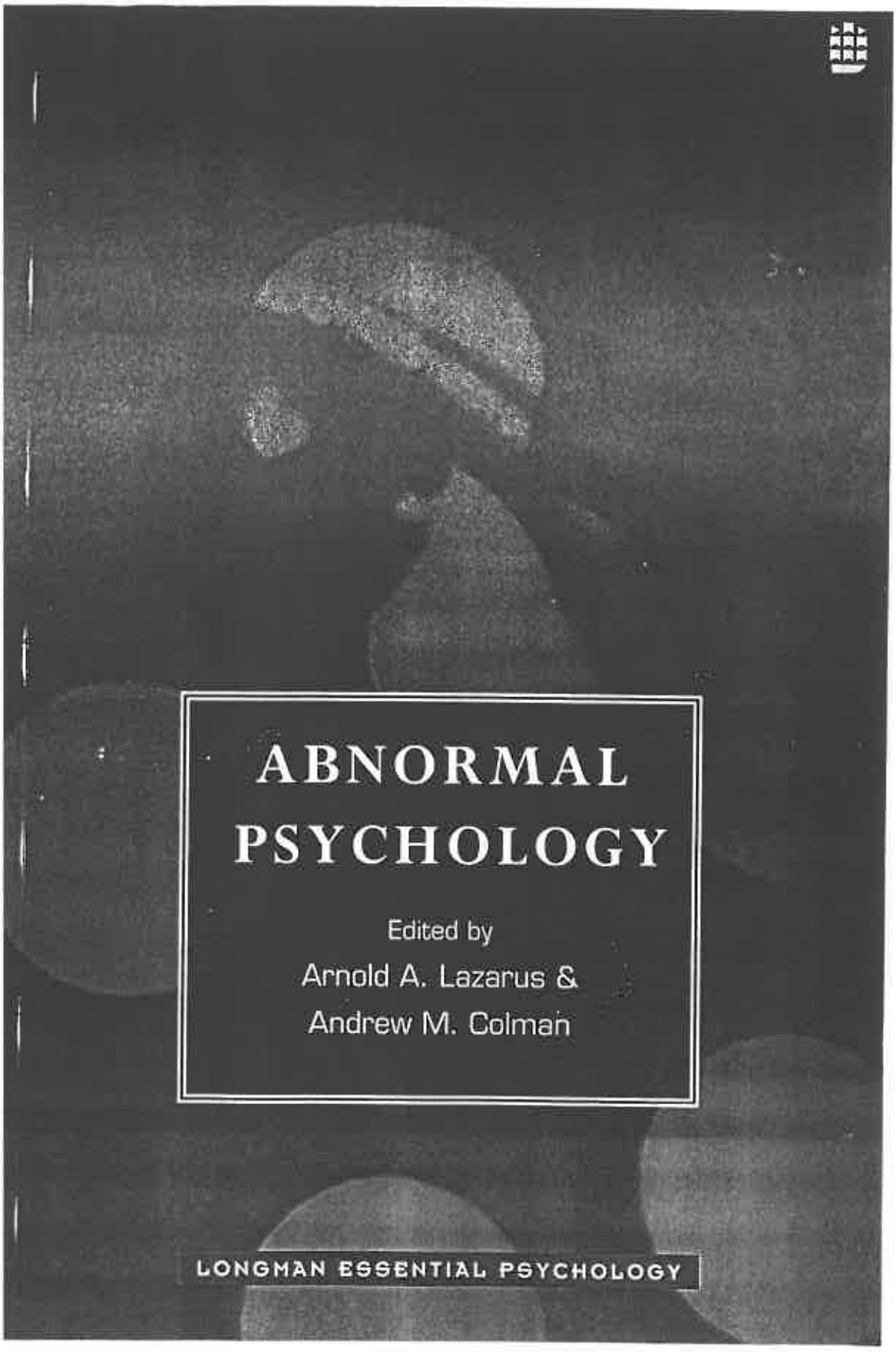

ABNORMAL PSYCHOLOGY

Edited by

Arnold A. Lazarus &

Andrew M. Colman

LONGMAN ESSENTIAL PSYCHOLOGY

3

INFANTILE AUTISM

Simon Baron-Cohen

University of London Institute of Psychiatry, England

Autism is often described as the most severe of all of the child psychiatric disorders. Why should this be? 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 sometimes 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. They may use sign-language, impoverished speech, or even simply eye-contact and gesture. This is not true of children with autism. For them, even understanding *what*

communication is for seems to be missing. Why? As I shall describe, this is part of the *social* difficulties that lie at the core of autism.

