REPRESENTATION OF MENTAL STATES
IN SCHIZOPHRENIA

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Submitted for the degree of
Doctor of Philosophy

September 1997
ACKNOWLEDGEMENTS

I am very grateful to Professor Chris Frith for his help and encouragement during the supervision of the work in this thesis. I would also like to thank Dr. Rhiannon Corcoran for the many useful discussions that we have had over the past four years.

I am indebted to a number of professionals within the Health Service, who helped to make this research possible. Firstly, I would like to thank Dr. Nicola Gray for showing great interest in the research, and for introducing me to the staff and patients at Horton Hospital. I also much appreciate her friendship and support during the final year of this work. I am grateful to the following psychiatrists for allowing me access to their patients: Professor Ann Mortimer at St. Bernard’s Hospital, Ealing; Dr. Pedro Mascuñan, Dr. Norman Harvey and Dr. Dora Kohen at the Whittington Hospital, Archway; Dr. John Cookson at St. Clement’s Hospital, Mile End; and Dr. David James and Dr. David Somekh at Horton Hospital, Epsom. Jeff Begum, the CPN associated with St. Clement’s, was particularly helpful in giving me access to all of his outpatients at health centres on the Isle of Dogs. My thanks also go to all the patients who agreed to take part in the research.

Many friends have helped by just being around, and providing me with a welcome respite from the hospital ward or computer screen. These include Emma Veitch, Dr. Carla Croft, Dr. Rabia Malik, Maria Woloshynowycz, Jim McGinlay, Robert Beattie, David Free, Brent Holder, Emily Hatchwell, Helen Rodriguez, Margaret Lewis, Penny Jennings, Andrew Brown, Anna Marx and Pauline, Nick Tagg and Mike, Graham Hemingway, Dr. Bryan Eaton, Richard Wells, Colin Davies, and Serena Surman. I would like to thank all the volunteers who acted as control subjects, and Jo Palmer for helping me to arrange the appointments. Most of all, I would like to thank my parents for their constant support and encouragement, and Bill Majrowski, whose presence made the work so much easier.
ABSTRACT

From a literature review, it was concluded that schizophrenia primarily involves deficits in conscious, controlled processing. This was shown to be compatible with Frith’s (1992) neuropsychological model of impaired metarepresentation in the disorder. There is strong evidence that patients with paranoid symptoms or behavioural signs have deficits in the representation of others’ mental states (‘theory of mind’; ToM), but two recent studies have produced conflicting results. Those findings were reconciled in the first study of the thesis, which showed that, on false belief tasks, patients have intact first-order ToM, but specific impairments at the second-order level. This was later confirmed using a ‘hints’ test of ToM. The results were contrasted with the case of autism. On a spatial reversal test of executive function, schizophrenics with behavioural signs made more perseverative errors than controls. No correlations appeared between ToM and executive function for any of the schizophrenic symptom groups. This was contrasted with the case of autism, and it was suggested that schizophrenia involves late-occurring, independent deficits in separate metarepresentational domains.

It was argued that Frith’s model of schizophrenia can be extended to include impaired representation of own knowledge, explaining the deficient use of context in the disorder. Some evidence was obtained that patients with primarily behavioural signs are impaired at naming objects in a picture context; this ability was unrelated to ToM, consistent with independent deficits in separate metarepresentational domains. It was suggested that ‘weak central coherence’ in autism may also reflect impaired representation of own knowledge, and some evidence was obtained that (like autistics), symptomatic schizophrenics show facilitation on embedded figures tests. Schizophrenic patients performed the same as controls, however, on an illusions task. For patients with behavioural signs, embedded figures accuracy was inversely related to the ability to name objects in a picture context, and it was argued that this supported task analyses suggesting a common cognitive process.
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CHAPTER ONE

INTRODUCTION

This thesis explores the neuropsychology of schizophrenia, especially Frith’s (1992; 1994; Frith & Frith, 1991) model of the disorder. In this opening chapter, the nature of schizophrenia is discussed, and key methodological issues are outlined.

1.1 The nature of schizophrenia

Signs and symptoms of the disorder

Schizophrenia is one of the most prevalent psychiatric disorders, occurring in about 0.85% of the population of all countries worldwide (Straube & Oades, 1992). Both sexes are equally susceptible, with the age of onset usually falling between 20 and 35 years (see McKenna, 1994). The disorder is characterised by a wide range of abnormal experiences (symptoms) and behaviours (signs), of which individual patients show only a subset. Common psychotic symptoms include hallucinations (especially in the auditory modality, where the patient hears ‘voices’ talking to or about him) and paranoid, grandiose or bizarre delusions. Patients may also experience so-called ‘passivity phenomena’; these include delusions of control (a belief that some external force is controlling the will), and abnormalities of thought possession. The latter include ‘thought insertion’ (a feeling that thoughts which are
not one’s own are being put into one’s head from outside), ‘thought withdrawal’ (one’s own thoughts are withdrawn by a force), and ‘thought broadcast’ (one’s thoughts are available to others) (see Straube & Oades, 1992). Research suggests that about 50% of schizophrenic patients experience auditory hallucinations at some stage of their illness, whereas more than 90% have delusions of one kind or another at some point (Cutting, 1995). In general, hallucinations and delusions are more prevalent in the early, acute stages of a schizophrenic illness (McKenna, 1994).

Common signs can be observed in the domains of language, motor behaviour and affective behaviour. For example, a patient might show ‘formal thought disorder’ (FTD), which manifests as a disturbance in the form (i.e. structure, organisation and coherence) of speech such that it becomes very difficult for the listener to follow what is being said (McKenna, 1994). Common features of FTD include ‘incoherence of speech’ (where the subject may fail to use logical connections between parts of a sentence), and ‘poverty of content’ (where the patient talks vaguely and rambles without coming to a point). Some schizophrenic patients show ‘poverty of speech’, saying very little and restricting their answers to a few words. In the domain of motor behaviour, as with speech, patients may be either inappropriate (being, for example, unduly familiar), or show poverty of behaviour (‘avolition’). Some of the key motor and volitional disorders are covered by the term ‘catatonia’; these include simple movement disorders (e.g. stereotypies, mannerisms and posturing), and complex behavioural disorders such as negativism, where the patient does the reverse of what is asked of him. Catatonic features can appear in the language domain as, for example, stereotyped speech or mutism (McKenna, 1994). Finally, patients with schizophrenia often show emotional disorders; the concepts of inappropriateness and poverty equate here with incongruous affect (i.e. not appropriate for the current situation) and blunt affect (i.e. expressionless face and voice) respectively. Patients sometimes report a loss of the normal ability to enjoy food, recreation etc. (‘anhedonia’). All of the signs detailed above (especially those such as poverty of
speech, avolition and affective blunting, which involve an absence of normal behaviour), tend to be more prevalent in the later, chronic stages of a schizophrenic illness (McKenna, 1994). A more detailed description of all the symptoms and signs can be found in the Glossary accompanying the manual for the Present State Examination Version 9 (PSE; Wing et al., 1974).

**Early attempts at defining schizophrenia**

Although it is clear that presence of the above abnormal experiences and behaviours suggests a psychotic illness, the precise definition of schizophrenia for diagnostic purposes has been much argued over. Modern ideas about the disorder are based on the work of Kraepelin and Bleuler. Kraepelin (1896) grouped all the patients under his care according to clinical presentation and illness course, and concluded that the various forms of ‘insanity’ (namely ‘catatonia’, ‘hebephrenia’ and ‘dementia paranoides’) all tended to show an early adult onset and deteriorating course. Whilst emphasising the diversity of signs and symptoms in these patients, he felt that the early onset and poor outcome were defining features of an illness which he called ‘dementia praecox’. He made clear from the start that he viewed dementia praecox as a brain disease involving lesions of the cerebral cortex (U. Peters, 1991). Bleuler’s (1911) approach was to concentrate on the signs and symptoms of patients diagnosed with dementia praecox, in an attempt to identify features specific to that group. He concluded that all patients showed a disturbance of thought processes involving ‘associative splitting’, whereby trains of thought disintegrated (C. Peters, 1991). This splitting was considered the primary defect amongst all subtypes of dementia praecox, so Bleuler renamed the illness ‘schizophrenia’ (literally ‘splitting of thought’). According to Bleuler’s model, the splitting gave rise to ‘fundamental’ symptoms (e.g. avolition, affective blunting and autism), which were considered a specific and permanent feature of the illness. Patients also showed ‘accessory’ symptoms (e.g. delusions and hallucinations), which occurred intermittently during
the illness course. These were known to occur in other disorders such as manic-depressive psychosis so were thought to be a secondary psychological reaction, and not caused directly by the biological process underlying schizophrenia (see Andreasen & Carpenter, 1993, for a full discussion).

The work of Kraepelin and Bleuler can be criticised on several fronts. For example, Boyle (1990) has pointed out that Kraepelin’s criteria of similar age of onset and similar outcome were insufficient to demonstrate a common pattern of illness amongst his patients, and were therefore inadequate as a basis for the hypothetical construct of dementia praecox. Bleuler, too, failed to show that his construct of schizophrenia was derived from an observed pattern involving co-occurrence of particular symptoms and signs. Instead, he accepted Kraepelin’s classification, and then searched for specific symptoms within the group of patients diagnosed with dementia praecox. However, despite these and other more general methodological flaws such as observer and sampling bias (see Shepherd, 1995), the work of Kraepelin and Bleuler is still noteworthy as the foundation of more recent attempts to produce a working definition of schizophrenia.

More recent definitions

Although Bleuler’s fundamental symptoms were used widely for diagnostic purposes, psychiatrists began to feel that they were too subjective and broad to be diagnostically useful. Schneider (1959) produced a list of reliably recordable ‘first rank’ symptoms which he felt to be specific to schizophrenia and therefore of special diagnostic importance. These included auditory hallucinations in the third person (i.e. voices talking about the patient or making a running commentary on the patient’s behaviour), delusions of control, and abnormalities of thought possession (thought insertion, withdrawal and broadcast). Schneider’s formulation was atheoretical in that he simply listed symptoms which he felt, from clinical experience, to be patho-
gnomonic of schizophrenia. As Boyle (1990) has pointed out, he was therefore as guilty as Kraepelin and Bleuler of failing to demonstrate a specific pattern of co-occurrence of symptoms and signs, from which one could infer the syndrome of schizophrenia. Indeed, subsequent research (discussed by McKenna, 1994) has not supported Schneider’s scheme, in that a minority of patients diagnosed with major affective disorders, such as manic-depressive psychosis, show first rank symptoms at some point during their illness.

Nevertheless, Schneider’s emphasis on the experiential abnormalities of psychosis, and on a cross-sectional rather than longitudinal evaluation, was very influential in moulding later diagnostic schemes. For example, the PSE of Wing et al. (1974), and its associated diagnostic computer program (CATEGO), considers the clinical picture over the past month only, and places great weight upon first rank symptoms. Since the early 1970s, the emphasis has been on producing descriptive criteria which, although essentially atheoretical, define schizophrenia rigorously, and lead to greater consensus in diagnosis. Such schemes include the Research Diagnostic Criteria (RDC; Spitzer et al., 1975), the Washington University Criteria (Feighner et al., 1972) and (most widely used of all), the Diagnostic and Statistical Manual (DSM) of the American Psychiatric Association (APA). The latter has gone through various stages of development and is currently at DSM-IV (APA, 1994). An abbreviated description of the DSM-IV criteria for schizophrenia is given below:

A. Characteristic symptoms and signs: At least two of the following, each present for a significant portion of time during a 1-month period (or less if successfully treated):
   (1) delusions
   (2) hallucinations
   (3) disorganised speech (e.g. incoherence)
   (4) grossly disorganized or catatonic behaviour
(5) affective flattening, avolition, poverty of speech

Only one A symptom is required if delusions are bizarre, or if hallucinations involve a running commentary on the person’s behaviour or thoughts, or two or more voices conversing with each other.

**B. Social / occupational dysfunction:** Since the onset of the disturbance, one or more areas of functioning such as work, interpersonal relations or self-care is markedly below the level achieved prior to onset.

**C. Duration:** Continuous signs of the disturbance persist for at least 6 months. This period must include at least 1 month of symptoms that meet criterion A, and may include prodromal and / or residual periods when the A criterion is not fully met.

**D. Affective components:** A major mood disorder (which would render a diagnosis of schizoaffective disorder or mood disorder with psychotic features more appropriate), has been ruled out.

**E. Substance / secondary exclusion:** The disturbance is not due to a substance-induced or secondary psychotic disorder.

1.2 Heterogeneity of the schizophrenia construct

**Validity of ‘schizophrenia’ as a diagnostic label**

Although a high degree of reliability could conceivably be achieved in diagnosis if all psychiatrists used the same operational criteria, the atheoretical nature
of these criteria means that the validity of the schizophrenia construct is open to question. Bentall (1990) examined both the predictive validity and construct validity of ‘schizophrenia’, and concluded that both are low. Predictive validity concerns the confidence with which one can predict variables such as outcome and treatment response on the basis of diagnosis. As Bentall pointed out, several studies have shown that outcome for patients with a diagnosis of schizophrenia can vary from complete recovery (about 33%), through partial recovery with occasional relapse (~33%) to deterioration (~33%). Similarly, it is not possible to accurately predict the response of patients to the various neuroleptic medications on the basis of diagnosis alone. Construct validity would be high if a particular set of symptoms and signs (a syndrome) tended to co-occur in patients. However, as already discussed, an individual schizophrenic subject typically shows several symptoms and signs taken from a very wide and heterogeneous possible set, and there is great variability in clinical presentation across subjects. Many studies have used correlational analyses of symptom scores to identify groups of symptoms that tend to co-occur in patients, and these studies have suggested that several different syndromes may in fact fall under the umbrella term ‘schizophrenia’. Each of these syndromes may have a separate pathophysiology, and at a psychological level of explanation, there may be several different psychopathologies, each underlying a particular symptom or syndrome (Carpenter et al., 1993). Until quite recently, many workers ignored this heterogeneity, comparing a ‘schizophrenic’ group with a control group on a particular dependent variable. In practice, this would have meant that patients showed widely different clinical presentations across studies, so it is perhaps not surprising that many of the findings in the schizophrenia literature have been difficult to replicate. Thus, whilst the existence of the various psychotic symptoms and signs is beyond doubt, examination of the validity of the schizophrenia construct suggests that future research should concentrate on sub-divisions of psychotic phenomena rather than on the psychiatric diagnosis per se (Frith, 1992; Persons, 1986).
Sub-syndromes of schizophrenia

A highly influential division of schizophrenic symptomatology has been the two-syndrome model of Crow (1980). He proposed that the symptoms and signs reflect two independent dimensions of psychopathology: type I (‘positive symptoms’), consisting of delusions, hallucinations and formal thought disorder, and type II (‘negative symptoms’), comprising features such as affective blunting and poverty of speech. Positive symptoms were said to be abnormal by their presence, relatively transient in nature, and more prevalent in the acute stages of illness. As they seemed to respond well to dopamine-blocking neuroleptic medication, Crow (1980) suggested that they may result from an excess of dopamine D\textsubscript{2} receptors. Negative symptoms (i.e. negative signs in the terminology adopted for this thesis), were said to reflect the absence or diminution of normal behaviour. They were found more commonly in chronic patients, and were more persistent than positive symptoms. They showed little response to typical neuroleptics, so Crow (1980) proposed that a structural cerebral abnormality underlay the type II syndrome. Andreasen (1982) extended the definition of the negative syndrome to cover avolition, anhedonia and attentional impairment as well as blunted affect and poverty of speech.

Much research effort has been put into exploring the validity of Crow’s classification. However, studies investigating the relationship between symptoms and brain abnormalities have tended to produce equivocal results. Lewis (1990) reviewed 18 studies from the literature, and found that only 5 of these showed a significant relationship between the presence of negative signs and enlarged cerebral ventricles. What has become apparent is that negative signs are associated with cognitive impairments such as low pre-morbid IQ and an IQ decline from pre-morbid levels (e.g. Frith et al., 1991c). Results are also equivocal regarding the suggested relationship between positive symptoms and elevated numbers of D\textsubscript{2} receptors. It is well
accepted that typical neuroleptics are effective because of their ability to block D₂ receptors, but it is not established that a primary disturbance of dopamine function is responsible for positive symptoms (Crow, 1994).

Correlational analyses have also been used to explore the validity of the type I/type II classification. Many studies have found no correlation between scores for positive and negative features, supporting Crow’s model. In addition, it is commonly found that scores for negative signs intercorrelate, as do scores for delusions and hallucinations. However, scores on measures of formal thought disorder (a positive sign) tend not to correlate with those for delusions and hallucinations (see McKenna, 1994, for a review of these studies). In line with this, a large number of factor analytic studies (which look for groups of signs and symptoms co-existing in patients), have found three factors (e.g. Bilder et al., 1985; Gureje et al., 1995; Liddle, 1987). Liddle named these as follows:

(a) psychomotor poverty (poverty of speech, blunt affect and decreased spontaneous movement);
(b) reality distortion (delusions and hallucinations);
(c) disorganisation (formal thought disorder and inappropriate affect or behaviour).

Thus, psychomotor poverty corresponds to Crow’s (1980) original definition of the negative syndrome, whereas reality distortion and disorganisation correspond to his definition of the positive syndrome. Several studies (e.g. Lindenmayer et al., 1994) have shown that if additional measures of psychopathology are entered into the factor analysis, two further factors emerge corresponding to depression and psychomotor excitation (i.e. hostility, excitement etc.). As these two dimensions are certainly not specific to schizophrenia, we can consider Liddle’s three syndromes as being the major clusters that derive from factor analysis of schizophrenic signs and symptoms.
Converging evidence from functional imaging and neuropsychology has provided support for the three-factor model. For example, in a study using Positron Emission Tomography (PET), Liddle et al. (1992) found that the three syndromes corresponded to distinct patterns of abnormal cerebral blood flow in the resting state (see Section 2.3 of this thesis). Liddle & Morris (1991) also found that each of the syndromes was associated with a distinct pattern of neuropsychological impairment (see Section 2.3). It is important to bear in mind that, in any given patient, the three factors can co-occur and overlap, so that each factor does not constitute a 'subtype of schizophrenia' (Liddle et al., 1992). Instead, a patient with high levels of symptoms from all three dimensions would be considered as having the most severe psychopathology. Moreover, when developing neuropsychological models of psychotic features, we should not automatically assume that all features within a given factor analytic cluster can be explained by the same impairment. Thus, although all delusions load onto one factor, it is still possible that different types of delusion may reflect distinct neuropsychological deficits (e.g. Frith, 1992).

1.3 Methodological issues in schizophrenia research

Selection of schizophrenic subjects

As a first step, patients should only be selected if they meet particular diagnostic criteria for schizophrenia (e.g. the DSM-IV criteria are used in this thesis). However, as already discussed, an additional feature of good research is the selection of groups of patients who are homogeneous along the dimension of interest. Usually this means choosing subjects who show particular symptoms and signs, or groups of symptoms and signs ('the symptom approach') (Frith, 1992; Persons, 1986). As an illustration, we might predict that only those subjects with hallucinations will show an effect on a given dependent variable. Comparison of a group of hallucinating patients with a control group of non-hallucinators would then reveal any effect,
whereas the effect may be lost within group means if a heterogeneous group of schizophrenics (only some of whom were hallucinating) was compared with controls. As Persons (1986) pointed out, the symptom approach facilitates theory development, since it is easier, and more meaningful, to propose a psychological model for just one symptom (or cluster) than for ‘schizophrenia’ itself. The importance of theory-driven research was also stressed by Carpenter et al. (1993); they pointed out that, although it is easy to show that a particular group of patients is impaired on a particular neuropsychological task, we are only able to develop cognitive models if we have an a priori hypothesis predicting an association between task performance and particular symptoms or signs.

Selection of control groups

One of the most useful control methodologies springs directly from the symptom approach, and involves contrasting schizophrenic patients with a particular symptom or sign with schizophrenics without that feature (e.g. Andreasen & Carpenter, 1993). This is particularly powerful in that both experimental and control groups can be matched on all possible confounding variables, including illness duration and dosage of neuroleptic and anticholinergic medication. There is some evidence that anticholinergic drugs such as procyclidine (given to treat neuroleptic-induced extrapyramidal symptoms) can impair memory in patients with schizophrenia (e.g. Fayen et al., 1988). Similarly, neuroleptics with anticholinergic properties can also impair memory (Perlick et al., 1986). Thus, if we can match subject groups on drug dosage, any differential task performance across groups can be attributed to factors other than medication effects.

Chapman & Chapman (1977) stressed the importance of also using a psychiatric control group (usually patients with unipolar depression), to control for factors such as hospitalization, the stigma of a psychiatric label, and the very presence of any
kind of psychiatric illness. However, it is important that this group is relatively homogeneous in terms of diagnosis, otherwise it might include some patients who show a given cognitive impairment, and others who do not (Chapman & Chapman, 1977). For example, if schizophrenics with hallucinations were compared with depressed patients on a cognitive task, it would be important to ensure that all controls met diagnostic criteria for unipolar depression, and that none had depression with psychotic features. Subjects with the latter diagnosis may themselves be hallucinating, so could conceivably show the same task effect as the schizophrenics. Such a scenario might result in a non-significant difference in mean scores between control and experimental groups, when a significant difference may have appeared with a more homogeneous control group. Chapman and Chapman also recommended the use of a control group of normal subjects in addition to the clinical controls. This is essential in order to give a baseline level of performance, so that any common deficits among the patient groups can be detected. The studies detailed in this thesis employ these various methodologies, using discrete symptom subgroups of schizophrenic patients and separate control groups of normals and patients with a non-psychotic psychiatric illness.

Exclusion criteria and group matching

Subjects are usually excluded from experimental or control groups if they have any known neurological illness or lesion. This includes patients who have undergone frontal leucotomy, which, until the 1970s, was a common operative procedure applied in schizophrenia. Exclusion of such subjects helps to maintain group homogeneity, and removes any possible confounding effects of the lesion on task performance. For similar reasons, subjects with a history of drug or alcohol abuse should also be excluded as several studies suggest a possible link between substance abuse and neurocognitive impairment (e.g. Brandt et al., 1983; Grant et al., 1978). In addition, subjects are not tested if they have a poor understanding of English or if
they are older than 65, as elderly subjects may present with dementia.

Whenever possible, groups should be matched closely on demographic variables such as age, sex, years of education, handedness, ethnicity and employment history, as studies have shown that these may be related to neuropsychological task performance (e.g. Heaton et al., 1986). Chapman & Chapman (1977) stressed the importance of matching very closely on educational level (i.e. within 0.2 years), because even if groups are non-significantly different on statistical tests, an extra year in one group could mean the difference between completing A-levels or not doing so; this is a potentially important intellectual difference. As already discussed, medication can be matched if schizophrenics without a target symptom are used as controls for those with the symptom. However, the issue of medication is much harder to resolve in the case of normal controls (who typically will be taking no drugs), and controls with other psychiatric diagnoses, who, although medicated, will not usually be taking neuroleptics. In practice, drug effects are usually examined post hoc, in terms of any correlation between schizophrenic performance and dose level. If necessary, dosage (usually the daily mg equivalent of 100 mg of chlorpromazine) can be entered into an analysis of co-variance to examine whether any significant group differences remain once medication has been controlled.

Matching groups on IQ is particularly important because schizophrenic subjects (especially older, chronic patients) often score poorly on IQ tests, showing a decline from pre-morbid levels (e.g. Frith et al., 1991c). It is thus important to show that any performance deficit on neuropsychological tasks is not just due to low general intelligence. The matching can be difficult in practice, as it is often hard to find controls (especially normals) whose current IQ levels fall within the schizophrenic range. Many studies in the literature have side-stepped this problem by matching patients and controls on pre-morbid IQ, measured using the National Adult Reading Test (NART; Nelson, 1982). This test requires subjects to correctly
pronounce 50 short, irregular words (e.g. ‘ache’, ‘prelate’), and it has been argued that the irregular nature of the words means that test performance depends more upon previously acquired knowledge than on current ability to work out a pronunciation (Nelson & O’Connell, 1978).

There are two major problems with the use of pre-morbid IQ as the matching variable in schizophrenia research. The first is that, as already mentioned, schizophrenics may have a current IQ that is considerably lower than the NART measure (Frith et al., 1991c). Normals, on the other hand, will have similar current IQ and NART scores. As performance on most neuropsychological tasks is likely to depend, to some extent, on current IQ, the matching of groups on NART scores leaves open the possibility that any deficit in the schizophrenic group is due to their lower current IQ compared to controls. The second problem regards the validity of the NART itself in schizophrenia research. Crawford et al. (1992) measured the NART IQ of two groups of schizophrenics - one from long-stay psychiatric wards, and the other from acute wards and out-patient clinics. All subjects in the latter group were normally resident in the community. Independent estimates of the pre-morbid IQ of each of the patients were obtained by giving the NART to normal controls individually matched with each patient on age, sex and years of education. Results of the study showed that, whilst the NART IQs of the community-resident schizophrenics were very similar to those of their matched controls, the NART scores of the long-stay schizophrenics were significantly lower than those of their controls. This latter result could simply reflect a lower than normal pre-morbid IQ in the long-stay patients, but it is also possible that the schizophrenic illness in that group had adversely affected their NART performance. Crawford et al. (1992) therefore suggested that the NART may not be a valid measure of pre-morbid intelligence in such patients, so can only be used with confidence in acutely ill subjects.

These arguments suggest that it is best to match schizophrenic and control
groups on current IQ (often measured by the Wechsler Adult Intelligence Scales - Revised (WAIS-R), Wechsler, 1981). As the WAIS-R takes approximately one hour to administer, many studies (including those detailed in this thesis) use a shorter estimate of current IQ such as the Quick Test (Ammons & Ammons, 1962). In this, subjects are shown four pictures and have to match each of fifty words to the most appropriate picture. The test is thought to rely heavily upon abstract reasoning rather than previously acquired knowledge (Ammons & Ammons, 1962). Since it is often difficult in practice to find normal controls who match the schizophrenic patients on current IQ measures, a common solution involves use of post hoc analysis of covariance to partial out any effects of higher current IQ in the control groups. This may be unreliable if the absolute IQ levels of groups differ greatly, so an alternative is to select a subgroup of controls which is matched with the schizophrenic group. In this case, it is obviously important to ensure that the number of subjects within the subgroup is large enough to give sufficient power for any statistical tests. Finally, when matching groups on intelligence, it is important to bear in mind that any subject may have a markedly different IQ score in one domain (e.g. verbal IQ) compared to another (e.g. performance IQ) (Wechsler, 1981). Because of this, the IQ test chosen for group matching should assess functioning in the domain required for the experimental tasks. Thus, if a major prerequisite of task understanding is the ability to read words and sentences, subjects should be matched on current verbal IQ (as measured by the Quick Test, for example) rather than on performance IQ.

In general, it should be realised that matching of groups on all variables is rarely possible. The primary aim of the experimenter is to find enough subjects for each group so that empirical data can be analysed meaningfully using standard statistical tests. As most studies in the field of schizophrenia use 15 or more subjects in each group, this invariably means making some compromises with matching so that research can be completed within a reasonable time. Experiments in which comparisons are made between groups of schizophrenics with specific symptoms or
signs may be especially time consuming, as several hospital sources may be needed to provide enough patients with the appropriate clinical presentation. However, as discussed above, it is believed that this extra investment of time pays dividends in terms of theory development (Frith, 1992; Persons, 1986).

Aspects of experimental design

Once experimental and control groups have been chosen and matched, the experimenter can begin administering any neuropsychological tasks. During testing, attempts should be made to ensure that all subjects are equally attentive and cooperative. This can be difficult, as schizophrenic patients often show poor attention (e.g. McGhie & Chapman, 1961), and are sometimes unwilling to perform tasks. One way of controlling for this is to give a clinical rating of attention, then examine post hoc whether this correlates with test scores. Alternatively, a number of control questions can be included at various stages of the test battery. The experimenter can then analyse scores on the measure of interest for only those subjects who pass all of these questions, enabling him to exclude subjects whose attention, memory or cooperation may have lapsed. This latter approach is used in some of the studies detailed in this thesis. Baron & Treiman (1980) stressed that groups should be equally familiar with the experimental tasks. For example, many findings of schizophrenic impairment relative to controls may simply reflect the fact that the patients often take longer than controls to learn to perform optimally on a given task. Such effects can be minimised by giving all subjects appropriate pre-training. The use of ecologically valid scenarios also aids familiarity. For example, the ‘theory of mind’ stories given to patients in the first study of this thesis (see Chapter 4), were all set in the highly familiar surroundings of a hospital ward in order to maximise their ecological validity.

Chapman & Chapman (1973) pointed out that schizophrenics generally
perform poorly on most cognitive tasks, so that if a specific (and theoretically meaningful) impairment is to be found in the schizophrenic group, it must be shown as a 'differential deficit'. This is a greater difference between schizophrenic and control groups in performance on an experimental task than on a control task. The two tasks are designed to be identical except for one feature, which is the psychological function of interest. An important requirement of such designs is that the two tasks are matched on discriminating power, so that any differential deficit cannot be ascribed to differences in, say, task difficulty. For example, a task (A) which 50% of normal subjects get right, has more discriminating power than an easier task (B) which 90% of normals pass. If these tasks were given to schizophrenic and control groups, any differential deficit in performance could simply be due to the greater difficulty of task A. Assuming that A and B measured different psychological skills, we might then conclude erroneously that schizophrenic patients were more impaired in the ability measured by task A than in that measured by B. The first study of this thesis (Chapter 4) uses a matched-tasks paradigm to explore whether certain schizophrenic symptoms and signs reflect a specific deficit in theory of mind ability.

Chapman & Chapman (1973) suggested that tasks should be matched on discriminating power using normal subjects with a wide range of ability. They argued that matching is successful when the task reliabilities, the means, variances and shapes of the distributions of item difficulty, and the means, variances and shapes of the distributions of scores, are equated. When the tasks are then administered in the experiment proper, they should remain matched for any normal control group, so that a differential deficit in the performance of a schizophrenic group can be attributed to a specific impairment rather than to task artefacts. Examination of the schizophrenia literature reveals many studies in which such differential designs have not been used, and where, as a result, the findings are difficult to interpret. A popular methodology has involved administration of a large battery of standard neuropsychological tasks to groups of schizophrenics and controls. As these tasks are not

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matched on discriminating power, a particular task deficit in the patients relative to controls should not be used to argue for a specific neuropsychological impairment. For example, Blanchard & Neale (1994) attempted to explore the hypothesis of lateraled neuropsychological impairment in schizophrenia by giving tests sensitive to left and right hemisphere function to groups of patients and normal controls. They found that the patients were significantly impaired relative to controls on both left and right hemisphere tests, and within the patient group, similar numbers of left and right hemisphere test scores met clinical criteria for impairment. However, as the authors themselves admitted, these data could reflect either a generalized or differential deficit in hemispheric function compared to controls. For example, the right hemisphere tests may have been more difficult than the left hemisphere tests, so the observed generalized performance deficit may have actually reflected a true greater schizophrenic impairment in left hemisphere function. The various possibilities could only have been teased apart by the use of psychometrically matched tasks.

Unfortunately, such close matching of tasks is very difficult to achieve in practice, and even when it is possible, further problems can be introduced. For example, Raulin & Chapman (1976) investigated the ability of schizophrenic and control subjects to recall lists of words with high or low contextual constraint. To equate task difficulty, the low constraint lists were made shorter, but this then meant that any differential deficit in performance could have been an effect of contextual constraint or list length. To resolve such ambiguities, extra versions of tasks are needed to examine the independent effects of each variable; this clearly makes an experiment more time consuming. Another problem with differential deficit designs is that if the schizophrenic group performs worse than the control group on both tasks, it is always possible to devise a scale which equalises the difference between the groups on each task, thereby removing any differential. The best way to overcome this is to equate the groups on performance on one of the two tasks, but as Baron &
Treiman (1980) pointed out, this is often difficult to achieve and may lead to the selection of atypical subjects.

Watts (1989) suggested an alternative methodology, which is to use unmatched tasks, followed by *post hoc* analyses in an attempt to draw conclusions from a significant task by group interaction. For example, a log transformation can be applied to data to remove inter-group differences in variance of scores. Confidence in an interaction is increased if it survives this transformation, although it is still possible that it reflects differences in discriminating power between tasks. Another *post hoc* method is to divide normal controls into high- and low-IQ subgroups, and see whether a differential deficit occurs between these subgroups on the two tasks. If it does not, then any differential deficit with schizophrenics and controls is more likely due to the nature of schizophrenia than to task differences. Finally, in experiments where the clinical group performs worse than controls on both tasks, the data can be manipulated in an attempt to produce a cross-over effect (with schizophrenics performing better than controls on task A, but worse on task B). This is done by analysing only the data from high-scoring schizophrenic subjects and low-scoring controls. If a cross-over is produced, it is unlikely to be an artefact of psychometric task differences (Watts, 1989).

**Alternative designs**

The problem of general intellectual impairment in schizophrenia, and the consequent need to show a differential deficit, can be avoided if a task is found on which schizophrenic patients reliably perform *better* than controls. In practice, this may mean that a cross-over between group performance on two tasks is found directly, without any need for *post hoc* manipulation of data. Beech *et al.* (1989) found such an effect when they tested acute schizophrenic patients on the ‘negative priming’ paradigm (Tipper, 1985; see Section 2.2 of this thesis). In this task, a
A distractor from a priming display becomes a target on an immediately following display. Beech et al. found that normals showed a longer reaction time (negative priming) to this target than in control trials where the target had not previously been a distractor. Schizophrenic subjects, however, were slower than normals in the control condition, but faster in the priming trials. This faster responding cannot be attributed to general intellectual or motor deficits, so was interpreted by Beech et al. (1989) as having theoretical importance. A similar methodology is adopted in Chapter 7 of this thesis, where schizophrenic patients are predicted to perform better than controls on two neuropsychological tasks.

Problems of general cognitive impairment in schizophrenia can also be avoided by studying analogues of psychotic symptoms in the normal population (e.g. Persons, 1986). However, great care is needed here as it may often be misleading to assume that a clinical symptom (e.g. social withdrawal by a paranoid patient) is simply an extension of a ‘normal’ phenomenon (e.g. social avoidance by a shy person) (Watts, 1989). The most useful symptoms for research are those which are qualitatively similar to psychotic symptoms, but yet occur in some normals. For example, Bentall & Slade (1985) were able to test a cognitive theory of hallucinations by comparing the task performance of groups of normals with high or low scores on a scale of hallucination proneness.

Group studies have been considered in detail in this chapter, but it is worth pointing out that single case methodology can also make a valuable contribution to schizophrenia research (e.g. Shallice et al., 1991). Case studies are useful for localising brain abnormalities in schizophrenia, as they typically involve the administration of many different neuropsychological tests to one patient. In theory, this enables us to show impaired performance on several tests thought to be subserved by one brain system, but intact performance on tasks involving a different region. Problems of task difficulty are still relevant, so it is important to ensure, where
possible, that the tasks used are equally discriminating. The administration of many
different tasks addresses the general problem that many tests of ‘higher’ cognitive
processes rely upon several different psychological functions for correct execution
(Keefe, 1995). For example, normal performance on the Wisconsin Card Sorting Test
of ‘executive function’ (see Section 2.3 of this thesis) requires, as well as executive
control, intact auditory and visual attention, motor skills, memory and abstraction.
It is possible that a subject with deficits in one or more of these non-executive
domains may score in the impaired range on the test and be wrongly classified as
having executive dysfunction. For us to conclude that executive function is impaired,
we need to administer several different executive tasks, and also test the component
psychological skills (using equally difficult tests of memory, attention etc.), in order
to rule out any deficits in these domains (Keefe, 1995). This technique is theoret­
ically possible with single case designs, whereas it is usually too time consuming
to be employed with large group studies (where it is more appropriate to look for
differential deficits on only a few tasks). Shallice et al. (1991) suggested that a
powerful methodology involves examination of several cases, and comparison of
these with data from group studies, in an attempt to find a common interpretation.

1.4 Conclusion

We have seen that a symptom-specific theory-driven approach is particularly
suitable in schizophrenia research, where patients selected according to established
diagnostic criteria present with heterogeneous signs and symptoms. Control groups
should be chosen carefully, and matched as closely as possible with patients on
demographic variables. The general intellectual impairment often found in schizo­
phrenia necessitates close matching of groups on current IQ, and the use of differ­
ential deficit designs where possible. However, such designs may introduce further
problems, and there is to date no strong consensus on the best way of overcoming
these. It seems that advances will be most likely made by using a method of ‘conv-
erging operations’, i.e. the drawing of common conclusions from as many different experimental approaches as possible. In the next chapter, we shall examine the progress that has actually been made in developing particular psychological and neuropsychological models of psychotic phenomena.
2.1 Introduction

As discussed by Frith (1992), the discovery of the therapeutic effects of some D₂ receptor blocking drugs, and the advent of sophisticated brain imaging techniques, have left no doubt that some form of brain disorder underlies the signs and symptoms of psychosis. However, we can attempt to understand signs and symptoms at various levels of dysfunction, such as the psychological (e.g. in terms of abnormal cognitive processes) or the physical (e.g. in terms of physiological processes). Frith stressed the importance of searching for complete explanations of phenomena at different levels, whilst looking for links between these levels, so that “what we know about physiology will influence our explanation at the psychological level and vice versa” (ibid., p. 28). One of the best ways of working at the psychological level is to propose a cognitive model of certain psychotic signs or symptoms, and then test specific predictions arising from the model using suitably designed experiments. Clearly this uses the theory-driven approach discussed in Chapter 1. Over the years, a number of candidates have been proposed for a specific cognitive impairment in schizophrenia, but the models have varied widely in their complexity and in their ability to explain particular psychotic features. In the first section of this chapter we shall review a
selection of approaches which are grouped together because of their emphasis on the psychological level of explanation. The aim of the review is to highlight common features present in many of the models, and to show which ones are best able to explain signs and symptoms. Many of the theories were stimulated by the information processing approach to normal cognition, where analogies were drawn between the brain and a computer. This approach was useful as it attempted to specify clearly the processes occurring between stimulation of the organism and its response; this had the potential to provide a precise specification of the relationship between task performance and a particular inferred cognitive function (e.g. Hemsley, 1988; 1993). However, as Hemsley discusses, there is no single agreed model of normal cognitive function, so any theory of schizophrenia had to be based on one chosen model. The cognitive approaches discussed in Section 2.2 generally fall short of the complete explanation at the psychological level advocated by Frith (1992). However, the most influential ones are reviewed, as they have often stimulated experiments whose results can be interpreted within the framework of more recent formulations.

An alternative approach, which has tended to run in parallel with the experimental psychological literature (often leading to duplication of results (Serper & Harvey, 1994)), has been that of classical neuropsychology. This involves the administration of psychological tests which are thought to be sensitive to the presence of lesions in certain brain areas in neurological patients. For example, a patient may be given a test of 'executive function', which is thought to require an intact frontal lobe (McCarty & Warrington, 1990). The assumption of the approach when applied to schizophrenia is that failure by a patient on a particular test indicates brain pathology in the area subserving test performance, so clearly workers in this area are interested in specifying both the psychological and physical bases of psychosis. However, as pointed out by Frith (1992), this method provides incomplete explanations of phenomena at both the psychological and physiological levels, and therefore tends to be unsatisfactory. A schizophrenic patient may show impaired executive
function, suggesting a frontal lobe deficit, but without a psychological model relating executive dysfunction to signs and symptoms, or a knowledge of how a frontal deficit relates to overall brain function, we are no further forward in our understanding of psychosis. Many studies have been carried out in which standard neuropsychological batteries were given to groups of schizophrenic subjects. Some of the better studies have tried to group patients homogeneously in terms of signs and symptoms, or have used a psychological model to relate task performance to particular psychotic features. A selective review of these neuropsychological studies (with an emphasis on the domains of memory and executive function) will be given in Section 2.3 of this chapter. A useful path for future research might involve a combination of the best elements of the cognitive and neuropsychological approaches, so that a cognitive neuropsychology of schizophrenia can be formulated (Frith, 1992). This requires a priori models relating psychotic features to particular cognitive processes; these processes can then be investigated in patients using specially designed neuropsychological tasks. The specificity of these tasks means in turn that it may be possible to relate the cognitive process of interest to underlying brain function using brain imaging techniques. Frith (1992; 1994) has taken such an approach in his model of schizophrenia, which forms the basis for much of the research in this thesis. His model, and the empirical support for it, will be described in full in Chapter 3.

2.2 Psychological Theories of Schizophrenia

Controlled and automatic processing

In the literature on normal cognitive functioning, Shiffrin & Schneider (1977) made the distinction between controlled and automatic processing. The former is thought to occur within conscious awareness: it uses limited resources, occurs relatively slowly in a serial manner, and involves flexible responding to the requirements of a given task. In contrast, automatic processing occurs outside conscious
awareness, with little or no demands on processing resources. It is fast and proceeds in a parallel manner, involving activation of a relatively inflexible set of mental operations in response to a particular input. With practice, tasks which initially required controlled processing may become automatic. Within this framework, Callaway & Naghdi (1982) proposed that schizophrenic patients have impaired controlled, but normal (or supernormal) automatic processing ability. In their terminology, schizophrenia is characterised by deficits in limited-channel-capacity processes (LCCPs), with normal (or faster than normal) parallel processing. Callaway and Naghdi took as evidence for this model the observation that the performance of schizophrenics on a capacity-demanding task (e.g. word recall) is more vulnerable than that of normals to external interference, indicating a shortage of available capacity for controlled processing in schizophrenia. In contrast, they suggested that the performance of schizophrenics on non capacity-demanding tasks is less vulnerable to external interference than for normals, indicating that “automatic processes seem more effective than normal” (ibid., p. 340). They quoted the example of ‘pre-pulse inhibition’, a phenomenon whereby a weak tone followed up to 400 ms later by a loud stimulus leads to inhibition in normals of an eye-blink to the loud stimulus. Schizophrenic patients typically show less inhibition of blink in this paradigm (e.g. Braff et al., 1978), and Callaway and Naghdi interpreted this as meaning that their automatic response to the loud stimulus is more resistant to interference from the weak tone than in normals.

In evaluation of this model, the first thing to note is that it gives no direct explanation of any specific signs or symptoms. However, in fairness to Callaway and Naghdi, we should acknowledge that primarily they were trying to account for abnormal task performance of patients rather than for particular psychotic features. They referred only to “loss of spontaneity”, a “reduced sense of voluntary control” and “disorders of . . . verbal associations” (ibid., p. 342) as phenomena explicable in terms of disordered LCCPs. Examination of the tasks discussed by Callaway and
Naghdi reveals several possible problems with their interpretation. Firstly, the authors failed to consider the issue of general performance deficit in schizophrenia, i.e. the fact that patients tend to perform badly on many tasks, with performance deteriorating as the task becomes more difficult. As tests of controlled processing will, by definition, be capacity-demanding, they are likely to be more difficult (and therefore more discriminating) than tests of automatic processing. From arguments given in Chapter 1, we should therefore not be surprised that schizophrenic patients perform particularly poorly on tests of controlled rather than automatic processing. The apparent schizophrenic impairment in controlled processing may, therefore, be simply an artefact of task difficulty. Also, as mentioned earlier, Callaway and Naghdi used results from interference paradigms in partial support of their model. Examination of these reveals that the paradigm used to explore controlled processing was conceptually quite different from that used for automatic processing. In the former, subjects had to recall words whilst concurrently listening to an interfering stimulus, but in the latter (pre-pulse inhibition), the blink response was recorded after presentation of the ‘interfering’ stimulus. The relative lack of interference found with schizophrenics in the eye-blink task could therefore reflect a failure of the normal mechanism by which the pre-pulse influences subsequent processing. We shall see later that Hemsley and others (e.g. Hemsley, 1994) have used similar results to argue for an impairment of automatic processing in schizophrenia, clearly a very different interpretation of the data to that given by Callaway and Naghdi.

Thus, whilst Callaway & Naghdi’s (1982) paper was important in highlighting a possible selective impairment in controlled processing in schizophrenia, their data were open to more than one possible interpretation. Nuechterlein & Dawson (1984) listed several possible explanations for an apparent reduction in controlled processing capacity on psychological tasks. Firstly, there could be a disorder of automatic processing so that controlled processing capacity was needed to complete operations which were usually carried out automatically outside conscious awareness. This
would leave less controlled processing capacity for task performance. Alternatively, there could be a normal pool of available processing capacity, but failure of an ‘executive’ process in schizophrenia so that capacity was not allocated appropriately. We shall see later that the distinction between automatic and controlled processing has recurred in several models of psychosis, and disturbances of either automatic or controlled processes in schizophrenia have been proposed independently by different research groups. Importantly, however, these groups have often specified the impairments within the context of an elaborated psychological model, and have tried to link them to specific signs and symptoms.

**Preconscious and conscious processes**

An early model proposed by Frith (1979) was related closely to the concepts of automatic and controlled processing. He made the distinction between preconscious processes, which occur automatically in parallel in a high capacity system outside awareness, and conscious processes which involve serial strategic processing, with low available capacity, in awareness. Studies suggest that in normal word recognition, for example, all possible meanings of a word are activated in preconsciousness, but that only one meaning reaches consciousness, the others being inhibited or filtered out. Frith proposed that certain positive symptoms and signs of schizophrenia (namely hallucinations, delusions and formal thought disorder) arise when this ‘filtering’ process fails, so that information which would normally only be present in the preconscious system is allowed to reach awareness. The particular preconscious processes proposed by Frith to intrude into awareness were those concerned with the selection of interpretations of stimuli or with the selection of appropriate responses. As a result, “the patient becomes aware of ambiguous and multiple interpretations of events” (ibid., p. 228). Within this framework, it is possible to explain the apparent reduction in schizophrenia of controlled processing capacity discussed above: If the irrelevant results of normal automatic processing
intrude into conscious awareness, then it is likely that resources will be needed to process this extra information, leaving less available capacity for tasks requiring controlled processing.

Auditory hallucinations were explained within Frith’s (1979) model in terms of an intrusion into conscious awareness of incorrect early interpretations of sounds. This formulation was useful because it made a testable prediction, namely that auditory hallucinations should be more common when the subject receives ambiguous auditory input (so is more likely to misinterpret sounds). Two studies (Margo et al., 1981; Gallagher et al., 1994) confirmed this prediction, showing that the intensity of ‘voices’ reported by schizophrenic patients was reduced as auditory input given over headphones became more structured. Within Frith’s model, delusions could arise by normal reasoning being applied to make sense of the auditory hallucinations, a suggestion first put forward by Matussek (1952, quoted in Frith, 1979). Thus, to explain his voices, the patient may, for example, develop the false belief that God is able to communicate with him. However, Frith’s model could also account for the clinical observation that delusions sometimes occur in non-hallucinating patients. If, due to the proposed cognitive impairment, a percept was to arrive in conscious awareness after correct interpretation of a particular stimulus, normal reasoning might suggest that the percept had some special significance in the current situation. The patient may then construct a delusion to explain this apparent significance. As we shall see later, a similar argument has been used by Anscombe (1987). Finally, Frith (1979) was able to account for formal thought disorder (FTD) in terms of his proposed cognitive impairment. He suggested that whereas hallucinations and delusions were primarily a result of misinterpreting stimuli, FTD could arise at the response level of information processing due to a failure to exclude from consciousness the multiple meanings of words activated in preparation for speech. This could lead to the production of neologisms (i.e. new words made up by the patient), or words inappropriate to the current situation.
The evidence for and against this model will not be discussed here in detail, because Frith (1992; Frith & Done, 1988) has since rejected it in favour of a different formulation. He felt that the 1979 model was too general, pointing out that the nature of the defective filtering of information needed to be specified more precisely. Also, as we shall see later, there is some evidence to suggest that schizophrenia is characterised primarily by deficits in output processing, with the processing of inputs being essentially intact. As the 1979 theory relied exclusively on deficits at the stimulus (input) level to explain delusions and hallucinations, Frith & Done (1988) developed a new model which concentrated much more on the output level of processing. This will be discussed fully in Chapter 3, but it should be noted that some current theories of psychosis still emphasise deficits at the input level, and it remains a matter of controversy whether the primary disturbance is at the input or output stage of processing. Like Frith’s 1979 model, several of these theories also proposed that a defective filtering of information underlies psychotic symptomatology. However, they were presented explicitly within the framework of selective attention theory, so will be discussed together later in this section.

Sustained attention

Since the time of Kraepelin and Bleuler, there has been a suggestion that attention may be impaired in patients with schizophrenia, and many workers have investigated whether poor performance on various cognitive tests of attention is a specific feature of the illness. Because attention is such a broad concept, we need to concentrate on a relatively well-defined aspect of it in order to make any progress. One such construct is that of ‘sustained attention’, which refers to the ability to maintain focus on a particular stimulus over a period of time. Nuechterlein and his colleagues (e.g. Nuechterlein & Dawson, 1984) linked sustained attention to the concept of controlled processing capacity discussed earlier. They suggested that schizophrenic patients have reduced available processing capacity for task-relevant
cognitive operations, so are less able to sustain attentional focus on a given stimulus. Nuechterlein and Dawson went on to propose that deficits in sustained attention may be a vulnerability marker for schizophrenia (i.e. they occur in symptomatic and asymptomatic patients as well as their relatives, and indicate a predisposition towards developing psychotic features), and a large body of literature has now developed around the measurement of sustained attention in schizophrenic patients. It should be emphasised that the main focus of this work is on finding a trait rather than state marker, so Nuechterlein and Dawson do not propose detailed mechanisms whereby impaired sustained attention could lead to particular symptoms and signs.

The most commonly used task in this literature is the Continuous Performance Test (CPT), in which stimuli are presented tachistoscopically at a fixed, rapid rate (e.g. 1 per second) over 5 - 15 minutes; the subject must respond to a predesignated letter or number (e.g. press a button every time 'a' appears). Recent variants of the task include a version with degraded stimuli, and a 'memory-load' version in which a conditional instruction must be followed (e.g. only respond to the number 7 if it was preceded by 3). In a brief review of the literature, Nuechterlein et al. (1994) provided evidence to support the notion that deficits in performance on the CPT are specific to schizophrenia, and do indeed provide a vulnerability marker for the illness. They argued that although several different psychopathological groups show a CPT impairment, signal detection analyses of scores reveal that only schizophrenic groups show deficits on a signal/noise discrimination (sensitivity, d') dimension, whilst maintaining normal response criterion levels (β). A recent longitudinal study of schizophrenic subjects (discussed by Nuechterlein et al., 1994), using the 'degraded stimuli' version of the CPT task, showed that impairment occurred relative to normals when patients were either symptomatic or in remission, suggesting that CPT deficits may be a stable vulnerability indicator. With the 'memory-load' version of the task described earlier, schizophrenics in either a psychotic or remitted state were again impaired relative to normal controls, but this time there was an additional
effect of clinical state in the patients, such that performance was worse in the psychotic state. The authors interpreted these results, (together with those from the ‘degraded stimuli’ task), as indicating that deficits in stimulus analysis on the CPT may constitute a relatively stable impairment in schizophrenia, whilst disturbance in the influence of memory as a guide to the interpretation of sensory input may be more related to the presence of positive psychotic symptoms. This last proposal will be discussed further in later parts of this thesis. Finally, a number of studies (reviewed by Nuechterlein et al., 1994) suggest that CPT deficits are present in the offspring of schizophrenic patients, but not in the children of patients with other psychiatric disorders; this is again consistent with the notion of CPT impairments as vulnerability markers for schizophrenia.

In any critique of this approach, it is important to remember that other psychiatric groups do in fact show impaired performance on the CPT, and can often only be distinguished from schizophrenic groups when patterns of impairment are analysed by signal detection methods. It is unclear how valid it is to propose a distinct schizophrenic deficit based solely upon these mathematical analyses. Furthermore, the studies which have examined symptomatic and remitted schizophrenic patients have often used normal controls rather than patients with non-schizophrenic psychiatric illnesses. Thus, it is still possible that symptomatic and remitted patients with, say, a diagnosis of unipolar depression, would show a CPT deficit. If this were the case, it would suggest that CPT impairments may be associated more with the presence of a psychiatric diagnosis than with schizophrenia per se. Some studies (e.g. Grove et al., 1991) have shown a relationship between impairment on the CPT and the presence in schizophrenic patients and their relatives of negative features such as affective flattening and anhedonia. This again suggests that patients with illnesses such as depression (who may also show affective flattening and anhedonia) might show CPT deficits, contrary to the notion of a schizophrenia-specific impairment.
If poor performance on the CPT is related to the presence of particular negative signs, it is unfortunately not clear what this means in terms of impaired cognitive processes. Many different psychological functions are likely to be required for normal task performance, so a poor score does not necessarily mean that a subject has a specific deficit in, say, sustained attention. These ambiguities are exacerbated by the use of new forms of the CPT involving, for example, degraded stimuli. An impairment on this type of task could reflect either a deficit in sustained attention, a deficit in perceptual discrimination, or both. Thus, whilst Grove et al. (1991) found a relationship between CPT impairment and the presence of some negative signs, it is not possible to conclude that sustained attention deficits are responsible for these signs. This discussion highlights how important it is for researchers to develop tasks that tap well-defined cognitive processes if they are to increase understanding of the cognitive basis of psychotic features; as it currently stands, the CPT is a relatively poorly defined task for this purpose.

Theories of impaired ‘filtering’ in selective attention

We saw earlier that Frith (1979) suggested an impairment in schizophrenia in the mechanism which filters information as it passes from preconscious to conscious awareness. Within the framework of selective attention theory, defective filtering of input was proposed as early as the 1960s. McGhie & Chapman (1961) carried out an interview study of newly admitted patients with diagnoses of schizophrenia, and suggested that an impairment of selective attention could account for many of the experiences reported by their subjects. For example, one patient said “Things are coming in too fast. I lose my grip of it and get lost. I am attending to everything at once and as a result I do not attend to anything” (ibid., p. 104). McGhie and Chapman suggested that this overload of incoming information could explain decreased concentration, heightened sensory awareness, and more specifically, formal thought disorder, which they felt reflected an inability to select information
pertinent to the logical sequence of thought. Several groups proposed a deficit in schizophrenia based on Broadbent’s (1958) ‘filter’ model of normal selective attention, which was influential at the time. This postulated a mechanism whereby stimuli were selected on the basis of their physical attributes such as colour, source or pitch (the ‘stimulus set’), so that irrelevant stimuli were filtered out and prevented from entering the limited capacity system. On the basis of this model, it was suggested that schizophrenic patients had an impaired filtering mechanism.

One of the main proponents of impaired filtering was Payne (e.g. Payne et al., 1970), who linked the notion of a defective filter to the concept of ‘overinclusive thinking’ first described by Cameron (1939, quoted in McKenna, 1994). A notable feature of Cameron’s work is that he was particularly interested in explaining formal thought disorder. He suggested that this could arise from an inability to restrict thought to the matter in hand, so that concepts related only loosely to the current discourse became incorporated into the patient’s speech, making it vague and difficult to follow for any listener. Payne proposed that such overinclusion may result from defective filtering, so that patients were unable to exclude irrelevant information when performing cognitive operations. To facilitate empirical investigation, Payne developed a number of tests of overinclusion; for example, subjects had to sort objects into categories, with overinclusion being shown by a tendency to include objects in a category which normals would exclude from it (e.g. putting a potato into the category of fruit). The results of Payne’s investigations will be discussed shortly.

As the defective filter theories outlined above are based on Broadbent’s (1958) model of attention, they clearly predict that schizophrenics (especially those with formal thought disorder), should have difficulty in the selection of information on the basis of physical features. Many experiments have investigated this using the dichotic listening paradigm, a typical version of which requires subjects (wearing headphones) to shadow words or digits read to them in one ear, whilst ignoring distracting
information presented to the other ear. As the to-be-shadowed information is selected simply on the basis of its source (e.g. left ear), rather than content, an intact filter mechanism is thought to be required to carry out the task. Hemsley & Zawada (1976) found that schizophrenic subjects did show evidence of defective filtering relative to normal controls, but the deficit was almost as prevalent in a control group of depressed subjects. The researchers therefore suggested that impaired filtering was related only to a “severity of illness” dimension (ibid., p. 460) rather than to schizophrenia per se. This was supported by a review of many of the studies in the field by Straube & Oades (1992), who concluded that schizophrenic patients show no evidence of a specific impairment relative to psychiatric controls when required to select information on the basis of physical cues. The relationship between particular psychotic features and defective filtering has not been investigated explicitly, although studies which have looked at the shadowing performance of subgroups of schizophrenics (defined in terms of the acute/chronic or paranoid/non-paranoid distinctions) have found no differences across these subgroups (e.g. Korboot & Damiani, 1976).

These results clearly undermine any theories of psychosis couched in terms of defective filtering. A specific prediction of Payne’s model was that overinclusive thinking (as measured by his tasks) should correlate with poor performance on dichotic listening tasks requiring filtering. However, given the above results showing that filtering deficits may simply reflect general psychiatric illness, it is perhaps not surprising that Payne et al. (1970) found no significant relationship between measures of overinclusion and impaired filtering. These results led them to suggest that overinclusion may reflect an inability to ignore both perceptual (physical) and conceptual features of a stimulus, so in Broadbent’s (1958) terms they were now proposing more than just a filtering deficit. A second prediction arising from the work of Payne and Cameron was that overinclusion should be related to the presence of formal thought disorder. McKenna (1994) reviewed the many studies relevant to this,
and concluded that there is evidence suggesting that overinclusive thinking (as measured by Payne’s tasks) is more common in thought disordered patients, whether they have schizophrenia or mania. Thus, whilst ‘defective filtering’ has little explanatory value, the concept of overinclusion may still be worth investigating. In terms of specific cognitive processes, it is clearly too vague a concept to be useful, but if we can elucidate the particular cognitive deficits which lead to abnormal performance on Payne’s tasks, we may move further towards understanding the cognitive basis of formal thought disorder. Recent work by Chen et al. (1994) linked overinclusive thinking to a proposed disorder of semantic memory in schizophrenia, and these workers did find that scores for formal thought disorder were related to one measure of abnormal semantic memory on their task. This area of research will be examined in more detail in Section 2.3 of this chapter.

Hemsley’s attentional model

Further advances were made in the selective attention field following Broadbent’s (1971) modification of his model of normal perception to include the concept of response set (‘pigeon-holing’) as well as stimulus set (filtering). This new model was probabilistic, so that the system received ‘evidence’ about the stimulus; the filter (which still dealt with physical characteristics of the stimulus), did not act in an all-or-none way as with the earlier model, but instead gave greater weight to some stimulus features and less weight to others. The pigeon-holing mechanism then acted on this evidence provided by the filter, integrating it with ‘larger classes’ of evidence such as the meaning or patterning of input. It was thus able to bias the interpretation of the current input, and the preparation of responses to it, in accordance with the probabilities of events as predicted by the integrated evidence. In the words of Gray et al. (1991, p. 2), pigeon-holing biases the interpretation of input “by integrating information from the present context and past experience of similar contexts”. Hemsley (1988) gave the example of a subject shadowing a series
of digits against a white noise background. If the sound ‘ee’ was presented in the middle of the series, then pigeon-holing may bias the subject to respond with ‘3’ because only digits were expected. Unlike the original model, Broadbent’s new formulation was now able to explain the finding that normal subjects, if told to listen only to the left ear presentation in a dichotic listening experiment, still heard their name if it was presented to the right ear. In terms of the model, the right ear information was still allowed into the system by the filter, albeit with a low weight. When pigeon-holing then acted on this evidence, the significance (meaning) of the name to the subject would give it a low selection threshold, increasing the probability of it being heard.

We have seen already that schizophrenic patients show little evidence of a specific deficit in filtering. However, Hemsley (1975) suggested that an impairment in pigeon-holing may be a fundamental feature of the illness. This would mean in practice that patients were “less able to make use of the redundancy and patterning of sensory input to reduce information processing demands . . . each stimulus [would be] treated by the system relatively independently of its context (both temporal and spatial)” (Hemsley, 1990, p. 65). One direct exploration of this hypothesis (Hemsley & Richardson, 1980) used a dichotic listening task in which subjects had to shadow one of two different passages of prose. Both passages were presented simultaneously to both ears, and were read in the same voice at the same rate and volume. The shadowing could therefore only be done using the content of the passages, (rather than any physical features of them), so Hemsley and Richardson suggested that it required an intact pigeon-holing mechanism. As they had predicted, schizophrenic subjects performed significantly worse than normal and depressed controls. However, it should be noted that this study failed to employ an equally difficult control task requiring only filtering, so failed to rule out the interpretation of a generalized deficit in the schizophrenic patients.
Throughout the early 1980s several research groups put forward candidates for a specific cognitive deficit in schizophrenia, and in a review of these, Hemsley (1987) suggested that many of them were very similar to his proposed impairment in pigeon-holing. In particular, there was an emphasis on possible abnormalities in the way in which regularities of past experience (both spatial and temporal) influenced current sensory processing. This led Hemsley (1987, p. 182) to propose that a basic feature of the schizophrenic condition was “a weakening of the influence of stored memories of regularities of previous input on current perception”. Hemsley (1994) clarified his position somewhat by emphasising that in his model the ‘memories of past regularities’ were still stored and accessible to controlled processing; he proposed simply that these stored memories had a weakened influence on the rapid, automatic processing of current sensory input. A later modification also removed the emphasis on perception, so that there was now considered to be a weakened influence of the memories of past regularities on current information processing (e.g. Gray, 1996).

The model has some utility in explaining particular symptoms and phenomena reported by schizophrenic patients. For example, McGhie & Chapman’s (1961) patient quoted earlier, who said “I am attending to everything at once and as a result I do not attend to anything”, could be said to be experiencing ‘over-attention’ (a term coined by Gray et al., 1991). In terms of Hemsley’s model, this could reflect an inability to select only those aspects of the environment which are relevant in light of past experience of similar contexts. This could lead to capture of attention by many irrelevant features. In a similar manner, Gray et al. (1991) proposed that formal thought disorder may arise when the subject fails to integrate information from the current context (e.g. a thought or phrase) with stored information relevant to that context. This would tend to produce a stream of discourse whose elements were only vaguely related to one another. The explanation of hallucinations offered by the model is the “least satisfactory” part of the hypothesis (Gray et al., 1991, p. 3);
Hemsley proposed that the ambiguous, unstructured sensory input resulting from defective pigeon-holing triggers the intrusion into conscious awareness of unexpected material from long-term memory (LTM). This is then attributed to an external source by the subject and is experienced as an hallucination. Much of the evidence for this proposal comes from work on sensory deprivation in normal subjects (discussed by Hemsley, 1990), where it seems that a lack of structured input is associated with hallucinatory experiences. Also, as we saw earlier, studies with schizophrenic patients experiencing auditory hallucinations have shown that the intensity of their ‘voices’ is reduced as auditory input given over headphones becomes more structured (e.g. Margo et al., 1981). (This latter result suggests, incidentally, that schizophrenic subjects are able to make some use of stimulus structure, implying that their pigeonholing is intact to some extent). Although there may well be a relationship between unstructured input and the experience of hallucinations, it is by no means obvious from Hemsley’s model why any intrusions of material from LTM should be experienced by the subject as alien and externally generated. In fact one might argue that information from one’s own memory stores should often seem familiar, and be ‘labelled’ as internally generated. This aspect of Hemsley’s model clearly requires further elaboration if hallucinations are to be convincingly explained.

According to Hemsley (1994), delusional beliefs can be formed by three different possible routes. The first involves a process of normal reasoning applied to hallucinatory experiences, (i.e. Matussek’s (1952) argument already discussed in the context of Frith’s (1979) model). The second route is related directly to the capture of the patient’s attention by incidental details of the environment, due to the hypothesised impairment in the automatic use of context to exclude irrelevant features. Hemsley (1994) cited Anscombe (1987), who suggested that in order to explain this capture of attention, the patient attributes a “spurious sense of significance” to these environmental features (ibid., p. 241). Again, we saw a similar argument applied earlier by Frith (1979), who suggested that patients attach signif-
icance to stimulus features which inappropriately pass from preconscious to conscious awareness. Anscombe (1987) went on to propose that an impairment in the ability to place information within the context of ‘background knowledge’ increases the chance of the patient reasoning abnormally (and forming a delusion) about their apparently ‘significant’ experience, rather than just thinking of it as an aberrant perceptual experience. The final way in which delusions can be formed, according to Hemsley (1994), is via abnormal reasoning arising directly from the hypothesised weakening of influence of past regularities on current information processing. Hemsley argued that normals make causality judgments by noting the co-occurrence of events in space and time, taking into account previous instances when the events did not co-occur. If the schizophrenic patient has a weakened influence of knowledge of previous non-co-occurrences, he may form a delusion of causality regarding two unconnected events which happen to co-occur once by chance. From the above analysis, it is clearly possible that delusions could be formed either by normal reasoning about abnormal percepts, by an abnormal reasoning process itself, or perhaps via both routes. Experimental evidence pertaining to these possibilities will be presented in a later section of this chapter on models of delusions.

It should be stressed that Hemsley’s model aims primarily to explain the positive symptoms of psychosis, although recently Hemsley (1994, p. 112) proposed that negative signs may reflect “a more drastic weakening of the influence of stored regularities on current perception [which] result[s] in a level of disorganisation such as to render difficult any goal-directed activities”. To the best of my knowledge, this formulation has not been developed further, but it is preferable to Hemsley’s (1977) earlier suggestion that negative signs (especially social withdrawal and poverty of speech) reflect a coping strategy for dealing with the state of ‘information overload’ resulting from impaired selective attention. As Frith (1992) pointed out, this is unlikely as negative signs can be present from the earliest stages of a schizophrenic illness, so are likely to be primary rather than secondary features of the illness.
Evidence for the model

A brief review will be now be presented of some of the experimental results which Hemsley and others have used in support of his model. A feature of many of these studies is the use of paradigms in which the cognitive deficit proposed by Hemsley predicts superior performance of schizophrenic patients relative to controls. This methodology has the potential to overcome the problem of generalised deficit discussed in Chapter 1, although as Hemsley (1988) points out, the generally lowered performance of schizophrenic patients means that it may also be of interest to consider cases where their performance on such tasks is equivalent to, as well as superior to, that of controls. The evidence presented in this section will be concerned solely with Hemsley’s suggestion of ‘a weakening of the influence of stored memories of regularities of previous input on current information processing’. Investigations of abnormal reasoning style in deluded patients will be discussed fully in the later section on delusions.

A study which was not designed to test Hemsley’s theory, but which is sometimes cited as supporting it (e.g. Hemsley, 1988), was carried out by Schwartz Place & Gilmore (1980), and replicated by Wells & Leventhal (1984). In Experiment 1 of their study, Schwartz Place and Gilmore tachistoscopically presented, for a time interval of about 20 ms, stimuli containing either lines alone (the no-noise condition), or lines and circles (noise condition), to 10 chronic schizophrenic subjects (of unspecified symptomatology) and 10 age-matched psychiatric controls. On each of the 152 experimental trials, subjects simply had to report the number of lines present. Results showed that schizophrenics performed significantly worse than controls only in the noise condition, and the authors interpreted this in terms of a two-stage model of information processing. They suggested that normals analysed the noise stimuli by first structuring them according to their global gestalt properties of similarity and proximity (i.e. spatial ‘regularities’ in Hemsley’s terminology), so that one group of lines and one group of circles were perceived. The lines could then be counted in
stage two by applying a detailed analysis only to the group of lines. In contrast, it was proposed that the schizophrenics failed to perform the initial global analysis, so in the noise condition had to examine each element to determine its identity as well as counting the lines. This would lead to their observed poor performance in the noise condition, but would have no effect in the no-noise (only lines) condition, where an initial global analysis would be superfluous. In Hemsley’s terms, the schizophrenics showed a weakening of the influence of knowledge of spatial regularities (i.e. gestalt properties) on the automatic processing of the stimuli, so perceived input whose structure was not constrained by contextual information.

The poorer performance of patients relative to controls in this study could, of course, be ascribed to a generalised deficit with the noise condition being more difficult. In Experiment 2, of their study, however, Schwartz Place & Gilmore (1980) used a paradigm in which the proposed schizophrenic deficit predicted superior performance. This time, subjects were presented with displays containing horizontal lines, vertical lines, or combinations of the two, and had to simply report the total number of lines present. The displays varied in their degree of gestalt grouping of elements, ranging from high gestalt (i.e. the ‘homogeneous’ condition where all lines were of the same orientation) to lower (i.e. the ‘heterogeneous’ conditions where both orientations were present, and lines of the same orientation were either adjacent or non-adjacent). Results showed that psychiatric controls performed worse on passing from the homogeneous to the heterogeneous conditions, whereas schizophrenic patients showed roughly equal performance over all conditions, giving them a higher overall accuracy than controls. This was interpreted in a similar way to Experiment 1, i.e. that controls were influenced by the gestalt properties of the stimuli, so initially grouped lines of the same orientation. When both orientations were present this would lead to slower counting, resulting in poorer performance relative to the schizophrenics, who were presumed to treat all the displays equally, irrespective of their organizational properties. Again this may be consistent with
Hemsley's notion of a weakening of spatial context in schizophrenia, and could be taken as evidence in favour of his model. However, there is a potential problem with the interpretation of this experiment given by Hemsley: his theory emphasises a "weakening of the influence of stored memories of regularities of previous input on current information processing" (Hemsley, 1994, pp. 100-101, my italics added). This suggests that an important feature is the weakened influence of previously learned information. However, it is widely agreed that gestalt principles are not learned, but are a 'hard-wired' feature of the normal human brain; the paradigm used by Schwartz Place and Gilmore arguably does not, therefore, require the use of previously learned information.¹

A second type of paradigm of possible relevance to Hemsley’s theory can be found within the large literature on reaction time in schizophrenia, reviewed by Straube & Oades (1992). Of interest here are studies in which subjects were given a warning signal, followed shortly afterwards by a stimulus to which they had to respond as quickly as possible. In the ‘embedded procedure’ introduced by Bellissimo & Steffy (1972), subjects were presented with a series of warning-stimulus pairings, most of which had different (irregular) time intervals between the warning tone and the stimulus. However, embedded in the long series of pairings were sequences of up to four pairings with the same (regular) time intervals. Normal subjects showed faster reaction times during the regular sequences, possibly because, at an automatic level of processing, they used the predictability of the time intervals to guide their responses. Schizophrenic patients, on the other hand, showed the so-called ‘crossover effect’: at time intervals of a few seconds, they responded like normal subjects (i.e. faster with regular intervals), but at longer time intervals (greater than 6s) they responded similarly to the regular and irregular sequences (in fact somewhat faster with the irregular sequences). This is consistent with a ‘weakened

¹ I am grateful to Chris Frith for this suggestion.
influence of memories of regularities of previous input’ in the situations with longer

time intervals, and indeed Bellissimo and Steffy offered a similar explanation in
terms of an inability to make use of redundancy in the input. Of course, the fact that
schizophrenic patients responded like normals at short time intervals would imply
that this cognitive impairment is relative rather than absolute in schizophrenia.

Regarding symptomatology, studies suggest that the cross-over effect is particularly
common in poor pre-morbid, chronic schizophrenic patients (Straube & Oades,
1992), who we might expect to show predominantly negative rather than positive
features. This is not consistent with Hemsley’s model, which might predict the effect
to be particularly marked in acute patients with mainly positive symptoms.

Furthermore, it is possible that effects other than a redundancy deficit could account
for the experimental results. For example, subjects may find the conditions with long
time intervals (i.e. greater than 6s) rather monotonous (especially when the time
intervals are regular), and perhaps lose concentration on the task. This would account
for the patients’ slower reaction times in the regular compared to the irregular
conditions, and might also explain why normals typically show a slight increase in
reaction time with long regular sequences (Straube & Oades, 1992).

The main studies cited by Hemsley and colleagues in favour of their model use
the Latent Inhibition (LI) and Kamin ‘blocking’ paradigms (see Gray et al., 1991;
Gray, 1995, and Hemsley, 1994, for reviews). The work with blocking will not be
reviewed here, as essentially the studies support the findings using LI. Within an LI
paradigm, a stimulus that is to be used as a CS is either pre-exposed or not, with pre-
exposure meaning presentation of the CS without consequence. The CS is then paired
with a US, and LI reflects the fact that the CS-US pairing is learnt more slowly in the
pre-exposed condition. In the studies discussed by Gray et al. (1991), the CS was an
auditory stimulus and the US was a number increment on a visual counter. The
paradigm can be related theoretically to Hemsley’s model because in pre-exposed
normal subjects, the CS-US learning is thought to be disrupted by the automatic
influence of previously acquired knowledge about the irrelevance of the CS, (i.e. the influence of 'stored memories of regularities of previous input' is important for LI to occur normally). The application of LI to schizophrenia was also suggested by findings that the indirect dopamine agonist amphetamine attenuates LI (i.e. reduces the 'influence of stored memories of regularities') in normal humans. As it is widely held that a hyperdopaminergic state is characteristic of the acute stages of psychosis (e.g. Davis et al., 1991), it was predicted that acute schizophrenic patients would show reduced LI, and perhaps learn the CS-US contingency faster than normal controls. Again, therefore, we have a paradigm with the potential to overcome problems of generalised deficit in schizophrenic subjects. The LI procedure is also useful in that it has been widely applied in studies with rats, where putative neural mechanisms underlying LI have been suggested (see Gray et al., 1991, for a review).

Results of experiments with the LI paradigm in schizophrenia have been generally mixed. Data from Gray's research group (reviewed by Gray, 1995) have shown that LI is abolished, as predicted, in the early stages of an acute psychotic episode, with schizophrenic patients showing faster learning of the CS-US association than normals in the pre-exposed condition. Furthermore, LI returns to normal levels in such patients after 8 weeks of treatment with neuroleptics (i.e. dopamine receptor antagonists), and is found at normal levels in medicated chronic schizophrenic patients (those defined by Gray and colleagues as having an illness of more than 3 months' duration). These findings have been taken to suggest a link between abolition of LI and positive symptoms (in accordance with Hemsley's model), because such symptoms are particularly prevalent in acute episodes and are the main target of typical neuroleptics. However, studies specifically examining the relationship between positive symptom scores and extent of abolition of LI, have produced conflicting results. For example, Baruch et al. (1988) found that scores for positive, but not negative, features on the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962) correlated with the reduction of LI, whereas N. S. Gray et
al. (1992) found that LI was abolished in acute, but not chronic schizophrenics, when both groups were matched on BPRS scores for positive symptoms. This latter result suggests that LI may be fairly directly related to dopamine receptor blockade rather than to positive symptoms (N. S. Gray, personal communication), and poses a major problem for Hemsley’s psychological model of schizophrenia. In addition, there is evidence from recent work by other research groups that the abolition of LI effect in acute patients may be difficult to replicate. For example, Swerdlow et al. (1996) used the same apparatus and paradigm as Gray’s group, but found that both acute and chronic schizophrenic patients showed normal LI.

Several other paradigms are similar conceptually to LI, and have been investigated in schizophrenia. For example, we saw earlier that in a standard pre-pulse inhibition paradigm, where a blink-eliciting stimulus is forewarned by a pre-pulse, schizophrenic patients typically show less inhibition of blink compared with controls (e.g. Braff et al., 1978). This is consistent with a weakened influence in schizophrenia of the memory of the pre-pulse on subsequent information processing, and accords with Hemsley’s model. The ‘negative priming’ (NP) paradigm, originally developed by Tipper (1985), has also been applied widely in schizophrenia research. In this, a distractor from a priming display becomes a target on an immediately following display. Normals show a longer reaction time (negative priming) to this target compared with trials where the target has not previously been a distractor. In terms of Hemsley’s model, this can be seen as the influence of previous learning that the target was irrelevant upon the subsequent response to that target. Hemsley might therefore predict that schizophrenic patients with positive symptoms would show less NP than controls, responding faster to the target. Beech et al. (1989) tested this prediction using medicated schizophrenic patients (all of whom showed at least one Schneiderian ‘first rank’ symptom) and non-psychotic psychiatric controls. The schizophrenic sample showed a reduction in negative priming relative to the control group, and did indeed respond faster to the target than the control group in the NP
condition. This finding has since been replicated with other groups of schizophrenic patients (e.g. Williams, 1996), and with spatial rather than verbal paradigms (Park et al., 1996). Some studies have also found that extent of abolition of negative priming is related to questionnaire ratings of positive (but not negative) features of schizotypy in healthy subjects (e.g. Park et al., 1996, Experiment 3; Peters et al., 1994).

Preliminary studies have shown that, as with latent inhibition, negative priming is abolished in human subjects if they are administered a 5 mg dose of the dopamine agonist amphetamine (Gray et al., 1991). Thus, whilst the negative priming data discussed above are consistent with Hemsley’s model, it still remains possible that NP effects (as with LI) may be more directly related to dopamine receptor blockade than to positive symptoms per se. As we saw earlier, N.S. Gray et al. (1992) found that acute and chronic schizophrenics matched on scores for positive symptoms showed differential abolition of LI. The corresponding experiment using the negative priming paradigm still remains to be done, but results so far do suggest that NP may be less sensitive to dopamine receptor blockade than is LI. For example, the patients with positive symptoms in the studies by Beech et al. (1989) and Williams (1996) were almost all medicated, and had probably been receiving medication long enough for it to have had some therapeutic effect. However, these subjects still showed a clear abolition of negative priming. Furthermore, Williams (1996) found that her subgroups of patients with positive or negative features were matched on medication dosage, yet only those with positive symptoms showed abolition of NP. Work in this area has been complicated by results from David (1995) who found that negative priming was normal in his sample of neuroleptic-free psychotic patients, but was abolished in the patients receiving medication. This is difficult to reconcile with Gray et al.’s (1991) finding that dopamine agonists abolish NP, and suggests that further research is needed (especially with non-medicated patients) to disentangle the precise relationship between negative priming, positive symptoms and dopamine receptor blockade.
A final theoretical point relating to negative priming concerns its relevance to Hemsley’s model. Tipper’s (1985) analysis suggests that negative priming occurs because the internal representation of the distractor stimulus becomes associated with ‘cognitive inhibition’. If the distractor stimulus is a word, for example, then we can imagine that inhibition occurs in the semantic network around the node associated with that word. Thus, in Tipper’s terms, abolition of negative priming in schizophrenia would reflect a lack of inhibition in the semantic network. This is subtly different from Hemsley’s model, which suggests that the schizophrenic subject learns normally that the distractor is irrelevant (i.e. the ‘past regularity’ is represented normally in memory), but that this knowledge fails to influence subsequent information processing. If Tipper’s analysis is correct, then it might suggest that the negative priming (and perhaps even latent inhibition) data, although often consistent with Hemsley’s model, may in fact be explicable in terms of a lack of inhibition being associated with the irrelevant information when it is presented in the first part of the NP or LI paradigm. A set of studies of semantic priming in schizophrenia (which will be discussed in Section 2.3) have some bearing on this argument. Several groups (e.g. Spitzer et al., 1994) have found that schizophrenic patients with the positive behavioural sign of formal thought disorder show more semantic priming than non-thought disordered patients and controls, i.e. they show a shorter recognition time for a word such as ‘wrist’ when it has been preceded by ‘hand’. This is compatible with increased activation (reduced inhibition) in the semantic network in these patients, and provides support for an analysis of the abolition of NP in schizophrenia in terms of reduced cognitive inhibition. Moreover, it is difficult to explain enhanced semantic priming in terms of Hemsley’s model; he would predict a weakened influence of previously presented information in schizophrenic patients with positive symptoms (including formal thought disorder), so might expect them to show reduced semantic priming relative to controls.

In summary, we have seen that a number of studies can be interpreted as
providing support for Hemsley's model of the positive symptoms of schizophrenia. However, there are possible problems with each set of evidence presented. In particular, the paradigms of LI and NP, whilst at first sight producing data consistent with the model, reveal subtle complications when examined in more detail. In the next section, studies are discussed which provide quite direct evidence against Hemsley's theory. Some of these also suggest an alternative approach to the signs and symptoms of psychosis.

Evidence against the model

Recent work by O'Carroll et al. (1993) directly investigated Hemsley's model using the proactive interference (PI) paradigm. This involves several successive presentations, with recall, of a word list (A), followed by a single presentation, with recall, of a different word list (B). PI occurs in normals when the immediate recall of list B is worse than the immediate recall, on the first trial, of list A. It is thought to reflect interference of list A learning with the acquisition of material from list B, and in terms of Hemsley's model is an example of the automatic influence of stored memories of previous input on current information processing. The prediction from Hemsley's theory would therefore be that acute schizophrenic patients with positive symptoms should show less PI than controls. Contrary to this prediction, O'Carroll et al. found that their unmedicated schizophrenic sample showed the normal proactive interference effect. In a further study, O'Carroll (1995) examined associative learning in acutely ill and remitted schizophrenics, as this is more obviously related to the latent inhibition paradigm than is PI. Subjects had to learn 10 simple paired associates (e.g. Metal - Iron) over 5 trials, then immediately afterwards had to learn new associations, each of which involved one item from the previous pairs together with a new partner (e.g. Metal - Copper). Again, Hemsley's theory would predict that acutely ill schizophrenic patients should show less interference of previous learning upon subsequent learning, so should learn the new pairings more easily than controls. This prediction was not supported - the acutely ill subjects performed more poorly
than remitted patients in learning the new word pairings.

A study not designed with Hemsley’s model in mind, but having some bearing upon it, was carried out by Gerver (1967). He presented subjects with word strings masked by white noise; these strings consisted of either grammatical and meaningful sentences, grammatical and meaningless sentences, or random words. Gerver found that chronic schizophrenic inpatients (of unspecified symptomatology) and controls both perceived the words more accurately as the cohesiveness of the strings increased. As word recognition in such tasks is widely held to occur at an automatic level of processing (e.g. Morton, 1979), this result can be interpreted in Hemsley’s terminology as showing a normal influence of contextual information on the automatic processing of stimuli by schizophrenic patients. Similarly, Rochester et al. (1973) used the ‘click paradigm’ to investigate speech perception in schizophrenic inpatients (of unspecified symptomatology), and found that they performed the same as normal controls. The paradigm involves auditory presentation of phrases, with a short burst of noise (a click) superimposed. Irrespective of the actual position of the click, Rochester et al. found that schizophrenic patients perceived it as being at, or near, a clause boundary to the same extent as normals. Again, this can be viewed as a normal influence in schizophrenia of stored memories of syntactical constraints upon the automatic processing of input, and is not consistent with Hemsley’s model of psychosis.

Done & Frith (1984) proposed on the basis of such evidence that the use of contextual information at an automatic level of processing is intact in schizophrenia. They devised a test of this hypothesis involving the use of context in auditory and visual word perception. In the visual paradigm, subjects were presented with sentences from which the final word had been deleted (e.g. “Coming in he took off his . . .”), and after a few seconds, the stimulus word (e.g. “coat”) was presented for a brief duration. The subject had to read aloud this stimulus word. The initial
exposure duration of the word was very short, but on each cycle through the procedure, it was exposed for increasingly long durations, so became easier to read. Thus, in early cycles subjects effectively had to guess a word which could fit the sentence context, whereas at a later stage, when perceptual threshold was reached, they could produce the correct word. The number of cycles for identification of the stimulus word was taken as a measure of the perceptual threshold, and any incorrect words produced before threshold were analysed for their suitability to the sentence context. The effect of context was investigated by using sentences of high and low contextual constraint.

