

Neurofeedback Outcomes in Clients with Asperger's Syndrome

Lynda Thompson · Michael Thompson ·
Andrea Reid

Published online: 12 November 2009
© Springer Science+Business Media, LLC 2009

Abstract This paper summarizes data from a review of neurofeedback (NFB) training with 150 clients with Asperger's Syndrome (AS) and 9 clients with Autistic Spectrum Disorder (ASD) seen over a 15 year period (1993–2008) in a clinical setting. The main objective was to investigate whether electroencephalographic (EEG) biofeedback, also called neurofeedback (NFB), made a significant difference in clients diagnosed with AS. An earlier paper (Thompson et al. 2009) reviews the symptoms of AS, highlights research findings and theories concerning this disorder, discusses QEEG patterns in AS (both single and 19-channel), and details a hypothesis, based on functional neuroanatomy, concerning how NFB, often paired with biofeedback (BFB), might produce a change in symptoms. A further aim of the current report is to provide practitioners with a detailed description of the method used to address some of the key symptoms of AS in order to encourage further research and clinical work to refine the use of NFB plus BFB in the treatment of AS. All charts were included for review where there was a diagnosis of AS or ASD and pre- and post-training testing results were available for one or more of the standardized tests used. Clients received 40–60 sessions of NFB, which was combined with training in metacognitive strategies and, for most older adolescent and adult clients, with BFB of respiration, electrodermal response, and, more recently, heart rate variability. For the majority of clients, feedback was contingent on decreasing slow wave activity (usually 3–7 Hz), decreasing beta spindling if it was present (usually between 23 and 35 Hz), and increasing fast wave

activity termed sensorimotor rhythm (SMR) (12–15 or 13–15 Hz depending on assessment findings). The most common initial montage was referential placement at the vertex (CZ) for children and at FCz (midway between FZ and CZ) for adults, referenced to the right ear. Metacognitive strategies relevant to social understanding, spatial reasoning, reading comprehension, and math were taught when the feedback indicated that the client was relaxed, calm, and focused. Significant improvements were found on measures of attention (T.O.V.A. and IVA), core symptoms (Australian Scale for Asperger's Syndrome, Conners' Global Index, SNAP version of the DSM-IV criteria for ADHD, and the ADD-Q), achievement (Wide Range Achievement Test), and intelligence (Wechsler Intelligence Scales). The average gain for the Full Scale IQ score was 9 points. A decrease in relevant EEG ratios was also observed. The ratios measured were $(4-8 \text{ Hz})^2/(13-21 \text{ Hz})^2$, $(4-8 \text{ Hz})/(16-20 \text{ Hz})$, and $(3-7 \text{ Hz})/(12-15 \text{ Hz})$. The positive outcomes of decreased symptoms of Asperger's and ADHD (including a decrease in difficulties with attention, anxiety, aprosodias, and social functioning) plus improved academic and intellectual functioning, provide preliminary support for the use of neurofeedback as a helpful component of effective intervention in people with AS.

Keywords Asperger's · Neurofeedback · EEG · Biofeedback · Intelligence · T.O.V.A. · IVA · Aprosodia · Anterior cingulate · Mirror neurons · Metacognition

Introduction

Background Regarding Asperger's Syndrome

People with Asperger's Syndrome (AS) “just don't fit in”. Their symptoms were first described by the Viennese

L. Thompson (✉) · M. Thompson · A. Reid
ADD Centre, 50 Village Centre Place, Mississauga,
ON L4Z 1V9, Canada
e-mail: addcentre@gmail.com

pediatrician, Asperger (1944). He described a group of boys who were like “little professors” with advanced knowledge in a special interest area and pedantic language that contrasted with delayed social skills and awkward motor skills. The syndrome came to bear his name after the British psychiatrist and autism expert Lorna Wing wrote about the constellation of symptoms in 1981, thus bringing it to the attention of English speaking psychiatrists. The American Psychiatric Association included Asperger’s Disorder in the 1994 revision of their Diagnostic and Statistical Manual (DSM-IV) and the rates of diagnosis of AS have been increasing since that time (Bashe and Kirby 2005; Nash 2002). There has also been an increase in diagnoses of autism since the early 1990s (Attwood 1997). *Asperger’s Disorder* shares with other disorders along the autism continuum (called Pervasive Developmental Disorders in the DSM-IV) deficits in social understanding, range of interests, and imagination (social imagination, flexible thinking, and imaginative play). It differs from autism in that there are no significant developmental delays in language or cognition (American Psychiatric Association 1994). *Asperger’s Syndrome*, on the other hand, does allow for language delay in the early years (though typically the child eventually develops advanced language skills, albeit with some differences in their speech, such as pedantic phrases and lack of prosody—intonation and rhythm) and it can be diagnosed in children with a wide range of intellectual functioning. Additionally, poor motor coordination (odd gait and poor fine motor skills) are among the criteria for AS but are not mentioned in DSM-IV criteria for Asperger’s Disorder (Attwood 1997; Gillberg and Billstedt 2000; Wing 2001). Prevalence for AS has been estimated at 36 per 10,000 in school-age children and the syndrome is much more frequent in boys, with at least a 4:1 ratio of males to females diagnosed (Attwood 1997; Ehlers and Gillberg 1993).

Clients with AS are usually very honest and take things literally. Social skills training helps but these skills often do not fully generalize. The first author has heard many tragic-comic stories from parents when taking histories; for example, the Kindergarten child who, when the class was learning about different professions, was told to be a dog in a skit about a veterinarian. He proceeded to run about on his hands and knees, bark, and then bite the other child (the “vet”) on the leg. Another boy had a mother who would assiduously teach her son the rules of social engagement for new situations. She carefully told him how to treat a new friend when they went on vacation: the first day he successfully made a connection with another boy at the resort, but the second day he ignored the boy. He told his astonished mother that he had not forgotten the rules she gave him the first day, but now this boy was not a “new” friend.

Such stories help distinguish between AS and Attention-Deficit/Hyperactivity Disorder, a common presenting

diagnosis. Overlap with symptoms of ADHD is so frequent that some authors recommend treating the symptoms of ADHD before making a diagnosis of AS (Fitzgerald and Kewley 2005). Starting in the early 1990s, this was the approach taken at the ADD Centre. Parents were told that there were good (though uncontrolled) case series published in professional literature showing that clients with ADHD became more attentive and less impulsive after about 40 sessions of neurofeedback (NFB). In more recent years we were able to say that NFB was an established intervention for ADHD (Yucha and Gilbert 2004). Parents were told that for their child with AS, though NFB would be considered experimental, it was logical to try NFB both because paying attention was part of his/her presenting problems and because the EEG patterns differed in similar ways when a single channel assessment was performed at the vertex; that is, the assessment revealed an immature pattern with excess slow wave activity.

As work proceeded with increasing numbers of clients with AS, 19-lead assessments were also performed in some cases. Comparisons using standard databases (SKIL [Serman-Kaiser Imaging Laboratory, Version 3.0 (2007). Copyright 2001] and/or Neuroguide) yielded additional findings of abnormal coherence patterns, in particular, lack of communication (hypocoherence) between left frontal and right temporal-parietal regions and too much common activation (hypercoherence) within the right (or left) hemisphere. There were also amplitude differences at various 10–20 electrode placement sites. The source of those abnormalities when LORETA analysis (Low Resolution Electromagnetic Tomography Analysis, Pascual-Marqui et al. 2002) was applied was most often the anterior cingulate. Other involved cortical areas implicated by LORETA included the superior temporal gyrus, amygdala, uncus, insula, fusiform gyrus, orbital and medial frontal lobe, hippocampal gyrus, and parahippocampal gyrus. Correspondence between symptoms and the functions of the areas found to have abnormalities are discussed in another paper (Thompson et al. 2009). The EEG findings, in conjunction with theories concerning AS, including Stephen Porges’ polyvagal theory (2003, 2004), were used to develop a rationale for implementing neurofeedback combined with general biofeedback and, in particular, respiration and heart rate variability. For a simple explanation of the polyvagal theory see the interview conducted with Stephen Porges by Dykema (2006).

Correlation of AS Symptoms, EEG Findings, and Functions of Different Brain Areas

Of particular interest with respect to neurofeedback are studies that investigate how brain anatomy and neurological functioning differ in those with Asperger’s. Sensory

aprosodia (difficulty interpreting tone of voice, body language, gesture and facial expression) frequently correlates with less activation at T6, as evidenced by increased theta and/or alpha activity or decreased 16–18 Hz beta activity (Thompson et al. 2009). Difficulties in the ability to understand motivations and intentions of others correlates with dysfunction in a frontal mirror neuron area near F5 (Dapretto et al. 2006; Iacoboni and Dapretto 2006) and these difficulties include problems with empathy (Pfeifer et al. 2005). Motor aprosodia (not expressing emotion in tone of voice, gestures or facial expression) frequently correlates with signs of inactivity at F6, which is also a frontal mirror-neuron area. Anterior cingulate functions underlie many of the symptoms, including problems with attention (Devinsky et al. 1995). Difficulties with disengaging and shifting attention (Landry and Bryson 2004) and symptoms related to elevated anxiety appear to correlate with EEG amplitudes outside Neuroguide database norms for theta, low alpha and/or high frequency beta with a source in the anterior cingulate. Anxiety may also correlate with beta spindling activity related to the anterior cingulate gyrus at Brodmann area (BA) 24. Difficulties modulating affect in our assessments appear to correlate with EEG amplitudes outside database norms in one or more of several limbic areas that have been identified by researchers as not functioning normally in persons with ASD. These include: anterior cingulate gyrus, medial aspect of the frontal lobe, superior temporal lobe, insula (Ramachandran and Oberman 2006), uncus and amygdala (Bachevalier and Loveland 2006), hippocampus and parahippocampal gyrus (Salmond et al. 2005), and the medial and orbital regions of the frontal lobe (Shamay-Tsoory et al. 2005). These findings are discussed in more detail in an earlier paper (Thompson et al. 2009).

Interventions for AS

Medications, social skills training, behavior therapy, and educational interventions have been the most commonly used interventions for children who present with the symptoms of Asperger's Syndrome. Gattegno and De Fenoyl (2004) propose group psychotherapy that involves teaching social abilities. Loffler (2005) and Blandford (2005) provide management advice to teachers. Another helpful publication for educators is *Asperger's Syndrome: A practical guide for teachers* (Cumine et al. 1998).

The multiplicity of attempted interventions attests to the observation that there is no universally accepted method for intervention with Asperger's. Given the correlation between EEG assessment findings in persons with AS and areas of cortical dysfunction found using other methodologies, it seems reasonable to attempt to apply a learning paradigm that allows a person to make changes that can be

seen in measurements of his/her brain's electrical activity and thereby achieve a change in functioning. Note that causation is not implied: the EEG *reflects* brain functioning and is thus a way to measure changes. We do not know the exact mechanisms for the changes. In the last decade, a few papers and presentations about intervention using neurofeedback have appeared (Coben 2005, 2007; Jarusiewicz 2002; Linden et al. 1996; Reid 2005; Solnick 2005; Thompson and Thompson 1995, 2003a, b, 2004, 2005, 2007a, b, c; Thompson et al. 2009). Results using this methodology with clients diagnosed with AS in a clinical setting over the last 15 years (1993–2008) is the subject of this review.