WHAT IS AUTISM?

Autism is a psychiatric disorder which begins during the first three years of life (American Psychiatric Association, 1987). It affects approximately 4 children in every 10,000, although some studies have suggested it may be as common as 15–20 per 10,000 (Frith, 1989). Boys are affected three times as often as girls; two-thirds of children 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 (Rutter, 1985). Even those whose intelligence is in the normal range show an unusual pattern of skills, with visuo-spatial intelligence usually being superior to verbal abilities (Frith, 1989).

Various sets of diagnostic criteria exist (American Psychiatric Association, 1987; Rutter, 1985), 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, non-reciprocal, 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 behaviours* (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 (Baron-Cohen, 1988; Hobson, 1986, 1993; Kanner, 1943).

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, while 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 below.

The final symptom is repetitive behaviour, 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 (Baron-Cohen, 1987). 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 (Leslie, 1987).

Tragically, while the symptoms may change in form as people with autism get older, and while with age a considerable amount of learning may be possible, autism appears to be a lifelong condition (Frith, 1989). 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.

CAUSES

Various possible causes of autism have been identified, all biological, and all of these are assumed to disturb the normal development of the central nervous system (Gillberg, 1990). The major causes for which there is scientific evidence are genetic, perinatal, viral, and a variety of medical conditions.

The genetic evidence centres on the higher concordance rate for autism among monozygotic (genetically identical) twins, where one has autism, than among dizygotic (genetically non-identical) twins, where one has autism (Bolton & Rutter, 1990). In addition, some 2–3 per cent of the siblings of children with autism also develop autism, and this is approximately 50 times higher than one would expect from chance alone (Bolton & Rutter, 1990). The perinatal evidence centres on the increased risk for autism produced by a range of complications during pregnancy and labour. The viral evidence centres on the statistically significant association between autism and infection by the rubella (German measles) virus during pregnancy (Wing, 1969).

Finally, the range of medical conditions associated with autism (and assumed to be causal) include genetic disorders (such as Fragile X Syndrome, phenylketonuria, tuberous sclerosis, neurofibromatosis, and other chromosomal anomalies); metabolic disorders (such as histidinaemia, abnormalities of purine synthesis and of carbohydrate metabolism); and congenital anomaly syndromes (such as Cornelia de Lange Syndrome, Noonan Syndrome, Coffin-Siris Syndrome, Williams Syndrome, Biedl-Bardet Syndrome, Moebius' Syndrome, and Leber's Amaurosis). These are reviewed by Gillberg (1990).

No single cause has been identified for all cases, and current theories suggest that there may instead be several separate causes of autism, any 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. Using neuroimaging techniques, brain abnormalities have been found in various regions of the brain in different cases, but again none of these is consistent across all individuals with autism (George, Costa, Kouris, Ring, & Ell, 1993). The exception to this is the finding that the cerebellum may show specific atrophy in all cases (Courchesne, Yeung-Courchesne, Press, Hesselink, & Jernigan, 1988). This work remains to be replicated. But the clearest evidence that there is brain dysfunction in autism stems from the fact that some 30 per cent of people with autism also develop epilepsy at some stage in their lives (Rutter,

1985). Finally, autism has not been demonstrated to be associated with either poor parenting, or social factors such as class. This last statement rules out some early theories of autism. For example, Bettelheim (1968) had proposed that the mothers of children with autism gave inadequate emotional input to their children, preventing the formation of the primary bond between mother and child, and thus preventing further social development or development of the child's concept of self. Tinbergen and Tinbergen (1983) argued for a similar characterization of autism, emphasizing traumatic factors that might have prevented the primary mother–child attachment relationship from forming. Finally, Kanner (1943) emphasized the predominantly intellectual, upper-middle-class nature of the parents of children with autism, implying that a lack of emotion in the parents may have caused the child's autism. None of these claims has been supported by subsequent work (Frith, 1989).

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 rate of acquisition of syntactical forms, although longitudinal studies show that the order of acquisition does not differ from that found either in normal children or in children with learning difficulties (Tager-Flusberg et al., 1990). 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 first uttered it.

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 (Happé, 1992).

Other semantic abnormalities are seen in the production of echolalia – either *immediate*, where the person with autism 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.