Results from this, and the similar auditory paradigm, showed that context had a normal influence on perceptual thresholds for both acute and chronic schizophrenic patients, i.e. thresholds were reduced with sentences of higher contextual constraint. These patients were all medicated, but still symptomatic, so if Hemsley’s model was correct, we would have expected certainly the patients with positive symptoms to have shown less influence of context on this automatic processing of stimuli. This was not found. Analysis of incorrect responses produced before threshold was reached showed that some schizophrenic subjects, especially those with negative signs, had a tendency to give responses which were inappropriate to the context. This led Done and Frith to suggest that schizophrenics only have problems in the use of context when automatic processes cannot operate (due to a lack of available ‘evidence’ in the stimulus), so responses have to be generated using strategic processing with guessing and editing procedures in conscious awareness. This has marked parallels with Callaway & Naghdi’s (1982) proposed impairment of controlled processing in schizophrenia, and places a firm emphasis on deficits at the output, rather than input, stage of processing. Within this framework, it is instructive to consider Hemsley & Richardson’s (1980) dichotic listening experiment discussed earlier. In this, subjects had to shadow one of two passages which differed only in their content, and the impairment shown by schizophrenics relative to controls was
interpreted by Hemsley and Richardson as reflecting a pigeon-holing deficit. However, in terms of the above discussion, this task can be seen as placing quite strong demands on controlled, rather than automatic processing. Thus, although all the necessary ‘evidence’ is in fact present within the two passages, subjects have to employ conscious monitoring processes in order to use the sentence context to produce an appropriate response. The poor performance of schizophrenic subjects on this task may, therefore, be compatible with Done & Frith’s (1984) analysis.

Many other studies indicate a schizophrenic deficit in the use of context in tasks requiring controlled processing. For example, de Silva & Hemsley (1977) tested patients on the ‘Cloze’ procedure in which subjects must guess the deleted words in passages of prose. This is clearly very similar to the pre-threshold situation in Done & Frith’s (1984) experiment. Passages of varying contextual constraint were prepared by deleting, for example, every tenth word (high context) or every fourth word (low context), and these were presented in a counterbalanced order to groups of acute and chronic schizophrenics (of unspecified symptomatology) and normal controls. Results showed that controls guessed more words correctly as contextual constraint increased, whereas the schizophrenics as a whole performed more poorly with increasing context. Analysis of subgroup data showed that the chronics tended to score about equally in each context condition, whereas the acute schizophrenics performed worse as contextual constraint increased. These results cannot be attributed to a generalised deficit in the schizophrenic patients, because they performed worse in the easier (high context) rather than harder conditions. Instead, the data are compatible with difficulties in the use of context to generate appropriate responses using strategic processing. We can perhaps speculate that the reason for poorer performance with increasing context is that the patients had to read and process more stimulus words in the high context conditions, so had more opportunity to make inappropriate associations to particular words in those conditions. If, as proposed, the patients had difficulty with the conscious monitoring and editing of
responses, then incorrect responses would be more likely in those higher context conditions, as found.

Naficy & Willerman (1980) used a similar paradigm, but as well as testing normal controls, they compared their group of schizophrenic subjects (with positive and negative features) with a group of manic patients exhibiting positive signs such as incoherent speech and inappropriate behaviour. All of the patients were medicated at the time of the study. Subjects were given sentences containing an underlined word, which had both a common and uncommon meaning, and had to choose the meaning appropriate to the sentence context from a list provided. In accordance with the notion that schizophrenic patients have problems in the use of context in controlled processing tasks, the schizophrenic group in this study was significantly more likely than normals to choose the common word meaning in sentences where the unusual meaning was required. Interestingly, the manic subjects showed the same tendency, suggesting that the effect may be related to the presence of particular symptoms and signs (e.g. incoherence).

Finally, we saw earlier that studies using the Continuous Performance Test (CPT) in schizophrenia have shown that the presence of positive symptoms is related to poor performance on the ‘memory load’ version of the task, where subjects must respond to, say, ‘7’ only if preceded by ‘3’. Again this task requires the ability to use contextual cues during conscious, controlled processing, and the results are consistent with an impairment of this ability in schizophrenic patients. The idea that schizophrenic cognitive deficits may be primarily associated with output generation in conscious awareness was elaborated by Frith (e.g. Frith, 1987; Frith & Done, 1988) into a neuropsychological model of the signs and symptoms of psychosis, which will be presented in Chapter 3. At that point, we shall return to the above studies of controlled contextual processing in schizophrenia, and an attempt will be made to explain their results in terms of Frith’s model. For now, however, it should be noted
that research is still needed to directly compare models of psychosis proposing contextual deficits in automatic processing, with those suggesting context deficits at only the conscious, controlled level. One such study (which to the best of my knowledge has not been done), could use the shadowing paradigm discussed earlier, in which subjects must shadow a very long series of numbers presented against a white noise background. It is likely that this paradigm has a large automatic processing component, so that a sound ‘ee’ presented during the number series would often be shadowed by normals as ‘3’ (reflecting intact pigeon-holing). Hemsley’s model would predict that psychotic patients would be more likely than normals to shadow the sound correctly (i.e. as ‘ee’), because of their proposed pigeon-holing abnormalities. In contrast, researchers such as Done & Frith (1984) who suggest that the context deficit in schizophrenia is apparent only at the strategic, controlled level of processing, would predict little difference between the shadowing performance of normals and schizophrenics.

 Specific models of auditory hallucinations

So far, we have discussed general models of psychosis which have tried to account for many of the signs and symptoms in terms of one cognitive deficit. However, some workers have proposed models of just one particular symptom or sign, and in this section we shall review specific models of auditory hallucinations; models of delusions will be discussed in the next section. Frith (1992) briefly reviewed some of the work on hallucinations, and pointed out that (as with much of the psychological literature on schizophrenia), most theories propose deficits in either input or output processing. The ‘input theories’ are those which suggest that auditory hallucinations arise when patients misperceive external stimuli. Thus, Frith’s (1979) early model of hallucinations, which we discussed earlier, is one example of an input theory. Misperception of stimuli could occur either by a failure of discrimination processes or through an abnormal bias towards interpreting stimuli in a particular
way (Frith, 1992). As Frith pointed out in his review, there is little evidence that schizophrenic patients show discrimination failures, and a recent unpublished study by Done and Frith showed that hallucinating schizophrenics had no bias towards hearing speech sounds as words. However, it is still possible that hallucinating patients have a bias towards hearing noises as speech sounds (Frith, 1992). For example, Bentall & Slade (1985) used a signal-detection paradigm in which ten hallucinating and ten non-hallucinating schizophrenic patients had to distinguish between 50 signal (a voice saying “who”) plus noise trials and 50 noise alone trials. Results showed that the hallucinating patients had a significant bias compared to their controls in reporting stimuli as signal rather than as noise only. Notably most of the subjects simply described the signal as a ‘voice sound’ or ‘grunt’, and did not identify the word ‘who’. Although these results are consistent with misperception of input in hallucinating patients, the authors interpreted the data in terms of deficient ‘reality testing’ in the hallucinators, i.e. a tendency to attribute internal (self-generated) events (such as voice sounds) to an external source. This makes Bentall and Slade’s model effectively an ‘output theory’ of hallucinations, as it proposes that a patient’s ‘voices’ are internally generated but misattributed to an external agent (see Slade, 1994).

Output theories of hallucinations received support from early studies showing that some schizophrenic patients showed increased electromyographic activity in the speech musculature at the time when auditory hallucinations were reported to begin. In addition, amplified subvocal speech was sometimes found to correspond closely with the reported content of hallucinations (see McKenna, 1994, p. 175, for a review of these studies). Thus, it is likely that in these cases, patients were perceiving their own subvocal speech as an externally generated voice. As discussed by Frith (1992), auditory hallucinations could also arise from ‘inner speech’ in the articulatory loop component of Baddeley’s (1986) working memory system. This would not give rise to any sound or muscle activity, but if attributed to an external source may still be
experienced as a voice. In support of this are findings from prelingually deaf schizophrenic people, who sometimes insist that they can ‘hear voices’ (e.g. Critchley et al., 1981, quoted in Frith, 1992). Their experience suggests that auditory hallucinations can arise from inner speech (or even thoughts) rather than from actual sounds. We saw in earlier sections that a study by Margo et al. (1981) found that the loudness, clarity and duration of patients’ hallucinations decreased as their auditory input became more structured. Whilst this is not inconsistent with input theories of hallucinations, it should be noted that the greatest reduction in the intensity of ‘voices’ in that study occurred when patients listened to a prose passage or read aloud. As these activities almost certainly disrupt inner speech processes, this result would be predicted from output models of hallucinations. A common factor in all of the output theories is that an internally generated word or phrase is somehow not recognised as one’s own, so is perceived as externally produced. Theorists differ as to how precisely this might occur, but one of the most fully worked out models has been developed by Frith (1987; 1992) as part of his attempt to explain many of the signs and symptoms of psychosis. This will be presented in Chapter 3.

An output theory of hallucinations with some similarities to Frith’s (1987; 1992) model was proposed by Hoffman (e.g. Hoffman, 1986; 1991). He noted that the content of patients’ ‘voices’ is often repetitive and stereotypic, as though stored information was being reproduced each time the patient hallucinated. Hoffman developed computer simulations of pathology in parallel distributed processing (PDP) systems, and suggested that auditory hallucinations reflected random intrusion into current thought processes of words and phrases from long-term memory (LTM). He proposed that the intrusions occurred because of a general impairment in discourse planning, and suggested that the patient experienced them as ‘alien’ (i.e. externally generated) because they were unintended and unrelated to his current thoughts. Furthermore, the patient’s real world knowledge that external voices come from other people might influence his perception of the ‘voice’, giving it ‘non-self’ acoustic
characteristics (Hoffman, 1991). (Incidentally, this last proposal is akin to saying that the automatic ‘influence of stored memories of regularities’ is intact in hallucinating schizophrenics, contrary to Hemsley’s model of psychosis). Hoffman (1986) also suggested that the same disruption of discourse planning could result in incoherent speech if the patient vocalised the inappropriate intrusions from LTM. As Frith (1992, p. 75) pointed out, this would predict a co-occurrence of hallucinations and formal thought disorder in schizophrenic patients, but as we saw in Chapter 1, most factor analytic studies find no such association (e.g. Liddle, 1987). However, Frith suggested that the two features may lie on a continuum of severity; thus in less severe cases, intrusions from LTM would remain as inner speech or thought, being experienced as auditory hallucinations. With greater severity, the phrases might actually be spoken aloud, giving rise to incoherence (Frith, 1992). This would predict that the two features should not usually co-occur (as is found empirically), but that patients who experience auditory hallucinations may be more likely to develop incoherence if their condition deteriorates. As far as I am aware, this prediction has not yet been investigated.

In summary, there is a general convergence of theory in favour of ‘output’ models of auditory hallucinations (see Slade, 1994). This is consistent with the results of many studies which suggest that the production of speech, rather than its perception, is abnormal in schizophrenia. For example, we have seen already that Done & Frith (1984) found normal context effects on perceptual thresholds for words, but abnormal use of context by schizophrenic patients when they produced speech. In addition, Cohen (1978) developed a paradigm in which a subject had to describe a coloured disk to a listener in such a way as to enable the listener to select the disk from several (of various colours and shades) placed before him. Results showed that when normal controls described the disks to schizophrenic patients, the patients were able to choose the correct disk (showing intact speech perception). However, when the roles were reversed, the schizophrenics were particularly poor
at describing the disks to normal listeners.

**Specific models of delusions**

As we have seen, two general approaches to delusions have been taken throughout the literature. Firstly, Matussek (1952) suggested that delusions essentially arise from a process of normal reasoning applied by the patient to explain aberrant percepts such as hallucinations. This was elaborated into a full account by Maher (e.g. Maher & Ross, 1984), who cited studies of normal subjects who developed irrational beliefs when placed under conditions such as hypnotically-induced sensory impairment. However, one of the main problems with Maher’s theory is that many patients have delusions in the absence of any reported perceptual abnormalities, and similarly, not all hallucinating patients are deluded. In addition, whilst the model has some utility in explaining the establishment of delusions, it is less obvious why patients with normal reasoning processes should continue to hold their false beliefs with such conviction, often in the face of much contradictory evidence. Maher’s theory is also unable to account for the often bizarre nature of patients’ delusions; one might expect that normal reasoning would produce explanations which were as rational and plausible as possible.

Whilst many workers accept that Maher’s model is probably adequate to explain some delusions, it is also possible that deluded patients show an abnormal reasoning style. For example, patients may give more weight to information which supports their delusional system, and ignore information (such as well-established facts of physical reality), which contradicts their false beliefs. This may explain the maintenance of delusions, and also the fact that they are often bizarre and at odds with everyday reality. As noted by Chapman & Chapman (1988), there is a common tendency within the normal population to ignore evidence which contradicts already established ideas; however, they suggest that delusional patients show a pathological
accentuation of this tendency. We have seen already that Hemsley’s model of psychosis (e.g. Hemsley, 1994) suggests abnormal reasoning as one way in which delusions could be formed. He points out that two uncorrelated events which happen to co-occur by chance may be perceived as related because of a weakened influence of previous experience of non co-occurrence of those events. Thus, Hemsley is also emphasising a failure by deluded patients to appropriately weigh counter-evidence.

One study which directly investigated the phenomenon of ‘illusory correlation’ was carried out by Brennan & Hemsley (1984). Subjects were presented repeatedly with cards showing word pairs, some of which were unrelated (e.g. boat - notebook), some of which were related but without any connection to paranoid delusions (e.g. bacon - eggs), and some of which were related with strong paranoid content (e.g. killer - victim). All permutations of the word pairs were presented an equal number of times in a random order. Normal subjects showed illusory correlation, i.e. they erroneously reported having seen word pairs with an associative connection more often than those without such a connection. In line with theories suggesting a failure by deluded patients to correctly weigh counter-evidence, a paranoid schizophrenic subgroup showed a stronger tendency than normals to report illusory correlations. Interestingly, this effect was particularly marked for those words related to delusional themes.

In an attempt to expose a general reasoning abnormality in deluded patients, Huq et al. (1988) tested deluded schizophrenics, non-deluded psychiatric controls and normals on a probabilistic inference task using emotionally neutral material. Subjects were shown two jars containing different proportions of coloured beads (jar 1: 85% pink, 15% green; jar 2: 85% green, 15% pink). They were told the proportions, the jars were hidden from sight, and the experimenter then selected a series of beads from one of the jars, continuing until the subject told him to stop. Subjects had to guess from which of the jars the beads were being selected, and rated their confidence in their decision. Results showed that deluded subjects requested less draws than controls before reaching a decision, and were relatively overconfident
about their decisions based on this limited information. Garety et al. (1991) extended this research by giving the same task to groups of patients who met diagnostic criteria for schizophrenia (with delusions) or delusional disorder. They too found that deluded subjects (irrespective of diagnosis) requested less information than controls before reaching a decision. In addition, the deluded subjects would more readily change their probability estimates when confronted with potentially disconfirmatory information. Notably, the subjects showing most evidence of abnormal reasoning in Garety et al.'s study were those who also reported perceptual abnormalities. Contrary to Maher's theory, this latter result suggests that in some cases, delusions may be the result of abnormal reasoning about abnormal percepts, or alternatively, both delusions and perceptual anomalies could arise from a single underlying cognitive deficit. Hemsley (1994) interpreted these reasoning studies as being consistent with his general model of positive symptoms. However, whilst they are consistent with the proposed failure of deluded patients to appropriately weigh contradictory evidence, it should be noted that the probabilistic inference task is likely to have placed a high demand on strategic processing, with subjects required to make guesses based on a conscious monitoring of all the presented information. In this sense, the task is not dissimilar to the pre-threshold context task used by Done & Frith (1984) (see the earlier discussion in this chapter), in which subjects had to produce an appropriate word to complete a sentence, using all available contextual information. By this analysis, the results of the reasoning tasks are consistent with theories proposing an impairment in psychotic patients of the ability to generate output using conscious, controlled processing of contextual information, and are not relevant to Hemsley's suggested impairments of automatic processing in schizophrenia.

A number of more recent studies of reasoning in schizophrenia are consistent with this interpretation. For example, John & Dodgson (1994) gave deluded schizophrenic and psychiatric and normal controls an inductive reasoning task based on the 'twenty questions' game. The experimenter posed a question, e.g. "Who am I
thinking of?”, and the subject had to ask up to twenty questions in an attempt to
discover the identity of the person. This task was considered more relevant to
everyday reasoning than the probabilistic inference task of Huq et al. (1988), but as
with that study, John & Dodgson (1994) purposefully avoided affect-laden stimuli
to see whether their subjects had a general reasoning abnormality. An efficient
strategy on the ‘twenty questions’ game would involve subjects asking a series of
‘constraint-locating’ questions, narrowing down possible answers before asking a
‘direct hypothesis’ question. Consistent with the findings of Huq et al. (1988) and
Garety et al. (1991), John and Dodgson found that their deluded subjects requested
less constraint-locating information, produced direct hypotheses (i.e. guesses) more
readily, and made poorer overall judgments than controls. This again suggests an
impairment in the ability to integrate sequentially presented information, and use it
in the strategic generation of responses. Bentall & Young (1996) investigated
hypothesis-testing in deluded, depressed and normal subjects by presenting problems
involving everyday situations with either positive or negative outcomes. Subjects had
to choose strategies to prove that one of three variables was responsible for the
outcomes, and results showed that all subjects employed ‘sensible reasoning’ in this
paradigm (e.g. by choosing to manipulate the variable hypothesised to be responsible
for the outcome more when the outcome was negative than positive). From a
consideration of this, and the studies discussed above, Bentall and Young concluded
that deluded patients may in fact have normal hypothesis-testing ability, so only show
deficits on reasoning tasks when they require the integration of relevant information
over time. As we have seen, several studies (e.g. Done & Frith, 1984) suggest that
this ability to integrate contextual information may be impaired in schizophrenic
patients with both positive and negative features, so we might predict that non-
deluded schizophrenics would also show deficits on the reasoning tasks discussed
above. This remains to be investigated in future research (although it will not be
examined in this thesis). The proposed explanation of abnormal reasoning in schizo-
phrenia in terms of deficient integration of contextual information will be discussed
further in Chapter 6 of this thesis, where links will be made to Frith’s (1992) neuro-psychological model of schizophrenia.

All of the above studies have shown impaired performance by deluded patients on affectively neutral tasks, but an important area of research concerns the patients’ reasoning about emotionally salient information. Because of its content-specificity, any deficits found in this domain can more obviously be linked to the formation and maintenance of persecutory delusions. Bentall and his colleagues (see Bentall, 1994, and Bentall et al., 1994, for reviews) have examined the processing of affective information by deluded subjects, and have found that such patients have an attentional bias towards threat-related words. For example, Bentall & Kaney (1989) gave deluded, depressed and normal subjects an emotional Stroop task, in which they had to name the ink colours of threat-related, depression-related and neutral words; results showed that deluded patients were specifically slowed in colour-naming the threat-related words.

This finding was incorporated by Bentall et al. (1994) into a recent model of the cognitive biases of deluded patients. The model uses social attribution theory, which provides a framework for understanding the explanations given by individuals for their own and others’ behaviour. Kaney & Bentall (1989) gave the Attributional Style Questionnaire (ASQ; Peterson et al., 1982) to patients with persecutory delusions, psychiatric controls and normal subjects. The questionnaire requires subjects to generate possible causes for hypothetical positive and negative events, (e.g. “You go out on a date, and it turns out badly”). Kaney and Bentall found that their deluded patients made more external, stable and global attributions for negative events compared to normal controls. In other words, they systematically blamed other people if something went wrong, and attributed this to factors which were unlikely to change, and which would affect all areas of life. The deluded subjects also made excessively internal, stable and global attributions for positive events compared to
normals (i.e. they systematically credited themselves if something went well). Bentall et al. (1994) pointed out that this cognitive bias is in some ways the opposite of that found in depressed patients, who typically make internal, stable and global attributions for negative events. It is found to a degree in normal subjects when faced with threatening situations, and is widely regarded as a means of maintaining self-esteem (e.g. Taylor, 1988, cited in Bentall et al., 1994). As studies show that deluded patients are particularly attentive to threat-related stimuli (e.g. Bentall & Kaney, 1989, discussed above), Bentall et al. (1994) suggested that persecutory delusions reflect an exaggerated form of this normal bias, serving to protect the individual against chronic feelings of low self-esteem. In terms of the model, threat-related information or negative life events are thought to activate actual self / ideal self discrepancies, leading to low self-esteem. The defensive explanatory biases are then adopted, which reduce the actual self / ideal self discrepancies, but open large self / other discrepancies. In effect, the subject gains a positive view of himself, at the expense of believing that others have a negative view of him (paranoid ideation). This leads to excessively external attributions for any negative events. The model is particularly good at accounting for the maintenance of delusions: if it is true that delusional thinking is, in effect, a coping strategy, then it is not surprising that subjects are often reluctant to challenge their false beliefs.

Bentall (1994; Bentall et al., 1994) reviewed the many studies providing support for his model, but only one study will be mentioned here as it may have some bearing on earlier discussions regarding abnormalities of controlled processing in delusional thinking. Kinderman (1994) gave an emotional Stroop task to deluded subjects, depressed patients and normal controls; subjects had to name the ink colours of neutral, low and high self-esteem adjectives, and in a later interview had to rate the degree to which they endorsed the adjectives as self-descriptive. On the Stroop task, the deluded patients were similar to the depressed subjects in showing a strong attentional bias towards low self-esteem adjectives. However, they were significantly
less likely than depressed patients to endorse negative adjectives as being self-descriptive. As the Stroop task is widely held to tap automatic levels of processing, whereas the endorsement of adjectives is a controlled process, this result is consistent with cognitive biases operating at a controlled level in the deluded patients, enabling them to overcome underlying feelings of low self-esteem. This has intriguing similarities to the studies described earlier implicating cognitive abnormalities at a controlled level of processing in schizophrenia. However, as discussed by Young & Bentall (1995), it remains to be seen whether the attributional biases of deluded patients have any connection with the abnormal performance of such subjects on affectively neutral reasoning tasks such as that developed by Huq et al. (1988). Young and Bentall advocated further research in this area, especially the development of standard hypothesis-testing paradigms using emotionally-salient (rather than neutral) material.

2.3 Neuropsychological Studies of Schizophrenia

Introduction

Although recent speculations have been made about the brain systems underlying some of the cognitive processes discussed in Section 2.2 (e.g. Gray et al., 1991), all of the above models had their origins in a predominantly experimental psychological approach to schizophrenia, and paid little attention to the physiological basis of the disorder. However, running in parallel with this work has been a large body of research based in classical neuropsychology. This has involved the administration of batteries of psychological tests thought to be sensitive to the presence of lesions in various cortical and subcortical areas in neurological patients. It is assumed that poor performance by schizophrenic patients on these tasks indicates pathology in the relevant brain areas. Thus the neuropsychological approach attempts to reveal both impaired psychological functions, and their corresponding neural bases.
Workers in this area have often ignored methodological issues such as matching of tasks on difficulty (Serper & Harvey, 1994), and attempts have seldom been made to relate any deficits to particular symptoms or signs. However, since general intellectual decline was recognised as a widespread feature of schizophrenia (e.g. Frith et al., 1991), one way forward has involved the search for specific neuropsychological deficits which occur over and above any generalised impairment. Researchers have focused on memory and executive function, which may be particularly compromised in schizophrenia (e.g. Elliott & Sahakian, 1995; McKenna, 1994). In the following sections, studies of memory and executive function in schizophrenia will be reviewed, and finally the implications of neuropsychological studies for the underlying brain pathology in the disorder will be discussed.

Studies of memory

Many experiments have found memory impairment in schizophrenic patients (see Cutting, 1985 and Straube & Oades, 1992, for reviews of the earlier studies), and there is now some consensus that chronic patients tend to show moderate to severe deficits on memory batteries, whilst acute patients show milder disturbances (McKenna, 1994). As human memory is not a unitary function (e.g. Baddeley, 1990), it is instructive to see whether particular aspects of memory are compromised in schizophrenia. In a recent study, Tamlyn et al. (1992) tested a group of 60 patients with a wide range of signs and symptoms on a large battery of memory tasks. The study is noteworthy because attempts were made to control for poor motivation and the use of neuroleptic and anticholinergic drugs. In line with results from several other groups (e.g. Kolb & Whishaw, 1983), short term memory (as measured by forward digit span and its non-verbal equivalent, the Corsi blocks test) was found to be intact, but widespread impairments in long term memory (LTM) were apparent. These LTM deficits correlated significantly with chronicity of illness, and with the presence of negative signs and formal thought disorder, and remained when general
intelligence was controlled.

**Episodic memory**

Tamlyn et al.'s (1992) investigation of LTM used the episodic-semantic framework proposed by Tulving (1983). According to this, episodic memory records personal experiences within a time-and-place context, whereas semantic memory holds general knowledge about the world without any reference to the context in which it was acquired. Episodic memory is typically tested using recall or recognition of previously presented information, and Tamlyn et al. (1992) found that their patients were impaired on a prose recall task, and on several recognition tests of words and faces. Many other studies have also found impaired recall in schizophrenic patients compared to controls (e.g. Clare et al., 1993; Gold et al., 1992; Goldberg et al., 1989), and there is some evidence from these that the deficit is more pronounced in patients with negative signs. Studies of recognition performance in schizophrenia have produced more equivocal results, with some workers suggesting intact recognition (e.g. Goldberg et al., 1989), and others suggesting an impairment (e.g. Gold et al., 1992). Calev (1984) matched recall and recognition tasks for difficulty, and found that severely disturbed, chronic schizophrenics (of unspecified symptomatology) showed impairments on both types of task (although performance was worse on recall). This is consistent with the findings of Tamlyn et al. (1992) and points to a general episodic memory deficit in schizophrenia.

More recent studies have confirmed and extended these findings. For example, Rizzo et al. (1996) used a recency judgment task to investigate the memory of chronic schizophrenic patients (of unspecified symptomatology) for temporal order. Patients and normal controls were repeatedly presented with six pictures of objects until they could freely recall all the pictures. Four more trials, each using six different pictures, were then presented in the same way, with subjects being required to reach a 100% recall criterion for each set of pictures. At various points, subjects had to
select, from the full set of pictures, the six which had been most recently presented. In accordance with studies showing impaired recall in schizophrenia, the patients required more trials than controls to reach the 100% recall criteria. More interestingly, however, having reached 100% recall, the schizophrenic subjects still gave significantly more incorrect recency judgments than their IQ-matched controls. This suggests a particular impairment in schizophrenia in one aspect of episodic memory, namely the memory for temporal context.

Huron et al. (1995) investigated conscious awareness in recognition memory using schizophrenic patients (of mixed symptomatology) and normal controls. Forty study words were presented to subjects on cards, and later the subjects were given a recognition test in which they had to pick the 40 previously presented words from a list of 80 words. For each word they recognised, subjects were asked to report whether they ‘remembered’ it (i.e. their recognition was accompanied by conscious recollection of some association, feeling or experience that they had when the word was first presented), or whether they simply ‘knew’ that it had been presented (i.e. recognition was accompanied by a feeling of familiarity without any conscious recollection). The schizophrenic patients showed poorer recognition overall compared to controls, and this deficit was related to a lower number of ‘remember’ but not ‘know’ responses. This suggests that patients with schizophrenia have a selective impairment in the ability to consciously recollect previous events, but that feelings of familiarity without conscious recollection are not compromised. As Huron et al. (1995) pointed out, this may explain why, on equally difficult recall and recognition tasks, schizophrenics are worse than controls at both tasks, but are especially poor at recall (Calev, 1984): Recall relies heavily upon conscious recollection of previous events, whereas recognition tasks can be performed to some extent by relying upon feelings of familiarity without conscious recollection.

Taken as a whole, these studies of episodic memory in schizophrenia are
consistent with models discussed earlier which suggest an impairment of conscious, controlled processing in the disorder. The recency judgment study of Rizzo et al. (1996) specifically implicates a problem in the use of contextual information in conscious awareness, an issue which recurred frequently in the studies discussed in Section 2.2. Although Tamlyn et al. (1992) found that episodic memory deficits were related to the presence of negative signs or formal thought disorder, this has not been replicated (e.g. Duffy & O’Carroll, 1994). The two most recent studies discussed above unfortunately made no attempt to investigate the relationship between task deficits and symptomatology, so further research is needed in this area, preferably within the framework of a model which can relate episodic memory deficits to particular symptoms and signs of psychosis. We shall return to the notion of impaired episodic memory in schizophrenia in Chapter 3 of this thesis, where tentative links will be made to Frith’s (1992) neuropsychological model.

**Semantic memory**

As we have already seen, Tulving (1983) proposed that normal memory has a semantic store, which holds all context-independent knowledge such as word meanings and concepts. McKenna (1991) suggested that impairments of semantic memory in schizophrenia could account for delusions because, by definition, these involve the patient ‘knowing’ information which is untrue. He suggested that “delusions consist of an inappropriate laying down of new semantic memories” (ibid., p. 39). Cutting & Murphy (1988) compared schizophrenic and control groups on a multiple-choice test of ‘real-world knowledge’, and found that 75% of their patients were impaired. However, the relationship of this impairment to the presence of delusions was not explored, and it should be noted that in an extension of this study, Cutting & Murphy (1990a) found that schizophrenics were particularly impaired relative to controls on a subtest of social real-world knowledge, (e.g. How do you tell a friend politely that they have stayed too long?). This suggests that these patients may have been showing a specific social deficit, rather than a problem with semantic
memory *per se*. This issue of specific social impairment will be discussed in more detail in Chapter 3 in relation to Frith’s (1992) neuropsychological model.

Tamlyn *et al.* (1992) tested semantic memory in their group of schizophrenic patients using a sentence verification task in which subjects had to classify as true or false 50 statements (e.g. ‘rats have teeth’ or ‘desks wear clothes’). As well as being significantly slower at verification than normals, most schizophrenic patients (especially chronic subjects with formal thought disorder) made multiple semantic errors. Other recent studies (e.g. Clare *et al.*, 1993; McKenna *et al.*, 1994) extended these results, showing that schizophrenics perform worse than normal controls on various other tasks thought to tap semantic memory, such as object naming and vocabulary scales. However, these two studies noted no relationship between semantic memory deficits and symptomatology; in particular, no evidence has emerged to support McKenna’s (1991) prediction of a relationship between delusions and impaired semantic memory. Chen *et al.* (1994) tested chronic schizophrenics and normal controls on a timed semantic categorisation task, in which subjects had to decide whether or not words were members of particular conceptual categories. The words were systematically varied in their degree of relatedness to the category (e.g. borderline relatedness: fish - eel; related words outside the category: fish - crab), and Chen *et al.* found that normals showed the longest reaction time for words of borderline relatedness, whereas schizophrenics were longest in responding to the related exemplars outside a category. The same trend was found when ‘yes’ or ‘no’ responses were analysed. The authors interpreted these results as reflecting an outward shift of the semantic category boundary in schizophrenia, and they suggested that the best way of thinking about ‘overinclusive thinking’ on Payne’s tasks (e.g. Payne *et al.*, 1970; see Section 2.2 of this chapter) was in terms of a disorder of semantic category structure. As we saw earlier, McKenna’s (1994) review of the literature on overinclusion suggested that the phenomenon may be related to the presence of formal thought disorder (FTD). Chen *et al.* (1994) did not find a simple
relationship between patients’ performance on their task and ratings of FTD, but interestingly when a deviance measure was calculated (based upon the degree to which each patient departed from ideal normal performance), this was found to correlate with the presence of formal thought disorder, but with no other ratings of symptoms or signs.

Taken as a whole, these studies suggest that the performance of schizophrenic patients on semantic memory tasks is impaired, and there is (admittedly weak) evidence that the impairment may be linked to the presence of negative signs or formal thought disorder. According to Shallice (1988), neurological patients with semantic memory deficits tend to show one of two possible patterns of impairment, referred to respectively as ‘degraded store’ (i.e. information is lost completely) and ‘impaired access’ (i.e. information still exists, but retrieval is inefficient). Several findings suggest that in schizophrenia, the semantic deficit is of the ‘impaired access’ type. For example, Allen et al. (1993) measured categorical verbal fluency in chronic schizophrenic patients and controls on five separate occasions. The task required subjects to name, within three minutes, as many different exemplars as possible for categories such as animals or fruit. The schizophrenic patients generated significantly fewer words than controls, and produced more words which were outside the specified category. However, comparison of data from the different testing sessions suggested that the schizophrenics had as many words available in semantic memory as controls, but were just inefficient in retrieving those words. More specifically, patients with negative signs were particularly poor at generating words, whereas those with incoherence were more likely to produce inappropriate words. This study has since been extended by Joyce et al. (1996), who employed a letter fluency task (i.e. subjects must generate as many words as possible beginning with a given letter), as well as a category fluency task similar to that used by Allen et al. (1993). Previous research has shown that in normals, category fluency is superior to letter fluency, and Joyce et al. (1996) found that their patients (despite being impaired on both tasks
relative to normals), showed this normal pattern of output. In addition, the patients showed an improvement in category fluency when a cueing technique was used, so like Allen et al. (1993), these workers concluded that schizophrenic patients have impaired access to an intact semantic store.

It should also be noted that most studies of semantic priming in schizophrenia are consistent with suggestions of intact semantic networks in the disorder. Manschreck et al. (1988) and Spitzer et al. (1994) administered lexical decision tasks to groups of controls and thought disordered (TD) and non-thought disordered (NTD) schizophrenics. Subjects had to recognise a word immediately preceded (primed) by either a semantically associated or unrelated word. For the TD schizophrenics, recognition was facilitated by a semantically associated prime significantly more than for the NTD schizophrenic and control groups (who performed similarly). These results suggest that in all schizophrenic patients, the structure of the semantic network is intact, and that formal thought disorder may reflect abnormally high activation, or reduced inhibition, within that network (Manschreck et al., 1988; Spitzer et al., 1994). As we saw earlier, Chen et al. (1994) interpreted the results of their semantic categorisation task in terms of an outward shift of category boundaries in schizophrenia. However, it is possible that their data reflected heightened activation of a normally structured semantic network, so that for a given category, activation had spread to include related non-exemplars within the network. This may have meant that these related non-exemplars lay on the boundary of the zone of activation, leading to the observed difficulties in categorisation of these items by schizophrenic patients.

In summary, the results of studies of semantic memory in schizophrenia are generally consistent with an intact semantic network, but deficits in conscious, controlled processing in the disorder. As we have seen, Allen et al. (1993) found that patients with negative behavioural signs produced fewer words than controls on a
verbal fluency task, whereas those with the positive behavioural sign of formal thought disorder produced inappropriate words. Also, semantic priming studies (Manschreck et al., 1988; Spitzer et al., 1994) imply that the semantic network may be more highly activated in thought disordered patients. Together, these findings suggest that negative signs may be related to a problem with initiating actions (e.g. retrieval of information from memory), and formal thought disorder may reflect an inability to inhibit output resulting from high semantic activation. Initiation and inhibition of thoughts and actions have been linked within Shallice’s (1988) model of executive function, and there is now a large literature suggesting an impairment of executive function in schizophrenia. This will be reviewed shortly.

Procedural memory

Procedural memory is involved in the learning of motor skills on tasks such as the pursuit rotor (where subjects must keep a stylus in contact with a moving target), or jigsaw assembly. These are all tasks on which subjects can show learning without needing to be consciously aware of having encountered the task before (Baddeley, 1990). Clare et al. (1993) found that the rate of learning of their group of schizophrenic patients was comparable to that of a group of normals on these two tasks, and Schmand et al. (1992) also found that their group of schizophrenics (with heterogeneous symptomatology) were as able as non-psychotic controls to learn sequences of button presses on a motor task. Closely related to procedural memory is the concept of implicit memory, which is tested in tasks where retrieval of material from long term memory does not depend on conscious recollection. (In contrast, explicit memory tasks such as recall and recognition require conscious recollection of the learning episode). A common implicit memory task is word stem completion priming, in which subjects are initially presented with a set of words (e.g. DEFENCE), which they rate as pleasant or unpleasant. In the subsequent test phase (in which no reference is made to the earlier presentation of words), word stems (e.g. DEF ---- ) are presented, and the subjects are asked to complete these with the first
word that comes to mind. Intact priming is reflected by the tendency to complete the stems with words previously presented. On this task, Clare et al. (1993) found that their schizophrenic subjects performed the same as normal controls, showing intact priming. This result has since been replicated by Gras-Vincendon et al. (1994), although the area is controversial, with some workers finding impaired word stem completion priming in schizophrenia (e.g. Randolph et al., 1991). Taken as a whole, the findings are consistent with suggestions that automatic processing is relatively intact in schizophrenia; performance deficits only seem to appear on tasks requiring conscious, controlled processing.

The pattern of deficits

The performance of schizophrenic patients on memory tasks suggests that implicit, procedural and short term memory are intact in the disorder, whereas episodic and semantic memory are impaired. Clare et al. (1993) pointed out that this pattern of performance is similar to that observed in the classical amnesic syndrome (which may be found, for example, in patients with Korsakoff’s disease), with the exception that semantic memory is not generally thought to be impaired in amnesia. In a direct comparison of schizophrenic and amnesic patients, Duffy & O’Carroll (1994) confirmed that similarities do exist between the memory profiles of these two groups. The major differences in performance were on the episodic and semantic memory tasks: both groups showed impaired episodic memory relative to standard norms, but the amnesic patients were significantly more impaired than the schizophrenic subjects in this domain. In contrast, the schizophrenics were significantly worse than the amnesics on tests of semantic memory. In general, studies of this type, showing similarities in task performance between schizophrenics and subjects with other brain disorders (whose pathophysiology may, to some extent, be known), have the potential to reveal details about the pathophysiology of schizophrenia. This will be discussed further in the neuropathology section at the end of this chapter.
In summary, the expanding literature on memory in schizophrenia is generally consistent with the notion that psychotic patients have problems on tasks requiring conscious, controlled processing and the use of contextual information. The automatic processing of information outside conscious awareness seems to be relatively intact. Impairments in the conscious recollection of information from long term memory seem particularly pronounced in patients with negative signs (who may have problems with the initiation of actions) and formal thought disorder (who may have difficulty inhibiting inappropriate responses). This general conclusion is compatible with a body of work which has suggested that schizophrenics have problems with the initiation and application of appropriate encoding and retrieval strategies in memory tasks (Koh, 1978). For example, Sullivan et al. (1992) tested recall of the Rey-Osterrieth complex figure in normals, psychiatric controls and schizophrenics (of unspecified symptomatology). They found that both the accuracy of the original copy and the strategy used for copying influenced recall in the controls, but for the schizophrenics, recall errors derived solely from the inadequate organizational strategy used in copying the figure. Difficulties in the initiation and inhibition of responses, and problems with the application of appropriate strategies, often fall under the umbrella term ‘executive dysfunction’. Studies of executive function in schizophrenia will now be reviewed.

Executive function and working memory

Executive function

The term ‘executive function’ is used to describe the processes whereby particular cognitive systems are co-ordinated so that complex tasks can be carried out successfully. It is thought to be required for the planning and execution of complex behaviours, for the generation of strategies, and for the inhibition of particular strategies and behaviours once they become inappropriate (McCarthy & Warrington, 1990). One of the most influential models of normal executive function, the
‘Supervisory Attentional System’ (SAS) was developed by Shallice (see Shallice, 1988). He proposed that highly automatic, routine programmes (schemata) underlie much of our behaviour. These are triggered by external stimuli, and a system of mutual inhibition leads to selection of the most activated schema. In novel situations, or where no external stimulus is available, the SAS is activated. This can bias the activation of the schemata so that, for example, the current activated schema is inhibited. In addition, when no external stimulus is present, it can produce a particular action sequence appropriate to the current situation. Thus, the SAS is crucial for self-generated actions, for the inhibition of inappropriate actions, and for the prevention of perseveration (which can be thought of as repeated execution of a particular automatic schema) (Shallice, 1988).

Patients with lesions of the frontal lobes (especially the prefrontal cortex) often show avolition (i.e. a lack of self-generated actions), inappropriate affect and behaviour, and perseveration. They are also generally impaired on standard tests of executive function (McCarthy & Warrington, 1990), so Shallice (1988) proposed that such patients have an impaired Supervisory Attentional System. As Frith (1992) pointed out, the negative signs of schizophrenia, and the positive behavioural signs such as inappropriate affect and formal thought disorder, are strikingly similar to the behaviours shown by frontal patients. Schizophrenics often show perseverative behaviour too, and Frith & Done (1983) recorded this objectively on a two-choice guessing task. They found that patients with negative signs produced stereotyped sequences with many perseverations (e.g. LLLL) or alternations (e.g. RLRL) when they had to guess whether a random cross would appear on the right (R) or left (L) of a computer screen. Thus, as part of his cognitive neuropsychological model of schizophrenia (which will be discussed fully in Chapter 3), Frith (1992) suggested that schizophrenic patients with negative or positive behavioural signs have impaired function of the SAS, and should therefore perform poorly on standard tests of executive function. They may also have structural or functional abnormalities of the
frontal lobe (Frith, 1992). A brief review of studies investigating the performance of schizophrenics on executive function tasks will now be presented.

Using a single-case design, Shallice et al. (1991) administered a large battery of neuropsychological tasks (assessing IQ, visuospatial function, language, memory and executive function) to five chronic schizophrenics, all of whom showed some negative signs. Three of the patients showed widespread neuropsychological impairment (including poor performance on the executive tasks), whereas the remaining two cases showed quite isolated executive dysfunction. Shallice et al. concluded from this study that, whilst only some schizophrenics have widespread cognitive impairment, all schizophrenic patients (when chronic and showing behavioural signs), have executive deficits. In a group study, Liddle & Morris (1991) tested 43 chronic schizophrenics on a battery of executive function tasks, and found that the group as a whole was impaired on all the tasks compared to standard criteria. When post hoc analyses were used to partial out the effects of illness severity, chronicity and general intellectual decline, many of the executive impairments remained significant. Furthermore, significant relationships were found between Liddle's (1987) three clusters of psychotic features (see Chapter 1), and particular neuropsychological impairments. Specifically, signs of ‘psychomotor poverty’ (i.e. poverty of speech, blunted affect and decreased spontaneous movement) correlated with slowness of mental activity (including slowness of generating words on a word fluency task), whereas measures of ‘disorganisation’ (i.e. inappropriate affect and formal thought disorder) were related to the inability to inhibit an inappropriate response on the Stroop task. Scores for ‘reality distortion’ (i.e. hallucinations and delusions) were not related to performance on the executive tasks. These findings are consistent with Frith's (1992) suggestion that the function of the SAS is impaired in patients with negative or positive behavioural signs of schizophrenia. They also complement the verbal fluency data of Allen et al. (1993) discussed earlier. These workers suggested that schizophrenics with negative signs have problems in the initiation of actions (e.g. retrieval
of words from memory), whilst those with incoherence have difficulties with the inhibition of inappropriate output.

Two specific components of executive function, namely planning and set-shifting ability, have been extensively researched in schizophrenia. A commonly used test of planning ability is the Tower of London (Shallice, 1982), in which subjects must devise a plan to move coloured balls which are lined up on a stick, in order to achieve a particular goal configuration on three adjacent sticks. The task has a range of difficulties, requiring only two moves to achieve the goal configuration at the simplest level, while more difficult levels require up to five moves. Morris et al. (1995) tested schizophrenic patients and normal controls on a computerised version of this task, and found that patients required significantly more moves to solve the problems, and solved significantly fewer problems than controls in the minimum possible number of moves. This poor planning performance was independent of the slower motor speed of the patients, so Morris et al. concluded that there is a specific planning deficit in schizophrenia. These results suggest that the patients had problems with the generation of actions, and may therefore have had impaired function of the Supervisory Attentional System. However, contrary to predictions from Frith's (1992) model, the planning deficit in this study was unrelated to the presence of negative signs. Further studies of planning in schizophrenia are needed to clarify the relationship of deficits to signs and symptoms.

A test of set-shifting ability is the Wisconsin Card Sorting Test (WCST), in which subjects sort patterned cards along one of three dimensions (shape, number, colour) according to a rule that one of the dimensions is correct. The tester gives feedback, and when criterion is reached, the sorting rule is changed without the subject being told. The previous sorting strategy now receives negative feedback, so the subject must change strategy (‘shift set’) to sort along a new dimension. Executive dysfunction is reflected in a tendency to continue to sort along the
previously correct dimension, and the number of perseverations on the task is taken as an index of prefrontal impairment. Some workers (e.g. Weinberger et al., 1986) have suggested that the WCST provides a relatively specific challenge of dorsolateral prefrontal cortex (DLPFC), although this specificity has been questioned (e.g. David, 1992). Studies using the WCST have commonly found that schizophrenic patients (especially when chronic), show significantly more perseverations than controls (e.g. Weinberger et al., 1986), and it is generally agreed that this impairment is disproportionate to overall intellectual decline (Elliott & Sahakian, 1995). The relationship between WCST perseverations and symptomatology is more controversial, however: Many studies have found a correlation with scores for negative behavioural signs (e.g. Brown & White, 1991; Butler et al., 1992), or with scores for negative and positive behavioural signs (e.g. Rosse et al., 1991), but others have either found no relationship, or a correlation between perseveration and scores for positive symptoms (e.g. Morrison-Stewart et al., 1992). As a whole, these data provide some support for Frith’s (1992) model, but one possible reason for the discrepancies is that the WCST is a very complex task, which taps many cognitive functions in addition to executive function (Keefe, 1995). If impairments in any of these ‘lower level’ processes were correlated with, say, the presence of positive symptoms, then a relationship between WCST score and positive symptoms might be observed, irrespective of executive function. Furthermore, a number of different versions of the WCST have been used in the schizophrenia literature (e.g. in one version, subjects are told when the sorting rule changes), so variation in results across studies may partly reflect methodological differences. Future research on set-shifting in schizophrenia should use simpler tasks where possible, and in fact the first study in this thesis uses a simple reversal learning paradigm, rather than the WCST, as a measure of set-shifting ability (see Chapter 4). A recent experiment by Elliott et al. (1995) used a computerised set-shifting task which began with a simple reversal learning stage before becoming more difficult. Elliott et al. found that many of their chronic schizophrenic patients (all of whom had negative signs) failed at this first stage, showing a tendency to perseverate on the
previously correct stimulus. In general, the performance of the schizophrenics on this task was similar to (but worse than) that of patients with frontal lobe excisions, in that they showed a large impairment in the ability to shift set onto a new stimulus dimension.

**Working memory**

Another psychological function thought to be mediated by the prefrontal cortex is ‘working memory’, which enables subjects to retain and use stimulus information for a short period after the stimulus itself has disappeared (Baddeley, 1986). Baddeley’s model of working memory incorporates two ‘slave systems’, the articulatory loop and visuospatial sketch pad, which are specialised for the short term retention of phonological and visual information respectively. The operation of these systems is co-ordinated by a limited capacity ‘central executive’, which has access to long term memory. The central executive can be thought of as being very similar in form and function to Shallice’s (1988) Supervisory Attentional System, and the poor performance of schizophrenic patients on executive function tasks may reflect an impaired central executive, with largely intact slave systems (Fleming et al., 1994). This is consistent with the ‘output’ models of auditory hallucinations discussed earlier, which suggest that patients’ ‘voices’ may arise from inner speech within an intact articulatory loop. It also fits with the findings of Tamlyn et al. (1992), who found that short term memory, as assessed by forward digit span, was intact in their schizophrenic patients. This task requires only the retention of phonological information, so is likely to rely largely upon an intact articulatory loop. In contrast, tasks such as the Tower of London or WCST, where the subject has to retain information *and* use it to perform cognitive operations, are likely to tax the central executive component of working memory. Backwards digit span, where the subject must remember a number sequence and then operate upon it in order to say it backwards, is likely also to tap the central executive, and has been found to be impaired in schizophrenic patients (e.g. Goldberg et al., 1993).
Goldman-Rakic (1987) emphasised the importance of working memory for holding information ‘on line’ and operating upon it over a short delay period. Much of her work has investigated spatial working memory in non-human primates, as measured by performance on various delayed-response (DR) tasks. In a typical DR paradigm, an object is placed in one of several wells in full view of the subject, and the wells are covered over. After a delay, the subject must remove a cover and retrieve the object from the correct well. This requires that he holds ‘on line’ the spatial representation of the object, and then uses this representation to make a goal-directed reach to the correct position. Many studies (reviewed by Goldman-Rakic, 1987) have implicated the dorsolateral prefrontal cortex in human performance of this kind of task. As schizophrenic patients show poor performance on tests of executive function, they should also be impaired on DR paradigms, and in fact several studies have now confirmed this prediction. For example, Park & Holzman (1992) tested schizophrenics (of unspecified symptomatology), and bipolar controls, on oculomotor and haptic spatial DR tasks. In the experimental condition, targets were presented in a particular position and then removed; after a delay of 5 or 30 seconds, the subjects had to make a motor response to the previous position of the target, so had to hold its spatial position ‘on line’ during the delay. In a condition controlling for the sensorimotor component of the task, the target remained present for the whole delay period, so the subjects’ response was not dependent upon working memory. The schizophrenics made more errors than controls only in the experimental conditions, and the deficit was more pronounced with longer delay periods. Park and Holzman concluded from this that schizophrenic patients have a modality-independent impairment in spatial working memory. This result has since been replicated using a similar paradigm (e.g. Spitzer, 1993), and a pen-and-paper analogue (Keefe et al., 1995). Spitzer (1993) found a relationship between number of errors and the presence of formal thought disorder in his patients, which is clearly consistent with those theories (e.g. Frith, 1992) suggesting that an impairment of executive function underlies the behavioural signs of schizophrenia.
Partiot et al. (1992) administered several delayed reaction tasks to a group of schizophrenic subjects (of unspecified symptomatology), a group of patients with vascular ischaemic lesions of the prefrontal cortex, and age-matched normal subjects. In line with results discussed above, both patient groups were impaired relative to controls on a DR task. In addition, the schizophrenic and frontal patients showed deficits on a spatial reversal (SR) task. As we saw earlier, reversal learning is similar to the WCST in that subjects must establish a response set to a particular position, and then shift set to a new position when the response rule changes. When Partiot et al. (1992) investigated correlations between task performance for the schizophrenic subjects, they found that scores on both DR and SR tasks correlated with the subjects’ performance on a standard WCST. These data therefore link together the working memory and set-shifting areas of executive function research, and suggest that impairments in the holding 'on line' of internal representations and the shifting of response set in schizophrenia may both reflect dysfunction of the prefrontal cortex.

In summary, much evidence now points towards an impairment in executive processes in some schizophrenic patients. As predicted by the model of impaired SAS function, the presence of negative or positive behavioural signs seems to be related, respectively, to problems in the initiation of goal-directed action or the inhibition of inappropriate action. More specifically, patients with these signs show impairments in set-shifting ability and, (despite little symptom-specific research using the DR task), may have a particular problem with the holding ‘on line’ of representations for use in subsequent cognitive operations. As with the other areas of research in schizophrenia, these data are consistent with deficits in conscious controlled processing in the disorder. As Fleming et al. (1994) pointed out, the data can also be used to explain the performance of schizophrenic patients on various other tasks. For example, we saw in Section 2.2 that schizophrenics tend to score poorly on the ‘memory load’ version of the CPT test of sustained attention, where subjects must respond, say, to the number ‘7’ only if preceded by a ‘3’ (Nuechterlein et al., 1994).
In terms of the working memory model, this task requires a subject to continuously hold ‘on line’ particular information to be used in guiding his response. The poor performance of schizophrenics on the task is therefore compatible with executive dysfunction in these subjects.

Relations to neuropathology

As many of the tasks discussed above have been used in studies of patients with localised brain lesions, the performance of subjects with schizophrenia can be used to make inferences about the neuropathology of the disorder. However, it is important to bear in mind that similar poor performance by two patients on a given task does not necessarily mean that they have the same brain pathology. For example, on complex tasks involving many cognitive processes, subjects may have different cognitive impairments (mediated by different brain structures), yet both show a task deficit. Thus, any inferences from neuropsychology are necessarily speculative, and should be supported by converging evidence from as many other research areas as possible (e.g. brain imaging). Using this approach, it is possible to identify the most likely areas of neuropathology in schizophrenia, and to attempt to relate these to specific signs and symptoms.

Fronto-posterior brain systems

The neuropsychological studies of executive function and working memory discussed above suggest that the prefrontal cortex (PFC) may be damaged or dysfunctional, especially in those schizophrenic patients showing negative or positive behavioural signs. Examination of the brain imaging literature reveals little evidence for structural abnormalities of the frontal lobes in schizophrenia, but there are data suggesting an association between the presence of behavioural signs and impaired function of the PFC. For example, Weinberger et al. (1986) measured the regional cerebral blood flow (rCBF) of schizophrenic patients (many of whom had negative
signs) and normal controls as they performed the Wisconsin Card Sorting Test. In carrying out this task, controls activated the dorsolateral prefrontal cortex (DLPFC), whereas patients showed a significantly lower activation of this region. In a similar study, Andreasen et al. (1992) measured the rCBF of chronic patients and normals as they performed the Tower of London planning task. Controls activated the left medial frontal cortex during task performance, but the schizophrenics showed significantly less activation of this region; the reduction of activation was correlated with the presence of negative behavioural signs, but not positive symptoms.

A criticism often levelled at such activation studies is that the relation between negative signs and reduced frontal activation may simply reflect the generally poor performance of patients with negative signs on any task. Liddle et al. (1992) avoided this problem by carrying out functional imaging of chronic schizophrenic patients in the resting state. They examined the relationship between rCBF and symptomatology using Liddle’s (1987) three-syndrome framework discussed in Chapter 1, and found that each of the three syndromes was associated with a specific pattern of blood flow: Psychomotor poverty was associated with decreased rCBF in the left dorsolateral PFC, medial PFC and parietal cortex, and with increased rCBF in the caudate nucleus. This is consistent with the findings of Weinberger et al. (1986) and Andreasen et al. (1992) discussed above. In addition, we saw earlier that the negative signs characteristic of the psychomotor poverty syndrome are associated with difficulties in the production of self-generated actions; Liddle et al.’s data are therefore consistent with several imaging studies of normals which implicate the left DLPFC in the self-generation of actions (e.g. Frith et al., 1991a, b). Scores for disorganisation (i.e the positive behavioural signs of inappropriate affect and formal thought disorder) were found by Liddle et al. (1992) to be associated with decreased rCBF in the right orbital PFC, and increased rCBF in the right anterior cingulate gyrus and dorsomedial thalamic nuclei. We saw earlier that the positive behavioural signs of psychosis are often associated with an inability to inhibit inappropriate
responses on neuropsychological tasks, and in an imaging study of normals performing the Stroop task, Pardo et al. (1990) found high activation of the right anterior cingulate when subjects had to suppress irrelevant responses. As it is widely held that the orbital PFC also plays an important role in the suppression of irrelevant behaviour (e.g. Fuster, 1989), one possibility is that the relatively low activation of right orbital PFC in Liddle et al.’s patients means that many inappropriate actions are not inhibited; as a result, the anterior cingulate is highly activated as the patient tries in vain to suppress these behaviours.

Finally, scores for the syndrome of reality distortion were found by Liddle et al. (1992) to correlate positively with rCBF in the left parahippocampal gyrus and striatum. This fits with a large body of post-mortem and imaging data (e.g. Bogerts et al., 1985; Suddath et al., 1989) implicating the left temporal lobe in the pathogenesis of schizophrenia. More specifically, it is consistent with several imaging studies exploring the physiology of auditory hallucinations. For example, Cleghorn et al. (1992) found that hallucinating patients showed activity in the left superior temporal gyrus (STG), and in a study using single photon emission computerised tomography (SPECT), McGuire et al. (1993) found auditory hallucinations to be associated with activity in Broca’s area (left inferior frontal gyrus), and (to a lesser extent), the left STG. In a recent imaging study of normals, McGuire et al. (1996) investigated the brain activity associated with inner speech or imagining the sound of someone else speaking. They found that inner speech activated Broca’s area, while imagining the sound of someone else speaking additionally activated the left STG, as well as left premotor cortex and supplementary motor area (SMA). Together with the imaging studies of auditory hallucinations, these data are consistent with those models suggesting that patients’ ‘voices’ arise from internally generated speech or thoughts (see Section 2.2).

Liddle et al.’s (1992) study of resting rCBF in schizophrenic patients is
important as it provides a physiological basis for the three-syndrome model, and a rationale for some of the neuropsychological test data. As discussed by Elliott & Sahakian (1995), it is also consistent with clinical data from various types of neurological patient. For example, psychomotor retardation and loss of motivation and initiative (signs of Liddle’s psychomotor poverty syndrome) are known to occur in patients with lesions of the DLPFC, whereas inappropriate social behaviour (part of the disorganisation syndrome) is often related to OFC damage. Delusions and hallucinations (Liddle’s reality distortion syndrome) are sometimes experienced by patients with temporal lobe epilepsy. Liddle et al.’s (1992) data are also important as they highlight the possible role of orbitofrontal cortex in the positive behavioural signs of schizophrenia. Thus, whilst many of the neuropsychological studies have emphasised DLPFC dysfunction in schizophrenia, it is important to bear in mind that the OFC may be just as relevant to the pathogenesis of the disorder. A study by Seidman et al. (1992) explored DLPFC and OFC function in schizophrenic patients and normal controls, using neuropsychological tasks thought to be specific markers of these regions. The putative test of OFC function was a standard multiple-choice odour identification test, and the WCST was used as a challenge of the DLPFC. Compared to controls, the schizophrenics were significantly impaired on both tasks, but unlike controls, task performance was uncorrelated within the schizophrenic group. The authors suggested that this reflected distinct subtypes of frontal pathology in schizophrenia, and on the basis of the test results, they divided their schizophrenic sample into patients showing an isolated DLPFC deficit, those with an isolated OFC deficit, and those with a generalised prefrontal impairment. Unfortunately, however, relationships between performance deficits and symptomatology were not explored; future studies should examine whether, for example, neuropsychological markers of OFC dysfunction are related to the presence of positive behavioural signs.

Liddle et al.’s (1992) rCBF data are also significant in that they show how the clusters of psychotic features reflect abnormal function in a distributed neuronal
network. Thus, whilst prefrontal cortex plays a part in the pathogenesis of some signs and symptoms, the structures to which PFC projects are also closely involved. One possibility is that a primary abnormality in subcortical structures leads to prefrontal hypofunction and consequent poor performance of patients on executive function tasks. In support of this was a study carried out by Raine et al. (1992), in which schizophrenic and control subjects were given Magnetic Resonance Imaging (MRI) and a battery of neuropsychological measures including DR and WCST tasks. Although the schizophrenics had smaller prefrontal areas than controls on MRI, and were relatively impaired on the executive tasks, no relationship was found between the structural and task measures. The authors speculated from this that executive dysfunction may have been secondary to structural damage at subcortical sites.

Research into the abnormal function of distributed networks in schizophrenia has concentrated mainly on fronto-striatal and fronto-temporal systems. Alexander et al. (1986) showed that the normal brain contains a number of parallel, segregated cortico-striatal 'loops' linking the prefrontal cortex, striatum and thalamus. At least five loops have been identified which pass through the basal ganglia from PFC, returning to the PFC via the thalami. They subserve motor, oculomotor and cognitive functions, and it is possible that abnormalities in the functions or interconnections of components of these loops may give rise to particular signs and symptoms of schizophrenia (Robbins, 1990). Two of the loops have particularly strong contributions from the dorsolateral and orbital regions of PFC, and disruption of these loops could underlie the psychomotor poverty and disorganisation syndromes respectively (Pantelis & Brewer, 1995). There is good evidence for fronto-striatal dysfunction in schizophrenia; for example, we saw earlier that Liddle et al.’s (1992) imaging study of patients in the resting state showed that psychomotor poverty was associated with decreased blood flow in prefrontal cortex and increased flow in the caudate nucleus. In addition, a functional imaging study by Buchsbaum et al. (1992) found reduced metabolism in both the prefrontal cortex and basal ganglia of neuroleptic-naive
schizophrenic patients performing the Continuous Performance Test (CPT). Pantelis et al. (1992) reviewed the similarities between patients with schizophrenia and those having subcortical dementias due to disorders of the basal ganglia or thalamus. Both types of patient often show a co-occurrence of movement, affective and cognitive disorders, and as all of these domains are likely to be affected by the integrity of the cortico-striato-thalamic loops, dysfunction of such loops would provide a parsimonious explanation for the co-occurrence of these signs in schizophrenia. Pantelis et al. (1992) concluded from their review that schizophrenia may be characterised by a degeneration of subcortical areas, leading to a loss of activating subcortical afferents to frontal cortex, and secondary frontal hypofunction.

Much research has also concentrated on fronto-temporal systems in schizophrenia, especially the connectivity between prefrontal cortex and the limbic system. We saw earlier that Liddle et al. (1992) found an association between the presence of hallucinations and delusions and increased blood flow in the para-hippocampal gyrus. The limbic system is also implicated by studies of memory in schizophrenia (e.g. Tamlyn et al., 1992; see earlier sections of this review), which suggest a neuropsychological profile similar to that of the classical amnesic syndrome, which is found, for example, in patients with septo-hippocampal lesions. In an influential study, Weinberger et al. (1992) used PET and MRI to study 9 pairs of monozygotic twins discordant for schizophrenia. Results showed that in the affected twins, there was a significant association between reduced activation of the prefrontal cortex during performance of the WCST, and reduced volume of both right and left hippocampi. No such relationship was found for the unaffected twins. The authors interpreted these data as reflecting disrupted connectivity between prefrontal cortex and limbic structures in schizophrenia.

Consistent with a fronto-temporal account of schizophrenia was a PET functional imaging study carried out by Frith et al. (1995), in which chronic,
medicated schizophrenic patients and normal controls performed a verbal fluency task. The authors equated the number of responses of patients and controls by using a slow, paced version of the task, in which subjects had to produce a word beginning with a given letter every 5 seconds. This overcame the problem discussed earlier, in which reduced prefrontal activation by chronic patients on such tasks may simply reflect their generally poorer performance. Both schizophrenic and control groups showed similar patterns of rCBF in that verbal fluency activated the left DLPFC, anterior cingulate and thalamus, and led to reduced activation in the posterior cingulate cortex and right superior temporal gyrus. This was very similar to the pattern of activation found in an earlier study of normals performing unpaced verbal fluency (Frith et al., 1991a). However, Frith et al. (1995) found a difference in activation between patients and controls in the left superior temporal gyrus, where schizophrenics failed to show the normal reduction of activity during task performance. Interestingly, no difference in activity was found between subgroups of schizophrenic patients formed using Liddle’s three-syndrome framework. The study shows that on a task in which all subjects perform similarly in terms of speed and strategy, all schizophrenics (even those with pronounced negative signs) are able to activate the left DLPFC. It may therefore be the case that many earlier studies showing reduced prefrontal activation by schizophrenics on tasks such as the WCST, were indeed reflecting the poorer performance of the patients (Frith et al., 1995).

Frith et al. (1995) proposed that in their study, the relatively higher left superior temporal activation in the schizophrenics compared to controls reflected reduced fronto-temporal functional connectivity in the patients. In their earlier imaging study of verbal fluency in normals (Frith et al., 1991a), they suggested that the increase in activity in left DLPFC, coupled (i.e. correlated) with reduced rCBF in left superior temporal cortex, corresponded to a strategic selection and inhibition process, whereby spreading activation amongst word representations in superior temporal cortex was both initiated and controlled, so that activated (but unsuitable)
words were inhibited. This suggests that schizophrenic patients with all types of signs and symptoms may show a failure of inhibitory processes in temporal regions due to impaired fronto-temporal connectivity. Frith et al. (1995) proposed that this may underlie the abnormal performance of schizophrenics with negative and positive behavioural signs on standard (unpaced) verbal fluency tasks (e.g. Allen et al., 1993). It may also underlie the enhanced semantic priming found in patients with formal thought disorder (see the earlier discussion of studies by Manschreck et al., 1988 and Spitzer et al., 1994). Frith et al. (1995) went on to suggest how reduced inhibition in posterior brain regions could also explain certain hallucinations and delusions; this explicitly relates to Frith’s (1992) model of these symptoms, and will be discussed fully in Chapter 3. Thus, Frith et al. (1995) concluded that although different signs and symptoms may be associated with different patterns of resting cerebral blood flow (Liddle et al., 1992), it is possible that a core abnormality of fronto-temporal connectivity is present in all schizophrenic patients. The results of Frith et al.’s (1995) study have now been replicated several times (e.g. Dolan et al., 1995; Grasby et al., 1994; Yurgelun-Todd et al., 1995); if it is true that a disturbance of fronto-temporal functional connectivity underlies both the negative and positive features of psychosis, then it is also possible (although as yet unproven) that this reflects a disruption of the projections between frontal and temporal areas (McGuire & Frith, 1996).

**Dopamine systems**

Disordered functional connectivity is likely to affect neurotransmitter systems, so fronto-striatal and fronto-temporal accounts of schizophrenia enable us to incorporate theories of abnormal dopamine transmission, which have been popular for many years (see Davis et al., 1991, for a review). As originally formulated, the ‘dopamine hypothesis’ of schizophrenia held that the disorder arose from hyper-dopaminergic activity. This was supported by data showing that dopamine agonists exacerbated the illness, and that the clinical efficacy of neuroleptic drugs correlated
strongly with their ability to bind to dopamine D₂ receptors (Seeman, 1980). In addition, much post-mortem evidence suggested an abnormally high density of D₂ receptors in the striatum of neuroleptic-naive patients (see Davis et al., 1991). Recent research has led to modifications and extensions of the hypothesis. For example, it has recently been proposed that excess dopamine activity specifically in the mesolimbic dopamine system is related to psychosis; this system is therefore thought to be the locus of action of typical neuroleptics (Davis et al., 1991). The mesolimbic system has its cell bodies in the ventral tegmental area (A10) of the midbrain, and projects to the amygdala and nucleus accumbens. Notably, Gray et al. (1991) suggested that the projections to accumbens from A10 interact with those from the septo-hippocampal system to accumbens, and that the resulting dopamine release in accumbens underlies the absence of latent inhibition (LI) in some psychotic patients (see Section 2.2 of this thesis).

A simple model of mesolimbic hyperdopaminergia in schizophrenia cannot, however, explain all the experimental data. For example, some studies (e.g. Davidson & Davis, 1988) have shown that plasma concentrations of the dopamine metabolite homovanillic acid (HVA) are lower in chronic schizophrenics (with predominantly negative signs) than normal controls, and low HVA concentrations have been found to be associated with prefrontal hypofunction in imaging studies (e.g. Weinberger et al., 1988). It has thus been suggested that hypodopaminergia in the mesocortical dopamine system (which projects from A10 to prefrontal cortex, nucleus accumbens and septum) may be related to the negative behavioural signs of schizophrenia, whereas mesolimbic hyperdopaminergia is associated with positive symptoms (Davis et al., 1991). One possibility is that in schizophrenic patients, the effects of primary impairments in functional connectivity between prefrontal cortex and striatal and temporal lobe structures are exacerbated by abnormalities in dopaminergic transmission. As Robbins (1990) suggested, the dopaminergic abnormalities may actually be caused by fronto-striatal dysfunction, or they may be due to the effects
of stress. Either way, they are likely to be modulators of psychotic symptomatology rather than the primary cause (Davis et al., 1991).

**Neurodevelopmental models**

Finally, the evidence implicating disrupted fronto-temporal and fronto-striatal functional connectivity in schizophrenia is consistent with a neurodevelopmental model of the disorder proposed by Breslin & Weinberger (1990). These authors reviewed research suggesting that the anatomical and functional characteristics of prefrontal areas and their connections with limbic system structures continue to develop up to early adulthood in animals and humans. For example, synaptic density in the prefrontal cortex has been found to peak at the age of 2 years in human infants, and then decrease up to the age of 16 years. This led Feinberg (1982-83) to suggest that problems with synaptic elimination in PFC during adolescence may explain the common onset of schizophrenia around this age. Research has also shown that myelination of PFC-temporal lobe connections, and changes in density of dopaminergic innervations to PFC, continue into adolescence for both animals and humans (see Breslin & Weinberger, 1990, for a review). Thus, a full functional interaction between frontal and posterior brain regions may only start to operate in normals in early adulthood. Breslin and Weinberger pointed out that structural neuroimaging studies of schizophrenic patients suggest that abnormalities such as ventricular enlargement and reduced hippocampal volume are present at the first psychotic episode, and show no correlation with measures of illness duration. On the basis of all this evidence, they therefore proposed that schizophrenia is associated with early (perhaps congenital) subtle neuropathology in frontal and temporal regions, but that this only manifests as psychotic symptomatology when certain anatomical and functional maturational processes fail to occur normally in PFC and its connections with temporal regions.

To date, this hypothesis remains unproven and, as we shall see in later
chapters, Murray et al. (1992, p. 326) have suggested that the picture may be more complex, with a subgroup of schizophrenic patients showing a particularly severe “neurodevelopmental” form of the illness (characterised by early onset, cognitive impairment, a predominance of negative signs and poor outcome), while others have “adult-onset” schizophrenia, characterised by generally better prognosis, periods of remission, and positive as well as negative symptoms. Murray et al. proposed that whilst the ‘adult onset’ cases may have experienced subtle neurodevelopmental abnormalities (e.g. in neurotransmitter systems), the true ‘neurodevelopmental’ cases may show grosser structural abnormalities as the result of aberrant brain development during fetal and neonatal life.

2.4 Conclusions

Many years of schizophrenia research within both the experimental psychological and neuropsychological traditions, have revealed a number of cognitive deficits which cannot be explained away in terms of generalised intellectual impairment. The often contradictory findings reflect the heterogeneity of the disorder, and only since the comparatively recent adoption by several research groups of the ‘symptom approach’ (e.g. Frith, 1992; Persons, 1986), has it been possible to begin to rationalise all the results in terms of the clinical presentation of schizophrenic patients. Research using a combination of carefully designed neuropsychological tasks and sophisticated brain imaging techniques now has the potential to elucidate the specific cognitive impairments associated with particular psychotic features, and to relate these to underlying brain systems.

The studies reviewed in this chapter have revealed several common themes, suggesting that one cognitive deficit may be associated with all the signs and symptoms of psychosis. Callaway & Naghdi (1982) proposed that schizophrenics have reduced controlled processing capacity, with intact automatic processing, and
although their own data were difficult to interpret, many separate studies have supported their conclusion. For example, in the areas of memory and executive function, patients (especially those showing behavioural signs) are impaired on resource-demanding tasks requiring the self-generation or inhibition of actions in conscious awareness. In contrast, tasks relying on automatic processes, (e.g. tests of implicit memory), are often performed normally by schizophrenic subjects. Studies investigating the use of spatial and temporal context also suggest that schizophrenics (especially those with behavioural signs), have particular problems with the integration of contextual information in conscious awareness. Tasks which involve the influence of context (or previously learnt information) on automatic processing are often performed normally by schizophrenic patients (e.g. Done & Frith, 1984), suggesting that the automatic ‘influence of stored memories of regularities of previous input’ may be relatively intact in schizophrenia (contrary to Hemsley’s (1994) proposal). Impairments in the conscious integration of context may be associated with certain positive symptoms as well as behavioural signs. For example, Bentall & Young (1996) suggested that a problem with the strategic integration of information over time may underlie the maintenance of delusions, as patients may have difficulty in correctly weighing evidence which contradicts their false beliefs.

Studies of neuropathology implicate distributed neuronal networks involving the prefrontal cortex and its connections to striatal and limbic structures. Whilst each of Liddle’s (1987) three syndromes of psychotic features is associated with a distinct pattern of resting cerebral blood flow (with, for example, an association between reduced prefrontal activation and certain behavioural signs), it is possible that a core deficit in fronto-temporal (e.g. Frith et al., 1995) and/or fronto-striatal (e.g. Robbins, 1990) functional connectivity underlies all the symptoms and signs of psychosis. We might speculate that the severity of disconnection, and/or modulatory effects of dopamine, underlie the clinical heterogeneity, so that negative signs, for example, may reflect a marked loss of fronto-posterior connectivity, with the patient behaving
in many ways like a person with frontal lobe lesions (Frith, 1994). In contrast, positive psychotic symptoms may reflect only subtle functional disconnection. In the next chapter, we shall discuss the cognitive neuropsychological model developed by Frith (1987; 1992). This is consistent with the studies already reviewed in that it emphasises deficits at the output rather than input stage of processing. It has the potential to account for the poor performance of schizophrenic patients on tasks requiring controlled processing and contextual integration, and is also compatible with a neuropathological account which emphasises fronto-posterior disconnection.
CHAPTER THREE

FRITH’S MODEL OF SCHIZOPHRENIA

3.1 Explaining the signs and symptoms of psychosis

Several of the psychological models presented in Chapter 2 were able to account for particular signs and symptoms of schizophrenia, but none could explain all the features of psychosis in terms of one underlying cognitive deficit. However, a model initially presented by Frith in 1987, and since elaborated (e.g. Frith, 1992; 1994), can account for most signs and symptoms in terms of impairment in a single cognitive system, that involved with the representation of mental states (‘meta-representation’). Frith’s contention is that, broadly speaking, the range of psychotic features reflects variation in the degree of severity of this cognitive impairment, with the most severe deficit manifesting as negative or positive behavioural signs, and less severe deficits giving rise to positive symptoms. As the model has developed over the course of several years, it will be presented in this first section in the same way as it appeared in the literature. This means that the ‘unifying theme’ of impaired meta-representation may only become apparent once most of the model has been described. Firstly, we shall examine Frith’s account of particular behavioural signs (which has already been mentioned in the executive function section of Chapter 2), and then his explanations of certain types of delusion and hallucination will be presented.
Negative and positive behavioural signs

Theory and empirical support

Frith (1987) argued that normally there are two routes by which actions can occur: they can be a response to an external stimulus, or can be self-generated (what Frith called a ‘willed action’). By the first route (labelled A in Figure 3.1), the subject perceives a stimulus and, in consultation with long-term memory (LTM), forms an intention to act (‘the stimulus intention’) on the basis of the stimulus meaning.

Figure 3.1. The two routes to action (adapted from Frith, 1992; Chapter 4).

For the second route (labelled B1 in Figure 3.1), the subject has a plan or goal, and in consultation with LTM forms a ‘willed intention’ to produce an action appropriate to that goal. Thus, route A leads to a stimulus-driven action, and route B1 leads to a willed action. If an ongoing stimulus intention is inappropriate to the current situation or conflicts with a willed intention, it can be inhibited by route B2.
We saw in Chapter 2 that much evidence points towards a schizophrenic impairment in the generation of output, whilst input processing may be relatively intact. In line with this, Frith (1987) proposed that route A (Figure 3.1) is largely intact in schizophrenic patients, whereas patients with negative behavioural signs are impaired in the generation of willed actions by route B1. This deficit may underlie the typical signs of poverty of action, speech and thought, flat affect and social withdrawal. It may also explain the observation that patients with poverty of speech are able to respond to questions (i.e. external stimuli), but usually give short answers without any (self-generated) extra comments (Frith, 1992). As we saw in Chapter 2, some of the negative signs have been investigated neuropsychologically, and results generally support Frith's proposal that they are associated with problems in the initiation of willed actions. For example, in their verbal fluency experiment, Allen et al. (1993) found that schizophrenic patients with negative features were impaired in the generation of words, and this was ascribed to a problem with word retrieval from an intact semantic store. Similarly, Liddle & Morris (1991) showed that the psychomotor poverty syndrome was associated with slowness of mental activity, including word generation on a verbal fluency task. Braun et al. (1991) found that chronic hospitalised schizophrenics (who may be assumed to have had some negative signs) had an impaired ability to express facial affect when given a verbal command, but were relatively less impaired in copying the affect demonstrated by a model. This effect was independent of non-affective bucco-facial dyspraxias such as grimacing and dyskinesia, and was not related to differences in task difficulty. As copying of demonstrated facial affect may be more 'stimulus-driven' than generation of affect on the basis of verbal cues, these data are consistent with Frith's hypothesis.

On the basis of Figure 3.1, Frith (1992) went on to propose that positive behavioural signs such as incongruous behaviour and certain forms of incoherent speech may reflect a deficit in route B2, so that inappropriate stimulus-elicited actions are not inhibited. This hypothesis can explain, rather specifically, phenomena
such as ‘distractible speech’ (where, for example, the patient may stop mid-sentence and comment about a nearby object or person), and ‘clanging speech’ (where sounds of words rather than their meanings govern word choice) (Frith, 1992). Some of the neuropsychological studies discussed in Chapter 2 suggested a relationship between positive behavioural signs and an inability to inhibit inappropriate responses, so are consistent with Frith’s hypothesis. For example, Allen et al. (1993) found that patients with these signs tended to produce inappropriate words on a verbal fluency task, and Liddle & Morris (1991) found that the disorganisation syndrome was associated with poor performance on the Stroop task and a modified version of the Wisconsin Card Sorting Test (WCST). In a Stroop task, the subject has to name the ink colour (e.g. blue) of a printed colour word (e.g. RED), so must suppress the tendency to respond to the meaning of the stimulus word. In card sorting tasks, the subject must change sorting strategy when informed that the previous response was incorrect. The task therefore requires inhibition of a prepotent response to a particular stimulus dimension.

Frith (1992) suggested that impairments in the willed action system may also explain stereotyped behaviour, where the patient repeats a previous response or a previous sequence of responses. In the domain of speech, this may manifest as ‘poverty of content’ if the patient produces speech but says little of any note because of frequent repetition of phrases. In terms of Figure 3.1, stereotyped behaviour may reflect impairments in routes B1 and B2. For example, the patient may utter a word or phrase, but then fail to generate any more output (failure in route B1); the utterance may itself then act as a stimulus, so that with failure of inhibition (route B2), repetition occurs. As we saw in Chapter 2, perseverative responding on set-shifting tasks such as the WCST is often associated with the presence of positive or negative behavioural signs (e.g. Brown & White, 1991; Rosse et al., 1991), and Frith & Done (1983) elicited stereotyped responding from chronic patients with negative signs on a two-choice guessing task. In the latter study, Frith and Done found that
chronic schizophrenics with intellectual impairment produced perseverations, so that when asked on several trials to predict whether a random cross would appear at the left (L) or right (R) of a computer screen, they produced responses such as LLLL. Notably, patients at an earlier stage of their illness (but still showing some negative signs), tended to produce less severe stereotypy such as response alternation (e.g. LRLR) (Frith & Done, 1983). In terms of the model in Figure 3.1, this may mean that the route to willed action (B1) was slightly less impaired in these patients than in those who perseverated.

In summary, Frith (1992) was able to account for three types of behavioural abnormality in schizophrenia (i.e. poverty of behaviour, inappropriate behaviour and stereotyped behaviour) in terms of impairments in the system underlying self-initiated (willed) action. The mechanisms leading to stimulus-driven actions were assumed to be largely intact. We saw in Chapter 2 that such behaviours are often shown by patients with frontal lobe lesions, and can be explained in terms of a dysfunctional Supervisory Attentional System (SAS; Shallice, 1988). The SAS, which can generate actions in the absence of external stimuli, or inhibit stereotyped behaviour or inappropriate responses to stimuli, is very similar to Frith’s willed action system. Moreover, the many highly automatic, routine programmes (schemata) which, in Shallice’s (1988) model are modulated by the SAS, correspond very closely to the stimulus-driven actions indicated in Figure 3.1. Frith (1992) suggested that a possible difference between patients with prefrontal lesions and schizophrenics with behavioural signs, lies in the severity of impairment of the willed action system. One possibility is that frontally lesioned patients have a severely damaged SAS, so have great problems in even generating appropriate goals or plans. Schizophrenics with behavioural features, on the other hand, may have goals, but are impaired in the translation of these into willed intentions. As Frith (1987; 1992) has pointed out, patients with Parkinson’s disease (who often show negative behavioural signs, especially poverty of movement), may have a deficit at an even later stage in the
willed action sequence, so that willed intentions can be generated, but are not then translated into action. In terms of the model shown in Figure 3.1, this implies an impairment of route C. Evidence for this comes from self-report of patients, who often say that they know what action they want to initiate, but cannot actually make the movement (Frith, 1987). In addition, the phenomenon of hemi-Parkinsonism, where the patient has problems in moving his limbs on only one side of his body, suggests that Parkinson’s disease may disrupt the willed action sequence at a relatively late stage when action is being coded in relation to a particular part of the body (Frith, 1987).

Relation to brain systems

Lesion studies of non-human primates (e.g. Passingham et al., 1989, cited in Frith, 1992) and several functional imaging studies of normal volunteers discussed earlier (e.g. Frith et al., 1991a; Pardo et al., 1990), suggest that the willed action system is mediated by prefrontal cortex, cingulate cortex, supplementary motor area (SMA), and the reciprocal interactions between these structures and posterior brain regions. Passingham et al. (1989) found that monkeys with SMA lesions were impaired at self-initiating actions, but could still perform tasks in which an external stimulus specified the required response. In their PET functional imaging study of word finding in normal volunteers (see Chapter 2), Frith et al. (1991a) found that self-generation of words (verbal fluency) activated the left dorsolateral prefrontal cortex (DLPFC), the left parahippocampal gyrus, and bilateral anterior cingulate cortex (ACC). It also led to reduced blood flow in bilateral superior temporal cortex. In a lexical decision task, in which the same subjects had to decide whether aurally presented stimuli were words or non-words, there was an increase in superior temporal blood flow, with no change in DLPFC. The authors suggested from these results that word representations are stored in neural networks in the superior temporal cortex, and that spreading activation in these networks is initiated when an external word stimulus is presented. In terms of Figure 3.1, this can be thought of as
a ‘stimulus-driven’ activation. The pattern of activation during intrinsic word
generation on the verbal fluency task (i.e. a willed action) was thought to reflect a
functional interaction between frontal and temporal regions, so that spreading
activation amongst word representations in superior temporal cortex was both
initiated and controlled (Frith et al., 1991a; see the earlier discussions in Section 2.3).
In a similar PET study of normals, Frith et al. (1991b) investigated the activation
associated with intrinsic generation of a finger movement, and found increased blood
flow in the DLPFC, with decreased activity in sensorimotor cortex in the position on
the sensorimotor strip where finger movement is known to be located. Together, these
studies imply that willed action involves interactions between frontal areas (e.g.
DLPFC and ACC) and posterior regions specific to the action being generated (i.e.
words or finger movements in these two experiments) (Frith, 1992).

In line with these results, Frith (1992) suggested that patients with impair­
ments of the willed action system may all have deficits (of varying severity) within
functional loops linking frontal and posterior regions. Patients with prefrontal lesions
may have the most severe deficit because structural damage will severely restrict the
function of the loop, meaning that on a psychological level, goals and plans may not
be generated. On the other hand, Parkinson’s disease is known to be associated with
a lack of dopamine in the striatum (Ehringer & Hornykiewicz, 1960, cited in Frith,
1992), so it may be this deficit which underlies the proposed inability of such patients
to convert willed intentions into motor output. We saw in Chapter 2 that several
strands of evidence suggest that the primary impairment in schizophrenia may be
fronto-temporal and/or fronto-striatal functional disconnection (e.g. Frith et al., 1995;
Robbins, 1990). Such a deficit fits rather well with Frith’s (1992) hypothesis that, on
a psychological level, the behavioural signs of schizophrenia arise from a willed
action impairment which is intermediate in severity between that of patients with
prefrontal lesions or Parkinson’s disease, namely an inability to translate goals into
willed intentions. It may well be that intact fronto-posterior connectivity is necessary
for such a sequence to be carried out.
Passivity experiences

Theory

Frith (1987) proposed that impairments in the willed action system could also be invoked to explain many of the positive symptoms of schizophrenia. He pointed out that an organism which can produce both willed actions and stimulus actions by the routes shown in Figure 3.1 needs some way of knowing whether a particular action was stimulus-elicited or self-generated. He suggested that there must, therefore, be a monitoring system which can distinguish between externally- and internally-generated action. The monitor is shown in Figure 3.2, where it can be seen that it has access to information about current goals and plans, stimulus intentions (i.e. intended actions appropriate to an external stimulus), willed intentions (i.e. intended actions appropriate to the goals and plans), and actions currently initiated.