Method

Participants

Participants were comprised of 159 clients seen consecutively over a 15-year period who received both assessment and neurofeedback training in a clinical setting. Within the group, 150 satisfied the criteria for AS and 9 were diagnosed with Autistic Spectrum Disorder (DSM-IV classifications of Autism or Pervasive Developmental Disorder, NOS). There were 117 children (ages 5–12 years), 30 adolescents (ages 13–19), and 12 adults (ages 20–58) with 139 males and 20 females. The male:female ratio was thus about 7:1. Given the cultural diversity of the Toronto area, the participants were mixed in terms of ethnic backgrounds, countries of origin, and socioeconomic status. Most were self-referred to the ADD Centre in order to deal with problems in attention and many had not previously been diagnosed as having AS. The most common previous diagnosis was Attention-Deficit/Hyperactivity Disorder (ADHD).

Assessment and Testing

The first author completed the initial portion of the assessment for establishing a diagnosis. The assessment entailed a half-day evaluation that included history taking, review of present and past symptoms via questionnaires, administration of computerized continuous performance tests (Test of Variables of Attention [T.O.V.A.: Universal Attention Disorders Inc., 4281 Katella Ave. #215, Los Alamitos, CA 90720] and, once it was available, the Integrated Visual Auditory continuous performance test, [IVA: BrainTrain, 727 Twin Ridge Lane, Richmond VA 23235.]), a single-channel EEG assessment collected at the vertex (CZ), and a brief neurofeedback training session. The sample NFB usually involved a baseline plus four 2-min feedback conditions with parameters based on the EEG findings from the single channel assessment.

History taking followed a set format for collecting information and resulted in generation of an initial assessment report that was shared with parents. The report has the following headings: Present Situation, Background Information (including developmental history and school history), Medical History (including details concerning medications, sleep, exercise, and diet), Family (including mention of parental occupations, siblings, and questions regarding symptoms present in extended family members), Summary and Opinion (gives diagnosis and recommendations), and Objectives for Training.

The questionnaires were completed by parents, usually while they sat in the same room with their child as he/she completed the T.O.V.A. Three scales were typically used for assessing symptoms of ADHD in children. The Conners' Global Index for Parents has 10 items rated on a 4-point scale from Never (0) to Almost Always (3) so scores can range from 0 to 30. The Conners' has been normed for children from age 5–17 and provides T-scores based on age and sex. Scores >65 are significant. Norms were developed in 1998 (Multi-Health Systems Inc.) and were updated in 2008. The same 10 questions, usually referred to as the Conners' Abbreviated Rating Scale, had been widely used in research since the 1970s simply using raw scores with a cut-off score of >15 as indicative of significant problems with respect to ADHD (Appendix, p. 238 in Wender 1995). The SNAP version of the DSM-IV (Swanson et al. 1993) also uses a 4-point scale (Not at All to Very Much) and has 23 items covering attention, impulsivity, hyperactivity and peer relationships. The scale is not normed and in this study raw scores were tracked (range 0–69). The ADD-Q (Sears and Thompson 1998) was developed for use at the ADD Centre and the 30-item, 4-point scale (Never or Very Rarely = 0 to Almost Always = 3) thus has a range of scores from 0 to 90. There are no norms but clinical experience suggests scores above 35 nearly always are associated with a diagnosis of ADHD. A questionnaire specific to AS, the Australian Scale for Asperger's Syndrome, was added after its publication in Tony Attwood's book, *Asperger's Syndrome: A guide for parents and professionals* (1998). For adults three questionnaires related to ADHD were used. The Wender-Utah Rating Scale (WURS) is retrospective ("As a child I was...") (Appendix, pp. 245–246 in Wender 1995). The DSM-IV criteria are similar to the questions used for children but reworded for adults. The ADD Centre Questionnaire (ACQ) was developed at the same time as the ADD-Q and is available through the ADD Centre. None of the adult scores have been normed but one research study validated the WURS against the other two and provided cut-off scores and ranges for adults with ADHD compared to non-ADHD adults (Collins-Williams 1997).

Intellectual and academic testing were completed by the first author during a second visit if this type of testing had not already been completed within the past 2 years. The appropriate, current version of the Wechsler Intelligence Scale was used for the intellectual measure (WISC-R, WISC-III, WISC-IV, for ages 6–16 and WAIS-R and WAIS-III for those 17 years and above). Canadian norms were used in the scoring when available. The academic screening measure used was the current edition of the Wide Range Achievement Test (WRAT-R, WRAT3, WRAT4). Clients were also asked to draw a person at the time of the initial testing and each time progress testing was done but the drawings were not scored so results are not reported. (They can be scored as an intelligence measure using the Goodenough-Harris scoring, but the Wechsler Scales are more appropriate for that purpose.) The d-a-p task does, however, always yield clinically interesting information for generating hypotheses about emotional functioning. In those with AS there is usually reluctance to draw a person, especially the face. Often the request will yield something other than a human figure, such as a detailed train with a little head to show the engineer, an animal, a goalie wearing a mask, a cartoon figure, or just a stick figure. Changes after training are observed, especially with respect to eyes, hands and feet, details which are often missing initially.

Psychophysiological stress assessments were conducted with most adult clients and were completed jointly by the second and third authors (see the chapter by Thompson and Thompson 2007a, b, c). These assessments were used only to determine which biofeedback modalities would be incorporated in neurofeedback; they were not repeated after training. Testing involving T.O.V.A., IVA, questionnaires, EEG ratios, I.Q., and WRAT3/4 was accomplished at intake, after 40 sessions of training, and, for those who completed more sessions, again after 60 sessions of training were finished. With respect to the Wechsler Intelligence Scales, testing was only completed twice: at baseline before training began (using scores obtained by the author or sometimes already by another psychologist) and then the appropriate version re-administered by the first author at the time of either the 40 session or 60 session progress testing. Intellectual assessment would be deferred at the time of the 40-session progress testing until after 60 sessions if discussion with parents after 40 sessions made it clear that further training would be undertaken. Training was typically scheduled twice per week, so 40 sessions required at least 20 weeks, which would typically be completed in 5–6 months depending on holidays. The pre-post test interval was thus 6 months or more.

For EEG ratios, the single channel A620 assessments (Stoelting Autogenics, 6200 Wheat Lane, Woodale, Illinois 60191) were collected by the first author using the methods

developed by Lubar and colleagues (Lubar 1991; Monastra et al. 1999). In the last 7 years, the ratios obtained from the Autogen assessment have been supplemented, for a reliability check, by the assessment program on the procomp+/Biograph or the BioGraph Infiniti (Thought Technology). The newer equipment has been used for the mini-training session that forms part of the initial assessment for the same length of time. For purposes of consistency, three assessment ratios from the Autogen assessment program are used in this paper: a ratio comparing theta (4–8 Hz) to beta (16–20 Hz) activity as a ratio in microvolts, the $(4-8 \text{ Hz})^2/(13-21 \text{ Hz})^2$ theta/beta power ratio in picowatts, and theta/sensorimotor (SMR) using $(3-7 \text{ Hz})/(12-15 \text{ Hz})$ in microvolts. Though not reported here, in adult clients seen within the last 9 years, ratios of high frequency beta (23–35 Hz) to sensorimotor rhythm (13–15 Hz) and high frequency beta (19–21 Hz) to high frequency alpha (11–12 Hz) have also been examined as they are thought to reflect ruminations and anxiety (Thompson and Thompson 2006).

Full-cap 19 channel assessments were carried out on selected clients using Lexicor, Neuronavigator, Mindset or Neuro-Pulse instruments for data collection. Analysis of 19 channel EEG was accomplished using SKIL and/or Neuroguide plus LORETA. The Mindset and its up-dated version, Neuro-Pulse, both collect data directly linked to the Neuroguide software program. Note that it is not always easy, possible, or even advisable to attempt 19-channel assessments during initial assessments in those with Asperger's because of their anxiety, discomfort in new situations, and tactile sensitivity. Having the child comfortable is important so that they will return for training. Once training becomes part of their routine, they are usually compliant and easy to work with and tactile sensitivity decreases as they receive SMR training.

Another reason that relatively few (just 17) of the Asperger's clients who completed a full course of neuro-feedback training had 19 channel assessments is that we apply the Principle of Parsimony: *first* do the least invasive, least disruptive, and least expensive intervention that is expected to help. (The authors were introduced to this principle by child psychiatrist Naomi Rae-Grant when she was head of Children's Services for the Government of Ontario in the 1970s.) If findings with a single channel assessment at Cz were significant based on the Monastra-Lubar norms for ADHD and the initial training plan after single lead assessment was apparent, we would proceed with training on the basis of single-channel assessment. By the time the symptoms of ADHD were addressed and the client had their progress testing, Asperger's symptoms, for the most part, had improved substantially and parents saw no reason for further assessment using QEEG. By using a single channel assessment only, one runs the risk of

missing something important, such as a simple partial seizure in an area that does not change the pattern seen at CZ, but time and cost are important factors to consider in clinical settings.

All charts were included where pre and post testing results were available for one or more of the following: questionnaires, Test of Variables of Attention (T.O.V.A.), Integrated Visual and Auditory Continuous Performance Test (IVA), Wechsler Intelligence Scale, Wide Range Achievement Test, and the electroencephalogram (EEG) assessment protocol using the Autogen A620 (Stoelting Autogenics). Of the 159 clients, 57 clients (9 adults and 48 children/adolescents) had pre and post test results on at least the IQ, academic, and TOVA measures plus the ADHD questionnaires. Contributing to incomplete test results were the following factors. Some measures, such as the Asperger's questionnaire and IVA, were not yet published or not yet in use at the center when the first clients were seen. Some clients were not able to complete the lengthy continuous performance tests (T.O.V.A. and/or the IVA) because they became frustrated, or they invalidated the T.O.V.A. scores with excess (>10%) anticipatory errors. On the IVA many clients with AS complained of not liking the voice, some became upset by hearing "oops" if they made a mistake during the practice section, and some even removed their headphones and thus had invalid results. Pre-training scores for the Wechsler Scales and WRAT were not always available or usable if testing had been performed by another psychologist; for example, they may have used the Kaufmann or the Stanford-Binet for the intelligence measure and a different academic measure, such as the Wechsler Individual Achievement Test. Not all adult clients were comfortable having intellectual and academic assessments completed and this was not a requirement for training for adults. Time constraints at post-test occasionally meant a test was omitted from the battery. Some children were un-testable on some measures at pre-test due to extreme anxiety, restlessness, inattention, frustration, lack of compliance or understanding (mainly with those with autism) or simply being too young.

EEG Instruments and Trainers

The instruments used for training the clients in this study were the F1000 (Focused Technology, P.O. Box 13127, Prescott, AZ 86304), the Autogen A620 (Stoelting Autogenics), Neurocybernetics (EEG Spectrum), and the procomp+/Biograph and BioGraph Infiniti (Thought Technology). Impedances were measured before training sessions using either an external impedance meter (Checktrode) or the EEG-Z preamp available for Thought Technology equipment. Impedances for 19-channel assessments were obtained either using equipment provided by Lexicor or by using the

Neuronavigator internal impedance meter. Impedances for all sites for assessments were less than 5 k Ω and, for training sessions, were usually below 5 k Ω but always below 10 k Ω . Electrode sites were prepared with Nu-prep and 10–20 EEG paste. Electrodes were always of the same metal for all sites: gold, silver-silver chloride, or tin.