But of all the language abnormalities in autism, the most severe are in the pragmatics of speech. By pragmatics is meant the rules governing the appropriate *use* of language in specific social contexts, and the rules for inferring a speaker's intended meaning. Almost every aspect of pragmatics that has been studied in people with autism has been found to be abnormal (see Baron-Cohen, 1988, for a review). Thus, the range of *speech acts* that they produce is quite limited – requests being the most frequent, informative 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 pragmatic 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 pragmatic 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 conventional uses of different parts of speech.

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 (Rutter, 1985). This theory lost credibility when studies compared children with dysphasia 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, more recent psychological theories suggest that language delay is an entirely independent disability which may co-occur in autism, while 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 below.

THE MIND-BLINDNESS THEORY

Experiments have 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 behaviour (Baron-Cohen, Leslie, & Frith, 1985; Baron-Cohen, 1993). This ability in normal people has been referred to as a "theory of mind" (Premack & Woodruff, 1978) because of how we use our concepts of people's mental states to explain their behaviour. Attributing mental states such as thoughts, desires, intentions, and so on to other people allows us to understand why people do what they do, and in keeping track of both other people's mental states and our own, we can mesh flexibly in social interaction.

Apart from using a theory of mind to make sense of the social world, and to participate in it (Dennett, 1978), a second key function of a theory of mind in normal people is to make sense of communication, and to communicate with others (Grice, 1975). In computing the meaning and relevance of another person's speech we constantly take into account their background mental states, and in making our speech meaningful and relevant to our listener, we do the same (Sperber & Wilson, 1986).

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 not only to understand and participate in social interaction, but also communication itself. It is in this sense that the deficits they show in pragmatics are thought to be intimately entwined with their social deficits. A number of experiments have demonstrated specific difficulties for people with autism in understanding the mental states of belief, knowledge, pretence, and intention (Baron-Cohen et al., 1985; Leslie & Frith, 1988; Goodhart & Baron-Cohen, 1992; Phillips, 1993).

One example of a test of understanding belief is shown in Figure 1. This core inability to appreciate other people's mental states has been termed "mind-blindness" (Baron-Cohen, 1990). Current research is elucidating 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 (Baron-Cohen, 1989a), and what the origins of their mind-blindness might be (Baron-Cohen, 1989b; Baron-Cohen, 1993; Hobson, 1993).

