Autism and Infant Attachment:

A Review of the Literature

Anna Rooney

Psychology 340

Professor Pederson

November 28, 2005

A Review of the Literature

Even when Stephen Bohay was just a few months old, his parents knew there was something odd about him. Instead of developing the normal one consonant/one vowel sounds characteristic of three to eight month infants, Stephen remained silent and, according to his mother, never cuddled, never wanted to be picked up, and "never looked at [her]—ever" (Holloway, 1981). Finally, when he was three years old, a psychiatrist diagnosed Stephen with autism. At that age he "wouldn't show any affection or love" and it "seemed to annoy him when [his mother] spoke to him" (Holloway, 1981). Then suddenly Stephen went through what his mother called a second baby-hood at six years. He started being very loving and affectionate: holding his arms out to her to be held, sitting on her lap, and wanting to be cuddled. It was only then Mrs. Bohay could say, "I really felt he loved *me*" (Holloway, 1981).

Since its discovery in 1943 by Leo Kanner, autism has puzzled neurologists and psychologists alike. While there have been strides in the early detection and biological understanding of the disorder, the inner and social worlds of autistic individuals remain a mystery due to the large range of cognitive, verbal, and social skills in the autistic population. However, as Kanner stated, the one common denominator is social withdrawal (Holloway, 1981). Because this withdrawal is so characteristic, as is the lack of affect described in the case of Stephen, one question that arises is whether autistic infants are able to form the attachments to their caregivers John Bowlby described as so critical for healthy social development (Bowlby, 1988). Does the nature of the disorder prevent such a reciprocal relationship from building or at least postpone it, as seems to be the case with Stephen and his mother? The following paper will present the information thus far known about the symptoms and neurological aspects of

autism that may hinder attachment and actual attachment patterns in autism as well as take a critical look at applying the Ainsworth categorization of attachment to these infants.

An Introduction to Autism

Although autism is commonly believed to be rare, actual estimated rates in children in the United States alone range from 7.8 to 40.5 per 10,000 (Goode, 2004), making it more common than both Down Syndrome and childhood cancer. In the past decades, the rate of diagnosis has increased dramatically which some experts attribute to an actual increase in cases due to environmental factors but other experts attribute to better public awareness and means of diagnosis and a wider definition of autistic disorders (Goode, 2004).

Doctors can now detect the signs of autism earlier in a child's life and parents often alert their doctors quickly when they observe slower than normal development. As infants, autistic children refuse physical contact and to sit on their parents' laps, pull away from being held, are stiff when held, avoid eye contact, fail to respond to their parents' voices, demonstrate undifferentiated smiling, lack social and emotional reciprocity, and often show developmental delays in verbal skills (American Psychiatric Association [APA], 1994; Hamblin, Buckholdt, Ferritor, Kozloff, & Blackwell, 1971, p. 217-218). However, it should be noted that these symptoms may be lacking in infancy. An infant or toddler may develop normally for up to two years and suddenly loose social and language skills they have thus far achieved (APA, 1994). As the autistic child continues to grow, he or she continues to display the above symptoms as the disorder manifests itself in new ways. He or she fails to develop appropriate peer relationships, does not display spontaneous interest in things by bringing them to his or her parents or pointing to them, lacks or has impaired symbolic play, exhibits echolalia or other unoriginal speech (if speech is present), refrains from holding or initiating conversation, remains preoccupied with

very specific objects or parts of objects for extended periods of time, resists change, is highly sensitive to auditory and visual stimuli, and displays stereotypies (repetitive motor mannerisms such as rocking or hand flapping) or hyperkinesis (APA, 1994; Seifert, 1990). In older ages, autism is generally accompanied by an associative diagnosis of mental retardation within a moderate IQ range of 35-50 (APA, 1994). However, some autistic individuals demonstrate savant-like skills in which they have markedly better, sometimes incredible, talent in specific areas (Carlson, 2005, p. 497; Holloway, 1981).

