

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

Attention Deficit Hyperactivity Disorder: Neurological Basis and Treatment Alternatives

Arreed Barabasz EdD PhD ^a Marianne Barabasz

^a Washington State University, Pullman, Washington

Published online: 18 Oct 2008.

To cite this article: Arreed Barabasz EdD PhD & Marianne Barabasz (1995) Attention Deficit Hyperactivity Disorder: Neurological Basis and Treatment Alternatives, Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience, 1:1, 1-10, DOI: 10.1300/J184v01n01_01

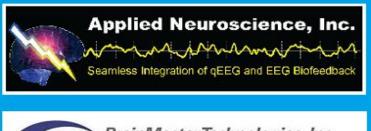
To link to this article: http://dx.doi.org/10.1300/J184v01n01_01

PLEASE SCROLL DOWN FOR ARTICLE

© International Society for Neurofeedback and Research (ISNR), all rights reserved. This article (the "Article") may be accessed online from ISNR at no charge. The Article may be viewed online, stored in electronic or physical form, or archived for research, teaching, and private study purposes. The Article may be archived in public libraries or university libraries at the direction of said public library or university library. Any other reproduction of the Article for redistribution, sale, resale, loan, sublicensing, systematic supply, or other distribution, including both physical and electronic reproduction for such purposes, is expressly forbidden. Preparing or reproducing derivative works of this article is expressly forbidden. ISNR makes no representation or warranty as to the accuracy or completeness of any content in the Article. From 1995 to 2013 the *Journal of Neurotherapy* was the official publication of ISNR (www. Isnr.org); on April 27, 2016 ISNR acquired the journal from Taylor & Francis Group, LLC. In 2014, ISNR established its official open-access journal *NeuroRegulation* (ISSN: 2373-0587; www.neuroregulation.org).

THIS OPEN-ACCESS CONTENT MADE POSSIBLE BY THESE GENEROUS SPONSORS







Attention Deficit Hyperactivity Disorder: Neurological Basis and Treatment Alternatives

Arreed Barabasz and Marianne Barabasz

Recent research indicates a neurological basis for attention deficit disorder, specifically, right frontal lobe dysfunction. Traditional treatments for ADD/ADHD, such as stimulant drugs, behavior modification, and cognitive-behavior therapy have had limited, short-term success and many drawbacks. Neurotherapy (or EEG feedback), which addresses the frontal lobe dysfunction, has shown significant, long-term results, by teaching patients to normalize their brainwave responses to stimuli. When Instantaneous Neuronal Activation Procedure (INAP) is used in adjunct to neurotherapy, treatment time is significantly reduced without losing long-term effects of the therapy. INAP was developed on the basis of research on hypnotic phenomena.

Attention deficit disorder (ADD) and attention deficit hyperactivity disorder (ADHD) are characterized by the inability to self-regulate focused attention. Children with hyperactivity are impulsive and behaviorally disinhibited. The condition is developmentally disabling which, if left uncontrolled, persists into adolescence and adulthood. This biologically based behavioral disability has a pervasive negative impact on a wide range of adaptive functioning. Although under diagnosed in the past, such is not the case today. Diagnoses are on the rise as public awareness increases. "Its ADHD's time in the sun and they are getting it" (Edwards, 1995, P.44).

Neurological Basis and Assessment

A plethora of correlational studies have led to speculations about the role of food additives, dietary sugar (Finegold 1973, 1975), blood lead levels (David, 1974), allergies (Marshall, 1989), smoking and alcohol use during pregnancy (Barkley, 1990), as causal factors in the etiology of ADD/ADHD. At the same time, serious research has been progressing systematically which forms the basis for a neurological understanding of attention deficit disorders. The data from these investigations clearly implicates frontal lobe involvement, providing a substantive rationale for the use of neurotherapy and a new adjunctive technique called Instantaneous Neuronal

Activation Procedure (INAP) (Barabasz, 1985, 1993, in press; A. Barabasz & M. Barabasz, 1993b, 1994a, 1994b, in press-a).