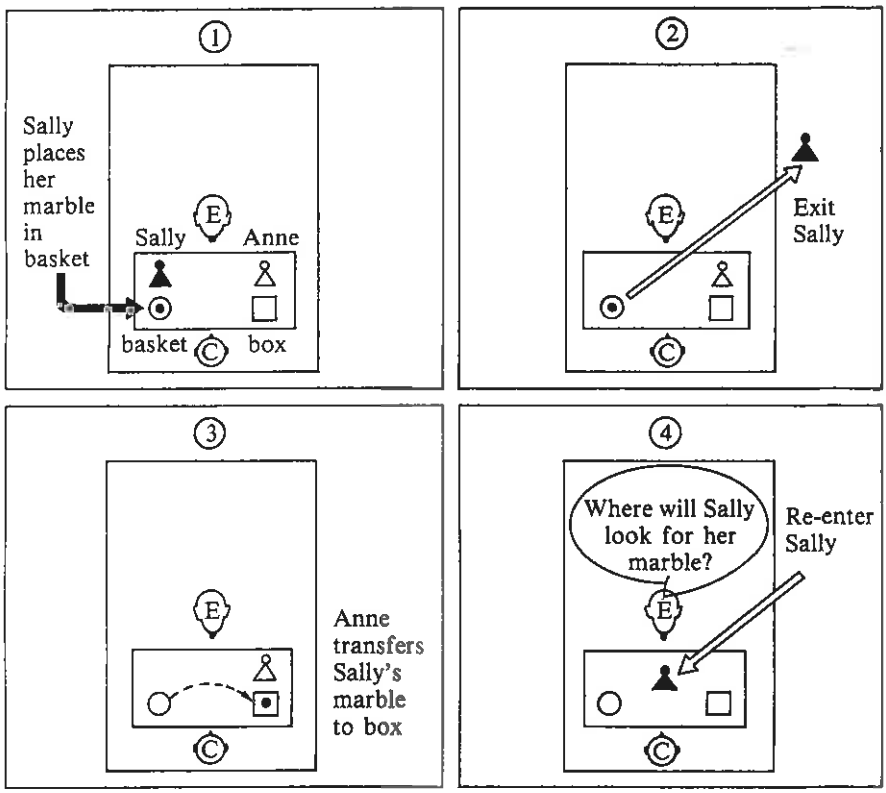

Figure 1 A test of children's understanding of belief. The story: *Sally puts her marble in the basket. Then she goes out. Anne takes Sally's marble, and puts it into her box. Then Sally comes back from her walk. Where will she look for her marble?* Normal 4-year-old children have no difficulty in correctly pointing to the basket, in answer to this question. In contrast, children with autism usually point (incorrectly) to the box

Source: Taken from Baron-Cohen, Leslie, and Frith, 1985, with permission

COGNITIVE MECHANISMS

The failure to develop a normal theory of mind in autism has been explained by several theories. Perhaps the most detailed account to date has been advanced by Leslie (1987; Leslie & Roth, 1993) who argues that in the normal case there is a specialized module called the *theory of mind mechanism* (ToMM) which matures around 12–18 months of age, and which processes information in the form of *metarepresentations*. These are essentially representations of mental representations, or representations of propositional attitudes. Leslie argues that this module for processing metarepresentations is not the same as a general capacity for representing *any* representation (such as a drawing, or a map, or a photograph). Rather, it is a highly specialized mechanism for representing *mental* representations. Evidence in favour of this specialized function comes from experiments showing that children with autism are able to represent non-mental representations such as photographs (Leekam & Perner, 1991) and drawings (Charman & Baron-Cohen, 1992), despite failing tasks of representing beliefs.

A second proposal, suggested by Frith (1989), is that the theory of mind deficit is just one part of a larger deficit in cognition, in the capacity for finding "central coherence": by this, she means the ability to use context to relate otherwise disparate sets of information. In the normal case, this ability to find central coherence can be seen in the non-social domain in our tendency to be distracted by overall *meaning* when perceiving a scene, rather than focusing on individual parts in the scene. Her work has shown that children with autism are more accurate in tasks such as the Children's Embedded Figures Test, in which the subject has to identify a target shape among a more complex, meaningful design (Shah & Frith, 1983), for example, identifying the triangle within the picture of the pram (depicted in Figure 2). By extension, she argues that in the social domain, theory of mind is par excellence an illustration of how we normally find central coherence. Rather than focusing on the myriad of individual behaviours, we focus on inferred mental states that we assume must underlie these behaviours. In Frith's theory, the superiority of children with autism on tasks like the Embedded Figures Test, and their deficits on theory of mind tasks, can be explained by reference to this single impairment in finding central coherence. Note that this explanation is opposed to Leslie's account, in that his account is highly modular, while hers is not. Frith's theory also predicts that children with autism should have difficulties in building *any* theory about some aspect of the world, not just a theory of mind. Tests of whether children with autism develop theories of biology would, for example, provide data with which to evaluate the coherence theory.

A third account that has been proposed is that the theory of mind deficits in autism may be secondary to deficits in *executive function* (Hughes, Russell and Robbins, 1993). By "executive function" is meant the ability to

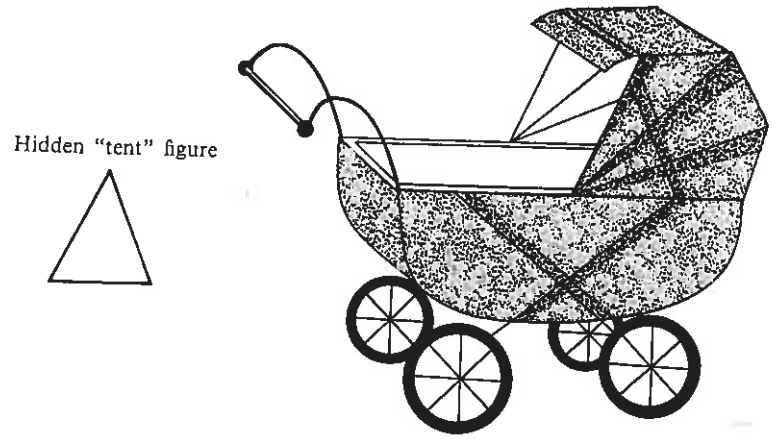

Figure 2 An example of an item from the Children's Embedded Figures Test
Source: Described by Shah and Frith, 1983; reprinted with permission