Although Kanner first described autism as having a biological origin (Carlson, 2005, p. 498), as is now widely accepted, it was once theorized that autism is caused by refrigerator parents, or by a lack of parental love and care. After witnessing the lack of affect and withdrawal exhibited by prisoners in German concentration camps that reminded him of autistic children, Bruno Bettelheim proposed that autistic symptoms were actually a coping mechanism to deal with the horrible emotional pain children endured at their parents' hands (Carlson, 2005, p. 498; Seifert, 1990). According to Bettelheim (1967), "autism has essentially to do with everything that happens from birth on" (p. 393). Despite proof otherwise, it was said that the autistic child is born with normal intelligence and the ability to communicate, but chooses not to in order to defend him or herself from future trauma (Seifert, 1990).

However, due to the lack of evidence pointing towards the refrigerator mom theory (personal communication, November 5, 2005) and scientific advances, autism is now considered a biologically based developmental disorder (Seifert, 1990). It is often defined as "a sign of central nervous system damage, in which withdrawal and affectlessness are secondary symptoms" (Seifert, 1990, p. 5). While the actual causes of the disorder are still unknown, all hypothesized causes are biological in nature. Strong evidence suggests a genetic role. Ritvo and

Freeman found an extremely high concordance rate in monozygotic twins (70%) with absolutely no concordance rate in dizygotic twins (Seifert, 1990). Furthermore, according to Carlson (2005, p. 499), 2-3% of the siblings of autistic individuals are also autistic. Prenatal biological influences such as rubella, thalidomide use during the first trimester, and the herpes virus were also found to correlate with autism as well as tuberous sclerosis (APA, 1994; Carlson, 2005, p. 499). The DeLong and Rapin studies (as cited in Carlson, 2005) suggest that approximately 20% of all cases of autism have a traceable biological cause.

Possible Hindrances to Attachment

As mentioned above, the Bettelheim hypothesis failed to produce any evidence indicating an environmental cause of autism. In fact evidence was found that demonstrated that parents with autistic children did not differ greatly in parenting styles or mental health than parents of normally developing children. Cox, Rutter, Newman, and Bartak (as cited in Carlson, 2005 and Seifert, 1990) demonstrated that parents with autistic children do not differ from parents with dysphasic children in early stress events— such as psychosis, illness, or financial strain— and more importantly in emotional responsiveness and sociability. Similarly, DeMyer, Barton, and Norton (as cited in Seifert, 1990) conducted interviews with parents of normally developing children and parents of autistic children. Assessing variables such as infant acceptance, warmth, nurturing, feeding patterns, and physical arousal, results indicated no differences in these two groups. It should be noted, however, that Massie (as cited in Seifert, 1990) measured the amount of eye contact and touch demonstrated by mothers of "mixed-type child psychotics" to be inadequate in her 1978 study using home movies of children younger than six months. But due to the lower correlations and variations in portions of the film, the psychiatric community deemed her methods (and thus results) questionable (Seifert, 1990).

As in all studies of parent-child dyads, the parent's personality is a subject of analysis for attachment theorists. While Emde and Brown (as cited in Watkins, 1987) note that if a mother with a strong desire to maintain control and order may feel helpless in face of disorders such as Down Syndrome, McAdoo and DeMyer (as cited in Seifert, 1990) found that parents with autistic children did not exhibit any striking pathological personality traits. Using the Minnesota Multiphasic Personality Inventories (MMPIs), they compared the parents of autistic children to parents of normally developing children being treated at an outpatient psychiatric clinic and failed to find any statistically significant similarities (Seifert, 1990).

However, these studies were not conducted during the infant's first few months of life. It is likely that feelings towards the child and parenting during those months were more withdrawn and negative. Instead of filling the baby book with pictures of their young child hugging his father and looking at his mother's eyes,' the parents of autistic children have a "distortion of [their] hoped-for child" (Watkins, 1987, p. 119) that they may feel ashamed of or simply depressed by. So strong are these feelings that Olshansky (as cited in Watkins, 1987) characterizes a parent's reaction to the mental retardation of their child as one of "chronic sorrow." Accompanying their mourning is the realization that their autistic child will always need assistance, which can lead to feelings of anger and resentment. According to Drotar, Irvin, Kennell, and Klaus (as cited in Watkins, 1987), a parent goes through five stages of coming to terms with and then attaching to their disabled infant. The first three stages are of particular interest when considering infant attachment: (1) "profound shock and disruption of normal emotions" in which the parent may consider leaving his or her child and the problem behind; (2) "a period of denial;" 3) "feelings of sadness, anger, and anxiety about the infants' condition" which can target a spouse or medical staff (Watkins, 1987, p. 119). If a caregiver feels as if he or

