

Functional Neuroanatomy and the Rationale for Using EEG Biofeedback for Clients with Asperger's Syndrome

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Abstract This paper reviews the symptoms of Asperger's Syndrome (AS), a disorder along the autism continuum, and highlights research findings with an emphasis on brain differences. Existing theories concerning AS are described, including theory of mind (Hill and Frith in *Phil Trans Royal Soc Lond, Bull* 358:281–289, 2003), mirror neuron system (Ramachandran and Oberman in *Sci Am* 295(5):62–69, 2006), and Porges' (Ann N Y Acad Sci 1008:31–47, 2003, *The neurobiology of autism*, Johns Hopkins University Press, Baltimore, 2004) polyvagal theory. (A second paper, *Outcomes using EEG Biofeedback Training in Clients with Asperger's Syndrome*, summarizes clinical outcomes obtained with more than 150 clients.) Patterns seen with QEEG assessment are then presented. Single channel assessment at the vertex (CZ) reveals patterns similar to those found in Attention-Deficit/Hyperactivity Disorder. Using 19-channel data, significant differences (z -scores > 2) were found in the amplitude of both slow waves (excess theta and/or alpha) and fast waves (beta) at various locations. Differences from the norm were most often found in mirror neuron areas (frontal, temporal and temporal-parietal). There were also differences in coherence patterns, as compared to a normative database (Neuroguide). Low Resolution Electromagnetic Tomography Analysis (Pascual-Marqui et al. in *Methods Find Exp Clin Pharmacol* 24C:91–95, 2002) suggested the source of the abnormal activity was most often the anterior cingulate. Other areas involved included the amygdala, uncus, insula, hippocampal gyrus, parahippocampal gyrus, fusiform

gyrus, and the orbito-frontal and/or ventromedial areas of the prefrontal cortex. Correspondence between symptoms and the functions of the areas found to have abnormalities is evident and those observations are used to develop a rationale for using EEG biofeedback, called neurofeedback (NFB), intervention. NFB training is targeted to improve symptoms that include difficulty reading and mirroring emotions, poor attention to the outside world, poor self-regulation skills, and anxiety. Porges' polyvagal theory is used to emphasize the need to integrate NFB with biofeedback (BFB), particularly heart rate variability training. We term this emerging understanding the Systems Theory of Neural Synergy. The name underscores the fact that NFB and BFB influence dynamic circuits and emphasizes that, no matter where we enter the nervous system with an intervention, it will seek its own new balance and equilibrium.

Keywords Asperger's · Neurofeedback · QEEG · EEG biofeedback · Anterior cingulate · Mirror neurons · Polyvagal theory · Systems theory of neural synergy

Introduction

Asperger's Syndrome (AS) comprises a triad of qualitative impairments in social interaction, repetitive and restricted special interests, and differences in imagination (Wing 2001). Language proficiency constitutes a main feature of those with Asperger's, though there may be some differences in their speech, such as pedantic phrases or a voice that is monotone and lacks prosody (intonation, loudness variation, pitch, rhythm). AS is considered to be along the spectrum of autistic disorders. As children, persons with AS are often inappropriately friendly and open with

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strangers, which is an example of problems with social boundaries. As they progress into adolescence and adulthood they often withdraw socially, perhaps as a reaction to rejection by peers. They are socially naïve, socially immature and thus often the target of teasing or mistreatment by bullies. Attwood (2007, p. 60) states that, in early elementary school years, “their level of social maturity is usually at least 2 years behind that of their age peers”.

Recognition of AS came earlier in Europe than in North America, in large part because nothing was available in English until Lorna Wing, a British psychiatrist and autism expert, wrote about the constellation of symptoms that was first described by the Viennese pediatrician Hans Asperger towards the end of WWII (Asperger 1944; Wing 1981; Wing and Gould 1979). Asperger used the term “autistischen Psychopathen” (autistic psychopathy), borrowing the autism term from Bleuler (1911) and selecting the term psychopathy to indicate it was a personality type. Asperger described a group of boys who had excellent language skills (albeit with pedantic use of language and unusual prosody) and expert knowledge in areas of intense special interests, yet revealed severe limitations in their social relationships, abnormal eye contact, motor clumsiness, behavioral problems (including both aggression and being bullied) and limited facial or gestural expressiveness. His paper was finally translated into English by Uta Frith of University College, London in 1991.

Asperger’s Disorder was included in the Diagnostic and Statistical Manual of the American Psychiatric Association for the first time in 1994 (DSM-IV code number 299.80). Those with *Asperger’s Disorder* show qualitative impairments in social interaction and restricted, repetitive, and stereotyped patterns of behavior, interests, and activities to a degree that causes significant impairment in social, occupational and other important areas of functioning. There are exclusion criteria; namely, no significant delay in language or cognitive development (DSM-IV, Text Revision, American Psychiatric Association 2000). This paper deals primarily with *Asperger’s Syndrome* as delineated by Asperger himself (1944) and with the symptoms articulated by experienced clinicians including Wing (2001), Gillberg (1991) and Attwood (2007). Asperger’s Syndrome is the authors’ preference, both because it has always been used at their ADD Centre, which pre-dated DSM-IV, and also because of perceived problems with the DSM-IV criteria of no significant language delay and at least average intellectual functioning. The authors’ experience aligns with Wing’s contention that a range of intellectual functioning can be found in association with AS and with Attwood’s view that language differences are important in AS and having language delay as an exclusion criterion is not clinically useful. The first author has seen rigid adherence to the DSM-IV, for example, lead to a diagnosis of

Pervasive Developmental Disorder (PPD), Not Otherwise Specified in a child who clearly had Asperger’s Syndrome. The psychiatrist, limited by DSM-IV, made the PDD diagnosis rather than Asperger’s Disorder because there had been a speech delay. Language functioning at age seven, when the diagnosis was made, was advanced, but there was a history of delay, likely because the child grew up in a tri-lingual household.

Prevalence is conservatively estimated at 2.5 per 10,000 in school-age children (Frombonne and Tidmarsh 2003). A much higher rate of 36 per 10,000 and a male:female ratio of 4:1 was found in a population study conducted in Sweden (Ehlers and Gillberg 1993). The male predominance of 4:1 was also found in 1000 cases seen at a clinic in Brisbane, Australia (Attwood 2007). Vocations which require logical, sequential thinking without much emotional content or social understanding, such as computer specialists, appear to have higher rates of AS. A review on autism in a TIME magazine article reported that *Wired* magazine in December 2001 dubbed the “striking combination of intellectual ability and social cluelessness the ‘geek syndrome’” and noted that rates of AS were rising in Silicon Valley, California (Nash 2002). Of course, this survey did not meet the standards of most epidemiological studies, but it is suggestive. Increased awareness after inclusion of Asperger’s Disorder in the DSM-IV may be contributing to some of the increase in diagnosis. Writing in 2007, Attwood noted that there were over 2000 articles and 100 books on AS.

AS is much more frequent in boys and Asperger himself suggested an extreme male analogy as a way of characterizing this syndrome (cited in Wing 2001, p. 43). Males, as contrasted with females, tend to be more interested in how things work than in how people feel, and those diagnosed with AS are at the extreme end of that continuum. One study that supported this idea compared responses of three groups (males with AS, males without AS, and females without AS) on empathizing and systemizing tasks. On the tasks requiring empathy, females had higher scores than males without AS and the latter had higher scores than males with AS. On the task requiring figuring out a logical system, the males with and without AS were equal and both out-performed the females (Lawson et al. 2004).

AS comprises a heterogeneous group of individuals. Alvarez (2004) noted the complex way in which a child’s personality interacts with the symptomatology of the disorder. The majority of those diagnosed as having AS do not have all of the traits, though they will have a sufficient number for the diagnosis to be made. Most clients with AS have very high IQ’s, as tested by the Wechsler Intelligence Scales, possibly due to the fact that most of the subtests, particularly on the latest version, the WISC-IV, can be

completed using verbal mediation and logical left hemisphere skills. The most common pattern is that Verbal IQ exceeds Performance IQ. However, some cases show the opposite pattern: strong Performance IQ (called Perceptual Organization on the WISC-IV) and the ability to excel in spatial reasoning and mathematics rather than in language based areas. Attwood cites a review of the cases seen over a 30 year period by Asperger and his colleagues showing 48 per cent had a higher Verbal IQ, 18 per cent had a higher Performance IQ, and 38 per cent showed no significant difference (2007, p. 229). A personal account of the pattern of AS plus math genius is found in the autobiography, *Born on a Blue Day* by Tammet (2007) and it is also portrayed in the novel, *The Curious Incident of the Dog in the Night-time* (Haddon 2002). Film depictions of AS include *About a Boy*, starring Hugh Grant (Weitz and Weitz 2002), and *Mozart and the Whale*, a love story about two young people with AS.

Emotional regulation is poor. Even in their teens, those with AS may suddenly over-react emotionally, going from placid to tears, or even extreme anger. Others observing the behavior may feel the precipitating incident was quite trivial. Anxiety may be most apparent with any transition or change in routine. One client at the ADD Centre had a morning routine of his brother being dropped off first before he was driven to school. If this routine were not maintained, even if the older brother did not have school that day, the child with AS would be unable to get out of the car. Behavior usually worsens if the family moves or if the child has to change schools.

It is still quite rare for clients to come for assessment and neurofeedback training complaining that they have Asperger's Syndrome. The most common presenting symptom pictures at the ADD Centre, in order of frequency, are: 1. ADHD symptoms, 2. Anxiety and Panic, especially social anxiety, 3. Learning problems, 4. Emotional Lability, 5. Depression, 6. Obsessive-Compulsive Symptoms, and 7. Acquired Brain Injury (ABI). When any of these are found in conjunction with Asperger's Syndrome, there is a core symptom of anxiety. In line with anxiety, the most common finding when LORETA analysis is performed using QEEG data is involvement of the anterior cingulate gyrus, as will be discussed later.

Sources of Social Difficulties in AS

Children who have Asperger's syndrome are at risk of being misunderstood and neglected because the syndrome is not always obvious. Within the general population, these people just "do not fit in" (Portway and Johnson 2005). Comorbidity with other disorders, such as

obsessive compulsive features, just makes their situation more difficult.

There are many differences in how those with AS process social-emotional information (Carothers and Taylor 2004). Nils Kaland and his colleagues have studied how strange stories are understood. They found that the Asperger's group could not correctly understand inferences in material that included pretence, joke, lie, white lie, figures of speech, misunderstanding, persuasion, irony, double bluff, contrary emotions, appearance versus reality, and forgetting (Kaland et al. 2005). Others have shown deficits in those with AS when they were asked to perform complex verbal tasks that involved cognitive switching and initiation of efficient word retrieval strategies (Kleinhans et al. 2005). Research by Emerich and colleagues found that the ability of adolescents with Asperger's syndrome to comprehend humorous material, such as picking funny endings for cartoons and jokes, was significantly impaired (Emerich et al. 2003). There may be memory difficulties with free recall, but not cued recall (Bowler et al. 2004), though those with AS have prodigious memories for things that interest them. Laurent and Rubin (2004) studied social communication problems and showed difficulties in verbalizing emotions and interpreting intentions. Barton et al. (2004) looked at facial recognition difficulties and emphasized the heterogeneity in the perceptual processing of faces rather than seeing them as a single pattern. Finally, Deruelle and colleagues studied face processing strategies and found all aspects, except for identity matching, were deficient (Deruelle et al. 2004).

