Autistic Disorder: A Neuropsychological Enigma

Ruth A. Huebner

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Autism is increasingly viewed as an expression of an unidentified neurological disorder. Because understanding of neurological dysfunction is basic to evaluation and treatment in occupational therapy, this article provides a comprehensive and critical review of the literature since 1985 concerning the neuropsychology of autistic disorder. The research is categorized into four basic types: (a) neuropsychological testing of functional abilities, (b) treatment studies based on neuropsychological hypotheses, (c) autistic-related diseases and genetic disorders, and (d) neuroanatomical and neurophysiological studies. The research shows a spectrum of neurological impairments within the brain stem, cerebellum, midbrain, and frontal lobe. These impairments are associated with deficits in socioemotional skills, sensory processing, motor planning, and cognitive flexibility. This research suggests that persons with autistic disorder need evaluation and treatment of a wide spectrum of functional deficits.

Ruth A. Huebner, MS, OTR, is Associate Lecturer, Department of Therapeutic Science, University of Wisconsin–Madison, 1087 Medical Science Center, 1300 University Avenue, Madison, Wisconsin 53706–1532.

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Autism is a syndrome of complex brain dysfunction expressed in a spectrum of functional deficits. Extensive neuropsychological research that can expand understanding of the treatment of persons with autism is published every year. As Clark (1983) submitted, "Intermeshing of occupational therapy and neurobiology is not a new idea, but when considered in relation to the syndrome of autism, the two disciplines would appear to be natural allies" (p. 17). Because occupational therapists use neurorehabilitation approaches to assessment and treatment, it is crucial that they sustain an understanding of prevailing neurological information. This article provides a comprehensive review of the literature since 1985 concerning the neuropsychology of autistic disorder. Nelson (1984) and King and Grandin (1990) have explored occupational therapy for children with autism in detail. The suggestions for occupational therapy included in this paper are intended to complement their work.

The population of people with autistic disorder is heterogeneous and identified only through clinical judgment. This ambiguity complicates generalization or even comparison of research evidence. In addition, diagnostic criteria and names have changed since Kanner (1943) first described autism from his study of 11 children. Neither the first nor second edition of the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1955, 1968) included autism; children were classified as having childhood schizophrenia. The Diagnostic and Statistical Manual of Mental Disorder (3rd ed.) (American Psychiatric Association, 1980) was the first to include infantile autism under a group of five overlapping pervasive developmental disorders. In 1987, the American Psychiatric Association published a revised edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R), which acknowledged only autistic disorder and pervasive developmental disorder.

DSM-III-R said 8 of 16 characteristics from three groups of criteria must be satisfied for a child to receive a diagnosis of autistic disorder. Under this criteria, a child must exhibit qualitative impairment in reciprocal social interaction and verbal and nonverbal communication and a markedly restricted repertoire of activities and interests. Within DSM-III-R criteria, there is no differentiation of autism associated with mental retardation, genetic disorders, diseases, neglect, or abuse. Thus a child with an autistic disorder may have related complications such as neurological, medical, or psychological disabilities that are not defined in the research samples.

Authors included in this review have dealt with this diagnostic ambiguity and confusion in several ways. Gillberg, Steffenburg, and Jakobsson (1987) described a primary, or Kanner-type, autism as autism without complications in persons with IQs above 70 on either full-scale verbal or performance IQ measures. But discrepancies between standard verbal and performance IQ scale scores...
are common in the autistic population (Kaufman, 1990), which complicates research comparison. In contrast to primary autism, secondary autism is associated with other complications, such as mental retardation.

Although not recognized in the DSM-III-R, Asperger syndrome, or autistic psychopathy, is often described as a mild form of autism in children with higher levels of intelligence. Children with Asperger syndrome fail to adjust socially, are clumsy, lack adaptive social language, and often inadequately respond to nonverbal expressions of others. Because these children require treatment, Tantam (1988) argued that they should be identified on the autistic spectrum. Although the distinctions among autistic disorder, pervasive developmental disorder, and Asperger syndrome are unclear, children with Asperger syndrome are often included in research populations on autism (Tantam, 1988).

Some population statistics are helpful in understanding autism, which affects only 4 to 6.7 children per 10,000 (Gillberg, 1988). Gillberg et al. (1987) described 50% of children with autism as beautiful, with a distinctive, intriguing aloofness; this compelling nature of children with autism is seldom captured in the descriptive literature. In their extensive review of the literature, Rumssey and Hamburger (1988) noted that 50% of children with autism fail to develop communicative speech, and 25% to 30% develop seizures by adulthood. Young, Newcorn, and Leven (1989) found that 70% to 80% of children with autism are mentally deficient. The ratio of boys to girls with autism is generally thought to be between 4:1 and 5:1 (Gillberg, 1988; Rapin, 1988). Only 33% of males with autism have IQs below 50, whereas nearly 85% of females with autism score below 50 (DeLong & Dwyer, 1988). Thus girls with autism are likely to have increased neurological and intellectual disorders. At puberty, possibly because of hormonal changes, the condition may worsen in both boys and girls with the onset of seizures or behavioral disturbances (Prior, 1987). Approximately 60% of children with autism remain severely disabled throughout life, although 35% show fair to good adjustment in some sheltered life-styles (Lovass, 1987). A few develop special talents and adjust well (Rutter, 1970).

Although semantic confusion and complexity permeate the study of autism, Maurer (1986) proposed a research hierarchy model in which (a) the population is described, (b) clinical abnormalities are mapped onto biological models, and (c) experimental research is initiated. The study of autistic disorder is still in the stage of linking clinical abnormalities with biological models. Occupational therapists could collaborate with neuropsychologists to link clinical and biological models.

Neuropsychological Testing of Functional Abilities

Because authors who study neoanatomy or neurophysiology often base their research design on the results of neuropsychological testing, this literature is examined first.

The data collected from many of the human research articles included in this paper are summarized in Tables 1, 2, and 3. These figures were generated to facilitate research comparisons and to provide concise references. Information from these figures is discussed in detail below. Those studies that carefully selected their subjects to diminish the effects of complications (such as seizures or retardation) were judged to be well designed. Unfortunately, much of the research is flawed by mixed or vague diagnostic criteria; these limitations have been noted in the figures. Because of diagnostic confusion, limited numbers of subjects with autism, and strong interest in research, weak research design is common in the literature on autistic disorder (Lord, 1991).