she is suddenly thrown into a world of confusing emotions, their ability to tend to the most basic physical, nonetheless emotional, needs of their infant could be adversely affected. Considering that these ideas of desertion and periods of anger may last for months, it seems highly probable that the caregiver's initial reactions may cause lasting affects on his or her relationship with their child and secure attachment hindered despite future appropriate care and support.

In addition, autistic disorders are marked by symptoms that may limit attachment. Watkins (1987, p.121) explained that "[a Down Syndrome] baby's placid nature, inability to express needs normally, infrequent crying, and poor quality of eye-contact all interfere with the reciprocity of early interactions." Because autistic infants also demonstrate "placid nature, inability to express needs normally, infrequent crying, and poor quality of eye-contact," Watkins' assertion applies to them as well. Siegel further emphasizes the importance of infant activity:

> Parents attune to the subtle changes in a baby's state of arousal, not merely the categorical affect that the infant may or may not be expressing. In fact, the expression of internal state through vitality affects is the primary mode of communication between an infant and a caregiver during the early years of life. These affective expressions reveal the profile or energy level of the state of mind at a particular moment. The profile contains within it a picture of how the individual's internal state is being expressed in changing state of activation of the face, motion in the body, and tone and intensity of the voice. Vitality affects reveal aspects of the primary nature of emotion—the changes in the system's state of arousal. (1999, p. 129)

Considering that the most dominate manifestations of autism (failure to respond to parent's voices or faces, looking away when held, resisting touch all together, avoiding eye contact, and

smiling at everyone—not just the caregivers) restrict exposure of overall arousal as well as produce an appearance of affectlessness, autistic infants inhibit this primary mode of communication. Thus the caregiver may halt any of their behaviors that would normally activate the infant's attachment system and thereby frustrate the natural formation of attachment.

At this point it is important to include evidence from Capps, Kasari, Yirmiya, and Sigman (1993) indicating that autistic individuals are not affectively flat, but display socially inappropriate extremes of emotions. Capps et al. conducted two separate studies (one with mentally retarded autistic children and the other with autistic children with IQs of 75 or higher) in which emotional expressiveness was examined by parental reports and experimental situations designed to elicit empathy. Contrary to both DSM-IIIR (the diagnostic manual used at the time of the study) and popular characterization of autism, the autistic individuals did not display less emotion or facial expression, but reacted more intensely than the normally developing subjects (Capps et al., 1993). However, the subjects used in the Capps et al. studies were not infants and thus do not have any bearing on infant attachment.

Recent findings indicate that autism may also inhibit the ability to recognize emotions, especially through facial expressions (Downs & Smith, 2004). Using a group of highly functioning autistic boys (with normal IQs) who had received intensive behavioral treatment, a group of boys diagnosed with either Attention-Deficit/Hyperactivity Disorder (ADHD) or Oppositional Defiant Disorder (ODD), and a control group of undisturbed boys, Downs and Smith (2004) studied emotional understanding. Five different levels were examined: (1) emotional facial expression recognition in photographs, (2) emotional facial expression recognition in schematic drawings, (3) situational emotional recognition in drawings, (4) recognition of emotions based on desire and (5) recognition of emotions based on belief. The

autistic group performed significantly worse than both the ADHD/ODD group and the control group on the level one tasks, indicating deficits in recognizing facial expressions (Downs & Smith, 2004). Because facial expressions make up a critical portion of social communication, it is clear how such a deficit impacts social understanding and relationship formation.