At our centre, a study was conducted to compare children with AS to a group of children without any identified problems, in terms of emotional reactions to stories. Subjects completed an adjective check-list describing their mood before and after reading a happy passage. Those with Asperger's responded differently in that they did not show the shift towards positive emotion found in the control group; indeed, some reported themselves as *less* happy. After NFB training, the children with Asperger's did identify more adjectives that signified positive mood after reading the story in the same way as matched, normal controls (Martinez 2003).

Clients with AS display symptoms of both sensory and motor aprosodia. Sensory aprosodia refers to an inability to correctly interpret social innuendo, either verbal or non-verbal. Sensory aprosodia resulting from neurological damage has been reviewed by Ross (1981) who describes how people who suffer an infarct to the right temporal-parietal area often cannot understand emotional tones of sadness or happiness in another person's voice. Motor aprosodia refers to lack of prosody; that is, an inability to use emotionally appropriate vocal intonation and volume control in conversation. When damage is right frontal,

people will show motor aprosodia. Similarly, those with AS often speak in a monotone voice or they may use a loud voice, especially when feeling stressed.

An example of these difficulties is provided by Brian, a 19-year-old client diagnosed by his college as having Learning Disabilities and at the ADD Centre as having AS plus LD. In his early sessions Brian would watch people and, if someone told a joke, he would see others smiling and laughing and then he would produce a forced laugh. After 40 neurofeedback sessions Brian not only picked up on humor and laughed appropriately, but was also telling truly funny stories and jokes. The main training objective had been to decrease high frequency beta activity, so-called beta spindling at frequencies above 20 Hz (Thompson and Thompson 2006a, b) whose source, according to LORETA analysis performed on a 19-lead EEG, originated in the anterior cingulate, specifically Brodmann area (BA) 24. This inappropriate activation was observed and successfully trained down, with the active surface electrode for recording EEG placed at a central location, FCz (half-way between CZ and FZ). In younger children similar patterns are seen at CZ.

Those with AS are often very honest (no social lies and sometimes too open about personal topics) and one often feels they would have a smoother time if the world were a better place; that is, if people would say what they mean, follow the rules, keep to routines, and be kind. It is important with clients with AS to have clear communication without the use of confusing figures of speech, pretence or sarcasm. Advise parents that yelling, anger and impatience are all counter-productive when dealing with someone with AS. If their child or adolescent with AS is out of control and digging in their heels, parents should understand that this usually is due to the child trying to control the situation to reduce anxiety. Parents and teachers must be flexible and model the calmness that they want to see in the child and not escalate the confrontation.

Children with AS typically interact well with those younger than themselves and with adults, but are usually not successful in maintaining friendships with peers. This is due to their difficulty in reading social cues and responding appropriately, which often leads to them being bullied, teased, or socially ostracized. There are problems with boundaries, both physical (bumping into others), and social (not understanding social boundaries); for example, being seen as disrespectful when they treat a teacher like an equal. They also may have difficulty in taking part in normal peer group activities. In team sports, motor clumsiness and spatial awareness problems may interfere and make it hard for them to obtain a sense of the game. In other peer

activities their superiority in verbal skills and impressive vocabularies would superficially appear to be an asset. However, people with AS tend to be literal and have difficulty with figurative language and with correctly using terms to describe emotions in themselves and others. They go on eloquently about their special interest area when the listener is clearly bored. They may have reading comprehension difficulties when the stories involve emotional insight, innuendo, or inference. Their literal interpretations can be quite marked. For example, a first grade child, told by his teacher that she did not want to see him out of his seat, took his chair with him when he got up. Another first grader, given the same directive, went under the desks to the pencil sharpener, presumably so the teacher would not see him out of his seat. Both were genuinely confused when sent to the principal's office. They believed they had done what they were told to do.

Children with AS are socially naïve: they lack “street smarts”. They are the ones ‘left holding the bag’ and other children can set them up to do things that get them laughed at or in trouble. They make easy victims for teasing, bullying or extortion. They may copy behavior from books or television, not realizing it is inappropriate outside of that context. Their attire may set them apart because they wear what is comfortable, rather than what is fashionable, due to a combination of sensory sensitivity and not reading fashion cues. Yet they possess great gifts in terms of acquiring knowledge in an area of interest. One 5-year-old, whose special interest was weather, not only explained what a barometer was, he gave instructions for making one. Instead of cartoons on television, he watched the weather channel.

In the teenage years, social difficulties may lead to withdrawal and even depression. In adulthood, fortunate people with AS develop their special interests into careers and may even become professors in a field where they possess vast, arcane knowledge. There has been a retrospective diagnosis of AS in Jonathan Swift, author of *Gulliver's Travels*, and the eccentric Dean of St. Patrick's Cathedral in Dublin (Fitzgerald 2000).

The pattern of social difficulties persists through adulthood. The most common temperament configuration found on the Temperament and Character Inventory for adults with AS (Soderstrom et al. 2005) was the triad of obsessional, passive-dependent, and explosive features. Subjects also scored high on measures of anxiety and the test indicated they had difficulties with social interaction and self-directedness.

Neurophysiologically, what many of the above behaviors have in common is a core symptom of anxiety. With LORETA analysis, involvement of the cingulate gyrus is the common finding.

Overlap With Other Disorders

Clinical experience indicates overlap in symptoms with a number of other diagnoses, especially attention-deficit/hyperactivity disorder (ADHD), which is a frequent presenting diagnosis in children (Klin et al. 2000). There may also be co-morbidity with specific Learning Disabilities. Often the LD problems will involve having difficulty with organization, boundaries (physical ones and social ones), and with many aspects of mathematics (geometry, concepts relating to time and space). These problems are related to right hemisphere dysfunction. There may also be white matter damage in the brain (Rourke and Tsatsanis 2000) and there is a smaller corpus callosum.

Autism

Autism is a disorder of neurodevelopment resulting in pervasive abnormalities in social interaction and communication, repetitive behaviors and restricted interests. There is evidence for functional abnormalities and metabolic dysconnectivity in “social brain” circuitry in this condition (McAlonan et al. 2005). The DSM-IV criteria for autism and Asperger’s Disorder are very similar with the main difference being that there are no significant delays in language development or cognitive development in AS, as discussed above. The differences are easily seen between autism and AS in lower functioning children with autism who have severe language limitations. High functioning autism (HFA) can seem close to AS so the two terms are often used almost interchangeably. There has been quite a lot of debate over whether Asperger’s and high functioning autism can be differentially diagnosed (Bregman 2005; Ghaziuddin and Mountain-Kimchi 2004; Macintosh and Dissanayake 2004; Mayes and Calhoun 2004; Rubin and Lennon 2004; Simpson 2004). Attwood (2007) states that there are no data that unequivocally confirm them as separate diagnoses. Yet some researchers have found fundamental differences between the two; for example, those with Asperger’s tend to have less severe deficits in theory of mind than HFA (Dissanayake and Macintosh 2003). Those with HFA have more difficulties in comprehension of humorous materials than Asperger’s, although both groups perform more poorly than controls (Emerich et al. 2003). The age of diagnosis is usually several years older for AS than for autism (Gillberg 1989), reflecting the fact that the symptoms are less severe, especially at home where the individual has their comfortable routines. Qualitatively, in comparison to autism, there is an increased likelihood of seeking social interaction in those with AS (Khouzam et al. 2004).

In the authors’ experience, clients with AS are quite different from those with autism in terms of their emotional

responsiveness and interest in others. The term Pervasive Developmental Disorder (PDD) is not appropriate for AS because it should be reserved for those few children who truly have a “pervasive” disorder in virtually all areas of functioning. Such children are described well in older literature on childhood psychoses and autism (Thompson and Havelkova 1983).

Attention-Deficit/Hyperactivity Disorder

The overlap with Attention Deficit/Hyperactivity Disorder is discussed in a number of recent publications (Bara et al. 2001; Corbett and Constantine 2006; Fitzgerald and Kewley 2005). Most clients presenting at the authors’ center come with a previous diagnosis of Attention-Deficit/Hyperactivity Disorder. Like those with ADHD, clients with AS are inattentive (more in their own world) and often do not seem to listen well, but their inattention is in part due to ego-centricity and not understanding social demands and also, in part, due to anxiety and ruminating. Impulsivity could relate to behavior that appears inappropriate to others, though the child may have his reasons; for example, an unprovoked attack in the schoolyard in September because the child with AS felt the other boy deserved it because of something he had done the previous June. (That example also illustrates the trait of exceptional memory. Excellent memory coupled with being literal and honest means these children will often correct their parents about details of past events during history taking, something a child with ADHD would not do.) Impulsive actions are often related to their own special interest area or to their anxiety. In younger children, bossy behavior, acting like little policemen, and tattle-tale actions, are all attempts to cope with social anxiety by being in charge and such behavior also illustrates a lack of understanding of appropriate social interaction. Those with ADHD can also be bossy and immature, but there is a different quality to it—and they do not become upset when others do not follow rules.

A British study found that children, on average, were first diagnosed as having AS at age 11 and that they had had three previous assessments, usually with a diagnosis of ADHD, before they were diagnosed correctly (Fitzgerald and Kewley 2005). They noted that, once medical treatment of ADHD is undertaken, the Asperger-type symptoms may also fade. They suggest that a diagnosis of Asperger’s syndrome when features of ADHD are also present be delayed until the ADHD has been effectively medically treated. The authors have had the same experience treating the ADHD symptoms with EEG biofeedback, namely, that the AS features are less bothersome. This is perhaps partly because there is less negative feedback from their environment when they are less fidgety and inattentive so there

is less to make them anxious. As will be discussed later, successful outcomes may be based on the involvement of the anterior cingulate in both disorders.

Nonverbal Learning Disorder

Some professionals will use the term non-verbal learning disorder (NLD) almost interchangeably in persons with Asperger's Syndrome (Attwood 1997) whereas others make a distinction (Klin and Miller 2004). Although it is not unusual to see people with both types of difficulty, they are not synonymous and a client can have either without the other. Those with NLD typically have a much higher verbal than performance IQ with related problems in mathematics and written language. Some aspects of mathematics can be difficult due to weak spatial reasoning skills and problems seeing the relationships in number patterns. Clients with NLD may have difficulties with social interactions related to problems with boundaries, but they have normal speech intonation and do not have such severe social deficits as do people with AS. Nor do they have narrow, special interest areas. In both NLD and AS there is an interaction between the child's personality and the disorder so there is great heterogeneity in the population for each diagnosis.

