted for utilization in his pretreatment baselines (See Table I).

Case 2's mean amplitude of SMR increased from a baseline of 10 $\mu$V to 15 $\mu$V by his 9th EEG biofeedback training session. Concurrent with this 50% increase in amplitude of sensorimotor rhythm, he exhibited notable improvement in auditory memory and reading ability. His handling of written material progressed to where he reported that 'I worked great for two solid hours in reading. I usually get tired after fifteen minutes. I feel great! I wasn't even tired. It was easier to think. Before, my brain would clunk out on me and then I'd talk instead of read. Now its (reading) comfortable'. Reports with respect to his class behavior echoed his assertions of improved function. One such report related that: 'Boy, has he picked up academically! He pays much more attention now. Before, he couldn't verbalize mathematics and be correct. Now, he's winning all the verbal tests in Math'. As of his 9th EEG sensorimotor rhythm biofeedback training session, he expressed a sense of academic well being and a wish to terminate his training sessions. His mother concurred and he was readministered the WISC-R at that time. His verbal I.Q. had increased, 21 points, from mid average (97) to the upper end of high average – bright (118). His performance I.Q. had increased, 11 points, from the lower end of the superior (121) to the very superior (132). His full scale I.Q. increased, 17 points, from the upper end of the average (109) to the upper end of the superior (128) range of intellectual functioning.

*Case 3*

Case 3 is a 10 year 8 month old Caucasian male, in a Grade 2 placement for the neurologically impaired. He did not speak until the age of 3 years 6 months, repeated kindergarten, and was placed in an 'educable class for minimal brain impaired children' (neurologically impaired) as of Grade 1. His neurological impairment was diagnosed as including an organic pattern of hyperkinesis, developmental delays in cognitive function, deficits in reading and language development in excess of 1 year below expectancy, deficits in both visual-motor and verbal-expressive modalities, and a borderline-retardate level of intellectual functioning. As of Grade 2, his 2nd year in a class for the neurologically impaired, his diagnostic classification was ammended to read multiply handicapped: emotionally disturbed and neurologically impaired. His intellectual ability was assessed to be at 'the top of the range of educable mental retardation'. Three months into his Grade 2 experience, he undertook EEG sensorimotor biofeedback training. He evidenced, upon intake, difficulties in saccadic fixation and ocular pursuit movements. Specifically, he was unable to move his eyes smoothly along a horizontal axis with his head in a stationary position. In addition, he could not move his head from side to side while keeping his eyes fixated on an object held before him. For this boy, attempts at reading entailed his moving his head side to side in order to read the words along a line of print. This visual approach style was evidenced by his skipping words, not noticing his missing the commas and periods, and a consistent inability to understand what it was that he had just 'read'. Even though he had been promoted to a Grade 2 class for the neurologically impaired, he had not yet attained a Grade 1 reading level. At this time, he had been maintained on Ritalin (methylphenidate) for his hyperactivity since the age of 5 years 9 months. He presented as alert, energetic, and posessed of a verbal-expressive ability which did not correspond to that expected of the educable mentally retarded. So, he was administered the WISC-R prior to his EEG biofeedback training. He was found to be functioning within the Borderline range of intellectual functioning. His verbal, performance, and full scale I.Q. scores were 75, 81 and 76, respectively. These scores were used as part of his pretreatment baselines (See Table I).

Case 3's mean amplitude of SMR increased from a baseline of 6.4 $\mu$V to 8.9 $\mu$V by his 18th EEG biofeedback training session. Concurrent with this 39% increase in amplitude of sensorimotor rhythm, he exhibited notable improvement in his behavioral, academic, and intellectual measures of performance. As of his 7th EEG sensorimotor rhythm biofeedback training session, where his average SMR amplitude had increased to 42% over baseline, it was noted that he had become able to move his head from side to side while keeping his eyes fixated on an object held before him. As of his 9th EEG biofeedback training session, where his average SMR amplitude had increased to 45% over baseline, it was noted that he could then be able to smoothly move his eyes along a horizontal axis with his head in a stationary position. Within 3 weeks of this improvement in fine motor/ocular control, his school progress was related as follows: 'His greatest gain is that now, he is reading a first grade book. He is becoming more self-confident and satisfied with this accomplishment'. Four weeks later, as of his 14th EEG biofeedback training session, his mother related that at home: 'He wants to read. He read three short stories last night in a Grade 2 book and was very anxious to read for me. He did very well'. In addition, as of his 14th EEG biofeedback training session, his mother reported that: 'I took him off his Ritalin 3 days ago and he's doing even better off it'. He continued to improve behaviorally and academically for the next 4 weeks, whereupon his mother decided to cease his EEG biofeedback training sessions with the onset of summer. The week following his 18th EEG sensorimotor rhythm training session, he was readministered the WISC-R. His verbal I.Q. had increased, 13 points, from mid borderline (75) to the upper end of low average (88). His performance I.Q. had increased, 15 points, from the lower end of low average (81) to the middle range of average (96). His full scale I.Q. increased, 15 points, from mid borderline (76) to the lower end of the average (91) range of intellectual functioning.