Clearly the behavior of an autistic child could inhibit attachment with his or her parent, but neurological science indicates that not all the factors that effect the ability to form attachment are behavioral—some are biological. The literature demonstrates an unquestionable confidence in a biological basis for the disorder. For example, Dawson, Meltzoff, Osterling, and Rinaldi (1998) write, "Indeed, autism most certainly involves dysfunction of brain circuits that support the functioning of a variety of brain regions" (p. 1276). One such dysfunction may have caused the results of Downs and Smith's aforementioned study of emotion recognition. Schultz, Gauthier, Klin, Fullbright, Anderson, Volkmar, Skuldarski, Lacadie, Cohen, and Gore as (as cited in Dawson et al., 1998) demonstrated that the neural processes active in highly functioning autistic individuals while looking at faces correlates to feature-based processing instead of holistic processing. When looking at a face, people with autism focus on one facial feature (like the nose) instead of looking at the whole face for information, making it difficult for them to identify emotions—as seen in Downs and Smith's study. This contributes to the hypothesis (Downs & Smith, 2004; Holloway, 1981) that autistic children fail to recognize humans as more important than other things in their environment.

The prevailing theory of autistic neurology identifies dysfunction of the limbic system as highly associated with occurrence of the disorder (Dawson et al., 1998). In 1994, Bachevaliar (as cited in Dawson et al., 1998) found that lesions in the limbic structures, the amygdala and hippocampus, resulted in long lasting serious cognitive and social impairments, as well as

repetitive behaviors. Bauman and Kemper's discovery of reduced neuron size and increased cell packing (as cited in Dawson et al., 1998), indicates further the abnormality in the limbic structures of autistic individuals.

Even more revealing, however, is the work done by Dawson, Meltzoff, Osterling, and Rinaldi (1998). They compared the severity of autistic symptoms, medial temporal lobe and orbital prefrontal cortex functioning, and dorsolateral prefrontal cortex functioning of children with autism, Down Syndrome, and normal development (Dawson et al., 1998). Testing of the dorsolateral prefrontal cortex was included in addition to the limbic structures because it is associated with working memory and higher cortical functioning, both of which have marked deficiency in autism. Medial temporal lobe and orbital frontal cortex functioning was assessed by a delayed response test in which the subject was shown an object that, when moved, would reward him with a piece of food. After a short delay, the subject was shown the sample object again, but it was paired with an unmatched novel object and only the movement of this novel object would produce the reward. Criterion performance was reached when the child reached for the new object first in five consecutive trials. Meanwhile the dorsolateral prefrontal cortex activity was measured by a delayed response test in which the experimenter showed the subject a toy and then hid it in a container. During a five second delay, a screen was lowered between the experimenter and the subject while the experimenter placed a second identical container on the table opposite from the original container with the toy in it. The child was then permitted to search for the toy. After successfully searching in two consecutive trials, the side of the table the correct container was on was reversed. The severity of the autistic symptoms was measured using a variety of tasks in addition to a Wing social subgroup classification system.

Dawson et al. (1998) determined that autistic children performed significantly lower on the delayed non-matching to sample test than both the Down syndrome and control groups (only 61% of the autistic group reached criterion performance while 100% of the control group did) and that such low performance was correlated to severity of symptoms and Wing subgroup classification. Meanwhile, although the autistic group performed significantly worse on the delayed response test also, these results did not correlate with severity of symptoms or Wing subgroup classification. Thus, Dawson et al. (1998) concluded that the manifestation and strength of early autistic symptoms may be associated with medial temporal lobe and orbital frontal cortex functioning which may, in turn, inhibit the development of social and motor skills. The lack of these skills may explain the main manifestations of early childhood autism. As explained by Siegel (1999, p. 131), malfunctioning of the medial temporal lobe structures (such as the amygdala) may result in social development delays because the social appraisal process is restricted or fully inhibited. Siegel also suggests (1999, p. 138) that there may be a cluster of cells that fire in response to eye contact and facial expressions in the amygdala and orbital frontal cortex. Since Dawson et al. demonstrated that these areas are malfunctioning in autistic children, perhaps the cluster of cells described by Siegel are damaged or nonexistent, causing the limited eye contact and deficits in facial emotion recognition.

