PART ONE

What Is Autism?
1 Meeting Autism

There she moved, every day, among us but not of us, acquiescent when we approached, untouched when we retreated, serene, detached. . . .
Existing among us, she had her being elsewhere.

(Park 1982, 12)

I visit the home of parents I recently met. Their five-year-old son is standing on his head on the couch. I go up to him, turn my head to the side, and say, “Hello, Kenneth.” “Hello, Kenneth,” he echoes.

I enter a room at a hotel where an informal meeting is in progress. As soon as I step through the doorway, a handsome, nicely dressed young man of perhaps seventeen walks up to me and tells me his name. The following exchange then takes place:
“What is your name?”
“Shirley.”
“What is your sister’s name?”
“Which sister?”
“How many sisters do you have?”
“Two.”
“What are their names?”
“Paula and Sandy.”
“What is your brother’s name?”
“How do you know I have a brother?”

After faltering for a second, he continues, “You don’t have a brother?”

His attention then immediately shifts to the person who entered the room after me, and the same questioning routine begins again.

I am at a national conference on autism. Walter, a man perhaps in his mid-twenties, draws my attention. He claps loudly when anyone is introduced, and as he does so his mouth opens, his head moves from side to side, and his eyes appear to focus at a point near the ceiling. Walter and his mother are sitting only a few feet from the bluegrass band that is to play at the conference reception. As soon as the loud and lively music starts, Walter’s hands begin to twist rapidly in arcs before him, the right one clockwise, the left counterclockwise. He keeps clasping his hands together, whether to stop their movement or to clap I don’t know, but his hands keep breaking loose. His head turns faster and faster, keeping time with both his hands and the music. When the music ends, Walter’s movements slow to a stop. He looks at the ceiling briefly and then sits quietly.

A woman who has a PhD is making a presentation: Temple Grandin has written two books about herself as well as numerous articles on autism. She is also the subject of an intensive case study in the book *An Anthropologist on Mars* (1995) by the neurologist Oliver Sacks. Temple Grandin is a person with autism.

We glimpse here a few of the many faces of autism. One of the most striking aspects of the condition (or conditions) referred to as “autism” is its variability. What then do people identified as having autism have in common? What does the term mean if it encompasses such heterogeneity? What is the concept behind the label? To answer this question we need to look across several perspectives—those of researchers, clinicians, parents, and adults with autism.

We can begin by studying the “bible” of diagnostic categories and labels, the latest version of the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental Disorders, DSM-IV-TR*, published in 2000. DSM-IV-TR doesn’t refer to autism; rather, it refers to *pervasive*
developmental disorders, which include five categories: autistic disorder, Asperger’s disorder, pervasive developmental disorder not otherwise specified, childhood disintegrative disorder, and Rett’s disorder. The term “pervasive developmental disorders” is meant to indicate “severe and pervasive impairment in several areas of development: reciprocal social interaction skills, communication skills, or the presence of stereotyped behavior, interests, and activities” (American Psychiatric Association 2000, 69). In practice, the term “autism spectrum disorders” has largely replaced the term “pervasive developmental disorders,” except in those instances when the very low-incidence conditions of Rett’s disorder or childhood disintegrative disorder are the focus.

Let’s look at what autistic disorder may mean through examples provided by parents.

Qualitative impairment in social interaction. Catherine Maurice describes the social isolation of her infant daughter:

Anne-Marie was not shy: she was largely oblivious to people, and would sometimes actually avoid them, including, a lot of the time, her own mother. She drifted toward solitary spaces: the corners of a room, behind the curtains, behind the armchair. If I was somewhere else in the apartment, she never sought me out....

Worst of all, perhaps, was the lack of that primary connection: the sweet steady gazing into one’s eyes that we began to see all around us in other toddlers. . . . Sometimes I would catch her gazing in my direction and would start up, eager to respond to her invitation, to meet her look. But her eyes, frighteningly, were focused upon some middle distance, somewhere between me and the wall behind me. She wasn’t seeing me at all. She was looking right through me! (Maurice 1993, 31, 33)

Qualitative impairments in communication. Clara Park describes her daughter’s language at age two and then at age twenty-three. At two, her daughter Jessy used words, but infrequently and not to communicate. “She had no idea of language as a tool that could cause things to happen” (1982, 74). As she reached twenty-three, Jessy’s communication problems were still striking:
Anybody who hears Jessy speak more than a word or two realizes that something is wrong. She has learned English as a foreign tongue, though far more slowly, and she still speaks it as a stranger. The more excited she is about what she has to say, the more her speech deteriorates; her attention cannot stretch to cover both what she is saying and how she is saying it. Pronouns get scrambled: “you” for “I,” “she” for “he,” “they” for “we.” Articles and tenses are confused or disappear; verbs lose their inflections or are omitted altogether. (Park 1982, 292)