NLD is usually diagnosed by psychologists with an interest in learning disabilities. It is based on a learning profile and is not a psychiatric diagnosis in the way Asperger's Disorder is. Sometimes AS is the more appropriate diagnosis but it is missed because the child interacts appropriately with the psychologist during testing and they do not ask about broader social functioning with peers. Resources for those interested in NLD include Stewart's book (1998) *Helping a Child with Non-verbal Learning Disorder or Asperger's Syndrome* and Pamela Tanguay's *Nonverbal Learning Disabilities at Home: A Parent's Guide* (2001).

Pragmatic Language Disorder and Dyspraxia

The diagnosis of Pragmatic Language Disorder (Attwood 2007) may be made by a Speech and Language Pathologist if they are the first person asked to assess the child with AS. Those with AS do have the symptoms of Pragmatic Language Disorder because their communication difficulties lie partly in the practical applications of language, such as in conversations with peers. Those with AS talk about their interests too much and they fail to read the nonverbal cues of the person(s) with whom they are talking. They may not maintain appropriate eye contact. Their tone of voice (loud or monotone) does not fit the subject matter. They do not keep their audience's viewpoint in mind when explaining things. Certainly a speech and language

pathologist can do useful work with a child who has either Pragmatic Language Disorder or AS if they focus on training the practical, social applications of language, like taking turns during a conversation.

An Occupational Therapist may be called upon for consultation because the child is clumsy and has poor printing skills. They may diagnose dyspraxia because of poorly developed fine motor skills. Researchers who spent 3 years investigating autism in Lancashire, England, meeting over 100 children with AS, note that those with dyspraxia differ in terms of having a relatively intact ability to form social relationships and being less rigid and obsessional in their interests (Cumine et al. 1998). Those with AS certainly have motor skills problems (just ask how old the child was before he could tie his shoelaces) but those children who just have dyspraxia do not have the same problems in social communication.

As an aside with respect to fine-motor skills, a person with AS can occasionally be artistic, but nearly all of those assessed by the first author for this study showed reluctance when asked to draw a person (d-a-p). Often they would produce a drawing with facial features blank or hidden, or they would draw a detailed object such as a train, bulldozer, or airplane with the person represented by a tiny circle in the window of the vehicle. One young boy, who will likely become an ornithologist, drew beautifully detailed birds but declined to draw a person. We postulate that the problem in drawing people could be related to problems in reading people. Changes in the d-a-p task are an interesting way to gauge clinical improvement. Though they are not quantifiable with respect to emotional functioning [drawings can be scored for IQ equivalence using the Goodenough–Harris method (1963)] they provide interesting hypotheses for clinical symptom correlation; for example, the person without hands indicating the child is not reaching out to others in his environment or the one without feet suggesting the child does not feel grounded.

Approaches to Intervention

Psychotherapy, behavior therapy, social training, group therapy and medications have been the most commonly used interventions for children who present with the symptoms of Asperger's syndrome. These interventions, plus speech therapy, are also commonly tried interventions for Autistic Spectrum Disorders (ASD) (Green et al. 2006). There is much less literature on intervention outcomes than there is on diagnosis. Blandford (2005), Cumine et al. (1998) and Loffler (2005) all offer information about Asperger's Syndrome and provide management advice to teachers. Gattegno and De Fenoyl (2004) propose group psychotherapy that involves learning social abilities. These

writings are helpful and are based on clinical experience, but they lack outcome data.

Diet should be discussed during the intake evaluation in clients with AS because there is preliminary evidence that ASD in some individuals may involve the digestive system and the immune system. The group called Defeat Autism Now! (DAN), co-founded by the late psychiatrist, Bernard Rimland, who had a son who was autistic, encourages practitioners to look at diet, often suggesting elimination diets that avoid wheat (because of gluten) and dairy products (because of casein). They focus on detoxification and decreasing what they believe to be neuro-inflammation. The basic theory is that some individuals with autistic spectrum disorders have a digestive problem such that their bodies cannot handle the proteins found in gluten and casein. The Online Asperger Syndrome Information and Support (OASIS) website has conducted a Survey on Alternative Treatments and concluded that, although some individuals reported benefits, special diet regimens probably have higher success rates for persons with autism than for AS (Bashe and Kirby 2005).

Although there is no specific pharmacological treatment for AS or, for that matter, any of the autistic spectrum disorders, psychotropic medications are frequently used to treat symptoms (Sloman 2005). Many children with AS show hyperactive behavior and are placed on medications that range from stimulants and antidepressants to antipsychotics. The stimulants target hyperactive behavior and the commonly used ones are methylphenidate, either Ritalin or the controlled release Concerta, and amphetamines, such as Dexedrine and Adderall. Common side effects are appetite suppression and insomnia. Stimulants reduce the seizure threshold and rates of seizure disorders are higher among people along the autistic spectrum, so that is one reason they should be used with caution. Some children with Asperger's have angry outbursts and over-react to frustrations that seem trivial to others. For symptoms of anger, temper tantrums, and aggression psychiatrists may prescribe Risperdal (risperidone), an anti-psychotic medication with calming properties. This medication, most commonly used in nursing homes to help staff deal with difficult elderly patients, is given to decrease agitation and aggressive outbursts and increase social interaction. Although it has fewer extrapyramidal (Parkinsonian) side effects when compared to other commonly used neuroleptics, such as haloperidol and thioridazine, it can cause significant weight gain (Committee on Children with Disabilities 2001). If targeting symptoms of anxiety, panic, obsessive-compulsive behavior and depression, the psychiatrist usually starts with an anti-depressant from the class of selective serotonin re-uptake inhibitors (SSRIs). Sloman (2005) notes that most of the psychotropic medications used in children have not gone through the

evaluation necessary to establish their efficacy, tolerability, and safety. There is also the limitation that, "Medication does not ameliorate the basic deficits in social interaction and communication." Even in ADHD, stimulants are only effective for the short-term management of behavior (Swanson et al. 1993).

Drugs are prescribed for those with AS when their symptoms bother other people and these difficulties arise most often in school settings where the child feels over-stimulated or confused. The list of psychotropic medications used includes antidepressants (SSRIs like Prozac, Celexa, Zoloft and Paxil; atypical antidepressants like Effexor and Wellbutrin; tricyclic antidepressants like Elavil); stimulants like Adderall, Concerta and Ritalin; the selective norepinephrine reuptake inhibitor, Strattera; antipsychotics, like Mellaril, and atypical antipsychotics, like Risperdal and Seroquel; mood stabilizers or anticonvulsants like Neurontin and Tegretol; anxiolytics like Ativan and Valium; and antihypertensives like Catapres (clonidine). Bashe and Kirby (2005) who run the OASIS website have compiled helpful information into a book that includes a comprehensive chapter on these medications with discussion of the symptoms they target and cautions concerning their use. The combination of clonidine and a stimulant, for example, has been associated with sudden death if one of the medications is stopped abruptly. The heterogeneity of the medications underscores the individual differences in people with AS and the range of comorbidity.

In the authors' experience, the results of medications used with children who have Asperger's syndrome are usually a lack of significant improvement and an array of unfortunate side effects. Our experience may be biased because some clients try neurofeedback when medications have failed or have produced side effects. "*Medications when necessary but not necessarily medications*" is a conservative guideline for managing ADHD (Sears and Thompson 1998) and this advice also applies to management of AS. With stimulant medications, some children do settle and produce more work with neater handwriting, just as is found in those with ADHD, but there may be an increase in anxiety and more of a tendency to become stuck on things. Indeed, we have observed that, when beta spindling is present, stimulants may make the patient's symptoms considerably worse. This is hypothesized to occur because stimulant drugs increase narrow focus, and that focus may be on an inner worry. Thus using stimulants for dealing with symptoms of ADHD that present in someone with AS may actually worsen behavior. Suffin and Emory (1995) have reported on EEG patterns predicting drug response in those with attentional and affective problems and these observations may perhaps be extended to those with AS; namely, frontal excess theta

responds best to stimulants, frontal excess alpha responds better to anti-depressants, and coherence problems (excess frontal theta coherence) respond best to anticonvulsants (seizure medications). Physicians interested in medication approaches may be able to improve their hit rate by prescribing based on QEEG analysis (Prichep et al. 1993; CNS Response 2008; McCann 2006). Using neurometrics to predict drug response was pioneered by psychologist E. Roy John of New York University's Psychiatry Department in Manhattan, who has published extensively on this subject, as well as Prichep. There now exists a publicly traded company, CNS Response, which markets this service to psychiatrists. Of course, QEEG and brain maps can also be used to guide neurofeedback interventions.

Over the past dozen years, a few papers and presentations about intervention using neurofeedback for clients with AS have appeared (Coben 2005, 2007; Jarusiewicz 2002; Reid 2005; Solnick 2005; Thompson and Thompson 1995, 2003a, 2007a; 2009). These papers all note favorable clinical outcomes using neurofeedback based on case series, some with large numbers of cases, such as the 150 cases reported on at the Biofeedback Foundation of Europe annual meeting in 2007 (Thompson and Thompson). More well controlled studies appear warranted. Later in this paper a rationale is developed for why neurofeedback could be of value to people with AS manage their symptoms and make changes in how they interact with the world.

Correlation of Symptoms, EEG Findings, and Functional Neuroanatomy

Right Frontal and Right Parietal-Temporal Junction

Of particular interest with respect to neurofeedback are studies that investigate how brain anatomy and neurological functioning differs in those with Asperger's. As noted previously, an early review by Ross (1981) showed that sensory and motor aprosodia may be acquired. In people with Asperger's they appear to be inborn. Motor aprosodia refers to flat vocal tones and/or inappropriate vocal tone. This can occur after an infarct in the right frontal lobe in an area approximately corresponding to Broca's area in the left hemisphere. This is very close to the area now implicated in mirror neuron functioning (Iacoboni and Dapretto 2006). Ross also noted that a stroke or infarct in the right posterosuperior-temporal-lobe and posteroinferior-parietal-lobe (an area in the right hemisphere that corresponds to Wernicke's area in the left hemisphere) may result in sensory aprosodia. Sensory aprosodia refers to the inability to correctly interpret social innuendo, either verbal or non-verbal, with difficulty copying emotional tones that express indifference, anger, sadness, or happiness. This area at the

junction of the parietal and temporal lobes includes part of the angular gyrus and the right hemisphere site homologous to Wernicke's area in the left hemisphere. It corresponds in part to Brodmann area 39. The angular gyrus merges with the supramarginal gyrus and is at the junction of visual, auditory, and touch centers. It is known to contain cells with mirror neuron properties (Ramachandran and Oberman 2006). We have observed decreased activity in these areas in the right hemisphere in clients with AS who display these symptoms of motor and sensory aprosodias (Thompson and Thompson 2003b).