The assessment program on the A620 provided the EEG ratios. The electrodermal response (EDR), a measure of skin conductance, finger temperature, and respiration training were performed with some clients using the F1000 prior to 1998 and with the Procomp+ and Infiniti instruments (Thought Technology) from 1998-onwards. The Thought Technology equipment has the capacity to simultaneously monitor and give feedback for EEG, and biofeedback variables of EDR, temperature, muscle tension, respiration, pulse, and heart rate variability. Which instrument was used depended on client needs, client preference, and availability of instruments. Most clients had experience with more than one instrument, though Thought Technology equipment has been used increasingly.

NFB training consisted of 40–60 fifty-minute sessions that combined neurofeedback with coaching in learning strategies. Although occasionally the symptoms of Asperger's appeared to be adequately treated within 40 sessions, these individuals usually benefited from more sessions than those needed for clients with Attention Deficit Disorder (ADHD, Inattentive or Combined Type). A small number of clients received more than 60 sessions of training but the pre-post measurements reported here do not reflect later assessments which were collected after each block of 20 sessions using EEG, continuous performance tests (CPT), questionnaires, and academic measures. In the early years, for adolescents and adults, the NFB was combined with BFB if anxiety and stress related tension were factors. In the last 5 years BFB, particularly diaphragmatic breathing and HRV, has been used with all clients who present with AS. All sessions were conducted one to one with a trainer. The trainers had backgrounds in psychology, teaching, nursing, medicine, occupational therapy, speech and language therapy, or social work. They all underwent training at the ADD Centre (see www.addcentre.com) in how to conduct NFB sessions. Trainers were chosen, however, not so much for their academic backgrounds as for their ability to relate to and coach students. At the center each student/client typically works with, and benefits from, exposure to a variety of trainers over the course of their training. Good rapport between a student and the trainer in each session is important, even though the training effects should be dependent on the neurofeedback effects and the strategies taught and not mainly on the relationship with a particular trainer. Clients with AS were usually found to be less flexible about working with different trainers than is the case for clients with ADHD, which is to be expected given

their dislike of change and greater comfort level with sameness and routines.

Neurofeedback

Neurofeedback was individualized based on assessment findings. For the most part, clients with Asperger's were trained to increase sensorimotor rhythm (SMR) at FCz (between Cz and Fz) for adults or Cz for children and to decrease the amplitude and variability of their dominant slow wave activity. Sometimes this theta-SMR training was conducted, for some sessions, at C2 or C4 or occasionally at C3. Excess slow wave activity targeted for treatment was usually activity in the 3–7 or 4–8 Hz bandwidth (theta), though in some clients it was 8–10 Hz alpha that was excessively high. Spindling beta was targeted for reduction when it was observed. It was usually seen between 19 and 36 Hz. Older equipment (A620 and Neurocybernetics) used an EMG inhibit range around 22–30 Hz, which (albeit unintentionally) would double as an inhibit for spindling beta. A high frequency range, usually 52–58 Hz, was used as an indicator of muscle tension (EMG) influence on the EEG and was used as a so-called EMG inhibit on feedback displays on Thought Technology equipment. (True electromyogram ranges used in EMG training are much higher, above 100 Hz, so these ranges are really frequencies within the EEG range that reflect EMG activity.) Placement was typically referential to the right ear lobe, but the reference electrode would also be placed on the left ear for some of the sessions if there were deficits in verbal or written comprehension. The ground was placed on the other ear lobe except with the F1000 equipment that used a wrist strap. Occasionally a bipolar placement was used, FCz–CPz, as suggested by Lubar in his publications on ADHD (Lubar 1991; Lubar and Lubar 1984). This was used mainly with children who were hyperactive so that common mode rejection would eliminate some of the muscle artifact. Left side placement at C3 was sometimes used if functions that predominantly involve the left hemisphere, such as language, needed to be strengthened in an individual. Dyslexia was rare in students with Asperger's but, when present, some sessions were designed to activate Wernicke's area while completing reading exercises.

Reward System

Subjects' EEGs were sampled at a rate of 128 samples per second for the A620, F1000, and Neurocybernetics systems or at 256 samples/second for the Thought Technology (TT) equipment. EEG activity influenced by EMG was defined for TT equipment as activity greater than 4 μ V occurring between 52 and 58 Hz. The EMG inhibit frequencies

varied according to the equipment being used. Monitoring the effects of EMG assisted the trainer in making sure that the feedback received by the student was due to increasing SMR or low frequency beta activity, rather than due to increased muscle tension.

Rewards were given by auditory and visual feedback from the computer, points accumulating on the monitor screen, and by praise and a token reward system administered by the trainer. Children earned tokens for effort and good performance and they had a bank account and could exchange tokens to purchase items from the ADD Centre store. Prizes ranged from balls and collector cards (Yugioh, etc.) to crafts, model cars, stuffed animals, toys, books, board games, and gift certificates for a local bookshop and music store. At first we were surprised at how well many of the children with AS, in contrast to those with ADHD, could delay gratification and save tokens. In retrospect, this was often a reflection of their difficulty in making choices and, perhaps, anxiety about making a wrong decision so they just kept accumulating tokens. Some of the children with AS would spend tokens on gifts for other people, in line with parental descriptions of their child being “a sweet, gentle kid”.

Points were given by the machines for each 0.5 second of activity (50 of 64 samplings on the A620) or by 0.5–2 s of appropriate activity (with the Biograph and Infiniti programs) during which the slow wave activity was maintained below threshold at the same time as fast wave activity (in 13–18 Hz range, such as 13–15 or 15–18) was maintained above threshold. In addition, immediate feedback was given by the TT equipment by means of a % of time >threshold (a constant numerical value) which was positioned beside the bargraphs for each frequency being monitored on the display screen. The “threshold constant” is a threshold figure that is independent of where the trainer sets a threshold on the display screen so it allows for comparisons across time. Thresholds on the screen could be changed according to how much reward seemed appropriate for the individual’s learning; for example, the trainer could make it easier on a day when the client was tired so that he/she would not become discouraged. We set the constants (for % of time >C) equal to the original assessment findings using the mean microvolt value for the frequency band being monitored. In this manner all of these figures would be about 50% when a client began training. Children were rewarded for bringing this % figure down for theta and up for SMR during each segment of each session. The thresholds on the feedback screens for each frequency range, shown on the bargraph, were initially set by the Center Director (first author) after the intake assessments. The slow wave and fast wave (high frequency beta) inhibit thresholds were set 1–2 μ V above the average activity level of the wave band. The fast wave

reward thresholds were set 0.2–0.6 μ V below the average activity level of those bands. These display screen thresholds could be altered to emphasize decreasing slow wave (and/or high frequency beta) or increasing fast wave (SMR and/or low frequency beta) activity according to the needs of a particular student or for purposes of ‘shaping’ the student’s responses. Thresholds could also be altered during an individual session in order to increase the motivation of a young client or to make it more challenging for clients as they became more proficient. Feedback was both auditory and visual on all of the EEG machines. The student would receive primarily auditory feedback when working on strategies. The F1000 used bargraphs for reward and inhibit frequency bands and an oval that would glow green and show points. Feedback displays on the A620 and Neurocybernetics were more like games, such as moving a fish through a maze or assembling puzzles. Feedback on the Infiniti (TT) could be games or bargraphs, linegraphs and various animations, like a triplane flying over an island. As clients improved they could be challenged to produce better scores without feedback for 3 min but with a review of inhibit and enhance frequencies plus EMG inhibit at the end of that time segment. This demonstrated to the client that they were capable of turning on the desired mental state without the external reinforcement and this encouraged transfer to home and school settings. The results of each few minutes (section) of training were reviewed with the client on a statistics screen (such as excel) that was kept running in the background. These learning curves could also be printed out or graphed after each training session.

Combining Neurofeedback and Biofeedback

Clients with Asperger’s Syndrome experience problems with attention and that is partly linked to alertness, which can be measured by electrodermal activity (EDR), where higher arousal reflects higher EDR (also referred to as skin conduction or SC). It may become labile or heightened with anxiety. However, the EDR response to a stressor may be flat (rather than showing an increase) when a client has undergone chronic stress. After a psychophysiological stress test was performed with an older adolescent or adult client, the decision was made as to whether EDR should be a feedback modality for that particular client and, if so, whether the trainer should encourage the client to maintain a high EDR (alertness) or whether the client needed to decrease EDR by becoming more relaxed (as when anxiety is dominant). The F1000 (unfortunately no longer manufactured) and Infiniti equipment both allow simultaneous auditory and visual feedback of brain waves, EDR and peripheral temperature. In clients who demonstrated an abnormal electrodermal response, EDR feedback was

given with the sensors on the left hand (index finger and ring finger) while they were also receiving neurofeedback. The goal was to make clients aware of their alertness level and empower them to control it. They were encouraged to use techniques such as sitting up straight to increase alertness or effortless diaphragmatic breathing to decrease arousal level and become calmer.

Clients with AS often show heightened anxiety, so self-regulation to manage stress and anxiety was part of their program. Clients were taught to breathe diaphragmatically in a comfortable manner and not to over-breathe (hyperventilation). Adolescents and adults were encouraged to breathe diaphragmatically at about 6 breaths per minute (BrPM). Children could breathe at a faster rate. As deemed appropriate after a stress assessment (Thompson and Thompson 2003c, 2007a, b, c), adult clients might receive feedback to increase heart rate variability (HRV), decrease tension usually of the frontalis and/or trapezius muscles, and/or increase their peripheral skin temperature. These variables were monitored using Focused Technology or Thought Technology equipment that combined NFB with BFB.

When adult clients observed how their physiology changed with stress and then how they could control these changes with breathing and muscle relaxation, they typically became enthusiastic about incorporating this BFB training into their program and, subsequently, into their daily lives. Usually only one or two biofeedback modalities had to be displayed on the screen with the EEG because often, when the breathing was diaphragmatic and regular, heart rate followed it and the hands became warm and muscles relaxed. Clients were taught to “generalize” relaxing into their daily life by breathing diaphragmatically at about 6 BrPM while consciously relaxing their shoulder muscles at the beginning of every daily routine such as: waking-up, getting out of bed, brushing their teeth, eating, opening the front door, traveling, answering the phone and so on. In most cases only about 10–15 sessions of combined feedback were needed before there were reports of decreased anxiety at home or work. Data on respiration, EDR, temperature, HRV, and EMG are not reported in this review but the authors cannot recall any clients who did not report positive changes with respect to stress management. Learning to regulate these physiological measures seemed easier than learning self-regulation of brain wave activity because it required fewer sessions. Note, however, that biofeedback does not produce lasting change without practice so clients needed to remind themselves on a daily basis to relax their shoulders and breathe diaphragmatically.

The importance of pairing stress management techniques with neurofeedback and, in particular, with increasing SMR, has been discussed in a previous paper reporting on a case study of a client with dystonia and Parkinson’s disease (Thompson and Thompson 2002). The mental state learned when combining NFB and BFB pairs

relaxing with a change in EEG activity, an application of classical conditioning that brings about an unconscious change in the EEG when diaphragmatic breathing is initiated. Relaxing using breathing techniques and muscle relaxation with hand warming can then trigger variables associated with both thalamic and anterior cingulate activity, such as an increase in SMR and a decrease in beta spindling respectively.