Social Affective Deficits

Several authors (Fein, Pennington, Markowitz, Braverman, & Waterhouse, 1986; Hobson, 1986; Leslie & Frith, 1990; Reed & Peterson, 1990; Temple & Vilarroya, 1990) have argued from multiple viewpoints that the underlying deficit of autistic disorder is either primarily cognitive or socioaffective in nature.

Hobson (1986) compared 23 children with autism to 23 nondysfunctional children and 11 children who were nonautistic but retarded, to determine if they could recognize emotions associated with facial expressions. All three groups successfully matched meaning and emotion with corresponding objects and sketched facial expressions, but the children with autism failed to perceive the meaning of expressed emotion presented in a dynamic videotape. The subjects with autistic disorder had difficulty recognizing emotional gesture, vocalization and tone, and context of emotional expression. Hobson concluded that this was due to difficulties in judging momentary and dynamic configurations of emotion, an affective disorder. Fein et al. (1986) maintained that social aloofness is fundamental in autism because it is rare (even in severely brain-damaged infants), resists treatment, and ultimately obstructs cognitive development.

In contrast, Baron-Cohen (1991) and Leslie and Frith (1990) argued that children with autism have a primary cognitive disorder characterized by an inability to establish a theory of mind. In Leslie and Frith's 1990 study of social situations, children with autism performed poorly compared with both nondysfunctional children and children with Down syndrome on social tasks that required taking into account someone else's knowledge or beliefs. This theory of mind comprises aspects of empathy, role taking, and predicting another's behavior. Children with autism did not demonstrate metarepresentation, or the ability to view a situation in alternative ways, particularly from another person's point of view. Lack of metarepresentation impairs a child's ability to engage in imaginary play (Baron-Cohen, 1991; Leslie & Frith, 1990). Further-
more, children with autism had a poverty of language related to another's beliefs or mental states, thus prompting these authors to argue that cognitive impairment hinders social interaction.

Reed and Peterson (1990) integrated the social and cognitive elements of the social deficits seen in those with autistic disorder by discriminating between visual and cognitive perspective taking. Visual perspective taking, which involves visual spatial identification of objects, was adequate for those with autism. However, cognitive perspective taking, which involves aspects of social empathy and cognitive adaptability, was a difficult task for such persons. Moreover, the autistic and nonautistic perspective of what is rewarding may vary. Koegel, Dyer, and Bell (1987) found that allowing children with autism to have choices and to initiate activities in play therapy greatly decreased their social avoidance behaviors. They suggested that sharing control with the child results in improved motivation and decreased social avoidance that may outlast treatment.

Social abilities are complicated skills, integrating many aspects of learning, behavior, and emotion. Because the inability to use flexible abstract thinking in particularly ambiguous situations may lead to a lack of understanding of complex emotional expressions, persons working with children with autism may need to exaggerate and maintain their facial expressions, especially during early learning. Grandin and Scariano (1986) described...
Table 2
Results of Neuroanatomical Research Studies

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>Subjects</th>
<th>Control Group</th>
<th>Method</th>
<th>Result A</th>
<th>Result B</th>
<th>Nervous System Deficit</th>
<th>Research Design</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holroyd et al. (1991)</td>
<td>2</td>
<td>DSM-III-R: Joubert syndrome</td>
<td>Non-disabled MRI; midsagittal</td>
<td>Agenesis of cerebellar vermis</td>
<td>Enlarged 4th ventricle</td>
<td>Cerebellum</td>
<td>1 Subject MR Joubert syndrome</td>
<td></td>
</tr>
<tr>
<td>Garber et al. (1989)</td>
<td>15</td>
<td>DSM-III-R: grouped by IQ</td>
<td>15 Nondisabled MRI; midsagittal</td>
<td>No difference</td>
<td>1 Nondisabled had small ventricle</td>
<td>No result</td>
<td>Well designed; precise measure</td>
<td></td>
</tr>
<tr>
<td>Murakami et al. (1989)</td>
<td>10</td>
<td>DSM-III-R: neurological disability</td>
<td>8 Nondisabled MRI; midsagittal</td>
<td>Small cerebellar hemispheres</td>
<td>Hypoplasia of lobules VI and VII</td>
<td>Cerebellum; lobe VI and VII</td>
<td>Well designed; precise measure</td>
<td></td>
</tr>
<tr>
<td>Bauman &amp; Kemper (1988)</td>
<td>3</td>
<td>Case history documentation</td>
<td>Nondisabled Autopsy all sections</td>
<td>Hippocampal &amp; amygdala abnormalities</td>
<td>Fewer Purkinje cells</td>
<td>Midbrain; cerebellum</td>
<td>MR population included</td>
<td></td>
</tr>
<tr>
<td>Courchesne et al. (1988)</td>
<td>18</td>
<td>DSM-III-R: VIQ 77; healthy</td>
<td>12 Nondisabled; 10 with central nervous system disorder MRI; midsagittal</td>
<td>Vermal lobules VI and VII smaller</td>
<td>CNS disorders; mix pathology</td>
<td>Cerebellum</td>
<td>Well designed</td>
<td></td>
</tr>
<tr>
<td>Jacobson et al. (1988)</td>
<td>9</td>
<td>DSM-III-R: IQ &lt; 70; healthy adults</td>
<td>13 Nondisabled CT scan all areas</td>
<td>Enlarged 3rd ventricle</td>
<td>Abnormal caudate nuclei</td>
<td>Midbrain; limbic lobe</td>
<td>Well designed</td>
<td></td>
</tr>
<tr>
<td>Rumsey &amp; Hamburger (1988)</td>
<td>10</td>
<td>DSM-III-R: MR adult males</td>
<td>10 Nondisabled CT scan; PET; glucose</td>
<td>No localized abnormalities</td>
<td>Other cognitive disorders</td>
<td>Frontal lobe</td>
<td>Well designed; high IQ levels</td>
<td></td>
</tr>
<tr>
<td>Gaffney et al. (1987)</td>
<td>13</td>
<td>DSM-III-R: Aged 5–22 years mean FSIQ 84.9</td>
<td>35 Aged 4–19 years MRI; midsagittal</td>
<td>Trend to small cerebellum</td>
<td>Enlarged 4th ventricle</td>
<td>Structures near 4th ventricle</td>
<td>3 Subjects had central nervous system disorder</td>
<td></td>
</tr>
<tr>
<td>Gilberg et al. (1987)</td>
<td>20</td>
<td>DSM-III-R: &amp; 3 with Asperger syndrome and IQs &lt; 65</td>
<td>Comparison to nondisabled CT Scan; EEG</td>
<td>½ CT scan abnormal</td>
<td>½ EEG pathology</td>
<td>Many sites of deficit</td>
<td>Well designed; 3 Asperger syndrome subjects</td>
<td></td>
</tr>
<tr>
<td>Courchesne et al. (1987)</td>
<td>1</td>
<td>DSM-III-R: Non-VIQ 113</td>
<td>Nondisabled MRI; midsagittal</td>
<td>Small vermis &amp; cerebellum</td>
<td>R hemi larger; large ventricle</td>
<td>Cerebellum</td>
<td>Early MRI use; single subject</td>
<td></td>
</tr>
<tr>
<td>Ritvo et al. (1986)</td>
<td>4</td>
<td>DSM-III-R: deceased</td>
<td>3 Nondisabled male brains Autopsy cerebellum</td>
<td>Number of Purkinje cells reduced</td>
<td>1 of 4 had CNS pathology</td>
<td>Cerebellum</td>
<td>Included MR/CNS pathology</td>
<td></td>
</tr>
<tr>
<td>Bauman et al. (1985)</td>
<td>1</td>
<td>DSM-III-R: retarded</td>
<td>None Autopsy all sections</td>
<td>Increased cell density; cell size decreased</td>
<td>Limbic, cerebellum, &amp; sensory association</td>
<td>Midbrain</td>
<td>MR subject</td>
<td></td>
</tr>
</tbody>
</table>