*Case 4*

Case 4 is a 11 year 10 month old Caucasian male, in Grade 6, with a history of dyslexia, visual-motor, perceptual, attentional, and verbal-expressive deficits since Grade 2. In the final quarter of Grade 5, he received his first tri-yearly re-evaluation, which further supported his classification of neurologically impaired. His WISC-R profile showed him to be functioning within the high average – bright range of intellectual ability, but there was a 31 point discrepancy between his verbal (100) and performance (131) I.Q. in favor of his performance I.Q. score. He was scheduled to be promoted to Grade 6, and to be continued in a regular class placement with assignment to a resource room for remediation in reading, spelling, and language arts. He undertook EEG sensorimotor rhythm biofeedback training in the 2nd month of his Grade 6 experience. At that time his reading level was assessed to be at the 4.5 grade level, his spelling ability to be at the 2.7 grade level, and arithmetic skills to be at the 4.3 grade level; as assessed by the Wide Range Achievement Test (WRAT). His school's re-evaluation WISC-R scores were accepted for utilization in his pretreatment baselines (see Table I).

Case 4's mean amplitude of SMR increased from a baseline of 4.2 $\mu$V to 5.8 $\mu$V by his 22nd EEG biofeedback training session. Concurrent with this 38% increase in amplitude of sensorimotor rhythm, the exhibited notable improvement in his dyslexia, verbal articulation skills, and reading ability. There were no noted behavioral changes until his 9th EEG biofeedback training session; whereupon, his Learning Disability Teacher Consultant phoned this boy's parents to report that, in English class, starting 3 weeks earlier, he had spontaneously 'understood what he had just read so much better — it was as if a light went on inside him'. During this same period of time, his average amplitude of SMR had increased from 19% to 33% over baseline. As of his 10th EEG training session, it was reported that 'he can now remember 7 digits in a row. Before, it was impossible. He'd always bomb out at 4 or 5 digits'. At this time, it was also noted that his handwriting skills had improved and that it no longer contained the letter reversals so characteristic before. These positive remediations continued up through his 22nd EEG sensorimotor rhythm biofeedback training session, where, it was decided to reassess his ability to reading, spell, and do arithmetic, as well as his WISC-R scores. His reading ability had increased from the previous 4.5 to the 6.0 grade level. His ability to spell increased from the previous 2.7 to the 3.3 grade level. His ability to do mathematics went from the previous 4.7 to the 8.1 grade level (WRAT). This testing was performed in the 8th month of Grade 5. In addition, his verbal I.Q. had increased, 25 points, from average (100) to superior (125). His performance I.Q. had increased, 7 points, (from 131 to 138) staying within the superior range. His full scale I.Q. increased, 20 points, from high average – bright (116) to the very superior (136) range of intellectual functioning.

*Case 5*

Case 5 is an 11 year 1 month old Caucasian male, in Grade 6 with a history of academic difficulty since Grade 1. He missed a significant amount of school instruction due to onsets of pneumonia, bronchitis, allergies, and asthma throughout Grades 1, 2, 3 and 4. In Grade 5, he had his adenoids and tonsils removed with a resultant increase in his school attendance. Even though he had attended summer school for every summer of his school experience, he was found to be functioning well below grade level in reading and mathematics. He was promoted to Grade 6 with assignment to a resource room two periods daily for remediation of language arts, reading, and spelling. He undertook EEG sensorimotor rhythm biofeedback training just prior to his entering Grade 6. On entrance, he was administered the test of written language (TOWL) which showed him to have a grade level ability of 3.4 in thematic maturity, 3.4 in spelling, 7.0 in word usage, 5.2 in style, 2.2 in thought units, and 4.6 in handwriting. Independent reading was at the 2.0 grade level for both silent and oral reading; with his instructional level being at the 3.5 grade level for both silent and oral reading (Houghton–Mifflin Individual Reading Inventory). His WISC-R profile showed him to be functioning within the average range of intellectual ability. His verbal, performance, and full scale I.Q. scores were 101, 104 and 102, respectively. His WISC-R scores were accepted for utilization in his pretreatment baselines (see Table I). He was also noted to evidence, upon intake, difficulties in saccadic fixation and ocular pursuit movements. Specifically, he could not move his eyes smoothly along a horizontal axis with his head in a stationary position. This ocular motor dysfunction was accompanied by his skipping words, missing commas and periods, and a consistent inability to understand what it was that he had just 'read'.