Frontal lobe functions are executive in nature and are involved in developing plans and organizing resources. They also are critical in mediating inhibitory behaviors such as controlling motor behavior and inhibiting attentional focus on distractor or irrelevant stimuli. The evidence suggesting right frontal lobe dysfunction as the basis of attention deficit disorders is considerable (Chelune, Ferguson, Koon & Dickey, 1986; Gualteri & Hicks, 1985; Hynd, Semrud-Clikeman, Lorys, Novey & Eliopulos, 1990; Lou, Henriksen, Bruhn, Bomer & Nielsen, 1989; Schaughency & Hynd, 1989; Voeller & Heilman, 1988)

Recent research using advanced neuro-imaging morphological procedures has shown that ADD/ADHD children fail to show the normal right-greater-than-left asymmetry in the mass of the frontal lobes (Hynd, Hem, Voeller & Marshall, 1991). Consistent with this finding, computerized quantitative electroencephalographic (EEG) analysis (referred to as "neurometric assessment" in the practice of psychology) shows significantly greater slow wave (theta) activity and significantly less fast wave (beta) activity predominantly in the frontal regions for ADD/ADHD boys (Mann, Lubar, Zimmerman, Miller & Muenchen,

1992) and for ADD/ADHD girls and boys (A. Barabasz, Crawford & M. Barabasz, 1993; in press; Barabasz & Barabasz, In press, b) when compared to age-and-sexmatched nonnals.

ADD/ADHD Treatments

Current ADD/ADHD treatments consist of the traditional approaches aimed at symptom management, using stimulant medication, behavior modification, and cognitive-behavior modification; and the more recent approaches focus on neuropsychological rehabilitation. These latter approaches now include neurotherapy alone and neurotherapy with Instantaneous Neuronal Activation Procedure (INAP). INAP is an adjunctive technique which enhances EEG Beta, increases responsiveness to feedback and substantially shortens total treatment time.

Stimulant drugs such as methylphenidate (Ritalin), are prescribed for 600,000 to 1 million school children in the U.S. These powerful drugs are believed to potentiate the dopamine and norepinephrine neurotransmitters. However, medication was found to do absolutely nothing for 25%-40% of children with the disorder. This striking finding was revealed by a "review of reviews" conducted by 15 co-authors (Swanson et al., 1993), that comprehensively examined 341 reviews of the effects of stimulant medication on children with attention deficit disorders. Furthermore, a large proportion of patients responding to Ritalin also show the same improvements on placebo (Swanson et al, 1993).

For those who respond to stimulant medication, temporary management of over-activity, inattention and impulsivity can be expected as well as temporary improvement in compliance. Aggression may be reduced and the amount of academic work completed may increase. Contrary to the hopes of parents and practitioners, there will be no significant improvement in reading, no significant improvement in athletic or game skills, no significant improvement in pro-active social skills and no significant improvement in learning other than improved attending. Furthermore, one should expect no improvement in long-

term adjustment such as improved academic achievement nor any reduction in anti-social behaviors or arrest rates (Swanson et al., 1993).

The side effects and limitations of stimulant medications include: 1) short length of action (4-5 hours) which critically limits its benefits and requires careful planning for administration at school and school-related overnight trips; 2) problems with the child's self esteem due to taunting by peers (e.g., "hyper diaper has to go get his pill"); 3) growth stunting unless medication vacations of adequate length are planned such as summer vacation and the longer school year holidays, (medication free weekends, frequently recommended by prescribers, are too short to counteract inhibition of normal growth); 4) insomnia and poor eating; 5) tics, cardiovascular problems, and Tourettes Syndrome occur in a small percentage of children; 6) high doses cause cognitive impairment; 7) no residual effect once medication is terminated; and 8) a small, but significant, number of cases show negative physiological side effects that do not diminish over time, despite cessation of the medication (Whalan & Henker, 1991).

Behavior modification is undoubtedly the next most widely used therapy to manage the disorder. One of the strengths of the behavioral approach is the typical collaborative involvement of both parents and teachers. Starting with the seminal work of B. F. Skinner, behavior therapy researchers have published in excess of 5,000 articles, many of which are applicable directly or indirectly to the management of ADD/ADHD. Therapists train parents to use token economies and positive attention for appropriate behaviors and time out or other punishments for non-compliance (Gaddes & Edgell, 1994; Whalan & Henker, 1991). Teachers use classroom contingency management where verbal praise and other rewards are administered for appropriate attentive behaviors and privileges withdrawn or punishments administered for undesirable behaviors. Behavioral interventions can be combined with stimulant drugs to provide a more comprehensive approach. (1990) believes that medication helps to potentiate the benefits of this approach.