Note. MRI = Magnetic resonance imagery. MR = Mental retardation. VIQ = Verbal IQ. CT = Computed tomography. PET = Positron emission tomography. FSIQ = Full-scale IQ. EEG = electroencephalogram. CNS = central nervous system. R = right.


Grandin's own struggle as an adult with autism learning social skills. To learn the subtle mechanics of body language and empathy, Grandin watched hours of emotional hyperboles on television soap operas. The research from neuropsychological testing suggests that occupational therapists may need to both direct and share control during a therapy session. They may need to teach pretending skills as well as skills in perceiving emotions and social responses. Occupational therapists may need to exaggerate their own emotions to teach a child through modeling and play.

Crucial questions remain. How do cognition, affect, social interaction, environment, neurobiology, and neurological structure and process dynamically interact in autistic disorder? Is the impairment in social skills, which is fundamental to autism, the result of cognitive inflexibility or a more subcortical impairment of sensory or emotional processing? Answers to these questions would help guide the focus and emphasis of occupational therapy intervention.

Arousal Deficits

Lovaas, Newsmom, and Hickman (1987) believed that autism results in a state of sensory deprivation because of the severe physical, behavioral, and interactive deficits.
inherent in the disorder. They studied self-stimulatory behavior in children with autism, which they theorized was a natural product of the children's attempts to maintain an optimal arousal state. Because self-stimulatory behaviors are consistent across cultures and even species, have a predictable developmental sequence, resist extinction, and persist without tangible or social reinforcers,Lovas et al. (1987) argued that these behaviors are maintained because of perceptual or internal reinforcement. They found that with treatment self-stimulatory behaviors became more sophisticated and complex—for example, singing or compulsively assembling puzzles replaced more primitive behaviors of hand flapping and rocking, but such behaviors did not disappear.

The intrinsic perceptual reinforcement of self-stimulatory behavior was demonstrated in studies (Lovas et al., 1987) that allowed time to engage in self-stimulation as a reward to shape alternative behaviors. Extinction of such behavior resulted in the development of a similar alternative self-stimulation pattern, and activities that simulated self-stimulation were effective in engaging a child's cooperation. Although the research of Lovas et al. (1987) was criticized for oversimplification and failure to consider the possible role of altered neurophysiology in self-stimulatory behavior (Lewis, Baumeister, & Mail, 1987), Lovas et al. (1987) offered excellent clinical descriptions of self-stimulatory behavior in the absence of definitive neurological data. King and Grandin (1990) offered another perspective, suggesting that stereotypic behaviors may be the result of the children's attempts to calm themselves or modulate their arousal levels during times of general overarousal.

Although the mechanisms for modulating arousal are not completely understood, these research and treatment hypotheses are consistent with other neuroanatomical and neurophysiological findings of brain stem, midbrain, and cerebellar sites of dysfunction. Conversely, as Peters (1986) suggested, stereotypies might be an extrapyramidal release symptom related to frontal lobe dysfunction.