inhibit responses to salient stimuli in the here-and-now, in favour of representations of objects, plans, or events that are not currently present. Individuals with autism, like many patients with frontal lobe damage, show impairments in tests of executive functioning (Hughes, Russell & Robbins, 1993; Ozonoff, Pennington, & Rogers, 1991), and this is the main evidence in favour of this account. An alternative possibility is that there is not a *single* cognitive deficit in autism, but rather there are several. It may be that the brain damage responsible for theory of mind impairments is localized in the same area of the brain that can also produce executive function deficits, namely, in the frontal lobe (Baron-Cohen et al., 1993). On this view, executive function and theory of mind deficits may be independent of one another, but tend to co-occur in the autistic syndrome by virtue of their neural proximity to each other. Testing the independence of these deficits is an important question for research in this area.

EARLY DIAGNOSIS

Leaving the question of the nature of the cognitive mechanism underlying the theory of mind deficit to one side, another area of research has been exploring developmental *precursors* to the theory of mind deficit in autism, partly towards understanding the ontogenesis of this psychological deficit, and partly to test if abnormalities in these precursors might be useful in the early diagnosis of autism. Candidate precursors of theory of mind are joint-attention skills (Baron-Cohen, 1989c, 1993) and pretend play (Leslie, 1987). Not only have these been found to be absent or impoverished in older

children with autism (Baron-Cohen, 1987; Sigman, Mundy, Ungerer, & Sherman, 1986), but their absence in a sample of 18 month olds at raised genetic risk for autism predicted which children were undiagnosed infants with autism (Baron-Cohen, Allen, & Gillberg, 1992).

TREATMENT

Currently, treatment centres on special education for children with autism, and the most effective techniques seem to include highly structured, individually tailored behaviour therapy, aimed at skill-building, reducing difficult behaviours, and facilitation of educational achievements (Howlin & Rutter, 1987). 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 behavioural, psychological, and biological abnormalities in this condition, as well as aiming at developing more effective treatment and diagnostic methods.

ACKNOWLEDGEMENTS

This work was written while the author was in receipt of grants from the Medical Research Council, the British Council, the Royal Society, and the Mental Health Foundation. Parts of this chapter appeared in J. Cooper (Ed.) (1992). *The Encylopaedia of Language and Linguistics*. Oxford: Pergamon and Aberdeen University Press. Permission to reprint these parts is gratefully acknowledged.

FURTHER READING

Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. J. (Eds) (1993). *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.

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

Howlin, P., & Rutter, M. (1987). *Treatment of autistic children*. Chichester: Wiley.

Schopler, E., & Mesibov, L. (1988). *Neurobiological issues in autism*. New York: Plenum.

REFERENCES

American Psychiatric Association (1987). *Diagnostic and statistical manual of mental disorders* (3rd edn). Washington, DC: APA.

Baron-Cohen, S. (1987). Autism and symbolic play. *British Journal of Developmental Psychology, 5*, 139–148.

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

Baron-Cohen, S. (1989a). The autistic child's theory of mind: A case of specific developmental delay. *Journal of Child Psychology and Psychiatry, 30*, 285–298.

Baron-Cohen, S. (1989b). Are autistic children behaviourists? An examination of their mental–physical and appearance–reality distinctions. *Journal of Autism and Developmental Disorders, 19*, 579–600.

Baron-Cohen, S. (1989c). Perceptual role-taking and protodeclarative pointing in autism. *British Journal of Developmental Psychology, 7*, 113–127.

Baron-Cohen, S. (1990). Autism: A specific cognitive disorder of "mind-blindness". *International Review of Psychiatry, 2*, 79–88.

Baron-Cohen, S. (1993). From attention-goal psychology to belief-desire psychology: The development of a theory of mind and its dysfunction. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds) *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.

Baron-Cohen, S., Allen, J., & Gillberg, C. (1992). Can autism be detected at 18 months? The needle, the haystack, and the CHAT. *British Journal of Psychiatry, 161*, 839–843.

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

Baron-Cohen, S., Ring, H., Moriarty, J., Schmitz, B., Costa, D., & Ell, P. (1993). *The role of the orbito-frontal region of the brain*. Unpublished manuscript, Institute of Psychiatry, University of London.

Bettelheim, B. (1968). *The empty fortress*. Chicago: Free Press.