Behavior modification's limitations include: 1) a substantial number of children do not respond to the treatment; 2) training does not generalize to non-trained behaviors; 3) there is no carryover to the classroom of behaviors learned only with parents and visa-versa; 4) as with medication, once the treatment is terminated, the behaviors rapidly return to baseline/pre-treatment levels; 5) a high degree of dependence on both parental and teacher cooperation. Undoubtedly the greatest limitation lies in the enormous complexity of the approach required for its application to ADD/ADHD. Consistent with behavioral interventions for other disorders, failures and relapses are blamed on non-compliance (Barabasz, 1987). Firestone, Kelly, Goodman & Davey (1981) reported that 50% of parents failed to continue behavior modification treat-

Cognitive-behavior therapy has greater flexibility than behavior modification and/or medication. For example, self talk coping skills can be taught which should generalize to a wide range of situations that the child might face. In theory, cognitive-behavior therapy should go beyond symptom management by providing a basis for continued growth and rehabilitation. Unfortunately, the present adaptations of Cognitive-Behavior Therapy to the treatment of ADD/ADHD have produced few positive outcomes or have completely failed to demonstrate any lasting effects (Conte, 1991; Gaddes & Edgell, 1994).

Neurotherapy (EEG feedback) is a rehabilitative approach to the treatment of ADD/ADHD. The goal is permanent normalization without dependence on drugs or continuous behavioral management therapy. Neurotherapy accepts the neurological basis of the disorder (i.e. frontal lobe dysfunction). Recognizing that the attention deficit disordered child, adolescent, or adult produces greater EEG slow wave (theta) activity (4-8 Hz) and less beta (14-32 Hz) activity compared to normal controls, neurotherapy is intended to teach patients to normalize their brainwave responses to stimuli (Barabasz et al, 1993, in

press; Mann et al, 1992). The procedure is based on an early study by Sterman and Friar (1972), who discovered that brainwave feedback made it possible to learn to inhibit epileptic seizures by enhancing low beta (12-16) which is referred to as sensory motor rhythm (SMR). As in current neurofeedback protocols for ADD/ADHD, Sterman and Friar's patients were also trained to simultaneously minimize theta. The first preliminary case study application of this procedure to hyperkinetic children was by Lubar and Shouse (1976).

When a normal person is presented with an attentional task, such as reading, doing simple arithmetic or listening to a story, his/her EEG's usually shift to the beta frequency band with an increase in magnitude with projection to the frontal (particularly right frontal) regions. In contrast, persons with attention deficit disorder typically do just the opposite. Instead, they shift down into the slow theta frequency band without any significant increase in frontal activity (A. Barabasz & M. Barabasz, 1994, in pressa, -b; Barabasz et al, 1993, in press; Lubar, 1991; Mann et al, 1992). The slow activity (e.g., remaining in alpha 8-14 Hz or dropping down to theta) is characteristic of the wandering mind, non vigilance, and unfocused thought.

Before commencing neurotherapy, a neurometric evaluation involving a computer analysis of a minimum of 19 active scalp electrode sites must be conducted to confirm or disconfirm the above reactivity to key attentional tasks. It is inappropriate to proceed with standardized single channel (single electrode site) EEG feedback on the basis of a DSM-III-R (American Psychiatric Association, 1987) or DSM-IV (American Psychiatric Association, 1994) diagnosis alone. The diagnosis is confirmed by complex digital analyses including evaluation of spectral arrays and comprehensive color topographic maps of brain activity. First, raw EEG is assessed to remove artifacts and for pathological activity. Then fast fourrier transforms are performed to provide averaging of EEG activity in a range of bands or sub-ranges within bands. Only patients who show EEG responses to attentional tasks that are characteristic of those with attention deficit

disorder are appropriate candidates for neurotherapy.

In neurotherapy, EEG responses to stimuli displayed on a computer screen are real time analyzed for frequency, amplitude and artifact characteristics. Then the computer provides feedback information, in the form of visual displays and stereophonic auditory tones or verbalizations showing how well the subject is doing. Unlike the EEG biofeedback apparatus of the 1970's and 80's, continuous computer analysis allows detection of muscle induced artifacts. Subjects are now only provided with reinforcement for EEG responses which, for example, may include augmentation of one band while simultaneously inhibiting another. Several companies compete for the most intriguing forms of feedback, varieties of data analyses provided for therapists and range of features and display alternatives. Recently, Allen Pope of NASA's Human Engineering Group and Edward Bogart of Lockheed Sciences, have gone so far as to develop the concept even further by producing a video game which becomes more difficult as the attention deficit disordered child's brain waves show attention is waning (Pope & Bogart, 1991, in press). As in the more established focus of neurotherapy, this approach teaches children to decrease the time spent in slow wave activity and increase the time spent in the fast activity required for focused attention and concentration on tasks. It takes 40-80 sessions (40 minutes to 1 hour each) for neurotherapy to produce lasting EEG and clinical changes. Follow-up neurometric and clinical assessments should be conducted upon completion of the course of treatments and at about 1 month, 6 months, and 1 year follow-ups.

