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Neurotherapy and Drug Therapy in Combination for Adult ADHD, Personality Disorder, and Seizure Disorder

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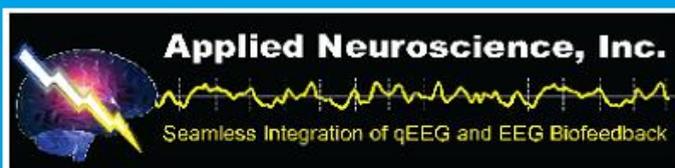
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Neurotherapy and Drug Therapy in Combination for Adult ADHD, Personality Disorder, and Seizure Disorder: A Case Report

Lisa M. Hansen, B.S., David L. Trudeau, M.D., and Dixie L. Grace, Ph.D.

This is a case report of an adult female patient with ADHD, temporal seizure disorder, and Borderline Personality Disorder treated with 30 weekly sessions of SMR neurofeedback and carbamazepine. Posttreatment measures showed improvements in T.O.V.A., self report, and QEEG. Both neurofeedback and carbamazepine showed the most effect in early treatment. Progress continued after discontinuance of the drug.

We had an opportunity to study a patient who presented for evaluation for ADHD, referred by her psychotherapist, also managed by a neurologist for seizure disorder. Because of the seizure disorder and the patient's initial reluctance to take her prescribed anticonvulsant, carbamazepine, we elected to treat this patient primarily with neurofeedback, rather than with stimulant medication, which is contraindicated in untreated seizure disorder. However, after neurofeedback therapy began, the patient began taking an anticonvulsant, carbamazepine, as recommended by her neurologist. Because the patient reached therapeutic carbamazepine levels, stopped the drug because of grogginess, then agreed to restart it, and then again discontinued it, we had the opportunity to study a naturalistic crossover effect. We were able to measure the effects of neurofeedback therapy with and without carbamazepine on attentional testing and QEEG.

Interestingly, this patient also had established diagnoses of Borderline Personality Disorder and seizure disorder with features of partial complex seizure with interictal thought intrusions. The occurrence of these comorbidities, as well as the effect of carbamazepine on attentional disorders have been described, but not in a single case.

There appears to be an abundance of literature on the comorbidity of behavioral disorders, both organic and psychological. Andrulonis et al. (1980) report a high co-occurrence of borderline syndromes with other syndromes, such as Minimal Brain Dysfunction (MBD), a term used in the 1960's prior to the definition of Attention Deficit Disorder (ADD), and episodic dyscontrol. A follow-up study by Andrulonis, Glueck, Stroebel, and Vogel (1982) attempted to find and test subcategories for Borderline Personality Disorder which involved nonorganic borderlines, trauma/epilepsy borderlines, and Attentional Deficit/Learning Disorder borderlines. Using a four-way discriminant analysis function, significant differences were found between these groups, and a high number of patients in the organic group were found to exhibit episodic dyscontrol syndrome, MBD, or a disorder of the limbic system. A study conducted of 286 individuals diagnosed with episodic dyscontrol syndrome found 94% to present with either MBD, head injury, and/or epilepsy (Elliot, 1984).

Andrulonis et al. (1980) report that borderline subjects often display attentional deficits as children. There appears to be a relationship between ADD and/or ADHD and head injury. A self-report survey of high school and university students by Segalowitz and Lawson (1995) found signif-

ficant correlations between mild head injury and ADD. The presence of ADHD was significantly reported by more females who had multiple head injuries over single injuries, and by more females with head injuries than males. Moreover, Rutter (1982) proposes a continuum notion of Minimal Brain Dysfunction, describing it as a more mild form of traumatic brain damage, which does not produce any specific identifiable clinical symptoms.

With regards to electroencephalogram (EEG) abnormalities, Andrulonis et al. (1980) report temporal and frontal EEG abnormalities in patients with episodic dyscontrol syndrome, and that many borderline subjects with limbic system disorders exhibit abnormal EEG's as well. Also, Mann, Lubar, Zimmerman, Miller, and Muenchen (1992) found more theta and less beta activity in a sample of ADHD males as compared to control matched males. As for treatment for ADHD, several studies report beneficial effects on attention and related factors after sensorimotor (SMR) neurofeedback training (Lubar & Lubar, 1984; Lubar & Shouse, 1976; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1992; Tansey, 1984).