Frontal and Prefrontal

Shamay-Tsoory et al. (2005) have hypothesized that prefrontal brain damage may result in impaired social behavior, especially when the damage involves the orbitofrontal and/or ventromedial areas of the prefrontal cortex (but not dorsolateral areas). These authors note that prefrontal lesions resulted in significant impairment in the understanding of irony and faux pas. In contrast to the patient who has damage to the amygdala, who cannot correctly understand the significance of another person's anger or aggressive behavior, the patient with orbital frontal damage recognizes the significance of other people's emotions but may fail to modulate their behavior as the social situation changes. This kind of impairment could lead to difficulty in correctly recognizing the intentions of others and thereby lead to inappropriate behavior (Bachevalier and Loveland 2006). In their paper, Bachevalier and Loveland posit that developmental dysfunction of the orbitofrontal-amygdala circuit is a critical factor in the development of autism and hypothesize further that the degree of intellectual impairment is directly related to the integrity of the dorsolateral prefrontal-hippocampal circuit of the brain. Wing (2001) notes that, as early as 1966, a Positron Emission Tomography (PET) study demonstrated that, unlike normal subjects, those with Asperger's syndrome did not show normal activation in the left medial prefrontal cortex during tasks that required them to consider what might be going on in another person's mind. Channon (2004) demonstrated that impairments in real-life problem solving are associated with left anterior frontal lobe lesions. Nikolaenko (2004) found that problems in metaphorical thinking are associated with decreased right hemisphere functioning.

Areas Related to Memory and Emotional Interpretation

Salmond et al. (2005) found that, in people with high functioning autistic spectrum disorders (ASD) there can be a profile of impaired episodic memory (hippocampus) with relative preservation of semantic memory (temporal lobe

cortex). Imaging studies have shown differences from normal in the density of gray matter at the junction of the amygdala, hippocampus and entorhinal cortex. These findings are said to be consistent with a recovering abnormality involving these areas. Structural abnormalities were also seen in these studies in the medial temporal lobes. These findings are of interest because, using LORETA, we consistently find EEG differences from the data base means (DBM) in the temporal lobe regions, including the hippocampus, in our clients with AS and autistic spectrum disorder (ASD). LORETA often shows EEG activity in a particular frequency band being more than 2 standard deviations from the Neuroguide database mean in these regions. Nacewicz and colleagues noted that: “Those in the autism group who had a small amygdala were significantly slower at identifying happy, angry, or sad facial expressions and spent the least time looking at eyes relative to other facial regions. Autistic subjects with the smallest amygdalae took 40 percent longer than those with the largest fear hubs to recognize such emotional facial expressions”. Their paper goes on to say that, “the autism subjects with small amygdalae had the most non-verbal social impairment as children” (Nacewicz et al. 2006). Irrational social behavior and social disinhibition result from amygdala damage (Adolphs 2003) and the human amygdala is critical for the retrieval of socially relevant knowledge on the basis of facial information (Adolphs et al. 2005). Using LORETA we often see abnormal EEG amplitudes in the right and/or left fusiform gyrus. The fusiform gyrus has been implicated in face recognition. Davidson’s research, performed at the Institute for the Study of Emotions, has shown that persons with autism have reduced activation of this face-processing area on both sides of their brains while performing a face-processing task, whereas their well siblings showed reduced activation only on the right side. He and his colleagues feel that this suggests an “intermediate pattern” in the siblings (Dalton et al. 2005).

Anterior Cingulate, Right Parietal Cortex, Attention, and Proprioception

Of interest, considering the importance of the anterior cingulate in fixation of attention and findings of abnormalities in the EEG in this area, are studies of attention. Landry and Bryson (2004) have shown that, once attention was first engaged on a central fixation stimulus, persons with autistic spectrum disorder had a marked difficulty in disengaging attention in order to shift attention to a second stimulus as compared to normal children and also compared to children with Down’s Syndrome. Belmonte and Yurgelun-Todd (2003) note that, in autism, physiological indices of selective

attention are abnormal even in situations where behavior is intact. They used functional magnetic resonance imaging (fMRI) while subjects performed a bilateral visual spatial attention task. In normal subjects, the task evoked activation in a network of cortical regions including the superior parietal lobe. Weimer’s research group has postulated that motor clumsiness associated with Asperger’s Syndrome may be caused by a deficit in proprioception due to an over-reliance on visual information to maintain balance and position in space (Weimer et al. 2001).

The ASD subjects’ differences from normal in the studies noted above correspond to our EEG observations where the clients with AS show EEG differences from the normal data base in the AC and in the right superior temporal lobe, hippocampus, and in the parietal cortex.

Theories for Grouping and Understanding the Symptoms of AS

Mirror Neuron System

Recent research concerning the mirror neuron system (MNS) is being applied to theories concerning what is different in the brain functioning of people with autistic spectrum disorders. The MNS is postulated to be involved in the imitation of movements, and perhaps also to copying appropriate social interactions, as well as being critical to understanding and predicting the behavior of others. The frontal MNS area may be responsible for understanding the intention of others. The frontal cortex mirror neurons are found in the pars opercularis: the dorsal portion has a ‘mirror’ function while the ventral portion may correlate with prediction of sensory consequences of a motor action. The pars opercularis is located in the posterior inferior frontal cortex (in the left hemisphere this is posterior to Broca’s area near F5 in the 10–20 electrode placement system) and the adjacent ventral prefrontal cortex. Parietal mirror neurons (emphasis on motoric description of action) are found in the rostral portion of the inferior parietal lobule. The visual input to the mirror neuron system (description of action, matching of imitation plan to the description of the observed action) comes from an area of the cortex in the posterior sector of the superior temporal sulcus (Iacoboni and Dapretto 2006). An fMRI study demonstrated that activity of the MNS is correlated with empathic concern and interpersonal competence (Pfeiffer et al. 2005). It has also been shown that children with ASD have reduced activity in MNS regions during tasks that require the child to mirror facial expressions of different emotions (Dapretto et al. 2006).

Mirror neurons have strong connections to the limbic system including the anterior cingulate (AC) (Iacoboni and Dapretto 2006). The cingulate and the insular cortices both contain mirror neuron cells (Ramachandran and Oberman 2006). The AC is well connected to the anterior insula and the amygdala and other areas of the limbic network and these areas are, in turn, connected to other areas involved in the mirror neuron system (Carr et al. 2003). The importance of imitation in social learning has been well described (Meltzoff and Prinz 2002). Imitation can be directly linked to the MNS and, significant for understanding ASD, structural abnormalities have been found in the MNS in ASD (Hadjikhani et al. 2006). A delayed conductivity in this MNS for imitation is also found in people with ASD (Nishitani et al. 2004). It is not surprising that deficiencies in this system are being hypothesized to be a core deficit in ASD. At our center, parietal and amygdala areas are both found to be outside the Neuroguide data-base norms when 19-channel QEEG and LORETA analyses are conducted.

Salience Landscape Theory

Although an inactive MNS could account for the lack of appropriate imitation of social behaviors, poor understanding of the actions of others, and lack of empathy, mirror neuron system deficiencies alone cannot account for some of the other symptoms that may be seen in children with disorders along the autistic spectrum, such as repetitive movements, or the need to maintain sameness, and hypersensitivity to sounds or to touch. Ramachandran and colleagues have therefore put forth a theory that they labeled the “salience landscape theory.” In the typical child, sensory information is relayed to the amygdala where it is compared to stored information and an appropriate emotional response is selected. The salience of the input is compared to an environmental landscape already in the child’s mind. They note the importance of the amygdala in this process and suggest that pathways from the sensory areas of the brain to the amygdala may be altered, resulting in extreme emotional responses to minimal stimuli. The amygdala may inappropriately trigger the autonomic nervous system so that the child’s heart starts racing and distress is experienced. Self-stimulation might actually dampen these responses and be self-soothing for the child (Ramachandran and Oberman 2006). For those with AS, engaging in activity related to their special interest area could provide the calming. The Mirror Neuron System theory, combined with salience landscape theory, thus is able to cover two groups of symptoms found in AS (and other ASDs) that involve brain areas that are functionally distinct and anatomically different.

Neuro-Cognitive Theories

Three older theories that attempt to explain ASD behaviors are described briefly below. More extensive explanations can be found in a publication by Hill and Frith (2003). The three neuro-cognitive theories are called theory of mind, central coherence (not to be confused with the term coherence used in QEEG analysis), and executive dysfunction.

Theory of Mind (ToM)

Theory of mind (which is sometimes more accurately called theory of others’ minds) involves the ability to “mentalize about both the self and others” (Abu-Akel 2003). In other words, it is the ability to comprehend the other person in order to make sense of their behavior and predict what they are going to do next. Ahmed Abu-Akel has created a neurobiological model to account for deficits in AS regarding the ability to construct a theory about what is going on in another person’s mind. This model implicates the posterior brain (parietal and temporal) in representational thinking and the prefrontal regions for the application and execution of theory of mind. ToM proposes that a fault in any component of the social brain can lead to an inability to understand aspects of social communication. Intuitive understanding of others, especially understanding what they are feeling or thinking, has always been understood to be a core deficit of the autistic spectrum disorders (Thompson and Havelkova 1983). As noted above, in neurological disorders resulting from infarcts, symptoms that correspond to difficulties seen in Asperger’s were well summarized under the terms *sensory, motor, and global aprosodias* by Ross in 1981, so those with AS function in some respects like people who have suffered brain damage to the right hemisphere. These children do not “read” the intentions of others and may be gullible, literal and concrete, symptoms described earlier in this paper. The examples given in Hill and Frith’s paper (2003) are well worth reading. These authors describe possible malfunction in the medial prefrontal cortex (anterior paracingulate cortex), the temporal-parietal junction, and the temporal poles.

The reader will note that these are also areas referred to in the above discussion of mirror neurons. The amygdala may also be involved and the reader will see the overlap here with the salience-landscape theory described above. They mention findings of less connectivity between the occipital and temporal regions and that is a finding that we observe using coherence analysis in the EEG with these subjects. Note that this theory does not account for the difficulty in recognition of faces, a symptom linked to dysfunction in the fusiform gyrus, which is another area

often observed to be outside database norms in our clients using surface EEG and LORETA.

Weak Central Coherence

The weak-central-coherence theory seeks to explain behaviors subsumed under the term ‘preservation of sameness’ and also to the special interests and talents of those with AS. The theory is that those with AS cannot draw together information and make sense of it in the usual way: they cannot come up with a coherent understanding of what is going on because they fail to take note of (or simply do not understand) how context changes the meaning or appropriateness of what is said or done. The child may only respond to part of what is said – the part that refers to his special interest area. With respect to context, think of the child who hugs you at the end of the interview (has not figured out that visits to an office and visits to a family friend’s home involve different etiquette), or who hugs classmates the way he saw football players hugging each other on television. Those behaviors were engaged in by the same youngster who was sent to the office for telling the supply teacher that she was not allowed to yell at the class (no yelling was his regular teacher’s rule). Other symptoms include rigidity, repetitive movements, and obsessive or preservative behaviors. Weak central coherence also relates to the observation that most people with AS have an incredible ability to recall details from past experiences that were important to them, even if they do not get the whole context correct. Weak central-coherence probably involves a lack of appropriate connectivity between areas of the brain. Connectivity in this discussion refers particularly to connections between the posterior sensory processing areas of the brain and the frontal areas that modulate responses to the sensory input (“top-down” modulation).