Case 5's mean amplitude of SMR increased from a baseline of 2.4 $\mu$V to 9.1 $\mu$V by his 34th EEG biofeedback training session. Concurrent with this 279% increase in amplitude of sensorimotor rhythm, he exhibited notable improvement in his academic and intellectual measures of performance. As of his 5th EEG sensorimotor rhythm biofeedback training session, where the average amplitude of SMR was 160% over baseline, it was noted that he had become able to smoothly move his eyes along a horizontal axis with his head in a stationary position. At the same time, he reported that 'my reading is getting better'. He continued to increase his average SMR amplitude over the course of his 34 EEG biofeedback training sessions, and this positive neurologic reorientation was accompanied by consistent reports of his improvement in reading, spelling, and mathematics. In the 8th month of his Grade 6 experience, he was retested at school. The school personnel were not aware that he had been receiving EEG sensorimotor rhythm biofeedback training sessions. His TOWL spelling level had increased from the 3.4 to the 7.2 grade level, with the word usage ability having increased from the 7.0 to the 8.5 grade level, the style subscale having increased from the 5.2 to the 9.3 grade level, the thought units measure having increased from the 2.2 to the 3.8 grade level, and the handwriting measure having increased from the 4.6 to the 9.5 grade level. His silent reading ability increased from the 2.0 to the 4.0 grade level in independent reading, from the 3.5 to the 6.5 grade level in instructional level, and from the 5.0 to the 8.0 grade level in frustration level. His oral reading ability increased from the 2.0 to the 5.0 grade level in independent reading, from the 3.5 to the 6.5 grade level in instructional level, and from the 5.0 to the 8.0 grade level in frustration level (Ekwall Individual Reading Inventory). Concurrent with this academic reevaluation, he was readministered the WISC-R. His verbal I.Q. had increased, 19 points, from average (101) to the lower end of superior (120). His performance I.Q. had increased, 14 points, from average (104) to the upper end of high average–bright (118). His full scale I.Q. had increased from average (102) to the lower end of the superior (122) range of intellectual functioning.

*Case 6*

Case 6 is a 10 year 2 month old Caucasian male, in Grade 5, with a history of poor academic performance since Grade 3. At the conclusion of Grade 4, a psychoeducational evaluation noted his having exhibiting deficits in visual-motor and fine motor skills. His reading ability was assessed to be at the 3.6 grade level, with his spelling ability assessed to be at the 2.1 grade level, and his mathematics ability assessed to be at the 4.9 grade level. His WISC-R profile showed him to be functioning within the average range of intellectual ability, but there was a 21 point discrepancy between his verbal (112) and performance I.Q. (91) in favor of his verbal I.Q. score. His WISC-R scores were accepted for utilization in his pretreatment baselines (See Table I). Classification and/or the scheduling of remedial services was deferred and he was promoted to a regular Grade 5 class. One month into his Grade 5 experience, he undertook EEG sensorimotor rhythm biofeedback training. He evidenced upon intake difficulties in saccadic fixation and ocular pursuit movements.

Specifically, he was unable to move his eyes smoothly along a horizontal axis with his head in a stationary position. His reading style was evidenced by his skipping words, not noticing his missing commas and periods, and a consistent inability to understand what it was that he had just 'read'. In addition, his handwriting was characterized by an inability to 'stay on the line'.