Chartier and Kelly (1991) reviewed the effects of neurofeedback for ADD/ADHD on over 200 children treated by Dr. Joel Lubar at the University of Tennessee, Dr. John Carter at the University of Texas and Dr. Michael Tansey of Sommerville, New Jersey. Consistent with our own findings, Chartier and Kelly found neurofeedback training to provide significant and sometimes "dramatic" clinical improvements in children with attention deficit disor-

der. Using an A-B-A design, Lubar and Shouse (1977) treated groups of ADD children with the standard protocol (reinforce Beta and inhibit Theta). The protocol was then switched to inhibit Beta and enhance Theta. The subjects, parents, and teachers were kept blind regarding the switch, but within two weeks they began reporting that the childrens' behaviors and attentional skills were deteriorating. Returning to the standard protocol at 4 weeks, the children, parents, and teachers noted resumption of academic and behavioral improvements. Gaddes and Edgell's (1994, p. 279-280) summary of studies reports 80% of children treated show significant measurable improvements on I.Q. tests, standardized tests of academic achievement, and teacher/parent ratings of behavior. Once the full course of treatments have been completed, long term follow-ups indicate that the effects last. Maintenance treatments are not necessary (Chartier & Kelly, 1991).

Presently, limitations of neurofeedback include: 1) the need for additional controlled experimental studies demonstrating effects which are independent of developmental maturation and the potentially confounding effect of the therapist's and parents' attention during the course of treatments; and 2) the large number of sessions (up to 80; 6-8 months) required for permanent clinical and academic changes to occur.

Instantaneous Neuronal Activation Procedure (INAP): Adjunct to Neurotherapy

INAP was developed on the basis of research on hypnotic phenomena (Barabasz, 1980a, 1980b, 1982; 1985; A. Barabasz, Baer, Sheehan & M. Barabasz, 1986; A. Barabasz & M. Barabasz, 1989, 1992, 1994b; A. Barabasz, Crawford, & M. Barabasz, 1993, in press; Barabasz & Lonsdale, 1983; Hilgard, 1979; Brown & Fromm, 1986; Hammond, 1990, p. 42; Holroyd, 1985-1986; Crawford & Gruzelier, 1992). Building on the data showing that hypnosis is a state of attention that may be focused, to exclude distracters, or diffuse depending on the specific instructions (suggestions) (Fromm, 1987; Hilgard, 1965, 1986), we

observed that the more general attentional processes involved in vigilance, such as military radar target detection, could be enhanced with alert hypnosis (Barabasz, 1980b). Later, military pilot instrument flight reliability was improved with an active alert attentional procedure to help optimize pilots' situational awareness with regard to cockpit navigation cues (Barabasz, 1985).

The attentional shifts made possible with hypnosis have been causally linked to specific EEG changes, which, as Hilgard foreshadowed (1965, 1986), vary depending upon the specific instructions given. In the first fully controlled experiment to demonstrate an EEG signature unique to hypnosis, Barabasz & Lonsdale (1983) discovered amplitude increases in the late components of EEG olfactory event related potentials (ERP) in response to specific instructions. The findings were consistent with a related field study conducted in Antarctica (Barabasz & Gregson, 1979) and subsequent cerebral blood flow investigations (Crawford, Skolnick, Benson, R. Gur & C. Gur. Later, Spiegel, Cutcomb, Ren & Pribram (1985) also showed highly significant ERP amplitude changes after specific hypnotic instructions. Spiegel and Barabasz, (1988) discussed how EEG ERPs could be modified dramatically in response to alternative instruc-

The alert attentional instructions used to produce human performance enhancement effects in radar operators and pilots (Barabasz, 1980b, 1985) have been applied in more refined and replicable forms to modify EEG topography and enhance focused attention for reading tasks. Using 19 active electrode sites we were able to demonstrate normalization of EEG during reading with shifts to Beta and shifts toward frontal regions following INAP in children with ADD/ADHD (A. Barabasz & M. Barabasz, 1993a, b; A. Barabasz, Crawford, & M. Barabasz, 1993, in press).

The INAP intervention has also demonstrated significant effects in a carefully controlled experiment using normal young adults. To help control for experimental demand effects, A. Barabasz & M. Barabasz (1994a, b)

embedded the INAP procedure into an EEG neurological screening aspect of another study. Eleven subjects were exposed to counterbalanced conditions of waking, attentional instructions, and INAP. A simple suggestion emphasizing speed and memory retention was given prior to subjects' exposure to parallel forms of a standardized reading comprehension test. Both attentional instructions and INAP significantly increased BETA magnitude at key frontal sites while significantly increasing reading speed, but only INAP served to significantly increase reading comprehension performance. Unfortunately, testing of long term effects was beyond the scope of the study.

