Infantile Autism Reviewed: A Decade of Research

by Marian K. DeMyer, Joseph N. Hingtgen, and Roger K. Jackson

Abstract

Progress in the study of infantile autism over the past 10 years was reviewed in terms of classification and diagnosis, relation to other disorders, demographics, parental characteristics, genetics, intelligence, language, perceptual processes, behavioral characteristics, neurobiological, biochemical and pharmacological aspects, behavioral/educational treatment methodologies, prognosis, and theoretical considerations. This decade's research led to the now generally accepted position that infantile autism is a type of developmental disorder accompanied by severe and, to a large extent, permanent intellectual/behavioral deficits. However, its relationship to other forms of developmental disorders and to mental retardation remains to be delineated. Perceptual/cognitive/language defects appear central to the autistic syndrome, but the specific underlying mechanisms are unknown. Most studies indicate that autistic children have more signs of brain dysfunction than do normal children and about the same number as mentally retarded children. The overwhelming evidence suggests that the treatment of choice for maximal benefit to autistic children is a systematic, intrusive behavioral/educational approach. Yet, in spite of significant gains in almost all children treated, the typical prognostic picture is poor in terms of achieving self-supportive adulthood. The parents of autistic children have been found to be essentially similar to parents of children with organic brain disorders, and manifest no psychopathology which conceivably could induce the disorder. The vast majority of theoretical articles appearing in the 1970s proposed some form of neurobiological defect as the causative factor in autism. One of the major goals for future research is to undertake more extensive comparative studies of nonautistic brain dysfunctional children and autistic children that could yield clearer differential behavioral profiles and testable neurobiological hypotheses.

It has been 9 years since a general survey of the most important developments in the study of early childhood psychosis was published in Schizophrenia Bulletin (Hingtgen and Bryson 1972). That review covered the period from about 1964 to 1970 and was based on a perusal of over 400 articles pertinent to infantile autism, childhood schizophrenia, and related disorders. Since over 1,100 additional publications appeared in the decade of the 1970s, including a new journal devoted to infantile autism, another review now seems appropriate to assess what.

This review article is dedicated to the memory of two recently deceased pioneers in the study of infantile autism: Leo Kanner, the famed child psychiatrist, who in 1943 was the first to describe and name infantile autism as a specific syndrome; and Charles B. Ferster, the equally prominent research psychologist, who in 1961 collaborated on a report that subsequently led to the current predominance of behavioral techniques in the treatment of autistic children.

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progress has been made in understanding the syndrome.

One of the major problems confronting the field has been the inconsistent use of terminology referring to the various disorders included under the general designation of early childhood psychosis—for example, infantile autism, childhood schizophrenia, early childhood schizophrenia, early infantile psychosis, and symbiotic psychosis. Indeed, the term early childhood psychosis is no longer considered useful by most researchers and clinicians, since it may mislead one into assuming an association between this condition and adult psychotic disorders, a possibility that appears more and more remote. Reflecting this attitude is the new DSM-III: Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association 1980), which uses the term pervasive developmental disorder and includes infantile autism as one category under this general classification. Schizophrenia in childhood, on the other hand, in DSM-III is classified under the same subcategories used for the adult schizophrenias.

In our original review we did not want to exclude any articles pertinent to infantile autism. Thus, we dealt with studies of "psychotic" children manifesting most of the symptoms associated with infantile autism regardless of the terminology used in the diagnosis. Now, 10 years later, this may be less of a problem since there are so few articles using the terms childhood psychosis or childhood schizophrenia. Indicative of this change in attitude is the change in name of the Journal of Autism and Childhood Schizophrenia to Journal of Autism and Developmental Disorders. Therefore, as much as possible, we will restrict the articles reviewed to those clearly dealing with infantile autism as described under pervasive developmental disorders in DSM-III. With a few exceptions, those studies that are more obviously related to what is now regarded as schizophrenia in children will not be considered here.

In our earlier review, we were able to cite almost all of the more than 400 articles reviewed. With over 1,100 papers published during the 1970s, we were required to be more selective in citing articles for the present review. For readers who are interested, a total list of all references reviewed, including those not cited in this article, is available on request from the authors. We have referred mainly to studies in which data were collected with some consideration for experimental control or were of special significance to the field. Case reports have been cited only when especially informative.

### Classification and Diagnosis of Infantile Autism

How investigators classify and diagnose infantile autism has depended heavily on the ideas of each investigator concerning etiology and the underlying meaning of various symptoms. There are nearly as many different systems as there are investigators. Nevertheless, some important steps have been taken, and important knowledge has been gained. Before giving these advances, it may be helpful to discuss the historical origin of controversies and issues in diagnosis and classification of infantile autism.

One of the chief problems has been how to handle the questions of mental retardation and organic brain disease, issues especially troublesome with regard to infantile autism. When Kanner (1943) first described the diagnostic features, he also remarked that the condition bore no resemblance to any known neurological condition and implied that autistic children had a basically normal intelligence. For over two decades afterwards, diagnosticians generally believed that the presence of mental retardation or neurologic signs ruled out the diagnosis of infantile autism in the Kannerian sense, even if the child met all behavioral criteria (Eisenberg 1966). Thus diagnosis was frequently one dimensional; a child was labeled as afflicted with either infantile autism or mental retardation, not both. On the other hand, Goldfarb (1961) early recognized two sub-classes of childhood schizophrenia, the organic and nonorganic, a two category system.

Several problems were met in using these ideas. First, organic brain dysfunction is difficult to rule out, especially if the neurological evaluation is not rigorous. Second, even if a patient has no positive neurological signs by examination or by history, the brain may still be malfunctioning in an organic sense. An insult to the brain or a maldeveloped brain may leave certain areas such as the motor system intact and yet seriously compromise other areas such as the limbic lobe or language center without leaving a detectable trace in even a rigorous neurological evaluation. Insults to the brain may occur in a "silent" fashion without an overt gross neurological change at the time the insult
occurred. Third, whether a child shows signs of organic dysfunction may change over time. Followup studies have revealed that many preschool autistic children may have negative neurological examinations only to develop signs of brain malfunction later in life. For example, Rutter and Lockyer (1967) reported that 18 percent of their followup sample had developed seizure disorders by adolescence. Fourth, long-term studies of intelligence showed that most autistic children whose mental retardation was considered functional during their preschool years remained retarded in later years, often despite improvement in social functioning (Knobloch and Pasamanick 1975).

The fact that most studies of infantile psychoses have taken place in psychiatric/psychological clinics/hospitals has affected classification and diagnosis. Children with psychotic symptoms have generally been prescreened, and those with obvious neurological signs tend not to be referred to such settings. As Knobloch and Pasamanick (1975) pointed out:

elimination of all patients with other specifically classified disorders might leave an apparently discrete condition. Such a selection process could explain why debate continues about criteria for diagnosis and organic versus psychogenic etiology. . . . [p. 183]

These two investigators worked in a children's hospital where they saw a wide spectrum of cases, most of whom were 2 years old or younger. Among 1,900 cases, 64 (3.4 percent) met Kanner's criteria, 14 of whom were phenylketonuric. These latter were removed from further comparison and 50 autisticics were compared with 50 children with some type of central nervous system (CNS) dysfunction but without autistic symptoms and 50 without neuropsychiatric disorder. In both the autistic and development disorder samples, there were equal signs of perinatal and neonatal complications and of other disorders implying neurological dysfunction (e.g., developmental quotient below 50, convulsive disorders, strabismus, and cerebral palsy). All 50 autistic children showed signs of CNS dysfunction and differed from the nonpsychotic comparison group only in their autistic symptom complex. In a followup of 39 autisticics whose mean age was 7 years, three-fourths had lost their autistic behavior but retained their mental retardation.

These kinds of findings led to changes in ideas of investigators who found it increasingly difficult to sort cases of autism that were "true" Kannerian cases from the ones that were "secondary" to brain dysfunction or mental retardation. They were beginning to suspect that more cases than heretofore supposed were complicated by neurological dysfunction or by mental retardation or both. Classification systems in the 1960s, and sometimes yet today, attempted to deal with these complex cases by providing such labels as "secondary autism" or "organic brain damage with autistic features" or some roughly equivalent terminology.

Another attempt to deal with complex cases was the multicategory system as in DSM-II (American Psychiatric Association 1968). For example, a schizophrenic child with an IQ of, say, 54 would have two diagnoses coded, namely schizophrenia, childhood type, and mental retardation, mild. Still a third attempt was the multi-axial system which differs chiefly from the multicategory system in requiring that certain dimensions of the illness always be coded. In the multicategory system, the coding of several diagnoses is in a sense discretionary.

The pioneer multi-axial system of the World Health Organization (WHO) was found superior to a multicategory one by 24 child psychiatrists (Rutter, Shatter, and Shepherd 1975). Separation of etiological factors made classification easier and more uniform especially considering continuing disputes over theory and causation. If the system is used conscientiously, investigators can protect themselves from bias and keep important dimensions separate from each other to study their interrelationships.

The most important development of the 1970s for the immediate future of classification and diagnosis is the new DSM-III (American Psychiatric Association 1980), which is multi-axial throughout, listing five separate axes (see table 1). While this was modeled on the WHO system, the intellectual diagnosis of mental retardation was eliminated as a separate axis. Instead, coding of both a psychiatric disorder and mental retardation is done on Axis I (see table 2). In the only reliability study to date (Russell et al. 1979), 1

1 The WHO system had four axes: (1) clinical psychiatric syndrome; (2) intellectual level; (3) associated biological factors; and (4) psychological/social factors. DeMyer (1975b) also has used a three-dimensional system (behavioral, intellectual, and neurological) since the early 1970s.
diagnosticians had low rates of agreement when two diagnoses were needed in a single category. To us, it appears more appropriate to require an intellectual diagnosis as a separate axis for all children's psychiatric disorders. This coding seems justified, particularly because of the importance of measured IQ as a prognosticator in infantile autism² (see the Prognosis section).

Age of onset has become an important factor. Kolvin (1971) classified child psychoses as early onset (before 30 months), middle onset (after 30 months up to 8 years), and late onset (prepubertal and pubertal). He found that early onset cases (which most would probably call infantile autism) were much more common than middle onset cases, and the former did not appear to him to be related to later developing schizophrenia. Prior et al. (1975) subjected data from Rimland's Behavior Rating Checklist to a numerical taxonomic analysis and found that age of onset was one of the primary features that distinguished the two resulting classes of children (class 1: onset before 2 years of age, autistic type behavior and severely impaired interpersonal relationships; class 2: later onset, less severe relationship impairment, and greater symptom variability). The age break demonstrated by Prior et al. (1975) was 6 months younger than Kolvin’s (1971).

DSM-III uses age of onset (i.e., 30 months) as the dividing line for diagnosing infantile autism versus childhood onset pervasive developmental disorder (see table 2). The new manual, however, does not make use of Kolvin’s (1971) findings concerning middle onset psychosis, but instead gives 12 years as the upper chronological age for diagnosing pervasive developmental disorder. It appears that relationships of exact age of onset to other important variables such as symptom picture and outcome have not yet been firmly established (Chess 1977). Chess (1977), in her study of autism in congenital rubella, found that typical autism could develop after age 2½ years. In any case, locating age of onset within an exact 6-month period is difficult to do, but no formal reliability studies on the subject have been conducted.

### Table 1. DSM-III axes of psychiatric diagnosis

<table>
<thead>
<tr>
<th>Axis</th>
<th>I. Clinical psychiatric syndrome(s) and other conditions (multiple diagnoses are possible)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Axis</td>
<td>II. Personality disorders (adults) Specific developmental disorders (children and adolescents)</td>
</tr>
<tr>
<td>Axis</td>
<td>III. Physical disorders</td>
</tr>
<tr>
<td>Axis</td>
<td>IV. Severity of psychosocial stressors</td>
</tr>
<tr>
<td>Axis</td>
<td>V. Highest level of adaptive functioning past year</td>
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</tbody>
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² The National Society for Autistic Children (NSAC) (Ritvo and Freeman 1978) has recommended a four-point diagnostic system. It will probably be superseded by DSM-III.
chotic children “early schizophrenia” because they thought it did not meet Kanner’s criteria to the letter. Because these children had developed serious symptoms in the first 2 years of life and seemed to fit what most other diagnosticians called “infantile autism,”

Table 2. DSM-III classification of disorders previously called by other terms (e.g., early child psychosis, early infantile autism, childhood schizophrenia, atypical child psychosis)

<table>
<thead>
<tr>
<th>Axis I 11</th>
<th>Generic term: Pervasive developmental disorders 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subclass term</strong></td>
<td><strong>Diagnostic criteria</strong></td>
</tr>
<tr>
<td>1. Infantile autism</td>
<td>All of the following:</td>
</tr>
<tr>
<td>Full syndrome</td>
<td>299.00</td>
</tr>
<tr>
<td>Residual</td>
<td>299.01</td>
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<tr>
<td>c. Language deficit—gross</td>
<td></td>
</tr>
<tr>
<td>d. Speech peculiar, if present</td>
<td></td>
</tr>
<tr>
<td>e. No delusions/hallucinations</td>
<td></td>
</tr>
<tr>
<td>2. Childhood onset pervasive developmental disorder</td>
<td>a. Severe disturbance of emotional relations</td>
</tr>
<tr>
<td>Full syndrome</td>
<td>299.90</td>
</tr>
<tr>
<td>Residual</td>
<td>299.91</td>
</tr>
<tr>
<td>3. Atypical pervasive developmental disorder</td>
<td>Distortion of social and language skills that cannot be classified as either 1 or 2 above</td>
</tr>
<tr>
<td>299.8x</td>
<td></td>
</tr>
</tbody>
</table>

| **Generic term: Schizophrenic disorders 3** | **Diagnostic criteria** |
| Schizophrenic disorders in childhood (5 subtypes as for adults) | As for adult schizophrenia, all of the following: |
| Full syndrome | 295.1x | a. At least 1 of 10 delusional, hallucinatory, associational, or affectional symptoms |
| through | 295.6x | b. Impairment in two or more routine functions |
| Residual | 295.6x | c. Continuous illness for at least 6 months |

1 If an individual has mental retardation in addition to a mental illness, this diagnosis is also coded on Axis I and is described in DSM-III beginning on p. 36. Borderline mental retardation is described on p. 332.

2 Described in DSM-III, pp. 86–92, under the section “Disorders Usually First Evident in Infancy, Childhood or Adolescence.”

3 Described in DSM-III, pp. 181–193, in the section “Schizophrenic Disorders.”

the authors dropped the term “schizophrenia” and called this group “high functioning infantile autism.”

Piggott and Simson (1975) presented some evidence that diagnosis itself may have changed in the 1970s. Using DeMyer and Churchill’s (DeMyer et al. 1971a) criteria and their chart information, they rediagnosed 33 children who had been originally diagnosed in the 1950s and 1960s. Childhood schizophrenia was the original diagnosis in 58 percent of cases but constituted only 18 percent on rediagnosis. One third of children originally diagnosed as psychotic did not retain this category on rediagnosis.

We hope that reviewers at the end of the 1980s will be able to report separately on the subcategories of major mental illness of childhood if investigators have followed the new DSM-III diagnostic system. We will have to get used to new terms. “Childhood psychoses” does not appear at all. Instead of the old generic term “childhood schizophrenia,” DSM-III gives us “pervasive developmental disorders” (see table 2). In fact, childhood schizophrenia is nowhere mentioned, but we are advised to speak of “schizophrenia in childhood.” DSM-III also resurrected an old term that has appeared seldom in the literature of the 1970s—namely, “atypical”—and applied it to pervasive developmental disorder whose relationship to “schizophrenia in childhood,” the authors say, is disputed. But what is its relation to infantile autism, with which it shares a generic heading? (see table 2). The answer awaits future research.
Studies of Diagnostic Reliability and Differential Diagnosis. In addition to the dimensions of intelligence and neurological signs, the behavioral features of infantile autism create many thorny reliability problems. Kanner's behavioral criteria for infantile autism—namely, severe social distance, desire for preservation of sameness, and failure to use language for communication—have been used frequently. This situation would seem to make for good diagnostic agreement, especially since the criteria seem to be relatively simple. Actually they are complex constructs that can be and are defined by many different behaviors. For example, various investigators differ in how many social avoidance behaviors need to be present to define autistic social distance. All of the rating scales, while having overlapping items, vary considerably in the number and types of items. For example, Rimland's (1971) scale has 80 items while Clancy, Dugdale, and Kendle-Short (1969) used 14. The categorical systems also vary in the features that must be present. The requisites for a useful diagnostic system are that different diagnosticians using it make the same diagnosis, that it differentiates related but separate conditions (see table 2 for differential diagnosis), and that it be related to other important aspects of the illness such as cause or type of therapy required. We reviewed the literature to see how well these conditions had been met by any of the diagnostic systems.

Rimland's scale, which has undergone three revisions (E-1, E-2, E-3), has received the most research attention. DeMyer et al. (1971a) found that it differentiated only partially between infantile autism and mental subnormality; that his E-1 version more closely coincided with their definition of primary autism than did his later E-2 version, but that only a small fraction of children diagnosed autistic by their criteria met Rimland's +20 cutoff score. They also compared Rimland's system with those of Polan and Spencer (1959), Lotter (1966), and the British Working Party (Creak 1964). While there were significant correlations among scores of all these scales, there was an overlap no greater than 35 percent, meaning that several children receiving high scores on some scales would receive low scores on others. None of the systems differentiated the subcategories of psychosis as defined by DeMyer and Churchill (DeMyer et al. 1971a) who achieved 96 percent agreement with each other. The authors concluded that investigators who work closely together can achieve good diagnostic agreement about subgroups of infantile psychoses. However, agreement lessens considerably when diagnosticians without constant feedback from one another compare diagnoses. Davids (1975) reported that Rimland's E-1 checklist differentiated chart diagnoses of autistic, other psychotic, and nonpsychotic groups. However, the checklist did not differentiate children with the full symptom complex of infantile autism from those appearing "autistic-like."

Questions have been raised about the reliability of Rimland's (1971) checklist because it relies solely on the parents scoring the items by themselves. Albert and Davis (1971) found high agreement between 31 sets of parents who rated their normal preschool children. In the usual rating situation of Rimland's scale, however, parents are asked to rate how the child was behaviorally in the first 5 years of life. If the child is, say, 8 years old, parents may remember differently than they would have when the child was 5 years old. Thus children of different ages might be rated differently because of differences in parents' memory factor. Interestingly, the normal children received mean autism scores of 21.2 compared to 20.9 for autistic children as reported by Douglas and Sanders (1968) and 23.6 reported by DeMyer et al. (1971a).

When parent ratings were compared with teacher ratings by Prior and Bence (1975), there were a total of 63 disagreements for nine autistic children. Generally the teacher assessed the children at a lower level than the parents in estimates of ability, social responsiveness, and bizarre behavior. In only one case, however, would the diagnosis have been changed. The authors criticized the weight given to speech items. Children with higher E-2 scores had speech patterns that added considerably to their scores so that they were not strictly comparable with the scores of the nonspeakers for severity of handicap. Masters and Miller (1970) have pointed out also that the instrument does not significantly improve upon base rate prediction.

Rimland (1971) himself subtracts the "nonautistic score" from the "autistic score" and calls only those with +20 scores "autistic," 10 to 20 "equivocal," and below 10 "definitely not autistic." Of 2,218 suspected autistics whose E-2 forms he analyzed, only 9.7 per-
cent met his cutoff +20 score. However, because these children were diagnosed by many different clinicians who used no uniform criteria, no effective diagnostic comparison groups were available to him.

Rimland’s checklist was compared with Ruttenberg’s Behavior Rating Instrument for Autistic and Atypical Children (BRIAAC) by Cohen et al. (1978) and with clinical diagnoses using DSM-III criteria. On clinical grounds 13 children received a diagnosis of “primary autism” and the remaining 14 other diagnoses. All were rank ordered by degree of severity. Only 2 of 13 children with a clinical diagnosis of primary autism met Rimland’s +20 score. Clinical assessment of severity revealed “relatively good agreement” on overall severity as measured by BRIAAC. The latter test, however, did not differentiate among clinical diagnostic groups. Thus Cohen et al. (1978), like other investigators, found that a large proportion of the children with equivocal and nonautistic Rimland scores appeared clinically quite similar to the children with scores above +20. In contrast to Rimland’s system, which may result in many false negatives, the 14-point scale of Clancy, Dugdale, and Rendle-Short (1969) may locate many false positives (Capute et al. 1975).

An important source of diagnostic unreliability comes from the relation of various symptoms to both mental age and chronological age. Freeman et al. (1978) compared autistic, normal, and retarded children, ages 2 to 5½ years, using 67 “objectively defined behaviors” under controlled observer conditions. An important strategy was that the two abnormal groups were matched for mental age and chronological age. A discriminant analysis revealed that only two behaviors (“repeats sounds” and “communicates speech”) defined the discriminant function and yielded only 63 percent overall correct classification of the three groups. While 78 percent of the normal children were correctly classified and only 12 percent of autistic and retarded children were misclassified as normal, it was not possible to separate the autistic from the retarded in the presence of the normal sample. There was some evidence that many of the items used on the rating scale were strongly related to both chronological and mental age. Such a situation would mean, for example, that some behavior held to be essential to the diagnosis of autism by one investigator might be strongly present at one chronological or mental age, but not at another and lead to a difference in diagnosis in the same child at different chronological ages. The authors concluded that several years of work were necessary to develop a rating scale which could distinguish reliably between young autistic children and other diagnostic groups.