There is some evidence that treating attentional deficits and other disorders with anticonvulsants, such as carbamazepine, may be promising. The literature offers varied opinions of this. The use of anticonvulsant medication is sometimes required when the ADHD patient also presents with seizure disorder (Millichap, 1982). Carbamazepine has also been used for rage outbursts. A study by Mattes (1990) found that patients exhibiting intermittent explosive disorder had significantly improved on carbamazepine, but that the patients with ADHD benefited more from propranolol. Remschmidt (1976) proposed that carbamazepine would be beneficial to patients with behavioral disorders, including ADHD. A case study by Gay and Ryan (1992) report administering carbamazepine to a patient who acquired paroxysmal kinesigenic dystonia after receiving methylphenidate for pos-

sible ADHD. The patient's symptoms ceased after carbamazepine was dispensed and recurred after discontinuance of it. Carbamazepine has also been applied as a treatment for hyperactivity. A study by Reid, Naylor, and Kay (1981) report improvements in five adult mentally handicapped patients with carbamazepine.

Regarding the question of what population will most likely react to treatment with carbamazepine, patients in whom hyperactivity was the primary problem seemed to respond to carbamazepine with lowered hyperactivity (Reid et al., 1981). Carbamazepine has been proposed as one of the chosen drugs for use with borderlines. Furthermore, Andrulonis et al. (1980) hypothesized that anticonvulsants would best treat borderline patients with ADHD or learning disabilities, exhibiting episodic dyscontrol syndrome. Also, there has been a procedure proposed which attempts to identify which patients exhibiting a form of limbic system dysrhythmia would benefit from anticonvulsants. Ryback and Gardner (1991) administered procaine to patients with normal EEG's. Many patients with ADHD who displayed a positive reaction to procaine, reported improvements in attention after carbamazepine.

Method

The subject is a 36-year-old female, diagnosed with temporally focused seizure disorder and an Axis II of Borderline Personality Disorder. She has had episodes of behavioral dyscontrol (i.e., destroying her medical records, throwing furniture around) which could be described as episodic dyscontrol syndrome, intermittent explosive disorder, or both. Under stress, the patient has reported severe psychotic thinking (i.e., fear of shooting others, belief she was a queen). The patient presented with attentional problems and was therefore administered the Wender Utah questionnaire and met criteria for childhood ADHD by achieving a score of at least 46 (Ward, Wender, & Reimherr, 1993). She also met DSM-IV criteria for adult residual ADHD (either 6/9

inattention symptoms and/or 6/9 impulsivity-hyperactivity symptoms occurring at least most of the time with dysfunction in work or family life occurring most of the time). The patient was administered Dupuy and Greenberg's (1993) Test of Variables of Attention (T.O.V.A.). The patient's performance on the T.O.V.A. was compared to norms constituted for gender and age to the ninth decade (Greenberg & Woldman, 1993). Test results were abnormal, $t \geq 65$, on omissions ($t=81.4$), reaction time ($t=67.1$), and variability ($t=64.6$). A self-report head injury history was also taken, and although the patient mentioned a few injuries, she did not meet criteria for significant mild traumatic brain injury (MTBI) by not meeting criteria of a Glasgow Coma scale of 13-15.

A nineteen-channel Quantitative Electroencephalogram (QEEG) using Neurolex 24 (Lexicor, Boulder, CO) was taken under five conditions at a sampling rate of 128 Hz: eyes closed for two 200-300 second recordings; eyes open at rest and with listening, reading, and mental math for 100-150 seconds each. Artifact was rejected and the remaining epochs were analyzed by spectral averages in topographic maps across bands and visually inspected. In addition, eyes-closed records were artifacted using high resolution graphics, analyzed, and compared to a normative data base (Thatcher, 1994a, 1994b; Thatcher, Walker, & Guidace 1987; Hudspeth, 1985; Hudspeth & Pribham, 1990, 1991) for relative power (Neurorep, Stockton, CA). High amounts of frontal alpha relative power were noted at sites F7 ($z=2.26$, $p=.025$), F3 ($z=2.36$, $p=.025$), F4 ($z=2.32$, $p=.025$), and F8 ($z=2.76$, $p=.005$).