One result of this dysfunction may be piecemeal recall, rather than recall that shows an understanding of the total context, the Gestalt. Hill and Frith (2003) state that one cause of this deficit could be a failure of normal developmental “pruning” in early life that eliminates certain brain connections and optimizes the coordination of neural functioning. This could be one neural basis for the apparent perceptual overload experienced by individuals with AS. This overload may, in turn, be partly responsible for their “autistic” withdrawal. Withdrawal from social interaction and a focus on a narrow area of interest results in a reduction in the quantity of unpredictable sensory inputs. Hill and Frith cite fMRI findings of right lingual gyrus activation while processing local features of a visual presentation and suggest that this activation is associated with left inferior occipital cortex activation. QEEG findings at our clinic have similarly found the lingual gyrus to be

among the areas identified as the source of abnormal activity in some children with AS. Perhaps these parietal-occipital areas may be overly involved while, at the same time, there may be a prefrontal failure of modulation of this incoming sensory information resulting in the tendency to focus on piecemeal and often inconsequential detail while missing the big picture. One test they found to be difficult for clients with autistic spectrum disorder uses homographs, words which can have more than one meaning but which have the same spelling in each case. The meaning of a word such as “tear,” for example (to tear a piece of paper or to shed a tear), depends on the context of the sentence in which it is used and can thus be a source of confusion.

Weak central coherence as a theory would be supported by the work of Michael Greenberg, director of neurobiology at Children’s Hospital in Boston. His animal research investigates how experience shapes synaptic connections and he suggests that, in those with ASD, the normal pruning process goes awry. This would result in too much information being relayed, which results in overload – too much information to integrate efficiently—so just little bits are processed and perceived.

Executive Dysfunction

The third cognitive theory that has been advanced to help explain features that do not appear to be subsumed under the former two theories is called the “executive dysfunction” theory. Executive functioning (including attention, planning, inhibition and mental flexibility) appears to be impaired in clients with autistic spectrum disorder. This dysfunction is not unique to ASD but is also found in clients with other frontal lobe problems including head trauma, ADHD, obsessive compulsive disorder (OCD), and Tourette’s syndrome.

One test that measures many of the functions subsumed under the term executive-functioning is the Tower of London test (sometimes called the Tower of Hanoi). This test is difficult for clients with AS. The ToL requires the subject to move colored rings that are placed over three pegs of progressively shorter height until they match the arrangement on the examiner’s pegs. The test requires the subject to inhibit immediate response, plan, shift mental-set, use working memory, initiate a response and then monitor and evaluate the results of that response. The required cognitive functions all depend on good prefrontal functioning, an area also seen to be outside EEG database norms in our clients with Asperger’s. Improvement in performance on ToL has recently been reported in children with AS who received neurofeedback training (Knezevic 2007).

Another test that seems to address several of these parameters is the Wisconsin Card Sorting test that requires

the subject to understand the whole context of the test in addition to the detail and to be able, mid test, to evaluate what is going on and make a decision to try sorting the cards according to a new 'rule' (from sorting by, say, shape, to sorting by color or number). The subject is required to shift mental set (without being told to do so) and sort the cards on a different theoretical understanding of what is required. Clients with ASD persevere and find it very difficult to shift mental set and thus do poorly on this test.

Polyvagal Theory

Porges (2004), director of the Brain-Body Center at the University of Illinois, Chicago, has developed a comprehensive polyvagal theory that can be applied to help explain the physiology underlying the social engagement and attachment problems in ASD as well as account for symptoms like tactile sensitivity and poor listening skills. It involves three circuits that developed phylogenetically and that regulate reactivity: the unmyelinated vagus, whose behavioral function includes immobilization (as in death feigning in animals and passive avoidance in humans); a sympathetic-adrenal component that facilitates mobilization (fight-flight); and the myelinated vagus that is involved in the functions of social communication, self-soothing and calming.

His theory has relevance to many psychiatric disorders that involve emotional dysregulation and social interaction problems. Of particular interest is Porges' explication of why a person has to feel safe in order to participate in social behavior. Feeling safe involves evaluating the environment and some of the neural structures involved include the fusiform gyrus and the superior temporal sulcus. (Recall Iacoboni's work, cited above, dealing with visual input to the mirror neuron system coming from the posterior portion of the superior temporal sulcus.) Because these are not activated in those with ASD there is lack of inhibition of the limbic defense system involving the amygdala and the person remains vigilant and experiences anxiety. Also present is difficulty regulating visceral states, such as vagal regulation of the heart to slow it down. Of particular interest for AS, and the symptoms of flat facial expression (Fitzgerald 2004), poorly modulated tone of voice, and poor listening skills, is his explanation of the neural pathways that regulate the striated muscles of the face and head. Reduced muscle tone in this circuit correlates with less expressiveness in voice and face, less eye contact (eyelids droop), and slack middle ear muscles (distinguishing human voices from background noise becomes more difficult). In addition, he discusses the neurophysiological interactions between what he terms the Social Engagement System and the hypothalamic-

pituitary-adrenal (HPA) axis, the neuropeptides of oxytocin and vasopressin, and the immune system (Porges 2003).

His is the only theory that links head, heart and gut via bi-directional vagal pathways, both myelinated vagus involved in calming and unmyelinated vagus associated with immobilization. It is thus a theory that supports using the combination of neurofeedback and biofeedback. Heart rate variability training, for example, which involves effortless diaphragmatic breathing, can have a beneficial effect on vagal tone (Gevirtz 2007; Gevirtz and Lehrer 2005).

EEG Findings Related to Core Symptoms of Asperger's

Attention Span

Four core symptoms found in Asperger's clients are attention span problems, difficulties with social interactions, anxiety, and executive functions. Attention span and executive functions are also compromised in ADHD so one would expect overlap in EEG patterns given the overlap in symptoms. Symptoms of ADHD are most often associated with increased slow 4–8 Hz. (theta) activity in frontal and central cortical regions (Jantzen et al. 1995; Lubar 1991; Lubar et al. 1995; Mann et al. 1992; Monastra et al. 1999, 2002; Thompson and Thompson 1998) in conjunction with low amplitude sensorimotor rhythm (SMR) 13–15 Hz and reduced beta 13–21 Hz. Patterns seen in Asperger's are similar at the central location (Cz) and are often more extreme than simple ADHD in terms of theta/beta power ratios. (See Monastra et al. 1999, 2001, for norms for theta/beta power ratios, discussion of their utility in diagnosing ADHD, and validity and reliability information.) These EEG differences provide the rationale for decreasing the theta/SMR ratio at Cz and FCz using neurofeedback. The goal of this form of NFB for the ADHD symptoms is to train the subject to maintain a calm, relaxed, alert and focused mental state while carrying out cognitive tasks. These techniques have been developed over the last 30 years and have been described in previous publications (Lubar 1991; Lubar and Lubar 1984; Monastra 2005; Rossiter and LaVaque 1995; Shouse and Lubar 1979; Serman 2000b; Thompson and Thompson 1998, 2003b).

Social Interactions

Symptoms of Asperger's that involve social interaction include: sensory and motor aprosodia (neither reading or expressing emotion appropriately), difficulty initiating and maintaining close social relationships, and a pattern of having an intense single interest area to the exclusion of other activities. These areas of interest may be interpreted,

in part, as a defensive withdrawal from reciprocal interactions with others (Thompson and Havelkova 1983). Social interaction difficulties suggest involvement of the limbic system, including the anterior cingulate (AC), and areas in the right hemisphere identified as important in the aprosodias. These symptoms provide the rationale for normalizing EEG differences at CZ (FCz in adults) to influence AC functioning, decreasing the dominant high amplitude slow wave activity (somewhere in the 3–10 Hz range), decreasing high frequency beta (20–35 Hz) and increasing beta (somewhere in the 13–18 Hz range). Note that the particular frequency ranges would vary with each client according to findings from the initial assessment, but there would always be a slow frequency range to inhibit and a faster frequency range to enhance. The amygdala is important but, until LORETA NFB for direct feedback is readily available, we have to assume that we may be influencing it through its connections to the medial prefrontal area and the AC. Findings would also support training to influence activation in the right hemisphere over the parietal area (P4 and T6) and the right frontal area (F4). There could also be training to normalize connectivity between these areas. Connectivity is defined by coherence in most databases (for example, Neuroguide), co-modulation if using the SKIL analysis and database (Serman 1999). (For a discussion of databases, see Thatcher et al. 2003.)

Anxiety and Executive Functions in AS

One of the most important factors affecting daily functioning in people with AS is their underlying anxiety. In part, anxiety may be related to difficulty in distinguishing abstraction, innuendo and social meaning, which results in defensive withdrawal from emotionally laden social situations. But, more importantly, there appears to be atypical activation in areas of the brain related to anxiety. Attempts to cope with anxiety may result in other presentations of symptoms, such as those found in obsessive compulsive disorder (OCD) and social anxiety disorder. The anterior cingulate may be thought of as being the “hub” of the emotional control system and thus central to affect regulation and control. It also has executive functions. It has connections to premotor areas, spinal cord, red nucleus, locus coeruleus, and many connections with the thalamus. It exerts control of sympathetic, parasympathetic, and endocrine responses through its connections to all parts of the limbic system including the amygdala, hypothalamus, periaqueductal gray matter and autonomic brainstem motor nuclei. It is engaged in both response selection and in cognitively demanding information processing and in discrimination tasks concerning the motivational content of internal and external stimuli (Devinsky et al. 1995). It has

strong connections to the medial and orbital cortex of the frontal lobes and, as noted above, there are connections with the anterior insula and the amygdala and thus to the mirror neuron system. All of the theories mentioned above, from Ramachandran and Iacoboni’s work on MNS to Porges’ polyvagal theory, would support training the AC and its connections.

The surface location that best corresponds to AC findings using LORETA analysis is between Cz and Fz (Neuroguide 2007). This is the area that we have been addressing in our work over the past 15 years, using the research-validated neurofeedback (NFB) approach advocated by Joel Lubar for dealing with symptoms of ADHD. Perhaps our success with clients with Asperger’s has been due to our (unwittingly in the early years) focusing our NFB work at these sites after doing single channel assessments at the vertex. Explaining to parents that part of their child’s difficulties had to do with paying attention and that we felt we could address those deficits, training typically involved reducing high amplitude theta (3–7 Hz) or low frequency alpha (8–10 Hz) while increasing sensory motor rhythm (12–15 or 13–15 Hz). All training was individualized to the frequencies found to be either too high or too low for each individual client; for examples, inhibit frequency ranges might be 2–5, 3–7, 4–8, 6–10, or whatever range differed for a particular individual. In adolescents and adults this training might include decreasing high frequency beta (20–35 Hz) (Thompson and Thompson 2007b).