The continuous nature of autistic symptoms across various diagnostic groups such as blindness, deafness, mental retardation, learning disorders (Wing 1969), disintegrative psychosis (Corbett et al. 1977), and even normality has posed many problems in achieving reliability in the differential diagnosis. There are very few symptoms, for example, that are found nearly exclusively in infantile autism. Even though the various types of childhood psychosis are supposed to be defined by a group of symptoms in each child so diagnosed, factor-analytic, cluster-analytic, and numerical taxonomic analyses, by and large, have not verified this assumption, no matter whose diagnostic system has been studied. In the largest study (DeMyer, Bryson, and Churchill 1973) examining these issues (normals, n=30; nonautistic neurological cases, n=36: autistics, n=70), the following findings emerged: (1) The psychiatric diagnostic groups lay on a severity continuum, with nonautistics having the fewest symptoms, high-functioning autistics next fewest, and lower functioning autistics the most. (2) The only symptom items differentiating the three psychiatric diagnostic groups were degree of social withdrawal and degree of communicativeness of speech. (3) A cluster analysis showed much symptom overlap, thus precluding any definition of cluster groups. However, this analysis did point out unequivocally the continuous nature of symptom distribution across normal, neurological, and autistic groups and confirmed the severity continuum of these groups. The latter feature was pointed out by Anthony in 1958.

Capute et al. (1975) have demonstrated how frequently autistic symptoms are found in developmentally disabled children by using the 14-point autistic checklist of Clancy, Dugdale, and Rendle-Short (1969). They found that as the severity of mental retardation increased, so did the frequency of autism as diagnosed by Clancy’s scale. While the authors did not compare symptom frequency with mental age, their findings strongly suggested a positive correlation and would tend to
support the findings of Freeman et al. (1978).

Clearly a crucial question still remains about which behavioral functions critically define the differences between infantile autism and related conditions. Research of the 1970s strongly suggests that degree of social distance and degree of communication of speech are the most consistent differentiating rating items. However, the optimum rating instrument has yet to be devised. Any rating instrument that cannot differentiate in 100 percent of cases the normal child from the autistic must be used in conjunction with other aids. It seems clear also that researchers in rating behavioral symptoms must find ways to control for both mental age and chronological age.

Rutter (1978) has suggested that the single most important factor for the diagnosis of infantile autism is that the social age be on a lower plane than the mental age, but much research must be done on such an assertion. For example, the mental age can be derived using several dimensions of intelligence. Which dimensions should be used in the comparison: verbal age, perceptual-motor age, gross motor age, or a general intelligence estimate? If the last dimension is chosen, what test or group of tests should be used? This is a crucial issue because of the wide scatter of many psychotic children on psychological tests. Another important question is how to measure social age, which in itself can be defined by many different behaviors. And thus we return full circle to the ever present problems in achieving diagnostic reliability.

The most recent classification system has been introduced by Schopler et al. (1980), who used their Childhood Autism Rating Scale (CARS) to compare their definition of autism with that of Ritvo and Freeman (1978) and of Rutter (1978). While there is agreement among all three systems about some behavioral factors that are important for diagnosis, there are important differences. Schopler et al., with 15 rating items, use the broadest concept of autism, and they include the specific items about which Ritvo and Freeman and Rutter disagree: “sensory peculiarities” (Ritvo and Freeman) and insistence on sameness and stereotypies (Rutter). Of the 266 children identified as autistic by the CARS, only 20.6 percent met Rutter’s criteria for autism, while 46.6 percent met Ritvo and Freeman’s criteria—a significant difference. On this basis, Schopler et al. argue that sensory peculiarities should be included as a primary diagnostic feature of autism.

Validity of Diagnoses. The ultimate test of usefulness of a diagnostic system is whether it is related to some other important features of the condition such as cause, outcome, or treatment. Unfortunately, none of the present systems can completely achieve this goal. No matter what system is used, a diagnosis, for example, of infantile autism, early childhood schizophrenia, or early childhood psychosis predicts a less than normal outcome in general (DeMyer 1979). With regard to etiological and therapeutic considerations, none of the checklists or categorical systems which have been proposed in the last decade appear unequivocally superior. Thus, continued work toward devising a more useful diagnostic system is required. To us, it appears that multi-axial systems have the greatest potential, especially if the influences of mental and chronological age on behavioral symptoms can be better delineated.

Relation to Other Disorders

The major conditions that infantile autism must be differentiated from according to DSM-III are listed in table 3 along with major differentiating features. As was discussed in the diagnosis and classification section, the various formal checklist diagnostic systems lacked diagnostic specificity and sometimes could not differentiate autistic from young normal children let alone from related pathologic groups. It is clear we still need a clinical diagnostic history and observation to use the various proposed rating scales intelligently. To recapitulate: The two features that most consistently separated nonautistic from autistic populations were degree of communicative skills and social relatedness. These differentiating features applied both to the mental retardation and expressive language disorder populations (see table 3). (For relationship to dysphasia, see the Language section.)

Mental Retardation. Bartak and Rutter (1976) found some symptom differences between autistics above and below 70 IQ (nonverbal). The more intellectually able autistics had better relationships with adults, higher language skills, better developmental milestones, and fewer ritualistic and compulsive behaviors. Using the BRIAAC, Wolf, Wenar, and
Table 3. *DSM-III* differential diagnosis of pervasive developmental disorders: Major differential features listed in *DSM-III*

**Axis I**

Infantile autism vs. childhood onset pervasive developmental disorder
Former onset before 30 months of age and latter after 30 months of age—chief differential criterion

Pervasive developmental disorders vs:
1. Mental retardation
   a. Full syndrome PDD rarely present
   b. When both disorders present, both diagnoses should be made
2. Schizophrenia in childhood
   a. Hallucinations, delusions, incoherence present
3. Hearing impairment
   a. History of responding only to loud noises
   b. Audiogram indicates deafness or hearing impairment
4. Developmental language disorder, receptive type
   a. Eye and social contact made
   b. Appropriate gestures used

Ruttenberg (1972) found that retarded children, particularly those with Down’s syndrome, were less emotionally disturbed and better integrated than autistic children who were unable to generalize “between key areas of function.” (See also the Intelligence section.)

**Borderline or Schizoid States.** According to Wolff and Barlow (1979), about 3 to 4 percent of new referrals to child psychiatry could be labeled as “schizoid” or resembling Asperger’s (Van Krevelen 1971) autistic psychopaths. They were described as verbal, solitary, unadaptable, overly sensitive, and paranoid with “odd ideation.” Schizoid children were intermediate in their overall functioning between autistics and normals and did poorly on cognitive and memory tasks. Autistics did relatively well on rote memory and visual-spatial tasks and poorly on language comprehension. Thus, there was a continuum effect, as well as an indication that the schizoid children had a somewhat different disability pattern. It is important to note that both experimental groups were not typical of the larger groups from which they were drawn; that is, the experimental autistic children were higher functioning than autistic children as a whole and the experimental schizoid were lower functioning than their larger group. This observation points out strongly that schizoid children are not so severely handicapped intellectually as autistic children and further substantiates the continuum of this handicap across diagnostic groups.

**Later Developing Schizophrenia.** Followup studies (Rutter and Lockyer 1967; DeMyer et al. 1973) have emphasized that lower functioning infantile autistic children grown into adulthood generally do not resemble adult schizophrenics but more often appear mentally retarded. Higher functioning autistic adults also are not typically diagnosed as schizophrenic but generally have improved their social symptoms which, while stopping short of normal, are not typically those of an adult schizophrenic. Those studies that have looked into the childhood symptoms of typical adult schizophrenia have failed to find many examples of typical cases of infantile autism or childhood schizophrenia (Roff, Knight, and Wertheim 1976; Watt 1978). Many cases of infantile autism as adults have been cared for in both institutions for the mentally retarded and mentally ill where, according to Kanner (1973, p. 185), many “settled down to a life not too remote from a Nirvana-like state,” presumably meaning a continuation of the early social isolation.

However, Bender and Faretra (1973, p. 57), who included infantile autism under the rubric of childhood schizophrenia, stated that “child schizophrenia invariably leads to adult schizophrenia although it may be expressed in a wide variety of clinical pictures.” Also Fish (1977) indicated that adult and child schizophrenics are part of the same spectrum on the basis of common perceptual-motor integration problems. However, Piggott and Gottlieb (1973) found childhood schizophrenics did not resemble adult schizophrenics on several biological measures. Chess (1979) presented evidence that children with congenital rubella who developed autistic symptoms in the postinfancy period looked the same as those becoming autis-
tic during infancy. To her this was evidence that autism is a separate syndrome. Whether infantile autism is the earliest expression of schizophrenia needs yet to be verified or refuted through systematic longitudinal study and comparison of adult autistics with adult schizophrenics, a study that has never been done.

After comparing results of all developmental studies on childhood psychosis, we can state that there is a strong continuum effect in general intellectual ability of the four diagnostic groups, namely: infantile autism is lowest, later developing psychosis (e.g., schizophrenia in childhood) intermediate, and borderline states and nonpsychotic learning disability children highest. One possible connection between all the psychotic and borderline states in all chronological age groups is the presence of cognitive disability. Here again, there is a possibility of continuum effect, with autistics and child schizophrenics having the most severe degree of cognitive defect and schizoid and adult schizophrenia populations having less severe degrees. Trunnell (1965), using a Piagetian frame of reference, found that adult schizophrenic patients were like 7- to 11-year-old children in forming logical concepts. But all mentally retarded individuals without psychosis are also retarded in this respect, so the explanation for psychotic symptoms would have to lie at least partially in some other sphere or in a particular and unique cognitive disability.

**Demography**

Wing et al. (1976) compared all prevalence studies in England and Wales, the U.S.A., and Denmark. Those studies in which a total population or a wide range of handicapped children were surveyed with case-note inspection and interviews all revealed the estimated prevalence of autism to be between 4 and 5 children per 10,000 aged under 15 years. In contrast, those studies employing diagnosed cases only or using administrative records only produced lower prevalence rates. Ornitz and Ritvo (1976, p. 615) consider that all the published estimates are low because “autistic children younger than 4 and older than 6 are often misdiagnosed.” Chess (1977) has shown that the prevalence rate in children with congenital rubella is 741 per 10,000 which is roughly comparable to one estimate of the rate in severely subnormal children (see Lotter below). In Denmark, Haracopos and Kelstrup (1978) estimated that about 25 percent of mentally retarded children displayed “psychotic behavior.” Lotter (1978) examined 1,300 mentally retarded children in Africa and found 2.3 percent had some autistic-like behavior and only 0.6 percent were autistic according to Western standards. While it was not a true prevalence study, the numbers of autistics found were far fewer than expected; e.g., in England, 5 to 8 percent of all severely subnormal children would have some marked autistic behavior. In comparison to Western autistics, rocking, head banging, and complex object use were rare. In sex ratio, occurrence of epilepsy, and social status, the African group “was broadly comparable” to the Western group.

In New South Wales, 21.9 percent of all subjects diagnosed as autistic over a 20-year period had at least one foreign-born, non-English-speaking parent (Harper and Williams 1976). Greeks and Germans were overrepresented in this sample. Parker (1978) from Australia reported that the season of birth did not differ between autistics and the normal population.

In a study of autistic, cerebral palsied, mongoloid, and normal children, Ando and Tsuda (1975) from Japan found that autistics did not differ from the normal control group or from general population statistics in maternal age, birth order, or age interval between the subjects and their closest sibling. Autistics had a 4.95:1 male/female ratio in contrast to cerebral palsied 1.75, mongoloids 1.18, and normals 1.1. The data on maternal age, birth order, and male/female ratio were consistent with those of most other investigators in the 1970s from the United States and England (Kolvin et al. 1971; Ritvo et al. 1971a; Spence et al. 1973). In contrast, Allen et al. (1971) reported that mothers of autistics were significantly older than a normal control group matched for age of child, birth order of child, and position of child in the family. Also O’Moore (1972) found the age of mothers of autistics in Dublin, Ireland, to be higher than the national average (32 years vs. 28).

Previously, most studies agreed that parents of autistics come from higher socioeconomic groups than the general population or from control populations (Treffert 1970; Allen et al. 1971; Kolvin et al. 1971; O’Moore 1972; Cox et al. 1975). Types of indicators of higher socioeconomic status (SES) in the autistic groups were higher incomes, more professional and managerial jobs, home and car ownership,
and spacious housing. Ritvo et al. (1971a), however, found similar SES characteristics between autistic children’s families and those of children with another psychiatric diagnosis. Schopler et al. (1980) reported that in their sample of 264 autistics from North Carolina, 74 percent came from lower SES families.

L. Wing’s (1980) epidemiological survey of autistic, other psychotic, and mentally retarded children of southeast London suggests the reason that so many study populations of autistics appear to come from high SES families. She found no SES differences among the three groups studied but did find that the higher SES fathers of autistics were more likely than the other fathers to apply for service from clinics with a special interest in autism. Membership in the National Association for Autistic Children was also related to higher SES.

All measures of SES also have implications for the IQ of the heads of household, with those from higher SES groups having higher IQs than lower SES groups. Findings from the United States and Japan (Allen et al. 1971; Ki-kuchi et al. 1971; Florsheim and Peterfreund 1974) indicated that only fathers of autistic children may have greater than average intellectual abilities (mean IQs about 116) while the mothers’ mean IQs were somewhat lower. In contrast, one U.S. study (Wolff and Morris 1971) of only five sets of parents of autistics found the fathers’ mean IQ to be 107.8 and the mothers’ 109.8. In view of L. Wing’s findings, it seems necessary that all such studies of parental intelligence must be suspect unless care is taken to match comparison groups for SES.

Parents and Families

Parents of autistic children have been portrayed as cold, aloof, lacking in real warmth, and unstimulating to their children. Kanner (1949) described them as obsessive, perfectionistic, humorless individuals who used set rules as substitutes for life’s enjoyments. Severe early stresses to the infants such as parental rejection, separation, or maternal depression have been linked to autism.

Cox et al. (1975) investigated early stress events and parental warmth, responsiveness, and sociability in parents of autistic children with normal nonverbal intelligence and no evidence of neurological disorders with parents of matched dysphasic children. The parents’ groups did not differ in incidence of psychiatric conditions, housing, illness, finances, or interpersonal relationships. Ratings of emotional warmth and sociability were similar except that parents of autistics spent more time with friends. Thus the parents of autistic children were as sociable, demonstrative, and emotionally responsive as the parents of dysphasic children.

McAdoo and DeMyer (1978a) compared the Minnesota Multiphasic Personality Inventories (MMPIs) of parents of autistics with a random sample of parents being treated in an adult outpatient psychiatric clinic. If the parents of autistics had severe psychopathology, then their MMPI profiles should have been similar to those of psychiatric patients. Instead, significantly more psychopathology was found in the identified patients. MMPI profiles of parents of autistics were similar to those of a random sample of child guidance clinic parents. The failure to find differences between the two parent groups might be expected if there were two different groups of autistic children (Goldfarb 1970). If most of the parents of organic, autistic children were like parents of normal children, and parents of nonorganic children had significant psychopathology, then combining these two groups might minimize even dramatic differences. McAdoo and DeMyer (1978a) found that MMPI profiles were no different in parents of “organic vs. nonorganic” autistic children. Goldfarb, Spitzer, and Endicott (1976) also failed to demonstrate parent group differences in psychopathology and functioning.

Infant care practices have been hypothesized to reflect deviant parent personalities, and autistic children have been viewed as “victims” of extreme parent procedures. DeMyer et al. (1972b) found through interviews that parents of autistics and extensively matched normal children did not differ in infant acceptance, warmth, nurturing, feeding, and tactile or general stimulation. A control parent group of nonautistic handicapped children of lower SES was judged less warm and stimulating. Normal infants were recalled as more alert and responsive than autistic or handicapped infants who were similar to each other. In contrast, Massie (1978), from home movies taken before age 6 months, judged that mothers of “mixed” type child psychotics showed less adequate eye gaze and infant touching than normal controls. Feeding measures...
for the two infant groups were judged not to differ. On the face of it, these findings offer support for the nurture causation theory of child psychosis, but low rater correlations (.39 to .54) and important variations in film segments cast doubt on the adequacy of the method. Nevertheless, interview methods have inherent limitations and the study bears replication with better standardized film.

Using the Ferreira and Winter Unrevealed Differences Task, Byassee and Murrell (1975) found similar family interactions in normal and autistic groups, while parents of emotionally disturbed children had fewer spontaneous agreements than either group. Cantwell, Baker, and Rutter (1978b) demonstrated that parent-child interactions of families with autistic and dysphasic children were similar in quality and intensity except that the autistic children received more interactions. Using Ittleson Center Scales, the authors found group similarities in spontaneity, decisiveness, anticipation, control, and meeting children’s demands. The mothers of autistics had the same linguistic clarity, complexity, and grammaticality to their speech. In contrast, Goldfarb, Yudkovitz, and Goldfarb (1973) judged mothers of schizophrenics inferior to normal controls in labeling and describing objects. The differences between the two studies may be the result of the criterion groups (autistics, childhood schizophrenics), the control groups (dysphasics, normals) or the differences in interactional periods (90 minutes, 10 minutes). King (1975), rating chart descriptions, reported more double-bind interactions in mothers of autistics than in controls. He proposed that because of the mother’s double-bind attitude, the autistic child “wants to escape.”

After reviewing recent family research, McAdoo and DeMyer (1978b) concluded that, as a group, parents of autistic children (1) display no more signs of mental or emotional illness than parents whose children have “organic” disorders with or without psychosis; (2) do not have extreme personality traits such as coldness, obsessiveness, social anxiety, or rage; and (3) do not possess specific deficits in infant and child care.

**Parent Reactions to Symptoms.** An autistic child’s social withdrawal, unusual object use, and lack of communication is also likely to produce parental uncertainty about how to respond. The long-term persistence of these behaviors produces stress, which eventually may result in personality changes or psychiatric symptoms. McAdoo and DeMyer (1978a) found no support for this hypothesis after comparing MMPI responses of parents of autistics and parents of nonpsychotic child guidance clinic children. Nevertheless, in both England and the United States (Cox et al. 1975; DeMyer 1979) evidence was found that, while severe depression was rare, rearing an autistic child was stressful and that depressive symptoms were common in mothers after months to years of unsuccessful struggle to socialize an autistic child. Before pregnancy with the index child, mental illness was no more common in parents of autistics than in a well-matched normal control group (DeMyer 1979). In the Cox et al. study, fathers of autistics felt more often than fathers of dysphasics that child-rearing difficulties had altered their wives’ general behavior. Nevertheless, mothers of autistics were judged as warm to their children as comparison mothers.

DeMyer (1979) collated from extensive interviews the maternal reactions to the difficulties of rearing an autistic child. All mothers described greater than usual tension and most expressed guilt over what their role might be in producing the child’s symptoms. About one third said they had become unsure of their mothering capacity. Other reactions were anger over irritating behaviors, emotional hurt over the child’s lack of affection, feeling old before their time, demoralization, frustration, and curtailment of life’s enjoyment. Fathers, as a group, were less expressive of their emotions but nevertheless were deeply affected, saying that the mothers’ emotional pain in turn depressed them and they worried over the ultimate effect the continuing stress would have on their wives’ health.

About half the normal comparison children had a generally positive effect on the marriages, while the autistic child’s more irritating symptoms created “nervous tension” which precipitated more quarreling and thoughts of divorce. About 70 percent of the mothers said they needed more “moral support” from the husbands in attempting to fulfill their difficult maternal role. In many cases (45 percent), the parents’ sexual relations were rated as diminished chiefly because of the mother’s worry about the autistic child. When either marriage partner expressed strong bitterness to-
ward the mate, divorce was likely to follow. Some parents said that the decisions they made together about the autistic child ultimately strengthened their ties to each other. When fathers shared the mothers' concerns about the child and gave adequate 'moral support' (about 33 percent of cases), then marital ties were not diminished. The following were evidence of fathers' lack of support: critical attitudes, failure to praise, not taking the family on outings, never helping with the physical care, unreasonable or too harsh child discipline, and not talking with wives about problems or belittling their efforts to obtain help.

An excellent summary of effects of living with and seeking help for an autistic child was given by Marcus (1977, p. 398):

parents are worried and confused by their child's condition; management problems have exhausted them; although vaguely aware that their child is suffering a developmental delay . . . the possibility of retardation has seemed remote; . . . [many symptoms] are annoying and at times maddening; . . . unresponsiveness to affection has caused personal pain; parents are fatigued and tense and the family . . . [in] considerable conflict; in some instances, the parents have not been supported or provided with . . . advice they need.

Marcus advised professionals to engage the family in a collaborative effort while giving the parents "the main facts of psychosis" and a "frank appraisal" of the child's cognitive impairment. "Although usually upsetting to the parent . . . [this discussion] begins the process of reducing expectations to more realistic proportions." Also parents need direction and hope, qualities that derive from increased control . . . over their dilemma. The clinician should have practical solutions to real problems, be willing to work directly with the child, work for community involvement such as appropriate school placement, and, in general, take the side of the family in its struggle with a life-long disorder. [p. 398].

Despite confusion and anxiety, parents have been found by Schopler and Reichler (1972) to be able to assess their child's level of development with reasonable accuracy, a result confirmed by Wing and Gould (1978). Schopler and Reichler (1972, p. 398) interpreted the finding as suggesting that:

parents can be trusted in their evaluation of their child and in the information . . . they provide. . . . Professionals, however, because of their distrust of parental judgment, frequently superficially reassure, or even worse, humiliate parents by labeling them overconcerned and overanxious, or blame the child's problems on parental management. Such criticism without appropriate evaluation and specific instructions in improved management can . . . leave parents on their own to cope with extremely difficult developmental problems, discouraging their seeking of further help, and harmful delays of appropriate interventions.

