Sensory Integration Used with Children with Asperger’s Syndrome
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Abstract

Sensory Integration Program on Children with Asperger’s Syndrome This literature review will document the effects of a parent implemented Sensory Integration Program upon children diagnosed with Asperger’s Syndrome in order to discern its influence upon these children’s overall ability to attend to learning and social development. The infrequency of research on Asperger’s Syndrome and sensory-based programs indicates a need for further study into the potential relationship between evaluation regarding Asperger’s Syndrome and sensory-based approaches. The following literature review reflects the findings of current research within the fields of psychology, education, neurology, medicine, and occupational therapy as they pertain to Asperger’s Syndrome.

Asperger’s Syndrome

Volkmar and Klin (2000) state that Asperger’s Syndrome (AS) is a neurodevelopmental disability that can be distinguished by limited interests and social deficits. Viennese pediatrician Hans Asperger (1943) identified the features of AS in the early 1940s and Wing (1981) gave further details about the disability’s features decades later. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR, 2004), Asperger’s is classified as a Pervasive Developmental Disorder (PDD). Although, Asperger’s differs from other PDDs in that children diagnosed with AS meet normal developmental milestones in the area of language acquisition and cognitive ability. There is a continued need to research Asperger’s Syndrome (Bashe, Kirby, & Attwood, 2001). Although an AS diagnosis does not look at sensory processing deficits (DSM-IV-TR, 2004), numerous studies note that sensory deficits are common in people with AS (Barnhill, 2001; Dunn, et. al, 2002; Mailloux, 2001).

Characteristics of Asperger’s Syndrome

Hans Kanner published a paper in 1943 that described the characteristics of people diagnosed with autism. Within a year of Kanner’s paper, Hans Asperger published his own report on a population of children that he described as
exhibiting difficulties with social interaction and motor skills (Volkmar, Klin, Schultz, Bronen, Marans, & Sparrow, 2000). Wing (1981) described commonly recognized features of AS as: lack of the ability to empathize with peers, limited social skills, limited nonverbal communication skills, poor motor coordination, and/or a rigid absorption in topics requiring rote memorization (Myles and Simpson, 2002; Mailloux, 2001).

Asperger (1968) focused the intent of his studies on the social and learning development of children which displayed the symptoms of Asperger’s Syndrome. Mailloux (2001) notes that Asperger focused on the AS child’s inability to read facial expressions and understand social concepts. Asperger thought that AS children were not incapable of interacting with the outside world. Instead, Asperger thought that children with Asperger’s Syndrome had peculiar methods of interacting with the world around them. Asperger (1991) acknowledged the AS child had sensory processing deficits that limited their access to maximal learning opportunities and delayed their social development (Falk-Ross, Iverson, Gilbert, 2004; Mailloux, 2001; Myles & Simpson, 2001).

**Diagnostic Criteria of Asperger’s Syndrome**

The World Health Organization (WHO) (1993) recognized AS as a separate disability category that fell under the heading of Pervasive Developmental Delays. Three years later, the disorder was recognized by the Diagnostic and Statistical Manual of Mental Disorders-IV Edition (2000; 2004). It took the majority of the medical community (Myles & Simpson, 2002) a while to recognize the disorder as a separate disorder, distinct from autism.

Klin and Volkmar (2000) acknowledged the listing of AS as a separate category disorder, distinct from autism and PDD, as reliable based upon the DSM-IV-TR (2004) criteria. The systematic classification of Asperger characteristics and symptoms developed by Wing was relied upon heavily (Gillberg & Gillberg, 1989; Tantum, 1988) by medical personnel, before the WHO and the DSM-IV recognized the disorder as a distinct disorder. There were attempts made to develop diagnostic scales for diagnosis based on symptoms (Szatmari, 1991; Gillberg & Gillberg, 1989). Attwood (1998) asserts that the criteria set forth by the Gillbergs for diagnosing Asperger’s Syndrome (1989) were widely accepted due to the criteria’s clarity, conciseness, and comprehensiveness. The International Statistical Classification of Diseases and Related Health Problems-10th edition, Szatmari’s Rating Scale, Gillbergs’ Asperger’s Rating Scale and the DSM-IV-TR criteria are all similar in their basic diagnostic function. Diagnostic rating scales seem to share many of the following criteria:

A. distinctive preoccupation with stereotypic patterns of behavior, such as repetitive body movements or repetitive movement of objects, or distinctive preoccupation with stereotypic patterns of thought processes.
B. delayed social impairment that is qualitative in nature; or emotional reciprocity; failure to develop relationships with peers; lack of want to share meaningful life moments with others.