Written informed consent was obtained for a neurofeedback treatment protocol approved by an institutional human subjects committee. The patient received 30-minute sessions for 30 weeks of sensorimotor rhythm (SMR) training at 12-15 Hz over 4 microvolts (μv). The training protocol also included reward inhibits for theta (4-8 Hz)

greater than 12 μv , EMG (24-30 Hz) greater than 10 μv , and delta (0-4 Hz) greater than 25 μv . Score was defined as the percentage of time SMR was over 4 μv and theta was under 12 μv , EMG under 10 μv , and delta under 25 μv . Three tones of feedback were given for SMR: 220 frequency for SMR over 4 μv , 440 frequency for SMR over 5 μv , and 660 frequency for SMR over 6 μv . So if a patient achieved 6 μv of SMR training, she would receive a three note chord of feedback. We used a visual display of SMR wave, theta wave, delta wave, and raw EEG using Biolex digital filters so that the patient received both sound and visual feedback. CZ was used as a training site for the first 26 sessions and C4 for the last 4. Materials used included a Neurosearch-24 EEG acquisition unit using Biolex software (Lexicor, Boulder, CO), 4 electrodes (linked ears for reference, a common forehead ground and an active), 10/20 conductive paste, and skin preparing gel.

During the course of neurotherapy the patient agreed to begin carbamazepine 200 mg tid as recommended by her neurologist. After being on that medication for approximately 8 to 9 weeks, she stopped taking it because it made her drowsy, presumably as medication blood levels slowly increased. After a one week wash out period, T.O.V.A. and QEEG were repeated. The patient then agreed to resume carbamazepine at lower levels and began taking it at 200 mg bid. Following stabilization with the drug for one week, T.O.V.A. and QEEG were again repeated. Subsequently at the end of therapy T.O.V.A. and QEEG were repeated, at which time the patient volunteered that she had secretly discontinued her medication several weeks after resuming it.

Throughout the course of neurofeedback therapy, the patient's score, an expression of the percentage of time she was able to exceed a 4 μv of SMR, was monitored as a measure of her success at training.

Results

Since neurofeedback, the patient reports

improved adjustment in her professional and personal relationships. The patient has been able to hold a job, whereas she had difficulties with maintaining employment in the past. The patient has completed remedial classes at a community college and has enrolled in more classes, whereas in the past there was a history of dropping out. In therapy the patient has been reported by her psychologist as still being overly talkative, but more appropriate, keeping appointments more regularly than in the past, reporting fewer and more milder outbursts, and having a stable mood since the training started.

After 17 sessions of SMR neurofeedback treatment, the patient increased her SMR over 4 μ v. The T.O.V.A. test was administered again and another QEEG was obtained, showing relative powers within normal ranges. The patient's T.O.V.A. revealed 2 scales of significance ($t \geq 65$): omissions ($t=69.1$) and commissions ($t=71.7$), as opposed to the 3 abnormal scales at pre-treatment. The patient, however, had previously discontinued taking carbamazepine approximately six days before, reporting the drug made her feel groggy and intoxicated. The patient agreed to take a lower dose of carbamazepine at 200 mg bid. One week later a third T.O.V.A. and QEEG were taken. The T.O.V.A. results revealed 0 of 4

scales of significance ($t \geq 65$), indicating no attentional problems as defined by this test. The QEEG still showed normal relative powers. On Session 27, the patient's neurofeedback training was then changed to every two weeks, this time with electrode placement at site C4. Approximately a month and a half later, the patient reported that she discontinued her carbamazepine approximately two weeks after her third T.O.V.A., due to undesirable side effects. The patient was administered a fourth T.O.V.A. that continued to reveal 0 of 4 scales of significance ($t \geq 65$).

Table 1 displays the T.O.V.A. results for baseline, after 17 sessions of neurofeedback but off carbamazepine, after 18 sessions of neurofeedback back on carbamazepine, and after 30 sessions of neurofeedback but off carbamazepine. It can be seen that there is an effect of neurofeedback alone on T.O.V.A. performance, that is substantially enhanced with the reinstatement of carbamazepine in the beginning part of treatment. At baseline three of four scales (omission, reaction time, and variability) are abnormal ($t \geq 65$), consistent with attentional deficit. Following neurofeedback, but off carbamazepine, there is improvement in reaction time, variability, and omissions, but commission errors increase and two scales are abnormal (commission and omission). The omission

Table 1
Results of T.O.V.A. (expressed as t-scores)

CONDITION	OMISSION	COMMISSION	REACTION TIME	VARIABILITY
BASELINE	81.4	56.3	67.1	64.6
NEUROFEEDBACK ALONE	69.1	71.7	45.6	49.7
NEUROFEEDBACK AND CARBAMAZAPINE	44.4	54.4	45.9	40.6
POST TRAINING AND DRUG FREE	44.4	50.5	42.3	41.3

score is consistent with attentional deficit, though improved. After reinstatement of carbamazepine and months later back off carbamazepine, all four scales are normal and the test is no longer consistent with attentional deficit.