Parental Cognitive Characteristics. Evidence is mixed as to whether parents of autistic children have different cognitive characteristics than controls. Schopler and Loftin (1969) showed that parents of autistic children who were interviewed about one of their normal children obtained the same Object Sorting Test (OST) scores as control parents, while those in psychotherapy attained higher scores. The authors concluded that anxious confusion about the autistic child could lead to atypical thinking. Netley, Lockyer, and Greenbaum (1975) found that more parents of autistic children were cognitively disordered than parents of nonautistics on the basis of the Grid Test of Thought Disorder scores. In contrast Lennox, Callias, and Rutter (1977), who used both the OST and Grid Test, found parents of autistics similar to parents of normals. The two tests failed to agree with each other, and there was no consistent association between thought disorder and anxiety. SES membership affected the scores. Clearly more work is needed to explain the contradictions (Netley and Lockyer 1978).

Genetics

In an excellent review of possible genetic components of infantile autism, Spence (1976) concluded (1) that comparisons of identical and fraternal twins suggested a possible genetic mechanism which would not involve a single gene or the chromosomes but would have to be polygenetic or multifactorial; (2) these hypotheses would be complicated to test because of insufficient family data and the different aggregation of symptoms in various autistics; (3) while weak evidence from twin studies supported a role for genes, no evidence pointed to a specific mechanism. Among those theories Spence hypothesized was that certain aspects, or symptoms, of autism were inherited while others were not.

Folstein and Rutter (1977) gave evidence tending to support this
hypothesis. They located 21 same-sexed twins, at least one of whom in each pair was diagnosed as autistic without associated diagnosable neurological disorder. Four of 11 sets of monozygotic (MZ) twins were both autistic, while none of the 10 dizygotic (DZ) co-twins were both autistic \((p = .055)\). Of most interest was that all 25 autistics met at least two of four criteria for cognitive and linguistic impairment in contrast to only six nonautistics. Nine of 11 MZ twins (in contrast to 1 of 10 DZ twins) were concordant for cognitive or language disability \(\text{(concordance rates 82 percent MZ and 10 percent DZ,} \ p = .0015)\). This difference more than met Spence’s (1976) ratio of 80:20 as a requirement for genetic involvement. Most importantly, the basic disability in autism was linked to a cognitive-language disorder, an idea convincingly demonstrated by Churchill (1972). (See also Language and Intelligence sections.)

In the 17 discordant pairs, the autistic twin suffered a biological hazard, usually perinatal, which did not affect the nonautistic co-twin. According to Rutter (1977), this latter finding is consistent with a multidetermined model of autism in which at least one of the factors is biological.

Kolvin et al. (1971) found that early onset cases differed from late onset cases in symptoms, outcome, and in familial rates of schizophrenia. Higher schizophrenia rates were found in families of late onset cases. The authors concluded that late onset child psychosis resembled adult schizophrenia genetically while infantile psychosis was unrelated to either adult schizophrenia or late onset child psychoses. Hanson and Gottesman (1976) agreed and gave some evidence that early onset psychosis closely resembled organic pathology of childhood. However, Lobascher, Kingerlee, and Gubbay (1970) found that alcoholism, psychiatric illness, and mental retardation occurred more often in families of autistics than in normal controls.

A family history of speech delay was reported in about 25 percent of autistic families by Bartak, Rutter, and Cox (1975) and by Rutter, Bartak, and Newman (1971). DeMyer (1979) found that siblings of autistics and of normal controls were not different in incidence of learning plus speech problems \(\text{(autistics 20 percent, normals 15 percent). When learning, speech, and physical defects were combined, however, sibs of autistics tended to have a greater incidence of such difficulties (autistic sibs 36 percent; normal sibs 21 percent). About 69 percent of autistic extended families had a member with a defect compared to 30 percent of normal families} \ (p < .01).\)

The low incidence of autism among the siblings of autistics appears to negate a genetic effect. When the estimated sibling incidence of about 2 percent \(\text{(Rutter 1968) is compared with the incidence in the general population of 4.5 per 10,000} \ (\text{Lotter 1966}), \text{however, the rate of autism in the siblings is clearly much higher.}\)

In conclusion, twin and other sibling studies lend weak support to genetic inheritance which may not involve the complete autistic syndrome, but only its language-cognitive components. As a cautionary note, Handford (1975) and Campion and Tucker (1973) have seriously challenged one key basis for the genetic hypothesis of schizophrenia: the increased concordance in MZ twins over DZ twins. The prenatal environments of MZ twins, contrary to common belief, may not be identical. About 70 percent of MZ twins \(\text{(in contrast to 30 percent of DZ twins) share a single chorion and therefore are subject to periodic reduction of blood supply to one or the other twin, thereby causing cortical infarction in near-term twins} \ (Towbin 1970). Also MZ twins have a greater incidence of neurological dysfunction than DZ twins \(\text{(Campion and Tucker 1973). The near similarity in concordance rates for single chorions and cognitive disabilities in MZ twins (70 and 80 percent) and the lower but similar rates in DZ twins (30 and 10 percent) make it imperative that the incidence rate of single chorion be determined in any twin study.}\)

As Handford (1975, p. 193) pointed out, “without corroborations of the twin studies, the case for a genetic basis of schizophrenia is greatly weakened.” The same can be said about infantile autism.

**Measured Intelligence**

In the largest followup study to date of intelligence estimates from standardized tests, DeMyer et al. (1974) tested 135 autistic children \(\text{(mean age 5.32 years) at initial evaluation and 70 of these same children 6 years later} \ (\text{mean age 11.45 years). In the preschool years, about 74 percent had a general IQ below 52 and only 2.6 percent had an IQ above 85. While “performance” IQs were higher than verbal IQs, nevertheless 78 percent of autistics had performance IQs below 68. Initial and followup IQs correlated .70} \).
(p < .001); and most of the children, even those who had shown considerable social improvement, remained in the retarded range. The mean IQs of those 25 autistic children who received several years of treatment and special education were the same at followup as the 30 untreated cases. This study confirms Hingtgen and Bryson’s (1972) summation of the 1960s: (1) Most psychotic children score in the mentally retarded ranges; (2) IQs are remarkably stable over time; (3) splinter skills do not reduce the validity of IQ scores; and (4) IQ scores are predictive of outcome. Two separate investigations (Rutter and Lockyer 1967; DeMyer et al. 1973) have found that an IQ below 40 is predictive of a poor outcome. While not all children with IQs above 60 or 70 have a good outcome, the ones who make accelerated intellectual progress and make a better social adjustment come from this higher IQ group.

Early onset cases may have lower mean IQs than later onset cases. Kolvin, Humphrey, and McNay (1971) reported that 51 percent of 47 early onset cases were either “untestable” or had IQs under 50 in contrast to about 3 percent of 30 late onset cases. Pollack et al. (1970) found the mean IQ of a mixed group of autistic and schizophrenic children (n = 76) to be 70.5, with 72 percent of scores below 90. The 113 siblings’ mean IQ was 111.6.

In a search for an intelligence testing instrument that would meet the testing needs (i.e., wide scatter and infancy basal mental ages) of the young autistic child, DeMyer, Barton, and Norton (1972) culled items from 13 standardized tests for infants and children. The “easiest” items in each of five classes could be successfully performed by infants, and the “most difficult” items could only be performed by children with a mental age of 6 years and higher.

Beginning with the easiest items and reinforcing for attempts to perform, the authors found that nearly every preschool autistic child was testable and that a profile of mental ages could be derived for language, motor, and perceptual-motor modalities. Of 97 consecutive referrals, nonautistic subnormal children tested higher than autistics in all modalities. The autistics did relatively better in perceptual-motor and motor tasks than in intellectual and verbal tasks. Lowest scores were verbal abstract reasoning and highest scores in fitting and assembly tasks—that is, Seguin formboard and Wechsler Intelligence Scale for Children (WISC) object assembly and block design subtests. In followup testing, mean age 11½ years, the entire WISC profile could be successfully used with only about 28 percent of autistics, while the remainder had to be tested with a battery suitable for children with mental ages below 6 years. Again at followup, autistic children’s highest scores were in object assembly and block design and the lowest in verbal comprehension.

Lockyer and Rutter (1970) first demonstrated in autistics, whose mental age was high enough to be testable using the WISC, that autistics achieved lower comprehension and higher block design and digit span scores than a control group of nonpsychotic behavior disturbed controls. The autistics were inferior to the controls in the Vineland Test of Social Competence. Lockyer and Rutter (1970, p. 152) hypothesized that autism represents “a central disorder of language and of perception of sounds.” Walker and Birch (1974) found in 120 schizophrenic boys, ages 10 to 15, that performance IQ was superior to verbal IQ in those with WISC IQ over 75. In children below IQ 75, verbal scores were relatively better than performance scores. Tymchuk, Simmons, and Neafsey (1977) compared WISC subtest scores between 20 adolescent autistics and 20 other extensively matched children of mixed nonpsychotic diagnoses (mean IQ: autistics 88, contrast group 89). The autistics achieved lower comprehension scores and higher block design scores than nonpsychotics. The autistics showed significant subtest variability with comprehension, coding, and picture arrangement being lower and digit span and block design being higher.

Profiles of intelligence test results meet objections that a single IQ score in face of the wide scatter found in many autistic children is not useful. While the general IQ is a powerful statistic in predicting the overall illness outcome, the more useful statistics in planning education programs for an individual child are the individual points of his profile of test scores.

In conclusion, the intelligence of each autistic child should be estimated using tests that are within his capability. The goal should be not to diagnose mental retardation versus infantile autism but to judge the degree of intellectual retardation or adequacy in the various facets of intelligence. As Wing (1979) pointed out, one of the biggest advances of the last
decade has been an increasing awareness of how autistic children compare with developmentally normal and other handicapped groups. Standardized intelligence tests have provided one good yardstick.

**Language**

Gross disturbances in language development are a generally accepted major characteristic of infantile autism. Indeed, some researchers regard disorders in language coding and speech to be the primary defect in autism (e.g., Rutter, Bartak, and Newman 1971; Churchill 1972, 1978). In an extensive study of 47 boys, aged 5 to 10, who all possessed a severe developmental language disorder with no demonstrable neurological dysfunction or hearing loss, the British group of Rutter and his colleagues (Bartak, Rutter, and Cox 1975) initially reported that within this group, those boys diagnosed as autistic had more deviant language development, more severe comprehension defects, and also showed more defects in social usage of the limited language that they did have. Since few differences were seen in the groups with regard to patterns of nonlinguistic skills, they concluded that a language disability is probably necessary for the development of the behavioral syndrome of infantile autism.

This group continued their reports on the same population of boys over the next few years (Cantwell, Baker, and Rutter 1978a), and the data have recently been reviewed by Rutter (1979). He formally broadens the concept of a central language deficit to be included under a cognitive deficit manifested by impaired language, sequencing, abstraction, and coding functions. When compared to a group of dysphasic children, the autistic children did not differ significantly in visual-spatial perception, articulation, or syntactical skills. Marked differences in the two groups, however, were observed in five areas: Autistic boys scored lower on understanding of language and language expression, lower on imaginative play and understanding of gesture, lower on spontaneous speech and speech for social communication, and higher on echolalia (especially self-echolalia and delayed echolalia); they also exhibited a highly variable pattern in verbal scale scores on the WISC. Rutter concludes that a particular kind of cognitive/linguistic deficit fundamental to autism is as much associated with language deviance as it is with language delay.

The lack of spontaneity in speech, even in those autistic children with some communicative speech, has been noted by many investigators. The Illinois Test of Psychological Abilities was used to assess and compare language-related functioning in 20 autistic and 20 retarded children (Prior 1977b). In this Australian population, it was found that higher functioning groups of both diagnoses differed from lower functioning groups overall; but for both high- and low-functioning autistic children, verbal and manual expressive performance was particularly impoverished and indicated a severe deficit in spontaneous communicative ability. The absence of spontaneous protodeclarative gestures, a preverbal form of intentional communication occurring around the first year of life for normal infants, in a group of 12 mute autistic children suggested a qualitatively distinct pattern of prelinguistic development (Curcio 1978). This severe paucity of gestural usage by autistic children also had been pointed out by others (Wing 1971; Bartak, Rutter, and Cox 1975).

Shapiro's work (Shapiro, Chiarandini, and Fish 1974; Shapiro and Huebner 1976) led him to conclude that various groups of autistic children can be categorized according to their language deficits, which he regards as central to the developmental disorder. In a 1976 followup study of five autistic children during adolescence, speech samples of the children between the ages of 12 and 19 were analyzed and compared to earlier speech patterns (8–12 years). The speech patterns of the children when older showed the expected varying combinations of constraint in length of utterance, echoing, syntactic disturbance, semantic concreteness, context inappropriateness, and disorders of prosody. The most dramatic finding was that early descriptions of these children were reliably matched to later speech samples by 10 psychiatrists (using blind matching techniques), indicating a continuity in style of speech deviance. Simmons and Baltaxe (1975) have also hypothesized the existence of distinct linguistic subgroups of autistic children, perhaps related to disabilities of rhythmic and prosodic perception.

Restricting their investigations to verbal autistic children, Bartolucci and his colleagues have made a comprehensive study of the language of these English-speaking Canadian children compared to that of mentally retarded and nor-
normal children, all matched for nonlinguistic mental age (approximately 6 years: Leiter Performance Scale). Autistic children used correct verb tense only 8 percent of the time during a structured picture and toy test session vs. 80 percent and 60 percent for normal and retarded children, respectively (Bartolucci and Albers 1974).

When the free speech syntax of autistic children was compared to retarded and normal groups, the former had lower developmental sentence and grammar complexity scores and higher error rates (Pierce and Bartolucci 1977). The grammatical system of the autistic group did appear rule governed, but simpler in construction than that observed in the other two groups. Although autistic children showed a more extreme and global delay in language development, they appeared similar to retarded children with regard to simple phonological perception or production, as well as in their frequency of omission of 14 normally occurring grammatical morphemes (Bartolucci and Pierce 1977; Bartolucci, Pierce, and Streiner 1980; Bartolucci et al. 1976).

Baltaxe and Simmons (1975) found some of the same deviances in their sample of verbal autistic adolescents. When compared to Down's syndrome adolescents, the deficits in the retarded group centered primarily in the area of articulation and syntax, whereas the autistic group's deficits related primarily to prosodic features of language, semantics, an inability to switch linguistic codes, and a manifestation of echolalia. Regarding articulation, Boucher (1976b) also found her sample of verbal autistic children to have superior ability to both retarded and dysphasic control groups.

Because of the prominence of echolalia in the speech of autistic children, this topic has fascinated many investigators. In a series of studies on echoing in autistic children, Shapiro and colleagues (Shapiro, Roberts, and Fish 1970; Shapiro 1977; Shapiro and Lucy 1978) have been able to make the following observations: (1) The total number of responses of a child who echoes is greater than that of a comparable nonechoing child at a similar developmental stage; (2) the mean length of utterance of echoes is longer than the mean length of other responses because echoes bypass intrinsic coding devices; (3) echoing occurs when a question taxes the child's receptive understanding; (4) echoes tend to cluster once an echo chain is set in motion; (5) there is a difference in reaction time for echoing and nonechoing (spontaneous) responses in autistic children, whereas this is not seen in normal children.

An experiment by Carr, Schreibman, and Lovaas (1975) demonstrated that echolalic children were most likely to echo questions and commands to which they had not yet learned an appropriate response, but rarely echoed questions and commands to which they had learned an appropriate response. Further, once an echolalic was taught an appropriate response to a previously echoed question, the child no longer echoed the question on future occasions. Although it appeared that echolalia could be reduced by this method, it was judged not feasible to teach a child a response to every verbal stimulus that might be encountered. Thus, in a subsequent study (Schreibman and Carr 1978), two echolalic children were taught to respond to a set of previously echoed questions with the sentence, "I don't know." At the same time, their nonechoing responding to those few questions to which they already had an appropriate response was maintained. Then the children were tested on a different set of previously echoed questions to see if the treatment intervention generalized to the new questions. It was found that each child discriminated correctly between those questions that had previously been echoed and those that had not. Followup probes showed that treatment gains were maintained at least 1 month later.

Other investigators have viewed echolalia in autistic children as a manifestation of late-onset imitative speech related to the central language disorder in autism (Philip and Dyer 1977). If echoic memory for words is regarded as a peak ability in the more able autistic child (Boucher 1978), similar to digit-span ability, this near normal facility, in contrast to most other behaviors at levels far below normal functioning, could account for the fixation at this stage of normal language development. In an interesting study comparing a group of 12 autistic and 12 dysphasic boys of matched ages (the same population as used in the Bartak study; see above), the data confirmed that echoing is prevalent in both these types of children and that the two groups cannot be distinguished by the total amount of echoing (Cantwell and Baker 1978a). The only significant differences between the two groups were in the amount of inappropriate repetitions of self and the amount of delayed echoing. For both of these types of echoes, the
incidence was higher in the autistic group, but there were no differences between the groups in the amount of exact, reduced, expanded, mitigated, or congruent echoes.

A number of attempts have been made to teach sign language to autistic children, especially to that group which shows little progress in expressive or receptive verbalizations even after intensive behavioral therapeutic intervention (Webster et al. 1973; Devilliers and Naughton 1974; Creedon 1975; Fulwiler and Fouts 1976; Benaroya et al. 1977; Carr et al. 1978). The majority of these investigations suggest that while the results are highly variable from child to child, children who have some imitative verbal skills before sign language training show gains after this training; whereas mute children, who usually do learn some signing, rarely learn to talk (Miller and Miller 1973; Salvin et al. 1977; Carr 1979). There is also some indication that abstract concepts, syntax, and generative skills can be taught through sign language, and that some children do show increases in spontaneous communication, decreases in self-stimulatory behavior, and some improvement in socialization after sign training (Casey 1978; Carr 1979). Despite the promise indicated by some of the earlier reports, however, sign language has, by and large, not produced the dramatic gains in language skills in autistic children that some researchers at first suggested. On the other hand, sign language or other types of nonspeech language training (McLean and McLean 1974; LaVigna 1977; Churchill 1978), as well as automated methods (Colby and Kraemer 1975; Hargrave and Swisher 1975), are all successful in increasing the total behavioral output of the autistic child, and to this extent should be considered for incorporation into the treatment program designed for each autistic child. In fact, recent evidence strongly suggests that a multisensory approach, combining both verbalization and sign techniques, is more effective than either approach by itself (Brady and Smouse 1978; Benaroya et al. 1979; Konstantareas, Webster, and Oxman 1979; Barrera, Lobato-Barrera, and Sulzer-Azaroff 1980).

Two widely accepted clinical impressions of the language of autistic children can no longer be considered tenable as a result of research conducted in the 1970s. It has been frequently stated that the autistic child’s language is atypical because of the manner in which his parents respond to his attempts to communicate. While many parents do develop strategies of communication in an attempt to circumvent the language disabilities of their autistic children (Ricks and Wing 1975), numerous studies have yielded no evidence that the speech of parents had a detrimental effect on that of their autistic children (Frank et al. 1976; Cantwell, Baker, and Rutter 1977; Cantwell and Baker 1979). An interesting sidelight to this issue is the observation that the child’s own verbal behavior provides a significant source of sensory reinforcement in autistic as well as in normal children (Lovaas et al. 1977). The second clinical view was that autistic children refuse to use the pronoun “I” because of a disturbance in perception of self. Bartak and Rutter (1974) showed that autistic children imitated pronouns at the ends of sentences, including “I,” no matter what pronouns were used. Silberg (1978) carried this research a step further and was able to demonstrate that in her sample of autistic children, those possessing some of the least verbal behavior used the pronoun “I’’ more frequently than the second or third person pronouns.

The extreme deficiencies in abilities for concept formation (Noach 1974) and the overwhelming dependence on the use of imitation as a major strategy in linguistic coding (Shapiro and Kapit 1978) in those autistic children who do possess some language skills present a rather pessimistic outlook for the eventual development of true spontaneous speech in the majority of autistic children, even with intensive behavioral procedures (Lovaas 1977; Carr 1979; Goetz, Schuler, and Sailor 1979; Prior 1979). In spite of this generally poor prognosis, the extensive studies of language and language training in autistic children certainly are justified by the improvements in communication that have been achieved (see Treatment section) and by the possibility for a better understanding of language development in both autistic and nonautistic disorders.

**Perceptual Process**

The extreme difficulty that most autistic children experience with even the most rudimentary intellectual and language developmental tasks has often been attributed to underlying perceptual disturbances. Indeed, studies in the 1960s strongly suggested this as a possible source for the autistic child’s behavioral deficits. Has this...
early suggestion been supported by the past 10 years of research? Some of the major experiments will be reviewed in order to answer this question.

One of the new areas of perceptual research with autistic children since 1970 has been the concept of stimulus overselectivity as proposed by Lovaas and his colleagues. This phenomenon refers to the fact that autistic children tend to respond only to a few cues from a larger range of available cues in a learning task. In an initial study (Lovaas et al. 1971), three groups of children (autistic, retarded, and normal) were rewarded for responding to a complex auditory-visual-tactile set of simultaneous stimulus cues. A discrimination having been established, elements of the complex stimulus were presented to assess which aspects of the complex had acquired control over the child's behavior. The autistic children responded to only one of the cues, whereas the normals responded uniformly to all three, with the retardates functioning at a level in between. When the stimulus complex was simplified to only two types (white noise and red floodlight), the autistics still demonstrated stimulus overselectivity because seven out of nine subjects responded to only one of the components (Lovaas and Schreibman 1971). In both studies, however, the previously nonfunctional stimulus was made functional when trained separately.