Restricted repetitive and stereotyped patterns of behavior, interests, and activities. “We watched him as he rocked his body and spun every round object he could find,” recalls Barry Kaufman, describing his not yet two-year-old son’s repetitive behavior (1976, 62). Judy Barron writes of her infant son:

He was drawn to odd things. He’d crawl past a brightly colored selection of toys to get to the furnace register. Once there he would stick his fingers into the slots and watch his fingers move. There was a hole in the wooden floor of his bedroom that riveted his attention. He’d put his finger into that and wiggle it around for hours. (Barron and Barron 1992, 13)

Six-year-old Paul McDonnell became obsessed with light bulbs, his mother, Jane, tells us:

His light bulb collection had grown to include not only incandescent household light bulbs, but also fluorescent bulbs, black lights, infrared and ultraviolet bulbs, and flashcubes: three hundred seventy-two in all. He kept many of his light bulbs in a basket by his bed, and every night he tried out different bulbs. (McDonnell 1993, 154)

As an adolescent Temple Grandin became fixated on squeeze chutes for cattle, an obsession that she later transformed into a therapeutic device to calm herself and others.

All autistic children do not act exactly like the children described here. Not all are as cut off from people as Anne-Marie Maurice was; not all have to struggle as hard as Jessy Park did to use language for communication; and the stereotyped interests of children with autism vary widely.
See appendix A for the specific combinations of behavioral indicators that professionals use to arrive at a diagnosis of autistic disorder.

High-functioning autism by another name is one way of thinking about *Asperger’s disorder*, but DSM-IV-TR treats Asperger’s disorder as a separate category of pervasive developmental disorders distinct in some ways from autistic disorder. Although the two conditions share some core characteristics—namely, severe impairment in social interaction and restricted repetitive patterns of behavior, interests, and activities—Asperger’s disorder, unlike autistic disorder, is not marked by severe delays in language acquisition or cognitive development during the child’s first three years. When the child with Asperger’s disorder begins preschool or approaches school age, problems with social communication often become quite apparent. The label “Asperger’s disorder,” more commonly referred to as “Asperger syndrome,” has come into wide use in the United States only since the mid to late 1990s. Prior to that, children and adolescents who today are identified as having Asperger syndrome would likely have been diagnosed as having emotional disturbance, behavior disorder, and/or attention-deficit/hyperactivity disorder. Other labels used in the past to classify those children and adolescents were schizoid personality and nonverbal learning disabilities.

Individuals with Asperger syndrome are often “loners.” Although most show interest in making friends after the early childhood years, their attempts to do so are frequently unsuccessful. Contributing to such failures are insensitivity to other people’s feelings and nonverbal communications, as well as long-winded, one-sided “conversations” about their own favorite interests. In spite of good academic performance in most areas, or even giftedness, children with Asperger syndrome are commonly viewed as odd, and they often lack even a rudimentary sense of what is socially appropriate. Kenneth Hall, who at age ten produced a book about himself (*Asperger Syndrome, the Universe and Everything*), writes: “If I am unhappy about something I tell the truth. Like if I am fed up with a visitor. Or if I dislike something. Or if I dislike a person. Or if someone does or says something stupid. . . . Sometimes adults get annoyed when I am honest” (2001, 66).
Individuals with Asperger syndrome also often appear to lack common sense, as in Rita’s case. Rita was not identified as needing special education services when she was a child; academically she functioned at grade level in elementary school. But in adolescence her behavior became increasingly age-inappropriate or eccentric, and she was referred for evaluation. Rita tried hard to copy the dress and chatter of her classmates, but she couldn't keep up with the rapid pace of their communications and couldn't master the process of achieving the “right” look. Her first attempts to use makeup produced exaggerated and grotesque results. At age eighteen, Rita moved to a group home where the staff focused on developing skills for independent living and work. On one of her weekend trips to her family home, Rita seemed to be trying to apply some of the skills she had been taught. She entered a fish store and, after a substantial period of consideration, indicated to the proprietor the particular fish she wanted. After the fish had been weighed and she had been given the price, Rita stated that she was now going home to ask her mother if she could buy it.