Table 3

Results of Studies Using Physiological Research

<table>
<thead>
<tr>
<th>Author</th>
<th>N</th>
<th>Subjects</th>
<th>Control Group</th>
<th>Method</th>
<th>Result A</th>
<th>Result B</th>
<th>Nervous System Deficit</th>
<th>Research Design</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wong &amp; Wong (1991)</td>
<td>10</td>
<td>DSM-III-R*</td>
<td>36 With autistic-like characteristics 19 MR/20 nondisabled</td>
<td>Auditory evoked potential</td>
<td>Increased latencies</td>
<td>Increased brain stem transmission time</td>
<td>Brain stem</td>
<td>Stratified populations</td>
</tr>
<tr>
<td>Ciesielski et al. (1990)</td>
<td>10</td>
<td>DSM-III-R* mean 83</td>
<td>13 Nondisabled matched PIQ</td>
<td>ERP recorded auditory/visual stimuli</td>
<td>P&lt;0.01 smaller posterior area</td>
<td>NC absent frontal area</td>
<td>Complex network of attention</td>
<td>Well designed</td>
</tr>
<tr>
<td>Courchesne et al. (1989)</td>
<td>11</td>
<td>DSM-III* mean 78</td>
<td>9 Language disabled 16 nondisabled</td>
<td>ERP recorded auditory/visual stimuli</td>
<td>P&lt;0.01 smaller posterior area</td>
<td>NC response reversed pole</td>
<td>Attention &amp; cognitive response</td>
<td>Well designed</td>
</tr>
<tr>
<td>Heh et al. (1989)</td>
<td>7</td>
<td>DSM-III* no MR complications</td>
<td>8 Nondisabled</td>
<td>PET cerebellum glucose metabolism</td>
<td>Autistic subjects, not lower metabolism</td>
<td>Equal/greater metabolism</td>
<td>Inefficient processing</td>
<td>Excluded MR population</td>
</tr>
<tr>
<td>Dawson et al. (1988)</td>
<td>17</td>
<td>DSM-III* 8-19 years IQ 53-91</td>
<td>17 Nondisabled</td>
<td>ERP auditory</td>
<td>&lt;$P_2$ at right hemisphere</td>
<td>&gt;$P_1$ to phonetic stimuli</td>
<td>Attention to specific stimuli</td>
<td>6 Subjects MR</td>
</tr>
<tr>
<td>Horwitz et al. (1988)</td>
<td>14</td>
<td>DSM-III* VQ 40-117</td>
<td>14 Nondisabled age matched</td>
<td>PET scan glucose— all</td>
<td>Metabolism altered between frontal &amp; parietal lobes</td>
<td>Metabolism altered with subcortex</td>
<td>Complex network of attention</td>
<td>5 Subjects MR</td>
</tr>
<tr>
<td>Gillberg et al. (1987)</td>
<td>20</td>
<td>DSM-III* &amp; 3 with Asperger syndrome &amp; IQ &lt;65</td>
<td>Comparison to nondisabled</td>
<td>ERP brain stem spinal fluid</td>
<td>25% Prolonged transmission time</td>
<td>Elevated spinal fluid homovanillic acid, 5-hydroxyindoleacetic acid</td>
<td>Brain stem</td>
<td>Thorough study, well designed</td>
</tr>
<tr>
<td>Courchesne et al. (1985)</td>
<td>10</td>
<td>Specific criteria mean VQ 71</td>
<td>10 Nondisabled</td>
<td>ERP recorded auditory/visual stimuli</td>
<td>Accuracy same</td>
<td>Difference in ERP task specific</td>
<td>Limited orientation to novel</td>
<td>No DSM-III* criteria; well designed</td>
</tr>
<tr>
<td>Ornitz, E. (1985)</td>
<td>22</td>
<td>DSM-III* 29-71 months</td>
<td>25 Nondisabled age matched</td>
<td>Vestibular nystagmus</td>
<td>Prolonged nystagmus</td>
<td>—</td>
<td>Brain stem</td>
<td>MR population included</td>
</tr>
</tbody>
</table>

Note: MR = Mental retardation. VIQ = Verbal IQ. PIQ = Performance IQ. ERP = Event-related potentials. $P_1$ = Positive electrical potential over the parietal scalp. NC = Negative electrical potential over frontal scalp. PET = Positron emission tomography. $P_2$ = Positive electrical potential. Homovanillic acid and 5-hydroxyindoleacetic acid are products found in cerebral spinal fluid resulting from breakdown of dopamine, serotonin, and norepinephrine.

Vital questions remain: Are children with autism chronically overaroused, underaroused, or variable in their arousal levels? Does stereotypic behavior result from neurochemical or neuroanatomical deficits? Do these behaviors induce neurochemical changes or alter neurological development? Answers to these questions would provide crucial data about the sensory needs of those with autism as well as support for the premise that decreasing self-stimulation would enhance learning (Clark, 1983).

Attention and Memory Deficits

Surprisingly, I found few studies that placed primary focus on attention and memory deficits, even though concerns over attention were mentioned in numerous articles. Fein, Lucci, and Waterhouse (1990) found excessive fragmentation and overlap of forms in drawings made by 34 children with autism. Although geometric design fragmentation was found equally in the nonautistic and autistic samples, the autistic group (mean age 10 years, 11 months) displayed significantly more design overlap. Most notably, their drawings of human figures showed overlap and fragmentation. Fein et al. concluded that this reflected an obsessive attention to detail, an extreme narrowness of attention, and a lack of whole body concept more compatible with frontal lobe dysfunction.

Memory deficiencies have been used to explain the rigidity of autistic behavior, and the hippocampus-amygdala neuronal network has been implicated in this memory deficit. However, Ameli, Courchesne, Lincoln, Kaufman, and Grillon (1988) found that subjects with autistic disorder showed a poorer memory for meaningless material than did nondysfunctional subjects. Even distraction intervals did not impair memory as much as lack of meaning. The researchers concluded that these findings were inconsistent with a hippocampus-amygdala complex site of defect and suggested instead poor cognitive flexibility or frontal lobe deficiency. Rumsey and Hamburger (1988) found that tasks demanding cognitive flexibility and inferential thinking were difficult for a person with autism, even when no memory components were involved; however, their research identified no global memory deficits.

Cognitive Deficits

Because language deficits are prominent in autism, early research suggested that the left hemisphere was impaired. Currently, however, there is little support for this theory. Van Lancker, Cornelius, Kreiman, Tonick, Tangany, and Schulman (1988) studied sound recognition but failed to find support for left hemisphere dysfunction. Children with autism, like nondysfunctional children, did better in recognizing words than sounds and demonstrated a strong ability to match visual and auditory patterns. However, the autistic group (n = 25) made the most errors on words denoting humans, again suggesting a deficit in understanding the interpersonal meaning and semantic use of social words. Interestingly, one-third of the subjects with autism had not established dominance (mean chronological age = 7.7 years), but any tendency toward laterality was correlated with better linguistic performance.

Lincoln, Courchesne, Kilman, Elmasian, and Allen (1988); Prior and Hoffmann (1990); Rumsey and Hamburger (1988); and Schneider and Asarnow (1987) all used cognitive neuropsychological tests to study older persons (8 to 39 years old) with autism. Using the Wechsler Intelligence Scale for Children—Revised (WISC-R) (Wechsler, 1974) and the Wechsler Adult Intelligence Scale—Revised (WAIS-R) (Weschler, 1981), Lincoln et al. (1988) found that 81% of their 33 autistic subjects had significantly higher performance IQs than verbal IQs. Additional factor analysis showed that children and adults with autistic disorder performed poorly on all subtests of Comprehension and Vocabulary (Weschler 1974, 1981); they did better on visual motor subtests of Block Design and Object Assembly (Weschler 1974, 1981). In a comparison of subjects with autism with other subjects, some of whom were nondysfunctional and some of whom had disabilities, the former had more uneven intellectual profiles; scored lowest in general; and tended to do most poorly on subtests requiring both verbal and nonverbal reasoning, concept formation, and comprehension.