In older equipment, like the Autogen A620 program (Stoelting Autogenics) designed with Lubar’s input, frequencies went up to 32 Hz and so the frequency range used to inhibit EMG artifact would also have inhibited high frequency beta spindling and contributed to reduced anxiety. Beta spindling (bursts of beta in a narrow, often single Hertz, frequency band, of high amplitude and synchronous morphology) has been found in people who tend to ruminate (have trouble letting go of something they are thinking or worrying about) and has been dubbed a “busy-brain” pattern (Thompson and Thompson 2006a, b). Beta spindling may correspond to an unstable, easily kindled cortex so reducing this beta might be expected to stabilize the cortex, and improve functioning (Johnstone et al. 2005). However, it should be noted that high frequency beta is not necessarily a negative finding. It may be associated with a highly productive but very “busy” brain. Increasing SMR might also be expected to stabilize the cortex as evidenced by research on epilepsy (Serman 2000a).

Combining NFB with biofeedback, in particular training to increase heart rate variability and skin temperature while decreasing muscle tension, is an integral part of intervention for these symptoms.

Implications of 19 Channel QEEG and LORETA Findings for NFB Training

By combining knowledge of functional neuroanatomy with the foregoing theoretical discussions, a picture emerges concerning the difficulties experienced by those with ASD. Encouragingly for those who use neurofeedback, quantitative electroencephalographic assessment (QEEG) can pinpoint cortical areas with abnormal activation as compared to database norms. These areas can then be addressed using the neurofeedback plus biofeedback approach.

Based on the foregoing review, one might postulate differences (an axis of disturbed functioning) in the right temporal-parietal cortex, the posterior cingulate (Brodmann Area [BA] 31), anterior cingulate (BA 24, 25), medial and orbital frontal cortex, prefrontal cortex, the amygdala, hippocampus, and the fusiform gyrus. EEG and LORETA findings may include very high or low amplitude delta, theta, alpha or beta (13–18 Hz) activity and/or high frequency spindling beta (a narrow frequency band within the 19–35 Hz range) in these areas. Decreased activation can be indicated by high amplitude slow wave activity and/or low amplitude beta (13–18 Hz). In addition to differences found at the vertex (CZ), and between CZ and FZ, abnormalities are frequently found at T6, or between T6 and P4 and, on occasion, at F4. Functions of the area near T6 include spatial and emotional contextual comprehension, and non-verbal memory.

Figure 1 provides a picture of high amplitude, low frequency alpha activity at T6 in the raw EEG with the traditional linked ears reference. In Fig. 2 the same pattern is even more clearly seen with a sequential montage. Figure 3 is an example of frontal and central beta spindling in an adult client with AS. Figure 4 is a LORETA image that indicates that the probable source of the beta spindling is the anterior cingulate (BA 24) in this client. These are representative samples taken from two clients, but the same pattern has been seen in dozens of clients evaluated at the ADD Centre. The observations have been replicated by others in the field: Linden (2006), Coben (2006) and the authors took part in symposia concerning ASD at annual meetings of the Association for Applied Psychophysiology and Biofeedback (AAPB) and the International Society for Neurofeedback and Research (ISNR) and EEG findings were convergent from these three clinical settings.

Note that in the following figures the original International 10–20 System (Jasper 1858) designations for electrode placements are used as they are throughout this paper in order to be consistent with the nomenclature of the Neuroguide database used in the illustrations. New terminology renames T3 and T5 as T7 and P7. The sites T4 and T6 are now referred to as T8 and P8 (Fisch 1999, Appendix II).

While these exemplars from two clients suggest a phenomenon, many more cases are required to show that it is a more generalized finding. Support for these patterns being typical of people with AS came from a review of all 19-channel assessments conducted with clients with diagnoses of Asperger's ($n = 58$) or autism ($n = 11$). These clients were assessed at the ADD Centre but did not necessarily go on to complete training; indeed, only 17 of these cases are included in the case series of outcomes using neurofeedback reported elsewhere (Thompson et al. 2008). Others were either consultations performed for people outside the center's geographic catchment area, families who wanted diagnosis only, often in order to access special education interventions at school, or children the first author deemed inappropriate for training at the time they were seen. (Criteria for the latter group included things like dysfunctional families, inability to co-operate and/or attend during the practice EEG session to the extent that little true feedback could occur, the opinion that, given a family's time and financial resources, other interventions, such as intensive speech therapy for an autistic child, should take precedence. One wants to select clients where a cost-benefit analysis justifies the neurofeedback intervention.)

In originally looking at the EEGs the emphasis was on raw EEG observations or QEEG findings that really stood out as being outside of expected norms and could therefore be addressed in the NFB training program. We were not searching for specific EEG findings, in large part because we were not sure what to look for when the early EEGs were completed. When Neuroguide plus LORETA were eventually used, it became easier to distinguish levels as outside the database mean (DBM) for children. Norms for Cz theta/beta ratios (Monastra et al. 1999) were used for the single channel EEG assessments. Note that when using comparison to a database mean (DBM), the controls for age and sex are already in the database. If 2sd is used as the cut off, then findings are in the extreme 2% of the population range and any records $>3sd$ means the finding would be expected in less than 1% of the general population.

In the 58 cases with Aspergers, 48 (83%) showed T6 to be less active than T5. In half of these the difference appeared to be very large. The slowing was usually indicated by excess alpha 8–9 Hz. In a few cases it was excess theta 3–5 Hz. In one case it was shown by very low amplitude beta, 15–18 Hz, at T6. In a small number of children, dyslexia may have accounted for slowing at T5 (and P3) that equaled the slowing observed at T6. Fifty of the records (86%) demonstrated theta or low alpha excess at Fz and Cz and theta/beta ratios at Cz above the Monastra et al. (1999) norms. Of those records that did not show this slow wave pattern (the remaining 14%), all showed beta spindling. Some records showed both excess slow wave activity and beta spindling, so the phenomena are not

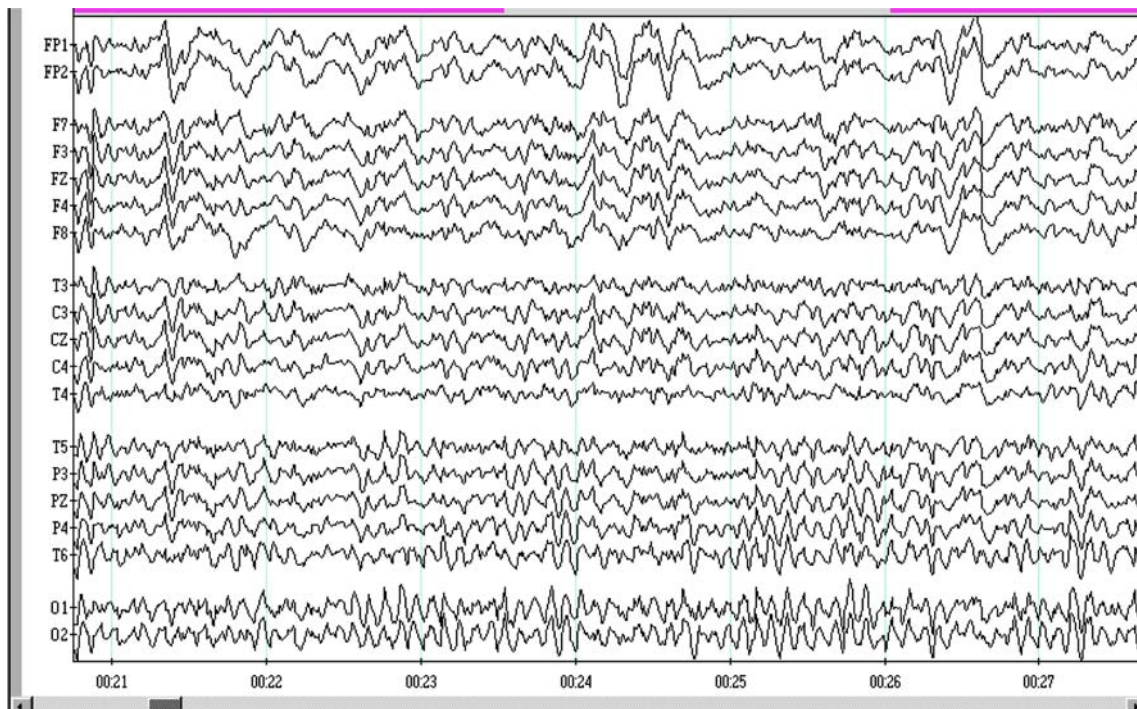
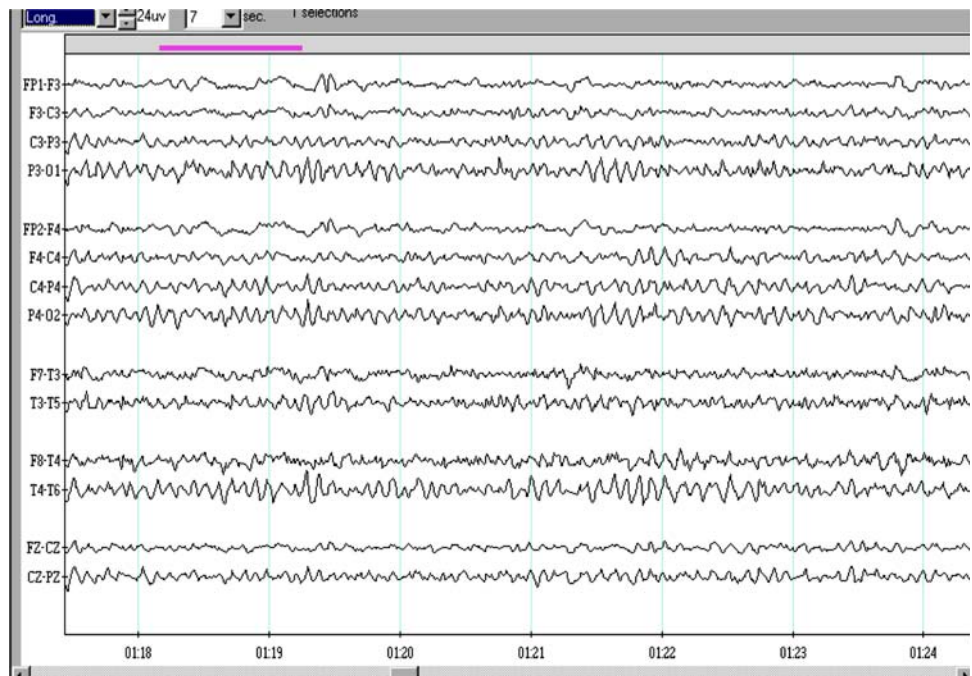


Fig. 1 Raw EEG: Eyes closed, linked ears reference. In the above figure compare T6 with T5. Note the alpha activity at T6

Fig. 2 Same client as Fig 1. Eyes closed, longitudinal sequential (bipolar) reference, EEG. In the above figure compare T4–T6 with T3–T5. Note the high amplitude alpha at T4–T6



mutually exclusive. This supplied the main rationale for training at FCz. The slow wave and high theta/beta ratio so often found at these central sites should not be a surprise because the most common EEG finding for ADHD is excess theta at the central location. Another, much smaller, subtype is excess beta (Thompson and Thompson 2006a, b).

Virtually all of our clients with Asperger’s or autism have inattention as a major symptom.

