SECTION VI

Language Pathology in Neuropsychiatric Disorders

Autism

S. Baron-Cohen

Autism is often described as the most severe of all of the child psychiatric disorders. Yet, surely each disability is severe in its own way? Autism has gained this reputation because, unlike all other childhood disorders, people with autism appear to be virtually cut off from other people—'in a world of their own.' It is in this sense that autism is also categorized as a psychosis: like schizophrenia, autism appears to be qualitatively unlike anything in the normal range of experience. In contrast, neurotic disorders (such as anxiety or depression) seem closer to experiences in the normal range.

Even the other communication disorders of childhood do not leave the sufferer isolated to quite the same degree as occurs in autism. Thus, although dysphasic disorders of childhood include language comprehension or expression deficits, somehow the social contact between the sufferer and other people is not severed: children with various dysphasias still find some way of making and developing relationships with others—family, teachers, and friends (see *General Aspects of Developmental Language Disorders*). They may use sign language, impoverished speech, or even simply eye contact and gesture. Not so children with autism. For them, even understanding what communication is for seems to be missing. As will be described below, this is part of the social difficulties that lie at the core of autism.

1. What is Autism?

Autism is a psychiatric disorder which begins during the first three years of life (possibly from birth, or even before). It affects approximately four children in every 10 000, although some studies have suggested it may be as common as 15–20 per 10 000. Males are affected three times as often as females, and two-thirds of people with the condition have learning difficulties in addition to the problems specific to autism. That is, two-thirds of children with autism have an IQ (or measured intelligence) below the average range. And even those whose intelligence is in the normal range show an unusual pattern of skills, with visuospatial intelligence usually being superior to verbal abilities.

Various sets of diagnostic criteria exist, but all of these share an emphasis on three key symptoms. First, the child fails to make normal social relationships, or to develop socially in the normal way. Instead, social interests tend to be one-sided, nonreciprocal, and exist only to satisfy the child's immediate wishes. Missing are any genuinely social games (or turn-taking), any attempt to share interests through joint-attention behaviors (such as using the pointing gesture to indicate things of interest to people, or showing people things of interest), normal use of eye contact, or any friendship beyond the most superficial acquaintance. A lack of empathy is often identified as the central feature of the social deficit.

Second, the child fails to develop language or communication in the normal way. This symptom can include a multitude of anomalies. For example, some children with autism are functionally completely mute (see *Mutism*), whilst others are slow learning to speak, and their language development severely limited. Yet others can speak in full sentences, but nevertheless show a range of speech abnormalities, and fail to use their speech appropriately to achieve communication or to use gesture in a normal way. These abnormalities are described in detail later.

The final symptom constitutes repetitive behavior, in conjunction with a lack of normal imagination. Thus, children with autism often carry out the same action over and over again, becoming quite distressed if other people attempt to prevent them from carrying out their repetitive rituals, and their play is often devoid of any apparent creativity or imagination. During play, for example, children with autism often simply arrange objects in strict geometric patterns in the same way every day, rather than transforming objects into *pretend* or symbolic play, as normal children do even from the age of about 18 months.

Tragically, whilst the symptoms may change in form as people with autism get older, and whilst with

age a considerable amount of learning may be possible, autism appears to be a lifelong condition. Some claims of 'cures' have been reported, but in none of these cases has recovery to a *normal* state been verified, and in the majority of cases individuals remain 'odd' and obviously disabled in adulthood.

2. Causes

Various causes of autism have been identified, all biological, and all of these are assumed to disturb the normal development of the central nervous system. The major causes for which there is scientific evidence are genetic, perinatal, viral, and a variety of medical conditions.

The genetic evidence centers on the higher concordance rate for autism among monozygotic (i.e., genetically identical) twins, where one has autism, than among dizygotic (i.e., genetically nonidentical) twins, where one has autism. In addition, some 2–3 percent of the siblings of children with autism also develop autism, and this is approximately 50 times higher than one would expect from chance alone. The perinatal evidence centers on the increased risk for autism produced by a range of complications during pregnancy and labor. The viral evidence centers on the statistically significant association between autism and infection by the rubella virus during pregnancy.