While the evidence of a neurological basis for the symptoms of autism is undeniable, it does not necessarily indicate a causal relationship. Instead, it reveals the reciprocal effects of neurology and experience. As Pipp-Siegel, Siegel, Clifford, & Dean describe (1999),

> Interactions between infants and parents may be influenced by the neurological condition of the infant and these interactions may influence the development of the immature neurological system. The resulting neurological impairment may then have further

influences on parental interactions that, in turn, sustain a particular pathway of neurological functioning. (p. 43)

Actual Attachment Patterns in Autism

With all these possible limitations on attachment, it seems highly improbable that autistic individuals are able to form attachments at all, especially during infancy. However, evidence indicates that autistic children do form attachments (albeit delayed) and these attachments may be just as secure as those of normally developing children.

Silencing assertions of autistic attachment ineptness, Marian Sigman and Judy A. Ungerer published the results of a comprehensive study in 1984. They compared the social behaviors and cognitive functioning of fourteen autistic children who had achieved object permanence and a control group of fourteen normally developing children with a mean mental age of 24.1 months. During the months leading up to the study, the autistic children stayed in hospitals with only limited contact with their parents. Sigman and Ungerer conducted the study in two phases (a play session and separation episodes) in a laboratory room with two chairs in diagonal positions and a variety of toys. In the play session the experimenter first modeled four different symbolic acts with the toys in an attempt to initiate symbolic play and then left the child to play freely with the toys for sixteen minutes. Neither the experimenter nor the caregiver initiated contact with the infant while the experimenters recorded the types of play as either functional (normal use of objects in a useful way) or symbolic (using objects as different objects, using inanimate things to be independent beings, or pretending objects without any physical existence are present in the room). Sigman and Ungerer's separation episodes were actually Mary Ainsworth's Strange Situation with modifications. The infamous Ainsworth Strange Situation is a laboratory experiment conducted to evaluate the attachment between infant and

mother as secure, anxious- ambivalent, anxious- avoidant, or disorganized/ disorientated. The infant is separated and then reunited with their mother twice after being at first alone and again later with a stranger. During all of Sigman and Ungerer's separations the child was never left alone due to ethical concerns. Before the episodes, the experimenter was changed to ensure the stranger was truly foreign to the child. Each separation episode lasted two minutes and was followed by two minutes of reunion.

Sigman and Ungerer (1984) found that autistic children exhibited more proximity-seeking during the two minute reunion with their mothers, averaging 25 additional seconds directed toward the mother and 0.6 seconds less towards the stranger. Contrastingly, the autistic group did not change their behavior during the absence of the stranger and sought out their mother more once the stranger returned. Because attachment behaviors were restricted to the separation and reunion with the mother, it is clear that the autistic child can differentiate his or her mother from others and may be attached to her. Given the long periods of hospitalization, the evidence is even more convincing that autistic children form strong attachments (Sigman & Ungerer, 1984). Meanwhile, the play sessions provided another insight: preferential social interaction (the difference between total social behaviors directed at the mother and those directed at the stranger) correlated to duration and diversity of symbolic play in autistic children. Although the autistic group demonstrated less symbolic play than the control group, the longer and more variedly they played symbolically, the more the child interacted with his mother. Inversely, the control group demonstrated a negative correlation between symbolic play and proximity-seeking directed towards the mother: "Unlike the autistic children, more sophisticated representational skills were associated with the demonstration of fewer attachment behaviors during reunification in the normal group" (Sigman & Ungerer, 1984, p. 241). The difference in

correlation suggests that higher levels of cognitive skills may be required for autistic attachment formation. Object permanence clearly is not sufficient since all the autistic subjects had obtained object permanence but only half of them demonstrated clear attachments. If autistic children "require higher levels of representation than normal children in order to demonstrate attachment responses" (Sigman & Ungerer, 1984, p. 242) it is understandable that these children do not exhibit attachment behaviors during infancy—no infant has the ability to play symbolically.