Recently, INAP has been used as an experimental adjunct to standard neurotherapy (enhance beta in range 14-20 Hz while inhibiting theta 4-8Hz) in the treatment of ADHD (A. Barabasz & M. Barabasz, in press a). Experimental cases were chosen from our clinical experience which provided direct comparisons in treatment process, progress, outcomes and 1-year follow-ups. Patients in both conditions were matched closely with respect to gender, age, WISC-III verbal and performance I.Q. scores and scores on the Stanford Hypnotic Clinical Scale for Children (Morgan & Hilgard, 1978). Patients in both conditions had failed a grade in school as a direct result of their ADHD and had been medicated for 2 or more years with Ritalin when beginning treatment with us. Families were intact. ADHD symptoms and severities were very similar. Histories included active parental and school teacher participation in behavioral modification programs with little positive effect.

Using the standard neurotherapy protocol without hypnosis, treatment was completed without need for maintenance sessions after 60-70 sessions (2-3 per week). Upon completion of treatment, EEG thetalbeta ratios were consistent with normals in response to a variety of attentional tasks. Medication was no longer used and significant gains in I.Q., school grades and parent/teacher deportment ratings had been realized. All gains were maintained at a 1 year follow-up.

INAP which takes about 45 to 90 seconds

per administration (described in detail in A. Barabasz & M. Barabasz, in press) used in conjunction with neurotherapy resulted in an outcome equivalent to or better than those obtained with standard neurotherapy. The standard INAP procedure, is conducted in two distinct phases (A. Barabasz & M. Barabasz 1994a, b). In the training phase, patients are instructed to roll eyes up. Eye focus is led to this position by instructions to focus on the psychologist's thumb. The thumb is then moved slowly from 10-15 cm in front of the patient's nose to the approximate center of the forehead. Speed of movement is carefully coordinated with the patient's ability to follow without swimming of the eyes or obvious loss of focus. When eyes dart, or focus seems lost, the procedure is reinitiated. Normal adults seldom have a problem with this procedure (A. Barabasz & M. Barabasz, 1994), but clinical experience and patience may need to be brought to bear in the treatment of hyperactive children to get eyes as fully rolled up as possible and then kept steadily rolled up as required for successful INAP effects. Once the eyes are fully and steadily rolled up, instructions are then given to take notice of breathing and the relaxation, confidence and special alertness felt at this point. Once subjective signs of hypnosis are observed by the experienced clinician, the patient is asked to raise a finger upon perception of the suggested responses "... just lift a finger on this hand (clinician touches patient's non-dominant hand) when you feel the comfortable relaxation and special alertness." Upon observation of the patient's signal, which should occur within 5-10 seconds, the patient is given the attentional process specific suggestions such as, "in this special state of alertness you will be able to focus your attention anyway you like, you can concentrate as completely as you desire." The addition of INAP to neurotherapy appeared to be the key therapeutic ingredient to produce these same lasting widely reported positive effects in only 30-32 (2-3 per week) sessions. It is noteworthy that the progress in the enhancement of frontal beta, stagnated in sessions when INAP was not used and resumed upon reinitiation of INAP. We speculate that had this A-B-A test not

been incorporated into the treatment program that the number of sessions required to meet EEG and behavioral normalization criteria might have been even further reduced.

Additional Findings

Our continued experimental clinical tests of INAP subsequent to our previous study (A. Barabasz, & M. Barabasz, in press a) have produced additional findings which may be useful to clinicians. Previous uses of INAP as an adjunct to neurotherapy have included a procedure which asks patients to confirm experiencing feelings of relaxation, confidence and special alertness by raising a finger during INAP. We no longer see the need for this additional technique since it appears to have no measurable effect on the 14-20 Hz Beta enhancement produced by INAP. An alternative procedure appears to be helpful when treating patients who have special difficulties with obtaining and/or maintaining this eyes rolled up position required for successful results. Normally the patient is asked to focus on the clinician's stationery thumb held 10-15 cm in front of the patient's nose. Then the patient's eyes are led to the rolled up position by following the clinician's slow movement to the center of the patient's forehead. A new alternative method we have found useful is to continuously wiggle the first digit (index finger) to help maintain the patient's focus/attentiveness throughout the upward movement of the hand which is kept at 10-15 cm from the patient's head rather than descending to the patient's forehead. This variation of the technique can be used alternately with the original procedure or exclusively with patients experiencing difficulty in maintaining eyes rolled up for the minimum period of 30 seconds.