Figure 3.2. The monitoring of action. Adapted from Frith (1992; Chapter 5).
In this system, the monitoring of stimulus intentions and willed intentions tells the organism about the causes of its actions, i.e. whether they are self-initiated or caused by external stimuli. The monitoring of currently initiated actions tells the organism about the causes of events, i.e. whether they were caused by it, or by an external influence (Frith, 1992). Frith therefore suggested that if something went wrong with this monitoring system, the subject might have problems in distinguishing between internally- and externally-generated actions and events. One consequence of this might be that a willed action, such as a limb movement, was experienced as externally generated. This may give rise to one of Schneider’s (1959) ‘first rank’ symptoms of schizophrenia, namely a delusion of control (where the subject believes that his actions are controlled by alien forces). If the monitoring problem extended to thoughts as well as motor output, then the patient might experience his own thoughts as externally generated. This odd experience may correspond to the symptom of ‘thought insertion’, where the patient reports that thoughts are put into his mind from outside. As we saw in Chapter 2, a considerable amount of evidence suggests that auditory hallucinations are caused when subvocal speech or inner speech is experienced as externally generated. Clearly, an impairment of Frith’s proposed monitoring system could also account for this phenomenon.

Convincing evidence for the existence of such a monitor in normals came from studies of error correction on motor tasks (see Frith, 1987, for a review). Frith pointed out that comparison by the monitor of information about intended acts and acts which had actually been initiated, would enable an organism to detect errors of action very rapidly before external feedback reached its perceptual system. Such a comparison would also provide information about whether the error was stimulus-elicited or self-generated. For example, if a subject produced a response to an external stimulus whilst concurrently holding a willed intention, then a mismatch between the willed intention and the centrally monitored action would locate the error as stimulus-elicited, and alert the subject to pay more attention to his willed
intentions. The speed with which normals can correct their errors on motor tasks is often so fast that the error detection could not have been based solely on external feedback, so presumably relied on an internal monitoring system of the kind discussed above (Frith, 1987).

In the model shown in Figure 3.2, there are two possible places at which a disconnection might lead to impaired monitoring of willed action. One of these is route D, by which an action that has just been initiated is centrally monitored. This process is thought to occur by ‘re-afference copying’ (von Holst & Mittelstaedt, 1950) or ‘corollary discharge’ (Sperry, 1950), which involves a copy of the signal sent to the effector system being fed back to the monitor (Frith, 1987). Corollary discharge is easily understood in relation to eye movements; for example, movement of an image across the retina could be caused either by movement of the eye itself, or by movement of an object in the world. In the former case, corollary discharge from the signal initiating eye movement enables the shift in retinal image to be interpreted and allowed for, so that the object in the world is still perceived as stationary. In the latter case, the absence of corollary discharge means that the movement is interpreted (correctly) as externally generated (Sperry, 1950). As discussed by Frith (1987), particular manipulations of this system can lead to situations in which the consequences of a subject’s own actions are experienced as externally generated. For example, if the eyeball is poked with a finger rather than moved by a direct command to the eye muscles, the room appears to move. This is because the absence of any corollary discharge leads to the interpretation that retinal image movement must have been externally caused. This effect also suggests that corollary discharges are much more local effects than willed intentions, because the subject’s knowledge of his intention to poke the eye clearly has no influence on his perception in this situation (Frith, 1987).

Feinberg (1978) proposed that corollary discharges may be associated with

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thoughts as well as motor actions, and argued that disruption of the mechanism could underlie typical passivity phenomena such as delusions of control and thought insertion. However, on its own, such a deficit may be insufficient to explain these symptoms. For example, Frith (1987) pointed out that performance of a simple motor act may be thought of as involving three stages: the willed intention to act, the action itself (whose initiation is monitored centrally by corollary discharge), and external feedback that the action occurred. In the absence of corollary discharge, the subject would have no awareness of the initiation of action, so would be conscious simply of an intention to act followed by external feedback that the action had occurred. Whilst this would be a rather odd experience, the subject's awareness of his intention would make him unlikely to attribute the movement to an external force. On the other hand, a failure to monitor the willed intention to act, with intact corollary discharge (i.e. a disruption only of pathway E in Figure 3.2), would give the experience of movement being initiated, but without any intention to move on the part of the subject. This corresponds well to the reported experience of patients with delusions of control, when they say that an alien force is making them carry out particular actions (Frith, 1987). Interestingly, impaired monitoring of both willed intentions and initiated action in this situation might correspond to a more severe form of the symptom, where the patient reports that he is neither intending nor launching an act, but is simply a 'puppet', being passively manipulated by external forces.

If we assume that thoughts are also normally monitored within the willed action system, then a failure to monitor the intention to think, together with an intact corollary discharge indicating that a thought had been initiated, might give the experience of being made to think particular thoughts. Impaired monitoring of both willed intentions and initiated action may correspond to the 'true' symptom of thought insertion as defined in the Present State Examination (PSE; Wing et al., 1974), where the patient reports that thoughts which are not his own are put into his mind from outside. Within the same framework, it is possible to speculate about the
symptoms of ‘thought block’ or ‘thought withdrawal’. In the former, the patient reports that his thoughts suddenly stop quite unexpectedly; perhaps this reflects impaired monitoring of the intention to stop thinking about something. Similarly, in thought withdrawal, the experience is of thoughts being taken out of the mind, as though by some external force (for a full description see the Glossary of the PSE; Wing et al., 1974). This may be the exact opposite of thought insertion, and could occur when faulty monitoring of willed intentions and initiated actions begins in the middle of a train of thought. The subjective experience associated with this may be of thoughts which were initially intended and labelled as self-initiated, suddenly becoming unintended and no longer associated with the self. The symptom of ‘thought broadcasting’, where the patient reports that his thoughts are shouted out of his head, and therefore available to the world in general, may be explained in a similar way. Finally, auditory hallucinations may involve impaired monitoring of the willed intention and action associated with subvocal speech or inner speech, so that the patient experiences an externally generated ‘voice’ (Frith, 1992). Notably, this is similar to Hoffman’s (1986) theory of auditory hallucinations (see Chapter 2), in which it was proposed that random intrusions of material from long term memory into current thought processes are experienced as alien because of their unintended nature. It is likely that all of the experiences discussed above result from an intermittent malfunction of the monitoring system, so that over time the severity of psychotic symptoms fluctuates. Such intermittent problems may explain why alien ‘voices’ or inserted thoughts are often reported as being brief and fragmentary (Frith, 1987).

Empirical support
A number of studies have investigated self-monitoring in schizophrenia, and in general the results have supported Frith’s hypothesis. As we saw earlier, evidence for the existence of a monitor in normals comes from studies showing that they can rapidly correct errors in the absence of external feedback. Thus, we might predict that
schizophrenics showing the various positive symptoms described above should be impaired at such error correction, but should perform normally when external feedback is provided. Malenka et al. (1982) investigated this using a task in which subjects had to follow a target on a computer screen using a joystick. They ensured that subjects made many errors by periodically reversing the contingency between joystick movement and the movement of the target. In line with Frith’s model, schizophrenic patients were less able than normal and psychiatric controls to rapidly correct errors in the absence of visual feedback. However, no relationship between task performance and symptomatology was reported, and as discussed by Frith (1987), the result of this study was weakened somewhat by the fact that the schizophrenics made more errors than controls overall. Frith & Done (1989) used a similar task in the guise of a video game, in which subjects used a joystick to fire bullets from guns to shoot birds. As in the study by Malenka et al. (1982), the relationship between joystick movement and the particular gun selected was periodically reversed, so subjects made many firing errors. In the first version of the task visual feedback was provided, so that for 2800 msec after the firing of a particular gun the subject had the chance to correct an error and fire the other gun. The second version of the task was identical, except that walls filled most of the screen between the gun and the bird so that the bullet was invisible for the first 2000 msec after it had been fired. The subject therefore received visual feedback only for the final 800 msec of the bullet’s trajectory. Results showed that schizophrenic patients and psychiatric and normal controls were equally adept at error correction in version 1 of the task, where visual feedback was provided. However, in version 2, the schizophrenic patients with passivity experiences (specifically delusions of control, thought insertion and thought blocking) corrected significantly fewer errors in the first 2000 msec (i.e. without visual feedback) than the other schizophrenics and control subjects. This was interpreted as showing that passivity symptoms are associated with impaired internal monitoring of intention and action, as predicted by Frith’s (1987) hypothesis.
Mlakar et al. (1994) developed a task in which subjects had to draw geometric shapes using a joystick, without any visual feedback appearing on the computer screen. As with version 2 of Frith & Done’s (1989) task, performance (which was evaluated using the responses recorded by the computer), was heavily dependent upon central monitoring ability. In line with Frith’s model, a group of 25 schizophrenic patients experiencing Schneiderian ‘first rank’ symptoms was significantly worse on several measures of task performance than schizophrenics without those symptoms, or normal controls. In a study using several tests of monitoring ability similar to those discussed above, Quraishi et al. (1996) substantially replicated the findings of earlier studies, and confirmed that poor performance is associated with positive symptoms such as delusions of control and thought insertion, but is unrelated to negative signs or general cognitive dysfunction. The only error-correcting study to date which has failed to show impaired self-monitoring was carried out by Kopp & Rist (1994). As we have seen, Frith’s theory suggests that monitoring of willed intention and action should be particularly impaired in patients with passivity experiences or auditory hallucinations. However, none of the schizophrenic subjects used by Kopp and Rist definitely had passivity symptoms at the time of the experiment, and in fact only 3 patients had reported them in the two weeks before testing. Furthermore, although several subjects did report auditory hallucinations, the relationship between these and error-correction was not specifically investigated in this study.

A second type of paradigm which has the potential to reveal impaired internal monitoring involves EEG responses. As discussed by Frith & Done (1988), a random tone normally elicits a large EEG response, but if the tone is self-generated by the subject, the response tends to be of much smaller amplitude. This may be because, in the self-generation condition, the subject ‘expects’ the tone because of internal monitoring of his intention or action to produce it (Frith & Done, 1988). This analysis would predict that if schizophrenics with certain positive symptoms have
problems in the monitoring of self-generated acts, they should show equally large EEG responses to self-generated and externally produced tones. A preliminary study discussed by Frith & Done (1988) supported this prediction by showing that only 20% of acute, drug-free psychotic patients had the normal EEG response to internally- and externally-generated stimuli. A similar result was also obtained by Braff et al. (1977). These findings are encouraging, but require further replication, with particular emphasis on the relationship between the EEG effect and passivity symptoms and auditory hallucinations.

Relation to brain systems

Frith (1992) reviewed a number of studies investigating self-monitoring in animals, and concluded that, as with the generation of willed action, interactions between frontal and posterior brain regions may also be involved in the internal monitoring of action. For example, Robinson & Wurtz (1976, cited in Frith, 1992) discovered cells in the superior colliculus of the rhesus monkey which responded when an external stimulus moved, but did not respond when the monkey moved its eye over a stationary stimulus. Such a system would allow the animal to distinguish between events caused by its own actions, and those due to external influences, and Robinson and Wurtz suggested that it operated by corollary discharge (indicating initiation of eye movement) passing from the frontal eye fields to the superior colliculus. Similarly, Ploog (1979, cited in Frith, 1992) noted that the auditory cortex of the squirrel monkey contains cells which respond to the vocalisation of other monkeys, but not to self-produced vocalisations. He suggested that inhibition of these cells during self-vocalisation was due to corollary discharge passing from anterior cingulate to auditory cortex. In both of the studies discussed here, the area suggested as the source of corollary discharge is the area responsible for the self-initiated action (i.e. frontal eye fields and anterior cingulate respectively). This is intuitively plausible, as it is likely that any copy of the signal to the effector system will be produced in the same place as the signal itself.
A number of findings from work with humans are also consistent with the notion that internal monitoring of action involves frontal and posterior brain regions and the interactions between these regions. For example, Goldberg et al. (1981) described two patients with unilateral medial frontal cortex damage (largely in the supplementary motor area), who both showed the ‘alien hand sign’. This is a phenomenon whereby the hand contralateral to the lesion shows apparently purposeful behaviour (e.g. grasping in the presence of a door handle), with the patient being totally unaware of the action unless he actually looks at the hand. It can be explained as a ‘stimulus-driven’ action (in response to the sight of the door handle), with an absence of corollary discharge associated with the hand movement (Frith, 1987). As the sign is found in patients with medial frontal damage, an analysis of it in terms of impaired corollary discharge is consistent with the notion that the discharges associated with stimulus-elicited limb movements may originate in the frontal cortex.

A number of studies suggest that impaired self-monitoring may also result from temporal lobe damage. For example, Trimble (1990) found that patients with temporal lobe epilepsy often reported ‘first rank’ symptoms such as thought insertion or delusions of control. As part of their neuropsychological model, Gray et al. (1991) proposed that defective monitoring by a ‘comparator’ in the septo-hippocampal system was responsible for many of the positive symptoms of schizophrenia. They suggested that the comparator continually monitors sensory input, and compares it with expected input so that suitable responses are produced. Any mismatch between perceived and expected information may mean that increased attention is paid to the input. Clearly this system could be the basis of Broadbent’s (1971) ‘pigeon-holing’ mechanism whereby information processing is modified by prior expectations, and failure of the system could underlie Hemsley’s (1987, p. 182) proposed “weakening of the influence of stored memories of regularities of previous input on current perception” in schizophrenia (see Chapter 2 of this thesis). Frith & Done (1988) pointed out that Gray et al.’s (1991) comparator primarily receives information about
stimulus intentions, whereas any monitoring of the action system needs information about both stimulus intentions and willed intentions in order to distinguish between stimulus-driven and self-initiated responses. They suggested that their own monitor (which clearly does have access to this information) may itself be located in the hippocampus, and that certain positive symptoms of schizophrenia reflect a failure of input to this area from other brain regions. In particular, they argued that information about initiated actions (corollary discharges) or intended actions (willed intentions) normally passes from prefrontal cortex (where planning and goal setting occur) to hippocampus, via either the parahippocampal cortex or the cingulate cortex and subiculum. They proposed that in schizophrenia, functional disconnection of these prefrontal-hippocampal routes may lead, on a psychological level, to impaired monitoring of willed action and the specific positive symptoms thought to be associated with this.

This general model is compatible with Frith et al.’s (1995) functional imaging study of verbal fluency in schizophrenia (see Chapter 2), which showed that when generating words on a paced task, schizophrenic subjects can activate frontal regions, but fail to show the normal reduction in blood flow in left superior temporal gyrus. This was interpreted as showing fronto-temporal functional disconnection in all schizophrenic patients, and Frith et al. (1995) related this disconnection to particular signs and symptoms. For patients with positive symptoms such as auditory hallucinations and passivity phenomena, one possibility is that the reduced inhibition in left superior temporal cortex reflects impairment of input from frontal regions into the proposed hippocampal monitoring system. This may mean in practice that, whilst self-initiated actions can still be produced in these patients, the corollary discharges and/or willed intentions associated with them are poorly monitored.

A closely related possibility arises from consideration of the pathways taken by some corollary discharge signals. We saw earlier that Ploog (1979) suggested that
corollary discharges associated with self-vocalisation in the squirrel monkey pass from cingulate cortex to auditory association cortex, where they inhibit the response of cells. It is now widely recognised that in both animals and humans, corollary discharges can pass directly from brain regions involved in initiating the motor components of actions to the areas which receive sensory stimulation as a result of those actions. Thus, as discussed by Frith et al. (1995), there is evidence that in normal humans, less activity occurs in the temporal cortex in response to the subject’s own voice than when he hears the voice of someone else. This may reflect inhibitory modulation of auditory cortex by corollary discharges associated with self-vocalisation. By this analysis, the reduced inhibition in temporal regions observed on functional imaging of schizophrenic patients may reflect impaired monitoring of self-initiated vocalisation or inner speech; this is precisely the abnormality suggested by Frith (1987; 1992) to be associated with auditory hallucinations.

Supporting evidence for this hypothesis was provided by a PET functional imaging study by McGuire et al. (1995), in which subjects had to imagine sentences being spoken in another person’s voice. Schizophrenic patients prone to auditory hallucinations (but not hallucinating at the time) showed reduced activation in the left medial temporal gyrus (MTG) and supplementary motor area (SMA) relative to non-hallucination-prone schizophrenics and normal controls. In line with the above discussion, McGuire et al. suggested that the reduced SMA activation in the hallucination-prone patients reflected a loss of corollary discharge associated with self-generated action, which may contribute to the tendency of those patients to attribute their own inner speech to an external source. McGuire et al. also pointed out that the lower MTG activation in these subjects suggests that their temporal cortex was responding as if they were speaking aloud. As there was no evidence that vocalisation occurred, the results are consistent with the hypothesis that hallucination-prone patients have a tendency to confuse imaginary alien speech with self-vocalisation (and vice versa). A remaining point regarding inhibitory modulation by corollary
discharge relates to positive symptoms other than auditory hallucinations. It is likely that self-initiated limb movements, for example, are associated with corollary discharges passing from frontal brain regions to posterior regions receiving sensory stimulation as a result of the movements. By this account, delusions of control could reflect disconnection of these pathways, so that self-initiated movements are experienced as externally generated.

A final issue relates to the role of dopamine in positive symptomatology. As we saw earlier, Frith (1987) suggested that Parkinson’s disease involves an impairment in the route by which willed intentions are translated into actions (route C in Figure 3.2). As Parkinson’s disease is known to be associated with a lack of dopamine in the striatum (Ehringer & Hornykiewicz, 1960), it is reasonable to assume that dopamine is required for that particular step in the willed action sequence. Now, the clinical efficacy of typical neuroleptic drugs is known to correlate strongly with their ability to bind to dopamine D₂ receptors (Seeman, 1980; see Chapter 2 of this thesis), so Frith (1987) proposed that in schizophrenic patients, these drugs reduce the translation of willed intentions into action. As positive symptoms such as delusions of control and auditory hallucinations are thought to arise from impaired monitoring of actions (and the intentions associated with them), a reduction in self-generated action will necessarily reduce the chance of aberrant monitoring (and positive symptoms) occurring. This model may explain, incidentally, why Parkinsonian signs are sometimes produced as a side-effect of neuroleptic medication. It also predicts that typical neuroleptics should, if anything, exacerbate the negative signs of schizophrenia (Frith, 1987).

Other positive symptoms

Theory

Within the framework of Frith’s model, many other positive symptoms can be
rationalised if we consider the monitoring of ‘stimulus intentions’ (i.e. the meaning of external stimuli) by schizophrenic patients (route F in Figure 3.2). One such symptom is ‘delusional misinterpretation’ where the patient feels that there are special meanings for him in particular objects, or in the way things are arranged (Wing et al., 1974). For example, a patient of mine told me that traffic lights turning from red to green had ‘forced’ him several times to start running down the street; as a result, he subsequently held the belief that every time a traffic light turned to green, it was a sign that he should start doing things more quickly. A possible interpretation of this within Frith’s model is that on several occasions, the patient started running when a traffic light turned to green, but failed to monitor his intention to run. As a result, he repeatedly experienced delusions of control by the traffic lights, and eventually concluded that these stimuli must have personal significance for him. It is interesting to note that the patient’s monitoring of the stimulus intention was partially intact, as he incorporated the correct meaning of a green traffic light (i.e. ‘speed up’ or ‘go!’) into his delusional misinterpretation. However, he incorrectly inferred that traffic lights had an additional ‘intention’ relevant only to himself, i.e. to make him speed up his daily activities.

Other people in the world around us can be thought of as external stimuli whose ‘stimulus intentions’ are especially complex and difficult to interpret. For example, we often need to predict or explain a person’s behaviour on the basis of subtle facial expressions or non-verbal gestures. Thus, someone who pulls a bored-looking face whilst saying of a lecturer, “He was exciting”, is likely to be interpreted as being sarcastic. Such interpretation requires us to make inferences about the mental states (e.g. desires, knowledge, intentions or beliefs) of the person producing the utterance, so that in this case we infer that he thinks the lecturer is rather boring, and wants to communicate that fact to us. The inability to make these inferences might lead to acceptance of the utterance at face value. Premack & Woodruff (1978) called the ability to predict and explain others’ behaviour on the basis of their mental
states 'having a theory of mind'\textsuperscript{2}. The attribution of mental states to others has also been called 'mentalising' (U. Frith \textit{et al.}, 1991). From their observation of apparently deceptive behaviour by chimpanzees, Premack and Woodruff suggested that, at some level, these animals could infer that their conspecifics had knowledge and beliefs which could be manipulated in order to produce a certain behaviour. The theory of mind ability of chimpanzees has probably only evolved to a rudimentary level, and indeed many other non-human primates may not have a theory of mind at all (Cheney & Seyfarth, 1990). However, in normal human adults the skill is highly developed, and underlies many of our most complex social behaviours. In the human literature, a lot of evidence suggests that before the age of about 4 years, normal children have a poorly developed theory of mind (see Astington & Gopnik, 1991, for a review). In addition, people with autism seem to specifically lack this cognitive skill (see Baron-Cohen \textit{et al.}, 1993\textit{b}, for a review). In both cases, the absence of a theory of mind makes it very difficult for the person to understand, predict or manipulate the behaviour of someone else solely on the basis of their mental states. Thus, young children and autistics are, for example, very poor at lying, because such a strategy requires an understanding of the fact that people’s behaviour can be manipulated by making them believe something which is, in fact, false (e.g. Sodian & Frith, 1992).

As part of his neuropsychological model, Frith (1992; 1994) suggested that a number of positive symptoms of schizophrenia can be explained in terms of a dysfunctional theory of mind, with patients incorrectly monitoring the mental states (i.e. stimulus intentions) of other people. He argued that these patients may have had a normal theory of mind during adolescence, but that it became dysfunctional at the time of their first breakdown. As a result, they still know that others have mental

\textsuperscript{2}In fact the term 'theory of mind', as defined by Premack and Woodruff, referred to the attribution of mental states to oneself as well as to other people. In the present discussion, however, 'having a theory of mind' will refer to the ability to attribute mental states to others.
states, but now infer these incorrectly or have great difficulty inferring them at all (Frith, 1994). The model most obviously accounts for ‘delusions of persecution’ where, by definition, the patient incorrectly believes that others intend to harm him. As discussed by Frith (1994), a difficulty in reading others’ intentions might lead to the conclusion that people were deliberately disguising their intentions for some secret reason; this could underlie a paranoid belief in a general conspiracy. Frith’s model can also explain ‘delusions of reference’, where the subject may believe that other people drop hints about him, say things with a double meaning, or even that people on the television or radio are speaking especially to him (Wing et al., 1974). In all of these cases, the patient incorrectly infers that people intend to communicate certain things, when in reality they have no such intention. A difficulty inferring the mental states of people might make them seem like actors or robots without any real feelings (the experience of ‘derealisation’). If this occurred with someone who the patient knew very well, then he might conclude that the real person had been replaced by an identical double without any real mental states (Capgras’ syndrome; Capgras & Reboul-Lachaux, 1923) (Frith, 1994).

While some patients with auditory hallucinations attribute the ‘voice’ they hear to an unspecified external source, or even report that they hear a ‘voice’ but know that it is not ‘real’, others have a strong belief that a particular external agent is speaking to (or about) them, and trying to influence them in some way (Frith, 1996). Similarly, although delusions of control and thought insertion are sometimes reported in rather abstract terms (e.g. the patient who said, “I am manipulated by cosmic strings”; Mellors, 1970), they often involve the belief that a specific agent is the controlling power or the one responsible for the alien thoughts. Thus, although, as discussed earlier, the core symptoms of auditory hallucinations and passivity phenomena probably reflect deficient self-monitoring of willed action, an additional impairment in the monitoring of others’ mental states may explain why patients sometimes believe that these experiences reflect intentional acts by particular external
agents (Frith, 1996).

This hypothesis is consistent with the results of a study by Cahill et al. (1996), who investigated how schizophrenic patients, when speaking, perceived their own voice when it was immediately fed back to them through headphones with distorted pitch. Subjects were asked to identify the source of the sounds they heard, and to explain their answers. This paradigm provides a strong measure not of self-monitoring (because subjects would receive some external feedback from orofacial movements and skull vibration to indicate that sounds were self-generated), but of the tendency to attribute events to external agents. Results showed that subjects did often attribute the speech sounds to an external agent (e.g. one said, “I think it’s an evil spirit speaking when I speak”). The frequency of these attributions was significantly correlated with severity of delusions ($\rho = 0.61; p < 0.01$), but there was no significant relationship with scores for hallucinations ($\rho = 0.40$) or negative behavioural signs ($\rho = -0.02$). Pilot data using the same paradigm showed that normal subjects and schizophrenics in remission consistently realised that the speech sounds were emanating from themselves. Although the particular types of delusion were not recorded in this study, the results are consistent with Frith’s proposal that many delusions reflect a tendency to incorrectly infer the intentions of other agents. By attributing the speech to an external agent, the subject was, in effect, inferring that that agent intended to communicate with him. On the other hand, the data suggest that auditory hallucinations are only sometimes associated with the tendency to misattribute others’ mental states. Thus, as discussed above, it is possible for patients to hear ‘voices’ (and therefore have impaired self-monitoring of initiated actions and willed intentions), but not attribute these to a specific external agent. Together, these results suggest that although various symptoms of schizophrenia may involve dysfunction of the cognitive systems underlying self-monitoring of willed intentions and attribution of others’ intentions, these systems are relatively independent of one another, and can be selectively impaired. As a result, some patients report hallucin-
ations in the absence of delusions (and vice versa), whilst others report both types of symptom (Frith, 1996).

A remaining theoretical point concerns the possibility of explaining certain behavioural signs in terms of an impaired appreciation of others’ mental states. In the domain of language, we saw earlier that deficits in the willed action system can explain poverty of speech, perseverative speech and distractible or inappropriate speech. However, schizophrenic patients sometimes produce speech which is difficult for the listener to comprehend because of a failure on the part of the patient to consider what the listener already knows. For example, Docherty et al. (1996) found that schizophrenic speech samples were significantly more likely than those from normal controls to contain ‘missing information references’, which were defined as unqualified references to things unknown to the listener. Thus, one patient produced the utterance, “They let George go home, so why not me?” (ibid., p. 359), without any prior mention of George, a person unknown to the listener. Similarly, Rochester & Martin (1979, cited in Frith, 1992) found that schizophrenics often produced speech with inadequate referents and cohesive ties, so failed to inform the listener how the conversational topics related to one another. Failure to take into account a listener’s knowledge could explain why schizophrenic patients sometimes use pronouns incorrectly (Frith & Allen, 1988). This manifests either as a failure to use a pronoun in place of a redundant noun (suggesting that the patient fails to consider that the listener already knows the subject of the discourse), or as use of a pronoun without any obvious antecedent (Frith, 1992). In the former case, the result is pedantic speech with unnecessary repetition of nouns, and in the latter case ambiguous, sometimes incomprehensible speech is produced. In general terms, a failure to correctly infer the knowledge of listeners may be one reason why schizophrenics show a particular impairment in the expressive, rather than receptive aspects of language. Thus, as we saw in Chapter 2, patients are impaired in accurately describing a coloured disk to a listener, but can easily understand the description provided
by a normal speaker (Cohen, 1978). This selective impairment is consistent with the patient failing to tailor his description to fit the probable knowledge base of the listener. In contrast, a normal speaker does take into account the listener’s knowledge, so produces a description which the patient can understand (Frith, 1992).

Finally, it is possible that severe impairments of the theory of mind system could underlie the negative behavioural signs of social withdrawal and blunt affect (Frith, 1992). Sperber & Wilson (1995) pointed out that for communication between two individuals to begin successfully, the listener must realise that the speaker intends to communicate with him. This intention is often signalled by subtle non-verbal cues such as a raised eyebrow or a smile. We saw earlier that a tendency to misattribute others’ intentions on the basis of these non-verbal signals could lead to delusions of reference. However, if the patient had a more severe theory of mind impairment, he may completely fail to attribute mental states such as intentions to others. As a result, he may fail to recognise when other people are trying to communicate with him; this could make him appear socially unresponsive and withdrawn. From this argument, it is possible that poverty of speech (although certainly explicable in terms of an impairment in generating willed action) could, in some cases, be explained in terms of a failure to recognise that other people are engaging in communication, and are therefore expecting an elaborated response to their questions or comments (Frith & Frith, 1991).

**Empirical support**

To date, only a few studies have explicitly investigated the theory of mind ability of schizophrenic patients. However, a number of experiments from other areas of the literature used tasks which required mental state understanding, so those studies will be reviewed first. It should be borne in mind throughout this review that Frith’s model predicts the poorest performance by patients with negative signs such as affective blunting, poverty of speech and social withdrawal, as it is proposed that
these people often completely fail to infer others' mental states. Patients with positive symptoms such as delusions of persecution should also show poor task performance, but their deficit may be less pronounced, as they are presumed to still infer others’ mental states, albeit with frequent errors. As Frith predicts the poorest theory of mind performance from the patients who tend to show the highest level of generalised cognitive impairment (e.g. Frith et al., 1991c), it is important that any empirical tests of Frith’s model control carefully for factors such as current IQ and memory.

We saw in Chapter 2 that Cutting & Murphy (1990a) gave multiple-choice questions to schizophrenic patients to test their knowledge of social situations (e.g. How would you tell a friend politely that they had stayed too long?), and situations relatively free of a social component (e.g. Why is it unsafe to drink tap water in some countries?) The schizophrenics showed a specific impairment on the social knowledge questions, relative to IQ-matched psychiatric controls, and it is interesting to note that a number of these questions required an intact theory of mind for their solution. For example, in order to tell a friend politely that he has stayed too long, we might lie to him to make him think that we have to attend an urgent appointment. Unfortunately, Cutting and Murphy did not report the symptoms of the schizophrenic patients in their study, so it is not possible to tell whether the most pronounced deficits were shown by those with negative signs, as predicted by Frith’s model. Pilowsky & Bassett (1980) asked schizophrenic subjects and psychiatric controls to describe photographs of people, and found that the schizophrenics were more likely than controls to mention physical appearance rather than ‘internal state of mind’. This general finding was replicated in several other studies, where a lack of ‘psychological’ language (referring to the emotions and mental states of the people in the photographs) was found to be associated with ratings of social withdrawal and affective blunting (Bodlakova et al., 1974; Williams & Quirke, 1972), and poverty of speech (Allen, 1984). Clearly this is consistent with Frith’s suggestion that of all the negative behavioural signs, these three signs should be particularly associated
with a problem in attribution of others’ mental states.

It is possible that a dysfunctional theory of mind might lead to difficulties in distinguishing facial expressions of emotion, especially complex emotions such as surprise, where interpretation requires an understanding of mental states (e.g. “He expected X, but received Y”). Also, as discussed earlier, the proposed cognitive deficit may lead to a poor understanding of subtle non-verbal facial or bodily signals, especially those reflecting underlying mental states (e.g. a person leaning forward shows that he intends to begin speaking). There is a large literature on facial processing in schizophrenia (see Morrison et al., 1988, for a review of the earlier experiments), and despite many methodological differences across studies, there is evidence for a deficit over and above general cognitive impairment. Few studies have explicitly investigated the relationship between this deficit and symptomatology, but poor task performance has been found in groups of acute (e.g. Cutting, 1981) and chronic patients (e.g. Salem et al., 1996), suggesting that poor facial processing may occur in patients with either positive or negative features of schizophrenia (in line with Frith’s model). The precise nature of the facial processing deficit has been debated for several years. For example, Cutting (1981) suggested that acute schizophrenics have a specific deficit in judgment of emotional expression, because his patients were impaired relative to remitted psychotics and psychiatric controls at judging the friendliness or meanness of faces in photographs, but were as good as controls at judging age (which required analysis of facial features). A similar result was obtained by Heimberg et al. (1992). Gessler et al. (1989) also used tasks in which subjects had to judge facial affect and age from photographs, but in this study, care was taken to match the two tasks on difficulty and discriminating power. With this manipulation, acute schizophrenics performed significantly worse than remitted schizophrenics and controls on both tasks, suggesting a problem with the processing of both facial affect and facial features. This conclusion was supported and extended by more recent studies which also used a matched-tasks design. For example, Kerr
& Neale (1993) found that chronic, unmedicated schizophrenics (of unspecified symptomatology) performed worse than normal controls on equally difficult tests of facial emotion perception (emotion identification and discrimination tasks) and non-emotion perception (a facial recognition task). This finding was replicated using the same tasks with chronic, medicated patients by Salem et al. (1996). Finally, Archer et al. (1992) found similar results when they compared schizophrenics with predominantly positive symptoms with current IQ-matched control groups of normal and depressed subjects. Thus, the general consensus now seems to be that schizophrenic patients with either positive or negative features show a general deficit in facial processing.

These results are broadly consistent with Frith’s theory, in the sense that a problem inferring others’ mental states may lead to a general difficulty with the extraction of information from facial stimuli. However, more conclusive evidence would be provided by studies showing that schizophrenics are differentially impaired at recognising emotions associated with mental states (e.g. surprise) compared to those which can be understood without mental state inference (e.g. happiness). Some of the more recent studies (e.g. Kerr & Neale, 1993; Salem et al., 1996) did use stimuli including ‘happy’ and ‘surprised’ faces, but as Frith’s hypothesis was not being directly investigated in those studies, separate scores for these two types of expression were not reported. In one sense, all of the studies discussed above may have unwittingly taxed the theory of mind system, because they used posed photographs of models, so required subjects to make judgments about intentional communication by actors rather than veridical reflections of reality (Gessler et al., 1989). By this analysis, the widespread impairments shown by acute and chronic schizophrenics are consistent with Frith’s hypothesis. One study of facial affect recognition which may have some bearing on this point was carried out by LaRusso (1978). She presented subjects with video clips of peoples’ faces as they either expected, or did not expect, to receive electric shocks. Groups of paranoid schizo-
phrenics (with delusions of persecution and reference) and IQ-matched normal controls viewed the tapes; half of each group received a version of the tape in which shocks had actually been administered, and the other half saw a version in which actors simulated facial expressions appropriate to expectation or non-expectation of shock. Subjects simply had to say, for each clip, whether the person expected to receive a shock or not. Results showed that normals viewing the simulated stimuli scored significantly higher than normals viewing the genuine stimuli. A possible reason for this is that, through theory of mind inference, the normals viewing the simulated tapes perceived the actors’ intention to communicate an emotion (for example by an exaggerated facial expression), so found this easier than the ‘genuine’ condition. In contrast, the schizophrenics who received the simulated tape were significantly less accurate than the normals in this condition, and performed at a similar level to the schizophrenics in the ‘genuine’ condition. This is consistent with the hypothesis that the patients in the ‘simulated’ condition had difficulty inferring the communicative intentions of the actors, so found the task more difficult than controls, responding to the stimuli in a similar way to that of the patients in the ‘genuine’ condition.

A related area of relevance to Frith’s model is the understanding by schizophrenic people of non-verbal facial and bodily signals and gestures. For example, the control of turn-taking in normal conversation is thought to rely heavily upon eye contact, with a person’s intention to either start or stop speaking often signalled by his gaze. This means that a listener must maintain frequent eye contact so that he can tell when it is his turn to speak, and indeed Hedge et al. (1978) found that normal people spend more time looking at a conversational partner while listening than while speaking. From this analysis, we might expect that if schizophrenic patients have problems inferring others’ intentions, they may show abnormal use of gaze during conversation. This was confirmed by Davison et al. (1996), who quantified a number of videotaped facial behaviours of chronic schizophrenic patients, and found that they
showed an abnormal pattern of eye contact, tending to look at the interviewer for longer periods while talking than while listening. A related study was carried out by Rosse et al. (1994), who administered a gaze discrimination task to chronic schizophrenics and normal controls. Subjects were shown slides of a person and asked, “Is the person looking directly at you?” Results showed that the schizophrenic patients (especially those with paranoid symptoms) were more likely than controls to perceive the person in the slide as looking at them, when in fact the person was looking away. One interpretation of this within Frith’s model is that the paranoid patients had a dysfunctional theory of mind, so incorrectly inferred a communicative intention in the stimulus, and consequently judged the eyes as looking directly at them. In light of this, it is interesting to note the results of a study by Phillips & David (1997), who used visual scan paths to reveal the direction and duration of gaze when deluded and non-deluded schizophrenics and normal controls looked at photographs of faces. Relative to the other two groups, deluded subjects had abnormal viewing strategies, fixating non-feature areas of the photographs to a significantly greater extent. A possible interpretation of this result (within the framework of Frith’s model), is that the deluded patients had a greater tendency than other subjects to perceive the faces as staring directly at them, or intending to communicate with them in some way. In view of the often paranoid nature of their delusions, this may have led these patients to avert their gaze from the features (such as eyes and mouth) thought to be responsible for these potentially aversive communications.

A number of other studies have investigated the understanding of non-verbal facial and bodily signals by schizophrenic patients. For example, Monti & Fingeret (1987) tested schizophrenic and mixed psychiatric control subjects on the Profile of Nonverbal Sensitivity (PONS) test (Rosenthal et al., 1979), which requires subjects to judge which of two alternatives best describes short non-verbal sequences presented on videotape. For example, in one case, subjects had to decide on the basis of non-verbal cues whether a woman was asking forgiveness or talking to a lost child.
It is conceivable that good performance on this task requires the ability to make inferences about the mental states (desires, intentions etc.) of the story characters, and in line with Frith’s theory, the schizophrenic patients (whose symptomatology was not specified) scored significantly worse than controls. However, the absence in this study of a suitable control task matched on difficulty with the PONS, means that we cannot rule out an interpretation in terms of generalised cognitive deficit. Using a similar paradigm, Berndl et al. (1986) tested schizophrenic patients on recognition of non-verbal gestures occurring in videotaped vignettes. They found that patients with all types of signs and symptoms performed significantly worse than normal controls, and from post hoc data analysis it was concluded that this was not a reflection of general cognitive impairment, but instead represented a “selective deficit in . . . [the] functions necessary for averbal social communication” (ibid., p. 282). It is possible that a problem in attributing the mental states of the story characters underlay this apparently selective deficit.

In other areas of the literature, research groups have looked at schizophrenic patients’ understanding of situations involving potentially ambiguous verbal information as well as non-verbal cues. For example, Corrigan & Green (1993) gave schizophrenics and normal controls true or false questions about social interactions which they had just watched on video. Some of the questions were defined as ‘concrete’, and tested memory of the actors’ behaviour and dialogue (e.g. “He saw the man put down his baseball mitt”), whilst others were ‘abstract’, and required inferences about the actors’ affect and goals (e.g. “Jane talked nicely to the hostess because she wanted an extra cookie”). The concrete and abstract questions were matched on difficulty, and Corrigan and Green found that, whilst the schizophrenics (whose symptoms were not specified) performed worse than controls on both types of question, they were significantly more impaired on the abstract questions (in line with Frith’s theory). In a follow-up study, Corrigan (1994) replicated this finding, and ruled out current verbal IQ as a possible confounding factor.
Corcoran et al. (1995) developed a ‘hints task’ to test whether schizophrenics could infer the intentions behind items of indirect speech such as, “My shirt is very creased”, meaning “Please iron it”. Ten short scenarios were read aloud by the experimenter; they were presented more than once if necessary, to control for poor attention and memory. Each scenario contained a target utterance, which was read with the appropriate prosody, and in each case the subject was asked to say what the story character really meant by the utterance. As this study was devised as an explicit test of Frith’s theory, subjects were divided into symptom subgroups appropriate to the theoretical framework of his model (i.e. negative signs, incoherence, paranoid symptoms, passivity experiences and patients in remission). These groups were structured hierarchically so that any patient showing symptoms from more than one group was placed into the group for which poorer task performance was theoretically predicted (e.g. patients with negative and positive features were placed into the negative signs group). Strong evidence was found in favour of Frith’s theory in that, regardless of current IQ, patients with negative signs, incoherence or paranoid symptoms all scored significantly worse than normal and psychiatric controls (although there were only 3 subjects in the ‘incoherence’ group). The patients with negative signs scored particularly badly, showing a trend to be worse than those with paranoid symptoms. This is consistent with the predictions from Frith’s model, in the sense that patients with negative signs are presumed to fail to attribute others’ mental states, whilst those with paranoid symptoms do infer others’ intentions, but often make errors in those attributions. It can be argued that the repetition of stories by the experimenter, as and when required by subjects, ruled out any explanations of these results in terms of poorer attention or memory in the patients with negative features (R. Corcoran, personal communication). The patients with only passivity symptoms performed as well as controls in this study, which is consistent with Frith’s (1992) suggestion that these people may have problems only with the representation of their own intentions, rather than others’ mental states. Notably, this also fits with the findings of Cahill et al. (1996) discussed earlier, which suggested that the cognitive
systems underlying the monitoring of one’s own and others’ mental states are
dissociable and can be selectively impaired in schizophrenia. Finally, Corcoran et al.
found that schizophrenics in remission (i.e. those without any current symptoms or
signs) scored as well as controls, supporting Frith’s contention that theory of mind
dysfunction is closely related to symptomatology. Corcoran et al.’s hints task will be
used as part of the experiment described in Chapter 5 of this thesis.

In a separate study, also designed as an explicit test of Frith’s model, Corcoran
& Frith (1996) tested whether schizophrenic patients apply the Gricean maxims of
quantity, quality, relation and manner, and the conversational convention of polite­
ness. Grice (1975) suggested that normal speakers adhere to the maxims in order to
ensure a meaningful exchange of information during conversation, so that the
exchange is no more informative than is required (quantity), is truthful (quality),
relevant (relation) and devoid of ambiguity and prolixity (manner). To conform to
these maxims, speakers must attribute mental states to their listeners; thus in order
not to say too much (quantity), or to be unambiguous (manner), speakers must
consider how much the listener already knows. In a similar sense, a subject’s decision
to be polite or impolite rests on his attribution of the likely mental states induced in
the listener by his utterances. In this study, schizophrenic subjects (grouped
according to signs and symptoms in the same hierarchical manner as described
above) and normal and psychiatric controls were given short scenarios, and had to
choose the most likely utterance given by a character in the scenario. One of the
choices for each story ran in contravention to a Gricean maxim, or was impolite,
whereas the other (correct) choice conformed to the maxim or the politeness
convention. In line with Frith’s model, Corcoran and Frith found that, irrespective of
current IQ, patients with negative behavioural signs were significantly worse than
controls or schizophrenics in remission at applying the maxims or the politeness
convention. As predicted, patients with paranoid symptoms showed a less severe
deficit, performing as well as controls on the maxims, but being significantly more
likely to choose impolite utterances. A possible explanation for this result is that the politeness questions used in the study required more complex theory of mind inference than the maxims, so the paranoid patients (whose theory of mind impairment is thought to be less than that of patients with negative signs) could understand the maxims but not the politeness scenarios. More specifically, a number of the maxim questions used by Corcoran and Frith could be solved by applying a so-called ‘first-order’ theory of mind (e.g. “He knows that . . .” or “she intends to . . .”), whereas correct answers to the politeness scenarios required a ‘second-order’ theory of mind, where the subject had to realise that a story character was thinking about another character’s thoughts (e.g. “He thinks that she thinks that . . .”). The results are therefore consistent with the suggestion that patients with negative signs have widespread theory of mind impairments at both the first- and second-order levels, whereas people with paranoid symptoms (and no negative signs) have more subtle deficits which may only appear when more complex theory of mind inference (e.g. at the second-order level) is required.

Within the framework of their Relevance Theory, Sperber & Wilson (1995) considered in detail the various types of communicative exchange in which listeners must infer a speaker’s intention in order to fully understand his utterance. For example, in order to understand a metaphor such as “David turned yellow when he faced the enemy”, we must infer that the speaker intended to suggest something like “David became cowardly”. Sperber and Wilson pointed out that attribution of this intention is essential in preventing us from interpreting the utterance literally. While the propositional content of a metaphor is a loose interpretation of what the speaker is actually thinking, the content of an ironic utterance such as, “That was exciting” in the context of a boring lecture, is clearly the opposite of what the speaker is actually thinking. In this case, Sperber & Wilson (1995) suggested that non-literal interpretation requires us to infer the speaker’s thought about an attributed thought. Thus, the speaker is mentioning a possible thought (that the lecture really was
exciting), and expressing his own (sarcastic) thoughts about that thought. As discussed by Happé (1993), this model suggests that understanding of metaphor requires a first-order theory of mind, whereas understanding of irony requires second-order inference (“He thinks that the thought . . . ”). Using groups of autistic and normal children, she found direct evidence for this, showing that performance on tasks specially designed to test first- and second-order theory of mind (Wimmer & Perner, 1983; Perner & Wimmer, 1985) was a good predictor of scores on metaphor and irony tasks respectively. One prediction from this research is that if schizophrenic patients have an impaired theory of mind, they should show poor understanding of metaphoric and ironic utterances. In particular, patients with negative signs (who, according to Frith, may simply not infer others’ mental states at all), should show widespread impairments on both metaphor and irony tasks, tending to accept the literal meaning of the utterances. As patients with positive symptoms such as paranoid delusions are thought to incorrectly attribute others’ mental states (Frith, 1992), they may show more subtle task impairments, perhaps having problems in deciding whether a speaker is being ironic or not.

A number of studies have investigated metaphor and irony in schizophrenia, but only one of these (Mitchley, 1993, discussed below) was devised specifically to test Frith’s hypothesis. Chapman (1960) gave a multiple-choice metaphor test to a group of chronic schizophrenic inpatients (with unspecified symptoms), and control groups of brain-damaged subjects and normals. As would be predicted from Frith’s model, the schizophrenics were significantly more likely than controls to misinterpret the metaphors literally. This appeared to reflect a relatively specific deficit rather than just random responding, as the schizophrenics were no more likely than controls to misinterpret a literally correct sentence in a metaphoric sense. In a similar multiple-choice paradigm, Cutting & Murphy (1990b) found that acute schizophrenics were significantly less likely than depressed controls to select a metaphorical meaning to an ambiguous adjective in a sentence (e.g. the meaning of the word

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‘slippery’ in the sentence, “When the bank manager visited the owner of the ice-rink, he found a slippery customer”). Again, the symptoms of the patients in this study were not reported, so it is not possible to judge whether the presence of negative features predicted particularly poor performance.

A study by Reilly & Muzekari (1979) tested understanding of irony and sarcasm by presenting chronic schizophrenic patients (of unspecified symptomatology) and normal subjects with ‘mixed messages’, in which an actress on video delivered verbal phrases (e.g. “You’re hopeless!”), whilst using contradictory non-verbal cues (e.g. a smile and warm vocal inflection). Subjects rated whether the message was positive or negative on a Likert scale, and these ratings were compared with those of independent judges. Results showed that patients were significantly more likely than controls to rate the message solely on the basis of its verbal content. This is consistent with the hypothesis that the patients had an impaired second-order theory of mind, so had problems making inferences about the speaker’s communicative intention, tending to rely more on the propositional content of the utterance to work out its meaning. Notably, Reilly and Muzekari also used a control group of normal children with a mean age of 8 years, and found that these gave very similar answers to those of the patients. This may be significant given that a number of more recent studies using specially designed tests of theory of mind ability (e.g. Perner & Wimmer, 1985) suggest that normal children may only begin to understand second-order mental state attributions around the age of 7 years. Thus, the relatively poor performance by 8-year old children on Reilly and Muzekari’s task is consistent with a task analysis in terms of second-order theory of mind. Finally, in a study designed to test understanding of irony in schizophrenia explicitly within the framework of Frith’s model, Mitchley (1993) presented chronic schizophrenic patients and psychiatric controls with written scenarios, half of which contained a target utterance meant in an ironic sense, and the other half of which contained an utterance that was meant literally. In line with Frith’s model, the schizophrenic patients were signif-
icantly more likely than psychiatric controls to misinterpret the ironic utterances literally, but were as good as controls at correctly interpreting the literal utterances. This difference remained when current IQ was controlled, and there was evidence that impairment on the task was associated with the presence of negative signs, but not positive symptoms. This is partially consistent with Frith’s theory, in the sense that he would predict the poorest performance from patients with negative features; he might also have predicted subtle deficits in patients with positive symptoms, but no such deficits were apparent in this study.

Irony is often used to convey humour, and in order to appreciate a joke we need to infer that a speaker or cartoonist intends to amuse us. Within Frith’s model, this suggests that schizophrenic patients should be impaired in the appreciation of humour because of their difficulties in inferring others’ intentions. Importantly, any studies investigating this prediction should always use a control group of depressed patients in order to rule out any impairment associated with secondary depressive features of the schizophrenic illness. An early study by Senf et al. (1956) asked acute and chronic schizophrenics and depressed controls to explain various cartoons. The patients’ explanations were analysed along dimensions such as ‘action’ (accurate description of the depicted action), ‘social roles’ (accurately labelling the relationships between the various characters in the story) and ‘motivation’ (discussing the desires and goals of the characters). It is likely that this task tested theory of mind ability at two levels: firstly, the subject had to infer an ‘intention to amuse’ on the part of the cartoonist, and secondly he had to infer particular mental states of story characters in order to give appropriate ‘motivation’ explanations. In line with Frith’s model, the chronic patients (who may be assumed to have had a number of negative behavioural signs) were significantly worse than acute schizophrenics and psychiatric controls at appreciating the purpose of the cartoon as a vehicle of humour, tending to see it instead as “a simple statement of a prosaic situation in which nothing is especially out of the ordinary.” (ibid., p. 50). The chronic patients were also signif-
icantly worse than all other groups at reporting the desires and goals of the cartoon characters, consistent with a theory of mind deficit. However, it should be noted that the chronic schizophrenics also gave poorer answers than the other groups in the 'action' and 'social roles' categories, so it is not possible to rule out generalised cognitive impairment as an explanation of the poor overall performance of these subjects.

Corcoran et al. (1997) also looked at the ability of schizophrenic patients to understand visual jokes, but in this study an attempt was made to reveal any specific theory of mind deficits by comparing patients' explanations of equally difficult sets of 'physical' jokes (whose understanding required only a behavioural analysis) and 'mental state' jokes (whose understanding required the attribution of mental states to particular characters in the scene). Frith's model was explicitly investigated here, so patients were divided into the hierarchical symptom subgroups discussed earlier (e.g. Corcoran et al., 1995). It was predicted that patients showing negative signs would have problems understanding all the cartoons (because of a failure to recognise the cartoonist's intention to amuse), but that they might show particular impairment on the 'mental state' jokes, because of the additional requirement in these cases for the subject to infer the mental states of particular characters. It was thought that patients with paranoid symptoms, on the other hand, would show more subtle deficits, probably recognising that all the cartoons had a humorous intention, but tending to misattribute particular characters' intentions and beliefs in the 'mental state' jokes. They were expected, therefore, to only be impaired in the 'mental state' condition. These predictions were largely supported, and the effects remained once current IQ had been controlled, suggesting that general cognitive impairment could not account for the results. Patients in remission scored as well as controls, (providing further evidence that an impaired theory of mind is closely related to symptomatology), but rather unexpectedly, the patients with only passivity symptoms showed a selective deficit on the 'mental state' jokes. This last result is difficult to
explain within Frith’s model because, as we have seen, the model predicts that such patients should only have problems with the representation of their own mental states (intentions), not those of other people. Corcoran et al. (1997) suggested that the result may be unreliable because of the small number of subjects in the ‘passivity’ group (N = 8), so clearly this study should be repeated with larger subject numbers.

Finally, two studies to date have investigated mentalising ability in schizophrenia using modified tests of first- and second-order theory of mind initially developed for use with normal children (Wimmer & Perner, 1983; Perner & Wimmer, 1985). These have become known as ‘false belief’ tasks, because to correctly answer a test question, subjects must understand that a story character holds a false belief about a situation. Versions of the tasks are described in full in Chapter 4 of this thesis. In a typical first-order task, the subject is told a story in which character A (often called Sally) places an object into a basket and then leaves the room. Character B (Anne) then enters the room, moves the object into a box, and leaves. The subject is asked to predict where character A will look for the object on her return, and the correct answer of “basket” requires the subject to infer that A has a false belief about the location of the object (i.e. “A thinks that . . .”). A second-order task involves similar reasoning, but in this case the subject has to infer what a story character (A) thinks about a character B’s thoughts (i.e. “A thinks that B thinks that . . .”). Bowler (1992) was the first researcher to give such tasks to schizophrenic patients, although in his study the schizophrenics were serving as controls for a group of subjects with Asperger’s syndrome (see Section 3.2 of this chapter). Bowler gave one first-order and two second-order tasks to 15 schizophrenics with negative signs and found, contrary to Frith’s hypothesis, that they performed as well as normal adults (a mean of 64.4% of the schizophrenics passed the tasks). Although IQ data were not reported for the normals in this study, it is highly unlikely that the result can be explained in terms of the patients having a higher IQ than the normals, because the mean WAIS-R (Wechsler, 1981) score of the schizophrenics was 84.7, a value more
than one standard deviation lower than the normal mean value of 100.0.

In contrast to Bowler’s results, Frith & Corcoran (1996) did find evidence for schizophrenic impairments on both first- and second-order tests of false belief and deception. Their study was designed as an explicit test of Frith’s model, so subjects were grouped according to their symptoms in the hierarchical manner described earlier (e.g. Corcoran et al., 1995). Six short stories were read out to each subject, and to aid concentration during each story, the subject looked at simple pictures depicting the action being described. After each story, subjects had to answer a ‘reality’ question (testing memory and comprehension of events in the story), and a question requiring inference about the mental state of one of the story characters. In line with Frith’s model, once current IQ and memory impairment had been controlled for, the patients with positive or negative behavioural signs, and those with paranoid symptoms, were significantly impaired on the theory of mind questions relative to normal and psychiatric controls. Patients with paranoid symptoms showed a less severe impairment than those with behavioural signs, but both groups still scored more poorly than controls on both the first- and second-order tasks. As found in the earlier study by Corcoran et al. (1995), patients with passivity symptoms showed an intact theory of mind, consistent with an impairment in the ability to represent their own, but not others’ intentions. Schizophrenic patients in remission again performed as well as controls. It is notable that the only two studies to date to have given first- and second-order false belief tasks to schizophrenic patients, have produced conflicting results. It is possible that methodological differences between the two studies can account for the discrepant findings, and in the first study of this thesis (detailed in Chapter 4), an attempt is made to explain the discrepancies. Overall, however, the large body of research discussed above provides strong evidence in favour of Frith’s model, suggesting that patients with certain signs and symptoms of psychosis do indeed have problems in attributing the mental states of other people.
**Relation to brain systems**

The above results suggest that theory of mind impairment in schizophrenia is often unrelated to general cognitive decline. We saw earlier that children with autism seem to have a specific theory of mind deficit, and this is also relatively independent of IQ (see Baron-Cohen *et al.*, 1993b, and Section 3.2 of this chapter for a review). This last observation led U. Frith *et al.* (1991) to suggest that mentalising ability may be subserved by a relatively specific brain system; this may be selectively impaired in autism, with many other functions remaining largely intact. In a similar way, it is possible that dysfunction of this neural system could underlie the particular positive and negative features of psychosis discussed in the above section. Frith & Frith (1991) pointed out that important differences in the age of onset of autism and schizophrenia may mean that theory of mind dysfunction gives rise to different clinical presentations in the two conditions; these issues will be discussed more fully in Section 3.2 of this thesis. We saw earlier that self-monitoring of one’s own actions and intentions may involve interactions between frontal brain regions and posterior regions specific to the action. As pointed out by Frith (1996), the problems associated with self-monitoring of action are very similar to those involved in interaction with other agents. Thus, by self-monitoring, we predict the results of our actions for ourselves (e.g. the resultant sensations or perceptions), and by employing theory of mind, we can predict the results of our actions on the mental states of other people. It is possible, therefore, that on a physiological level, theory of mind ability also requires frontal-posterior interactions (Frith, 1996). If this is the case, a theory of mind impairment in schizophrenia would clearly be consistent with the studies discussed earlier which suggested that both positive and negative features of the illness are associated with fronto-temporal or fronto-striatal functional disconnection (e.g. Frith *et al.*, 1995; Robbins, 1990).

A number of functional imaging studies have investigated the neural substrates of theory of mind ability in normals, and as predicted these studies have implicated
frontal and posterior brain regions. In the earliest experiment, Baron-Cohen et al. (1994) carried out a single photon emission computerised tomography (SPECT) investigation of normal volunteers as they performed a task in which they had to judge whether or not presented words had a mental state content (e.g. ‘think’, ‘expect’) or were just foils (e.g. ‘table’, ‘horse’). Relative to a control task, in which judgments were required as to whether presented words were body-related (e.g. ‘hand’, ‘artery’) or foils, the mental state task led to a relative increase in blood flow in the right orbito-frontal cortex compared to the left frontal-polar region. While this study is consistent with suggestions of a frontal involvement in mental state understanding, there were a number of methodological problems which limit its interpretation. For example, the task used did not actually require inferences to be made about others’ mental states (i.e. true theory of mind ability), but simply required knowledge of mental state terms. In addition, these terms were more abstract than the ‘body-related’ words used, so the observed activations may be related more to a dimension of abstraction than to knowledge of mental states per se. Finally, Baron-Cohen et al. used a ‘regions of interest’ analysis, concentrating only on orbito-frontal cortex and frontal polar regions, so these workers may have missed other important areas of activation.

These problems were overcome in three later studies which avoided SPECT and used the more sensitive technique of positron emission tomography (PET). Goel et al. (1995) imaged the brains of normal volunteers as they performed a control task (in which they had to infer the function of an unfamiliar object from its form), and a theory of mind task, in which they had to imagine what function Christopher Columbus would ascribe to the object based on his knowledge. Clearly the latter task required inferences about another character’s mental states, and as the control task required non-mental state inference, the PET subtraction methodology had the potential to reveal brain regions specific to theory of mind. Consistent with a neural account of mentalising in terms of frontal-posterior interactions, the theory of mind
condition in this study activated Brodmann’s Area (BA) 9 (part of the left medial frontal cortex) and the left temporal cortex relative to the control condition.

In the light of earlier discussions suggesting that the understanding of metaphor may require a first-order theory of mind (Happé, 1993), it is interesting to note the results of a PET study by Bottini et al. (1994), which investigated the comprehension, by normals, of new metaphors. Relative to a control task requiring the comprehension of sentences containing non-figurative language, Bottini et al. found that their metaphors task activated specific areas of the right hemisphere, notably prefrontal regions (BA 8 and 46), the medial temporal gyrus, right anterior and posterior cingulate, and precuneus (BA 31). This is again consistent with an account of mentalising in terms of frontal-posterior interactions. Interestingly, the activation of precuneus (which is an area implicated in the retrieval of information from episodic memory (Andreasen et al., 1995; Shallice et al., 1994)), suggests that metaphor comprehension may require both theory of mind and the application of episodic memory. Thus, to solve a metaphor task we need to infer an intention behind the utterance and apply context-specific knowledge of our own to make sense of it.

In perhaps the best controlled imaging study of theory of mind so far, Fletcher et al. (1995) used PET to study the brain activity in normal volunteers while they performed story comprehension tasks which required inferences about the mental states of story characters. Subjects also carried out equally difficult comprehension tasks requiring non-mental state inferences about very similar situations to those given in the theory of mind condition. Results showed that, relative to the control stories, the theory of mind condition activated the left medial frontal gyrus (mainly in BA 8 but extending into area 9, very close to the areas activated by Goel et al. and Bottini et al. in the above studies) and the posterior cingulate cortex (BA 23/31). As discussed by Fletcher et al. (1995), several studies have implicated the posterior cingulate in memory function, and Shallice et al. (1994) suggested that it may be
particularly involved at the encoding stage. The greater activation of this region during the theory of mind condition may therefore have reflected a greater requirement for the encoding of cohesive narrative structure in these stories. This analysis suggests that BA 8 (the only other area which showed significant activation relative to the control condition) may be the brain region which subserves the representation of others’ mental states. As this region is known to have widespread connections to parietal, temporal, occipital and anterior cingulate cortices (see Fletcher et al., 1995), it is possible that theory of mind inference involves interactions between BA 8 and regions specific to the inference being made. Thus, for example, an inference of the form, “He intends to run away” may involve interactions between BA 8 and regions involved with the representation of movement (Frith, 1992). Similarly, mental state inferences based on others’ facial expressions, gaze or affect might involve interactions between BA 8 and the structures thought to store information about faces and gaze (superior temporal sulcus) and emotion (the amygdala) (Frith, 1992). Notably, this analysis is very similar to that provided earlier for self-monitoring of action, where it was suggested that monitoring involves interactions between frontal areas (from where corollary discharges and willed intentions may originate), and posterior regions specific to the initiated action. It is possible that the severity of theory of mind impairment in schizophrenia reflects the degree of functional disconnection between BA 8 and posterior brain structures. Thus, patients with negative features such as affective blunting and social withdrawal, who are presumed to lack a theory of mind, may have almost complete functional disconnection, being unable to relate others’ mental states to relevant information stored in posterior regions. Patients with paranoid symptoms, on the other hand, may have only an intermittent and less severe functional disconnection, being able to relate others’ thoughts to relevant information, but frequently making errors in those inferences (Frith, 1994).
A synthesis of Frith’s ideas

Frith’s model, as described so far, has accounted for some of the positive and negative behavioural signs of schizophrenia in terms of impairments in the willed action system, so that negative signs reflect problems in the generation of action, while positive signs reflect an inability to inhibit ongoing action sequences or stimulus-driven actions. At the same time, Frith has explained passivity symptoms such as delusions of control and thought insertion in terms of impaired monitoring of willed action (through a failure to represent one’s own willed intentions), and he has accounted for a number of other behavioural signs and positive symptoms in terms of impaired representation of others’ mental states (theory of mind). To achieve a possible synthesis of these ideas in terms of only one cognitive system, Frith (1992; Frith & Frith, 1991) suggested that the generation of willed action and inhibition of inappropriate action depend crucially on the representation of one’s own goals and plans. Thus, in Figure 3.1 at the start of this chapter, it is proposed that for a goal or plan to be translated into a willed intention, or for the goal or plan to be used to inhibit a stimulus intention, the subject must be aware of the goal - he must know what he wants to do. By this analysis, the positive and negative behavioural signs previously explained in terms of impairments in the willed action system, can be rationalised in terms of impaired representation by the patient of his own goals. By extension, it is therefore possible that all the signs and symptoms discussed previously reflect deficient representation of mental states (goals, intentions, beliefs etc.) of oneself or other people (Frith, 1992).

In support of his contention that willed action requires the representation of goals, Frith (1992) cited theoretical work by Perner (1991) from the normal developmental literature. On the basis of research with infants, Perner suggested that below the age of 2 years, children can show goal-directed activity (such as searching for a sweet which is hidden in either a red or a blue box), but have no awareness that they
have that goal. The child is able to learn by trial and error that the sweet is always in, say, the red box, so develops a response pattern whereby he always opens the red box. If, out of sight of the child, the sweet is then placed consistently into the blue box, the child gradually modifies his responses by trial and error, so that eventually he always opens the blue box. In effect, he shows a response pattern characterised by initial perseveration to the original position (Perner, 1991). As we saw earlier, such perseveration has been ascribed by Frith (1992) to impairment of the willed action system. When the search task is given to children over the age of 2 years, they show surprise on the first trial in which the red box is found to be empty, and soon start to open the blue box on every trial, showing little sign of perseveration to the old position. Perner’s (1991) analysis of this result (cited by Frith, 1992), is that children older than 2 years are aware of their goal, so on the first trial in which the red box is empty, they know that they have not achieved that goal. As a result, they quickly modify their strategy and begin responding to the new position. Children younger than 2, however, fail to represent their goal, so show no surprise when the position of the sweet changes, and only modify their response pattern gradually. This analysis implies that representation of one’s own goals may be necessary for efficient operation of the willed action system. Frith’s suggestion (see Figure 3.1) that the representation of goals is necessary for the formation of willed intentions and the inhibition of stimulus intentions, is consistent with other results which imply that, developmentally, normal children can represent goals before they can represent their own intentions. For example, Shultz et al. (1980, cited in Frith, 1992) showed that children aged 5 years could distinguish between an intentional leg movement and a reflex elicited by the experimenter, but children who were only 3 years old could not make this distinction. Together with the results from the sweet-searching task, these data imply a developmental decalage in representational understanding (i.e. goals represented at 2 years, intentions at 5 years), and are consistent with Frith’s model of willed action, in the sense that he makes representation of goals a prerequisite for the representation of willed intentions. Frith (1992) suggested that in the sweet-
searching task, the most efficient performance would be shown by a child who could represent both his goals and intentions, because on the first trial in which he found no sweet, the child would be aware both that he had not achieved his goal, and also that his previous intended action was responsible for this failure. Such awareness would facilitate a shifting of response to the alternative position.

The search task discussed above is an example of an executive function task in that the subject must shift his response set and inhibit a prepotent (but now incorrect) response in order to learn a reversal. Frith’s analysis of this task in terms of a requirement for representation of goals and intentions therefore suggests that the impaired executive function shown by many schizophrenic patients with positive or negative behavioural signs (see Chapter 2), can be explained in terms of deficient mental state representation. It should be noted at this point that Russell (1996) has argued for a rather different interpretation of the literature, suggesting that mental state representation need not, in fact, underlie executive function. Russell’s thesis is that normal mental state understanding springs from the development of executive function which itself is one aspect of ‘agency’, the child’s first person experience that it is an agent acting in the world. Russell argues that one of the integral features of agency is ‘action monitoring’, which he defines as “locating the cause of altered inputs in one’s body rather than in the world” (ibid., p. 76). He suggests that it is the development of action monitoring which enables the child to regulate its behaviour in order to achieve goals and make judgments about situations (i.e. executive function), and that this experience leads to an understanding that other people also have mental states such as goals. A crucial difference between Frith’s and Russell’s hypothesis is that Russell suggests that action monitoring, in itself, does not require representation of mental states whereas, as we have seen, Frith proposes that the monitoring of willed action does involve the representation of goals and intentions. While these two positions are still very much open to debate, it is less than clear from Russell’s account how an implicit experience of one’s own agency can lead to an
explicit understanding of others’ mental states. Perhaps a more plausible account would be that one explicitly understands others’ goals, intentions etc., at around the same time developmentally that one explicitly represents one’s own goals or intentions. In line with Frith’s account, this may be the time at which the child begins to show signs of inhibiting perseverative responses on tests of executive function such as the reversal task discussed above.

Frith’s analysis of willed action will be accepted for the purposes of this discussion, and the utility of his explanation of certain behavioural signs in terms of impaired representation of goals will be examined specifically for the case of speech abnormality in schizophrenia. We saw earlier that poverty of speech, distractible speech, and some examples of poverty of content (e.g. repetitive, stereotyped speech) are explained well in terms of deficits in the willed action system. Thus, poverty of speech reflects an inability to generate utterances, whilst distractible or stereotyped speech reflect difficulties with the inhibition of inappropriate comments or earlier utterances. Such deficits are easily incorporated into the model of mental state representation by ascribing them all to problems with the representation of goals in speaking. Thus, a patient who wants to say something, but fails to represent that goal, will either not say anything at all (poverty), or will produce stimulus-elicited (distractible or stereotyped) speech inappropriate to their goal. Within this framework, it is possible that some patients do represent their goals, but do so less effectively than normals. A manifestation of this impairment may be speech which is generally relevant to the task in hand, but in which information is conveyed in an inefficient way, with no ‘editing out’ of ambiguous phrases. Such utterances were produced by schizophrenic patients in Cohen’s (1978) study described earlier, in which subjects had to accurately describe a coloured disk to a listener. As cited by Frith (1992, p. 105), a typical schizophrenic speaker in that study, when describing a light green disk, produced the utterance, “Clean green. The one without the cream... it looks like moss, boss”. Frith pointed out that such utterances consisted of a
series of inadequate phrases, and he suggested that patients may have recognised the inadequacy of each phrase after it had been emitted (i.e. with external feedback), and therefore produced another phrase in an attempt to improve the situation. However, a failure to efficiently monitor the goal of the task may have meant that inappropriate information in these subsequent phrases was not 'edited out' before speaking, so that each phrase added little information of relevance.

A recent study by Leudar et al. (1994) looked at the relationship between symptoms and the performance of schizophrenic patients on a task similar to that used by Cohen. In Leudar et al.'s experiment, subjects carried out the 'reporter test', in which they had to describe the actions of the experimenter so that they could be performed accurately by an imaginary listener. The self-repair of subjects (i.e. their recognition and correction of speech errors), was studied by recording whether they interrupted their speech flow early (e.g. within an error word) or late (e.g. after the error word had been emitted). In terms of Frith’s analysis discussed above, early interruptions may reflect internal monitoring of goals, whereas late repairs may reflect monitoring solely on the basis of external feedback. Results of the study showed that schizophrenic patients (who all had some negative behavioural signs) made significantly less within error-word interruptions than normal controls, and were more likely than controls to interrupt sentences after error words had been emitted. This is consistent with Frith’s suggestion that negative behavioural signs are associated with a problem in the self-monitoring of goals, and in fact Leudar et al. concluded that internal monitoring of goals (or ‘phonetic plans’ as they called them) was not completely absent, but was less efficient in the patients than in controls. It should be noted that the same pattern of results was shown by a subgroup of Leudar et al.’s patients who reported mainly auditory hallucinations (and showed only a few negative signs). As other studies (discussed earlier in this chapter) suggest that auditory hallucinations are associated with impaired representation of one’s own intentions, this result may reflect a reliance of early self-repair on the monitoring of
both goals and intentions during speech. Finally, we might expect poor self-repair of speech to be associated with incoherence of speech, so it would be useful to repeat Leudar et al.’s experiment with an explicit investigation of the relationship between task performance and scores for positive behavioural signs rather than negative signs.

**Impaired metarepresentation in schizophrenia**

As the above synthesis has shown, Frith’s model proposes that many signs and symptoms of psychosis are explicable in terms of impaired representation of one’s own or others’ mental states. Such representation is a crucial component of self-awareness, and as it involves the representation of representations (e.g. knowing that I want something or thinking that he believes something), it has been termed ‘metarepresentation’. In Frith’s (1992) terminology, then, schizophrenia involves disordered self-awareness due to impaired metarepresentation. As metarepresentation is clearly a high-level conscious process, Frith’s model ties in well with one of the major conclusions from Chapter 2, namely that schizophrenic patients show marked deficits in conscious, controlled processing. Thus, delusions of control, for example, involve the patient failing to consciously represent his own intention to act, and consequently losing the ‘awareness of effort’ usually associated with self-generated action (Frith, 1992). It is important at this stage to clarify the role of corollary discharges within Frith’s model. As we saw earlier, these signals are generated when actions are initiated, and they enable a system to predict the perceptual outcomes of those actions (Sperry, 1950). Such signals must be able to operate in the absence of metarepresentation, as they are known to occur in animals which lack a metarepresentational capacity, such as monkeys (Ploog, 1979; Robinson & Wurtz, 1976) and insects and fish (see Feinberg, 1978, for a review). One possibility is that in animals, corollary discharge simply involves representation of the predicted perceptual change resulting from an action. While this is a representation that is not derived directly from external stimuli, it is not a metarepresentation (i.e. a represent-
ation of a representation). In humans, however, Hershberger & Misceo (1983, cited in Frith, 1992) have suggested that corollary discharge can occur either in an automatic, unmonitored form or in a conscious, monitored form. The former may be analogous to the signal found in animals, whereas the latter may involve conscious awareness that an action has been initiated, a process which involves metarepresentation. As Frith’s model proposes impaired metarepresentation in schizophrenia, it is likely that the impaired monitoring of initiated action thought to be associated with passivity phenomena and auditory hallucinations, involves dysfunction of the normally conscious form of corollary discharge. This leaves open the possibility that the automatic, unmonitored form of corollary discharge may be intact in schizophrenic patients. Although not directly tested, this is plausible, because a failure at the automatic level would be likely to leave a patient very disoriented, with problems in functioning at even the most elementary levels. Clinical experience tells us that, on the contrary, many schizophrenic patients often report their symptoms against a background of generally intact functioning.

The cognitive mechanism of metarepresentation

Frith’s (1992) model suggests impaired metarepresentation in schizophrenia, but in cognitive terms, the precise nature of the metarepresentational mechanism was first explored by Leslie (1987; 1994a) in the normal developmental literature. Leslie suggested that an early sign of mental state representation may be found during the second year of a child’s life when it begins to show pretend play, and shows evidence of understanding pretense in others. To understand its mother when, for example, she speaks into a banana as though it were a telephone, the child has to infer the intention behind its mother’s action, so has to metarepresent (Leslie, 1987). Leslie pointed out that pretense cannot involve manipulation of two primary representations (e.g. ‘banana’ and ‘telephone’), because this would easily lead to confusion in the child’s knowledge of the world. Instead, he suggested that pretend representations must be
‘marked off’, or ‘decoupled’ from primary representations, so that both primary and pretend representations (e.g. ‘banana = yellow fruit’ and ‘banana = telephone’) could co-exist without confusion. He proposed that this decoupling is achieved by a domain-specific cognitive system or ‘module’ (the Theory of Mind Mechanism or ToMM; Leslie & Thaiss, 1992; Leslie, 1994a) specialised for the representation of mental states. Within this model, the metarepresentation^{3} computed by ToMM^{4} is a data structure providing an agent-centred description of the current situation, in which the agent takes an attitude towards a proposition. For the above case of pretense, this would take the form:

I (or She) PRETEND “this banana is a telephone”.

Here, the contents of the quotation marks (the proposition) are false in reality, but the decoupling of the proposition within the metarepresentation means that the child can simultaneously represent a banana as itself and as a telephone, without confusion. As discussed by Leslie (1987, p. 416), this “suspension of normal reference relations” occurs whenever a mental state term is used, (e.g. I WANT “the sun is shining”; She BELIEVES “it is raining”), because in all cases of mental state usage, it is unimportant whether the contents of the quotation marks are true or false in reality. The importance lies in the attitude that the agent takes towards the proposition, as it is this attitude which enables us to understand the agent’s action or to predict his future action in a given situation. Thus, Leslie (1987) suggested that development of

^{3} Leslie & Thaiss (1992) subsequently replaced the term ‘metarepresentation’ by ‘M-representation’ in response to a theoretical discussion prompted by Perner (1991). However, the term ‘metarepresentation’ will be maintained throughout this thesis to mean representation of mental states, as conceptualised by Frith (1992) and Leslie (1987).

^{4} More accurately, the discussion in this thesis relates to ToMM system_{2} as conceptualised by Leslie (1994b).
the ToMM module is responsible for the normal child’s emergent understanding of all mental states, and can explain, for example, the widely replicated ability of normal 4-year olds (but not 3-year olds) to pass first-order false belief tasks (e.g. Astington & Gopnik, 1991).

Within Frith’s (1992) model of schizophrenia, impaired metarepresentation can be thought of as dysfunction of the ToMM module, so that the patient has difficulty in constructing decoupled representations. In all cases, the patient will have intact primary representation (in the above example, knowledge that a banana is a yellow fruit), but metarepresentation will be impaired so that, in effect, he has difficulty representing two things at the same time (e.g. a banana as a yellow fruit and as a telephone). In the most severe cases, metarepresentations will not be formed at all so that, for example, a patient will see someone else’s facial movements (primary representation), but will fail to interpret them as intentions to engage in communication; as a result, he will show negative signs such as social withdrawal or poverty of speech. In less severe cases, metarepresentations will be formed, but incorrectly, so that, for example, the patient interprets a person’s facial expression as, ‘She THINKS “I am stupid”’ (when in fact the person had no such belief), and as a result experiences positive symptoms such as delusions of reference or persecution. One form of dysfunctional metarepresentation might involve complete detachment of an agent’s attitude (mental state) towards a proposition, leaving a data structure consisting only of the proposition itself (Frith, 1992; 1994). Thus, a third person auditory hallucination of the form “he’s something special”, might reflect a ‘free-floating’ proposition due to breakdown of the metarepresentation “He THINKS “he’s something special””, formed by the patient in reference to someone else (Frith, 1994). Similarly, the earlier conceptualisation of delusions of control as a failure to represent one’s own intentions to act, can be thought of as a breakdown of decoupling in a metarepresentation such as ‘I INTEND “my arm is lifting up”’. In this case, the ‘free-floating’ proposition corresponds to the experience of one’s arm lifting unintent-
ionally. Leslie’s framework suggests a possible link between metarepresentation and the brain which is compatible with the imaging studies presented earlier in this chapter. We saw earlier that evidence suggests frontal-posterior interactions as being crucial to metarepresentation, with the mental state information (e.g. ‘He THINKS’) being possibly represented in frontal regions, and information specific to the inference being represented in posterior regions (Frith, 1992). Within Leslie’s model, therefore, the attitude of an agent towards a proposition may be frontally mediated, with the content of that proposition being subserved by posterior regions specific to the nature of the proposition; full metarepresentation would then depend upon functional interactions between these two areas (Frith, 1992; 1994).

A possible extension of Frith’s model to memory systems

Theory and empirical support

In Figures 3.1 and 3.2, long-term memory (LTM) is shown as having some input into the willed action system, and one obvious way in which this might be useful is in the provision of context, so that the notion of ‘appropriateness’ of an action can be defined. Only with this knowledge is it possible to generate actions which are appropriate to a situation, or to inhibit actions which are inappropriate. In Figure 3.2, no provision was made for the monitoring of LTM, but when we consider that one function of the monitor is to distinguish between internally- and externally-generated action, it is likely that a similar monitor must apply to memory; only with monitoring of memory would we know, for example, whether stored concepts were self-generated (e.g. our own original ideas) or externally-generated (e.g. ideas provided by a teacher). In terms of metarepresentation, monitoring of memory can be thought of as the representation of knowledge, so that a subject knows that, or how, he knows something (e.g. ‘I KNOW “X”’), in the same way that representation of goals involves a subject knowing that he wants to do something (‘I WANT “X”’).
As Frith’s model proposes impaired metarepresentation in schizophrenia, we might expect some patients to show impaired monitoring of their own knowledge. This may manifest as impaired *source memory* with, for example, the patient having difficulty in deciding whether particular words had been self-generated in an earlier session, or had been provided by the experimenter, a task which normal people find relatively easy (Johnson et al., 1993). This prediction was supported by a study from Bentall et al. (1991), in which patients were asked to generate words from a particular category and also to read out words (provided by the experimenter) from the same category. One week later, subjects were presented with all the words from the previous session, and had to say for each one whether it had been self-generated or not. The psychotic patients with all types of signs and symptoms performed worse than normal volunteers on this task, and there was a tendency for patients with auditory hallucinations to say that self-generated words had in fact been provided for them on the reading list. In a similar study, Frith et al. (1991c) asked schizophrenic patients to generate items from a particular category; the subject then listened while the experimenter generated more items from the same category. After ten minutes, words were read out, and the subject had to say for each one whether it was new, or had been self-generated or experimenter-generated. Again, schizophrenic patients performed poorly on this task, but this time those with incoherence of speech scored particularly badly. Thus, studies of source memory do suggest that some schizophrenic patients are impaired, but the relationship between task performance and symptomatology remains equivocal and requires further research. One possibility is that the different associations between performance and symptoms in the above two studies reflected the slight differences in the paradigms used; a useful replication, therefore, would involve administration of the two paradigms to the same patient sample within a single study.

The representation of one’s own knowledge and experiences, or the subject ‘knowing that he knows’ something, was the definition used by Tulving (1983) for
episodic memory. Thus, according to Tulving, a subject's episodic memory involves 'autonoetic consciousness', with the subject representing his own act of experiencing certain past events. For example, memory of what one ate this morning for breakfast may be accompanied by recollective experience of the situation at breakfast time (e.g. how one felt, the weather, etc.). In this case, the memory consists of a particular piece of information (a type of food), together with an associated context, and in general, episodic memory can be thought of as information stored together with details about the context in which it was acquired. In terms of metarepresentational theory, we might speculate that episodic remembering involves the construction of metarepresentations such as 'I KNOW "I had cornflakes for my breakfast" (because the sun was shining at the time)'. In this case, the subject is certain that he ate cornflakes (i.e. he knows that he knows) because he can recall the context associated with the particular situation. Within Frith's model, it is possible that an impairment in the ability to represent knowledge in this way could underlie schizophrenic patients' poor performance on episodic memory tasks (e.g. Tamlyn et al., 1992; see Chapter 2). Calev (1984) showed that chronic schizophrenics were worse than controls at both free recall and recognition, but that free recall was particularly impaired. As Tulving (1983) has pointed out, free recall depends much more heavily than recognition on the availability of 'episodic trace information', (i.e. information about the subjective experience of seeing particular items on a word list), because in free recall no explicit retrieval cues are given.

Similarly, as we saw in Chapter 2, Huron et al. (1995) found that schizophrenic patients showed a selective impairment compared to controls in the ability to consciously recollect whether words had been previously presented (as measured by 'remember' responses). Their ability to recognise words associated with a feeling of familiarity in the absence of recollective experience (as measured by 'know' responses) was not compromised. By definition, 'remember' responses in this task required recollection of the subjective experience of seeing words on the list, so
relied heavily upon episodic trace information (and by extension, therefore, may have required metarepresentation). In contrast, ‘know’ responses can be thought of as relying only on ‘primary representations’ (e.g. feelings of familiarity) without any awareness of contexts associated with particular word presentations. As interpreted here, these results are all consistent with the proposed impairment in schizophrenia in the representation of own knowledge and experience. Regarding associations with symptomatology, the study of Tamlyn et al. (1992) suggested that particularly poor episodic memory was found in patients with negative or positive behavioural signs. In light of this, it is interesting to recall that Frith et al. (1991c) found positive behavioural signs (e.g. incoherent speech) to be associated with poor source memory which, as we have seen, may also involve deficient monitoring of one’s own knowledge.

On the basis of the above discussion, we might speculate that impaired metarepresentation underlies a number of other experimental findings discussed in Chapter 2. For example, Done & Frith (1984) found that schizophrenic patients (especially those with negative signs) were poor at using context to guess an appropriate word to complete sentences such as, “Coming in, he took off his . . .”. Similarly, de Silva & Hemsley (1977) showed that both acute and chronic patients were impaired on the ‘Cloze’ procedure, involving the filling in of missing words in passages of text. In both of these cases, subjects had to use long-term memory to generate context-appropriate responses, and we might explain the poor performance of schizophrenic patients at two levels within Frith’s model. Firstly, patients may have difficulty representing their goals when performing this type of task, so either fail to generate a response at all, or fail to inhibit the first (often inappropriate) response which occurs to them. On the other hand, patients who are, perhaps, less cognitively impaired, may be able to represent their goals, but have difficulty in the representation of their own knowledge. It is likely that task performance involves retrieval of information from the semantic network (which, as discussed in Chapter
2, seems to be largely intact in schizophrenia). Thus, in the above sentence-completion example from Done and Frith's study, subjects may generate several possible responses from semantic memory, such as 'coat' or 'Wellington boots', either of which could be considered appropriate. However, in order to choose between these possible responses, and produce an optimal response on the task (i.e. one rated as highly appropriate by an independent judge), it is likely that subjects need to be simultaneously aware of the contextual constraints of the sentence while retrieving the words. This may require metarepresentation, so that for the above example, a subject who has generated 'coat' or 'Wellington boots' as possible answers, might simultaneously represent 'I KNOW "The answer is not Wellington boots" (because people usually take off coats rather than Wellington boots when entering rooms)'; as a result he would be likely to produce the highly appropriate response of 'coat'. In terms of the discussions earlier in this chapter, the various apparently 'appropriate' items retrieved from semantic memory can be thought of as 'primary representations', whereas the simultaneous representation of one of these items as 'inappropriate' (i.e. Wellington boots in this example) requires metarepresentation. At one extreme, we can think of the metarepresentation in these tasks as involving episodic remembering, so that, for example, the subject thinks back to particular occasions on which he himself entered a room or observed someone else doing so, and concludes as a result that coats are much more commonly removed in this situation than are Wellington boots. In most cases, however, subjects will not need to employ episodic memory to carry out the task, but will simply represent a particular contextual constraint at the same time as they retrieve information from semantic memory; this will be generally sufficient to produce optimal responding.