Case 6's mean amplitude of SMR increased from a baseline of 3.4 $\mu$V to 7.5 $\mu$V by his 19th EEG biofeedback training session. Concurrent with this 120% increase in amplitude of SMR, he exhibited notable improvement in his fine motor skills and reading ability. As of his 5th EEG sensorimotor rhythm biofeedback training session, he received his report card for the first academic quarter. His reading grade was a B. It was noted by his regular school teacher that: 'his reading ability has definitely improved — especially comprehension in the first 5 weeks. His memory is much improved. He now knows what he reads'. For that same initial 5 week period, his mean amplitude of SMR had increased to 50% over baseline. Two weeks later, his father noted that: 'his Hebrew teacher said that, over the past 4 weeks, he had a significant improvement in his Hebrew reading'. This boy continued to improve in both his regular and Hebrew school functioning. As of his 8th EEG biofeedback training session, it was noted that: 'now he writes on the lines — before it was all over', as of his 9th EEG biofeedback training session, that he became able to smoothly move his eyes along a horizontal axis while keeping his head stationary, and of his 10th EEG biofeedback training session that 'he no longer skips words as he reads'. As of his 19th EEG sensorimotor rhythm biofeedback training session, his functioning seemed to have stabilized and his reading, spelling, mathematics, and intellectual abilities were reassessed. His reading ability had increased from the 3.6 to the 7.2 grade level, his spelling ability had increased from the 2.1 to the 6.4 grade level, and his mathematics ability had increased from the 4.9 to the 5.5 grade level (WRAT). This testing was performed in the 9th month of Grade 5. His verbal I.Q. had increased, 13 points, from high average–bright (112) to superior (125). His performance I.Q. had increased, 24 points, from the lower end of average (91) to high average–bright (115). His full scale I.Q. had increased, 21 points, from average (102) to the superior (123) range of intellectual functioning.

## DISCUSSION

The results of this study verify and extend previous findings with respect to this EEG biofeedback training procedure and its effects on learning disabilities (Tansey and Bruner, 1983). The results, themselves, have been presented in a manner so as to adequately convey both the quantitative performance measure changes (SMR mean amplitude and WISC-R/academic testing) and the qualitative effects of EEG sensorimotor rhythm biofeedback training on the idiosyncratic psychoeducational status of each subject. Overall, the main effect of this procedure, for the biofeedback and subsequent conditioning of increased 14 Hz neural discharge patterns over the Rolandic cortex, seems to increase bilateral sensorimotor transactions resulting in substantive reductions/remediation in the learning disabilities of the recipients of such training.

It is noteworthy that those subjects, with either a significant ($> 15$ points, $P < 0.01$) verbal greater than performance or performance greater than verbal I.Q. score discrepancy, exhibited no less than a 60% greater increase in the lower of the two I.Q. scores. This finding is, in itself, a strong argument for the bilateral effects of this EEG biofeedback training procedure. The WISC-R verbal and performance I.Q.'s have long been acknowledged as reflecting left and right cerebral hemispheric function, respectively; with significant differences between them reflective of a greater functional dependency on one or the other cerebral hemisphere underlying all behavior. Thus, ongoing research has linked perceptual-motor dysfunction with significant verbal greater than performance I.Q. discrepancies (Kinsborne and Warrington, 1963; Rourke and Telegdy, 1971). Cases 1 and 6 exhibit such a pattern of learning disability and WISC-R profile. Of note is that the neurologically depressed performance I.Q. scores of both cases 1 and 6 showed 60% and 84% greater increase, respectively, than their verbal I.Q. score, and that this trend towards functional cerebral equalization concurred with a substantive reduction/remediation of their learning disabilities. Research has further linked the opposite state of affairs, a significant performance greater than verbal I.Q. discrepancy, with a verbal-expressive dysfunction (Hewitt and Massey, 1969; Weiner, 1969; Killian, 1971). Cases 2 and 4 exhibit such a pattern of learning disabilities and WISC-R profile. Again, the neurologically depressed I.Q. score — this time verbal — for Cases 2 and 4 showed 90% and 257% greater increase, respectively, than their performance I.Q. score, and that this trend towards functional cerebral equalization concurred with a substantive reduction/remediation of their learning disabilities, Overall, these results indicate that this EEG sensorimotor rhythm training procedure not only seems to facilitate increased sensorimotor transactions (SMR amplitude) but also to promote increased functional symmetry in the interhemispheric interactions between the right and left cerebral hemispheres for this population of learning disabled boys.