In their testing of 10 adults, Rumsey and Hamburger (1988) found no difference in performance and verbal IQs; however, they did find dramatic deficits in both verbal and nonverbal problem-solving abilities. The autistic subjects performed most poorly on the Comprehension and Picture Arrangement subtests of the WAIS-R, both of which involve social knowledge and conceptual thinking rather than rote skills. Similarly, Schneider and Asarnow (1987), who used neuropsychological tests such as the Wisconsin Card Sort Test (Heaton, Grant, & Berg, 1981) and Rey's Tangled Line Test (Rey, 1964), found the poorest performance on both visual and language abstract problem-solving tests but intact performance on rote sorting and visual spatial tasks.

Prior and Hoffmann (1990) suggested that frontal lobe dysfunction was evident in the autistic subject's in-
ability to plan strategies, profit from feedback, show cognitive flexibility, or conceptualize information. Their tests on 12 autistic subjects of average intelligence supported this theory because the subjects perseverated with maladaptive strategies despite training and task variation. All of these studies suggest disabilities consistent with frontal lobe deficits, but questions remain: Is frontal lobe dysfunction primary, causing the behavioral deficits seen in autism? Or, are frontal lobe deficits a secondary developmental problem due to inadequate functioning of more primitive neurological systems?

Therapeutic implications of this research are many. One may need to gradually expand the repertoire of skill expectations through a carefully graded introduction of task complexity and include step-by-step problem-solving challenges. This could be included in sensory-integrative therapy or during functional skill building by changing task demands in subtle yet challenging ways.

Dyspraxia

Some children with autism have motor and visuomotor deficits, and most authors implicate an impediment in cognitive flexibility to explain this. Jones and Prior (1985) questioned whether the poverty of play activities seen in children with autism was due to dyspraxia or deficits in symbolic comprehension. In their study, Jones and Prior compared 10 children with autism with two control groups; one control group had 10 subjects matched on mental age and the other had 10 subjects matched on chronological age. They found that the autistic subjects had the most difficulty in imitating arm and hand gestures, thus suggesting dyspraxia. Furthermore, the typical child with autism displayed an average of four soft neurological signs, such as choreiform movements, difficulty with balance, labored thumb-finger touching, poor discrimination of tactile stimuli, incoordination, or inadequate speech sound production. The evidence of soft neurological signs implicates brain stem and basal ganglia involvement. None of their subjects with autism demonstrated decreased muscle tone. These authors suggested that approximately one-third of the autistic population has significant visuomotor dyspraxia that limits functional and play skills. Because occupational therapists frequently assess and treat children with dyspraxia, these findings support the need for occupational therapy intervention in some children with autistic disorder.

Treatment-Based Studies

Although implications for medical treatment were drawn in many of the studies cited above, a few authors used treatment studies to denote nervous system processes in autism.

Relational Treatment

Zappella (1990) speculated that autism could be the result of an organic process in some children and a relational problem in others; thus, children without neurological disorders could be treated with the use of a family therapy approach. Fifteen children who did not have neurological or metabolic disorders but who were diagnosed with autism under DSM-III criteria were included in this study. They ranged in age from 2 years 10 months to 4 years 6 months. The children’s language and play skills were absent or sparse, and they had delayed toilet training. The therapist met once a month for an average of 19 visits over 2 years with the child and his or her entire family. Between sessions, family members performed therapy at home. The subjects’ relational deficits included: (a) frustrated parent-child communication with increased child power, (b) parental interpretation of the child’s behavior as arising from trauma or suffering, and (c) prevalence of intensive defensive patterns in the child. Therapy was aimed at empowering the parents, developing direct emotional communication with the child, gaining cooperative parental interaction, and dissipating other conflicts (e.g., marital strife). After 2 years, 40% of the 15 children achieved normal development (presumably as judged by Zappella), were mainstreamed into regular classrooms, and obtained a mean IQ of 94 on the Wechsler Intelligence Scale for Children (WISC) (Wechsler, 1949). Thirteen percent showed minimal to no improvement. Zapella reported that when using the same treatment methods on older children and those with neurological impairments, only 12% began to function and develop normally. Despite the compelling nature of the study, Zappella failed to use a control group or correlate selection criteria with outcome results.

Although the assumption that autism is the result of relational conflicts is no longer accepted, Zappella’s (1990) treatment techniques and results are interesting. Her emphasis on early intervention and family involvement are consistent with current trends to treat this problem in populations from birth to 3 years of age. In particular, a holding technique was an integral and unique component of this therapy. Prior to this study, Zappella (cited in Zappella, 1990) had used holding in her work with deprivation-induced autistic rhesus monkeys as they went through stages of rage, relaxation, and normal behavior. In her therapy with children, Zappella (1990) interpreted this holding technique as an intense emotional communication activity that could be used during a child’s periods of overarousal and rage. This holding technique can also be viewed as intense sensory input with powerful emotional components that can be comforting and integrating.

In some respects, Zappella’s (1990) holding technique is similar to Grandin and Scariano’s (1986) squeeze machine that was used to produce an overall intense body
pressure that Grandin found calming and organizing for herself. King and Grandin (1990) advocated a comparable technique that used deep-pressure touch, in addition to other treatment, to reduce hypersensitivity in children with autism. Similarly, Peters (1986) argued that children with autism need more social arousal and loving, but, because of their deficits in sensory processing and reciprocal social interaction, receive and perceive less emotional stimulation.

Thus, children with autism may benefit from intense and positive emotional input from their families. This research also provides tentative evidence that in some cases deep pressure may be a useful technique for calming an overaroused child and engaging the child in social interaction.

**Behavior Modification**

Lovaas (1987) reported a 47% success rate when using intensive behavioral modification techniques with 19 young (under age 46 months) children with autism who had achieved a mental age of at least 11 months at a chronological age of 30 months. These 19 subjects received 40 hr per week of one-on-one in-home treatment from a teacher or therapist. Treatment continued for at least 2 years or until first grade. A control group of 19 subjects received a maximum of 10 hr per week of similar treatment, and another control group of 21 subjects received no treatment. The use of two control groups in the study was an attempt to control for spontaneous recovery, diagnostic error, and the placebo effect. Strikingly, 47% percent of experimental children with autistic disorder successfully passed regular first grade and achieved average IQ scores on the WISC-R, in contrast to only 2% of the combined control groups. Despite the well-conceived research design, pretreatment testing of the multiple expressions of autistic disorder was not reported. Data from a neurological examination, a family history, developmental testing, and a behavioral battery would have been invaluable in predictive research.