This analysis is obviously speculative, but it provides a link by which impaired representation of knowledge can explain the poor performance of schizophrenic patients on context-dependent tasks. Thus, patients lacking the ability to metarepresent will be unable to simultaneously represent contextual constraints while
generating responses from semantic memory, and as a result will be more likely to produce inappropriate responses. This model would predict that some schizophrenics should be differentially impaired in the generation of responses in a context condition, relative to a no-context condition, and this prediction will be investigated in Chapter 5 of this thesis.

**Relation to brain systems**

We have seen already that many studies suggest that frontal and posterior brain regions, and the interactions between them, are required for the representation of others’ mental states or one’s own goals and intentions; we might expect, therefore, that the representation of one’s own knowledge involves a similar distributed brain system. Although animals are thought to have little, if any, metarepresentational capacity, some animal tests of memory and learning require the use of internal representations to guide responses. As such representation may be an evolutionary precursor of metarepresentational ability (Frith, 1992), lesion studies using these types of task will be discussed briefly here. In conditional learning tasks, the correct response depends upon context, so that a subject has to learn a rule such as, ‘if there are two red plaques, search under the right one for reward; if there are two green plaques, search under the left plaque’. Here the positional response is determined solely by internally represented information so, for example, a response to the right is associated with the colour red. Stamm (1973) found from work with monkeys that performance on such tasks is impaired by frontal or hippocampal lesions. Similarly, Eacott & Gaffan (1992) found that in rhesus monkeys, visuomotor conditional learning (stimulus A = motor response X; stimulus B = motor response Y) required intact frontal cortex, inferotemporal cortex and the connections between these areas. Interestingly, an auditory analogue of this task recruited frontal and superior temporal cortex rather than inferotemporal regions. As information about visual and auditory stimuli is thought to be stored in inferotemporal and superior temporal regions respectively, this suggests (by analogy with earlier discussions) that
conditional learning involves interactions between frontal cortex and posterior regions specific to the nature of the task.

Work with human subjects supports these conclusions. For example, Petrides (1985) found that patients with left or right frontal lesions were impaired on both spatial and non-spatial conditional learning tasks, whilst those with hippocampal lesions showed deficits that were material-specific and varied with the side of the lesion. In particular, right hippocampal damage was associated with impaired spatial learning, and left hippocampal lesions were associated with non-spatial deficits. Petrides concluded from these results that conditional learning in humans is subserved by a neural circuit that includes the frontal cortex and hippocampal regions specific to the task being used. Notably, in a recent functional imaging study of conditional learning in normal volunteers, Petrides et al. (1993) found that medial frontal regions were highly activated during task performance. As we saw earlier, Fletcher et al. (1995) found evidence for an association between medial frontal activity and theory of mind understanding. If an analysis of conditional learning in terms of the representation of knowledge is correct, this suggests that medial frontal regions (e.g. BA 8) may have a role in the representation of both others’ mental states and one’s own knowledge. It is intriguing, therefore, that in a number of studies discussed by Zelazo & Frye (1996), the performance of normal 3- and 4-year old children on false belief tasks was found to correlate with their scores on non-social tests of conditional reasoning. Finally, a number of PET functional imaging studies of episodic memory in normal human volunteers have also suggested the involvement of frontal and posterior brain regions. For example, Shallice et al. (1994) investigated acquisition and retrieval from verbal episodic memory, with subjects being required to learn, and then recall, paired associates consisting of categories and relatively uncommon exemplars of those categories (e.g. furniture - sideboard). The use of appropriate control tasks enabled the subtraction of semantic memory activations, so that episodic encoding was found to specifically activate the left dorsolateral
prefrontal cortex and its reciprocal connections to hippocampus (in the retrosplenial region), whilst retrieval activated the right prefrontal cortex and medial precuneus.

A possible extension of Frith’s model to explain ‘poor insight’ in schizophrenia

Because of the association between metarepresentation and self-awareness, disordered metarepresentation may be a useful concept in explaining the lack of awareness of illness often shown by schizophrenic patients. Studies suggest that this ‘poor insight’ is particularly common in patients diagnosed with schizophrenia compared to those with other serious mental illnesses such as major depressive disorders (e.g. Amador et al., 1994). The degree of insight is generally thought to lie on a continuum, and David (1990) has argued that it may comprise three different, but overlapping dimensions: the ability to relabel unusual mental events (e.g. hallucinations) as pathological; the recognition by a patient that he is suffering from a mental illness; and treatment compliance. The notion that insight is not a unitary construct has been supported by a number of correlational studies (e.g. David et al., 1992).

Two broad accounts of variation in insight have emerged in the literature, and these might be called ‘cognitive deficit’ and ‘motivational’ theories (see Amador et al., 1991, for a review). The cognitive deficit models are most closely allied to Frith’s model of psychosis in that they propose that brain dysfunction gives rise to a deficit of self-awareness. They draw support from studies of anosognosia (lack of awareness of disease) in neurological patients, which suggest that damage to right parietal areas, or frontal lobe dysfunction, may cause deficits in awareness (McGlynn & Schacter, 1989; Stuss & Benson, 1986). It may be that functional interactions between frontal and posterior brain regions are necessary for full self-awareness, which of course concurs with Frith’s view of the neural substrates of metarepresentation. Cognitive deficit models would predict impaired insight to be associated with poor performance
on neuropsychological tests of frontal or parietal function, and in particular, Frith’s model might predict an association between insight and executive function, given that the latter construct can be conceptualised in terms of representation of one’s own mental states (see earlier sections of this chapter). Several studies have investigated these predictions, but the results have been equivocal. For example, with current IQ controlled, Young et al. (1993) and Lysaker & Bell (1994) found significant associations between measures of poor insight in schizophrenia and increased numbers of perseverative responses on the Wisconsin Card Sorting Test (WCST) of executive function. However, with a large battery of neuropsychological tests (including measures of executive function), Cuesta & Peralta (1994) found no relationship between impaired insight and poor task performance; in fact, poor insight was associated with better performance on various tests of memory in that study.

Within Frith’s model, these results as a whole provide some support for an association between poor insight and impaired representation of one’s own mental states. However, an examination of the relationship between symptomatology and measures of insight suggests that a full explanation may be more complex. As we have seen, Frith’s (1992) hypothesis explains a number of behavioural signs in terms of impaired representation of one’s own goals, whereas many positive symptoms (e.g. paranoid delusions) are explained by impaired representation of others’ mental states. If insight is conceptualised solely in terms of self-awareness, we might expect from this analysis that patients with behavioural signs should have the poorest insight. Some studies have found such an association (e.g. Kemp & Lambert, 1995), but this finding is by no means common. For example, Cuesta & Peralta (1994) and Lysaker & Bell (1994) found no association between measures of insight and symptoms, whilst Amador et al. (1994) found poor insight to be particularly associated with the severity of delusions. On this basis, therefore, it is probably inadequate within Frith’s framework to conceptualise impaired insight solely in terms of an inability to represent one’s own mental states.
Startup (1996) has recently proposed a reconciliation of the empirical findings which, as well as drawing upon 'cognitive deficit' accounts, incorporates the 'motivational' theories discussed by Amador et al. (1991). The motivational models hold that in some sense the patients are aware of their illness, but deny (both to themselves and to others) that they are ill, as a coping strategy to avoid low self-esteem or the social stigma associated with mental illness (see Johnson & Orrell, 1995). The motivational models suggest, within Frith’s framework, that patients do have some ability to represent their own mental states. Their poor insight may therefore reflect an attributional style involving overly positive self-evaluation, rather than an absence of self-awareness per se. In an investigation of the association between insight and executive function (measured by tests such as verbal fluency and the Stroop task), Startup (1996) found a quadratic relationship, with very low and very high insight associated with intact executive function, and intermediate levels of insight associated with impaired executive function. He suggested that the patients with intermediate levels of insight could best be classified within a ‘cognitive deficit’ model; these people may have deficient self-awareness because of brain dysfunction, and be unable to construct overly positive self-evaluations because of their cognitive deficits. Within Frith’s model, these may be the patients with impaired representation of their own mental states, and perhaps a predominance of behavioural signs. Startup proposed, however, that the patients in his study with intact executive function but very poor insight, could best be classified within a ‘motivational’ model; according to him, these people may have some awareness of their illness, but because of their generally intact cognitive ability, can adopt and maintain overly positive self-evaluations, giving the impression of very poor insight. In terms of Frith’s model, the intact executive function shown by these patients is consistent with a generally intact ability to represent their own mental states (e.g. goals). As we saw in Chapter 2, Bentall (1994; Bentall et al., 1994) suggested that paranoid delusions may arise from a very similar process to that proposed by Startup to be operating in his patients classified within the ‘motivational’ model. In Bentall’s terms, patients maintain self-
esteem, particularly in response to threat, by adopting internal attributions for positive events and external attributions for negative events. As a result of this, they experience persecutory ideation. This suggests that patients classified by Startup within the ‘motivational’ model should have shown marked paranoid symptomatology; it would have been interesting, therefore, if he had reported whether symptoms varied with insight in his experiment.

A recent study by Kinderman et al. (submitted) found an association in normal volunteers between poor performance on theory of mind tasks and the tendency to provide external attributions for negative events. Whilst this is a preliminary result with normals rather than patients, it suggests (in the context of the above discussion) that very poor insight could sometimes be associated with impaired representation of others’ mental states. Thus, in Startup’s study, the patients classified within the ‘motivational’ model may have had an impaired theory of mind despite being able to represent their own mental states. In light of this, it is interesting to note that in their study of psychotic patients, David et al. (1992) found that one dimension of insight was strongly associated with a measure of ‘hypothetical contradiction’ (Brett-Jones et al., 1987). This construct is defined as “the subject’s capacity to take into account another person’s failure . . . to hear the ‘voice’ or detect the [delusional] plot . . . and for this to engender self-doubt” (David et al., 1992, p. 601). Clearly this ability would require an intact theory of mind. The various speculative associations discussed above are open to direct investigation through a replication of Startup’s study, with the inclusion of theory of mind tasks, attributional style questionnaires (e.g. the ASQ; Peterson et al., 1982) and symptom measures, in addition to executive function tasks and insight scales. On the basis of the above discussions, we might expect patients with very low insight to have an impaired theory of mind, overly positive self-evaluations on the ASQ, a predominance of paranoid symptoms, and intact executive function. In contrast, patients with intermediate levels of insight should have a normal attributional style, but impaired theory of mind and executive function.
function, and a predominance of behavioural signs. Although this replication of Startup’s study will not be done in this thesis, the study discussed in Chapter 4 will attempt a preliminary investigation of the relationship between insight, theory of mind, and executive function.

In summary, Startup’s (1996) model explained the curvilinear relation between executive function and insight in terms of a trade-off between cognitive deficit and motivational factors; patients with very low insight and intact executive function were presumed to have generally intact self-awareness, but to adopt a self-protecting attributional style. In contrast, those with high insight and intact executive function were hypothesised to lack the motivation to make such attributions. Patients with intermediate levels of insight were thought to lack self-awareness because of brain dysfunction, and to be unable to implement self-protecting attributions because of their cognitive deficits. This model has the potential to explain the contradictory empirical findings discussed earlier, with some workers showing a positive correlation between poor insight and executive dysfunction (Lysaker & Bell, 1994; Young et al., 1993), and others finding a positive correlation between poor insight and intact cognitive function (e.g. memory; Cuesta & Peralta, 1994). Startup pointed out that these results can be explained in terms of the different research groups sampling patients from different regions of the executive function / insight curve. For example, Cuesta and Peralta’s patients may have been in the region of low insight, where increasing insight scores are associated with an increase in cognitive deficits, whereas the patients sampled by Lysaker & Bell (1994) and Young et al. (1993) may have come from higher insight regions where the opposite relation obtains.

Summary

We have seen throughout this chapter how impairments, at varying degrees of severity, within a precisely defined metarepresentational system can explain many
of the signs and symptoms of schizophrenia. Historically, however, the notion of impaired metarepresentation was first applied to children with autism in an attempt to explain their characteristic behavioural signs (e.g. U. Frith, 1989), and their poor performance on tests of theory of mind (reviewed by Baron-Cohen et al., 1993b). To conclude this chapter, a brief review of the case for impaired metarepresentation in autism will be presented, with particular emphasis on the clinical and theoretical similarities and differences between autism and schizophrenia. Some of the empirical results from the autism literature will be used in Chapter 4 to make specific predictions relevant to the first study of this thesis.

3.2 Impaired metarepresentation in early childhood autism

Explaining the characteristic signs

   Autism is now known to be a biologically caused disorder, although a complete picture of its biological basis has not yet emerged (see Bailey et al., 1996, for a review). It occurs more commonly in males than in females, and this sex ratio is higher in intellectually unimpaired cases (Lord & Schopler, 1987, cited in Happé & Frith, 1996). In an influential epidemiological study, Wing & Gould (1979) showed that three behavioural features (the ‘autistic triad’) cluster together to form the autistic syndrome. These are:

   1) Impaired socialization (e.g. behaving in a withdrawn, aloof, passive or ‘active-but-odd’ way);
   2) Impaired communication, both receptive (e.g. failure to understand others’ body language or the figurative aspects of others’ speech) and productive (e.g. muteness and the absence of facial expression and gesture, or various forms of inappropriate language such as verbosity or neologism);
   3) A lack of imagination shown, for example, in stereotyped and repetitive activity with a marked absence of pretend play.
Happé & Frith (1996) reviewed a number of studies in which these particular characteristics were investigated empirically. In line with Wing and Gould's triad of impairments, the DSM-IV criteria of the American Psychiatric Association (APA; 1994), define autistic disorder as "the presence of markedly abnormal or impaired development in social interaction and communication, and a markedly restricted repertoire of activity and interests" (ibid., p. 66). For DSM-IV diagnosis, such behaviour should be apparent before the age of 3 years. As discussed by Happé & Frith (1996), the condition known as 'Asperger's syndrome' falls within the autistic spectrum, but people with this disorder tend to be less aloof than those with autism, showing more social interaction and language (although language is typically verbose or pedantic). The "restricted repertoire of activity and interests" often manifests here as an obsessional interest in unusual or arcane topics.

On a psychological level, a number of cognitive theories of social impairment have been advanced to explain the characteristic autistic behaviours (see Happé, 1994a and Happé & Frith, 1996, for reviews). For example, Hobson (e.g. Hobson, 1993) proposed that autism is characterised by an innate inability to engage emotionally with others, leading to a paucity of social experience during infancy and thus to enduring social deficits. His theory remains unproven as the crucial experiments investigating affective interaction in the earliest months of the autistic child's life have not yet been carried out (Happé & Frith, 1996). Other workers have suggested that the absence of early joint attention behaviours (Mundy et al., 1993), or a failure of early imitation of facial expression (Meltzoff & Gopnik, 1993) could underlie the social impairments characteristic of autism. However, as discussed by Happé & Frith (1996), although there is empirical evidence that these deficits are indeed present in autism, their causal status is as yet unresolved.

Probably the most influential cognitive theory of social impairment in autism is the theory of mind deficit account, which suggests that Wing & Gould's (1979)
triad of impairments can be accounted for by a failure of metarepresentation - the inability of the autistic child to represent its own and others' mental states (e.g. U. Frith et al., 1991). Leslie's (1987) metarepresentational account of pretend (see earlier discussions) means that impaired mental state representation in autism is easily able to account for the absence of pretend play, and the inability of autistic people to understand pretend in others. In the domains of socialization and communication, Frith & Frith (1991) drew parallels between the behavioural signs of schizophrenia and the impairments shown by autistic people. Thus, in the social domain, autistic people may show social withdrawal and blunt affect, or socially inappropriate behaviours, and in the domain of communication, they may show poverty of speech or inappropriate language, and often fail to understand non-verbal gestures and figurative speech. As we saw for the case of schizophrenia, all of these behavioural signs can be explained in terms of an inability to represent one's own and others' mental states, so Frith & Frith (1991) suggested that this cognitive impairment may be a core deficit in autism as well as in schizophrenia.

As mentioned in Section 3.1, a major difference between autism and schizophrenia lies in age of onset, with autism being diagnosed in early childhood, and schizophrenia typically manifesting in the second or third decade of life, with an acute psychotic breakdown. Frith & Frith (1991) pointed out that these differing ages of onset can explain why a metarepresentational impairment gives rise to both positive symptoms and behavioural signs in people with schizophrenia, but only behavioural signs in people with autism. They suggested that in patients with schizophrenia, the metarepresentational system usually develops normally throughout childhood, so that the person is able to correctly represent his own and others' mental states. However, dysfunction of this system at the time of psychotic breakdown means either that the patient continues to represent his own and others' mental states, but does so incorrectly (leading to positive symptoms), or fails to represent these mental states at all (leading mainly to behavioural signs). In contrast, early childhood
autism is thought to be characterised by a marked impairment in the development of the metarepresentational system, so that the child fails to fully acquire the ability to mentalise. In its simplest form, this theory suggests that autistic people fail to represent both their own and others' mental states, so show behavioural signs; as they fail to acquire the ability to infer goals, intentions and beliefs, they will not make false inferences about others' mental states, so do not report positive symptoms (Frith & Frith, 1991).

Within this model, it is possible that some children show a delay in metarepresentational development, but do eventually acquire a limited ability to represent mental states. One manifestation of this may be an autistic child who shows an ability to employ theory of mind in some real-life situations, and indeed, U. Frith et al. (1994) found that a subgroup of their sample of autistic children did show some social behaviours (e.g. deception) thought to require mentalising ability. However, the children in this subgroup (who had better verbal and communicative abilities than the other autistic children), still showed relatively poor social adaptation for their age and developmental level. A second possibility is that some of the children who show delayed development of the ability to represent mental states may subsequently experience dysfunction of their emerging metarepresentational system, and thus report positive psychotic symptomatology. Clinically, this may appear as an autistic child progressing to a diagnosis of schizophrenia in later life. We might expect this to be a relatively rare phenomenon, as it will occur in only a subgroup of those autistic children who show evidence of emerging mentalising ability. In line with this, there are isolated reports in the literature of autistic children becoming schizophrenic in later life (e.g. Rodriguez et al., 1994). Petty et al. (1984) presented case reports of 3 children, all of whom were diagnosed as having autism prior to 30 months of age, but who by early adolescence had received a diagnosis of schizophrenia. Volkmar & Cohen (1991) examined the case records of 163 adolescents and adults with well-documented histories of autism, to check whether any characteristic schizophrenic
symptoms had been shown at any time. Although only one subject met diagnostic
criteria for schizophrenia, 40 other patients (24%) showed some evidence of
schizophrenic symptomatology. Moreover, almost 48% of the patients in this study
were completely mute, so we cannot rule out the possibility that a number of these
experienced one or more positive symptoms, but failed to report them. Finally,
Watkins et al. (1988) examined the case records of 18 children with childhood-onset
schizophrenia, and found that in the 0 - 30 month age range, 39% of the sample had
shown signs of infantile autism. In addition, onset of schizophrenia occurred at an
earlier age for those children with a history of autistic signs than for other children
in the sample.

All of these studies may be consistent with suggestions by Murray et al.
(1992) that there is a ‘neurodevelopmental’ subtype of schizophrenia characterised
by early onset, poor pre-morbid social adjustment and restricted affect. For example,
a subgroup of patients with these features (and a male:female ratio of 7:3) emerged
from a latent class analysis of 447 schizophrenic patients by Castle et al. (1994). This
subgroup contrasted markedly with a second ‘paranoid’ type characterised by later
onset, persecutory delusions and an almost equal sex ratio. It is possible that the
relatively small number of schizophrenic patients in the ‘neurodevelopmental’
category had shown some signs of autism early in their development, as their clinical
history was characterised by poor pre-morbid social skills and a predominance of
negative signs, and they were more likely to be male than female. Within Frith’s
model, these patients may have had a delayed or dysfunctional metarepresentational
system since early childhood, whereas the majority of Castle et al.’s schizophrenics
(i.e. those labelled within the ‘paranoid’ subtype) probably showed a normal ability
to represent mental states during adolescence, with impairment only appearing at the
time of their first breakdown.
Empirical evidence for impaired theory of mind in autism

As already mentioned at various points in this chapter, much empirical evidence now supports the notion of impaired theory of mind in childhood autism. Extensive reviews of the area are provided by Baron-Cohen et al. (1993b) and Happé (1994a), but a selection of studies will be presented here to give some idea of the variety of paradigms used. It should be noted that many of these paradigms have subsequently been used to investigate theory of mind in schizophrenic populations; those studies were discussed earlier in this chapter (see Section 3.1).

The first study to test autistic children on a version of Wimmer & Perner's (1983) first-order false belief task, was carried out by Baron-Cohen et al. (1985). These workers contrasted the performance of autistic children with that of control groups of normal children and mentally retarded children with Down's syndrome. The autistics were selected to be of higher mental age (MA) (e.g. mean verbal MA = 5;5) than the control children, to rule out explanations of task failure in terms of IQ, and control questions were also used to check the children's memory for the story. The results were clear: 80% of the autistic sample failed the false belief question, whereas it was passed by 86% and 85% respectively of Down's syndrome and normal 4-year old children. Perner et al. (1989) introduced a different first-order false belief task, the so-called 'Smarties task' (Perner et al., 1987) into the autism literature. In this task, the child is shown a cardboard tube, (readily recognisable as the packaging for the well-known brand of sweets known as 'Smarties'), and is asked what the tube contains. After he has replied "Smarties", the tube is opened to reveal a pencil. The child is then asked the false belief question, namely "What will your friend say is in the tube when he comes into the room?". Perner et al. (1989) found that only 15% of their autistic children answered this question correctly (by saying "Smarties"), whereas is was passed by 92% of an MA-matched control group of children with specific language impairment. Baron-Cohen (1991) used a very similar
paradigm to test autistic children's ability to represent their own mental states. Thus, instead of asking about the mental state of a friend, he posed the question, "When I first asked you, before we opened the carton, what did you think was inside?". Results showed that only 27% of the autistic children passed this task; this was a significantly poorer performance than that shown by mentally handicapped controls of lower verbal MA. Taken together, then, these data are consistent with an impairment in autism in the representation of both own and others' beliefs.

These findings have been replicated and extended in many further studies. A notable methodological feature running through a number of experiments has been use of the 'fine cuts' technique (e.g. U. Frith & Happé, 1994), which employs two very similar tasks, only one of which requires the ability to represent mental states. By analogy with the arguments of Chapman & Chapman (1973) in the schizophrenia literature (see Chapter 1 of this thesis), this technique has the potential to reveal a specific autistic deficit in mental state understanding, as the use of a non-mental state task controls for deficits in other cognitive functions such as attention, memory and general problem-solving ability. Thus, with MA co-varied, Sodian & Frith (1992) found that autistic subjects were significantly worse than normal and retarded controls at deception (i.e. lying to a 'nasty' puppet, in order to stop it opening an unlocked box, by saying that the box was actually locked), but performed as well as controls in a sabotage condition, (in which they prevented the 'nasty' puppet gaining access to the box by actually locking it). Similarly, Baron-Cohen et al. (1986) found that high-MA autistics performed significantly worse than lower-MA normal and

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5 It should be noted that a number of workers (e.g. Leslie & Thaiss, 1992; Naito et al., 1994; Perner et al., 1989) found that autistic children performed well on this type of task, but in all of these studies, the question was posed in the form "When I first asked you, . . . what did you say was inside?". As pointed out by Leslie & Thaiss (1992), this question could have been answered correctly by the autistic children simply repeating their earlier response without any awareness that that response reflected a false belief. Thus, Baron-Cohen's (1991) use of the think question seems to provide a more stringent test of representation of one's own beliefs.
Down’s syndrome children at sequencing pictures requiring mental state understanding, but performed as well (or better) than controls when the story sequences could be understood in terms of physical-causal relations, or by reference to a character’s overt behaviour.

One example of the ‘fine cuts’ method involved contrasting the child’s understanding of false beliefs and ‘false’ photographs, a distinction first used by Zaitchik (1990) in the normal developmental literature. In the Zaitchik task, an object is placed in location A, and a polaroid photograph is taken of it. The photo is then laid face down, the object is moved to location B, and the child is asked, “In the photograph, where is the object?”. This has the same structure as Wimmer & Perner’s (1983) false belief task but, rather than involving an out-of-date belief held by a story character, it involves out-of-date representation by a non-mental medium (the photo). Leslie & Thaiss (1992) predicted that if autistic children have a specific impairment in mental state representation, they should fail a false belief task but pass the ‘false’ photo task, as only the former requires representation of the attitude of an agent towards a situation (i.e. metarepresentation). This prediction was supported by Experiment 1 of their study (Leslie & Thaiss, 1992): high-MA autistic children performed almost at ceiling on the photo task, but were significantly worse at false belief. In contrast, normal 4-year old children showed similar scores on belief and photo tasks, with about 70% passing in each case. These results were replicated (Leekam & Perner, 1991) and extended using a ‘false’ map (Leslie & Thaiss, 1992; Experiment 2) and out-of-date drawings (Charman & Baron-Cohen, 1992). These latter two paradigms are employed in the first study of this thesis (see Chapter 4) to provide a stringent test of Frith’s (1992) prediction that certain schizophrenic patients should be differentially impaired in mental state representation.

By analogy with the evidence discussed earlier for impaired theory of mind in schizophrenia, empirical studies in autism have also revealed deficits in the
processing of facial affect, in the understanding and production of gestures and non-verbal signals, and in the comprehension of figurative language. For example, Baron-Cohen et al. (1993a) employed the ‘fine cuts’ technique by testing autistic children’s recognition of the facial emotions ‘happy’, ‘sad’, and ‘surprise’. In accordance with the theory of mind deficit model, the autistics were impaired relative to verbal MA-matched normal and mentally handicapped controls at the recognition of surprise (an emotion usually caused by beliefs), but showed an intact ability to recognise happiness and sadness (emotions often caused by situations rather than mental states). In a study investigating the production of gestures, Attwood et al. (1988) found that autistic people specifically lacked the types of gesture associated with mental states (e.g. expressions of embarrassment), whereas gestures used to manipulate behaviour (e.g. a signal to be quiet) were as common as in a group of mentally handicapped controls. As with research in schizophrenia, studies of eye-contact in autism suggest an abnormality, and Baron-Cohen et al. (1995) concluded that autistic people have a specific deficit, relative to normal and mentally handicapped controls, in the use of eye-direction as a cue for inferring mental states such as intentions, goals and desires. These workers suggested that this deficit may underlie the lack of joint attention behaviours in autism, so it is notable that the absence of early joint attention (proposed by Mundy et al., 1993, to be the primary impairment in autism) may itself be rationalised within a metarepresentational account of autism (see also Leslie & Happé, 1989). Finally, studies exploring the understanding of intentional speech again suggest a specific autistic deficit. Thus, in line with her analysis of metaphor and irony comprehension as requiring first-order and second-order theory of mind respectively, Happé (1993) found that autistic children who failed false belief tasks at these two levels of complexity, showed an inability to understand the corresponding figurative utterances. Similarly, by analogy with the schizophrenia research described earlier, Surian et al. (1996) showed that autistic children were impaired relative to controls at choosing utterances which conformed to Grice’s (1975) conversational maxims and to the conversational
convention of politeness. In contrast, the autistics performed as well as controls on a task requiring the selection of grammatical rather than non-grammatical utterances. Surian et al. suggested that their maxims task required an intact theory of mind, and indeed the scores of autistic children were related to their performance on a first-order false belief task. This study provides further evidence, therefore, for a specific mentalising deficit in autism.

A notable feature of research in this area is that, despite showing the marked social impairments characteristic of their disorder, a small number of autistic subjects (usually the older ones) pass theory of mind tasks. In an analysis of pooled data from a number of studies, Happé (1995) showed that these autistic ‘passers’ tend to be those subjects with a higher verbal mental age, and as discussed by Happé & Frith (1995), two possible models are consistent with these findings. Firstly, U. Frith et al. (1991) suggested that the passers may not in fact have an understanding of mental states, but are able to use experience and general IQ-dependent problem-solving skills to work out a solution to the tasks as they would for any other puzzle. The other possibility (e.g. Leslie & Frith, 1990) is that passers do have some mentalising ability (despite gross developmental delay of their metarepresentational system), enabling them to pass simple theory of mind tasks, but at a later chronological age than normal children. Such people are presumed to have only a limited understanding of mental states, so should still fail more complex theory of mind tasks. This second model is consistent with U. Frith et al.’s (1994) study discussed earlier, which revealed a subgroup of autistic children who engage in some social behaviours thought to require a theory of mind, but who are still socially impaired for their age. The developmental delay model also fits data from Baron-Cohen (1989a), who followed up his earlier study of first-order theory of mind in autism (Baron-Cohen et al., 1985) by testing the 20% of autistic passers from that study on Perner & Wimmer’s (1985) second-order false belief task. All of the autistic subjects failed this task, whereas normal and Down’s syndrome controls performed significantly better. In further
work, Ozonoff et al. (1991a, b) and Bowler (1992) found that some high-functioning autistic people, and patients with Asperger’s syndrome, were even able to pass second-order tasks, but Happé (1994b) extended these findings by showing that such passers were still impaired relative to normal adult controls on more naturalistic and complex tasks requiring understanding of concepts such as persuasion, white lies and double bluff. Within this model, then, these people may have some understanding of mental states, but have great difficulty applying this knowledge to novel, complex situations. Happé (1995) suggested that the strong association between verbal ability and theory of mind performance in autism may reflect the fact that autistic passers acquire their ability to represent mental states through verbally mediated routes not used by normals or nonautistic mentally handicapped people. It is important to note that no autistic child has yet shown normal theory of mind task performance at the appropriate age or mental age, so even within the developmental delay model, it is still possible that impaired metarepresentation is a core feature of all cases of autism (Happé & Frith, 1996).

Executive function in autism

In light of the above discussion, Frith’s (1992) suggestion that aspects of executive function may require the ability to represent one’s own goals and intentions (see Section 3.1 of this chapter), is consistent with a large body of data showing impaired executive function in autism (for a review, see Pennington & Ozonoff, 1996). As patients with impaired executive function due to frontal lobe damage are known to show perseverative and repetitive behaviour (see Chapter 2), executive deficits in autism may go some way towards explaining the restricted repertoire of activities and interests, and the stereotyped behaviour, which make up part of the DSM-IV criteria for an autistic diagnosis (APA, 1994). Empirical investigations have revealed that autistic people show specific deficits in planning ability relative to normal and mentally handicapped controls, both on the computerised Tower of
London task (Hughes et al., 1994) and on its variant the Tower of Hanoi (Ozonoff et al., 1991a, b). They are also impaired, relative to controls, at set-shifting as measured by perseverative responses on the Wisconsin Card Sorting Test (e.g. Ozonoff et al., 1991a, b; Prior & Hoffmann, 1990), a simple reversal task (McEvoy et al., 1993) and a computerised intra-dimensional / extra-dimensional set-shifting task (Hughes et al., 1994). In addition, Hughes & Russell (1993) found that autistic children were impaired relative to mentally handicapped controls at disengaging attention from a desired object and pointing to an empty location. Finally, Ozonoff et al. (1994) showed that high-functioning autistic children were impaired relative to age- and IQ-matched control groups of normals and children with Tourette syndrome, on a test of 'cognitive flexibility' involving frequent shifting of response patterns in a computerised Go-NoGo task.

Preliminary investigations of working memory (Baddeley, 1986) in autism have provided some evidence for an intact articulatory loop, with possible impairments in processes thought to involve the central executive system. For example, Bennetto et al. (1996) showed that high-functioning subjects with autism performed as well on digit span tasks as clinical controls matched on verbal IQ. Similarly, Russell et al. (1996) found that the ‘word length effect’ (a marker of articulatory loop function) was similar in autistic children and normals matched on verbal MA; that is, on a task requiring recall of previously presented words, both groups remembered fewer words when those words took a longer time to articulate (e.g. ‘helicopter’) than when they could be articulated quickly (e.g. ‘cat’). In the domain of central executive function, Bennetto et al. (1996) showed that their autistic subjects were impaired relative to matched clinical controls on tasks requiring the processing of information and its concurrent storage for later recall. For example, in a counting task, the subjects had to count aloud the number of dots of a particular colour on each of a set of cards, then subsequently recall in order the number of dots on each card. Russell et al. (1996) also found autistic impairments on this type of task, but they questioned
the specificity of this deficit, as their control group of children with moderate learning difficulties was equally impaired. They suggested that, as their tasks were predominantly a test of central executive capacity, the specific autistic deficits in executive functions such as planning discussed earlier (e.g. Hughes et al., 1994; Ozonoff et al., 1991a, b) may not reflect executive capacity limitations. Instead, they argued that problems in the cognitive inhibition of prepotent responses, or an inability to choose one from a number of possible responses, may be the primary executive impairment(s) in autism.

Interestingly, suggestions that only certain components of executive function are impaired in autism, may explain why a number of other clinical groups (e.g. subjects with early-treated phenylketonuria or attention deficit hyperactivity disorder (ADHD)) also perform poorly on executive tasks, yet show different clinical presentations to people with autism. By this argument, each clinical group may have specific impairments in different aspects of executive function, and it is these specific deficits which relate to the characteristic features of their clinical presentation. In their review of this area, Pennington & Ozonoff (1996) provided some evidence for this, pointing out that children with ADHD seem to show particular impairments on tasks requiring voluntary motor inhibition, whereas autistic people have a notable deficit on tests of cognitive flexibility such as the WCST. In the light of Russell et al.’s (1996) data discussed above, it may be that the specific executive deficits of autistic people largely involve impaired cognitive inhibition of prepotent actions, giving rise to poor cognitive flexibility. Further research is clearly needed in this area, but this proposed deficit in cognitive inhibition is certainly compatible with the notion that impaired metarepresentation underlies the executive deficits in autism. By the same argument, if inhibitory deficits at the motor output (rather than cognitive) level are a major feature of ADHD, then it is unlikely that metarepresentational impairments underlie executive failures in that condition. We saw in Chapter 2 that schizophrenic patients (especially those with behavioural signs) show a similar
pattern of executive impairment to that shown by autistics, scoring particularly poorly on tests of planning and set-shifting, and on tasks thought to tap the central executive of working memory. Thus, although researchers have not yet explored in any detail the intact and impaired components of executive function in schizophrenia, the data so far are consistent with Frith’s (1992) suggestion that impaired metarepresentation is a core feature of schizophrenia, and underlies the executive dysfunction often shown by these patients. Future research might explore these ideas by comparing the performance of schizophrenic patients with behavioural signs on tasks requiring either cognitive inhibition or motor inhibition. By analogy with the autism literature, we might expect them to be differentially impaired on the former type of task.

Memory in autism

A metarepresentational account of autism makes predictions about the memory function of subjects with the disorder. As we saw earlier in this chapter, it is possible, within Frith’s (1992) model, to conceptualise episodic memory in terms of metarepresentation. Thus, Tulving (1983) defined episodic memory as memory associated with recollective experience of context, so the subject ‘knows that he knows’ something; in Frith’s terms, this clearly involves the subject representing his own knowledge. By analogy with the schizophrenia research described earlier, we might therefore expect impaired metarepresentation in autism to give rise to poor episodic memory; subjects should also show poor source memory and memory for temporal order, as both of these require the representation of associated contextual information. A review of the autism literature provides some support for these predictions. For example, Boucher & Warrington (1976) found that autistic children were impaired relative to age-matched normals (and control children matched on verbal and non-verbal ability), on tests of free recall and forced-choice recognition. The autistic subjects did, however, perform normally on cued recall tasks, which (in Tulving’s terminology) may depend less than free recall on the availability of
‘episodic trace information’. Similarly, Bennetto et al. (1996) showed that high-functioning autistic subjects performed worse than verbal IQ-matched clinical controls on tests of supraspan free recall, source memory and temporal order memory, but were intact on long-term recognition and cued recall tasks. Boucher (1981) also found that autobiographical memory (i.e. the subject’s memory for recent events in his own life) was poorer in autistic children than in normal and mentally handicapped controls, with the autistics having particular problems recalling the order or context in which events had occurred. Finally, in view of the similarities (in terms of cognitive processes) between episodic memory and conditional learning (see Section 3.1), it is intriguing that autistic children show intact object discrimination learning (i.e. learning that response to a particular stimulus always gives reward; Prior & Chen, 1975), but are impaired relative to MA- and IQ-matched retarded children at conditional learning (Prior, 1977). Taken together, these results are certainly persuasive, but they should be viewed in the light of a number of potentially contradictory findings. For example, studies by Rumsey & Hamburger (1988) and Minshew & Goldstein (1993) both found no marked evidence of poor free recall in high-functioning autistic subjects. Similarly, one of the ‘islets of ability’ often shown by autistic people is good rote memory (Lockyer & Rutter, 1970). Further research is therefore needed to elucidate the exact pattern of memory impairment in autism, but clearly an account in terms of deficient episodic memory would suggest that rote memory in autism is not subserved by the encoding of context-specific personal information, but by some other cognitive mechanism.

Relation to brain systems

We saw in Chapter 2 that a number of workers have suggested that schizophrenia may involve functional disconnection between frontal and temporal brain regions (e.g. Frith et al., 1995), and/or between frontal and striatal regions (e.g. Robbins, 1990). On a psychological level, Frith (1994) linked such disconnections
to impairments in the ability to metarepresent, and suggested that the degree of
disconnection may be related to the severity of the metarepresentational deficit. Thus,
patients with negative and positive behavioural signs are presumed to have marked
disconnection, whereas it may be less severe (or only intermittent) in patients with
positive symptoms. As there is convincing evidence for a severe metarepresentational
deficit in autism, and since Frith & Frith (1991) likened the signs of autism to the
behavioural signs of schizophrenia, we might therefore expect frontal-posterior
functional disconnection to be a marked feature of the autistic brain. To date, very
few functional imaging studies have been carried out in which autistic individuals
have performed cognitive tasks, so this prediction remains to be tested. Interestingly,
however, an early neurological model of autism proposed by Damasio & Maurer
(1978) suggested that dysfunction of the mesolimbic system (which includes the
cingulate gyrus, parahippocampal gyrus and subicular region of the hippocampal
formation), and its connected structures in the frontal lobes and basal ganglia, could
underlie the impaired communication, goal-directed behaviour and attention often
found in autistic people. In support of this, a number of post-mortem and structural
imaging studies have provided some evidence for medial temporal lobe abnormalities
in autism, and several functional imaging studies in the resting state reported reduced
perfusion in the temporal and frontal lobes in autistic subjects (see Bailey et al.,
1996, for a review of this work). However, many studies have not been replicated,
and other workers have implicated brain areas such as the parietal lobes or cerebellar
vermis, rather than frontal or temporal areas. As Happé & Frith (1996) pointed out,
a consideration of the plasticity of the developing brain makes it unlikely that autism
is characterised simply by certain brain regions failing to function. Instead, in line
with the above discussion, they emphasised the possibility of damage to the neural
connections between particular brain structures; such disconnection might give a
pattern resembling failure of these regions, whilst explaining the absence of gross
anatomical abnormalities in the autistic brain (Happé & Frith, 1996).
Bailey et al. (1996) pointed out that much of the neuropathology research in autism has suffered from methodological shortcomings such as the use of heterogeneous groups of subjects (in terms of aetiology or disease severity), and the lack of a theory-driven approach. In this respect, it is similar to early research in schizophrenia (see Chapter 1 of this thesis). In the light of Frith’s (1992) suggestions of similarities between autism and schizophrenia, one way forward might therefore be to explore the theoretical prediction of frontal-posterior functional disconnection in autism, using the same kind of functional imaging techniques as have been applied in schizophrenia research. For example, there is some evidence that verbal fluency is impaired in autism (Rumsey & Hamburger, 1988; Turner, 1996, cited in Happé & Frith, 1996), so one useful study might compare autistic and verbal IQ-matched controls on the verbal fluency paradigm used by Frith et al. (1995; see Chapter 2) with schizophrenic patients. This matches the index group and controls on level of performance, and has the potential to reveal fronto-temporal functional disconnection.

It is possible that autistic individuals matched on performance with controls may pass some cognitive tasks using different brain systems than normals. In the theory of mind domain, this is similar to U. Frith et al.’s (1991) argument discussed earlier, whereby autistic ‘passers’ of theory of mind tasks may use general problem-solving skills rather than any understanding of mental states. It would also be compatible with Happé’s (1995) suggestion that autistic passers may have achieved some understanding of mental states using verbally mediated routes not employed by normals.

As a caveat, we should remember that autism is a neurodevelopmental disorder, whereas in Frith’s (1992) model of psychosis, most cases of schizophrenia are thought to begin in early adulthood, following a period of relatively normal brain development. Thus, whilst functional disconnection in schizophrenia is consistent with malfunction of a fully developed metarepresentational system, autism may involve a failure to acquire metarepresentational skills. The neural correlates of this developmental abnormality may not, therefore, be easily comparable with effects in the adult brain (see Bailey et al., 1996, for a discussion of this issue).
normal children. Interestingly, a recent functional imaging study of theory of mind in subjects with Asperger’s syndrome (Happé et al., 1996) provided some support for these models. Using the theory of mind and control stories developed for use in normals (Fletcher et al., 1995; see Section 3.1 of this thesis), Happé et al. (1996) found that their volunteers with Asperger’s syndrome (who passed many of the theory of mind tasks), did not show the normal activation of left medial frontal cortex (Brodmann Area 8) in the theory of mind condition, but instead activated the neighbouring left medial areas BA 9 and 10; these subjects also showed smaller differences in activation (relative to data from normal controls) between the theory of mind and control conditions. On the basis of a number of imaging studies of problem solving ability, (which have implicated BA 9), Happé et al. concluded that their Asperger subjects may have been using a general purpose reasoning system to solve the theory of mind tasks, rather than the specific system for mental state representation used by normals (e.g. Fletcher et al., 1995).

**The relation between executive function and theory of mind in autism**

The research reviewed above has revealed specific autistic deficits in both executive function and theory of mind. It should be noted that Russell and his colleagues (e.g. Russell et al., 1991; Hughes & Russell, 1993) originally suggested that the poor performance of autistic people on some false belief tasks (e.g. the first-order ‘Sally/Anne’ object transfer tasks) may itself be explicable in terms of executive dysfunction. By this argument, the autistic child is unable to ignore the salience of the true location of the object, so answers incorrectly that Sally believes the object to be in that location. However, the many experiments discussed earlier using the ‘fine cuts’ methodology, provide very strong evidence for a specific mentalising deficit, and it is now widely accepted that this co-exists with, (rather than simply reflects), a specific executive dysfunction in autism.
Frith's (1992) suggestion that aspects of executive function require the subject to represent his own goals and intentions, enables us to rationalise the impaired theory of mind and executive function in both autism and schizophrenia in terms of impaired metarepresentation. As autism is presumed to involve the inability to represent both one's own and others' mental states (Frith & Frith, 1991), we can think of autistic subjects as having a grossly impaired metarepresentational system, with the most severely affected cases having problems in the representation of all mental states. As autism is a neurodevelopmental disorder, it is possible that a given subject may show similar degrees of impairment in the representation of own and others' mental states because of a problem, for example, in the development of a single cognitive system necessary for all types of mental state representation. This model would predict that scores on executive function and theory of mind tasks should correlate within a group of autistic subjects, and a number of studies have indeed found such correlations. For example, Ozonoff et al. (1991a) administered a battery of executive function tasks (the Tower of Hanoi and Wisconsin Card Sorting Test), theory of mind tasks (including first- and second-order false belief) and emotion perception tasks to groups of high-functioning autistic individuals and clinical controls matched on verbal IQ. The autistics scored significantly worse than controls in all of these domains, and (unlike the clinical controls), showed a strong correlation between executive function and theory of mind scores. Similarly, in a study comparing autistic children with clinical and normal controls, McEvoy et al. (1993) found that the autistics showed fewer joint attention behaviours in interactions with the experimenter, and made more perseverative errors on a spatial reversal test of set-shifting ability. In view of the fact that joint attention may be a marker of metarepresentational ability (Leslie & Happé, 1989), it is notable that a significant negative correlation emerged between frequency of joint attention behaviours and number of perseverations in this study, (although small subject numbers meant that the correlation was performed over all subject groups combined).
Bearing in mind the earlier discussion of episodic memory in autism, it is also of note that Ozonoff *et al.* (1991a) found robust correlations for their autistic sample (but not for the controls) between scores on a free recall task and performance on executive function and theory of mind tasks. In addition, in their investigation of memory and executive function in autism, Bennetto *et al.* (1996) found that measures of free recall and temporal order memory correlated significantly with executive function (as measured by the WCST and Tower of Hanoi) for the autistic subjects, but not for the clinical controls. Together, these results do indeed suggest, within Frith's model, that a given autistic subject may have similar degrees of impairment in the representation of own and others' mental states. As a note of caution, it is worth mentioning data from Turner (1996, cited in Bailey *et al.*, 1996); she found that, while the degree of repetitive behaviour in autistic individuals was related to performance on executive function tasks, there was little evidence of links with theory of mind ability. Clearly, further investigations of these relationships are required in autism, perhaps using a large battery of theory of mind and executive function tasks matched on difficulty and discriminating power. For the case of schizophrenia, the relationship between executive function and theory of mind will be investigated in the first study of this thesis (see Chapter 4); the predicted outcome of that study is based both on Frith's (1992) model and the above results from the autism literature.

Finally, as part of their study discussed above, Ozonoff *et al.* (1991a) explored the universality of cognitive deficits in autism, and showed that 96%, 87% and 78% of autistics scored below the control group mean on executive, second-order theory of mind and free recall tasks respectively. In contrast, only 52% and 65% of the autistics were below the control mean on first-order theory of mind and emotion perception respectively. In addition, in a companion paper in which they contrasted the performance of a subgroup of their autistic sample (having a diagnosis of Asperger's syndrome) with the remainder of the sample, Ozonoff *et al.* (1991b)
showed that both subgroups performed similarly on executive function tasks, but the Asperger’s subjects scored significantly better on the theory of mind and free recall tasks. Ozonoff et al. (1991a, b) concluded from their work that executive dysfunction may be more universal than theory of mind impairment in subjects with autism and Asperger’s syndrome. However, within Frith’s metarepresentational framework, an alternative explanation is that both deficits reflect the same cognitive impairment, and that the apparently greater prevalence of executive dysfunction in Ozonoff et al.’s patients simply reflected the greater difficulty of the executive tasks relative to the theory of mind tasks. This model predicts that individual autistic subjects should be equally impaired on executive and mentalising tasks when these tasks are matched on discriminating power; a future study might investigate this prediction using tasks from the advanced theory of mind battery developed by Happé (1994b), which can discriminate able autistic individuals from normal adult controls.

3.3 Conclusions

In this chapter, a detailed description has been presented of Frith’s (1987; 1992; 1994; 1996) neuropsychological model of schizophrenia. We have seen how impairments at varying degrees of severity within the cognitive system of meta-representation can explain many of the positive and negative signs and symptoms of the disorder. Thus, Frith’s (1987) early conceptualisation of some negative and positive signs in terms of problems in the generation and inhibition of action, can be recast in terms of the patient’s failure to represent his own goals. Similarly, the impaired self-monitoring of willed action, presumed to give rise to passivity symptoms such as thought insertion and delusions of control, can be thought of as impaired representation of willed intentions. Finally, positive symptoms involving the patient’s relations with other people (e.g. persecutory delusions and delusions of reference), and a number of behavioural signs, can be rationalised in terms of impairments in the representation of others’ mental states (theory of mind). At the level of
brain systems, these cognitive deficits may all reflect functional disconnection between frontal and posterior brain regions, with the severity of that disconnection being mirrored in the severity of the cognitive impairments and symptomatology. Thus, marked disconnection may manifest as an inability to represent mental states and the presence of behavioural signs, whereas less severe (or intermittent) disconnection, may manifest, for example, as a patient who can represent others’ mental states, but who does so incorrectly, experiencing positive symptoms as a result.

In later sections of the chapter, we saw how Frith’s (1992) conceptualisation of willed action in terms of representation of one’s own goals and intentions, can be used to explain the poor executive function often shown by schizophrenic patients. It was also suggested that impaired representation of own knowledge could underlie the poor episodic memory of schizophrenic patients and, more speculatively, their poor performance on tasks requiring the use of context (e.g. Done & Frith’s (1984) sentence-completion task). In addition, it was pointed out that a recent model of ‘poor insight’ in schizophrenia (Startup, 1996) has a role for cognitive deficits in self-awareness, which can, of course, be thought of in terms of impaired metarepresentation.

Finally, we saw how an impairment in the representation of both own and others’ mental states in autism can explain many of the characteristic behavioural signs of that disorder. Frith & Frith (1991) likened the features of autism to the negative and positive behavioural signs of schizophrenia, and explained the similarities and differences in clinical presentation between the two disorders in terms of age of onset. Thus, autism is a neurodevelopmental disorder characterised by the absence, or delayed emergence of metarepresentation; schizophrenia, on the other hand, can be thought of (in most cases) as a late-acquired disorder, characterised by dysfunction of an already-developed metarepresentational capacity. We saw how many studies have provided evidence for impaired theory of mind,
executive function (and, to some extent, episodic memory) in autism, and as with schizophrenia, all of these findings can be rationalised in terms of impaired meta-representation. Correlational studies provide some support for this model, in that executive dysfunction and theory of mind impairments seem to correlate within groups of autistic subjects, but not within groups of controls. The first study of this thesis, which follows in Chapter 4, uses these results to make predictions about the possible relationships between theory of mind and executive function in patients with schizophrenia.

It should be noted that although theory of mind and executive function in autism have been discussed in detail in this chapter, they cannot, at first sight, account for one notable feature of autism, namely the tendency of the autistic child to concentrate on details at the expense of gestalt (see Shah & Frith, 1983; 1993, for empirical confirmation of this clinical observation). U. Frith & Happé (1994) explained this phenomenon in terms of 'weak central coherence' in autism, a cognitive style which they felt to be independent of theory of mind deficits in the disorder. We saw in Chapter 2 that there is some evidence for a similar style of processing in some patients with schizophrenia (e.g. Schwartz Place & Gilmore, 1980; Wells & Leventhal, 1984), which is intriguing given the proposed similarities between the two disorders discussed in this chapter. The performance of schizophrenic patients on tests of 'central coherence' will be investigated in Chapters 6 and 7 of this thesis, where its relationship, if any, to theory of mind ability will be explored.
CHAPTER FOUR

THEORY OF MIND AND EXECUTIVE FUNCTION IN SCHIZOPHRENIA

4.1 Introduction

Frith’s model

Frith’s (1992) neuropsychological model of impaired metarepresentation in schizophrenia was discussed in detail in Chapter 3. According to that model, negative behavioural signs (e.g. avolition, poverty of speech and blunted affect), and the positive signs (such as incoherent or inappropriate speech), can be explained in terms of the patient failing to represent his own mental states (especially goals and intentions) and the mental states of others. Positive symptoms whose content involves the patient’s relations with other people (e.g. persecutory delusions and delusions of reference), are thought to reflect a specific dysfunction in the representation of others’ mental states. While the behavioural signs reflect an absence of metarepresentation, these positive symptoms involve the patient representing others’ mental states, but doing so incorrectly. In Frith’s model, the positive symptoms thought to reflect an absence of self-monitoring of willed action (e.g. passivity phenomena such as delusions of control and thought insertion, and auditory hallucinations where the ‘voice’ is not perceived as coming from an external agent),

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can be conceptualised as a specific failure to represent one’s own willed intentions. Patients with these symptoms, but no behavioural signs or paranoid symptoms, are presumed to have intact representation of their own goals and the mental states of other people. Finally, within the model, patients in remission (i.e. those with no current signs or symptoms), are presumed to have intact metarepresentational abilities. These features of Frith’s model are summarised below:

Table 4.1. Metarepresentational skills as predicted by symptoms in Frith’s model.

<table>
<thead>
<tr>
<th>Group</th>
<th>Signs and symptoms</th>
<th>Metarepresentational skills</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Own goals</td>
</tr>
<tr>
<td>1</td>
<td>Behavioural signs</td>
<td>✓</td>
</tr>
<tr>
<td>2</td>
<td>Paranoid symptoms &amp; no behavioural signs</td>
<td>✓</td>
</tr>
<tr>
<td>3</td>
<td>Passivity phenomena &amp; no paranoid symptoms or behavioural signs</td>
<td>✓</td>
</tr>
<tr>
<td>4</td>
<td>Remission</td>
<td>✓</td>
</tr>
</tbody>
</table>

Representation of others’ mental states in schizophrenia

The above model predicts that only schizophrenic patients in Groups 1 and 2 should perform poorly on tests requiring the representation of others’ mental states. Moreover, those in Group 2 should perform better than those in Group 1, because they are presumed to still represent others’ mental states, albeit with frequent errors. As we saw in Chapter 3, a number of studies have supported these predictions, using tasks involving hints (Corcoran et al., 1995), jokes (Corcoran et al., 1997) and the
appreciation of Gricean maxims (Corcoran & Frith, 1996). However, the two published studies which gave first- and second-order false belief tasks to schizophrenic patients have produced conflicting results. For example, Frith & Corcoran (1996) found that patients who fell into Groups 1 and 2 according to the above model, scored significantly worse than normal and psychiatric controls on a battery of false belief and deception tasks. In contrast, Bowler (1992) found that a group of chronic schizophrenics (all of whom had negative behavioural signs) scored as well as normal adults on similar first- and second-order tasks, with a mean of 64.4% of the schizophrenics passing the tasks. This discrepancy may reflect differences in task difficulty across the studies, and is one focus of the experiment discussed in this chapter. For example, examination of the methodology of the two studies reveals that Bowler enacted his stories using models, and asked up to nine ‘prompt’ questions throughout each story to control for attention and memory. In contrast, Frith and Corcoran read aloud their false belief and deception stories, whilst showing subjects cartoons depicting the action sequences. They only asked one memory control question at the end of each story, and reported that patients (especially those with behavioural signs) often had problems remembering the stories. Frith and Corcoran felt that this may have had some bearing on subjects’ performance, so it remains a possibility that the difference in performance of patients with behavioural signs across these two studies was partly a reflection of subjects’ ability to remember the content of the stories. In the experiment presented in this chapter, this factor is controlled by using Bowler’s technique of enacting the tasks using models, and giving control questions at intervals throughout the stories, to maximise attention and memory. By analysing theory of mind scores for only those subjects who then pass all the memory control questions, we can rule out the possible confounding effects of memory impairment.

A second possible reason for the different results of Bowler (1992) and Frith & Corcoran (1996), lies in the structure of the second-order theory of mind stories used. Bowler used two tasks closely modelled on the ‘ice-cream van’ story of Perner
& Wimmer (1985). Close examination of Perner & Wimmer’s narrative (and the texts used by Bowler), reveals that the story could, in fact, be solved using only a first-order theory of mind. In brief, the ice-cream van story involves two characters (John and Mary) who both see that an ice-cream seller has parked his van in the park. They return home separately, and find out independently that the ice-cream van has now moved to a location outside the church. In the afternoon, John calls for Mary, and is told that she has gone to buy an ice-cream, and the subject has to say where John thinks Mary has gone (Answer: the park, because he doesn’t know that Mary has found out the new location). Now, in the early part of Perner & Wimmer’s (1985) narrative, Mary says (in the presence of John) that she “will be back [in the park] in the afternoon to buy some ice-cream” (ibid., p. 441). Thus, in the theory of mind question, a subject could correctly answer that John thinks Mary is in the park because John knows that Mary said she would be in the park later. This is only a first-order attribution. In contrast to this narrative, the ‘second-order’ tasks used by Frith & Corcoran (1996) could only be solved by employing a second-order theory of mind. It is conceivable, therefore, that their subjects performed worse than Bowler’s because of the greater complexity of theory of mind reasoning required by their tasks. In the present study, this ambiguity was avoided by the use of first- and second-order stories which necessitated theory of mind reasoning at the appropriate level of complexity.

As a final stringent test of Frith’s model, the present study employed non-theory of mind control tasks matched in difficulty with the theory of mind tasks. As advocated by Chapman & Chapman (1973; see Chapter 1 of this thesis), this methodology should reveal any specific schizophrenic deficits in theory of mind as a ‘differential deficit’. The tasks chosen had the same structure as the false belief tasks, but involved ‘non-mental’ representation rather than representation of mental states. The first-order tasks were taken directly from the autism literature (see Chapter 3), and involved a map or drawing becoming an out-of-date representation of the current
situation, in much the same way that a character’s false belief misrepresents the current situation. As we saw in Chapter 3, several studies have shown that autistic people perform well on these map and drawing tasks, while being specifically impaired on the corresponding false belief tasks (Charman & Baron-Cohen, 1992; Leekam & Perner, 1991; Leslie & Thaiss, 1992). The second-order control task was designed especially for the present study, and involved a drawing of a map (i.e. a second-order non-mental representation) becoming out-of-date when the map itself was changed. If Frith’s model is correct, then those schizophrenic patients falling into Groups 1 and 2 in Table 4.1 (but not those in Groups 3 and 4) should show differential deficits in performance on the theory of mind tasks relative to performance on the control tasks.

Executive function and theory of mind

As discussed in Chapter 3, Frith (1992) conceptualised certain aspects of executive function in terms of the ability to represent one’s own goals and intentions. Thus, in a reversal task for example, the inhibition of prepotent responses is presumed to require representation of the goal of the task and of the intended actions during the task. The model described in Table 4.1 therefore predicts that schizophrenic patients in Group 1 (i.e. those with behavioural signs who are presumed to have impaired representation of their own goals and intentions) should show executive dysfunction, and as discussed in Chapter 2, a number of studies have supported this (e.g. Brown & White, 1991; Butler et al., 1992; Elliott et al., 1995; Rosse et al., 1991). We might also expect from Frith’s model that patients in Group 3 (i.e. those with passivity symptoms who are presumed to have impaired representation of their own intentions) should also show some executive deficits. These impairments should be much less severe than for patients with behavioural signs, as patients in Group 3 are thought to still have intact representation of their own goals. The study discussed in this chapter investigates the performance on a simple reversal
task of schizophrenic patients grouped according to the scheme in Table 4.1. Patients in Group 1 should be impaired (and those in Group 3 may show some deficits), while those in Groups 2 and 4 should have intact executive function, as they are presumed to correctly represent their own goals and intentions.

A final aim of the present study is to explore the relationship between theory of mind ability and executive function in schizophrenia. We saw in Chapter 3 that for people with autism, scores in these two cognitive domains correlate highly, whereas normal and clinical controls show no correlation between test scores (e.g. Ozonoff et al., 1991a). This result is compatible with the hypotheses that both theory of mind and executive function require metarepresentation (Frith, 1992), and that autism involves the selective impairment of a single cognitive system underlying all forms of metarepresentation. For the case of schizophrenia, predictions about the relationship between theory of mind and executive function are rather more complex. From Table 4.1 we can see that, according to Frith’s model, only patients with behavioural signs should have impairments in both executive function and theory of mind, as only for these patients is the representation of own goals and intentions and others’ mental states presumed to be impaired. If we assume (as suggested by Frith & Frith, 1991) that these patients are very similar to autistic people in having widespread and severe metarepresentational deficits, then we might predict theory of mind and executive function scores in this subgroup to be intercorrelated. As patients in the paranoid and passivity symptom groups are presumed to be selectively impaired in the representation of others’ mental states and own intentions respectively, no correlations should appear for these patients between scores in the theory of mind and executive domains. A similar lack of correlation should occur for schizophrenics in remission, as these people are predicted by Frith’s model to have the intact metarepresentational abilities characteristic of normal and clinical controls.
4.2 Methods

Subjects

Forty-one people with a DSM-IV diagnosis of schizophrenia (American Psychiatric Association; APA, 1994) took part in this study. General inclusion criteria were competence in the English language, an age in the range 16 to 65, no history of leucotomy, neurological disability or drug/alcohol abuse, and a current IQ as measured by the Quick Test (Ammons & Ammons, 1962) of 70 or higher. About half of the schizophrenic subjects were inpatients at one of two psychiatric hospitals in London. The other half lived in the community and attended psychiatric outpatient clinics. Demographic details of the patients are given in Table 4.2. All but one of the schizophrenic subjects were taking neuroleptic medication at the time of testing. The daily dose of neuroleptic was converted to chlorpromazine equivalents using the tables provided by Foster (1989). The patients receiving anticholinergic medication (26% of the sample), were all taking 15 mg/day or less of procyclidine (mean daily dose was 10.0 mg, range 5 - 15 mg). Tamlyn et al. (1992) have reported that in such doses, procyclidine does not affect memory performance in schizophrenic subjects. However, for the sake of rigour, a number of the neuropsychological tasks in this study incorporated memory control questions to check that subjects were correctly encoding important aspects of the tasks. Clinical details of the schizophrenics are given in Table 4.3.

In line with Frith's (1992) symptom-specific predictions about performance on theory of mind tasks (see Table 4.1), the schizophrenic patients were allocated to one of four groups according to their signs and symptoms as revealed by a Present State Examination (PSE; Wing et al., 1974) on the day of testing. These groups are the same as those used in previous investigations of Frith’s model (Corcoran et al., 1995; 1997; Corcoran & Frith, 1996; Frith & Corcoran, 1996), to enable us to easily
Table 4.2. Demographic details of subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age Mean (S) [Range]</th>
<th>Ethnicity White:Black</th>
<th>Hand Right:Left</th>
<th>Years of Education Mean (S) [Range]</th>
<th>Percent Employed</th>
<th>Pre-morbid IQ (NART) Mean (S) [Range]</th>
<th>Current IQ Mean (S) [Range]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics</td>
<td>29:12</td>
<td>38.2 (12.4) [17 - 60]</td>
<td>33:8</td>
<td>36:5</td>
<td>11.3 (1.6) [9 - 17]</td>
<td>7.3</td>
<td>106.0 (12.0) [76 - 122]</td>
<td>92.9 (10.5) [71 - 123]</td>
</tr>
<tr>
<td>Behavioural Signs</td>
<td>12:4</td>
<td>37.3 (14.6) [17 - 59]</td>
<td>14:2</td>
<td>14:2</td>
<td>11.1 (0.8) [10 - 13]</td>
<td>0.0</td>
<td>101.9 (12.6) [78 - 120]</td>
<td>87.8 (8.6) [71 - 100]</td>
</tr>
<tr>
<td>Paranoid Symptoms</td>
<td>10:6</td>
<td>40.9 (11.3) [25 - 60]</td>
<td>12:4</td>
<td>15:1</td>
<td>10.8 (1.3) [9 - 14]</td>
<td>6.3</td>
<td>105.5 (12.0) [76 - 117]</td>
<td>92.6 (8.9) [77 - 116]</td>
</tr>
<tr>
<td>Passivity Phenomena</td>
<td>0:1</td>
<td>22.0</td>
<td>1:0</td>
<td>1:0</td>
<td>13.0</td>
<td>0.0</td>
<td>119.0</td>
<td>104.0</td>
</tr>
<tr>
<td>Patients in Remission</td>
<td>7:1</td>
<td>36.4 (9.1) [23 - 52]</td>
<td>6:2</td>
<td>6:2</td>
<td>12.5 (2.6) [10 - 17]</td>
<td>25.0</td>
<td>113.5 (7.1) [104 - 122]</td>
<td>102.4 (11.2) [92 - 123]</td>
</tr>
<tr>
<td>Psychiatric Controls</td>
<td>7:11</td>
<td>43.6 (9.5) [26 - 60]</td>
<td>16:2</td>
<td>16:2</td>
<td>11.4 (1.4) [10 - 14]</td>
<td>33.3</td>
<td>105.7 (9.6) [92 - 121]</td>
<td>90.1 (10.4) [73 - 104]</td>
</tr>
<tr>
<td>Normal Controls</td>
<td>19:16</td>
<td>43.3 (13.3) [23 - 62]</td>
<td>32:3</td>
<td>28:7</td>
<td>12.1 (1.7) [11 - 17]</td>
<td>94.3</td>
<td>112.3 (12.4) [74 - 123]</td>
<td>102.8 (13.8) [71 - 125]</td>
</tr>
</tbody>
</table>

S = standard deviation
<table>
<thead>
<tr>
<th>Subjects</th>
<th>Age at first Psychotic Episode</th>
<th>Duration of Illness</th>
<th>Medication Details</th>
<th>Percentage taking Neuroleptics</th>
<th>Daily Dose in Chlorpromazine Equivalents</th>
<th>Percentage taking Anticholinergics</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Schizophrenics</td>
<td><strong>24.0 (8.8)</strong> [13 - 57]</td>
<td><strong>14.6 (11.9)</strong> [1 - 39]</td>
<td>98</td>
<td><strong>888 (911)</strong> [0 - 5000]</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>Behavioural Signs</td>
<td>20.4 (5.0) [13 - 30]</td>
<td>16.9 (12.7) [1 - 38]</td>
<td>100</td>
<td>724 (470) [50 - 1600]</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Paranoid Symptoms</td>
<td>29.0 (10.7) [18 - 57]</td>
<td>14.2 (11.3) [1 - 35]</td>
<td>94</td>
<td>715 (439) [0 - 1700]</td>
<td>20</td>
<td></td>
</tr>
<tr>
<td>Passivity Phenomena</td>
<td>19.0</td>
<td>3.0</td>
<td>100</td>
<td>Not Known</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Patients in Remission</td>
<td>24.3 (9.1) [13 - 43]</td>
<td>12.1 (11.8) [1 - 39]</td>
<td>100</td>
<td>1561 (1773) [262 - 5000]</td>
<td>50</td>
<td></td>
</tr>
</tbody>
</table>

S = standard deviation
compare results across studies. As with those previous studies, allocation to groups was carried out in a hierarchical manner, so that a patient with symptoms or signs from more than one group was allocated to the group for which poorer task performance was predicted. Thus, a patient with blunt affect (a behavioural sign) and paranoid delusions would be allocated to the behavioural signs group, while a patient with only paranoid symptoms and passivity experiences would be allocated to the paranoid group. For the 41 patients in this study, group membership broke down as follows:

**Behavioural signs.** There were 16 patients with negative (e.g. poverty of speech, blunt affect, social withdrawal) or positive (e.g. inappropriate speech, incongruous affect) behavioural signs. This was not considered a large enough group to subdivide into positive and negative features.

**Paranoid symptoms.** 16 subjects described positive symptoms involving other agents (e.g. persecutory delusions or delusions of reference), but showed no behavioural signs.

**Passivity experiences.** Only 1 patient reported experiences thought to reflect impaired representation of willed intentions (e.g. delusions of control, thought insertion or auditory hallucinations) in the absence of delusional beliefs about other agents, or behavioural signs.

**Remission.** 8 patients showed no behavioural signs, and reported no positive symptoms, on the day of testing.

Two control groups were recruited for the study, as advocated by Chapman & Chapman (1977). The clinical control group consisted of 18 non-psychotic psychiatric patients with primary diagnoses of anxiety or unipolar depression. The group was a mixture of inpatients and outpatients, but all subjects were taking antidepressant or anxiolytic medication. The other control group comprised 35 normal volunteers, none of whom reported any history of psychiatric disorder. Demo-
graphic details of the controls are given in Table 4.2.

**Neuropsychological tasks**

*Theory of mind and 'non-mental' representation tasks*

All of these tasks were read out and enacted by the experimenter, using Playmobil characters or other props to aid the subjects' concentration and comprehension. Most of the stories were set in a hospital, or involved familiar objects, in order to maximise the ecological validity for institutionalised schizophrenic patients. Control questions were asked throughout to ensure that subjects could remember the key facts about the story. The 'test question' was the measure of representational understanding.

*First-order tasks*

There were two first-order false belief tasks, both modified versions of tasks from the normal developmental literature. The first was a version of Wimmer & Perner's (1983) object transfer task, in which the subject must recognise that a story character has a false belief about the *location* of an object. The second was a variant of the 'Smarties task' (Perner et al., 1987), where the subject must attribute someone's false belief about the *identity* of an object. Two first-order 'non-mental' representation tasks were also used, based on the tasks developed by Leslie & Thaiss (1992) and Charman & Baron-Cohen (1992) in the autism literature. These had the same structure as the false belief tasks, but as they did not require an understanding of mental states, they provided a control for generalised cognitive impairment, enabling us to test the prediction of specific theory of mind deficit in schizophrenia. The first of these tasks corresponded to the false belief object transfer task, and involved a map showing the *location* of an object in a room. The object was then moved so that the map became out-of-date and falsely represented the object's current location. The subject's task was to say where the map showed the object to
be (Leslie & Thaiss, 1992). The second non-mental representation task corresponded to the ‘Smarties’ paradigm: a drawing was made of a particular object on a table, and this object was then swapped for a different object. As a result, the drawing became an out-of-date representation of the object’s identity. The subject’s task was to say which object the drawing depicted (Charman & Baron-Cohen, 1992). Leslie & Thaiss (1992) and Charman & Baron-Cohen (1992) found that autistic children performed well on both of these non-mental tasks, whilst failing the corresponding false belief tasks. As Leslie and Thaiss pointed out, previous studies suggest that the order of test and control questions does not affect performance in these types of task. However, for the sake of rigour, the test question was asked first in one of the false belief and one of the non-mental tasks, and last in the other two tasks. The specific task protocols were as follows:

**False belief, location.** The subject was shown a card depicting an office, common room and dining room in a hospital. He was introduced to a toy character (Andrew), who was a patient in the hospital. A story was then enacted in which Andrew had a book. Andrew left his book in the common room, and went for lunch into the dining room. Control question 1 was then asked: “Where is Andrew’s book?” While Andrew was away, the nurse came into the common room, picked up the book, and put it into her office for safe-keeping. The subject was then asked control question 2: “Where did Andrew leave his book?”; control question 3: “Where is the book now?”; and the test question: “Where does Andrew think his book is?”. Subjects were asked to explain their answer to the test question.

**False non-mental representation, location.** The subject was presented with a cardboard model room containing three items of toy furniture: a dresser, a table and an armchair. Following the procedure of Leslie & Thaiss (1992), a diagrammatic map was introduced. This had a black border to represent the three walls of the room, and two blue crosses to represent the windows. Coloured outlines of shapes represented
the positions of the dresser (a circle), table (a square) and armchair (a triangle) in the room. Leslie and Thaiss' pre-training procedure was carried out. A toy cat was then introduced and placed on the table in the model room. The experimenter took a sticker, emphasising that it “meant” the cat, and told the subject that he was putting the sticker in the right place on the map “to show where the cat is”. The sticker was placed on the map in such a way that the subject could not see it. The map was then turned face down, and the cat was moved from the table to the armchair. The subject was asked control question 1: “Where was the cat when I put the sticker on the map?”; control question 2: “Where is the cat now?”; and the test question: “In the map, where is the cat?”.

False belief, identity. The subject was shown a cigarette packet, and was asked control question 1: “What does this contain?”. The packet was then opened, revealing that it actually contained a pencil. The pencil was replaced in the box, and the lid was closed. The subject was then asked the test question: “If someone came in now, who had not seen this box before, and I showed it to them with the lid closed, what would they think is in here?”. Control question 2 was then asked: “When I showed you this box in the beginning, what did you say was in here?”; and control question 3: “What is really inside the packet?”.

False non-mental representation, identity. Following Charman & Baron-Cohen (1992), two trials were run in this task: in one, the experimenter did the drawing, and in the other, the subject did the drawing. The order of these trials was counter-balanced across subjects. At the start of a trial, the subject was presented with an object (e.g. an orange), and was asked to name it (control question 1). The object was

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7 In pre-training, the subject was told for each feature of the map that it “meant” its corresponding feature in the room. A toy glass was then introduced, together with a sticker that “meant” the glass. The glass was placed on the dresser, and the subject had to place the sticker on the map “to show where the glass is in the room”. All subjects passed the pre-training first time.
then drawn, and the drawing was put to one side, face down. The object was removed and replaced with a second object (e.g. a cup), and the experimenter said, “I’m putting this cup here in place of the orange”. The drawing was retrieved, still face down, and the subject was asked the test question: “What is in the drawing?”, control question 2: “What object was here before?”; and control question 3: “What object is here now?”. The objects used in the other trial were a pen and a spoon.

**Second-order tasks**

The false belief task used here was a modified version of Perner & Wimmer’s (1985) ‘ice cream van’ story. As discussed in the Introduction, it was believed that Perner and Wimmer’s story was constructed in such a way that it could have been solved using only a first-order theory of mind. In the present study, care was taken, therefore, to use a story in which second-order reasoning was definitely required. The non-mental representation task had the same structure as the false belief task, and served as a control for general cognitive impairment. As with one of the first-order tasks, it involved a map showing the location of an object in a room. However, in this case a drawing was made of the map, so that when the object was subsequently moved (and the map changed), the drawing became a false representation of the map (i.e. a false representation of a representation). The subject’s task was to say where, in the drawing of the map, the map showed the object to be. This type of second-order task has not been used before, but it was considered an important part of this study in that it provided a stringent control measure, enabling us to reveal specific deficits in theory of mind understanding. The task protocols were as follows:

**False belief.** The subject was shown a card depicting a hospital lounge and dining room. The story characters were introduced, and a model television set was placed in the lounge. The following text was read out and enacted by the experimenter using Playmobil characters:
Anne and Stephen are two patients on the same hospital ward. One day they are watching television together in the lounge.

Control question 1: Which room is the television in?
Anne says, “I’m going for a walk outdoors”, and she leaves the lounge. Stephen is now alone in the lounge.

Control question 2: Where is Stephen?
A nurse comes in and tells Stephen that she is going to move the TV into the dining room downstairs, to make more space in the lounge for chairs.

Control question 3: Which room is the TV going to be taken to?
Now, Anne doesn’t know that the nurse has talked to Stephen.
Stephen stays in the lounge, but the nurse leaves with the TV set. Just as she is taking it into the dining room downstairs, she passes Anne who is still on her way out for her walk. The nurse says to Anne, “The TV will be in the dining room from now on”, and she wheels it into the dining room. Anne then goes out for her walk.

Control question 4: Which room has the nurse put the TV in?
Now, Stephen doesn’t know that the nurse has talked to Anne.
An hour later, Stephen is on the ward looking for Anne. One of the other patients says to Stephen, “I’ve only just seen Anne. She has just come back from a walk, and said that she was going to watch television”. Stephen hurries off to find Anne.

Test question: Which room does Stephen think Anne has gone to to watch television?
Explanation question: Why?
Control question 5: Where has Anne really gone to watch television?
Control question 6: Where was the television at the beginning of the story?

Non-mental representation task. The model room used in the first-order map task was reintroduced. This time, it contained only two items of furniture - a fireplace (against one of the walls) and a dresser. Two maps were presented; these both

8 The two maps were the two non-mental representational media corresponding to the two minds of the story characters Anne and Stephen in the second-order false belief
showed the positions of the walls, windows and furniture in the room diagrammatically, and differed only in that one was coloured white and the other blue. Each feature of the maps was pointed out, and the subject was told for each feature that it “meant” its corresponding feature in the room. Blue and white envelopes were then introduced, with the explanation that each map would be placed later into its corresponding coloured envelope.

A toy feather was placed on the dresser in the room, and was pointed out to the subject. The experimenter took a sticker, emphasising that it “meant” the feather, and told the subject that he was going to put it in the right place on the white map “to show where the feather is.” The sticker was placed on the white map without the subject seeing it, and the map was turned face down. The experimenter now said, “I’m now going to draw a picture of this white map, as it looks now, with the sticker on it.” This was done so that the subject did not see the drawing; the drawing was then turned face down, and the white map was placed face down in the white envelope. The experimenter took another sticker, emphasising that it “meant” the feather, and told the subject that he was going to put it in the right place on the blue map “to show where the feather is.” The sticker was placed on the blue map without the subject seeing it, and the blue map was turned face down. The blue map and the drawing of the white map were placed together face down into the blue envelope. The subject was then asked control question 1: “Where is the feather in the room?”; and control question 2: “In the blue map, where is the feather?”

By this stage in the protocol, each map represents the original position of the feather (in the same way that both Anne and Stephen in the false belief task initially represented the original position of the TV set). The drawing of the map corresponds to Stephen’s original representation of Anne’s knowledge about the location of the television.
The feather was now moved onto the fireplace, and the subject was asked control question 3: “Where has the feather been moved to?”. The experimenter said, “I will now change the blue map so that it shows where the feather is at the moment”. This was done without the subject seeing the map, and the map was replaced in its blue envelope. The same was done with the white map, so that once replaced in its envelope it showed the true location of the feather\(^\text{10}\). The subject was then asked control question 4: “Where is the feather now in the room?”; and the test question: “I will now take out the drawing that I did earlier. In this drawing of the white map, where does the white map show the feather is?”; explanation question: “Why?”; control question 5: “Where does the real white map show the feather is?”; control question 6: “Where was the feather at the beginning of the story?”.

**Executive function task**

A spatial discrimination task, very similar to that used by McEvoy *et al.* (1993) with autistic children, was administered to the subjects in this study. The task required the subject to establish, and then reverse, a response set. The subject was seated at a table, and two metal tins were placed in front of him. The tins were padded with newspaper, and had covers made from strips of paper. Objects could thus be dropped inside noiselessly, and once inside were invisible. The experimenter explained that each time the subject put a bead into one of the tins, he had the chance of winning 5 pence. The aim was to try and win the money every time. The subject’s choice of tin on the first trial (right or left) became the non-rewarded side for original learning. Thus, on the first trial, the experimenter always said, “no money this time”. Trials then continued (with the experimenter giving the subject 5 pence for each correct response), until the subject made 5 consecutive responses to the rewarded side; this was the criterion for reaching the simple discrimination (SD). Number of

\(^{10}\) By this stage, both maps have been changed to show the new location of the feather, in the same way that both Anne and Stephen in the false belief task independently updated their knowledge about the location of the TV set.

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errors to criterion was recorded. Failure to reach criterion in 30 trials led to termination of the task.

Once SD was reached, a simple reversal (SR) was introduced (without the subject being explicitly told), so that the previously unrewarded tin now became the correct response position. As before, every time the subject responded to the incorrect position, he was told, “no money this time”. Trials again continued until the subject reached the criterion of 5 consecutive correct responses. The number of errors to criterion was recorded, which here corresponded to the number of perseverative responses to the original position. The task was terminated if the subject failed to learn the reversal within 30 trials. For all subjects, a ‘failure to maintain set’ variable was also calculated; this was the number of times over the whole task that the subject made 3 or 4 consecutive correct responses, but failed to make the 5 needed for criterion.

Procedure

Subjects were tested individually in a quiet room, and were paid £5.00 for their participation. All patients gave written, informed consent to take part in the study, which had been approved by the Ethical Committees of the relevant hospitals. Examples of the patient information sheet and consent form are given in Appendix I. At the start of the session, all schizophrenic subjects were given the Present State Examination Version 9 (PSE; Wing et al., 1974), to assess current symptoms and signs. Normal and psychiatric controls were not given the PSE, but the absence of any history of psychosis was checked verbally and from case notes where relevant. Thereafter, all subjects received the same test protocol. This consisted of an assessment of pre-morbid IQ with the National Adult Reading Test (NART; Nelson, 1982), followed by a test of current IQ with Form 2 of the Quick Test (Ammons & Ammons, 1962). These standard IQ tests were described in some detail in Chapter 1. Handed-
ness was also assessed using the Annett criteria (Annett, 1970), which require the subject to state his preferred hand for carrying out each of 6 common activities. The neuropsychological tasks were then presented in the same standard order for all subjects. The whole assessment lasted up to 1.5 hours; any subjects who felt unable to complete everything in one session were asked if they would finish the tasks on the following day.

4.3 Results

Demographic and clinical variables (see Tables 4.2 and 4.3)

The single schizophrenic patient describing passivity experiences in the absence of behavioural signs or paranoid symptoms was treated as a single case, and is described towards the end of the Results section. This left three schizophrenic symptom subgroups. As data were non-parametric, Kruskal-Wallis one-way analyses of variance (ANOVA) were used to compare these subgroups on duration of illness and daily dose of neuroleptic and anticholinergic medication. The subgroups were matched on all of these variables, but when they were compared on the mean age of onset of the schizophrenic illness, it was found that the patients with behavioural signs had a significantly earlier age of onset (p < 0.02) than those with paranoid symptoms.

When Kruskal-Wallis one-way ANOVA was used to compare the three schizophrenic subgroups and two control groups on demographic variables, all five groups were found to be matched on sex, age, handedness and ethnicity. The normal control group had significantly more years of education (p < 0.05) than the paranoid schizophrenic group, and when compared to all other groups, normal subjects were significantly more likely to be employed [Kruskal-Wallis: $\chi^2(4) = 58.8; \ p < 0.0001$].
Comparison of the five groups on NART pre-morbid IQ revealed a significant difference \( \chi^2(4) = 17.6; p < 0.002 \). Post hoc comparisons showed this to be due to normal controls having a significantly higher NART IQ than the psychiatric controls and schizophrenics with behavioural signs. Examination of current IQ scores from the Quick Test showed them to be approximately normally distributed, with similar variances across the groups. Group differences were therefore analysed using parametric analysis of variance, which revealed a highly significant difference \( F(4, 88) = 7.31; p < 0.0001 \). The ANOVA table for this analysis is given in Appendix II. Post hoc multiple comparisons using Tukey’s honestly significant difference test (with \( p < 0.05 \)) showed the normals to have a significantly higher current IQ than the psychiatric controls and schizophrenics with behavioural signs or paranoid symptoms. The schizophrenic patients in remission also had a higher current IQ than those with behavioural signs. For the sake of rigour, between-group differences in current IQ were checked using non-parametric ANOVA with post hoc comparisons, and the same results were obtained [Kruskal-Wallis: \( \chi^2(4) = 25.7; p < 0.0001 \)]. As we shall see later in this Results section, any possible confounding effects of IQ on task performance were investigated by using subsamples of subjects matched on current or NART IQ. To enable matching on current IQ, the normal controls and schizophrenics in remission with the highest current IQs, were systematically eliminated from the analysis until just enough had been removed to give a matched current IQ across all five groups. This meant that the number of normal controls was reduced to 17 [mean (and standard deviation, S) of current IQ in this subsample = 92.0 (9.7)], and the number of schizophrenics in remission was reduced to 6 [mean and S of current IQ = 96.7 (3.9)]. Subject numbers in the other groups remained unchanged. For matching on NART IQ, the number of normal controls was reduced in a similar way, so that 26 remained [mean (S) of NART IQ in this subsample = 109.2 (13.0)]; subject numbers in all other groups remained constant.
Mental and 'non-mental' representation tasks

First-order tasks

In all of these tasks there was no effect of the order in which control questions were asked. Subjects performed comparably on the two first-order tasks in each of the mental and non-mental domains, so within each domain scores were combined for analysis. Each subject was given a percentage score for performance on the memory control questions within a given domain. Similarly, a percentage score for performance on the test questions was calculated. In the calculation of these latter scores, all tasks were omitted for which the subject had failed any control questions; this ensured that failure on test questions reflected impaired representational understanding, rather than just a memory deficit.