C. no significant delays in oral language acquisition exist.

D. definitive impairment in social and/or occupational and/or other areas of life functioning (Myles & Simpson, 2002). This is a tendency to perseverate on specific areas of interests to such an extent that the individual’s social and/or professional life is adversely affected.

Prevalence of Asperger’s Syndrome

Asperger’s Syndrome has a prevalence rate in children of 9 out of 2500 births (Cumine, Leach, & Levinson, 1998), according to the most recent research. In other studies, prevalence rates were shown to be at 0.024 to 036 percent (NAMI, 2004). The ratio of boys to girls is found to be at 9:1. Kadesjö, Gillberg, and Hagberg completed a study in 1999 that surprisingly found that a higher amount of children are diagnosed as having AS by using Szatmari’s scale than when using the Gillbergs’ scale.

In a study conducted by Ehlers and Gillberg (1993), the prevalence of children with Asperger’s Syndrome in Sweden were 3.6:1,000. According to Ehlers and Gillberg the male to female ratio was 4:1. The study’s authors concluded that the prevalence of AS would be higher if all possible instances of AS could have been included. Ehlers and Gillberg think the prevalence rate is actually at 7:1,000. Ehlers, Gillberg, and Wing (1999) believe the prevalence of AS is higher than they can estimate. Once a diagnostic rating scale for Asperger’s Syndrome can be universally agreed upon (Attwood, 1998) there will be a more uniform prevalence rate.

Etiology of Asperger’s Syndrome

The exact cause of Asperger’s is not known (Ozbayrak, 2004). Asperger's Syndrome is a neuro-developmental disability (Volkmar & Klin, 2000) that can be distinguished by social deficits and a rigid absorption in topics that require rote memorization (Mailloux, 2001). Lincoln and colleagues (1998), report that Asperger’s Syndrome is caused by abnormal brain development and function. Autopsy studies conducted by Courchesne and Pierce (2000) revealed abnormalities of the brain. Lincoln et al. (1998) compared the results of studies conducted that utilized magnetic resonance imaging (MRI) and noted that Asperger patients have severe neurological abnormalities of the brain, such as an enlarged brain volume in the frontal and cerebellar regions of the brain. Brain abnormalities and anomalies have been linked to the Asperger child’s prenatal,
perinatal, and neonatal development (Tsai, Jacoby, Stewart, & Beisler, 1982).

Hans Asperger (1944) first noted a genetic link in his patients occurring between the male members of families. Asperger recorded that the male family members of his patients had strikingly similar characteristics. As AS research has progressed, researchers have continued to suggest that there is a genetic link in Asperger cases (DeLong & Dwyer, 1988; Gillberg, 1989; Kranowitz, 2006).

DeLong and Dwyer conducted a study, in 1988, on the family history of autistic patients and concluded that almost seventy-percent of the autistic subjects had a close relative that was diagnosed with Asperger’s Syndrome. Kerbeshian and Burd found in their 1986 study that fifty-percent of their Asperger diagnosed subjects had family members that had characteristics that were similar to AS symptoms. Wing and Burgosine studied triplets, in 1983, which were diagnosed with AS and reported findings that yield to a genetic link. Volkmar and Klin (2000) reported that a study on monozygotic twins supports genetic links in Asperger’s. With the research done, the possibility of genetic transmission of Asperger’s Syndrome is plausible.