Response to INAP is easily determined on the basis of EEG criteria. Using a simple EEG feedback protocol, and frontal electrode placement, the increase in Beta from pre- to post-INAP should be obvious in terms of time in Beta production and Beta amplitude. This response is so robust as to be used as a training criteria for clinicians adding INAP to their neurotherapy protocols.

To date, one of the criticisms of neurotherapy has been the lack of control for maturation during the, heretofore, lengthy 40-80 session and five to eight month course of treatment. It has been argued that the successful alleviation of the negative responses of those with attention deficit disorder may not be due to neurotherapy, but rather simply to a "growing out of it" effect coincident with the long treatment. This issue has not been raised with respect to the traditional stimulant medication and/or behavior modification treatments because the goal of these treatments has been limited to immediate symptom management. Even after "successful" treatment, the patient rapidly returns to baseline behavior upon cessation of medication/behavior modification. Assuming that further clinical trials and experimental research supports the effect of INAP's reduction of treatment time to 25-35 sessions and only two to three months, the maturation argument becomes even less defensible.

Perhaps the greatest limitation of our knowledge of INAP effects in the treatment of attention deficit disorder is in our inability to directly tie its clearly demonstratable effect on EEG Beta production with hypnosis. because we administer a procedure that was conceptualized within a type of active alert hypnotic induction and then observe effects in the predicted direction, by no means demonstrates that such effects are due to hypnosis per se. As noted in our work on experimental and clinical research design considerations (A. Barabasz & M. Barabasz, 1992) little can be said about the specificity of hypnosis if hypnotizability is not measured. This theoretical issue may make little difference to the clinician so long as the results are in the desired direction, but does point to the need for further research. Our ADD/ADHD cases have all been at least moderately high in hypnotizabilty as determined by the standardized tests. Children with ADD/ADHD and low hypnotizability scores are difficult to find and the only rigorously controlled experimental research on INAP to date (A. Barabasz, & M. Barabasz, 1994a, b) used moderately hypnotizable subjects. This means that thus far we do not know whether hypnosis per se can be viewed as the

causal factor. Perhaps the entirely nonhypnotic aspects of INAP account for the significant contributions to the positive effects on neurotherapy outcomes.

References

- American Psychiatric Association (1987).

 Diagnostic and statistical manual for mental disorders, 3rd Edition, Revised.

 Washington, D.C.: American Psychiatric Association.
- American Psychiatric Association (1994). Diagnostic and statistical manual for mental disorders, 4th Edition. Washington D.C.: American Psychiatric Association.
- Barabasz, A. (1980a). EEG alpha, skin conductance and hypnotizability in Antarctica. International Journal of Clinical and Exprerimental Hypnosis, 28, 63-74.
- Barabasz, A. (1980b). Effects of hypnosis and perceptual deprivation on vigilance in a simulated radar target detection task. *Perceptual and Motor Skills*, 50, 19-24.
- Barabasz, A. (1982). Restricted environmental stimulation and the enhancement of hypnotizability: EEG alpha, skin conductance and temperature responses. *International Journal of Clinical and Experimental Hypnosis*, 30, 147-166.
- Barabasz, A. (1985). Enhancement of military pilot reliability by hypnosis and psychophysiological monitoring: In-flight and simulator data. *Aviation, Space and Environmental Medicine, March, 248-250.*
- Barabasz, A. (1993). Presidential Address: Antarctic isolation and attentional processes: Research implications for practitioners. Presented at the Fifth International Conference on REST, Seattle, WA, Feb. 26-28.
- Barabasz, A. (in press) Instantaneous neuronal activation procedure (INAP): Reduced stimulation and psychophysiological monitoring in the treatment of phobias. Experimentalle und Klinishe Hypnose.
- Barabasz, A., & Barabasz, M. (In press a). Neurotherapy and alert hypnosis in the

- treatment of attention deficit disorder. In S. Lynn, 1. Kirsch & J. Rhue (Eds) *Clinical Hypnosis Handbook*, Washington, D. C.: The American Psychological Association.
- Barabasz, A., & Barabasz, M. (In press b). Diagnosis, etiology and treatment of attention deficit hyperactivity disorder. In M. Barabasz (Ed.) Special Issue on Attention Deficit Disorders, *Child Study Journal*.
- Barabasz, A., & Barabasz, M. (1989). Effects of restricted environmental stimulation: Enhancement of hypnotizability for experimental and chronic pain control. International Journal of Clinical and Experimental Hypnosis, 37, 217-223.
- Barabasz, A., & Barabasz, M. (1992). Research Design Considerations, In E. Fromm & M. Nash (Eds.), *Contemporary Hypnosis Research*. New York, NY: Guilford, 173-200.
- Barabasz, A., & Barabasz, M. (Eds) (1993a).