It was clear that most subjects found all the first-order tasks very easy. All the between-group comparisons reported below were carried out using Kruskal-Wallis one-way ANOVA. On the memory control questions, all groups performed equally well in both mental (mean = 98.1% correct) and non-mental (mean = 97.9% correct) domains. In addition, all groups scored equally highly on the test questions in both the mental (mean = 99.5% correct) and non-mental (mean = 92.1% correct) tasks.

For answers to the explanation question in the false belief location change task, subjects were given a score of 0 if they gave a physical explanation (e.g. “Andrew left his book there”), and a score of 1 if they gave a mental state explanation (e.g. “He doesn’t know it’s been moved). There were no group differences in scores on this question, although means ranged from 0.11 to 0.50, as shown in Table 4.4:
Table 4.4. Mean scores by group on the first-order false belief explanation question

<table>
<thead>
<tr>
<th>Subject Group</th>
<th>Mean (S) score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>0.15 (0.38)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms and no behavioural signs</td>
<td>0.19 (0.40)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>0.50 (0.54)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>0.11 (0.32)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>0.31 (0.47)</td>
</tr>
</tbody>
</table>

S = standard deviation

Second-order tasks

Control Questions

For each of the two second-order tasks, subjects were given a score out of 6 for performance on the memory control questions. In practice, every subject scored either 5 or 6 on a given task. Ten data points were missing on the non-mental task (which was always presented at the end of the test battery), because 4 psychiatric controls and 6 schizophrenics did not wish to complete it. Mean values of memory score on the two second-order tasks are given in Table 4.5.

To check that the two second-order tasks were matched for difficulty of memory control questions, a within-subjects analysis of scores was performed for the normal controls only. This confirmed that the two tasks were matched on memory load (McNemar test of change, binomial, p = 0.13, two-tailed). It should be noted, however, that at the one-tailed level these data suggest a trend for the non-mental task to have higher memory demands than the theory of mind task - of the 35 normal subjects, 4 showed poorer memory on the non-mental task than on the theory of mind task, but 0 showed the opposite pattern.
Table 4.5. Mean memory score on the second-order tasks by group

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) non-mental control score</th>
<th>Mean (S) theory of mind control score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>5.58 (0.52)</td>
<td>5.56 (0.51)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms &amp; no behavioural signs</td>
<td>5.57 (0.51)</td>
<td>5.81 (0.40)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>5.75 (0.46)</td>
<td>6.00 (0.00)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>5.50 (0.52)</td>
<td>5.72 (0.46)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>5.86 (0.36)</td>
<td>5.97 (0.17)</td>
</tr>
</tbody>
</table>

$S$ = standard deviation

For each of the second-order tasks, group differences in memory performance were tested by Kruskal-Wallis one-way ANOVA. There was no significant difference between groups on the non-mental task [$\chi^2(4) = 8.6; p = 0.07$] but, with correction for ties, a significant difference in memory scores did appear on the theory of mind task [$\chi^2(4) = 15.9; p < 0.004$]. All post hoc analyses failed to reach significance, but there was a trend (at $p < 0.10$, one-tailed) for the schizophrenic patients with behavioural signs to show poorer memory than normal controls on the theory of mind task. This trend disappeared when current IQ was controlled using the matched subsamples of subjects discussed earlier, although it remained with NART IQ controlled.

Test Questions

As with the first-order analysis, a subject was only given a test score for a task if he had passed all memory control questions on that task; a subject who failed any control questions on a given task was thus treated as a missing value for the test score variable on that task. This criterion reduced subject numbers considerably so that, for example, 8 and 6 schizophrenics in remission gave test score values on the theory of
mind and non-mental tasks respectively, while the corresponding numbers for the paranoid group were 13 and 7, and for the behavioural signs group, 9 and 6. A test score of 0 was given if the subject answered the test question incorrectly, and a score of 1 was given on the few occasions when a subject gave the correct response to the test question but provided an explanation which was clearly wrong or contained inaccurate information. A score of 2 was given on the very rare occasions in which the subject correctly answered the test question and provided an explanation which, although accurate in content, was devoid of any explanatory power. Thus, for example, one subject explained his correct response to the test question in the false belief task by saying, “The nurse moved the TV”, and in the non-mental task, one subject simply responded with, “You drew the map”. Finally, a score of 3 was given when the subject answered the test question correctly and provided a suitable and accurate explanation. At this stage of analysis, explanations in the false belief task which adequately accounted for the response to the test question, but which failed to use mental state language, were considered just as valid as those which explicitly used mental state terms. Thus, an explanation of the form, “Stephen last saw Anne when the TV was in the lounge”, was as acceptable as one such as, “He thinks that Anne thinks the TV is in the lounge”. This rationale was adopted because it is clearly possible for subjects to employ theory of mind reasoning without necessarily using mental state terms in their speech, and any stringent test of Frith’s model must allow for this possibility. We shall examine subjects’ use of mental state language in their responses to the false belief task in a later part of this Results section.

Mean values of test score by group for each of the two tasks are shown in Table 4.6 and Figure 4.1 (where error bars represent the standard error of the mean). To check that the two second-order tasks were matched for difficulty of test and explanation questions, a within-subjects analysis of test scores was performed for the normal controls only. This confirmed that the two tasks were indeed matched for difficulty on this variable (Wilcoxon signed ranks test, p = 0.11, two-tailed). It should
Figure 4.1. Mean test scores on the second-order tasks by group

- Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics;
- Beh = schizophrenics with behavioural signs
be noted that at the one-tailed level, these data suggest a trend for the theory of mind task to be harder than the non-mental task - of the 30 normal subjects who produced test scores for both tasks, 4 scored lower on the theory of mind than the non-mental task, but only 1 showed the opposite pattern. Thus, whilst on memory load there was a trend for the non-mental task to be harder, there was a trend for the theory of mind task to be more difficult on the test and explanation questions.

Table 4.6. Mean values of test score by group on each of the second-order tasks

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) non-mental test score</th>
<th>Mean (S) theory of mind test score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>2.00 (1.27)</td>
<td>0.22 (0.44)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms and no behavioural signs</td>
<td>3.00 (0.00)</td>
<td>1.46 (1.39)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>2.67 (0.82)</td>
<td>2.75 (0.71)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>2.83 (0.41)</td>
<td>2.54 (1.13)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>2.83 (0.59)</td>
<td>2.62 (0.95)</td>
</tr>
</tbody>
</table>

S = standard deviation

As data were non-parametric, group differences in test score on the two second-order tasks were analysed using Kruskal-Wallis one-way ANOVA. There were no significant differences on the non-mental task \(\chi^2(4) = 8.0; p = 0.09\), but on the second-order false belief task, a highly significant group difference appeared \(\chi^2(4) = 32.0; p < 0.0001\). Post hoc analyses showed this to be due to the schizophrenics with behavioural signs scoring significantly worse on theory of mind than normal and psychiatric controls and schizophrenics in remission. Scores of patients with paranoid symptoms were not significantly different from those of controls, although they showed a trend to be lower than those of the normal subjects (at \(p <\).
For the behavioural signs and paranoid groups, within-subjects analyses of test scores on the non-mental and theory of mind tasks were also carried out. Subject numbers were very small for these analyses (only 3 patients with behavioural signs and 6 from the paranoid group provided test scores on both tasks), but despite these small numbers there was a trend at the one-tailed level for the behavioural signs patients to score worse on the theory of mind task (Wilcoxon signed ranks test, \( p = 0.09 \)), and at the one-tailed level the paranoid patients did score significantly worse on the theory of mind task (Wilcoxon, \( p = 0.03 \)).

To explore the relationship between current IQ and theory of mind test score, the Spearman's rank correlation coefficient (\( \rho \)) between these two variables was calculated for each of the subject groups. In all cases, \( \rho \) was non-significant, although it approached significance (at \( p = 0.06 \), one-tailed) for the normal controls. Any possible confounding effects of between-group differences in IQ were investigated using the IQ-matched subsamples of subjects. As we saw earlier, the matching was achieved by eliminating a number of normal controls and (for the case of current IQ) 2 schizophrenics in remission. Thus, in all IQ-matched between-group comparisons detailed in this Results section, the number of psychiatric controls and schizophrenics with paranoid symptoms or behavioural signs is unchanged from the original non IQ-matched comparison. With current IQ matched, Kruskal-Wallis one-way ANOVA of theory of mind test score across group remained highly significant \( [\chi^2(4) = 22.6; \ p = 0.0001] \). Post hoc analyses confirmed that the schizophrenics with behavioural signs scored significantly worse than normal and psychiatric controls and schizophrenics in remission. However, the paranoid patients no longer showed a trend to score more poorly than normals (\( p \) was now greater than 0.15). With NART IQ matched, the same effects were obtained.

**Mental state analysis of explanations**

The presence of mental state terms (e.g. 'knows', 'thinks', etc.) in subjects’
explanations of the second-order false belief task was analysed for all subjects who had passed the memory control questions on the task. This meant in practice that subjects were included even if they had a test score of 0, but this was considered valid because inspection of the data suggested a degree of independence between the tendency to answer the test question correctly, and the tendency to use mental state language in the explanation\textsuperscript{11}. This method also enabled direct comparison with the results of Bowler (1992), who similarly analysed the explanations of all subjects, regardless of their success on the test question. In the first stage of analysis, subjects were given a score of 1 if they had used any mental state terms in their answer, and a score of 0 if they had used only physical state language (e.g. mentioning only the location of objects). Kruskal-Wallis one-way ANOVA of scores across groups was highly significant $\chi^2(4) = 27.5; p < 0.0001$, with post hoc analyses revealing that the schizophrenics with behavioural signs used significantly less mental state language than normal controls and schizophrenics in remission. These effects remained after controlling for current and NART IQ using the matched subsamples of subjects [e.g. with current IQ controlled, $\chi^2(4) = 18.5; p = 0.001$].

The order of subjects' explanations on the false belief task was now investigated using Bowler's (1992) criteria: explanations that referred to neither story character's mental state were coded as zero-order, whilst those that referred to either character's mental state (or to both mental states in parallel) were coded as first-order. Explanations which embedded one character's mental state within the other's (e.g. "he thinks that she thinks"), were second-order. Following Bowler (1992), a strict criterion was used whereby subjects had to explicitly use the mental state term rather than just imply it. Mean values of the order of explanation for each group are given in Table 4.7.

\textsuperscript{11} For example, one subject produced the incorrect response of 'dining room' to the test question, and justified this by saying, "because Anne knows the TV has been moved".
Table 4.7. Mean values of order of explanation on the false belief task by group

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) order</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>0.00 (0.00)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms and no behavioural signs</td>
<td>0.50 (0.52)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>1.25 (0.71)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>0.77 (0.60)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>1.18 (0.58)</td>
</tr>
</tbody>
</table>

S = standard deviation

Kruskal-Wallis one-way ANOVA of scores across groups was highly significant \(\chi^2(4) = 25.9; p < 0.0001\), with post hoc analyses showing that the schizophrenics with behavioural signs gave significantly lower order explanations than normal controls and schizophrenics in remission. In addition, the paranoid schizophrenic group scored significantly lower than normal controls, and there was a trend (at \(p < 0.15\), one-tailed) for the paranoid schizophrenics to give lower order explanations than the schizophrenics in remission.

To explore the relationship between current IQ and order of explanation, the Spearman’s rank correlation coefficient (\(\rho\)) between these two variables was calculated for each of the subject groups. Only for the normal controls was the value of \(\rho\) significant \(\rho(34) = 0.35; p = 0.02\,\text{one-tailed}\). As before, current and NART IQ were controlled using the matched subsamples of subjects. With current IQ matched, Kruskal-Wallis one-way ANOVA of scores across groups remained highly significant \(\chi^2(4) = 18.5; p = 0.001\), and post hoc analyses confirmed that the schizophrenics with behavioural signs gave significantly lower order explanations than normals or schizophrenics in remission. However, there was now no difference in score (even at a trend level of \(p < 0.15\)) between the paranoid schizophrenic patients.
and normal controls or schizophrenics in remission. All of these effects were also found when NART IQ was controlled. Finally, to investigate a suggestion from Bowler (1992) that there is little relationship between theory of mind competence and the tendency to use higher-order explanations, Spearman’s rank correlation coefficients ($\rho$) between second-order theory of mind test score and order of explanation, were calculated for each of the subject groups in turn (the subsamples of subjects which gave an overall match on current IQ were used). For normal controls, the value of $\rho$ was highly significant [$\rho(17) = 0.62; p = 0.004$, one-tailed], and for the schizophrenics in remission or with paranoid symptoms, $\rho$ approached significance [respectively, $\rho(6) = 0.71; p = 0.06$, one-tailed; $\rho(12) = 0.46, p = 0.07$, one-tailed].

**Composite scores**

To give an index of subjects’ overall competence in the theory of mind and non-mental domains, composite scores were calculated for each subject using data from the two first-order and one second-order tasks within each domain. For a given domain, only subjects who passed all control questions on all three tasks were included. In practice this caused no further reduction in the number of schizophrenic subjects for analysis in the theory of mind domain, although a few patients with paranoid symptoms ($N = 2$) or behavioural signs ($N = 3$) were lost from the non-mental domain, because of failure on one or more control questions in the first-order tasks. Within the theory of mind domain, the maximum possible composite score was 8, for which the subject had to give the correct response to the 2 first-order test questions, give a mental state explanation in the first-order location change task, and have a test score of 3, with a second-order explanation, in the second-order task. For the non-mental domain, the maximum possible composite score was 5, for which the subject had to give the correct response to the 2 first-order test questions and have a test score of 3 in the second-order task. Mean values of composite score by group for each of the two domains are shown in Table 4.8 and Figures 4.2 and 4.3 (where error bars represent the standard error of the mean).
Figure 4.2. Mean composite scores in the non-mental domain by group

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs
Figure 4.3. Mean composite scores in the theory of mind domain by group

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs
Table 4.8. Mean composite scores in the non-mental and theory of mind domains

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) non-mental composite</th>
<th>Mean (S) theory of mind composite</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>3.67 (1.53)</td>
<td>2.33 (0.50)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms and no behavioural signs</td>
<td>5.00 (0.00)</td>
<td>4.08 (1.75)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>4.33 (0.82)</td>
<td>6.50 (1.51)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>4.80 (0.45)</td>
<td>5.33 (1.44)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>4.77 (0.63)</td>
<td>6.12 (1.34)</td>
</tr>
</tbody>
</table>

S = standard deviation

Group differences were again analysed using Kruskal-Wallis one-way ANOVA. There were no significant differences on the non-mental composite [$\chi^2(4) = 8.4; p = 0.08$], but a highly significant group difference appeared on the theory of mind composite [$\chi^2(4) = 34.2; p < 0.0001$]. Post hoc comparisons revealed that the schizophrenics with behavioural signs scored significantly lower than normal and psychiatric controls and schizophrenics in remission. In addition, the schizophrenic group with paranoid symptoms scored significantly lower than normal controls and schizophrenics in remission.

To explore the relationship between current IQ and theory of mind composite score, the Spearman’s rank correlation coefficient ($\rho$) between these two variables was calculated for each of the subject groups. Values of $\rho$ were only significant for the normal controls [$\rho(34) = 0.41, p = 0.01$, one-tailed], and for the schizophrenics in remission [$\rho(8) = 0.76, p = 0.01$, one-tailed]. Any possible confounding effects of IQ were again controlled using the IQ-matched subsamples of subjects discussed earlier. With current IQ matched, Kruskal-Wallis one-way ANOVA of theory of
mind composite score across group remained highly significant \( \chi^2(4) = 24.6; p = 0.0001 \), and post hoc analyses confirmed that the schizophrenics with behavioural signs scored significantly more poorly than normal and psychiatric controls and schizophrenics in remission. However, matching current IQ removed the significant differences between paranoid schizophrenics and other groups, so that only trends were apparent. More specifically, the paranoid patients now showed a trend to score lower on the theory of mind composite than normal controls (at \( p < 0.10 \), one-tailed), and schizophrenics in remission (at \( p < 0.07 \), one-tailed). In contrast, when NART IQ was matched across groups, all the significant post hoc comparisons from the original analysis of variance remained significant [Kruskal-Wallis: \( \chi^2(4) = 30.4; p < 0.0001 \)]. This last point will be further explored in the Discussion.

**Executive function task**

With the exception of one subject, all controls reached criterion on both stages of the spatial discrimination task. The single failer (a normal subject) achieved the simple discrimination (SD), but then wished to terminate the task. She was excluded from any subsequent analysis because it was not possible to rule out lack of motivation as the reason for her not wishing to continue. Of the schizophrenic subjects, 3 failed to reach one of the criteria. Two of these expressed a desire to terminate the task before the requisite number of trials had been completed, so they too were excluded from any analysis to safeguard against amotivation. The other patient failed to reach the SD criterion within 30 trials, so the task was terminated before moving on to the reversal stage. As this patient showed no evidence of amotivation, and in fact wished to continue at this point, analysis of group differences in errors to SD was carried out both with and without the inclusion of her data; this manipulation had no effect on the final result. Mean number of errors to criterion at the simple discrimination (SD) and simple reversal (SR) stages of the task are shown for each subject group in Table 4.9 and Figure 4.4 (error bars represent standard error of the mean).
Figure 4.4. Mean errors to SD and SR criterion in the executive function task

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs
Table 4.9. Mean errors to SD and SR criterion on the executive function task

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) errors to SD criterion</th>
<th>Mean (S) errors to SR criterion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>2.57 (2.90)</td>
<td>3.79 (4.19)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms and no behavioural signs</td>
<td>3.25 (3.57)</td>
<td>0.80 (1.27)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>1.63 (2.07)</td>
<td>0.88 (1.73)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>2.11 (3.01)</td>
<td>0.78 (1.44)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>2.12 (2.95)</td>
<td>0.85 (1.26)</td>
</tr>
</tbody>
</table>

S = standard deviation

Data were non-parametric, so for each subject group, performance at the two stages of the task was compared using the Wilcoxon signed ranks test. At the one-tailed level, all groups (except schizophrenics in remission) showed a significant difference in performance at the SR compared to the SD stage. Specifically, the schizophrenics with behavioural signs performed significantly worse at reversal than at discrimination, while the other groups performed better at the SR stage than at SD. Kruskal-Wallis one-way ANOVA was then used to explore group differences in number of errors at each stage of the task. At the SD stage, all groups were matched on errors to criterion [$\chi^2(4) = 3.5; p = 0.47$], but at the simple reversal (SR) stage, a significant group difference appeared [$\chi^2(4) = 12.1; p = 0.02$]. Post hoc comparisons showed this to be due to the schizophrenic patients with behavioural signs making more perseverative errors than normal and psychiatric controls.

To explore the relationship between current IQ and number of perseverative errors at the SR stage of the task, Spearman’s rank correlation coefficients ($\rho$) were calculated between these two variables for each of the subject groups. In all cases,
values of $p$ were non-significant, although $p$ approached significance [$p(15) = -0.42; p = 0.06$, one-tailed] for the paranoid schizophrenic group. Any possible confounding effects of group differences in IQ were investigated using the IQ-matched subsamples of subjects discussed earlier, and with current IQ controlled, the significant group difference remained in errors at the SR stage [Kruskal-Wallis: $\chi^2(4) = 10.6; p = 0.03$]. Post hoc comparisons confirmed that schizophrenics with behavioural signs made more perseverative errors than psychiatric controls, but the difference between patients with behavioural signs and normal controls now only reached a trend level of significance (at $p < 0.15$). The same effects were obtained with NART IQ matched across groups.

In an investigation of the performance of schizophrenic patients on a computerised spatial reversal task, Elliott et al. (1995) attempted to control for generalised cognitive impairment by performing a separate analysis using only those schizophrenics with current IQs of 85 or higher (i.e. within one standard deviation of the population mean). To provide a direct comparison with their results, group differences in errors to reversal in the present experiment were also analysed using only the schizophrenics with a current IQ of 85 or higher (together with appropriate IQ-matched subsamples of other subjects). With this manipulation, Kruskal-Wallis one-way ANOVA of errors to reversal across groups was highly significant [$\chi^2(4) = 14.7; p = 0.005$], and post hoc multiple comparisons showed that the schizophrenics with behavioural signs made significantly more perseverative errors than both psychiatric and normal control groups. This result for the comparison between normals and schizophrenics with behavioural signs contrasts with the earlier non-significance of the same comparison when all the behavioural signs patients were compared with IQ-matched normals. The difference in results is mainly a reflection of an (unexpected) tendency for the normal controls with lower IQs to make more errors on the task; as the IQ-matched subgroup of normals used for comparison with all the behavioural signs patients, had a lower mean IQ than the normal subgroup used for comparison.
with patients whose IQ was greater than 85, there was a tendency for the former group of normals to make more errors than the latter group, leading to the observed non-significance of the comparison between all the behavioural signs patients and normal controls. This issue will be explored further in the Discussion.

Finally, Kruskal-Wallis one-way ANOVA across groups of the ‘failure to maintain set’ variable was just significant when corrected for ties \( \chi^2(4) = 9.3; p = 0.05 \). All post hoc comparisons failed to reach significance, although there was a trend (at \( p < 0.15 \), one-tailed) for the schizophrenics with behavioural signs to score more poorly than psychiatric controls on this variable. With current IQ controlled, the overall ANOVA failed to reach significance \( \chi^2(4) = 8.7; p = 0.07 \); controlling for NART IQ had the same effect.

**Single case with passivity symptoms**

In the PSE interview, one schizophrenic patient reported delusions of control in the absence of any paranoid or behavioural features. Frith’s (1992) model makes specific predictions about the metarepresentational ability of such subjects, so the data from this case were excluded from all of the above analyses, and are presented now. The subject was a 22-year old unemployed female with 13 years of education. She had a NART IQ of 119 and a current IQ of 104. Three years had elapsed since her first psychotic episode. On the neuropsychological battery she performed normally, making no errors in learning the reversal, and scoring almost at ceiling on the representational tasks (her theory of mind composite score was 7 out of a possible 8, and her non-mental composite score was 5/5).

**Relationship between executive function and theory of mind**

As a whole, the data suggest that executive deficits may be relatively specific
to schizophrenics with behavioural signs, whereas theory of mind impairments may be more widespread within the schizophrenic group. For example, when all schizophrenics were taken together and compared with a combined control group of normal and psychiatric controls, it was found that 61.3% of the schizophrenics scored below the control mean on the theory of mind composite measure, whereas only 42.1% of them scored below the control mean on the reversal stage of the executive task. To investigate the relationship between executive function and theory of mind ability, Spearman's rank correlation coefficients ($\rho$) between number of errors to reversal and theory of mind composite score, were calculated for the whole subject sample taken together. The value of $\rho$ was non-significant (at $p > 0.4$, two-tailed). The same correlation was then performed for the combined control group and for the combined schizophrenic group, and in both cases, values of $\rho$ were non-significant (at $p > 0.6$, two-tailed). Finally, the correlation was performed for each of the control groups taken separately, and for each of the schizophrenic symptom subgroups, and all values of $\rho$ were again non-significant. In particular, it is interesting to note the result for the subgroup of patients with behavioural signs which, as we saw in the Introduction, may have theoretical importance. In this case, $\rho(8) = +0.34; p = 0.40$ (two-tailed test). In order to remove any measure of the order of theory of mind explanations (which Bowler (1992) suggested may only be a weak index of theory of mind ability), the corresponding correlations were then performed between number of errors to reversal and theory of mind test score from the second-order task. All values of Spearman’s $\rho$ were again non-significant. Finally, for the sake of rigour, all of the above correlations were checked using the IQ-matched subsamples of subjects discussed earlier; all values of $\rho$ again failed to reach significance.

**Signs and symptoms as predictors of theory of mind deficit**

A regression technique was used to investigate which groups of schizophrenic symptoms most strongly predicted a poor theory of mind composite score. It was not
possible to use standard multiple regression, as the theory of mind composite was not normally distributed. Instead, the composite scores were dichotomised into 'deficit' and 'non-deficit' categories, so that logistic regression could be performed. The criterion for deficit was a composite score less than two standard deviations below the control group mean of 6.12; this meant in practice that subjects were classified as having a deficit if their composite score was less than 3.43. Of the 31 schizophrenics who provided a theory of mind composite score, this gave 16 in the 'deficit' category and 15 without a deficit. For the coding of symptoms, the hierarchical subgroups based on the predictions from Frith's model (and used throughout all the above analyses), were not used. Instead, the symptom ratings from the PSE interviews were used to give a score for each subject on each of 5 dimensions; these were 1) passivity symptoms; 2) paranoid symptoms; 3) negative behavioural signs; 4) positive behavioural signs and 5) insight. Thus, while a patient with, say, 5 paranoid symptoms and only 2 negative behavioural signs would have been classified in earlier analyses into the negative behavioural signs subgroup (in accord with Frith's model), the present technique had the potential to reveal whether that patient's paranoid symptomatology also made a significant contribution to any theory of mind deficit.

For dimension 1, 5 core passivity symptoms were selected from the PSE; these were thought insertion (symptom number 55), thought echo (57), thought block or withdrawal (58), delusions of control (71) and delusional explanations in terms of physical forces such as X-rays and electricity (80). As the PSE gives a score of 0, 1 or 2 for each of these symptoms (1 = 'partial delusions' expressed with doubt and 2 = 'full delusions' of which the subject is fully convinced with no insight), each subject could score a maximum of 10 on the passivity dimension. Similarly, for dimension 2, 5 core paranoid symptoms were selected; these were delusions of thoughts being read by other people (59), delusions of reference (72), delusional misinterpretation or misidentification (73), delusions of persecution (74), and delusions concerning hypnotism or telepathy from other people (79). Again, the
maximum score was 10 on this dimension. It should be noted that these two positive symptom dimensions did not include any ratings for auditory hallucinations because (as explained in Chapter 3), Frith’s model suggests that ‘hearing voices’ is only sometimes associated with incorrect attribution of others’ mental states. For the negative behavioural signs dimension, the 5 signs selected were social withdrawal (28), self-neglect (108), slowness and underactivity (110), blunt affect (128) and poverty of speech (134). As these were rated as 0, 1 or 2 in the PSE according to severity, a maximum score of 10 was possible on this dimension. The 5 selected positive behavioural signs were irreverent or embarrassing behaviour (signs 113 and 115 averaged to give a maximum score of 2 as a measure of inappropriate behaviour), stereotypy (117), incongruous affect (129), incoherent speech or flight of ideas (signs 136 and 137 averaged to give a maximum score of 2 as a measure of inappropriate speech), and poverty of content of speech (138). Again, a maximum score of 10 was possible. Finally, the insight dimension had scores ranging from 0 to 3, in response to PSE question 104: “Do you think there is anything the matter with you?” In accord with PSE criteria (see Wing et al., 1974, p. 223), 0 was given for full insight, 1 for a subject who had “as much insight into the nature of the condition as social background and intelligence allow”, 2 for a subject who “agrees to a nervous condition, but examiner feels that subject does not really accept the explanation in terms of a nervous illness (e.g. by giving a delusional explanation)”, and 3 for a subject who completely denied any illness.

As it is widely held that only N/10 predictors (where N = total number of subjects) can be entered simultaneously into a logistic regression analysis (Hosmer & Lemeshow, 1989), it was not possible to simultaneously enter all 5 symptom scores and other variables of interest (e.g. current IQ). Initially, therefore, a separate regression analysis was performed for each variable of interest, to rule out those which had no predictive value. These calculations revealed that adding scores for age of onset, insight, passivity symptoms or paranoid symptoms led to no significant
improvement in the fit of the data relative to a model with no predictors. However, adding scores for negative behavioural signs (neg beh) did lead to a significant improvement in fit:

\[ \chi^2(\text{no predictors}) = 42.94; \chi^2(\text{predictor} = \text{neg beh}) = 32.51 \]

=> Model \( \chi^2(1 \, \text{df}) = 42.94 - 32.51 = 10.43; p = 0.0012 \)

This gave an optimal logistic regression equation of:

\[ \ln(\text{odds}) = -1.3689 \, \text{neg beh} + 0.6996 \]

This means that a 1 point increase in neg beh decreases the ln (odds) of intact theory of mind by 1.3689. As \( \exp(-1.3689) = 0.2544 \), a 1 point increase in neg beh multiplies the odds of intact theory of mind by 0.2544. Thus, for example, a schizophrenic subject with no negative behavioural signs has \( \ln(\text{odds}) = 0.6996 \), and therefore odds of intact theory of mind of \( \exp(0.6996) = 2.01 \) (i.e. he is about twice as likely to have intact theory of mind as to have a deficit). However, a subject with a score of 1 for negative behavioural signs will have \( \ln(\text{odds}) = -0.6693 \), and odds of intact theory of mind of 0.51. He will therefore be about twice as likely to have a theory of mind deficit as not.

It should be noted that for neg beh, Wald’s \( \chi^2(1) = 3.69; p = 0.055 \). This value disagrees with the whole model \( \chi^2 \), but as there is only one predictor we would expect these two values to be equal (as occurs in linear regression). As discussed by Howell (1997), Hosmer & Lemeshow (1989) have raised questions over the behaviour of the Wald criterion, and they suggest relying upon the whole model \( \chi^2 \) instead. This convention will be adopted for the remainder of this analysis.

A similar logistic regression using scores for positive behavioural signs (pos
beh) also led to a significant improvement in fit relative to no predictors:

\[ \chi^2(\text{no predictors}) = 42.94; \chi^2(\text{predictor = pos beh}) = 38.67 \]

\[ \Rightarrow \text{Model } \chi^2(1) = 4.27; p = 0.0388. \] [Wald’s \( \chi^2(1) = 0.03; p = 0.86, \text{ns} \)].

Thus, based on data from the present experiment, it appears that scores for positive behavioural signs are considerably weaker predictors of theory of mind deficit than are scores for negative signs.

A similar logistic regression was carried out using current IQ scores, and this also led to a significant improvement in fit relative to no predictors:

\[ \chi^2(\text{no predictors}) = 42.94; \chi^2(\text{predictor = current IQ}) = 31.63 \]

\[ \Rightarrow \text{Model } \chi^2(1) = 11.31; p = 0.0008. \] [Wald’s \( \chi^2(1) = 5.61; p = 0.0179 \)].

Having identified the three variables (neg beh, pos beh and current IQ) which act as predictors of theory of mind score, both current IQ and neg beh were entered simultaneously into a logistic regression to see whether \( \chi^2 \) reduced further from its value of 31.63 with current IQ alone. This revealed \( \chi^2(\text{predictors: current IQ and neg beh}) = 26.56 \). Thus, model \( \chi^2(2) = 16.38; p = 0.0003 \), relative to the case of no predictors. This means that the use of neg beh as a predictor as well as current IQ increased the model \( \chi^2 \) by \( (16.38 - 11.31) = 5.07 \). From tables this is a significant change in \( \chi^2 \) at \( p < 0.05 \). Finally, current IQ, neg beh and pos beh were entered simultaneously into a logistic regression to see whether \( \chi^2 \) reduced even further. This revealed \( \chi^2(\text{predictors: current IQ, neg beh and pos beh}) = 20.85 \), with model \( \chi^2(3) = 22.10; p = 0.0001 \), relative to the case of no predictors. This means that the addition of pos beh increased the model \( \chi^2 \) by \( (22.10 - 16.38) = 5.72 \). This is again a significant change in \( \chi^2 \) at \( p < 0.05 \). [On this final regression, all values of Wald’s \( \chi^2 \) were non-significant: current IQ: \( \chi^2(1) = 3.13; p = 0.08 \); neg beh: \( \chi^2(1) = 2.64; p = 0.10 \); pos beh: \( \chi^2(1) = 0.02; p = 0.88 \). The optimal logistic regression equation resulting
from this analysis was:

\[
\ln(\text{odds}) = 0.1703 \times \text{(current IQ)} - 1.3879 \times \text{(neg beh)} - 10.0538 \times \text{(pos beh)} - 14.9673
\]

Thus, increases in current IQ increase the odds of a schizophrenic subject showing no theory of mind deficit on the present tasks, whereas increases in scores for negative or positive behavioural signs increase the odds that the subject will show a deficit.

**Signs and symptoms as predictors of executive deficit**

Logistic regression was similarly used to investigate which groups of schizophrenic symptoms predicted poor executive function as measured by perseverative errors at the simple reversal (SR) stage of the spatial discrimination task. Subjects were coded into ‘deficit’ or ‘non-deficit’ categories on the basis of their error scores, with the criterion for deficit being a number of errors greater than one standard deviation above the normal control group mean of 0.85\(^{12}\). In practice this meant that those schizophrenic patients who made more than 2.11 errors were coded into the ‘deficit’ category. Of the 38 patients who provided an error score, this resulted in 10 with a deficit and 28 without. The symptom scores used in the above logistic regression calculations were also used here. As before, a separate regression analysis was performed for each variable of interest, to rule out those which had no predictive value. These calculations revealed that adding scores for insight, paranoid symptoms, positive behavioural signs or current IQ led to no significant improvement in the fit of the data relative to a model with no predictors. However, adding scores for negative behavioural signs (neg beh) did lead to a significant improvement in fit:

\[\ldots\]

\(^{12}\) This criterion was used in preference to one of 2 standard deviations above the normal mean, as the latter would have resulted in only 6 patients with a deficit.
A similar logistic regression was carried out using scores for passivity symptoms (passiv), and this also led to a significant improvement in fit relative to no predictors:

\[ \chi^2(\text{no predictors}) = 43.80; \chi^2(\text{predictor = passiv}) = 39.04 \]

\[ \Rightarrow \text{Model } \chi^2(1) = 4.76; p = 0.0292. \text{ [Wald's } \chi^2(1) = 1.89; p = 0.1695, \text{ ns}]. \]

Having identified the two variables (neg beh and passiv) which acted as predictors of executive deficit in the present study, both were entered simultaneously into a logistic regression to see whether \( \chi^2 \) reduced further from its value of 37.26 with neg beh alone. This revealed \( \chi^2(\text{predictors: neg beh and passiv}) = 35.13 \). Thus, model \( \chi^2(2) = 8.67; p = 0.0131 \), relative to the case of no predictors. This means that the use of passiv as a predictor in addition to neg beh only increased the model \( \chi^2 \) by \((8.67 - 6.54) = 2.13\), which from tables is a non-significant change in \( \chi^2 \) at \( p < 0.05 \). These analyses therefore suggest that the optimal logistic regression equation for predicting the executive function of schizophrenic patients in the present study, contains only the score for negative behavioural signs. This equation takes the form:

\[ \ln (\text{odds}) = -0.5125 \text{ (neg beh)} + 1.7659 \]

As \( \exp (-0.5125) = 0.60 \), this means that a 1 point increase in the score for negative behavioural signs multiplies the odds of intact executive function by 0.60.

**Relation of insight scores to other variables**

Although the present study was not designed with the aim of investigating the correlates of insight, a few exploratory calculations were carried out to examine the
relationships between the schizophrenic patients’ insight scores on the PSE (ranging from 0 to 3), and their PSE scores for particular groups of symptoms (e.g. paranoid symptoms, passivity symptoms, etc.). For each group of symptoms, Spearman’s rank correlation coefficients between symptom score and insight failed to reach significance. As might be expected from the above logistic regression analyses, Spearman’s rank correlations between measures of insight and theory of mind competence or executive function also failed to reach significance. We saw in Chapter 3 that Startup (1996) recently found a quadratic relationship between executive function and measures of insight. One way of exploring that relationship in the present study would be to perform polynomial regression on the number of perseverative errors in the spatial discrimination task, testing whether $(\text{insight})^2$ predicted the number of errors. We saw earlier, however, that number of errors was non-normally distributed, so that standard multiple regression techniques could not be used. One approach, therefore, might be to adopt the logistic regression procedure, testing whether $(\text{insight})^2$ significantly predicts an executive or theory of mind deficit (as defined in the earlier analyses). Although logistic regression assumes a linear relationship between $\ln (\text{odds})$ and the predictor variables, we could define our predictor ($X$) in this calculation so that $X = (\text{insight})^2$. This would then give us a logistic regression equation of the form:

$$\ln (\text{odds}) = A + BX.$$ 

Using this method with data from the present study, $(\text{insight})^2$ was found not to be a significant predictor of either executive or theory of mind deficits.

### 4.4 Discussion

**Clinical details of the schizophrenic patients**

The results of this study illustrate clearly the advantages of using well-defined
subgroups of schizophrenic patients rather than one heterogeneous group. For example, the task performance deficits which have emerged can now be linked theoretically to specific psychotic signs and symptoms rather than simply to the atheoretical construct of 'schizophrenia'. In addition, the matching of symptom subgroups on medication dosage has meant that medication effects can be ruled out as an explanation of differences in task performance between those subgroups. It is of interest that one of the few significant differences in clinical details between the schizophrenic subgroups lay in age of onset, with patients in the behavioural signs group having an earlier mean age of onset than those with paranoid symptoms and no behavioural signs. This result is consistent with a number of studies suggesting an association between early onset and negative symptomatology (e.g. Johnstone et al., 1995) and indeed, as we saw in Chapter 3, several workers have proposed that a subset of schizophrenic patients with negative signs are suffering from a 'neurodevelopmental' form of the illness characterised by early onset (e.g. Castle et al., 1994; Murray et al., 1992). We shall return to the concept of a neurodevelopmental subgroup of schizophrenic patients later in this Discussion.

Performance on the theory of mind and 'non-mental' tasks

The first thing of note about subjects' performance on these tasks was the generally high score by all groups on the memory control questions, both in the first- and second-order tasks. Thus, even at the second-order level, the worst performance by any subject on the memory questions was a score of 5/6. Between-group comparisons showed that there were no significant differences between groups in memory score, and the only trend towards a difference (between behavioural sign schizophrenics and normals for memory in the second-order theory of mind task), was found to be an artefact of current IQ. These high memory scores are in marked contrast to the performance of subjects on Frith & Corcoran's (1996) theory of mind stories, where schizophrenic patients with behavioural signs or paranoid symptoms
were more likely than other groups to fail memory questions on the first- and second-order tasks. The memory performance of subjects in the present study was very similar to that of Bowler's (1992) schizophrenic patients (of whom none failed any control questions on a first-order theory of mind task, and only 3/15 failed one or more control questions on the second-order task). As both the present study and Bowler's experiment involved enacting the stories using models, and the use of a number of control questions spaced at intervals throughout each story, it is likely that these features considerably improved subjects' memory for the story material. This is clearly a desirable effect, as it reduces the number of subjects who must be excluded from subsequent analyses because of memory failure. Future studies might therefore adopt the present methodology and avoid simply reading aloud the stories with few aids to memory.

The use of a matched-tasks design in the present experiment was a further improvement on previous studies in that, as predicted, a differential performance deficit was shown by certain schizophrenic symptom groups on a second-order theory of mind task, relative to an equally difficult 'non-mental' task. This use of a differential deficit design (as advocated by Chapman & Chapman, 1973) provides strong evidence that schizophrenic patients with those particular symptoms have a specific problem in understanding mental representation, rather than a more general problem in understanding representation by any media (e.g. maps and drawings). It should be noted that although the two second-order tasks in the present study were matched (for normals) on difficulty of memory and test questions, there were trends for them to differ on these two performance measures. Future studies might attempt, therefore, to use even more closely matched tasks, and one possibility may be the theory of mind and 'physical' stories developed by Fletcher et al. (1995) for use in their functional imaging study of mentalising. Within a given pair, these stories were very similar in length and involved very similar narrative themes; they differed only in that the theory of mind stories required consideration of characters’ mental states.

*Footnote:* It should be noted that the map of a map in the non-mental task could be seen as a copy rather than a second-order representation, depending on one's analysis of embedding (Perner, 1991).
Both story types involved interactions between people, which is clearly an improvement on the present study where the non-mental task did not involve any story characters. Furthermore, both types of story in the Fletcher et al. study required an inference to be made beyond the information explicitly stated in the story. This again may be an improvement on the present study, where it could be argued that an inference was only required in the theory of mind task, and not in the non-mental task (where the subject had only to remember that a drawing done earlier showed a particular state of affairs). In a pilot study in which both sets of their stories were given to 60 normal volunteers, Fletcher et al. confirmed that they were matched on difficulty. In view of the problems already discussed in simply reading aloud stories to schizophrenic patients, any future study using Fletcher et al.’s materials might either present the stories in a written format for subjects to read themselves, or might use videotapes of actors to illustrate the narratives.

Regarding subjects’ performance in the theory of mind domain, the present study provides a partial replication of both Bowler (1992) and Frith & Corcoran (1996), and enables a reconciliation of all findings from these studies. At the first-order level, this study replicated Bowler’s findings, showing that when the stories are enacted to ensure good memory and comprehension, schizophrenic patients (even those with behavioural signs) show an intact theory of mind. This is contrary to predictions from Frith’s model, and suggests that the first-order deficits found by Frith & Corcoran (1996) in patients with paranoid symptoms or behavioural signs may have been artefacts of subjects’ poor memory for the stories. This is in line with Frith and Corcoran’s own conclusions, particularly regarding the behavioural sign patients, who they felt “clearly had problems remembering the stories. In these circumstances it is difficult to interpret their poor performance on the theory of mind questions despite attempts to deal with this confounding variable by examining theory of mind performance only when the memory questions were answered correctly” (ibid., p. 527). The implication of this statement is that correct responses
to the memory questions in Frith and Corcoran’s study may not have been a stringent enough measure of intact memory; this is highly plausible given that only one memory question was asked per story. It should also be noted that when Frith and Corcoran examined IQ-matched subsamples of their subjects, they found no significant difference between first-order theory of mind scores of schizophrenics and controls. As this IQ matching would have selected subsamples of similar cognitive ability, including, perhaps, memory function, this result is consistent with an explanation of Frith and Corcoran’s first-order results in terms of memory impairment.

For the test score on the second-order theory of mind task, the present study found clear evidence of impairment in schizophrenics with behavioural signs. This effect remained highly significant even with current IQ controlled, and is in line with Frith’s prediction that such patients should be impaired in the representation of others’ mental states. This result is a replication of Frith & Corcoran (1996), and shows that a real second-order deficit is present, even when memory is controlled much more rigorously than in their study. The result fails to support Bowler’s (1992) finding that schizophrenics with behavioural signs score no more poorly than controls on second-order theory of mind tasks. As discussed in the Introduction, it was possible for Bowler’s ‘second-order’ tasks to be answered correctly using only first-order reasoning, whereas those used by Frith and Corcoran (and the story designed for the present study) could only be solved with an intact second-order theory of mind. It is possible, therefore, that Bowler’s patients scored well on his tasks because of their generally intact first-order theory of mind, but that they would have failed any tasks which necessitated second-order reasoning.

The present study found that schizophrenic patients in remission had test scores on the second-order theory of mind task comparable to those of controls, and significantly higher than patients with behavioural signs. This is again a replication of Frith & Corcoran (1996), and supports Frith’s (1992) suggestion that theory of
mind deficits are related to symptomatology rather than to a schizophrenic diagnosis per se. This finding also fits with the other studies of theory of mind in schizophrenia which, without exception, have found patients in remission to score well on tasks requiring the representation of others' mental states (Corcoran et al., 1995; 1997; Corcoran & Frith, 1996). The single case reporting only passivity experiences in the present study had a second-order theory of mind test score of 3/3, so showed an intact understanding of others' mental states as measured by that task. Again this fits with earlier studies which found patients with passivity symptoms to have an intact theory of mind (e.g. Corcoran et al., 1995; Frith & Corcoran, 1996). It is also compatible with Frith's (1992) suggestion that such patients have a specific deficit in the representation of their own intentions, which does not generalise to the representation of others' mental states.

Frith's model predicts that the theory of mind performance of patients with paranoid symptoms and no behavioural signs should be better than that of patients with behavioural signs, but worse than that of controls. Data from Frith & Corcoran's (1996) study supported this prediction, and while the present test score results at the second-order level showed a similar tendency (for example, see Figure 4.1), the difference in test scores between normal controls and paranoid patients failed to reach significance. It should be noted at this point that with the exclusion of patients failing memory control questions, only 13 paranoid patients were entered into the second-order theory of mind analysis in the present study. In order to test whether this was an adequate sample size to reveal any effect, raw data were obtained for the performance of Frith & Corcoran's (1996) paranoid patients and normal controls on the single second-order false belief task in that study (R. Corcoran, personal communication). Taking only those subjects who passed the memory control question on that task gave 18 paranoids and 19 normal controls. The mean (and estimated population standard deviation, $\sigma$) scores for the normals and paranoids were 0.947 (0.223) and 0.667 (0.471) respectively (the maximum possible score on the task was
1.00). This gave an estimate of the effect size, $d$ of:

$$d = (0.947 - 0.667)/0.347 = 0.807$$

where 0.347 is an estimate of the total population standard deviation. To relate effect size to the harmonic mean of total sample size ($N_h$), we use the $\delta$ statistic, where:

$$\delta = d \sqrt{N_h/2}.$$

In the present study, the IQ-matched subsample analysis of second-order theory of mind test score included 17 normal controls and 13 paranoid schizophrenics. This means that based on an effect size of 0.807, $\delta$ for that comparison was 2.19. From tables, $\delta = 2.19$ corresponds approximately to a power of 0.70 for a one-tailed test at $p < 0.05$. Thus, the power of the present study for investigating the comparison of interest was lower than the conventionally accepted value of 0.80. This is unfortunate, but within the time constraints of the present study, was unavoidable, as data collection had to be terminated before any more patients falling into the paranoid group became available for testing\textsuperscript{13}. Future work should extend the present findings by recruiting sufficient paranoid subjects to give a power of 0.80. Assuming that the number of normal controls remained constant at $N = 17$, 9 more paranoid patients would be required. As these may take many months to recruit, an alternative would be to increase the number of normal controls (with low current IQs to keep them matched with the patients), as well as the number of paranoid patients. Thus, for example, if 6 more normal controls were obtained, only 4 more paranoid patients would be required.

\textsuperscript{13} In fact, any paranoid patients recruited for the subsequent studies detailed in this thesis (Chapters 5 and 7) were also tested on the neuropsychological battery from the present study. The $N = 13$ reported in the present analyses is the value of $N$ with those patients included.
In summary, then, results for the second-order theory of mind test score are generally in accord with Frith’s model, suggesting that schizophrenic patients with behavioural signs have clear deficits, while those in remission or with passivity features have no impairments. The data from paranoid patients are inconclusive based on the sample size presented here, although given the trends in the data, a significant deficit relative to normals may well appear when more patients are tested. Similar effects emerged from analysis of subjects’ explanations for their responses to the second-order theory of mind test question, and from analysis of the theory of mind composite scores. Thus, schizophrenics with behavioural signs gave significantly lower order explanations, and had a lower theory of mind composite than normal controls or patients in remission. Notably, Bowler (1992) also found that his schizophrenic patients (all of whom had behavioural signs) were significantly more likely than normals to give low order explanations for their responses. The single case with passivity symptoms in the present study produced a second-order explanation and had a high composite score, so again showed no evidence of theory of mind deficit. With current IQ controlled, the paranoid schizophrenics did not differ significantly from normal controls in the order of explanations given or in theory of mind composite score, although, as with the second-order test scores, trends were apparent which may become significant with larger subject numbers. Bowler (1992) suggested that there is little relationship between theory of mind competence and the tendency to produce explanations containing mental state terms. However, in the present study, the correlations between second-order theory of mind test score and order of explanation were significant for the normals, and approached significance for two of the patient groups. Examination of Bowler’s data reveals that he also

[14] With subsamples matched on pre-morbid (NART) IQ, the paranoid schizophrenics did have a significantly lower composite score than normals and patients in remission. However, this highlights the methodological weaknesses inherent in matching groups on pre-morbid IQ (see Chapter 1). Such matching ignores the often substantial pre-morbid / current IQ difference in patients, and makes it easier to obtain significant group differences relative to normal controls.
found trends in this direction, so it may be that he failed to find an effect because of the relatively small sample sizes in his study. Given, then, that order of explanation may well be an indicator of theory of mind ability, the group differences in explanations and composite scores found in the present study are consistent with Frith’s model.

Consideration of all theory of mind scores of the schizophrenic patients in the present study suggests a high level of competence at the first-order level, with symptom-specific deficits only appearing at the second-order level. The differences between these results and those of Frith & Corcoran (1996) have already been rationalised, but we should also examine whether the three other published studies of theory of mind in schizophrenia (Corcoran et al., 1995; 1997; Corcoran & Frith, 1996) are consistent with generally intact first-order ability. On the ‘hints task’, Corcoran et al. (1995) found poor performance by patients with behavioural signs and paranoid symptoms, and consideration of the form of the task suggests that many of the hinting scenarios required second-order theory of mind reasoning. For example, in the story where Paul says to his wife Jane, “I want to wear that blue shirt, but it’s very creased”, the subject has to infer that Paul intends that his wife knows that the shirt is to be ironed. By this argument, then, the results of Corcoran et al.’s hinting study are consistent with an impaired second-order theory of mind in schizophrenia, as found in the present study.

It was argued in Chapter 3 that in Corcoran & Frith’s (1996) maxims study, correct application of the Gricean maxims involved a first-order theory of mind, whereas politeness required mentalising at a second-order level. If this is the case, then the poor performance of paranoid patients in that study on only the politeness questions, is consistent with the notion of a specific second-order deficit in that group. Corcoran and Frith also found, however, that patients with behavioural signs scored poorly on both the maxims and politeness tasks, suggesting first- and second-
order deficits in that group. This is difficult to reconcile with the present findings, but there were several methodological problems with the maxims study. Firstly, the tasks were presented in a forced-choice format, but with only two choices given (correct or incorrect); the omission of a third (nonsense) choice therefore failed to control for the random responding often found in patients with behavioural signs. In addition, no control questions were posed in the maxims or politeness tasks, so as with Frith & Corcoran’s (1996) false belief study, poor memory may have been a contributory factor to the poor performance of patients with behavioural signs.

Finally, in the remaining published study of theory of mind to date, Corcoran et al. (1997) investigated subjects’ understanding of visual jokes requiring either a ‘physical’ or a ‘mental state’ explanation. As argued in Chapter 3, understanding of all the jokes in this study would probably have required a first order theory of mind, simply in order to appreciate the cartoonist’s ‘intention to amuse’. Correct responses to the mental state jokes would then have required further mentalising about the specific interactions shown in those cartoons. By this argument, Corcoran et al.’s finding that paranoid patients were selectively impaired on only the mental state jokes, can be taken as evidence for an intact first-order theory of mind (and deficits only at higher-order levels) in that patient group. The finding, however, that patients with behavioural signs had some problems with even the physical jokes, again suggests that that group may have had first-order theory of mind deficits, contrary to the findings of the present study. Examination of Corcoran et al.’s methodology, however, reveals that their subjects were simply shown the cartoons and asked to explain them. As this rather open-ended format provides little control for amotivation or inattention, it is perhaps not surprising that patients with behavioural signs (who are often unmotivated or inattentive) scored poorly on all the tasks. It is possible, given the results of the present study, that a replication of Corcoran et al.’s (1997) experiment with more rigorous control measures, would find behavioural signs patients only to be impaired on the mental state jokes.
The argument that schizophrenic patients show few (if any) first-order theory of mind deficits clearly runs counter to one aspect of Frith's (1992) model, namely that schizophrenics with behavioural signs are very similar to autistic people in lacking the ability to metarepresent. As we saw in Chapter 3, autistic people often do show first-order deficits, even on well-controlled, enacted tasks (e.g. Baron-Cohen et al., 1985), so the data from the present study provide a marked contrast with those findings. A possible reason for the difference is that many patients presenting with behavioural signs will have had an illness course characterised by acute onset in early adulthood, followed by deterioration to a state showing predominantly negative features. These people may be assumed to have had relatively normal theory of mind development during childhood and adolescence, and this early experience of mentalising is likely to have left them with some residual theory of mind skills, even at chronic stages of their illness. We might expect from this argument that the only schizophrenics to score as poorly as autistics on theory of mind tasks would be those whose illness followed the 'neurodevelopmental' course suggested by Murray et al. (1992). As we saw in Chapter 3, these are the patients who may have presented early in life with an autistic-like picture (so may have had problems in development of the theory of mind system), and who then go on to show an early-onset schizophrenic illness with a predominance of negative signs. This analysis predicts that a sample of patients with childhood-onset schizophrenia should perform poorly even on first-order theory of mind tasks; this remains to be investigated in future work. Interestingly, although none of the patients in the present study showed childhood onset, two who had the earliest onset (both at age 16 years) showed negative signs and had a theory of mind composite score of only 2 out of a possible 8 (less than one standard deviation below the mean for all the schizophrenics taken together). It may well be the case, therefore, that the earlier the schizophrenic illness begins, the more likely the patient is to show negative signs and a theory of mind deficit. In this context, it is notable that logistic regression analysis on the present data showed that of all the symptom scores, negative symptomatology was the strongest predictor of theory of
mind deficit in schizophrenia (although in the present analysis, age of onset was not a significant predictor).

**Executive function**

In the present study, executive function was tested using a simple reversal learning task in which the subject had to modify an established response set on the basis of external feedback, inhibiting prepotent responses to a previously-correct position. Frith (1992) suggested that efficient reversal learning requires the subject to represent his own goals and intentions, so a prediction from Frith’s model was that schizophrenic patients with behavioural signs (who are presumed to lack these representational abilities) should be worse than controls at learning a reversal. This was supported in the present study, where patients showing those features made more perseverative errors to the previously-correct position than both psychiatric and normal controls. There were no group differences on the ‘failure to maintain set’ variable (which can be thought of as a measure of the subjects’ ability to consistently maintain the correct problem-solving set), suggesting that the executive deficit in the schizophrenic patients was specifically one of perseveration on a previously-correct response. This result is in line with the findings of Partiot et al. (1992), who showed that a group of schizophrenic patients (whose symptoms were not specified) produced more perseverative errors than controls on a similar spatial reversal task. It also adds to the large body of evidence suggesting an association between the presence of behavioural signs and perseverative responding (e.g. on tasks such as the Wisconsin Card Sorting Test; Brown & White, 1991; Butler et al., 1992; Rosse et al., 1991). In particular, the present result supports the findings of Elliott et al. (1995), who showed that a group of schizophrenic patients with negative behavioural signs persevered at the simple reversal stage of the visual discrimination paradigm in the computerised CANTAB battery (Sahakian et al., 1990).
A number of methodological issues in the present study suggest that the specific deficit in reversal learning shown by patients with behavioural signs, was not an artefact of task difficulty or amotivation in that group. For example, every subject group, with the exception of behavioural sign schizophrenics, showed better performance at the simple reversal (SR) stage of the task relative to the simple discrimination (SD) stage, suggesting that SR was easier than SD. In addition, all groups were matched in performance on the harder (SD) stage of the task, suggesting in particular that the schizophrenics with behavioural signs were attending to the task in hand, and were as motivated as the other subjects to perform well. Elliott et al. (1995) suggested that the reversal deficit shown by their patients may have reflected general intellectual decline, as the size of the effect was greatly reduced in their sample when only those patients with current IQs of 85 or higher were included in the analysis. In the present study, however, the effect remained when a comparable analysis was performed, and the only evidence of IQ effects came from the comparison between all the schizophrenics with behavioural signs and the appropriate IQ-matched subgroup of normals, which showed the two groups to be matched in performance on the SR stage of the task. Inspection of the data showed that the behavioural signs patients in this analysis were performing just as badly as those with IQs of 85 or higher, so that the difference in results was a reflection of a tendency for the normals with lower IQs (who predominated in the IQ-matched comparison with all the behavioural signs patients) to make more errors on the SR stage of the task than those with higher IQs. This was not predicted, but it should be noted that at the end of the task a number of normal controls (but no schizophrenics) said that they had expected the task to be much more complex than it actually was, so that at the point when the reversal occurred they had felt that a simple change in response position was “too obvious”. This may explain why some of them produced rather complicated response patterns, and consequently had a relatively high error score. Such an analysis might predict that the sample of all patients with behavioural signs would have performed significantly more poorly than the IQ-matched normals on a more complex reversal
task, such as one requiring the learning of a number of successive reversals.

The schizophrenic patients in remission, or with paranoid symptoms, performed as well as controls on the executive task. This was predicted from Frith’s model, as both these groups are presumed to have an intact ability to represent their own goals and intentions. As discussed in the Introduction, it was predicted that patients with passivity symptoms would show some executive dysfunction (although much less than the behavioural signs patients) because they are thought to have an impaired ability to represent their own intentions, despite being able to represent goals. In view of this, it is interesting that although the single case with passivity experiences showed normal executive function on the present task, the logistic regression analysis revealed that passivity symptom scores for all the patients had some ability to predict executive function (although the main predictor was clearly the score for negative signs). In future investigations, it may be worth using a more complex executive task (e.g. the WCST or tests from the CANTAB), as this may be more sensitive than a simple reversal task to the cognitive deficits associated with passivity symptoms. Finally, in view of Frith’s (1992) suggestions that both positive and negative behavioural signs reflect an inability to represent one’s own goals, it is intriguing that logistic regression analysis on executive function revealed scores for negative signs to have strong predictive power, whilst scores for positive behavioural signs were not good predictors. A possible, but tentative, explanation for this is that, as we saw in Chapter 3, some cases of incoherent speech may involve relatively mild, rather than severe, impairments in the representation of goals. Such mild deficits may manifest as speech which is generally relevant to the task in hand, but in which information is conveyed in an inefficient way with no ‘editing out’ of ambiguous phrases. By this argument, it may be that scores for negative signs in the present study gave a stronger index of severe impairments in the representation of goals, so acted as better predictors of executive dysfunction.
Correlations between theory of mind and executive function

For the control subjects in the present study, no correlations appeared between measures of executive function or theory of mind ability (replicating the findings of Ozonoff et al., 1991a). In addition, as predicted on the basis of Frith’s (1992) model, schizophrenic patients in remission were the same as controls in showing no significant correlation between these two domains. Patients with paranoid symptoms also showed no correlation, which is consistent with a selective theory of mind deficit and intact executive function in that group. As discussed in the Introduction, Ozonoff et al. (1991a) found that for autistic subjects, scores on executive function and theory of mind tasks intercorrelated highly, suggesting (within Frith’s model) that autism may involve impairment of a single cognitive system underlying all forms of meta-representation. On the basis of the parallels drawn by Frith & Frith (1991) between autism and schizophrenia with behavioural signs, it was therefore predicted that a similar correlation would appear in the present study for the behavioural signs group. That no such correlation did appear may not now be too surprising, given the earlier discussion of theory of mind ability in patients with behavioural signs. It was pointed out above that many patients presenting with behavioural signs will have had an illness course characterised by acute onset in early adulthood, followed by deterioration to a state showing mainly negative features. Within Frith’s model, these people can be assumed to have experienced relatively normal development of their meta-representational abilities, with cognitive impairments only appearing at illness onset. As Frith’s model, and its supporting data, suggests that deficits in the representation of one’s own and others’ mental states can occur independently in schizophrenia, it is likely that although many patients with behavioural features do have impairments in both of these domains, the deficits are relatively independent of one another. Thus, at acute onset the patient may present mainly with paranoid delusions (i.e. impaired representation of others’ mental states), but after several years negative signs may emerge (as representation of own mental states is compromised). The independence
of these deficits may explain the absence of any correlation in the present study between theory of mind and executive function in patients with behavioural signs. One prediction arising from this model is that Murray et al.'s (1992) proposed 'neurodevelopmental' subgroup of schizophrenics with behavioural signs should show a correlation between theory of mind and executive function. If the preceding discussions are correct, these are people who present with an early autistic-like disorder, which then progresses to schizophrenia later in life. As autistic people appear to show a strong correlation between theory of mind ability and executive function (perhaps reflecting impaired development of the whole metarepresentational system), then schizophrenic patients with an early history of autistic-like behaviour may also show such a relationship.

**Correlates of insight**

Finally, various correlates of insight were investigated in the present study, to compare findings with the earlier studies reviewed in Chapter 3. No association was found in the present experiment between any symptom scores and the insight measure (replicating Cuesta & Peralta, 1994 and Lysaker & Bell, 1994), although other studies have found associations (e.g. with delusions: Amador et al., 1994; with behavioural signs: Kemp & Lambert, 1995). Similarly, no association was found in the present study between executive function and insight. Again this replicates Cuesta & Peralta (1994), but is contrary to the findings of Lysaker & Bell (1994) and Young et al. (1993), who did find such an association using the WCST as the executive function measure. As discussed in Chapter 3, a proposed quadratic relationship between insight and executive function (Startup, 1996) has the potential to reconcile the many contradictory results in this area, and it is unfortunate, therefore, that the present study failed to find any evidence in favour of that model. It should be noted, however, that the insight measure used in the present experiment was very crude (ratings of 0 to 3 on the PSE) in comparison to Startup’s measure (ratings of 0 to 22),

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so it may be that more fine-grained assessments of insight are required to reveal any effects.

4.5 Conclusions

The present study has enabled the reconciliation of a number of findings from existing studies of theory of mind in schizophrenia, and within Frith’s (1992) model has highlighted the similarities and differences between autism and schizophrenia. Thus, using a matched-tasks design, we have found that schizophrenic patients with behavioural signs (and possibly those with paranoid symptoms, although more subjects are needed) have a specific deficit in theory of mind. This deficit only appeared on second-rather than first-order tasks, so it was argued that, contrary to Frith’s model, most patients with behavioural signs may have more subtle theory of mind deficits than autistic people. On a reversal learning test of executive function, patients with behavioural signs showed more perseverative errors than controls, and it was suggested that this was consistent with Frith’s hypothesis of an impairment in the representation of own goals and intentions in that patient group. No correlations between theory of mind ability and executive function were significant for any of the subject groups, and in particular, the absence of a correlation for the behavioural sign schizophrenics was contrasted with the highly significant correlation between these two domains in autism (Ozonoff et al., 1991a). It was suggested that this highlights the important difference in age of onset between autism and most cases of behavioural sign schizophrenia, so that while theory of mind and executive deficits are linked in the former through impaired development of the metarepresentational system, they are relatively independent in the latter, where deficits in a developed system may only start to appear at the first breakdown.

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CHAPTER FIVE

THEORY OF MIND AND CONTEXT-RELATED MEMORY IN SCHIZOPHRENIA

5.1 Introduction

The experiment to be presented in this chapter will be a preliminary investigation of the proposal put forward in Chapter 3 that Frith's (1992) neuropsychological model of schizophrenia can be extended to explain the poor performance of some schizophrenic patients on tasks requiring the use of context. Performance of patients on a new context-sensitive task will be compared with that on a control task not requiring contextual knowledge, in an attempt to reveal a specific deficit in the use of context in schizophrenia. Scores in the context domain will then be compared with performance on the 'hints task' (Corcoran et al., 1995) in an attempt to relate any contextual deficits to theory of mind impairments.

Frith's model and the representation of knowledge

As discussed in Chapter 3, a core feature of Frith's (1987) model of the normal willed action system is a 'monitor' which enables us to distinguish between internally- and externally-generated actions. Many core features of schizophrenia can be explained in terms of impaired monitoring (or representation) by the patient of
various mental states so that, for example, impaired monitoring of own goals means either that actions are not generated (giving rise to negative signs), or inappropriate actions are not inhibited (positive signs). Impaired monitoring of own intentions results in self-generated actions being perceived as externally caused, and underlies passivity experiences (Frith, 1987; 1992). It was suggested in Chapter 3 that a similar monitor may apply to knowledge stored in long-term memory, as only then can we know, for example, whether stored concepts were self- or externally-generated. This monitoring of memory can be thought of as the representation of knowledge, so that a subject knows that (or how) he knows something, in the same way that the representation of goals involves him knowing that he wants to do something. As discussed in Chapter 3, impairments in this form of metarepresentation may underlie the poor source memory found in many schizophrenic patients (e.g. Bentall et al., 1991; Frith et al., 1991c), and may also explain their often poor performance on tests of episodic memory such as free recall and recognition (e.g. Calev, 1984; Clare et al., 1993; Huron et al., 1995; Tamlyn et al., 1992; see Chapter 2 of this thesis).

Studies of context-related memory in schizophrenia

A useful feature of the above model is that it may allow results from a number of other studies of schizophrenic patients to be explained in terms of impaired metarepresentation. For example, as discussed in Chapter 2, Done & Frith (1984) presented patients and controls with sentences from which the final word had been deleted such as, “Coming in, he took off his . . .”. After a few seconds, the stimulus word (e.g. “coat”) was presented for a brief duration, and the subject had to read this aloud. The exposure duration of the stimulus word increased with each cycle through the procedure, but in early cycles the duration was so short that subjects effectively had to guess a word to fit the context using their own knowledge. Done and Frith found that for all schizophrenic patients, variations in contextual constraint of the sentences had a normal influence on the threshold at which the stimulus word was
perceived (suggesting normal operation of automatic processes), but that some patients (especially those with negative signs) produced unsuitable responses before threshold was reached, suggesting a problem with the use of context in controlled processing. In addition, both acute and chronic patients had a tendency to produce perseverative responses (i.e. words elicited in earlier parts of the task) and neologisms, before threshold was attained. Similar conclusions were reached by de Silva & Hemsley (1977), who used the ‘Cloze’ procedure, in which subjects must guess the deleted words in passages of prose. These workers found that variation in contextual constraint had little effect on the performance of chronic schizophrenics, who scored poorly in every condition. Acute patients also performed poorly, and in fact their score fell as contextual constraint increased. Finally, Naficy & Willerman (1980; see Chapter 2) gave subjects sentences containing an underlined word which had both a common and uncommon meaning, and asked them to choose the context-appropriate meaning from a list provided. In line with the above results, schizophrenic patients (especially those with behavioural signs) were more likely than normals to choose the common word meaning when the unusual meaning was required.

**Extending Frith’s model to explain these findings**

A feature of all three of the above studies is that they required subjects to use knowledge stored in long-term memory to generate context-appropriate responses. As discussed in Chapter 3, poor task performance could conceivably be explained at two levels within Frith’s (1992) model. Firstly, patients may have difficulty representing their own goals, so either fail to generate responses, or fail to inhibit the first (often inappropriate) response which occurs to them. On the other hand, patients with intact representation of goals may still be impaired on the tasks because of deficits in the representation of their own knowledge. It is likely that all three of the tasks discussed above required retrieval of information from the semantic network,
which as we saw in Chapter 2, seems to be largely intact in schizophrenia. Thus, in order to complete Done & Frith’s (1984) sentence, “Coming in, he took off his . . .”, subjects may generate several possible responses from semantic memory (e.g. ‘coat’, ‘Wellington boots’), any of which could be considered appropriate. In order to choose between these possible responses, and give an optimal response, however, it is likely that subjects need to be simultaneously aware of the contextual constraints of the sentence while retrieving the words. This may require metarepresentation, so that the subject simultaneously represents something like, ‘I KNOW “The answer is not Wellington boots” (because people usually take off coats when entering rooms)’, and as a result produces the highly appropriate response of ‘coat’. In effect, the subject represents ‘Wellington boots’ as both a potentially appropriate answer and as an inappropriate answer given the current context. This cognitive process can be thought of as analogous to that discussed in Chapter 3 in relation to pretense (Leslie, 1987) where, for example, a banana can be represented both as a yellow fruit and as a telephone in a context in which it is held by someone and spoken into (e.g. ‘I KNOW “the banana is a telephone” (because people often speak into telephones’)).

At the extreme, we might think of subjects’ metarepresentation in context-dependent tasks as involving episodic remembering, so that for the example from Done and Frith’s study, the subject thinks back to particular occasions on which he himself entered a room, and concludes that coats are much more commonly removed in these situations than are Wellington boots (e.g. ‘I KNOW “The answer is coat” (because I took off my coat when I came in this morning)’). In most cases, however, subjects will not need to employ episodic memory to carry out the tasks, as representation of the contextual constraint at the same time as retrieval from semantic memory will be sufficient to promote optimal responding.

From this (admittedly speculative) analysis, the poor performance of some schizophrenics on context-dependent tasks can be explained in terms of impaired representation of own knowledge, with patients failing to simultaneously represent
contextual constraints while retrieving information from semantic memory. This model would predict patients to be differentially impaired in the generation of responses in a context condition, relative to a no-context condition, and this prediction is investigated in the present study.

Design of the study

In the present experiment, a new task requiring contextual processing (the ‘picture pieces task’ designed by F. Happé, personal communication) was given to patients with schizophrenia with the aim of revealing specific deficits in the use of context. Subjects had to name objects in four picture scenes, and in most cases the object could only be named appropriately by taking into account the whole context of the scene. For example, one picture showed a beach scene, and the subject had to name an ambiguous curved object that was lying on the beach. By analogy with the above arguments, subjects might generate possible responses such as, “shell” or “glove”, and then be aided in choosing the optimal response by metarepresentation of the form, ‘I KNOW “The answer is shell” (because shells are very commonly found on beaches)’. This paradigm clearly broadens the scope of earlier studies with schizophrenic patients, which have mainly involved the processing of words within sentence contexts. As discussed earlier, poor task performance could reflect either impaired representation of goals (i.e. difficulty in generating or inhibiting responses), or impaired representation of knowledge, so in an attempt to reveal specific deficits in the latter domain, a control condition not requiring contextual processing was also used. This required subjects to name (before viewing any of the picture scenes) the drawings, in isolation, of each of the objects that they would subsequently see in the scenes. Most of these drawings were ambiguous, in that they could have represented a number of different possible objects. Independent raters judged subjects’ responses for suitability based solely upon the appearance of the drawing in isolation, so it was predicted that any problems with the representation of goals would manifest at this
stage as either a failure to respond, or as a tendency to produce unsuitable answers. It was then expected that any additional impairment in the ability to use context would appear as a differential deficit relative to controls on the second stage of the task, where subjects had to name the objects in the picture scenes.

**Symptom-specific predictions**

Frith's (1992) model suggests that schizophrenic patients with behavioural signs are impaired in the generation and inhibition of responses (because of a failure to represent their own goals), and as discussed in Chapter 3, this has been confirmed empirically on tasks such as verbal fluency (e.g. Allen *et al.*, 1993). It was therefore predicted that, in the present study, behavioural sign patients would perform significantly worse than controls at naming the objects in the ‘isolation’ condition of the picture pieces task. Schizophrenic patients with positive symptoms, or those in remission, were expected to be unimpaired. As we saw earlier, most existing studies of schizophrenic patients that can be interpreted as requiring the representation of knowledge, suggest that impairments are associated with the presence of behavioural signs (e.g. studies of source memory: Frith *et al.*, 1991c; studies of episodic memory: Tamlyn *et al.*, 1992; studies of context-related memory: de Silva & Hemsley, 1977; Done & Frith, 1984; Naficy & Willerman, 1980). It was therefore predicted that behavioural sign patients would be impaired in the ‘context’ condition of the picture pieces task, with other patients showing intact performance. As the context condition is thought to require both the generation and inhibition of responses and the representation of one’s own knowledge, it was expected that behavioural sign patients would be more impaired at this stage of the task, relative to controls, than they had been in the isolation condition of the task.
Relation to performance on the ‘hints task’

The present study also tested subjects’ theory of mind ability using the ‘hints task’ (Corcoran et al., 1995). In line with the suggestion that this task is mainly a test of second-order theory of mind (see the Discussion in Chapter 4), it was predicted on the basis of Frith’s (1992) model, and the results from Chapter 4, that schizophrenics with behavioural signs or paranoid symptoms would perform more poorly than patients with passivity symptoms, patients in remission, or controls. As the hints task requires subjects to represent others’ mental states, while the picture pieces task may be a test of the ability to represent one’s own mental states (i.e. goals in the isolation condition, and goals and knowledge in the context condition), it is interesting to speculate about the predicted relationships between task performance for the various subject groups. We saw in Chapter 3 that Ozonoff et al. (1991a) found that for people with autism, theory of mind ability correlates with executive function (which within Frith’s model involves the representation of own goals), whereas normal and clinical controls show no correlation between test scores. This is compatible with impairment in autism of a single cognitive system underlying all forms of metarepresentation, and based on Frith & Frith’s (1991) suggestion that schizophrenics with behavioural signs are very similar to autistic people, we might expect theory of mind ability and scores on both stages of the picture pieces task to correlate for that group in the present experiment. However, based on the finding from the study in Chapter 4 of this thesis, that theory of mind ability and executive function are not correlated in schizophrenics with behavioural signs, (and the explanation of this in terms of differences in age of onset of autism and most cases of schizophrenia), we might expect impairments in theory of mind and the representation of own mental states to be relatively independent for behavioural sign schizophrenics in the present study. In addition, as patients with other symptoms, or those in remission, are expected to have an intact ability to represent their own goals and knowledge, no correlations should appear for them (or for control subjects) between
theory of mind ability and scores on either stage of the picture pieces task.

5.2 Methods

Subjects

Forty people with a DSM-IV diagnosis of schizophrenia (APA, 1994) were interviewed and tested as part of this study and the experiment discussed in Chapter 7. However, only 32 of these patients provided data on either or both of the neuropsychological tasks in the present study, so the following details only refer to those 32 patients. All subjects met the general inclusion criteria of competence in the English language, an age in the range 16 to 65, no history of leucotomy, neurological disability or drug/alcohol abuse, and a current verbal IQ on the Quick Test (Ammons & Ammons, 1962) of 70 or greater. About three-quarters of the schizophrenic subjects were inpatients at one of three psychiatric hospitals in London, whilst the rest lived in the community and attended psychiatric outpatient clinics. Demographic details of the patients are given in Table 5.1. All but one of the schizophrenics were taking neuroleptic medication at the time of testing; the daily dose of neuroleptic was converted to chlorpromazine equivalents using the tables provided by Foster (1989). The patients receiving anticholinergic medication (31% of the sample), were all taking 15 mg/day or less of procyclidine (mean daily dose was 12.0 mg, range 5 - 15 mg). As discussed in Chapter 4, Tamlyn et al. (1992) reported that in such doses, procyclidine does not affect the memory performance of schizophrenic subjects. However, in case some subjects did have memory deficits, the hints task in the present study was administered in such a way as to minimise its demands on memory. Clinical details of the schizophrenic patients are shown in Table 5.2.

As in the study detailed in Chapter 4, the schizophrenic subjects were allocated to one of four groups according to their signs and symptoms as revealed by
Table 5.1. Demographic details of subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age</th>
<th>Ethnicity</th>
<th>Hand</th>
<th>Years of Education</th>
<th>Percent Employed</th>
<th>Current Verbal IQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics</td>
<td>28:4</td>
<td>35.3 (9.7) [19 - 61]</td>
<td>22 : 10</td>
<td>29 : 3</td>
<td>11.1 (1.1) [9 - 14]</td>
<td>0.0</td>
<td>87.9 (9.9) [70 - 108]</td>
</tr>
<tr>
<td>Behavioural Signs</td>
<td>8:1</td>
<td>33.8 (10.0) [23 - 58]</td>
<td>7 : 2</td>
<td>8 : 1</td>
<td>11.8 (1.2) [11 - 14]</td>
<td>0.0</td>
<td>87.9 (9.9) [74 - 104]</td>
</tr>
<tr>
<td>Paranoid Symptoms</td>
<td>13:2</td>
<td>37.3 (10.6) [19 - 61]</td>
<td>10 : 5</td>
<td>14 : 1</td>
<td>10.7 (0.9) [9 - 12]</td>
<td>0.0</td>
<td>88.4 (8.2) [71 - 100]</td>
</tr>
<tr>
<td>Passivity Phenomena</td>
<td>1:0</td>
<td>34.0</td>
<td>0 : 1</td>
<td>1 : 0</td>
<td>12.0</td>
<td>0.0</td>
<td>70.0</td>
</tr>
<tr>
<td>Patients in Remission</td>
<td>6:1</td>
<td>33.0 (8.4) [25 - 46]</td>
<td>5 : 2</td>
<td>6 : 1</td>
<td>11.0 (1.0) [10 - 13]</td>
<td>0.0</td>
<td>89.1 (12.6) [71 - 108]</td>
</tr>
<tr>
<td>Psychiatric Controls</td>
<td>6:9</td>
<td>44.2 (11.4) [24 - 62]</td>
<td>15 : 0</td>
<td>11 : 4</td>
<td>12.1 (2.2) [10 - 16]</td>
<td>40.0</td>
<td>97.5 (13.0) [73 - 125]</td>
</tr>
<tr>
<td>Normal Controls</td>
<td>15:15</td>
<td>41.2 (14.6) [22 - 64]</td>
<td>26 : 4</td>
<td>25 : 5</td>
<td>12.4 (2.5) [10 - 19]</td>
<td>93.3</td>
<td>99.9 (11.5) [71 - 116]</td>
</tr>
</tbody>
</table>

S = standard deviation
Table 5.2. Clinical details of the schizophrenic patients

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Age at first Psychotic Episode</th>
<th>Duration of Illness</th>
<th>Medication Details</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (S) [Range]</td>
<td>Mean (S) [Range]</td>
<td>Percentage taking Neuroleptics</td>
</tr>
<tr>
<td>All Schizophrenics</td>
<td>23.2 (8.0) [12 - 57]</td>
<td>11.9 (8.6) [0.5 - 36]</td>
<td>97</td>
</tr>
<tr>
<td>Behavioural Signs</td>
<td>21.2 (4.8) [12 - 28]</td>
<td>12.6 (10.3) [2 - 36]</td>
<td>100</td>
</tr>
<tr>
<td>Paranoid Symptoms</td>
<td>24.3 (10.9) [15 - 57]</td>
<td>13.2 (7.5) [2 - 27]</td>
<td>100</td>
</tr>
<tr>
<td>Passivity Phenomena</td>
<td>29.0</td>
<td>5.0</td>
<td>100</td>
</tr>
<tr>
<td>Patients in Remission</td>
<td>23.3 (5.9) [17 - 34]</td>
<td>9.8 (9.2) [0.5 - 29]</td>
<td>86</td>
</tr>
</tbody>
</table>

S = standard deviation
a Present State Examination (PSE; Wing et al., 1974) on the day of testing. The
groups were formed in line with predictions from Frith’s (1992) model about the
symptom-specific performance of patients on theory of mind tasks; they were the
same groups as used in previous investigations of Frith’s model (Corcoran et al.,
1995; 1997; Corcoran & Frith, 1996; Frith & Corcoran, 1996). Allocation to groups
was carried out hierarchically, so that patients with symptoms from more than one
group were allocated to the group for which poorer task performance was predicted.
Thus, for example, a patient with behavioural signs and paranoid symptoms would
be allocated to the behavioural signs group. For the 32 patients in this study, group
membership broke down as follows:

**Behavioural signs.** There were 9 patients with negative (e.g. poverty of
speech, blunt affect, social withdrawal) or positive (e.g. inappropriate speech,
incongruous affect) behavioural signs. As the group was small, it was not subdivided
into positive and negative features.

**Paranoid symptoms.** There were 15 subjects describing positive symptoms
involving other agents (e.g. persecutory delusions or delusions of reference), but
showing no behavioural signs.

**Passivity experiences.** As with the previous study, only 1 patient reported
experiences thought to reflect impaired representation of willed intentions (e.g.
delusions of control, thought insertion, auditory hallucinations) in the absence of
delusional beliefs about other agents, or behavioural signs.

**Remission.** 7 patients showed no behavioural signs, and reported no positive
symptoms, on the day of testing.

Two control groups were recruited. The clinical group consisted of 15 non-
psychotic psychiatric outpatients with primary diagnoses of anxiety or unipolar
depression. They were all taking antidepressant or anxiolytic medication. The normal
control group comprised 30 normal volunteers, none of whom reported any history
of psychiatric disorder. Demographic details of the controls are given in Table 5.1.

The tasks

Hints task

This was devised by Corcoran et al. (1995), and tests subjects' ability to infer the real intentions behind items of indirect speech. As argued in the Discussion in Chapter 4, it is likely that it tests theory of mind ability at the second-order level. The task consists of ten short passages involving an interaction between two story characters (see Appendix III for full scripts of the stories). In each of the stories, one character drops a hint, and the subject is asked what the character really meant by the utterance. If the subject answers correctly at this stage, he receives 2 marks, and the next story is presented. If the subject gives an incorrect response (e.g. by just paraphrasing the hint), an extra line is read out by the experimenter, containing a more obvious hint, and the subject is asked what the story character wants the other one to do. A correct response at this stage receives 1 mark, whereas an incorrect response scores 0. Each story was read aloud by the experimenter in order to convey the appropriate prosodic information, but simultaneously the story was presented on a card in front of the subject to minimise memory problems. The stories were repeated for any subjects who requested it, so that all subjects were given the best chance of answering correctly. In this way, the task was a conservative test of theory of mind ability. In the present study, the task was presented in 2 sections of 5 stories each, as a ‘filler’ given between parts 1 and 2 and parts 3 and 4 of the picture pieces task (see below). Within each section, the 5 stories were given in the same order for all subjects, but within each subject group (and for the schizophrenic patients, within each symptom subgroup), the order of presentation of the sections was counterbalanced to avoid fatigue effects. A scoring protocol for each story was developed in consultation with R. Corcoran (personal communication), so responses can be assumed to have been judged in a similar way to those in Corcoran et al.'s (1995)
original study. The scoring protocol is given in Appendix III.

*Picture pieces* task

This was devised by F. Happé (personal communication). The task materials consisted of 4 picture scenes taken from an existing test battery (a beach (A), a living room (B), a mother and son doing the washing up (C), and a family preparing to go away on holiday (D)), each of which contained 7 to-be-named objects. The 4 scenes were divided into two pairs of two (A and C; B and D), and are shown in Appendix IV. There were also 28 cards, each of which had a picture of one of the to-be-named objects (in isolation from any context) stuck onto it. These pictures were simply cut out from photocopies of the scenes, so were exactly the same in size and appearance as the objects in the scenes. The task was presented in four sections, as part of the complete battery for the present study and the study detailed in Chapter 7. In the first section, the subject was presented one-by-one with 14 of the cards showing objects taken from two of the scenes, and was asked to guess what the pictures showed. Subjects were not told that the objects would be presented again later in a scene. The order of presentation was the same for every subject, but care was taken not to present all the objects from a given scene one after the other, in an attempt to avoid possible ‘priming’ effects, with subjects recognising a particular ‘theme’ (e.g. ‘beach objects’) and responding accordingly. After the subject had named all 14 objects, an intervening task (half of the hints task) was given as a filler. This was an attempt to avoid perseveration effects, because if the context scene had been presented immediately after the objects, subjects prone to perseveration may simply have given the objects in the scenes the same labels as when presented in isolation. In the second part of the picture pieces task, the two scenes were presented from which the 14 objects in part 1 had been taken, and subjects were simply asked to name each of the objects as it was pointed to. The order in which the objects were named was the same for every subject, but was different from the order in which they had been presented in isolation.
A filler task was then given, followed by parts 3 and 4 of the picture pieces task, which were presented in the same way as parts 1 and 2. Thus, subjects were asked to name the 14 objects in isolation taken from the remaining two scenes, and then an intervening task (the rest of the hints) was given. The two remaining scenes were then presented, with subjects being asked to name each of the objects in the scene as it was pointed to. Within each subject group (and for the schizophrenics, within each symptom subgroup), the order in which the two pairs of scenes were used was counterbalanced across the two halves of the task to avoid fatigue effects. The scoring scheme for subjects’ responses on this task is described in the Results section.