**Co-occurrence of Asperger’s Syndrome**

Children who are diagnosed with Asperger’s often are diagnosed with multiple disabilities or disorders at the same time (Ozbayrak, 2004). Volkmar and Klin (2000) related the co-occurrence of AS with obsessive-compulsive disorder. There has also been a noted association between AS and schizophrenia (Tantum, 1988) and AS with nonverbal learning disorders (Raja, et al., 1998). Green, et al. found in 2000 that children with Asperger’s are at high-risk of developing depression, oppositional-defiant disorders, and anxiety disorders (Fonseca & Perrin, 2001). In 1944, Asperger reflected that patients with AS are subject to affective disorders. Other researchers have noticed a link between Tourette’s Syndrome and AS (Kerbershian & Burd, 1986). It does appear that a large number of children diagnosed with Asperger’s Syndrome are diagnosed with other disabilities (Ozbayrak, 2004), though no general assumptions can be made at this time.

**Misdiagnosis of Asperger’s Syndrome**

Asperger’s Syndrome had been misdiagnosed throughout the 1940s, and for several decades thereafter, as early infantile autism and schizophrenia (Frith, 1991). At other times, the AS child is first diagnosed with a nonverbal learning disability (McKelvey, Lambert, Mottson, & Shevell, 1995; Ozbayrak, 2004), a pragmatic language disorder (Attwood, 1998; Myles & Simpson, 2002), or a
semantic language disorder (Bishop, 1989; Brook & Bowler, 1992), or both. Some medical professionals have diagnosed the AS child with a personality disorder (Frith, 1991).

Wing (1981) was the first to publish a clinical report of the Asperger characteristics. Bowler (1992) felt that Wing’s account of the disorder gave the first systematic description of a child diagnosed with AS. It is due to the clinical report Wing published in England that the medical community began to accept Asperger’s Syndrome as a distinct diagnosis and sought to learn more about the characteristics of the disorder.

**Sensory Processing Difficulties and Asperger’s Syndrome**

Many researchers have identified that children with Asperger’s Syndrome have sensory processing difficulties as a specific characteristic (Asperger, 1944; Attwood, 1998; Barnhill, 2001; Gillberg & Gillberg, 1989; Mailloux, 2001). Though many researchers identify sensory processing difficulties as a major characteristic (Dunn, et. al, 2002; Falk-Ross, et. al, 2004; Iwanaga, et. al, 2000), it is still not used as a criterion for diagnosing the disorder. Klin, et al. (2000) discovered that subjects with AS had significantly impaired sensory processes.

Children with Asperger’s are particularly susceptible to having sensory responses that would not be perceived as normal (Attwood, 1998; Iwanaga, 2000). They are either hypersensitive or hyposensitive. AS children can have an extremely high pain tolerance (Attwood, 1998; Ayers, et. al, 2004) or express a need for the feeling of particular textures (Grandin, 1988; Ayers, et. al, 2004). As well, the AS child may only want to eat particular foods (Barron & Barron, 1992; Grandin, 1988) or shy away from certain smells and odors (Cesaroni & Garber, 1991; Grandin, 1988). Smith-Myles, Cook, Miller, Rinner, and, Robbins (2000) reflect that the child with Asperger’s also engages in self-stimulatory behaviors that limit him in his developmental skills. It is the limitations in sensory processing (Ayers, et. al, 2004; Grandin, 1990) of an AS child that does not allow the child to access maximal learning opportunities and delays the social development of the child.

**Sensory Integration**

Sensory Integration (SI) involves the stimulation of the neuronal response mechanisms of an individual to sensory stimuli with the expressed intent of maturizing this aspect of brain function (Ayers, et. al, 2004; Bundy, et. al, 2002). Neuronal responses can be processed through the five senses of the body or through the proprioceptive, vestibular, or tactile senses (Ackerman, 1990). Kranowitz, Sava, Haber, Martin, and Szklut (2001) explain the proprioception
sense involves position of the joints and contraction of the muscles. The vestibular sense involves the awareness of gravity and its force upon the body and the movements of the head (Kranowitz, et. al, 2001). The tactile sense involves sensory stimulations received through the skin (Barnhill, 2001; Dunn, et. al, 2002). Most often the senses work together to allow a person to function (Miller & Summers, 2001).