Although the early studies indicated that reinforcement during successive training trials was instrumental in reducing stimulus overselectivity, a later study (Schreibman, Koegel, and Craig 1977) demonstrated that training per se, rather than reinforcement, was an important factor. Nineteen autistic children were trained on a discrimination task with a cue complex composed of two visual cues. When they reached criterion, they were presented a testing phase involving probe trials in which the cue components were presented singly. The following results were obtained: 16 children initially showed some overselectivity, but 13 of these children decreased their level of overselectivity within less than 48 probe trials. Since there was no reinforcement for responses to the single component during probe trials, and yet a reduction in overselectivity occurred, the results suggested that repeated exposure to testing, rather than reinforcement, was the important factor in reducing overselective attention to cues. This finding is especially interesting because overselectivity was also significantly reduced when partial reinforcement (variable ratio 3) was used during part of the training, compared to continuous reinforcement used throughout the training (Koegel et al. 1979).

Additional studies have shown that stimulus overselectivity is clearly related to a lowered mental age (Wilhelm and Lovaas 1976) and thus is probably closely associated with behavioral retardation across different types of children rather than being a characteristic response tendency of only the autistic child (Koegel and Lovaas 1978). However, it may still reflect a critical perceptual deficit in infantile autism, even though its initial cause may differ from that in nonautistic retarded children.

It should be noted that at least one negative study has been reported (Litrownik et al. 1978) which suggested that when autistic children are compared to normal children of the same approximate mental age, they perform more similarly to the normal children on matching-to-sample tasks than do Down's syndrome children. These researchers claimed that their results indicated that overselective attention is more characteristic of retardate than autistic responding, with the autistic response deficits attributable to a possible stimulus control or lack-of-retention factor that develops over a series of training trials.

Since this study had many procedural differences from the previous experiments, the data are subject to multiple interpretations, but they do point out the need for further controlled studies of the overselectivity phenomenon in autistic children.

Perhaps related to the stimulus overselectivity response in autistic children is their apparent inability to use prompt fading techniques to facilitate learning. Prompt fading (the introduction of an additional cue to aid discrimination, with the gradual removal of that cue when correct responding occurs with high frequency) has been used with some success for teaching simple and complex discriminations to retarded children. When used with autistic children, however, it appears to slow learning rather than improve it. Schreibman (1975) reported that whereas autistic children usually failed to learn stimulus discriminations without a prompt, they always failed to learn with an extra-stimulus prompt (the type of prompt fading described above). When within-stimulus prompts were used (an exaggeration of the
relevant component of the training stimulus, thus not requiring the child to respond to multiple cues), the children usually learned the discrimination task (similar to the study of Sherman and Webster 1974). Those results were confirmed by later studies (Koegel and Rincover 1976; Arick and Krug 1978) and indicated that extra-stimuli prompt fading was actually detrimental to learning in autistic children, and even had a negative influence on transfer of therapeutic training effects from one setting to another (Rincover and Koegel 1975). Extensive studies of the important variables in prompt fading that might reduce the attentional requirements for discrimination learning in autistic children, such as the recent experiments of Rincover (1978b), are needed to understand better the bizarre nature of multiple stimulus organization in these children and to facilitate the design of more effective training programs. Errorless criterion-related stimulus shaping (rather than stimulus fading) should also be considered as a potentially useful procedure with autistic children (Eitel and LeBlanc 1979).

There is some evidence that autistic children impose their own perceptual patterns on incoming sets of stimuli. In two studies of pattern detection (Frith 1970a, 1970b), one dealing with immediate recall of auditory sequences and the other dealing with reproduction of color sequences, when normal and autistic children of comparable performance levels were tested, the latter subjects tended to show a marked response bias, imposing their own independent patterns on input stimuli rather than using the structures present in the stimulus patterns. Subsequent studies have supported these conclusions (Frith 1972; Hermelin 1972; Hermelin and O'Connor 1971). Also indicative of the unusual nature of perceptual processing in autistic children is the fact that simple photic stimulation can serve as a powerful reinforcer for lever-pressing in relation to the frequency of stimulation, which was not the case for retarded children (Frankel et al. 1976). Similar results were also reported for vestibular stimulation (Freeman, Frankel, and Ritvo 1976).

Multiple and complex cues pose special problems for autistic children. Although autistic subjects were able to successfully acquire a simple object discrimination learning set (Prior and Chen 1975), they failed to solve a conditional matching learning set problem requiring the use of an abstract symbol as a cue for the correct response (Prior 1977a). This failure was in contrast to the success of their normal and retarded counterparts, all three groups being matched for mental age (about 5 years), and demonstrated the difficulties that autistic children have in dealing with complex symbolic input. When presented double cue visual discrimination problems (using varying stimuli of color, form, and size) on a modified Wisconsin General Test Apparatus, nonverbal autistic children performed significantly more poorly than either verbal autistic or Down's syndrome children (Kovattana and Kraemer 1974). Using a stimulus generalization paradigm, autistic children performed as well as normal children in a test with single stimuli, but they overgeneralized when tested with complex stimuli by responding to fewer features of the stimulus (Fein, Tinder, and Waterhouse 1979).

Abnormalities in audition have been found in some groups of autistic children (Chess 1977; Hayes and Gordon 1977). In a group of dysfunctional autistic-like children, Condon (1975) reported multiple responding to auditory stimuli as if the children were perceiving the sound as being presented more than once. He also saw dysynchronous or awkward overall behavioral response organization in these children. This latter finding was supported by the research of Oxman, Webster, and Konstantareas (1978), but they could not replicate the multiple responding observation. Further indirect evidence for atypical auditory perception was provided by Fassler and Bryant (1971), who found that reduction in auditory input through the use of ear protectors improved classroom attention and task performance in autistic children.

Disturbances in perceptual input mechanisms could be related to cross-modal association and/or deficits that some studies find evidence for in autistic children. In addition to severe problems with learning auditory-visual associations, Bryson (1972) found that most of the autistic children tested also had substantial visual-visual short-term memory deficits, an observation made earlier by Hermelin and O'Connor (1970). However, when autistic, retarded, and normal children were matched for mental age (approximately 5 years), there were no differences in visual short-term memory among the three groups, although recall deteriorated with increase in
the delay interval (30 to 120 seconds) for all groups (Prior and Chen 1976). The children also did equally well on a visual serial memory task. Further evidence for auditory-visual association defects was provided by Morton-Evans and Hensley (1978) in their study of four groups of children (autistic, aphasic, retarded, and normal) matched for nonverbal mental age (4.4 years). The autistic and receptive aphasic children were both slower in associating sounds with their visual counterparts, but the aphasic children overcame this deficit at a significantly faster rate than the autistic children. No significant differences were found among the groups of children in learning visual-visual associations. Abnormalities in developing specific visual-motor associations by autistic children have also been described (DeMyer 1975a; Fulker- son and Freeman 1980).

In a recent study, Koegel, Dunlap, and Dyer (1980) found intertrial interval duration to be directly related to frequency of correct responding. Intervals of 1 second led to higher levels of correct responding and improving trends in performance, whereas intervals of 4 seconds showed large numbers of errors with no sign of improvement even over a long series of trials. It should be noted that reinforcement was always presented immediately after the correct response; only the beginning of the succeeding trial was delayed. The activities that occurred during the intertrial intervals, such as self-stimulatory behaviors (Lovaas, Litrownik, and Mann 1971), could have played an important role in interfering with normal memory processes, or the data could indicate a deficit in the memory process itself.

Lack of eye-to-eye contact, failure to recognize family or self, and lack of interest in or withdrawal from interpersonal relationships are commonly described features of autistic behavior and have been hypothesized to be related to perceptual deficits. Although autistic children are clearly deficient in body imitation (DeMyer et al. 1972a), which strongly suggests a self-perception disturbance, the evidence for low frequency of eye-to-eye contact and visual self-recognition is negative. Churchill and Bryson (1972) failed to find any differences in 14 autistic and schizophrenic children when compared to matched normal controls in terms of looking at and approach toward a strange adult. Both groups also responded more to an attentive adult than to a nonattentive adult. Another study (Neuman and Hill 1978) found that autistic children above the age of 5 were capable of self-recognition and displayed an intense interest in their own images and attempted to manipulate and control them.

An overview of the evidence for severe perceptual deficits in autistic children appears to make a solid case for the presence of a significant disturbance in perceptual processing. Whether the underlying mechanism is related to an attentional factor, a complex perceptual processing mechanism, a memory factor, or some other variable, however, has not yet been clearly established. In addition, the relationship of these defects to mental retardation needs to be further delineated.

**Behavioral Characteristic**

Hingtgen and Bryson (1972) summarized behavioral studies of autistic children in the 1960s as follows: (1) Behavior remains stable and relatively unaffected by events or fatigue except that avoidance and stereotyped behavior may be related to success/failure ratio in structured tasks. (2) Repetitive, nonfunctional behavior with objects and their own bodies constitutes a large portion of the autistic child’s repertoire. (3) While there was conflicting evidence about whether autistics avoid eye-to-eye contact, it appeared that they look less than nonautistics at all environmental objects. When looking at adults they lack other behaviors considered indicative of attention in normal children. The behavioral studies of the 1970s were few in number, but the findings were important because the authors took care to look into the developmental nature of the symptoms they studied.

**Play.** The poverty of symbolic play seen regularly in infantile autism was postulated by Wing et al. (1977) to come from an inability to abstract concepts from experience and to store these abstractions in symbolic form. From structured parent interviews and direct child observations of 108 autistic and retarded children, there emerged three types of play groups: (1) 43 with flexible and varied symbolic play, occurring only in those with language ages above 19 months and in no autistic child; (2) 23 with stereotyped, repetitive coping play; and (3) 42 with no symbolic play, all of whom had a language age below 20 months. Most chil-
Children in groups 2 and 3 were autistic or had "marked autistic features." Thus the conditions of stereotyped play (found also in normal 2-year-olds) was related both to the diagnosis of autism and to developmental age.

Observing five autistic boys in four different environments, Black, Freeman, and Montgomery (1975) confirmed the repetitive, nonfunctional object use and the paucity of peer interaction. In a confined space, the boys "modeled and imitated" at low developmental levels, but only two children engaged in any mutual play which nevertheless was parallel in nature and thus not higher than a mental age of 2 years.

Pantomime, Hand Use, and Imitation. Normal children of 3 years pantomime nonabstractly, and at 6 years they begin to represent an object abstractly. In a group of 24 "psychotic children" who met Creak's (1964) definition of childhood schizophrenia, Curcio and Piserchia (1978) found that subjects with echolalia could produce almost no abstract forms of pantomime while those with higher levels of speech produced some. Introducing a model increased the pantomimic scores. Those who discriminated a teacher from a child in a drawing task attained higher pantomime scores. All children were below the pantomimic norms for their ages.

Abnormal hand gestures, including fixed posturing and movement gestures such as flapping, clapping, rubbing, and pinching, were displayed by 16 of 18 autistic children during uncontrolled environmental conditions (Masagatani 1973). Mental retardates often hold their thumbs in continuous flexion (Peiper 1963), a condition also found in 9 of 18 autistics. The other half displayed conditions resembling a partial primitive grasp. Those children with fewer abnormal hand movements had more spontaneous speech. Masagatani stated that the reflexive, clumsy, and fixed patterns observed in the autistic children (ages 2½ to 13 years) should have been replaced by more skillful voluntary movements by 12 months of age. The author advised that the term "primitive reflex patterns" with physiological implications should be applied to the autistics' abnormal hand gestures rather than the term "bizarre."

Imitation is an important activity in normal children because much learning comes from reproducing parental vocalizations and body actions. Douglas and Sanders (1968) found that failure to imitate before 3 years of age differentiated retardates from autistics. A more severe motor imitative dyspraxia was found in 12 autistic children in comparison to 9 subnormal nonautistic children (DeMyer et al. 1972a). This result suggested to the authors that most autistic children may have not only a severe abstract language defect but also a dyspraxia, both defects contributing to their inability to learn the meaning of and reproduce body language which forms a large part of the infant and the young child's communication mode with others. The authors also suggested that the basis of the praxia defect might be in poor visual memories or an inability to transfer a visual memory to the motor system (agnosia) and that such deficiencies might be one basis for failures of autistic children to think abstractly and to symbolize.

The seeing of common and different properties in the "mind's eye" of various objects is essential for abstract and symbolic mental operations. It is no accident that the colloquial term "I see" means "I understand." [DeMyer et al. 1972a, p. 284]

Maintenance of Sameness and Negativism. Among 32 psychotic children, 10 children with > 20 Rimland E-2 scores exhibited higher "sameness" scores which reflected a greater amount of stereotypy in general behavior in contrast to those children with < 20 Rimland scores (Prior and Macmillan 1973). However, two children with minus E-2 scores also had very high sameness scores. Those children with ability to create and maintain more complex sameness patterns appeared to have higher cognitive abilities as measured by presence of speech and school attendance than those children with less complex stereotypies.

Negativism, defined as the consistent avoidance of a correct response, was not demonstrated in any of 27 autistic children tested by Clark and Rutter (1977) contrary to findings of Cowan, Hodinott, and Wright (1965). Of all possible explanations for the difference, the authors felt that a lack of school exposure might be responsible for the negativism of Cowan's group since all of Clark and Rutter's subjects were well used to various kinds of school work. Successful task performance was related to language competence.

Motor and Perceptual-Motor Development. The gross motor development of many individual autistic infants and children has been described as within normal limits.
However, when groups of autistic children were compared with normal children, the autistic children showed significantly lower levels of motor skills. In 65 autistic children of preschool and primary school age, gross motor abilities, as tested by adequacy of stair climbing, were found to be below developmental norms (mean developmental quotient 52; DeMyer, Barton, and Norton 1972). When perceptual motor skills were tested by such standardized tasks as drawing geometric figures and ball play, autistic children were behind their normal counterparts (mean developmental quotients 40 and 42). Thus gross motor skills seem relatively more intact than complex eye-hand-body coordination skills. In a study of infants born to schizophrenic mothers in a state hospital, Fish and Hagin (1973) found that failure in hand-to-hand performances at 4 to 9 months of age was related to subsequent emotional illness at followup, age 10–11 years.

Nevertheless, superior drawing skills have been reported in individual cases (DeMyer, Barton, and Norton 1972). DeMyer (1975a) reported graphic skills as a splinter skill in about 3 percent of autistics. However, the commonest perceptual-motor splinter skills appear to be in fitting and assembly tasks, such as the Seguin form board, WISC object assembly and block design subtests (see Intelligence section). These tasks can be accomplished by recognition of similar form and outlines and by the picking up and release of objects, an infant motor skill. Whenever a more advanced motor skill or cognitive judgment, as in the coding subtest of the WISC or draw-a-person test, was called for, autistic children did relatively worse.

Parental Reports of Developmental Milestones. Two separate studies investigated developmental milestones of autistic children in comparison to matched normal control groups. Using a parental paper-pencil questionnaire, Ornitz, Guthrie, and Farley (1977) found in 74 autistic children, mean age 45.2 months, that nearly all aspects of development were delayed. Receptive and expressive speech development was delayed beginning at age 2 months. By age 6 months, continuing through the second year, significant motor delays were reported—for example, holding head erect, first rolling onto back, sitting without support, crawling, and walking. An interesting finding of this carefully done study was that autistic children without a concomitant neurological condition did not differ from those who had such overt pathology, illustrating the difficulties of basing presence or absence of brain dysfunction solely on outward signs of such pathology.

Using face-to-face interview methods and all available records such as baby books and photographs, DeMyer (1979) also found 33 autistic children as a group to be delayed in passing developmental milestones. In addition to delay in language and the better known motor milestones, autistics were behind agemates in use of eating utensils, drawing geometric figures, and dancing and rhythmic performances. Music skills in both listening and performance aspects were also behind the normal comparison group.

Even the autistic children with exceptional splinter skills such as "perfect" music pitch lacked other abilities such as good rhythm or hand coordination or cognitive abilities which thus made impossible an age-appropriate music performance. Most autistic children (90 percent) were interested in listening to music, but even in this regard they were less advanced than the matched normals. Autistic children generally preferred simple music while many normal children listened to a wide variety of music, including classical music. Only the autistic children with advanced tonal skills, about 9 percent of the sample, listened to classical music.

In conclusion, autistic children, like normal and retarded children, obey the laws of child development in that specific behavioral features such as the ability to pantomime, imitate, and engage in symbolic play and successful task performance are directly related to the mental age of the child. Some evidence has accrued, however, that autistic children, in contrast to nonautistic mental retardates, may have relatively more serious problems in (1) symbolic representation necessary to the development of symbolic play and abstract thought; and also (2) visual-motor dyspraxia which precludes the learning of pantomime and body imitation at levels sufficient to participate in everyday nonverbal communication. The extremely low developmental levels of both verbal and nonverbal communication in turn may be related to the phenomenon of psychotic social distance in the autistic population. The failure to find the trait of negativism in autistic children is of utmost importance. It indicates that the scores of autistic children on various developmental and in-
From the 1960s, Hingtgen and Hood psychosis. Investigators of complicated in some cases of child-
gestated to the authors that autism
ported that signs of neurological examinations, including
local evidence of brain disease,” 28
25 autistic children had “unequiv-
ly in autistic children than in nor-
and autistic children with 113
autistics they examined showed
signs of neurological dysfunction
(see Classification and Diagnosis
of Childhood Psychoses section).
In a comparison of 76 schizophrenic
and autistic children with 113
of their biological sibs, 70 percent
of index cases had soft signs and 6
percent hard signs while only 15
percent of sibs showed soft signs
and 2 percent hard signs (Pollack
Other authors using more cir-
cumscribed evaluation methods
have reported increased incidence
of “minor” physical anomalies
(e.g., low set ears, electric hair,
high arched palate) among psy-
chotic groups vs. normal groups
(Steg and Rapoport 1975; Walker
1977b). In 52 autistic children,
Campbell et al. (1978b) demon-
strated significantly more anoma-
lies than in either a sibling or
nonrelated normal control group
while the latter two groups did not
differ. A presumptive connection
exists between presence of minor
physical anomalies and first tri-
monther pregnancy complications.
Anomaly scores tended to be
higher in 17 patients with a history
of such complications than in 35
without them. A later study by
Campbell et al. (1980) reported
that young autistic children were
significantly shorter than a control
population.
Abnormal finger print patterns,
formed by the 18th week of gesta-
tion would signify that something
gone awry in the first or early
second trimester. Dermatoglyphic
patterns are largely under genetic
control, but other factors such as
rubella virus and thalidomide can
cause abnormal patterns. The fin-
gerprint patterns of 20 schizophre-
nic children were described by
Hilbun (1970) as not abnormal in
and of themselves, but the distri-
bution of the types of patterns was
significantly different than that of
a randomly selected normal popu-
lation (Alter and Schulenberg
1966). Walker (1977a) reported that
73 of 78 autistic cases showed one
or more dermatoglyphic
abnormalities, most of which were
extensive, in contrast to 43 of 78
normal patterns in a normal
group. Also, ridge pattern fre-
quencies differed markedly be-
tween Walker’s autistic group and
other diagnostic groups.

**Neurobiological Studies**

From the 1960s, Hingtgen and
Bryson (1972) concluded that
neurobiological factors may be im-
pliied in some cases of child-
hood psychosis. Investigators of
the 1970s using extensive neuro-
logical examinations, including
electroencephalograms, have re-
ported that signs of neurological
dysfunction appear more frequen-
tly in autistic children than in nor-
mal children and that autistic chil-
dren are much like nonpsychotic
subnormal children in this regard.
In Kolvin, Ounsted, and Roth’s (1971)
series from England, 54 per-
cent of infantile psychosis and 15
percent of late onset psychosis
cases had some evidence of brain
dysfunction. Gubbay, Lobascher,
and Kingerlee (1970), from West-
ern Australia, found 56 percent of
25 autistic children had “unequiv-
ocal evidence of brain disease,” 28
percent had probable brain dis-
ease, leaving only 16 percent with
no such evidence. The multiple
types of neurological findings sug-
gested to the authors that autism
is not “produced by the malfunc-
tion of a specific anatomical region
of the brain” and also “that only
the occurrence of the characteristic
types of behavior patterns seemed
to justify [the] separation diagno-
sically (of autistic children) from
any heterogeneous group of re-
tarded children” (p. 427). Harper
and Williams (1974) reported that
47 percent of 131 autistics had evi-
dence of organicity.
In studies from the United
States, DeMyer et al. (1973) using
a weighted scoring system showed
the mean brain dysfunction index
of 48 normal children to be 11.6, of
155 autistic children 45.9, and of 26
subnormal children 52.5. Only
14.2 percent of the autistics’ scores
fell within one standard deviation
of the normals. Knobloch and
Pasamanick (1975) reported find-
ings with similar implications and
stated unequivocally that all young
autistics they examined showed
signs of neurological dysfunction
(see Classification and Diagnosis
of Childhood Psychoses section).
In a comparison of 76 schizophre-
nic and autistic children with 113
of their biological sibs, 70 percent
of index cases had soft signs and 6
percent hard signs while only 15
percent of sibs showed soft signs
and 2 percent hard signs (Pollack

**Handedness and Right-Left Ori-
entation.** Normal children develop
an adult type hand preference at 3
to 4 years, while subnormal chil-
dren usually have mixed handed-
ness (ambidextrousness) until later
ages (Annett 1967). Apparently
autistic children resemble subnor-
mal children in this regard. Only
35 percent of preschool autistics
had developed a right-hand domi-
nance in comparison to 88 percent
of normal controls (Colby and
Parkison 1977). In children ages 6
to 19 years, right-hand dominance
was displayed by 59 percent of autistics and 74 percent of normals (Barry and James 1978). Dominant hand usage (whether right or left) was related significantly to chronological age in all children, showing its developmental nature.