Procedure

Subjects were tested individually in a quiet room, and were paid £10.00 for their participation. All patients gave written, informed consent to take part in the study, which had been approved by the Ethical Committees of the relevant hospitals. Copies of the patient information sheet and consent form are given in Appendix V. At the start of the session, all schizophrenic patients received an abbreviated Present State Examination Version 9 (PSE; Wing et al., 1974), to assess their current signs and symptoms. An abbreviated form was given because the length of the test battery precluded a full PSE; the primary aim of the PSE interview was therefore to accurately place the patient within the correct symptom subgroup. Normal and psychiatric controls were not given the PSE, but the absence of any history of psychosis was checked verbally and from case notes where relevant. Thereafter, all subjects received the same test protocol. This consisted of an assessment of handedness using the Annett criteria (Annett, 1970) and a test of current verbal IQ using Form 2 of the Quick Test (Ammons & Ammons, 1962). The picture pieces and hints tasks were then presented as above, as part of a larger battery including tasks discussed in Chapter 7. The whole assessment lasted up to 2 hours; any subjects who felt unable
to complete everything in one session were asked if they would finish the tasks on
the following day.

5.3 Results

Demographic and clinical variables (see Tables 5.1 and 5.2)

The single schizophrenic subject with passivity experiences in the absence of
behavioural signs or paranoid symptoms was treated as a single case, and is described
towards the end of the Results section. This left three schizophrenic symptom sub-
groups. The duration of illness data for these groups were normally distributed, and
parametric analysis of variance revealed the groups to be matched on this variable.
The remaining clinical data were non-parametric, so Kruskal-Wallis one-way
analyses of variance were used to explore group differences. These revealed the three
symptom groups to be matched on age of onset of illness and daily dose of
anticholinergic medication. The groups differed, however, in their chlorpromazine
equivalent daily dose of neuroleptic medication [$\chi^2(2) = 7.9; p = 0.02$]. Post hoc
comparisons revealed that the schizophrenics in remission were taking significantly
less medication than both the paranoid and behavioural signs groups. To avoid any
possible confounding effects of these differences in medication dosage, some of the
later analyses in this section will use subgroups of patients matched on dosage. These
were formed by systematically eliminating those schizophrenics with paranoid or
behavioural features who were taking the highest doses of medication, until all three
groups were matched. In practice, there were two data points missing for the medica-
tion of patients in remission, and one point missing for a patient with behavioural
signs, so the subgroups of schizophrenics matched on medication dosage comprised
5 patients in remission (rather than the total of 7), 13 with paranoid symptoms and
7 with behavioural signs.
For the three schizophrenic symptom groups and the two control groups, any differences in demographic variables were examined using Kruskal-Wallis one-way ANOVAs. These showed all 5 groups to be matched on sex, age, handedness, ethnicity and years of education. As is commonly found, the normals were significantly more likely to be employed than all other groups \( \chi^2(4) = 53.2; p < 0.0001 \). Examination of current verbal IQ scores for the whole sample revealed them to be normally distributed, so group differences were explored using parametric analysis of variance, with post hoc multiple comparisons using Tukey’s honestly significant difference (HSD) test. This revealed a highly significant group difference \( F(4, 71) = 4.29; p = 0.004 \); see Appendix VI for the full ANOVA table], with normal controls having a significantly higher verbal IQ than the schizophrenics with paranoid symptoms or behavioural signs. To avoid any possible confounding effects of these group differences, some of the later analyses in this section will use subgroups of subjects matched on verbal IQ. These were formed by systematically eliminating normal controls with the highest verbal IQs until all groups were matched. In practice, this meant that four normals were eliminated, so that the mean (and standard deviation) of verbal IQ in the remaining group of 26 normals was 97.6 (10.6). Subject numbers in all other groups remained unchanged.

**Hints task**

All subjects who attempted the hints task completed all five stories in both halves of the task. Of the 32 schizophrenic patients taking part in this study, only 30 gave data on the hints task, as the case with passivity symptoms and one patient with paranoid symptoms did not wish to attempt it. As discussed earlier, responses on each story were given a score of either 0, 1 or 2 using the scoring protocol in Appendix III; the maximum score possible for each subject was therefore 20. For the normal controls only, scores on both halves of the task were compared and found to be matched [paired samples t-test: \( t(29) = 1.84; p = 0.08 \), two-tailed], although there was
a trend for stories 6 to 10 to be more difficult than stories 1 to 5. Scores from both halves of the task were therefore combined for each subject in the study, and mean scores by group are shown in Table 5.3 and Figure 5.1 (where error bars represent the standard error of the mean).

Table 5.3. Mean scores on the hints task by group

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) hints score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>14.4 (3.0)</td>
</tr>
<tr>
<td>(N = 9)</td>
<td></td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>16.1 (1.9)</td>
</tr>
<tr>
<td>(N = 14)</td>
<td></td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>16.1 (2.9)</td>
</tr>
<tr>
<td>(N = 7)</td>
<td></td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>17.2 (1.2)</td>
</tr>
<tr>
<td>(N = 15)</td>
<td></td>
</tr>
<tr>
<td>Normal controls</td>
<td>17.7 (1.4)</td>
</tr>
<tr>
<td>(N = 30)</td>
<td></td>
</tr>
</tbody>
</table>

S = standard deviation

As data were non-parametric, with marked differences in variance across groups, group differences were analysed using Kruskal-Wallis one-way ANOVA. This revealed a highly significant group difference \[\chi^2(4) = 16.1; p = 0.003\], and post hoc multiple comparisons showed that normal controls scored significantly higher on the task than schizophrenics with behavioural signs or paranoid symptoms. The difference in scores between schizophrenics in remission and those with behavioural signs did not reach significance, even at trend level. To investigate the relationship between task performance and verbal IQ, score was correlated with IQ for each of the subject groups in turn; this correlation was only significant for the normal controls \[\rho(30) = 0.32; p = 0.05\,\text{one-tailed}\]. Contrary to the results of Corcoran et al. (1995), there was no significant association between verbal IQ and hints score for the whole schizophrenic sample taken together. To avoid any possible confounding effects of IQ, group differences were then compared using the verbal IQ-matched subgroups of subjects discussed earlier. The comparison was still significant \[\chi^2(4) = 13.6; p =\]
Figure 5.1. Mean scores on the hints task by group

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs
0.009], and post hoc investigations revealed that normal controls still scored significantly higher than schizophrenics with behavioural signs. The comparison between normals and patients with paranoid symptoms now only approached significance at p < 0.12, one-tailed test. Finally, the relationship between task performance and medication dosage for the combined schizophrenic sample, and for each of the three schizophrenic groups in turn, was investigated by correlating score with chlorpromazine equivalent daily dose. The correlations in all cases were non-significant, so it can be assumed that performance differences between the schizophrenic groups on the hints task were not artefacts of medication differences.

**Picture pieces task**

Most subjects provided data on both halves of the picture pieces task, but due to oversight by the experimenter, one normal control was not asked for context responses on pictures B and D. One psychiatric control completed only half the task (pictures A and C and their isolation objects), and one gave no context responses on pictures B and D. All the schizophrenic patients completed the task, except for one who only did half of it (pictures A and C and their objects). For these few incomplete data sets, missing points were treated as missing values in the analysis. Inspection of the data revealed no evidence of priming effects occurring in the naming of objects in isolation. Thus, no subject reported noticing a ‘theme’ (e.g. ‘beach objects’ for the items from picture A), and none of the responses within a given section of the task showed marked thematic similarity.

**Scoring scheme**

For the scoring of responses to the objects in isolation, three judges were given each of the 28 pictures of the objects, and a list of all of the names given by all subjects for each object (a total of 492 different names). At this stage, none of the judges had seen the objects in the context scenes, so had no idea what they ‘really’
were. The judges were separately asked to rate each response for its 'suitability' as a description of the object in the drawing, based simply on the appearance of the drawing on the card. A five-point scale was used, ranging from 0 to 4 (0 = very unsuitable; 1 = fairly unsuitable; 2 = can't say/don't know; 3 = fairly suitable; 4 = very suitable). Spearman's rank correlation coefficients between ratings were all highly significant, at \( p < 0.001 \) \[ \rho(1 \text{ vs. } 2) = 0.60; \rho(1 \text{ vs. } 3) = 0.59; \rho(2 \text{ vs. } 3) = 0.64 \]. Using the formula given by Howell (1997), Kendall's coefficient of concordance (W) was calculated for all three judges simultaneously. For this calculation, \( \sum T_j^2 \) (i.e. the sum of the squares of the sums of ranks for each rated item) was \( 3.302 \times 10^6 \), which gave \( W = 0.685 \). As all three judges were similar in their ratings, these were combined to give a single 'index of suitability' (ranging from 0 to 12) for each isolation response. All responses to the objects in isolation, together with their suitability indices, are given in Appendix IV. The judges were then given the four context scenes, and a list of all names given by all subjects for the objects in context (a total of 160 different responses). The judges were then separately asked to rate each response for its 'suitability' as a description of the object in the context of the scene, using the same five-point rating scale as above. Spearman's rank correlation coefficients between ratings were all highly significant at \( p < 0.001 \) \[ \rho(1 \text{ vs. } 2) = 0.65; \rho(1 \text{ vs. } 3) = 0.75; \rho(2 \text{ vs. } 3) = 0.73 \], and for the calculation of Kendall's W, \( \sum T_j^2 \) was \( 1.164 \times 10^7 \), giving \( W = 0.752 \). As all three judges were similar in their ratings, these were combined to give a single 'index of suitability' (minimum 0, maximum 12) for each response in context. All context responses, and their suitability indices, are given in Appendix IV.

**Analysis of suitability of responses**

Subjects' responses to objects in isolation and in context were scored using the appropriate suitability indices. Initially, scores were calculated separately for the two halves of the task (i.e. isolation and context with pictures A and C, then isolation and context with pictures B and D), as results from the normal control group showed
one half of the task to be easier than the other (see below). Occasions on which a subject said “don’t know” were treated as missing values, so that for each subject, mean suitability scores were calculated by dividing the sum of scores by the number of responses given. Mean suitability scores for the isolation and context conditions on each half of the task are shown by group in Table 5.4.

Table 5.4. Mean suitability scores by group on each half of the picture pieces task

<table>
<thead>
<tr>
<th>Subject Group</th>
<th>Mean (S) isolation score (A &amp; C)</th>
<th>Mean (S) context score (A &amp; C)</th>
<th>Mean (S) isolation score (B &amp; D)</th>
<th>Mean (S) context score (B &amp; D)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behavioural signs</td>
<td>8.73 (0.70)</td>
<td>9.51 (2.18)</td>
<td>9.20 (1.55)</td>
<td>10.89 (1.06)</td>
</tr>
<tr>
<td>Paranoid symptoms</td>
<td>8.71 (1.37)</td>
<td>10.00 (0.60)</td>
<td>9.10 (0.95)</td>
<td>10.94 (0.53)</td>
</tr>
<tr>
<td>Patients in remission</td>
<td>9.64 (0.83)</td>
<td>10.01 (0.88)</td>
<td>9.51 (0.58)</td>
<td>11.26 (0.60)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>9.38 (0.89)</td>
<td>10.63 (0.67)</td>
<td>9.04 (1.11)</td>
<td>11.59 (0.30)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>9.55 (0.59)</td>
<td>10.69 (0.60)</td>
<td>9.68 (0.77)</td>
<td>11.59 (0.34)</td>
</tr>
</tbody>
</table>

S = standard deviation

Analysis of responses from the normal controls revealed that the isolation conditions in the two halves of the task were matched on difficulty [paired samples t-test: t(29) = 0.69; ns], but the context condition with pictures B and D was easier than that with pictures A and C [t(28) = 6.72; p < 0.001]. More importantly, within each half of the task, the isolation condition was significantly more difficult than the context condition [A and C: t(29) = 7.97; p < 0.001; B and D: t(28) = 13.19; p <
0.001], so the ‘ideal’ design of having isolation and context conditions matched on difficulty was not achieved in this study.

Parametric analyses of variance were used to investigate between- and within-group differences in scores because most distributions of scores within groups were approximately normal. However, as variances were markedly heterogeneous, and there was some skew in the distributions of scores within the context conditions, all data were logarithmically transformed before the ANOVAs were calculated. For scores on the first half of the task (objects and pictures A and C), a mixed model ANOVA was firstly performed, using group as the between-subjects factor and amount of contextual information (i.e. zero in the isolation condition and high in the context condition) as the within-subjects factor. This revealed a significant main effect of group \([F(4, 71) = 5.52; p = 0.001]\); there was also a significant main effect of contextual information \([F(1, 71) = 22.90; p < 0.001]\), reflecting the fact that subjects scored higher in the context condition than in the isolation condition. The group x contextual information interaction failed to reach significance \([F(4, 71) = 0.97; p = 0.43]\), suggesting that, overall, there was no marked difference between groups in the influence of context. Between-group one-way ANOVAs were then performed separately on isolation and context responses, with Tukey’s HSD tests (at a significance level of 0.05) used for post hoc multiple comparisons. These calculations revealed a significant group difference in performance on the isolation condition \([F(4, 71) = 3.61; p = 0.01]\), with normal controls scoring significantly higher than patients with paranoid symptoms. Similarly, there was a significant group difference on the context condition \([F(4, 71) = 3.23; p = 0.02]\), with normal and psychiatric controls scoring higher than schizophrenics with behavioural signs. Output tables for all of the above ANOVA calculations are given in Appendix VI. For each group in turn, scores in the two conditions were then compared using paired-sample t-tests; for normals, psychiatric controls and paranoid schizophrenics, the context condition was significantly easier than the isolation condition (all \(p <

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However, the patients in remission, and those with behavioural signs, scored comparably in the two conditions \([t(6) = 0.91; p = 0.40, \text{two-tailed}, \text{and } t(8) = 0.48; p = 0.65, \text{two-tailed}, \text{respectively}]\). As a measure of whether the isolation and context conditions were tapping different cognitive skills, scores in both conditions were correlated for each subject group in turn; all values of Pearson’s \(r\) failed to reach significance \((p > 0.3)\).

To investigate the relationship between verbal IQ and scores in the context and isolation conditions on the first half of the task, scores were correlated with IQ for each of the groups in turn\(^{15}\). Although all values of \(p\) failed to reach significance with a Bonferroni-corrected \(p\)-value of 0.005 (i.e. 0.05/10), some correlations were significant at the conventional \(\alpha\)-level [e.g. for paranoid schizophrenics, \(\rho(15) = 0.45; p = 0.05, \text{one-tailed, in the isolation condition}\), so it was decided to repeat the above ANOVA calculations using the verbal IQ-matched subgroups of subjects. All effects remained with this manipulation [main effect of group: \(F(4, 67) = 4.95; p = 0.001, \text{with the same significant post hoc comparisons on Tukey’s HSD test; main effect of contextual information: } F(1, 67) = 21.07; p < 0.001; \text{group x context interaction: } F(4, 67) = 0.92; p = 0.46\)]. To investigate the possible confounding effects of group differences in medication dose, scores were then correlated with dosage for each of the schizophrenic groups in turn, and for the schizophrenic sample as a whole. All correlations were non-significant, so it can be assumed that differences in task performance between the schizophrenic groups were not artefacts of medication differences.

Scores on the second half of the picture pieces task (i.e. objects and pictures \(B\) and \(D\)) were analysed in the same way as those on the first half of the task. A

\(\text{\textsuperscript{15}}\) The correlations were done on non-transformed data, so non-parametric correlation coefficients (Spearman’s \(\rho\)) are reported here (and in subsequent investigations of IQ effects).
mixed model ANOVA with group as the between-subjects factor and amount of contextual information as the within-subjects factor, again revealed a significant main effect of group [F(4, 67) = 3.16; p = 0.02] and a significant main effect of contextual information [F(1, 67) = 198.27; p < 0.001]. As with the first half of the task, the interaction between group and amount of contextual information failed to reach significance [F(4, 67) = 0.84; p = 0.50]. Between-group one-way ANOVAs were then performed separately on isolation and context responses; these revealed groups to be matched in the isolation condition [F(4, 69) = 1.69; p = 0.16], but there was a significant group difference in the context condition [F(4, 67) = 5.64; p = 0.0006], with Tukey’s HSD tests revealing that the normal and psychiatric controls scored significantly higher than the schizophrenics with paranoid symptoms or behavioural signs. Output tables for these ANOVA calculations are given in Appendix VI. For each group in turn, scores in the two conditions were then compared using paired-sample t-tests. For all groups, the context condition was significantly easier than the isolation condition, but the significance levels varied, with p < 0.001 for all groups other than the behavioural sign schizophrenics, for whom p < 0.01. As a measure of whether the isolation and context conditions were tapping different cognitive skills, scores in both conditions were correlated for each subject group in turn; all values of Pearson’s r failed to reach significance (all p > 0.18, two-tailed).

To investigate the possible confounding effects of verbal IQ, scores in the isolation and context conditions from the second half of the task were correlated with IQ for each of the subject groups in turn. Although all values of ρ failed to reach significance with a Bonferroni-corrected p-value of 0.005, some correlations were significant at the conventional α-level [e.g. for the schizophrenics in remission, ρ(7) = 0.70; p = 0.04, one-tailed, in the context condition], so it was decided to repeat the above ANOVA calculations using the verbal IQ-matched subgroups of subjects. All effects remained with this manipulation [i.e. main effect of group: F(4, 63) = 2.68; p = 0.04, with the same significant post hoc multiple comparisons on Tukey’s HSD
test; main effect of context: $F(1, 63) = 192.44; p < 0.001$; group x context interaction: $F(4, 63) = 0.77; p = 0.55$. Finally, for each schizophrenic group in turn, and for the whole schizophrenic sample, scores were correlated with medication dosage to investigate any possible confounding effects of this variable. All values of $p$ failed to reach significance with a Bonferroni-corrected $p$-value of 0.006 (i.e. 0.06/8), but some of the correlations were significant at the conventional $\alpha$-level [e.g. for the schizophrenics with behavioural signs, $\rho(8) = -0.78; p = 0.02$, two-tailed, in the context condition], so it was decided to repeat the above ANOVA calculations using the subgroups of schizophrenics matched on medication dose (together with the full control groups). All effects remained with this manipulation [main effect of group: $F(4, 61) = 2.82; p = 0.03$, with the same significant post hoc multiple comparisons on Tukey’s HSD test; main effect of amount of contextual information [$F(1, 61) = 154.37; p < 0.001$; group x contextual information interaction: $F(4, 61) = 1.00; p = 0.42$].

With both halves of the task combined, mean scores were finally calculated for each subject in both isolation and context conditions by dividing the sum of scores in each condition by the number of responses given in that condition. The means of these scores for each subject group are shown in Table 5.5 and Figure 5.2 (where error bars represent the standard error of the mean). Between- and within-group differences were again analysed using parametric analyses of variance on log-transformed data. A mixed model ANOVA was firstly performed, using group as the between-subjects factor and amount of contextual information as the within-subjects factor. This revealed a significant main effect of group [$F(4, 71) = 5.83; p < 0.001$], and a significant main effect of contextual information [$F(1, 71) = 139.81; p < 0.001$]; the group x contextual information interaction failed to reach significance [$F(4, 71) = 1.11; p = 0.36$]. Between-group one-way ANOVAs were then performed separately on isolation and context responses, with Tukey’s HSD tests used for post hoc multiple comparisons. These revealed that the groups differed significantly in
performance on the isolation condition \[ F(4, 71) = 3.43; p = 0.01 \], with normal controls scoring significantly higher than schizophrenics with paranoid symptoms. Similarly, there was a significant group difference in the context condition \[ F(4, 71) = 4.82; p = 0.002 \], with normal and psychiatric controls scoring higher than the schizophrenics with behavioural signs. Output tables for the above ANOVA calculations are given in Appendix VI. For each group in turn, scores in the two conditions were then compared using paired-sample t-tests. For all groups except the behavioural sign schizophrenics, the context condition was significantly easier than the isolation condition at \( p < 0.002 \); for the patients with behavioural signs, this difference only just reached significance \[ t(8) = 2.09; p = 0.04, \text{ one-tailed} \]. As a measure of whether the isolation and context conditions were tapping different cognitive skills, scores in both conditions were correlated for each subject group in turn; all values of Pearson's \( r \) failed to reach significance (all \( p > 0.18 \), two-tailed).

### Table 5.5. Mean suitability scores in isolation and context conditions for whole task

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) isolation suitability score</th>
<th>Mean (S) context suitability score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>8.91 (0.83)</td>
<td>10.18 (1.55)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms &amp; no behavioural signs</td>
<td>8.93 (0.98)</td>
<td>10.46 (0.34)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>9.58 (0.43)</td>
<td>10.64 (0.61)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>9.22 (0.87)</td>
<td>11.02 (0.44)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>9.61 (0.48)</td>
<td>11.13 (0.32)</td>
</tr>
</tbody>
</table>

\( S = \) standard deviation

As before, verbal IQ was correlated with scores for each of the subject groups in turn, and because some values of \( p \) were significant at the conventional \( \alpha \)-level
Figure 5.2. Mean suitability scores in the isolation and context conditions

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs
[e.g. for the paranoid schizophrenics, ρ(15) = 0.53; p = 0.02, one-tailed, in the context condition], the above ANOVA calculations were repeated using the IQ-matched subgroups of subjects. With this manipulation, all effects remained [main effect of group: F(4, 67) = 5.04; p = 0.001, with the same significant post hoc comparisons on Tukey’s HSD test; main effect of contextual information: F(1, 67) = 131.03; p < 0.001; group x contextual information interaction: F(4, 67) = 1.06; p = 0.38]. Finally, the ANOVA calculations were repeated using the three schizophrenic groups matched on medication dosage (together with the full control groups), and all effects again remained [main effect of group: F(4, 65) = 5.81; p < 0.001, with the same significant post hoc multiple comparisons on Tukey’s HSD test; main effect of contextual information: F(1, 65) = 169.08; p < 0.001; contextual information x group interaction: F(4, 65) = 0.86; p = 0.49].

**Analysis of extreme responses**

For each subject, the percentage of responses scoring either 0 or 1 (i.e. those rated the most unsuitable of all by the judges), was then calculated separately for the isolation and context conditions of the whole picture pieces task. In the context condition, these responses are a particularly strong measure of the failure to use contextual knowledge. For each subject group, the mean percentage of highly unsuitable responses is shown in Table 5.6 and Figure 5.3 (where error bars represent the standard error of the mean).

Data were non-parametric, so were transformed in order to allow investigation of possible interaction effects. As the scores consisted of proportions, an arcsine transformation was used, with \( Y = 2 \arcsin \sqrt{q} \), where \( q = (\text{percentage score}/100) \) (Howell, 1997). Between- and within-subjects effects were then explored using parametric analysis of variance in the same way as with earlier analyses. A mixed model ANOVA was firstly performed, using group as the between-subjects factor and amount of contextual information (i.e. zero in the isolation condition and high in the
context condition) as the within-subjects factor. This revealed a significant main effect of group \( [F(4, 71) = 6.74; p < 0.001] \). There was no main effect of contextual information \( [F(1, 71) = 0.01; p = 0.99] \), however, and the interaction between group and amount of contextual information also failed to reach significance \( [F(4, 71) = 1.28; p = 0.29] \). One-way between-subjects ANOVAs were then performed separately for scores in the isolation and context conditions of the task, with Tukey’s HSD tests used to investigate *post hoc* multiple comparisons. These calculations revealed that groups differed significantly in the isolation condition \( [F(4, 71) = 3.19; p = 0.02] \), with the paranoid schizophrenic group having a significantly higher mean percentage of very unsuitable responses than normal controls. Groups also differed significantly in the context condition \( [F(4, 71) = 5.73; p = 0.0005] \), with schizophrenics in remission, and those with behavioural signs, giving a higher proportion of very unsuitable responses than normal or psychiatric controls. The output tables for the above ANOVAs are given in Appendix VI.

Table 5.6. Mean percentage of highly unsuitable responses by group over whole task

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean percent (S) of isolation responses</th>
<th>Mean percent (S) of context responses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>3.64 (3.10)</td>
<td>5.76 (7.97)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms &amp; no behavioural signs</td>
<td>4.29 (4.05)</td>
<td>2.17 (2.28)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>1.59 (1.99)</td>
<td>3.61 (2.97)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>1.44 (3.26)</td>
<td>0.48 (1.26)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>1.49 (2.99)</td>
<td>0.83 (1.54)</td>
</tr>
</tbody>
</table>

S = standard deviation

For each group in turn, paired-sample t-tests were then used to compare the
Figure 5.3. Mean percent of highly unsuitable responses in each task condition

- Context
- Isolation

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs
percentage of highly unsuitable responses in the isolation and context conditions. Only for the schizophrenics in remission was this comparison significant \( t(6) = 2.38; p = 0.03, \) one-tailed], with these patients giving a higher percentage of very unsuitable responses in the context condition than in the isolation condition. For the other subject groups, none of the comparisons approached significance (all \( p > 0.18, \) two-tailed). For each of the groups in turn, percentage of highly unsuitable responses in the isolation and context conditions were then correlated together; only for the schizophrenics in remission was this correlation significant \( r(7) = +0.70; p = 0.04, \) one-tailed].

To investigate possible confounding effects of verbal IQ, percentage of highly unsuitable responses in the isolation and context conditions were correlated with IQ for each of the subject groups in turn. For the paranoid schizophrenic group, the correlation was significant in the context condition \( p(15) = -0.60; p = 0.01, \) one-tailed], so the above ANOVA calculations were repeated using the IQ-matched subgroups of subjects. For the mixed model investigation, all effects were unchanged \( [\text{main effect of group: } F(4, 67) = 6.25; p < 0.001; \text{main effect of contextual information: } F(1, 67) = 0.01; p = 0.99; \text{group }\times\text{contextual information interaction: } F(4, 67) = 1.27; p = 0.29], \) but it should be noted that separate one-way between-group comparisons for the isolation and context conditions now revealed the groups to be matched in the isolation condition, although they still differed in the context condition \( [\text{context: } F(4, 67) = 5.51; p = 0.0007, \) with the same significant \textit{post hoc} comparisons on Tukey’s HSD test as before]. Thus, the main effect of group in the mixed model ANOVA was almost exclusively due to differences in the context condition of the task. Finally, for the whole schizophrenic sample, and for the three symptom groups in turn, medication dosage was correlated with scores in both the isolation and context conditions. In all cases, values of \( p \) were non-significant, so we can assume that differences between the patient groups in task performance were not artefacts of medication differences.
Analysis of repetitions

As schizophrenic patients have often been reported to perseverate or repeat words and phrases in their speech (e.g. Manschreck et al., 1981; see Done & Frith, 1984), the frequency of repetitions was calculated for subjects in the present study. As the four sections of the picture pieces task were spaced at wide intervals over a test period of about 1 hour, responses were only considered as repetitions if they were produced more than once within a given section. For each subject, the percentage of repetitions in the isolation and context conditions was calculated by dividing the number of repetitions in each of those conditions by the total number of responses in each condition. Mean percentage of repetitions is shown by group in Table 5.7.

Table 5.7. Mean percentage of repetitions by group over the whole task

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean percent (S) of isolation repetitions</th>
<th>Mean percent (S) of context repetitions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>1.65 (2.66)</td>
<td>0.00 (0.00)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms &amp; no behavioural signs</td>
<td>2.51 (2.29)</td>
<td>0.25 (0.96)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>4.91 (4.23)</td>
<td>0.53 (1.40)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>2.69 (4.19)</td>
<td>0.00 (0.00)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>3.04 (3.41)</td>
<td>0.12 (0.65)</td>
</tr>
</tbody>
</table>

S = standard deviation

Data were arcsine transformed to allow parametric analyses of variance to be carried out. A mixed model ANOVA using group as the between-subjects factor and amount of contextual information as the within-subjects factor, revealed no significant main effect of group [F(4, 71) = 1.55; p = 0.20]. There was similarly no significant interaction between group and amount of contextual information [F(4, 71) =
0.50; \( p = 0.74 \), although the main effect of contextual information was highly significant \( [F(1, 71) = 44.25; p < 0.001] \), reflecting the fact that subjects produced a smaller mean percentage of repetitions in the context condition compared to the isolation condition. An output table for this ANOVA calculation is given in Appendix VI. For each group in turn, percentage of repetitions in the two conditions were compared using paired-sample t-tests. These revealed that, at the one-tailed level, all groups made significantly more repetitions in the isolation condition than in the context condition, although the significance levels varied widely (e.g. \( p < 0.002 \) for normal controls and paranoid schizophrenics; \( p = 0.04 \) for the behavioural sign schizophrenics). For all groups, the correlation between percentage of repetitions in the isolation and context conditions failed to reach significance.

**Neologisms**

Following Done & Frith (1984), all responses were examined for the presence of new words made up by the subject. None were produced by any subjects.

**Single case with passivity symptoms**

As with the study discussed in Chapter 4, one schizophrenic patient reported delusions of control in the absence of any paranoid symptoms or behavioural signs. He was excluded from all of the above analyses, as Frith's (1992) model makes specific predictions about the metarepresentational abilities of such patients. He was a 34-year old, unemployed male with 12 years of education and a verbal IQ of 70. Five years had elapsed since the onset of his illness. He did not wish to do the hints task, so unfortunately no measure of theory of mind ability was taken for this patient. He did, however, perform half of the picture pieces task (objects and pictures A and C), and on this his mean suitability score in the isolation condition was 9.31 (a value similar to that of 9.38 for the psychiatric controls; see Table 5.4). In the context condition, his mean suitability score was 8.62, a value lower than the lowest group
mean in that condition (9.51 for the patients with behavioural signs). He gave no highly unsuitable responses in either condition of the picture pieces task, and produced no repetitions on that task.

**Relation between theory of mind and responses on the picture pieces task.**

For each of the subject groups in turn, scores on the hints task were correlated with mean suitability scores and the percentage of highly unsuitable responses, in both the isolation and context conditions of the picture pieces task. As 20 correlations were performed, a Bonferroni-corrected p-value of 0.0025 was used; with this criterion all correlations failed to reach significance. With a less conservative \( \alpha \)-level of 0.05, most correlations were still non-significant, although for the schizophrenics in remission, hints scores correlated with percentage of highly unsuitable responses in both the isolation condition \([p(7) = -0.67; p = 0.05, \text{one-tailed}]\) and the context condition \([p(7) = -0.71; p = 0.04, \text{one-tailed}]\).

**5.4 Discussion**

**Performance on the hints task**

Inspection of scores on the hints task reveals that schizophrenic patients with behavioural signs performed somewhat better in the present study than the corresponding patients in Corcoran et al.'s (1995) original investigation (the mean scores being 14.4 and 12.8 respectively), whereas the normal controls in both studies performed similarly. This difference may reflect the fact that, in the present study, the hints stories were simultaneously read aloud to subjects and given in a written form, whereas Corcoran et al. only presented the stories orally. It is likely that giving stories in both formats optimises memory and comprehension, and this manipulation may have a greater effect on the performance of patients with generalised cognitive
impairment (e.g. those with behavioural signs), than for subjects whose memory and attention are intact (e.g. normal controls). Despite the generally higher hints scores of the behavioural signs patients in the present study, however, these subjects still performed significantly worse than normal controls (with verbal IQ controlled), and in this respect, Corcoran et al.'s (1995) findings were replicated. As discussed in Chapter 4 of this thesis, all of the hinting stories involve interactions between two story characters, one of whom always intends that the other recognises a particular demand. As a result, the stories probably require theory of mind reasoning at the second-order level, so the poor performance of patients with behavioural signs in the present study is consistent with their second-order deficits on false belief tasks discussed in Chapter 4. Moreover, the results from the present study are clearly compatible with Frith's (1992) model, which suggests that behavioural signs are associated with deficits in the representation of others' mental states.

The present experiment also replicated Corcoran et al.'s (1995) finding of no significant difference between the scores of remitted patients and controls; this is consistent with other studies of theory of mind in schizophrenia (Corcoran et al., 1997; Corcoran & Frith, 1996; Frith & Corcoran, 1996), and supports Frith's (1992) suggestion that theory of mind deficits are symptom-specific. However, the significant difference found by Corcoran et al. (1995) between scores of patients with behavioural signs and those in remission, was not replicated here. To enable a power analysis of the present data, the effect size, d, for the comparison between behavioural sign and remitted patients, was calculated using the mean scores (and standard deviations) of those groups in Corcoran et al.'s study. These values were 12.8 (4.5) and 18.0 (2.6) respectively, giving:

$$d = \frac{18.0 - 12.8}{3.55} = 1.465$$

To relate effect size to the harmonic mean of total sample size ($N_h$), we use $\delta$, where:
\[ \delta = d \sqrt{\frac{N/2}{d}}. \]

The present study involved data from 9 patients with behavioural signs and 7 in remission, so with \( d = 1.465 \), \( \delta \) for that pairwise comparison was 2.91. From tables, \( \delta = 2.91 \) corresponds approximately to a power of 0.90 for a one-tailed test at \( p < 0.05 \), so we might have expected a significant group difference to have emerged in the present experiment. One explanation for the absence of such a difference is that some of the patients classified as remitted may in fact have had underlying paranoid symptomatology which was not identified by the PSE interview. Frith’s (1992) model would predict such patients to perform poorly on theory of mind tasks, which would have the effect of lowering the group mean and reducing the chance of a significant difference relative to patients with behavioural signs\(^{16}\). Inspection of the case notes of all 7 ‘remitted’ patients in the present study did, in fact, reveal two whom the Consultant found to be frequently guarded, or unwilling to discuss their thoughts, and who it was felt “may still have underlying paranoid delusions, despite denial of any symptoms”. These two patients had low hints scores (14 and 11) which, within Frith’s model, would be compatible with the presence of paranoid symptomatology. Group differences on the hints task were therefore re-analysed with these two patients allocated to the paranoid, rather than remission group. Kruskal-Wallis one-way ANOVA with IQ-matched groups then gave \( \chi^2(4) = 17.0; \ p = 0.002 \), and post hoc analyses revealed that the difference between the behavioural sign patients and those in remission now approached significance at \( p < 0.10 \), one-tailed. This result is much more compatible with the findings of Corcoran et al. (1995), especially when we consider the additional factor (discussed earlier) of generally better performance by the behavioural signs patients in the present study relative to those in Corcoran et al.’s experiment, which would have had the effect of further reducing the difference in scores relative to patients in remission. In general, this analysis highlights one

\(^{16}\) In this context, it is notable that the group mean for the remitted patients in the present study was 16.1, compared to 18.0 in Corcoran et al.’s (1995) study.
potential problem with all symptom-related research in schizophrenia, namely that
the identification of positive symptoms relies heavily upon the patient’s truthful self-
report at interview, something which may be incompatible with a paranoid patient’s
view of the interview process.

Regarding the relationship between positive symptoms and hints score in the
present study, the single patient with passivity experiences would not do the task, so
it was not possible to confirm Corcoran et al.’s (1995) finding of intact theory of
mind in such cases. In line with Frith’s (1992) model, Corcoran et al. found that
patients with paranoid symptoms scored significantly worse than normal controls on
the hints task. However, the present study only revealed a trend in that direction,
once verbal IQ was controlled, so a power analysis of the present data was carried
out by calculating the effect size, d, for the comparison between paranoid patients
and normals in Corcoran et al.’s study. As the mean (and standard deviation) hints
score of the paranoid group in that study was 15.4 (3.6), this gave:

\[ d = \frac{(18.1 - 15.4)}{2.6} = 1.038 \]

As the present study involved 26 normal controls in the IQ-matched analysis, and 14
paranoid patients, we had \( \delta = 3.13 \); from tables, this corresponds to a power of
approximately 0.93 for the normal/paranoid comparison, so again we might have
expected a significant difference to have emerged here. These results can again be
rationalised by assuming that two of the patients classified as remitted on the basis
of the PSE, would have been better placed in the paranoid symptoms group. When
post hoc multiple comparisons were applied to the Kruskal-Wallis ANOVA on the
newly classified patients, a significant difference between normal controls and
paranoid patients did indeed emerge. If we accept, then, that two of the patients
classified as remitted in the present study may have had underlying paranoid
symptoms, the results are wholly compatible with Frith’s (1992) model, and suggest
that paranoid delusions are associated with second-order theory of mind deficits. Furthermore, the paranoid patients in the present study were less impaired on the hints task than those with behavioural signs, which is again consistent with Frith's suggestion that behavioural signs reflect the most severe deficits in theory of mind.

A final point involves the relationship between verbal IQ and performance on the hints task. Corcoran et al. (1995) found that for their whole schizophrenic sample taken together, IQ and hints score were highly intercorrelated, whereas no such relationship appeared for a combined group of psychiatric and normal controls. On this basis, Corcoran et al. suggested that, because of their impaired theory of mind, patients were relying upon general intellectual abilities to perform the task. In the present study no such correlation appeared, despite many patients having clear theory of mind deficits, and it remains possible that the association found by Corcoran et al. was artefactual, perhaps reflecting the fact that their remitted patients had higher IQs than their patients with behavioural signs.

Performance on the picture pieces task

As mentioned in the Results section, the performance of normal controls on this task revealed that the context condition was significantly easier than the isolation condition. This is perhaps not too surprising given that, relative to the isolation condition, the context condition provided more information to help in the naming of objects. Thus, although it has been suggested here that, on a cognitive level, the context condition required one more 'component process' than the isolation condition (i.e. representation of goals and knowledge, compared to only representation of goals), it is likely that subjects who could effectively implement that extra process (e.g. controls) benefitted from the additional information provided. As indicated by Chapman & Chapman (1973; see Chapter 1), such differences in task difficulty mean that the results of the present study must be interpreted cautiously, with consideration
given to possible psychometric artefacts. A possibility for future work might, therefore, be the use of a paradigm in which both conditions were matched on difficulty; this would necessarily require the naming of different objects in the context condition than in the isolation condition, because the use of the same objects in both conditions always faces the problem of the context condition being easier. Thus, one possible study might use the same isolation stimuli as the present task, together with context pictures which were harder than in the present experiment.

Examination of suitability scores in the isolation condition revealed that, despite this condition being harder than the context condition, all subject groups had mean scores of 8.7 or higher (out of a possible 12.0), so there was certainly no psychometric problem of floor effects in this condition. Furthermore, the isolation condition had some power to discriminate between groups because, with data from both halves of the task combined (and also with only the data from object sets A and C), normal controls had significantly higher suitability scores than patients with paranoid symptoms. In itself, this group difference was unexpected, as it had been predicted that only patients with behavioural signs would have problems in generating suitable isolation responses, because of difficulties in the representation of their own goals. While inspection of the data in Tables 5.4 and 5.5 reveals that the behavioural signs patients scored similarly to the paranoid patients in both suitability of isolation responses and percentage of highly unsuitable isolation responses (so that group differences between patients with behavioural signs and controls may well have appeared with larger subject numbers), the fact remains that it was the paranoid patients who, in the present study, performed significantly worse than controls in the isolation condition. All group differences were checked with re-classification of the two ‘remitted’ patients discussed earlier (who it was felt may have had underlying

17 Paranoid patients were also more likely to produce highly unsuitable responses in this condition than were normal controls, but that difference disappeared when IQ was controlled.
paranoid symptoms), and the paranoid/normal difference in mean suitability of isolation responses remained. This result is difficult to explain in terms of the task analysis presented here (and in terms of Frith’s model, which suggests that patients with paranoid symptoms and no behavioural signs should have an intact ability to inhibit inappropriate responses), and should be replicated before its significance is accepted. One possibility is that it reflected a non-specific dimension of ‘illness severity’ (which was not controlled in this study) so that, for example, paranoid patients may have been, on average, more severely ill than the other groups, and as a result less able to apply intact cognitive skills to the task in hand.

Regarding suitability scores in the context condition, all groups had a mean score less than 11.6 (out of 12.0), so there were no psychometric problems with ceiling effects here. Although, for normals, the context condition was significantly easier than the isolation condition (so may have had more discriminating power), it is notable that with data from both halves of the task combined (and also with only the data from pictures A and C), the schizophrenics with behavioural signs were the only patients to have significantly lower context suitability scores than controls. When only data from pictures B and D were examined, both patients with behavioural signs and those with paranoid symptoms had significantly worse context suitability scores than controls. As data from normals showed context conditions B and D to be easier than conditions A and C, this latter result is consistent with pictures B and D being more discriminating than A and C, and therefore having the power to reveal context impairments in both behavioural signs and paranoid groups. Notably, all of the above effects remained when the data were re-analysed after reclassification of the two ‘remitted’ patients thought to have underlying paranoid symptoms. Overall, by contrasting group differences in the isolation and context conditions, we can see that the isolation condition revealed only deficits in the paranoid patients, whereas the (more discriminating) context condition revealed deficits primarily in the patients with behavioural signs; context deficits in the
paranoid patients only appeared when the context condition was at its most discriminating (pictures B and D). A possible interpretation of these findings is that, while paranoid patients had impairments in the isolation condition, patients with behavioural signs (and to a lesser extent, those with paranoid symptoms), had deficits in the context condition which were unrelated to those in isolation. In line with the proposed task analysis, these context deficits may reflect particular problems with the representation of own knowledge, independent of any deficits in the representation of own goals.

This interpretation is generally supported by the other statistical analyses, which showed, for example, that suitability scores in the isolation and context conditions were uncorrelated for all the subject groups, suggesting that both conditions were tapping separate cognitive abilities. In addition, while all groups scored significantly better in the context condition than the isolation condition, the schizophrenics with behavioural signs differed from all other groups in that, for them, this difference between conditions only just reached significance (e.g. \( p = 0.04 \), one-tailed, in the combined analysis of both halves of the task). While these results (and the lack of any significant interaction between scores in the context and isolation conditions) suggest that, contrary to predictions, behavioural signs patients could use contextual information to some extent, the data are compatible with a smaller degree of ‘contextual advantage’ in behavioural signs patients than in other subjects, consistent with some problems in the representation of their own knowledge. Finally, examination of percentage of extreme responses showed that, of the schizophrenic groups, both the patients with behavioural signs and those in remission had a significantly higher proportion of very unsuitable context responses than controls. For the patients in remission this result may have been unreliable, as it disappeared when the

\[18\] This absence of correlation is particularly notable for the paranoid schizophrenic group, as it implies that the context deficits in that group did not simply reflect the same factors (e.g. illness severity) which caused deficits in the isolation condition.
data were re-analysed (with re-classification of the two ‘remitted’ patients suspected of having underlying paranoid symptomatology), so within the theoretical framework presented here, these findings are again consistent with behavioural signs being particularly associated with problems in the representation of own knowledge.

Examination of percentage of repetitions (i.e. occasions on which subjects produced the same answer more than once within a given section of the task) showed no group differences in either condition, and each group showed some advantage of context in that the percentage of repetitions in the context condition was always smaller than that in the isolation condition\(^\text{19}\). In addition, no subjects produced any neologistic responses in either condition of the task. These results are at odds with those of Done & Frith (1984), who found strong trends for both acute and chronic schizophrenics to produce repetitions in their sentence-completion task (which can be thought of as analogous to the context condition of the present task); in addition, 8 chronic patients in Done and Frith’s study produced neologisms in the visual version of the task. These different results probably reflect methodological features of the paradigms used. Thus, whilst both Done and Frith’s study, and the present experiment, required subjects to generate context-appropriate items from long-term memory, the paradigm used here aided subjects’ choice of response by providing drawings of the to-be-named objects. Although these drawings were often ambiguous, they narrowed down the possible range of responses, reducing the chance of repetitive responses. In contrast, in the very early stages of Done and Frith’s task, before perceptual threshold was approached, subjects had the freedom to produce any response at all, without the constraints of stimulus features; this may have increased the chance of repetitive responses. Similarly, the production of neologisms in Done

\(^{19}\) The difference between conditions was smallest for the patients with behavioural signs, but this cannot be interpreted as further evidence of reduced ‘contextual advantage’ in those patients, as it was primarily a reflection of their low mean number of repetitions in the isolation condition compared to other groups.
and Frith’s experiment probably reflected the fact that, as perceptual threshold was approached, subjects generated answers based on an incorrect perception of the stimulus word; thus, for example, subjects may have perceived the stimulus word ‘coat’ incorrectly as ‘cout’. As no such paradigm was used in the present study, the production of neologisms was not encouraged here. Given the operation of these processes in Done and Frith’s study, the production of repetitions and neologisms by their schizophrenic patients may have reflected problems with the inhibition of inappropriate responses, an impairment known to be associated with the presence of positive behavioural signs (e.g. Allen et al., 1993; Liddle & Morris, 1991; see Chapters 2 and 3). In contrast, control subjects would have been more likely than patients to inhibit any repetitive or neologistic responses, because of their presumed intact ability to suppress inappropriate output.

Similar arguments can be used to explain the differences in performance of acute and chronic schizophrenic patients on the ‘Cloze’ procedure in the study by de Silva & Hemsley (1977). As discussed in the Introduction, the scores of chronic patients in that study were relatively uninfluenced by increasing contextual constraint, whereas the performance of acute patients actually deteriorated as context increased. If we assume that patients in both groups had some positive or negative behavioural signs, then we might expect (on the basis of earlier arguments) that both groups had some problems with the representation of their own knowledge; this may explain the generally poor performance of both groups as context increased. In addition, however, patients with positive behavioural signs (who would be more likely to be in the acute group), can be assumed to have had a further impairment in the ability to inhibit inappropriate output resulting, for example, from associations made to external stimuli. As higher context conditions in de Silva and Hemsley’s task would have involved the presentation of greater numbers of external stimuli (as words in the Cloze passages), the poorer performance of acute patients with increasing context can be explained in terms of a failure to inhibit increasing numbers of
inappropriate associations.

In summary, the results of the present study have provided some evidence for impairments in the use of context in schizophrenic patients with behavioural signs (and to a lesser extent, paranoid symptoms\(^{20}\)), and it has been argued that this may reflect a problem with the representation of own knowledge, independently of any impairments in the representation of own goals. Although the symptom-specific predictions for the present study were based solely upon the poor performance of patients with behavioural signs on other tasks thought to require the representation of knowledge (e.g. source memory: Frith et al., 1991c; episodic memory: Tamlyn et al., 1992; context-related memory: de Silva & Hemsley, 1977; Done & Frith, 1984; Naficy & Willerman, 1980), we might speculate about how an impairment in the ability to represent one’s own knowledge could contribute to the aetiology of behavioural signs or positive symptoms. One possibility is that the production of appropriate behaviour (e.g. walking quietly around a supermarket rather than singing loudly while shopping), requires both the ability to generate action and inhibit inappropriate action (through the representation of goals), and an awareness of what actually constitutes appropriate behaviour in that context (through the representation of knowledge). The latter might involve metarepresentation of the form, ‘I KNOW “people don’t sing while shopping”’, or even episodic remembering such as ‘I KNOW “people I saw here last time weren’t singing”’. Thus, someone with intact abilities to generate or inhibit actions could still conceivably show behavioural signs, because of a lack of awareness of the action appropriate to a particular situation. For patients with paranoid symptoms, the evidence from the present study suggests that any contextual deficits are less pronounced than for patients with behavioural signs. Thus, paranoid patients may, for example, have an awareness of what constitutes

\(^{20}\) The single case with passivity symptoms also had a very low suitability score in the context condition, so there may be an association between impaired use of context and the presence of delusions in general, rather than just paranoid delusions.
appropriate behaviour in many everyday contexts, but they may lack the ability to represent knowledge in particular situations relevant to their own delusions. One example of this may be the patient who believes (erroneously) that certain colleagues at work intend to harm him. It may be that, in times past, these colleagues have been pleasant and helpful to the patient, but a failure of episodic memory (e.g. 'I KNOW “they helped me out yesterday”') would mean that those past instances were not considered in evaluation of the current situation. Interestingly, this may equate with the ‘failure to weigh contradictory evidence’, suggested by Chapman & Chapman (1988; see Chapter 2) as being an important factor in delusional thinking.

The above suggestions are obviously speculative and, as discussed earlier, the proposed relationships between symptoms and impaired use of context should be tested using context and no-context tasks matched in difficulty. A possible extension to such a study is suggested by work from Reich & Cutting (1982), who asked schizophrenic patients and controls to describe the contents of a picture placed before them. Notably, the picture was the same as picture D (a family going away on holiday) used in the present study. Reich and Cutting found that their schizophrenic subjects (who were all classified as ‘acute’ by the Research Diagnostic Criteria of Spitzer et al., 1975), were more likely than normal controls to begin their descriptions with ‘local’ elements of the picture (e.g. individual objects), and to end with more ‘global’ elements such as the ‘theme’ of the picture. Normal subjects, on the other hand, were significantly more likely than the schizophrenics to begin with a global analysis, and then provide the detailed evidence on which it was based. Reich and Cutting suggested that this implied a ‘bottom-up’ cognitive strategy in schizophrenics, compared to a ‘top-down’ strategy in normals, and in terms of the analysis given in the present study, it may have reflected schizophrenic deficits in the representation of context during task performance. A useful extension to any replication of the present study might, therefore, ask subjects to describe pictures as well as naming objects in isolation and context situations. The above arguments
would suggest that impairments in the naming of objects in context would correlate with the tendency to adopt a ‘bottom-up’ descriptive strategy.

**Correlations between performance on the hints and picture pieces tasks**

As discussed in the Results section, all correlations between scores on the hints task, and scores in the isolation and context conditions of the picture pieces task, failed to reach significance for all subject groups with application of appropriate Bonferroni corrections. Notably, the only correlations to be significant with a conventional $\alpha$-level of 0.05 (those for the schizophrenics in remission), may have been unreliable, as they disappeared when the data were re-analysed with re-classification of the two ‘remitted’ patients thought to have had underlying paranoid symptoms. As discussed in Chapter 3, research with autistic individuals has shown that theory of mind ability (Ozonoff et al., 1991a) and free recall and temporal order memory (Bennetto et al., 1996; Ozonoff et al., 1991a) are highly correlated with executive function, whereas no such correlations appear for normal or clinical controls. If we accept that all of these cognitive skills require metarepresentation (e.g. Frith, 1992), then the results are consistent with impairment, in autism, of a single cognitive system underlying all forms of metarepresentation. Now, based on the suggestion by Frith & Frith (1991) that schizophrenic people with behavioural signs are similar to autistics in lacking the ability to metarepresent, we might have expected correlations to have appeared for that group, in the present study, between hints score (reflecting theory of mind ability), suitability scores in the isolation condition of the picture pieces task (thought, like executive function, to reflect the representation of own goals), and suitability scores in the context condition of the picture pieces task (thought to reflect the representation of own goals and own knowledge). That no such correlations did appear is consistent with the finding in Chapter 4 of this thesis, that theory of mind and executive function are uncorrelated in schizophrenics with behavioural signs. This was interpreted as reflecting the fact that, unlike autistics, most
schizophrenic people with behavioural signs will have experienced relatively normal
development of their metarepresentational abilities during childhood, so that impair­
ments in the representation of own goals and others’ mental states occur relatively
independently at the onset of illness. The findings of the present study confirm these
results, and suggest that impairments in the representation of own knowledge (as
measured by performance on a context task) are also independent of other meta­
representational deficits in patients with behavioural signs and paranoid symptoms.
In addition, the lack of correlation between task scores for the control groups and
remitted schizophrenics in the present study, is consistent with the similar absence
of correlation for the controls in studies by Ozonoff et al. (1991a) and Bennetto et
al. (1996).

By analogy with the arguments in Chapter 4 of this thesis, we might expect
that the only schizophrenic patients to show intercorrelations between performance
on the various tests of metarepresentation used here, would be those whose illness
followed the ‘neurodevelopmental’ course suggested by Murray et al. (1992). These
patients may have shown an initial autistic presentation, followed by an early-onset
schizophrenic illness characterised by a predominance of negative signs; unlike the
majority of schizophrenic patients, they may be assumed to have had problems in the
development of their metarepresentational abilities.

5.5 Conclusions

In the first part of the present study, theory of mind in schizophrenia was
explored using Corcoran et al.’s (1995) hints task. The findings of Corcoran et al.’s
study were substantially replicated, in that patients with behavioural signs, and those
with paranoid symptoms, scored significantly worse than normal controls. It was
pointed out that, as the hints task may be a test of second-order theory of mind, these
findings are consistent with the results in Chapter 4 of this thesis, which suggested
second-order mentalising deficits in schizophrenia. In the second part of the present study, it was argued that deficient representation of own knowledge in schizophrenia may be able to explain a number of existing results showing impaired use of context in guiding retrieval from long-term memory. It was predicted that schizophrenics (especially those with behavioural signs) would be more impaired than controls on a task condition requiring the naming of objects in context, than on a condition requiring the naming of objects in isolation. Some evidence was found in favour of this proposal, although all subject groups showed some advantage of context, suggesting that any deficits in schizophrenia are relative rather than absolute. Tentative proposals were advanced to relate symptomatology to deficits in the representation of knowledge, and it was pointed out that a more rigorous test of the theoretical model would require the use of context and no-context tasks matched on difficulty. Finally, no correlations emerged for any of the subject groups between scores in the theory of mind domain and the ability to appropriately name objects in isolation or context. It was suggested that, within the theoretical framework developed here, these results are consistent with the finding in Chapter 4 of no correlations between theory of mind and executive function in schizophrenia. In particular, the absence of any correlations for the patients with behavioural signs contrasts markedly with the case of autism (e.g. Bennetto et al., 1996; Ozonoff et al., 1991a), and is consistent with the suggestion in Chapter 4 that autism may involve impaired development of the metarepresentational system, while most cases of schizophrenia involve dysfunction of a fully-developed metarepresentational capacity, with impairments in separate domains of mental state representation being relatively independent of one another.
CHAPTER SIX

‘WEAK CENTRAL COHERENCE’ IN AUTISM AND SCHIZOPHRENIA

6.1 The case of autism

Focus on detail rather than gestalt

As mentioned at the end of Chapter 3, a notable feature of autism, which at first sight is difficult to explain in terms of impaired metarepresentation, is the tendency of autistic individuals to concentrate on details at the expense of gestalt. Thus, in his early observations of autistic people, Kanner (1943, reprinted in Kanner, 1973) felt that a central feature of the disorder was “the inability to experience wholes without full attention to the constituent parts” (p. 38). As discussed by Bailey et al. (1996), the ease with which autistic children detect changes in detail may have some bearing on their resistance to change, and the “markedly restricted repertoire of activity and interests” used as a diagnostic feature in DSM-IV (APA, 1994, p. 66; see Section 3.2 of this thesis). A focus on detail may also have some bearing on the relatively high incidence in autism of ‘islets of ability’ (e.g. good rote memory), or in-depth knowledge of unusual topics; in addition, it may be relevant to the ‘idiot savant’ phenomenon, whereby a small proportion of (usually autistic) people develop an isolated talent in areas such as music, drawing or mathematics, to a level higher than that found in normals (see Bailey et al., 1996; Happé & Frith, 1996).
In addition to the above clinical features, a large body of research using a number of different paradigms has suggested that autistic people pay more attention to parts than wholes, and fail to extract the global meaning from stimuli (see U. Frith & Happé, 1994, for a review). For example, Hermelin & O’Connor (1967) found that children with autism performed similarly on tasks requiring immediate recall of meaningful sentences or random word strings; in contrast, mentally handicapped controls scored significantly better in the sentence condition, which suggests that meaning influenced their performance. Similarly, Tager-Flusberg (1991) found that normal and learning disabled controls showed significantly better recall of semantically-related nouns than unrelated nouns, whereas the performance of verbal MA-matched autistic children was little affected by word meaning. As discussed by U. Frith (1989), autistic people show a characteristic ‘spiky’ performance profile on the subtests of the Wechsler intelligence scales (e.g. Wechsler, 1981), and this may also partly reflect their failure to appreciate global meaning. For example, peak autistic performance is usually found on the Block Design subtest, in which subjects must use patterned blocks to construct (as quickly as possible) a specific gestalt pattern provided for them on a card. U. Frith (1989) suggested that this good performance may reflect the relative ease with which autistic people can ‘mentally segment’ the provided gestalt into its constituent parts (thereby ignoring the global pattern), and as discussed later in this section, this was empirically confirmed by Shah & Frith (1993).

On the basis of such findings, U. Frith (1989, p. 101) proposed that a cognitive deficit specific to autism was failure of the “normal operation of central coherence [which] compels us human beings to give priority to understanding meaning”. At this stage, she suggested that this ‘weak central coherence’ may underlie the poor theory of mind ability of autistic people, as impaired mentalising could reflect a failure to search for global meaning in social situations. However, on the basis of several later studies (discussed below) showing that weak central coherence is a characteristic of
autistic subjects irrespective of their performance on standard first- and second-order theory of mind tasks, U. Frith & Happé (1994) revised this proposal, suggesting that impaired theory of mind and weak central coherence may be two separate cognitive characteristics of autism: Impaired mentalising may reflect damage to a modular system, and can explain Wing & Gould’s (1979) autistic ‘triad’ of deficits in socialization, communication and pretend play (see Section 3.2 of this thesis), whereas weak central coherence may be more akin to a cognitive style (with autistic people at one end of a normal continuum), that can explain several of the abnormal non-social features of autism, such as the focus on detail rather than gestalt.

In the present chapter, it will be suggested that it may still be worth trying to explain the social and non-social abnormalities of autism in terms of only one cognitive deficit: impaired metarepresentation. Thus, in Chapter 5, we saw that some people with schizophrenia have problems in using context to guide retrieval of suitable information from long-term memory (LTM), and it was proposed that such a deficit could be explained in terms of impaired metarepresentation. By analogy with the above discussion, a failure to use context can be thought of as the inability to use global meaning to guide responses, so impaired use of context in schizophrenia may be a manifestation of ‘weak central coherence’ in that disorder. In the sections that follow, some of the data regarding weak central coherence in autism will be discussed, and a possible re-interpretation proposed in terms of metarepresentational theory. This extension is clearly speculative, but it has the advantage of being more parsimonious than an account of autism in terms of two independent cognitive abnormalities; as discussed below, it also makes several specific predictions, so is empirically falsifiable.

Correct pronunciation of homographs in context

U. Frith & Snowling (1983) devised a task in which subjects had to read aloud
sentences containing homographs with two possible pronunciations (e.g. ‘tear’ as a rip in cloth, or moisture from the eyes). The sentences were devised so that correct pronunciation of the homographs depended upon context, so subjects’ pronunciations gave a measure of their understanding of the word meaning in context. Importantly, before reading the sentences, subjects were given no warning that some of the words had two possible pronunciations. U. Frith & Snowling (1983) found that autistic subjects were specifically impaired, relative to dyslexic or normal controls matched on reading age, at giving the correct word pronunciation in context, (and in a separate experiment, at choosing a suitable word to fill gaps in stories), so clearly these findings are compatible with the notion of weak central coherence in autism. In a more recent study, Happé (1997) extended these results, using conditions in which the homograph came either before or after the sentence context. She predicted that young normal controls would show an effect of context position, pronouncing more of the homographs correctly when they appeared after the context than when they appeared before it; in contrast, autistic subjects were predicted to be relatively insensitive to context position. These predictions were confirmed, and furthermore, Happé found that insensitivity to context was a feature of all subgroups of her autistic sample formed according to competence on standard first- and second-order theory of mind tasks. She concluded (in line with U. Frith & Happé, 1994), that weak central coherence is a feature of all people with autism, and is independent of theory of mind ability.

By analogy with the schizophrenia research discussed in Chapter 5, U. Frith & Snowling’s (1983) homograph task is similar to the paradigm used by Naficy & Willerman (1980), in which subjects had to choose the most suitable meaning of a word given a sentence context. In addition, it is similar to the sentence completion studies of de Silva & Hemsley (1977) and Done & Frith (1984), in that in all cases, context had to be used to guide retrieval of appropriate information from LTM. In Chapters 3 and 5, it was argued that success on this type of task requires meta-
representation (specifically, representation of own knowledge), with the subject being simultaneously aware of the context whilst generating possible responses. Thus, a normal subject in the homograph task reading aloud a sentence such as, “in Lucy’s dress, there was a big tear” may produce the correct pronunciation of ‘tear’ because of metarepresentation such as ‘I KNOW “tear means a rip here”’. In effect, the subject has the common meaning and pronunciation of ‘tear’ (as moisture from the eyes) available to him, but is simultaneously able to represent ‘tear’ as a rip because of awareness of the sentence context. This is analogous to the cognitive process suggested by Leslie (1987; see Section 3.1 of this thesis) to be involved in pretense where, for example, the subject has the common meaning of ‘banana’ as a yellow fruit available to him, but is simultaneously (and without confusion) able to represent ‘banana’ as a telephone, because of awareness of the context (in which his mother is speaking into the fruit). By these arguments, therefore, the tendency of autistic subjects to score poorly on the homograph task (e.g. by giving the common, and incorrect, pronunciation of ‘tear’ in the above case), may reflect impaired metarepresentation.

It should be noted that Happé (1997) considered the possibility that the homograph task required metarepresentation, but rejected this as highly unlikely. She argued that “the task [is] an on-line task, which does not require meta-knowledge or the making of reflective judgments, abilities which may make demands on the metarepresentational system” (ibid., p. 4). However, regarding the ‘on-line’ nature of the task, we should note that everyday social interactions involving deception, jokes, irony etc., operate ‘on-line’ in real time, yet involve metarepresentation; thus, ‘on-line’ errors, such as the failure to immediately appreciate a joke can be taken as a sign of impaired mentalising. Moreover, in his definition of the Theory of Mind Mechanism, Leslie (1994a, p. 213) pointed out that theory of mind enables “time-pressured, on-line processing”, so clearly he views metarepresentation as occurring in real time. The notion of ‘on-line’ processing is important in explaining the task
performance of Happé’s (1997) autistic subjects. Thus, as discussed earlier, only a subset of those subjects showed theory of mind impairment on standard first- and second-order tasks, yet all of them scored poorly on the homograph task. At first sight, this is compatible with the notion of independent deficits in theory of mind and central coherence in autism (U. Frith & Happé, 1994), but when we contrast the task demands of the theory of mind and homograph paradigms, it is possible to explain all the findings in terms of impaired metarepresentation. The crucial point is that the homograph task places much greater demands on ‘on-line’ processing than standard first- and second-order theory of mind tasks, which give subjects more time to consider their responses. Thus, it is possible, for example, for theory of mind tasks to be solved using problem-solving strategies in the absence of metarepresentation (e.g. U. Frith et al., 1991), whereas the homograph task may be much more sensitive to metarepresentational impairments. By this argument, Happé’s (1997) findings are compatible with a degree of impaired metarepresentation in all of her autistic subjects, and the effective employment of either problem-solving strategies or rudimentary metarepresentational skills by a subset of subjects on the theory of mind tasks.

A test of this model would be provided by comparing autistic subjects’ performance on the homograph task with scores on more naturalistic theory of mind tasks (e.g. those developed by Happé, 1994b), which may be more demanding of ‘on-line’ metarepresentation than the standard first- and second-order tasks. Ideally, homograph performance of autistic people should be compared with ratings of their competence in real-life social interactions, as such ratings are likely to be very sensitive to on-line metarepresentational impairments. In line with results suggesting an intercorrelation of metarepresentational deficits in autism (e.g. Ozonoff et al., 1991a; Bennetto et al., 1996; see Section 3.2), one prediction of the above analysis would then be that scores on the homograph task should correlate with mentalising ability for autistic subjects. The use of executive function tasks (which within Frith’s
(1992) model require the representation of own goals), may also provide a test of the above account. Notably, Ozonoff et al. (1991a) found that autistic subjects were almost universally impaired on tests of executive function, whereas only a subset showed deficits on first- and second-order theory of mind tasks. This is intriguingly similar to the pattern of performance of autistics on Happé’s (1997) homograph and theory of mind tasks, and while, in Chapter 3, Ozonoff et al.’s results were rationalised in terms of the executive function tasks being more difficult than the theory of mind tasks, it may be that the executive tests had more discriminating power because they required more ‘on-line’ processing than the theory of mind tasks. A prediction from this analysis, therefore, would be that a group of autistic subjects would be almost universally impaired, relative to controls, on both the homograph task and an executive function task, and that scores in these two domains would be intercorrelated for the autistic group\textsuperscript{21}.

**Embedded Figures and Block Design tasks**

The ‘weak central coherence’ of autistic children has also been noted in their performance on perceptual tasks such as the Children’s Embedded Figures Test (CEFT; Witkin et al., 1971), which requires the child to find a ‘tent’ or ‘house’ shape (shown by a cardboard cutout) hidden within each of 25 meaningful pictures (e.g. a pram, a clock). Thus, Shah & Frith (1983) found that autistic children performed significantly better on this task than MA-matched normal or mentally retarded children, showing greater accuracy of performance and a tendency to spot the hidden

\textsuperscript{21} Happé (1997) pointed out that executive deficits in self-monitoring (giving rise to an observed paucity of self-correction in the autistic subjects in her study), could not explain her results, as autistics still performed more poorly than controls when only the first attempts were scored, without consideration of self-corrections. However, it should be noted that executive function may still be related theoretically to first attempt homograph errors, as an error on executive tasks such as the WCST is classified as a perseveration, even though it may occur on only the first attempt after a subject has been informed of a change in the sorting rule.
figure faster than the other groups. Shah and Frith suggested that autistic children performed so well on this task “because the overall meaning of the complex figure . . . was not very relevant or dominant for them” (ibid., p. 618); clearly this is consistent with less influence of context, and a weak drive for central coherence in the autistic individuals.

As pointed out by Happé (1997), the CEFT falls within the perceptual domain (so superior autistic performance suggests weak coherence at low levels), whereas other tests discussed earlier (e.g. those involving semantic relations; Tager-Flusberg, 1991) suggest weak coherence at higher, conceptual levels in autism. However, like the other context-dependent tasks, embedded figures tests are carried out in conscious awareness (with controlled processing as the subject actively searches for the hidden figure), so in that sense they may be amenable to analysis in terms of metarepresentational theory (see Frith, 1992, and Chapter 3 of this thesis). By analogy with earlier discussions of the cognitive basis of pretense (Leslie, 1987), it is possible to conceptualise the cardboard cutout (e.g. the ‘tent’) presented to subjects in the CEFT as a ‘primary representation’, whereas a normal subject’s simultaneous awareness of the tent as part of a larger context, may involve metarepresentation (e.g. ‘I KNOW “the tent is part of a clock”’). In this way, the subject represents the tent as itself, and simultaneously as part of a clock, without confusion. One consequence of such metarepresentation will be that normal subjects are continuously aware of the gestalt (i.e. the clock) while searching for the tent; this awareness may hinder their search, leading to errors and a long search time. If, on the other hand, we assume that, because of impaired metarepresentation, autistic individuals are not simultaneously aware of the gestalt while searching (so have in mind only the ‘primary representation’ of the cardboard cutout), then their search will be easier than that of normals, leading to the observed task superiority.

While this analysis is clearly speculative, it may enable us to explain why
good performance on embedded figures tests is a characteristic of most autistic people, as is poor performance on the homograph task. Thus, it was argued above that correct pronunciation of homographs in context requires ‘on-line’ metarepresentation, a skill in which most autistic people are deficient. By the same token, however, awareness of gestalt in tasks such as the CEFT is also likely to be an on-line process, so autistic people would be expected to be almost universally impaired at representing the gestalt while searching for hidden figures. Notably, this analysis still allows for Shah & Frith’s (1983) observation that autistic subjects were able to name the complex figures (e.g. “clock”) after searching was complete; the present model predicts that autistic awareness of the gestalt would only be reduced during the search, as only then does the task require representation of one of the component parts (thereby precluding simultaneous representation of the whole for autistics). Again this analysis is falsifiable as it makes specific predictions; for example, those autistic individuals who are particularly impaired on the homograph task or executive function tasks, should score particularly well on the CEFT. Similarly, the few autistic people who show some theory of mind ability in real-life situations (e.g. U. Frith et al., 1994) might be expected to perform worse on the CEFT than those with little or no real-life competence, as real-life ability may reflect a degree of on-line metarepresentation, a factor which would increase awareness of the gestalt during CEFT performance.

Similar arguments can be used to explain autistic subjects’ relatively good performance on the Block Design subtest of the Wechsler intelligence scales (and the characteristic ‘spiky’ IQ profile in autism). In a review of studies in this area, Happé (1994c) concluded that results consistently show an autistic performance peak on Block Design, and a trough on the Comprehension subtest of the verbal measures. In a detailed investigation of autistic Block Design performance, Shah & Frith (1993) found that autistic subjects performed better than normal and mildly retarded controls in a standard condition in which the gestalt to be copied was presented whole. In
contrast, when the to-be-copied design was pre-segmented, all groups performed similarly, reflecting the fact that controls showed a greater improvement than autistics in that condition. Shah and Frith concluded (in line with the weak central coherence account of autism), that autistic superiority on the standard version of the task reflected the relative ease with which autistic people can ‘mentally segment’ the to-be-copied design into its component parts.

If we accept that ‘mental segmentation’ of the provided gestalt is a key step in successful performance on the Block Design task, then the superiority of autistic people may be explained within the above metarepresentational account. Thus, segmentation of a diamond shape, for example, may involve breaking it down into four blocks each showing a triangle. The normal subject will ‘see’ triangles, but because of metarepresentation, will be simultaneously aware that the triangles are part of a larger context (e.g. ‘I KNOW “the triangle is part of a diamond”’); as a result, segmentation will be made more difficult because of ‘interference’ from this awareness of the gestalt. In contrast, having adopted the strategy of mentally segmenting the gestalt, autistic subjects will find it easier to ‘see’ the diamond as separate triangles because of a lack of simultaneous awareness of the gestalt (through impaired metarepresentation). As a result, they will find it easier than controls to construct the gestalt from its constituent blocks.

By analogy with earlier arguments, it is possible to explain experimental findings from Happé (1994c) within a metarepresentational account of the Block Design task. Happé found that autistic people showed good Block Design performance irrespective of their scores on standard first-order theory of mind tasks; in contrast, performance on the theory of mind tasks was a relatively good predictor of scores on the Wechsler Comprehension subtest (which U. Frith (1989) has argued may load heavily upon mentalising ability). These findings are similar to those discussed earlier, showing universally poor homograph task performance in autism,
irrespective of theory of mind task score (Happé, 1997), and as with those findings, it may be that the pattern of results is explicable in terms of task demands. Thus, the normal operation of metarepresentation in the Block Design task (as conceptualised above) is 'on-line', with the normal subject being continuously aware of the gestalt during initial segmentation; as a result, task performance is very sensitive to on-line metarepresentational impairments. In contrast, standard theory of mind tasks are less demanding of on-line processing, as they give subjects time to consider their responses, so can be solved by people with only rudimentary metarepresentational skills, or by those who adopt task-specific strategies in the absence of metarepresentation. By this argument, Happé’s (1994c) results are consistent with impairment of on-line metarepresentation in all of her autistic subjects, giving rise to good Block Design scores in all cases. The subset of subjects passing theory of mind and Wechsler Comprehension tests can then be assumed to have adopted rudimentary mentalising skills or task-specific problem-solving strategies. This analysis is again speculative, but it is empirically falsifiable in that it predicts negative correlations for autistic people between scores on the Block Design task and scores on other tasks thought to require on-line metarepresentation (e.g. tests of real-life social competence, executive function, or the ability to disambiguate homographs in context).

**Visual illusions**

Weak central coherence in autism has also been investigated in terms of the susceptibility of autistic individuals to particular visual illusions whose effect depends upon the integration of contextual cues. Happé (1996) presented six well known illusions (the Müller-Lyer, Hering, Kanisza triangle, Poggendorff, Ponzo and Titchener circles\(^{22}\) illusions) to autistic subjects and non-autistic control groups of young normals and children with moderate learning difficulties.

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\(^{22}\) The latter was used as part of the study in Chapter 7, and is shown in Appendix VIII.
In Happé’s study, subjects’ perception of the illusions was tested by asking them to make size judgments about relevant parts of the stimuli. Thus, in the Ponzo illusion, for example (see Figure 6.1), subjects were asked whether the two horizontal lines were the same length or not. As a control measure for language, motivation and general spatial ability, subjects were also shown the relevant parts of the stimuli without any inducing features (e.g. just the two horizontal lines in the Ponzo example), and were asked to judge their relative size. It was predicted that, because of weak central coherence, autistic subjects would be less susceptible than controls to the inducing effects of the contextual cues, so would make more accurate size judgments when presented with the illusions. Overall, this prediction was supported, and in addition, a ‘3-D’ condition (in which the to-be-judged parts of the stimuli were highlighted using brightly coloured plastic strips or shapes), significantly improved the accuracy of controls relative to the standard ‘2-D’ condition, but had little effect on the performance of autistics. This is clearly consistent with autistic superiority in the 2-D condition being due to their ability to ‘mentally segment’ the to-be-judged parts of the stimuli from their inducing contexts.

In Happé’s (1996) study, the Müller-Lyer figure (see Figure 6.2) produced different results from the other illusions, in that most of the autistic subjects (as well as controls) succumbed to the 2-D version. As Happé pointed out, this may reflect the fact that Müller-Lyer stimuli cannot easily be split into an ‘induced’ figure and
'inducing' context, as the arrow heads are connected to the to-be-judged lines, so form part of the same 'object'.

Figure 6.2. The Müller-Lyer illusion: the upper horizontal line appears longer than the lower one

As discussed by Happé (1996), the visual illusions used in her study may involve the integration of contextual information at very low levels, so by that analysis it may be difficult to explain her results in terms of the metarepresentational account discussed earlier. However, when we consider that the illusions task requires subjects to actively consider stimuli and make judgments about them (presumably using controlled processing in conscious awareness), it may be possible to extend the earlier discussion to explain these findings. The key point is that judgment of relative sizes of parts of the stimuli requires 'mental segmentation' of the whole (in the same way that Block Design requires segmentation of the provided gestalt). This segmentation involves the subject 'seeing' parts of the stimulus (e.g. the central circles in the Titchener illusion) as separate from the rest, and will be facilitated if the context (e.g. the 'surround' in the Titchener illusion) can be ignored. Now, by analogy with earlier discussions, normal subjects faced with these task demands will focus on the central circles, but because of metarepresentation may simultaneously be aware that the circles are part of a larger design (e.g. 'I KNOW "the circles are part of a pattern"'). This simultaneous awareness of the gestalt will make segmentation more difficult for them, increasing the chance of their perception being influenced by the inducing context, and making it more likely that they will experience the illusion. In contrast,
having chosen to focus on the central circles, autistic people will not simultaneously represent those circles as part of a larger pattern (because of impaired metarepresentation); as a result they will be less susceptible to the inducing effects of the 'surround', and more able to give accurate size estimations. Again, if this argument is correct, correlations should be observed for autistic people between accuracy of judgment on the illusions tasks, good performance on Block Design and the CEFT, and low scores on the homographs task and tests of executive function or real-life social competence.

Other results linking the social and non-social domains.

Findings from two other areas of research provide support for the hypothesis that, as well as explaining autistic failure in the social domain, impaired metarepresentation may underlie autistic performance on non-social tasks such as the CEFT and Block Design. The two areas are: 1) work on the ‘appearance-reality distinction’ in both normal children (e.g. Flavell et al., 1983) and autistic children (Baron-Cohen, 1989b), and 2) a large body of work on ‘field-dependence’ and ‘field-independence’ (e.g. Witkin et al., 1962). In the following section, these two areas will be briefly discussed.

The Appearance-Reality (A-R) Distinction

The ‘A-R distinction’ refers to the normal person’s ability to distinguish between real and apparent properties of objects, such as colour, size or identity. Flavell and his co-workers (e.g. Flavell et al., 1983) have investigated the phenomenon extensively in normal children, and for the case of object identity, for example, a typical version of their paradigm would be as follows: The child is presented with a sponge, painted and shaped so that it is indistinguishable from a rock. He is allowed to squeeze and examine the object to determine its true identity, and is then asked two questions: 1) What is the object really - is it really a rock or
really a sponge?; 2) When you look at it with your eyes, does it look like a rock or does it look like a sponge? Using many variations of this paradigm, including versions with pre-training and memory control questions, Flavell et al. (1983) found that normal 3-year olds performed poorly, tending to give the same answer (either “rock” or “sponge”) to both questions. In contrast, performance was markedly better for children aged 4 years or higher. Flavell et al. analysed the task in terms of a requirement for simultaneous representation of an object in two conflicting ways, and as discussed by Baron-Cohen (1989b), we can think of it as requiring ‘primary representation’ (e.g. ‘object = rock’) and metarepresentation (e.g. ‘I KNOW “object = sponge”’). Notably, this is very similar to the analyses earlier in this chapter of other ‘non-social’ tasks (e.g. the homographs task, the CEFT, Block Design and illusions tasks), where performance was presumed also to be influenced by the ability to simultaneously represent something (e.g. a word’s meaning, an object or a shape) in two conflicting ways. A metarepresentational analysis of the A-R distinction is supported by results from Gopnik & Astington (1988), who found that for normal children aged 3 to 5 years, scores on false belief and A-R tasks were intercorrelated. Furthermore, Baron-Cohen (1989b) found that autistic children scored significantly worse on a battery of A-R tasks than control groups of mentally handicapped and normal children. In addition, the A-R performance of the autistics in this study was related to their scores on a separate task requiring the ability to distinguish physical actions (e.g. ‘someone holding a biscuit’) from mental phenomena (e.g. ‘someone thinking about a biscuit’), a task which may have tapped mentalising ability. In line with the account presented here, it might be predicted that for autistic people, poor performance on the A-R task would correlate with low scores on the homograph task and tests of executive function and real-life social competence. Moreover, poor A-R scorers should be particularly good at the CEFT, Block Design and illusions tasks.

Field-dependence/independence

Research with normal people in the area of ‘field-dependence/independence’
(e.g. Witkin et al., 1962) has revealed relationships between various skills in the social and non-social domains and, although it is almost certainly over simplistic to rationalise all of these findings in terms of metarepresentation, a few of the results will be noted here, as they are consistent with the hypotheses proposed in this thesis. As reviewed by Witkin et al. (1971), many studies using an Embedded Figures Test (EFT) developed for adults, have revealed variation in normals in the ability to separate a perceptual field into discrete units. In Witkin et al.'s terminology, people who can easily segment the field are classified as ‘field-independent’, while those who score more poorly on the EFT are ‘field-dependent’. A number of workers have investigated the relationships between field-dependence/independence and specific aspects of behaviour and personality (see Witkin et al., 1971, for a review); thus, it has been found, for example, that field-dependent people show better memory for ‘social’ words than field-independent people, while the two groups do not differ in memory for ‘neutral’ words (Eagle et al., 1969). Similarly, field-dependents are particularly attentive to the faces of others, in that they look more at faces and are better at remembering them (e.g. Messick & Damarin, 1964). The latter finding is particularly interesting given that 1) attention to facial cues may reflect ‘on-line’ theory of mind ability, and 2) it was suggested above that on-line metarepresentation may hinder performance on embedded figures tests. In this context, it is notable that autistic people (whose particularly severe social deficits can be explained in terms of impaired metarepresentation) would be classified as extremely field-independent in Witkin et al.'s terminology on the basis of their scores on the CEFT. A final point relates to sex differences; Witkin et al. (1971) noted that males are generally more field-independent than females on the EFT, and as it is being suggested here that field-independence may reflect a relative reduction in ‘on-line’ metarepresentation, it is notable that, with age and verbal MA partialed out, Happé (1995) found female sex to be a significant predictor of greater theory of mind ability in a group of 70 normal 3- to 5-year old children.
In conclusion, while the concept of field-dependence/independence is complex, and may be only partly susceptible to analysis in terms of the cognitive processes discussed here, it is notable that research in this area suggests an inverse relationship, for normal people, between various measures of social awareness and the ability to disembed parts from wholes. This is consistent with the metarepresentational analysis of 'non-social' tasks presented above, and adds further weight to the argument that both the social and non-social abnormalities of autism may reflect impaired metarepresentation.

Summary

In this section, we have reviewed the case for 'weak central coherence' in autism, and it has been suggested that the empirical findings can be re-interpreted in terms of impaired metarepresentation in the disorder. Thus, by analogy with the schizophrenia research discussed in Chapter 5, poor autistic performance on U. Frith & Snowling’s (1983) homograph task may reflect a failure to simultaneously represent the contextual constraints of sentences while retrieving information (about word meaning and pronunciation) from long-term memory. Similarly, superior autistic performance on the CEFT, Block Design and illusions tasks, may reflect a failure to represent context while focusing on particular parts of the stimuli whose relevance is determined by the task demands (e.g. the central circles in adjacent Titchener stimuli, whose size is to be compared). It was argued that some support for this metarepresentational analysis is provided by the literature on the appearance-reality distinction and field-dependence, and it was suggested that the theoretical model presented here is empirically falsifiable as it predicts that autistic performance on a number of different tasks should intercorrelate.
6.2 The case of schizophrenia

Research reviewed in Chapters 3 and 5 suggested that, in schizophrenia, problems with the retrieval of context-appropriate information from long-term memory are particularly associated with the presence of behavioural signs (e.g. Done & Frith, 1984; Naficy & Willerman, 1980). In addition, there was some evidence from the study in Chapter 5 that behavioural signs (and to a lesser extent, delusions) are associated with context-inappropriate responses on the ‘picture pieces’ task. If it is true that the same cognitive deficits (e.g. metarepresentational impairments) can explain both these findings and the performance of autistic people on the homographs task and other tests of ‘central coherence’, then we might expect schizophrenic patients with behavioural signs or delusions to show an advantage similar to that of autistic subjects on tasks such as Block Design, illusions and embedded figures tests. In addition, schizophrenics with those signs and symptoms should be clinically similar to autistics in showing a focus on detail rather than gestalt. In the following sections, the schizophrenia research relevant to these predictions will be briefly reviewed.

Clinical features

Although the clinical presentation of schizophrenia is clearly different from that of autism (perhaps reflecting differences in the age of onset of the two disorders; see Frith & Frith, 1991), it is notable that a number of workers have identified clinical features of schizophrenia which suggest a focus on detail rather than the ‘whole picture’. Thus, in proposing problems in schizophrenia with “the maintenance of a major set”, Shakow (1950) suggested that schizophrenics “could not see the wood for the trees [and examined] each tree with meticulous care”. As discussed by Cutting (1985), distortions of perception are common in the early stages of a schizophrenic illness, and in an interview study of recently-diagnosed schizophrenic
patients, Chapman (1966, p. 229) reported one patient who said, “Everything I see is split up. It’s like a photograph that’s torn in bits and put together again”. Similarly, another of Chapman’s patients reported that, “I have to put things together in my head. If I look at my watch I see the watch, watchstrap, face, hands and so on, then I have got to put them together to get it into one piece” (p. 229). Visual hallucinations are relatively rare in schizophrenia (Cutting, 1985), but it is possible that these also sometimes reflect a relative focus on detail rather than gestalt. Thus, Cutting reported that schizophrenic visual hallucinations usually involve changes in only one aspect of the environment, and as an example, he discussed a patient who perceived a huge eye in an otherwise normal face; such experiences are generally found to co-occur with delusions (Cutting, 1985). Finally, the catatonic sign of ‘hypermetamorphosis’ sometimes observed in both schizophrenia and autism (e.g. Wing, 1996), may be a clinical manifestation of the tendency to concentrate on details. Patients showing this type of behaviour tend to be attracted by small objects which they examine extensively and, as discussed by Wing (1996), such behaviour may explain the observation that some schizophrenic patients in the community spend many hours examining the contents of rubbish bins for no apparent reason.

Performance on embedded figures tests

As discussed above, if impaired representation of own knowledge underlies autistic superiority relative to controls on the Children’s Embedded Figures Test (CEFT), then schizophrenic patients with behavioural signs (and to a lesser extent, delusions) should also perform well on such tasks. A number of studies have investigated schizophrenic performance on Witkin et al.’s (1971) adult version of the Embedded Figures Test (EFT), and although there has been no exploration of the relationship between EFT scores and symptoms, results have generally shown that schizophrenics perform poorly on the task. Thus, Magaro & Vojtisek (1971) found that male chronic patients took longer than male acute patients to find the hidden
figures on the EFT, and both patient groups scored significantly worse on the task than normal controls. Similar results were found by Vojtisek & Magaro (1974), who also showed that psychiatric controls performed significantly better than schizophrenics on the EFT. Finally, Grunebaum et al. (1978) investigated the performance of schizophrenic females on the task, and again found that they took significantly longer than normal females to find the hidden figures.