Proprioceptive Sense
Kranowitz and colleagues (2001) explains the proprioceptive sense involves position of the joints and contraction of the muscles. It is this sensory system that allows an individual to maintain his balance. This system is automatic for most individuals. For the majority of children diagnosed with AS, the proprioceptive sense does not occur automatically, but has to be taught (Anderson, 1998).

Vestibular Sense
The vestibular sense involves the awareness of gravity and its force upon the body and the movements of the head (Kranowitz, et. al, 2001). Myles and colleagues (2000) noted that most of the AS children they studied experienced dysfunction of the vestibular sense. Myles and colleagues (2000) also noted that the vestibular system “is involved in movement, posture, vision, balance, and coordination on both sides of the body” (p. 28). Individuals who experience vestibular dysfunction can be hyposensitive or hypersensitive in relation to this system (Kranowitz, 2006). Children who experience vestibular hyposensitivity are in a state of constant motion (Kranowitz, 2006). Children who experience vestibular hypersensitivity have difficulty with activities that require or involve movement. Asperger children can be either hyposensitive or hypersensitive in relation to the vestibular sense (Myles, et al., 2000).

Tactile Sense
The tactile sense involves sensory stimulations received through the skin (Barnhill, 2001). Asperger (1944; 1968; 1991) noted that his subjects had particular sensations to tactile stimulations. As with the vestibular sense, AS children can also be hyposensitive or hypersensitive (Koomar & Friedman, 1992; Kranowitz, et. al, 2001) in relation to the tactile sense. The tactile sense is a large sensory system to address, since sensations can be felt along all parts of the human’s skin. Temple Grandin (1990) states she experienced both extremes, being either tactile hyposensitive or tactile hypersensitive. Hyposensitivity and hypersensitivity both have a variety of problems that limit the AS child from being successful in school and socially (Ayers, et. al, 2004; Grandin, 1990; Kranowitz, et. al, 2001).

The AS child, who has tactile hypersensitivity, may actually feel pain when
receiving sensory stimuli (Ayers, et. al, 2004). Ayers and Mailloux (2004) refer to this condition as tactile defensiveness. Tactile hypersensitivity has negative impacts in the school and home setting for the AS child (Dunn, et. al, 2002; Falk-Ross, et. al, 2004; Fondacaro, 2001). The AS child socially isolates himself at school, so he will not have to take part in activities he knows will cause him distress and have other children react with negative social responses (Smith-Myles, et al., 2000). In the home environment, AS children may have preferences that affect their clothing, eating, sleeping, and bathing habits (Ayers, et. al, 2004; Kranowitz, 1998). Tactile defensiveness affects all aspects of the AS child’s daily functioning.

The AS child, who is tactile hyposensitive, is slow to respond to tactile stimulation (Grandin, 1990) and has a low tolerance for pain. The child with this specific type of sensory dysfunction may use the sense of touch to familiarize himself with his environment or to be aware of his presence in space. AS children with tactile sensitivity sensory dysfunction often seek tactile sensory stimulation.

Sensory Systems and Sensory Integration Processing

All people have specialized neural cells known as receptors that transport sensory information to the central nervous system (Miller & Summers, 2001). In 1993, Anzalone stated a Sensory Integration Program focuses on the stimulation of the neuronal responses of the tactile, vestibular, proprioceptive senses to stimuli. All behaviors of an individual are generated from the sensory stimuli a person receives and how the brain processes that information (Myles, et al., 2000).

The sensory systems of an individual must process sensory information correctly. All three of the sensory systems: tactile, vestibular, and proprioceptive, must work together for information to be integrated and interpreted (Kranowitz. 2006). Children who can readily process sensory information in an integrated fashion are able to benefit from maximal learning opportunities. Asperger's children are known for displaying varying types of sensory deficits (Dunn, et. al, 2002; Iwanaga, et. al, 2000), thus limiting their access to maximal learning opportunities and delaying their social development.