Of a population of 80 schizophrenic boys (8 to 12 years old and IQs above 70), 32 percent always used their right hand, 12 percent always used their left hand, and 55 percent were mixed (Walker and Birch 1970). In contrast, only 8 percent of normal boys had mixed dominance (Belmont and Birch 1963). Mixed eye/foot dominance was also more common in the patients. In tests of right-left awareness, 97 percent of normals and 62 percent of schizophrenics achieved all items correct. Among the schizophrenics, right-left awareness scores correlated positively with chronological age. The range of IQ scores was so narrow that no positive correlation between awareness and IQ could be expected to develop. The more informative test would have been correlation of mental age with right awareness scores. Wing (1971) also found problems with right-left orientation in 75 percent of speaking autistic children. Boucher (1977) showed slight but insignificant tendencies for sinistrality in autistics and dextrality in their parents.

In summary, right or left dominance and right-left awareness develops later in psychotic children than in normals but at rates similar to those seen in mental retardates. In one study in which perfect scores were required as evidence for presence of dominance, schizophrenic boys remained inferior to their normal counterparts through age 12. Thus we do not know whether psychotic children always remain behind normals in dominance and right-left awareness or whether they may catch up in later adolescence. Comparison of lateral dominance and awareness scores with mental ages would demonstrate more adequately whether incompetence in lateral dominance is related more to developmental level than to psychiatric diagnosis.

**Electroencephalographic (EEG) Studies.** The EEG has been used as a tool for studying the intactness of the central nervous system of autistic subjects. Like many such tools, the EEG must be used properly if results are to be trusted. Small (in press) assessed the technical adequacy and the findings of 13 studies reported in the 1960s and 1970s. Incidence of EEG abnormalities ranged from 10 to 83 percent, with an average of 52 percent. Only 3 of 13 studies met at least three of the four requirements for a technically adequate study, plus including control groups. In these three studies, autistics had as many or more EEG abnormalities than children with other psychiatric diagnoses. In all six studies in which a normal control group was provided, autistics had significantly more EEG abnormalities. Small concluded that “such data . . . have added significantly to the growing body of evidence that infantile autism is an organic disorder characterized by severe central nervous system impairment.” There is much disagreement about the kinds of EEG abnormalities and clinical correlations probably due “in part to technical difficulties in obtaining adequate records during waking, activation procedures, and sleep in extremely disturbed uncooperative children.” The differences might also be explained as reflecting differing amounts, types, and sites of brain dysfunction in various autistic children.

**Cerebral Asymmetry, Pneumoencephalograms, and Computerized Tomographic (CT) Scans.** Blackstock (1978) demonstrated that autistic children listened to music in preference to spoken material and generally preferred their left ears while normal controls had variable preferences. A second matched control group of nonautistic subnormal children would have given added information about the specificity of this phenomenon to autistic children. Blackstock believed his results supported the idea that autistics are “predominantly right hemisphere processors, a concept also supported in Prior’s (1979) review.”

Thorough neurological studies by Hauser, DeLong, and Rosman (1975) of 17 autistic children revealed consistent abnormalities only in the pneumoencephalographic (PEG) examinations. Fifteen cases showed “some enlargement of the left lateral ventricle (with respect to the right) and, particularly, enlargement of the left temporal horn . . . [which] reflected primarily flattening and atrophy of the hippocampal contours . . .” (p. 683). A variety of other PEG abnormalities appeared in 10 cases. Degree of temporal horn abnormalities, however, was not associated with severity of lan-

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3Because the neurophysiology of autism has been reviewed in detail by James and Barry (1980) for the *Schizophrenia Bulletin*, only a brief review is given here.
guage or behavioral symptoms, with presumed etiology, or with severity of intellectual deficit. The authors felt this finding supported medial temporal lobe disease being responsible for autistic symptoms. The lesion was thought to be "asymmetrically bilateral" but mainly located in the left hemisphere.

Using computerized tomography (CT) scans, Hier, Lemay, and Rosenberger (1978) found that 7 of 9 autistics had a right parieto-occipital region larger than the left in contrast to 11 of 41 nonautistic retardates (p < .02). This reversed pattern of asymmetry suggested to the authors a left hemisphere dysfunction in autistics. Mild ventricular abnormalities such as abnormal bifrontal dimensions in 5 of 17 of an autistic group, reversal of ventricular asymmetry in 6 of 17, greater than normal asymmetry in 6 of 17, and "clipping" of a frontal horn in 4 of 17 were reported by Damasio et al. (1980). Three patients had localizable lesions and five showed clear evidence of bilateral ventricular enlargement. There was no single abnormal pattern and those that were present were probably "consequent to a variety of (CNS) disease(s)."

Cerebral Spinal Fluid Studies. Cerebral spinal fluid (CSF) analyses by Young et al. (1977) for glucose, protein, cells, and folate revealed no abnormalities in 11 autistic children, 3.8 to 9.11 years old. The possibility that autism might be caused by a slow virus led these authors to measure CSF immunoglobulins. No abnormal levels were found, thus not supporting the slow virus hypothesis. As the authors pointed out, however, additional studies are needed since in many slow virus infections, CSF immunoglobulins are generally normal.

Antigen Studies. To test the viral etiology theory of autism, Stubbs and Magenis (1980) typed 20 autistic children and their parents for histocompatibility blood antigens types HLA-A and HLA-B and compared them with antigens of 757 controls. While no significant differences were found, the authors felt the autistic group was too small to come to any conclusions and that other new measures of the immune response should be used.

Neuropathological Studies. Darby (1976) located 29 literature cases of autism and four unpublished cases, all with documentation of neuropathological changes. Cerebral lipoidoses were found in 21 percent, tuberous sclerosis were found in 6 percent, and a variety of neuropathological findings occurred in other abnormal cases (54 percent). Darby believed that both degenerative child psychosis, formerly called Heller's disease, and other child psychoses "constitute the overt behavioral expression of a number of disorders ... analogous to the 'final common pathway' concept espoused by Bellak in 1958 for adult schizophrenics" (p. 49). In contrast, Hauser and Williams (1979) reported all three autistic cases studied showed no postmortem evidence of neuropathologic changes.

Diagnosable Neurological Conditions. It often occurs that many children have complaints that are originally diagnosed as due to a psychiatric disorder only to be rediagnosed later as neurological. Of 12 such cases presenting in a year's time to a hospital pediatric neurology service, two cases originally called "child psychosis" had the following ultimate neurological diagnoses: (1) sex-linked diffuse cortical sclerosis with Addison's disease and (2) subacute sclerosing panencephalitis following measles (Rivinus, Jamison, and Graham 1975).

In a "neuro-epileptic group" of 34 children (ages 5 to 14 years) with overt neurological disorders "above the brain stem," Rutter (1977) reported an increased incidence of hyperkinesis and psychosis over nonbrain-damaged children with psychiatric disorder (17 vs. 2 percent). In this group, psychosis was found more often in low IQ than high IQ cases.

Coleman (1979) has tabulated 149 cases of infantile autism accompanied by a diagnosable neurologic condition encompassing metabolic, infectious, chromosomal, structural, and nonspecific types. The most common individual illnesses of the 29 listed were phenylketonuria in 50 autistic cases and rubella in 29 cases, the latter clearly documented by Chess (1977). Autism also has been found in histidinemia, celiac disease, tuberous sclerosis, purine disorder, toxoplasmosis, congenital syphilis, encephalitis, infantile spasms, Cornelia deLange Syndrome, and cytomegalic inclusion disease, among others.

In the future, study of the neuropathology of phenylketonuria should reveal much about the connections among autistic behavioral symptoms, biochemistry, and anatomical sites of the brain lesions. Recent studies reveal that the buildup of phenylalanine is proba-
likely toxic to the brain (Rosenberger and Nigam 1979). In view of the high incidence of autistic behavior in phenylketonuric subjects, the anatomical substrate of autism might be made clearer through biochemical-anatomical autopsy studies of the phenylketonuric brain. Comparison should be made between brains of phenylketonuric people with and without autistic symptoms. A similar neuropathological study of congenital rubella brains with and without autistic symptoms to locate differences in the sites of lesions also might be productive.

**Biological Problems During Gestation, Birth, and Infancy.** A high incidence of postmaturity (65 percent) in a Western Australian sample of autistics was reported by Lobascher, Kingerlee, and Gubbay (1970). Also there were more complications of labor and more frequent neonatal abnormalities over normal controls. The latter finding was confirmed by DeMyer (1979) in the United States. Others reporting excess of perinatal risk factors were Bender and Faretra (1973), Pollack et al. (1970), and Ruttenberg (1970) while Bomberg, Szurek, and Etemad (1973) and Treffert (1970) did not. O’Moore (1972), in Dublin, Ireland, found a high incidence of pregnancies terminating unsuccessfully in mothers of autistics (8/25), some degree of fetal anoxia during parturition (21/25), and forceps deliveries (13/25).

In a detailed study of a large sample (105) of autistics admitted to Bellevue Hospital in New York City, Campbell, Hardesty, and Burdock (1978) found bleeding to be the most frequent complication of pregnancy (about 13 percent) which occurred more often with autistics than with their siblings. Using a weighted scoring system, the authors found that autistic children ($n = 46$) had more prenatal and perinatal complications than their normal siblings ($n = 20$) but fewer than the siblings with minimal brain damage ($n = 12$).

DeMyer (1979) identified a possible biological etiological factor in about 88 percent of cases in the first 3 years of life (troubled pregnancy 46 percent, difficult labor 37 percent, first year illness followed by behavioral change 15 percent, second year illness 18 percent, and third year illness 6 percent). About a quarter of the 33 children had more than one possible cause. Chronic constipation or diarrhea in infancy or early childhood was more common in autistics (75 percent) than in normal controls (33 percent).

**Implications of Neurobiological Findings.** A wide variety of neurobiological studies revealed that autistic children in comparison to normal children had more physical signs carrying the implication that something had gone wrong or could have gone wrong with the central nervous system. Studies encompassed such techniques as standard and circumscribed neurological evaluations, fingerprint patterns, hand dominance, right-left orientation, EEG, PEG, CT scans, neuropathology, early-life medical events, and analyses of body tissues and cerebral spinal fluids. In contrast, autistics and retardates had nearly equal signs of neurological import. Nevertheless, an anatomical and functional explanation must be found as to why some neurologically damaged children have the autistic syndrome and others do not. Logically we could assume that the lesion(s) in autistic children with normal performance IQs may be more subtle than in lower IQ children. Attempts to localize the lesions within any precise area of the brain of autistics have not been successful, although it does appear that the left side of the brain may be more heavily involved than the right side. Rutter’s (1978) conclusion that the lesion “must be bilateral” is given some credence. Localization studies are in their infancy and so far nonautistic retardate controls have been included in only one CT scan study. Such controls are a requisite for specifying neuroanatomical differences between autistics and retardates.

Those theorists who invoke perceptual inconstancy (see James and Barry 1980 for a review) as the basic neurological dysfunction have hypothesized that the lesion is located in the reticular activating system, vestibular and brainstem area. Data from most localizing studies suggest that theorists should begin to think about more widespread lesions in many cases that have affected not only thinking and language but have involved vaso-vegetative centers as well. The perceptual inconstancy theory is further weakened by observations that individual autistic children are quite consistent in the items they pass or fail on various developmental tests. Also while autistic children vary one from another on which neurological modalities are most dysfunctional, each child is quite consistent over time in his own dysfunctional modality (see especially the language studies of Churchill 1978).
One brain center that routinely seems fairly well spared in many autistic children is the motor system controlling large muscle activity. Most autistic children walk adequately if not completely normally. (See section on Behavioral Characteristics.) Most retarded children whose verbal IQs are as low as those of most autistics (profound and severely retarded) might be found either not to walk at all or to begin walking after 3 or 4 years of age. The autistic child's motor abnormalities are more consistently found in activities involving complex visual-motor modalities such as motor imitation and ball play. However, the better motor function in autistic children may itself be an artifact of diagnosis. If the motor system deficit is prominent, the child is likely to be labeled as cerebral palsy or profound retardation even though he has typically autistic behavioral features. Ordinarily such children are excluded from studies of autistic children.

To make further progress in locating the basic neurological dysfunction crucial for producing the autistic syndrome requires that investigators study multiple functions at the same time in populations of well-described autistics and mental-age-matched normals and subnormals. Only then will we begin to sort out which of the many disabilities of the autistic child differentiate this category from the nonautistic neurologically handicapped child. The site of the lesion(s) will be better known as our noninvasive brain lesion localizing techniques improve and as our neuropathological studies take advantage of phenylketonuria, rubella, and other neurological disease populations in which the incidence of autism is relatively high.

Biochemistry

In the last 10 years, many investigators have tried to elucidate possible biochemical etiologies of infantile autism. While various approaches have been used, including examination of metabolism of biogenic amines, amino acids, hormones, and trace elements, no specific biochemical markers have been found. Attempts to find such markers have been confounded by the lack of diagnostic specificity and by the numerous other factors such as age, sex, intellectual status, and activity level that also affect the various body biochemicals. While it is possible that biochemically distinct subgroups of psychotic children do exist, studies to date have not demonstrated them with any certainty.

The serotonergic system has received the most widespread attention by measurement of serum and platelet levels, platelet uptake and efflux studies, and assaying CSF and urine for serotonin and indoleamine metabolites. Studies of peripheral serotonin (5-HT) levels done by independent investigators suggest that 5-HT levels are higher than normal in autistic children. It is not possible to conclude that this increase is a specific pathogenetic factor in autistic illnesses because of the high individual variability of levels within diagnostic categories as well as the observation of elevated 5-HT levels in nonautistic individuals. The most compelling evidence supports the idea that blood serotonin levels are more strongly related to intellectual status than to psychiatric diagnosis. Higher levels of serotonin were found consistently in lower IQ groups. Thus it is not surprising that in autistic children who customarily also have low IQs serotonin would be increased over groups of normal or mildly subnormal children. This finding makes it especially important that IQ be controlled in all future studies of the serotonergic system in autistic and control populations.

Serotonin was measured by Campbell et al. (1974) in small groups of emotionally disturbed and neurologically impaired children matched for age, sex, and verbal IQ. No significant difference in group mean 5-HT level was found, but low IQ was related to elevations of serotonin. In a followup study by Campbell et al. (1975), childhood schizophrenics (n = 23) and normal controls (n = 16) were compared. Serotonin levels were not different, age and 5-HT levels were not correlated, and neuroleptic drugs did not seem to alter 5-HT levels. Interestingly, comparison of low IQ (IQ = 3-54, mean = 30.6, n = 16) to high IQ (IQ = 70-135, mean = 89.9, n = 7) intellectual function yielded 5-HT levels of .324 ± .214 µg/ml and .139 ± .102 µg/ml, respectively (p < .05). A similar relationship of low intellectual function to elevated 5-HT was shown by Hanley, Stahl, and Freedman (1977) in a study of autistics (n = 27), severe retardates (n = 25), mild retardates (n = 23), and normals (n = 6). Mean 5-HT levels for the autistics and severe retardates were significantly higher than those of the mild retardates and normals. Neither autistics and severe retardates nor mild retardates and normals were significantly different in 5-HT levels. "Hyperserotonemia" was then
defined as a 5-HT level more than 1.67 SD above the mean for the mildly retarded group. Using this definition, 30 percent of autistics, 52 percent of severe retardates, 9 percent of mild retardates, and 0 percent of normals were defined as “hyperserotonemic.” Another study correlating low intellectual function to alterations in 5-HT was contributed by Sankar (1977). He reported that autistics and retardates with psychosis had lower platelet 5-HT uptake than nonautistic schizophrenics and children with behavior disorders. Low IQ was correlated with low platelet 5-HT uptake in children with various diagnoses.

Ritvo et al. (1970) measured 5-HT and platelets in autistics and normal controls and found an inverse relationship between age and these variables. The group mean 5-HT level for autistics was significantly higher than that of age-matched controls, but the 5-HT platelet ratio was not significantly different. In a related study, Yuwiler et al. (1971) looked for circadian rhythmicity in 5-HT and platelets in autistics and nonautistics. The groups did not differ significantly with respect to 5-HT and platelets and no rhythmicity was demonstrated.

Several studies have been done on the nature of the relationship of platelets to serotonin metabolism because platelets are thought to be a model of serotonergic neurons in the central nervous system. Boullin, Coleman, and O'Brien (1970) reported that autistics (n = 6) had (1) significantly higher platelet counts (4.38 ± 0.36 × 10⁶ vs. 3.51 ± 0.22 × 10⁶, p < 0.05); (2) platelets that accumulated serotonin to a slightly greater degree; and (3) platelets with a decreased ability to retain 5-HT (as evidenced by a two-fold increase in efflux) when compared to normals (n = 6). In a followup study, Boullin et al. (1971) studied disturbed children (n = 10) and normals (n = 8). Using a three-fold increase in the normal rate of 5-HT efflux as a criterion for autism, they found a biochemical correlate that agreed with the Rimland E-2 score prediction for autism in 9 of 10 cases. The discordant case was a child rated as autistic by the Rimland score but who was not observed to have an elevated rate of efflux.

Groups of autistics, hospitalized comparison patients, and normal controls were studied by Yuwiler et al. (1975). While autistics were found to have significantly higher whole blood 5-HT levels and 5-HT platelet ratios when compared to normals, no significant differences in uptake or efflux of 5-HT were shown among the three groups.

Higher platelet serotonin levels were confirmed by Takahashi, Kanai, and Miyamoto (1976) in a study of autistics (n = 30), nonautistics (n = 45), and normal controls (n = 30). Autistics had higher levels than controls (980 ± 357 vs. 807 ± 202 ng 5-HT/mg protein, p < 0.025). Nonautistics were not significantly different than either of the other groups.

Hyperactivity was correlated to increased platelet 5-HT in the nonautistic group. Eleven of 45 nonautistics were rated as hyperactive, and this subgroup had a higher 5-HT level compared to the remainder of the group (1,012 ± 420 vs. 745 ± 288 ng 5-HT/mg protein, p < 0.05). They suggested that hyperactivity might be a factor in elevated serotonin levels seen in some disturbed children.

Studies of platelet monoamine oxidase (MAO) have generally agreed that there is no significant difference in the activity of this enzyme in disturbed children (Belmaker, Hattab, and Ebstein 1978; Boullin et al. 1975; Campbell et al. 1976a; Cohen, Young, and Roth 1977; Lake, Ziegler, and Murphy 1977; Roth, Young, and Cohen 1976; Takahashi, Kanai, and Miyamoto 1977). Roth, Young, and Cohen (1976) did find that females had higher MAO activity than males (29.96 ± 1.34, n = 42 vs. 22.05 ± 1.39, n = 37, nmoles/mg protein/hour, p < 0.001). Also, they noted that among females, younger individuals had higher MAO activity.

Another subject of interest in the biochemistry of autistic children has been the catecholamines. Young et al. (1978) studied urinary free catecholamines in normal (n = 9) and autistic (n = 5) boys and found autistics had markedly lower levels of urinary amines (10.8 ± 2.3 vs. 32.6 ± 18.6 µg/24 hours, p < 0.01). In a study of platelet uptake and efflux of dopamine, Boullin and O'Brien (1972) found no difference between normals and controls.

The research history of dopamine-β-hydroxylase (DBH), a synthesizing catecholamine enzyme catalyzing the conversion of dopamine to norepinephrine, epitomizes the need for multiple biological controls if biochemical research is to be meaningful. Coleman et al. (1974) found DBH to be slightly but nonsignificantly lower in autistic children than in normal controls, but these findings were reversed in a later report (Goldstein 1976). Lake, Ziegler,
and Murphy (1977) measured plasma norepinephrine (NE) and dopamine-β-hydroxylase (DBH) activity in autistics (n = 11), family members of autistics (n = 39), and controls (n = 12). Significantly higher NE levels were observed in autistics vs normals both in basal (462 ± 42 vs. 233 ± 42 pg/ml, p < .001) and standing (676 ± 41 vs. 480 ± 70 pg/ml, p < .025) positions. Significantly lower levels of DBH activity were observed both in autistics and their family members when compared to controls. Plasma DBH was found by Belmaker, Hattab, and Ebstein (1978) to be elevated in children with functional psychosis when compared to those with organic psychosis. No differences were observed in catechol-O-methyltransferase activity.

In 1980 Young et al. found that 22 autistic children did not have DBH levels different from other psychiatric patients or normal controls. However, there was increasing DBH activity as chronological age increased among the controls but not in the autistics. The authors felt this finding might be due to the smaller age range of autistics, or alternatively, might be related to a decrease in urinary free catecholamine and MHPG (Young et al. 1978). They also found a strong genetic effect of DBH activity.