**Pharmacology**

Pharmacological treatment based on neurological hypotheses has been tried with limited success. Attention-deficit hyperactivity disorder is recognized in some children with autism, and the benefits of stimulant medication (e.g., methylphenidate hydrochloride, pemoline, dextroamphetamine sulfate) has been examined (Allen & Rapin, 1990) and found to be helpful in a few children (Sloman, 1991). Both Young et al. (1989) and Sloman (1991) explored pharmacotherapy in depth. Although Young et al. (1989) found that haloperidol can be beneficial in reducing stereotypic behaviors of hyperactive persons with autism and with a low IQ, especially when it is paired with behavior therapy, Sloman (1991) cautioned that movement disorders may result from this medication. Propranolol hydrochloride and desipramine hydrochloride have been used to reduce explosive behavior and temper tantrums (Allen & Rapin, 1990). Other antidepressants, anxiolytic medications, and lithium have proven to be useful in some carefully evaluated and selected children (Allen & Rapin, 1990; Sloman, 1991). Although one third of children with autism have elevated blood serotonin levels, treatment with fenfluramine hydrochloride or levodopa has had only limited success (Young et al., 1989). Sloman (1991) suggested that the use of fenfluramine hydrochloride may ultimately reduce natural production of serotonin. Currently, medication use with autism is still experimental, and its criteria are not established.

The subjects involved in the relational treatment, behavior modification, and pharmacotherapy studies reviewed here all demonstrated some positive response to intervention. However, there is a profound need for careful documentation of each subject’s behaviors and symptoms before initiation of a therapeutic intervention. A comparison of factor and multivariate analyses of pretreatment data including intervention and outcome research would enhance our ability to predict which child is likely to benefit from which treatment.

**Diseases and Genetic Disorders**

Although a number of genetic disorders produce severe global retardation, only a few correlate with autistic disorder. Several studies (Bregman, Dykens, Watson, Ort, & Leckman, 1987; Gillberg, 1988; Reiss, Feinstein, & Rosenberg, 1986; Young et al., 1989) have looked at autism’s genetic aspect. These studies showed that 12.3% to 21% of children with fragile-X syndrome, a genetic disorder of the X chromosome, were also diagnosed with autism. Folic acid (Vitamin B) has been shown to improve attention and decrease hyperactivity in some children with fragile-X syndrome. A variety of other X and Y chromosomal abnormalities are found in small numbers of subjects with autistic disorder. Autism is associated with neurofibromatosis, phenylketonuria, Cornelia de Lange syndrome, and Williams syndrome. Gillberg (1988) found that approximately 5% of persons with autism have tuberous sclerosis. In a study of 20 subjects with autism who had no clinical neurological abnormalities, computerized tomography showed one child having tuberous sclerosis (Gillberg et al., 1987).

Some of these genetic disorders share a common pathological process such as deficits in myelination (phenylketonuria and Cornelia de Lange syndrome), microcephaly, or metabolism (Reiss et al., 1986). Other disorders associated with autism are hydrocephalus, Duchenne muscular dystrophy, and Rett syndrome (Reiss et al., 1986). Congenital viral infections such as rubella and cytomegalovirus have been documented as causes of au-
Autism (Gillberg, 1988). Hearing or vision deficits that may mimic autistic disorder need to be recognized before a diagnosis of autism is applied (Allen & Rapin, 1990).

Autism is associated with increased medical and psychopathological disorders in immediate and extended family members. Young et al. (1989) reported that 15.5% of siblings of children with autism had cognitive disabilities, in contrast to only 3% of siblings of children with Down syndrome. DeLong and Dwyer (1988) studied 51 persons with autism, ranging in age from 3 years old to 35 years old, who were grouped into level of functioning by IQ or language ability. After gathering extensive family and developmental histories as well as medical and neurological data on all subjects, they found that neurological abnormalities negatively correlated with IQ scores. It was found that 68% of high functioning persons with autism had one or more immediate family members (parents or siblings) or near relatives (grandparents, aunts, uncles, cousins) with Asperger syndrome. Those families in which a member was diagnosed with Asperger syndrome were also more likely to have a member diagnosed with bipolar disorder; although, the overall incidence of bipolar disorder was five times the average. These findings suggest that neurological deficits are more likely to underlie autism in low-functioning persons, whereas familial genetic factors are more likely to underlie autism in high-functioning persons. In addition, there may be a common underlying genetic disorder associated with high functioning autism, Asperger syndrome, and bipolar mood disorder.

These findings of associated child and family conditions are important for the occupational therapist as he or she attempts to understand the complexity of issues surrounding autistic disorder. During both evaluation and treatment, the therapist can be sensitive to issues of differential diagnosis as well as to the onset of more subtle complications in the child or adult with autism.

Neuroanatomical and Neurophysiological Studies

Other researchers have attempted to discover the neurological substrate of autism through more intrusive methods, such as computerized tomographic scanning, magnetic resonance imaging, positron emission tomography, and event-related potentials measurements. Although evolving technology offers hope for more sensitive data collection, it also reduces our ability to generalize the results of studies performed with varying technology. Furthermore, integrating research on functional deficits with specific central nervous system sites is limited by an incomplete understanding of neurological structures and processes. The data collected from neuroanatomical studies are summarized in Table 2. Neurophysiological research data are summarized in Table 3.

Cerebellum

Anatomical abnormalities of the cerebral cortex were not found in any study. However, the cerebellum was found to have the following characteristics: (a) smaller lobules VI and VII (Courchesne, Yeung-Courchesne, Press, Hesselink, & Jernigan, 1988; Maurakami, Courchesne, Press, Yeung-Courchesne, & Hesselink, 1989); (b) small overall size (Courchesne, Hesselink, Jernigan, & Yeung-Courchesne, 1987; Gaffney, Kuperman, Tsai, Minchin, & Hassenstein, 1987); (c) increased cell density (Bauman & Kemper, 1985; Jacobson, Le Courteur, Howlin, & Rutter, 1988); (d) decreased neuronal size (Bauman & Kemper, 1985); (e) fewer Purkinje cells (Bauman & Kemper, 1988; Ritvo et al., 1986), and (f) agenesis of the cerebellar vermis (Holroyd, Reiss, & Bryan, 1991).

Garber et al. (1989), however, did not find cerebellar abnormalities in a study using magnetic resonance imaging on 15 subjects with autism. In a study that used positron emission tomography on seven subjects, Heh et al. (1989) failed to find the decrease in cerebellar glucose metabolism that one might expect if there are fewer Purkinje cells or a smaller cerebellum. Creasey et al., (1986) speculated that their negative computerized tomography findings on 12 men with autism could be due to deficits too subtle for detection with computerized tomography scanning. Thus, cerebellar dysfunction is associated with autistic disorder in some but not all studies.