Finally, the range of medical conditions which can be associated with autism (and which, in those cases, are assumed to be causal) include genetic disorders (such as Fragile X Syndrome, phenylketonuria, tuberous sclerosis, neurofibromatosis, and other chromosomal anomalies); metabolic disorders (such as histidinemia, abnormalities of purine synthesis and of carbohydrate metabolism); and congenital anomaly syndromes (such as Cornelia de Lange Syndrome, Noonan Syndrome, Coffin Siris Syndrome, William's Syndrome, Biedl-Bardet Syndrome, Moebius' Syndrome, and Leber's Amaurosis).

No single cause has been identified for all cases, and theories suggest there may instead be several separate causes of autism, any one of which may affect the part of the brain that produces the condition. This view has come to be known as the 'final common pathway' hypothesis. Abnormalities have been found in various regions of the brain in different cases, but again none of these are consistent across all individuals with autism. The exception to this is the finding that the cerebellum may show specific atrophy in all cases. This new work remains to be replicated. But the clearest evidence that there is brain dysfunction in autism stems from the fact that some 30 percent of people with autism also develop epilepsy at some stage in their lives. Finally, autism has not been demonstrated to be associated with either poor parenting (contrary to early theories) or social factors (such as class or culture).

3. What are the Language Abnormalities in Autism?

Language abnormalities exist in all of the subsystems of language. In syntax, for example, there can be considerable delays in the rate of acquisition of syntactical forms, although longitudinal studies show that the order of acquisition does not differ to that found either in normal children or children with learning difficulties. Thus, children with autism who develop speech usually go through a one-word and a two-word phase, their 'mean length of utterance' (MLU) usually increases in normal ways, and the syntactical forms used seem to appear in the same order as in normal development. In phonology, intonation can sometimes be rather monotonous and 'mechanical' sounding, but otherwise is often normal, if not superior. Thus, when children with autism produce *echolalia* (echoing someone else's speech), it is often with identical intonation to the person who spoke first (see *Phonological Disorders of Language*).

In semantics, words are clearly referential, but neologisms may be present. Thus, the child may use a word that is not a conventional one, but which nevertheless has a meaning for that child. For example, one boy with autism referred to a cat as a 'milk outside.' When the origin of such neologisms is traced, they are often found to derive from incomplete learning during the first usage of the term. In the example above, the boy's mother often used to say 'Let's put the milk outside for the cat.' Kanner, the psychiatrist who first described autism in 1943, characterized such neologisms in the speech of children with autism as 'metaphorical,' although it is worth stressing that these do not conform to cases of true metaphor. Indeed, semantic abnormalities in the speech of people with autism include difficulties in understanding or creating true metaphors and other forms of figurative language (such as irony or sarcasm).

Other semantic abnormalities are seen in the production of echolalia: these may be either *immediate*, where the person repeats straight back what the other person has just said, or *delayed*, where the person repeats back a segment of conversation that was overheard some time before. In delayed echolalia, the speech echoed may be part of a television jingle, or lyrics from a song, and often testifies to excellent long-term memory in people with autism.

However, of all the language abnormalities in autism, the most severe are in the pragmatics of speech. Almost every aspect of pragmatics that has been studied in people with autism has been found to be abnormal. Thus, the range of *speech acts* that they produce is quite limited—requests being the most frequent, informatives or humorous speech acts being quite rare. They also appear not to realize how to use language in a way that is sensitive to the social context. For example, they tend to say things that are rude, not because of any wilful desire to offend, but simply because they are blind to the polite/rude distinction

(e.g., one child with autism correctly noticed but then said out loud 'That woman has dyed her moustache!'). Furthermore, they often do not distinguish old and new information in a conversation, failing to take into account what the listener already knows or does not know. For example, they may repeat things they have already told the listener, or they may refer to things that the listener could not possibly know about, without explaining these. It is also rare for them to introduce their topic so that the listener can appreciate its relevance (e.g., by using phrases such as 'You know I was in France for my holidays, well...').