Sensory Integration Dysfunction

Just as Sensory Integration is the involvement of the stimulation of the neuronal response mechanisms of an individual to sensory stimuli (Ayers, et. al, 2004); Sensory Integration Dysfunction is the opposite of such (Ayers, et. al, 2004; Miller & Summers, 2001). Kranowitz (1998) defines Sensory Integration Dysfunction as “the inability to process information received through the senses”
(p. 8). Dr. Jean Ayers was the first to identify the problem of Sensory Integration Dysfunction. Ayers’ research concluded that the dysfunction occurs in the central nervous system when the body's sensory systems do not properly integrate sensory data. A child with Sensory Integration Dysfunction is unable to learn to his full potential and will experience delayed social development (Anderson & Emmons, 1996; Ayers, et. al, 2004). The child diagnosed with Asperger’s Syndrome needs to be taught to manipulate his sensory systems (Dunn, et. al, 2002; Falk-Ross, 2004) to maximize his learning potential and social development (Smith-Myles, et al., 2000).

Sensory Integration and Asperger's Syndrome

Sensory processing takes place for an individual when one is able to process and comprehend information received from the senses. A child with Sensory Integration Dysfunction (SID) may have difficulty limiting information extracted from incoming stimuli (Ayers, et. al, 2004; Fondacaro, 2001; Myles & Simpson, 2001). Asperger's children are known for displaying varying types of sensory anomalies (Myles & Simpson, 2002; Williams, 1995). Since all children with Asperger’s Syndrome demonstrate some degree of sensory deficits (Myles & Simpson, 2002; Ozbayrack, 2004), it may be assumed that all children with an AS diagnosis will have Sensory Integration Dysfunction. It is due to the sensory deficits that all AS children demonstrate that SI would be beneficial to them (Ayers, et. al, 2004; Miller & Summers, 2001).

Sensory Integration was first defined by Ayers (1972) as the communication and synchronization of two (+) functions in a method that improves the acclimation of the brain. Ayers (1979) was one of the first in the field of autistic studies to note the profound positive effects that Sensory Integration could have on an individual diagnosed with an autism spectrum disorder. Ayers (1989) notes that the reason autistic individuals engage in self-stimulatory behaviors is due to a deficiency in the body's ability to process sensory information. This delay is referred to as SID (Ayers, et. al, 2004). Mailloux (2001) noted stereotyped behaviors as indicative of AS characteristics and a symptom of Sensory Integration Dysfunction.

According to Sensory Integration theorists, sensory events that occur early in life affect the development of advanced level processing abilities of an individual (Ayers, 1972, 1979; Ayers, et. al, 2004; Fisher, Murray, & Bundy, 1991; Knickerbocker, 1980). Therefore, it can be noted that development of the sensory skills affects the entire sequence of normal developmental milestones. Difficulties with SI can impede the development of social skills, learning acquisition, and motor coordination (Dunn, et. al, 2002; Falk-Ross, 2003; Fondacaro, 2001). The delay in these key developmental milestones will impact the degree of a person's involvement in the routine of day-to-day living (Ayers, et. al, 2004; Fondacaro, 2001).
Asperger's Syndrome is a neuro-developmental disability that is distinguished by limited interests and social deficits (Volkmar & Klin, 2000). It is well documented that children with Asperger’s Syndrome display symptoms that are universal to other disabilities (Myles & Simpson, 2002). As a result, children with Asperger’s Syndrome are often misdiagnosed. Accordingly, further study to assist accurate diagnosis is necessary.

Sensory Integration (SI) involves the stimulation of the neuronal response mechanisms of an individual to sensory stimuli (Kranowitz, et. al, 2001). Neuronal responses can be processed through the five senses of the body or through the proprioception, vestibular, or tactile senses. All of the body’s senses will work together most of the time to allow a person to function.

Since all children with Asperger’s Syndrome have sensory deficits (Ayers & Tickle, 1980; Myles & Simpson, 2002), it may be assumed that all children with an AS diagnosis will have Sensory Integration Dysfunction (Ayers, et. al, 2004). Due to the sensory deficits that AS children demonstrate, a Sensory Integration Program would be beneficial to them (Ayers, et. al, 2004; Dunn, et. al, 2002; Lincoln, Courchesne, Harms, & Allen, 1995; Ornitz, 1989; Wainwright-Sharp & Bryson, 1993).

References


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