The question remains as to what type of attachment these older autistic children form with their caregivers. Using the Ainsworth categories of attachment, van IJzendoorn, Schuengel, and Bakersmans-Kranenburg (as cited in Pipp-Siegel, et al., 1999), found a significantly high percentage of disorganized/disoriented (D) attachment in autistic and Down Syndrome children—35% as compared to the accepted norm of 15%. In another study by Capps et al. (1994) all autistic subjects were assigned to the D category. However, these results came under heavy fire from the scientific community because of the questionable appropriateness of the Ainsworth categorization in describing the attachment between caregiver and autistic child (Pipp- Siegel, et al., 1999). Numerous behavioral manifestations of autism appear to be signs of anxious attachment (for example, resistance to being held or cuddled and eye contact avoidance) and even indications of disorganized/disoriented attachment (such as stereotypies and disorganized wandering). Realizing that many of the behaviors they used to assign the autistic subjects to the D category are characteristic of autism, Capps et al. reanalyzed the assessments and excluded stereotypies. After doing so only three of the fifteen autistic children were classified as truly having disorganized/disoriented attachments (Pipp- Siegel, et al., 1999).

Implications of the Research

While the research is in its beginnings, a diagnosis of autism does not necessarily equate with an attachment-less life. Considering the possible adverse reaction of the parent, the prevalence of symptoms that contribute to a lack of emotional reciprocity, and neurological malfunctioning in areas critical in relationship formation it seems hopeless for a parent to attempt to form a secure attachment with their autistic child. Yet, as Stephen Bohay demonstrates, a parent should not desert their dreams of eventually forming a close, happy relationship with their child. Autistic children can and do form attachments to their parents and these attachments are just as likely to be secure as the attachments of normally developing children. However, these attachments generally have a late onset due to an apparent need for higher mental representational skills. But once these skills develop, it is quite possible that the parent of an autistic child may be able to exclaim, "I really felt he loved *me*" (Holloway, 1981) also.

References

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders Forth Edition*. Washington, DC: Author.

Bettelheim, B. (1967). *The Empty Fortress*. New York: Macmillan Publishing Co., Inc.

Bowlby, J. (1998). *A Secure Base*. New York: Basic Books, Inc.

Capps, L., Kasari, C., Yirmiya, N., & Sigman, M. (1993). Parental Perception of Emotional Expressiveness in Children With Autism. *Journal of Consulting and Clinical Psychology, 61*, 475-484.

Carlson, N. R. (2005). *Foundations of Physiological Psychology*. Boston: Pearson Education, Inc.

Dawson, G., Meltzoff, A. N., Osterling, J., & Rinaldi, J. (1998). Neuropsychological Correlates of Early Symptoms of Autism. *Child Development, 69*, 1276-1285.

Downs, A. & Smith, T. (2004). Emotional Understanding, Cooperation, and Social Behavior in High-Functioning Children with Autism. *Journal of Autism and Developmental Disorders, 34*, 625-635.

Goode, E. (2004, January 26). More and More Autism Cases, Yet Causes Are Much Debated. *The New York Times*, p. A1.

Hamblin, R. L., Buckholdt, D., Ferritor, D., Kozloff, M., & Blackwell, L. (1971). *The Humanization Processes: A Social, Behavioral Analysis of Children's Problems*. NY: John Wiley and Sons, Inc.

Holloway, C. (producer). (1981). *The Autistic Child* [motion picture]. San Diego: University Media.

Pipp-Siegel, S., Siegel, C. H., & Dean, J. (1999). Neurological Aspects of the Disorganized/Disoriented Attachment Classification System: Differentiating Quality of the Attachment Relationship from Neurological Impairment. *Monographs of the Society for Research in Child Development. 64*(3), 25-44.

Seifert, C. D. (1990). *Theories of Autism.* Lanham, MD: University Press of America.

Siegel, D. J. (1999). *The Developing Mind.* New York: The Guilford Press.

Sigman, M., & Ungerer, J.A. (1984). Attachment Behaviors in Autistic Children. *Journal of Autism and Developmental Disorders, 14(3),* 231-244.

Watkins, K. P. (1987). *Parent Child Attachment: A Guide to Research*. New York: Garland Publishing, Inc.