If cerebellar dysfunction is present in some persons with autism, how does this affect function? Leaton and Supple (1986) found that lesions to the cerebellar vermis in rats substantially impaired integration of the acoustic startle response. Hypersensitivity to noise is seen clinically in some autistic and neurologically impaired children. Lesions to the cerebellum in adults is associated with hypotonia, ataxia, poor equilibrium, loss of coordination, intention tremor, and difficulty with rapid alternating movements. Speech is affected by hesitancy, stammering, and an explosive quality (deGrott & Chusid, 1988). In addition, there are numerous connections from the cerebellum to sites of sensory processing, including tactile, proprioception, visual, and auditory systems; therefore, the cerebellum serves a role in sensory modulation and integration of activity of the cerebral cortex (Westman, 1990). Although knowledge of the secondary effect of early cerebellar deficits and of deviant development on the immature brain is limited, occupational therapy directed toward lessening the effects of muscle tone abnormalities and dysmetria and improving overall sensory modulation may be helpful for those children with autism who show signs of cerebellar dysfunction.

Brain Stem, Midbrain, and Limbic Lobe

Substantial indication exists that the brain's subcortical centers, including the brain stem systems of the reticular
formation and vestibular nuclei, midbrain structures, and limbic lobe functions, may be major sites of deficits.

From an anatomical perspective, Bauman and Kemper (1985, 1988) found abnormally small neurons and increased cell density in the hippocampal complex as well as in some of the amygdala nuclei, the mammillary bodies, and the medial septal nucleus in all of their subjects (N = 1 and N = 3, respectively). Similarly, Jacobson et al. (1988) found that all nine high-functioning adult males with autism in their study showed increased size of the third ventricle and abnormalities in the caudate nucleus. Gaffney et al. (1987) found a significantly enlarged fourth ventricle on the magnetic resonance imagery scans of 13 subjects with autism when compared with 35 control subjects. They suggested that impairment with atrophy of the pons, medulla, and cerebellum surrounding the fourth ventricle would explain this compensatory enlargement. Four of the 18 subjects with autism studied by Gillberg et al. (1987) using computerized tomographic scans showed abnormalities of either the midbrain or frontal horns. Thus, subtle abnormalities in subcortical central nervous system centers are found in some subjects.

Several studies (Ciesielski, Courchesne, & Elmasian, 1990; Courchesne, Lincoln, Kilman, & Galambos, 1985; Courchesne, Lincoln, Yeung-Courchesne, Elmasian, & Grillon, 1989; Dawson, Finley, Phillips, Galpert, & Lewy, 1988) examined the processes of selective attention using event-related potentials. They found that the event-related potentials for those with autism were smaller or showed polarity to those of the control subjects, who were either nondysfunctional or language-disordered. In his study of 10 subjects with autism, Courchesne et al. (1985) proposed that persons with autism have a limited capacity to respond to novel stimuli because their event-related potentials are similarly small for both novel and background stimuli. Even when the behavior and accuracy of a few highly successful subjects with autism matched that of the nondysfunctional subjects, the autistic subjects showed depressed or reversed event-related potential polarity (with the most difference noted in frontal lobe processing) for visual and especially auditory stimuli (Ciesielski, et al., 1990). In other words, the autistic brain functioned in a manner nearly opposite that of the normal brain even when overt behavior was the same. However, the autistic group tended to be slower in responding, made more errors of commission, and persevered on inaccurate responses. Similarly, when compared to subjects with receptive developmental language disorders (Courchesne et al., 1989), autistic subjects showed opposite responses and neurological functioning despite similar overt behavior. These authors unanimously proposed that these differences implicated the neurophysiological mechanisms of selective attention, including the reticular activating system as the initial site of cortical de-polarization and the frontal and parietal lobes as secondary sites. Furthermore, Dawson et al. (1988) concluded that attention deficits may be specific to language because they found decreased event-related potentials to phonetic sounds but not to piano chords in their 17 subjects with autism.

Wong and Wong (1991) present the most compelling evidence of brain stem impairment. They studied brain stem auditory evoked potentials in 109 children (103 boys and 6 girls, aged 18 to 96 months) with autistic disorder (using DSM-III-R criteria) and compared them with 20 nondysfunctional children, 38 children with autistic-like symptoms (but not full blown autism), and 19 children with retardation. After thorough factor analysis for the effects of age, sex, retardation, and autistic-like characteristics (varying degrees of deviance in language, social interaction, and behavior), they found that the autistic characteristics were the only factor significantly related to an overall increased latency, or slower brain stem transmission time. Thus, autism, not mental retardation, was correlated with slower brain stem responses. Similarly, Ornitz (1985), who found a prolonged postrotary nystagmus in 22 subjects with autism compared with 25 age-matched control subjects, proposed that this response was caused by deficiencies in sensory habituation of brain stem pathways, which resulted in inadequate inhibition of the reticular formation and vestibular system.

Deficits in neurotransmitter levels have also been implicated in autism; however, this extensive research is beyond the scope of this paper. For more information on this topic, see Coleman and Gillberg (1985) and Sloman (1991).

From the research studies cited, one could conclude that impairment of the brain stem is found in a substantial number of persons with autism. This impairment might account for arousal deficits, sleep disorders, abnormal auditory-evoked responses, widespread attention limitations, and sensory processing deficits in those with autism. These brain stem and subcortical centers have numerous connections with all parts of the brain. Deficits in limbic lobe structures might account for the emotional disorder present in autism because the limbic system determines what is pleasurable or distressing. The limbic system has a role in memory, facilitating attention, and registration or orientation to information (Westman, 1990). Bauman and Kemper (1985) suggested that the abnormalities of the midbrain and limbic system that they found (n = 1) were enough to explain autistic features and that similar lesions produced autistic-like symptoms in monkeys. Because occupational therapists use sensory integration that aims to improve function through improved sensory responsiveness, these findings add support to the notion that subcortical abnormalities are present in persons with autistic disorder.