Young children who do not strictly meet the criteria for autistic disorder or any other pervasive developmental disability are often assigned the label “PDD,” which is a shorthand version of the term "pervasive developmental disorder not otherwise specified" (PDD-NOS). This label indicates that the child displays impairment in reciprocal social interaction and in one of the two other defining areas for pervasive developmental disorders without fitting the picture of another pervasive developmental disorder. By age six or seven, many children with this label have been reclassified as having either autistic disorder or Asperger syndrome.

Sometimes well-meaning professionals use the term “PDD” or “PDD-NOS” with very young children when they feel that a diagnosis of autistic disorder or autism would be premature, and they want to protect families from the frightening associations commonly linked to these terms. PDD is presented as a serious, multifaceted problem in development, but one that is not quite as severe as autism. At times, the concern that leads to the use of the PDD label can have unintended negative effects, such as leaving parents confused or making it more difficult to obtain intensive
early intervention services for the child. Another reason for use of the PDD label is parents’ growing awareness of the importance of early identification of developmental disabilities and the availability of intervention services for very young children with disabilities. While it is possible to identify many children with pervasive developmental disorders at eighteen to twenty-four months of age or even younger, at such an age the distinction between the various categories within autism spectrum disorders is not always clear.

Most people, when they have heard anything at all about autism, have a particular image of it that represents only one of the faces of autism. While the full range of variations in autism may not be apparent in early childhood, one difference can be clearly seen: some children with autism appear different almost from birth, while others appear to develop normally for a period of time and then begin to regress. By the time her daughter was one month old, Annabel Stehli (author of Sound of a Miracle) was sure that something was wrong. Time only added confirmation. Georgie did not grasp her mother’s finger or look at her or smile or snuggle against her when she was being held. Judy Barron (author of There’s a Boy in Here) reported a similar experience with her son Sean, who never seemed to want to be held, squirming, twisting, and pushing against her as if he felt trapped when she picked him up.

Some professionals who are considered experts on autism have in the past stated that what seems to be late-onset regressive-type autism may in fact reflect delay in detection owing to parental denial or lack of sophistication. That may sometimes be the case. I think back to what the mother of my student Nellie said to me over thirty years ago. Nellie was doing all right until she went to nursery school, her mother told me, implying that something had happened at nursery school to cause her daughter’s autistic behavior. Yet home films viewed by the clinical staff had shown a two-year-old Nellie, before any nursery school experience, hiding behind furniture and people, not looking or smiling at anyone, not playing with anyone. Nellie’s mother was trying to account for an unexplainable loss that caused great grief. However, recent studies of the early development of children with autism that have analyzed video-
tapes taken between eight and twelve months of age demonstrate that many parents were right: about 25 percent of these children appeared to be developing typically until sometime during their second year (Werner and Dawson 2005). It was the professionals who doubted parental reports who were proven wrong about late-onset autism.

There’s no question of delayed detection creating a false appearance of late onset in Jordan Schulze’s case. His early development was too advanced and its reversal too sharp a contrast. His parents’ sense of loss was excruciatingly exacerbated by development suddenly halted and then reversed.

A clear-eyed boy of nineteen months is pointing to the gaily decorated tree in my living room. “Christmas lights, Daddy,” he says as he reaches up to touch the bulbs. “This one is green. This one is blue.” Jordan goes through all the colors, carefully pointing out the names of each that he touches. From across the room my wife, Jill, beams as she listens to these first words of the season from our first child. . . .

The here-and-now version of the happy toddler is the seven-year-old boy who, on this day alone, has bitten the school bus driver, flung himself on the floor in a fit of rage in the grocery store, spent nearly half an hour tapping toys on his teeth, and occupied himself in his last waking hour flushing the toilet over twenty times. (Schulze 1993, i–ii)

Childhood disintegrative disorder, or Heller’s syndrome, is a rare pervasive developmental disorder, and it has a very poor prognosis. According to DSM-IV-TR its essential feature is “a marked regression in multiple areas of functioning following a period of at least two years of apparently normal development.” The areas affected include language, social skills or adaptive behavior, play, motor skills, and bowel or bladder control; and this condition is “usually associated with severe mental retardation” (American Psychiatric Association 2000, 77, 78). There are no known biological markers that can be used to differentiate childhood disintegrative disorder from autistic disorder.

