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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

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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

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 overstimulated 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 Manhatten, 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 nonverbal, 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 autistism 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 perseverate 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; Sterman 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 (Sterman 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 two 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 (Sterman 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

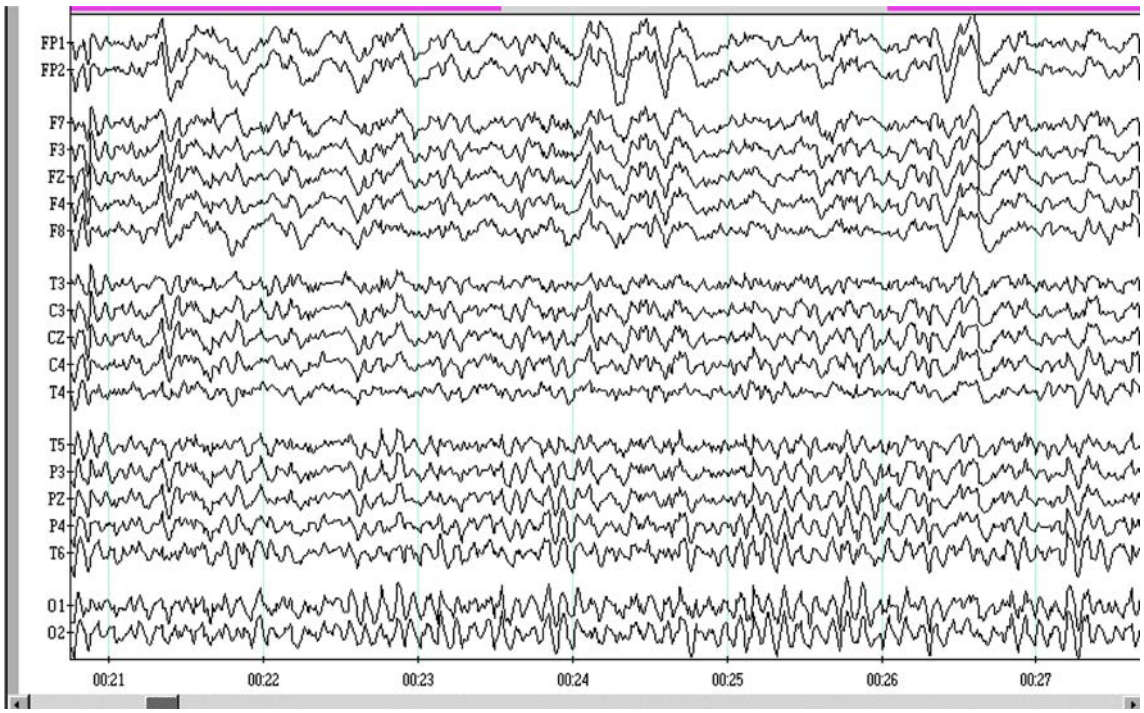

Fig. 1 Raw EEG: Eyes closed, linked ears reference. In the above figure compare T6 with T5. Note the alpha activity at T6

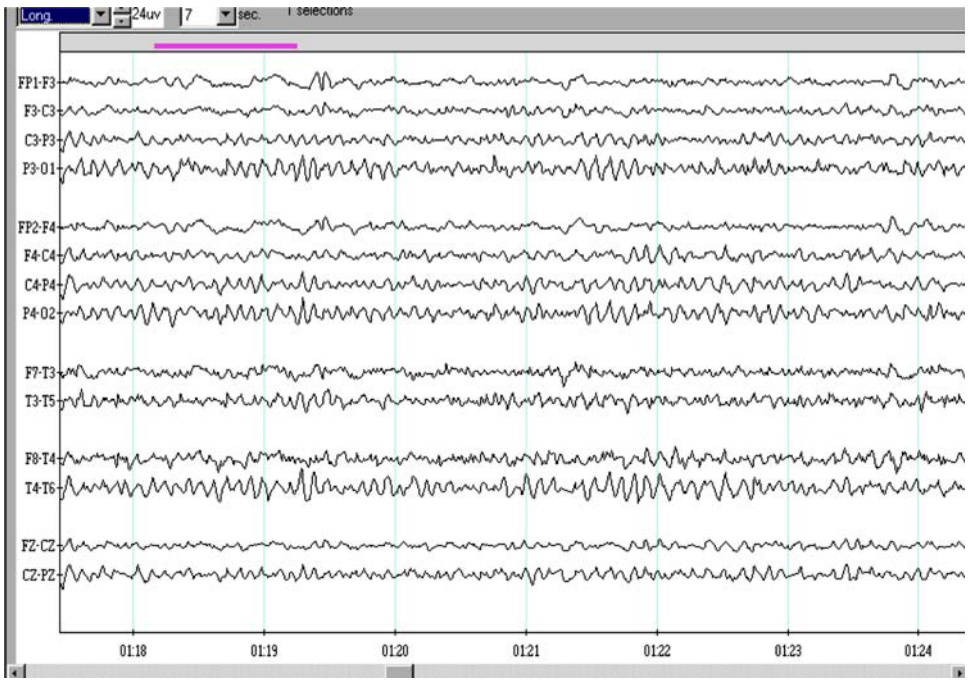

Fig. 2 Same client as Fig 1. Eyes closed, longitudinal sequential (bipolar) reference, EEG. In the above figure compare T4–T6 with T3–T5. Note the high amplitude alpha at T4–T6