Levels of biogenic amine metabolites in CSF have been studied as a result of the suspected relationships of these levels to the turnover rates of the parent compounds in the central nervous system. Cohen et al. (1974) measured CSF levels of homovanillic acid (HVA), and 5-hydroxyindoleacetic acid (5-HIAA) in psychotic children, epileptics, and children with movement disorders. Elevated levels of these metabolites were seen in psychotics when compared to epileptics. The relationship between levels of metabolites and probenecid suggested the need to measure 5-HIAA, HVA, and probenecid simultaneously. In a related study of children with autism, nonautistic psychosis, central processing disturbances, and aphasia and pediatric controls, Cohen et al. (1977) found autistics had lower CSF 5-HIAA levels than nonautistic psychotics.

Studies of abnormal metabolites of biogenic amines in childhood psychoses have been prompted by observations of possible abnormalities in adult schizophrenics. Widlitz and Feldman (1969) studied childhood schizophrenics for the presence of "pink spot" in urine. The incidence of "pink spot" was nearly equal in normals and schizophrenics but "... the intensity of the 'spot' was greater in schizophrenics" (p. 29). The qualitative assay for "pink spot" has not been useful as a marker for childhood psychoses.

Bufotenin and other N,N-dimethyl derivatives of 5-HT have been measured in urinary free catecholamine and MHPG (Young et al. 1978). They also found no differences when compared to normals. Perry, Hansen, and Christie (1978) found a significant difference in the level of CSF ethanolamine between controls and psychotics, but it is not certain that elevated ethanolamine is unique to psychotic illness.

Several studies examining the endocrine system of psychiatrically disturbed children have been done. Brambilla, Viani, and Rosso (1976) found that elevated ethanolamine was seen in 50 percent. In general, children with more debilitating psychoses had higher rates of endocrinopathy. Of the patients studied, 87 percent had demonstrable endocrinopathies thought to be related to problems in the pituitary or hypothalamus and not in the peripheral target organs, although this result was not well documented.

Yamazaki et al. (1975) studied the stress response and circadian rhythmicity of 11-hydroxycortico-
steroids in autistics. They found the stress response to be preserved, but the development of normal circadian rhythmicity was disturbed in autistics, suggesting an abnormality in the development of the cerebrohypothalamic system. Mahler et al. (1975) studied an insulin stress response and found controls recovered normal glucose levels more quickly, autistics had more prolonged cortisol elevation in response to stress, and no difference in growth hormone levels was seen between the groups. From these results, the authors suggested a functional abnormality in the endocrine stress-coping mechanism in autistics.

Diurnal cortisol and temperature variations were measured in autistics and normals by Hill et al. (1977). Normal children (n = 3) were similar to adults in cortisol and temperature variations, while four of six autistics showed abnormalities. One autistic child on antipsychotic medication had diurnal rhythms similar to adults.

Free fatty acid (FFA) metabolism was studied in autistic, schizophrenic, and emotionally disturbed children by DeMyer et al. (1971b). The following were observed: (1) Plasma FFA levels were more variable in psychotics than other groups; (2) 66.6 percent of autistics had normal lowering of FFA in response to exogenous insulin, which was contrary to reports on adult schizophrenics; (3) no difference was observed in the FFA response to endogenous insulin as tested by glucose stimulation. A poorly functioning regulatory feedback mechanism was suggested to explain these data.

Reviews of biochemical studies in psychiatrically disturbed children have been done by Guthrie and Wyatt (1975), Cohen and Young (1977), and Ritvo (1977). They found that cross-comparison of biochemical and endocrine experiments was difficult because of differences in diagnostic categories considered and experimental methodology. In addition, most experimenters have used rather small groups of patients because of the difficulty of any one center's accumulating a relatively large autistic population. The aggregate of results strongly implies that factors such as age, sex, activity level, intellectual function, organic neurological or medical disease, treatment modalities, physical environment, and severity of illness need to be taken into account as any of these variables may cause alterations in the parameters studied. Higher than normal serotonin levels are found to be more strongly related to low intelligence than to the diagnosis of infantile autism. A collaborative effort controlling for these variables in larger populations would be able to answer better the question of which, if any, of the many interesting biochemical deviations reported in autistic children are specific to the syndrome.

**Pharmacotherapy**

Drug treatment of autistic children has proved difficult because of uncertain efficacy of drugs used and numerous side effects. Controlled drug studies for the most part have involved small, nonhomogeneous populations, making interpretation of the results difficult. Agents studied have included antipsychotics, biogenic amine precursors, stimulants, psychedelics, antidepressants, vitamins, thyroid analogs, and lithium. While these agents have been associated with improvement in some individuals, no group of drugs is clearly superior in treating the complex symptoms of this syndrome, and improvement may be seen in some symptoms while others are aggravated.

Antipsychotics have been associated with improvement in some children. Rock (1974) reported that trifluoperazine, when used early, decreased autism, improved the ability to relate to adults and other children, decreased hyperactivity, improved the ability to perceive surroundings, and arrested the development of secondary retardation as measured by WISC scores. Further, serious side effects were not observed with trifluoperazine.

Faretra, Dooher, and Dowling (1970) found haloperidol and fluphenazine to be equally effective in decreasing anxiety and autism with little change noted in assaultiveness and regressive behavior after 8 weeks of treatment. Haloperidol exerted its effects somewhat more rapidly than did fluphenazine. Engelhardt et al. (1973) found haloperidol and fluphenazine to be equally effective in improving problems with increased psychomotor activity, stereotypic behavior, responsiveness, sleep disturbance, and feeding. They did observe a higher incidence of extrapyramidal symptoms with fluphenazine than haloperidol.

A study by Rimland using parent replies to questionnaires

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4 Information from July 1980 bulletin of the Institute for Child Behavior Research, San Diego, California; Bernard Rimland, director.
showed that of 15 psychopharmacologic agents, haloperidol was perceived by parents as resulting in some improvement in the greatest percentage (58 percent) of cases in which drug therapy was tried. In comparison, all drugs averaged were perceived as helpful in 28 percent of cases, psychotherapy in 34 percent, high-dose vitamins in 67 percent, day school in 70 percent, and operant conditioning in 82 percent.

Claghorn (1972) compared haloperidol and thioridazine in an outpatient group and found both drugs to be beneficial. Haloperidol was better in controlling behavioral symptoms and more rapid in onset of action. Haloperidol and behavior therapy were studied by Campbell et al. (1978a). Haloperidol was clearly superior to placebo in controlling withdrawal and apathy. In a retrospective study of 125 children, McAndrew, Case, and Treffert (1972) reported weight gain, tardive dyskinesia, impaired learning, and ocular changes as significant complications of antipsychotic treatment. Using patients as their own controls to assess the need for further drug treatment was suggested as a means of reducing cumulative dose, which is thought to affect the rate of side effect occurrence.

The biogenic amine precursors, dihydroxyphenylalanine (L-dopa) and 5-hydroxytryptophan (5-HTP), have been shown to have little effect on psychotic children. Zarcone et al. (1973) reported increased rapid eye movement sleep and no behavioral change in two schizophrenic boys treated with 5-HTP. Sverd et al. (1978) also found no significant change in behavior with 5-HTP treatment. Ritvo et al. (1971b) found no significant alteration in symptomatology despite significant lowering of serum 5-HT levels with chronic L-dopa administration. L-Dopa and amphetamine were compared in a crossover study by Campbell et al. (1976b). Amphetamine had only transient beneficial effects which were outweighed by worsening of preexisting symptoms. L-Dopa treatment resulted in stimulation of motor behavior, language production, and affective responsiveness in 4 of 11 subjects studied.

Although megavitamin therapy has been suggested as a possible treatment for schizophrenia, few studies involving children have been done. Niacinamide was evaluated by Greenbaum (1970) and found to have no significant therapeutic effect after 6 months of treatment. Rimland, Callaway, and Dreyfus (1978) studied vitamin B6 (pyridoxine) in a population of children who had previously responded favorably to B6 treatment and reported “behavior . . . deteriorating significantly during B6 withdrawal” (p. 472). The authors discussed several problems with the experimental design, problems which raise
questions about the validity of the observations.

Trials of psychedelic drugs have been limited. In a review of seven studies involving 91 patients, Mogar and Aldrich (1969) suggested further studies based on observation of increased speech production, affective responsiveness, and decreased ritualistic behaviors in patients treated with psychedelics. Simmons, Benor, and Daniel (1972) reported increased affective response and contact with adults and decreased responsiveness to external stimuli and motor activity with LSD-25. These changes were of short duration, and the effects of chronic LSD administration are unknown.

Treatment with thyroid hormone initially was promising, but later results failed to uphold these findings. Campbell et al. (1972a) compared triiodothyronine (T3) and amphetamine and found T3 to have stimulating and antipsychotic effects leading to significant improvement in a heterogeneous population. T3 was found to be effective in patients classified as euthyroid, leading the investigators to suggest a greater “need” for thyroid hormone for adequate CNS function in this population. Campbell et al. (1973) reported similar results with liothyronine treatment. As a followup, Campbell et al. (1978d) conducted a placebo-controlled crossover study of T3 in 30 autistics and found no significant difference between T3 and placebo.

Abassi, Linscheid, and Coleman (1978) measured T3, T4, and TSH in 13 autistics and found no evidence for hypothyroidism. Two patients developed clinical hyperthyroidism with administration of exogenous T3. The hypothesis of thyroid dysfunction in autistics was not supported.

Results after trials with other drugs have generally been unrewarding. In a study by Campbell et al. (1971b) imipramine was not useful generally for treating psychotic children, although further study of its application in retarded, mute, anergic, and borderline psychotic children was suggested. Results of lithium and chlorpromazine administration suggested lithium may be useful in treatment of aggression, explosive affect, and hyperactivity (Campbell et al. 1972b).

In summary, of the drugs studied, non-sedating antipsychotics seemed to offer the most promise, whereas stimulants were usually found to cause greater disorganization. Mixed results have been obtained with vitamins, thyroid hormone, and psychedelics. Biogenic amine precursors have not been found to be therapeutic. Antidepressants and lithium may be useful in treatment of specific symptoms: when any of these agents are used, the benefits must be carefully weighed against potential side effects, especially in the pediatric population with its potential for prolonged exposure.

Behavioral/Educational Treatment Programs

During the last 10 years, the most significant development in the treatment of infantile autism has been the continued growth in the use of behavioral/educational approaches. This development has been due to the generally accepted position that traditional forms of psychotherapy have not been effective in reducing the severe behavioral deficits associated with this syndrome. Following the first direct use of behavior modification methods with early childhood psychotics, as reported by Ferster and DeMyer in 1961, over 100 papers in this area had appeared by 1970. The next decade has seen the publication of over 200 more papers using various modifications of these techniques. Although behavioral methods are by no means a cure for infantile autism, there is a consensus that the use of systematic-intrusive approaches can lead to significant expansions in the behavioral repertoires of the children, allowing them to reach a higher level of functioning than if they were untreated or were treated with traditional therapeutic procedures. In this section some of the more extensive studies reporting the use of behavioral/educational programs with autistic children will be reviewed.

Verbal Behavior. One of the most important applications of behavioral methods has been to develop language skills in autistic children, since severe deficits in verbal behavior are often regarded as the major characteristic of this syndrome. Lovaas and co-workers (1973) have continued their early work in developing verbal skills with autistic children. After an initial period of reinforcing verbal imitation, they developed more spontaneous speech using a shaping process involving labeling objects (at first with prompts which are gradually faded) and then using simple statements about objects in an appropriate context. While this procedure is effective with either autistic children who are completely mute or with those exhibiting some spontaneous speech, therapeutic progress is
more rapid and achieves a higher level of functional speech with children who begin the treatment with some verbal skills. Of 20 children treated, all showed gains in verbal behavior, with spontaneous language being developed in the children with some speech when treatment was initiated. However, elementary vocabularies and object identification characterized the older and mute autistic children. One- to 4-year followups on these children indicated that those who were institutionalized tended to regress in verbal behavior as well as in all other behaviors while the children receiving further behavior therapy from their parents continued to show improvement. A brief reinstatement of behavioral methods temporarily reestablished some of the original verbal gains in the institutionalized children. These findings point out the importance of intensive and continued work with the individual autistic child if behavioral procedures are to be effective (Lovaas, Schreibman, and Koegel 1974). Much ground is lost rather quickly once the reinforcement contingencies and/or the concentrated training procedures are no longer in force. It also suggests that spontaneous developmental processes necessary for language acquisition are not being triggered or stimulated (if, indeed, they are even potentially available in these children) by behavioral methods in the case of most autistic children (see the Language section).

Many other workers have had success with expanding verbal skills in autistic children which is similar to that reported by Lovaas’ group. Halpern (1970), for example, studied 15 children over a 4-year speech treatment period. When the children started the program, nine were completely nonverbal and six showed some minimal verbal output. By the end of the treatment, 13 of the children demonstrated some useful speech to the degree that 11 of these were subsequently enrolled in special public school classes. Using prompting and fading procedures, Marshall and Hegrenes (1970) worked with four previously nonverbal autistic children and successfully developed object name and description skills. Working with children possessing some basic verbal skills, other researchers have been able to develop more complex language behaviors, such as simple and compound sentence usage (Stevens-Long and Rasmussen 1974), prepositional usage (Sailor and Taman 1972), and simple reading comprehension (Rosenbaum and Breiling 1976). In the Sailor and Taman study, it was found that the use of nonambiguous stimulus conditions was far superior as a training procedure, thus demonstrating the continued need for concrete references in language even for autistic children with some verbal skills. Also, short verbal commands (one to four words) were more effective in eliciting correct responses from autistic children than longer commands (Browning 1974).

In spite of the substantial gains in verbal output which the behavioral methods yield, it is obvious that only in a small number of cases have these procedures led to the development of true language in autistic children. Even Lovaas’ group no longer expresses the optimism regarding long-range goals for speech development that they once did (Lovaas et al. 1977). As more and more researchers come to the conclusion that perceptual language disorders play a critical role in the autistic syndrome, it appears that more realistic goals for verbal behavior in these children are being defined. However, any speech, even if it is a primitive rote speech, is better than complete mutism, and behavioral methods appear to be some of the most effective for working with the mute autistic child. The autistic child already possessing some verbal skills apparently can benefit from a number of intensive behavioral/educational programs and has some chance to develop a more functionally useful language than his mute autistic counterpart.

**Special Behavioral/Educational Approaches.** The one-to-one therapist-child relationship is usually considered the ideal, if not the essential, situation if significant progress is to be achieved in working with autistic children. However, there is some evidence that a 1:1 ratio is not always superior to a group approach (Frankel 1976; Frankel and Graham 1976). In addition, it is not practical or financially feasible to maintain this schedule for the many hours per day, and for the many years, required to expand the behaviors of autistic children. Thus, a number of researchers have attempted to adapt behavioral/educational methods for classroom use over long-term treatment programs. Koegel and Rincover (1974), starting with a 1:1 student-teacher ratio, gradually phased in larger classroom ratios and found that multiple baseline behaviors generally increased for both verbal and nonverbal (e.g., attending to...
teacher upon command) responding.

In a subsequent study (Rincover and Koegel 1977) the effectiveness of unsupervised responding in a classroom setting as a variable influencing behavioral progress was investigated. Initially each child in a four-child class was given individual instruction on a rotating basis. Typically one child stopped responding when the teacher moved on to the next child. However, when prompt fading, chaining, and programmed instructional material were introduced to teach each student to continue working for an extended period of time after the individual instruction, significant academic progress was observed. By the end of the training sessions one teacher was able to teach (various tasks related to tracing and drawing letters) all four children simultaneously with each child learning at his own rate with minimal supervision.

Although substantial gains can be achieved with intensive behavioral/educational treatment, learning deficits will likely continue to interfere with the acquisition of new responses, thus requiring strict adherence to structured home or institutional programs to maintain treatment gains (Brown 1971). The enormity of the task of training these children should be faced realistically. In a 1-year study of 14 autistic children receiving programmed instruction with a student-teacher ratio of 10:1, 11 children completed only an average of 10 percent of the course material with three children making much faster progress (Fischer and Glanville 1970). If the slower children progressed at the same rate for successive years of schooling, the authors estimated that it would take about 10 years before they were ready to enter a normal school program. However, it should be pointed out that other workers (e.g., Koegel, Egel, and Dunlap 1980) take a more optimistic view and stress the fact that some progress can be made even with the slowest children.

In a comprehensive study of 50 autistic children treated in three different special educational treatment settings with a 3½- to 4-year followup (Bartak and Rutter 1971, 1973; Rutter and Bartak 1973), all children made considerable progress in educational, cognitive, linguistic, social, and behavioral status sufficient to justify the effort and expense involved. The most important finding appeared to be that in comparing the three types of treatment settings (general regressive therapeutic approach with little attention to development of skills vs. permissive classroom environment combined with regressive therapeutic methods vs. structured classroom training emphasizing specific perceptual, cognitive, and motor skills), the latter experience was the most effective in fostering the greatest scholastic progress, as well as yielding as much progress in general social and behavioral development as the other two methods.

The advantage of organized educational programs in combination with intrusive behavioral methods was also supported by Rutter and Sussenwein (1971), Schopler et al. (1971), Graziano (1970), and Ward (1972, 1978). They can effectively be taught to teachers and a variety of nonprofessionals, especially parents (Schopler and Reicher 1971; Kozloff 1973; Marcus et al. 1978), enabling more “therapist”-child interactions in varied settings, one factor which seems critical for significant progress to be attained and maintained (Nordquist and Wahler 1973; Gallagher and Wiegerink 1976; Schopler 1976; Zifferblatt et al. 1977; Singh 1978).

Related to this, the importance of systematic teacher education should not be overlooked. Empirical assessment of teacher training in behavior therapy is possible. Not surprisingly, systematic improvement in the autistic child’s behavior was found to be related to the training level of the child worker (Koegel, Russo, and Rincover 1977). In addition, the importance of the teacher-child relationship should not be underestimated. Although automated instruction may be of some aid in training autistic children, at least one study has indicated that teaching machines are effective with autistic children only when the teacher is present, whereas the teacher did not require the machine to teach the task successfully (matching-to-sample) to the child (Russo, Koegel, and Lovaas 1978).

In spite of their emphasis on mastery of skills, the structured behavioral approaches have not penalized the autistic child in general behavioral-social development, when compared to unstructured methods. One study did fail to find differences in therapeutic effectiveness among various approaches: behavior modification, educational programs, relationship therapy, and activity therapy (Wenar and Rutter 1976). However, the weight of evidence from the greatest number of investigations indicates that the method of choice for maximal behavioral expansion in autistic
children is a systematic behavioral/educational program involving as many child contact hours as possible and using therapists who have been trained in the use of behavioral methods.

Other Aspects of Behavioral/Educational Programs. In the last 10 years some additional studies have been published describing important variables that may influence the learning that should occur during behavioral/educational programs with autistic children. These include research on time-out stimuli, responding to multiple cues, maintenance and generalization of training, sensory stimulation as reinforcers, and the reduction of self-stimulatory behavior.

Time-out procedures might normally be expected to serve as powerful aversive stimuli for autistic children. On the other hand, if they provide the opportunity for preferred, nonproductive self-stimulatory responses, they might actually increase the undesired responses, rather than reduce them. This appeared to be the case with tantrums; however, when self-stimulation was prevented during time-out, these methods did reduce the tantruming (Solnick, Rincover, and Peterson 1977). Numerous other studies have questioned the routine use of time-out as a training procedure with autistic and/or retarded children. Generally, they have demonstrated that it is of extremely limited training value with this population and is a poor use of valuable training time (Husted, Hall, and Agin 1971; Martin 1975; Plummer, Baer, and LeBlanc 1977).

The inability to respond to multiple cues has been suggested as one of the underlying deficits in autistic behavior (see the Perceptual Processes section). Koegel and Schreibman (1977) demonstrated that four autistic children were able to learn to discriminate a multiple-cue complex from each of its two component cues. However, the autistic children did not learn the discrimination in the same manner as normal children, responding at a higher level to one of the cues in the early trials. Only after a long series of successive discrimination problems were they able to develop a set for approaching new discriminations by responding equally on the basis of both component cues.

Maintenance and generalization of the learned behavior are crucial considerations for any training program involving autistic children. Desired behaviors that are emitted only in the restricted training setting during the actual training period are of limited practical value compared to those that are taken up as part of the general repertoire of the child. Thus, the delineation of variables that influence generalization and/or maintenance of the newly acquired responses is essential for improving the success rate of training programs. In two experiments (Rincover and Koegel 1975; Koegel and Rincover 1977) it was clearly demonstrated that training which failed to generalize could be made to generalize when the antecedent stimuli that were functional during training were identified and introduced into the extratherapy setting. Even when generalization occurred, however, maintenance was not usually noted. The dual use of partial reinforcement schedules in the original treatment environment, and noncontingent reinforcers in the extratherapy environment significantly increased generalization. The authors concluded that there were two distinct parameters of extratherapy responding: generalization and maintenance, both requiring separate assessment for developing a behavioral program that can be carried out effectively outside of the actual therapy sessions. This may also be the case with regard to developing appropriate social behaviors in autistic children, since these responses are resistant to generalization (Romanczyk et al. 1975; Strain, Kerr, and Ragland 1979).

Since food reinforcers, typically used in training autistic children, have the disadvantage of a low satiation threshold, other reinforcers are always being sought out. Sensory stimulation (music, a visual flickering stimulation, or a visual movement) was successfully used with four autistic children to maintain high rates of responding (bar pressing on an FR 5 schedule) over a long period of time (Rincover et al. 1977). The reinforcement strength of the sensory events varied from child to child, and when a child appeared to satiate for a particular sensory event, a relatively small change in the event was sufficient to produce a high rate of responding once again. Similar results were found with strobe light stimulation used as a reinforcer (Frankel et al. 1976). Of special importance is the recent quantitative evidence that successful completion of tasks by autistic children can itself increase motivation to continue to respond effectively to those tasks (Koegel and Egel 1979).