Figure 1 shows the progress made by the subject over neurofeedback sessions, the first 26 sessions done at CZ with the following 4 at C4, with SMR reward which inhibit of reward for movement, EM and excess theta. The score for each session is depicted sequentially, score being defined as the percentage of time during the session the subject created SMR greater than 4 μv . As can be seen in Figure 1, the subject generally improved over time. Between Sessions 7 and 8 the subject began carbamazepine 200 mg tid, and after Session 16 discontinued it. The subject was then studied by T.O.V.A. and QEEG after a 10 day wash-out period, and then agreed to resume carbamazepine at 200 mg bid, which she continued for

Sessions 18 through 20, and then discontinued.

Table 2 displays Z scores for alpha relative power across bands for 16 of the 19 international 10-20 sites pre and posttreatment. High frontal alpha relative power at baseline normalizes following neurofeedback, whether on or off carbamazepine. The posttreatment record is essentially the same for relative power on QEEG obtained after Session 17, after Session 18, and after Session 30, and only the record following Session 17 is shown.

Discussion

This case report illustrates the importance of integrating neurofeedback therapy with pharmacotherapy and psychotherapy. In this particular case, the relationships between each of the indicated therapies and each of the diagnostic formulations is important.

Figure 1
Progress over sessions expressed as a percentage of time over SMR threshold.

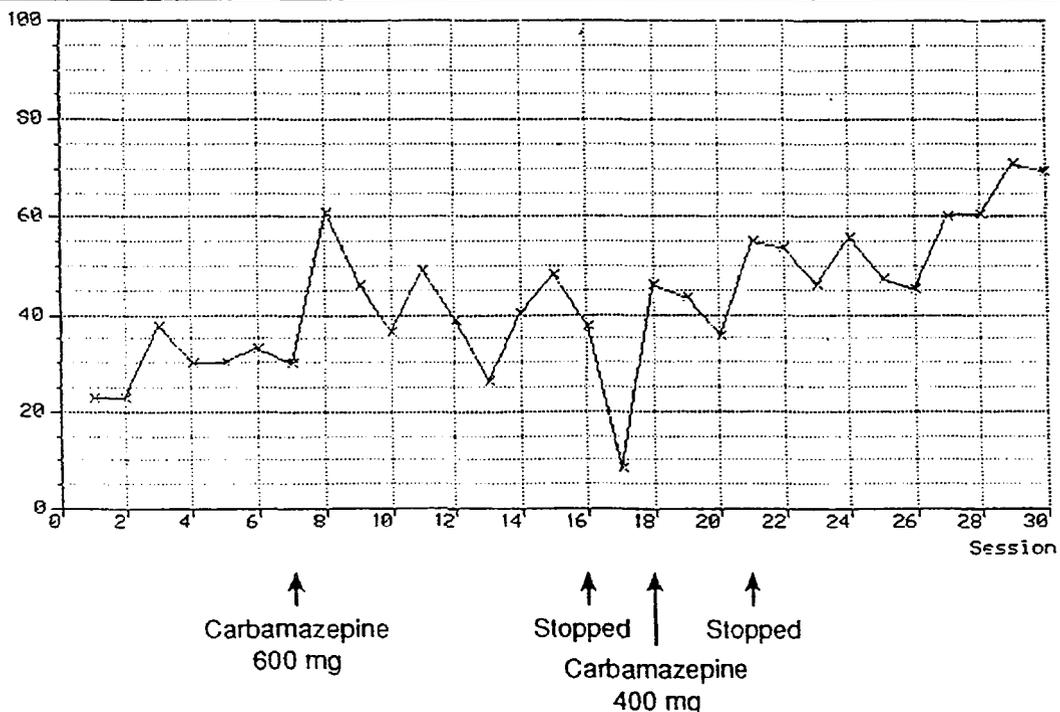


Figure 1 adapted from Biolex report of track of sessions (© Lexicor 1992)