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

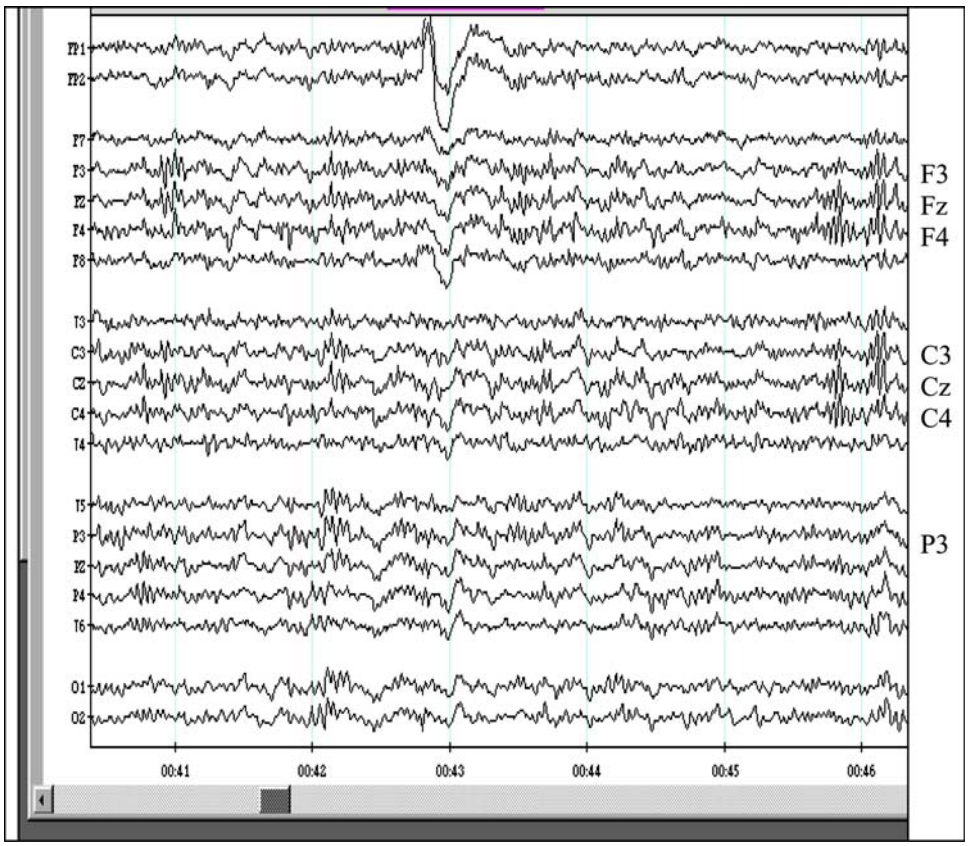

Fig. 3 Raw EEG: Eyes open condition, linked ears reference. In the above EEG note beta spindling at F3, Fz, F4, Cz. In LORETA this 20 Hz beta was >2SD above the Neuroguide data base mean. Source was Brodmann area 24 in the anterior cingulate

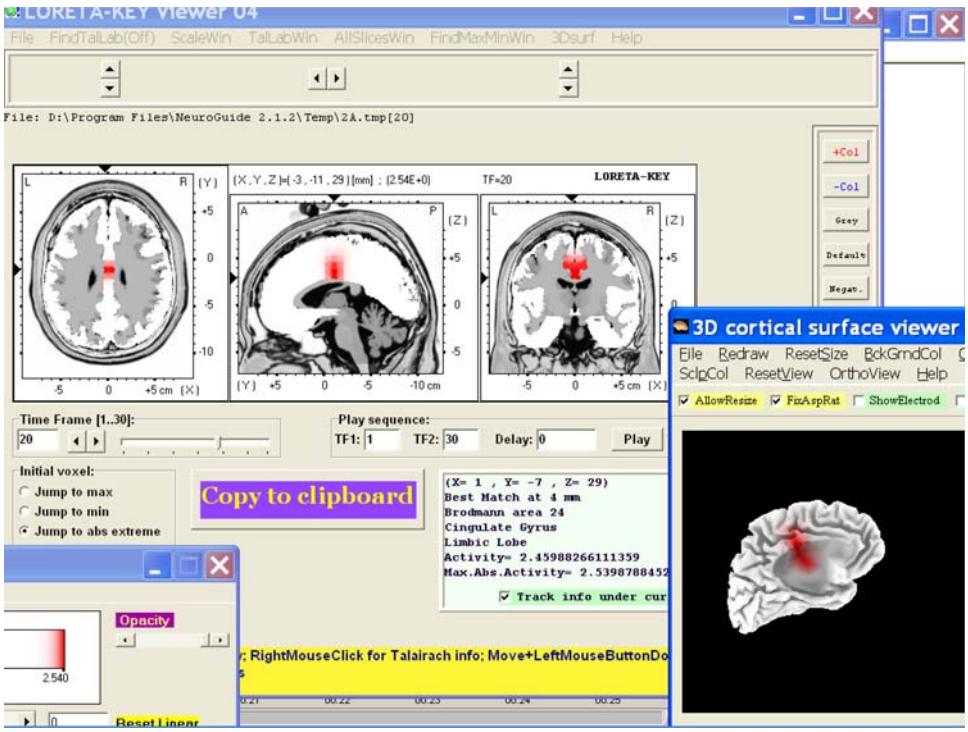

Fig. 4 LORETA analysis on eyes open, linked ears reference EEG shown in Fig. 3. LORETA shows 20 Hz is 2.5 SD above the Neuroguide data base mean, Brodmann area 24 in the anterior cingulate gyrus is suggested as the source

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 (Sterman 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 solitarus 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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