The results of this study also replicate earlier findings with respect to the effect of increased sensorimotor transactions and oculo-motor dysfunction (Tansey and Bruner, 1983). Specifically, Cases 3, 5 and 6 manifested a remediation of specific oculomotor dysfunction following this EEG sensorimotor rhythm biofeedback training procedure. As stated in that study, while there is little argument as to the discomfort and fatigue that ocular anomalies lend to a reading task, there is a growing literature concluding that reading disorders stem from deficits in information processing, in essence a symbolic learning disorder rooted in cerebral dysfunction, rather than being caused by oculomotor dysfunction. At very best, the literature shows oculomotor anomalies to be a concurrent dysfunction whose manifestation is determined by deficits of volitonal control and internal coordination of the eye muscles. Developmental reading disorders are then viewed in terms of the efficacy of cerebral mechanisms that store visually derived information and its selective retrieval (Benton, 1975; Critchley, 1970; Fox et al., 1975; Lawson, 1968). In this context, we may entertain the possibility that internal stimulation of the sensorimotor cortex had, as a side benefit, the increasing integration/coordination of fine motor skills.

This study presents a clinical treatment regime for pathological interhemispheric dysfunction with respect to a population of learning disabled boys. In this, the treatment does not rely on complicated and exorbitantly expensive equipment neither must it be performed in an isolated laboratory setting. This EEG biofeedback training procedure utilizes compact, portable equipment, and the training itself was performed in a clinical office setting. It is hoped that this paper will serve as a useful pilot for further studies with larger populations and, in this manner, increase our knowledge of the link between the brain and human behavior.

## REFERENCES


Aaron, P.G. (1978) Dyslexia, an imbalance in cerebral information-processing strategies. *Percept. Mot. Skills*, 47: 699–706.

Badian, N.A. and Wolff, P.H. (1977) Manual asymmetries of motor sequencing in boys with reading disability. *Cortex*, 13: 343–349.

Bakker, D.J. (1979) Hemispheric differences in reading strategies: Two dyslexias? *Bull. Orton Soc.*, 29: 84–100.

Bakker, D.J., Moerland, R. and Goekoop-Hoetkins, M. (1981) Effects of hemisphere-specific stimulation on the reading performance of dyslexic boys: a pilot study. *J. clin. neuropsychol.*, 3: 155–159.

Bakwin, H. (1973) Reading disability in twins. *Develop. Med. Child Neurol.*, 15: 184–187.

Batey, O.B. and Sonnenschein, S. (1981) Reading deficits in learning disabled children. *J. appl. develop. Psychol.*, 2: 237–246.

Bauer, R.H. (1977) Memory processes in children with learning disabilities: evidence for deficient rehearsal, *J. exp. Child Psychol.*, 24: 415–430.

Benton, A.L. (1975) Developmental dyslexia: neurological aspects. In W.J. Friedlander (Ed.), *Advances in Neurology, Vol. 7* Raven Press, New York.

Benton, A.L. (1980) Dyslexia: evolution of a concept. *Bull. Orton Soc.*, 30: 10–26.

Bravo, V.L. (1978) Dyslexia: Neuropsychological and psycholinguistic focus. *Revista Chilena de Psicologia*, 1: 7–16.

Byrne, B. (1981) Reading disability, linguistic access and short-term memory: comments prompted by Jorm's review of developmental dyslexia, *Austr. J. Psychol.*, 33: 83–95.

Cohen, R.L. and Netley, C. (1978) Cognitive deficits, learning disabilities, and WISC Verbal-Performance consistency, *Develop. Psychol.*, 14: 624–634.

Cohen, R.L. and Netley, C. (1981) Short-term memory deficits in reading disabled children, in the absence of opportunity for rehersal strategies, *Intelligence*, 5: 69–76.

Cole, E.M. (1979) Dyslexia: implications for diagnosis and treatment. *Bull. Orton Soc.*, 29: 205–224.

Critchley, M. (1970) *The Dyslexic Child*, Thomas, Springfield, IL.

Croxen, M.E. and Lytton, H. (1971) Reading disability and difficulties on finger localization in right–left discrimination, *Develop. Psychol.*, 5: 256–262.

Cummings, E.M. and Faw, T.T. (1976) Short-term memory and equivalence judgments in normal and retarded readers, *Child Develop.*, 47: 286–289.

Davis, S.M. and Bray, N.M. (1975) Bisensory memory in normal and reading disabled children. *Bull. Psychon. Soc.*, 6: 572–574.

Dean, R.S. (1979) Cerebral laterality and verbal-performance discrepancies in intelligence. *J Sch. Psychol.*, 17: 145–150.

Deegener, G. (1979) Functional hemispheric asymmetry of dyslexic children: evaluation of a dichotic listening test. *Prax. Kinderpsychol. Kinderpsychiatr.*, 28: 254–260.

DeFries, J.C., Singer, S.M., Foch, T.T. and Lewitter, F. (1978) I. Familial nature of reading disability, *Brit. J. Psychiat.*, 132: 361–367.