Frontal and Parietal Lobes
A study by Horwitz, Rumsey, Grady, and Rapoport (1988) is particularly important because it was the first to docu-
ment frontal lobe physiological abnormalities in persons with autism and link this to subcortical systems. Using positron emission tomography, the authors studied 14 men with autism (aged 18 to 39 years) with widely scattered IQ scores and 14 nondisabled subjects. They found a disruption between frontal and parietal lobe interaction and between the frontal and parietal lobes' interaction with the thalamus, caudate nucleus, and lenticular nucleus. Horwitz et al. (1988) explained that these results are compatible with neurological impairment in the complex attention network. That network has three basic components: (a) the reticular structures, which are responsible for arousal, (b) the limbic lobe, which provides the motivational element, and (c) the frontal and parietal lobes, which are responsible for integrating sensory and motor components of directed attention. According to Westman (1990), the four functions of the frontal lobes are: (a) to maintain focused attention and regulate awareness, (b) to organize strategies and execute behavior, (c) to sequence and plan skilled acts or thoughts, and (d) to make choices and initiate action.

When considering the literature and the children seen in treatment, these explanations of frontal lobe function and the attention network are uncanny in their relevance and clarity. A more complete picture of the etiology of autistic disorder emerges when one considers the role of the cerebellum in coordination, maintenance of muscle tone, and sensory modulation and notes the cerebellar deficits found in some children with autism. Furthermore, the results of these neurological studies of anatomy and physiology seem to be correlated with the affective, arousal, attention, cognitive, and praxis deficits noted in functional studies.

Summary

Although a tremendous amount of research on autism exists, many questions regarding etiology and treatment remain. What, then, can we conclude about the neuropsychology of autism? Figure 1 is a summary of the medical, functional, and neurological findings. Frontal lobe, brain stem, midbrain, limbic lobe, and cerebellar deficits are proposed as being primary to the understanding of autism. These neurological deficits are expressed in primary or secondary impairment in social, cognitive, attention, sensory processing, and praxis skills. However, it should also be clear that subjects with autistic disorder, across all studies, demonstrated heterogeneous rather than homogeneous deficits. Genetic disorders and diseases are considered primary distinguishable causes of autism and may represent separate subsets of autism.

The etiology of these heterogeneous disorders, all of which are labeled autistic disorder, is still uncertain. Perhaps autism is one of several currently undefined disorders that have similarities of expression but varying causes. For example, Fein, Waterhouse, Lucci, and

Figure 1. Summary of medical, functional, and neurological findings in studies of autistic disorder.
Synder (1985) found three distinct cognitive subtypes, independent from language or social interaction, in about half of the autistic population that they studied. Thus, cognitive variations can be found despite similar social and language features. Conversely, as Goodman (1989) suggested, autism may result from multiple primary deficits. For example, the critical neurodevelopmental period theory was used to explain simultaneous cerebellar and subcortical abnormalities resulting from late intrauterine disruption of development; therefore, the timing of an insult may affect several vulnerable central nervous system sites simultaneously (Courchesne et al., 1988).

Most researchers agree that autism is a case of deviant structure, functional organization, or both that occurs before birth, rather than cell atrophy following damage. Is there a common link between the various sites of dysfunction? Ornitz (1985) proposed a sequence of disorder that has a primary site of impairment in the midbrain reticular formation and other subcortical structures and that subsequently affects the cortex. Both Ornitz (1985) and Horwitz et al. (1988) believed damage to a specific site that provides input to cortical systems could impair cortical development and functioning in the absence of structural cortical defects. Other researchers proposed that deficits in neurotransmission and the resultant abnormal neuronal polarization at several vulnerable sites may be crucial, as is the failure of sites stimulated by neurotransmitters to fully develop (Jacobson et al., 1988). Another research approach advocated by Siegel (1991) viewed autism as a system of atypical ontogeny; hence, one could chart the sequence of developing psychopathology to aid in prognosis and timely intervention. For example, the onset of stereotypic behavior could be systematically recorded so that early signs of such behavior could be recognized in other children with suspected autistic disorder. Finally, Hobson (1991) asked “Is enough known about the brains of children with pervasive disorders to provide them with therapy that is truly neurologically oriented in nature?” (p. 236). Although our understanding of neurological function has progressed, clearly we do not fully understand the neuropsychology of autistic disorder.

The treatment of a child with autism must involve a team of professionals who can address the potential social, cognitive, motor, attention, medical, and sensory deficits presented. Caution must be used in projecting the potential benefits of any single treatment approach. As Sloman (1991) stated, “parents . . . [and] professionals . . . sometimes react to the severity of this disorder with unrealistic expectations for therapeutic intervention” (p. 166). Despite the suggestions of King and Grandin (1990) and Nelson (1984), current practice in occupational therapy for those with autism is undefined.

In addition to treatment, occupational therapists can collaborate in rehabilitation research projects. Acker (1986) proposed that rehabilitation professionals serve as a bridge pairing neuropsychological findings with a description of the effect of neurological deficits on daily life functioning. Perhaps there is an analogy between research on autism and occupational therapy; both are at the stage of mapping functional models onto biological models. Clearly, occupational therapists could collaborate with neuropsychologists to define functional deficits, abilities, and treatment methods and to measure treatment outcome. There are numerous tools, too many to mention in this paper, that could be used to gather information on functional skills and behavior. For example, the recently developed Childhood Autism Rating Scale (Schopler, Reichler, & Renner, 1988) is used to clarify behaviors and classify subjects with severe, moderate, or mild expressions of autism. This scale can be used to rate 15 aspects of autism, including social relating, object and body use, sensory responses, communications skills, and general intellectual abilities. Consistent use of a standard set of functional skills and behavioral rating scales would establish a baseline for treatment, enhance integration of neu-
ological and functional findings, and supply data about the expression of autism. It would be useful for occupational therapists to define a recommended set of well standardized assessment tools to facilitate research on autistic disorder.

New research findings are continually evolving. In addition to human research, there is research being done on primates that holds promise for occupational therapists working with autistic populations. For example, Schneider, Kraemer, and Suomi (1991) found that vestibular stimulation in rhesus monkeys resulted in improved motor maturation rates and cognitive test scores. Kraemer (in press) proposed a neurobiological basis for attachment in rhesus monkeys, but drew implications to human autism. He proposed a cascading effect from the lack of ability to form social attachments to subsequent deficits in sensory motor skills, arousal, and behavior, which results in a locking of neurobiological systems with decreased neuronal plasticity that prevents further development.

Although the specific neuropsychology of autism remains hazy, a data explosion is under way. Ongoing research may link neuropsychology, functional deficits, and treatment, thus improving the prognosis for persons with autistic disorder.

References


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- Relaxation training as a self-help approach for children with headaches
- Occupational therapists in private practice
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