 Clinical and Experimental Restricted

 Environmental Stimulation: New Developments

 and Perspectives. New York: Springer-Verlag.
- Barabasz, A., & Barabasz, M. (1993b). Neurometric assessment of attention deficit disorders, neurofeedback and active alert hypnosis. Presented at the Portland Academy of Hypnosis, Portland Oregon, November 19. (Invited Address)
- Barabasz, A., & Barabasz, M. (1994a). EEG responses to a reading comprehension task during active alert hypnosis and waking states. Presented at the 45th Annual Scientific Meeting of the Society for Clinical and Experimental Hypnosis, San Francisco, Oct. 4-8.
- Barabasz, A., & Barabasz, M. (1994b). Effects of focused attention on EEG topography, Symposium: Behavioral Medicine, Psychophysiology and Hypnosis, Presented at the 102nd Annual Convention of the American Psychological Association, Los Angeles, August 12-16.
- Barabasz, A., Baer, L., Sheehan, D. V., & Barabasz, M. (1986). A three year follow-up of hypnosis and restricted environmental stimulation therapy for smoking.

- International Journal of Clinical and Experimental Hypnosis, 34, 169-18 1.
- Barabasz, A., Crawford, H., & Barabasz, M. (1993). EEG topographic map differences in attention deficit disordered and normal children: Moderating effects from focused active alert instructions during reading, math and listening tasks. Presented at the 33rd Annual Meeting of the Society for Psychophysiological Research, Rottach-Egem, Germany, October, 27-31.
- Barabasz, A. Crawford, H. & Barabasz, M. (In press). Quantitative EEG markers in children with attention deficit disorder: Moderating effects of active alert instructions during reading and math tasks, *Child Study Journal*.
- Barabasz, A., & Gregson, R. A. M. (1979). Antarctic wintering-over, suggestion and transient olfactory stimulation: EEG evoked potential and electroderrnal responses. *Biological Psychology*, 9, 285-295.
- Barabasz, A., & Lonsdale, C. (1983). Effects of Hypnosis on P300 olfactory evoked potential amplitudes. *Journal of Abnormal Psychology*, **92**, 520-525.
- Barabasz, M. (1987). Trichotillomania: A new treatment. *International Journal of Clinical and Experimental Hypnosis*, 35, 146-154.
- Barkley, R. A. (1990). Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment. New York, NY: The Guilford Press.
- Brown, D. & Fromm, E. (1986). Hypnotherapy and Hypnoanalysis. Hillsdale, N.J.: Erlbaum.
- Chartier, D., & Kelly, N. (1991).

 Neurofeedback treatment of attention deficit-hyperactivity disorder. Grand Rounds Presentation, Rex Hospital, Raleigh, N.C. August.
- Chelune, G. J., Ferguson, W., Koon, R., & Dickey, T. 0. (1986). Frontal lobe disinhibition in attention deficit disorder. *Child Psychiatry and Human Development*, 16, 221-232.
- Conte, R. (1991) Attention disorders. In B.

- Wong (Ed.) Learning about learning disabilities. New York: Academic Press, 60-96.
- Crawford, H. J., & Gruzelier, J. H., (1992) A midstream view of the neuropsychophysiology of Hypnosis: Recent research and future directions. In E. Fromm & M. Nash (Eds.) Contemporary Hypnosis Research, New York: Guilford, 227-266.
- Crawford, H. J., Skolnick, B. E., Benson, D. M., Gur, R. E., & Gur, C. (1985, August). Regional cerebral blood flow in hypnosis and hypnotic analgesia. Paper presented at the 10th International Congress of Hypnosis and Psychosomatic Medicine, Toronto.
- David, 0. (1974). Association between lower lead concentrations and hyperactivity in children. *Environmental Health Perspectives*, 7, 17-25.
- Edwards, R. (1995) Is the hyperactivity label applied too frequently? American Psychological Association Monitor, 26, 44-45.
- Feingold, B. (1973). Food additives and child development. *Hospital Practice*, 8, 11-12, 17-18, 21.
- Feingold, B. (1975). Why your child is hyperactive. New York, NY: Random House.
- Firestone, P., Kelly, M. J., Goodman, J. T., & Davey, J. (1981). Differential effects of parent training and stimulant medication with hyperactives. *Journal of the American Academy of Child Psychiatry*, 20, 135-147.
- Fromm, E. (1987) Significant developments in hypnosis during the past 25 years. International Journal of Clinical and Experimental Hypnosis, 35, 215-230.
- Gaddes, W. H. & Edgell, D. (1994) Learning Disabilities and Brain Function. New York: Springer-Verlag.
- Gualteri, C. T., & Hicks, R. E. (1985). Neuropharmacology of methylphenidate and a neural substitute for childhood hyperactivity. *Psychiatric Clinics of North America*, 8, 875-892.
- Hilgard, E. R. (1965). *Hypnotic Susceptibility*, 4, New York: Harcourt.