Metacognitive Strategies

Metacognition refers to thinking skills that go beyond basic perception, learning and memory. It is the executive function that consciously monitors our learning and planning. Metacognitive strategies increase awareness of thinking processes (Cheng 1993; Palincsar and Brown 1987). They help students think about thinking and reflect on what they know about how they know and remember things. The kinds of strategies taught varied according to the needs of the individual client. Strategies included the following: active reading strategies; listening skills; organizational skills; reading comprehension exercises; approaches to exam questions; tricks for times tables; solving word problems in math; organizing study time; creating mnemonic devices; preparing study notes and, of particular importance to those with ASD, recognizing and labeling of emotions. The techniques emphasized (1) remaining alert while listening or studying and (2) organizing and synthesizing material to aid recall. In essence, students learned to be active learners. This is essential for those with symptoms of ADD as they are not naturally reflective about the learning process and tend to become bored easily. It is also important for these students who had symptoms of Asperger’s because, in general, they had difficulties with “right-brain” functions. They worked on the social and emotional aspects of learning such as understanding the emotional content of reading passages and tone of voice. In some cases spatial reasoning skills were also emphasized and visual-motor tasks were practiced like printing, handwriting and tangram puzzles. Discussion and examples of metacognitive strategies are found in *The A.D.D. Book* (Sears and Thompson 1998) and (Thompson and Thompson 2003b).

Training Paired with Metacognitive Strategies

Strategies were taught while students were simultaneously receiving feedback. Trainers were instructed to emphasize the neurofeedback with the student watching the screen for two to four 2–5-min periods initially each session. The next section of the training session would last from 3 min for very young students to as much as 10 min for older students. During this section academic challenges were

introduced. These tasks were appropriate to the needs of the student as determined by the intake evaluations. As noted above, different from our students who present only with ADHD, with Asperger's clients the tasks were more often tasks that emphasized right hemisphere functioning. These included visual-spatial activities and tasks that involved emotional comprehension in listening, viewing pictures and reading passages. During tasks the feedback was auditory. The ADD Centre is a learning centre with books and strategies laid out for the trainers to use to meet the individual needs of students from age 5 through adulthood. The academic task was paused by the trainer if clients lost their focus, concentration or calmness, as indicated by neurofeedback measures. They needed to regain their calm, relaxed, focused and concentrating mental state before they continued the task. Task and mental state were, in this manner, coupled together (a classical conditioning procedure). This process of alternating pure feedback with feedback combined with cognitive activities was continued for the remainder of the session. The idea behind this approach is as follows: once the student is relaxed, alert and focused, one has a useful moment for discussing learning strategies. In addition, pairing the desired mental state with the kind of activities that occur outside the centre, at school or work, means that the activity itself becomes an unconscious stimulus for putting the student into the desired mental state (operant conditioning combined with classical conditioning as described in *The Neurofeedback Book*, Thompson and Thompson 2003b). It is a tool for generalizing the training effects.

Results

Statistical analysis was coordinated by the third author. Statistical significance was assessed using *t*-tests and a Bonferroni correction was used to allow for repeated *t*-tests. With 17 *t*-tests being conducted, a $P < .003$ was required for significance.

Results on Test of Variables of Attention (T.O.V.A.) (Fig. 1; Table 1)

All four sub-tests in the T.O.V.A. showed significant improvement. Twenty-one clients were untestable or had invalid test results (invalid = >10% anticipatory errors) at the time of the initial interview. This was usually due to an inability to remain in the chair and press the button for the duration of the test. These clients were usually testable after 40 sessions but there was no baseline for comparison. A further twelve clients received training in the early

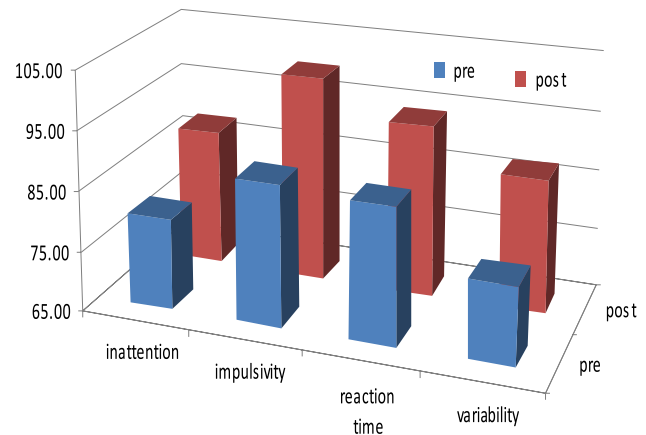


Fig. 1 T.O.V.A. the test of variables of attention is a continuous performance test. Graphic representation of changes in mean standard scores on the T.O.V.A.

1990s before we settled on a test battery that included the T.O.V.A.

There was a dip in alertness level in the afternoon for most people and this was reflected in the EEG. In the ADD Centre setting, first assessments are completed in the morning when clients are fresh and the best results possible may be expected. The progress testing is completed in the afternoon. There is more slow wave activity in adults in the afternoon (Cacot et al. 1995) than at other times of the day. The gains in T.O.V.A. scores and in EEG measures are the more impressive considering that positive results would theoretically be harder to achieve in the afternoon. In contrast to stimulant medications, which produce improvements on the T.O.V.A. only while the medication is at a therapeutic level in the blood stream (Brown et al. 1986), neurofeedback appears to produce more lasting changes (Gani et al. 2008; Monastra et al. 2002).

IVA (Fig. 2; Table 2)

On the Integrated Visual Auditory continuous performance test (IVA) the changes in the Attention Quotient, both Auditory and Visual, were significant but Response Control Quotients were not. Because the initial scores for response control were within one standard deviation of the mean (for Auditory and for Visual) this does not seem to be the major area of concern for those with AS. As on the T.O.V.A. these are standard scores with a mean of 100 and a standard deviation of 15. Speed is factored into the Attention Quotient. People with Asperger's tended to be slow and careful. Having a slow response time but with few commission errors meant scores for Response Control were higher and for attention were weaker.

Table 1 Mean T.O.V.A. scores

	Pre	Post	Gains	<i>n</i>	<i>P</i>
Inattention	80.15	88.07	7.92	128	<.003
Impulsivity	88.42	99.71	11.29	128	<.003
Reaction time	87.60	93.73	6.13	128	<.003
Variability	77.95	87.20	9.24	128	<.003

(One tailed *t*-tests) A Bonferroni correction for repeated *t*-tests meant that, for statistical significance, the probability level had to be set at $P < .003$

After a multiple *t*-test correction using Bonferroni the adjusted *P*-value is $\alpha/n = .05/17 = .003$

Statistically Significant stats after correction: ($P < .003$)

Conners

All WISC data

All WRAT data

All TOVA data

IVA auditory attention

IVA visual attention

EEG uv ratio 4–8/16–20 Hz

EEG uv ratio 3–7/12–15 Hz

Not significant: IVA visual response control, IVA auditory response control, EEG theta/beta power ratio

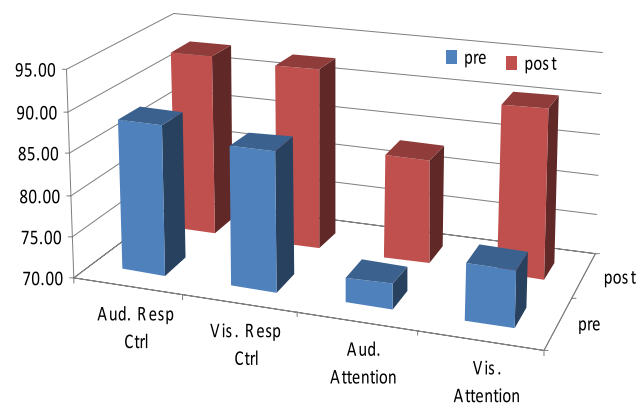


Fig. 2 The Integrated Visual Auditory (IVA) continuous performance test. Graphic representation of pre-post changes in mean standard scores on the IVA

The Wide Range Achievement Test (WRAT-3) (Fig. 3; Table 3)

Results were significant (after Bonferroni correction) for the children and adolescents. Only the children who completed this test on both the initial and the progress testing interviews were included in the analysis. Many students with outside testing performed before training did not have the WRAT measures available for pre-test as other academic tests had been used. A small number of children were untestable on the initial testing interview. Only one adult completed this test. Academic levels for Reading

Table 2 Mean IVA scores

	Pre	Post	Gains	<i>n</i>	<i>P</i>
Auditory response control	88.21	92.69	4.49	107	<.05
Visual response control	86.61	92.44	5.83	107	<.05
Auditory attention	72.96	82.82	9.86	107	<.003
Visual attention	76.59	90.43	13.84	107	<.003

See Table 1 footnote

(decoding), Spelling, and Arithmetic calculations using the Wide Range Achievement Test showed significant gains. As new editions of the WRAT became available, they were used, thus standard scores from the WRAT-R, WRAT 3, and WRAT 4 scores were used.

Results on Wechsler Intelligence Scales (Fig. 4; Table 4)

Only the clients who completed a Wechsler evaluation before training and at the time of progress testing were included in the analyses. A number of children had intelligence tests administered elsewhere for pre-test and sometimes not all the subtests were reported. A small number of children were untestable at the initial interview. For one child only the verbal score was available at both pre and post tests. Gains on the Wechsler Intelligence Scales were significant. The WISC-R, WISC-III, and WISC-IV for children and the WAIS-R and WAIS-III for adults were the tests used according to which version was in use at the time of the two testings. Canadian norms were utilized. The Verbal Concepts Index and Perceptual Reasoning Index of the WISC-IV were used for Verbal and Performance scores respectively. These are not strictly comparable to WISC-R and WISC-III because they are comprised of slightly different subtests, but very similar domains are assessed.

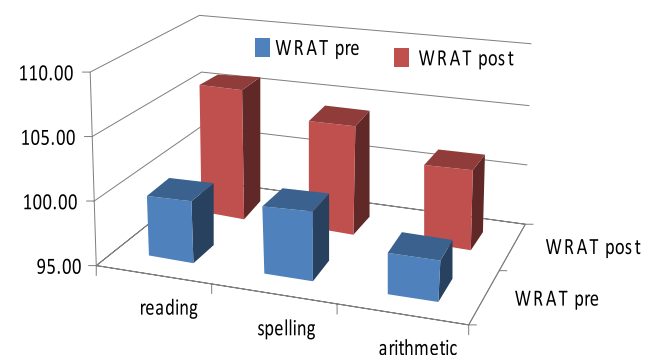


Fig. 3 Graphic representation of pre-post changes in mean standard scores on the WRAT. For the total group $P < .01$ for changes on each of the three variables

Table 3 Mean WRAT scores

	Pre	Post	Gains	n	P
Reading	99.93	105.86	5.93	83	<.003
Spelling	100.37	104.00	3.63	83	<.003
Arithmetic	98.06	101.48	3.42	83	<.003

See Table 1 footnote

EEG Changes (Fig. 5; Table 5)

Only those clients who completed pre and post testing on the same EEG instrument were included in the above analyses. All clients measured demonstrated a decrease in at least one ratio, though not necessarily in all three. In the table, 4–8/16–20 Hz and 3–7/12–15 Hz are microvolt ratios. Subjects in this review were tested before and after training using the EEG assessment program designed by Lubar for the Autogenics A620 instrument (see Table 1). Note that other investigators, such as Monastra et al. (1999), have used Lubar’s power ratios of $(4-8)^2/(13-21)^2$. These power ratios in picowatts will have larger numbers than ratios in microvolts. The power ratio is the square of the microvolt ratio. Both ratios are available using the standard A620 software or the Infiniti software.