Bolton, P., & Rutter, M. (1990). Genetic influences in autism. *International Review of Psychiatry, 2*, 67–80.

Charman, T., & Baron-Cohen, S. (1992). Understanding beliefs and drawings: A further test of the metarepresentation theory of autism. *Journal of Child Psychology and Psychiatry, 33*, 1105–1112.

Courchesne, E., Yeung-Courchesne, R., Press, G., Hesselink, J., & Jernigan, T. (1988). Hypoplasia of cerebellar vernal lobules VI and VII in infantile autism. *New England Journal of Medicine, 318*, 1349–1354.

Dennett, D. (1978). *Brainstorms: Philosophical essays on mind and psychology*. Sussex: Harvester.

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

George, M., Costa, D., Kouris, K., Ring, H., & Ell, P. (1993). Cerebral blood flow abnormalities in adults with infantile autism. *Journal of Nervous and Mental Diseases*.

Gillberg, C. (1990). What is autism? *International Review of Psychiatry, 2*, 61–66.

Goodhart, F., & Baron-Cohen, S. (1992). *Do children with autism understand how knowledge is acquired?* Unpublished manuscript, Institute of Psychiatry, University of London.

Grice, H. P. (1975). Logic and conversation. In R. Cole & J. Morgan (Eds) *Syntax and semantics: Speech acts*. New York: Academic Press (original work published in 1967).

Happé, F. (1992). *A test of Relevance Theory: Communicative competence and theory of mind in autism*. Unpublished manuscript, MRC Cognitive Development Unit, London.

Hobson, R.P. (1986). The autistic child's appraisal of expressions of emotion. *Journal of Child Psychology and Psychiatry, 27*, 321–342.

Hobson, R. P. (1993). Understanding persons: The role of affect. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen, (Eds) *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.

Howlin, P., & Rutter, M. (1987). *Treatment of autistic children*. Chichester: Wiley.

Hughes, C., Russell, J., and Robbins, T. (1993). Autistic children's difficulty with mental disengagement from an object: Its implications for theories of autism. *Developmental Psychology*.

Kanner, L. (1943). Autistic disturbance of affective contact. *Nervous Child, 2*, 217–250. Reprinted in L. Kanner (1973). *Childhood psychosis: Initial studies and new insights*. New York: Wiley.

Leekam, S., & Perner, J. (1991). Does the autistic child have a metarepresentational deficit? *Cognition, 40*, 203–218.

Leslie, A. M., (1987). Pretence and representation: The origins of "theory of mind". *Psychological Review, 94*, 412–426.

Leslie, A. M., & Frith, U. (1988) Autistic children's understanding of seeing, knowing, and believing. *British Journal of Developmental Psychology, 6*, 315–324.

Leslie, A. M., & Roth, D. (1993). What autism teaches us about metarepresentation. In S. Baron-Cohen, H. Tager-Flusberg, & D. J. Cohen (Eds) *Understanding other minds: Perspectives from autism*. Oxford: Oxford University Press.

Ozonoff, S., Pennington, B., & Rogers, S. (1991). Executive function deficits in high-functioning autistic children: Relationship to theory of mind. *Journal of Child Psychology and Psychiatry, 32*, 1081–1106.

Phillips, W. (1992). *Comprehension of desires and intentions by children with autism*. Unpublished PhD thesis, Institute of Psychiatry, University of London.

Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a "theory of mind"? *Behavioral and Brain Sciences, 4*, 515–526.

Rutter, M. (1985). Infantile autism and other pervasive developmental disorders. In M. Rutter & L. Hersov (Eds) *Child and adolescent psychiatry*. Oxford: Basil Blackwell.

Shah, A., & Frith, U. (1983). An islet of ability in autism: A research note. *Journal of Child Psychology and Psychiatry, 24*, 613–620.

Sigman, M., Mundy, P., Ungerer, J., & Sherman, T. (1986). Social interactions of autistic, mentally retarded, and normal children and their caregivers. *Journal of Child Psychology and Psychiatry, 27*, 647–656.

Sperber, D., & Wilson, D. (1986). *Relevance: Communication and cognition*. Oxford: Basil Blackwell.

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*, 1–22.

Tinbergen, N., & Tinbergen, E. (1983). *Autistic children: New hope for a cure*. Oxford: Pergamon.

Wing, L. (1969). The handicaps of autistic children: A comparative study. *Journal of Child Psychology and Psychiatry, 10*, 1–40.