These results are contrary to predictions from the present work, but several methodological features of the above studies should be considered. Firstly, all of the existing experiments with schizophrenic patients have administered the EFT using Witkin et al.'s (1971) standard procedure. In this, subjects are shown the to-be-found simple figure for 10 seconds, this is then removed, the complex figure is presented, and the subject begins searching. This methodology clearly relies upon the subject's ability to remember the simple figure during searching (by, for example, maintaining it in working memory), but given the widespread schizophrenic impairments in working memory (e.g. Goldman-Rakic, 1987; see Chapter 2 of this thesis), it is perhaps not surprising that schizophrenic patients perform poorly on the EFT. Autistic children also have working memory deficits (e.g. Russell et al., 1996; see Chapter 3), and in this regard, it is notable that Shah & Frith (1983), in their study of autistic subjects, departed from Witkin et al.'s (1971) recommended administration procedure for the CEFT, by ensuring that the to-be-found simple figure was always present during searching. A prediction from this analysis, therefore, would be that schizophrenic patients should perform better than controls on the EFT if the simple figures are present during searching.

Another methodological issue relates to general cognitive impairment; thus, the findings of poorer EFT performance by chronic patients compared to acute patients, and poorer scores by both patient groups relative to controls (Magaro & Vojtisek, 1971; Vojtisek & Magaro, 1974) may simply have reflected lower spatial
IQs in the lower-scoring groups (as IQ was not controlled in either of those studies). In addition, Vojtisek & Magaro (1974) suggested that after failing to find two or more hidden figures, their patients often lost interest in the task. This poor motivation may have compounded the poor scores of their schizophrenics, and Vojtisek and Magaro suggested that a way of minimising this effect would be to present the easiest stimuli first, thereby encouraging patients to continue because of early success. In the final empirical study of this thesis (to be discussed in Chapter 7), the performance of schizophrenic patients on a new embedded figures task was investigated. In line with the above discussion, the simple figure was always present during searching, any confounding effects of spatial IQ differences were removed, and stimuli were presented in order of increasing difficulty. As we shall see, the results were markedly different from those of earlier workers.

Performance on Block Design tasks

By analogy with the above discussion of embedded figures tests, impaired representation of own knowledge in schizophrenics with behavioural signs or delusions might be expected to lead to relatively good performance on the Block Design subtest of the Wechsler Adult Intelligence Scales (e.g. WAIS-R; Wechsler, 1981). One problem with investigation of this prediction is that, as well as involving ‘mental segmentation’ of a gestalt, Block Design requires subjects to carry out mental rotations and manipulate obliques (i.e. lines at a 45° angle relative to the frame of the whole design) (Shah & Frith, 1993). Shapiro (1952, cited in Shah & Frith, 1993) has shown that schizophrenic patients score poorly on tests involving oblique lines or rotations, so any advantage that schizophrenics have in mental segmentation on the Block Design task may be offset by their poor performance on the other task components. In addition, Block Design is quite a difficult task, which requires the subject to be motivated and attentive throughout; as the behavioural signs of schizophrenia are often associated with a lack of motivation, any advantage which these
patients have in segmentation may be cancelled out by this factor.

With these points in mind, it is of note that several studies have found schizophrenic scores on the Wechsler scales to show a relative peak at the Block Design subtest. For example, Robertson & Taylor (1985) found that although their sample of 61 schizophrenic males scored more poorly than 41 normal controls on all subtests of the WAIS (except the Vocabulary subtest), schizophrenic performance was relatively spared on the Block Design and Similarities subtests, with the normals and schizophrenics not differing significantly. Interestingly, when patients were divided into symptom subgroups, the most clear evidence of a relative Block Design peak was found in the ‘deluded’ group (patients with delusions or hallucinations, but no behavioural signs); in line with the above discussion, these may be people whose relative advantage in ‘mental segmentation’ was not offset by amotivation or in­attention. In a similar study, Green & Walker (1985) gave a large battery of neuropsychological tasks to a heterogeneous group of schizophrenics, and found that the patients scored significantly worse than controls on all tests except Block Design and a test of motor control, (for which there were only trends towards poorer patient performance). Thus, while relatively weak, these data certainly suggest that schizophrenic patients may find Block Design tasks less difficult than many other standard neuropsychological tests.

Visual illusions

By analogy with the work of Happé (1996) with autistic people, several studies have investigated the susceptibility of schizophrenic patients to visual illusions. Unfortunately for the present discussion, much of the schizophrenia research has used the Müller-Lyer illusion (see Figure 6.2), a figure which cannot easily be split into ‘induced’ and ‘inducing’ parts, and which may therefore be of little use for the investigation of mental segmentation effects (Happé, 1996). As
discussed in Section 6.1, Happé found that despite being less susceptible to a number of other illusions, autistics were as likely as controls to succumb to the ‘2-D’ Müller-Lyer illusion; furthermore, more autistics than controls experienced the illusion in the ‘3-D’ condition of her study. The relevant studies in the schizophrenia literature report similar effects with the Müller-Lyer figure; thus, Orme et al. (1968) found that chronic schizophrenics (whose symptoms were not specified) showed a trend to be more susceptible to the Müller-Lyer illusion than depressed controls, and other workers found that this effect reached statistical significance (e.g. Capuzzoli & Marsh, 1994, with acute paranoid patients; Broota & Kaur, 1987; Mrinal, 1980; and Weckowicz & Witney, 1960, all with heterogeneous groups of schizophrenics). While these findings are generally compatible with Happé’s (1996) autistic results in showing schizophrenic susceptibility to the Müller-Lyer illusion, it should be noted that all of the above studies with schizophrenic patients failed to use a control task. As illusion susceptibility in these studies was shown by greater error in judging the actual length of the horizontal lines in the Müller-Lyer figures, it therefore remains possible that the results with schizophrenic subjects simply reflected nonspecific features of the illness such as poor motivation or inattention.

Such problems make it theoretically more interesting to investigate the illusions to which schizophrenics fail to succumb - i.e those where they make more accurate size judgments than controls. Happé (1996) found that autistic people were less likely than controls to succumb to the Ponzo, Kanisza triangle, Titchener, Hering and Poggendorff illusions; these stimuli can all be separated into an ‘inducing’ and ‘induced’ component, and Happé interpreted her findings in terms of weak central coherence in autism, with autistics being better than controls at ‘mentally segmenting’ the stimuli. From earlier discussions suggesting that a common cognitive deficit (impaired metarepresentation) underlies both these findings and the failure of schizophrenics with behavioural signs or delusions to use context, we might expect symptomatic schizophrenics to similarly show better size judgments than controls.
when presented with such stimuli. Little research has so far been done in this area, but notably Capozzoli & Marsh (1994) found that the same patients who were more likely than normal controls to experience the Müller-Lyer illusion, were significantly less susceptible than normals to a ‘perspective drawing’ illusion. Although this figure was not described or illustrated in their paper, it was probably similar to the Ponzo illusion (see Figure 6.1), where perspective cues from the two inwardly tilting lines lead the normal subject to think that the upper horizontal line is longer than the lower one. In this regard, therefore, Capozzoli and Marsh’s findings support the prediction that schizophrenics would be less susceptible than controls to the Ponzo illusion.

The only other study of relevance (Letourneau & Lavoie, 1973) found that schizophrenic patients were more susceptible than normal controls to the Poggendorff illusion (unlike Happé’s autistics, who were less susceptible than controls). However, the control subjects used by Letourneau and Lavoie had full-scale IQs significantly higher than those of the patients, so the finding that patients made greater errors in judging the Poggendorff stimuli may simply have been an artefact of this intellectual difference. In the study to be discussed in Chapter 7 of this thesis, the susceptibility of schizophrenic patients to the Titchener illusion (see Appendix VIII) was investigated. Any confounding effects of spatial IQ differences were explored, and it was predicted that schizophrenics with behavioural signs or delusions would be less likely than controls to succumb to the illusion.

Other relevant empirical findings

In the final part of this chapter, it will be suggested that results from several other areas of research in schizophrenia can be rationalised in terms of patients failing to be aware of context (through impaired representation of their own knowledge). This account is clearly speculative, but it has the advantage of unifying several rather disparate strands of psychological research.
Abnormal reasoning styles in schizophrenia

As reviewed in Chapter 2 (see Section 2.2), several studies have found that deluded patients show abnormal reasoning styles on probabilistic inference tasks involving affectively neutral stimuli. Huq et al. (1988) and Garety et al. (1991) used a paradigm in which subjects were shown two jars containing different proportions of coloured beads. Subjects were told the relevant proportions, the jars were hidden from sight, and the experimenter then selected a series of beads from one of the jars, asking the subject to guess from which jar the beads were being selected, and to rate his confidence in his judgments. Results showed that deluded subjects requested less draws than controls before reaching a decision, were relatively over-confident about their judgments, and more readily changed their estimates when given potentially disconfirmatory evidence. As discussed in Section 2.2, these results can be interpreted in terms of problems with the integration of sequentially presented information in conscious, controlled processing, and indeed Bentall & Young (1996) suggested that the main reason patients performed poorly on such tasks was that they were impaired in the integration of information over time.

In terms of the discussion in the present chapter, normal performance on the probabilistic inference task can be thought of as involving awareness of the importance of previous draws in making an informed judgment. These previous draws can be thought of as an informational ‘context’ which, when combined with information from the current draw, enables the subject to give his ‘best guess’ of the jar from which the beads are being drawn. As an example, the subject may have been told that the two jars contain 85% blue beads (jar A) and 15% blue beads (jar B) respectively, with the remainder of the beads being pink. A normal subject presented with three consecutive blue beads and then a pink bead is likely to decide that beads are being drawn from jar A, despite the current bead being pink, because he is aware of the ‘context’ of the previous draws. In terms of metarepresentational theory (e.g. Leslie, 1987), the subject may have a ‘primary representation’ (e.g. ‘pink is import-
ant’) based on his current percept, but simultaneously forms a metarepresentation (e.g. ‘I KNOW “pink is unimportant” (because all the other draws have been blue’)). Subjects with impaired metarepresentation will be likely to have in mind only the primary representation (e.g. ‘pink is important’ in the above case), so will be more likely than controls to base their decisions upon the current, rather than previous, draws. This may explain the tendency of deluded patients to request less draws than controls, to be over-confident in their judgments, and to be more likely to change their decisions based solely on the most recent draw. This analysis suggests that a similar response pattern would be observed for schizophrenics with behavioural signs, as well as for those with delusions, as previous research (see the study in Chapter 5, and work by de Silva & Hemsley, 1977; Done & Frith, 1984; Naficy & Willerman, 1980) has suggested that both groups show a relative lack of awareness of contextual information.

*Studies using the Continuous Performance Test (CPT)*

Similar arguments can be used to explain the poor performance of schizophrenic patients with positive symptoms on the ‘memory load’ version of the CPT (e.g. Nuechterlein et al., 1994; see Section 2.2 of this thesis). In this variant of the task, subjects must implement a conditional rule, responding to, say, ‘7’ only if preceded by ‘3’. The preceding digit can be thought of as providing a ‘context’ which determines the subsequent response, so in terms of metarepresentational theory (e.g. Leslie, 1987), good performance (e.g. a non-response when ‘7’ is preceded by ‘4’) may involve both ‘primary representation’ (e.g. ‘7 is important’) and metarepresentation (e.g. ‘I KNOW “7 is unimportant” (because 4 has just been presented’)). Subjects with impaired representation of their own knowledge (e.g. those with positive symptoms such as delusions) will be likely to have in mind only the primary representation during task performance (e.g. ‘7 is important’ in this case), which will increase their chance of responding to ‘7’ irrespective of the preceding figure. Notably, Cohen & Servan-Schreiber (1992) also analysed the ‘memory load’ version
of the CPT in terms of a requirement for awareness of context, and in a neural network simulation, they suggested that deficits in the use of context were a major contributory factor to schizophrenic cognitive impairment. One prediction of the present model is that poor performance on the ‘memory load’ CPT should be related to the presence of either delusions or behavioural signs (as both are presumed to be associated with problems in the representation of context). Research to date has suggested a relation primarily with positive symptoms (e.g. Nuechterlein et al., 1994; Servan-Schreiber et al., 1996), but it should be noted that no CPT studies have yet employed symptom subgroups of patients formed according to Frith’s (1992) model. The present analysis would predict that patients with either behavioural signs or paranoid symptoms, grouped in that way, would be impaired on the task.

*Studies involving the sorting of schematic and abstract stimuli*

Studies by Griffith et al. (1980) and Frith et al. (1983) gave a sorting task to groups of schizophrenic patients and psychiatric controls. In Griffith et al.’s study, subjects had to sort two sets of stimuli, one consisting of 30 schematic faces and the other comprising 30 abstract histoforms; in both sets, the stimuli varied in terms of 9 different features, and subjects were simply asked to sort the stimuli into as many subgroups as they wished, grouping stimuli which they thought had similar characteristics. As discussed by Griffith et al., the quality of sorting was assessed by measuring the extent to which the groups produced by each subject differed on each of the 9 features. Results showed that controls sorted faces better than the abstract shapes, whereas schizophrenic patients performed similarly with both sets of stimuli, and Griffith et al. suggested that this may be because the gestalt properties of the faces aided controls (but not schizophrenics) by enabling them to extract more of the component features simultaneously. With similar sets of stimuli, Frith et al. (1983) replicated Griffith et al.’s results, showing in addition that for controls, a distracting feature greatly impaired sorting performance with faces but not with histoforms; for the schizophrenics, the distractor had similar effects on performance for both types
of stimuli. Frith et al. suggested that this reflected the fact that the gestalt quality of the faces made it more difficult for controls to ignore the distractor, and in line with Griffith et al., they concluded that gestalt properties were used by controls, but not schizophrenics, during sorting.

In showing a relative lack of use of contextual information (gestalt properties) in schizophrenia, both of these studies may be amenable to analysis in terms of impaired metarepresentation. Thus, in sorting either faces or histoforms, we can think of control subjects as concentrating on the component features of the stimuli (e.g. various lines of different length), but being simultaneously aware that the features are part of a larger context (e.g. 'I KNOW “these lines are part of a pattern”'). In the case of the faces (but not for the histoforms), this ‘pattern’ has strong gestalt properties, so control subjects’ awareness of it will mean that these properties can influence performance, leading to the observed superiority of controls in the face condition relative to the abstract condition. In contrast, we might hypothesise that impaired metarepresentation in the schizophrenic patients means that, having focused on the component features of the faces or histoforms, they are unable to simultaneously represent those features as being something else (i.e. part of a larger pattern), so as a result fail to benefit from the gestalt properties of the faces during sorting. In effect, in sorting both faces and abstract shapes, the patients have available to them only ‘primary representations’ (i.e. details of the component features), so use the same sorting strategy for both types of stimuli. This analysis would predict sorting abnormalities in schizophrenia to be particularly associated with the presence of behavioural signs or delusions, as earlier discussions have suggested that such features are related to problems with the representation of context. This prediction has not been directly investigated, but it is notable that all of Frith et al.’s (1983) patients had some positive symptoms, and Griffith et al. (1980) found that schizophrenic patients both with, and without, formal thought disorder, performed poorly at face sorting.
Studies of 'perceptual organisation' in schizophrenia

As reviewed in Chapter 2 (Section 2.2), Schwartz Place & Gilmore (1980, Experiment 2) tachistoscopically presented, to chronic schizophrenic patients and psychiatric controls, line displays varying in the degree of gestalt grouping of their elements. An example of a high gestalt condition was the 'homogeneous' display, where all lines were of the same orientation, and a lower gestalt condition was the 'heterogeneous' display, where lines were at two different orientations. Subjects simply had to report the total number of lines present, and results showed that controls performed more poorly as gestalt organisation decreased, whereas schizophrenics were unaffected by this manipulation, scoring better than controls overall. Schwartz Place and Gilmore concluded that controls were influenced by the gestalt properties of the stimuli, so grouped lines of the same orientation before starting to count. When both orientations were present this would lead to slower counting, resulting in poorer performance relative to the schizophrenics, who were presumed to treat all the displays equally, irrespective of their gestalt properties.

As we saw in Chapter 2, Hemsley (1988) argued that this study supported his model of schizophrenia, which posits a weakening of the automatic influence of stored information on current sensory processing. However, it was argued throughout Chapter 2 that much evidence suggests that automatic processing is intact in schizophrenia, and that deficits only appear when conscious, controlled processing is required. In this regard, it is notable that a number of recent studies (briefly reviewed by Silverstein et al., 1996) have implied that perceptual organisation, as assessed by tasks such as that used by Schwartz Place & Gilmore (1980), may be partly guided in normals by attentional strategies or contextual information stored in working memory. This suggests that Schwartz Place and Gilmore's data may be compatible with controlled processing deficits in schizophrenia, and indeed Silverstein et al. (1996, Study 2) found that a task manipulation thought to aid (controlled) contextual processing removed the perceptual organisation deficit in a group of schizophrenic
patients. From these arguments, it may be possible to extend metarepresentational theory to explain the abnormal performance of schizophrenic subjects on tests of perceptual organisation. Thus, by analogy with the sorting studies discussed above, normal or psychiatric controls presented with Schwartz Place & Gilmore’s (1980, Experiment 2) displays, will focus on the component lines in order to count them, but because of intact metarepresentation, may be simultaneously aware that these lines are part of a larger context (e.g. ‘I KNOW "these lines are part of a pattern" ’). As the patterns have gestalt properties, awareness of the context will increase the chance of these properties influencing task performance, leading to the observed lower scores with ‘heterogeneous’ than ‘homogeneous’ displays. In contrast, having focused on the component lines for counting, schizophrenic patients presented with the same displays may fail to simultaneously represent the lines as part of a larger pattern (because of impaired metarepresentation), so will be uninfluenced by the gestalt properties of the stimuli, showing better task performance than controls as a result. In Leslie’s (1987) terminology, we can think of the patients as having only ‘primary representations’ available to them (i.e. the perceptual details of the component lines), without simultaneous metarepresentation.

This analysis would predict that superior performance by schizophrenic patients on tests of perceptual organisation would be associated with the presence of behavioural signs (and to a lesser extent, delusions), as earlier discussions suggest that these features may be particularly related to impairments in the representation of context. This has not been investigated directly, but Silverstein et al. (1996) did find the effect to be most pronounced in inpatients with poor pre-morbid social function, who may be assumed to have shown some negative signs. A final point relates to the influence on patients’ performance of factors designed to increase the salience of the contextual information in this type of paradigm. As mentioned above, Silverstein et al. (1996) found that when a task manipulation was used which facilitated contextual processing, the performance of schizophrenic patients was
normalised. In terms of the account given above, this suggests that patients can represent context to some extent, so that any deficit may be relative rather than absolute. This is compatible with the results of the study in Chapter 5 of this thesis, where it was found that all subject groups showed some advantage of context, but that the advantage was smaller in the schizophrenics with behavioural signs. These findings are also consistent with results from Rief (1991), who used a similar paradigm to that of Schwartz Place & Gilmore (1980, Experiment 2) and found that, when the gestalt properties of the stimuli were made particularly strong, schizophrenic patients did use those properties during line counting. Rief concluded that perceptual organisation abnormalities in schizophrenia are only apparent when contextual effects are relatively weak.

6.3 Conclusions

The first section of this chapter reviewed the empirical findings relevant to the concept of 'weak central coherence' in autism, and it was suggested that this phenomenon may be a manifestation of impaired metarepresentation. By analogy with arguments in Chapter 5 suggesting a metarepresentational explanation for schizophrenic impairments in the use of context to guide retrieval from LTM, it was proposed that poor autistic performance on the 'homograph task' (e.g. U. Frith & Snowling, 1983) may similarly reflect a lack of awareness of context. The argument was extended to explain superior autistic performance on embedded figures, Block Design and illusions tasks, in terms of a failure to simultaneously represent context while focusing on aspects of the stimuli relevant to the task in hand. It was suggested that evidence in favour of this application of metarepresentational theory to 'non-social' tasks, is provided by the literature on the appearance-reality distinction and field-dependence.

The second part of the chapter focused on schizophrenia, and it was argued
that if the same cognitive deficit (e.g. impaired metarepresentation) underlies both weak central coherence in autism, and the failure of schizophrenic people with behavioural signs or delusions to use context, then these symptomatic schizophrenics should perform well on embedded figures tests, Block Design, and illusions tasks. The relevant research was reviewed, and it was suggested that results from studies using Block Design and illusions paradigms are encouraging. Studies using embedded figures tests have shown generally poor schizophrenic performance, but this may reflect methodological issues specific to Witkin et al.’s (1971) administration procedure for the EFT. It was finally argued that impaired metarepresentation in schizophrenia may also underlie the abnormal reasoning styles of deluded patients, the poor performance of schizophrenics on the ‘memory load’ CPT, and the failure of patients to use gestalt on sorting tasks and tests of ‘perceptual organisation’. If correct, this model of impaired metarepresentation provides a useful way of synthesising a large body of research in hitherto disparate areas.
CHAPTER SEVEN

EMBEDDED FIGURES AND ILLUSIONS IN SCHIZOPHRENIA

7.1 Introduction

Awareness of context in autism and schizophrenia

It was pointed out in Chapter 6 that autistic children and some schizophrenic patients show similarly poor performance on tests in which context must be used to guide the retrieval of information from long-term memory. For example, autistic people score poorly on the ‘homograph task’, where the correct pronunciation of words is determined by sentence context (U. Frith & Snowling, 1983; Happé, 1997), and this has been taken as evidence for ‘weak central coherence’ in autism (e.g. U. Frith & Happé, 1994). Similarly, schizophrenic patients with behavioural signs (and possibly delusions) perform poorly on tasks in which context-appropriate words must be produced or selected (e.g. de Silva & Hemsley, 1977; Done & Frith, 1984; Naficy & Willerman, 1980; the ‘picture pieces’ study in Chapter 5 of this thesis). In Chapter 6, it was suggested that a common cognitive deficit underlies all of these findings, and a specific candidate for that deficit (impaired metarepresentation) was proposed. Now, regardless of the accuracy of the metarepresentational model, a ‘common deficit’ account would predict that schizophrenic patients with behavioural signs or delusions should perform similarly to autistic people on other tests of ‘central
coherence’ such as embedded figures tasks (e.g. Shah & Frith, 1983) or Happé’s (1996) illusions task (see Chapter 6). In the first part of the present study, that prediction is investigated. Regarding the accuracy of the metarepresentational model, relevant data can be obtained by examining correlations between schizophrenic subjects’ scores on tests of central coherence and their performance on other tasks thought to require metarepresentation (e.g. the ‘hints task’; Corcoran et al., 1995). These correlations are explored in the second part of the present study.

Embedded figures in autism and schizophrenia

As reviewed in Chapter 6, Shah & Frith (1983) showed that autistic children performed very well on the Children’s Embedded Figures Test (CEFT; Witkin et al., 1971), finding significantly more of the hidden objects than controls, and tending also to find the objects faster. Investigations of schizophrenic performance on the adult Embedded Figures Test (EFT; Witkin et al., 1971) have generally revealed lower scores than controls (Grunebaum et al., 1978; Magaro & Vojtisek, 1971; Vojtisek & Magaro, 1974), but it was argued in Chapter 6 that these studies may have been confounded by methodological problems. For example, all of the studies with schizophrenic patients administered the EFT using Witkin et al.’s standard procedure, in which the card showing the to-be-found simple figure is removed while the subject searches the complex figure. This relies upon the subject’s ability to maintain a representation of the simple figure in working memory during searching, so impaired working memory (e.g. Goldman-Rakic, 1987; Park & Holzman, 1992) may be one reason for the poor EFT performance of schizophrenic patients. In their study of autistic performance on the CEFT, Shah & Frith (1983) overcame this problem by ensuring that the to-be-found simple figure was always present during searching, and in fact subjects were allowed to use the figure to aid their search. In the study to be discussed in the present chapter, a similar procedure was therefore adopted with schizophrenic patients.
A second problem with existing EFT studies in schizophrenia is a lack of control for spatial IQ, so that poor scores by patients relative to controls may have simply reflected general cognitive impairment in the patients. In their study of autistic children, Shah & Frith (1983) matched all groups on spatial IQ as measured by Raven's Coloured Progressive Matrices (Raven, 1962), and in the present study, post hoc statistical analyses were used to investigate any effects of spatial IQ on subjects' performance. Vojtisek & Magaro (1974) noted that their schizophrenic patients often lost interest in the EFT after failing two or more items, so these workers recommended that items be given in order of increasing difficulty, so that early success on the easier items may encourage patients to continue with the task; this procedure was adopted in the present study. With these methodological modifications, it was predicted that, because of reduced awareness of context, schizophrenic patients with behavioural signs (and to a lesser extent, those with delusions), would score better than normal and psychiatric controls on an embedded figures test. Schizophrenics in remission were predicted to score similarly to controls because of intact use of context. These predictions are rather bold because, as we have seen throughout this thesis, schizophrenic patients (especially those with behavioural features) generally perform badly on neuropsychological tasks. However, the predictions follow directly from the work of Shah & Frith (1983), where autistics were shown to perform better than controls on the CEFT. A new embedded figures task was devised for the present study (see Appendix VII), because pilot work with normal controls showed that a number of Witkin et al.'s (1971) standard EFT stimuli could be solved very quickly (i.e. within 10 seconds) when the simple figure was present during searching. As it was predicted that some patients would perform faster than controls, more difficult stimuli were therefore developed to avoid the possibility of ceiling effects in the data.

Visual illusions in autism and schizophrenia

As we saw in Chapter 6, Happé (1996) found that autistic children were less
susceptible than controls to particular visual illusions whose effect depends upon the integration of contextual cues (e.g. the Poggendorff, Ponzo and Titchener illusions), and Happé interpreted this in terms of weak central coherence in autism. Very few comparable studies have been carried out with schizophrenic patients, and as reviewed in Chapter 6, most of the studies that have been published may be confounded by methodological problems. Thus, Letourneau & Lavoie (1973) found that schizophrenics were more susceptible than normal controls to the Poggendorff illusion, but the greater judgment errors made by the patients in that study may have been an artefact of their lower IQs relative to controls. In a notable recent study, however, Capozzoli & Marsh (1994) matched patients and normal controls on age, sex and educational background, and found that their group of acute paranoid schizophrenics was significantly less susceptible than controls to a ‘perspective drawing’ illusion. In as much as this figure may have been similar to the Ponzo illusion (see Figure 6.1), these findings are similar to those of Happé (1996) showing that autistics were less susceptible than controls to the Ponzo illusion.

In the present study, the susceptibility of schizophrenic patients and controls to the Titchener illusion (see Appendix VIII) was investigated. On the basis of earlier arguments suggesting an impaired awareness of context in schizophrenia, it was predicted that patients with behavioural signs (and to a lesser extent, delusions) would be less susceptible to the illusion than controls, providing more accurate judgments of the size of the central circles in the illusion stimuli. Schizophrenic patients in remission were predicted to score similarly to controls because of their presumed intact use of context. Post hoc statistical analyses were used to rule out any confounding effects of low spatial IQ on performance of the schizophrenic patients. In addition, an attempt was made to overcome problems of inattention or poor motivation by the use of a control condition (e.g. Happé, 1996), in which subjects judged the size of circles in the absence of any ‘inducing’ context.
Relation to metarepresentational theory

As discussed earlier, if the above predictions about schizophrenic performance on embedded figures and illusions tasks are confirmed, we shall have further evidence that a common cognitive deficit underlies the failure of autistic or schizophrenic individuals to use context. Evidence that this deficit is one of impaired metarepresentation can, however, only be obtained by correlating subjects' scores on the embedded figures or illusions tasks with scores on other tasks known to require metarepresentational ability. Now, the subjects tested in the present study were the same as those detailed in Chapter 5, so a number of them also gave data on the 'hints task' (Corcoran et al., 1995), which is an accepted test of theory of mind ability (probably at the second-order level; see the Discussion in Chapter 4). The present study therefore examined the relationship between scores on the hints task and scores on the embedded figures and illusions tasks, for each subject group in turn. The hints task tests the ability to represent others' mental states, and it was suggested in Chapter 6 that embedded figures and illusions tasks normally involve representation of one's own knowledge, with the subject being consciously aware of context. Thus, on the basis of the findings in Chapter 4 that executive function (thought to involve the representation of own goals; Frith, 1992) and theory of mind are uncorrelated in patients with schizophrenia (and the subsequent explanation of that in terms of late-occurring, independent impairments in a fully-developed metarepresentational system), we might expect an absence of correlation for all schizophrenic groups between performance on the hints task and performance on embedded figures or illusions tasks. In addition, we might similarly expect an absence of correlation for the control groups in the present study, given Ozonoff et al.'s (1991a; see Chapter 3) finding of no correlation between theory of mind and executive function in clinical controls.23

23 The prediction for controls is complicated by the results of studies on field-dependence (see Chapter 6), which suggest that increased social awareness may be
The above predictions are relatively weak, in that they involve the absence of a significant result rather than a predicted relationship, but the finding of no correlations between hints score and performance on embedded figures or illusions tasks, would at least be consistent with the theoretical framework developed here. Notably, consideration of scores on the 'picture pieces' task (which, like the hints task, was performed by a number of the subjects from the present study), enables us to predict a significant correlation. In Chapter 5, it was suggested that the 'isolation' condition of the picture pieces task requires representation of own goals, so that subjects are able to generate or inhibit responses. The 'context' condition of the task, however, may involve both representation of own goals and own knowledge, so that subjects generate or inhibit responses whilst being aware of the contextual constraints of the picture scene. Now, from the arguments in Chapter 6 that the normal operation of metarepresentation in embedded figures or illusions tasks involves the representation of own knowledge, we might expect poor performance on the context condition (but not the isolation condition) of the picture pieces task to correlate with good performance on the embedded figures or illusions tasks. Given the expected superior performance of schizophrenics with behavioural signs and delusions in the present study, and their poor scores in the context condition of the picture pieces task, the correlation may only reach significance for those two groups. While still not proving the metarepresentational account proposed here, the finding of significant correlations would add weight to the argument developed in Chapter 6, that similar cognitive processes underlie performance on tasks involving the retrieval of context-appropriate information from memory (e.g. the picture pieces task), and tasks traditionally thought to operate at a more perceptual level, such as embedded figures tests.

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associated with poorer performance on embedded figures tests. This will be explored further in the Discussion.
7.2 Methods

Subjects

Forty people with a DSM-IV diagnosis of schizophrenia (APA, 1994) were tested as part of this study and that discussed in Chapter 5. Only 37 of these patients provided data on either or both of the neuropsychological tasks in the present study, so the following details only refer to those 37 patients. All subjects met the general inclusion criteria of competence in the English language, an age in the range 16 to 65, no history of leucotomy, neurological disability or drug/alcohol abuse, and a current spatial IQ on Raven’s Advanced Progressive Matrices, Set I (Raven, 1958) of 70 or higher. About three-quarters of the schizophrenic subjects were inpatients at one of three psychiatric hospitals in London, whilst the rest lived in the community and attended psychiatric outpatient clinics. Demographic details of the patients are given in Table 7.1. All but one of the schizophrenics were taking neuroleptic medication at the time of testing. The daily dose of neuroleptic was converted to chlorpromazine equivalents using the tables provided by Foster (1989). The patients receiving anticholinergic medication (32% of the sample), were all taking 15 mg/day or less of procyclidine (mean daily dose was 11.4 mg, range 5 - 15 mg). The clinical details of the schizophrenic patients are given in Table 7.2.

As with previous studies in this thesis, the schizophrenic subjects were allocated to one of four groups according to their signs and symptoms as revealed by a Present State Examination (Wing et al., 1974) on the day of testing. The groups were formed in line with predictions from Frith’s (1992) model about the symptom-specific performance of patients on theory of mind tasks; they were the same groups as used in previous investigations of Frith’s model (Corcoran et al., 1995; 1997; Corcoran & Frith, 1996; Frith & Corcoran, 1996). Patients were allocated to groups hierarchically, so that those with symptoms from more than one group were placed
<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age</th>
<th>Ethnicity</th>
<th>Hand</th>
<th>Years of Education</th>
<th>Percent Employed</th>
<th>Current Spatial IQ (Raven's)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics</td>
<td>32:5</td>
<td>36.0 (9.8) [19 - 61]</td>
<td>26:11</td>
<td>32:4 [1 missing value]</td>
<td>11.3 (1.5) [9 - 17]</td>
<td>0.0</td>
<td>89.5 (12.8) [75 - 120]</td>
</tr>
<tr>
<td>Behavioural Signs</td>
<td>7:2</td>
<td>34.6 (10.9) [23 - 58]</td>
<td>8:1</td>
<td>7:2</td>
<td>11.7 (1.3) [10 - 14]</td>
<td>0.0</td>
<td>91.7 (12.2) [80 - 115]</td>
</tr>
<tr>
<td>Paranoid Symptoms</td>
<td>15:3</td>
<td>36.3 (10.0) [19 - 61]</td>
<td>12:6</td>
<td>17:1</td>
<td>11.2 (1.7) [9 - 17]</td>
<td>0.0</td>
<td>87.2 (10.6) [75 - 105]</td>
</tr>
<tr>
<td>Passivity Phenomena</td>
<td>1:0</td>
<td>34.0</td>
<td>0:1</td>
<td>1:0</td>
<td>12.0</td>
<td>0.0</td>
<td>80.0</td>
</tr>
<tr>
<td>Patients in Remission</td>
<td>9:0</td>
<td>36.9 (10.0) [25 - 51]</td>
<td>6:3</td>
<td>7:1 [1 missing]</td>
<td>11.0 (1.3) [9 - 13]</td>
<td>0.0</td>
<td>93.0 (17.5) [75 - 120]</td>
</tr>
<tr>
<td>Psychiatric Controls</td>
<td>7:10</td>
<td>45.5 (11.3) [24 - 62]</td>
<td>17:0</td>
<td>13:4</td>
<td>11.5 (2.6) [6 - 16]</td>
<td>35.3</td>
<td>103.8 (17.2) [80 - 130]</td>
</tr>
<tr>
<td>Normal Controls</td>
<td>15:15</td>
<td>41.2 (14.6) [22 - 64]</td>
<td>26:4</td>
<td>25:5</td>
<td>12.4 (2.5) [10 - 19]</td>
<td>93.3</td>
<td>110.0 (13.1) [80 - 130]</td>
</tr>
</tbody>
</table>

S = standard deviation
Table 7.2. Clinical details of the schizophrenic patients

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Age at first Psychotic Episode</th>
<th>Duration of Illness</th>
<th>Medication Details</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (S) [Range]</td>
<td>Mean (S) [Range]</td>
<td>Percentage taking Chlorpromazine Equivalents</td>
</tr>
<tr>
<td>All Schizophrenics</td>
<td>23.7 (8.1) [12 - 57]</td>
<td>12.3 (8.5) [0.5 - 36]</td>
<td>97</td>
</tr>
<tr>
<td>Behavioural Signs</td>
<td>22.4 (6.0) [12 - 34]</td>
<td>12.1 (10.5) [2 - 36]</td>
<td>100</td>
</tr>
<tr>
<td>Paranoid Symptoms</td>
<td>23.2 (10.2) [13 - 57]</td>
<td>13.1 (6.7) [2 - 27]</td>
<td>100</td>
</tr>
<tr>
<td>Passivity Phenomena</td>
<td>29.0</td>
<td>5.0</td>
<td>100</td>
</tr>
<tr>
<td>Patients in Remission</td>
<td>25.1 (6.4) [17 - 34]</td>
<td>11.8 (10.1) [0.5 - 29]</td>
<td>89</td>
</tr>
</tbody>
</table>

S = standard deviation
in the group for which poorer task performance was predicted. For the 37 patients in this study, group membership broke down as follows:

**Behavioural signs.** There were 9 patients with negative (e.g. poverty of speech, blunt affect, social withdrawal) or positive (e.g. inappropriate speech, incongruous affect) behavioural signs. As the group was small, it was not subdivided into positive and negative features.

**Paranoid symptoms.** There were 18 subjects describing positive symptoms involving other agents (e.g. persecutory delusions or delusions of reference), but showing no behavioural signs.

**Passivity experiences.** Only 1 patient reported delusions of control, thought insertion or auditory hallucinations in the absence of delusional beliefs about other agents, or behavioural signs.

**Remission.** 9 patients showed no behavioural signs, and reported no positive symptoms, on the day of testing.

Two control groups were recruited. The clinical group consisted of 17 non-psychotic psychiatric outpatients with primary diagnoses of anxiety or unipolar depression. All were taking antidepressant or anxiolytic medication, and 15 of the subjects were the same as those tested for the study in Chapter 5 of this thesis. The normal control group comprised 30 normal volunteers, none of whom reported any history of psychiatric disorder. They were the same subjects as tested for the study in Chapter 5. Demographic details of the controls are given in Table 7.1.

**The tasks**

**Embedded figures test**

This was devised especially for the present study because, as mentioned in the Introduction, more difficult materials were needed than those in Witkin et al.’s
(1971) adult EFT. The task materials consisted of 8 full-colour A4-size pictures showing 1) a public building; 2) a digger; 3) a ship; 4) a peacock; 5) a schematic water jug and glasses; 6) shoes on a table cloth; 7) a stately home; and 8) a house and trees. These are shown in Appendix VII. Pictures 2), 5) and 6) were modelled on similar stimuli in the Children’s Embedded Figures Test (CEFT; Witkin et al., 1971), but were made more difficult by the addition of extra components. Pictures 1), 3), 4), 7) and 8) were modified from drawings in the British Picture Vocabulary Scale (BPVS; Dunn et al., 1982). Each of the 8 pictures had an object concealed within it; these were, respectively, a mug; a kite; an ice-cream cone; a bell; a house; a crown; a cigarette; and a candle. These hidden objects are also shown in Appendix VII. All of the hidden objects were different from each other (unlike in the CEFT where, in every case, the hidden object is either a house or a tent) to minimise practice effects. A laminated black and white cardboard cutout of each of the hidden objects was prepared so that, when searching, subjects would know the appearance of the to-be-found object. These cutouts were always exactly the same shape and size as the corresponding hidden object in the picture. Three practice pictures were also prepared; these were easier than all of the 8 experimental stimuli, and were formed by minor modification of three pictures from the CEFT (the doll, house and pram stimuli). These 3 pictures and their corresponding hidden objects (a trumpet, bow tie and tent) are also shown in Appendix VII. In an attempt to control for attention, motivation and psychomotor skills, a control condition was also developed. This used each of the above pictures, but instead of having to find a hidden object within the picture, subjects simply had to place (as fast as possible) a coloured plastic counter onto any part of the picture that was of a similar colour to the counter. A different coloured counter was used for each picture, to minimise practice effects.

In the present study, the task was presented as part of a larger battery including the tasks discussed in Chapter 5, and the Titchener illusion to be discussed later. It was presented in one complete run, and as it was quite long (and perhaps
tiring), it was given at the start of the battery for all subjects, to minimise fatigue effects. At the beginning of the task, the subject was told that he would be shown some pictures, and his aim would be to find, as fast as possible, an object hidden within each picture. It was pointed out that the hidden object would always be illustrated by means of a cardboard cutout, which would be the same shape and size, and the same way up, as the object in the picture. As an example, a cardboard cutout of a tent was produced, and following Witkin et al.'s (1971) training procedure for the CEFT, it was checked that the subject could discriminate the tent shape from three similar triangles on each of cards D_1 to D_4 of the CEFT training materials. All subjects passed this screening test. The three practice stimuli were then given - in each case the subject was presented with a picture (e.g. the pram), and was then given the relevant cutout (e.g. the tent), and was asked to find the object within the picture as quickly as possible, while the experimenter began timing with a stopwatch. Following Shah & Frith (1983), all subjects were encouraged to place the cutout shapes on top of the hidden shapes during training, to remove any ambiguity in task understanding. When the subject had found the hidden shape within a given practice picture, the control condition for that picture was presented. Subjects were told that they would be given a coloured plastic counter, and simply had to place the counter, as fast as possible, onto any part of the picture that was roughly the same colour as the counter. All subjects understood this requirement, and performed the control conditions well.

The 8 experimental pictures were then given one-by-one. The order of presentation (pictures 1 through 8) was the same for every subject, and was in order of increasing difficulty based on pilot work with 8 normal volunteers. This followed the recommendation of Vojtisek & Magaro (1974), and was an attempt to increase the chance of subjects completing the whole task because of success with the earlier items. For each of the experimental stimuli, the subject was given the relevant picture and cutout, and the experimenter began timing while asking the subject to find the
hidden object as fast as possible. The cutout was always presented in the same orientation as the hidden object in the picture to avoid any need for rotation of objects, something which schizophrenic patients are known to do poorly (Shapiro, 1952). Following Shah & Frith (1983), but in a departure from Witkin et al.’s (1971) standard procedure for the EFT, the cutout always remained present during searching. This meant that three search strategies could be objectively distinguished (see Shah & Frith, 1983): 1) where the subject saw the target figure immediately without any obvious search; 2) where the subject pointed to the target figure after visual search without using the cutout shape; 3) where the subject used the cutout for his search, either by sliding or repeated placements (concrete search). Because subjects sometimes made search errors, pointing to incorrect shapes within the pictures, the experimenter only stopped timing when the correct hidden object had been identified. All search errors, together with the strategies used in making them, were recorded on the score sheet. The task was not terminated when search errors were made, to avoid the possibility of unmotivated subjects taking the ‘easy option’ of making errors in order to bring the task to an early close. Subjects were given 2 minutes to find the hidden figure within each of the 8 experimental stimuli; if, after that time, they had failed to find it, the location was pointed out by the experimenter.

After the hidden object had been located within a given picture, the control condition for that picture was given (so the format of the task was experimental picture - control; experimental - control, etc). In the control condition, the experimenter handed a coloured plastic counter to the subject and began timing while asking him to place the counter, as fast as possible, onto a similarly coloured part of the picture. All subjects correctly placed all 8 counters, and the mean time taken by subjects in placing the counters was used as an index of motivation, attention and psychomotor speed. At the completion of each picture, the subject was asked to say what the picture showed. The resulting dependent variables included the number of hidden objects correctly located within 2 minutes (maximum 8), the mean time taken
to find these objects, the percentage of each type of strategy for cases where the hidden object was either found or not found, the number of search errors made, the mean time for correctly placing the coloured counters in the control condition, and the number of pictures (maximum 8) correctly named.

*The Titchener illusion*

The test stimuli were devised especially for the present study, and are shown in Appendix VIII. There were 9 illusion stimuli, 5 of which consisted of both the 'underestimation illusion' (i.e. large context circles causing a central black circle to appear smaller than in reality) and the 'overestimation illusion' (i.e. small context circles causing the central black circle to appear larger) next to one another. These will be referred to as 'combined illusion' stimuli. The remaining 4 stimuli consisted of 2 underestimation illusions and 2 overestimation illusions, with the illusory figure placed next to a solitary black circle in all 4 cases. These will be referred to as 'single illusion' stimuli, and it was expected that control subjects would be less likely to succumb to these than to the combined illusion stimuli, because the single stimuli may only have about half the 'illusory strength' of the combined stimuli. For each of the 9 illusion stimuli, a control condition was devised consisting simply of the same two (central) black circles from the illusion condition without any 'inducing' context. As discussed by Happé (1996), asking subjects to judge the relative size of two circles in such a condition allows some control for attentional and motivational factors. Four practice stimuli were also prepared; these consisted of one combined illusion, one single illusion, and two control stimuli. These are also shown in Appendix VIII.

In devising the illusion stimuli, it was considered important to maximise the strength of the illusions, thereby increasing the chance of controls succumbing to them, and making it easier to reveal the predicted schizophrenic superiority in judging the actual sizes of the central circles. Accordingly, a number of existing
studies of the Titchener illusion in normals were reviewed (e.g. Jaeger & Grasso, 1993; Massaro & Anderson, 1971), and these suggested that the number of ‘inducing’ contextual circles, and the size difference between the contextual and central circles, should be large in order to increase the illusory effect. In addition, it seems that the strength of the overestimation illusion is increased when the distance between the context and central circles is decreased, whereas the strength of the underestimation illusion is increased when the central and context circles are further apart (see Jaeger & Grasso, 1993, for a brief review). There is also some evidence to suggest that the strength of the underestimation illusion may be increased by decreasing the lightness contrast of the central and context circles. These factors were all considered in development of the present stimuli, and it was found that with large context circles for the underestimation illusion, a maximum of 6 circles would fit around the central circle. Similarly, with small context circles for the overestimation illusion, a maximum of 10 would fit around the central circle. For the underestimation illusion, the surrounding circles were shaded grey to decrease their lightness contrast relative to the central circle. In line with existing studies, it was also ensured that the two central black circles were the same distance apart in all of the stimuli (125 mm in the present study), so that comparative size judgments had to be made across the same distance in all cases.

The diameters of the two central black circles in the stimuli varied from 18 mm to 24 mm, but in order to optimise illusory effects, the maximum difference in their diameter on any given stimulus was only ever 2 mm; in 5 of the 9 stimuli the central circles differed by this amount, and in the remaining 4 stimuli the central circles were actually equal in diameter. The appearance of the 9 stimuli was varied systematically so that the underestimation and overestimation illusions were placed roughly an equal number of times at the top or the bottom of the displays. Similarly, the stimuli were arranged so that in the experimental and control conditions, an equal number of correct responses referred to the central circles being equal, the top one
being larger or the bottom one being larger. Thus, any subject who perseverated on the same response for every stimulus would score correctly on only 33% of the stimuli in each condition. In addition, control stimuli were sometimes presented upside down relative to their corresponding experimental stimuli so, even though subjects were never told that the control circles were the same as those in the previous stimulus, no advantage could be gained by giving the same response (e.g. "top circle larger") to both stimuli within a pair. Finally, it was decided that the cards on which the stimuli were mounted should be non-laminated, to avoid possible perceptual problems due to light reflection from the cards.

The Titchener illusion task was presented to subjects as part of a battery including the tasks discussed in Chapter 5, and the embedded figures test discussed earlier. It was presented at the end of the battery for all subjects, following the final section of the 'picture pieces' task. Subjects were told that they would be shown some cards, each of which had 2 black circles on it. Sometimes the black circles would have other circles around them, but the subject's task was simply to say whether the two black circles (this was emphasised) were equal, or whether one was bigger than the other (and if so, which one). The 4 practice stimuli were presented first, and if subjects made any errors in judging the 2 practice control stimuli, these errors were corrected by pointing out the actual relative sizes of the circles. All responses to the 2 practice illusion stimuli were said to be correct whether correct or not, to avoid any mention of illusions or visual tricks. The 9 illusion and 9 control stimuli were then presented one-by-one, in the same order for every subject (and in the same order that they appear in Appendix VIII), with an experimental stimulus always followed by its control. Based on existing studies with normals (e.g. Pressey, 1977), the subject - stimulus distance was the same for all stimuli and for all subjects (60 cm in the present study). A gap of a few seconds was always left between each stimulus presentation to avoid possible problems with visual after-effects from the preceding stimulus. Subjects’ responses were noted on a standard score sheet, and
responses to the 5 combined illusions and their controls were subsequently analysed separately from those to the 4 single illusions and their controls. For experimental or control stimuli, subjects scored 2 marks if they correctly judged the relative size of the two circles. They scored 0 if they judged the relative sizes incorrectly by one increment (e.g. by saying that the two circles were equal when, in fact, one was larger than the other). Two marks were deducted if the subject judged the relative sizes of the circles incorrectly by two increments (e.g. by saying that one circle was larger than the other when, in fact, it was smaller than the other).^24

**Procedure**

Subjects were tested individually in a quiet room, and were paid £10.00 for their participation. All patients gave written, informed consent to take part in the study, which had been approved by the Ethical Committees of the relevant hospitals. The patient information sheet and consent form are shown in Appendix V. At the start of the session, all schizophrenic patients received an abbreviated Present State Examination Version 9 (PSE; Wing *et al.*, 1974), to assess their current signs and symptoms. Normal and psychiatric controls did not receive the PSE, but the absence of any history of psychosis was checked verbally and from relevant case notes. Thereafter, all subjects received the same test protocol. This consisted of an assessment of handedness using the Annett criteria (Annett, 1970) and a test of current spatial IQ using Raven’s Advanced Progressive Matrices, Set I (Raven, 1958). The latter consists of 12 geometrical patterns, each of which has a missing part. For each pattern, the subject must choose the correct part to fit the missing area from 8 possibilities given at the bottom of the page. The test is untimed, and the score can be converted to a spatial IQ score using age-related norms provided by Raven (1958). The possible spatial IQ scores on the test range from about 65 to 130, so the test is

^24 In the illusion condition, such a response clearly indicates a large illusory effect.
adequate to identify any subjects with the exclusion criterion of a spatial IQ less than 70. The embedded figures and Titchener illusion tasks were then presented as above, as part of a larger battery including the tasks discussed in Chapter 5. In total, the assessment lasted up to 2 hours; any subjects who felt unable to complete everything in one session were asked if they would finish the tasks on the following day.

7.3 Results

Demographic and clinical variables (see Tables 7.1 and 7.2)

As with other studies in this thesis, the single schizophrenic patient with passivity symptoms in the absence of behavioural signs or paranoid symptoms, was treated as a single case and is described towards the end of the Results section. This left three schizophrenic symptom subgroups. The age of onset and duration of illness data for these groups were normally distributed, and parametric analyses of variance revealed the groups to be matched on these variables. The remaining clinical data were non-parametric, so Kruskal-Wallis one-way ANOVAs were used to explore group differences. These revealed the three symptom groups to be matched on daily dose of anticholinergic medication, but the groups differed in their chlorpromazine equivalent daily dose of neuroleptic medication \[\chi^2(2) = 8.5; p = 0.01\]. Post hoc comparisons revealed that the schizophrenics in remission were taking significantly less medication than the paranoid group. To avoid any possible confounding effects of this difference in medication dosage, some of the later analyses will use subgroups of patients matched on dosage. These were formed by systematically eliminating those schizophrenics with paranoid symptoms who were taking the highest doses of medication, until all three groups were matched. In practice, 2 paranoid patients (both taking an equivalent of 1500 mg/day of chlorpromazine) were eliminated, and as there was one medication data point missing for each of the remission and behavioural signs groups, the subgroups of schizophrenics matched on medication dosage
comprised 8 in remission, 16 with paranoid symptoms, and 8 with behavioural signs.

For the three schizophrenic symptom groups and the two control groups, any differences in demographic variables were examined using Kruskal-Wallis one-way ANOVAs. These showed all 5 groups to be matched on age, ethnicity, handedness and years of education. When corrected for ties, the overall ANOVA on sex was significant \( \chi^2(4) = 14.9; p = 0.005 \), but uncorrected for ties it barely reached significance \( \chi^2(4) = 10.3; p = 0.04 \). With post hoc multiple comparisons, none of the groups differed significantly on sex, but any possible confounding effects of the trend towards more males in the schizophrenic sample, will be investigated later for performance on the embedded figures test. As was found in earlier studies in this thesis, normal controls were significantly more likely to be employed than all the other groups \( \chi^2(4) = 58.5; p < 0.0001 \). Examination of current spatial IQs for the whole sample showed them to be normally distributed, so group differences were explored using parametric analysis of variance with post hoc multiple comparisons using Tukey’s honestly significant difference (HSD) test. This revealed a highly significant group difference \( F(4, 78) = 9.26; p < 0.0001; \) see Appendix IX for the full ANOVA table, with normal controls having a significantly higher spatial IQ than the schizophrenics in remission, those with paranoid symptoms, and those with behavioural signs. Psychiatric controls also had a significantly higher spatial IQ than the schizophrenics with paranoid symptoms. To avoid any possible confounding effects of these group differences, some of the later analyses in this section will use subgroups of subjects matched on spatial IQ. These were formed by systematically eliminating normal and psychiatric controls with the highest spatial IQs, until all groups were matched. In practice, this meant that 18 normals were eliminated (all those with IQ > 109) as were 6 psychiatric controls (those with IQ > 119), so that the mean (and standard deviation) of spatial IQ in the remaining group of 12 normals was 97.1 (8.6), and in the remaining group of 11 psychiatric controls, was 93.6 (11.9). Subject numbers in all other groups remained unchanged.
Embedded figures test

All of the control subjects performed this test, as did 36 of the schizophrenic patients (one patient in remission refused to do it). All successfully solved both experimental and control conditions in the three practice stimuli, and all attempted the 8 experimental stimuli and their corresponding control conditions.

Accuracy scores

The first dependent variable to be examined was the number of stimuli (maximum 8) in which the subject correctly found the hidden figure. Following Shah & Frith (1983), this is termed the accuracy, and mean scores by group are shown in Table 7.3 and Figure 7.1. As can be seen, there was no ceiling effect for the scores of the normal controls so, as planned, the materials were more difficult than the EFT stimuli of Witkin et al. (1971).

Table 7.3. Mean accuracy scores by group on the embedded figures test

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) accuracy (max = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>5.9 (1.3) (N = 9)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>5.8 (1.8) (N = 18)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>4.9 (1.9) (N = 8)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>5.4 (2.4) (N = 17)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>7.0 (1.0) (N = 30)</td>
</tr>
</tbody>
</table>

S = standard deviation

Scores were approximately normally distributed, but as variances were rather heterogeneous, group differences were firstly analysed using Kruskal-Wallis one-way ANOVA. This was highly significant [$\chi^2(4) = 15.3; p = 0.004$], and post hoc multiple comparisons showed that normal controls scored significantly higher than schizo-
Figure 7.1. Mean accuracy on the embedded figures test by group

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs. Error bars represent the standard error of the mean.
phrenics in remission. The difference between normal and psychiatric controls also approached significance at \( p < 0.075 \) (one-tailed). These results were checked with parametric analysis of variance\(^{25}\), and the same main effect of group was obtained \( [F(4, 77) = 4.20; p = 0.004; \text{see Appendix IX for the full ANOVA table}] \), together with the same post hoc multiple comparisons (using Tukey’s HSD test). Thus, although the results did not support the prediction that schizophrenics with behavioural signs or paranoid symptoms would score significantly higher than controls, it is notable that these two symptomatic groups scored somewhat better than remitted schizophrenics or psychiatric controls.

Any possible confounding effects of medication differences between the three schizophrenic groups were investigated by correlating dosage with accuracy score for each group in turn. The correlation was significant for the paranoid schizophrenics \( [\rho(18) = -0.51; p = 0.02, \text{one-tailed}] \), so the Kruskal-Wallis one-way ANOVA of accuracy across all 5 groups was repeated using the schizophrenic subgroups matched on medication dosage (discussed earlier). The ANOVA was still significant \( [\chi^2(4) = 12.7; p = 0.01] \), and the significance levels of the post hoc multiple comparisons were unchanged.

The effects of spatial IQ differences between groups were also investigated by correlating accuracy score with spatial IQ for each of the groups in turn. A number of values of Spearman’s \( \rho \) approached significance, but the value only reached significance for the paranoid schizophrenics \( [\rho(18) = 0.53; p = 0.01, \text{one-tailed}] \). Group differences in accuracy were investigated using the spatial IQ-matched subgroups of subjects discussed earlier; the Kruskal-Wallis one-way ANOVA just

\(^{25}\) Attempts were made to transform the data, but common transformations including logarithmic and square root manipulations did not lead to greater homogeneity of variance. The greatest proportional difference between group variances therefore remained at about 5.8 (normals vs. psychiatric controls).
failed to reach significance \( \chi^2(4) = 8.6; p = 0.07 \), with *post hoc* multiple comparisons showing that the difference between normal controls and schizophrenics in remission approached significance at \( p < 0.075 \). Thus, even with the drastic reduction in subject numbers required for the IQ matching (e.g. only 12 normal controls remained in this analysis), there was still a strong trend for the remitted schizophrenics to score more poorly than normals. These effects were confirmed with parametric analysis of covariance (ANCOVA) on the *whole* sample, which showed that with spatial IQ partialled out, the group difference in accuracy score remained significant \( [F(4, 76) = 3.00; p = 0.02; \text{see Appendix IX for the output table}] \). The adjusted group means from the ANCOVA are shown in Table 7.4, from where it is clear that the symptomatic schizophrenic patients show a tendency towards higher accuracy than those in remission.

**Table 7.4. Adjusted mean accuracy by group with spatial IQ co-varied**

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean accuracy (max = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>6.1 ( (N = 9) )</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>6.2 ( (N = 18) )</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>4.9 ( (N = 8) )</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>5.2 ( (N = 17) )</td>
</tr>
<tr>
<td>Normal controls</td>
<td>6.6 ( (N = 30) )</td>
</tr>
</tbody>
</table>

An attempt was made to control for attention, motivation and psychomotor ability using the reaction time data from the control condition of the task, in which subjects had to place coloured counters as fast as possible onto relevant parts of the pictures. It should be noted that this condition was much easier than the condition in which subjects had to find the hidden objects (as shown by the fact that all subjects correctly placed all 8 counters, and did so in considerably less time than they spent
on the embedded condition), so the task was a less than ideal control measure. It is likely that greater deficits would have appeared on a more difficult task, so we can assume that attention may not have been fully controlled in the present study. The mean reaction times on the control condition are shown in Table 7.5, and Kruskal-Wallis one-way ANOVA revealed the groups to be matched on reaction time \( \chi^2(4) = 6.9; p = 0.14 \), although there was a trend (at \( p < 0.15 \), one-tailed) for the schizophrenics with behavioural signs to have a longer reaction time than normals.

Table 7.5. Mean reaction times by group on the embedded figures control condition

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) reaction time/secs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>3.86 (2.34) (N = 9)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>3.24 (1.54) (N = 18)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>3.41 (1.41) (N = 8)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>3.10 (1.70) (N = 17)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>2.49 (1.01) (N = 30)</td>
</tr>
</tbody>
</table>

S = standard deviation

To investigate the effects of attentional impairments on task performance, accuracy score was correlated with control reaction time for each of the subject groups in turn. Most correlation coefficients were significant, but when ANCOVA was used to partial out the effects of attention, the significant group difference in accuracy score remained \[ F(4, 76) = 3.17; p = 0.02; \text{see Appendix IX for the output table} \]. Finally, ANCOVA was performed on accuracy score with both spatial IQ and attention partialled out simultaneously. The significant group difference in accuracy remained \[ F(4, 75) = 2.80; p = 0.03; \text{see Appendix IX for the output table} \], and the adjusted group means from this ANCOVA are shown in Table 7.6, from where it is clear that the scores of the schizophrenics with behavioural signs are now slightly higher than those of the normal controls.
Table 7.6. Adjusted mean accuracy by group with spatial IQ and attention co-varied

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean accuracy (max = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>6.4 (N = 9)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>6.2 (N = 18)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>5.0 (N = 8)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>5.1 (N = 17)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>6.2 (N = 30)</td>
</tr>
</tbody>
</table>

As discussed in Chapter 6, Witkin et al. (1971) reported that males generally perform better than females on embedded figures tests. This was investigated in the present study by comparing the mean accuracy of males and females in the normal control group. Although the mean for the males (7.1) was higher than that for the females (6.8), the difference was not significant \(t(28) = 0.91; p = 0.19\), one-tailed.

As noted earlier, all 5 subject groups in the present experiment were just matched on sex, but it could be argued that a contributory factor to the generally good task performance of the behavioural signs and paranoid schizophrenic groups, was the fact that many of the schizophrenics were male, whereas a large proportion of the controls were female. To investigate this, the mean accuracy scores for each group were calculated with only male subjects included. In practice, this meant that data from 15 normals and 7 psychiatric controls were analysed, together with data from 8 schizophrenics in remission, 15 paranoid schizophrenics, and 7 patients with behavioural signs. The new mean accuracy scores are shown in Table 7.7.

Group differences were firstly analysed non-parametrically, and Kruskal-Wallis one-way ANOVA of accuracy across groups was still highly significant \(\chi^2(4) = 13.9; p = 0.008\). Post hoc multiple comparisons showed that this difference was due solely to normal controls scoring significantly higher than schizophrenics in
remission; there was now no trend for normal and psychiatric controls to differ.

Table 7.7. Mean accuracy by group with only male subjects included

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) accuracy (max = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>6.4 (0.8) (N = 7)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>6.1 (1.4) (N = 15)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>4.9 (1.9) (N = 8)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>5.3 (2.4) (N = 7)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>7.1 (0.6) (N = 15)</td>
</tr>
</tbody>
</table>

S = standard deviation

Thus, the use of only males was even more clear-cut than analysis of the whole sample, in revealing a specific deficit in the remitted schizophrenic patients. The analysis was checked with parametric ANOVA and Tukey’s HSD tests, and the same significant main effect and post hoc multiple comparison were obtained [F(4, 47) = 4.04; p = 0.007; see Appendix IX for the output table]. As with analysis of the whole sample, ANCOVA was used to partial out the effects of spatial IQ or attentional differences between groups, and in both cases the main effect remained significant [IQ: F(4, 46) = 3.84; p = 0.009; Attention: F(4, 46) = 3.65; p = 0.011; see Appendix IX for the output tables]. When ANCOVA was used to control for both spatial IQ and attention simultaneously, the main effect of group again remained significant [F(4, 45) = 4.52; p = 0.004; see Appendix IX]; the adjusted group means from this ANCOVA are given in Table 7.8. Again, it can be seen that the symptomatic schizophrenic groups score slightly higher than controls and remitted schizophrenics, when IQ and attention are taken into account.
Table 7.8. Adjusted mean accuracy with spatial IQ & attention co-varied, males only

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean accuracy (max = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>7.0 (N = 7)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>6.4 (N = 15)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>5.1 (N = 8)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>5.1 (N = 7)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>6.2 (N = 15)</td>
</tr>
</tbody>
</table>

A final point relates to two patients in the remission schizophrenic group, who (as will be remembered from the Discussion in Chapter 5) may have had underlying paranoid symptoms which were not exposed by the Present State Examination (PSE). All of the above analyses allocated these patients to the remission group because, objectively, that was the correct classification based on the PSE. However, it should be noted that the accuracy score of both of those patients on the embedded figures test was 7/8, so if they had both been placed in the paranoid group, the group differences described above would have been accentuated.

Analysis of search errors

Another variable of interest is the number of times over the whole embedded figures test that subjects incorrectly reported finding the hidden object. The mean number of these search errors for each subject group is shown in Table 7.9. Data were non-parametric, so group differences were analysed using Kruskal-Wallis one-way ANOVA. This revealed a highly significant main effect of group [$\chi^2(4) = 17.8; p = 0.001$], and post hoc multiple comparisons showed that normal controls made significantly less search errors than schizophrenics with behavioural signs or paranoid symptoms. The difference between normals and remitted schizophrenics approached significance at $p < 0.10$, one-tailed.

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Table 7.9. Mean number of search errors by subject group over the whole task

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) number of search errors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>3.3 (1.1) (N = 9)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>4.9 (5.8) (N = 18)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>3.4 (2.1) (N = 8)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>1.9 (2.0) (N = 17)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>1.5 (1.6) (N = 30)</td>
</tr>
</tbody>
</table>

S = standard deviation

One interpretation of the greater number of search errors in the schizophrenic groups, is that patients were more likely than controls to make guesses based on line formations in the pictures which approximated to the outline of the to-be-found object on the cardboard cutout. If correct, this may explain the relatively good performance of the symptomatic schizophrenics in the present study, because people who make more guesses may be more likely to eventually locate the hidden object correctly. Such a rationale would negate a theoretical account of the present findings in terms of metarepresentational theory, so to investigate this, correlations were computed between number of search errors and accuracy for each of the schizophrenic groups. All values of Spearman’s ρ were non-significant, and in fact the trends suggested that subjects who made more search errors had a lower accuracy. The interpretation of these findings, therefore, must be that symptomatic schizophrenics had a relatively high task accuracy despite making more search errors than normal controls.

Search strategies

Following Shah & Frith (1983), the mean percentage of each strategy used was calculated separately for correct responses (i.e. those where subjects successfully located the hidden figure in a picture) and for incorrect responses (i.e. search errors
and those cases where subjects failed to find the hidden figure after 2 minutes). Percentage scores were calculated in order to remove the differences in performance level. The scores are given for each subject group in Table 7.10.

Table 7.10. Mean percentage (and standard deviation) of each search strategy used

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Correct responses</th>
<th>Incorrect responses</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Instant find</td>
<td>Visual search</td>
</tr>
<tr>
<td></td>
<td>(0.0)</td>
<td>(33.4)</td>
</tr>
<tr>
<td>Behavioural sign schizophrenics</td>
<td>0.0</td>
<td>35.0</td>
</tr>
<tr>
<td></td>
<td>(0.0)</td>
<td>(33.4)</td>
</tr>
<tr>
<td>Paranoid schizophrenia</td>
<td>1.6</td>
<td>36.0</td>
</tr>
<tr>
<td></td>
<td>(4.6)</td>
<td>(36.4)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>0.0</td>
<td>4.2</td>
</tr>
<tr>
<td></td>
<td>(0.0)</td>
<td>(7.7)</td>
</tr>
<tr>
<td>Psychiatric control patients</td>
<td>0.8</td>
<td>31.2</td>
</tr>
<tr>
<td></td>
<td>(3.5)</td>
<td>(42.7)</td>
</tr>
<tr>
<td>Normal control subjects</td>
<td>1.9</td>
<td>25.9</td>
</tr>
<tr>
<td></td>
<td>(6.2)</td>
<td>(28.3)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Visual search</td>
<td>Concrete search</td>
</tr>
<tr>
<td></td>
<td>(9.3)</td>
<td>(22.2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(18.6)</td>
<td>(33.1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(0.0)</td>
<td>(0.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(26.6)</td>
<td>(42.6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(9.5)</td>
<td>(19.5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(89.6)</td>
<td>(19.6)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

All data were analysed non-parametrically because of the large differences in variance between groups. For correct responses, hardly any subjects produced immediate responses, and there were no significant differences between groups in the percentage of immediate responses. All groups used the concrete search strategy more often than the visual strategy for correct responses (most effects significant with a one-tailed Wilcoxon signed ranks test). Groups did not differ in the proportion of correct responses for which a visual search was used [$\chi^2(4) = 6.1; p = 0.19$], although post hoc multiple comparisons showed that there was a trend for the schizophrenics
in remission to use visual search less often than the paranoid schizophrenics (at p < 0.15, one-tailed test). Similarly, there were no significant group differences in the percentage of correct responses for which a concrete search was adopted \( \chi^2(4) = 6.6; p = 0.16 \), although post hoc multiple comparisons revealed a trend (at p < 0.10, one-tailed) for the schizophrenics in remission to use concrete search more often than the paranoid schizophrenic group.

For incorrect responses, the immediate strategy was hardly ever used, so a separate category was not formed for analysis. All groups again used the concrete strategy more often than the visual strategy (all effects significant on a one-tailed Wilcoxon signed ranks test). Groups did not differ significantly in the percentage of incorrect responses for which either a visual search or a concrete search was used, and there were no trends in the data towards group differences. Inspection of Table 7.10 reveals that subjects tended to use concrete searches more often for incorrect responses than for correct responses, and this effect was highly significant for the normal controls (Wilcoxon: p = 0.0005, one-tailed).

**Search times**

Using only the search time data from pictures on which subjects correctly located the hidden object, a mean location time was calculated for each subject. These values are given by group in Table 7.11.

Group differences were analysed non-parametrically because of heterogeneity of variance, and Kruskal-Wallis one-way ANOVA revealed the groups to be matched on location time \( \chi^2(4) = 5.7; p = 0.22 \). Post hoc comparisons revealed a trend (at p < 0.12, one-tailed) for the remitted schizophrenics to take longer to find the hidden objects than normal controls.
Table 7.11. Mean time taken to locate the hidden objects for each group

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) location time/secs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>30.4 (8.0) (N = 9)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>35.3 (15.8) (N = 18)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>45.7 (13.1) (N = 8)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>36.4 (17.5) (N = 17)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>32.2 (15.2) (N = 30)</td>
</tr>
</tbody>
</table>

S = standard deviation

_Naming of the picture scenes_

Finally, it was found that all subjects successfully named 7 of the 8 picture scenes in the embedded figures test. Many people had problems naming number 5 (the schematic water jug and glasses), but all groups were equally likely to make errors in the naming of this picture [$\chi^2(4) = 7.4; p = 0.12$]. For the sample as a whole, and for each subject group taken individually, there was no significant correlation between the ability to correctly name picture 5, and the ability to locate the hidden object within that picture (all Spearman’s $\rho$ non-significant).

_Titchener illusion task_

A few subjects in each group did not perform this task, expressing a desire to terminate the testing session because of fatigue. Specifically, one normal and two psychiatric control subjects, and one remitted schizophrenic and one with behavioural signs, did not carry out the task. Four paranoid patients refused to do the task, but the reason here seemed more to do with their symptomatology, as three of these people felt that their answers might, in some way, give the experimenter an insight into their own thoughts or personalities.

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Of those subjects who attempted the task, all understood the task demands and completed all 9 experimental stimuli and their corresponding controls. As discussed earlier, for either control or experimental conditions, subjects were given 2 marks each time they correctly judged the relative size of the two target circles in a stimulus. A score of 0 was given if the relative sizes were judged incorrectly by one increment (e.g. by saying that the circles were equal when one was bigger than the other), and two marks were deducted if the relative sizes were judged incorrectly by two increments (e.g. by saying that one circle was larger than the other in the opposite direction to the true difference). All cases of errors on the illusion stimuli were in the direction of subjects succumbing to the illusion so that, for example, with two equal circles in a ‘combined illusion’ stimulus, subjects never said that the circle at the centre of the underestimation illusion was larger than the other. Data were firstly analysed separately for the ‘combined illusion’ and ‘single illusion’ stimuli, as the former were expected to be harder to judge accurately than the latter.

**Combined illusion stimuli**

As there were 5 combined illusion and corresponding control stimuli, the maximum score was 10 in the illusion condition and 10 in the control condition. In the illusion condition, some subjects had a negative score, reflecting the fact that they judged several pairs of circles incorrectly by two increments (i.e. they succumbed very strongly to the illusion). The mean scores are shown by group in Table 7.12 and Figure 7.2.

Scores in the control condition were not normally distributed, so group differences in this condition were analysed using Kruskal-Wallis one-way ANOVA. This revealed all groups to be matched on control scores, with no trends for groups to differ. In the illusion condition scores were normal, with homogeneous variances, so group differences were analysed using parametric ANOVA. This was again non-significant, but it is notable that the paranoid schizophrenics scored slightly higher.
Figure 7.2. Mean scores in control & illusion conditions for combined illusions

- Control
- Illusion

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs. Error bars represent the standard error of the mean.
than all other groups, and of all the schizophrenics, only they showed any sign of not succumbing completely to the illusions. Accordingly, when group differences in illusion score were analysed for only the three schizophrenic groups, the paranoid patients showed a trend (at $p < 0.075$, one-tailed) to score higher than those with behavioural signs. To compare the difference between scores in the control and illusion conditions, Wilcoxon signed ranks tests were performed for each subject group in turn. These were all highly significant (at $p < 0.005$, one-tailed), reflecting the fact that all groups found the illusion condition much harder than the control condition. Correlations between scores in the control and illusion conditions were non-significant for all groups, suggesting that the two conditions may have been tapping different cognitive processes.

Table 7.12. Mean scores by group on the combined illusion stimuli

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) control score (max = 10)</th>
<th>Mean (S) illusion score (max = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behavioural signs schizophrenics</td>
<td>8.25 (1.67)</td>
<td>-1.75 (2.71)</td>
</tr>
<tr>
<td>Paranoid schizophrenics</td>
<td>8.71 (1.49)</td>
<td>0.57 (2.77)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>8.75 (2.12)</td>
<td>-1.00 (1.85)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>9.07 (1.98)</td>
<td>0.13 (3.16)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>9.52 (0.87)</td>
<td>0.41 (3.09)</td>
</tr>
</tbody>
</table>

S = standard deviation

For each subject, illusion scores were then examined for only those stimuli on which the control condition was passed. An 'accuracy index' was calculated by dividing the score in the illusion condition by the control score (so the maximum value of the index was 1.00), and the mean values of this are shown for each group in Table 7.13. The accuracy indices were normally distributed, and parametric ANOVA of scores across groups was non-significant. It is notable that the paranoid
patients had the highest mean accuracy index, while the schizophrenics with behavioural signs had the lowest. In line with this, analysis of differences between only the three schizophrenic groups revealed a trend (at \( p < 0.075 \), one-tailed) for the paranoid and behavioural signs groups to differ. Any possible confounding effects of spatial IQ differences between groups were examined by correlating the accuracy index with spatial IQ for each group in turn. All values of Pearson’s \( r \) were non-significant. Similarly, for each of the schizophrenic groups, there was no correlation between accuracy index and chlorpromazine equivalent daily medication dosage.

Table 7.13. Mean values by group of the ‘accuracy index’ for the combined illusions

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) illusion index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>-0.21 (0.34)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>0.09 (0.36)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>-0.06 (0.15)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>0.04 (0.31)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>0.04 (0.31)</td>
</tr>
</tbody>
</table>

\( S = \) standard deviation

**Single illusion stimuli**

As there were 4 single illusion and control stimuli, the maximum score was 8 in the illusion condition and 8 in the control condition. The mean scores by group are given in Table 7.14 and Figure 7.3. As with the combined stimuli, scores in the control condition were non-normal, so group differences were analysed using Kruskal-Wallis one-way ANOVA. This was non-significant, and there were no trends for groups to differ. In the illusion condition scores were normal, and group differences were analysed with parametric ANOVA. This was again non-significant, but it is notable that the schizophrenic patients with behavioural signs scored lower than all other groups, seeming particularly susceptible to the illusions. Accordingly,
Figure 7.3. Mean scores in control & illusion conditions for single illusions

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs. Error bars represent the standard error of the mean.
comparison of only the three schizophrenic groups revealed a trend (at $p < 0.075$, one-tailed) for the patients with behavioural signs to score lower than those with paranoid symptoms. To compare differences between scores in the control and illusion conditions, Wilcoxon signed ranks tests were performed for each subject group in turn. These were all significant, reflecting the fact that all groups found the illusion condition harder than the control condition. The difference between scores in the two conditions was smaller for the three schizophrenic groups (all $p < 0.02$, one-tailed) than for the two control groups (both $p < 0.0007$, one-tailed), and this was largely a reflection of the fact that the schizophrenics scored slightly worse than controls in the control condition of the task, but (if paranoid or remitted) had similar scores to controls in the illusion condition. As with the combined stimuli, correlations between scores in the control and illusion conditions were non-significant for all groups.

Table 7.14. Mean scores by group on the single illusion stimuli

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) control score (max = 8)</th>
<th>Mean (S) illusion score (max = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behavioural signs schizophrenics</td>
<td>6.25 (1.98)</td>
<td>3.00 (1.07)</td>
</tr>
<tr>
<td>Paranoid schizophrenics</td>
<td>6.71 (1.49)</td>
<td>4.71 (2.30)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>7.25 (1.04)</td>
<td>5.00 (1.85)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>7.60 (0.83)</td>
<td>4.67 (1.63)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>7.72 (0.70)</td>
<td>4.48 (1.98)</td>
</tr>
</tbody>
</table>

$S =$ standard deviation

For each subject, illusion scores were then examined for only those stimuli on which the control condition was passed. As with the combined illusion stimuli, an ‘accuracy index’ (max = 1.00) was calculated by dividing the score in the illusion condition by the control score, and the means of this index are given for each group.
in Table 7.15. The accuracy indices were normally distributed, and parametric ANOVA revealed no significant differences between groups. However, comparison of only the three schizophrenic groups did show a trend (at \( p < 0.10 \), one-tailed) for the paranoid patients to have a higher accuracy index than those with behavioural signs. For all subject groups there was no correlation between accuracy index and spatial IQ.

Table 7.15. Mean values by group of the ‘accuracy index’ for the single illusions

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) illusion index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>0.43 (0.21)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>0.65 (0.31)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>0.63 (0.28)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>0.55 (0.23)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>0.56 (0.25)</td>
</tr>
</tbody>
</table>

S = standard deviation

Inspection of tables 7.13 and 7.15 shows that for all groups, the accuracy indices for the combined illusions were much smaller than for the single illusions, reflecting the greater strength of the combined illusions. Thus, for the normal controls, for example, the difference between indices for the two sets of stimuli was highly significant, with \( t(28) = 11.5; p < 0.001 \).

Analysis of the complete set of stimuli

Finally, all 9 illusions and their controls were analysed together, so that the maximum score in both the illusion and control conditions was 18. The mean scores by group are shown in Table 7.16 and Figure 7.4. In the control condition, Kruskal-Wallis one-way ANOVA revealed no group differences, and no trends for groups to differ. In the illusion condition, group scores were compared using parametric
Figure 7.4. Mean scores in control & illusion conditions for all stimuli

Norm = normal controls; Psych = psychiatric controls; Rem = schizophrenics in remission; Par = paranoid schizophrenics; Beh = schizophrenics with behavioural signs. Error bars represent the standard error of the mean.
ANOVA, which was also non-significant. However, it is notable that the paranoid schizophrenic group had the highest score in this condition, while the patients with behavioural signs had the lowest score. In line with this, comparison of only the three schizophrenic groups revealed a trend (at p < 0.075, one-tailed) for the paranoid patients to score higher than those with behavioural signs. To compare performance in the two conditions, Wilcoxon signed ranks tests were carried out for each subject group in turn. These were all significant (at p < 0.01), reflecting the fact that all groups found the illusion condition harder than the control condition. There were no correlations between scores in the control and illusion conditions for any groups.

Table 7.16. Mean scores by group on the complete set of illusion stimuli

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) control score (max = 18)</th>
<th>Mean (S) illusion score (max = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behavioural signs schizophrenics</td>
<td>14.50 (3.34)</td>
<td>1.25 (3.01)</td>
</tr>
<tr>
<td>Paranoid schizophrenics</td>
<td>15.43 (2.77)</td>
<td>5.29 (4.41)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>16.00 (2.83)</td>
<td>4.00 (2.83)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>16.67 (2.47)</td>
<td>4.80 (4.13)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>17.24 (1.12)</td>
<td>4.90 (4.55)</td>
</tr>
</tbody>
</table>

S = standard deviation

Finally, illusion scores were examined for only those stimuli on which the control condition was passed. As before, an ‘accuracy index’ (maximum 1.00) was calculated for each subject by dividing the score in the illusion condition by the score in the control condition, and the mean values of this by group are given in Table 7.17. Values were normally distributed, and parametric ANOVA revealed all groups to be matched on the accuracy index. However, as before, it is clear from Table 7.17 that patients with behavioural signs had the lowest accuracy of all groups, while those with paranoid symptoms had the highest accuracy. Accordingly, comparison of only
the three schizophrenic groups showed a trend (at $p < 0.075$, one-tailed) for the paranoid patients to score higher than those with behavioural signs. For all groups, there were no significant correlations between accuracy index and spatial IQ.

Table 7.17. Mean values by group of the ‘accuracy index’ for all illusions

<table>
<thead>
<tr>
<th>Subject group</th>
<th>Mean (S) illusion index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenics with behavioural signs</td>
<td>0.06 (0.24)</td>
</tr>
<tr>
<td>Schizophrenics with paranoid symptoms</td>
<td>0.34 (0.32)</td>
</tr>
<tr>
<td>Schizophrenics in remission</td>
<td>0.25 (0.17)</td>
</tr>
<tr>
<td>Psychiatric controls</td>
<td>0.28 (0.23)</td>
</tr>
<tr>
<td>Normal controls</td>
<td>0.27 (0.25)</td>
</tr>
</tbody>
</table>

S = standard deviation

Single case with passivity symptoms

This case was the same as that discussed in Chapter 5; he was excluded from all of the above analyses to provide consistency with the earlier studies in this thesis. His spatial IQ was 80, a value lower than the means of all other subject groups in this study. On the embedded figures test, he had an accuracy of 6/8, a value comparable with the means of the behavioural signs and paranoid schizophrenic groups (5.9 and 5.8 respectively). His mean location time was 31.10 seconds, a value similar to that for the schizophrenics with behavioural signs (30.4 seconds), and he always used a concrete strategy when searching. He made 4 search errors (comparable with the other schizophrenic groups), and his relatively good performance on the test was notable given that his mean reaction time in the attention control condition was 5.12 seconds, a value higher than the means for all other groups in the study.

On the Titchener illusion task, he performed very well, scoring 10/10 and 6/10
respectively on the control and illusion conditions of the combined illusion stimuli. This gave him an accuracy index with these stimuli of 0.60, a value much higher than the highest of the other groups (0.09 for the paranoid schizophrenics). Similarly, with the single illusion stimuli, he scored 8/8 and 6/8 respectively on the control and illusion conditions. His accuracy index with these stimuli was therefore 0.75, a value higher than the highest of the other groups (0.65). Finally, with all stimuli taken together, his accuracy index was 0.67, a value again much higher than the highest of the other groups (0.34).

Correlations between scores on the various tasks

To explore the relationship between performance on the embedded figures test and metarepresentational ability as measured by the ‘hints task’ (Corcoran et al., 1995), embedded figures accuracy was correlated with scores on the hints task (from the study in Chapter 5 of this thesis) for each subject group in turn. As 5 correlations were performed, a Bonferroni-corrected p-value of 0.01 was used, but even with a conventional significance level of 0.05, all values of Spearman’s ρ were non-significant. Scores on the hints task were also correlated with the accuracy index for the whole set of illusions from the present study, and all values of ρ again failed to reach significance for all subject groups.

To investigate the relationship between task performance in the present study and performance on the ‘picture pieces task’ (see Chapter 5), picture pieces scores from the ‘isolation’ condition (i.e. where subjects had to name objects in isolation from any context), were firstly correlated with embedded figures accuracy for each subject group in turn. A Bonferroni-corrected significance level of 0.01 was used, but even with a conventional α-level of 0.05, all correlations were non-significant. Similar correlations between picture pieces scores in the ‘isolation’ condition and the illusion accuracy index (for the whole set of illusions) were also non-significant for
all subject groups at \( p = 0.05 \).

Picture pieces scores in the 'context' condition (i.e. where subjects had to name objects in the context of a scene) were then correlated with embedded figures accuracy for each group in turn. All correlations were non-significant at \( \alpha = 0.01 \), but notably the correlation for the behavioural signs patients was the only one to reach significance with an \( \alpha \)-level of 0.05 \( [\rho(8) = -0.69; \ p = 0.03, \ one-tailed] \). The negative value of \( \rho \) reflects the fact that, as predicted, subjects who were poor at naming objects in context were relatively good at finding hidden objects within a picture. Finally, picture pieces scores in the 'context' condition were correlated with the illusion accuracy index (for the whole set of illusions), for each group in turn. All correlations were non-significant at \( \alpha = 0.01 \), but the correlation coefficient for the paranoid patients was the only one to reach significance at \( \alpha = 0.05 \) \( [\rho(14) = +0.54; \ p = 0.02] \). This correlation remained significant when the analysis was checked with re-classification of the two 'remitted' patients suspected of having underlying paranoid delusions (see earlier discussions). The positive value of the coefficient was not expected, as it suggests that paranoid patients who were poor at naming objects in context were more likely to be influenced by context in the illusions task. Together, these results suggest that rather different cognitive processes may underlie the performance of schizophrenic patients on the embedded figures and illusions tasks, and in line with this, correlations between embedded figures accuracy and the illusion accuracy index were non-significant for all subject groups (all \( p > 0.2 \)). This issue is explored further in the Discussion.