Questionnaires (Fig. 6; Table 6)

ASAS refers to the Australian Scale for Asperger’s Syndrome (published in Attwood 1998). ACQ refers to a questionnaire developed at the ADD Centre for adults with ADHD (available at www.addcentre.com). The ADD-Q is a questionnaire developed at the ADD Centre for children and published in *The A.D.D. Book* (Sears and Thompson 1998). DSM refers to the SNAP version of the questionnaire developed by James Swanson for assessment of ADHD and is based on the symptom list of the DSM-IV. Conners’ refers to the Conners’ short form (10 item) questionnaire for ADHD (Conners’ Global Index for

Table 4 Changes in I.Q. on the Wechsler Intelligence Scale (WISC)

	Pre	Post	Gains	n	P
Full Scale IQ	101.11	110.11	9.00	65	<.003
Verbal IQ	101.48	107.74	6.26	66	<.003
Performance IQ	99.03	108.57	9.54	65	<.003

See Table 1 footnote

Parents). The Conners’ raw scores were converted to T-Scores with scores above 65 (1.5 standard deviations) considered significant for ADHD. For the other three questionnaires raw scores are presented and no statistical analyses were performed.

Medications

All decisions concerning medication were made by the individual’s prescribing physician in consultation with the client and/or the client’s parents. Data concerning medication use in the 159 clients was as follows. Ninety-eight had never used psychotropic medications and a further 7 had previously tried stimulant medications that either did not work or produced unacceptable side effects so they were not being used at the time the client began training. One client who was off medication initially was placed on 5 mg of Adderall after he changed schools. Of the 39 clients taking a single stimulant medication when they began training, 27 were weaned completely off the stimulant during the course of training while a further 10 clients reduced their dosage levels. The most popular stimulant was methylphenidate (either Ritalin or Concerta) with a few clients being prescribed amphetamines (Dexedrine or Adderall). Two clients had no change in their stimulant medication. Two clients with epilepsy continued taking their anti-seizure medication. The remaining 13 clients were on a range of medications, or on a combination of medications, including anxiolytics, anti-depressants, and anti-psychotic drugs in addition to stimulants and anti-seizure medications. Drugs being used were

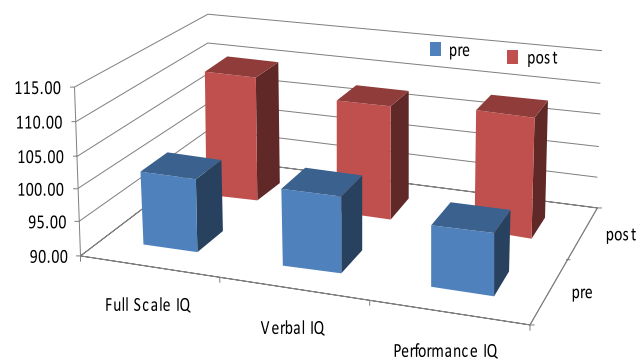


Fig. 4 Wechsler Intelligence Scale. Changes in the sum of scaled scores on Wechsler Intelligence Scale

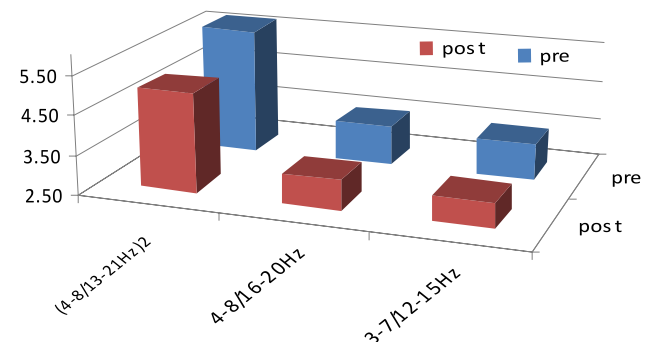
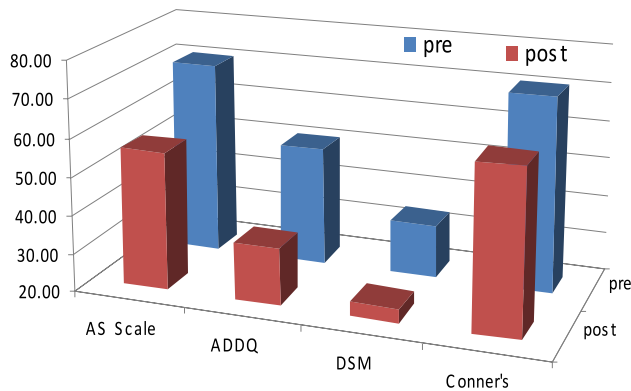


Fig. 5 Single Channel EEG CHANGES. Graphic representation of pre-post changes showing decrease in mean scores on theta/beta power ratio, theta/beta microvolt ratio, and theta/SMR ratio

Table 5 Changes in mean theta/beta power ratios and microvolt ratios

	STATS-EEG				
	Pre	Post	Decrease	Percentage decrease	<i>n</i> <i>P</i>
(4–8/13–21 Hz) ²	5.69	5.00	0.69	12.07	125 <.01
4–8/16–20 Hz	3.49	3.24	0.25	7.05	123 <.003
3–7/12–15 Hz	3.41	3.14	0.27	7.90	120 <.003

See Table 1 footnote

**Fig. 6** Questionnaire data. Graphic representation of pre-post decreases in mean scores on questionnaires for Asperger's and ADHD**Table 6** Questionnaires

Questionnaires	Questionnaires				
	Pre	Post	Decrease	Percentage	<i>n</i>
AS Scale	70.55	55.94	14.61	20.71	84) no
ADDQ/ACQ	50.95	34.66	16.28	31.96	116) stats
DSM	33.89	23.61	10.28	30.32	109) done
Conners' global	70.91	62.63	8.28	11.68	102 <i>P</i> < .003

See Table 1 footnote

No statistical analysis done on raw scores where questionnaires have not been normed. Conners' is normed and T-scores were used

Adderall, Ativan, Celexa, Clopixel, Dexedrine, Dilantin, Effexor, Lorazepam, Paxil, Risperdal, Seroquel, Tegretol, and Zoloft. Three of this group of 13 came off medications entirely and three more reduced the dosage and/or number of medications. One was on seven medications before training and another child was on five different medications. The very number of medications being tried perhaps speaks to the heterogeneity of symptoms in those with ASD and the lack of effectiveness of any particular medication(s) for most clients with ASD. Excluding the two clients who had co-morbidity with epilepsy, 52 clients (about 1/3 of the total sample) were on medications initially and 30 (58%) became medication free and a further 14 (27%) reduced their dosage levels. Thus

85% of those taking medication either came off drugs entirely or reduced their dosage.

Discussion

This is a clinical outcome study based on a review of the records from clients trained in a private educational/therapeutic setting. The results reported herein are helpful in two ways: first, they provide initial evidence that a training program, which includes neurofeedback, biofeedback, and instruction in metacognitive (learning) strategies, can be associated with positive clinical outcomes in clients with Asperger's Syndrome and, second, they demonstrate that a private center, which is not set up primarily for research, can, nevertheless, carry out systematic data collection. Sharing results will hopefully encourage others in both clinical and research settings to replicate and extend this work.

The EEG data must be viewed cautiously because many variables contribute to EEG activity. Lubar et al. (1995) referred to the work of Etevenon (1986) and of Fein et al. (1983) who reported that multi-channel EEG brain mapping demonstrates stability in the EEG over time. Thatcher (1997) has suggested that EEG changes in young children occur with maturation about every 2 years so perhaps, in some cases, we may have been adding to changes that would have occurred just with the passage of time. However, although one does expect theta reductions as a child ages, 5 months would not typically be a long enough period for changes due to chronological age. Changes observed in the single channel assessments reported in this paper after training are therefore considered most likely to be due mainly to a training effect. Activity in adults is known to vary depending on the time of day when it is measured, as noted above when discussing T.O.V.A. results (Cacot et al. 1995). In planning studies, one would ideally conduct assessments and re-assessments at the same time of day, which was not possible in our clinical setting. In addition to diurnal variations, EEG can vary with fatigue and boredom. Relative amounts of slow and fast wave activity also vary with age, with higher slow wave activity found in younger children. Activity may also vary dramatically within a single session. Nevertheless, there was considerable consistency in the results obtained on the EEG measures with a given participant completing the same tasks under the same conditions; namely artifacted data from a 3-min sample, one minute sitting quietly and instructed to watch the screen and 2 min of silent reading of material suited to their reading level. Those participants who were given a second EEG assessment at intake on different equipment, the procomp-Infiniti or the BioGraph Infiniti, demonstrated consistency of theta to beta ratios

between the two measurements on the different instruments. Conducting 19 channel QEEGs on all clients with Asperger's would be ideal; however, for clinical reasons, these cannot always be completed in the initial interviews because anxiety and tactile sensitivity are too high and rapport would be lost. Cost is also a factor in performing 19 channel assessments.

Lubar (1997) has reported, from his work with hundreds of children who have ADHD, that those who achieve EEG changes are the ones who also show positive behavioral/psychological effects of training that appear to last. Our subjective impression was that changes in school performance often began before we were able to see changes in the theta/beta ratio. The coaching in strategies might have contributed to that early improvement.

One goal of this chart review was to identify EEG and QEEG differences from data base norms that corresponded to known functions of the cortex and to symptoms observed in clients with Asperger's Syndrome. Based on functional neuroanatomy, we expected to find differences in the right temporal-parietal cortex, the cingulate (Brodmann areas 25, 23, 24, 31), anterior cingulate (BA 24, 25), medial and orbital frontal cortex, prefrontal cortex, amygdala, uncus, superior temporal lobe and the fusiform gyrus. For comparisons, differences from a normal database provide helpful clinical correlations (Thatcher et al. 2003) and QEEG and LORETA findings did include amplitude differences in delta, theta, alpha or beta activity (either less 13–18 Hz and/or more spindling beta with frequencies usually above 19 Hz) related to these areas. Less activation at T6 compared to T5 was expected based on the work of Ross (1981) concerning sensory aprosodia because those with Asperger's are poor at interpreting nonverbal communications and that was found. Details about QEEG findings are reported in another paper concerning the theoretical underpinnings for NFB work in ASDs (Thompson et al. 2009).

With respect to changes in EEG ratios, a primary symptom in AS is anxiety and we have often seen a rise in 19–22 Hz beta at CZ in these clients who have anxiety (Thompson and Thompson 2007a). This would lower the initial 4–8/13–21 power ratio in anxious clients so it should not be surprising that this is the one ratio that did not yield a significant drop after a Bonferroni correction was applied for repeated *t*-tests.