In those 29 records where LORETA was applied, we found that the anterior cingulate (Brodmann Area 24) was identified as the source for the beta spindling activity in 22 cases (76%). In addition, one or more of the following

Fig. 3 Raw EEG: Eyes open condition, linked ears reference. In the above EEG note beta spindling at F3, Fz, F4, Cz. In LORETA this 20 Hz beta was $>2SD$ above the Neuroguide data base mean. Source was Brodmann area 24 in the anterior cingulate

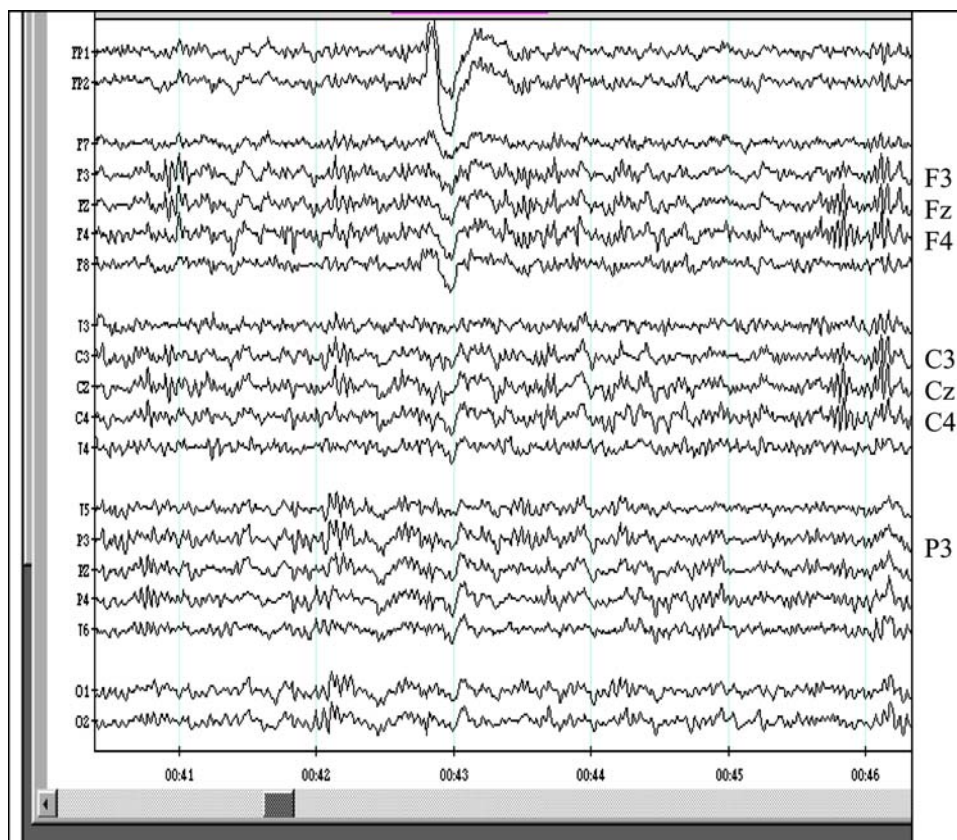
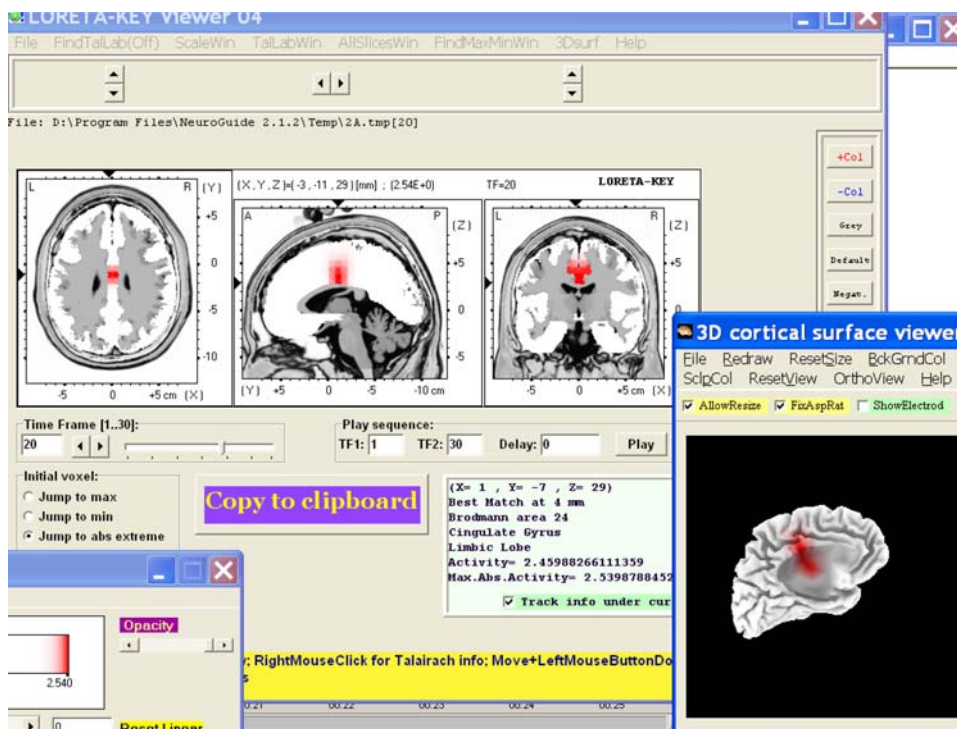


Fig. 4 LORETA analysis on eyes open, linked ears reference EEG shown in Fig. 3. LORETA shows 20 Hz is 2.5 SD above the Neuroguide data base mean, Brodmann area 24 in the anterior cingulate gyrus is suggested as the source



areas were $>2sd$ above the DBM: insula and fusiform gyrus in about 52% of records, amygdala and uncus in 24%. The medial and orbital prefrontal areas, the hippocampus and

parahippocampus were also sites of significant differences noted in individual clients. At least one of those areas was noted to be $>2sd$ above the DBM in 83% of these 29

records analyzed using LORETA. We reviewed whether right or left side predominated for any one of these areas and found it was, for the most part, both sides that were affected with a slight trend for left amygdala, uncus and fusiform gyrus and right insula (which influences sympathetic system activity) to be implicated as the source of abnormal activity.

Half of the children with autism demonstrated T6 slowing compared to T5. With this group generalized parietal slow wave activity and coherence values well outside the database for Neuroguide in the parietal region were the common finding. In addition, compared to our ADHD population who show high amplitude slow wave at Fz and Cz, the group with autism showed high amplitude theta at Pz and Cz.

In visual analysis of the raw EEG mu was noted to be present in 7 cases (just 10%), as determined by morphology of the waves and the lack of suppression during the eyes open conditions. We feel this may be an underestimate as we were not looking for this as a factor in the early years. In addition, previously undetected seizure activity was seen in 3 cases. The cases with seizure activity were referred to a neurologist.

NFB Training

Training recommendations, based on the findings shown in Figs. 1, 2, 3, 4, suggest that normalizing the EEG could involve using two-channel NFB training. One channel would have the active electrode placed at FCz (to influence the AC) and the second channel would have the active electrode at T6 (to decrease the sensory aprosodia symptoms). Both would be referenced to linked ears (or to a common reference point even less influenced by surrounding electrical activity, such as the nose) and have a common ground (Thompson and Thompson 2007a, b). This training might reasonably be expected to ameliorate some of the core symptoms of AS (inattention, anxiety, not reading emotions/nonverbal communication) (Thompson et al. 2008). Alternatively, one could use single channel training and work first at the central location and then later at T6 if the ability to pick up on nonverbal communication continued to be a problem for the client.

Increasing sensorimotor rhythm (SMR) using neurofeedback may have a stabilizing effect on a cortex that is unstable and easily kindled (Serman 2000a). As previously noted, beta spindling is one indication of a cortex that may be easily kindled, irritable, even unstable, in other words, a cortex that may not be functioning properly. Beta spindles are high amplitude, narrow band (often a single Hz),

synchronous beta (Johnstone et al. 2005; Thompson and Thompson 2006a, b). Beta spindling is an EEG finding that may be observed in a number of disorders where anxiety is a symptom. In our experience using LORETA, spindling beta has most often been associated with a source in the cingulate gyrus. Perhaps our past success in these disorders, when we emphasized normalizing theta, alpha, and beta amplitudes and increasing SMR rhythm at CZ, was only in part due to re-setting thalamic pacemakers. Perhaps it was also due to normalizing anterior cingulate (AC) activity with all its connections to various elements in the mirror neuron system in addition to the hypothalamus and the brainstem nuclei controlling the autonomic nervous system.

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 of these clients. In our experience mu, which is considered a normal variant, is not observed in the majority of clients. Therefore using this as a training parameter for NFB, as suggested in an article in *Scientific American Mind* (Ramachandran and Oberman 2006), would not be our initial approach. Additionally, it can be difficult to distinguish between mu, alpha, and sensorimotor rhythm at the C3 and C4 sites given that the frequency ranges overlap. One must examine the raw EEG to check morphology of the wave forms (mu being a wicket rhythm with one end sharp and the other rounded, whereas alpha and SMR are sinusoidal) and/or check for responsiveness (alpha is attenuated by eye opening and SMR and mu respond to movement of the hands).

With QEEG, Chan and colleagues at the University of Hong Kong have recently attempted to find an EEG marker for ASD. They found that children with ASD showed significantly less relative alpha power. This was not specific to restricted location(s) but was a widespread pattern (Chan et al. 2007). Less alpha may indicate a brain that does not rest appropriately, which is perhaps the flip side of the picture of spindling beta and low SMR frequently found in clients with AS.

Both hyper and hypo coherence abnormalities are found in clients with autistic spectrum disorders. Care must be taken in evaluating theta and alpha hypercoherence because sometimes it may be due to ear reference contamination; for example, very high amplitude alpha at a site such as T6 near the linked ear reference will make it appear as though many other sites have alpha. Coherence often changes after amplitude training is done to increase and decrease frequencies found to differ from database norms.

Coben (2007) has demonstrated good outcomes with normalization of coherence after training that used sequential (bipolar) placement at pairs of sites that showed hypercoherence on QEEG assessment.