### 7.4 Discussion

**Performance on the embedded figures test**

Pilot work with normal volunteers showed that Witkin *et al.*'s (1971) standard
EFT stimuli were often solved very easily when the to-be-found figure was displayed during searching. A new embedded figures test was therefore developed for the present study, and as planned, this proved much more difficult than Witkin et al.'s task. However, data from normal controls revealed no floor or ceiling effects in performance, and there was some variance in scores, so the task had a reasonable ability to discriminate between subjects. In the present study all subjects completed the whole task, and this may well have reflected the fact that the easier stimuli were given first (as recommended by Vojtisek & Magaro, 1974), so subjects were encouraged to continue because of early successes.

By analogy with Shah & Frith's (1983) finding of superior autistic performance relative to controls on the Children's Embedded Figures Test (Witkin et al., 1971), it was predicted that, because of a lack of awareness of context (e.g. Done & Frith, 1984; Naficy & Willerman, 1980; the 'picture pieces' study in Chapter 5 of this thesis), schizophrenic patients with behavioural signs or delusions would score significantly better than controls or remitted schizophrenics on the new embedded figures test. This prediction was not supported, but trends in the data suggested that effects were operating in the predicted direction. Thus, the only significant difference in the accuracy data showed poorer performance by patients in remission relative to normal controls; the two groups of symptomatic schizophrenics performed similarly to normals, and somewhat better than psychiatric controls and remitted schizophrenics. In itself, this result is notable, because many of the studies reviewed in this thesis (e.g. the theory of mind studies of Corcoran et al., 1995 and Frith & Corcoran, 1996), have consistently found remitted schizophrenics to perform on a par with normals, while symptomatic patients perform poorly. Furthermore, the remitted patients in the present study were the same subjects who scored similarly to controls on the 'hints task' in Chapter 5, and the symptomatic schizophrenics were the same ones who scored poorly on the hints task in that study. These results suggest that (as predicted) the positive or negative features of schizophrenia may be associated with
facilitation on embedded figures tests relative to the remitted schizophrenic condition, and it is possible that a significant difference would have appeared between the symptomatic and remitted groups if larger subject numbers had been used.

There are several possible reasons for the poorer performance of the remitted schizophrenics relative to normal controls, and these were mentioned in the post hoc analyses of the accuracy data. Firstly, most of the remitted patients were receiving neuroleptic medication, whereas all of the normals were unmedicated. Although the correlation between dosage and accuracy only approached significance for the remitted group \( \rho(7) = -0.56; p = 0.09, \text{ one-tailed} \), the negative value of \( \rho \) reflects the lower task accuracy associated with higher doses. It is certainly possible, therefore, that medication effects were a contributory factor to the significant difference between the normals and remitted schizophrenics\(^26\). A second possible reason for the group difference is the significantly higher spatial IQ of the normals compared to the remitted patients. However, post hoc analyses showed that the correlations between spatial IQ and task accuracy only approached significance for most groups, and although partialling out IQ reduced the difference in accuracy between normals and remitted schizophrenics, the difference still remained significant. Thus, spatial IQ differences only had a weak influence on group differences in accuracy. The third (and most important) contribution to the difference in accuracy between normal and remitted schizophrenics probably came from relatively poor attention, motivation and/or psychomotor skills in the remitted group, features which are often found in patients with a psychiatric history. An attempt was made to control for these factors using the task condition in which subjects had to place coloured counters, as fast as possible, onto the pictures. Reaction times for this were highly correlated with accuracy in the embedded condition, but partialling out reaction times left intact the significant normal/remitted difference in accuracy. A major problem

\(^{26}\) In support of this, the psychiatric controls were also medicated (although not with neuroleptics), and they showed a trend towards lower accuracy than normals.
with the attention control condition was that it was much easier than the embedded condition, so may have been comparatively insensitive to motivational or attentional problems in the subjects. Future work should, therefore, use a more difficult control task (e.g. a ‘finger tapping’ task, where subjects must tap a key as fast as possible for a minute or more), preferably matched on difficulty with the embedded figures task; we might expect that the partialling out of subjects’ scores on such a control task would considerably reduce the difference between normals and remitted schizophrenics in embedded figures accuracy.

The schizophrenics with behavioural signs and paranoid symptoms in the present study were also medicated, had lower spatial IQs than controls, and had attention control scores similar to those of the remitted schizophrenic patients. Their relatively high embedded figures accuracy should, therefore, be seen in the light of these factors, and it is possible that with adequate control measures, these patients would show higher accuracy than normals, as predicted. The post hoc analyses performed on the present data gave some support for this, as controlling simultaneously for attention and spatial IQ pushed the means of the symptomatic schizophrenic groups slightly higher than those of the control groups, whilst leaving the mean of the remitted patients almost unchanged. Partialling of a more difficult attention control measure (as discussed above) would have a much more marked effect, and while increasing the mean accuracy of all the schizophrenic groups, this would give a particularly strong boost to the scores of patients with behavioural signs, as these people are generally recognised as having the worst attention and motivation (e.g. Frith, 1992).  

And in the present study, the patients with behavioural signs showed a trend towards a longer control reaction time than normals.

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Thus, for those hidden objects which they accurately located, remitted patients showed a trend towards a longer search time than normals. No other groups showed trends to differ on this variable. Analysis of search strategies showed that very few subjects used the immediate strategy, and this probably reflected the difficulty of the materials, with the hidden figure hardly ever being immediately apparent. All subjects used the concrete strategy (involving sliding or repeated placement of the cutout shape) more often for incorrect responses (e.g. those where they were unable to locate the hidden figure within 2 minutes) than for correct responses. One interpretation of this may be that subjects used the concrete strategy when they were having particular difficulty in locating the object, and in this regard, it is notable that schizophrenics in remission showed a trend (for correct responses) to use the concrete strategy more often than paranoid patients. This may reflect a trend for the remitted patients to find the task more difficult than the paranoid subjects. As mentioned in the Results section, all three schizophrenic groups made more search errors (saying that they had found the hidden object, when in fact they had not) than normal controls, but there was no relationship between number of search errors and task accuracy. A possible interpretation, therefore, of the relatively high accuracy of the symptomatic schizophrenics compared to the remitted patients, is that the former two groups scored well despite their tendency towards search errors, an argument again consistent with particular deficits in the remitted group. It should be emphasised that after making any search errors, all subjects were allowed to continue searching for the hidden object up to the 2 minute time limit. This was considered an important safeguard against the possibility of unmotivated patients taking the ‘easy option’ of quickly identifying an incorrect location within the picture, in order to prematurely terminate the task.

The finding, from the present study, that symptomatic schizophrenic patients score similarly to normal controls on an embedded figures test, contrasts markedly with results of earlier studies, which have consistently found groups of acute and
chronic schizophrenic patients (many of whom were presumably symptomatic) to perform significantly worse than both normals (e.g. Magaro & Vojtisek, 1971) and psychiatric controls (e.g. Vojtisek & Magaro, 1974) on Witkin et al.'s (1971) EFT. The present findings suggest that spatial IQ differences between groups are only weakly associated with differences in task performance, so from the arguments given in the Introduction, it is likely that a more important contributory factor to those earlier results, was the methodological feature of the EFT whereby the to-be-found simple figure is always removed during searching. This is likely to place high demands on working memory, with the subject being required to hold in mind a representation of the simple figure, so poor schizophrenic performance on the EFT may simply reflect impaired working memory in the disorder (e.g. Goldman-Rakic, 1987; Park & Holzman, 1992). From this argument, any studies attempting to reveal facilitatory effects on embedded figures tests in symptomatic patients, should always have the simple figure present during searching, as was done in the present experiment.

In summary, the present study has shown that schizophrenic patients with behavioural signs or paranoid symptoms score similarly to normal controls on an embedded figures test, while remitted patients score worse than normals. It has been argued that, while all the schizophrenic patients were medicated and had deficits in motivation and attention, the symptomatic patients alone showed facilitation on the task because of reduced awareness of context. It was therefore suggested that with adequate control for attention and motivation, scores of remitted patients would be brought to the level of normals, while the symptomatic patients would score better than normals. The single case with passivity symptoms also showed good embedded figures performance so, as predicted, it is possible that many types of delusion, as well as behavioural signs, are associated with some facilitation on embedded figures tests. This is clearly a preliminary result which should be replicated with larger subject numbers, but at this stage we have some evidence that symptomatic schizo-
phrenics show a similar pattern of performance to autistic people on embedded figures tasks. This adds weight to the argument that a common cognitive abnormality underlies the failure of these different subject groups to use contextual information. In Chapter 5, we saw that contextual deficits in schizophrenia are quite small effects (with patients showing a relative, rather than absolute, lack of awareness of context), so from that analysis the trends in the present study are even more notable.

On their own, the embedded figures results shed no light on the suggestion that the common cognitive deficit responsible for the lack of awareness of context in autism and schizophrenia, is one of impaired metarepresentation. However, the correlations between embedded figures accuracy and subjects’ performance on the ‘hints task’ and ‘picture pieces’ task in Chapter 5, provide some information on this issue for the case of schizophrenia. As discussed in the Introduction, the hints task tests the ability to represent others’ mental states, whereas normal performance on the embedded figures test (as conceptualised in Chapter 6) may involve the representation of own knowledge, with normals being continuously aware of the picture context (e.g. ‘I KNOW “the mug is part of a building”’) while searching for the hidden object (e.g. the mug). From findings earlier in this thesis suggesting that impairments in separate domains of metarepresentation are uncorrelated in schizophrenia (e.g. Chapter 4: own goals and others’ mental states; Chapter 5: own goals and knowledge and others’ mental states), we might therefore expect hints task performance to be uncorrelated with embedded figures accuracy for all schizophrenic groups. This was found to be the case in the present study, so the data are consistent with the theoretical framework developed throughout this thesis. Ozonoff et al. (1991a) found that scores in separate domains of metarepresentation (own goals and others’ mental states) were also uncorrelated for clinical controls, and indeed hints scores and embedded figures accuracy were unrelated for the control groups in the present study. It should be noted that some of the work on ‘field-dependence’ in normals suggests a relationship between social awareness and performance on
embedded figures tasks (e.g. Messick & Damarin, 1964; see Chapter 6), so from that body of work, we might have predicted a significant correlation between hints score and embedded figures accuracy for the normals in the present study. That no such correlation did occur (p was greater than 0.2, and Spearman's p was positive rather than negative), may reflect the fact that, with the relatively small number of subjects used in the present study, significant relationships between task scores may only be expected to appear if subjects show marked abnormalities on the tasks (giving rise to larger variance in scores). Most of the normals in the present study performed reasonably well on both the hints and embedded figures tasks, and the variance in scores of the normal group was smaller than that of all schizophrenic groups for both tasks, so by that argument it is not too surprising that correlations between task performance were non-significant for the normals.

It was argued in Chapter 5 that good performance on the 'isolation' condition of the 'picture pieces' task requires representation of own goals, whereas performance on the 'context' condition involves the representation of own goals and own knowledge. From the above arguments suggesting a lack of correlation, in schizophrenia, between impairments in separate domains of metarepresentation, we might therefore expect isolation scores in the picture pieces task to be unrelated to embedded figures accuracy for all schizophrenic groups. This was indeed found, so the results are again consistent with the theoretical framework developed here. As it has been argued that high scores by symptomatic schizophrenic patients on the embedded figures task reflect impairments in the representation of own knowledge, we would predict a significant negative correlation for them between embedded figures accuracy and context scores in the picture pieces task. This was found, but only for the behavioural signs patients (and only at p = 0.03); the absence of any correlation for the paranoid patients may reflect their low variance on the context condition of the picture pieces task (where $S^2$ was 21 times smaller than for the patients with behavioural signs; see Table 5.5). The finding of a predicted significant
relationship is encouraging, as it adds weight to earlier arguments that the context condition of the picture pieces task (which requires the production of context-appropriate responses using controlled processing), taps the same cognitive processes that are involved in the embedded figures task (where subjects must find hidden figures using controlled processing, and are hindered by greater awareness of context). The results do not prove the suggestion that these cognitive processes are metarepresentational, but they support the notion that a task involving the retrieval of information from long-term memory can be analysed theoretically in a similar way to one traditionally thought to operate at a more perceptual level.

Future work might investigate, more directly, whether metarepresentation is involved in the performance of embedded figures tests and the context condition of the picture pieces task, by correlating the scores of schizophrenic patients on these tasks with performance on other tests thought to require the representation of own knowledge (e.g. tests of source memory and episodic memory, or Done & Frith’s (1984) sentence-completion task). The theoretical framework developed here would predict that performance on these various tasks should intercorrelate for groups of symptomatic schizophrenic patients. The most stringent test of the metarepresentational model will again be found using the small subgroup of schizophrenic patients whose illness may have followed a neurodevelopmental course (e.g. Murray et al., 1992; see Chapter 3). These patients may be similar to autistic people in having impaired development of the metarepresentational system, so as with autistics (e.g. Ozonoff et al., 1991a), they might be expected to show significant correlations between performance in different metarepresentational domains. For them, therefore, scores on embedded figures tests, executive function tasks, and tests of real-life social competence, should be significantly related.
Performance on the Titchener illusion task

By analogy with Happé’s (1996) finding that autistic children were superior to controls in judging the relative sizes of the two central circles in adjacent Titchener stimuli, it was predicted that, because of a lack of awareness of context, schizophrenic patients with behavioural signs or delusions would show a similar advantage compared to normal and psychiatric controls. Schizophrenics in remission were expected to perform similarly to controls, and succumb to the illusion. The latter prediction, but not the former, was confirmed, with analysis of all five groups together showing that for both combined and single illusion stimuli, all groups succumbed strongly to the illusion, and did so to the same extent statistically. Inspection of the data showed that for all illusion stimuli, the schizophrenics with paranoid symptoms were slightly more likely than all other groups to correctly judge the relative sizes of the circles, while the patients with behavioural signs were slightly less likely than all other groups to accurately judge the stimuli. However, these effects only reached a trend level of significance when the three schizophrenic groups were analysed separately from the controls, and then the paranoids showed a trend at $p < 0.075$ to be more accurate than patients with behavioural signs.

These results contrast markedly with those from the embedded figures test, where several of the observed trends conformed to theoretical predictions. However, consideration of the nature of the Titchener illusion task enables us to explain the present findings without undermining the metarepresentational model developed throughout this thesis. Data analysis showed that all subjects found correct judgment of the control circles much easier than judgment of the circles in the illusion stimuli, so there was a psychometric problem of differences in difficulty between the control and illusion conditions. As the control condition was used to control for poor attention or a lack of motivation, this means that (as with the embedded figures test), these factors were incompletely controlled in the present task. Now, when presented
with the illusion stimuli, it seems likely that subjects had two options available to them in providing a response: they could either attempt to ignore the inducing context and try to accurately judge the relative sizes of the two central circles, or they could take the easier option of giving their 'first impression' of the relative sizes, making no attempt to ignore the inducing context. The first option is the route needed for accurate responding, and it was argued in Chapter 6 that, having adopted this route, people with impaired representation of their own knowledge are facilitated relative to those with intact metarepresentation, because when they attempt to compare the relative sizes of the circles (using controlled processing), they are unhindered by simultaneous awareness of the surrounding context. The second route will almost certainly lead to incorrect responses, as it involves perceptual processing (probably at an automatic level), with no attempt to ignore the context and critically judge the stimuli.

Data from the attention control measure of the embedded figures task (see Table 7.5) suggested that normal controls in this study had somewhat higher attention and motivation than the schizophrenic groups (the difference was significant at trend level relative to patients with behavioural signs), and although the Titchener illusion task was given to all subjects about one hour after the embedded figures task, we can assume that these differences were still present at this stage of the battery (and may well have been considerably increased). The performance of normal subjects in the illusion condition was quite poor, with many succumbing to the illusions, so if we assume that many of these relatively highly motivated people took the effortful first route to giving a response, and actively attempted to ignore the inducing contexts, then (within the theoretical framework developed here), their low scores can be seen as a reflection of intact metarepresentation, with performance being hindered by awareness of context. As the three schizophrenic groups had lower attention and motivation than normal controls, we can assume that these patients were more likely than normals to take the second (easier) route to a response, using only automatic
perceptual processing without any attempt to ignore the inducing contexts. As much evidence suggests that automatic processing is intact in schizophrenia (see Chapter 2 of this thesis), patients taking this route would be expected to succumb to the illusion. This argument can explain why patients with behavioural signs scored worse than all other subject groups on the illusion condition of the task: these people may have had the lowest motivation, so were most likely to adopt the automatic route to a response. Many of the patients in remission, or with paranoid symptoms, will have also taken this route (thereby succumbing to the illusion), but it is possible that those patients with higher motivation adopted the first route to a response, actively attempting to ignore context when judging the stimuli. We might then expect from the metarepresentational model that patients with paranoid symptoms (but not those in remission) who adopted this route, would show high task accuracy because of reduced awareness of surrounding context. This may explain the trend, within the schizophrenic sample, for the paranoid patients to score higher in the illusion condition than those with behavioural signs. Similarly, the excellent performance of the single case with passivity symptoms in the illusion condition, may have reflected his adoption of the effortful route to a response, together with reduced awareness of context due to impaired metarepresentation.

This analysis is clearly speculative, but it highlights how the problem of poor motivation in schizophrenia can confound results if inadequately controlled. It will be remembered that the attention and motivation control task in the embedded figures test was also too easy, and therefore inadequate. However, the results from that task conformed loosely to theoretical predictions, and that may be because subjects only had one possible route available to them in making a response (the route where they actively searched for the hidden object, and where intact metarepresentation was presumed to hinder performance); subjects who took the ‘easy option’ on that task and pointed indiscriminately to any part of the picture (thereby making a search error) soon learned that they were still required to continue searching for the full 2
minutes. These arguments suggest that all future studies of illusion susceptibility in schizophrenia must control very carefully for motivation. This can only be done by having a control task which is as difficult as the illusion task, so for the case of the Titchener illusion, one possibility might be to use difficult control stimuli (where the two circles are very similar in size), and illusion stimuli where the actual difference between the two central circles is comparatively large. These two conditions would then need to be matched psychometrically using a group of normal volunteers. It is interesting to note that the only study to date to have found schizophrenic patients to be less susceptible than controls to illusions (Capozzoli & Marsh, 1994), used a relatively homogeneous group of acute paranoid patients. These may have been subjects for whom the facilitatory effect of reduced awareness of context was not offset by motivational problems. If Capozzoli and Marsh had used a group of patients with mainly behavioural signs, problems of poor motivation may well have resulted in the patients succumbing to the illusion.

Finally, the absence of any correlation between performance of schizophrenic patients on the embedded figures and illusions tasks, is consistent with the above suggestion that all of the patients adopted a controlled processing strategy on the embedded figures test (because the task demands required it), while only some adopted such a strategy on the illusions task, the remainder judging the illusions using automatic, perceptual processing. These arguments would predict that correlations between performance on the context condition of the picture pieces task, and accuracy on the illusions task, would be non-significant for all schizophrenic groups, as the former is thought to reflect the ability to represent own knowledge, whereas the latter will only reflect that ability for subjects who attempted to actively judge the stimuli using controlled processing. The correlations were indeed non-significant for remitted schizophrenics and patients with behavioural signs, but for the paranoid patients there was a positive correlation between scores on the two tasks. This was unexpected, but a possible explanation is that the paranoid patients who scored...
poorly on the context picture pieces condition (and who therefore had more impaired representation of their own knowledge), were those patients with the most severe symptoms (e.g. more extensive delusions). Because of their greater illness severity, these patients may have been less motivated to try to judge the illusion stimuli by actively ignoring the context, choosing instead the 'easy option' of automatic perceptual processing. As a result, they would be highly likely to succumb to the illusions, as found. The theoretical framework developed in this thesis would predict that if these patients had chosen to process the stimuli in a controlled way, they would have judged them more accurately than controls.

7.5 Conclusions

The present study investigated the prediction that, because of reduced awareness of context, schizophrenic patients with behavioural signs or delusions would perform similarly to autistic people on embedded figures and illusions tasks, being more accurate than controls both in finding hidden figures and in accurately judging the relative sizes of parts of illusion stimuli. Results from the embedded figures test provided some evidence in favour of the predictions, because patients in remission scored significantly worse than normals, while patients with delusions or behavioural signs scored similarly to normals. It was argued that with adequate controls for attention and medication effects, scores of remitted patients would have been similar to those of normals, while performance of the symptomatic patients would have been better than that of normals. Results from the illusion task did not conform to predictions, as all subjects succumbed strongly to the illusions. However, there was a trend for paranoid schizophrenics to give more accurate judgments of the stimuli than patients with behavioural signs. It was suggested that the results could be explained by assuming that most of the behavioural signs patients adopted an automatic route to a response, making no attempt to accurately judge the stimuli because of poor motivation. In contrast, while many of the paranoid patients may have also taken this
route, the more highly motivated members of that group may have attempted to accurately judge the stimuli; these people then gave correct responses because of their reduced awareness of context.

Correlations between performance on the embedded figures test and scores on the hints task and picture pieces task (see Chapter 5), were consistent with the metarepresentational analyses of these tasks presented earlier. Thus, in line with earlier findings that impairments in separate domains of metarepresentation are uncorrelated in schizophrenia (see Chapters 4 and 5), performance of patients on the embedded figures test (thought to reflect representation of own knowledge) was unrelated to performance on the hints task or the isolation condition of the picture pieces task. For the schizophrenics with behavioural signs, performance on the context condition of the picture pieces task was inversely related to accuracy on the embedded figures test, a finding consistent with analysis of both of these tasks in terms of similar cognitive processes.
CHAPTER EIGHT

CONCLUSIONS

This thesis has explored in some detail Frith’s (1992) neuropsychological model of impaired mental state representation (metarepresentation) in schizophrenia. A broad range of literature covering the psychology and neuropsychology of schizophrenia was reviewed, and attempts were made to find common themes, and links to Frith’s model. It was concluded from the review that many schizophrenic patients have deficits in conscious, controlled processing, while automatic processing outside conscious awareness is relatively intact. It was noted that this general conclusion is compatible with Frith’s model, in that metarepresentation operates in conscious awareness, and may underlie many of the ‘higher’ cognitive functions such as executive function, episodic memory and theory of mind ability.

The first empirical section of the thesis investigated the ability of schizophrenic people to attribute mental states to other people (theory of mind), and their ability to represent their own goals (as measured by executive function). The results were broadly in line with predictions from Frith’s model, although it seems that first-order theory of mind may be intact in schizophrenia; this suggests that any ‘mentalising’ deficits in the disorder may be less pronounced than in autism. In later sections of the thesis, it was suggested that Frith’s model may be extended to include impaired representation of own knowledge in schizophrenia, and that, as well as explaining
poor episodic memory in the disorder, this may account for the impaired use of context by many schizophrenic patients. It was speculated that such a lack of awareness of context may underlie the phenomenon of ‘weak central coherence’ in autism, and that schizophrenic patients might also be expected to show weak coherence on embedded figures and illusions tasks. These predictions were investigated empirically, and the findings integrated into the metarepresentational account of schizophrenia.

In the remainder of this chapter, each of the main themes of the thesis will be briefly discussed, and the conclusions brought together. Suggestions for future work will be made within each section.

**Controlled and automatic processing**

From a review of psychological research in schizophrenia (see Chapter 2), it was concluded that patients with many different signs and symptoms have deficits in controlled processing (as defined by Shiffrin & Schneider, 1977), while automatic processing is relatively intact. Thus, in the domain of memory, for example, patients (especially those with behavioural signs) are often impaired on resource-demanding tasks requiring the generation of responses in conscious awareness (e.g. tests of explicit memory such as free recall), whereas performance on tests of implicit memory (e.g. word-stem completion) is intact. A particular controlled processing deficit often noted in schizophrenia, is impairment in the use of contextual information in conscious awareness; this deficit has been associated with the presence of behavioural signs, and also with delusions (where it may underlie the abnormal reasoning style of many deluded patients). In contrast, a number of studies suggest that the *automatic* influence of context in schizophrenia is relatively intact (contrary to Hemsley’s (1987; 1994) model of psychosis). It should be noted that the issue of controlled *versus* automatic processing deficit in schizophrenia is still a controversial...
one, and future research should attempt to conclusively resolve the relative impairments in these two domains by giving both automatic and controlled processing tasks to the same patient sample within one study. Importantly, any such study should attempt to match the two tasks on difficulty using normal volunteers, so that in the domain of memory, for example, a free recall task could be matched in difficulty with a word-stem completion task by manipulating the number of to-be-remembered words in the recall task.

Frith's (1987; 1992) neuropsychological model of schizophrenia

Frith’s model suggests that many psychotic signs and symptoms reflect impaired representation of own or others’ mental states (metarepresentation). As metarepresentation is a crucial component of self-awareness, Frith’s theory is consistent with the deficits in controlled processing discussed above. According to Frith, some negative and positive behavioural signs (e.g. avolition and inappropriate speech) reflect, respectively, impairments in the generation of willed action and impairments in the inhibition of inappropriate action; in terms of metarepresentation, these both correspond to deficits in the representation of own goals. Passivity symptoms such as delusions of control, on the other hand, are thought to reflect deficient self-monitoring of willed action, which can be thought of as impaired representation of own intentions. Finally, positive symptoms involving the patient’s relationship with other people (e.g. paranoid delusions), and behavioural signs such as social withdrawal or affective blunting, may reflect impaired representation of others’ mental states (i.e. impaired theory of mind). It is presumed that deficits in these various metarepresentational domains can occur independently so that, for example, patients with paranoid delusions and no passivity symptoms have a selective theory of mind deficit, with intact representation of their own intentions. Moreover, the nature of the symptomatology reflects the severity of metarepresentational deficits so that, for example, social withdrawal reflects an inability to
represent others’ mental states, while paranoid delusions reflect incorrect attribution of others’ mental states. Patients in remission (i.e. those without symptoms or signs) are presumed to have a fully intact ability to metarepresent. At the neuropathological level, impaired metarepresentation may reflect a functional disconnection between frontal and posterior brain regions, with the extent of disconnection being related to the severity of metarepresentational deficit (Frith, 1994). This is consistent with a large number of imaging studies which have suggested impaired fronto-temporal (e.g. Frith et al., 1995) and/or fronto-striatal (e.g. Buchsbaum et al., 1992) functional disconnection to be a common feature of the schizophrenic brain.

Frith & Frith (1991) compared the case of schizophrenia with that of early childhood autism, where impaired representation of own and others’ mental states has also been invoked to explain the characteristic impairments in socialization, communication and pretend play (e.g. U. Frith et al., 1991). Frith & Frith (1991) likened these features of autism to the behavioural signs of schizophrenia, and suggested that both cases reflect an absence of metarepresentation. Thus, for autistic people, the ability to represent mental states has never developed properly, whereas for schizophrenics with behavioural signs, a developed cognitive system has completely failed to function as a result of the illness. Frith and Frith pointed out that such failures in a developed system, if intermittent or only partial, would manifest as positive psychotic symptoms (with, for example, the patient representing others’ mental states, but doing so incorrectly); the absence of positive symptoms in autism is therefore a reflection of the fact that such people have never had a fully-developed metarepresentational capacity.

**Representation of other peoples’ mental states (theory of mind) in schizophrenia**

Frith’s model would predict theory of mind impairments in schizophrenic patients with behavioural signs, and to a lesser extent in those with paranoid
symptoms and no behavioural signs. From a review of relevant studies, it was concluded in Chapter 3 that there is strong evidence for this, but it was noted that two studies using standard false belief tasks have produced conflicting results. Thus, Frith & Corcoran (1996) found that patients with behavioural signs or paranoid symptoms were impaired on both first- and second-order tasks, whereas Bowler (1992) showed intact performance by behavioural signs patients on both types of task. The first empirical study of this thesis enabled a reconciliation of these findings: Schizophrenic patients were given first- and second-order false belief tasks, using Bowler’s methodology of enacting the stories with models and providing memory control questions throughout. In addition, subjects were given equally difficult first- and second-order ‘non-mental representation’ tasks, in an attempt to reveal specific deficits in theory of mind. At the first-order level, Bowler’s findings were replicated and extended, as it was shown that all groups of symptomatic schizophrenic patients scored as well as remitted patients and controls on all tasks. It was therefore suggested that Frith & Corcoran’s (1996) first-order theory of mind results were artefacts of poor control for attention and memory in that study. At the second-order level, the present work replicated Frith and Corcoran’s findings, showing theory of mind deficits in patients with behavioural signs, and to a lesser extent in those with paranoid symptoms. In addition, the matched-tasks methodology showed that these deficits were specific to the theory of mind domain, and did not reflect more general problems in understanding any type of representation. It was argued that Bowler’s (1992) ‘second-order’ theory of mind stories could have been solved with only first-order reasoning (hence the good performance of his patients), and that Frith & Corcoran (1996) revealed real second-order deficits despite their poor control for memory and attention. On the basis of all these findings, it was suggested that Frith’s (1992) model should be modified to allow for intact first-order theory of mind in most cases of schizophrenia. Thus, even patients with behavioural signs usually have more subtle deficits than autistics (who often fail first-order tasks); this implies that the behavioural signs patients still have some residual theory of mind skills, despite
severe impairment of their once fully-developed metarepresentational system.

Murray et al. (1992) have suggested that a small subgroup of patients with predominantly negative signs, has a ‘neurodevelopmental’ form of schizophrenia. Within the framework of Frith’s model, these may be people who had an initial autistic presentation, but who then went on to show an early-onset schizophrenic illness. They can be assumed to have experienced impaired development of the metarepresentational system (like autistics), so we might predict that they would be the only schizophrenic patients to show some theory of mind deficits at the first-order level. This should be investigated in future research, using a carefully selected group of patients with childhood-onset schizophrenia and a predominance of negative signs. These arguments would predict that early onset and negative symptomatology should be strong predictors of theory of mind deficit in schizophrenia, and in this regard it is notable that logistic regression analysis on theory of mind scores from the present work, found scores for negative signs to be the strongest predictor of deficit. Age of onset was not a significant predictor, but this may reflect the fact that no patients with childhood-onset were included in the present investigations, the earliest age of onset being 16 years.

In a later study from the present thesis, schizophrenic patients were given Corcoran et al.’s (1995) ‘hints task’. It was argued that this tests theory of mind ability at the second-order level, and in line with the above discussions, Corcoran et al.’s findings were substantially replicated, with patients with behavioural signs (and to a lesser extent, those with paranoid symptoms), showing worse performance than controls and schizophrenics in remission. Future studies of theory of mind in schizophrenia might employ the stories developed by Fletcher et al. (1995) for use with normals, as the ‘physical’ control stories from that study were extremely well matched with the theory of mind stories in terms of length, thematic content, and difficulty. In addition, both the physical and theory of mind stories in Fletcher et al.’s
work required an inference to be made, whereas it could be argued that the ‘non-mental representation’ control tasks from the first study in this thesis differed from the theory of mind tasks in not requiring inferential reasoning. A study using Fletcher et al.’s stories with schizophrenic patients would therefore enable us to rule out general reasoning problems as an explanation of apparent theory of mind deficits in the disorder. Finally, the theory of mind ability of remitted schizophrenic patients, and those with only passivity symptoms, should be further investigated in future research. Frith’s model suggests that these groups of patients have a fully intact theory of mind, and while this has been supported by most studies to date (including those in the present thesis), it would be useful to give the complex, naturalistic theory of mind stories developed by Happé (1994b), to these groups of schizophrenics. If Frith’s model is correct, these patients should perform as well as normal controls even on such difficult tests of mentalising ability.

Executive function and Frith’s model

Frith’s (1992) model also suggests that executive dysfunction in schizophrenia reflects impaired representation of own goals (and to a smaller extent, own intentions), so it was predicted in the present thesis that patients with behavioural signs (and to a lesser extent, those with only passivity symptoms), would show executive deficits on a simple reversal task. Patients with paranoid symptoms and no behavioural signs, and those in remission, were predicted to be unimpaired on the task because of intact representation of own goals and intentions. Most of these predictions were supported; thus, paranoid and remitted patients performed well on the task, while the group with behavioural signs showed significantly more perseverative errors than controls. Logistic regression analysis confirmed that scores for negative signs were a strong predictor of executive deficit. Scores for passivity symptoms had some ability to predict executive dysfunction in the logistic regression, but this predictive power was very small, and the single case showing only passivity...
symptoms performed well on the reversal task. It was therefore suggested that the
task used here may have been too insensitive to detect the subtle executive deficits
associated with passivity symptoms, so future work should use a more difficult task
(perhaps one involving successive reversals), in an attempt to reveal these impair­
ments.

Executive function and theory of mind

Autistic people show a strong correlation between executive and theory of
mind deficits (e.g. Ozonoff et al., 1991a), and it was argued that, within Frith’s
theoretical framework, this is consistent with severe impairments in a single cognitive
system underlying all forms of metarepresentation. From Frith & Frith’s (1991)
suggestion that schizophrenic people with behavioural signs are similar to autistics
in having severely impaired metarepresentation (i.e. deficient representation of both
own and others’ mental states), it was therefore predicted that the behavioural signs
patients in the present investigations would also show a correlation between theory
of mind and executive abilities. No such correlation was found, and (as with earlier
discussions of intact first-order theory of mind in schizophrenia), this was explained
in terms of the different ages of onset of autism and most cases of schizophrenia.

Thus, autism may involve impaired development of a single cognitive system
necessary for all types of metarepresentation, leading to an association between
executive and theory of mind deficits. In contrast, most cases of schizophrenia
involve late-occurring impairments in a fully-developed metarepresentational system.

As we have seen, impairments in different domains of mental state representation can
occur independently in schizophrenia (e.g. paranoid patients have problems in
representing others’ mental states, whilst having intact representation of their own
goals), so while patients with behavioural signs have impaired representation of both
own goals and others’ mental states, these deficits probably occurred independently
of one another (hence the lack of correlation between executive function and theory
of mind in that group). Such a model would predict that the small ‘neurodevelopmental’ subgroup of schizophrenics with predominantly negative signs (e.g. Murray et al., 1992) would show a significant correlation between theory of mind and executive ability, because of impairments in the development of their metarepresentational system; this prediction should be investigated in future work.

‘Poor insight’ in schizophrenia

It was argued that Frith’s (1992) model might be extended to explain the concept of ‘poor insight’ (i.e. lack of self-awareness) in schizophrenia, so that patients with impaired representation of their own mental states (e.g. goals) may have the worst insight. However, a review of studies suggested that there are no linear relationships between, for example, insight and executive function. Startup (1996) recently found a quadratic relationship between these two variables, and he attempted to explain this in terms of a two-factor model incorporating ‘motivational’ as well as ‘cognitive deficit’ accounts of poor insight. With this in mind, logistic regression was used in the first study of the present thesis to test whether (insight)^ predicted executive function. It was found not to do so, but this may well have been because the PSE insight scale used here was rather insensitive. A future regression investigation of Startup’s model should, therefore, test patients on a sensitive insight scale (e.g. David’s (1990) ‘schedule for the assessment of insight’), as well as a difficult (and therefore discriminating) executive task.

Awareness of context in retrieval from memory

It was suggested that Frith’s (1992) neuropsychological model might also be extended to include the monitoring of knowledge stored in long-term memory (LTM). Such representation of knowledge, with the subject knowing how or that he knows something, may underlie normal source memory and episodic memory, so an impair-
ment in this domain of metarepresentation may explain the particular deficits shown by many schizophrenic people in these aspects of memory (e.g. Frith et al., 1991c; Tamlyn et al., 1992). It was pointed out that intact source memory and episodic memory involve the representation of information together with its associated contextual details, so that impairments in the representation of own knowledge in schizophrenia may also explain the poor performance of patients on tasks requiring the retrieval of context-appropriate information from LTM (e.g. Done & Frith, 1984). By this argument, a normal subject asked, for example, to name an ambiguous curved object in a beach scene (e.g. in the ‘context’ condition of Happé’s ‘picture pieces’ task), will produce the optimal response of “shell” because of awareness of the picture context while retrieving information from semantic memory. Most studies in schizophrenia which can be conceptualised as requiring representation of own knowledge, suggest that impairments are particularly associated with the presence of behavioural signs. In the second study of the present thesis, therefore, it was predicted that patients with behavioural signs would be differentially impaired relative to controls in the ‘context’ condition of the picture pieces task relative to the ‘isolation’ condition (requiring the appropriate naming of objects in the absence of context). The association between contextual ability and metarepresentation was tested by correlating scores on the picture pieces task with those on the hints task, for each subject group in turn.

Contrary to predictions, results showed no statistically significant interaction between performance in the isolation and context conditions of the task. However, examination of group differences within conditions, and of differences for each group between conditions, gave some evidence for small deficits in the context condition for schizophrenics with behavioural signs (and to a lesser extent, for those with delusions). It is important to emphasise, however, that all groups showed some benefit of context, so any context deficits in schizophrenia are relative rather than absolute. It was noted that the context and isolation conditions of the picture pieces
task were not matched on difficulty for normal controls, so future investigations should use matched tasks to avoid psychometric problems in interpretation of the data. Such a paradigm would necessarily require the naming of different objects in context from those in isolation, and one possibility would be to use the same isolation objects as used in the present work, but harder context scenes to those used here. In addition, following Reich & Cutting’s (1982) finding that, unlike controls, schizophrenic patients tended to ignore the larger context or ‘theme’ when describing a picture (giving instead descriptions of details), it would be useful to investigate whether this effect correlates with the tendency to give context-inappropriate names to objects in the picture pieces task.

For all subject groups, correlations between performance on the hints task, and scores in the isolation and context conditions of the picture pieces task, were non-significant. For the two groups who showed deficits on both tasks (i.e. schizophrenics with paranoid symptoms and those with behavioural signs), it was argued that this was consistent with the earlier finding of no significant correlation between theory of mind and executive deficits for patients with behavioural signs. Thus, if performance in the context condition of the picture pieces task does require the representation of own knowledge, then impairments in this metarepresentational domain occur independently of theory of mind impairments in most schizophrenic patients. This fits with the notion that most cases of schizophrenia involve independent, late-occurring deficits in a fully-developed metarepresentational system. One prediction from this model is that the small ‘neurodevelopmental’ subgroup of schizophrenics with mainly negative signs (e.g. Murray et al., 1992) would show a significant correlation between theory of mind deficits and low scores in the context condition of the picture pieces task. In these patients, the ability to represent own knowledge and others’ mental states may be linked because of impaired development of the metarepresentational system. This prediction should be investigated in future research. Autism also provides a test case for the above model, as autistic people would similarly be
expected to show a correlation between performance on tasks requiring theory of mind or the use of context in retrieval from LTM. Finally, if it is true that the same cognitive process (representation of own knowledge) underlies source memory, episodic memory, and the retrieval of context-appropriate information from LTM, then deficits in these three areas shown by schizophrenic patients with behavioural signs, should intercorrelate. This again should be explored in future work.

‘Weak central coherence’ in autism and schizophrenia

In Chapter 6 of the thesis, the case for ‘weak central coherence’ in autism (e.g. U. Frith, 1989) was reviewed, and it was suggested that this phenomenon may be explicable in terms of impaired metarepresentation. Thus, it was noted that one of the empirical findings taken as support for the weak central coherence account, is the poor ability of autistic people to use sentence context to disambiguate homographs (e.g. Happé, 1997). By analogy with the above discussions, this task may require the subject to be aware of context while retrieving information from LTM, a process which may involve representation of own knowledge. It was argued that superior autistic performance on other tests of weak central coherence (e.g. embedded figures, Block Design, and illusions tasks), may also be explained in terms of impaired metarepresentation, as in all cases performance is facilitated if a subject lacks awareness of context while attending to particular stimulus features relevant to the task. This account is clearly speculative, but it makes specific predictions, so is empirically falsifiable. Thus, if the homograph task does require representation of own knowledge, then autistic task performance should correlate with scores on other tasks thought to tap this metarepresentational domain (e.g. tests of source memory or episodic memory). Similarly, low scores by autistic people on the homograph task should correlate with high scores on embedded figures, Block Design and illusions tasks. From results suggesting that autistic impairments in separate metarepresentational domains are intercorrelated (e.g. Bennetto et al., 1996; Ozonoff et al., 1991a),
we might also expect autistic performance on tests of executive function or real-life social competence to be related to scores on the various tests of central coherence. These predictions should be investigated in future research.

In the second part of Chapter 6, the relevant schizophrenia research was reviewed, and it was suggested that if the same cognitive deficit underlies the failure of autistic and schizophrenic people to use context, then symptomatic schizophrenic patients (especially those with behavioural signs, but to a lesser extent those with delusions) should show similar performance to autistics on tests of central coherence. There is some evidence for this in the literature, but performance on embedded figures tests has generally been found to be poor in schizophrenia. This issue was explored in the final study of the thesis, and it was found that remitted schizophrenics performed significantly worse than normal controls on a new embedded figures task, while symptomatic schizophrenics scored similarly to normals. It was argued that, as predicted, this reflected a symptom-related facilitation on the task, so that with adequate control for poor motivation and attention, the remitted patients would have performed similarly to normals, while those with symptoms would have scored better than normals. This prediction should be investigated in future work using a difficult attention control condition (e.g. a ‘finger tapping’ test), psychometrically matched in difficulty with the embedded condition. It was suggested that earlier studies showing poor schizophrenic performance on Witkin et al.’s (1971) EFT were confounded by impaired working memory in the patients (as the task requires subjects to remember the to-be-found figure while searching). Future studies should therefore investigate the prediction that schizophrenics would score well on the EFT if the picture of the to-be-found figure was present during searching.

The final study of the thesis also investigated the susceptibility of schizophrenic patients to the Titchener illusion. It was predicted that because of reduced awareness of context, symptomatic patients would be more likely than remitted
patients or controls to accurately judge the relative sizes of parts of the illusion stimuli. Little evidence was found for this, although there was a trend for patients with paranoid symptoms to show the highest task accuracy; schizophrenics with behavioural signs performed worse than all other groups on the task. It was argued that these unexpected results could be explained without abandoning a metarepresentational account of the task, if it was accepted that there were two possible routes to a response, performance on only one of which (the more effortful route) was facilitated by impaired metarepresentation. This analysis exposes the vulnerability of the task to motivational problems in the subjects, so any future studies of illusion susceptibility in schizophrenia, should use an attention and motivation control task matched in difficulty with the illusion condition. Such rigorous methodology would be much more likely than the present work to reveal the predicted task superiority of symptomatic patients.

The results from the embedded figures task in the present work give some support for the idea that a common cognitive deficit underlies the failure of autistic and symptomatic schizophrenic people to use context. Evidence that this deficit is one of impaired metarepresentation (representation of own knowledge), was sought from correlations between embedded figures accuracy and performance on the ‘hints task’ (Corcoran et al., 1995). In line with earlier suggestions of independent impairments in separate domains of metarepresentation in most cases of schizophrenia, these correlations were non-significant for all schizophrenic groups. Future work should investigate the same correlation using a sample of patients whose illness may have followed the ‘neurodevelopmental’ course proposed by Murray et al. (1992). This group would be expected to show a significant correlation between performance on hints and embedded figures tasks, because (as with autism) impaired development of the metarepresentational system may have linked deficits across separate metarepresentational domains in these patients.
Finally, it was predicted in the present work that for symptomatic schizophrenics, good performance on the context condition of the ‘picture pieces’ task would be inversely correlated with embedded figures accuracy, as it is proposed here that both tasks tap the same cognitive process. This was indeed found for the schizophrenics with behavioural signs. Direct evidence that this common process involves the representation of own knowledge, should be sought in future studies by correlating embedded figures and ‘picture pieces’ context scores for symptomatic schizophrenics, with their performance on other tasks thought to involve this metarepresentational domain (e.g. tests of source memory or episodic memory, Done & Frith’s (1984) sentence-completion task, or U. Frith & Snowling’s (1983) homograph task); these correlations should all be significant. The most rigorous test of the model would again use a group of patients whose illness may have followed a neurodevelopmental course. They would be expected to show correlations between performance on tasks from different metarepresentational domains so that, for example, ‘picture pieces’ context scores would correlate with executive function and real-life mentalising ability.

**Impaired metarepresentation in schizophrenia**

We have seen that a metarepresentational account of schizophrenia can explain many of the signs and symptoms of the disorder, and many of the empirical findings from the psychological literature. A common theme of the present work has been that metarepresentational deficits in schizophrenia are often quite subtle, and perhaps less pronounced than in autism; this suggests that future tests of the theoretical ideas in this thesis might be easiest to carry out using groups of autistic subjects. Only when the proposed links have been revealed in that group, should similar experiments be attempted in schizophrenia.
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inhibition in schizophrenia. *Schizophrenia Research, 20,* 91-103.


APPENDICES
Appendix I: Patient information sheet and consent form for the study in Chapter 4

PATIENT INFORMATION SHEET

The tests which you are going to do investigate how you understand what other people mean or believe in situations when the meaning is not immediately obvious. We are interested in investigating this type of ability because we believe it could have something to do with the difficulties you may sometimes have. We also need to know what sort of problems you have been having recently, and we need some idea of how you do on other simple tests of reading and comprehension.

Our results are only for the purposes of this research, and your answers will not influence your treatment in any way. You can withdraw from the research at any time, so if you wish to do so, please tell us.

All answers that you give to Graham Pickup (the researcher) will be in confidence. The only other people who will have access to your responses are your Consultant and the project supervisor (Professor C. Frith). If you require any further information, or have any questions, Mr. Pickup or your Consultant will do their best to provide it.
PATIENT CONSENT FORM

A study of ‘theory of mind’ by Graham Pickup, University College London, Gower Street, London WC1E 6BT. Tel: 0171 380 7777 extension 5412.

To be signed by the patient:

I have read the information sheet concerning this study and I understand what will be required of me if I take part in the study.

My questions concerning the study have been answered by Graham Pickup.

I understand that at any time I may withdraw from this study without giving a reason, and without affecting my normal care and management.

I agree to take part in this study.

Signed ........................................ Date ..................................

Consultant ........................................

Signature of investigator ........................................
Appendix II: Table for the parametric ANOVA analysis of group differences in current IQ, from Chapter 4

<table>
<thead>
<tr>
<th>Source of variance</th>
<th>df</th>
<th>Mean squares</th>
<th>F ratio</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Groups</td>
<td>4</td>
<td>955.86</td>
<td>7.31</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>Within Groups</td>
<td>88</td>
<td>130.77</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>92</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

450
Appendix III: The ‘hints task’ stories and scoring protocol used in Chapter 5

The following stories were read out to subjects (and simultaneously presented on cards, so that subjects could follow the story themselves). The stories are the same as those used by Corcoran et al. (1995).

**Story 1**

George arrives in Angela’s office after a long and hot journey down the motorway. Angela immediately begins to talk about some business ideas. George interrupts Angela, saying: “My, my! It was a long, hot journey down that motorway!”

Question: What does George really mean when he says this?

If subject fails to respond, or gives the wrong answer, add: George goes on to say, “I’m parched!”

Question: What does George want Angela to do?

**Scoring protocol**

The correct answer involves some indication that George wants a drink after his journey. It is not adequate to say things such as, “he’s fed up”, “he’s tired”, “he wants a break”, “he needs to have a rest”, “he doesn’t want to talk about business ideas”.

**Story 2**

Melissa goes to the bathroom for a shower. Anne has just had a bath. Melissa notices the bath is dirty so she calls upstairs to Anne: “ Couldn’t you find the Ajax, Anne?”
Question: What does Melissa really mean when she says this?

If subject fails to respond, or gives the wrong answer, add: Melissa goes on to say, “You’re very lazy sometimes, Anne!”

Question: What does Melissa want Anne to do?

*Scoring protocol*

The correct answer refers to the fact that Melissa wants Anne to clean the bath. Acceptable answers also include, “why didn’t you clean it?”, “can’t you clean the bath after you?”, “couldn’t you have cleaned the bath?”. Unacceptable answers include, “the bath needs cleaning”, “she didn’t clean the bath”, “are you feeling lazy tonight?”, as these do not explicitly refer to the fact that Anne is required to clean the bath.

*Story 3*

Gordon goes to the supermarket with his mum. They arrive at the sweetie aisle. Gordon says, “Cor! Those treacle toffees look delicious!”

Question: What does Gordon really mean when he says this?

If subject fails to respond, or gives the wrong answer, add: Gordon goes on to say, “I’m hungry, mum”.

Question: What does Gordon want his mum to do?

*Scoring protocol*

The correct answer involves Gordon indicating that he wants his mother to
buy some sweets. Acceptable responses include, “Gordon wants some sweets”, “he wants some”. It is unacceptable to say, “He needs something sweet in his diet”.

**Story 4**

Paul has to go to an interview and he’s running late. While he is cleaning his shoes, he says to his wife, Jane; “I want to wear that blue shirt but it’s very creased.”

Question: What does Paul really mean when he says this?

If subject fails to respond or gives the wrong answer, add: Paul goes on to say, “It’s in the ironing basket”.

Question: What does Paul want Jane to do?

**Scoring protocol**

The correct answer is that Paul wants Jane to iron his shirt. It is acceptable for the subject to say, “he wants it ironed”. Unacceptable responses include, “Get the iron out”, “he means his clothes should look neat for the interview”, “his shirt hasn’t been ironed”.

**Story 5**

Lucy is broke but she wants to go out in the evening. She knows that David has just been paid. She says to him; “I’m flat broke. Things are so expensive these days.”

Question: What does Lucy really mean when she says this?

If subject fails to respond, or gives the wrong answer, add: Lucy goes on to say; “Oh
well, I suppose I’ll have to miss my night out.”

Question: What does Lucy want David to do?

**Scoring protocol**

The correct response is that Lucy wants David to give/lend her some money. An acceptable response would also be, “She wants him to pay for the evening out”. Unacceptable responses include, “Would you take me out?”, “she hasn’t got enough money”, as these don’t explicitly refer to David giving or lending Lucy money.

**Story 6**

Donald wants to run a project at work but Richard, his boss, has asked someone else to run it. Donald says; “What a pity, I’m not too busy at the moment.”

Question: What does Donald really mean when he says this?

If subject fails to respond, or gives the wrong answer, add: Donald goes on to say; “That project is right up my street.”

Question: What does Donald want Richard to do?

**Scoring protocol**

The model answer is that Donald wants Richard to give the project to him to run. Acceptable answers include, “Donald would rather run the project”, “he wants it himself”, “he wants to do it/run it”, “why don’t you get me to do it?”, “he means that he should be allowed to run it”. Unacceptable answers are those which don’t imply that Donald wants the project or is pushing strongly for it, e.g. “He could do with the work”, “he means he could do it”, “he’s got more time to do it”, “he thinks
he can run it better”, or those which imply that Donald would have liked the project, but it’s now too late, e.g. “He means he could have done it”, “he would have liked to have run it”, “he wanted to run it”, “he wishes he was the one doing it”, “why didn’t you ask me to do it?”.

Story 7

Rebecca’s birthday is approaching. She says to her dad; “I love animals, especially dogs.”

Question: What does Rebecca really mean when she says this?

If subject fails to respond, or gives the wrong answer, add: “Rebecca goes on to say; “Will the pet shop be open on my birthday, dad?”

Question: What does Rebecca want her dad to do?

Scoring protocol

The correct answer is that Rebecca wants her dad to buy her a dog for her birthday. It is also acceptable for the subject to say, “she wants a dog”.

Story 8

Betty and Michael moved into their new house a week ago. Betty has been unpacking some ornaments. She says to Michael; “Have you unpacked those shelves we bought, Michael?”

Question: What does Betty really mean when she says this?
If subject fails to respond, or gives the wrong answer, add: “Betty goes on to say; “If you want something doing you have to do it yourself!”

Question: What does Betty want Michael to do?

Scoring protocol
The correct answer must refer to Betty wanting Michael to put the shelves up. Acceptable alternatives include, “assemble the shelves” or “fix the shelves”. Unacceptable answers include, “unpack the shelves”, “get the shelves together”, “put the ornaments on the shelves”, or “she wants somewhere to put her ornaments”.

Story 9

Jessica and Max are playing with a train set. Jessica has the blue train and Max has the red one. Jessica says to Max; “I don’t like this train.”

Question: What does Jessica really mean when she says this?

If subject fails to respond, or gives the wrong answer, add: “Jessica goes on to say; “Red is my favourite colour.”

Question: What does Jessica want Max to do?

Scoring protocol
The correct answer is that Jessica wants Max’s red train to play with. Acceptable alternatives include, “I want your train”, “Do you want to swap trains?”. Unacceptable responses are, “She doesn’t like the colour of her train”, or “She prefers Max’s train”.

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Story 10

Patsy is just getting off the train with three heavy cases. John is standing behind her. Patsy says to John; “Gosh! These cases are a nuisance.”

Question: What does Patsy really mean when she says this?

If subject fails to respond, or gives the wrong answer, add: “Patsy goes on to say; “I don’t know if I can manage all three.”

Question: What does Patsy want John to do?

Scoring protocol

The correct answer is that Patsy wants John to carry the cases for her. Alternative acceptable responses are, “she wants some help”, “John should help her”, “she’s asking him to give her a hand”.

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Appendix IV: The ‘picture pieces’ task & scoring protocol from Chapter 5

Figures IV.1 to IV.4 show the context scenes A and C and B and D, as used in the task. The to-be-named objects are numbered for reference in these scenes, although it should be noted that none of these numbers were present when the task was given. The isolation condition involved presentation of each of the numbered objects stuck onto a card, without any of the surrounding context.

For each of the objects *in isolation*, all responses given by subjects are shown below, with the relevant ‘suitability index’ (the sum of judgments of 3 independent raters on a scale of 0 to 4) in parentheses.

<table>
<thead>
<tr>
<th>Object 1</th>
<th>Object 2</th>
<th>Object 3</th>
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<td>ball (12)</td>
<td>lady (11)</td>
<td>plant (10)</td>
</tr>
<tr>
<td>ring (11)</td>
<td>face (12)</td>
<td>spider (11)</td>
</tr>
<tr>
<td>circle (10)</td>
<td>woman (11)</td>
<td>candle &amp; fireworks (6)</td>
</tr>
<tr>
<td>spot (8)</td>
<td>face of girl (11)</td>
<td>spider smoking cigarette (5)</td>
</tr>
<tr>
<td>hole (7)</td>
<td>boy’s face (6)</td>
<td>palm tree (9)</td>
</tr>
<tr>
<td>orange (6)</td>
<td>person (10)</td>
<td>flower (9)</td>
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<td>pea (6)</td>
<td>haircut (2)</td>
<td>insect (8)</td>
</tr>
<tr>
<td>moon (6)</td>
<td>man’s face (6)</td>
<td>small sketch (4)</td>
</tr>
<tr>
<td>pizza (1)</td>
<td>magistrate (1)</td>
<td>a star (6)</td>
</tr>
<tr>
<td>clock with no hands (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>omelette (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>snowball (10)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a dot (7)</td>
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<table>
<thead>
<tr>
<th>Object 4</th>
<th>Object 5</th>
<th>Object 6</th>
</tr>
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<tbody>
<tr>
<td>door lock/handle (4)</td>
<td>animal’s paw (11)</td>
<td>wood (4)</td>
</tr>
<tr>
<td>window (7)</td>
<td>glove (9)</td>
<td>blinds (8)</td>
</tr>
<tr>
<td>letter &amp; stamp (12)</td>
<td>foot (9)</td>
<td>spring (10)</td>
</tr>
<tr>
<td>envelope (11)</td>
<td>hand (9)</td>
<td>pile of plates (12)</td>
</tr>
<tr>
<td>postcard (12)</td>
<td>jumper (1)</td>
<td>pile of papers (12)</td>
</tr>
<tr>
<td>shed &amp; window (9)</td>
<td>shell (9)</td>
<td>paper folded up (9)</td>
</tr>
<tr>
<td>door (4)</td>
<td></td>
<td>heart (9)</td>
</tr>
<tr>
<td>oblong &amp; square (6)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure IV.1: Context scene A and its to-be-named objects
Figure IV.2. Context scene C and its to-be-named objects.
keyhole (1) monster's foot (10) pile of sandwiches (10)
bed (1) lampshade (5) pile of towels (12)
a shape (4) skirt (2) pile of linen/sheets (12)
flag (4) cardigan (1) records (7)
door of cat flap (3) a mark (2) cotton reel (8)
heater (3) footstool (4) part of a screw (7)
stamp (5) duck's foot (6) rubber tubing/tubing (8)
swimming pool (2) roll of cable (11) coils (10)
spy-hole (4) corrugated paper (12) bed quilt (2)
bed & pillow (3) whisky bottle top (7)

Object 7

blob (9) hut (9)
bun (10) gazebo (7)
pile of dough (11) curtains (9)
bread bap (10) column (7)
rock (11) tent (7)
stone (11) blinds (7)
beret/cap (8) side of dog kennel (4)
hat (8) coving (6)
island (5) kiosk (9)
plasticine (9) flag (5)
skull cap (7) end of a seat (7)
shoe (1) front of marquee (10)
sweet (4) side of windows (6)
car cover/tarpaulin (6) side of shed (9)
helmet (6) bus shelter (8)
pile of sand (5) shop front canopy (8)
plaster (4) piece of wood (2)
dome (4) football rattle (5)
upside down basin (3) bathing hut/chalet (11)
<table>
<thead>
<tr>
<th>Object 10</th>
<th>Object 11</th>
<th>Object 12</th>
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<td>slicer (5)</td>
<td>match (12)</td>
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<td>window (6)</td>
<td>paintbrush (7)</td>
<td>cigarette (7)</td>
</tr>
<tr>
<td>ticket (7)</td>
<td>can of oil (8)</td>
<td>thermometer (9)</td>
</tr>
<tr>
<td>rectangle &amp; dot (7)</td>
<td>football racket (8)</td>
<td>pencil (3)</td>
</tr>
<tr>
<td>door &amp; knob (3)</td>
<td>trowel (12)</td>
<td></td>
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<tr>
<td>envelope (4)</td>
<td>scoop (12)</td>
<td></td>
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<tr>
<td>notice on board (10)</td>
<td>bottle (4)</td>
<td></td>
</tr>
<tr>
<td>spray can (4)</td>
<td>shovel (11)</td>
<td></td>
</tr>
<tr>
<td>advent calendar door (9)</td>
<td>meat cleaver (8)</td>
<td></td>
</tr>
<tr>
<td>rectangle (3)</td>
<td>chopper (10)</td>
<td></td>
</tr>
<tr>
<td>label (8)</td>
<td>hack saw (7)</td>
<td></td>
</tr>
<tr>
<td>notepaper &amp; hole (8)</td>
<td>potato peeler (9)</td>
<td></td>
</tr>
<tr>
<td>front of cupboard (7)</td>
<td>coat hook (4)</td>
<td></td>
</tr>
<tr>
<td>wood nailed to wall (8)</td>
<td>light bulb (3)</td>
<td></td>
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<tr>
<td>small box (6)</td>
<td>lollipop (8)</td>
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</tr>
<tr>
<td>letter (5)</td>
<td>spade (8)</td>
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<td>lock (3)</td>
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<tr>
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<td>fire extinguisher (4)</td>
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</tr>
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<td>belt (0)</td>
<td>ashray (7)</td>
<td></td>
</tr>
<tr>
<td>dustbin (2)</td>
<td>corkscrew (3)</td>
<td></td>
</tr>
<tr>
<td>part of a key ring (8)</td>
<td>object with a bit sticking out (2)</td>
<td></td>
</tr>
<tr>
<td>part of a xylophone (9)</td>
<td>wire-connector (4)</td>
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<tr>
<td></td>
<td>wasp-swatter (7)</td>
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</tr>
<tr>
<td></td>
<td>key (2)</td>
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</tr>
<tr>
<td></td>
<td>part of a tap (2)</td>
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<tr>
<td></td>
<td>garden fork (7)</td>
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</table>

<table>
<thead>
<tr>
<th>Object 13</th>
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<th>Object 15</th>
</tr>
</thead>
<tbody>
<tr>
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<td>umbrella (11)</td>
<td>lampshade (12)</td>
</tr>
<tr>
<td>slipper (9)</td>
<td>parasol (12)</td>
<td>lightshade (12)</td>
</tr>
<tr>
<td>pompom on slipper (11)</td>
<td>beach umbrella (12)</td>
<td>cake &amp; candle (11)</td>
</tr>
</tbody>
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Figure IV.3. Context scene B and its to-be-named objects
Figure IV.4: Context scene D and its to-be-named objects
<table>
<thead>
<tr>
<th>Object 16</th>
<th>Object 17</th>
<th>Object 18</th>
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<tbody>
<tr>
<td>book (12)</td>
<td>coin/money (12)</td>
<td>pillow (12)</td>
</tr>
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<td>manhole cover (11)</td>
<td>cushion (12)</td>
</tr>
<tr>
<td>bit of wood (8)</td>
<td>tambourine (8)</td>
<td>bag of crisps (5)</td>
</tr>
<tr>
<td>lid (8)</td>
<td>biscuit (11)</td>
<td>Weetabix (4)</td>
</tr>
<tr>
<td>slab (11)</td>
<td>lid (11)</td>
<td>tubing (5)</td>
</tr>
<tr>
<td>sanding block (10)</td>
<td>cloth cap (5)</td>
<td>pillow case on line (11)</td>
</tr>
<tr>
<td>tombstone (8)</td>
<td>hamburger (5)</td>
<td>flannel (10)</td>
</tr>
<tr>
<td>bookend (7)</td>
<td>bottle top (6)</td>
<td>flag (6)</td>
</tr>
<tr>
<td>cushion (4)</td>
<td>flat bun (5)</td>
<td>newspaper (4)</td>
</tr>
<tr>
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<td>button (11)</td>
<td>curtains (9)</td>
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<td></td>
<td>shoe (0)</td>
<td>handkerchief (8)</td>
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<td></td>
<td>hub-cap (5)</td>
<td>sliced bread (7)</td>
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<tr>
<td></td>
<td>sandwich (4)</td>
<td>plates (2)</td>
</tr>
<tr>
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<td>plate (5)</td>
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<td>disc (6)</td>
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<td>slice of bread (4)</td>
<td>towel/tea towel (6)</td>
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<td>powder compact (7)</td>
<td>toast rack (5)</td>
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<td>pebble (5)</td>
<td>dress (1)</td>
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<td>ice hockey puck (7)</td>
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<td>dustbin lid (8)</td>
<td>lines (2)</td>
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<tr>
<td>Object 19</td>
<td>Object 20</td>
<td>Object 21</td>
</tr>
<tr>
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<td>------------------------------</td>
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<tr>
<td>basket (10)</td>
<td>jug (9)</td>
<td>lampshade (10)</td>
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<td>glass (6)</td>
<td>vase (11)</td>
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<td>skittle (8)</td>
<td>Terry’s chocolate orange (11)</td>
</tr>
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<td>bucket (12)</td>
<td>penguin (2)</td>
<td>tangerine (12)</td>
</tr>
<tr>
<td>face (1)</td>
<td>flame (8)</td>
<td>ball (7)</td>
</tr>
<tr>
<td>fairy cake (7)</td>
<td>fish (3)</td>
<td>pumpkin (12)</td>
</tr>
<tr>
<td>doll (1)</td>
<td>vegetable (2)</td>
<td>melon (10)</td>
</tr>
<tr>
<td>cup (5)</td>
<td>a seal (3)</td>
<td>gooseberry (9)</td>
</tr>
<tr>
<td>hamburger (1)</td>
<td>part of a leg (3)</td>
<td>bananas (9)</td>
</tr>
<tr>
<td>light bulb (3)</td>
<td>bottle (5)</td>
<td>orange (12)</td>
</tr>
<tr>
<td>microphone (6)</td>
<td>toilet chain handle (7)</td>
<td>beach ball (10)</td>
</tr>
<tr>
<td>lamp (4)</td>
<td>pigeon (1)</td>
<td>balloon (6)</td>
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<tr>
<td>watch (5)</td>
<td>lamp (5)</td>
<td>sea urchin (10)</td>
</tr>
<tr>
<td>back of chair (2)</td>
<td>condom (3)</td>
<td>fruit (7)</td>
</tr>
<tr>
<td>cigarette butt (5)</td>
<td>handle (5)</td>
<td>peach (4)</td>
</tr>
<tr>
<td>gear stick (3)</td>
<td>jar (3)</td>
<td>humbug (3)</td>
</tr>
<tr>
<td>thimble (4)</td>
<td>sock (2)</td>
<td></td>
</tr>
<tr>
<td>car (1)</td>
<td>bannister (1)</td>
<td></td>
</tr>
<tr>
<td>camel’s head (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>cork (4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>mask (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>finger (0)</td>
<td></td>
<td></td>
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<tr>
<td>chef (0)</td>
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<td></td>
</tr>
<tr>
<td>bottom of a table leg (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>screw (2)</td>
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<td></td>
</tr>
<tr>
<td>tip of a match (6)</td>
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<td></td>
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<tr>
<td>pepper pot (5)</td>
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<table>
<thead>
<tr>
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<th>Object 23</th>
<th>Object 24</th>
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<td>lock (7)</td>
<td>folder (9)</td>
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<td>button (6)</td>
<td>switch (11)</td>
<td>book (8)</td>
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<tr>
<td>sunhat (7)</td>
<td>mug (3)</td>
<td>sandwich (6)</td>
</tr>
<tr>
<td>porthole (9)</td>
<td>square &amp; circle (4)</td>
<td>van window (9)</td>
</tr>
<tr>
<td>ring (12)</td>
<td>button (8)</td>
<td>sweet (5)</td>
</tr>
<tr>
<td>Object 25</td>
<td>Object 26</td>
<td>Object 27</td>
</tr>
<tr>
<td>-------------------</td>
<td>----------------------------------------</td>
<td>----------------------------------</td>
</tr>
<tr>
<td>car (12)</td>
<td>mirror (11)</td>
<td>chair (12)</td>
</tr>
<tr>
<td>toy car (12)</td>
<td>windscreen (12)</td>
<td>book (0)</td>
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<tr>
<td></td>
<td>suitcase (6)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>television (10)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>toaster (10)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>rear-view/wing mirror (12)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>caravan window (11)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>window (11)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>tray (10)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>bread bin (7)</td>
<td></td>
</tr>
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<td></td>
<td>racing track (5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>bread tin (7)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>sardine tin (8)</td>
<td></td>
</tr>
</tbody>
</table>

plate (10) lightswitch (12) window pane (7)
door bell (8) lift button (12) tile (8)
washer (11) electrical socket doorway (7)
eye (5) screw in a bit of plastic (5) packet (10)
wheel (10) square knob (6) switch (4)
doughnut (10) a safe (6) a page (3)
egg (7) camera (6) newspaper (3)
leg of table (2) piece of Lego (2) lighter (7)
frisbee (8) drawer (6) television (2)
saucer (10) knob (7) book stand (4)
tyre (9) bell-push (12) box (6)
two plates (9) picture frame (4) toast (2)
moon (2) drawer handle (5) cup (0)
bullseye (7) keyhole (5) a square & a bit on side (4)
Polo mint (9) lighter (2) square jug (3)
circles (6) cake (2) flag (2)
ashtray (8) flag (1) catflap (7)
magnifying glass (6) eye (4) stove (2)
the sun (2) sweet (1) card (3)
top hat/hat (6) card (2) wood block (6)
doner kebab (2) fried egg (8)
snail’s shell (3) a spiral (0)
cup of tea (3)

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**Object 28: What is she doing?**

- pressing on something (7)
- baking (9)
- washing/cleaning something/clothes (10)
- making something (10)
- rolling pastry (11)
- looking through a book (1)
- wringing a cloth (7)
- making bread (12)
- tickling something (3)

- wrapping something (8)
- scrubbing (5)
- washing the floor (5)
- cleaning (9)
- polishing (7)
- washing up (5)
- folding something (8)
- fixing washing machine (2)
- making food (6)

---

For the objects *in context* in the picture scenes, all responses given by subjects are shown below, with the relevant ‘suitability index’ (the sum of judgments of 3 independent raters on a scale of 0 to 4) in parentheses.

<table>
<thead>
<tr>
<th>Object 1</th>
<th>Object 2</th>
<th>Object 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>ball (11)</td>
<td>face (9)</td>
<td>palm tree/tree (12)</td>
</tr>
<tr>
<td>circle (0)</td>
<td>woman’s/lady’s face (12)</td>
<td>windmill (0)</td>
</tr>
<tr>
<td>beach ball (12)</td>
<td>person (6)</td>
<td>plant (2)</td>
</tr>
<tr>
<td></td>
<td>housewife (11)</td>
<td>a flower on the horizon (4)</td>
</tr>
<tr>
<td></td>
<td>girl’s face (4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>head (4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>lady’s head (7)</td>
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<table>
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<tr>
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<th>Object 6</th>
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<tbody>
<tr>
<td>drawer (12)</td>
<td>shell (12)</td>
<td>plates piled up/plates (12)</td>
</tr>
<tr>
<td>cupboard (7)</td>
<td>footprint in the sand (5)</td>
<td></td>
</tr>
<tr>
<td>fridge (2)</td>
<td>flipper (9)</td>
<td></td>
</tr>
<tr>
<td>a square (0)</td>
<td>jellyfish (7)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>glove (4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>jumper (0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>baseball glove (4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>a foot (0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>football (0)</td>
<td></td>
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<td></td>
<td>starfish (3)</td>
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</tr>
<tr>
<td></td>
<td>cardigan (0)</td>
<td></td>
</tr>
<tr>
<td>Object 7</td>
<td>Object 8</td>
<td>Object 9</td>
</tr>
<tr>
<td>---------</td>
<td>----------</td>
<td>----------</td>
</tr>
<tr>
<td>hill (2)</td>
<td>hut (10)</td>
<td>card/postcard (11)</td>
</tr>
<tr>
<td>island (8)</td>
<td>blind (2)</td>
<td>letter (6)</td>
</tr>
<tr>
<td>rock (12)</td>
<td>cigarette &amp; matches (0)</td>
<td>box (3)</td>
</tr>
<tr>
<td>boat (0)</td>
<td>beach hut/chalet (12)</td>
<td>book (6)</td>
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<tr>
<td>sand crater (3)</td>
<td>side of a hut (11)</td>
<td>notepad (7)</td>
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<tr>
<td>boulder (12)</td>
<td>sunshade/shelter (12)</td>
<td>ice-cube tray (5)</td>
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<tr>
<td>humpback whale (0)</td>
<td>booth (11)</td>
<td>part of the oven (2)</td>
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<tr>
<td>lilo (0)</td>
<td>tent (7)</td>
<td>magazine (5)</td>
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<table>
<thead>
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<th>Object 12</th>
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<td>vent (3)</td>
<td>shovel (10)</td>
<td>table leg (12)</td>
</tr>
<tr>
<td>picture (3)</td>
<td>spade (12)</td>
<td>ironing board leg (8)</td>
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<tr>
<td>calendar (6)</td>
<td>kids play with it</td>
<td>match (0)</td>
</tr>
<tr>
<td>notepad (9)</td>
<td>in sand (7)</td>
<td>cigarette (0)</td>
</tr>
<tr>
<td>notice/paper pinned on wall (12)</td>
<td>digger (5)</td>
<td>bit of table (8)</td>
</tr>
<tr>
<td>something on wall (5)</td>
<td>sand shovel (11)</td>
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<td>message board (9)</td>
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<td></td>
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<td>chopping board (7)</td>
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<td>mirror (8)</td>
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<td>cupboard (1)</td>
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<td>lightswitch (1)</td>
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<tr>
<td>label (0)</td>
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</tr>
<tr>
<td>diary (0)</td>
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<tr>
<td>towel (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>curtain pattern (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>window (0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>painting (3)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Object 13: slipper (12)
you wear it when walking around at home (6)
shoe (4)
fluffy thing on shoe (5)

Object 14: umbrella (7)
parasol (12)
sunshade (11)

Object 15: lampshade (12)
light on ceiling (11)
chandelier (5)

Object 16: back of chair (12)
settee material (4)

Object 17: makeup compact (12)
mirror (11)
plate (1)
coaster (2)
soap (7)
coin (0)
bread (0)

Object 18: cushion (12)
pillow (7)
pillowcase (3)

Object 19: bucket (12)
basket (5)
pot (3)
bag (5)

Object 20: vase (12)
jar for flowers (10)
flower pot (5)

Object 21: ball (11)
girl’s body (5)
girl’s jumper/blouse (5)
pumpkin (1)
beach ball (12)
parcel (2)
anorak (4)
melon (0)
a fruit (0)
banana ball for seaside (10)

Object 22: plate (12)
trophy (9)
ornament (8)

Object 23: lock (11)
clip/catch on case (12)
handle (2)

Object 24: book (12)
bookend (10)
end of bookcase (2)

Object 25: car (12)

Object 26: suitcase lid (26)

Object 27: chair (12)

Object 28: What is she doing?
packing suitcase (11)

rolling up clothes at side of
getting passport out of her pocket (2)
wrapping up gear (8)
unpacking the case (2)
washing garment with bar of soap (0)
scrubbing (0)

the case (11)
cleaning the case (4)
putting a towel over the book (1)
folding something to put in the case (11)
Appendix V: Patient information sheet and consent form for the studies in Chapters 5 and 7

PATIENT INFORMATION SHEET

We invite you to participate in a research project which we believe to be of potential importance. In order to help you understand what the research is about, we are providing you with the following information which we want to be sure you understand before you formally agree to participate. Be sure to ask any questions you have about the information which follows, and we will do our best to explain and to provide any further information you require.

The present research is investigating how you view certain pictures and patterns. We are also interested in how you understand what other people think in certain social situations. We are interviewing you and the other patients on this ward, as we believe your answers may help us understand why you sometimes feel unwell.

The interview will begin with some questions about how you have been feeling recently, and we shall then ask you to do a few simple tests of reading and comprehension. You will then look at some pictures, read some stories, and be asked a few questions about the pictures and stories. It is possible that anyone might find some of these tasks frustrating or boring. If you do, please tell us, and we will stop the task immediately.

The study is solely for the purposes of research, and will not affect your own treatment at all. There are no potential hazards involved in the research, and you will not be required to give up any of your other ward activities in order to take part in the study.
All answers that you give to Graham Pickup (the researcher) will be in confidence. The only other people who will have access to your responses are your Consultant and the project supervisor (Professor C. Frith). If you require any further information, Mr. Pickup or your Consultant will do their best to provide it.

You are free not to participate, and may withdraw from the study at any time. This will not jeopardise the ordinary course of your medical treatment.

General information on patients’ rights, particularly regarding participation in research projects, may also be obtained from your local Community Health Council.
PATIENT CONSENT FORM

Title of research proposal: An Investigation of ‘theory of mind’.

Ethics Committee No.

Name of subject:  
Address:

I have read the attached information on the research in which I have been asked to participate, and have been given a copy to keep. I have had the opportunity to discuss the details and ask questions about this information.

The Investigator has explained the nature and purpose of the research, and I believe that I understand what is being proposed. For example, I understand that this trial is part of a research project designed to promote medical knowledge, and that it has been approved by the local Research Ethics Committee.

I understand that my personal involvement and my particular data from this trial will remain strictly confidential. Only researchers involved in the trial will have access.

I hereby fully and freely consent to participate in the study, which has been fully explained to me.

SUBJECT’S NAME: ............................................................
SUBJECT’S SIGNATURE ................................................
SUBJECT’S WITNESS’ NAME ........................................
WITNESS’ SIGNATURE ..............................................
INVESTIGATOR’S NAME ............................................
INVESTIGATOR’S SIGNATURE ....................................
DATE ...............................................................................

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The following should be signed by the clinician responsible for obtaining consent. As the clinician responsible for this research, I confirm that I have explained to the patient named above the nature and purpose of the research to be undertaken.

CLINICIAN’S NAME ........................................................
CLINICIAN’S SIGNATURE .............................................
DATE .......................................................................................

Declaration by the Consultant or Principal Investigator in charge of the research:

I fully accept responsibility:

1. To inform all medical and nursing staff (including GP with the subject’s consent) at each location where a patient/volunteer may be treated, that a subject is enrolled in a trial or experiment, what drugs (if any) or invasive procedures will be used (or not as may be), and what precautions they should take, if any. In some cases it will be necessary to give special training to nurses or junior staff to prepare them to undertake procedures.

2. To ensure that the details of each procedure to be done or drug to be given are entered in the clinical notes and that the date and time when the procedure was done and/or drug given are subsequently noted.

3. To make three copies of the patient information sheet and the signed consent form. One copy of each should be kept by the patient/volunteer, one copy should be included in the patient’s clinical notes, and one copy should be kept by the Consultant/Chief Investigator responsible for the research.

4. To ensure that each subject is verbally warned not to take part in more than one
study at any time.

5. To inform the Committee of any adverse or unforeseen circumstances arising out of this research.

6. For clinical research, to provide the Committee with one brief report of progress half way through the project, and another at its completion.

PRINCIPAL INVESTIGATOR ..............................................

SIGNATURE ......................................................
Appendix VI: Tables for the parametric ANOVA analyses in Chapter 5.

Demographic details

1. Comparison of current verbal IQ across the 5 groups

<table>
<thead>
<tr>
<th>Source of variance</th>
<th>df</th>
<th>Mean squares</th>
<th>F ratio</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Groups</td>
<td>4</td>
<td>535.75</td>
<td>4.29</td>
<td>&lt; 0.004</td>
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<tr>
<td>Within Groups</td>
<td>71</td>
<td>124.93</td>
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<tr>
<td>Total</td>
<td>75</td>
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</table>

Picture pieces task

1. Mixed model investigation of effects of group and within-subjects effects of contextual information ('cxt') in conditions A & C (data are log transformed)

<table>
<thead>
<tr>
<th>Source of variance</th>
<th>df</th>
<th>Mean squares</th>
<th>F ratio</th>
<th>p</th>
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<tr>
<td>BETWEEN</td>
<td>75</td>
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</tr>
<tr>
<td>Treatment</td>
<td>4</td>
<td>14.02 x 10^{-3}</td>
<td>5.52</td>
<td>0.001</td>
</tr>
<tr>
<td>Error</td>
<td>71</td>
<td>2.54 x 10^{-3}</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WITHIN</td>
<td>76</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cxt</td>
<td>1</td>
<td>0.052</td>
<td>22.90</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Group x Cxt</td>
<td>4</td>
<td>2.18 x 10^{-3}</td>
<td>0.97</td>
<td>0.429</td>
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<tr>
<td>Error</td>
<td>71</td>
<td>2.25 x 10^{-3}</td>
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<tr>
<td>TOTAL</td>
<td>151</td>
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</table>
2. **Comparison of isolation suitability responses for conditions A & C across groups** *(data are log transformed)*

<table>
<thead>
<tr>
<th>Source of variance</th>
<th>df</th>
<th>Mean squares</th>
<th>F ratio</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Groups</td>
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<td>0.0071</td>
<td>3.61</td>
<td>0.01</td>
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<tr>
<td>Within Groups</td>
<td>71</td>
<td>0.0020</td>
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<tr>
<td>Total</td>
<td>75</td>
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</table>

3. **Comparison of context suitability responses for conditions A & C across groups** *(data are log transformed)*

<table>
<thead>
<tr>
<th>Source of variance</th>
<th>df</th>
<th>Mean squares</th>
<th>F ratio</th>
<th>p</th>
</tr>
</thead>
<tbody>
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<td>3.23</td>
<td>0.02</td>
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<tr>
<td>Within Groups</td>
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<td>0.0028</td>
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<tr>
<td>Total</td>
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4. **Mixed model investigation of effects of group and within-subjects effects of contextual information ("cxt") in conditions B & D** *(data are log transformed)*

<table>
<thead>
<tr>
<th>Source of variance</th>
<th>df</th>
<th>Mean squares</th>
<th>F ratio</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>BETWEEN Treatment</td>
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<td>0.02</td>
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<td>Treatment Error</td>
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<td>1.49 x 10^{-3}</td>
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</tr>
<tr>
<td>WITHIN Cxt</td>
<td>1</td>
<td>0.207</td>
<td>198.27</td>
<td>&lt; 0.001</td>
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<tr>
<td>Group x Cxt</td>
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<tr>
<td>Error</td>
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<td>1.04 x 10^{-3}</td>
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<tr>
<td>TOTAL</td>
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</tbody>
</table>
5. Comparison of isolation suitability responses for conditions B & D across groups (data are log transformed)

<table>
<thead>
<tr>
<th>Source of variance</th>
<th>df</th>
<th>Mean squares</th>
<th>F ratio</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
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6. Comparison of context suitability responses for conditions B & D across groups (data are log transformed)

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7. Mixed model investigation of effects of group and within-subjects effects of contextual information ('cxt') for the whole task (data are log transformed)

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8. *Comparison of isolation suitability responses across groups for the whole task (data are log transformed)*

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9. *Comparison of context suitability responses across groups for the whole task (data are log transformed)*

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10. *Mixed model investigation of effects of group and within-subjects effects of contextual information ('cxt') on the percentage of highly unsuitable responses over the whole task (data are arcsine transformed)*

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11. *Comparison of percentage of highly unsuitable responses across groups for the isolation conditions over the whole task (data are arcsine transformed)*

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12. *Comparison of percentage of highly unsuitable responses across groups for the context conditions over the whole task (data are arcsine transformed)*

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13. *Mixed model investigation of effects of group and within-subjects effects of contextual information ('cxt') on the percentage of repetitions over the whole task (data are arcsine transformed)*

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<td>Cxt</td>
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<tr>
<td>TOTAL</td>
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Appendix VII: Experimental and practice stimuli for the embedded figures test in Chapter 7

Copies of the 8 experimental stimuli (full size A4) and 3 practice stimuli (smaller size) are provided in the pocket on the inside back cover of the thesis. These copies are the same size and colour as the actual stimuli used in the task. They are numbered in order of increasing difficulty, so the numbering order is the same as the order of presentation in the task. Line drawings of the hidden objects are shown below, in actual size. During the task, these were given to subjects as cardboard cutouts to aid searching.

Hidden objects for experimental stimuli

1: Building
Mug

2: Digger
Kite

3: Ship
Ice-cream cone

4: Peacock
Bell

5: Water jug
House

6: Shoes
Crown
Hidden objects for the practice stimuli

(N.B. The practice pictures were modified from Witkin et al.’s (1971) Children’s Embedded Figures Test (CEFT), but the hidden objects were not the same as those used in the CEFT).
Appendix VIII: Experimental and practice stimuli for the Titchener illusion task in Chapter 7

The experimental stimuli are presented on the following pages in the same size and order as used in the task. An illusion presentation is always followed by a control presentation with the same size circles as in the illusion.
The four practice items for the Titchener illusion task are given on the following pages. There are 2 illusion stimuli and 2 non-illusion stimuli, to demonstrate to subjects the various types of presentation that they will receive during the task.
Appendix IX: Tables for the parametric ANOVA analyses in Chapter 7

Demographic details

1. *Comparison of current spatial IQ across the 5 groups*

<table>
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Embedded figures task

1. *Comparison of accuracy on the task across the 5 groups*

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2. *Task accuracy across groups with spatial IQ co-varied*

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3. **Task accuracy across groups with attention co-varied**

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4. **Task accuracy across groups with spatial IQ and attention co-varied**

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5. **Comparison of accuracy across groups with only males included**

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### 6. Task accuracy across groups of males with spatial IQ co-varied

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### 7. Task accuracy across groups of males with attention co-varied

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### 8. Task accuracy across groups of males with spatial IQ and attention co-varied

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PRACTICE PICTURE 1
PRACTICE PICTURE 2
PRACTICE PICTURE 3.