Questionnaire results must always be reviewed carefully. They are subjective and may tell more about the bias of the person completing the rating than the behavior of the person being rated. The Australian Scale for Asperger's Syndrome, published in 1998 in Attwood's book, was not added to the assessment measures until 1999 so there is a smaller "n" for that measure. The pretest Asperger's questionnaire ratings often seemed to underestimate the

child's social difficulties, probably because the parents had usually brought their child to the centre due to ADHD and were not so focused on peer interactions and the social and emotional symptoms. Once the diagnosis of AS was made parents started observing social interactions more closely and the questionnaire might have been answered quite differently, showing greater severity. It might thus be helpful to have the AS questionnaire administered twice initially, once at the first interview and a second time a few weeks later. Considering this factor, it is interesting that the percentage improvement was as high as it was. WURS results are not presented as they were not expected to change because they were a retrospective self-rating of behavior in childhood. The ADD-Q was developed because we found many years ago that the Conners' and the DSM emphasized symptoms observed in behavior problem children rather than reflecting pure symptoms of ADHD. Our population perhaps differs from that which presents to a mental health centre in that the families that come to a private learning centre are usually stable, the parents are very involved in helping their child, and there is less comorbidity with secondary behavior problems. This may be a non-specific factor influencing the results seen in the program.

The results reported in this paper provide initial support for neurofeedback (EEG biofeedback) as an intervention for achieving self regulation of brain wave activity and decreasing three principle symptoms found in Asperger's Syndrome: social ineptitude, anxiety, and attention span. There were also significant gains on measures of intelligence and academic performance. However, these data cannot be used to determine the precise mechanism(s) of the effect. It is the nature of clinical practice that a variety of interventions that are judged to be of possible utility are combined. In this study these multi-factor interventions included neurofeedback, biofeedback, and coaching in metacognitive strategies. There was also discussion of diet, sleep and exercise at the time of initial assessment and parents may have effected change in those areas, too. Other possible factors contributing to positive outcomes might include familiarity with the tests, examiner, and test setting at the time of post-test. It should be noted that this would not necessarily be positive: for example the clients with AS often handle the continuous performance tests well initially but are not enthusiastic about completing them again. T.O.V.A. and IVA changes were smaller for our clients with AS than for our ADHD population and the deficits were not as great to begin with. (Results for ADHD may be found in Thompson and Thompson 1998.) Still other factors that could contribute to a positive outcome include medication (though all testing was done off stimulant medication); increased parental support and attention; spending time twice a week with an enthusiastic adult who

provided praise and encouragement; high intelligence in some clients (always a protective factor); placebo effects associated with positive expectations (e.g., Roberts and Kewman 1993), and other nonspecific effects, as well as a host of extra-therapy influences.

Our impression is that the positive outcomes using neurofeedback and biofeedback plus metacognitive strategies affect a wider area of functioning and generalize better than other interventions for people with Asperger's. This impression is based on prior experience with other interventions in clinical settings that did not use NFB. We are not advocating for using neurofeedback alone. A multimodal approach is always advisable. Combining metacognitive strategies with neurofeedback and biofeedback increases the client's ability to produce an ideal performance state. An ideal performance state for this particular group of clients (AS) is not only characterized by being relaxed, alert, calm, aware, reflective, focused, and concentrating but also by being able to understand emotional communication, social innuendo and nuance, and demonstrate empathy and conduct their interactions with others in a manner that shows that they understand how the other person is thinking and feeling. After training, clients should be more flexible in terms of shifting their mental and psychophysiological state as task demands change and be able to plan and monitor their behavior using strategies learned in treatment.

Improvements in a client's objective test scores were paralleled by subjective self-reports and, with children, parent and teacher reports of their success and by questionnaires for many of the clients. To enhance our evaluation efforts, we are considering adding an adjective checklist test administered before and after reading a happy passage, as used in a student research study at our centre (Martinez 2003), to our pre-post test battery.

Children with Asperger's and children with learning disabilities often require more than 40 sessions to derive full benefit from NFB training. In a clinical setting the number of sessions must be determined on an individual basis based on response to treatment. In this report, all clients had at least 40 sessions but many continued onto 60 sessions (or more). Improvements start slowly and the main improvements may only emerge after 50–60 sessions. Though special education support stopped or slowed the falling behind of students with Asperger's who also had a learning disability, catch up usually only occurred after neurofeedback was added. We suggest that remedial instruction performed when a child is paying attention would have a greater effect than those same attempts when the child's mind is wandering or, as with the Asperger's children, when the child's mind is fixated on worry or on their area of special interest. Again, research incorporating appropriate control groups would be necessary to

determine whether neurofeedback is the active, efficacious, training component.

Another group of people that require more training sessions is those with a diagnosis of autism. These children may require well over 100 sessions. In part this is because it is difficult for some of these children to sit without producing EMG artifact and to attend to the feedback. Much of the therapy session is often spent in efforts to engage them in the task. None of the children with autism in our trial had been able to maintain appropriate friendships prior to NFB and as NFB training proceeded there were clear and observable changes in the children's social behavior. All of them were socializing, and some were having friends call on them and even invite them to events such as birthday parties. This does not mean that they appeared entirely normal. In fact, most did not. It does mean, however, that they are now being better accepted by their peer group. The second author has been involved in treating autistic children since the 1970s and has co-authored a chapter in a child psychiatry textbook on these children (Thompson and Havelkova 1983) and, in his experience, he has never seen results (quality and quantity) of this nature using other methodologies.

The parents of children who are autistic are often good trainers for their own child, possibly because they have always carried out a triple role of parent/teacher/therapist. We have been successfully training some of these parents to conduct NFB training at home and that is another direction for intervention and research.

The IQ tests demonstrate a general improvement on all sub-scales. This was a very diverse group of clients with some classic cases of Asperger's with very high IQs contrasting with other individuals who were very low functioning. The gains are not attributable to practice effects because, when working on Canadian norms for the WAIS III, one investigator found practice effects, when comparing WISC-R and WISC-III results, were negligible with a 6 month interval (D. Saklofski, Department of Psychology, University of Saskatchewan, personal communication, 1997). Similarly, Linden et al. (1996) found a non-significant one point increase in IQ for a waiting list control group who were retested on the Kaufman-Brief Intelligence Test after 6 months, whereas the group with ADHD who received NFB showed about a 10 point gain. Our clients with AS had a 9 point gain on Full Scale I.Q. The students generally appeared more reflective, less anxious, and better in terms of having answers that were less verbose and more to the point after training. Importantly, they could better deal with questions that involved social understanding on the Comprehension sub-test. Feeling more comfortable with the examiner and familiar with the setting could contribute to these effects, but the changes were large for these factors alone to be the cause. The

coaching in thinking skills would also contribute to gains but, in the first author's experience as a school psychologist and as the director of learning centers, significant IQ gains are not expected with tutoring alone. Tutoring is effective in the specific subject area being targeted. The results found in this work with neurofeedback are associated with gains across many areas of intellectual, academic, and social functioning. Neurofeedback appears to increase functioning in many domains, sports (Landers et al. 1991) as well as academics and intelligence (Linden et al. 1996; Lubar 1997; Thompson and Thompson 1998). Academic performance and intellectual levels after training may be more in line with potential that was always there but had not shown itself previously.

In children with Asperger's the underachievement was perhaps due to a lack of social awareness and also perhaps due to anxiety both of which affect classroom behavior and learning. Gains may result from combining neurofeedback, biofeedback (for anxiety symptoms), instruction in meta-cognitive strategies to assist social understanding of written material, and the one-to-one work with a trainer who would help the child to interact in a socially appropriate manner. It would seem useful to conduct a controlled scientific study, perhaps in a school setting where all training was without charge, to examine more closely the contribution of various factors, the characteristics of children who benefit most from this approach, and the areas of functioning that may reasonably be expected to demonstrate improvement. The population coming to a private educational center is perhaps skewed towards children who do not exhibit major behavior problems, just as the population in mental health clinics is skewed toward those who have extensive co-morbidity. This does not mean that all the students in this study were uncomplicated cases. Many presented with complex problems, and neurofeedback was a last resort after medications, therapy, private schools, and counseling had all been tried with limited success.

In the group of clients with Asperger's, anxiety and a desire to please may have contributed to the T.O.V.A. showing less dysfunction than in our previous review of outcomes in clients with ADHD (Thompson and Thompson 1998). A second continuous performance test, the IVA yielded results similar to those found with the T.O.V.A. for attention but the Response Control did not improve as much as the T.O.V.A. Impulsivity scores. This is perhaps because the Response Control Quotient is based not just on accuracy ("prudence" defined as few commission errors) but also on consistency of response time and stamina (Sanford and Turner 2002; Corbett and Constantine 2006). Those with Asperger's often showed very high stamina (comparison of response times at the beginning and end of the test) and most were careful.

Social interactions uniformly improved. The children with Asperger's went from having virtually no friends to initiating and maintaining peer friendships. The largest improvements, it seems to us, were usually in those who received the highest number of sessions.

We have observed that a small number of patients with autism (as distinct from those with Asperger's) may appear to show an increase in difficult behavior in the early stages of NFB treatment. Two possible reasons for this observation may be considered. First, in children with abnormal development, deviant amplitude and coherence z-scores might, in part, reflect compensatory mechanisms. Thus care should be taken when attempting to "normalize" QEEG findings. Second, the child with autism has arrested development. Treatment allows these children to begin to progress through the normal stages of development that should have been negotiated at an earlier age. As these children move through the equivalent of rapprochement they may enter what has been termed an "aggressive-depressed" stage. The child may begin to test limits. At this juncture the caregivers must be careful not to reverse the child's forward movement in development. The caregiver, while carefully setting appropriate limits, should reinforce the child's sense of independence while still meeting their needs for dependence. These children may be going through what is commonly called "The Terrible Twos" but at a much later age making their behavior more difficult to deal with because they are much bigger and stronger and even more determined and emotionally vulnerable (anxiety). Thus, when a child moves forward in stages of Separation-Individuation they will appear to be acting out, but really he/she is exploring autonomy and power and control in the world. One should not "put the child down" but rather join and then redirect. You join in what he/she is doing then introduce what you are now going to do together. Thus, you meet his/her dependency needs while allowing some independence and control (Thompson and Patterson 1986).

Increasing sensorimotor rhythm (SMR) using neurofeedback may have a stabilizing effect on a cortex that is unstable and easily kindled (Sterman 2000a, b). Beta spindling is one indication of a cortex that may be easily kindled, irritable, or even unstable; in other words, a cortex that is not functioning properly. Beta spindles are high amplitude, narrow band (1 Hz), synchronous beta (Johnstone et al. 2007; Thompson and Thompson 2003c). Beta spindling is an EEG finding that may be observed in a number of the disorders that have anxiety as a symptom. LORETA analysis usually shows spindling beta associated with a source in the cingulate gyrus. Perhaps the success when increasing SMR rhythm at CZ was, in part, due to resetting thalamic pacemakers and, in part, due to normalizing anterior cingulate (AC) activity.