Craig Schulze is an educator with a PhD in human development whose son was diagnosed as autistic, but who came to believe that his son had childhood disintegrative disorder. In his 1993 book Schulze describes the deterioration of his young son and his own fruitless strug-
gle to return Jordan to his first life: “It is as if he has died from one existence and returned in another form. . . . Psychologists, neurologists, self-styled gurus, teachers (American and Japanese), relatives and friends, even our own intuitions had joined in a continuing chorus of ‘We Shall Overcome.’ Now the harmony is gone, and the tune is flat, and the voices are silent” (42, 147).

“He” is the common personal pronoun in books on autism, but that usage does not reflect gender bias. Autism is largely a male disorder, approximately four times more prevalent among boys than among girls. But that is not the case for the last category of pervasive developmental disorders. Rett’s disorder, or Rett syndrome, the fifth pervasive developmental disorder presented in DSM-IV-TR, shares several features with both autistic disorder of late onset and childhood disintegrative disorder, but it is virtually exclusive to females. Rett syndrome is a progressive, genetically based neurological disorder in which infants who seemed to be developing typically begin to lose ground sometime after five months of age, with resultant severe impairments in language, cognition, hand usage, and gross motor functioning. An X-linked gene whose mutations lead to Rett syndrome has been identified.

Why are there differences in time of onset and the course of development in autism spectrum disorders? The answer to this question is still elusive. But one of the factors that seems to account for some differences in the course of development is the presence or absence of mental retardation. Some children with autism function as if they have mental retardation, even with intensive early intervention, while others show clear signs of good cognitive ability in multiple areas. However, it is often difficult to determine the cognitive ability of a young child with autism. Aside from the broader issues about the conceptualization of intelligence and the meaning of IQ, how are we to assess intelligence in children who may have no functional speech or any other organized communicative system, who rarely look at people or attend to verbal directions or imitate movements?

Occasionally a child will show unusual abilities that seem to provide clues. Some young autistic children are hyperlexic—that is, they learn to
read at an early age without formal teaching, in spite of a general delay in language development, although this reading may be largely word naming with little comprehension. Other children may show different kinds of evidence of prodigious rote memory. Astounding abilities in art, music, and calendar calculations have been noted in autistic individuals who otherwise appear to be moderately to severely mentally retarded; and “islets” of competence in such areas as mathematics are not uncommon in autistic individuals who otherwise function at a level considered to indicate mild mental retardation. (Sometimes early signs of autism are not recognized because of such islets of competence in the young child.)

IQ in young children does seem to matter. A performance IQ approaching 70, combined with some functional language by age five, seems to be associated with a greater likelihood that the child will have a better developmental outcome. But autistic children are idiosyncratic; they break all rules. Some autistic children are nonverbal until well beyond age five and are considered to be mentally retarded, and then they begin to communicate and prove everyone wrong. Yet the variability of intelligence among individuals with autism is unquestionable. Many young children with autism make slow and limited progress in learning even with intensive intervention, while others have gone from treatment programs to challenging educational programs and on to college studies in such areas as mathematics or science.

**IS AUTISM BECOMING MORE COMMON?**

“There are, due to a tragic accident of nature, children with autism who live in society, but who for some as yet ill-understood reasons cannot profit much from the social stimulation provided by loving and caring parents” (Volkmar 1993, 40). Who are these children? How many are there?

In 2005 the Centers for Disease Control and Prevention of the U.S. Department of Health and Human Services estimated that up to 500,000 individuals between birth and age twenty-one had an autism spectrum disorder, although many of the younger children in this population had not yet been diagnosed and classified. This estimate was arrived at by using a rate of occurrence of 1 in 166 or about 60–62 per 10,000, which
was the rate found in several recent studies that considered the entire autism spectrum. This rate is dramatically higher than those found in earlier studies. For years, the generally accepted rate of occurrence was 4–5 per 10,000, although individual studies sometimes reported higher figures; by the mid to late 1990s, the widely accepted rate was 10–15 per 10,000. While large increases in prevalence have been reported for autistic disorder, the greatest increase in prevalence has occurred in the milder forms of autism spectrum disorders (Chakrabarti and Fombonne 2001).

Autism used to be considered a low-incidence disorder. Now it is referred to as one of the most common childhood conditions within the category of serious developmental disorders, surpassing such conditions as cerebral palsy and Down syndrome. Clearly, something real has been happening to bring about such a large rise in the reported prevalence of autism spectrum disorders. The question still to be resolved is how much of this rise represents a true increase in prevalence and how much reflects other factors such as earlier identification, greater willingness by physicians and psychologists to use diagnostic labels on the autistic spectrum with young children, and modifications in diagnostic categories and criteria. The large increase in Asperger syndrome, in particular, may at least to some extent reflect more familiarity with this diagnostic label and better identification.