Another instance of the pragmatics deficit in the language of people with autism is seen in the lack of normal turn-taking in conversation. Instead, they may talk at the same time as the other person, or deliver extended monologues, or simply not reply at all when a reply is expected. This can appear as a failure to recognize the intention behind a question. For example, when asked 'Can you pass the salt?,' a person with autism may simply reply 'Yes.' Such a limited reply is not a sign of wilful rudeness, but simply due to a failure to recognize the question as a request for an object.

The pragmatics deficit is also seen in the use of a pedantic style of language that is inappropriate for the social situation. For example, one girl with autism asked 'Do you travel to work on a driver-only operated number 68 bus?' Also, many people with autism do not establish eye contact with the listener before speaking, or use eye contact to regulate any conversational turn-taking. Finally, some studies have shown that they tend to ask questions to which they already know the answers, thus violating the rules about the conventional uses of different parts of speech.

4. Relationship between the Language and the Social Abnormalities

During the 1960s and early 1970s one major theory of autism argued that the social abnormalities in this disorder were secondary to the language problems. This theory lost credibility when studies compared children with dysphasias (see *General Aspects of Developmental Language Disorders*) and children with autism. Such studies demonstrated that language disabilities did not inevitably produce social disabilities, in that children with even severe dysphasia nevertheless often showed surprisingly intact social skills and sensitivities. In contrast, late twentieth-century psychological theories suggest that language delay is an entirely independent disability which may cooccur in autism, whilst the abnormalities in pragmatic competence are an inevitable consequence of the social disability in people with autism, and are seen in all cases. One such psychological theory is elaborated in Sect. 5.

5. The Mindblindness Theory

In the early 1990s experiments demonstrated that people with autism are severely impaired in their understanding of mental states, such as beliefs and thoughts, and in their appreciation of how mental states govern behavior. This ability in normal people has been referred to as a 'theory of mind'—because of how people use their concepts of other people's mental states to explain their behavior. Attributing mental states such as thoughts, desires, intentions, etc., to other people allows the individual to understand why people do what they do, and in keeping track of both other people's mental states and their own, to mesh flexibly in social interaction.

Apart from using a theory of mind to make sense of the social world, and to participate in it, a second key function of a theory of mind in normal people is to make sense of communication, and to communicate with others. In computing the meaning and relevance of another person's speech the listener constantly takes into account the speaker's background mental state, and in making speech meaningful and relevant to a listener the speaker does the same.

Given these two functions of a theory of mind, it is clear that, if people with autism are unable to appreciate that other people have different mental states, this would severely impair their ability to understand and participate not only in social interaction, but also in communication itself. It is in this sense that the deficits they show in pragmatics are thought to be intimately entwined with their social deficits. This core inability to appreciate other people's mental states has been termed 'mindblindness.' Research is elucidating which mental states are easier to understand (e.g., for people with autism, desire seems to be easier to understand than either pretence or belief); whether this problem constitutes a case of specific developmental delay (in that some children with autism do eventually develop a theory of mind, years after it emerges in normal development); and what the origins of their mindblindness might be.

6. Treatment

Treatment in the 1990s centers on special education for children with autism, and the most effective techniques seem to include highly structured, individually tailored behavior therapy, aimed at skill-building, reducing difficult behaviors, and the facilitation of educational achievements. Other specialist therapies also play important roles, and these include speech and music therapies. Sign languages, such as Makaton or Paget-Gorman, are also used with some children with autism, if speech is particularly limited. However, none of these treatments claims any dramatic success in removing the core social abnormalities, although these may become less intrusive and disabling over time. Medical treatments exist for specific difficulties, such as epilepsy and hyperactivity, but at present there

are no medical treatments which are useful in ameliorating the language or social difficulties in people with autism. Current and future research is aiming to find the links between the behavioral, psychological, and biological abnormalities in this condition, as well as seeking to develop more effective treatment and diagnostic methods.