Table 2
Relative Amplitude Z-Scores Pre and Post Treatment

	DELTA	THETA	ALPHA	BETA
F1	1.07	-0.42	0.61	-1.80
F2	0.66	-0.40	0.93	-1.84
F7	-1.26	-0.96	2.23	-1.37
F8	-1.45	-1.07	2.71	-1.90
F3	-1.22	-0.93	2.41	-1.77
F4	-1.12	-0.96	2.33	-1.93
T3	-1.07	-0.85	1.32	-0.17
T4	-0.77	-0.50	1.64	-1.02
C3	-1.08	-0.76	1.71	-1.25
C4	-0.92	-0.68	1.54	-1.27
T5	-0.28	-0.32	0.75	-0.85
T6	-0.54	-0.06	0.71	-1.09
P3	-0.48	-0.43	0.81	-0.89
P4	-0.20	-0.24	0.44	-0.58
O1	-0.14	-0.11	0.51	-1.05
O2	0.03	0.18	0.24	-0.96

	DELTA	THETA	ALPHA	BETA
F1	-0.61	-0.00	1.20	-1.19
F2	-0.88	0.02	1.41	-1.11
F7	-0.77	-0.16	1.14	-0.88
F8	-1.19	-0.21	1.67	-1.30
F3	-0.96	-0.06	1.30	-1.06
F4	-0.88	-0.20	1.36	-1.26
T3	-0.84	-0.44	0.72	0.06
T4	-0.75	-0.43	1.58	-1.03
C3	-0.52	-0.16	0.68	-0.58
C4	-0.53	-0.17	0.72	-0.66
T5	-0.69	-0.03	1.01	-1.33
T6	-1.07	-0.24	1.24	-1.52
P3	-0.23	-0.16	0.47	-0.74
P4	-0.34	-0.23	0.59	-0.87
O1	-0.20	-0.06	0.50	-1.01
O2	-0.16	0.07	0.48	-1.19

Z scores are expressed as deviations from age and gender adjusted normals as derived from the Life Span QEEG Reference Database. Relative power is the percentage of power in each frequency band. All sites are Universal 10-20 Montage, except that PF1 and PF2 are named P1 and P2, respectively.

The subject's clear lifetime diagnosis of attention deficit disorder is apparent from the childhood history (Wender Utah), the current symptoms (DSM-IV criteria for ADHD), and the subject's attentional performance (T.O.V.A.). The attentional problems prevented progress in psychotherapy issues around the subject's personality disorder and lifetime abuse issues. This in turn led to a referral for ADHD evaluation and treatment. As part of that evaluation, stimulant medication was considered, but relatively contraindicated because of the presence of seizure disorder. The subject's reaction to anti-seizure medication was in turn colored by both her personality disorder in that she distrusted it, and by her ADHD in that usually effective doses made her groggy and impaired her attention even further. The seizure disorder treatment in turn seemed to have an impact on the personality disorder, in that the subject described intrusive homicidal impulses that disappeared on the

carbamazepine, and she also described features of episodic anger dyscontrol that improved on carbamazepine. Furthermore, we found that carbamazepine enhanced her ability to meet neurofeedback goals, as well as improve her T.O.V.A. performance.

Thus, we began our treatment of her ADHD with neurofeedback alone. When she had developed trust in that process and some clinical improvement, she became willing to take the drug. Her progress accelerated, but then faltered because of sedation due to the drug. She discontinued that, affording us an opportunity to study her postneurofeedback, off carbamazepine. What we found was that QEEG changes were independent of carbamazepine, but followed neurofeedback, with substantial reduction in frontal alpha relative power. Attentional performance improved as a result of the neurofeedback treatment condition, with some improvement of T.O.V.A. Initially the patient's performance on the T.O.V.A. as

well as her neurofeedback scores were enhanced by medication. As she continued with neurofeedback training, after 20 sessions she was able to discontinue medication entirely, sustaining her improvements in attentional performance and medication stabilization.

This case study illustrates the importance of evaluating comorbidities of personality disorder and seizure disorder with attentional deficit disorder. The use of concurrent medication treatment initially enhanced the subject's ability to do neurofeedback. Following neurofeedback of the attentional enhancement type, there were substantial improvements in behavioral dyscontrol that had been thought to be due to the seizure disorder or the personality disorder. Even though the patient was able to stop medication and sustain these improvements, these improvements may be explained by her improved attention and focus as well as her enhanced ability to respond to psychotherapy.