One mother raised a question about classification and labeling within the autism spectrum that cannot be answered until the condition we now call autism is better understood. She questions whether high-functioning individuals with autism should be grouped with individuals who have severe autistic disorder and whose behavior and functioning are quite different (Gilbert 1995, 4). The issue here is whether these are distinct conditions or different intensities of the same disorder. Do they have the same etiology, or are the underlying factors different? The answers to these questions are still unknown.

**Etiology: The Search Continues**

The etiology of autism is still not well understood, but autism appears to have a strong and complex genetic foundation. It is more common in
some families than in most others. Studies have shown that the prevalence of autism among siblings of autistic individuals is about 6 percent, substantially higher than in the general population; and the concordance rate for autism in identical twins is much higher than in nonidentical twins. There is no difference in prevalence rate between nonidentical twins and other siblings; but if one identical twin has autism, it is very likely that the other has an autism spectrum disorder as well. This may be the case as much as 90 percent of the time. A higher than expected frequency of Asperger syndrome has been found in the families of high-functioning individuals with autism, and Oliver Sacks refers to an entire family of individuals on the autism spectrum—the gifted parents and older son with Asperger syndrome, the younger son with classic autism—who, “between the serious business of life,” flapped their arms, jumped on a trampoline, and screamed (1995, 244). Moreover, parents or other close relatives with mild versions of autism have been identified in a good number of families of children with autism; and even when no other person in a family has an autism spectrum disorder, the family may have a higher than expected prevalence of individuals with social and communicative impairments. These findings strengthen the hypothesis that autism has a genetic base.

If autism spectrum disorders are, in fact, more common today than they were in earlier decades, what is causing this increase? The answer presented most frequently during the past few years by parents is vaccines. Why vaccines? The hypothesized genetic basis of autism did not seem to adequately account for the increase in prevalence reported in the past few years. What many parents began to notice was a close connection in time between the administration of a triple live-virus vaccine for measles, mumps, and rubella (MMR) when their children were twelve to fifteen months old and a deterioration in their babies’ functioning. The medical community explained this association as a noncausal coincidence in time, but Andrew Wakefield, an English doctor, hypothesized that the MMR vaccine might be triggering regressive autism in genetically vulnerable children.

Vaccines had long been recognized as causing problems in a tiny fraction of young children, and the Centers for Disease Control and Preven-
tion and the U.S. Food and Drug Administration maintain a Vaccine Adverse Event Reporting System. Before the MMR vaccine became the focus of controversy in regard to autism, the pertussis toxin in the diphtheria-tetanus-pertussis (DTP) vaccine was identified as posing a risk of brain damage for a very tiny percentage of young children, and action was taken to modify the pertussis formulation in the version of this vaccine that is now in use (DTaP). However, the number of children harmed by vaccines was considered so low relative to the imposing benefits of vaccination that this problem of adverse reactions received only limited attention from the medical community.

Parents and their supporters have raised multiple questions about vaccines. While the role of the MMR vaccine as a possible trigger for autism was being examined and debated, another possible trigger associated with vaccines was identified. This time the target of parental concern was the inclusion of thimerosal, a substance containing a form of mercury, as a preservative in some of the vaccines administered to children during their first and second years. Although a review conducted by the Food and Drug Administration concluded that no harm had taken place because of vaccines containing thimerosal, the agency nonetheless recommended that, as a precautionary measure, the use of thimerosal should be reduced or eliminated. In 1999 an agreement was forged with vaccine manufacturers to accomplish that goal. As of 2005, only one childhood vaccine, the flu vaccine, still contained thimerosal, and a thimerosal-free version of this vaccine had become available.

While most children who received vaccines containing thimerosal had no long-term adverse outcomes, many parents whose autistic children had been given such vaccines believed that the mercury in thimerosal had overwhelmed their children’s immune systems and led to autism. These parents argued that the total amount of mercury to which their babies had been exposed through vaccines far exceeded safety standards. Researchers pointed out that the standards parents referred to were for a different type of mercury—methylmercury rather than the ethylmercury in thimerosal—and that methylmercury crosses the blood-brain barrier more easily. Moreover, research studies of the MMR vaccine (which did not contain thimerosal) and of vaccines containing thimerosal appeared