EEG differences observed in clients with Asperger's Syndrome provide a rationale for using neurofeedback. As reviewed elsewhere (Thompson et al. 2009) there is correspondence between EEG findings and symptoms: to wit, excess slow wave activity corresponds to being "more in their own world"; excess slow wave and/or beta spindling at Fz (found to originate with LORETA in the medial frontal cortex with its connections to the amygdala and to the anterior cingulate) may correspond to difficulties modulating emotions; low SMR is consistent with fidgety, impulsive behavior, tactile sensitivity, anxiety and/or emotionally labile behavior; high left prefrontal and frontal slow wave activity is consistent with a lack of appropriate inhibition and modulation of sensory inputs; less activation, as evidenced by high slow wave activity and/or low, low frequency beta activity, in the right parietal-temporal area is consistent with difficulty interpreting social cues and emotions (sensory aprosodia); high slow wave activity and/or low, low frequency beta activity in the right frontal cortex (homologous site to Broca's area), is consistent with under-activation and inability to appropriately express emotion through tone of voice (motor aprosodia); deviations from a normal data base in frequencies whose source was identified by LORETA to be in the anterior cingulate (including beta spindling) corresponded to anxiety related symptoms; temporal lobe and, in particular, the superior temporal gyrus showing abnormal activity may indicate difficulty inhibiting the central nucleus of the amygdala (Porges 2007), which can have an adverse effect on vagal calming and allow increased sympathetic drive. Finally, abnormalities in coherence suggest that training for normalizing connectivity between the parietal lobes and the temporal and frontal regions may prove to be beneficial. (This has not been carried out on a large enough group of clients to report on at this time.)

Changes in physiological variables with minor stressors and the client's inability to rapidly recover after stress provide a rationale for using biofeedback. Learning comfortable, slow diaphragmatic breathing gives those with AS a portable stress management technique. Using NFB plus BFB and coaching in strategies exemplifies the dictum *skills not pills*.

Neuroanatomically, the common area that is posited to be influenced by neurofeedback in all clients was the cingulate gyrus, usually the anterior cingulate (AC), an area that is central to affect regulation and control. It has executive functions and it is critical in the areas of attention and concentration. But the AC is also well connected to the insula and the amygdala and to the mirror neuron system (Carr et al. 2003). Cz and FCz are the surface sites that best reflect activity in the "affective" area of the AC (Neuroguide, Thatcher 2007). Interestingly, we had been having success when we used a Cz or FCz site to train down frequencies that

were high amplitude compared to the rest of the client's EEG (theta 3–7 Hz or low alpha (8–10 Hz), and/or high frequency beta (in the range 20–35 Hz) and train up sensorimotor rhythm (12–15 or 13–15 Hz) based on the findings of single channel EEG assessments. In theoretical terms, given the clear relationship of the mirror neuron system (MNS) to ASD it seems reasonable to hypothesize that influencing what we have termed the "hub" of the affective nervous system, the AC, may have been responsible for improvement in 'reading' and copying nonverbal information (so-called social cues). Perhaps the NFB has had its positive effects by changing the responsiveness of the MNS. We postulate that this may be why, in most cases, we have not had to directly activate the T6 area using NFB. Training at the Cz and FCz sites is hypothesized to influence the AC and its affective, executive, and attentional functional networks. The connections from the AC to functionally corresponding areas of the basal ganglia and thalamic neuron groups would then be involved in feedback loops affecting functionally related cortical areas. This may help explain why good results were achieved with most clients with training at a single site. We must also take into account that many of the clients had biofeedback training to encourage effortless diaphragmatic breathing and, more recently, heart rate variability training. The vagal feedback through the medulla to the limbic system (including the anterior cingulate gyrus) could theoretically be an additional important factor in the positive outcomes. The combination of NFB affecting cortex-basal ganglia-thalamus cortical networks, with peripheral BFB augmenting the NFB effects on these functional networks, fits our systems theory of neural synergy (Thompson et al. 2009).

In addition to the low activity observed at T6, another factor that may, in the future, prove to be a helpful "marker" for ASD could be the "mu" rhythm response. In ASD there is evidence of a reduction in mu rhythm suppression during action observation (Oberman et al. 2005). However we did not investigate this relatively new finding in our analysis. In our experience mu is not observed in the majority of clients. Therefore using this as a major training parameter for NFB, as suggested in an article in *Scientific American* (Ramachandran and Oberman 2006), would not be our initial approach.

Conclusion

In this series of 159 cases, 40–60 sessions of neurofeedback, combined with training in metacognitive strategies, and with biofeedback added for the adolescent and adult clients, was associated with a decrease in symptoms of Asperger's and improvements in social, intellectual, and academic performance. Significant changes were measured on standardized tests (T.O.V.A., Attention Quotients on the

IVA, WRAT Reading, Spelling and Arithmetic, Wechsler Intelligence Scales) and improvements were also tracked by means of Asperger's and ADHD questionnaires and EEG ratios. The neurofeedback was targeted to improve symptoms of Asperger's that included poor attention, social difficulties, anxiety, and executive functions.

These data are important because they provide clinical outcome information on a large series of clients across a variety of measures. The significant improvements are a hopeful finding because Asperger's is a condition for which there is no other established, efficacious treatment. Additionally, the beneficial effects were achieved without any negative side effects. It may be particularly attractive when clients, or parents of clients, want to work on long-term change based on self-regulation skills. By giving clients feedback about their brain-wave patterns (NFB) and physiological variables (BFB), they learn how to maintain the state of being calm, relaxed, alert and concentrating. Anxiety is reduced and they notice and respond more appropriately to social cues and seem less ego-centric. Coaching in metacognitive strategies while in the calm, focused state, in order to increase conscious awareness of thinking and behavior, is hypothesized to further contribute to efficient learning and to social awareness.

The conclusions that can be drawn from these data are limited because, due to the lack of a control group and the use of multiple interventions, it cannot be determined what the efficacious components of the training were. The review does, however, provide pilot data that appears to justify further controlled studies. Such studies could address the question of which specific factors produced the significant positive results. In the meantime, an approach using neurofeedback that is individualized according to EEG assessment is proposed to be worth considering as part of a multimodal treatment plan for people with both Asperger's Syndrome and with autism.

References

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Authors.
- Asperger, H. (1991). Autistic psychopathy in childhood. In U. Frith (Editor and Translator), *Autism and Asperger's Syndrome* (pp. 37–92). Cambridge, UK: Cambridge University Press. Originally published as Asperger, H. (1944). Die "Autistischen Psychopathen" im Kindesalter. *Archiv fuer Psychiatrie und Nervenkrankheiten*, *117*, 76–136.
- Attwood, T. (1997). *Asperger's Syndrome: A guide for parents and professionals*. London: Jessica Kingsley Publications.
- Attwood, T. (1998). *Asperger's Syndrome: A guide for parents and professionals*. London: Jessica Kingsley Publications.
- Bachevalier, J., & Loveland, K. A. (2006). The orbitofrontal-amygdala circuit and self-regulation of social-emotional behavior in autism. *Neuroscience and Behavioral Reviews*, *30*, 97–117.
- Bashe, P. R., & Kirby, B. L. (2005). *The oasis guide to Asperger Syndrome*. New York: Crown Publishers.
- Blandford, S. (2005). Children can learn with their shoes off: Supporting students with Asperger's Syndrome in mainstream schools and colleges. *Educational Psychology in Practice*, *21*(1), 76–78.
- Brown, R. T., Borden, K. A., Wynne, M. E., & Shleser, S. R. (1986). Methylphenidate and cognitive therapy with ADD children: A methodological reconsideration. *Journal of Abnormal Child Psychology*, *14*, 481–497.
- Cacot, P., Tesolin, B., & Sebban, C. (1995). Diurnal variations of EEG power in healthy adults. *Electroencephalography & Clinical Neurophysiology*, *94*, 305–312.
- Carr, L., Iacoboni, M., Dubeau, M. C., & Mazziotta, J. C. (2003). Neural mechanisms of empathy in humans: A relay from neural systems for imitation to limbic areas. *Proceedings of the National Academy of Sciences of the USA*, *100*, 5497–5502.
- Cheng, P. (1993). Metacognition and giftedness: The state of the relationship. *Gifted Child Quarterly*, *37*(3), 105–112.
- Coben, R. (2005). Assessment guided neurofeedback for Autistic Spectrum Disorder. Presentation at the Society for Neuronal Regulation 13th Annual Conference, Denver, CO.
- Coben, R. (2007). Connectivity-guided neurofeedback for Autistic Spectrum Disorder. *Biofeedback*, *35*(4), 131–135.
- Collins-Williams, A. (1997). Validation of the WURS in Adults with ADHD. Unpublished Masters Thesis. University of Toronto/Ontario Institute for Studies in Education.
- Corbett, B. A., & Constantine, L. J. (2006). Autism and attention deficit hyperactivity disorder: Assessing attention and response control with the integrated visual and auditory continuous performance test. *Child Neuropsychology*, *12*(4–5), 335–348.
- Cumine, V., Leach, J., & Stevenson, G. (1998). *Asperger Syndrome: A practical guide for teachers*. London: David Fulton Publishers Ltd.
- Dapretto, M., Davies, M. S., Pfeifer, J. H., Scott, A. A., Sigman, M., Bookheimer, S. Y., et al. (2006). Understanding emotions in others: Mirror neuron dysfunction in children with autism spectrum disorders. *Nature Neuroscience*, *9*(1), 28–30.
- Devinsky, O., Morrell, M., & Vogt, B. (1995). Contributions of anterior cingulate cortex to behavior. *Brain*, *118*, 279–306.
- Dykema, R. (2006). "Don't talk to me now, I'm scanning for danger." How your nervous system sabotages your ability to relate: An interview with Stephen Porges about his polyvagal theory. *NEXUS, March/April 2006*. CO.
- Ehlers, S., & Gillberg, C. (1993). The epidemiology of Asperger's Syndrome. A total population study. *Journal of Child Psychology and Psychiatry*, *34*, 1327–1350.
- Etevenon, P. (1986). Applications and perspectives of EEG cartography. In F. H. Duffy (Ed.), *Topographic mapping of brain electrical activity* (pp. 113–141). Boston: Butterworth.
- Fein, G., Gain, D., Johnstone, J., Yingling, C., Marcus, M., & Kiersch, M. (1983). EEG power spectra in normal and dyslexic children. *Electroencephalography and Clinical Neurophysiology*, *55*, 399–405.
- Fitzgerald, M., & Kewley, G. (2005). Attention-Deficit/Hyperactivity Disorder and Asperger's Syndrome. *Journal of the American Academy of Child & Adolescent Psychiatry*, *44*(3), 210.
- Gani, C., Birbaumer, N., & Strehl, U. (2008). Long term effects after feedback of slow cortical potentials and of theta-beta-amplitudes in children with Attention-Deficit/Hyperactivity Disorder (ADHD). *International Journal of Bioelectromagnetism*, *10*(4), 209–232.
- Gattegno, M. P., & De Fenoyl, C. (2004). Social abilities training in people with Asperger syndrome/L'entraînement aux habiletés sociales chez les personnes atteintes du syndrome d'Asperger. *Journal de Thérapie Comportementale et Cognitive*, *14*(3), 109–115.