References

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Andrulonis, P. A., Glueck, B. C., Stroebel, C. F., & Vogel, N. G. (1982). Borderline personality subcategories. *Journal of Nervous & Mental Disease*, 170, 670-679.
- Andrulonis, P. A., Glueck, B. C., Stroebel, C. F., Vogel, N. G., Shapiro, A. L., & Aldridge, D. M. (1980). Organic brain dysfunction and the borderline syndrome. *Psychiatric Clinics of North America*, 4, 47-66.
- Dupuy, T. R., & Greenberg, L. M. (1993). *T.O.V.A. manual*. Los Alamitas, CA: Universal Attention Disorders, Inc.
- Elliot, F. A. (1984). The episodic dyscontrol syndrome and aggression. *Neurologic Clinics*, 2, 113-125.
- Gay, C. T., & Ryan, S. G. (1994). Paroxysmal kinesigenic dystonia after methylphenidate administration. *Journal of Child Neurology*, 9, 45-46.
- Greenberg, L. M., & Woldman, I. W. (1993). Developmental normative data on the Test of Variables of Attention (T.O.V.A.) *Journal of Child Psychology and Psychiatry*, 34, 1019-1030.
- Hudspeth, W. (1985). Developmental neuropsychology: Functional implications of quantitative EEG maturation. *Journal of Clinical and Experimental Neuropsychology*, 7, 606.
- Hudspeth, W., & Pribham, K. (1990). Stages of brain and cognitive maturation. *Journal of Educational Psychology*, 82, 881-884.
- Hudspeth, W., & Pribham, K. (1991). Physiological indices of cerebral maturation. *International Journal of Psychophysiology*, 12, 19-29.
- Lubar, J. O., & Lubar, J. F. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorder. *Biofeedback and Self Regulation*, 9, 1-23.
- Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): A preliminary report. *Biofeedback and Self Regulation*, 3, 293-296.
- Mann, C. A., Lubar, J. F., Zimmerman, A. W., Miller, C. A., & Muenchen, R. A. (1992). Quantitative analysis of EEG in boys with attention deficit hyperactivity disorder: Controlled study with clinical implications. *Pediatric Neurology*, 8, 30-36.
- Mattes, J. A. (1990). Comparative effectiveness of carbamazepine and propranolol for rage outbursts. *Journal of Neuropsychiatry & Clinical Neurosciences*, 2, 159-164.
- Millichap, J. G. (1982). The hyperactive child. *Triangle*, 21, 59-63.
- Oettinger, L., Jr. (1977). Pediatric psy-

- chopharmacology. A review with special reference to deanol. *Diseases of the Nervous System*, 38, 25-31.
- Reid, A. H., Naylor, G. J., & Kay, D. S. (1981). A double-blind, placebo controlled, crossover trial of carbamazepine in overactive, severely mentally handicapped patients. *Psychological Medicine*, 11, 109-113.
- Remschmidt, H. (1976). The psychotropic effects of carbamazepine in non-epileptic patients, with particular reference to problems posed by clinical studies in children with behavioral disorders. In W. Birkmayer (Ed.), *Epileptic seizures-behavior-pain* (pp. 253-258). Baltimore, MD: University Park Press.
- Rutter, M. (1982). Syndromes attributed to "minimal brain dysfunction" in childhood. *American Journal of Psychiatry*, 139, 21-33.
- Ryback, R. S., & Gardner, E. A. (1991). Limbic system dysrhythmia: A diagnostic electroencephalogram procedure utilizing procaine activation. *Journal of Neuropsychiatry & Clinical Neurosciences*, 3, 321-329.
- Segalowitz, S. J., & Lawson, S. (1995). Subtle symptoms associated with self-reported mild head injury. *Journal of Learning Disabilities*, 28, 309-319.
- Tansey, M. A. (1984). EEG sensorimotor rhythm biofeedback training: Some effects on the neurologic precursors of learning disabilities. *International Journal of Psychophysiology*, 1, 163-177.
- Thatcher, R. W. (1994a). Cyclic cortical reorganization in early childhood. *Brain and Cognition*, 20, 24-50.
- Thatcher, R. W. (1994b). Cyclic cortical reorganization: Origins of human cognitive development. In G. Dawson & K. Fischer (Eds.), *Human behavior and the developing brain* (pp. 232-266). New York: Guilford Press.
- Thatcher, R. W., Walker, R. A., Gerson, I., & Geisler, F. (1989). EEG discriminant analysis of mild head trauma. *EEG and Clinical Neurophysiology*, 73, 93-110.
- Thatcher, R. W., Walker, R. A., & Guidace, S. (1987). Human cerebral hemispheres develop at different rates and ages. *Science*, 236, 1110-1113.
- Ward, M. F., Wender, P. H., & Reimherr, F. W. (1993). The Wender Utah rating scale: An aid in the retrospective diagnosis of childhood attention deficit hyperactivity disorder. *American Journal of Psychiatry*, 150, 885-890.

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