The frequency of excessive self-stimulatory behavior in the autistic child can interfere with efficient
training of more normal responding (Lovaas, Litrownik, and Mann 1971). These repetitive responses are apparently affected by some types of environmental stimulation and may be related to the mental age of the autistic child (Colman et al. 1976; Frankel et al. 1978). Methods that can eliminate or reduce these behaviors are of great importance. It has been shown that simple removal of the sensory reinforcement of various self-stimulation responses in three autistic children was effective in extinguishing the behavior (Rincover 1978a)—for example, removing auditory feedback of plate spinning by installing carpeting on the table where the child would normally spin plates.

An overcorrection method was used to eliminate self-stimulatory hand clapping in an autistic 7-year-old boy (Foxx and Azrin 1973). The procedures involved 5-minute periods of functional movement training contingent upon hand clapping. The child was instructed and manually assisted in moving his hands to one of five different positions. Eventually the training required only a verbal cue. Within a few days hand clapping was reduced to near zero levels, thus permitting more appropriate and functional use of the hands. In general, the authors found overcorrection methods to be more effective than alternative procedures (e.g., physical punishment) in eliminating self-stimulatory behavior. Another group (Wells, Forehand, and Hickey 1977; Wells et al. 1977) also used overcorrection as a method for decreasing self-stimulatory responding (e.g., mouthing and twirling objects, flapping and shaking hands) in autistic children. In addition, they often found increased spontaneous toy play accompanied significant reductions in self-stimulatory responding. This latter observation was supported by Harris and Wolchik (1979) and had been earlier reported (Koegel et al. 1974) under conditions in which physical suppression of self-stimulation was used to reduce the inappropriate behaviors. In contrast to the usual attempts to reduce self-stimulation, Hung (1978) showed that the opportunity to engage in self-stimulatory behavior, judiciously used, could serve as a reinforcer for spontaneous sentences of two autistic boys.

Self-stimulatory behavior may take the form of self-injurious behavior in some autistic children. In addition to the obvious danger to the child that this responding presents, it is extremely disruptive to training sessions. Although some researchers clearly feel that self-mutilation is reinforceable, just as any other operant (Lovaas, Schreibman, and Koegel 1974), others hypothesize that self-injurious responding is simultaneously maintained by both intrinsic and extrinsic reinforcement (Carr 1977). High demand situations (such as usually found in training sessions) also could lead to increased rates of self-mutilation (Carr, Newsom, and Binkoff 1976). Frankel and Simmons (1976) propose both operant and respondent paradigms as the best model to deal with data from both human and animal studies of self-mutilation.

Lovaas' group has been predominant in the use of response contingent electric shock to suppress the self-abusive responses. Considering the risks to the child from injuries that they can self-inflict, Lovaas feels that this danger justifies the use of drastic measures such as shock. In fact, he feels that it is one of the most effective and reliable of all the behavioral methods used with autistic children (Lovaas, Schreibman, and Koegel 1974). Although most therapists hesitate at using such techniques, especially in view of possible unforeseen side effects, a recent review of studies employing electric shock with autistic children actually reports more positive side effects (response generalization, increases in social behavior, and positive emotional behavior) than negative side effects as the result of its use (Lichstein and Schreibman 1976). While not ruling out a role for punishment in suppressing self-injurious responding, Frankel and Simmons (1976) felt that not enough emphasis has been placed on programs that train adaptive and appropriate behaviors which are strengthened by social reinforcement.

The widespread use of behavioral/educational methods with autistic children in the last 10 years has led to significant improvements in the overall behavioral repertoire of almost all the children given advantage of these programs (Treffert, McAndrew, and Dreifuerst 1973; Margolies 1977; Egel, Koegel, and Schreibman 1980; Koegel, Egel, and Dunlap 1980). Except in the case of those children who already have a more extensive behavioral background, however, these therapeutic advances usually do not result in a change in the overall poor prognostic outlook for most autistic children (Prior 1979). In spite of this fact, significant ad-
Table 4. Percent distribution of autistic (psychotic) population by study

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<tbody>
<tr>
<td>Overall outcome</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Good and very good</td>
<td>10</td>
<td>14</td>
<td>5</td>
<td>18</td>
<td>14</td>
<td>19</td>
</tr>
<tr>
<td>Fair</td>
<td>16</td>
<td>25</td>
<td>22</td>
<td>27</td>
<td>24</td>
<td>17</td>
</tr>
<tr>
<td>Poor and very poor</td>
<td>74</td>
<td>61</td>
<td>73</td>
<td>55</td>
<td>62</td>
<td>64</td>
</tr>
<tr>
<td>Speech</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Normal</td>
<td>6</td>
<td>16</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Useful (communicative)</td>
<td>49</td>
<td>46</td>
<td>51</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Develop useful speech after age 5</td>
<td>11</td>
<td>11</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Worsening</td>
<td>11</td>
<td>11</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>None</td>
<td>32</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Education</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Age appropriate (within 2 years)</td>
<td>10</td>
<td>9</td>
<td></td>
<td></td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Regular school (2 or more years)</td>
<td>70</td>
<td>2</td>
<td></td>
<td></td>
<td>90</td>
<td></td>
</tr>
<tr>
<td>Other variables</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gainful employment</td>
<td>0³</td>
<td>2⁴</td>
<td></td>
<td>18</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Loss of autism</td>
<td>5</td>
<td>14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long-term institutionalization</td>
<td>42</td>
<td>44</td>
<td>54</td>
<td>54</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>n of autistic (psychotic) subjects</td>
<td>126</td>
<td>63</td>
<td>63</td>
<td>63</td>
<td>11</td>
<td>47</td>
</tr>
<tr>
<td>Mean chronological age at followup (years)</td>
<td>12</td>
<td>15</td>
<td>15</td>
<td>35</td>
<td>17</td>
<td>19</td>
</tr>
</tbody>
</table>

1 Sources which detail the diagnostic criteria and procedures are identified in the References.
2 Less than 50 percent.
3 Over 15 years of age.
4 Over 16 years of age.
5 Twenty to 75 percent depending on diagnostic group.

advantages associated with the behavioral/educational methods remain. They provide maximal opportunities to those children with the greatest potential, while those children with the least potential receive at least some important behavioral gains.

Prognosis

The outcome for most autistic people is a life of complete or semidependence. Three studies (Rutter and Lockyer 1967; Eisenberg 1956; and DeMyer et al. 1973) showed fairly close agreement about the poor chance of the autistic child to become completely normal. Only 1 or 2 percent achieve such status. In table 4 (adapted from DeMyer et al. 1973) are the findings of six outcome studies of the mid-1950s to the mid-1970s that lend themselves to meaningful comparison. About 5 to 19 percent of autistics reached the borderline of normality, while 16 to 27 percent had a fair outcome, and 60 to 75 percent had a poor or very poor outcome.

Rees and Taylor (1975) found that 36 percent of 54 mixed psychotics had a "successful outcome"; that is, were within 1 year of appropriate school grade or gainfully employed. Of Etemad and Szurek's (1973) sample of 84 mixed psychotic children, only about 29 percent at age 19 years were not hospitalized and were semi-independent. Eggers (1978) from Germany reported a 20 percent remission rate in 57 childhood schizophrenia cases. All patients with onset before age 10 years had a poor outcome. Bender (1973) indicated 33/100 child schizophrenics...
(25-50 years old) made a satisfactory community adjustment. Thus childhood psychosis developing at or near puberty may have a somewhat better prognosis than earlier onset cases.

The most potent factor associated with a relatively good outcome is a relatively good general ability as measured by IQ tests on initial evaluation. DeMyer et al. (1973) found the best predictors of outcome in children initially examined at mean age 5.5 years were IQ, severity of illness, social rating, speech, and the number of neurological signs. Lotter (1974) found speech and IQ to be the best predictors (r = .89), with others being severity of illness, delay in passing milestones, sex, neurological findings, and years of school. In his series, no autistic girl had a good or fair outcome; however, one of the two normal outcome cases in the sample of DeMyer et al. was a girl. From Lotter's reviews of other studies, he concluded that relatively more male autistics may have a good outcome than female autistics. Other authors reporting IQ tests and verbal skills as good predictive items were Campbell et al. (1978c) and Rees and Taylor (1975). Those important factors reported not to have prognostic implications were family history of neuropsychiatric disorder, family atmosphere (Eggers 1978), and adequacy of parental functioning (DeMyer et al. 1973).

If an autistic child has an initial IQ over 60 or 70, his chances for a relatively good outcome are better than for lower IQ children. In the followup sample of Lockyer and Rutter (1970), 42 percent of higher IQ autistics were working or still in school when reexamined 10 years later. All of DeMyer's better outcome autistics had initial IQs over 60. However, not all higher IQ children will be successful—a finding Lotter (1974) attributed to lack of social intelligence. Adult autistics tend to be impractical and lacking in ability to understand and respond to feelings of others. Some demonstrate lack of emotional control and others a lack of ambition (DeMyer 1979).

While overall outcome is not good for independent living, many autistic children improve symptomatically. Table 5 adapted from DeMyer et al. (1973) shows changes in percentage distribution toward the more able rating alternatives for three important aspects of behavior, namely conversational speech, social relationships, and work/school rating from mean age 5.5 years to 12 years. Unfortunately very few of the autistics were given normal or nearly normal ratings at followup and some autistic children (about 10 percent) lost ground. Those with

| Table 5. Changes in percentage distribution of speech, social, and work/school ratings among autistic children over a mean 6-year followup¹ |
|------------------|------------------|

<table>
<thead>
<tr>
<th>Evaluation period</th>
<th>Initial</th>
<th>Followup</th>
</tr>
</thead>
<tbody>
<tr>
<td>% of subjects</td>
<td></td>
<td>subjects</td>
</tr>
<tr>
<td>Conversational speech rating alternatives</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Normal for age</td>
<td>0.0</td>
<td>3.7</td>
</tr>
<tr>
<td>2. Expresses ideas but below chronological age</td>
<td>5.9</td>
<td>26.2</td>
</tr>
<tr>
<td>3. Expresses immediate needs + echolalia</td>
<td>21.0</td>
<td>15.9</td>
</tr>
<tr>
<td>4. Echolalia only or few words</td>
<td>28.6</td>
<td>15.0</td>
</tr>
<tr>
<td>5. Mute</td>
<td>44.5</td>
<td>39.2</td>
</tr>
<tr>
<td>Total n</td>
<td>119</td>
<td>107</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Social behavior</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Nonpsychotic</td>
</tr>
<tr>
<td>a. Normal</td>
</tr>
<tr>
<td>b. Immature/hostile/shy</td>
</tr>
<tr>
<td>2. Psychotic</td>
</tr>
<tr>
<td>c. Loner</td>
</tr>
<tr>
<td>d. Severe withdrawal</td>
</tr>
<tr>
<td>e. Oblivious</td>
</tr>
<tr>
<td>Total n</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Work/school rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Normal</td>
</tr>
<tr>
<td>2. Borderline</td>
</tr>
<tr>
<td>3. Educable retarded</td>
</tr>
<tr>
<td>4. Trainable retarded</td>
</tr>
<tr>
<td>5. Subtrainable retarded</td>
</tr>
<tr>
<td>Total n</td>
</tr>
</tbody>
</table>

¹ Adapted from DeMyer (1973).
extreme hyperirritability including self-injurious behavior may regress from a previously higher functioning level (DeMyer 1979). Many parents whose autistic children remain difficult to care for during adolescence and show minimal improvement in intelligence and self-care become worn out psychologically and physically, the burn-out syndrome. In one large followup study, 59 percent of autistics over 14 years old were residing in chronic care institutions (DeMyer et al. 1973).

Theories of Etiology and Pathogenesis

At the beginning of the 1970s, Hingtgen and Bryson (1972) identified three basic types of theories related to infantile autism: nonorganic, organic-experiential, and experiential. Most nonorganic theories then, as now, were psychodynamic, assumed the infant was normal at birth, and attributed the development of symptoms to defective "nurture" from the parents, mainly the mother (see table 6 for examples). Many versions of deficient parental nurture have been proposed, but these can be grouped into two chief types. In the first variety, the parents of autistics were considered to have greater amounts of psychopathology than other parent groups. In the second one, they were viewed as having extreme personality types such as being cold, full of rage, or without sense of self.

In either version the infant was viewed as basically normal biologically, but normal identification was blocked or the parents failed to give optimum warmth and stimulation. We use the word "infant" to emphasize that most investigators thought the seeds of infantile autism were sown during infancy and not childhood. While some theorists have not been explicit as to how the parent's personal deficiencies were translated into poor child care practices, others have blamed poor feeding or poor speech practices, for example.

Any parental deficiency, whatever its specific qualities, theoretically led to the infant's social withdrawal which in turn led to failure to acquire normal speech and other intellectual and social skills. Hence, the "nurture" theorists believed that normal biological intelligence was locked within the autistic child. If the right treatment key could be found, the child would accelerate in progress and eventually become normal or even supernormal in intelligence.

There were also two varieties of the "nature-nurture" interaction theory (table 7). In one version all

### Table 6. Examples of nurture theories of Infantile autism

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Theory Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fraknoi and Ruttenberg (1971)</td>
<td>Inadequate stimulation from mother leads to unrelieved tension and then to a state of frozen balance between aggressive and libidinal drives</td>
</tr>
<tr>
<td>Szurek (1973)</td>
<td>Ungratified infant impulses become repressed, then fused with anxiety/rage, press for discharge, meet with more parental repression/tension and become &quot;insatiable longings of a secondary narcissism of a dissociated human character&quot;</td>
</tr>
<tr>
<td>Williams and Harper (1973)</td>
<td>Sensory deprivation at critical developmental periods</td>
</tr>
<tr>
<td>Ekstein and Friedman (1974)</td>
<td>Mother has a lack of &quot;sending power&quot;</td>
</tr>
<tr>
<td>King (1975)</td>
<td>Double-bind attitude (superficial warmth combined with severe coldness and rejection) of the mother makes the infant &quot;want to escape&quot;</td>
</tr>
<tr>
<td>Massie (1978)</td>
<td>Active avoidance of eye-to-eye contact and deficient touching of the infant by the mother</td>
</tr>
</tbody>
</table>

### Table 7. Examples of nature-nurture theories of Infantile autism

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Theory Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Despert (1971)</td>
<td>Disturbance processing &quot;l-other&quot; concept leading to resistance in developing social contacts</td>
</tr>
<tr>
<td>O'Moore (1972)</td>
<td>Autism tends to develop in language-impaired children, most likely caused by fetal anoxia plus psychological trauma</td>
</tr>
<tr>
<td>Miller (1974)</td>
<td>Evidence for brain abnormality inconclusive. Possibly interference in early, subtle organization of newborn behavior leads to a perceptual disorder due to inadequate sensorimotor, tactile, and kinesthetic handling</td>
</tr>
<tr>
<td>Tinbergen and Tinbergen (1976)</td>
<td>Causes organic and experiential. Some cases caused more by organic factor while others have more psychogenic causes</td>
</tr>
</tbody>
</table>
autistic infants were seen as biologically deficient and the parents viewed as failing to give proper emotional support to a "vulnerable" infant. In the second version, some infants were viewed as "organically" damaged and others as biologically normal. In this second version, the parents of "nonorganic" infants were described as failing to a greater degree than parents of "organic" infants.

In the pure "nature" or organic theory (table 8), the parents were viewed as contributing little more to the child's illness than symptom variations. The illness was considered basically the expression of a biological abnormality. As in the "nature-nurture" interaction theory, investigators disagreed about the kind of biological dysfunction that might be present in the infant.

Types of Consistencies in Theories. Through all the variations of parental deficiency theories ran a consistent thread of emphasis on the parental failure to adequately and warmly stimulate their autistic infants. In some theories, this adequate stimulation failure was implied, for some theorists dwelt on the greater degrees of psychopathology in the parents and did not state explicitly the mechanism of the developmental failure of the autistic child. Logically, such developmental failure could come from inadequate handling practices making life uncomfortable and incomprehensible to the infant. Alternatively, the infant could mimic the sick personality of the parents.

In the many theories concerning the kind of biological defect, there was rather consistent emphasis on a deficient nervous system. It is interesting to note that only a few authors considered the deficiency to be the rather common neurological dysfunction such as might affect the infant's general intelligence, as well as specifically the language centers. Most often, some more esoteric and difficult to test biological deficiency was pronounced concerning the reticular activating system, the limbic lobe, or the whole timing mechanism of somatic development, for example.

Clearly, to test these three opposing theories and even a few of their variations, researchers needed to consider both the psychological nature of the parents and their child-rearing practices and the neurological status of the children. The reason it was logical to begin with such a homely biological procedure as a neurological evaluation was the proclivity for a maldevelopment or insult to any portion of the nervous system to be accompanied by other physical maldevelopments. These signs, small as they might be, such as deformed earlobes or cafe-au-lait skin spots, would be the outward tracks that accompanied the biological dysfunction in the brain. These kinds of outward tracks are common in other groups of neurologically dysfunctioning children such as the generally retarded (Kennard 1960). Such a neurological study, in addition to giving us indirect evidence of CNS dysfunction, or of normal function, might also give clues as to the nature of the insult in any given child or subgroups of children. It also might help define neurologically normal and abnormal subgroups of autistic children.

Types of Control Groups Needed. Two sets of control groups were necessary to test the three theories: (1) parents whose children were all normal and one of their normal children, and (2) parents who had at least one nonpsychotic child, subnormal in general intelligence or having a specific learning disability such as dysphasia. The control parents would need to be matched to autistic parents on important demographic variables. The children should be age- and sex-matched. All the subnormal children should have use of all four extremities and special senses so that the same developmental tests could be used on all children.

Findings That Would Prove or Disprove Theories. It is possible to outline a series of logical constructs of the findings from the 1970s that would prove or disprove each one of the opposing three theories (table 9, parts A and B). If the pure nurture theory were correct, investigators should find that parents of autistic children in comparison to control parents mishandled their autistic infants or provided more inappropriate identification models. Unhealthy personality types, mental illness, or extreme character traits should be more common in parents of autistics. Because most autistic children have severe symptoms before their third birthday, this parental deficiency would have had to occur before this time. Investigators should find no more signs of neurological dysfunction in autistic than in normal children.