Ellis, N.C. and Miles, T.R. (1977) Dyslexia as a limitation in the ability to process information. *Bull. Orton Soc.*, 27: 72–81.

Finley, W.W., Smith, H.A. and Etherton, M.D. (1975) Reduction of seizures and normalization of the EEG in a severe epileptic following sensorimotor biofeedback training: preliminary study. *Biol. Psychol.*, 2: 189–203.

Finley, W.W. (1977) Operant conditioning of the EEG in two patients with epilepsy: methodologic and clinical considerations. *Pavlovian J. biol. Sci.*, 12: 93–111.

Fox, F.J., Orr, R.R. and Rourke, B.P. (1975) Short-comings of the standard optometrics visual analysis for the diagnosis of reading problems. *Canad. J. Optometry*, 2: 189–203.

Freeman, B. and Beasley, D.S. (1978) Discrimination of time altered-sentential approximations and monosyllables by children with reading problems. *J. Speech Hear. Res.*, 21: 497–506.

Gardner, H. (1973) Developmental dyslexia: the forgotten lesson of Monsieur C. *Psychol. Today*, 7: 62–67.

Garren, R.B. (1980) Hemispheric laterality differences among four levels of reading achievement. *Percept. Mot. Skills* 50: 119–123.

Geschwind, N. (1979) Asymmetries of the brain — new developments. *Bull. Orton Soc.*, 29: 67–73.

Gomez, M.R. (1972) Specific learning disorders in childhood. *Psychiat. Ann.*, 2: 49–65.

Gordon, H.W. (1980) Cognitive asymmetry in dyslexic families. *Neuropsychologia*. 18: 645–656.

Gross, K., Rothenberg, S., Schottenfield, S. and Drake, C. (1978) Duration thresholds for letter identification in left and right visual fields for normal and reading-disabled children. *Neuropsychologia*. 16: 709–715.

Grotberg, E.H. (1970) neurological aspects of learning disabilities: a case for the disadvantaged. *J. Learn. Disabil.*, 3: 321–327.

Guyer, B.L. and Friedman, M.P. (1975) Hemispheric processing and cognitive styles in learning-disabled and normal children. *Child develop.*, 46: 658–668.

Hartlage, L.C. (1975) Neuropsychological approaches to predicting outcome of remedial educational strategies for learning disabled children, *J. Ped. Psychol.*, 3: 23–24.

Hewitt, P. and Massey, J.O. (1969) *Clinical clues from the WISC*. Consulting Psychologists Press, Palo Alto, CA,

Hier, D.B. (1979) Sex differences in hemispheric specialization: hypothesis for the excess of dyslexia in boys. *Bull Orton Soc.*, 29: 74–83.

Hulme, C. (1981) The effects of manual tracing on memory in normal and retarded readers: some implications for multisensory teaching. *Psychol. Res.*, 43: 179–191.

Hynd, G.W. and Obrzut, J.E. (1981) Development of reciprocal hemispheric inhibition of normal and learning-disabled children. *J. gen. Psychol.*, 104: 203–212.

Kagan, J. and Moore, M.J. (1981) Retrieval and evaluation of symbolic information in dyslexia. *Bull. Orton Soc.*, 31: 65–73.

Kaufman, A.S. (1979) *Intelligent Testing with the WISC-R*. Wiley, New York.

Kershner, J.R. (1977) Cerebral dominance in disabled readers, good readers, and gifted children: search for a valid model. *Child Develop.*, 48: 61–67.

Kershner, J.R. (1979) Rotation of mental images and asymmetries in word recognition in disabled readers. *Canad. J. Psychol.*, 33: 39–50.

Killian, L.R. (1971) WISC, Illinois Test of Psycholinguistic Abilities and Bender Visual-Motor Gestalt Test performance of Spanish-American kindergarten and first grade school children. *J. Consult. clin. Psychol.*, 37: 38–43.

Kinsborne, M. and Warrington, E.K. (1963) Developmental factors in reading and writing backwardness. *Brit. J. Psychol.*, 54: 145–146.

Lawson, L. (1968) Ophthmalogical factors in learning disabilities. In H.R. Myklebust (Ed.), *Progress in Learning Disabilities, Vol. 1*, Grune and Stratton, New York.

LeMay, M. (1981) Are there radiological changes in the brains of individuals with dyslexia? *Bull. Orton Soc.*, 31: 65–73.