- Hilgard, E. R. (1979). Consciousness and control: Lessons from hypnosis. Australian Journal of Clinical and Experimental Hypnosis, 7, 107-115.
- Hilgard, E. R. (1986). Divided Consciousness: Multiple Controls in Human Thought and Action (rev. ed.) New York; Wiley.
- Holroyd, J. (1985-1986). Hypnosis applications in psychological research. *Imagination, Cognition and Personality*, 5, 103-115.
- Hammond, D.C (1990). Handbook of hypnotic suggestions and metaphors, New York: W.W. Norton.
- Hynd, G. W. Hem, K. L., Voeller, K. K., & Marshall, R. M. (1991). Neurobiological basis of attention-deficit hyperactivity disorder (ADHD). School Psychological Review, 20,174-186.
- Hynd, G. W., Semrud-Clikeman, M., Lorys, A., Novey, E. S., & Eliopulos, D. (1990). Brain morphology in developmental dyslexia and attention deficit disorder/hyperactivity. Archives of Neurology, 47, 919-926.
- Lou, H. C., Henriksen, L., Bruhn, P., Bomer, H., & Nielsen, J. (1989). Striatal dysfunction in attention deficit and hyperkinetic disorder. Archives of Neurology, 46, 48-52.
- Lubar, J. F., (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Self-Regulation*, 16, 201-225.
- 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, 293-306.
- Lubar, J. F. & Shouse, M. N. (1977). Use of biofeedback and the treatment of seizure disorders and hyperactivity, *Advances in Child Clinical Psychology*. N.Y.: Plenum, 1, 204-251.
- 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.
- Marshall, P. (1989). Attention deficit disorder and allergy: A neurochemical model of the relation between the illness. *Psychological Bulletin*, **106**, 434-446.
- Morgan, A. H. & Hilgard, E. R. (1978). The stanford hypnotic clinical scale for children. *American Journal of Clinical Hypnosis*, 21, 148-169.
- Pope, A. T. & Bogart E. H. (In press). Extended attention span training system: Video game neurotherapy for attention deficit disorder. In M. Barabasz (Ed.) Special Issue on Attention Deficit Disorders, *Child Study Journal*.
- Schaughency, E. A., & Hynd, G. W. (1989). Attention and impulse control in attention deficit disorders (ADD). Learning and Individual Differences, 1, 423-449.
- Spiegel, D., & Barabasz, A. (1988). Effects of hypnotic hallucination on P300 evoked potential amplitudes: Reconciling conflicting findings. American Journal of Clinical Hypnosis, 331, 11-17.

- Spiegel, D., Cutcomb, S., Ren, C., & Pribram, K. (1985). Hypnotic hallucination alters evoked potentials. *Journal of Abnormal Psychology*, 94, 249-255.
- Sterman, M. B., & Friar, L. (1972). Suppression of seizures in an epileptic following sensorimotor EEG feedback training. Electroencephalography & Clinical Neurophysiology, 33, 89-95.
- Swanson, J., Mcbumett, T., Wigal, T., Pfiffner, L., Lemer, M., Williams, L., Christian, D., Tamm, L., Willcutt, E., Crowley, K., Clevenger, W., Khouzam, N., Woo, C., Crinella, F., Fisher, T., (1993). Effect of stimulant medication on children with attention deficit disorder: A "review of reviews." *Exceptional Children*, 60, 2, 154, 162.
- Voeller, K. K. S.. & Heilman, K. (1988). Attention deficit disorder in children: A neglect syndrome? *Neurology*, 38, 806-808.
- Whalen, C. K. & Henker, B. (1991). Therapies for hyperactive children: Comparisons, combinations, and compromises. *Journal of Consulting and Clinical Psychology*, **59**, 1, 126-137.



Correspondence should be addressed to Prof. Arreed Barabasz, Ed.D., Ph.D., A.B.P.P., Attentional Processes Laboratory, Cleveland Hall, Washington State University, Pullman, WA 99164-2136