Bibliography

Baron-Cohen S 1988 Social and pragmatic deficits in autism: Cognitive or affective? *Journal of Autism and Developmental Disorders* **18(3)**: 379–402

Baron-Cohen S 1995 *Mindblindness*. MIT Press/Bradford Books, Cambridge, MA

Baron-Cohen S, Leslie A M, Frith U 1985 Does the autistic child have a 'theory of mind?' *Cognition* **21(1)**: 37–46

Frith U 1989 *Autism: Explaining the enigma*. Blackwell, Oxford

Howlin P, Rutter M, et al. 1987 *Treatment of Autistic Children*. Wiley, Chichester

Kanner L 1943 Autistic disturbance of affective contact. *Nervous Child* **2**: 217–50

Tager-Flusberg H, Calkins S, Nolin T, Baumberger T, Anderson M, Chadwick-Dias A 1990 A longitudinal study of language acquisition in autistic and Down's Syndrome children. *Journal of Autism and Developmental Disorders* **20(1)**: 1–22

Schopler E, Mesibov G B 1987 *Neurobiological Issues in Autism*. Plenum Press, New York

Mutism

Y. Lebrun

Mutism is a condition in which there is no, or very little, oral–verbal expression, while comprehension of speech (and possibly also of written language) is normal or, at least, is at a considerably higher level than expressive speech. It is precisely the large discrepancy between the nonexistent or very scanty oral–verbal output and the normal or nearly normal receptive abilities that is characteristic of mutism.

Mutism does not necessarily mean the total absence of sounds produced with the vocal tract. Mute people may make noises either deliberately in attempts to communicate or spontaneously in reaction to various stimuli, for example, pain. But these noises are not speech sounds. Mutism thus refers to the absence of articulate speech. Also, mutism does not imply total lack of self-expression. Mute individuals may use nonvocal systems of communication. In fact, if literate, they often resort to writing.

1. Degree of Intentionality

At times, mutism is totally deliberate. When a prisoner resolves to keep his own counsel and not to answer the questions of those who have captured him, his silence is completely voluntary. Similarly, cloisterers may deliberately refrain from talking in order to commune better with God.

However, silence may also be completely unintentional. Following brain damage, an individual may be unable to express himself orally. He would like to communicate verbally but cannot because an organic lesion prevents him from speaking.

Silence, then, may be fully deliberate or, on the contrary, completely involuntary. Between these two extremes there are intermediate stages where the degree of intentionality is less easy to ascertain.

2. Functional versus Organic Mutism

In clinical practice a distinction is usually made between functional and organic mutism. The diagnostic label 'functional mutism' (or 'psychogenic mutism') is used when an individual cannot bring himself to speak under certain circumstances or in the presence of certain people, or when speech is completely suppressed, although on examination the peripheral speech organs appear normal and no central lesion can be discovered that would reasonably account for the absence of oral–verbal expression. 'Organic mutism' is spoken of when there is an organic lesion that can legitimately be made responsible for the speechlessness.

At times, organicity and functionality combine, as when a patient who, following brain damage, has severe speech difficulties, chooses to remain silent rather than to attempt painstakingly a few words.

Functional mutism is usually subdivided into selective (or elective) mutism and total (or hysterical) mutism.

3. Selective Mutism

Selective mutism denotes a condition in which the individual keeps completely silent in certain places or in the presence of certain people, while he uses speech in other places or when interacting with other people. Patients with selective mutism can use speech for communicative purposes but fail to do so in a number of circumstances where to speak would be the appro-