Leong, C.K. (1980) Laterality and reading proficiency in children. *Read. Res. Quart.*, 15: 185–202.

Levine, M. and Fuller, G. (1972) Psychological, neurological, and educational correlates of reading deficit. *J. Learn. Disabil.*, 5: 563–571.

Lewitter, F.I., DeFries, J.C. and Elston, R.C. (1980) Genetic models of reading disability. *Behav. Genet.*, 10: 9–30.

Lovegrove, W. and Brown, C. (1978) Development of information processing in normal and disabled readers. *Percept. Mot. Skills*, 46: 1047–1054.

Lowenberg, E.L. (1979) An investigation into a possible relationship between specific learning disabilities and temporoparietal brain dysfunction in the school-going child. *J. Res. Hum. Sci.*, 5: 23–31.

Lubar, J.F. and Bahler, W.W. (1976) Behavioral management of epileptic seizures following EEG biofeedback training of the sensorimotor rhythm. *Biofeedback Self-Regulat.*, 1: 77–104.

Lubar, J.F. and Shouse, M.N. (1976) EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR). *Biofeedback Self-Regulat.*, 3: 293–306.

Ludlam, W.M. (1981) Visual electrophysiology and reading/learning difficulties. *J. Learn. Disabil.*, 14: 587–590.

McManis, D.L., Figley, C., Richert, M. and Fabre, T. (1978) Memory-For-Designs, Bender-Gestalt, Trail Making Test, and WISC-R performance of retarded and adequate readers, *Percept. Mot. Skills*, 46: 443–450.

Mindell, P. (1978) Dyslexia and sequential order errors in reading. *Bull. Orton Soc.*, 28: 121–141.

Mollgaard, K., Diamond, M.C., Bennett, E.L., Rosenzweig, M.R. and Linder, B. (1979) Quantitative synaptic changes with differential experience in the rat brain. *Int. J. Neurosci.*, 2: 113–128.

Moore, M.J., Kagan, J., Sahl, M. and Grant, S. (1982) Cognitive profiles in reading disability. *Genet. Psychol. Monogr.*, 105: 41–93.

Morrison, F.J., Giordani, B. and Nagy, J. (1977) Reading disability: an information-processing analysis, *Science*, 196: 77–79.

Naylor, H. (1980) Reading disability and lateral asymmetry: an information processing analysis. *Psychol. Bull.*, 87: 531–545.

Nelson, H.E. and Warrington, E.K. (1980) An investigation of memory functions in dyslexic children. *Brit. J. Psychol.*, 71: 487–503.

Newell, D. and Rugel, R.P. (1981) Hemispheric specialization in normal and disabled readers. *J. Learn. Disabil.*, 14: 296–297.

Nielson, H. (1976) Dyslexia as the result of retarded development of hemispheric interaction. *Nordisk Psykologi*, 28: 152–163.

Parnavelas, J.G., Globus, A. and Kaups, P. (1973) Continuous illumination from birth affects spine density of neurons in the in the visual cortex of the rat. *Exp. Neurol.*, 40: 742–747.

Pettigrew, J.D. (1974) The effects of visual experience on the development of specificity by kitten cortical neurons. *J. Physiol. (Lond.)*, 237: 49–74.

Pirozzolo, F.J. and Raynor, K. (1979) Cerebral organization and reading disability. *Neuropsychologia*, 17: 485–491.

Riege, W.H. (1971) Environmental influences on brain and behavior of year-old rats. *Develop. Psychobiol.*, 4: 157–167.

Rosenzweig, M.R., Bennett, E.L. and Diamond, M. (1975) Brain changes in response to experience. In R.C. Atkinson (Ed.), *Psychology in Progress*, Freeman, San Francisco, CA, pp. 5–12.

Rossi, A.O. (1972) Genetics of learning disabilities. *J. Learn. Disabil.*, 5: 489–496.

Rourke, B.P. (1975) Brain–behavior relationships in children with learning disabilities. *Amer. Psychol.*, 30: 911–920.

Rourke B.P. and Telegdy, G.A. (1971) Lateralizing significance of WISC Verbal-Performance discrepancies for older children with learning disabilities. *Percept. Mot. Skills*, 33: 875–883.

Rourke, B.P., Yanni, D.W., MacDonald, G.W. and Young, G.C. (1973) Neuropsychological significance of laterlized deficits on the Grooved Pegboard Test for older children with learning disabilities. *J. Consult. clin. Psychol.*, (1975) 41: 128–134.