If the pure nature theory were
Table 8. Examples of nature theories of Infantile autism

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Theory</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hutt and Hutt (1970)</td>
<td>Hyperactive reticular activating system leading to reactive effort to reduce sensory input</td>
</tr>
<tr>
<td>Frith (1971), Hermelin (1971)</td>
<td>Imbalance between extracting external rules and internal rules interfering with organism-environment interchange</td>
</tr>
<tr>
<td>Money, Bobrow, and Clarke (1971)</td>
<td>Autoantibodies to CNS</td>
</tr>
<tr>
<td>Myklebust, Killen, and Bannochie (1972)</td>
<td>Dysfunction of right hemisphere with improper nonverbal processing</td>
</tr>
<tr>
<td>Bender (1973)</td>
<td>Early decompensation in genetically vulnerable individual due to organic stress in utero, perinatality, or early childhood</td>
</tr>
<tr>
<td>Zarcone et al. (1973)</td>
<td>Low 5-HT in rapid eye movement sleep center (brainstem) results in a mixed dream-wake state</td>
</tr>
<tr>
<td>Baltaxe and Simmons (1975)</td>
<td>May be left hemisphere. Underlying deficit not language specific but cognitive</td>
</tr>
<tr>
<td>DeMyer (1975b)</td>
<td>Profound language difficulty may be sole cause in some children but not in others who may also have a visual motor dyspraxia leading to the characteristic verbal and non-verbal communication problems</td>
</tr>
<tr>
<td>Hauser, DeLong, and Rosman (1975)</td>
<td>Asymmetrically bilateral temporal lobe disease but main location left hemisphere</td>
</tr>
<tr>
<td>Hertzig and Walker (1975)</td>
<td>Brain organization deficiency leading to disorder in information processing</td>
</tr>
<tr>
<td>Simon (1975)</td>
<td>Autistic is dysprosodic and cannot get meaning of intonation and stress features of speech. Location may be inferior colliculus of brainstem, a major center for selective attention to sounds. Variety of causes (birth anoxia, circulatory toxins, or phenylketonuria)</td>
</tr>
<tr>
<td>Boucher (1976a)</td>
<td>Language impairment is symptom of general cognitive difficulties</td>
</tr>
<tr>
<td>Cohen, Caparulo, and Shaywitz (1976)</td>
<td>Midbrain and brainstem dysfunction involving catecholaminergic pathways but need more evidence</td>
</tr>
<tr>
<td>Darby (1976)</td>
<td>Cause is due to a number of diseases; symptoms due to a &quot;final common pathway&quot;</td>
</tr>
<tr>
<td>Ornitz and Ritvo (1976)</td>
<td>Neuropathophysiological process affecting developmental rate, sensorimotor integration, language, cognition, intelligence, and ability to relate. Cause either idiopathic or known brain disease of various kinds</td>
</tr>
<tr>
<td>Porges (1976)</td>
<td>Imbalance in autonomic activities; possibly abnormal serotonin levels in CNS</td>
</tr>
<tr>
<td>Chess (1977)</td>
<td>Final behavioral consequence from many different causes and can follow chronic viral encephalitis. Onset not always before age 30 months</td>
</tr>
<tr>
<td>Fish (1977)</td>
<td>Pandevelopmental retardation is an &quot;infancy marker&quot; for an inherited neurointegrative defect</td>
</tr>
<tr>
<td>Churchill (1979)</td>
<td>Language deficits are necessary and sufficient cause of those phenomena common to all autistics. Other types of disabilities may account for some of the differences among them</td>
</tr>
<tr>
<td>Damascio and Maurer (1978)</td>
<td>A variety of agents cause lesions in mesolimbic cortex in mesial frontal and temporal lobes, neostriatum, and anterior and medial nuclear groups of thalamus</td>
</tr>
<tr>
<td>Hier et al. (1978)</td>
<td>No single abnormal brain location. Lesion(s) consequent to a variety of disease</td>
</tr>
<tr>
<td>Rutter (1978)</td>
<td>Nonspecific syndrome of biological impairment is most likely explanation. Bilateral lesion</td>
</tr>
<tr>
<td>Coleman (1979)</td>
<td>Not unitary disease. If localized lesion, it is in thalamus</td>
</tr>
<tr>
<td>Lovaas, Koegel, and Schreibman (1979)</td>
<td>Stimulus overselectivity: Low performance level of autistic children related to their tendency to respond to only limited number of cues in their environment</td>
</tr>
</tbody>
</table>
Table 8. Examples of nature theories of infantile autism—Continued

| Prior (1979) | Left hemisphere dysfunction with some language and cognitive functions possibly taken over by "relatively" strong right hemisphere mediation |
| Wing (1979) | Deficit in cognitive skills |

correct, we should find that autistic children differed in some important biological way from normal children and possibly also from the subnormal children. There should be no important deficiency in the way autistic parents handled their autistic infants in comparison to parents of subnormal infants. If the nature-nurture theory were correct, the autistic and subnormal children should be similar in neurological status, but parents of the latter group should provide more adequate infant care practices than parents of autistics. The alternate nature-nurture interaction theory was that there were two classes of autistic children—the neurologically normal and the abnormal. In this case, we should find that those autistic children who were normal

Table 9. Results needed to support each of three basic theories of causation

<table>
<thead>
<tr>
<th>For nurture theory to be correct</th>
<th>For nature theory to be correct</th>
<th>For nature-nature interaction theory to be correct</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children</td>
<td></td>
<td>Version A</td>
</tr>
<tr>
<td>1. Autistic children should have no more signs of neurological dysfunction than normal children but fewer signs than subnormal children</td>
<td>1. Autistic children should have more signs of neuropathology than normal children</td>
<td>1. Autistic children should have equal amounts and types of neurological dysfunction as subnormal children</td>
</tr>
<tr>
<td>2. They could or could not resemble subnormal children in overt neuropathology but should have some different modalities affected than subnormal children</td>
<td>2. They could or could not resemble subnormal children in overt neuropathology but should have some different modalities affected than subnormal children</td>
<td>2. Normal children should have lesser amounts of neurological dysfunction than either autistic or subnormal children</td>
</tr>
<tr>
<td>Parents</td>
<td></td>
<td>Version B</td>
</tr>
<tr>
<td>1. Greater amounts of psychopathology or</td>
<td>1. Equal amounts of psychopathology or</td>
<td>Parent type a should have a neurologically normal autistic child.</td>
</tr>
<tr>
<td>2. More deficient childcare practices (during infancy especially) in parents of autistics than parents of normals or subnormals</td>
<td>2. Child-care practices should be insignificantly different in autistic vs. normal and autistic vs. subnormal parents</td>
<td>Parent type b should have a neurologically abnormal child</td>
</tr>
</tbody>
</table>

1 Adapted from DeMyer (1975b).
neurologically would have had less adequate parenting than those who had signs of neurological dysfunction.

Major Import of Findings From the 1970s. Little support has been found for either the nurture or the nature-nurture interaction theories. Parents of autistic children were discovered to be similar in parenting practices and emotional health to parents of both normal and biologically deficient control children. Only a higher incidence of reactive depression from the stress of rearing a deviant child was found in both mothers of autistics and retardates in comparison to normal control groups. These findings have become so firmly established that nurture and nature-nurture interactional theory articles were infrequently published during the 1970s.

Evidence for the nature theory is strong. The results of most carefully designed studies using the two necessary control groups have shown that more autistic children than normal controls have come from troubled pregnancies and difficult labors, and have had more signs of neurological dysfunction at all periods of life. In contrast, nearly every study showed how much autistic children were like retardates and other neurobiologically handicapped groups with regard to incidence of neurological dysfunction and measured intelligence. Even in those very few autistic children who possess overtly a clean neurological bill of health and "normal" intelligence, some theorists (Cohen, Caparulo, and Shaywitz 1976) are speculating that new diagnostic techniques are likely to locate heretofore hidden brain deficits.

Yet to be explained is what makes one brain dysfunctional child autistic and another one retarded and yet another one special learning disabled. Investigators have yet to locate the exact functional and anatomical or biochemical differences between autistics and other neurologically handicapped groups. The brain is generally assumed to be the site of the lesion. The cause of the lesion is assumed to be any of the many agents that are known to damage the brain. A sign of the times is that defective biogenic amine metabolism is involved as a possible causative agent by some theorists. Currently some investigators (Ornitz and Ritvo 1976) speak of autism as classifiable on the basis of etiology as (1) idiopathic (due to unknown cause) and (2) associated with diagnosable disease. The anatomic site(s) offered as possibilities cover many areas of the central nervous system from the limbic lobe to the higher frontal and parietal cortical association centers (see summary of the Neurobiology section).

The question of the pathogenetic route of the language symptoms has intrigued several theorists. Some state or imply that pathology in the brain centers controlling language might be the core lesion while others believe that language symptoms primarily result from cognitive deficiencies. Any theorist who grapples with this controversy must also contend with the fact that neither "language" nor "cognition" has a good operational definition (Churchill 1978). Furthermore, the brain structures subserving all aspects of language, let alone cognition, have not been defined. Until such definitions are forthcoming and brain localization studies make enormous strides, these two competing theories cannot be put to a good test. The perceptual inconstancy theorists have not discussed in any detail the mechanism by which language symptoms develop, but they generally locate the lesion in the midbrain or brainstem.

The connections between the social symptoms and cognitive and language symptoms have been discussed relatively less often by organic theorists, although social symptoms have usually been regarded as secondary to language/body communication deficits or to cognitive or perceptual organization deficits. In Kanner's original theory, he saw the social deficits as inborn, a provocative idea that may apply to some but not all autistics.

As Hingtgen and Bryson noted (1972), there are still basic deficiencies in the theories, the most notable being the proclivity for theorists to reason from an incomplete data base. Either they seize upon partial aspects of the syndrome or they base their ideas on limited observations of a few children—the most noteworthy example being that of Tinbergen (1974) in his Nobel Prize acceptance speech. He largely took the side of the "nurture" theory based on observation of three autistic children and an unknown number of normal children. The errors in his theory and Tinbergen's ignorance of important facts concerning autism were knowledgeably rebutted by Schopler (1974) and Wing and Ricks (1976).

As we begin the 1980s, all but a
few researchers hypothesize that autism is an organic disorder involving the brain. While many organic theorists believe that any event that can damage the brain can cause the symptomatic picture of autism, few people agree on the site or sites of the lesion(s). A key controversy is whether language or cognitive defects are the central functional deficit. One major goal for the 1980s is to begin more extensive investigations comparing nonautistic brain dysfunctional children to autistic children. Data from these studies should help to shed light on important etiological factors that lead to major differences in social and communication differences between the two groups.

Current Status

In our earlier review (Hingtgen and Bryson 1972) two dominant hypotheses were said to have influenced much of the research up to that time: (1) that the childhood psychoses, including infantile autism, were thought to represent the earliest manifestations of adult schizophrenia, and (2) that psychotic children were believed to be potentially capable of normal functioning in virtually all areas of development. Neither hypothesis has been supported by the research data, and during the decade of the 1970s it was the rare investigator who even gave lip-service to such previously held notions. The great body of evidence generated during the last 10 years of research has indicated that infantile autism is a type of developmental disorder accompanied by severe and, to a large extent, permanent intellectual/behavioral deficits. It is viewed as a distinct entity with clearly no relationship to the adult psychoses. This profound change in attitude has received the strongest endorsement in two prestigious national publications. The leading journal in this area changed its name in 1978 from Journal of Autism and Childhood Schizophrenia to Journal of Autism and Developmental Disorders; and, of perhaps even greater significance, the new DSM-III (American Psychiatric Association 1980) no longer includes infantile autism under the heading of childhood schizophrenia, but rather under the category of pervasive developmental disorders.

Still unresolved, however, is the relationship between infantile autism, other forms of developmental disorders, and mental retardation. Although the current research singles out degree of social distance and degree of communicative speech as the two most consistent differentiating items, the optimal rating instrument, with appropriate controls for both mental and chronological age, remains to be constructed. In addition, of the many classification systems introduced in the last 10 years, no one system clearly stands out as having superior heuristic value regarding etiological, prognostic, or treatment considerations. Perhaps the multi-axial systems still provide the greatest hope for future work.

The prevalence rate of infantile autism is probably between 4 and 5 per 10,000 population under 15 years of age, while in subnormal populations such as those with congenital rubella and mental retardation, it is probably much higher: 500 to 800 per 10,000. There is nearly universal agreement that more males have autism than females, the ratio being about 4 to 1, and that autism is not related to birth order. There is less agreement about maternal age and national origin of parents. Twin and other sibling studies lend weak support to genetic inheritance of the complete autistic syndrome, although specific deficits in language-cognitive components may be genetically linked. The previous common finding of higher socioeconomic status being related to autism was probably due to referral bias. Higher socioeconomic fathers are more likely than lower class fathers to apply to clinics specializing in autism where most studies on autism are done.

In sharp contrast to early portrayals of parents of autistic children as "refrigerator" personalities, the last decade of investigation has characterized these parents as similar to those with children exhibiting other severe childhood disturbances. Thus parents of autistic children have been found to display no more signs of mental or emotional illness than parents of children with organic disorders (with or without psychosis). In addition, they do not manifest extreme personality traits such as coldness, obsessiveness, social anxiety, or rage, nor do they possess specific deficits in infant and child care.

Although rearing an autistic child is stressful, and mild depressive symptoms are not uncommon, especially in mothers, there is no evidence that parents of autistic children develop severe personality changes or major psychiatric symptomatology. While there appear to be conflicting data about the presence or absence of thought
disorders resulting from anxious confusion about their autistic child, parents of autistic children are now generally perceived to be trustworthy in their evaluation of their child's problems and are typically involved as co-therapists in various treatment programs.

A major thrust of the 1970s was the growth of parent groups under the aegis of the National Society for Autistic Children. Parents meet together for emotional support, sharing of home care techniques and diagnostic/treatment/school resources, and to support research. The Society has done much to relieve parents of unwarranted guilt feelings and focus national attention on the realities of autism.

The studies of measured intelligence of the 1970s confirmed the findings of the 1960s that most autistic children score in the mentally retarded ranges, that these scores are stable over short and long periods of time, and that splinter skills do not reduce the validity of the IQ, which is a good predictor of ultimate outcome. In addition, the conditions of social improvement or several years of treatment in autistic children are not, as a rule, accompanied by an increase in measured intelligence. An IQ of 40 or below, even when determined during preschool years, is predictive of a poor outcome. While not all children with IQs initially above 60 or 70 have a good outcome, those who make accelerated intellectual progress and a better social adjustment come from this higher IQ group. Autistic children in both preschool and later years are testable if appropriate mental age test items are used. As a group, autistics score nearer age norms on certain types of perceptual-motor tests items, namely, fitting and assembly tasks (e.g., Seguin form board and WISC object assembly) than they do on verbal items, particularly verbal comprehension and abstraction. This inability to use language abstractly on IQ tests parallels the disability in other language studies.

Current thinking assumes that gross disturbances in language development are a major characteristic, if not the primary defect, of infantile autism. If autistic children are not completely mute, as is frequently the case, their receptive and expressive language is primitive, echolalia occurs with high frequency, there is poor comprehension of nonverbal gestures, and they have low levels of spontaneous speech and speech for social purposes. Cognitive/language deficits appear fundamental to the autistic syndrome. Indeed, vocal articulation appears as the sole area in which the autistic children with some language skills do not score lower than control groups from retarded or dysphasic populations. Some initial studies using sign language provided hope that nonvocal communicative skills could be developed in autistic children. Unfortunately, sign language as used in more carefully controlled later studies failed to yield dramatic gains in language skills for most autistic children. However, these techniques could be effectively incorporated into an overall multisensory treatment program to increase the total behavioral (both verbal and nonverbal) repertoire significantly.

Important studies during the decade of the 1970s have successfully demolished two widely held clinical impressions—namely, that autistic children have atypical language because of parents' response to the child's attempts to communicate, and that autistic children refuse to use the pronoun "I." Neither hypothesis is taken seriously any longer by the great majority of researchers. Extensive use of behavioral procedures has produced significant improvements in speech in many autistic children. Nevertheless, in spite of many promising new treatment methods, the overall poor prognosis for development of true spontaneous, communicative language in most autistic children remains.

Since perceptual disturbances have been singled out as a possible underlying deficit responsible for the development of the autistic syndrome, research in perceptual processes appears crucial for ultimately understanding this disorder. The phenomenon of stimulus overselectivity (i.e., in a specific learning task autistic children tend to respond only to a few cues from a larger range of available cues) has received a great deal of attention since 1970. Although it is now apparent that stimulus overselectivity, also having been observed in nonautistic retarded children, is closely related to lowered mental age, such a perceptual strategy could account for much of the paucity of behavior associated with infantile autism. A number of training methods have been devised to reduce the autistic child's overselective attention to cues for specific learning tasks.

Another interesting feature of learning in the autistic child is that some types of prompt fading techniques, which are usually quite effective with retarded children, often impede, rather than facilitate, learning simple and complex discrimination tasks. Other prob-
lems in perceptual processing manifested by autistic children involve the imposition of idiosyncratic perceptual patterns on incoming sets of stimuli, severe disturbances in handling multiple and complex cues, specific abnormalities in audition, cross-modal stimulus association deficits, and failure to tolerate intertrial intervals of more than a few seconds. Although previously assumed to be an important aspect of the autistic syndrome, evidence of lack of eye-to-eye contact and lack of self-recognition is now seriously questioned. The major impact of research on perceptual processes in autistic children points to serious disturbances, but the underlying mechanism(s) have yet to be delineated.

In behavioral studies, autistic children demonstrated levels of play (typically stereotyped or nonsymbolic), pantomime, hand use, body imitation, drawing geometric figures, and ball play that were typical of much younger children, even infants. Those autistic children with higher achievement levels in other test scores and in school also had higher levels of performance in all behaviors studied. Parental reports of developmental milestones from autistic infancy through later childhood confirmed what the researchers measured: that autistic children were delayed in passing many milestones not only in language and motor skills but in use of eating utensils, drawing figures, dancing, and rhythmic and music performance. Autistic children's inability to symbolize and imitate bodily and thus in turn to their social distance.

With the growing conviction during the 1960s that many, if not all, autistic children had neurological abnormalities, investigators of the 1970s used every feasible technique to study this subject—for example, standard neurological evaluations, EEG, pneumoencephalograms, and computerized tomography. Autistic children in comparison to normal children had more physical and laboratory signs of possible brain dysfunctions. In contrast, autistic and mentally retarded children had nearly equal numbers of signs of neuropathological import. Attempts to localize the lesion(s) are just beginning, but initial studies indicated that both sides of the brain might be affected, the left side probably more severely involved than the right side. In many cases, however, a variety of brain abnormalities were reported in various sites, suggesting that the autistic syndrome could be related to many different kinds of neurobiological insults.

The possibility that abnormal neurochemistry is responsible for the autistic picture has been heavily investigated by chemical analyses of the various body fluids and their cellular constituents. The serotonergic system received the most research attention. Several investigators working independently found higher than normal levels of peripheral serotonin in groups of autistic children; however, this abnormality may have little to do with producing primary symptoms of autism but more to do with mental retardation aspects of autism. Autistics with low IQs resembled nonautistic persons with similarly low IQs in having higher than normal peripheral serotonin values. Also autistics with borderline or normal IQs had serotonin values within the normal range. Studies of other biogenic amines, amino acids, hormones, and trace elements have revealed no consistent biochemical abnormalities specific to autism. Nevertheless some provocative differences between autistics and other diagnostic groups have been reported in urinary catecholamines and bufotenin; plasma norepinephrine, dopamine-β-hydroxylase activity; CSF levels of homovanillic acid, 5-hydroxyindolacetic acid, and ethanolamine; serotonin efflux from red cells; and the endocrine system. These differences need to be confirmed and require further exploration in experiments carefully controlled for other biological factors.

While drug treatment is only partially successful in controlling some autistic symptoms in some individuals, the nonsedating antipsychotic agents (haloperidol: Haldol; fluphenazine: Prolixin; thioridazine: Mellaril; thiothixine: Navane; and molindone) appear more successful than the sedating agents (chlorpromazine: Thorazine; and promazine: Sparine). Other less successful agents studied include biogenic amine precursors, amphetamines, and thyroid hormones. Antidepressants and lithium may have limited usefulness for certain symptoms. Efficacy of high-dose vitamins is controversial, and well-controlled studies need to be done. Since side effects are common and can be serious and long lasting, the potent antipsychotic agents should be used mainly for treatment of severe symptoms uncontrolled by
behavioral techniques and for relatively limited periods of time.

Since traditional forms of psychotherapy have not proved effective with autistic children, systematic, intrusive, behavior- al/educational treatment programs were introduced in the early 1960s. This trend has increased in intensity in the 1970s and virtually displaced most other treatment approaches used for the disorder. Application of behavioral methods to verbal behavior has been extensively studied both in mute autistic children and in those who had some language skills before treatment began. The latter group appears to benefit most from these methods, often developing functionally useful language, if not true spontaneity of speech. In the former group of mute children, considerably less improvement is realized. Even here, however, object identification and elemental vocabularies are significant advances over the complete absence of verbal communication. Intensive and continued work during treatment and followup are crucial, especially for children possessing the lowest levels of verbal skills, if treatment gains are to be maintained.

Since one of the most important goals for most treatment programs is to prepare the autistic child to enter, at the very least, a special educational setting, a highly valuable recent finding is the fact that a 1:1 therapist-child ratio is no longer regarded as essential for the most effective treatment approach. Equivalent gains have been obtained in structured small classes. In addition, many studies have now indicated that greater progress can be achieved in the educational, cognitive, and linguistic status of autistic children, when treated in an educational setting with intrusive behavioral methods than when treated with nonin- trusive approaches. Of equal importance is the fact that the former methods have in no way penalized the child with regard to general behavioral-social development. Thus, the overwhelming evidence suggests strongly that the treatment of choice for maximal expansion of the autistic child's behavioral repertoire is a systematic behavioral/educational program, involving as many child contact hours as possible, and using therapists (including parents) who have been trained in the use of behavioral techniques.

Numerous improvements in specific behavioral techniques have resulted from intensive studies of the critical variables influencing learning in autistic children. Many studies of factors such as time-out stimuli, re- sponding to multiple cues, maintenance and generalization of training, sensory stimulation as reinforcers, and reduction of self- stimulatory behavior have resulted in the fine-tuning of behavioral methodology and the achievement of additional therapeutic gains. Although the rapid expansion in the use of behavioral/educational methods in the last 10 years has led to significant improvements in behavioral output in almost all children given the advantage of such programs, the overall prog- nostic outlook for a self-supportive adult life remains poor, except for those children already demonstrating key behavioral skills before treatment. In spite of this rather pessimistic ultimate expectation, the advantage of behavioral treatment programs remains im- pressive. They provide maximal opportunities for those autistic children with the greatest potential, and, at the very least, furnish those children with the least po- tential the opportunity to acquire some significant behavioral skills.

A review of the major outcome studies of the last three decades shows that the majority (60 to 75 percent) of autistic people live in complete or semidependence with relatives or in long-term institutions. Only about 1 or 2 percent achieve a normal, independent status and 5 to 19 percent a borderline normal status. The factor most heavily associated with a good outcome is a relatively good general ability as measured during initial evaluation (e.g., IQ, verbal skills). Those who make acceler- ated progress, both in intelligence and decrease in symptom severity, come from the group of autistic children with IQs of 60 or above. Low-functioning autistics, while often making symptomatic gains, do not reach normal or nearly nor- mal levels in adulthood no matter what type of treatment or educa- tion they receive. The ultimate outcome for higher functioning autistics may be enhanced by good behavioral/educational programs. Family adequacy is not related to outcome. Parents frequently become worn out psychologically and physically by the time of adolescence of the autistic child and seek placement. Of course, in every case in which the autistic offspring remains dependent, eventual placement must be sought because of parental illness or advancing age.

Theoretical articles supporting a nurture or nature-nurture position were rarely published in the 1970s. The consensus of most investiga-
tors leans toward the nature theories and attributes infantile autism to some type of neurobiological defect that affects the normal functioning of the brain. Localization studies have revealed different types of lesions in various parts of the brain, suggesting that any illness or agent that can damage the brain can also probably cause the autistic syndrome. The specific site(s) of the lesion(s) has (have) not been agreed upon. In addition, a major controversy is whether language or cognitive defects are the central functional deficit. A major goal for the 1980s is more extensive studies of nonautistic brain dysfunctional children for comparison to autistic children. These studies should enable researchers to explain why the two groups differ in social and communicative skills and how this might be correlated to neurobiological defects.

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