Rationale for Neurofeedback Intervention

EEG differences observed in clients with Asperger's syndrome provide a rationale for using neurofeedback. Specifically, these findings include

1. excess slow wave activity frontally corresponds to "being more in their own world";
2. excess slow wave and/or beta spindling at Fz, found to originate using LORETA in the medial frontal cortex with its connections to the amygdala and to the AC, may correspond to difficulties modulating emotions;
3. low SMR is consistent with fidgety, impulsive behavior, tactile sensitivity, anxiety, and emotionally labile behavior;
4. excess left prefrontal and frontal slow wave activity is consistent with lack of appropriate inhibition and failure to properly modulate sensory inputs;
5. excess right prefrontal beta activity is consistent with lack of appropriate affect modulation and the inhibition of impulsivity;
6. less activation, as evidenced by high slow wave activity and/or low, low frequency beta activity (beta frequencies <20 Hz) in the right parietal-temporal area (homologous site to Wernicke's area), is consistent with difficulty interpreting social cues and emotions (sensory aprosodia);
7. excess slow wave activity and/or reduced low frequency beta activity in right frontal cortex (homologous site to Broca's area), is consistent with underactivation and inability to appropriately express emotion through tone of voice (symptoms of motor aprosodia);
8. deviations from a normal data base in frequencies whose source was identified by LORETA to be in the anterior cingulate (including beta spindling) may correspond to anxiety related symptoms (including obsessive-compulsive tendencies).
9. temporal lobe and, in particular, the superior temporal gyrus and fusiform gyrus activity outside of data base norms may indicate difficulty inhibiting the central nucleus of the amygdala (Porges 2007), which can result in an adverse effect on vagal calming and allow increased sympathetic drive
10. abnormalities in coherence suggest that training for normalizing connectivity between the parietal lobes

and the temporal and frontal regions may prove to be beneficial.

Rationale for Biofeedback Intervention

Changes in physiological variables with minor stressors and the client's inability to rapidly recover after stress, as may be demonstrated with a stress test (Thompson and Thompson 2007b), provide one rationale for using biofeedback. Learning comfortable, slow diaphragmatic breathing at about six breaths per minute (faster in children) gives those with AS a portable stress management technique. In addition, vagal afferents from the heart feed back to the nucleus of the solitary tract in the medulla which is connected to the parabrachial nucleus and the locus coeruleus. These nuclei connect to the forebrain with links to the hypothalamus, amygdala, thalamic connections to the insula, orbitofrontal and prefrontal areas, all of which give feedback to the anterior cingulate (Porges 2003, 2007). Theoretically this could synergistically assist in normalizing the activity of the AC and its connections through both the mirror neuron system and the limbic system. Using NFB plus BFB and coaching in strategies exemplifies a holistic approach that emphasizes *skills not pills*.

Summary and Discussion

Neuroanatomically, the common areas that are posited to be influenced by neurofeedback plus biofeedback in clients where FCz was the site for the NFB training and BFB including HRV are first, the cingulate gyrus, usually the anterior cingulate (AC) with all its connections through the limbic system and the mirror neuron system and, second, the autonomic nervous system with all its brainstem connections to the amygdala and the forebrain. As previously noted, the AC is central to affect regulation and control. It has executive functions and it is critically involved in the processes of attention and concentration. Additionally, the AC is also well connected to the insula and the amygdala and to the mirror neuron system (Carr et al. 2003).

As is reported in another paper (Thompson et al. 2008) we have had 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 (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 non-verbal information (so-called social cues) when working with clients with Asperger's or autism. 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, even though it is an area that initially shows less activation.

It would be interesting to conduct an experiment using only BFB and, in particular, heart rate variability training to see what effect this might have on clients with AS. Performing another experiment with just NFB would also be of interest to tease out specific effects. However, as a clinician, one looks for the combination that best helps a client realize their potential. In real life a brain-body separation is artificial. As Walter Hess said in his 1949 Nobel prize acceptance speech, "Every living organism is not the sum of a multitude of unitary processes, but is, by virtue of interrelationships and of higher and lower levels of control, an unbroken unity". For our purpose of explaining why neurofeedback plus biofeedback can re-balance and thus improve mind-body functioning, we have developed a Systems Theory of Neural Synergy. The name is used in order to emphasize that, no matter where we enter the nervous system with our interventions, the neural system will adjust its own new balance and equilibrium (Von Bertalanffy 1976). Whether we train the brain (NFB) or we train the heart (BFB) the neural pathways do connect across the forebrain, the midbrain and the hindbrain. In particular, the anterior cingulate connects with the brainstem, as fits with Porges' polyvagal theory. The AC connects to the nucleus ambiguus giving it control over aspects of the vagal parasympathetic efferents controlling such important physiological functions as heart rate while it receives vagal afferent feedback from such organs as the heart via connections relayed through the nucleus solitarius in the medulla of the brain stem. The vagal afferent sensory information is conveyed from the medullary nucleus of the solitary tract to the parabrachial nucleus and the locus coeruleus. As noted under the Rationale for BFB Intervention above, these nuclei connect to the forebrain with links to the hypothalamus, amygdala, thalamic connections to the insula, orbitofrontal and prefrontal areas, all of which give feedback to the anterior cingulate. The central nucleus of the amygdala also directly connects to the brainstem autonomic nuclei. Our conclusion is that all of these considerations support the importance of recognizing the interconnectedness of the entire central nervous system (CNS) and, in our work with the ASDs, the combined use of NFB and BFB.

Studies have shown that neurofeedback is effective in reducing the symptoms of ADHD and evidence is

mounting that EEG biofeedback can similarly play an important role in helping those with Asperger's Syndrome learn self-regulation skills that address their unique challenges (Coben 2007; Jarusiewicz 2002; Knezevic 2007; Linden et al. 1996; Reid 2005; Thompson and Thompson 1995, 2007a, b). This paper provides a theoretical basis, derived from knowledge of functional neuroanatomy, for the improvements being reported in clinical practice.

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Neurofeedback Outcomes in Clients with Asperger's Syndrome

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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

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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 neurofeedback 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 μV above the average activity level of the wave band. The fast wave

reward thresholds were set 0.2–0.6 μV 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

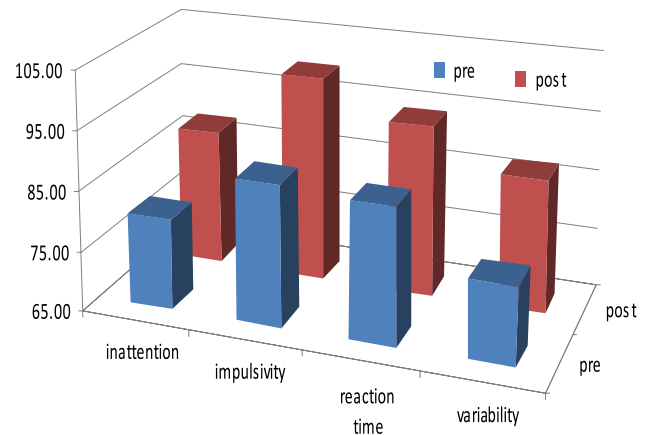


Fig. 1 T.O.V.A. the test of variables of attention is a continuous performance test. Graphic representation of changes in mean standard scores on the T.O.V.A.

1990 s 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

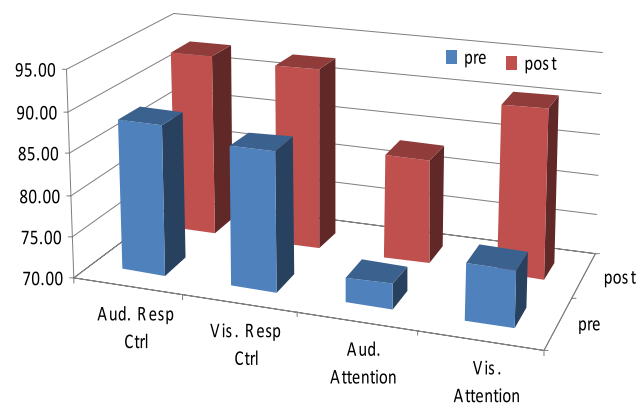


Fig. 2 The Integrated Visual Auditory (IVA) continuous performance test. Graphic representation of pre-post changes in mean standard scores on the IVA

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.

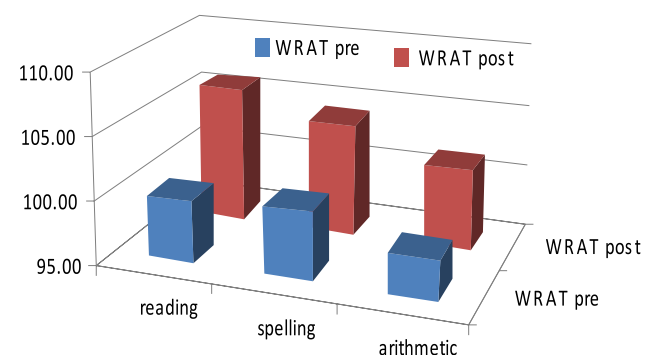


Fig. 3 Graphic representation of pre-post changes in mean standard scores on the WRAT. For the total group $P < .01$ for changes on each of the three variables

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

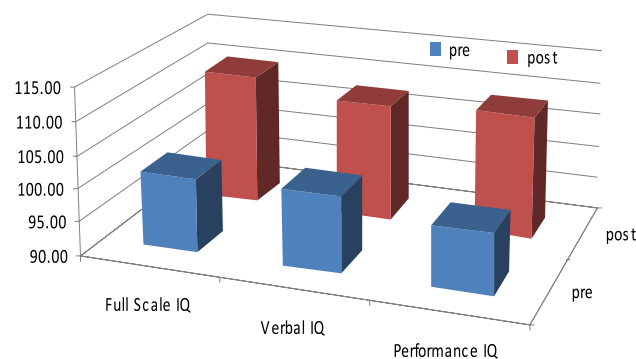


Fig. 4 Wechsler Intelligence Scale. Changes in the sum of scaled scores on Wechsler Intelligence Scale

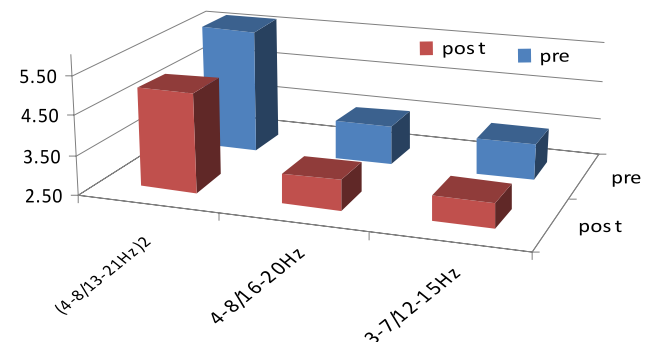
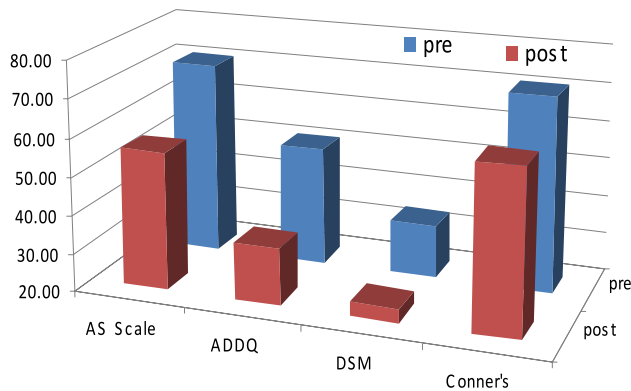


Fig. 5 Single Channel EEG CHANGES. Graphic representation of pre-post changes showing decrease in mean scores on theta/beta power ratio, theta/beta microvolt ratio, and theta/SMR ratio

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 Zolofit. 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.

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