Rourke, B.P. and Finlayson, M.A. (1975) Neuropsychological significance of variations in patterns of performance on the Trail Making Test for older children with learning disabilities. *J. abnorm. Psychol.*, 84: 412–421.

Rugel, R.P. (1974) WISC subscale scores of disabled readers: a review with respect to Bannatyne's recategorization. *J. Learn. Disabil.*, 39: 931–938.

Satz, P., Rardin, D. and Ross, J. (1971) An evaluation of a theory of specific developmental dyslexia. *Child Develop.*, 42: 2009–2021.

Seifert, A.R. and Lubar, J.F. (1975) Reduction of epileptic seizures through EEG biofeedback training. *Biol. Psychol.*, 3: 157–184.

Shouse, M.N. and Lubar, J.F. (1979) Operant conditioning of EEG rhythms and Ritalin in the treatment of Hyperkinesis. *Biofeedback Self-Regul.*, 4: 299–312.

Silver, L.B. (1971) A proposed view on the etiology of the neurological learning disability syndrome. *J. Learn. Disabil.*, 4: 123–133.

Silver, L.B. (1971) Familial patterns in children with neurologically-based learning disabilities. *J. Learn. Disabil.*, 4: 349–358.

Snef, G. and Freundl, P.C. (1971) Memory and attention in specific learning disabilities. *J. Learn. Disabil.*, 4: 94–106.

Spring, C. (1976) Encoding speed and memory span in dyslexic children. *J. spec. Educat.*, 10: 35–40.

Sterman, M.B. (1973) Neurophysiologic and clinical studies of sensorimotor EEG biofeedback training: some effects on epilepsy. In L. Birk (Ed.), *Biofeedback: Behavioral Medicine*, Grune and Stratton, New York.

Sterman, M.B. (1977) Sensorimotor EEG operant conditioning and experimental and clinical effects. *Pavlovian J. biol. Sci.*, 12: 65–92.

Swanson, L. (1978) Verbal encoding effects on the visual short-term memory of learning disabled and normal readers. *J. educat. Psychol.*, 70: 539–544.

Swanson, L. (1981) Vigilance deficit in learning disabled children: a signal detection analysis. *J. Child Psychol. Psychiatry & allied Discipl.*, 22: 393–399.

Tallal, P. (1980) Auditory temporal perception, phonics, and reading disabilities in children. *Brain Lang.*, 9: 182–198.

Tansey, M.A. (1982) EEG sensorimotor biofeedback training and the treatment of a six-year-old asthmatic child. *Amer. J. clin. Biofeedback*, 5: 145–149.

Tansey, M.A. and Bruner, R.L. (1983) EMG and EEG biofeedback training in the treatment of a 10-year-old hyperactive boy with a developmental reading disorder. *Biofeedback Self-Regul.*, 8: 25–37.

Thompson, L.J. (1973) Learning disabilities: an overview. *Amer. J. Psychiatry*, 130: 393–399.

Thompson, M.E. (1976) A comparison of laterality effects in dyslexics and controls using verbal dichotic listening tasks. *Neuropsychologia*. 14: 243–246.

Van der Honert, D. (1977) A neuropsychological technique for training dyslexics. *J. Learn. Disabil.*, 10: 15–21.

Van Eyck, J.W. (1980) Motor development and learning difficulties: II. *Academic Therapy*, 15: 577–587.

Vellutino, F.R., Smith, H., Steger, J.A. and Kaman, M., Reading disability: age differences and the perceptual-deficit hypothesis. *Child Develop.*, 45: 487–493.

Wechsler, D. (1974) *Manual for the Wechsler Intelligence Scale for Children - Revised*. Psychological Corporation, New York.

Weiner, P.S. (1969) The cognitive functioning of language deficient children. *J. Speach Hear. Res.*, 12: 53–64.

Wheeler, T.J., Watkins, E.J. and McLaughlin, S.P. (1977) Reading retardation and cross-laterality in relation to short-term information processing tasks. *Brit. J. educat. Psychol.*, 47: 126–131.

Witelson, S.F. (1977) Developmental dyslexia: two right hemispheres and none left. *Science*, 195: 309–311.

Worden, P.E., Malmgren, I. and Gabourie, P. (1982) Memory for stories in learning disabled adults. *J. Learn. Disabil.*, 15: 145–152.

Yeni-Komshian, G.H., Isneberg, D. and Goldberg, H. (1975) Cerebral dominance and reading disability: left visual field deficits in poor readers. *Neuropsychologia*, 13: 83–94.