- Gillberg, C., & Billstedt, E. (2000). Autism and Asperger Syndrome: Coexistence with other clinical disorders. *Acta Psychiatrica Scandinavica*, *102*, 321–330. Referenced in: Fitzgerald, M., & Corvin, A. (2001). Diagnosis and differential diagnosis of Asperger Syndrome. *Advances in Psychiatric Treatment*, *7*, 310–318.
- Iacoboni, M., & Dapretto, M. (2006). The mirror neuron system and the consequences of its dysfunction. *Nature Reviews and Neuroscience*, *9*, 942–951.
- IVA. Intermediate Visual and Auditory Continuous Performance Test, Available through BrainTrain, 727 Twin Ridge Lane, Richmond VA 23235.
- Jarusiewicz, E. (2002). Efficacy of neurofeedback for children in the Autistic Spectrum: A pilot study. *Journal of Neurotherapy*, *6*(4), 39–49.
- Johnstone, J., Gunkelman, J., & Lunt, J. (2007). Clinical database development: Characterization of EEG phenotypes. *Clinical EEG* (in press).
- Landers, D. M., Petruzzello, S. J., Salazar, W., Crews, D. J., Kubitz, K. A., Gannon, T. L., et al. (1991). The influence of electrocortical biofeedback on performance in pre-elite archers. *Medicine and Science in Sports and Exercise*, *23*(1), 123–128.
- Landry, R., & Bryson, S. E. (2004). Impaired disengagement of attention in young children with autism. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *45*(6), 1115–1122.
- Linden, M., Habib, T., & Radojevic, V. (1996). A controlled study of EEG biofeedback effects on cognitive and behavioral measures with attention-deficit disorder and learning disabled children. *Biofeedback and Self-Regulation*, *21*(1), 35–49.
- Loffler, D. (2005). Asperger Syndrome: What teachers need to know. *Educational Psychology in Practice*, *21*(1), 80–81.
- Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback treatment for attention deficit/hyperactivity disorders. *Biofeedback and Self-Regulation*, *16*(3), 202–225.
- Lubar, J. F. (1997). Neocortical dynamics: Implications for understanding the role of neurofeedback and related techniques for the enhancement of attention. *Applied Psychophysiology and Biofeedback*, *22*(2), 111–126.
- Lubar, J. F., & Lubar, J. (1984). Electroencephalographic biofeedback of SMR and beta for treatment of attention deficit disorder in a clinical setting. *Biofeedback and Self Regulation*, *9*(1), 1–23.
- Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC-R performance. *Biofeedback and Self Regulation*, *20*(1), 83–99.
- Martinez, Y. (2003). The comparison of the effects of literature on emotion in children diagnosed with Asperger's syndrome before and after Neurofeedback training. Honours thesis for undergraduate degree in Psychology, University of Waterloo. Available from the ADD Centre.
- Monastra, V. J., Lubar, J. F., Linden, M., VanDeusen, P., Green, G., Wing, W., et al. (1999). Assessing attention deficit hyperactivity disorder via quantitative electroencephalography: An initial validation study. *Neuropsychology*, *13*(3), 424–433.
- Monastra, V. J., Monastra, D. M., & George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of Attention-Deficit/Hyperactivity Disorder. *Applied Psychophysiology and Biofeedback*, *27*(4), 231–249.
- Nash, J. M. (2002). The secrets of autism. *Time (Canadian Edition)*, *159*(18), 36–46.
- Neuroguide Delux, 2.3.7, (2007). Robert Thatcher, *Applied Neuroscience Inc.* (www.appliedneuroscience.com).
- Oberman, L. M., Hubbard, E. M., McCleery, J. P., Altschuler, E. L., Ramachandran, V. S., & Pineda, J. A. (2005). EEG evidence for motor neuron dysfunction in Autistic Spectrum Disorders. *Brain Research & Cognitive Brain Research*, *24*, 190–198.
- Palincsar, A. S., & Brown, D. A. (1987). Enhancing instructional time through attention to metacognition. *Journal of Learning Disabilities*, *20*(2), 66–75.
- Pascual-Marqui, R. D., Esslen, M., Kochi, K., & Lehmann, D. (2002). Functional imaging with low resolution electromagnetic tomography (LORETA): A review. *Methods and Findings in Experimental and Clinical Pharmacology*, *24C*, 91–95.
- Pfeifer, H., Iacoboni, M., Mazziotta, C., & Dapretto, M. (2005). Mirror neuron system activity in children and its relation to empathy and interpersonal competence. In Abstract Viewer/Itinerary Planner. *Society of Neuroscience Abstracts*, *660*(24).
- Porges, S. W. (2003). Social engagement and attachment: A phylogenetic perspective. *Annals of the New York Academy of Sciences*, *1008*, 31–47.
- Porges, S. W. (2004). The vagus: A mediator of behavioral and physiologic features associated with autism. In M. L. Bauman & T. L. Kemper (Eds.), *The neurobiology of autism* (pp. 65–78). Baltimore: Johns Hopkins University Press.
- Porges, S. W. (2007). The polyvagal perspective. *Biological Psychiatry*, *74*, 116–143.
- Ramachandran, V. S., & Oberman, L. M. (2006). Broken mirrors. *Scientific American*, *295*(5), 62–69.
- Reid, A. (2005). *Autistic Spectrum Disorders, assessment and intervention results after neurofeedback in 146 cases*. Student Award Presentation, International Society for Neuronal Regulation annual meeting, Denver, CO.
- Roberts, A. H., & Kewman, D. G. (1993). The power of nonspecific effects in healing: Implications for psychosocial and biological treatments. *Clinical Psychology Review*, *13*, 375–391.
- Ross, E. D. (1981). The Aprosodias: Functional-anatomic organization of the affective components of language in the right hemisphere. *Archives of Neurology*, *38*, 561–569.
- Salmond, C. H., Ashburner, J., Connelly, A., Friston, K. J., Gadian, D. G., & Vargha-Khadem, F. (2005). The role of the medial temporal lobe in Autistic Spectrum Disorders. *European Journal of Neuroscience*, *22*(3), 764–772.
- Sanford, J. A., & Turner, A. (2002). *Integrated visual and auditory continuous performance test manual*. Richmond, VA: Brain Train.
- Sears, W., & Thompson, L. (1998). *The A.D.D. book: New understandings, new approaches to parenting your child*. New York: Little, Brown and Co.
- Shamay-Tsoory, S. G., Tomer, R., Berger, B. D., Goldsher, D., & Aharon-Peretz, J. (2005). Impaired “Affective Theory of Mind” is associated with right ventromedial prefrontal damage. *Cognitive & Behavioral Neurology*, *18*(1), 55–67.
- Solnick, B. (2005). Effects of electroencephalogram biofeedback with Asperger's Syndrome. *International Journal of Rehabilitation Research*, *28*(2), 159–163.
- Sterman, M. B. (2000a). Basic concepts and clinical findings in the treatment of seizure disorders with EEG operant conditioning. *Clinical Electroencephalography*, *31*(1), 45–55.
- Sterman, M. B. (2000b). EEG markers for attention deficit disorder: Pharmacological and neurofeedback applications. *Child Study Journal*, *30*(1), 1–22.
- SKIL, Sterman-Kaiser Imaging Laboratory, Version 3.0. (2007). Copyright 2001.
- Swanson, J. M., McBurnett, K., Wigal, T., Pfiffner, L. J., Williams, L., Christian, D. L., et al. (1993). The effect of stimulant medication on children with attention deficit disorder: A “Review of Reviews”. *Exceptional Children*, *60*(2), 154–162.
- Thatcher, R. (1997). Cited in Karen Wright's article, “Babies, Bonds and Brains”. *Discover Magazine*, Oct. 1997.
- Thatcher, R. W., Walker, R. A., Biver, C. J., North, D. N., & Curtin, R. (2003). Quantitative EEG normative databases: Validation

- and clinical correlation. In J. F. Lubar (Ed.), *Quantitative electroencephalographic analysis (QEEG) databases for neurotherapy: Description, validation, and application*. New York: Haworth Press.
- Thompson, M. G. G., & Havelkova, M. (1983). Childhood psychosis. In P. Steinhauer & Q. Rae-Grant (Eds.), *Psychological problems of the child in the family* (pp. 293–330). New York: Basic Books, Inc.
- Thompson, M. G. G., & Patterson, P. G. R. (1986). The Thompson-Patterson Scale of Psychosocial Development: I; Theoretical basis. *Canadian Journal of Psychiatry*, 31(5).
- Thompson, L., & Thompson, M. (1995). Exceptional results with exceptional children. *Proceedings of the Society for the Study of Neuronal Regulation*. Annual Meeting: Scottsdale, AZ.
- Thompson, L., & Thompson, M. (1998). Neurofeedback combined with training in metacognitive strategies: Effectiveness in students with ADD. *Applied Psychophysiology and Biofeedback*, 23(4), 243–263.
- Thompson, M., & Thompson, L. (2002). Biofeedback for movement disorders (Dystonia with Parkinson's Disease): Theory and preliminary results. *Journal of Neurotherapy*, 6(4), 51–70.
- Thompson, M., & Thompson, L. (2003a). Neurofeedback for Asperger's Syndrome: Theoretical rationale and clinical results. *The Newsletter of the Biofeedback Society of California*, 19(1).
- Thompson, M., & Thompson, L. (2003b). *The neurofeedback book: An introduction to basic concepts in applied psychophysiology*. Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.
- Thompson, M., & Thompson, L. (2003c). *Asperger's Syndrome*. Citation paper presented at the Association for Applied Psychophysiology and Biofeedback, 34th Annual Meeting, Jacksonville, FL.
- Thompson, L., & Thompson, M. (2004). *Autistic Spectrum Disorders: A rational approach to combined neurofeedback/biofeedback interventions*. Paper presented at the Association for Applied Psychophysiology and Biofeedback, 35th Annual Meeting, Colorado Springs, CO.
- Thompson, L., & Thompson, M. (2005). *Invited address: ADHD and Asperger's syndrome, comparison of EEG profiles and outcomes after NFB*. Istanbul, Turkey: Society for Applied Neuroscience.
- Thompson, M., & Thompson, L. (2006). Improving attention in adults and children: Differing electroencephalography profiles and implications for training. *Biofeedback Magazine*, 34(3), 99–105.
- Thompson, L., & Thompson, M. (2007a). *Autistic Spectrum Disorders: Assessment and intervention with results in 146 cases*. Paper presented at the Association for Applied Psychophysiology and Biofeedback, 38th Annual Meeting, Monterey, CA.
- Thompson, M., & Thompson, L. (2007b). Neurofeedback for stress management. In P. Lehrer, R. Woolfolk, & W. Sime (Eds.), *Principles, practice of stress management* (3rd ed., pp. 249–287). New York: Guilford Publications.
- Thompson, M., & Thompson, L. (2007c). Setting-up-for-clinical-success: Scripts. Biofeedback Foundation of Europe, BFE.org.
- Thompson, L., Thompson, M., & Reid, A. (2009). Functional neuroanatomy and the rationale for using EEG biofeedback for clients with Asperger's Syndrome. *Applied Psychophysiology and Biofeedback*. doi:10.1007/s10484-009-9095-0.
- Wender, P. (1995). *Attention-deficit hyperactivity disorder in adults*. New York: Oxford University Press.
- Wing, L. (1981). Asperger's Syndrome: A clinical account. *Psychological Medicine*, 11, 115–129.
- Wing, L. (2001). *The Autistic Spectrum: A parents' guide to understanding and helping your child*. Berkeley, CA: Ulysses Press.
- T.O.V.A., Test of Variables of Attention. Available from Universal Attention Disorders Inc., 4281 Katella Ave. #215, Los Alamitos, CA 90720.
- Yucha, C., & Gilbert, C. (2004). *Evidence based practice in biofeedback*. Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.